2012, Vol. 83, No. 3

ATLANTA Medicine

JOURNAL OF THE MEDICAL ASSOCIATION OF ATLANTA

Rheumatology

Are You Concerned About...?

- Sluggish Economy
- Increasingly Complex Healthcare Environment
- Unstable Reimbursement Climate
- Rise in Practice Costs
- Administrative Burden
- Healthcare Reform Uncertainty

Piedmont Healthcare Can Help

We offer solutions to these and many other issues facing physicians today. In fact, Piedmont Physicians Group has been identified as a *Better Performer* by the Medical Group Management Association (MGMA) for the past nine years.

Driven to Deliver Exceptional Quality

Our focus on continuous quality improvement – coupled with an exceptional professional staff, state-of-the-art technology and the knowledge that compassion and caring are integral to the practice of medicine – reflects our commitment to exceed expectations.

To learn more about ways to affiliate with Piedmont Healthcare, contact:

Cindy Baeder
Director, Physician Recruitment
2727 Paces Ferry Road • Suite 1-1100
Atlanta, Georgia 30339
770.801.2703
cindy.baeder@piedmont.org

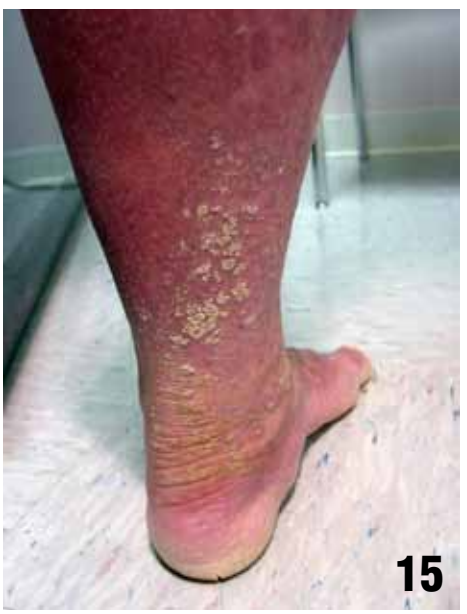
PIEDMONT HEALTHCARE

piedmont.org

contents

Vol. 83, No. 3, 2012

PHOTO © DEBRA JANSEN, DEBJANSENPHOTOGRAPHY.COM



Atlanta Medicine Editorial Board

MEDICAL ASSOCIATION OF ATLANTA Est. 1854

For information about ATLANTA Medicine magazine, contact the Medical Association of Atlanta.

1150 Lake Hearn Drive, STE 130, Atlanta, GA 30342, Phone: 404-881-1020
FAX: 404-872-0601 or email: info@maa-assn.org

Rob Schreiner, M.D., FACP, FCCP

MAA President's Message

I"m delighted to have the privilege of leading our Association for the coming year. Dr. Hilton, and all former MAA presidents, created a stronger, more relevant association during their tenure. I intend to continue that momentum.

As I've listened to many of you during the last three years, I've heard you say three things should be the strategic focus of our Association, particularly during this time of unprecedented change:

1. Create influence in business, regulatory and social circles, for the purpose of protecting and promoting the interests of patients and physicians.
2. Create a unified (or at least coordinated) voice of physicians in Atlanta, in order to be taken seriously by regulators and policy decision-makers.
3. Maintain or increase the current level of membership (>1,300 members), for the purpose of maintaining leadership in MAG and relevance in the Atlanta medical community.

As such, all activities we engage in this year will in some way serve one or more of those strategic goals. I'll communicate specifics following the upcoming Annual Board Strategic Retreat in August.

In the meantime …

In 2010, the Medical Group Management Association (MGMA) reported that, for the first time in American history, the percentage of employed physicians in the U.S. exceeded the percentage of physicians who owned their practice. Across Atlanta, physicians are joining larger healthcare organizations as a way to "get back to the bedside," as well as participate in more coordinated care. The MAA provides unbiased information and collegial advice for physicians who are interested in exploring career options beyond the traditional solo- or small-practice model. Our career center and CME symposiums offer connections to a broad spectrum of practice opportunities, including hospital-owned practices, integrated delivery systems, single and multi-specialty group practices, nonprofit clinics, military practice – even cruise ship medicine.

MAA is also an excellent organization for physicians in leadership roles. I firmly believe physicians are best suited to lead health care organizations, as we possess unique subject-matter expertise and credibility with patients, staff and other physicians during the inevitable change management process. We understand firsthand the complexity of healthcare delivery at the bedside and in the exam room. That awareness is crucial to crafting and executing strategies that transform the quality, service and affordability of health care, especially given the substantial changes that are coming as part of the Affordable Care Act (ACA). MAA provides opportunities to further develop your leadership skills as an officer or board member, while you build relationships with fellow physician leaders.

Finally, MAA gives individual physicians a voice in our medical and non-medical communities by promoting and protecting the practice of medicine. No one is better suited to inform legislators, government leaders and the media than physician-leaders who understand and organize the care. Our local and national elected officials will make better policy and legislative decisions if they deeply understand all aspects of high-quality care and what's required to create it. MAA provides a channel to help achieve that understanding and frame the healthcare debate in the context of patient interests and values.

If you are already a member of MAA, we thank you. If you are not, we welcome your active participation in Atlanta's vibrant medical community. ■

Rob Schreiner, M.D., is the executive medical director for The Southeast Permanente Medical Group (TSPMG) and 2012-2013 Board President of the Medical Association of Atlanta. Dr. Schreiner joined TSPMG as a pulmonary and critical care medicine physician in 1994.

Concord
A COMMUNITY PHARMACY

30 YEARS OF HISTORY SERVING ATLANTA'S MEDICAL COMMUNITY

At Concord Pharmacy, your patients' well-being is our top priority. We work closely with physicians to optimize your patients' personal health strategy.

- HARD TO FIND/SPECIALTY PHARMACEUTICALS -
- SPECIALTY COMPOUNDING - PAIN MANAGEMENT - FERTILITY -

LAKE HEARN PAVILION
404-781-2229

EMORY MIDTOWN HOSPITAL
404-523-4908

CARLTON'S
770-394-3233

AUSTELL
770-819-1584

NORTHSIDE DOCTORS CENTER
404-252-2280

NORTH FULTON HOSPITAL
770-664-5428

PEACHTREE DUNWOODY MEDICAL CENTER
404-250-9656

LAWRENCEVILLE/TERRACE PARK
678-990-8920

MEDICAL QUARTERS
404-252-3607

ELLIJAY RIVERSIDE DRUGS
706-635-5151

JOHN'S CREEK
678-417-0122

LAWRENCEVILLE
770-338-0971

www.concordrx.com

MAA Elects New Officers for 2012–2013

The Medical Association of Atlanta (MAA) installed its new officers on June 26 at its annual meeting held at The Biltmore Ballroom in Atlanta.

Rob Schreiner, M.D., FACP, FCCP, was elected president of the MAA for a one-year term. He has served as Executive Medical Director for The Southeast Permanente Medical Group, Inc. (TSPMG) in Atlanta since 2008. In this role, he leads one of Atlanta's largest multi-specialty medical groups, with overall executive responsibility for the cost and quality of healthcare provided to more than 240,000 Kaiser Permanente members in 28 medical facilities and four hospitals in a 28-county metropolitan service area, and more than 740 TSPMG physicians, associate practitioners and professional staff.

"I am honored to participate in the leadership of this important organization at a time when there is so much interest in the delivery of health care," says Dr. Schreiner. "Effective physician leadership is particularly necessary during this time of rapid change in our field. The interests and needs of patients must be the center of civil debate that includes the experienced, balanced, altruistic voice of the physician."

Dr. Schreiner also serves on the board of directors of Trees Atlanta, a non-profit organization that has enhanced and preserved Atlanta's urban forest since 1985. He is a member of the Metro Atlanta Chamber (MAC) Board of Advisors, and also serves on the Environmental Policy and Sustainability Committee. Dr. Schreiner is a Director of the Buckhead Coalition, a longstanding non-profit organization dedicated to improving life in the Buckhead Community of Atlanta. For more than 10 years, Dr. Schreiner served in several physician-leadership roles on the Medical Executive Committee (MEC) of Northside Hospital Atlanta. He is also a longtime volunteer at Our Lady of Perpetual Help Home (OLPHH), an Atlanta-area hospice that provides no-cost care for patients with terminal cancer.

Other officers include:

Chairman of the Board – **Dr. Michael Hilton.** Dr. Michael Hilton has been in the private practice of General and Forensic Psychiatry for 22 years. He is currently on the Board of Directors for the Medical Association of Georgia and is the outgoing President of the Medical Association of Atlanta.

President-elect – **Dr. Lisa Perry-Gilkes.** Dr. Perry-Gilkes has been a member of the MAA board for more than five years. She is also a member of the board of governors for the American Academy of Otolaryngology Head & Neck Surgery. She is president of the Georgia Physicians Association. Dr. Perry-Gilkes has been in solo private practice for more than five years and currently practices in Atlanta in the Camp Creek Market Place near the airport.

Treasurer – **Dr. W. Hayes Wilson.** Dr. Wilson is the President of Piedmont Rheumatology Consultants. He is chief of the Division of Rheumatology at Piedmont Hospital, chairperson of the Medical and Scientific Committee of the Arthritis Foundation, , and serves on the Board of Directors of the Arthritis Foundation National, medical advisory board for the Lupus Foundation of America, Georgia Chapter. Dr. Wilson is also a Fellow of the American College of Rheumatology and serves as Vice Chairman of the board of directors for the BreakThru House Ministry.

Secretary – **Dr. Quentin Pirkle.** Quentin R. Pirkle Jr., M.D., serves as Vice President and Chief Medical Officer of Piedmont Medical Care Corporation and is Chairman of the board of directors of Piedmont Clinic. Dr. Pirkle is a member of the American College of Physician Executives, the Leader's Board for Group Practice Executives and the Medical Association of Georgia. He also serves on the board

of directors of the Emory University School of Medicine Alumni Board and Developmental Disabilities Ministry, Inc.

"Atlanta is fortunate to have these physician leaders serve and continue the tradition of organized medicine that began in 1854," says David Waldrep, executive director/CEO for the Medical Association of Atlanta.

The MAA is a non-profit association dedicated to the advancement of organized medicine in the Atlanta area. "Representing over 1,400 physician members, the medical association promotes the health and safety of our community through physician leadership and serves its members with integrity by proactive leadership to influence the profession of medicine," says Waldrep. "The Medical Association of Atlanta is the largest component society of the Medical Association of Georgia and represents Atlanta physicians establishing the policies of the Medical Association of Georgia."

According to Waldrep, MAA members have united their individual voices and efforts into one cohesive body of progress, representing the new face of organized medicine in areas of legislation, community service, and individual patient care. In an era when outside influences are disrupting and dividing the practice of medicine more than ever before, MAA members stand as a united front, working to affect needed changes in today's healthcare systems, while remaining steadfast in support of the historic image of the skilled, caring, hands-on physician. ■

For more information on the Medical Association of Atlanta, please visit www.maa-assn.org or call 404.881.1020.

Harvey Awarded MAA's Aven Cup

The Medical Association of Atlanta (MAA) gave its highest award, the Aven Cup, to Dr. John S. Harvey at its annual meeting.

Dr. Harvey was awarded the Aven Cup for his service to the Atlanta community and the Medical Association of Atlanta. Dr. Harvey serves the community as a volunteer in the Georgia State Defense Force; he serves as the Command Surgeon. Notable missions include the Katrina/Rita hurricane victim airlift reception and the Haiti earthquake victim airlift reception in Atlanta. During the 1996 Olympics, he served as a Medical Command Officer dealing directly with the medical and multi-agency response to the Centennial Olympic Park bombing.

Dr. Harvey serves on the board of directors for the Medical Association of Atlanta and the Medical Association of Georgia, where he serves as Speaker of the House of Delegates.

The MAA has a tradition of its physician leaders serving the community. "Dr. Harvey is one of many

leaders that continue the tradition serving our community that began in 1854," says David Waldrep executive director/CEO. ■

W. Hayes Wilson, M.D., F.A.C.R.

Rheumatology, Mystery that Affects Everyone

This Edition of Atlanta Medicine is devoted to Rheumatology. Rheumatology is one of the smallest of subspecialties in Internal Medicine and Pediatrics despite the fact that Arthritis is the number one cause of disability in the United States of America. It has been estimated that the cost to the America is 128 billion dollars a year and one in five Americans has a chronic joint symptom.

The Arthritis Foundation estimates that 27 million Americans have Osteoarthrits, the most common type of the over 100 different forms of Arthritis and Arthritis related diseases. With such a pervasive problem it would seem that the practice of Rheumatology would be served by more than 0.3 percent of all specialists.

Rheumatology is a field that has more clues than diagnostic tests and there are more diagnoses than therapies. One of the confounding issues is that all Rheumatologic maladies have common systemic symptoms which can be overlapping and the diagnosis in an individual patient may evolve over time as new symptoms appear. There are also legitimate overlap syndromes that are not uncommon, such as scleroderma and polymyositis.

It is not the intention to uncover all the mysteries of Rheumatology in this edition of Atlanta Medicine but rather to touch on some of the more important issues that are relevant to any practicing physician. For instance, there are many nuances of laboratory testing which raise more questions than providing real definitive answers. Some of the more common testing will be discussed in this issue, with a little political commentary on the reality that laboratory testing only opens the door and that it takes a thorough history and physical combined with a generous quotient of patience to arrive at the correct diagnosis and treatment plan.

Likewise, our diseases have systemic manifestations that are not only important clues for a diagnosis but cross specialties requiring collaboration with all specialties. There are far reaching consequences to systemic inflammation. The most obvious is joint deformity and destruction, but the deadlier consequence can be associated cardiovascular disease.

There are common challenges that arise in the everyday practice of medicine, for instance gouty arthritis, that was described in ancient times and we still use an ancient medication to treat it. Unfortunately there are pitfalls in the treatment of gout that can easily complicate therapy.

Included inside this issue are guidelines and cautions that are pertinent to any physician that treats gout, one of the most inflammatory and thus painful types of arthritis. When my father started practicing Rheumatology in the 1960's there was little to do for patients beyond aspirin, steroids and gold salts. In the late 1990's there was a Renaissance in Rheumatology with the development of Biologic Response Modifiers.

Rheumatology has always been an intellectually stimulating subspecialty for all the reasons mentioned above, but with the advent of Biologic Response Modifiers it became very rewarding to treat previously unrelenting destructive and disabling diseases which often can be managed even to the extent that a patient can continue to play professional golf. With significant modulation of the immune system, there are potential consequences as well. Live vaccines are potentially a threat to our patients. Since they are often given by their family physician, guidelines for vaccination are offered.

If I could offer one message only to our reader it would be this, arthritis is unacceptable. There is no reason why a physician or a patient would decide to just live with arthritis rather than pursuing proper diagnosis and treatment. Arthritis is a life altering disease, slow, progressive and unrelenting. There are many therapies that are available today so to ignore arthritis would be tantamount to knowing that you had heart disease or cancer and you were just going to live with it. No one in their right mind would ignore serious life threatening disease and therefore arthritis and the related illnesses should be properly diagnosed and treated.

Take Control, We Can Help. Arthritis is Unacceptable. ■

Dr. Wilson is the President of Piedmont Rheumatology Consultants. He is chief of the Division of Rheumatology at Piedmont Hospital, chairperson of the Medical and Scientific Committee of the Arthritis Foundation, , and serves on the Board of Directors of the Arthritis Foundation National, medical advisory board for the Lupus Foundation of America, Georgia Chapter. Dr. Wilson is also a Fellow of the American College of Rheumatology and serves as Vice Chairman of the board of directors for the BreakThru House Ministry.

Laboratory Testing in Rheumatology

John A. Goldman, M.D.
MACR, FACP, CCD

Many times as rheumatologists, we are asked about lab testing in patients with rheumatic problems.

This article will mix some of the political, operational and mechanical issues within this topic.

Most important above the entire lab testing is the history and physical examination. I cannot emphasize this enough. The labs tests are a guide that helps define our patients, but the history and physical are the main guides to proper management.

The Centers for Medicare & Medicaid Services (CMS) took away the consultation code for specialists in January 2010. Nevertheless, it is careful examination and clinical evaluation that saves more in costs.

Enough of my editorial comments, let's discuss the lab tests available and how they may help confirm a diagnosis.

PHOTO CREDIT: DEBRA JANSEN, DEBJANSENPHOTOGRAPHY.COM

THERE ARE FIVE MAIN TESTS USED IN THE RHEUMATOLOGY FIELD TO HELP MAKE A DIAGNOSIS:

1. Westergren Sedimentation Test: This simple test for inflammation, if done correctly and fresh and NOT sent to a central lab like QUEST, Solstas, or LabCorp, can give us a lot of information about the status of the patients and their rheumatic complaints. It is not specific for a diagnosis - in fact, it was originally developed in the 1930s as a pregnancy test because it would rise during pregnancy and then fall after delivery. Even then, in the 1930s, they knew that any delay in performing the test would lessen numerical final result. In my office, we run fresh sed rates despite the fact that some of the insurance companies would not pay for the correct test. I am surprised the central labs have not made that clearer on their lab reports.

2. C Reactive Protein (CRP): This, too, is a simple test for inflammation. It is reported either as mg/dL or for high sensitivity mg/L. Therefore, it is about a 1:10 difference. We look at this test to judge inflammation. Of interest in Rheumatoid Arthritis (RA), there is about a 30+ percent discordance in that some people will have the sedimentation rate elevated or the CRP elevated, and about two-thirds will have both elevated. They measure different things. The high sensitivity is used by some to look at cardiovascular risks. In our Rheumatoid Arthritis (RA) patients, it is already high, consistent with the increased cardiovascular disease associated with inflammatory disease. Patients with RA have a higher cardiovascular risk, and people with RA die up to 10 years earlier with cardiovascular disease compared to the general population. Of note: some of our medications including methotrexate and the biological Tumor Necrosis Factor inhibitors are making a dent in this risk and improving cardiovascular risk in RA.

3. Rheumatoid Factor (RF): This is found in about 80 percent of people with Rheumatoid Arthritis (RA). It is not specific for RA and is the body's response to a chronic inflammatory condition. The most common cause of elevation of the RF in the world is malaria. We use it in rheumatology to help validate the diagnosis of RA using criteria for classification of RA by the American College of Rheumatology Criteria. It can be elevated in chronic infections such as endocarditis, and as the endocarditis is cured with antibiotics the elevated RF can normalize.

4. Cyclic Citrullinated Protein (CCP): This test is used now to help us diagnose Rheumatoid Arthritis, and also to judge the severity of RA. Those with the genetic risk of RA have potentially a higher risk of developing RA if they have smoked and carry either a single or double HLA-DR shared epitope(SE) gene and are anti-CCP positive. If you have smoked, you are at greater risk of having more severe RA, and it may not respond as well to therapy. Unfortunately, stopping smoking does not seem to help. The presence of both a positive RF and a positive CCP indicates a risk for more severe RA no matter else. Rheumatologists often order both the RF and CCP in combination for diagnosis and prognosis.

5. Antinuclear Antibody (ANA): This is a test indicating the presence in the body of an antibody against the nucleus of the cell. Everyone has a positive ANA, but people with lupus and other members of the connective tissue disease family have a higher positive ANA. This test, when positive, is one of the criteria for the diagnosis of Systemic Lupus Erythematosus. Its presence is not specific for lupus. In fact, a positive ANA can be seen in people who have no demonstrable disease. A positive ANA can also be seen in allergic reactions, other connective tissue disease and certain types of autoimmune hepatitis. A Rheumatologists will help guide the patient to understanding why the test is positive and whether it suggests lupus or not.

PHOTO CREDIT: DEBRA JANSEN, DEBJANSENPHOTOGRAPHY.COM

There are pitfalls in interpreting the ANA test. A few years ago the central labs like Quest and LabCorp and others like the Mayo Clinic changed the technique for identifying the ANA using an enzyme linked test (ELISA test) rather than the classic immunofluorescence (IFA) test. This was changed because they could automate their testing process. Central labs have developed the multiplex automated screens to process the large volume of clinical specimens. Tests using ELISAs and coated beads are not accurate ANA screening tests. The classic ANA immunofluorescent Assay(IFA) test is the gold standard for testing for the ANA, and it uses a biological system, usually mammalian cells, including a kidney cell, liver cell or now the Hep 2 cancer cell (with its large nucleus). HEP 2 cells have hundreds of antigens (proteins and nucleic acids) that are distributed in orderly domains in a tissue culture cell and thus can tests for hundreds of different types of these antinuclear antigens. The ELISA test only identifies about 12 or so antigens, and thus many people with lupus were not readily identified.

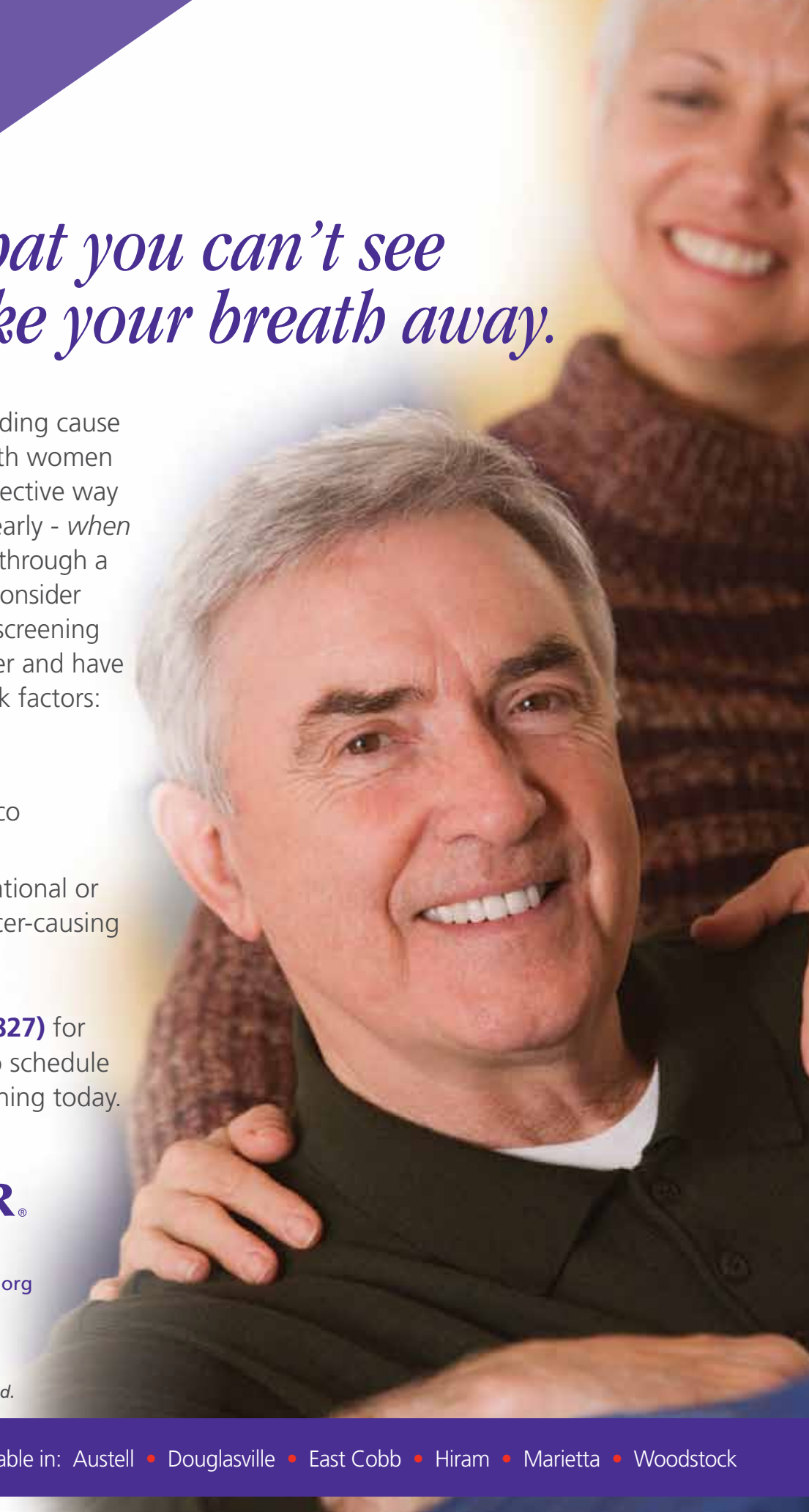
Multiplex ANAs have a good ability to detect specific antigens. The automated tests may have a dozen or so nuclear antigens coated on the walls in plastic plates or on beads. These tests are good and specific for the antigens tested but not for screening. Through the American College of Rheumatology ANA task force, this was reviewed and eventually a position paper was published. This went to the College of American Pathologists (CAP) (who certify the labs) and to the labs themselves and encouraged them to do the right thing. Quest now has the ANA by immunofluorescence as a standard test, and it's easily found on its website. However, LabCorp still uses the ELISA test to screen for ANA, and it's not readily apparent on its website that it has the IFA. The correct ANA test code to screen for ANA by immunofluorescence at QUEST is 249, LABCORP is 164947 and FANA for Solstas Lab Partners.

These are just a few of the tests in our field. After the appropriate evaluation, these tests are used to help guide the correct diagnosis and treatment in our patients. It is important to emphasize, despite the regulations of CMS to the contrary, that the most cost-effective diagnosis is the correct diagnosis. As rheumatologists we do this. ■

John A. Goldman, M.D. is chief of rheumatology at St. Joseph's Hospital, active staff at Northside Hospital and president of The Medical Quarters, P.C. He is rheumatology medical director of the Atlanta Center for Clinical Research. Dr. Goldman is a former clinical professor of medicine at Emory University School of Medicine. Current and former chairman of the Southeast Committee on Rheumatologic Care Network, Southeast Regional Advisory Council and the ACR Committee on Rheumatologic Care.

Lung Cancer Screenings
$199 per individual
or **$299 per couple.***

What you can't see can take your breath away.



Lung cancer is the leading cause of cancer death for both women and men. The most effective way to detect lung cancer early - *when it is most treatable* - is through a screening. You should consider WellStar's lung cancer screening if you are age 50 or over and have any of the following risk factors:

- History of smoking
- Secondhand tobacco smoke exposure
- Exposure to occupational or environmental cancer-causing substances

Call **770-956-STAR (7827)** for more information or to schedule your lung cancer screening today.

WELLSTAR®

770-956-STAR ★ wellstar.org

**A physician referral is not required.*

Lung Cancer Screenings available in: Austell • Douglasville • East Cobb • Hiram • Marietta • Woodstock

Kelly O. Weselman, M.D., F.A.C.R.

Overview of the Systemic Manifestations of Rheumatic Diseases

Although many think of rheumatic diseases as causing joint pain and swelling, it is important to remember that these illnesses cause pathology in multiple other organ systems. Since these patients frequently present to primary care physicians and virtually all specialists, it is important to be familiar with the myriad manifestations of these diseases.

In patients with a known rheumatic disease and new symptoms, consider the disease or its medications as a potential cause. In patients without a known rheumatic disease, keep these illnesses in your differential. In working with your rheumatology colleagues, remember to look for objective evidence of organ involvement by carefully interpreting the history, exam, labs and imaging data. Let's consider the effects of some of the common rheumatic diseases on each organ system.

DERMATOLOGIC FINDINGS

Skin rashes and lesions are seen commonly in rheumatic diseases such as lupus, scleroderma, dermatomyositis and some types of vasculitis. Rashes can be specific to a disease or represent nonspecific pathology.

Eighty-five percent of lupus patients will have skin manifestations. These can include photosensitivity rashes, discoid lesions, oral and nasal ulcers, malar rashes as well as subacute cutaneous lupus. Remember that the malar rash of lupus spares the nasolabial folds and crosses the bridge of the nose, which distinguishes it from the more common rosacea and seborrheic dermatitis.

Dermatomyositis rashes may be subtle but provide important clues to the diagnosis. Particular sites of involvement are the face, eyelids, anterior chest, upper back, extensor surface of the elbows, extensor surface of the knees, and the knuckles of the hands. Erythroderma may also be present.

Findings suggestive of scleroderma include tight skin on the hands and face, areas of skin with mixed hypo- and hyper-pigmentation, and digital ulcers.

Some less-common vasculitides can also cause skin ulcers and rashes.

HEMATOLOGIC ABNORMALITIES

Abnormalities on the Complete Blood Count (CBC) are quite common in many of the rheumatic diseases. Anemia of chronic disease is especially frequent in Rheumatoid Arthritis (RA) and Systemic Lupus Erythematosus (SLE). Leukopenia and thrombocytopenia are common in SLE. Thrombocytosis and anemia are commonly seen together in RA, Polymyalgia Rheumatica (PMR) and Temporal Arteritis. Unexplained hematologic abnormalities should prompt a careful search for not only malignancy but also a rheumatic cause. I addition, cytopenias are an important adverse effect from immunosuppressive medications. Glucocorticoids in any form commonly cause leukocytosis.

PULMONARY DISEASE

The lungs are commonly involved in many of the rheumatic diseases. In fact, lung disease may be the presenting manifestation. Pathology can be a direct result of the disease or medications used in treatment. It could also result from pulmonary infections associated with immunosuppression. Although most rheumatic diseases can affect the lungs, there are characteristic patterns that tend to occur with certain diseases.

Interstitial lung disease (ILD) is one of the most frequent pulmonary complications in these patients. ILD is most commonly associated with RA, SLE, Sjogren's syndrome, scleroderma and inflammatory myopathies. Findings can include shortness of breath, dry cough, rales and a restrictive pattern on PFTs. A chest CT most commonly demonstrates ground-glass changes or honeycombing. Patients with rheumatic disease who develop a persistent cough and shortness of breath and are not responding to conventional treatment may have ILD.

Pleuritis can occur in RA and SLE and usually presents with pleuritic chest pain and dyspnea. It is often confused with costochondritis. A less common pulmonary complication is alveolar hemorrhage associated with vasculitis, SLE or scleroderma.

Polymyositis and dermatomyositis may cause respiratory muscle weakness and resultant restrictive changes on PFTs but a normal chest CT. Usually this is associated with a very high CPK and an increased risk for aspiration pneumonia.

Organizing pneumonia is commonly associated with RA and can rarely be associated with methotrexate use.

Nodular disease is commonly seen in RA and Wegener's

granulomatosis. However, it is important to search for malignancy since many rheumatic diseases increase the risk of lung cancer and lymphoma.

CARDIOVASCULAR DISEASE

Cardiovascular disease and its prevention are increasingly important in the care of rheumatology patients. Rheumatic diseases can affect the heart in many ways, and we are learning that some are associated with a higher risk for acute cardiovascular events.

Pericardial effusion is the most common cardiac manifestation of RA and SLE. In RA patients, effusions rarely cause symptoms. Lupus patients can develop pericarditis, myocarditis and endocarditis. Both RA and SLE patients have increased risk for atherosclerotic disease. Control of risk factors should be emphasized as it is in patients with diabetes. Data in RA patients suggest fewer cardiovascular events occur with use of biologic agents and better disease treatment.

Scleroderma patients are at increased risk for pulmonary arterial hypertension. We are discovering the importance of screening these patients with PFTs and echocardiograms. The gold standard is to measure RVSP and PAP by right heart catheterization for early diagnosis and treatment.

All types of vasculitis can be important causes of cardiac and vascular symptoms. Consider temporal arteritis, Takayasu's arteritis, Behcet's disease and Kawasaki's disease in patients with a vasculitis affecting the large vessels. Although these diseases can be difficult to diagnose, clues to an inflammatory process such as fever, anemia, thrombocytosis, elevated CRP and/or ESR can sometimes suggest a rheumatic cause. Additionally, an unusual presentation may be a clue to underlying rheumatic disease.

In patients with a known rheumatic disease, consider an EKG or echocardiogram for new cardiac symptoms. In new patients with an atypical presentation of cardiac disease, consider a rheumatic disease.

RENAL MANIFESTATIONS

Renal disease is an important manifestation of lupus and some types of vasculitis. The most common finding is severe glomerulonephritis, which can lead to renal failure. Scleroderma patients specifically can develop renovascular disease leading to renal crisis. Additionally, many of the medications used to treat rheumatic diseases can have significant adverse renal effects such as tubulointerstitial disease.

Workup for lupus or vasculitis as a cause of renal disease requires a urinalysis and urine protein to evaluate for an active sediment and proteinuria to help determine whether a rheumatic disease might be involved. Often these patients require renal biopsy to assess the effect of their disease on the kidney.

It is important to monitor kidney function in many patients with rheumatic diseases. Patients with rheumatic disease without renal manifestations must be monitored for potential adverse medication effects.

EYE DISEASE

The rheumatic diseases most often associated with inflammatory eye disease include Sarcoidosis, Behcet's, reactive arthritis, inflammatory bowel disease-related arthritis, Ankylosing Spondylitis, RA and SLE. It is important for patients with a painful red eye to see an ophthalmologist promptly. Inflammatory eye diseases such as uveitis require prompt treatment and often long-term immunosuppressive medications to prevent loss of vision.

GASTROINTESTINAL DISEASE

Common GI symptoms such as diarrhea, abdominal pain, hematochezia and melena can all be associated with rheumatic disease.

Inflammatory bowel diseases, reactive arthritis, spondyloarthropathies, Behcet's disease, celiac disease and Whipple's disease can all cause intestinal inflammation and dysmotility resulting in diarrhea.

Abdominal pain can occur with or without diarrhea and is seen in inflammatory bowel disease and vasculitic syndromes such as Henoch-Schonlein Purpura (HSP), Polyarteritis Nodosa, RA, SLE, as well as scleroderma, Behcet's and Whipple's disease.

Gastrointestinal bleeding is usually due to a specific lesion in the gut and can occur in ulcerative colitis, HSP, vasculitis and Whipple's disease.

Workup up these symptoms includes EGD and/or colonoscopy. Sometimes a CT or a motility study can be helpful. Remember, many of the medications used to treat rheumatic diseases can have an adverse effect on the gut.

While this article is not meant to be a comprehensive review, it can serve as a framework to add rheumatic diseases to a physician's thought process in the evaluation of new signs and symptoms not primarily attributed to this subspecialty.

Think of rheumatic diseases as systemic diseases, not just causes of joint pain or arthritis. Even in a patient with no musculoskeletal symptoms, keep these disorders in the differential diagnosis. In a patient with a known rheumatic disease, consider that new signs or symptoms may be a new manifestation of the existing diagnosis. Finally, the medications used to treat these disorders should always be considered as a cause of new symptoms. ■

Dr. Weselman earned her Bachelor of Science in Biology from the College of William and Mary in Williamsburg, Va. She earned her Doctorate of Medicine from Baylor College of Medicine in Houston, Texas where she also completed her internship and residency in internal medicine. Dr. Weselman completed her fellowship in rheumatology at Emory University School of Medicine in Atlanta.

Dr. Weselman is a fellow of the American College of Rheumatology and a member of the Georgia Society of Rheumatology, American College of Physicians and American Medical Association. She is board certified in rheumatology and internal medicine.

Our Advanced Certifications Mean Better Results

Not all manual therapy is the same. Advanced training in the field of medicine means better results, and BenchMark has more therapists with advanced certifications in manual therapy and orthopedics, including board certified Orthopedic Specialists, than any other physical therapy practice in the region. Our patient outcomes prove it!

Use our convenient, toll-free
central scheduling: (866) 577-7342
We'll do all the rest!

BenchMark® PHYSICAL THERAPY

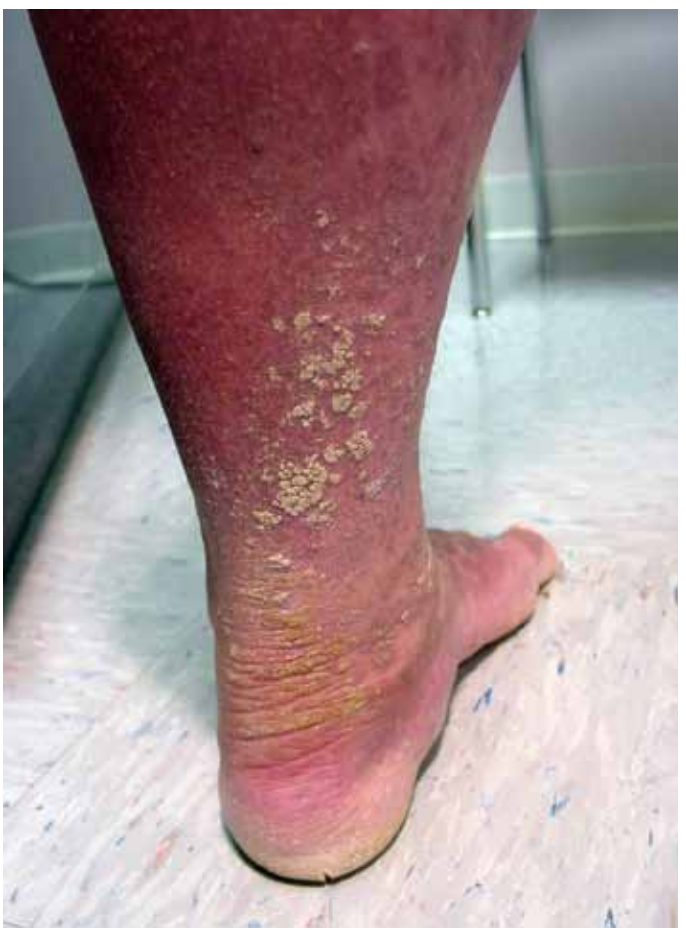
W. Hayes Wilson, M.D., F.A.C.R.

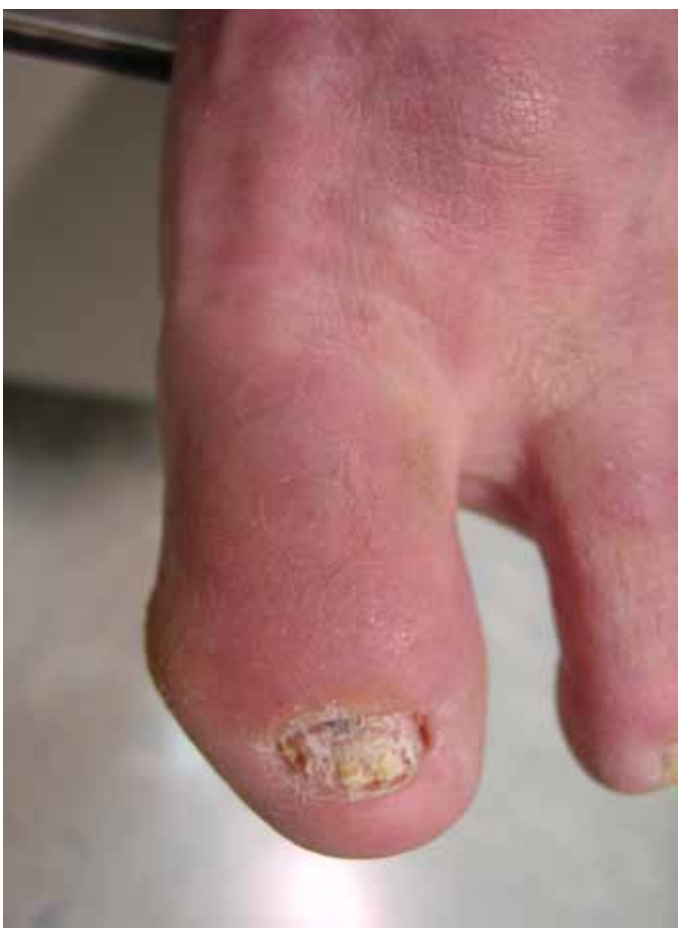
Psoriatic Arthritis

Phil Mickelson has put Psoriatic Arthritis (PsA) in the public spotlight. When he first announced that he had the disease and that his biologic therapy had allowed him to continue playing competitive golf, he had no idea the impact that he would make. There is now awareness that not only is Psoriatic Arthritis a life altering disease, but that there are very effective treatments available. The Arthritis Foundation tag line is "Take Control, We Can Help." In the end, Phil has to sink the putt himself, but to the extent possible he is better able to accomplish this without red, hot, painful and swollen joints because he is on a Biologic Response Modifier (BRM).

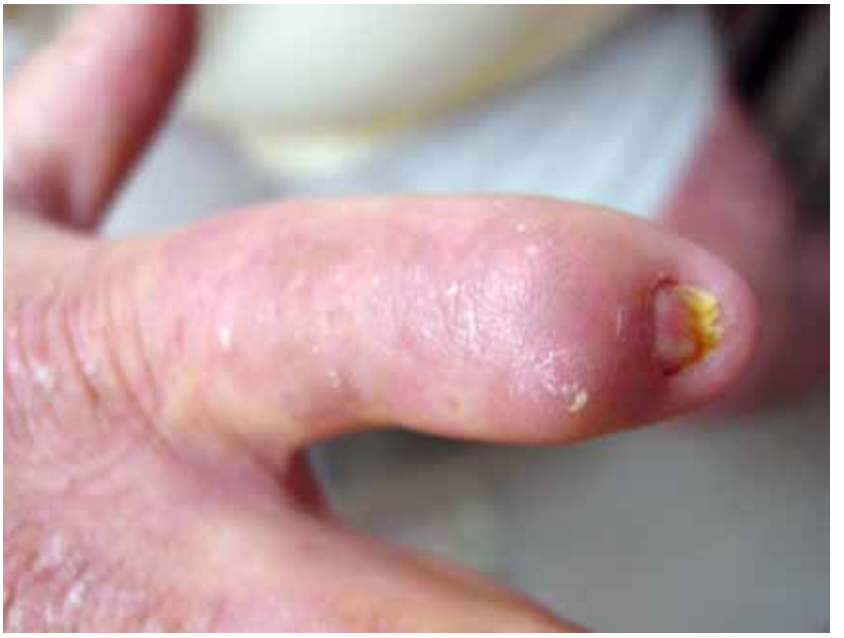
Psoriatic Arthritis is in the category of seronegative spondyloarthropathies, meaning that the Rheumatoid Factor is negative and Psoriatic Arthritis has a predilection for affecting the axial joints including the spine. Psoriatic Arthritis has the dubious distinction of having a variant called Arthritis Mutilans. It is always bad to have a disease with mutilation as half of its name. According the Arthritis Foundation and the National Psoriasis Foundation, Psoriatic Arthritis is quite common. An estimated two percent of the American population has Psoriasis, and 20 to 30 percent of them may come down with Psoriatic Arthritis. The onset of Psoriatic Arthritis is about 10 years after the onset of the Psoriasis, though the arthritis can precede the skin disease. Most patients are diagnosed between 15 and 35 years of age.

There are five different types of Psoriatic Arthritis. A symmetric type looks like Rheumatoid Arthritis with both sides of the body about equally affected. The most common is an Asymmetric variant, which can affect random joints on





either side of the body, often with sausage digits. There is Distal Interphalangeal (DIP) predominant PsA, where mostly the last joint of the fingers are involved. Spondylitis is more common in individuals who have the HLA B27 phenotype. Arthritis Mutilans is found in approximately five percent of patients with Psoriatic Arthritis. Most patients with Psoriatic Arthritis, 95 percent, have peripheral involvement, often times with sausage digits. It is not uncommon for a patch of Psoriasis on one finger with diffuse swelling whereas the digit next to it to appear normal with no inflammation.



The nails may have pitting and/or Onycholysis, which can easily be confused with a fungal infection. One of the more painful aspects of Psoriatic Arthritis is the Enthesitis, causing inflammation at the attachment of the muscle on the bone. Achilles tendinitis, plantar fasciitis, and epicondylitis are common examples of enthesitis. Uveitis can be associated with Psoriatic Arthritis, particularly in patients that are HLA B27 positive. The Psoriasis is sometimes missed because it is in the scalp, behind the ears, in the umbilicus or in the center between the gluteal muscles.

The Renaissance of Rheumatology occurred in the late twentieth century with the advent of the Biologic Response Modifiers (BRM). We still use Disease Modifying Anti-Rheumatic Drugs (DMARDs), but the BRMs have made the most dramatic effect. Often times our patients will tell us that they can endure the joint pain, but clearing up their skin and getting their energy back really is what they look forward to the most. Methotrexate in general helps the peripheral arthritis, but it is not very effective on the axial arthritis or spinal symptoms. The Biologic Response Modifiers work not only on the axial arthritis, but also on the Enthesitis, Dactylitis and quite dramatically on the skin.

Fair balance requires that I point out that we screen all of our patients for Tuberculosis and monitor them for symptoms while on the biologic medications. While our patients are on Biologic Response Modifiers, they are at increased risk for opportunistic fungal infections including Histoplasmosis and Coccidiomycosis. We are particularly concerned if the patient is from, or has travelled extensively in, the Ohio River Basin or the San Joaquin Valley.

We tell all of our patients to treat any infection seriously, and if they are sick enough to be on an antibiotic, then they should hold their immune modulating medication(s), including any Disease Modifying Anti-Rheumatic Drug or Biologic Response Modifier. In the end, we as rheumatologists try to decrease the autoimmune response, normalizing immune function while not compromising the ability to defend against infection – much like a cardiologist would try to normalize the blood pressure in hypertension, without causing hypotension.

The TNF inhibitors can dramatically affect the destructive inflammatory effects of Psoriasis and Psoriatic Arthritis. Most importantly, there are very effective therapies that can make the difference between existing with a disease that changes your patient's life, and changing a disease so your patient can live a full and productive life. Arthritis is unacceptable. Take all measures to regulate the immune response to normal and preserve the joints. Your patients can find more information at www.arthritis.org, www.rheumatology.org, or www.psoriasis.org. ■

Understanding the Immune Response

Gary E. Myerson, M.D., F.A.C.R.

Centuries ago, geography itself served as the primary barrier to disease exposure. But man's exploration of the planet, enhanced by progressive technological expertise, has permitted both exposure to and expansion of diseases worldwide. However, our increasing knowledge of science overall and the specific components of the immune response has led to numerous diseases either being reduced or eliminated.

The immune response is an ancient system. Certain components, however, have not significantly changed in the millions of years of existence of species with vertebrae. The homo-sapiens and homo-erectus, nearly 250,000 years ago, possessed an immune system similar to ours. Innate immune response has been present throughout this entire period. It has been our "equalizer" in dealing with the microscopic environment that envelops us. We have made" friends" with many microbes, resulting in relationships that are symbiotic, while others are indeed antagonistic or parasitic. It is this inherent, genetically provided, innate response that is our defense against invaders both physically and chemically. The Adaptive or acquired immune response, our body's ability to develop antibodies, has allowed us to improve or enhance our own innate immune response. Biologic therapy with monoclonal antibodies now allows us to manipulate specific proteins important in the immune response.

Our first line of defense includes the skin and lining surfaces of the internal body organs. There are enzymes in body secretions including lysozymes, phospholipases and defensins, which disrupt cell membranes promoting cell death. Periodically, the anatomical barriers are penetrated and an "acute inflammatory reaction" follows. The release of acute phase reactants: transferrin, CRP, interferon and interleukins, results in the cardinal signs of inflammation: calor/hot, rubor/red, dolor/painful and tumor/swelling. The phagocytic cells including the neutrophils, macrophages, dendritic cells, natural killer or NK cells and eosinophils are the major players in identifying and responding to microscopic invaders. The phagocytic cells are directed to specific locations via cytokines, protein communicators of inflammation that identify, engulf and destroy pathogens. These cells are primary players of the innate immune response - the oldest form of the host defense system.

The innate immune response is triggered when microbes (bacteria, virus and fungi) are detected. On or attached to the cell membranes of pathogens are commonly shared molecules referred to as pathogen-associated molecular patterns (PAMPS). These include lipopolysaccharides (LPS), lipoteichoic acid, flagellin, RNA of viruses, amongst others. These molecules are recognized by receptors on the phagocytic cells called pattern recognition receptors (PRR). One large group of the PRRs is a specific subgroup entitled Toll-like receptors (TLR). These highly specific receptors, numbered 1 through 13, identify and bind PAMPS, resulting in cytokine production triggering inflammation.

The complement cascade is also a major component of the innate immune response. The Complement system serves as an identifier and clearing house of pathogens by promoting vascular permeability, recruitment of phagocytic cells and ultimately the opsonization of bacteria and immune complexes. Opsonization refers to the coating and subsequent marking of bacteria for future destruction. A large component of opsonization occurs in the spleen, which is why splenectomized patients particularly need vaccinations. Complement CH50 is a measure of total complement activity. Individuals with active autoimmune disease may have low levels of C3 and C4 due to overactivity of the complement cascade. This results in immune complex formation and the consumption of complement. When the inflammation resolves, complement levels return to the normal range. Therefore, low levels of CH50 and C3 and C4 reflect active autoimmune disease, but higher levels do not.

Innate immune response is a non-antigen specific, immediate reaction. It does not produce immunological memory. For long-term protection, there is our adaptive or acquired immune response. Immunological memory is accomplished by the development of antibodies as our ready reserve defense force. Subsequent encounters with the same organism result in an enhanced response. There is a period of approximately 10-14 days for antigen-specific antibodies to be developed.

All cells of the immune system have their origin in the bone marrow. The myeloid series primarily produces the cells of the innate immune system including the neutrophils, monocytes and dendritic cells. While the lymphoid series (lymphocytes) produces cells for the acquired or adaptive immune response, the T cells undergo differentiation into their distinct types under the influence of the thymus gland. B cells become mature in the lymph nodes and the spleen. There is a tremendous amount of interaction between both

systems, utilizing the myeloid series innate immune system for stimulation and activation of the acquired immune response.

T-cells are involved in the cell-mediated immune response. They have no cytotoxic activity and do not kill infected cells or clear pathogens directly. Instead, they control the immune response by directing other cells to perform these tasks. The cytokines the T-cells produce are the "protein communicators of inflammation." Three important cytokines include TNF alpha and interleukins IL-1 and IL-6. These have been identified as primary players in autoimmune diseases including rheumatoid arthritis, psoriasis and psoriatic arthritis, Ankylosing spondylitis and inflammatory bowel disease (specifically Crohn's disease) as well as Uveitis and Sarcoidosis.The T-cell begins as a "naïve" cell, which requires antigen presentation in order for it to become activated. These antigen-presenting cells (APC) include the macrophage, dendritic cell and the B cell. The APC presents the antigen to the naive T-cell via its major histocompatibility complex (MHC) T-cell receptor (TCR). Depending on the APC cell type and the predominance of surrounding cytokines, T-cells differentiate in one of four directions:

1. TH 1 cells. APCs include the macrophages and dendritic cells. IL 12 and interferon (INF) gamma are the cytokines that drive differentiation. This type of T-cell usually develops from exposure to intracellular bacteria, fungus or viruses.

2. TH 17 cells. Neutrophils; IL 23, IL-1, IL-6. Usually from exposure of extracellular bacteria and fungi.

3. TH 2 cells. Eosinophils and basophils. IL-4. Usually from exposure to parasites.

4. T reg (regulatory) cells. Response to self-antigens. IL-10 and TGF beta. Down regulates autoimmune response.

There is however cross stimulation and inhibition depending on which interleukin predominates. For instance, IL6 inhibits TGF beta, therefore driving TH 17 production and reducing T reg production, Alternatively, IL-4 while driving TH2 production inhibits TH 17 production.

Cell membranes of T-cells have cluster differentiation (CD) glycoproteins CD4 and CD8. Cells with the CD4 glycoprotein are referred to as helper cells, and those with CD8 glycoprotein are referred to as cytotoxic T-cells. CD4 helper cells assist in the maturation of B cells into plasma cells and memory B cells. They also assist in the activation of cytotoxic T-cells and macrophages. Cytotoxic CD8 cells destroy viral-infected and tumor cells and also play a role in transplant rejection.

The humoral immune response is directed by B lymphocytes (B cells) differentiating into plasma cells which produce antibodies. The B cell develops through several stages. A B cell begins as a progenitor cell, then progresses to a pro-B cell and finally a pre-B cell. At this point, it leaves the bone marrow to mature in the lymph nodes and spleen where it becomes exposed to the pathogenic environment. As it matures, it develops three surface receptors – Blys, TACI and the B-cell receptor (BCR). When these receptors are bound, the cell matures and continues to exist. Failure to bind these receptors results in apoptosis or programmed cell death. This has become a recent important discovery since Benlysta, a new drug for the treatment of lupus, works by inhibiting Blys, the cell surface receptor.

B cells exist as clones. All B cells derived from a specific clone can produce "progenies" that can recognize specific antigens and produce specific antibodies for generations. With each cycle of exposure, the number of surviving memory cells continues to increase This is the basis of "immunogenic" memory. There are 3 B cell types: Plasma B cells, memory B cells and B-1 and B-2 cells.

Plasma B cell produces large amounts of antibodies or immunoglobulin including IgG, IgA, IgM. and IgE. These immunoglobulins are a hallmark of our defense system. We're aware that deficiencies in any one of them or their subtypes results in the development of recurrent infections. Specific types of intravenous and subcutaneous gammaglobulin are available for those deficiency states.

Memory B cells are formed from activated B cells that are specific to the antigen encountered during the primary immune response. These cells are capable of living "a long time" and can respond quickly following a second exposure to the same antigen.

B-1 cells and B-2 cells, B-1 cells express high levels of IgM greater than IgG and are polyspecific – meaning that they have the ability to produce low-level response to many antigens.

Unlike the T cell, the B cell does not need the antigen presented to it. It recognizes the antigen in the blood or lymphatic system and engulfs it. It can then act as an antigen-presenting cell itself by displaying its antigen bound to its unique MHC on the cell surface allowing a T-cell to bind to it. Through a co-stimulatory mechanism necessitating a second binding site to be activated, cytokines can then be released. The cytokines released by that T-cell further propagate the B cell into its mature state, producing plasma cells and more immunoglobulin. Antibodies bind to their specific antigens forming immune-complexes. These complexes are then "cleaved" by the complement system and eliminated through the reticuloendothelial system.

Autoimmune diseases result from aberrant antibody production. These autoantibodies are actually produced on a regular basis by all individuals. Fortunately, more than 90 percent of them undergo spontaneous apoptosis and never progress. Anti-B cell medications predominantly work by interfering with the B cell during its maturation. Examples of these medications are being utilized in both oncology and rheumatology.

A baby's immune defenses are passively transferred from the mother at birth. The baby's own immune defenses take approximately three months to begin to function.

In the H.G. Wells classic science fiction novel "War of the Worlds" circa 1890s, the narrator eloquently describes on day 15 "the far advanced Martians invaded the earth, began to destroy mankind and make the planet its own. As their overturned war machines ... laid stark and silent, the Martians were dead - slain by the putrefactive and disease bacteria against which their systems were unprepared ... after all man's devices had failed, by the humblest thing that God, in his wisdom had put on this earth ... these germs of disease had taken toll on humanity since the beginning of things – taken toll on her pre-human ancestors since life began here. But by virtue of this natural selection of our kind, we have developed resisting power; to no germs do we succumb without a struggle - our living frames are altogether immune. But there are no bacteria on Mars ... our microscopic allies began to work their over-throw... it was inevitable!! ■

Kimberley E. Wilson, M.D., F.A.C.R.

The Risk of Cardiovascular Disease in Chronic Inflammatory Arthritis

Rheumatoid Arthritis (RA) is a chronic progressive inflammatory disease that strikes up to 1 percent of the general population. This debilitating arthropathy is associated with a significantly higher rate of mortality and morbidity, primarily from cardiovascular disease (CVD). The increased risk of CVD in RA is now thought to be equivalent to the risk seen in Type II diabetes. In fact, the risk of ischemic stroke in RA is increased by 50 percent, and the risk of myocardial infarction is increased by 60 percent when compared to the general population.[1] Likewise Psoriatic Arthritis (PsA), Ankylosing Spondylitis (AS) and Systemic Lupus Erythematosus (SLE) all carry an increased risk of cerebrovascular stroke, hypertension and heart disease.[2]

Epidemiology studies reveal that the increased risk of CVD seen in inflammatory arthritis is not fully explained by the standard risk factors cited, neither in the Framingham Heart Study nor in the European SCORE study, (Systematic Coronary Risk Evaluation). In these two prospective studies, RA patients stopped smoking and achieved ideal lipid levels, yet their cardiovascular risk was still increased. This persistent increased risk may be due to metabolic abnormalities inherent to the chronic inflammation itself.

Low levels of HDL (high density lipoproteins) and high levels of total cholesterol, LDL (low density lipoproteins) and triglycerides have long been associated with increased CVD risk in the general population. Investigations looking at metabolic disturbances in untreated early RA conversely revealed low levels of LDL and total cholesterol in these newly diagnosed RA patients compared to age- and sex-matched healthy controls. Although the total cholesterol in inflammatory arthritis patients was low, the HDL levels were comparatively lower, thereby resulting in a high total cholesterol/HDL ratio. This high ratio is a specific predictor for future CVD disease.[3]

In seropositive RA patients, not only is an initial lowering of total cholesterol seen, but an elevation of triglycerides occurs as well.[4] After adjustment for age, sex, hypertension and statin use, clinical immunologists at the Oklahoma Medical Research Foundation found that 60 percent of RA patients who developed CVD had similar high levels of triglycerides. These RA patients also had higher VLDL, LDL, ApoB, ApoC-III and ApoB/ApoA-I. Many of these same lipoproteins are found to be associated with atherosclerosis. [5]

With regard to HDL, current opinion holds that HDL function may be as important as the HDL level in the development of CVD. A study published in The New England Journal of Medicine suggests that decreased phospholipase activity and diminished hydrolysis of VLDL may not only lead to the reduced number of HDL particles, but an alteration in HDL composition and function as well. This alteration of HDL, along with the lipoprotein dysfunction and elevated triglycerides, can lead to increased cardiovascular risk.[6]

So what is the link between inflammation and CVD? Endothelial cell activation is "characterized by loss of vascular integrity, increased expression of leukocyte adhesion molecules, change in phenotype from antithrombotic to thrombotic, production of several cytokines, and up-regulation of HLA class II molecules."[7] Vaudo, et al found a strong association between endothelial dysfunction and chronic inflammatory markers such as C-reactive protein (CRP) levels.[8] "CRP level down-regulates endothelial nitric oxide synthase transcription and also decreases both basal and stimulated nitric oxide release."[9] In that manner, CRP level is known to promote atherosclerotic processes and endothelial cell activation. Recent reports have further implicated chronic inflammation in the pathogenesis of atherosclerosis. In June of this year, researchers documented higher levels of serum interleukin-6, a known mediator of inflammation in RA, in patients who have elevated triglycerides and lipoprotein dysfunction.[10] Other studies

have shown an increased frequency of carotid plaques in RA and PsA patients with more severe disease.[11] Carotid artery studies on SLE patients without traditional cardiovascular risk factors exhibited unexpected early atherosclerotic changes.[12] Simply put, chronic inflammation leads to endothelial dysfunction, which in turn contributes to CVD, independent of known risk factors.

It has long been established that early aggressive control of the inflammatory process improves the outcome of RA. Risk factors for poor outcome in inflammatory arthritis include disease activity scores, seropositivity, inflammatory markers, extra-articular manifestations, physical disability and destructive joint changes on X-ray. These inflammatory risk factors correlate with increased risk of CVD. Evidence now suggests that medications employed to treat inflammation may also have a positive effect on cardiovascular risk.[13] While it is important to pay close attention to the traditional CVD risk factors in chronic arthritis patients, these preventive measures alone do not adequately improve their morbidity and mortality from CVD. A combined approach aimed at the prevention of CVD and adequate treatment of the chronic inflammatory arthritis is warranted.

The first step in management is to annually assess and provide counseling with regard to smoking, physical activity, nutrition, weight and blood pressure. The second intervention is to pursue cardioprotective therapy, such as statins, ACE inhibitors or angiotensin II blockers, if the patient's cardiovascular risk rises above the acceptable threshold. Finally, aggressive control of the inflammatory process is critical.

Primarily concerned with pain and limitation in function, arthritis patients often are more compliant with therapy for their chronic disease than with preventive care measures. It is important that inflammatory arthritis patients remain involved with their primary care physician to ensure regular monitoring for cardiovascular disease. ■

References:

1 Ann Cardiol Angeiol, 2012 Apr; 61(2): 111-7
2 Oxford Journals-Rheumatology, 2008; 47 (5): 718-723
3 Circulation, 2008; 117: 743-53
4 Curr Pharm Des, 2012 April; 18(11): 1465-77
5 Scand J Rheumatol, 2010; 39: 447-53
6 New England Journal of Medicine, 2011 Jan; 364: 127-135
7 Arthritis Res Ther, 2005; 7: R634-43
8 Arthritis Care Res, 2007 March 15; 57(2): 189-354
9 J Hypertens, 2006; 24: 643-5
10 Ann Cardiol Angeiol, 2012 June; 64(8): 872-6
11 J Invest Derm, 2009; 129: 2411-8
12 J Rheumatol, February 2002; 29(2):292-297
13 Seminars Arthritis Rheum, 2007 February; 36(4): 203-209 have shown

Kimberley Wilson, M.D. practices Rheumatology with Piedmont Rheumatology Consultants. She is the Co-Chairperson of the Lupus Foundation of America, Georgia Chapter's Medical Advisory Board. Dr. Wilson was voted "Top 100 Female Physicians" by the Editorial Board of Woman Magazine in 2002 and she received the Service of the Year Award by the Georgia Chapter in 2003. Dr. Wilson also received the 2009 Excellence in Patient Care Award from the Georgia Chapter.

Vaccines Contraindicated in Adult Patients with Autoimmune Inflammatory Rheumatic Diseases

Yellow Fever
MMR (Measles, Mumps, Rubella)
Oral Typhoid
Herpes Zoster
Flu Mist (nasal spray)
Small Pox

The Herpes Zoster vaccine may be used with caution in selected cases. Consider administration at least two weeks prior to initiation of immunosuppressive therapy or three months after last immunosuppressive dose. Watch for increased risk of disseminated infection.

Recommendations on Routine Adult Immunization Schedule-

(Taken from www.cdc.gov/flu)

Annual intradermal vaccination for H. Influenza is strongly recommended.

If possible, administer the Pneumococcal vaccine at least two weeks prior to initiation of immunosuppressive therapy. Repeat Pneumococcal vaccine every five years.

Dan Appelrouth, M.D., F.A.C.R.

Preventing Office Theft

Eight years ago I wrote an article about preventing office theft in this Journal. Much has stayed the same during this time, and much has changed. What has not changed is how to deal with personnel issues:

LESSONS TO BE LEARNED

1: Be aware of all personnel who handle money. The general rule used by fraud experts is that 80 percent of employees will steal if given the opportunity, 10 percent will always steal, and 10 percent will never steal. Thus you have to be aware that potentially 90 percent of your employees would steal. Bond all employees handling money – from those opening letters to inputting data to taking the checks to the bank. Other examples of theft include: Taking property from the practice; identity theft by stealing patients credit card numbers and social security numbers; and payroll theft by creating fictitious employees, unauthorized bonuses, cheating on a time card or changing pay rates.

2: Embezzlement is still the No. 1 financial crime, yet only 10 percent of thefts are reported. The person who embezzles is often the last person you suspect. Warning signs include: 1. A very dedicated employee, working for you the longest, whom you implicitly trust and who is in a position of authority. This person works weekends, holidays and nights, never takes vacations and does not want any help; 2. An employee whose spouse may have lost their job; who personally may have health problems, and yet who is living above their means; 3. Be wary when there are increased patient complaints about billing errors as insurance payments could be pocketed by the employee. Theft schemes usually take one to two weeks to be identified when not maintained. Therefore insist that every employee who handles your money takes a minimum of a one week vacation every six months

How to Reduce the Risk of Theft and Embezzlement

The Hiring Process: Be vigilant. Check all resumes and references. Call previous employers – especially if it is someone whom you know. If you do not know the person, ask the previous employers "Would you rehire the person?" If there is silence or reluctance, or the answer is "No," do not hire the applicant. I have had applicants with wonderful written resumes only to find out that the referring doctor never wrote the resume; the office stationery was stolen and nothing but falsehoods were written. Do background checks that include social security numbers, state criminal records and national felony warrant searches. Do credit checks for employees who will be handling money. Do not wait until after the person is hired to check references – it then may be too late to prevent theft.

CREATE SECURITY AWARENESS IN THE OFFICE

1: Your office manual should document your company's policy on security and the consequences of theft. ALWAYS get the police involved in cases of office theft as many personnel are repeat offenders. You must have a police report to show to the insurance company who bonds your staff.

2: Have identification badges made for all employees.

3: Engrave your property in two locations: one obvious and one hidden. Record serial numbers for all expensive equipment and furniture and personal property in the office. Keep a record of your inventory in a separate and safe location.

4: Office computers and laptops should not be allowed out of the office. Only authorized personnel should be allowed in rooms where expensive equipment is stored. Rooms should remain locked when no one's inside, and the names of all employees, and their times of entry and exit, should be kept in a log book.

RULES FOR BETTER OFFICE EFFICIENCY

1: Have a policy on Internet browsing and enforce it. Many offices block social media sites such as Facebook. Inform employees in your manual that email and Internet traffic are monitored and audited periodically. Anyone who is abusing your policies should know the consequences of their actions.

2: Have a sense of your employees. Do they ask for money? Are they in debt? Are they getting a divorce? Do they use drugs? All of this information is crucial to understand potential problems.

3: Checks coming into the office should immediately be stamped "For Deposit Only." Processed checks should be stamped "Void."

4: Have a security box or safe to store checks, cash and receipts.

5: Do not give your office manager the authority to sign checks.

6: Have your office manager write out each check you will sign and have it attached to its bill. Sign the checks when you have uninterrupted time, are not harried and can pay attention to your task. If there is no bill attached, do not sign the check.

7: Do not allow any employee to take home mail, checks or money

8: Have your bank send your monthly office bank statement to your home or a P.O. box, and not to the office. Make sure every check has the correct signature and that the amount of the check has not been changed. Reconcile the account within 30 days from when the statement was mailed.

9: If you expect to receive a large amount of money in a check, also have the check sent to your home or P.O. box. After depositing the check into your account, then give the receipt to the office manager to reconcile. If you are asked the question, "Don't you trust me?", you can answer with "I trust you but I need to verify."

10: Don't us a rubber stamp for your signature.

11: Don't leave blank checks in the printer overnight. Theft by cleaning crews does occur.

12: Balance receipts and deposits daily.

13: When adding a vendor, be mindful. Employees will set up phony companies in order to pay themselves with your money.

14: Use a bank that gives you a record of both the front and back of your check. The police and insurance industry requires this for proof of theft.

15: If there are problems with credit card transactions, you may need to install office cameras to identify the cause.

16: At least once a week, meet with your Accounts Receivable person. Make sure you get answers to your questions.

17: Use a diamond-cutting shredder. Shred all documents that have full credit card numbers to prevent identity theft.

With change in your office policies and procedures, you may meet resistance from your staff. However these implemented changes do make your practice more secure. However, no system is foolproof. There are businesses that specialize in practice protection: If you are so inclined, do not hesitate to use them.

It is incumbent that you be proactive and on top of your business and staff issues. You deserve to reap the benefits of your labors. I hope I have provided you with basic guidelines that will help you to protect your practice investment. ■

The Art of Medicine

DEBRA JANSEN

www.debjansenphotography.com

MEDICAL ASSOCIATION OF ATLANTA
EST. 1854

The Pulse of Atlanta Medicine

2011-2012 Board of Directors

Officers

Rob Schreiner, M.D. President

Lisa Perry-Gilkes, M.D. President Elect

Hayes Wilson M.D., Treasurer

QuentinPirkke M.D., Secretary

Michael Hilton M.D., Chariman of the Board

Directors

Robert Albin, M.D.
Larry Bartel, M.D.
Thomas Bat, M.D.
Sara Caceres-Cantu, M.D.
Larry Cooper M.D.
Barbara Croft, M.D.
Rutledge Forney, M.D.
Matt Gwynn, M.D.
John Harvey, M.D.
Brian Hill, M.D.
Mark Hutto, M.D.
Albert Johary, M.D.
Paul King, M.D.
Deborah Martin, M.D.
Cody McClatchey, M.D.
Dickie McMullan, M.D.
Dorothy Mitchell-Leef, M.D.
Bidemi Olaniyi-Leyimu, M.D.
Alan Redding, M.D.
Gary Richter, M.D.
Randy Rizor, M.D.
Maurice Sholas, M.D.
William Silver, M.D.
Barry Silverman, M.D.
Sumayah Taliaferro, M.D.
Earl Thurmond, M.D.
Steve Walsh, M.D.
Charles Wilmer, M.D.

Join the MAA today!
For membership information, contact David Waldrep, Executive Director at 404-881-1020.

The Medical Association of Atlanta (MAA) is a non-profit association dedicated to the advancement of organized medicine in Atlanta.

The Medical Association of Atlanta's Sponsors

PLATNIUM

THE DOCTORS COMPANY FPIC

First Professionals Insurance Company, Inc. is a leading provider of professional liability insurance for physicians, dentists, and other healthcare providers, primarily in Florida, Georgia, and Arkansas.

www.firstprofessionals.com

KAISER PERMANENTE
The Southeast Permanente Medical Group, Inc.

With more than 400 primary- and specialty-care practitioners, The Southeast Permanente Medical Group (TSPMG) is part of Kaiser Permanente's integrated health care delivery system. Our physicians are connected through one of the largest electronic medical record systems in the U.S., helping us lead the way in improving clinical practice and overall health care quality.

physiciancareers.kp.org/ga

MAG MUTUAL
Because your patients come first.

MAG Mutual is dedicated to helping physicians and others in the medical community focus on providing quality patient care by providing innovative business, financial and insurance solutions to help those we serve meet their professional, business and personal goals.

www.magmutal.com

SILVER

Arnall Golden Gregory LLP ***www.agg.com***
Bank NY Mellon ***www.bnymellon.com***
Blue Wave Computing ***www.bluewavecomputing.com***
Cbeyond ***www.cbeyond.com***
Shandy Creative Solutions ***www.shandycreative.com***
Centene Corporation/Peach State Health Plan ***www.pshpgeorgia.com***
Galen Advisors ***www.galenadvisors.com***
Habif, Arogeti, & Wynne, CPAs ***www.hawcpa.com***
Favorite Healthcare Staffing, Inc. ***www.favoritestaffing.com***
Northside Hospital ***www.northside.com***
Pfizer ***www.pfizer.com***
Piedmont Hospital ***www.piedmonthospital.org***
Potter Holden, & Company ***www.potterholden.com***
Saint Joseph's Hospital ***www.stjosephsatlanta.com***
Southern Protective Group ***www.southernprotectivegroup.com***
Suntrust ***www.suntrust.com/medical***

The Pulse of Atlanta Medicine

Join us at a joint legislative meeting of the
Coalition of Atlanta Medical Societies

Cobb Medical Society
DeKalb Medical Society
Gwinnett-Forsyth Medical Society
Medical Association of Atlanta

Speaker

Rob Schreiner, M.D.
President, The Southeast Permanente Medical Group
"Clash of the Titans: why Hospitals, Physicians and Plans are learning to work together"

Legislative Panel Discussion Federal and State
PPACA post SCOTUS

Monday, September 24, 2012

6:00 pm Reception
7:00 pm Dinner

Crowne Plaza Atlanta Perimeter at Ravinia

4355 Ashford Dunwoody Road
Atlanta, GA 30346

Free for members and guest
Cash Bar

Reservations Required
RSVP by Monday September 17, 2012
404-881-1020

Medical Association of Atlanta

2013 Membership

10% Discount

Renew your membership or join the MAA by October 5, 2012 and receive a 10% discount!

Contact the MAA office at 404.881.1020 or pay online at www.maa-assn.org to receive your 10% membership discount now.

Gout: Simple Rules for Proper Management

Gary R Botstein, M.D., F.A.C.R.

There is exciting new research about gout regarding mechanisms and treatment modalities. However, the biggest challenge we face about gout is the educating of the medical community (and our patients) in how to manage the disease with the familiar medications available.

In my 35 years of rheumatology, I see the same errors appearing over and over, often by people I know are good physicians. Patient compliance is notoriously poor (at least between attacks). There would be little debate in the rheumatology community about the following simple rules in the management of gout:

1. ALWAYS aim for a uric acid target of less than 6.0 (perhaps 5.0 for tophaceous gout). Most patients can achieve these levels with gradual dose escalation of Allopurinol or Probenecid. (I must admit, if I have a very fragile patient whose uric acid gets to 6.5 and hasn't had an attack in years, I might accept that level rather than take the small risk associated with increased doses of medication.)

2. ALWAYS get to the target uric acid level gradually - over a time period of 3-4 months, not weeks. Rapid lowering of uric acid often precipitates severe flares, which may become polyarticular, sometimes associated with fever and requiring moderate dose steroids or hospitalization. There is no rush to achieve the target, and in most cases, patients may continue to have some gout attacks during the first year of lowering uric acid. Remember, conceptually we are trying to dissolve the uric acid deposits that coat the joint and set the stage for gout attacks. This is a slow process, and the patient will remain at risk for attacks until the crystalline urate burden has been decreased.

3. ALWAYS use colchicine on a daily basis when initiating urate lowering treatment (Probencid, Allopurinol or Uloric). This should be at 0.6 mg once or twice daily (if tolerated and not contraindicated) for at least 6 months. This will limit or minimize attacks during the period of urate lowering.

4. ALWAYS examine the skin and joints for tophi, which might allow you to confirm the diagnosis of gout without the discomfort of arthrocentesis. Tophi may appear white, flesh colored or yellow. Stretching the skin over a flesh-colored nodule may make it more yellow in appearance. I have found this kind of nodule, often over small joints, to be tophaceous. Inserting a small gauge needle into a tophus and squirting the contents of the needle on the slide almost always yields sheets of uric acid crystals under the microscope, even in the absence of any free fluid.

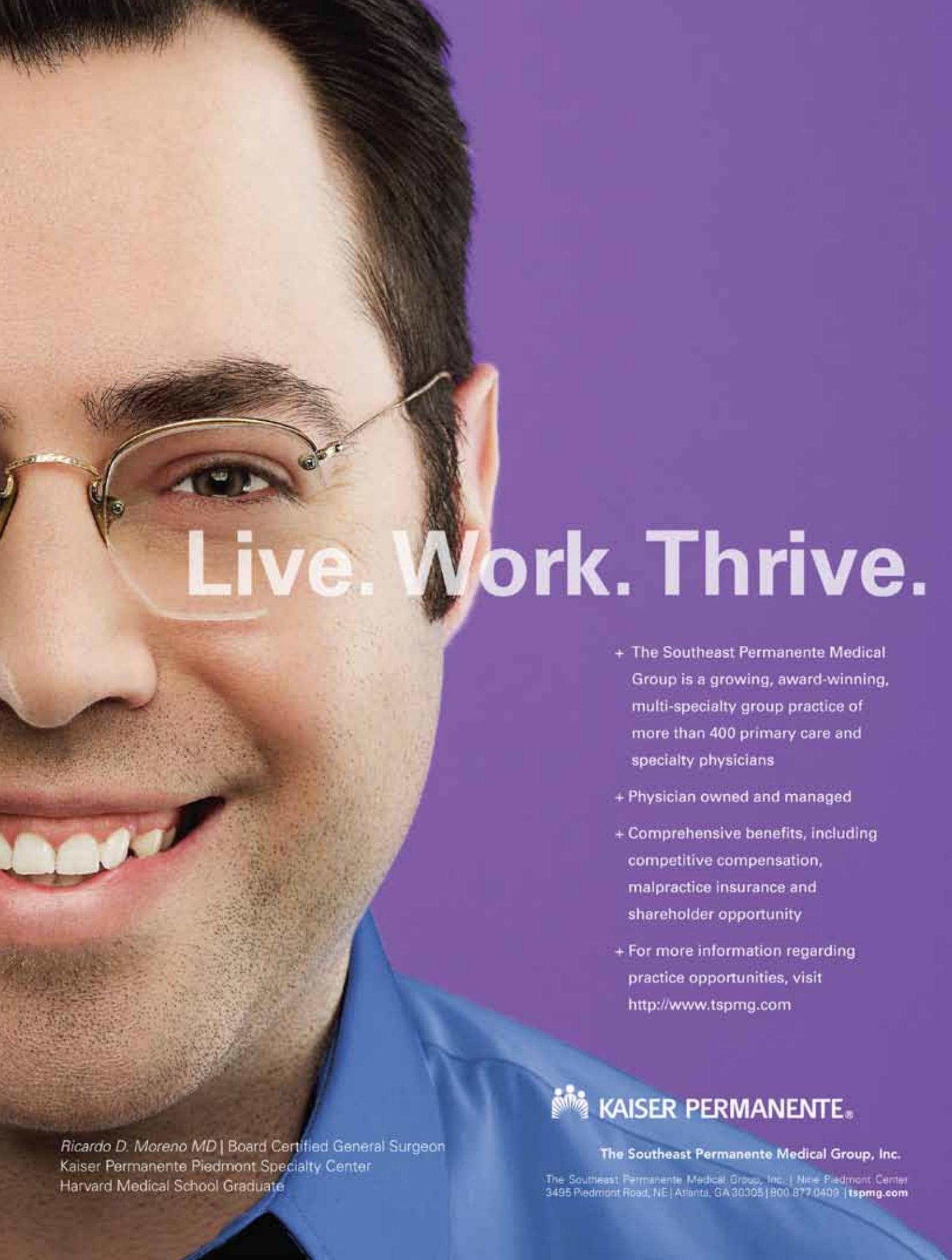
5. ALWAYS initiate urate lowering treatment after approximately three gout attacks. Symptomatic treatment with non-steroidal anti-inflammatory drugs (NSAIDs), colchicine or steroids is not sufficient.

6. NEVER initiate uric acid lowering during an acute attack. The most common error I see is starting Allopurinol, Benemid or Uloric during an acute attack. This often results in severe flares, both protracted monoarticular attacks as well as polyarticular attacks. I see this several times per year. It then becomes difficult to get patients to start uric acid lowering treatment because they associate the flare with these drugs. Follow two simple rules: "go low, go slow" and try to wait six weeks after an acute attack has resolved. (This is not always possible when attacks are happening very frequently.) I personally start Allopurinol at 50 mg daily for a month, and then increase to 100mg daily and then titrate monthly until reaching the target uric acid of less than 6.0. It is the rare patient who cannot achieve target with this regimen.

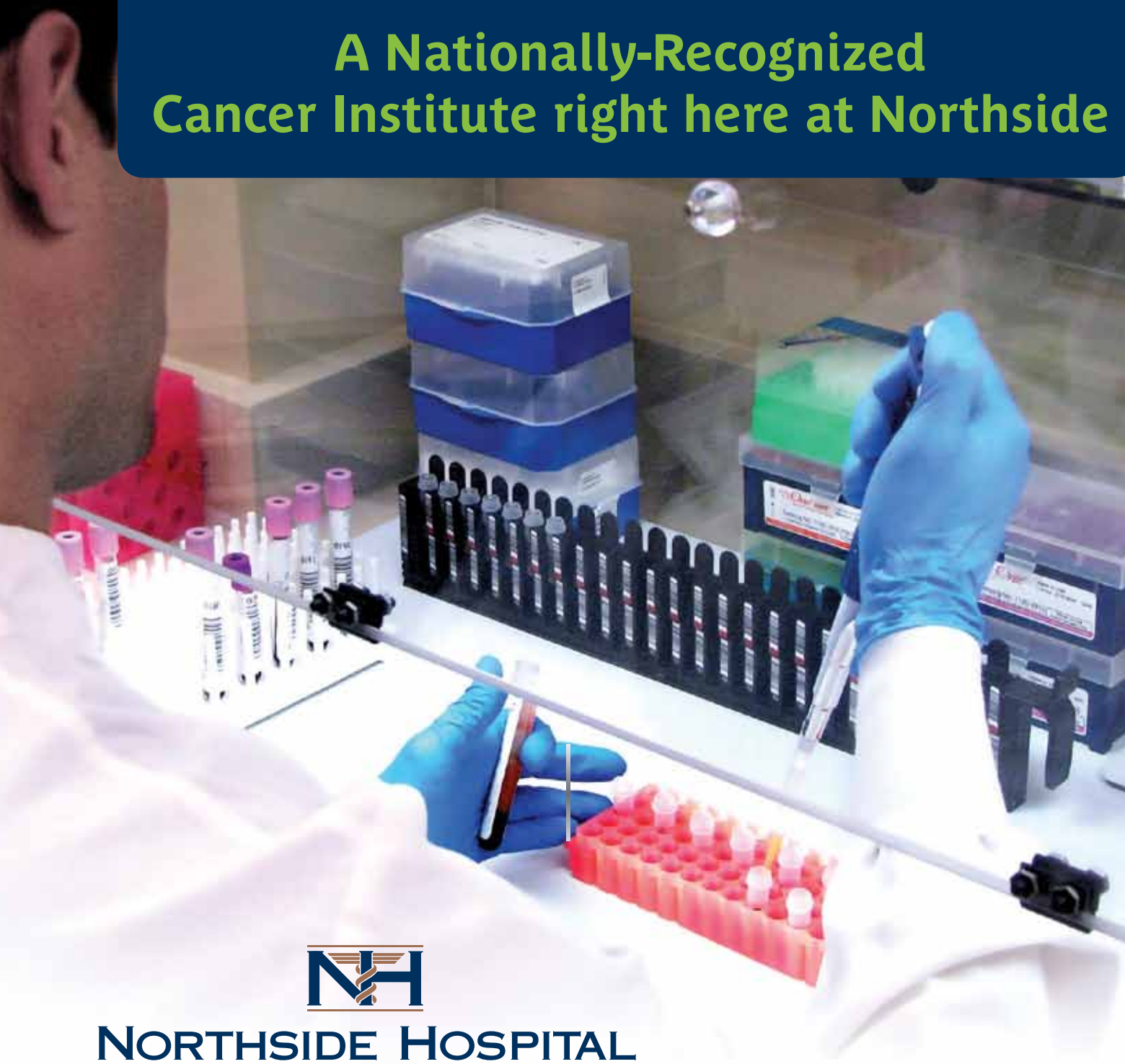
7. NEVER stop Allopurinol or any urate-lowering treatment during a flare. The enemy of gout is abrupt change in uric acid, either up or down. Aim for a steady, slow drop with no fluctuations.

8. NEVER treat with long-term daily NSAIDs or intermittent colchicine without addressing the hyperuricemia. Rheumatologists see patients who have been treated this way who go on to permanent destructive and crippling arthritis, even without recollection of recurring attacks.

It is rare that we can say "NEVER" or "ALWAYS" in the field of medicine. However, these simple guidelines could put an end to the unnecessary pain and crippling of this potentially "curable" disease. ■


Live. Work. Thrive.
+ The Southeast Permanente Medical Group is a growing, award-winning, multi-specialty group practice of more than 400 primary care and specialty physicians
+ Physician owned and managed
+ Comprehensive benefits, including competitive compensation, malpractice insurance and shareholder opportunity
+ For more information regarding practice opportunities, visit http://www.tspmg.com
KAISER PERMANENTE®
Ricardo D. Moreno MD | Board Certified General Surgeon
Kaiser Permanente Piedmont Specialty Center
Harvard Medical School Graduate
The Southeast Permanente Medical Group, Inc.
The Southeast Permanente Medical Group, Inc. | Nine Piedmont Center
3495 Piedmont Road, NE | Atlanta, GA 30305 | 800.877.0400 | tspmg.com

A Nationally-Recognized Cancer Institute right here at Northside



NORTHSIDE HOSPITAL
CANCER INSTITUTE

The Northside Hospital Cancer Institute is recognized nationally as a leader in cancer diagnosis, treatment and research. In fact, Northside is the only hospital in metro Atlanta chosen to be a Community Cancer Center by the National Cancer Institute. It is a privilege to serve this community, and we are proud to be able to bring you the latest that the world of medicine has to offer. When we say we offer a lifetime of care, we mean a long, long lifetime.

Northside.com | *Where the Extraordinary Happens Every Day*