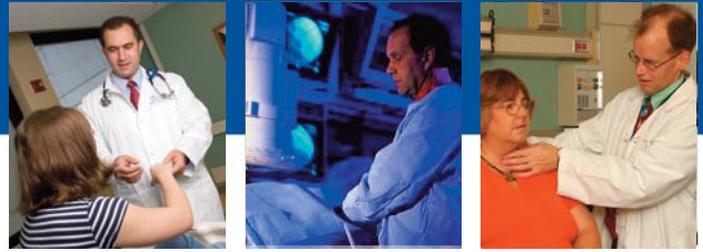


Academic Physician *Quarterly*

A DEPARTMENT OF MEDICINE BULLETIN



UF UNIVERSITY of
FLORIDA
College of Medicine
Jacksonville

FOCUS

Page 2

GME CORNER

Page 4

RX UPDATES

Page 5

NEWS AND NOTES

Page 7

MEET YOUR COLLEAGUES

Page 7

SHANDS BRAND

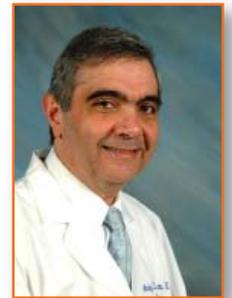
Page 8

CHAIRMAN'S MESSAGE

Dear colleagues:

Once again spring season is upon us. This year after an unseasonably cold winter in Jacksonville, we are all looking forward to warmer days. This is the season of renewed life and hope.

Despite the economic challenges facing the world economies and despite shrinking resources to support research and education, we are determined to forge ahead with innovative programs, creative research and delivery of first rate training to our students and residents.



Clinically, we have started a new musculoskeletal ultrasound unit under the directorship of Dr. Gurjit Kaeley. In this issue he describes the scope of the clinical applications of this technology. This is the first such unit in Northeast Florida and we expect it will generate interest among referring physicians.

Educationally, in January the Department sponsored an exceptionally successful three day conference on new innovations in Internal Medicine. During the proceedings of this conference a special dinner was organized to honor Dr. Malcolm Foster. Many of his past trainees who are currently prominent physicians and scholars in their own right attended this dinner. An education fund in Dr. Foster's name was established to help promote the involvement of young physicians in organized medicine.

Research continues to thrive on our campus and our faculty are successful in attracting research funds and publishing their research findings in high profile journals.

I hope you will find this issue of Academic Quarterly meeting your expectations. If you have any suggestions for improvement please do not hesitate to contact me.

Arshag D. Mooradian, M.D.
Professor of Medicine
Chairman, Department of Medicine



Gurjit Kaeley, M.D.
 Assistant Professor of Medicine
 Director of Musculoskeletal
 Ultrasound Unit
 Division of Rheumatology

Musculoskeletal Ultrasound: The scope of its clinical applications

The use of Musculoskeletal ultrasound has been rapidly expanding among Rheumatologists in the USA. Its appeal stems from the immediate bedside application of this technique after a thorough history and physical has been undertaken. It can be effective in determining the source of a regional pain disorder, detection of an underlying inflammatory disorder as well as injection guidance.^{1,2}

One of the common regional pain disorders seen in the Rheumatology clinic is shoulder pain. Sonography can assist in depicting abnormalities such as supraspinatus tears (fig 1) and subacromial subdeltoid bursitis (fig 2). When compared to MRI, Sonography is reliable in detecting erosions, complete supraspinatus tears as well as detecting synovitis.

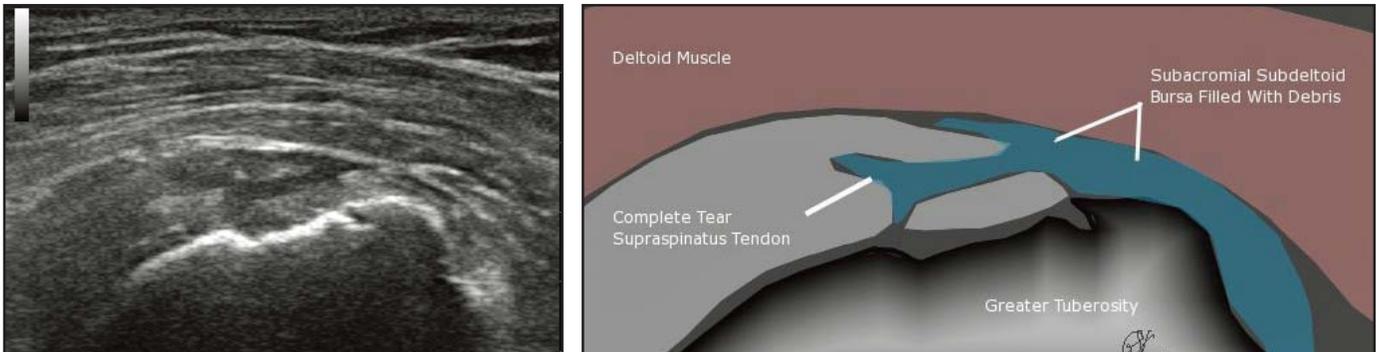


Fig. 1.: B-Mode Longitudinal View And Schematic Of Depicting Complete Tear Of The Supraspinatus Tendon.

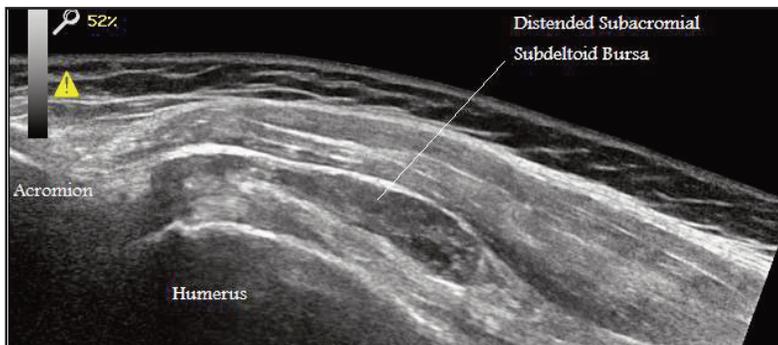
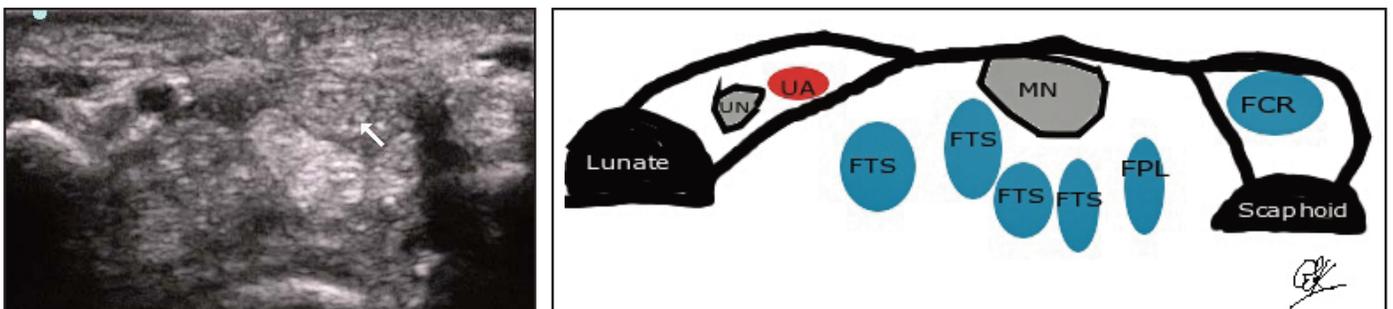


Fig. 2. (Left): Lateral Longitudinal View Revealing Distension of the Subacromial Subdeltoid Bursa

Entrapment neuropathies such as that at the carpal and cubital tunnels may also be readily evaluated. The characteristic pre and post stenotic dilation of the nerve can be demonstrated. (fig 3 and 4)

Fig 3. (Below) B-Mode Transverse view of the carpal tunnel and schematic of corresponding superficial structures. Arrow points to the dilated edematous median nerve (MN).



Continued on Page 3

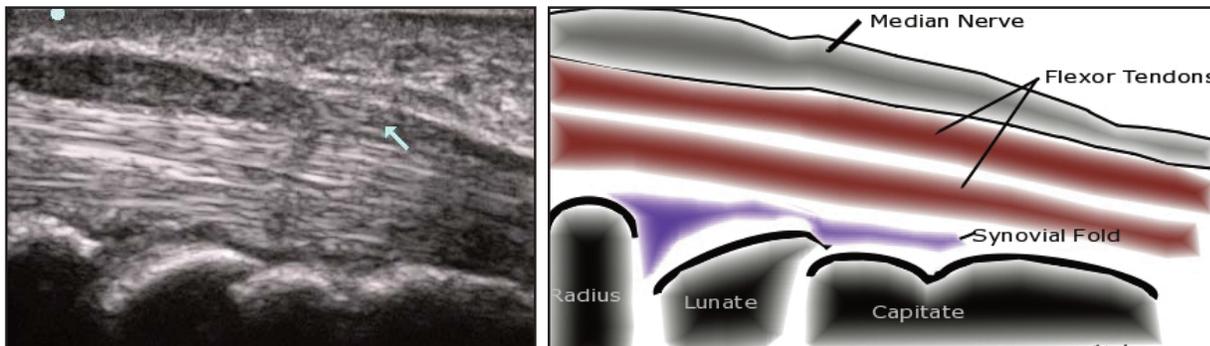


Fig 4. B-Mode Longitudinal view of the carpal tunnel and schematic of corresponding structures. Arrow points to the dilated median nerve.

With the advent of highly effective disease modifying agents, it has become important to detect inflammatory arthropathies early. Several studies have demonstrated that clinical exam underestimates the presence of inflammation. Sonography is more sensitive than radiography in detecting erosions, and has the added bonus of detecting synovial hypertrophy. The activity of the synovial tissue can be inferred by detecting increased blood flow by Power Doppler and does not require administration of contrast unlike MRI. These parameters can be followed longitudinally to determine response to therapy. Recent literature also suggests that tenosynovitis may precede synovitis. Ultrasound has excellent resolution in evaluating tendinous structures. (fig 5)

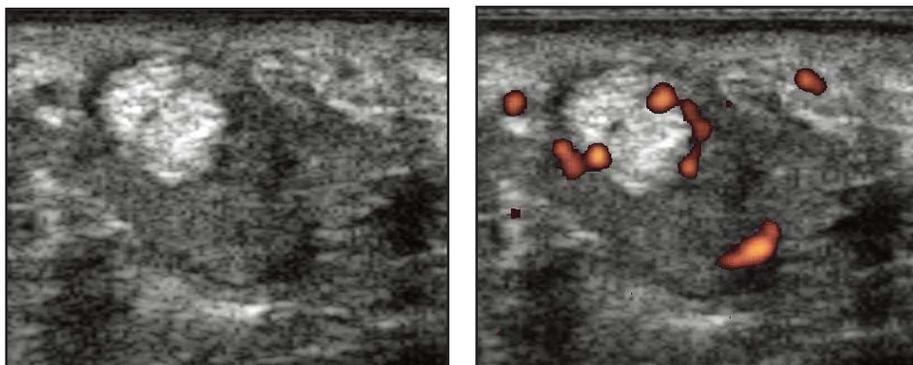


Fig 5. B-Mode And Doppler Transverse Images Of The Right 3rd Proximal Tendon Sheath Revealing Synovial Hypertrophy Within The Sheath With Associated Increase In Vascularity.

Sonography can also be valuable in the evaluation and diagnosis of crystalline arthropathy. In acute arthropathy, sonographic guidance can assist in aspirating small joints such as the 1st MTP. (fig 6)



Fig. 6. 1st MTP Aspiration In A Patient With Acute Podagra - Found To Have Classic Intracellular CPPD Crystals.

Urate crystals deposit on the surface of cartilage whereas CPPD deposits within cartilage. The characteristic deposition of these crystals can be depicted by sonography. In addition, subclinical tophaceous deposits can be demonstrated intra- and extra- articularly. ^{4,5} (fig 7,8)

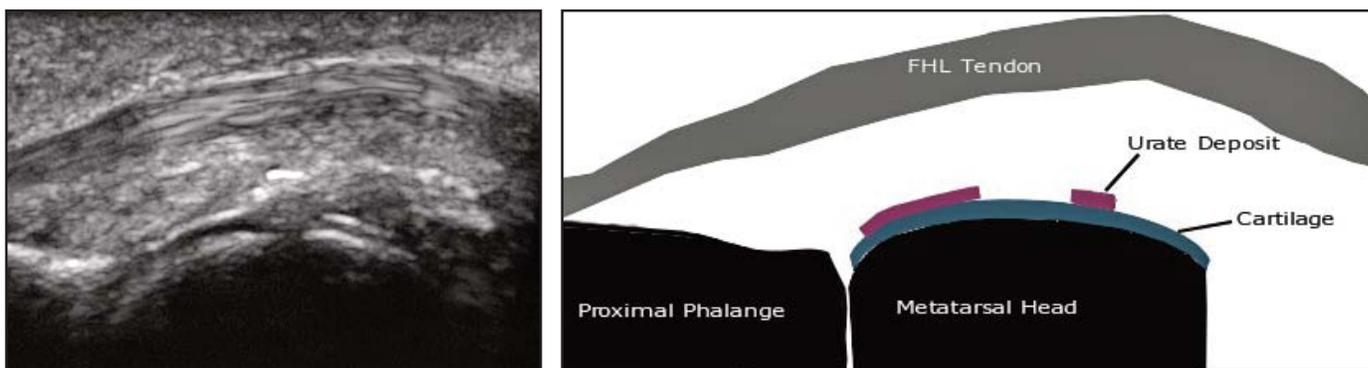


Fig. 7. B-Mode Longitudinal Plantar View and schematic of Urate Deposit on the surface of cartilage depicting the "double contour" sign.

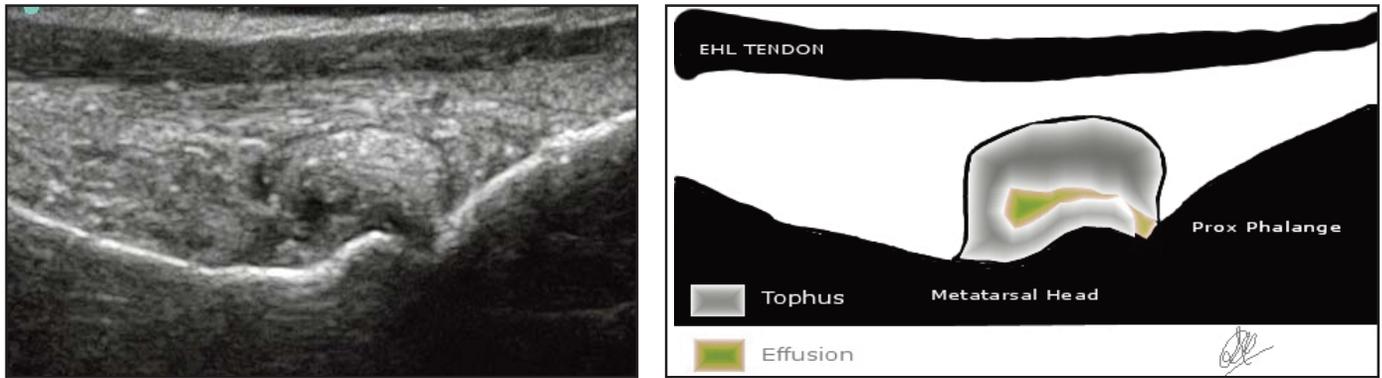


Fig. 8. B-Mode Longitudinal Dorsal View and schematic of Tophaceous Deposit within 1st MTP joint capsule..

Sonographically guided injections may improve outcomes compared to blind injections.⁶⁻⁸ It also offers better patient tolerance, and can be used to place medication with precision in small spaces such as synovial sheaths.

In conclusion, Musculoskeletal Ultrasound offers a powerful extension to the clinical evaluation of a patient. It is helpful in deciding the cause of a regional pain disorder, detecting signs of inflammatory arthropathies and assisting in injection guidance.

1. Grassi W, Filippucci E. Ultrasonography and the rheumatologist. *Current opinion in rheumatology* 2007;19(1):55-60.
2. Kane D, Grassi W, Sturrock R, Balint PV. Musculoskeletal ultrasound—a state of the art review in rheumatology. Part 2: Clinical indications for musculoskeletal ultrasound in rheumatology. *Rheumatology (Oxford)* 2004;43(7):829-38.
3. Bruyn GA, Naredo E, Moller I, et al. Reliability of ultrasonography in detecting shoulder disease in patients with rheumatoid arthritis. *Annals of the rheumatic diseases* 2008;ard.2008.089243.
4. Grassi W, Meenagh G, Pascual E, Filippucci E. "Crystal Clear"—Sonographic Assessment of Gout and Calcium Pyrophosphate Deposition Disease. *Seminars in Arthritis and Rheumatism* 2006;36(3):197-202.
5. Fodor D, Albu A, Gherman C. Crystal-associated synovitis- ultrasonographic feature and clinical correlation. *Ortop Traumatol Rehabil* 2008;10(2):90-102.
6. Chen MJ, Lew HL, Hsu TC, et al. Ultrasound-guided shoulder injections in the treatment of subacromial bursitis. *Am J Phys Med Rehabil* 2006;85(1):31-5.
7. d'Agostino M-A, Xavier Ayril Gabriel Baron Philippe Ravaud Maxime Breban Maxime Dougados. Impact of ultrasound imaging on local corticosteroid injections of symptomatic ankle, hind-, and mid-foot in chronic inflammatory diseases. *Arthritis Care & Research* 2005;53(2):284-92.
8. Naredo E, Cabero F, Beneyto P, et al. A randomized comparative study of short term response to blind injection versus sonographic-guided injection of local corticosteroids in patients with painful shoulder. *The Journal of rheumatology* 2004;31(2):308-14.

GME CORNER



Senthil Meenrajan, M.D., M.B.A.

**Assistant Professor of
Medicine, General Internal
Medicine**

**Associate Program Director,
Internal Medicine Residency**

Great Teachers

A clinical educator might ask himself or herself how and why I am here. This question might elicit a different response from each of us. All the same we all have chosen to be in the academic setting, being teachers and role models for a number of young doctors who are still looking for their mold.

Being physicians in and of itself puts us in a position that requires, at the very least, knowledge, compassion, good communication skills and beyond everything else professionalism. Think about the other role that we have to don, a teacher, and what attributes we need for this. The list is

thankfully not very different from the one we are required to possess already. Only in this role we influence not a few lives, but score of them separated in time and distance, by influencing the doctors that treat them. Think about the opportunity at hand and the responsibility that comes with it.

It is said "The mediocre teacher tells, the good teacher explains, the superior teacher demonstrates, and the great teacher inspires." In this seemingly simplistic statement lies the truth about everything we strive to be in our careers as academic physicians. Just being here makes us mediocre teachers, telling residents what they should and should not do and giving lectures. Some of us actually get to the level of a good teacher, at least explaining to them 'why' things should and should not be done a certain way. Rarely do we find teachers who can live by what they preach and actually teach by example, all the things they want their students to learn. The level that is above all this is when you have students not just following but who want to emulate what you do and be everything that you are and more. This may not be as easy as it sounds. This year I have had plenty of opportunity to hear first hand from a number of medical school graduates during interviews. It is not uncommon to hear they

Continued on Page 5

chose medicine because their medicine attending was so great they wanted to be like him/her as well. It is also not uncommon for students to choose medicine because the attendings in other rotations were terrible! In our own residency we have people choose their subspecialty solely based on their experience with the attending physicians and how they were either positively or negatively impacted by them.

The word 'Guru' brings a number of thoughts to mind. Generally speaking a guru is thought to possess great wisdom, knowledge and authority in a certain field and uses it to inspire others. In Eastern traditions the guru is seen as a conduit for sacred wisdom and guidance, and finding a true guru is often held to be a prerequisite for attaining self-realization. The gurudev is the concept of one's highest consciousness as an inner teacher or intuition within the student. There is a saying that compares teacher and parents to God, since they both 'give life' in a certain way. It is indeed a great privilege to be the life giver for someone who will positively affect so many other lives.

All this privilege brings tremendous responsibility with it and we need to periodically ask ourselves if we are living up to it. In this capacity it is not enough to be mediocre or good or even superior teachers. We have to be absolutely 'GREAT TEACHERS'. We have to be able to inspire our residents, each and every one of them, all the time. Inspiring and getting results has more to do with us than the students and residents themselves. If we can only inspire and motivate 'some' residents, 'some' times and in 'some' settings,

then that speaks more to the quality of the resident than our own abilities as a teacher. The GREAT TEACHER can teach, guide and motivate the weakest in the class to be the best they can be. For this to happen the team (residents, students and other learners) looks for four elements from the teacher - Hope, Trust, Opportunity and Enjoyment. The team needs to know from the teacher that there is hope for them, for their continued improvement and for the progress of the entire team. They need to know they can trust their teacher to be 'available' to them, to 'back' them and know that they will not be 'let down' at the drop of a hat. Trust is always bidirectional. There has to be opportunity - an environment that is conducive to learning, doing new things, asking questions. Finally all of this has to be an enjoyable experience. Residents and students should realize that learning can be an enjoyable experience as well. It does not have to be under situations where they are ridiculed and chastised. High pressure and high stakes do not have to make the experience miserable for the participants. A closely fought football game that goes 'down to the wire' might be stressful for the team and the coach but does not have to be miserable for either. The result is also immaterial in so long as everyone knows that the team did its best and had hope and trust instilled in it by the leader.

In the high stakes environment in which we operate we cannot expect anything less from each one of us. We have the opportunity to affect lives, make them better and create 'GREAT TEACHERS' for future generations.

RX UPDATES



Ronald Mars, M.D.
Associate Professor of Medicine
Division of Nephrology and Hypertension

Treatment of Secondary Hyperparathyroidism

The kidney is a remarkably versatile organ. When diseased the kidney undergoes functional adaptation to maintain the internal milieu within a narrow physiologic range, but often at the expense of maladaptive results elsewhere in the body.

Secondary hyperparathyroidism (s-HPTH) represents just such a circumstance whereby patients with progressive chronic kidney disease (CKD) develop the simultaneous events of hyperphosphatemia, hypocalcemia, vitamin D deficiency, elevated [calcium] x [phosphorous] product ($Ca \times$

P) which together and independently may result in excess production and secretion of parathormone (PTH), or the condition known as s-HPTH. The consequences of s-HPTH may include metabolic bone disease (osteodystrophy), metastatic calcification or calciphylaxis, cardiovascular disease, stimulation of the renin-angiotensin system with difficult to control hypertension, or impaired erythropoiesis and resistant anemia. Successful strategy designed to treat s-HPTH requires an understanding of the pathophysiologic events of progressive CKD.

When patients develop Stage-3 CKD (GFR = 30 - 59 cc/min) or lower, they have reduced secretion of phosphate. The resulting hyperphosphatemia binds ionized calcium, taking it out of solution, causing transient hypocalcemia. Hyperphosphatemia and hypocalcemia independently and together have the ability to stimulate post transcriptional PTH production by increasing and stabilizing PTH mRNA. Notably, the effects of calcium on parathyroid cells is mediated by a cell membrane bound calcium-sensing receptor (CaSR) - a recent discovery on which the use of calcimimetics, CaSR agonists, have been recently introduced.

Simultaneous to developing hyperphosphatemia and hypocalcemia is the impaired renal production of calcitriol [1,25(OH)2D3], the metabolically active form of vitamin D.

Continued on Page 6

The physiologic effects of 1,25(OH)₂D₃ are to normally reduce PTH mRNA levels (and gene transcription) via an intracellular vitamin D receptor (VDR). But, when vitamin D deficiency develops, as in stages 3 – 5 CKD, parathyroid cell proliferation occurs along with increased PTH synthesis. The additional effects of hypocalcemia, resulting from impaired vitamin D intestinal calcium absorption, further enhance PTH production.

It is through an understanding of these pathophysiologic events that has allowed the development of multifactorial strategies to treat s-HPTH.

The treatment of s-HPTH in CKD is, therefore, directed at normalizing phosphorous, calcium and PTH levels. Clinical practice guidelines established by the National Kidney Foundation Kidney Dialysis Outcome Quality Initiative (K/DOQI) are clearly defined for stage 5 CKD (GFR < 15 cc/min, on or off dialysis). At this level of reduced kidney function dietary phosphorous should be restricted to 800 – 1000 mg/day with the goal for phosphorous no > 5.5 mg/dl. For patients receiving either three times weekly hemodialysis, or daily peritoneal dialysis, dietary phosphate restriction alone is generally inadequate to control the positive phosphate balance. Consequently phosphate binders are invariably required. The available phosphate binders in the USA may be either calcium-containing (e.g., calcium carbonate/acetate) or non-calcium-containing (e.g., sevelamer hydrochloride or lanthanum carbonate). The use of aluminum containing phosphate binders is not recommended for long term use because of the risk of aluminum toxicity. The phosphate binders work best when taken with meals to maximize binding of phosphorous in the gut. While calcium carbonate (500 mg elemental calcium in a 1250 mg tablet) and calcium acetate (169 mg elemental calcium in 667 mg tablet) are useful phosphate binders, because of the increasing risk of vascular calcification in stage 5 CKD, the total dose of calcium based phosphate binders should be limited to 1500 mg/day of elemental calcium with a total elemental calcium intake (including diet) not to exceed 2000 mg/day.

The growing concern re: consequences of vascular calcification have now lead to the more popular use of non-calcium-containing sevelamer hydrochloride and lanthanum carbonate. Sevelamer acts as an ion exchange polymer to bind phosphorous in the gut and has the additional benefit of lowering LDL and increasing HDL lipoproteins. These combined effects may account for the fewer arterial calcifications when compared with calcium-containing phosphate binders. Standard sevelamer dosing may range from 800 – 4000 mg TID with meals and/or snacks.

Lanthanum carbonate, like aluminum, is a trivalent compound that chelates dietary phosphate, but has low systemic absorption. It is approximately four times as effective as sevelamer on a dose for dose basis with dosing ranging between 1000 - 1500 mg TID with meals. It is currently marketed as a wafer sized tablet that must either be chewed or crushed and sprinkled on food.

By K/DOQI guidelines, successful use of phosphate binders will lower the (Ca x P) product to $\frac{2}{3}$ 55.

Control of phosphorous and calcium is usually insuffi-

cient to lower PTH in stage 5 CKD to the K/DOQI recommended range of 150 – 300 pg/ml. So treatment with calcitriol or a vitamin D analogue (paricalcitol or doxercalciferol) will be required in the majority of dialysis patients and will suppress PTH production by binding to the VDR in parathyroid tissue. Vitamin D analogues, available in oral or IV form, may be more advantageous than calcitriol because of a greater PTH suppression and a lower risk for hypercalcemia. While the desired range of PTH in dialysis patients (150 – 300 pg/ml) exceeds by 2 – 3 fold the upper limit of normal, the risk for adynamic bone disease is low when PTH does not fall < 150 pg/ml. Not to be trivialized are the other novel biologic activities of vitamin D that directly or indirectly may affect cardiovascular function, blood pressure, immune and neurologic function. These too are positively impacted by use of calcitriol/analogues with data showing improved morbidity and mortality in treated patients.

Calcimimetics are an emerging class of calcium receptor-sensing agonists that modulate the activity of the calcium sensing surface receptor (CaSR) by allosterically increasing the sensitivity of the receptor to calcium. Available in the USA as cinacalcet HCl, treatment is generally reserved for stage 5 CKD and can profoundly lower PTH without elevating serum calcium or phosphorous. Although the cinacalcet may provide an estimated 30 – 40% drop in PTH levels, its greatest benefit is when used in combination with phosphate binders and vitamin D/anologue therapy. Monitoring for hypocalcemia (< 8.5 mg/dl) is critical and may require further adjustment of either the calcimimetic and/or vitamin D/anologue.

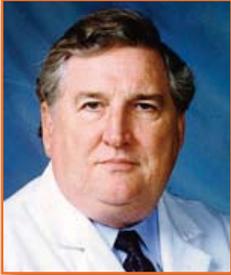
While subtotal (7/8th) parathyroidectomy with autotransplant of 1/8th gland to the subcutaneous forearm is an absolute strategy to reduce PTH levels, this is rarely indicated for s-HPTH and generally reserved for patients with tertiary hyperparathyroidism and symptoms of refractory bone pain or pruritis. Post parathyroidectomy the risk for symptomatic hypocalcemia is high for patients demonstrating the “hungry bone” syndrome. In this situation, phosphate binders are withheld, dietary calcium and phosphorous are liberalized, large doses of IV calcium may be required (especially if paresthesiae and neuromuscular activity spontaneously increases) and calcitriol may be indicated to enhance intestinal absorption of calcium.

Treating patients with stage 3 – 4 CKD, not yet on dialysis, should focus on prevention of parathyroid gland hyperplasia by way of phosphate restriction, phosphate binder and calcium supplementation as needed. When to start treatment with calcimimetics or active vitamin D/analogues has not been firmly established but should generally be used in the later stages of their disease.

Summary:

s-HPTH begins as early as stage 3 CKD. It is a progressive condition and becomes full blown as patients reach stage 5 CKD. Successful treatment outcomes rely on the therapeutic combination of phosphate binders, calcitriol or vitamin D analogues, and calcimimetics.

Establishment of a fund honoring Dr. Malcolm Foster



Dr. Malcolm T. Foster, Jr. was honored for his contributions to medicine at a dinner at Amelia Island Plantation on Friday, January 23, 2009. The dinner was held in conjunction with the University of Florida College of Medicine's Department of Medicine annual Update in Internal Medicine. Over 80 people came to celebrate Dr. Foster's contributions to medicine and in many cases to their professional and personal growth. Twelve of Dr. Foster's previous chief residents during his tenure as Chairman of the Department of Medicine at the University of Florida-Jacksonville were in attendance and had the opportunity to renew old acquaintances and make new ones. Based on the wonderful turnout and the comments made, it is evident that Doctor Foster

has touched the lives of many individuals and continues to do so.

To recognize Doctor Foster's many contributions to medicine, a scholarship fund in his name has been established through the University of Florida Foundation. Annually, a scholarship will be awarded to a deserving resident in Internal Medicine to attend the annual meeting of the American College of Physicians or another scholarly endeavor. If you would like to honor Doctor Foster by making a contribution to the fund, please contact the Office of Development and Alumni Affairs of the College of Medicine-Jacksonville at 904-244-1062.

Diabetes & Endocrinology Update 2009

Presented by the Department of Medicine and Division of Endocrinology, Diabetes & Metabolism

Date: Saturday, May 2, 2009

Time: 7:30am - 4:00pm

Location: Hyatt Regency Jacksonville Riverfront

This second annual event will showcase current and emerging trends in Endocrinology with practical recommendations and guidelines from clinical experts. Target audience includes primary care physicians and allied healthcare providers.

For more information, please contact Barbara Jones at 904-244-2380 or barbara.jones@jax.ufl.edu



MEET YOUR COLLEAGUES

Editor's note: Periodically the "Academic Physician Quarterly" will introduce our readership to new faculty members who have exceptional clinical skills. In this issue we highlight a new member of the Division of Cardiology who will also serve as Assistant Program Director of the Electrophysiology Fellowship Program.



**Daniel Katz, M.D., Assistant Professor of Medicine, Division of Cardiology
Assistant Program Director for the Electrophysiology Fellowship Program**

Dr. Katz earned his medical degree from the University of Medicine and Dentistry of New Jersey. He completed his residency in Internal Medicine and his fellowship in Critical Care Medicine at Brown University and his fellowships in Cardiovascular Disease and Clinical Cardiac Electrophysiology at Albert Einstein College of Medicine of Yeshiva University. Dr. Katz is board certified in Internal Medicine, Cardiovascular Disease and Clinical Cardiac Electrophysiology.

Stroke Program Receives High Marks During Recertification

The Shands Jacksonville Comprehensive Stroke Program successfully completed its second recertification as a Primary Stroke Center by the Joint Commission in November. Shands Jacksonville is also certified as a Comprehensive Stroke Center by the Florida Agency for Health Care Administration.

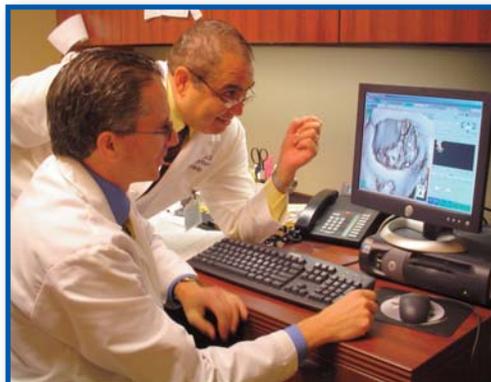
The hospital is one of only two Comprehensive Stroke Centers in North Florida. The successful review noted that there were no deficiencies or need for follow-up. The stroke center exceeded the national average in 2008 with 100 percent compliance in eight out of 10 measures.

The Neuroscience Institute received positive comments on its community and EMS educational initiatives. UF physicians are involved in multiple research projects to expand and improve care for stroke victims. Research is also underway on the effectiveness of traditional stroke education to post stroke patients who are likely to be cognitively impaired during hospitalization.

Shands Jacksonville currently sees 50 to 80 new stroke patients each month. With the help of TraumaOne flight services, Shands has expanded the coverage area of the stroke center to include rural areas surrounding

Jacksonville. Patients have successfully received the disability reducing treatment tPA after being transported from as far away as 100 miles.

The window for IV tPA has expanded from three



hours to four-and-a-half hours, enabling the stroke team to effectively treat patients who did not identify symptoms early or

who have come from farther distances. Additionally, the measure for DVT prophylaxis will expand from DVT prophylaxis considered, to DVT prophylaxis being considered, ordered and administered.

Call Scott Silliman, MD, UF associate professor of neurology, or Nader Antonios, MD, UF associate professor of neurology, at 244-3960 if you have questions about treatments or education programs available to your patients.

UF UNIVERSITY of
FLORIDA

College of Medicine

Jacksonville

653-1 West Eighth St.

Department of Medicine

Jacksonville, FL 32209-6511

904-244-8846; fax: 904-244-8844

NON-PROFIT ORG.
U.S. POSTAGE
PAID
JACKSONVILLE, FL
PERMIT NO. P173