Athens Kidney Center
1440 North Chase Street
Athens, GA 30601

RETURN SERVICE REQUESTED

THE KIDNEYS OF BLACK FOLKS

There are several theories why Black kidneys are particularly prone to disease and failure (see the August edition of Second Opinion). Now comes data indicating that renal risk is extended to those altruistic Black souls who offer their kidney to save another's life. Using retrospective medical insurance data, Lentine et al, N Engl J Med, 2010, find that compared to their white peers, Black (and Hispanic) kidney donors had a 1.5x higher risk of arterial hypertension, 2.3x risk of developing drug-requiring type 2 diabetes mellitus, and 2.3x higher risk of future CKD. Strikingly, this appears to be par for the course, even without kidney donation: only arterial hypertension appears to be a specific result of unilateral renal loss, being higher in kidney donors than in the general population. Moral: minorities should not be discouraged from kidney donation, but we are obliged to follow their post-donation metabolic status much more closely.

BALANCED POLYMORPHISM IN KIDNEY DISEASE

In an intriguing study published by Genovese et al, Science, 2010, patients of African ancestry who commonly carry a variant of the apoplipoprotein L1 gene (called APOL1), a normal component of HDL cholesterol as well as mediator of autophagy (a cell-cycling process) are at higher risk (up to 10.5x) for developing serious kidney disease. It appears that this gene variant has been selected out through generations, as it confers protection against sleeping sickness, carried by the parasite Trypanosoma brucei. By avoiding potentially life-threatening parasitic infection, patients of African ancestry pay a price of higher rates of kidney damage. Interestingly, the parasite is slowly striking back: the new East African species of parasite, T. brucei var rhodesiense, has a mutation enabling it to overcome the cytocidal effect of APOL1. This scenario is reminiscent of sickle cell gene which protects against malaria at the high cost of chronic hemolytic anemia.

DEPARTMENT OF DANGEROUS DRUG COMBINATIONS

Periodically, this section will highlight some commonly used drugs that could, in combination, result in potentially dangerous adverse effects. By highlighting some of these potential problems, we hope to lessen the risk of therapeutic misadventure in the office or at your clinic.

Protease inhibitors (used as anti-retroviral therapy) + cytochrome CYP3A4 isoenzyme inducers: PIs block and are metabolized by isoenzyme, so inducers (anti-epileptics, smoking, dexamethasone) can lead to drop in serum levels of PI drugs.

SSRI antidepressants + monoamine oxidase inhibitors + cocaine + Ultram/Tramadol + MDMA/Ecstacy: all can singly or in combination elicit the serotonin syndrome.

Coumadin + Bactrim: excessive bleeding risk, possibly from altered GI flora by Bactrim and loss of endogenous vitamin K synthesis.

Herbal remedies: hawthorn can provoke bradycardia in digitalized patients; garlic is potentially hypoglycemic in treated diabetics; ginseng can worsen coumadin-associated bleeding risk.

ONE FOR THE PALLIATIVE CARE TEAM

Palliative care in the right hands and well administered, can be a saving grace. Common practice is to institute palliative care as a last resort, after conventional treatments have been exhausted. A new study of people with metastatic non-small cell cancer of lungs suggest that candidates who began palliative care at the same time as cancer diagnosis did better on Quality of Life indices and also had overall less aggressive care compared to those who only received palliative care after exhausting all other aggressive treatment for cancer (Temel et al, N Engl J Med, 2010).

STAGING NON-SMALL CELL LUNG CANCER: SURGICAL VS MEDICAL APPROACH

Node sampling is requisite for staging non-small cell lung cancer. The gold standard has historically been surgical, using cervical mediastinoscopy, which has a high diagnostic yield, 2% morbidity, 0.08% mortality, and provides surgical access to 4 out of 9 lymph node stations (i.e. highest mediastinal node group, upper paratrachael node group, lower paratrachael node group including the azygos nodes, and anterior compartment of the subcarinal node group)- but not to the aortic (sub-aortic/para-aortic) and most of the inferior mediastinal (posterior compartment of the subcarinal, as well as the paraesophageal and pulmonary ligament node groups). The advent of outpatient bronchoscopic and endoscopic procedures, such as endobronchial U/S-guided fine needle aspiration (EBUS-FNA) and endoscopic ultrasound-guided fine needle aspiration (EUS-FNA) has challenged the primacy of mediastinoscopy. Both EBUS-FNA and EUS-FNA provide real-time U/S guidance for mediastinal node sampling to the same anatomical extent as mediastinoscopy (including the ability to occasionally sample retro-trachael nodes), being even more extensive with EUS-FNA, which can additionally sample all of the inferior mediastinum (subcarinal, paraesophageal and pulmonary ligament) as well as subaortic node groups, areas that are not accessed by either mediastinoscopy or EBUS-FNA. Also, both EBUS-FNA and EUS-FNA have comparable diagnostic yields as mediastinoscopy, and though there are no head-to-head studies comparing morbidity/mortality, it is virtually certain that complications resulting from EBUS-FNA and/or EUS-FNA are less than those associated with mediastinoscopy. In a study of 66 patients with known or suspected non-small cell cancer of the lungs, Ernst et al, J Thorac Oncol, 2008, found EBUS-FNA had a higher overall yield (91%) compared to mediastinoscopy (78%) in per lymph node analysis. There was disagreement in yield between both procedures in subcarinal lymph node analysis, favoring EBUS-FNA; the difference in yield at other sites was not significant. Sensitivity, specificity and negative predictive value for EBUS-FNA were 87%, 100%, 78% and 68%, 100% and 59% for mediastinoscopy; there were no significant differences between the 2 methods in determining true pathologic stage. In another study by the same group, lymph nodes measuring 0.5-1.0 cm were studied in non-small cell cancer patients with radiologically non-enlarged mediastinal and hilar nodes, where the negative predictive value of EBUS-FNA for detecting malignancy was 96.3% with no false negative biopsies of true mediastinal nodes (Ernst et al, Eur Respir J, 2006). Another study of 138 patients using a combination of EBUS-FNA and EUS-FNA performed under conscious sedation in an average time of 52 minutes for lymph node staging, showed a specificity of 100%, sensitivity of 97%, missing only 1 node located anterior to the aortic arch; there were no significant complications. Therefore, EBUS-FNA is at least equal to, or better than, cervical mediastinoscopy for nodal staging, whilst the combination of EBUS-FNA plus EUS-FNA appears to be superior. Factors related to safety, cost-effectiveness, negative predictive value and operator variability have not yet been systematically studies in this regard. Contributed by Milos Tucakovic, MD (Pulmonary & Critical Care Medicine)

the SECOND OPINION

October 2010 *A monthly medical newsletter for the Athens medical community* Volume 1, #10

FROM THE EDITOR

Appearances notwithstanding, Stephen Hawking, the 17th Lucasian professor of mathematics at Cambridge, is no atheist. A worthy heir in the intellectual tradition of previous Lucasian professors, such as Charles Babbage, the grandfather of computers, and Sir Isaac Newton, the granddaddy of everything else, he now faces an inquisition of sorts. This contretemps was triggered by his recent tome, "The Big Bang", a layman's exposition on the Big Bang, the fons et origo of creation itself. Prior to those celestial fireworks of 13.7 billion years ago, all matter was packed into an infinitesimal dot, with neither space, dimension, time nor texture. Ever since, the universe has gradually expanded to encompass our world, and presumably, the world to come. Hawking's heresy has to do with his new account of the Big Bang: first, there wasn't just 1 big bang, but several; we do not have a unique universe, but an infinite multiverse; and, adding incendiary salt to ecclesiastical injury, the miracle of creation could perfectly be explained solely by the laws of physics, with no need to invoke a creator.

The ensuing reverberations have been felt all the way from Cambridge to Kandahar. Channeling the spirit of LaPlace, whose experiments did not, as he patiently explained to Napoleon, require the invocation of a creator, Hawking has stood his ground. As his science falls outside the literal prescription of Genesis, the devoutly pious have effectively dubbed him a heretic. As with any contrived marriage, the union of religion and science- the ecumenical orgy notwithstanding- is never pleasant. Each bears its own pedigree: science on experimentation and proof, laying no claims to absolutism; religion on faith, conviction and virtue, expressing only contempt for doubt. But neither tradition should preclude intelligent thought; the antithesis of knowledge is savagery, not religion. Indeed, religion, which is a search for meaning, cannot be based on superstition and illogic.

Even more to the point, the absence of evidence of a creator cannot be construed as evidence of the absence of an enabling force. En arche en ho logos. In the beginning was the word, knowledge, spirit. Within that doctrinal statement of Biblical exegesis lies insight into the mystery of creation. Of course, part of the problem is the popular conception of God as man-in-the-sky, depicted in medieval lithography as a greying, hirsute, somewhat obese, Caucasian male sitting on a throne in the skies. If that isn't apostasy, I don't know what is. Having remade God in man's image, little wonder the piously devout feel constrained to defend his honor at every turn. God, as St. Augustine wrote in his deeply introspective book, The Confessions, has no need for our lies. I'll see you Friday lunch-time, at the CME lounge.

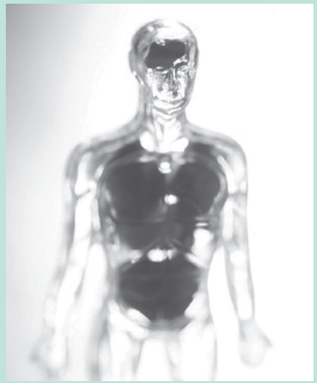
Beze Adogu, MD, PhD, FACP

TREATING HYPOTENSION

The definition of what constitutes hypotension is controversial, but we all accept that a fall in ambient BP of >20 mmHg systolic or >10 mmHg diastolic, particularly if associated with signs/symptoms of cerebral hypoperfusion (dizziness, fatigue, confusion, headache, syncope, visual obscurations, etc) or hemodynamic compensation from sympatho-adrenal discharge (palpitations, tachycardia, cold extremities, angina, tremors, nausea, etc) is pathologic. Etiology includes neurogenic failure (autonomic dysfunction, either in isolation or associated with systemic disease), hypovolemia (from severe dehydration or blood loss), end-organ failure (ablation of the afferent/effector loop of the baroreceptor reflex, secondary to cardiac failure/aortic stenosis, adrenal disease, systemic vasodilation/venous blood pooling) or iatrogenic (from prescription pills). The common iatrogenic causes are diuretics, neuroleptics/anti-psychotics, anti-depressants, narcotics, nitrates, anti-hypertensives and anti-parkinsonism medications). Less commonly, autonomic dysfunction may result from chronic exposure to vinca alkaloids, cis-platin, insulin and amiodarone. Treatment depends on cause: correct the initiating factor, if possible; remove any offending drugs; correct volume deficits; treat any fevers; avoid heavy carbohydrate meals and alcohol ingestion; institute regular, low-grade physical activity; avoid heat exposure and straining whilst standing (Valsalva); abdominal compression with corsets; elevate head of bed at night (avoids nocturesis); consider medications, if intractable. Commonly used medications are pyridostigmine 30-60 mg p.o. TID (neurogenic causes), fludrocortisone 0.1-0.6 mg p.o. q daily (for volume-related causes) and midodrine at 5-10 mg p.o. TID (for neurogenic causes, but effectiveness has become recently controversial).

LEST WE FORGET

There is an endogenous digoxin-like substance in the sera of (CKD) renal patients, leading to substantial variation in detected levels of "serum digoxin" using different assays based on radioimmunoassay methodology (Steven Graves et al, Annals Intern Medicine, 1983). This poorly identified substance in uremic sera leads to false-positive digoxin levels in sera, and potentially compromises the interpretation of routine digoxin assays in renal disease.



Contents Within:

Editor:
Beze Adogu, MD, PhD, FACP

Associate Editors:
Khudr Burjak, MD & Harini Chittineni, MD

Athens Kidney Center
1440 North Chase St • Athens, GA 30601
706-227-2110 (p)
706-227-2116 (f)
www.athenskidneycenter.com

DRIVE-BY RENAL BIOPSIES: HOW LONG IS LONG ENOUGH?

Financial and scheduling pressures are forcing same-day percutaneous renal biopsies, with some assurance from historical reports of patient safety (Alebiosu & Kadiri, J Natl Med Assoc, 2004; Maya & Allon, Semin Dial, 2009). With adoption of modern biopsy techniques, it appears that complication rates have dramatically improved, with absence of any delayed complication reported by Carrington et al, Nephrol Dial Transplantation, 2010. But even in countries with more centralized medical systems, such as France, there are wide variations in unit post-biopsy protocols: only 4% of all units routinely discharge patients on the same day after native-kidney biopsies, and 28% effect a same-day discharge after allograft biopsies (Bollee et al, Nephrol Dial Transplant, 2010). The most important complication of renal biopsy is serious bleeding in 3-12% of procedures, which in rare cases might lead to emergency nephrectomy or death. Bleeding is more likely in patients with advanced renal disease, uncontrolled arterial hypertension or renal amyloidosis. Total biopsy-associated mortality is estimated at 0.07%. Data from Maya & Allon suggests that patients can be safely discharged 8 hours post-biopsy, whilst a larger study from Rush-Presbyterian had indicated that as only 2/3 of major complications are evident within 8 hours of biopsy, with 89% being detectable by 12 hours and 91% by 24 hours respectively (Whittier & Korbet, J American Soc Nephrol, 2004), in low risk patients without known bleeding disorders, post-biopsy surveillance should be 12 hours at least. Based on more recent studies, the pressure towards even shorter observation times are unlikely to relent.

UNDERSTANDING ERECTILE DYSFUNCTION

ED is a failure of neurovascular competence, involving parasympathetic nerve dysfunction and/or failure of carvernous endothelial relaxation primed by nitric oxide release, which may be the end-result of localized (such as Peyronie's disease, prostatitis, urethral disease), neurologic (e.g. paraplegia, multiple sclerosis, stroke), vascular, hormonal (e.g. hypogonadism, insulin resistance, hyperprolactinemia), psychological or systemic disease. At least 75% of ED result from organic disease, and 15-25% are either psychogenic or "idiopathic" in origin. Common underlying medical diseases include diabetes mellitus (which increases the odds of developing ED by 4), prostatic disease (increases odds by 2.9), peripheral artery disease (odds 2.6), and arterial hypertension, cardiac disease, obesity, smoking and hyperlipidemia (each independently increasing the odds by ~1.7). Indeed, these common medical diseases predict ED: 85% of patients with non-cardiac peripheral artery disease have ED. Conversely, ED predicts present or future coronary artery disease typically within 5 years of onset (Montorsi et al, Int J Impot Research, 2002): erectile failure, which in relative terms requires an equivalent surge of blood flow as needed for coronary perfusion in moderate exercise, is as powerful in signaling future cardiovascular disease as a personal history of current smoking or a family history of early MI. In other words, ED is as potent as exercise intolerance in a patient's history; both are primarily from cardiovascular disease, but either may be related to non-vascular factors too. Therefore, every case of ED ought to be screened for underlying CVS disease as well as previously undiagnosed CV risk factors (Roumeguere et al, European J Urol, 2003) especially dyslipidemia and diabetes mellitus, either of which are present in 70% of all cases, with previously undiagnosed cases found on routine screening in at least 25% of newly diagnosed ED patients. Treatment of ED should parallel treatment of cardiac (risk factors): what helps 1, assists the other.

NEPHROGENIC SYSTEMIC FIBROSIS

This autoimmune connective tissue condition only occurs with kidney disease, showing no age, sex or gender bias. Probability of disease is strongly linked to exposure to gadolinium contrast dye used for MRI as well as severity of kidney failure (worse in ESRD), with weaker links to modality of renal treatment (more common in peritoneal dialysis) and possibly, Procrit or IV iron treatment. Patients develop a characteristic darkened, scaling, hardened, stiff, tightening of the skin which is typically associated with distal skin blotches (non-truncal telangiectasias), sclerodactyly (with loss of digital pulp) and systemic fibrosis of lungs, muscle and viscera. Differential diagnosis include scleroderma (positive ANA, positive Raynaud's reaction), eosinophilic fasciitis (blood and tissue eosinophilia, sparing of digits), scleromyxedema (blood and tissue plasmacytosis, monoclonal gammopathy, mucin-rich papular eruption of head/digits) and porphyria cutanea tarda (elevated uroporphyrin levels). Treatment is based on prevention: avoid gadolinium where serum creatinine is above 1.5 mg/dL, and if there are no viable options, consider the use of low-dose ProHance (gadoteridol, a macrocyclic gadolinium chelate) followed by immediate post-exposure hemodialysis.

SEPTIC SHOCK

Treatment of septic shock, as with therapy for many acute disease processes is time-driven: consider "the STEMI" (ST elevation acute myocardial infarction), acute ischemic strokes, acute kidney injury or acute poisoning. Treatment protocols for STEMI are designed to cut door-angioplasty times to the barest minimum: time is (heart) muscle- or (brain) neurone, or (kidney) nephron. Both mortality and morbidity are dramatically reduced with timely intervention. In septic shock, timing is also everything. Survival worsens with delay of antibiotic therapy, survival declining by the minute. (Kumar et al, Crit Care Med, 2006). Septic shock is responsible for an estimated 225,000 deaths in the US yearly, a number similar to deaths attributed to acute myocardial infarction. Early recognition of sepsis can be challenging, but its increasing frequency has driven the development of ICU protocols aimed at improving survival through evidence-based medicine. The ongoing Surviving Sepsis Campaign highlights these difficulties and also heightens our awareness of this common, potentially fatal disorder (Dellinger et al, Crit Care Med, 2008). Septic shock describes a systemic condition, with or without evidence of end-organ damage and/or systemic hypo-perfusion due to infectious agents or their toxins. Early treatment goals include appropriate identification of bacterial source or infection portal with removal of same where possible (e.g. indwelling vascular catheter), rapid antibiotic initiation based on probable cause, and reversal of visceral hypo perfusion with fluids and appropriate vasopressors. The Surviving Sepsis Campaign identifies 6 cardinal treatment goals in 6 hours: (1) obtain cultures, (2) start antibiotics, (3) fluid resuscitation, (4) monitor CVP, (5) measure lactate levels, and (6) assess mixed venous oxygen saturation. Rapid volume replacement is encouraged until an adequate CVP is established (>12 cm H20), after which fluid volume can be safely reduced, and blood pressures maintained with pressor agents. The physiologic goals are a mean arterial BP >65 mmHg, urine output >0.5mL/kg, and a mixed venous oxygen saturation of >70%. The vasopressor agent of choice is norepinephrine, and in some instances, dopamine. Steroids are indicated only for systemic BP unresponsive to IV pressor agents. Ambient blood glucose should be <150 mg/dL. DVT prophylaxis with SQ LMWH as well as anti-ulcer prophylaxis with H2-blockers should be instituted; daily antibiotic review with appropriate changes dictated by culture results is crucial. Patients with respiratory failure requiring intubation are best managed with a tidal volume of 6 mL/kg, plateau pressure <30 torr, and PEEP adjusted with ambient oxygenation requirements. There is evidence that intermittent or continuous sedation with daily interruption and spontaneous breathing trials reduce the duration of ventilator-dependence. The challenge for the physician is to recognize the septic patient and then institute timely management. As our population ages, treatment becomes more invasive, and survival from serious illness more common, the incidence of sepsis will expectedly rise in coming years, and treatment will continuously evolve both here and elsewhere: stay tuned. Contributed by Hugh Jenkins, MD (Pulmonary and Critical Care Medicine)

PROPER TREATMENT OF CALCIPHYLAXIS

1. Aggressive local wound care, including tissue debridement, and where necessary, vacuum device dressing, autologous split-skin grafting and amputation.
2. Correction of hyperparathyroidism with oral Sensipar or surgical parathyroidectomy.
3. Optimization of calcium x phosphorus product: intensive low-calcium bath hemodialysis, oral non-calcium phosphate binders (Fosrenol or Renvela), proscription of vitamin D products, reduced calcium intake, IV or oral biphosphonates (Aredia, Didronel or Boniva).
4. Soft tissue calcium chelation with sodium thiosulfate, given at 5-25g IV or intra-peritoneally during dialysis treatments; major side-effect is nausea.
5. Hyperbaric oxygen treatment, vitamin K supplements (if calciphylaxis was triggered by coumadin treatment), avoidance of oral anticoagulants, weight loss, proscription of steroid use, avoidance of soft tissue trauma or systemic hypotension.

UNRESPONSIVENESS TO ANTI-PLATELET DRUGS

Recurrent ischemic cardiac events in properly treated patients are largely attrributed to poor responsiveness to asnti-platelet drugs, though disease progression remains a plausible alternative explanation. A recent French study suggests that patients who might be resistant to dual aspirin-Plavix combinations could be clinically predicted based on the presence of diabetes mellitus, obesity and high CRP levels. The obvious suggestion is to use higher doses of aspirin >160 mg/day or perhaps, a triple coumadin-aspirin-Plavix combination as is sometimes deployed for atrial fibrillation (Fontana et al, J Thromb Haemost, 2010).

HOSPITAL ACQUIRED INFECTIONS

The days of hospital-associated infections are about to be numbered -by official mandate. What really will happen is that hospitals will hemorrhage money through uncompensated medical care delivery for infections that occur within their domain. The rationale for this measure is that CMS has finally realized the huge cost (and extended hospital stay) directly associated with nosocomial infections, and more importantly, that infection rates show significant country-specific and hospital-specific patterns and incidence, with clear evidence that hospital-triggered initiatives can substantially reduce the incidence of those infections. And nosocomial infections are a (Joe Bidenesque expletive deleted) big deal: 65% of hospital deaths are caused or associated with nosocomial infections, mostly pneumonia, catheter-related blood stream infections, urinary tract infections and localized surgical site infections. Nosocomial infections rate is primed to overtake cardiac indicators as the #1 index for institutional grading of hospitals. Risk factors for nosocomial infections include host-defense failure (loss of skin/mucosal integrity, immuno-compromise, malnutrition, indwelling devices favoring colonization, underlying medical disease), pathogen-related aggression (drug-resistant organisms) and a favorable environment (fomite spread, healthcare provider as vector, airborne/water transmission). Management should focus on modifiable risk factors: contact isolation where necessary; scrupulous hand hygiene; aspiration precautions; oral antiseptic irrigation (reduces colonization); adequate glycemic control; stress ulcer prophylaxis; restricted use of leucocyte-poor RBC transfusions; avoidance of parenteral nutrition (with enteral feeds as preferred choice); avoidance of nasotrachael intubation or nasogastric lines; avoidance of promiscuous antibiotic prophylaxis; DVT prophylaxis; limited sedation and daily interruption protocol; continuous or intermittent aspiration of oral/subglottic secretions; strict asepsis during catheter care/manipulation; use of dedicated "catheter care team"; avoidance of decubiti ulcers.

CHOLESTEROL EMBOLIZATION SYNDROME

Iatrogenic (following vascular instrumentation or initiation of anticoagulation) or spontaneous disruption of atherosclerotic plaques often lead to embolization of cholesterol crystals as showers into the circulation. Clinical effects include constitutional symptoms, inflammatory response syndrome (fever, leucocytosis, high ESR, low complement levels), eosinophilia, acute kidney failure, digital infarcts (classically, as "blue toe" syndrome), intestinal ischemia (manifest as GI bleed), livedo reticularis of skin, Hollenhurst plaques of retina (clinically, retinal infarcts present as amaurosis fugax), brain infarcts (most common destination of emboli, manifesting as diffuse encephalopathy, e.g. confusion or amnesia, rarely as focal deficits) and acute pancreatitis. Treatment with anticoagulants is very controversial, but supporting data for either opinion is limited.

STATINS FOR LUNG INJURY?

The pleiotropic effects of statins (i.e. those beneficial properties which are not readily explained through its lipid-lowering effects) are multiple and often attributed to their anti-inflammatory qualities. So far, there is no effective drug treatment for acute lung injury. A new randomized, double-blinded, placebo-controlled study suggests that statins may have a modest effect in treating this deadly illness, and that such clinical use is relatively safe (Craig et al, American J Resp Crit Care Med, 2010). Interestingly, another study examined statin effects in asthma and concluded that whilst there might be some minor symptomatic and functional improvement with simvastatin, it did not exhibit any steroid-sparing effects (Cowan et al, Thorax, 2010).

BEWARE THE OVERTIME PAY

America is the land of workaholics, overtime pay, and short vacations. Now comes data from England (now, that's a surprise!) that overtime work is probably not good for you. Tracking British civil servants over 11 years, it was shown that 3-4 hours of overtime work per day was associated with a 1.6x higher risk of later development of coronary heart disease (Virtanen et al, European Heart J, 2010). Moral: you might not live long enough to enjoy the overtime paycheck, especially if you are a British civil servant.

MORTALITY WITH PPIS IN THE AGED

An observational study from Finland indicates that PPI use was associated with higher all-cause mortality in institutionalized elderly (Bell et al, Arch Intern Med, 2010). The study did not provide any answers for this finding, but reiterates previously described associations of PPI use with Plavix antagonism, bacterial pneumonia, C. difficile enterocolitis and bone fractures. Should we return to H2 blockers as anti-ulcer treatment of choice in vulnerable populations?

THE AGE OF CPOE

There are 50 billion reasons to use CPOE (computerized physician order entry): that is how much government is investing in information technology, the bulk of which is expected to go to hospitals (Bates DW, Arch Intern Med, 2010). Consequently, CPOE will become mandatory at ARMC from 10.10.10. But there are problems: physicians are reluctant users; clinical alerts are often non-specific and non-tailored to "real-life" situations; practitioners tend to "over-ride" red-flag alerts during routine use; the learning curve is steep; potability of systems is impractical (each hospital has its own format); compliance is time-intensive; and there is some data that age is often a barrier to proficiency. All these snafus can be corrected, though unintended consequences are rife. A new study by Strom et al, Arch Intern Med, 2010, shows that when "over-ride" prompts cannot be ignored by doctors, potentially fatal treatment delays may result. Moral: you can take a horse to water, but you can't force it to drink.

DELIRIUM: NOW WE GET IT

Delirium a.k.a. ICU psychosis is common, under-diagnosed, and serious. A new study (Witlox et al, JAMA, 2010) meta-analyzed 21 previous papers on the subject amongst the elderly. The results were startling: 1.4x risk of death in succeeding 2 years, 3x risk of institutionalization over next 1 year; 8x risk of dementia in following 4 years compared to age-matched controls without delirium. Moral: dementia must be prevented (see last month's edition of Second Opinion). For debate: is delirium the actual trigger for future dementia, or does it mark a pre-dementia state amongst the elderly?

DEMENTIA: CLUES TO VASCULAR ORIGIN

Cognitive loss is common in the elderly, and is often assumed to be secondary to Alzheimer's disease. Not necessarily. At least 15% of dementias are from vascular disease, typically involving extensive small vessel disease. The 3 diagnostic clues are prior CVA (60% of stroke victims develop cognitive dysfunction apparent within 3 months), advanced age (which is not specific for vascular dementia) and presence of cardiac failure. Suggestive findings on evaluation include absence of early memory deficits, abnormal brain MRI findings, presence of post-stroke stigmata including focal neurologic lesions, clinical depression, nocturnal wandering/confusion, relative preservation of personality and emotional responses, urinary/fecal incontinence, increased somatization and emotional lability, and of course, the characteristic "step ladder" deterioration in cognitive function with intervals of extended neuro-stability..

NO SNITCHING

Doctors across all specialties do not like reporting their peers who are either incompetent or impaired (DesRoches et al, JAMA, 2010). Only 64% would report a colleague or take any action, the rest would see no evil, speak no evil, and hear no evil. The reasons are as varied as there are doctors: "it's someone else's problem"; "nothing will come out of it"; "it could easily be me", and, of course, the old staples, including fear of retribution, fear of excessive punishment meted to offender, and not knowing how/whom to report. Not surprisingly, minorities and foreign medical graduates were least likely to report their peers, whilst doctors in academic medical centers were most likely to blow the whistle. With performance quality metrics (why did you think CPOE was started in the first place?) it will now be easier to pinpoint errant doctors with better accuracy.

STEROIDS BEFORE TRANSPLANTATION

Early post-transplantation allograft failure (acute renal failure within a few days of kidney transplant) is not uncommon. It is more common if donor kidney is "marginal", i.e. the elderly, cadaveric donors, or donors with pre-transplant kidney disease. In an attempt to prevent early renal failure following transplantation, Kainz et al, Annals Intern Med, 2010, administered IV Solumedrol in heart-beating cadaveric donors to suppress systemic inflammation, alleged to be a prime cause of renal failure in this situation. Unfortunately, IV steroids given to the "marginal" donor did not improve the incidence or shorten the duration of renal failure in allograft recipients.