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***Guest Editor*: Yul D. Ejnes, MD, FACP**

COLUMNS

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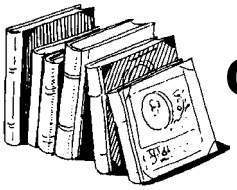

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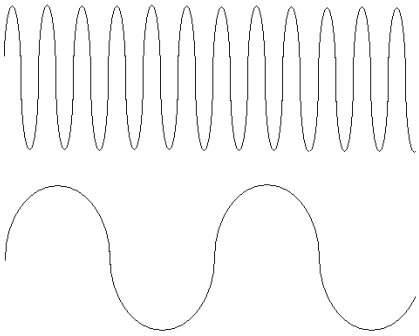

Brain Waves and the Era of Cosmetic Neurology

I was about to begin this column focusing on a brochure my daughter, who lives in LA, sent me, advertising "Yoga for brain education" and a book called, Brain Wave Vibration, which "helps you reawaken your natural healing abilities by awakening your brain stem." "Your brain stem is critical to your health because it is the area of your brain that controls breathing, heart rate, stress response and other automatic functions of the body." The brochure noted that Los Angeles had proclaimed May 2nd, Brain Education Day."

As luck would have it I then stumbled on an editorial in Neurology (Hamilton et al. Rethinking the Thinking Cap. 2011;76:187), the journal of the American Academy of Neurology, about the potential use of brain stimulation to enhance memory and cognition. "Cosmetic Neurology," is their term. This type of brain stimulation is like a watered down and somewhat focal electroconvulsive therapy (ECT), but does not induce seizures. This "cosmetic" research is in addition to the experimental trials on mood and motor function for people with Parkinson's disease, again based on effects of ECT.

Discussions of brain wave vibration, trans-magnetic stimulation and relaxing the brainstem are reminders of the common belief that we all only use a percentage of our brains, the rest of it presumably lying fallow. Popular myth holds that what distinguished Albert Einstein from the rest of us was his ability to harness his whole brain power. The rest of us are innately like him, but due to some trick or peculiarity of wiring he could harness a 200 horse power motor awhile the rest of us are in the double digits (or less). But, if we can vibrate our brains, or parts of the brain we might go from our V6 to a V8 version.

Having read about brain wave vibrations and studied electroencephalography, I have come to believe that electrical synchrony may save the planet. While it is true that when portions of the brain fire in synchrony epileptic seizures occur, near-synchronous firing may be ideal. Perhaps seizures simply represent too much of a good thing, which brings me to quantum neurology.

Most of us don't understand quantum mechanics, and even fewer the notion of quantum computing, but here, at the very core of physics, may be the key to understanding the mental universe, which is, by definition, the actual and only universe we all live in. The core of quantum mechanics, of course, is probability; objects and waves transform into each other, and no object is in any particular location, but rather has a probability or energy distribution. This is why people have memory lapses and why some people are smarter or more coordinated than others. Their electrons are on the wrong side of the bell shaped curve.

Imagine that each electron in your brain has a probability of doing something or being somewhere. The brain works by chemical-electric connections. So, if you can't count on each electron doing its job, these synaptic connections are only partly reliable. Many people have used a variety of drugs to alter the physiology of their brains, with the ultimate goal of changing the reliability of transmission. Imagine if all your electrical impulses actually triggered their intended connections! Perhaps you'd think more clearly. Perhaps you wouldn't forget the thing you walked into the living room to retrieve but then forgot. On the other hand, if the connections all fired at the same time, the energy released is overwhelming, but short of a critical mass and an explosion, the brain generates an electrical seizure.

For the past few years we've been working on cosmetic neurology. Originally used to describe the use of botulinum toxin to reduce age-related wrinkles, cosmetic neurology now focuses on intelligence, memory and, hopefully, personality. Of course part of the explanation for this is that when botulinum injections for wrinkles proved to be a money-making activity, it was taken over by dermatologists so neurologists needed something else for the cosmetic effect. Being neurologists we have taken a "deeper," more philosophical approach to cosmesis, by tapping American's concerns to be smarter and to think quicker. When we offered our first study for "cosmetic Neurology" intervention we were swamped with interest, but when we described our study, and people learned that it was not for botulinum, we did, indeed, have trouble recruiting. More people are interested in wrinkles than intelligence. However, we have concluded our study and showed that people who wear magnets on their left parietal scalps improve on tests of verbal fluency. We are studying the effect of magnets on frontal lobe function for apathy, and reverse magnets (patent pending) which reduce electrical activity in places we think may be overactive.

The idea behind magnetic stimulation is that we can overcome the randomness imposed by quantum mechanics, with directed electromagnetic forces that cause electrons to move in ways that produce higher degrees of wave synchrony, thus relaxing the brainstem and also making the interactions between the two cerebral hemispheres less oppositional, allowing them to work in concert, thus increasing brain power. We have found that focused electromagnetic stimulation increased brain metabolism as measured by PET scan. This in turn has led to significant weight loss for many subjects, as they think harder, and use greater energy. This, in turn, has greatly improved our recruitment for our studies, for we can now advertise weight loss as a potential

benefit. It appears that many more people are interested in wearing magnets and reverse magnets on their heads to lose weight than to think better. We can again talk about "cosmetic neurology," although once reimbursement codes are determined it will undoubtedly become a behavioral medicine procedure.

April Fool.

– Joseph H. Friedman, MD

Disclosure of Financial Interests

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A Tale of Two Stricken Cities

It was a magnificent harbor where the Tagus River emptied into the eastern Atlantic Ocean. Archeologists tell us that Celtic tribes had peopled its shores, building great stone dolmens to honor their gods. About three millennia ago it became the site of a Phoenician trading post called *Allis Ubbo* (safe harbor.) The port assumed great mercantile importance since the Phoenicians—disseminators of civilization—used the harbor as their point of departure to the fabled tin mines in what is now southwestern England. Greek mariners knew the community as Olissipo; and the Romans, now designating the Iberian province as Lusitania, called the community, Olissipona. And when Portugal was conquered by the Islamic Moors in 711 CE, they referred to the site as *al-'Isbunah*, a name than morphed finally to its current title, Lisbon, when the coastal city was recaptured by Christian forces in the Reconquista of 1147 CE.

Mid morning of November 1, 1755, All Saints Day: The citizens of Lisbon, Portugal, were not unfamiliar with periodic earth tremors; and fourteen times in the prior five centuries the tremors were sufficiently intense to do structural damage within the city limits. But it had been many years since the last earthquake of note had touch the city; and Lisbon, on this holy day, was unprepared for the intensity of this quake, which in retrospect, was the most damaging quake afflicting a European city in recorded times.

The citizens of Lisbon witnessed the physical destruction of much of their baroque city; but nature had not yet completed its veil of lethal terror. The epicenter was located on the floor of the Atlantic Ocean some 200 kilometers southwest of the Portuguese shores. The temblors were experienced as far northeast as Finland. The Atlantic shores of Portugal, however, were then struck by a massive tidal wave, a tsunami, that engulfed the Algarve coast, destroying many fishing villages and flooding habitations along the Tagus River. Superimposed upon the destruction wrought by quake and flood were devastating fires beginning shortly after the quake-initiated destruction. The separate fires coalesced, razing much of the center of Lisbon including its central hospital, the Royal Hospital of All Saints. An estimated 40,000 Portuguese died.

The three-fold tragedy befalling Lisbon—earthquake, tidal wave and fire—brought the Portuguese to reappraise their political views, their religious tenets, and certainly their newly experienced intimacy with a nascent science called seismology. The ruling monarch, King Joseph, had his palace utterly destroyed, and for the remainder of his life he refused to dwell within customary masonry structures, remaining instead in a small tent city. Widespread fear dominated Lisbon's urban population, many believing that their unhappy fate was an act of divine punishment for their emerging intimacy with the Age of Enlightenment.

Lisbon's tragedy was well known to the remainder of the western world . Voltaire wrote poetic condolences and a century later in the United States, Oliver Wendell Holmes Sr.,—poet and physician of renown—makes mention of the Lisbon earthquake beginning in the second stanza of his enduring poem, "The Deacon's Masterpiece," which begins with the memorable lines: "Have you heard of the wonderful one-hoss shay, That was built in such a wonderful way, It ran a hundred years to a day ?"

Holmes' second stanza declares, somewhat irreverently:

Seventeen hundred and fifty-five.
George Secundus was then alive,
Snuffy old drone from the German hive.
That was the year when Lisbon town
Saw the earth open and gulp her down.

Over a millennium before, in the year 79 CE, another proud city was felled by natural forces. Pompeii, a prosperous city of some 20,000, situated on the southern reaches of the Bay of Naples, provided many summer villas for the Roman elite. The city stood figuratively in the shadow of Mount Vesuvius, a volcano with a reputation for unanticipated eruption. In November of the year 79—historians think that it was November 23—Vesuvius erupted sending a cloud of superheated ash in a southeastern direction killing the inhabitants of Pompeii and neighboring cities such as Herculaneum. Pompeii was buried under a 22 meter layer of hot ash; and it was then lost to memory except as a footnote in the writings of the late Roman scribes. The physical existence of Pompeii was only verified, by accident, in 1599.

Two cities, one pious and one voluptuary, were felled by indifferent natural forces. In nature, said Ingersoll, there are neither rewards nor punishments, there are only consequences.

– Stanley M. Aronson, MD

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Disclosure of Financial Interests

Stanley M. Aronson, MD, and spouse/significant other have no financial interests to disclose.

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Giving Thought to Primary Care

Yul D. Ejnes, MD, FACP

This is the second of two *Medicine and Health Rhode Island* issues on Primary Care, in which each expert addresses three questions, selected by the guest editor, that arise frequently in everyday practice.

No journal issue on the topic of primary care would be complete without mention of the current crisis in primary care. In 2006, the American College of Physicians warned of the imminent "collapse" of primary care.[1] Along with many other stakeholders, medical schools have contributed to this collapse of primary care and have a major role in its rebuilding.[2] This summer, Mullen *et al* published a study of medical schools' commitment to the "social mission" of medical education—defined as "graduating physicians who practice primary care and work in underserved areas and recruiting and graduating young physicians who are underrepresented minorities."[3] The article generated the expected responses: medical schools that were rated highly celebrated their standing, and the Association of American Medical Colleges (AAMC) and schools that did not fare as well criticized the study.

As an alumnus of Brown (twice) and current faculty member, I was curious to see where we ranked. Brown ranked 97th out of 141 in primary care physician output, 66th out of 141 in HPSA (health professional shortage area) physician output, and on a "social mission score" that comprised the previous categories plus a measure of underrepresented minorities, Brown also ranked 66th.

Brown's scores in these areas, given the signals from College Hill over the past several years, did not surprise me. The decreased standing within the Medical School of the Program in Liberal Medical Education, the spotlight on research, grants, technology, and buildings in the Medical School's regular publications, and the pursuit of a top ranking in the *US News and World Report* list seem, at least to me, to matter more than training primary care physicians. Brown was once recognized as a primary care center of excellence and the Medical School took pride in that reputation. Now, I am not so sure of either.

With its renowned family medicine and primary care internal medicine residencies, its dedicated clinical faculty, and innovative programs such as the Doctoring course, the Medical School has an opportunity to lead by contributing to the revitalization of primary care. That would be more valuable than a top ranking on the *US News* or any other list.

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Asymptomatic Carotid Stenosis: Screening and Management

Jonathan A. Grossberg, MD, N. Stevenson Potter, MD, PhD, and Mahesh V. Jayaraman, MD

The management of asymptomatic carotid stenosis can be a dilemma to the primary care physician. In this review, we hope to update the reader on current insights into asymptomatic carotid atherosclerosis.

Carotid stenosis is defined as the atherosclerotic narrowing of the proximal internal carotid artery exceeding 70% in severe cases and 50% in moderate cases. The prevalence of carotid stenosis in Western countries increases with age. A recent Western European population study placed the prevalence of moderate asymptomatic stenosis at 4.2% and severe stenosis at 1.7%. The prevalence was found to increase with age and be higher in males, with 12.5% of men being diagnosed with moderate stenosis.[1]

Fisher first described the pathophysiology of carotid atherosclerosis in 1951.[2] Since then it has increasingly been recognized as a risk factor and cause of stroke. A population based study of subjects older than 55 found that the presence of a severe carotid plaque burden increased the relative risk of a non-lacunar infarction in the anterior circulation by 3.2 times and increased the risk of a lacunar infarction by 10.8 times.[3] Accordingly, asymptomatic carotid stenosis has become an important cerebrovascular topic for the primary care physician.

What is the best screening test for carotid artery stenosis?

The diagnosis of asymptomatic carotid stenosis classically begins in the outpatient office. Many primary care physicians will examine patients for the presence of a carotid bruit, which is thought to signify turbulent blood flow across a stenotic plaque. While the Framingham Heart Study found that patients with a carotid bruit had double the stroke rate of controls, the majority of these strokes were in vascular territories unrelated to the stenosis.[4] Another large meta-analysis found a significantly increased risk of myocardial infarction and cardiovascular death in patients with a carotid bruit, thus implying that the bruit is best used as a marker for total body atherosclerotic disease and not as a direct marker of carotid risk.[5] A recent cohort study examined the incidence of carotid bruit and clinically significant stenosis on carotid duplex and found that bruit auscultation had a sensitivity of 56% and a specificity of 98%. From the analysis of their data, the authors concluded that the auscultation was not sufficient to exclude stenosis, and further invasive testing should be considered in high-risk patients.[6]

Since physical exam has a low probability of diagnosing asymptomatic carotid stenosis, the diagnosis is often made through a variety of non-invasive radiological exams, namely **duplex ultrasonography (DUS)**, **computed tomographic angiography (CTA)**, and **magnetic resonance angiography (MRA)**. DUS is often the first screening test done due to its ease of use, lack of radiation or need for contrast material and cost. A review of non-invasive imaging found that DUS had an 86% sensitivity and 87% specificity for diagnosing clinically significant (>70%) stenosis.[7] One drawback of DUS is that it is operator dependent, and operator experience can affect the accuracy of results. In addition, the utility of DUS in screening patients without any risk factors for atherosclerotic disease is low, and screening the general population may not be cost-effective. Better results were found using MRA for the detection of clinically significant stenosis with a sensitivity of 95% and a specificity of 90%.[7] In another recent meta-analysis, CTA was found to have a sensitivity of 76% and a specificity of 94%. This study also found that contrast-enhanced MRA had the best sensitivity (94%) and specificity (93%) when compared to DUS, CTA, and non-contrast MRA.[8] One potential advantage of MRA over CTA is that dense calcification from some carotid plaque can limit luminal evaluation on CTA. While catheter angiography is the gold standard for evaluation of carotid stenosis, the cost, more invasive nature and potential for neurologic complications have relegated it to a problem-solving role when significant discrepancies exist among the non-invasive imaging techniques. In patients with known carotid disease, the optimal interval for repeating DUS is unknown, but it is likely reasonable to repeat the study annually to monitor for interval change.

How should a patient with asymptomatic carotid artery stenosis be followed and when should they be referred for revascularization?

Medical Therapy

For the majority of asymptomatic patients with carotid stenosis the ideal treatment is a combination of risk factor modification and anti-platelet medication. The risk factors targeted are familiar to all clinicians and include hypertension, smoking, physical inactivity, obesity, hyperlipidemia, and glycemic control.

While a number of these risk factors can be modified through lifestyle changes, particular attention has been paid to pharmacological treatments for hyperlipidemia and hypertension and their direct effect on carotid atherosclerosis. It is well known that the use of statins to meet cholesterol goals reduces the risk of stroke, but there is also evidence that statins may impact carotid plaque itself.[9] A recent study reviewed the effect of statins on atherosclerotic burden by randomizing patients to low-dose versus high-dose statin therapy. All patients in the trial had a significant reduction in radiologic atherosclerotic burden by 12 months, and post-hoc analysis revealed that the change was more related to the low LDL level induced by statin therapy than to the medication itself.[10] Another more recent study found that statin use in symptomatic carotid patients was associated with a decreased incidence of negative plaque features on MR imaging. These negative plaque features are thought to be predictive of future cerebrovascular events and include intraplaque hemorrhage, necrotic

core, and a thin fibrous cap.[11] In addition, there is some evidence that certain anti-hypertensive medications can have positive effects on the morphology of carotid plaques. A more detailed review of the effect of various medications on plaque morphology is beyond the scope of this paper and can be found in the review by Daskalopoulou et al.[12] The literature, however, is not conclusive so the recommendation is that patients be treated with anti-hypertensive medications that best fit their other co-morbidities.

In addition to risk factor and lifestyle modifications, it is the consensus of the **American Stroke Association (ASA)** that patients with asymptomatic carotid stenosis be treated with aspirin.[13] While good data exist for recommending aspirin and other anti-platelet agents to patients with a history of an ischemic cerebrovascular or cardiovascular event, there are no adequate studies examining aspirin in patients with asymptomatic carotid stenosis.[14] This ASA recommendation for aspirin derives from the fact that nearly all trials comparing outcomes of medical management to surgical management for carotid stenosis treat patients with aspirin. While the ASA does give a class I evidence rating to the use of aspirin, there are no studies that provide class I data for the use of other antiplatelet agents in the management of asymptomatic carotid stenosis.

Carotid Revascularization: Endarterectomy or Stenting

The final potential treatment for asymptomatic carotid stenosis is the invasive option in the form of either surgical **carotid endarterectomy (CEA)** or **carotid angioplasty and stenting (CAS)**. The two keystone studies comparing endarterectomy to maximal medical management in asymptomatic patients found a small benefit of surgery when performed by an experienced surgeon with a low complication rate. The ACST examined asymptomatic patients with at least 60% stenosis on DUS and found a five-year stroke rate of 6.4% for patients treated with CEA versus 11.8% for patients under maximal medical management.[15] The ACAS study also evaluated asymptomatic patients with at least 60% stenosis and found similar results: patients treated surgically had a stroke or death rate of 5.1% compared to 11% for patients treated with medical management.[16] As a result the ASA recommends endarterectomy in selected asymptomatic patients when performed by a surgeon with a <3% complication rate.[13] It should be noted that the patients in these trials were a highly selected group and there were many exclusion criteria that make the studies less generalizable. For example, patients with contralateral carotid occlusion were often excluded from these trials, as were patients with asymptomatic restenosis in the setting of prior carotid revascularization. In addition, the medical management arms of these trials were not standardized and often did not include current aggressive anti-hypertension and anti-hyperlipidemia pharmacological treatment.

The question often facing the primary care physician dealing with asymptomatic carotid stenosis is when to refer for invasive treatment and when to treat medically.

While the evidence for CEA in carotid stenosis is well established, the data involving CAS is only beginning to emerge. The most significant recent publication is the CREST trial, which randomized both symptomatic and asymptomatic patients to either CEA or CAS. Looking at both symptomatic and asymptomatic patients, the trial found that CEA and CAS were statistically equivalent in the composite rate of stroke, myocardial infarction, and death at four years. Where the two differed, however, was in a higher rate of peri-procedural myocardial infarction with CEA and a higher rate of stroke with CAS. For the asymptomatic subgroup, there was a non-statistically significant trend towards lower stroke and death rate at four years in the CEA group, and the writers noted the effect of stroke on quality of life. The CREST trial lacks a medication-only asymptomatic subgroup.[17] There are a number of similar trials comparing endarterectomy to carotid artery stenting that go beyond the scope of this paper. On the basis of the early trials, the ASA recommends that CAS be considered only in patients with symptomatic stenosis who have either a medical or surgical condition that makes surgery high risk (such as prior CEA, radiation therapy to the neck, or significant co-morbidities to surgery).[13] Despite this recommendation, a recent study detailed that in New York and Florida, nearly 92% of CAS performed were for asymptomatic disease.[18] In addition, many of the same critiques of the CEA trials (such as patient selection and variable medical management of the non-operative arm) can be applied to the stenting trials, which make recommending CAS for the modern asymptomatic patient a difficult decision.

How should asymptomatic carotid stenosis be treated?

The question often facing the primary care physician dealing with asymptomatic carotid stenosis is when to refer for invasive treatment and when to treat medically. A recent article in *Stroke* by Abbot attempted to answer the question by reviewing the stroke rate of patients with asymptomatic carotid stenosis in published studies over a two-decade span. The study found that with the advances in modern medical therapy, the stroke rate of patients with asymptomatic carotid stenosis treated medically overlaps that of the patients treated surgically in the above-detailed studies.[19] After analyzing the raw study data, Abbot found that the risk of ipsilateral stroke was 1.5% per year in the patients undergoing CEA in ACAS and 2.3% for the patients treated with maximal medical treatment in the same study. Abbot compared those ACAS rates published in 1995 with the ASED and SMART trials, published over the past 5 years, which found annual ipsilateral stroke rates of 0.6%-1.2% in asymptomatic patients managed medically.[19,20,21] These conservative management stroke rates are similar to the recent CREST trial which found a 4.5% four year risk of ipsilateral stroke for patients treated with CAS and a 2.7% four year risk of stroke for patients treated with CEA.[17, 19]

In addition, Abbot's study found that medical management is three to eight

times more cost-effective than surgical management. This study suggests that the best treatment for most patients with asymptomatic carotid stenosis is the medical treatment detailed above: aggressive treatment of risk factors such as smoking, hyperlipidemia, hypertension, and glycemic control, combined with anti-platelet therapy, such as aspirin. The author does indicate that her numbers are based on a regression analysis and that there is no study that directly measures the impact of best medical practice on stroke rates in asymptomatic carotid stenosis.[19]

Should asymptomatic carotid disease be screened for?

Given the questions regarding stroke rates in asymptomatic disease for medically treated patients, perhaps the bigger question is whether carotid atherosclerotic disease should be screened for with DUS. A large trial of over 5000 asymptomatic individuals analyzed with DUS found that increased carotid intima and media thickness was significantly associated with an increased risk for myocardial infarction and stroke.[22] Even in patients with known peripheral artery disease or coronary disease, screening for carotid disease with DUS should be considered. While the results of screening might not result in referral for carotid revascularization, they do provide valuable insight into the patient's overall atherosclerotic plaque burden, a "window into their arteries."

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Gastroesophageal Reflux Disease:
Endoscopy, Duration of Treatment, and Choice of PPI

Neil R. Greenspan, MD, FACG

When is endoscopy indicated in the diagnosis and follow-up of Gastro-esophageal Reflux Disease (GERD)?

In the Montreal definition based on a consensus of experts, GERD was defined as "*a condition that develops when the reflux of stomach contents causes troublesome symptoms and/or complications*."[1] It is important to note that the diagnosis may be symptom-based and made independent of endoscopy and other diagnostic testing. Patients diagnosed with GERD, therefore, will include patients with erosive esophagitis as well as those with **non-erosive reflux disease (NERD)**. Patients with NERD may account for two-thirds of all patients with GERD and, as they will appear normal endoscopically, the sensitivity of endoscopy for making the diagnosis of GERD is low.

Endoscopy does play a significant role in excluding other serious disorders such as esophageal or gastric cancer in a patient presenting with reflux-like symptoms. The concept of "alarm" signs or symptoms comes in to play here. Although not supported by literature, it is certainly appropriate to consider endoscopy in any patient presenting with reflux and dysphagia, odynophagia, weight loss, abdominal mass, GI bleeding or anemia.[2]

The issue of GERD, Barrett's esophagus and esophageal cancer remains controversial. Carcinoma of the esophagus is a clinically important disease. Worldwide esophageal cancer is diagnosed in almost a half a million people annually where it is the sixth leading cause of death in men (ninth leading in women). The incidence of adenocarcinoma of the esophagus is rising rapidly in the United States and other "Western" countries.[3] Over the period from 1976-1987, the incidence of adenocarcinoma of the esophagus in US white males increased more rapidly than that of any other cancer. With its incidence increasing almost 20% annually, adenocarcinoma has replaced squamous cell carcinoma as the most prevalent type of esophageal cancer in this population.[4]

The prognosis of esophageal adenocarcinoma is strongly associated with the stage at diagnosis. According to **Surveillance Epidemiology and End Results (SEER)** data from 2005, one-year survival in patients with local, regional, and distant (metastatic) disease were 68%, 54%, and 28%, respectively. The five-year survival for esophageal cancers confined to the mucosa (T1m, N0, M0) may approach 90%.[5] Therefore, there is great interest in identifying risk factors for esophageal cancer and focusing screening and surveillance efforts on populations at high risk for esophageal cancer. A large population-based Swedish study showed that the relative risk of esophageal cancer may be more than seven-fold greater in patients with recurrent reflux when compared to patients without reflux. The more frequent, more severe, and longer-lasting the symptoms of reflux, the greater the risk.[6] While this may make GERD patients appear to be a good target population for endoscopic esophageal cancer screening, enthusiasm for devoting limited health-care dollars on this effort is tempered by the realization that the majority of patients with adenocarcinoma of the esophagus do not report a history of reflux. Additionally, only a small percentage of all patients with reflux ever develop esophageal adenocarcinoma.

Like adenocarcinoma, GERD is a strongly associated with Barrett's esophagus. It is well accepted that Barrett's esophagus, defined as metaplasia of esophageal mucosa and presumably related to chronic exposure to acid, is a pre-malignant condition for adenocarcinoma of the esophagus. The risk estimated to be about 0.5% annually is substantially higher in patients with low or high grade dysplasia.[7] There are controversies in the diagnosis of Barrett's esophagus and dysplasia that are beyond the scope of this article. The American College of Gastroenterology has published guidelines addressing the management of patients with Barrett's esophagus.[8] In addition, a variety of exciting new ablative therapies and endoscopic mucosal resection techniques for Barrett's esophagus with dysplasia or early adenocarcinoma have been developed. These can eliminate dysplastic epithelium and, under acid suppression, permit restoration of squamous mucosa. The fact remains however, that at this time, no endoscopic screening protocol for Barrett's has been shown to improve survival by preventing deaths from esophageal cancer.

How long should daily drug treatment (for GERD) continue and how should it be stepped down?

As with all therapies, the decision as to choice and duration of therapy for GERD involves consideration of risks and benefits. In all patients with GERD, **Proton Pump Inhibitors (PPIs)** have been shown to be more efficacious in controlling acute symptoms and maintaining a symptom-free state than **Histamine 2 Receptor Antagonists (H2RA)** which have, in turn, proven to be more effective than placebo. PPIs are also the most effective agents for healing esophagitis. Regarding the dosing frequency, once daily dosing of PPIs has been used most often in studies, though for non-responders, a trial of twice daily dosing is certainly appropriate and supported by the pharmacodynamics of the drugs.[9]

Assuming the patient improves, how long should therapy continue? Without the benefit of maintenance therapy (typically one-half the dose of healing therapy), the recurrence of esophagitis is high, occurring in up to 80% of patients within 6 months of completing acute therapy. This potentially puts the patient at risk for complications including esophageal stricture. H2RA and placebo are less effective than PPIs in preventing recurrent esophagitis in a patient healed with PPIs. Similarly, on demand therapy with PPIs can not be recommended for patients with documented esophagitis as the recurrence rates are unacceptably high.[10]

The greater efficacy of PPIs must be weighed against the possible risks of therapy including bone fractures, inter-

ference with anti platelet therapy, and an increased risk of infections. In May 2010 the **Food and Drug Administration (FDA)** required a change in the labeling of prescription and over-the-counter PPIs to include information about a possible increased risk of fractures of the hip, wrist, and spine in patients treated with PPIs. This was based on a Drug Safety Communication that quoted seven of eight retrospective studies showing an increased risk of fractures in patients treated with one of these drugs.[11] Interestingly the eighth study which did not show an association between PPI and bone fractures was published this year by Tarownik, who previously reported an association using a different data base.[12] Three of the studies that showed an association between PPI and fractures did not document a change in bone mineral density in patients taking these medications. As with other retrospective studies, causation is unproven.

There is evidence that some PPIs may interfere with the action of clopidogrel (Plavix®). Clopidogrel is a pro-drug that is converted to the active form by the enzyme CYP2C19. The FDA has changed the labeling of clopidogrel to include a "Black Box warning" warning that 2-14% of people who, as a result of a variant of CYP2C19, are 'poor metabolizers' of clopidogrel. As a result, poor metabolizers are more likely than normal metabolizers to suffer from adverse cardiovascular events when treated with clopidogrel for acute coronary syndrome or percutaneous coronary intervention.[13] Omeprazole is an inhibitor of CYP2C19 thereby reducing by about 45% the *in vitro* efficacy of clopidogrel on platelets Although not mentioned in the "Black Box" warning, omeprazole's adverse effect on clopidogrel's metabolism is mentioned in the prescribing information with a recommendation that coadministration be avoided. Observational studies have indicated a possible increased of cardiovascular events in patients treated with clopidogrel and PPI in the setting of an acute coronary syndrome.[14] As with other observational studies, this may be subject to certain biases including confounding risk variables. Unfortunately, the only randomized trial, **The Clopidogrel and the Optimization of Gastrointestinal Events (COGENT)**, to study this interaction was stopped prematurely when the sponsoring company went bankrupt.

PPIs have also been associated with a slightly increased incidence of pneumonia, *Clostridium difficile* infection and bacterial gastroenteritis. The studies are all retrospective, and the risk modest.

For the larger population of patients with non-erosive disease who presumably do not carry the same risk of esophageal stricture, the cost and risk of therapy may play a more important role in decision making about maintenance therapy. Patients with NERD who improve on daily PPI are also more likely to remain asymptomatic when continued on PPI compared to those given H2RA or placebo.[15] The cost of therapies differs but in general, H2RA are less expensive than PPIs and have been shown to be a class of medications with a very favorable side-effect profile. Therefore a trial of step-down therapy is a reasonable approach in this setting. "On demand" PPI therapy for recurrent symptoms has been shown to improve the health-related quality of life and patient satisfaction but the comparison to daily maintenance therapy has not yet been published.

The greater efficacy of PPIs must be weighed against the possible risks of therapy including bone fractures, interference with anti platelet therapy, and an increased risk of infections.

Are there clinically significant differences among the proton pump inhibitors?

When reviewing the primary literature comparing the efficacy of PPIs in GERD it is important to make sure that the endpoints are not only clearly defined but clinically relevant. For example, a recently published article stated "significantly greater acid control" of one PPI over another because the authors demonstrated a greater percentage of time of gastric pH> 4 (45.7 vs. 36.8).[16] While this may have reached statistical significance at the p<0.0001 level, it is not at all clear that this minuscule change in gastric pH has any relationship to a better outcome for the patient.

Acid suppressive therapy is 'big business.' In 2000 the economic impact of GERD in the United States was estimated to be $9.3 billion dollars. More than 60% of that cost was attributed to anti-secretory therapy.[17] The potential influence of the pharmaceutical industry on the research of antisecretory therapy is clear when reading the conflict of interest statements of many authors in the field. The majority receive some support from the pharmaceutical industry. I think it is likely that many of the studies performed today regarding the treatment of GERD are adversely affected by the same influences that prompted a recently retired editor of the NEJM to conclude that:

> *Given the conflicts of interest that permeate the clinical research enterprise, it is not surprising that industry-sponsored research has consistently been shown to favor the sponsor's drug—partly because negative results are often not published partly because positive results are repeatedly published in slightly different forms, and partly because a positive spin is put on even negative results… Clinical research that is published is often biased, usually by designing the studies in ways that will almost inevitably yield favorable results for the sponsor.* [18]

The evidence supporting the superiority of one PPI over another is very limited, especially when one takes into account different doses and dosing schedules. In my own practice, I typically prescribe a generic PPI and patients are given a prescription that permits the pharmacist to substitute another PPI that is lowest cost to the patient. An exception to this is a patient requiring antiplatelet therapy with clopidogrel in whom a new prescription for omeprazole or esomeprazole may be eschewed in favor of another PPI such as pantoprazole that is metabolized through a different pathway. Otherwise, changes from one PPI to another are usually driven by the patient's report of side effects

though there are few data to support the efficacy of this practice. Changes in PPI driven by insurance company mandates may precipitate more severe symptoms and decreases patient satisfaction. [19]

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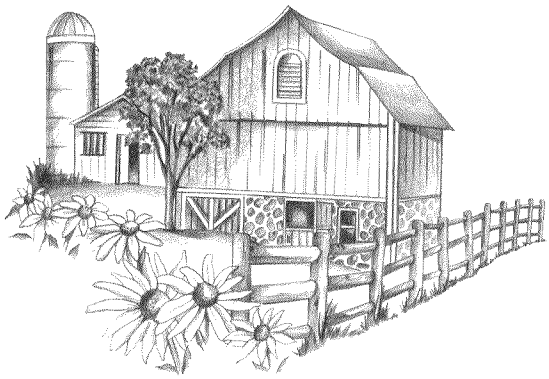

Osteoporosis: Screening, Evaluation, and Monitoring

Michael W. Schaefer, MD, and Geetha Gopalakrishnan, MD

Osteoporosis is the most common skeletal disorder. It is characterized by a decrease in bone mineral density resulting in fractures. According to the **National Osteoporosis Foundation (NOF)**, 10 million Americans are affected by osteoporosis and an additional 34 million are at risk for osteoporosis. Although osteoporosis is more common among women, men account for approximately 20% of all cases. It is estimated that 50% of women and 25% of men over the age of 50 years will have an osteoporosis related fracture in their lifetime. Osteoporotic fractures typically involve the spine, hip and forearm and result from minimal or no trauma. The morbidity, mortality, and cost associated with these fractures are significant. Mortality rates as high as 25% are reported one year after a hip fracture. If patients survive, 25% will require long-term nursing home care and only 40% will achieve their pre-fracture level of independence. The estimated cost of these fractures to our health care system is more than $17 billion per year and this cost is anticipated to triple by 2040. Therefore, prevention of osteoporosis, particularly fractures, is a public health priority.[1]

What are the current evidence-based recommendations on screening for osteoporosis?

Choice of Screening Test

The goal of screening is to identify individuals at risk for osteoporotic fractures. **Dual Energy X-ray Absorptiometry (DXA)** is the gold standard for diagnosing osteoporosis and monitoring changes in **bone mineral density (BMD)**. BMD measurement by DXA strongly predicts fracture risk. It is estimated that each **standard deviation (SD)** decrease in BMD increases the risk of fracture by 2-3 fold.[2] BMD can be expressed as a number of SD away from a sex-matched young normal adult population (T-score) or a population of the same age and gender (Z-score). Osteoporosis is defined by a clinical history of fragility fractures or by a DXA assessment of the lumbar spine, hip, and forearm BMD (see Table 1).[1]

Other screening tools include quantitative ultrasound (QUS), quantitative CT (QCT), and peripheral QCT. Although these scans also predict fracture risk, they are not currently recommended for screening purposes. Cost, radiation exposure and lack of normative data limit the use of these tests for screening and monitoring therapy (see Table 2).[3]

Population to be screened

Due to the increased risk of fractures noted with age, the NOF currently recommends screening all postmenopausal women over the age of 65 years and all men over the age of 70 years. This recommendation is based on an outcomes analysis conducted by the **United States Preventive Service Task Force (USPSTF)**. Based on prevalence rates of osteoporosis, screening 10,000 postmenopausal women between the age of 65 and 69 would identify approximately 1,200 individuals at high risk for fracture (T-score ≤ -2.5). If this high-risk population were offered treatment, it is estimated that 14 hip fractures and 40 vertebral fractures could be prevented, assuming 70% compliance. These conclusions were based on published evidence that bisphosphonates reduce the risk of hip fractures by 37% and vertebral fractures by 50%. In this analysis, fracture prevention utilizing anti-resorptive therapy became more favorable with increasing age.[6]

Furthermore, the NOF recommends screening in younger postmenopausal women and men aged 50 to 69 years based on the presence of risk factors (see Table 3). The USPSTF analyzed the use of risk factors as a guide to screening. In women aged 60-64, screening would prevent only

TABLE 1: WORLD HEALTH ORGANIZATION CLASSIFICATION [1]

Normal	T-score at -1.0 and above
Low bone mass (Osteopenia)	T-score between -1.0 and -2.5
Osteoporosis	T-score at or below -2.5
Established Osteoporosis	One or more fragility fractures

T-score: a number of standard deviations (SD) away from a sex-matched young normal adult population

TABLE 2: SCREENING TOOLS FOR OSTEOPOROSIS [2-5]

Type of Test	Ability to Predict Fracture	Advantages
Dual Energy X-ray Absorptometry (DXA)	+	Gold standard for screening and monitoring changes in BMD. Advantage: Low cost, ease of use, excellent precision and minimal radiation exposure.
Quantitative Ultrasound (QUS)	+	Can be considered for screening but should not be used to diagnose or monitor therapy. Individuals with a low QUS measurement need to be reassessed by DXA for diagnostic purposes. Advantage: Low cost, ease of use and lack of radiation exposure.
Quantitative computed tomography (QCT) & peripheral QCT	+	Utilized in research studies. Higher radiation exposure and expense has limited use in clinical settings. Advantage: Provides a detailed assessment of the geometrical and structural elements of bone.

TABLE 3: COMMON RISK FACTORS FOR OSTEOPOROSIS & FRACTURE [1, 7]	
General	History of prior low trauma fracture as an adult, Low BMD in patient with or without fractures, Low body mass, Frequent falls
Lifestyle factors	Low calcium intake, Smoking, Alcohol use (> 3 drinks per day), High caffeine intake, Inadequate physical activity
Medications	Glucocorticoids (> 5mg Prednisone daily for at least 3 months), Aromatase inhibitors, Anticonvulsants, Selective serotonin reuptake inhibitors, Proton pump inhibitors, Anticoagulants, Depo-medroxyprogesterone
Medical conditions	Thyrotoxicosis, Hyperparathyroidism, Diabetes Mellitus, Vitamin D deficiency, Cushing's syndrome, Hypogonadism, Rheumatoid arthritis, Malabsorption, Multiple Myeloma
Genetic factors	Hip fracture in a first degree relative

five hip fractures over five years. However, in the presence of one risk factor, the rate of fracture prevention doubled and approached that of the group over age 65. Based on these estimates, the presence of a risk factor should prompt screening in younger postmenopausal women and men over the age of 50 years.[6]

Lastly, individuals with secondary causes of osteoporosis such as steroid use or hyperparathyroidism should be screened for osteoporosis irrespective of age. Routine BMD assessment is not recommended for healthy young men, premenopausal women, or children.[1]

Frequency of Testing

Despite having established guidelines on when to start screening for osteoporosis, limited data is available on how often to repeat BMD assessments after the initial exam. Hillier and colleagues prospectively followed 4,124 postmenopausal women with a mean age of 72 and a total hip T score of -1.3 for a period of eight years. On average, the subjects showed a 0.59% loss in BMD per year and the mean T score at follow up decreased to -1.64. After eight years, there were 877 non-vertebral fractures including 275 hip fractures. After a detailed ROC analysis, the BMD at follow up could not predict fractures any better than the original BMD evaluation. The authors concluded that repeat DXA analysis was not useful in further estimating fracture risk in this population.[8]

Considering the low rate of bone loss noted in this study, these findings are not surprising, and therefore should not be extrapolated to other populations with higher rates of bone loss, such as early menopausal women or individuals with osteoporotic risk factors. The American Association of Clinical Endocrinologists (AACE) currently recommends that in patients with a normal BMD (T-score > -1) at baseline, repeat screening should be done every three to five years. If a patient has a BMD significantly above the lower limits of normal, further testing may not be needed. For patients at risk for osteoporosis (i.e., patients with risk factors or low bone mass), DXA should be performed every one to two years until stability in BMD is established, after which testing frequency can decrease.[9]

TABLE 4: FDA- APPROVED PHARMACOLOGIC AGENTS FOR THE PREVENTION AND TREATMENT OF OSTEOPOROSIS [1]			
Class	Agents	Prevention	Treatment
Bisphosphonates	Alendronate	+	+
	Risedronate	+	+
	Ibandronate	+	+
	Zoledronic Acid	+	+
Calcitonin	Salmon Calcitonin	-	+
Estrogen Agonist/Antagonist	Raloxifene	+	+
Hormone Replacement Therapy	Estrogen	+	-
Parathyroid Hormone	Teriparatide	-	+
RANK Ligand (RANKL) Inhibitor	Denosumab	-	+

How should response to treatment be assessed?

Once a diagnosis is established, calcium and vitamin D supplementation along with pharmacological therapy can be considered for the prevention and treatment of osteoporosis (see Table 4).

Even though treatment can be associated with a decrease in the incidence of osteoporotic fractures, development of a new fracture does not necessarily represent failure of therapy. At best, pharmacological agents reduce fracture rates by 30-70% [10]. Therefore, monitoring changes in BMD can help determine the effectiveness of a treatment strategy and guide management decisions.

The NOF, AACE, and **International Society for Clinical Densitomtery (ISCD)** all recommend using DXA for monitoring treatment. In order to compare BMD, the measured change in BMD must be greater than the precision error of the machine.[9] In general, precision errors range between 2-4% for the spine and 3-6% for the hip. Changes that are greater than the precision error are considered clinically significant.[1] However, changes in BMD with aging or upon initiation of therapy tend to be small in magnitude compared with the expected error in measurement, and therefore, waiting at least a year before repeating a BMD measurement (and preferably two years) is recommended.[2]

In addition to serial DXA measurements, assessment of bone turnover can be considered in certain circumstances. Bone resorption makers such as **N-terminal crosslinking telopeptide of type 1 collagen (NTX-1)** and **C-terminal crosslinking telopeptide of type 1**

collagen (CTX-1) are elevated in high turnover states like menopause.[10] They have been shown to predict fracture risk independent of BMD in postmenopausal women[11]. These markers decrease rapidly with antiresorptive therapy and can be used to monitor response to treatment. Urine NTX is the most common biochemical marker used in clinical practice. Considering the biological variability of this marker, levels should be obtained in the morning after an overnight fast. Large change (i.e. > 50% reduction) is required for clinical significance. Routine measurement of bone turnover markers is not recommended. [10] Measurement of bone turnover can be considered in cases when a discrepancy exists between serial DXA measurement and the clinical senario (i.e. loss in BMD despite therapy with bisphosphonates). In these cases, bone turnover markers can assess degree of compliance or absorption of oral medications.

Who should undergo workup for secondary causes and what should that workup include?

Traditionally, menopausal or age-related osteoporosis is considered primary osteoporosis. Secondary osteoporosis results from a variety of medical conditions and external factors. All patients with osteoporosis should have a comprehensive history and physical examination to identify risk factors for osteoporosis (i.e. family history, medication use, medical history). A more extensive work-up to evaluate secondary causes of bone loss may be indicated in patients with low BMD (Z-score < -2.0), fragility fractures, failure to respond to therapy, or a decline in BMD at a rate greater than expected for age.[12]

The AACE recommends that all patients with osteoporosis, regardless of suspicion for secondary causes, have a routine baseline laboratory evaluation. These studies include serum calcium, phosphorous, creatinine, electrolytes, total protein, albumin, liver enzymes and complete blood count. A 25-hydroxy Vitamin D level should also be checked in any osteoporotic patient, given the prevalence of Vitamin D deficiency. The AACE recommends more extensive laboratory workup depending on history and or physical exam findings including urinary calcium excretion, TSH, PTH, serum and urine protein electrophoresis, urinary free cortisol or other assessment of cortisol excess (1 mg overnight dexamethsone suppression test).[8]

Conclusion

Osteoporosis is a common condition with a significant impact on morbidity, mortality, and health care costs. With this in mind, it is important to screen not only postmenopausal women, but also older men and younger individuals with risk factors for bone loss and osteoporotic fractures. DXA is not only useful as a screening tool, but is an excellent way to monitor response to therapy. Finally, all patients with osteoporosis should have a routine comprehensive evaluation including history, physical, and laboratory assessment; however, a subset of these patients should have a more detailed laboratory workup to rule out secondary causes.

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Venous Thromboembolism:
Duration, IVC Filters, and Hypercoagulable Workup

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Venous thromboembolism (VTE), which includes **deep venous thrombosis (DVT)** and **pulmonary thromboembolism (PE)**, is the third most common cause of cardiovascular mortality in the U.S., after coronary heart disease and stroke.[1] VTE is a systemic disease which may develop spontaneously (idiopathic, unprovoked) or secondary to some identifiable provocative or environmental risk factor.[2] Classifications into unprovoked, surgical, and provoked are useful, since such a classification correlates with the cumulative risk of recurrence and, therefore, influences the duration of therapy and the appropriateness of laboratory investigation.

VTE most commonly affects the deep venous system of the pelvis or lower extremity, but can also occur in less common sites such as the upper extremity, mesenteric veins, ovarian veins, cerebral veins and retinal veins. The anatomic site(s) is of importance as it may also influence diagnostic testing and duration of therapy.

The management of VTE (symptomatic or asymptomatic) can be divided into immediate (up to the first 10 days), early long-term (up to three months), and late long-term (beyond three months) therapy. The objective of immediate management is to shut down thrombin generation and to prevent thrombus extension and embolisation. The objective of early long-term therapy is to prevent thrombus recurrence, embolisation and the **post thrombotic syndrome (PTS)** and to promote the lysis of the thrombus; recurrence in this phase is particularly problematic since there is a higher case fatality rate. The objective of late long-term management is to prevent thrombus recurrence and embolisation and to prevent or reduce PTS[4] since ipsilateral recurrence is a particular risk factor for PTS. Unprovoked VTE may be a manifestation of an inherited or acquired thrombophilic state and, hence, esoteric laboratory testing may be indicated in some cases.[5] This article will address only three of these considerations—the role of late long-term anticoagulation, utilization of an IVC device to interdict embolisation and the judicious use of esoteric laboratory testing.

What is the optimal duration of oral anticoagulation with VTE?

Early long-term anticoagulation is recommended for all VTEs, provoked or unprovoked, symptomatic or asymptomatic, upper or lower extremity. The target INR is 2.5 with a suggested range of 2-3. Shorter courses of anticoagulation have been shown to be inferior.[6]

The decision to use late long-term anticoagulation is more difficult and is essentially a risk-benefit analysis that balances the risk of recurrent thrombosis with the risk of severe bleeding. The risk of recurrent thrombosis is determined by a history of a previous thrombotic event, the circumstances associated with the occurrence of the thrombosis, the presence of residual or persistent thrombosis and laboratory data. The risk of bleeding is determined by patient clinical co-morbidities, the ability to control the anticoagulant effect of warfarin, which is determined by patient compliance, the quality of medical oversight, and patient age. Thus, it is clear that any absolute time period is inappropriate and individualization of therapy required.[7]

General Approach

1. The overall cumulative two-year recurrence rate for surgically provoked thrombosis is 0%- 5%. Hence, there is general agreement that surgical induced VTE should *not* be treated with late long-term anticoagulation.[8]
2. For non-surgical, provoked DVT the recurrent rate at two years has been estimated as high as 8.8%. Hence, continuation of anticoagulation to at least six months may be reasonable, in the absence of any contraindication.[8]
3. For unprovoked VTE that involves the distal lower extremity, three months is adequate. For unprovoked VTE involving the pelvic veins or proximal lower extremity, extended late long-term anticoagulation is preferred, unless there is a major contraindication. Six months is a common minimum and indefinite anticoagulation is a consideration. The overall two-year cumulative recurrence in this setting is 20%; but at five to eight years, it is 30%, and 35% at 10 years.[7] This

Table 1. Suggested Duration of Oral Anticoagulant Therapy for VTE

Target INR = 2.5; range 2-3

Condition	Duration of Therapy
Surgically provoked DVT	3 months
Unprovoked lower extremity distal DVT	3 months
Non-surgical provoked proximal DVT or PE	3 months minimum; 6 months may be preferred, if no contraindications.
Unprovoked proximal DVT or PE	3 months minimum: 6 months is clearly preferable and extension beyond 6 months requires a careful risk-benefit analysis – (see text).
Cancer associated VTE	6 months therapy with LMWH or fixed dose UF. Warfarin – after 6 months on a risk-benefit analysis.

represents substantial risk for recurrence, although the case fatality rate for recurrent DVT (3.6%) is much lower than recurrent DVT while on early long-term anticoagulation (13%).[9] Certain features will be useful in making this decision and are summarized below.

Assessment of the risk of VTE recurrence versus the risk of a major or life threatening bleeding event.

Factors that favor extension of VKA therapy beyond 6 months:

a.) Previous VTE – A history of a previous VTE is generally considered an indication for *indefinite therapy*, unless contra-indicated.

b.) Lupus anticoagulant, anticardiolipin antibodies and anti-β_2 glycoprotein 1 – Among these tests, the lupus anticoagulant (functional assay) is far more important than the anticardiolipin antibodies (immunochemical assays): however, a positive anti-β_2 glycoprotein 1 antibody correlates with thrombotic risk. The relative risk for recurrence varies, but is of the order of 3-5. Hence, treatment for *two to five years* should be considered, unless contra-indicated.

c.) Hereditary thrombophilia: (see Table 2).

i. FV Leiden and prothrombin gene polymorphism: These increase VTE recurrence by approximately 1.4-1.7 and, therefore, are not 'hard' findings to influence duration.[5, 16]

ii. FVIII:C levels: high levels of FVIII appear a stronger risk factor for recurrence—2-4 fold.

iii. ATIII and protein C deficiency (and protein S deficiency) are rare disorders and recurrence risk is difficult to estimate. Family studies suggest that ATIII deficiency particularly and PC deficiency to some extent, are indicative of an increased risk.[17]

iv. Multiple (combined deficiencies e.g. FV Leiden plus PT polymorphism, PC and FV Leiden) are highly predictive of VTE recurrence.[18]

In summary, ATIII deficiencies, patients with multiple defects and, perhaps, PC deficiency could be considered for *anticoagulation_* for four to five years or possibly indefinite;[17] high FVIII:C *for two to five years*; other isolated findings are minimally predictive of recurrent risk.[16]

d.) Assessment of D-dimer: D-dimer levels are useful in the initial assessment of the acute situation, where a level below 0.5 FEU µg/mL is helpful in the exclusion of DVT and (less data), PE. D-dimer levels return to "normal" (negative) in 85% of patients on warfarin. The presence of a high D-dimer (>0.5 FEU ug/ml) 1 month after discontinuing VKA is indicative of an increased risk for recurrence on the order of 2-4 fold. Hence, elevated D-dimer levels would be an indication *for one to two more years*, at a minimum.[11]

e.) Short aPTT: A short aPTT (< 24 seconds) can also be indicative of an increase risk of recurrence (~ 2.0) independent of the elevated FVIII:C (FVIII:C is a major driver of the aPTT).[13]

f.) Pulmonary embolism: Patients with PE are at high risk of recurrence whenever anticoagulation is discontinued. About 50% of recurrences are PE's, and 10% of these will be fatal. Hence, *indefinite anticoagulation* (or *at least five years*)

Table 2.

I. Laboratory tests used to detect a hereditary or acquired thrombophilic state:

1. Activated protein C resistance or the FV Leiden mutation (G1691A):
2. Prothrombin Gene Polymorphism (G20210A):
3. Lupus anticoagulant and anticardiolipin antibodies
4. Protein C
5. Protein S – both Total and Free PS
6. Antithrombin III
7. FVIII:C
8. Fasting plasma homocysteine
9. HIT antibody in the correct context (heparin exposure and abrupt onset of an unexplained decrease in the platelet count—whether thrombocytopenic or not)

II. Laboratory tests used to assess the risk of VTE recurrence:

1. Lupus anticoagulant
2. D-dimer
3. aPTT
4. FVIII:C

III. Other tests which might be considered:

1. DNA tests for the methylenetetrahydrofolate reductase thermolabile polymorphism(MTHFR C677T and A1298C) or the PAI-1 gene 4G/5G polymorphism
2. CBC to detect Polycythemia Vera or essential thrombocytosis
3. Testing for PNH: red cell flow cytometry for CD 55 and CD59
4. Assay of FIX and FXI
5. Testing for dysfibrinogenemia – PT and Reptilase time
6. Testing for Wegener's granulomatosis (Pulmonary embolism)—cANCA against proteinase 3.

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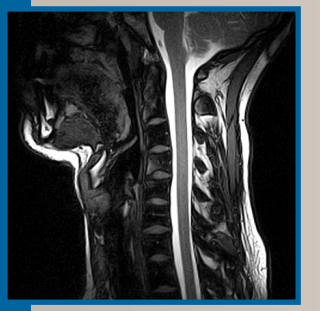

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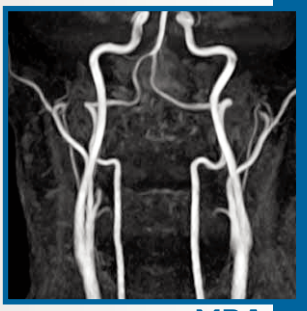

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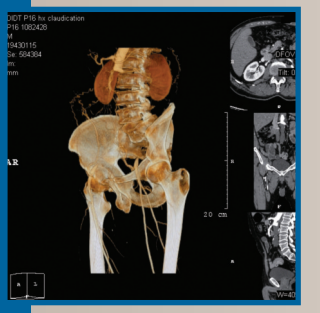

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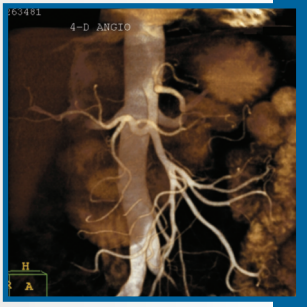

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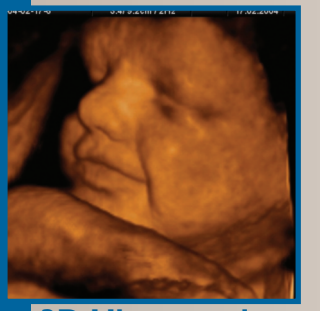

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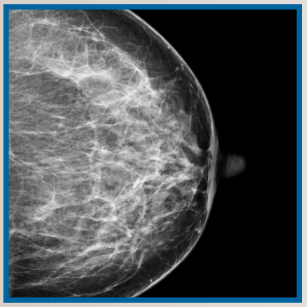

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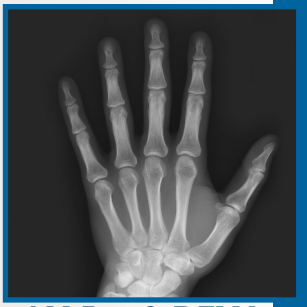

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should be considered in this population, especially if other risk factors for recurrence are present.[15]

g.) The presence of residual (unresolved) thrombosis by ultrasound after "completion" of therapy.[14]

h.) Presence of other persistent morbidity: Cancer is the most important, but SLE (especially with nephrotic syndrome), **paroximal nocturnal hemoglobinumic (PNH)**, inflammatory bowel disease, Cushing's syndrome and, in some, myeloproliferative disorders. These comorbidities could influence late long-term management decisions, e.g. for PNH-indefinite.

Factors that raise concern of an increased predisposition to bleeding:

a.) Hypertension, renal insufficiency, diabetes mellitus, hepatic disease, anemia and recent peptic ulcer may be associated with an increased risk of bleeding, as may age, cancer and ischemic stroke, although the last three are also associated with increased thrombotic risk. This risk is particularly present in the first 18 months;[7] hence, if warfarin has already been extended to 18 months without any bleeding, the risk of bleeding appears less after this time.

b.) Patient education, compliance and quality of medical supervision.

Patient preference:

Last, but by no means least, patient preference is a strong consideration. Some patients will have a morbid fear of recurrent VTE (especially PE) and will be prepared to accept late long-term or indefinite anticoagulation.

The above sections are intended as suggestions only based on known risk assessments, but are not supported by high quality evidence and, therefore, individualization of therapy is required.

In summary, there is no "one size fits all" duration for warfarin therapy and it is clear from the foregoing that multiple factors influence this decision. Failure to prescribe late long-term anticoagulation will result in a recurrent VTE in some patients; continuing to prescribe warfarin will result in major bleeding events in others.[9] The converse is, however, never obvious, as the prevention of VTE in individual patients on warfarin is, by definition, silent, as is the avoidance of major bleeding events that might have occurred in the presence of the VKA. Therefore, only *treatment-decision failures are evident* and physician and patient alike must accept this situation in order to put expectations in perspective and avoid misunderstandings.

This raises the important question of who to test?

What are the Indications for Vena Caval Filters?

The most feared complication of DVT is **pulmonary embolisation (PE)**. Initial treatment of DVT with heparin decreases the risk of fatal PE by 75% and the risk of recurrent PE from 25% to 2%. When heparin is completed, early long-term anticoagulation begins, but there is still a residual risk of recurrent DVT during this period. Furthermore, the case fatality rate for PE is higher during this phase.[9] Since most DVT's occur in the pelvic or lower extremity veins, interruption of venous flow by ligation of the common femoral vein, with or with thrombectomy, was performed in the 1950's. This approach was associated with limb edema. Later, ligation of the IVC was performed, with the ligation preferred below the renal veins. Edema remained a complication, however. This approach was modified in the 1960's with the use of suture plication and caval clips, which reduced the occurrence of edema. The initial IVC filters were developed in 1967 and released into wider use in 1972.

Approximately 13 Vena caval filters are available for use in the U.S. and an additional seven in Europe. The overwhelming majority are inferior vena caval filters introduced via the femoral vein. The best-known filter is the standard Greenfield Filter introduced in 1973. This filter is conical with six strands of zigzag shaped legs, each with a hook that anchors the filter to the IVC. The standard filter however, was ferro magnetic and has been replaced with a titanium Greenfield filter. A 20-year experience report shows a PE recurrent rate of 4% and a caval patency rate of 96%.[19] This is similar to subsequent reports and a relative risk of 0.41 at eight years for recurrent PE is achieved. More recent developments have resulted in the availability of several retrievable or optional IVC filters, although optional is a preferred term since they are intended for either temporary or indefinite placement.

Insertion of an IVC filter is never considered a routine approach. Long-term follow up shows that there are complications of early hematoma formation, late thrombosis at the site of insertion and the incidence of DVT is increased (RR about 1.8).

Therapeutic Indications for IVC Filters

1. Anticoagulation is contraindicated: Situations such as hemorrhagic stroke, active internal bleeding requiring transfusion, pregnancy, recent neurosurgical procedure or intracranial neoplasm, or hereditary or acquired bleeding disorder.

2. Anticoagulation complication: either bleeding or HIT, in a patient with an active DVT/PE.

3. Anticoagulant failure: recurrent PE despite apparently adequate anticoagulant therapy.

4. Incipient risk for embolisation. Presence of free-floating ileofemoral or lower IVC thrombosis.

5. Emergent surgery in a patient with DVT.

6. Patients with chronic pulmonary (thromboembolic) hypertension with marginal pulmonary reserve.

From Greenfield's series, the most common indication is #1 (about 50%), with #2 and #3 accounting for the rest.[19]

"Prophylaxis" (#5, 6) accounts for a minority (15%). It needs to be reinforced that high quality data (RCTs) is lacking to justify any of the above indications (where an IVC filter is used without concurrent systemic anticoagulants), but an RCT of IVC filters versus no filters in anticoagulated patient with acute DVT supports the contention that both PE and fatal FE are decreased by the IVC filter.[20] Given this, it is unlikely that a RCT comparing IVC insertion versus no filter will ever be performed.

What are the components of a "hypercoagulable workup" and when should one be performed?

Laboratory testing in the context of DVT can be divided into tests which are useful in *initial assessment*—mostly the D-dimer and fibrinogen concentrations - where the diagnosis is being considered or systemic profibrinolytic therapy is anticipated, and *later testing* where the objective is to determine the presence of an underlying hereditary or acquired thrombophilic state or to assess the risk of recurrence, although these are interrelated. These tests are shown in Table 2. The purpose of this section is to discuss appropriate testing and not to give a descriptive of each of these abnormalities—for this, the reader is referred elsewhere.[5]

In general, use of the tests to detect a thrombophilic state should only be performed on some patients with an unprovoked VTE or a non-surgical provoked VTE. Testing should be discouraged at the time of diagnosis or during immediate therapy or early long-term therapy. At those times, the results will not influence treatment, as erroneous results can occur because of the anticoagulant effects, which may cause confusion. Therefore, the tests should optimally be performed after completion of therapy . The patient should be off warfarin for a minimum of 2 weeks before any testing is performed, but one month is preferred. This clearly may be associated with some risk, if the patient is in a higher risk category. Because of this, some tests can be performed when the patient is on warfarin: the DNA based tests (FV Leiden and PT polymorphism); the immunochemical tests (anticardiolipin antibodies) and the functional assays for ATIII and FVIII:C. Some important assays cannot be properly interpreted when the patient is taking warfarin—of importance are the lupus anticoagulant assay and some assays for APC resistance. For this reason, it is "cleaner "to test after warfarin discontinuation and recommence warfarin for some defined time period if high risk is detected. It is recommended to perform all tests in the proband (but not necessarily family members) rather than selected high prevalent tests since multiple deficiencies are not uncommon[18] and the high prevalence defects (e.g. FV Leiden) provide much less information in themselves regarding continuation of therapy.[16]

This raises the important question of who to test? The following are suggested as general guidelines. Patient preference is again an important consideration:

1. A young patient (<50 years) with an unprovoked VTE or non-surgical provoked VTE.[5]

2. A patient with an unprovoked VTE or non-surgical provoked VTE in an unusual site—mesenteric veins, cerebral veins, possibly retinal veins.[5] Testing in patients with upper limb DVT should largely be confined to patients who do not have an anatomical thoracic outlet obstruction and where there is no recent history of upper limb strain such as heavy lifting or stretching (e.g. basketball). The true Paget-Schroeter Syndrome should be "spontaneous."

3. A woman who presents with pregnancy or puerperal associated VTE. This is of particular importance since it may influence the management of subsequent pregnancies. ATIII deficiency, although rare, typically presents in this manner and, in addition, it may influence a decision to test family members. Furthermore, certain findings may be important in understanding recurrent abortions in the proband or family members.

4. A patient with an unprovoked VTE. The tests indicated in table 2 (section II) can be helpful in risk assessment if anticoagulation is being considered for extension beyond six months. These are inexpensive tests that may guide the decision process.

5. Family members: Considerable caution needs to be exercised regarding the testing of family members and the preferences of each family member are important. Testing should be limited to any abnormality(ies) found in the proband and to hereditary traits, although rare familial lupus anticoagulants have been described. Family members need to be counseled that any finding (e.g. FV Leiden) may not be predictive of a future event in any clinically meaningful way, i.e., would not influence a decision regarding anticoagulation regimen or duration. As indicated above, ATIII deficiency would be an exception.

In general, patients with surgically provoked VTE, older patients, or patients with cancer should be discouraged from the testing described in Table 2 (section I), as should patients with recurrent DVT, since they are candidates for indefinite therapy regardless.

The role of the tests described in Table 2 (section III) is unknown at this time—a CBC is a simple test but others are more involved (Flow cytometry) or may be difficult to interpret (FIX and FXI) in terms of risk stratification.

Conclusion

VTE is a common systemic disease predominantly occurring in the later decades of life. Treatment is primarily systemic with oral anticoagulation for at least 3 months. Extension beyond three months is dependent on the circumstances surrounding the event, persistence of residual thrombus, any previous history of VTE, the results of laboratory testing, assessment of the bleeding risk, patient preference and anticipated compliance and the quality of medical supervision. There is a limited role for mechanical interruption of embolising thrombus with vena caval filters, primarily in patients with an active DVT for whom systemic anticoagulation is contraindicated. Esoteric testing should be largely reserved for patients who present in the earlier decades of life and may

be useful in determining the duration of therapy in some. Recently, more simple tests have shown clinical usefulness in risk assessment for recurrence.

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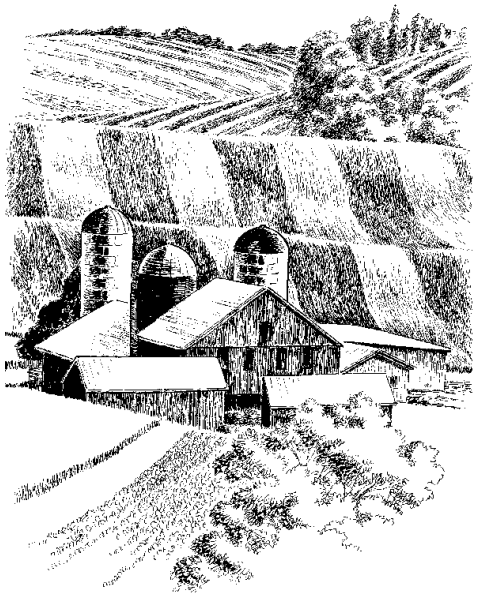

Acute Low Back Pain:
Imaging, Treatment, and Referral

Deus Cielo, MD, Heather Spader, MD, and Jonathan Grossberg, MD

Back pain is a very common complaint. It accounts for two to three percent of all physician visits in the US. According to two national surveys, the 2002 National Health Interview Survey and National Ambulatory Medical Care Survey, 26.4% of respondents reported a whole day of low back pain in the last three months and 13.8% had an entire day of neck pain during the same period.[1] Because low back pain is so common, it is important to be able to differentiate the features that distinguish serious from not serious conditions. This will lead to proper evaluation and treatment.

What imaging should be used to evaluate and manage acute low back pain and when should it be obtained?

One of the main goals in the evaluation of acute low back pain is to identify red flags that require a more detailed work-up and imaging. These red flags include the following:[4]

- Age under 20 or over 50. Age under 20 is a red flag because low back pain in this age group is unusual and therefore warrants further workup. Back pain in persons over age 50 is more common, but in this age group, malignancies are more common, therefore making pain in this age group concerning.
- History of cancer. The most common causes of metastatic tumors of the spine are lymphoma, lung, breast, and prostate cancers.
- Unexplained weight loss
- Immunosuppression. This includes patients with HIV/AIDS, transplant recipients, diabetics, and patients on chronic steroids, immunomodulators, and chemotherapeutic agents.
- Known infection
- Intravenous drug abuse
- Fever or chills
- Back pain not improved with rest
- History of significant trauma
- Prolonged use of steroids
- Acute onset of urinary retention or overflow incontinence
- Fecal incontinence or loss of anal sphincter tone
- Saddle anesthesia
- Global or progressive weakness of lower extremities

Any of the above conditions should warrant a more detailed evaluation for systemic disease as well as an MRI of the spine. Contrast enhancement is indicated only when an infection or tumor is part of the differential diagnosis.

One of the main goals in the evaluation of acute low back pain is to identify red flags that require a more detailed work-up and imaging.

Plain lumbosacral x-rays can be used in an acute trauma if no CT scanner is available. However, for fractures of the spine, CT is preferable, and for imaging of the spinal cord and soft tissue, MRI is the best imaging modality.

Of note, of the patients presenting with acute low back pain, over 95% do not need any imaging within the first four weeks of symptoms. The incidence of significant pathology in the remainder is as follows:[3]

- Compression fracture (4%)
- Cancer (0.7%)
- Cauda equina syndrome (0.04%)
- Spinal infection (0.01%)

The Joint Clinical Practice Guidelines from the American College of Physicians and American Pain Society make a strong recommendation against clinicians routinely obtaining imaging in patients with nonspecific low back pain.[3] In a meta-analysis of imaging strategies for low back pain performed by Chou *et al*, the authors found that increased frequency of lumbar MRI's is associated with higher rates of spine surgery, without a clear difference in patient outcomes.[2] Therefore, the treatment strategy for these patients is conservative management for the first four to six weeks.

Which treatments for low back pain are proven effective?

During the first four to six weeks of acute low back pain, patients are advised to remain active. In addition, books about self-care for back pain have been shown to be as effective as other therapies such as supervised exercises, acupuncture, massage, and spinal manipulation. Additional strategies include heating pads and firm mattresses.[3]

Although the evidence on the efficacy of these modalities is mixed, there is some proven benefit to spinal manipulation during the first four weeks of pain.[3] In a meta-analysis of exercise therapy for nonspecific low back pain, Hayden *et al* found that exercise therapy was as effective as either no treatment or other conservative treatments in acute low back pain.[5] Beyond the initial four-week period, the evidence for the best treatment modalities is very mixed with success found with physical therapy, acupuncture, massage, spinal manipulation, and self-education.

First line medications for acute low back pain should include nonsteroidal anti-inflammatory drugs (NSAIDs) and acetaminophen. Opioids can be used for severe pain that is unresponsive to NSAIDs.

When should a patient with low back pain be referred for specialty care?

After conservative therapy for four to six weeks, up to 90 percent of patients recover from acute low back pain. In one

study by Coste *et al*, a cohort of 103 patients with acute back pain was followed, and 90 percent recovered within two weeks. Of these 103 patients, only two developed chronic low back pain.[1] Of the patients who do not recover, those with the following symptoms would warrant further workup with MRI imaging:[4]

- Related leg symptoms and signs of nerve root compression
- Neurogenic claudication, which includes pain with ambulation that is improved by bending forward. It does not improve with rest, as does vascular claudication.
- Symptoms consistent with lumbar spinal stenosis, which includes pain or numbness radiating down the leg

MRI abnormalities may indicate the need for an orthopedic or neurosurgery evaluation. The absence of MRI explanations for the pain make a surgically remediable cause highly unlikely. If surgery is not recommended, a referral can be made to an interventional pain specialist. If the MRI has no findings, a consultation with a rheumatologist may be beneficial.

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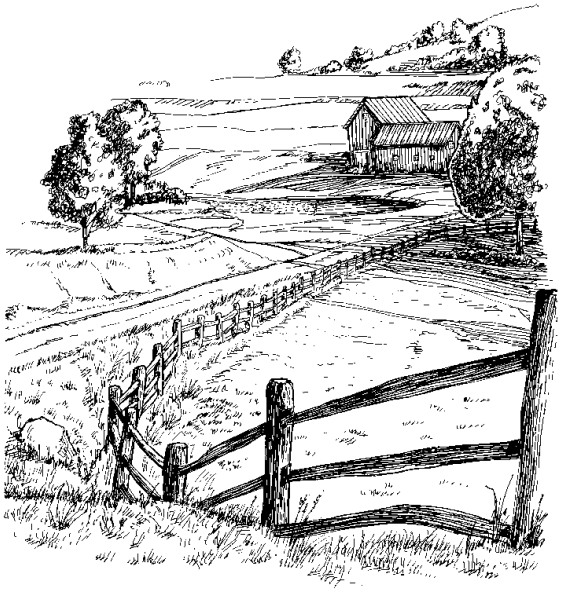

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EDITED BY ANA TUYA FULTON, MD

Electroconvulsive Therapy in Older Adults

W. Bogan Brooks, MD

CASE

Your patient is a 78 year old widowed woman with a history of hypertension, hyperlipidemia, osteoporosis, and major depressive disorder. She was doing well on verapamil, hydrochlorothiazide, aspirin, atorvastatin, calcium with vitamin D, nortriptyline, and trazodone until the death of her brother. During a family squabble over the allocation of the brother's assets, your patient develops chest pain, diaphoresis, and left arm heaviness & numbness. She is admitted to a hospital where workup including cardiac isoenzymes and dobutamine stress test is negative. She is discharged with a diagnosis of Atypical Chest Pain and referred for psychotherapy. Over the next few months, your patient develops poor sleep and low appetite. She can't concentrate to pay her bills, and stops going to the senior center for aerobics classes. She believes she is dying, fears being alone, and starts relentlessly telephoning you, her therapist and her son. You switch her from nortriptyline to mirtazepine, but she only gets worse. When you see the patient with her son in your office, she is contemplating suicide. Her son asks, "Isn't there anything you can do to help my mother?" Since your patient has failed trials of medication & psychotherapy and is suicidal, you refer her to a geriatric psychiatry inpatient unit for electroconvulsive therapy.

BACKGROUND

Electroconvulsive therapy (ECT) was invented in 1938 when two Italian physicians, Cerletti and Bini, used electricity to induce a seizure in their successful treatment of a schizophrenic patient suffering from catatonia.[1] In the early days of ECT, practitioners were reluctant to use ECT in older adults because of the multiple medical problems frequently found in this age group. But with procedural innovations such as oxygenation, general anesthesia, muscle relaxants, and physiological monitoring, ECT is now a safe and effective treatment for a number of medical conditions including: major depressive disorder, bipolar disorder, schizophrenia, schizoaffective disorder, neuroleptic malignant syndrome, and catatonia.[2] Despite its efficacy, the mechanism of action of ECT is unknown. One theory postulates that ECT works by targeting the hypothalamic-pituitary axis.[3] Another theory holds that ECT increases the amount of neurotransmitters in the central nervous system.[4] A third theory maintains that ECT produces changes in neurotransmitter receptor activity and density.[5]

"Is this the right treatment for my mother?" the son asks.

While several clinical considerations must be weighed in deciding whether ECT is appropriate for any given elderly patient, if a rapid response is needed because of severe psychiatric or medical morbidity, then ECT is a first line treatment.[6] ECT has the highest rate of response and remission of any form of antidepressant treatment, with up to 90% of patients showing improvement.[7] ECT should be considered for patients with major depressive disorder who have the following.

- Not responded to psychotherapeutic and/or pharmacologic interventions
- Psychotic features
- Catatonia
- Suicidal risk
- Food refusal leading to nutritional compromise
- Previous positive response to ECT
- Preference for ECT[8]

"What is the treatment like?"

A patient under consideration for ECT is seen in consultation by a psychiatrist, internist or family practitioner, and an anesthesiologist. If the patient is deemed to be suitable for ECT, the risks, benefits, and alternatives to the procedure are discussed. A consenting patient is not permitted to eat or drink after midnight on the evening before treatment. Some medications such as antihypertensives are permitted with a sip of water, while other medications such as anticonvulsants may be withheld. The patient lies on a stretcher and is attended by an anesthesiologist, a nurse, and a psychiatrist. Blood pressure, pulse oximetry, heart rate, and respiration are monitored. Electrocardiogram and electroencephalogram leads are attached. An intravenous line is started and a short acting anesthetic like methohexital is given to sedate the patient. Succinylcholine is given to induce paralysis, and a bite block is inserted to prevent dental injury. Electrodes are positioned in either bitemporal or right unilateral configuration, an electrical stimulus is applied, and the patient is carefully monitored. Just after the electrical stimulus, there is a brief parasympathetic outflow, which can cause bradycardia, hypotension, and asystole. [9] This vagal response can be attenuated by pre-treatment with an anticholinergic like glycopyrolate, which is the standard practice. The transient parasympathetic phase is followed by a sympathetic discharge, which can cause tachycardia and hypertension lasting up to twenty minutes. Excessive tachycardia can be alleviated with a beta blocker like esmolol, and excessive hypertension with nitroglycerine or other intravenous anti-hypertensive. Approximately 10% of patients will be given intravenous medications such as beta blockers to control these vital sign changes.

"Are there any side effects?"

There is a post-ictal confusion that lasts at least one hour after the seizure. Any post-ictal agitation can be alleviated with midazolam. Headache and muscle ache are common complaints that can be ameliorated with ibuprofen. Nausea is another common side effect that can be remedied by ondansetron. A less rare complication is dental injury and even rarer, is skin singing. These are carefully watched for, and a pre-ECT dental evaluation is always completed by the anesthesiologist and consulting physicians who do screening workup.

ECT is associated with a memory impairment that nearly always subsides. Anterograde amnesia typically resolves within one week to one month after the last ECT treatment, whereas retrograde amnesia may continue for up to six months. Rarely, mild residual memory complaints may persist in some patients but formal testing of patients has not documented persistent memory loss. Since older adults tend to have greater and more prolonged cognitive impairment with ECT,[10] it is sometimes necessary in elderly patients, particularly in those with pre-existing cognitive impairment, to employ unilateral electrode placement, lower electrical stimulus, and less frequent treatments.[11]

"What about her high blood pressure?"

Older adults referred for ECT frequently have pre-existing medical illnesses. While some illnesses increase the risk of ECT, none should be considered an absolute contraindication. The decision of whether to pursue a course of ECT should involve a careful weighing of the risks and benefits of treatment. The overall mortality of ECT is roughly one death per 80,000 treatments, a rate comparable to the use of general anesthesia in minor surgery.[12]

The majority of serious complications associated with ECT are cardiovascular in nature. In general, most adverse events can be prevented or limited by providing adequate oxygenation and strict control of heart rate and blood pressure. While individuals with congestive heart failure, active cardiac ischemia, severe valvular disease, uncontrolled hypertension, high grade atrio-ventricular block, and arrhythmias are at increased risk of cardiac complications following ECT; ECT has nonetheless successfully been carried out in many of them.[13] Other conditions that increase the risk of ECT include:

- Space occupying cerebral lesions or other conditions with increased intracranial pressure;
- Recent cerebral hemorrhage or infarction;
- Bleeding or otherwise unstable vascular aneurysm or malformation;
- Retinal detachment;
- Pheochromocytoma;
- Anesthetic risk rated at ASA level 4 or 5;
- Severe pulmonary condition[12]

In older patients, dosages of anticholinergic, anesthetic, and relaxant agents may need modification because of the physiologic changes associated with aging. Stimulus intensity should be selected with an awareness that the seizure threshold increases with age. Because patients with diabetes are prone to have hypoglycemia with fasting before ECT, insulin doses may need to be adjusted. In patients with asthma or COPD, the risk for post-treatment bronchospasm can be mitigated by pre-treatment with bronchodilators. Patients with osteoporosis and unstable fractures can safely be treated with ECT by using an increased dose of succinylcholine to ensure adequate relaxation.

"How long is the treatment?"

Evaluation of the patient's symptoms and any adverse reactions to treatment is needed to determine the efficacy of a course of ECT.[14] In an index episode of illness, ECT is ordinarily administered three times per week on nonconsecutive days, and is continued until the patient reaches a plateau in improvement over two treatments. Most patients reach this plateau within six to twelve treatments.

Without further treatment, half of all patients will relapse within six months, so continuation/maintenance therapy, typically consisting of psychotropic medication or ECT, is indicated for virtually all patients.[12] Continuation ECT refers to the practice of giving additional treatments at a reduced frequency for six months after discontinuation of the index course of treatment. Maintenance ECT refers to treatments administered beyond the continuation phase.

In a study of the efficacy of continuation ECT and antidepressant drugs compared to long-term treatment with antidepressants alone, the findings provided strong support for the efficacy of continuation ECT plus long-term antidepressant treatment in preventing relapse and recurrence in chronically depressed patients who have responded to acute treatment with ECT.[15] In a study of maintenance medication comparing placebo, nortriptyline alone, or nortriptyline combined with lithium in depressed patients after index phase ECT, only the combination of nortriptyline and lithium significantly reduced relapse rates.[16] Further research focusing on augmentation strategies to protect against relapse following ECT in depressed older adults is needed.

Conclusion

Your patient elects to pursue ECT. After six treatments, her depression improves and she is no longer suicidal; but she develops confusion, which abates when the frequency of her treatments is decreased from three times weekly to twice weekly. After the ninth treatment, her depression resolves. Your patient is started on venlafaxine and discharged home. When you see her in your office, your patient is tolerating the venlafaxine and doing well on an outpatient course of continuation ECT. Her goal is a trial of venlafaxine alone after she completes continuation ECT, with an option of switching to maintenance ECT if the venlafaxine trial is unsuccessful.

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Rhode Island Department of Health • David Gifford, MD, MPH, Director of Health
Edited by Samara Viner-Brown, MS

Sexual Behavior and Contraceptive Use among Rhode Island Adolescents

Hyun (Hanna) Kim, PhD, Rosemary Reilly-Chammat, EdD, and Tricia Washburn, BS

Teen pregnancy rates in the United States have declined substantially since the early 1990s in all states including Rhode Island, mainly due to positive changes in teens' sexual behavior and contraceptive use.[1,2] However, recent data show that the significant progress the nation has made in reducing teen sexual activity, improving contraceptive use, and providing better sex education in school has stagnated.[3,4] Many teenagers engage in behaviors that put them at risk of pregnancy and sexually transmitted diseases (STDs). Nationally, about 750,000 teens become pregnant each year, almost one third of girls become pregnant at least once by age 20, and approximately one in four sexually active young adults ages 15 to 24 contracts an STD annually.[1,5] Teen pregnancy and STDs are an important public health issue since they pose a serious risk to the health and well-being of teens and their babies, and to society as a whole.

This report describes the sexual and contraceptive behaviors that contribute to teen pregnancy and STDs among Rhode Island public high school students.

Methods

Data from the 2009 Rhode Island Youth Risk Behavior Survey (YRBS) were analyzed to determine sexual and contraceptive behaviors among high school students. The YRBS, developed by the Centers for Disease Control and Prevention (CDC), monitors health risk behaviors related to the major causes of mortality, morbidity, injury, and social problems among high school students.[4] The Rhode Island Department of Health, sponsored by the CDC, conducts the YRBS every two years from a representative sample of public high school students using self-administering survey. In 2009, a total of 3,213 students 9th through 12th grades participated in the Rhode Island YRBS with a 67% response rate.

Sexual behaviors included in this report were: 1) whether students ever had sex; 2) whether they had sex in the past 3 months; 3) whether they had first sex before age 13; 4) whether they had sex with 4 or more partners in life; 5) whether they were ever forced to have sex; and 6) whether they were ever taught about AIDS/HIV infection in school. The prevalence of each of these indicators for Rhode Island students was compared to national rates. Contraceptive behavior was determined by asking "The last time you had sexual intercourse, what one method did you or your partner use to prevent pregnancy?" This question was asked only of those reporting they ever had sexual intercourse.

The percentage of students who ever had sexual intercourse and the percentage of student who did not use birth control method at the last time they had sex were examined by race/ethnicity, gender, grade, primary language used at home, academic performance, sexual orientation, and disability status, and were presented with the p-values from the Chi-square tests. Data analyses were performed using the SUDAAN software, which accounts for the complex sample design of the survey. All percentages presented here were weighted to represent the public high school student population statewide. Unknown and refused responses were excluded from the analyses.

Results

Sexual Behaviors

Overall, 44.2% of RI public high school students reported that they ever had sexual intercourse (U.S.: 46.0%); 32.3% had sex in the past 3 months (U.S.: 34.2%); 5.2% had first sex before age 13 (U.S.: 5.9%); 11.2% had sex with 4 or more people in life (U.S.: 13.8%); 7.1% were ever forced to have sex (U.S.: 7.4%); and 13.3% were never taught about AIDS or HIV infection in school (US: 13.0%). (Figure 1) There were no statistically significant differences between Rhode Island and U.S. students in any of these measures.

The percentage of Rhode Island high school students reporting they ever had sexual intercourse was significantly higher among non-Hispanic Blacks (53.2%), Hispanics (52.4%), 11th-12th graders (57.5%), students with mostly D's and F's (71.4%), students identifying themselves as lesbian, gay, bisexual, or unsure sexual orientation (LGBU: 59.3%), and students with disabilities (50.7%), compared to their counterparts. (Table 1)

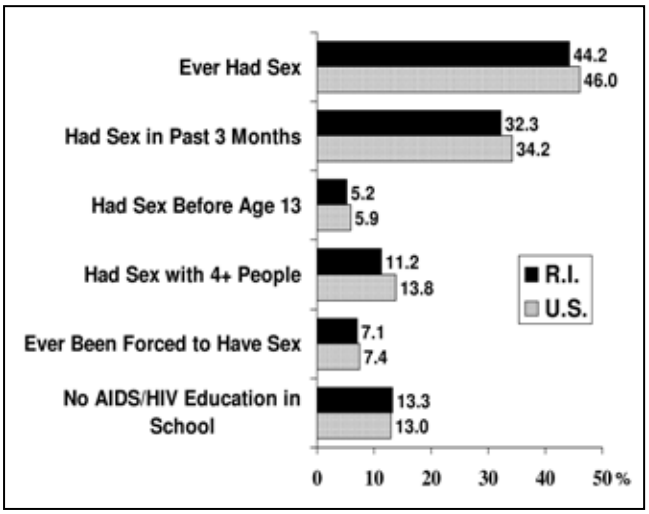

Figure 1. Sexual Behaviors among High School Students, Rhode Island vs. United States.

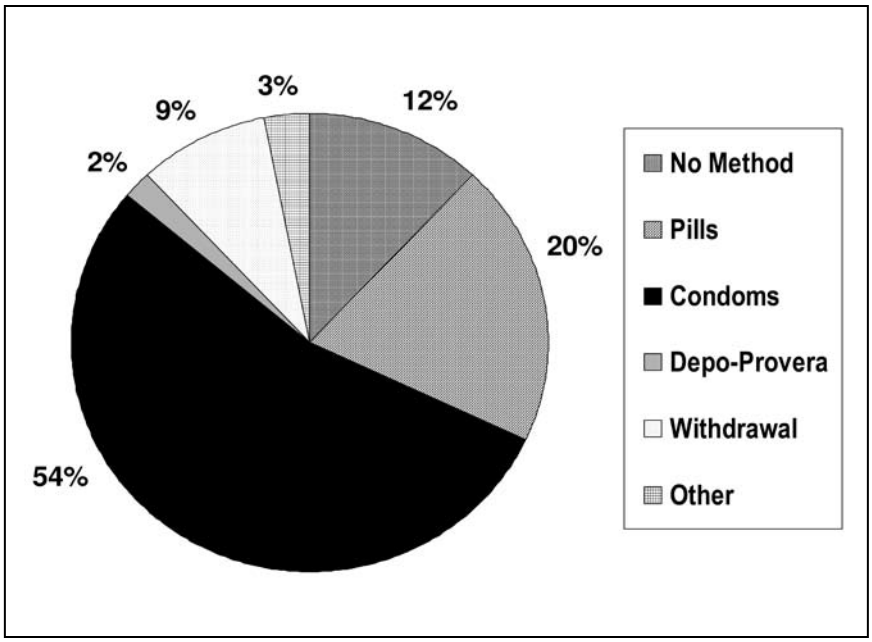

Figure 2. Birth Control Methods Used at Last Sex Among High School Students Who Ever Had Sexual Intercourse.

Table 1. Percentages of high school students who ever had sexual intercourse and who did not use contraceptive method at the last time they had sex by selected characteristics, Rhode Island, 2009

	Ever Had Sexual Intercourse		Not Used Contraceptive Method at Last Sex[1]	
	%	*P*-Value	%	*P*-Value
Overall	**44.2**		**12.3**	
Race/Ethnicity				
White, non-Hispanic	41.2	0.0324	8.7	0.0002
Black, non-Hispanic	53.2		17.9	
Hispanic	52.4		21.3	
Other[2]	41.1		16.6	
Sex				
Male	45.6	0.0567	11.0	0.1170
Female	42.8		13.6	
Grade				
9th-10th	33.4	<0.0001	14.4	0.1308
11th-12th	57.5		10.7	
Language used at home				
English	43.5	0.1240	10.8	0.0052
Non-English	48.1		20.6	
Academic performance				
Mostly A's & B's	35.3	<0.0001	9.3	0.0219
Mostly C's	57.5		14.0	
Mostly D's & F's	71.4		17.5	
Sexual orientation				
Strictly hetero-sexual	42.9	0.0028	11.4	0.0446
LGBU[3]	59.3		20.3	
Disability status				
No disability	42.6	0.0491	11.6	0.2186
Have disability	50.7		15.1	

Data Source: Rhode Island Youth Risk Behavior Survey, 2009
[1] Among students who have ever had sex
[2] Other category includes American Indian/Alaska Native, Asian, Native Hawaiian/other Pacific Islander and multiple race, non-Hispanic
[3] LGBU includes lesbian, gay, bisexual, and unsure sexual orientation

Contraceptive Behaviors

Among Rhode Island high school students reporting they ever had sexual intercourse, 12% did not use any contraceptive method at the last time they had sex; 20% used birth control pills; 54% used condoms; 2% Depo-Provera (injectable birth control); 9% withdrawal; and 3% other method. (Figure 2) It should be noted that about 20% of students refused to respond to this question, and they were excluded in the analysis.

The percentage of Rhode Island high school students who did not use contraceptive method at the last time they had sexual intercourse was significantly higher among non-Hispanic Blacks (17.9%), Hispanics (21.3%), students whose primary language used at home is not English (20.6%), students with mostly D's and F's (17.5%), and students identifying themselves as LGBU (20.3%), compared to their counterparts. (Table 1)

Discussions

Our findings indicate that many Rhode Island high school students are engaging in sexual risk-taking behaviors and in unprotected sex that can lead to pregnancy and STDs. Early onset of sexual activity, involuntary sexual activity and having multiple sex partners all increase the risk of becoming pregnant and contracting an STD. Contraceptive methods used by teens should be carefully reviewed. Latex condoms are the only form of birth control that reduce the risk of STD transmission and pregnancy, and must be used every time. Other contraceptive methods, such as birth control pills and Depo-Provera (injectable birth control) may help prevent pregnancy, but they don't protect against STDs.

One important limitation of this report is that the YRBS did not include adolescents who dropped out of high schools, and these adolescents may be much more likely to engage in behaviors that put them at risk of pregnancy and STDs. Further study is needed to understand the risk behaviors of these adolescents as well as those who refused to answer the contraceptive method question on the YRBS.

Access to comprehensive and confidential health services is the key to preventing unintended pregnancies and STDs among adolescents. Medical

homes, where teens are known well by their primary care provider, can offer a safe environment to promote healthy behaviors and reduce risk-taking behaviors especially as it relates to sexual behavior and birth control methods. Primary care providers can familiarize themselves with community based resources such as Title X providers and mental health providers, along with support for school and community based efforts around comprehensive sex and family life education and community service programs, all of which can contribute to optimal health outcomes for teens.[6]

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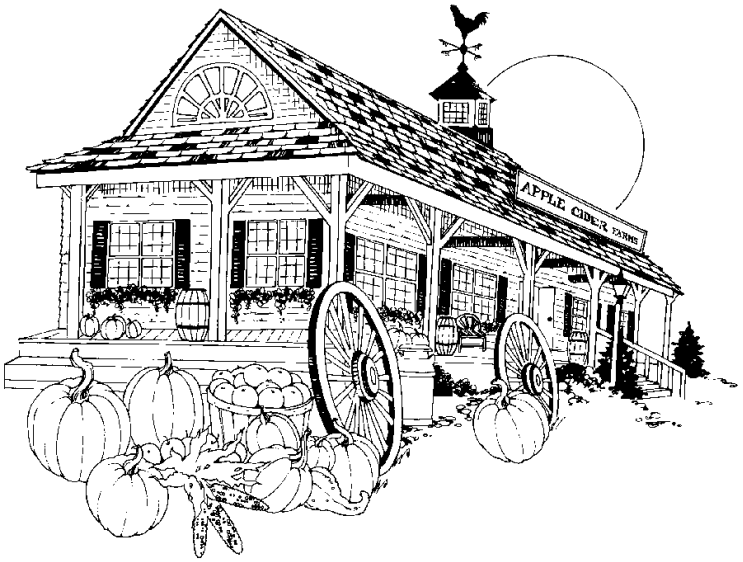


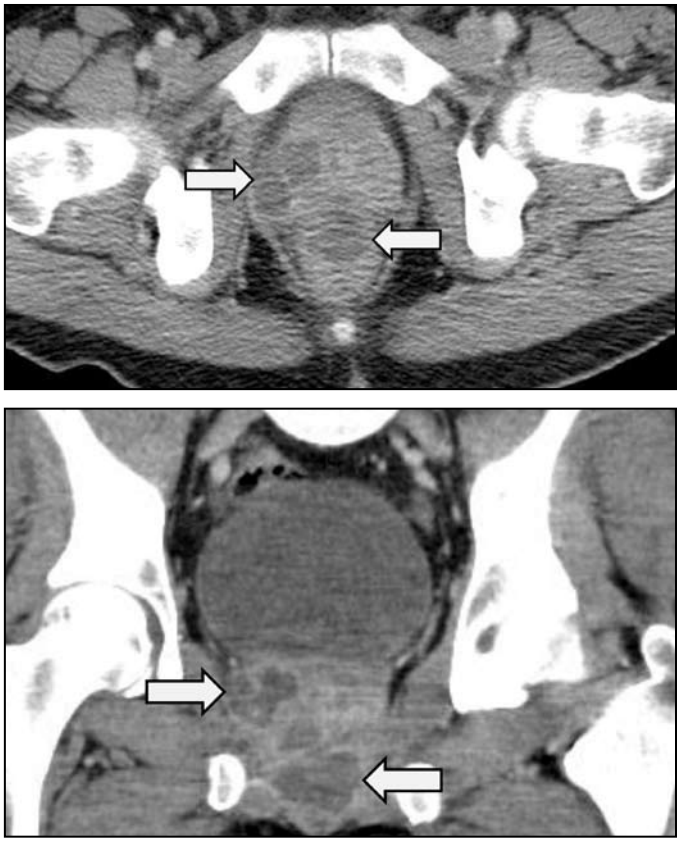
Images In Medicine

Prostatic Abscess

Sammy Elsamra, MD, Patrick Kelty, MD, and Steven Schechter, MD

A 44 year-old man presented to the Emergency room with abdominal cramping pain, fever, and diarrhea. He had an unremarkable medical and surgical history. The patient denied smoking, alcohol abuse, and illicit drug use, is married, and works as an accountant. His physical exam was remarkable for a fever to 102.7 °F, tachycardia, and a tender anterior rectal wall and boggy prostate on digital rectal exam. His **white blood cell count (WBC)** was 19,000/mcL and **urine analysis (UA)** was negative for leukocyte esterase, nitrites, blood, bacteria, WBC or **red blood cells (RBCs)**. A **computed tomography (CT)** of the abdomen and pelvis was performed and revealed multiple low attenuation densities involving the right lobe of the prostate gland as well as the right peri- prostatic and infra prostatic regions extending into the base of the penis. He was diagnosed with a prostatic abscess, placed on **intravenous (IV)** antibiotics, and taken to the operating room for a cystoscopy, suprapubic tube insertion, and transuretheral resection of a prostatic abscess. Examination under anesthesia revealed only a boggy prostate. Blood cultures and urine cultures were remarkable for methicillin sensitive staphylococcus aureus. Post-operatively, the patient was noted to have elevated blood sugar levels and was diagnosed with untreated diabetes and placed on insulin per medicine consultation. Infectious Disease consultation was obtained. He was found to be HIV, gonorrhea, chlamydia, and **rapid plasma reagin (RPR)** negative and with a negative echocardiogram. He was treated with IV cefazolin for two weeks.

We present this case as prostatic abscesses are uncommon in the modern antibiotic era. In addition, prostatic abscess symptoms can be confused with rectal symptoms and careful attention to physical exam and CT imaging should help clinicians properly diagnose this uncommon illness. Rectal examination in a systematic fashion may more precisely identify the cause of the patient's symptoms.[1] Currently, prostatic abscess is diagnosed in only 0.2% of patients with urological symptoms and in 0.5 to 2.5% of those hospitalized for these symptoms. Risk factors cited are diabetes and immunosuppression. The most common pathogens are E coli and staphylococcus.[2] Typically these abscesses are treated with IV antibiotics and one of three modalities of drainage—percutaneous drainage (typically CT guided), transrectal ultrasound guided drainage, or transuretheral drainage by cystoscopic unroofing of overlying prostate.



Axial (upper) and coronal (lower) CT images with arrows depicting prostatic abscess fluid loculations.

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Sammy Elsamra, MD, and/or their spouse/significant other has no financial interests to disclose.

Patrick Kelty, MD, is on the Speakers Bureau for Pfizer.

Steven Schechter, MD, is on the Speakers Bureua for GlaxoSmithKline, and received Grant Research Support from Ethicon.

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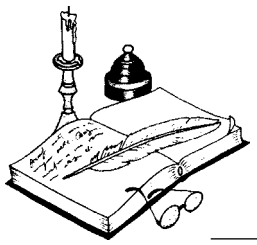

Physician's Lexicon

A Medical Menagerie

Through analogy and metaphor the names of certain denizens of earth, air, and sea have found their way into the physician's lexicon. Lupus erythematosus, with its characteristic butterfly lesion, takes its name from that attacker of flesh, the wolf (L. *lupus*). The rough and furrowed skin of leprosy reminded Aretaeus of Cappodocia of an elephant's hide so he called the disease *elephantia*, a term no longer used for that affliction but surviving today in elephantiasis, the grotesque swelling caused by a filarial worm. Scaly skin caused by a variety of diseases is captured by the term icthyosis (Gr. *ichthys*, fish), while circumscribed regions of baldness looked to the Greeks like mange on a fox (Gr. *alopex*), hence alopecia. Latin *tenia* (worm) is found in the names of diseases caused by real worms but ringworm, *tenia capitis*, is a fungal infection. The Arthopodia give us formication (L. *formis*, ant), the feeling that ants are crawling on the body, and the web-like varicosities in the skin called spider veins. Cancer and carcinoma descend from the Greek word for crab (*karkinos*) and one variety is the rodent ulcer, the ulcer that gnaws, as do members of the order Rodentia, one of whom contributes the term hare-lip. From the barnyard come chickenpox, swine flu, and mad-cow disease, and vaccination from Latin *vacca*, cow. A deletion at chromosome 5p is responsible for the cri-du-chat syndrome and a deformity of the thorax that pushes the sternum forward (L. *pectus carinatum*) carries the vernacular name pigeon breast. Phocomelia, a developmental anomaly characterized by flipper-like limbs, combines two Greek words: *phocia* (seal) and *melea* (limb). To the animal kingdom we owe, lexically at least, our canine teeth and ears with a concha (Latin, mollusk) on the outside, a cochlea (Latin, snail) on the inside and the hirsute tragus that reminded someone of the tuft on the goat's (Gr. *tragos*) chin. Not to be left out, the skull can claim a seahorse in the temporal lobe (Gr. *hippocampus*), a worm in the cerebellum (L. *vermis cerebelli*), a horses tail in the spinal canal (L. *cauda equina*), a rooster's crest on the sphenoid bone (L. *crista galli*), and his spur on the occipital cortex (L. *calcar avis*).

– James McIlwain, MD

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Rhode Island Department of Health
Michael Fine, MD
Interim Director of Health

Vital Statistics

Edited by Colleen Fontana, State Registrar

Rhode Island Monthly Vital Statistics Report Provisional Occurrence Data from the Division of Vital Records

Underlying Cause of Death	Reporting Period			
	April 2010	12 Months Ending with April 2010		
	Number (a)	Number (a)	Rates (b)	YPLL (c)
Diseases of the Heart	188	2,327	220.9	3,124.5
Malignant Neoplasms	190	2,238	212.5	6,232.5
Cerebrovascular Diseases	38	459	43.6	840.0
Injuries (Accidents/Suicide/Homicde)	52	619	58.8	10,652.5
COPD	38	492	46.7	550.0

Vital Events	Reporting Period		
	October 2010	12 Months Ending with October 2010	
	Number	Number	Rates
Live Births	1,006	11,863	11.2*
Deaths	818	9,102	8.5*
Infant Deaths	(1)	(72)	6.0#
Neonatal Deaths	(4)	(62)	5.2#
Marriages	828	6,114	5.7*
Divorces	338	3,300	3.1*
Induced Terminations	297	4,196	352.0#
Spontaneous Fetal Deaths	27	586	49.2#
Under 20 weeks gestation	(20)	(522)	57.4#
20+ weeks gestation	(7)	(64)	5.4#

(a) Cause of death statistics were derived from the underlying cause of death reported by physicians on death certificates.

(b) Rates per 100,000 estimated population of 1,053,209. (www.census.gov)

(c) Years of Potential Life Lost (YPLL).

Note: *Totals represent vital events that occurred in Rhode Island for the reporting periods listed above. Monthly provisional totals should be analyzed with caution because the numbers may be small and subject to seasonal variation.*

* Rates per 1,000 estimated population
\# Rates per 1,000 live births

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Ninety Years Ago, April 1921

Dr. Creighton W. Skelton brings to light before the March meeting of the Rhode Island Medical Society a number of disturbing cases regarding factory owners under-reporting workplace accidents by having nurses and other first-aid specialists see patients outside official doctors' hours in which to avoid having said incidents recorded. He notes that reporting this practice to the management resulted in his dismissal and while a formal report to the Board of Health resulted in a return to proper procedures, it was only a short-lived return. He then cites a number of cases in which when a patient is seen only by a nurse or a first aid attendant, and then returned to the production floor, the patient often worsens and what would have been a simple visit with the company doctor has turned into something much more serious.

Dr. Skelton also makes a call for higher quality and greater diversity in papers presented to the Medical Society. Among his entreaties are: " Let the x-ray men show their light, and let not the Otologist forget that the general man is as much interested in otitis media as he himself is. As a diversion a paper by Garvin on Single Tax would be entertaining. You could play safe by limited him to thirty-five minutes."

In a journal article, Dr. Jacob Kelley encourages the use of x-rays to discover fractures frequently overlooked and of the importance of an experienced Roentgenologist in reading and interpretation.

In an editorial there is a call for reform in the State of Rhode Island in regards to the duties and powers of Medical Examiners and the law under which they operate. Too often, Medical Examiners make pronouncements of death by natural causes without the benefit of autopsies or detailed examinations. This practice is due mostly to the costs associated with autopsies which cannot be performed without the approval of the Attorney General who, in turn, will not grant permission unless strong evidence is presented of death by anything other than natural causes. The author calls for a greater role for the State Pathologist in performing autopsies as typical Medical Examiners are not qualified, and that the cost is not too great for the State.

Dr. Robert B. Greenough of Boston addressed the Providence Medical Society on "The Modern Use of Radium," which was "a careful, straight-forward statement on the different effects of radium on living tissue the manner of using it and the present view of its value in the treatment of cancer and other neoplastic conditions in the human body."

Fifty Years Ago, April 1961

In a report prepared by the Washington Office of the American Medical Association, it states that the US Public Health Service and the National Foundation are working together in an all-out drive to get as many persons as possible to take Salk vaccine shots before the summer polio season starts. It further notes that the Sabin live polio vaccine is in short supply and that the Salk vaccine drive is aimed mostly at children and young adults in lower economic groups.

Dr. Laurence A. Senseman suggests that psychiatry is being oversimplified—mostly by drug companies eager to sell their products. The current rage is for "psychic energizers" from a group known as mono-oxidase inhibitors. Dr. Senseman goes on to identify different types of depression and treatments.

Dr. Seebert J. Goldowsky presents excerpts from his "Israel Diary" in which he relates his experiences as part of a hospital-to-hospital medical exchange program between Miriam Hospital and Poriah Government Hospital in Tiberias, Israel. He is impressed both by the industry of the people, and the sights and foods of the area.

Dr. Reuben C. Bates discusses certified milk in Providence and outlines the various classifications and standards available.

In "Through the Microscope," it is reported that Dr. David Littman of West Roxbury, MA VA hospital and Harvard Medical School and Dr. Hubert V. Pipberger of Mt. Alto VA hospital in Washington, DC and Georgetown School of Medicine have used a computer to screen electrocardiograms for heart abnormalities with a 95-100% accuracy rate in diagnostic decisions.

Twenty-Five Years Ago, April 1985

Dr. Richard H. Egdahl asks "Should We Shrink the Health Care System?" and suggests that a way to do so would be to reduce the number of hospital beds and healthcare providers. The author notes how many companies are restructuring their medical plans to include incentives to reduce the use of copayments, deductibles, and less comprehensive coverage.

Robert J. Canny, Executive Director of Hospice Care of Rhode Island, writes about the history of hospice care benefits under the Medicare program. He notes their ten-year history in the United States, and how Hospice Care of Rhode Island was the first certified Medicare program in New England.

In a recent issue of the American Journal of Diseases of Children, Dr. Karen M. Kaplan of the University of Pennsylvania School of Medicine suggests that herpes infections in children should "always be considered as a possible indicator of child abuse." She and her colleagues note that sexual abuse was documented in four of six children under the age of 13 years who were treated for genital herpes simplex virus.

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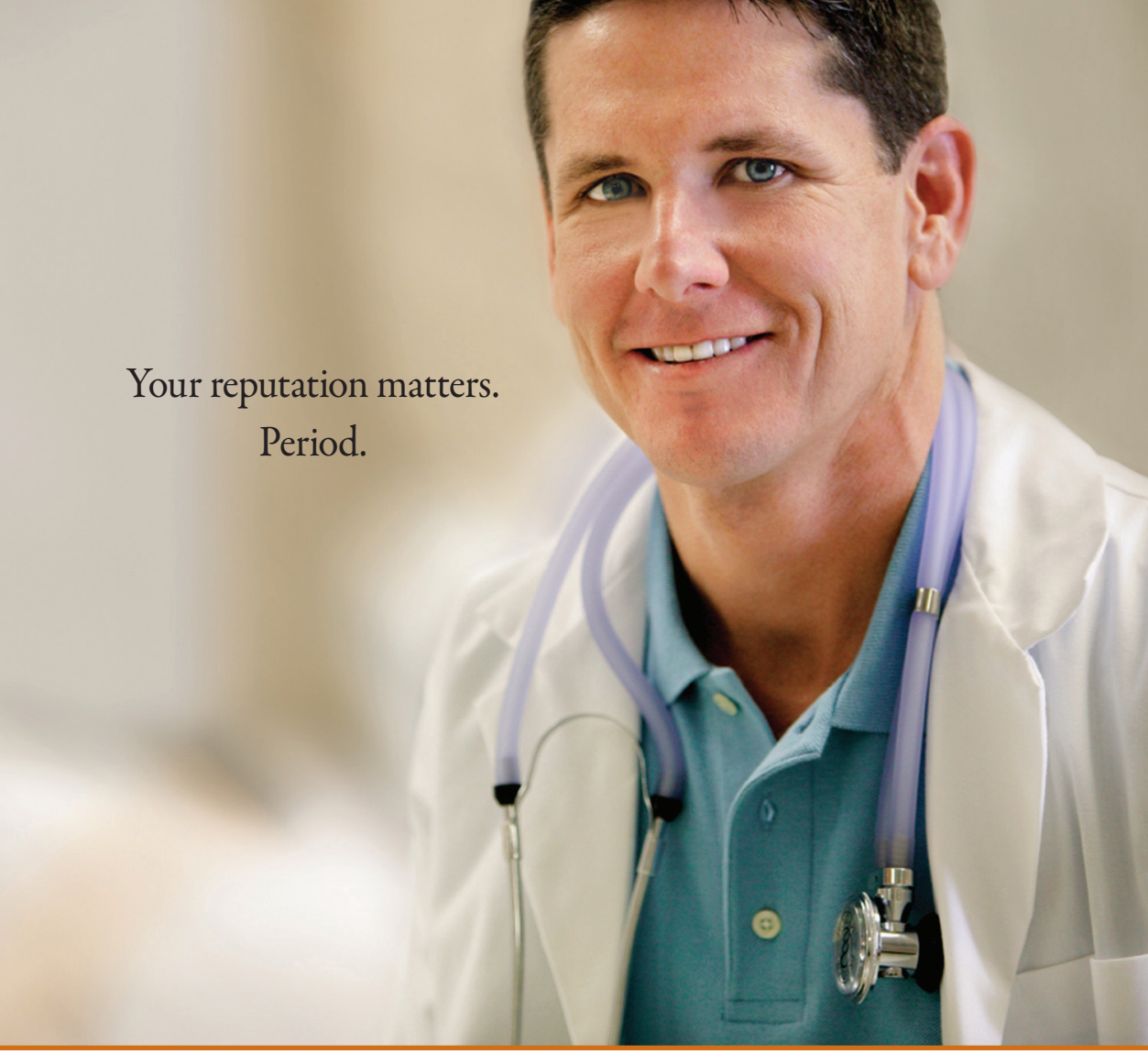

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