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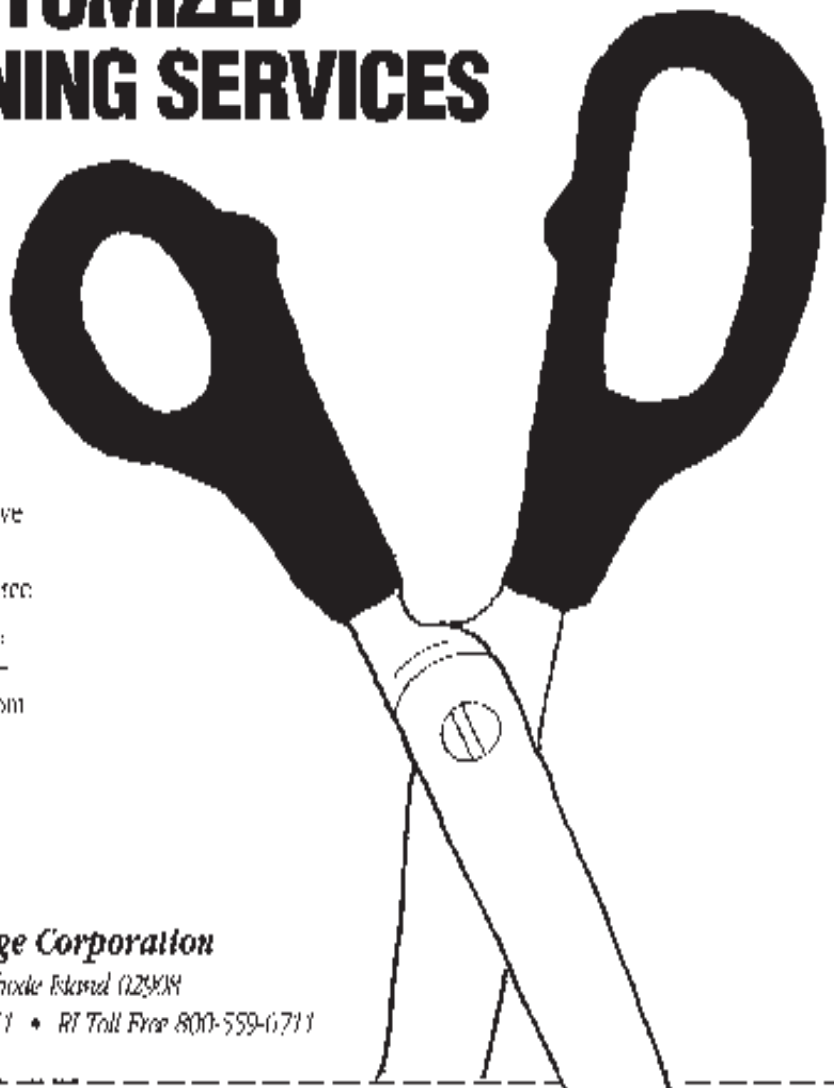

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Cover: "Nana," mixed media, by Toba Weintraub, a Rhode Island artist and educator, who conducts art programs with seniors.
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Commentaries

Slights

Slights weigh on my mind. I think this is a moral shortcoming, another flaw in my character. I'm referring to professional insults. I really don't get upset when people who should know me fail to say hello or look through me at the supermarket or the hospital hallway, or even when my doctor's secretary calls my name, "Joe," as the next patient, or when I'm overlooked at a posh store because I look unlikely to be purchasing something expensive.

A few years ago a doctor called to make an appointment to meet with me to discuss a research project. I was pleased and we scheduled a time a week hence. A couple of hours before the appointment the doctor called. "Joe, I was supposed to meet with you and then with Dr. X at your hospital, but Dr. X is out of town so I'm not coming. Why don't you come here?" Dr. X was a VIP while I was new to the area and a junior person. Yet I hadn't made this appointment. I had not asked him to free up time for me. The Yiddish word for this is "chutzpah," loosely synonymous with gall or arrogance. Obviously the appointment with me was an "add on," made only because of a more important appointment at my hospital. Clearly I didn't count. "Oh, by the way, I would be willing to meet with you if you came here, but you're not important enough to merit a visit from me, even though I scheduled it."

Once I called another doctor about a VIP patient he had referred to me for chronic headaches. I reported an early, dramatic success but cautioned that it was probably a temporary, placebo effect, a "honeymoon" happening. "You're not so dumb," was the response. I guess it was meant in jest, but it still strikes me, years later, as an odd comment to make to a consultant who took on a difficult patient on an urgent basis.

More recently I was invited to undertake a major project and needed a partner in another discipline. I had asked a moderately senior physician to help but he declined because of other commitments. However, he offered to think of appropriate substitutes. A week later I got a call from a resident physician. "Dr. Y told me you needed help on this project…" One professor asks another to partner a project, on equal terms, supervising a large number of senior physicians, and the professor suggests a doctor in training. The inappropriateness of his proposal had two aspects. On the face of it, it was ludicrous. One would not submit a grant proposal, co-edit a book, or develop a research center with a house officer. On the other hand it was insulting. It was clear that I was being told, in not so subtle terms, what my project was worth.

We all need respect. We are taught to respect our patients but we need to respect our colleagues and our staff as well. We all have flaws and some will hurt others accidentally through oversight, arrogance and insensitivities of one sort or another. I can write these columns about them, which helps me. People I've insulted may read this and think that the writer meant well, even if not actually achieving it. On the other hand the reader may say, "Boy, he sounds like a sensitive guy but he's really a jerk in real life."

The real-life people I described might read this, but they won't see themselves in this article if they do. That's the nature of insensitivity. I know that one of my flaws is the inability to forget (and probably to forgive fully). But what can I do about the people I've hurt without recognizing it?

I received a letter recently in response to a consultation note. The doctor wrote a very cogent note explaining that he understood my recommendations, but that he hadn't instituted some for a variety of reasons and could not institute others. My job is to assess and either make recommendations or institute treatment myself. When I consult for other specialists about problems that are in an overlapping terrain I almost always make suggestions based on extensively explained reasons. I try to make clear that I am a neurologist speaking only from my narrow perspective, and that I understand there are wider imperatives. So I was unnerved by this letter, because there was an implication that was clear to me, although possibly not to others, that the doctor was insulted by my note. In some way I was either suggesting a level of incompetence on his part, or an arrogance on mine, that I knew better, so he "should" do what I suggested. Hopefully specialist consultants do know more than the referring doctor, that's why the consultation was requested. However, the consultant isn't always right, and frequently, even when making the "medically correct" recommendation in his field, may still not be suggesting the best choice for this particular patient. I try to take pains to explain why I think certain things need to be done, to outline choices and to describe what the different choices may lead to. All doctors do this. When we become patronizing, we lose referrals. When we are specialists in disciplines so narrow that our referring doctors have no choice in the RI area but to refer to us we have to take special pains to avoid being seen as stiff-necked and arrogant. I must have fallen down on the job.

– Joseph H. Friedman, MD

DEDICATION

This edition of *Medicine & Health/Rhode Island* is dedicated to the memory of Dr. Elise Coletta, who passed away on October 1st, 2003 after a brief illness. Dr. Coletta devoted her medical career to teaching residents and medical students about geriatrics, women's health and primary care. In her tenure at Memorial Hospital of Rhode Island, Dr. Coletta was the Director of Medical Care of the Rehabilitation Unit from 1986 to 1993, Assistant Director of the Division of Gerontology and Rehabilitation from 1989 to 1993, and Chief of Gerontology from 1993 to 2000. In 2001, Dr. Coletta became Medical Director of the Memorial Hospital of Rhode Island Homecare Program and Associate Medical Director of Medicare at Blue Cross/Blue Shield of Rhode Island. In addition, she was an active member of the Rhode Island Academy of Family Physicians and of the Rhode Island Medical Women's Association. She was Clinical Associate Professor in the Department of Family Medicine at Brown Medical School. Her pursuit of excellence and unrelenting service to the care of older adults and the training of resident physicians made her an exemplary Family Physician.

To contribute to the Elise Coletta, MD Education Leadership Fund, please contact Brown University, Box 1877, Providence, RI 02912.

Come Ye To the Waters

Before there was light or dark, before there was a firmament, before there were creatures on the land or birds in the sky, there was water. Indeed, other than chaos and nothingness, there was little more primeval in man's cosmology than the waters. The human fetus is nurtured in its amniotic waters; and water is a companion to every major event in man's extrauterine life from baptism at its onset to the ceremonial ablution at its completion.

Life began as one-celled creatures within the salt-laden waters. Much of life then evolved in these saline waters; but it required millions of years before a few bold marine creatures dared to venture beyond the protective environment of the oceans. And even when these pioneering species abandoned the sustaining waters to live upon the dry land, they took with them their briny environment in the form of their internal biological fluids which simulated the chemical composition of ocean water.

Water represented a precious commodity in regions remote from rivers and fresh lakes. Accordingly, man protected the functioning springs in his region as irreplaceable tribal assets eventually viewing them as sacred trusts. Geographic names such as Hollywell [the holy well] hint at how revered some sources of water had been in the remote past.

First came man's need for water without which he could not survive; then came man's pleasure in bathing in fresh water; and still later came the belief that certain waters, especially from fresh springs, might be endowed with healing powers bestowed upon it by divine benediction.

Roman civilization, with its sophisticated aqueductal system, had made water both abundant and common place; and Rome's urban baths were then relegated to centers for relaxation, rehabilitation and even social intercourse. The Roman baths, famous as imposing marble structures, no longer made claims for miraculous cures; they were quite content with fulfilling their banal function as centers for communal and hygienic activity. Elsewhere in Western Europe and the British Isles, however, certain pools and springs clung to their magical aura.

Pilgrims, particularly those with chronic disabilities such as rheumatism, sought out the springs of Europe in the hopes that the waters might be curative. Nobody seems to have kept records of those deemed to be cured and those unaffected by the mineral waters, but certain springs evolved into holy shrines for miraculous cures often dedicated to a local deity or saint. The Reformation condemned such springs as pagan and many sites altered their avowed purpose from spiritual to health-oriented goals. Local entrepreneurs then built elaborate structures to house the springs. They also constructed bathing chambers, particularly if the spring waters were naturally heated. And there emerged from some of these sites [such as Evian in the Haute-Savoie Alpine district of southeastern France; Vichy in southern France; Seltzer, Wiesbaden, Marienbad and Baden-Baden in Germany; Spa in Belgium; and Bath and Malvern in England] health-related facilities for an emerging middle class not yet accustomed to the social merit of a planned vacation. It was a time, too, when organized medicine had few effective pharmaceuticals to offer. Indeed, 18th Century physicians could claim little beyond occasionally taking credit for spontaneous recoveries.

Initially, these spas [the word is derived from a small Belgian town with a famous natural spring] were known only to the locals and to a few itinerant pilgrims. But with time they became widely acclaimed and the original rude shelters for the religious pilgrims were supplanted by sumptuous resorts, often with elaborate casinos. The tribal spring, venerated initially as a place of magic, a sanctuary for the believers, had been transformed into a profitable institution attracting masses of adults seeking relaxation and earthly diversion. And if, by chance, they felt better, relieved of some of their sundry ailments, so much the better.

Many of these sites, such as Baden-Baden in Germany, identified themselves primarily as health centers, providing their clientele a full range of medical facilities. Over the centuries physicians, qualified and unqualified, gravitated to the European spas recognizing that there was a profitable component to what became known as "taking the waters" or going for "the water cure." Indeed, many health care providers opened clinics at the spas to exploit the many credulous souls who sought out places for cures of their bodily ills.

The spas, especially in the gilded Edwardian Age, offered cure-packages for those convinced that water possessed restorative properties and they voluntarily submitted themselves to rigorous schedules of mineral waters by mouth, for bathing and even for colonic irrigation. By the 19th Century, the advent of qualitative analytic procedures allowed for the identification of the various salts, which imparted the uniquely bitter taste to some of these waters. And each spa proudly boasted of the mineral composition of its springs. Some even announced the degree to which the waters bore radioactive elements hinting that radioactivity taken internally was a health-promoting attribute. A new, alternative form of medical treatment was evolving in these resorts, something called hydrotherapy with its cadre of faithful practitioners. Said one of these more pious adherents: "The disposing hand of Providence hath settled me in Lewisham, the place which God out of his liberal bounty hath blest with this Medicinal water."

Some skeptics, such as the Swiss chemist Jacob Schweppe, declared that mineral water was nothing more than the sum of its chemical constituents: some dissolved salts, a few earthern chemicals and natural gas all incorporated in simple water. Once these elements were identified, why not then reconstitute the mineral waters in a factory? Or, alternatively, even bottle the effluent of the natural springs? The faithful, however, insisted that their spring water contained a healing life-spirit which could neither be measured by chemical analysis nor survive the bottling process.

Secular America no longer finds miraculous endowments in spring water. But some remote tribal memory must resonate whenever Americans opt for Vichy, Seltzer, Evian, Perrier or a wagon-load of other spring waters rather than drink the abundant and free tap water from their kitchen sinks.

– Stanley M. Aronson, MD, MPH

Primary Prevention For Older Persons

John B. Murphy, MD, and Ramona Rhodes, MD

In the twentieth century the US population aged 65 and older increased 12-fold from roughly 3 million to 35 million. This is four times the rate of growth for the population under age 65. By the mid twenty-first century the population age 65 and older will more than double to 80 million with most of that growth occurring between 2010 and 2030.

In Rhode Island, 15% of the population (roughly 150,000 people) are age 65 and older; by 2020, it is projected that there will be 210,000 Rhode Islanders age 65 and older. Not only is the older population in Rhode Island growing, it is growing older. Rhode Islanders age 75 to 84 increased by 18% in the period 1990-2000; those aged 85 years and older increased by 30%.

Preventive health interventions are key to maintaining the health and optimum function of persons of all ages, including older persons. While data supporting or refuting the validity of certain interventions in older populations are lacking (e.g., PSA screening for prostate cancer), data support many interventions.

Primary prevention is the prevention of disease before it ever occurs. Secondary prevention refers to detecting disease in the asymptomatic phase (i.e., screening); tertiary prevention, to preventing progression of disease once symptomatic. This report will focus on primary prevention for those aged 65 and older; the recommendations will address average-risk individuals. Individuals with specific risk factors may benefit from interventions beyond those noted here.

The discussion will focus on immunoprophylaxis [Table 1] and chemoprophylaxis [Table 2]. Counseling interventions with older persons (e.g., stopping smoking, increasing physical activity and discussing advance directives) are beyond the scope of this paper.

Immunoprophylaxis

Influenza

Influenza A and B affect 10% of the population in the typical epidemic year and as many as 40% of the population in a pandemic year. Persons aged 85+ are 32 times more likely to die of an influenza-associated illness than those aged 65-69.[1] The aging of the US population is thought to account for the doubling of influenza-associated deaths over the past two decades.[2] It is estimated that as many as 50,000 Americans die each year of influenza, and influenza is responsible for as many as 120,000 hospitalizations per year.

Influenza vaccine is the primary intervention for the prevention of influenza. There are two vaccines, an inactivated injectable vaccine composed of subviron components and a live attenuated intranasal vaccine. The live virus vaccine was FDA-approved in June of 2003 only for persons aged 5-49.

The efficacy of the vaccine depends upon the match between the strains in a given year's vaccine and the virus circulating that year. Studies in young, healthy adults have demonstrated vaccine efficacy of 70-90% while efficacy in older populations ranges from 40-60%.[3] In spite of lower seroconversion rates in older populations, influenza vaccine unequivocally decreases morbidity and mortality in older groups. Influenza vaccine has resulted in 40% reductions in hospitalization due to influenza, pneumonia, heart disease and stroke, as well as 50-60% reductions in mortality.[4,5]

The inactivated vaccine is well tolerated, and for individuals who have previously received the vaccine, placebo and vaccine side effect rates are equivalent. The vaccine is grown in eggs; thus, individuals with egg allergy should not be vaccinated. Controversy exists over the likelihood of an association between influenza vaccine and Guillian-Barre¢ syndrome. However, if there is an association it is small (one case per million vaccine recipients). [6]

To protect older populations, it is imperative to vaccinate all formal and informal health care providers, including all who live with an older person (excluding infants 0-6 months). Staff vaccination rates in nursing homes are more highly correlated with good outcomes for residents than are vaccination rates for the residents themselves.

The vaccine requires a one to two-week lead time to work and maintains peak effectiveness for approximately 6 months. In Rhode Island there is seldom an epidemic prior to December 1st or after March 30th. Thus, the optimum window for vaccination is October 1st through November 15th. In years when vaccine is available as early as September 1st, vaccination should occur prior to October 1st if there is a contact between health care professional and an at-risk individual, because the risk of under-immunization (early vaccination-late outbreak) is much less than the risk of being unvaccinated. Medicare pays for the influenza vaccine.

Tetanus Vaccine

The incidence of tetanus in the United States has dropped dramatically since the universal administration of tetanus toxoid to infants in the United States in the 1940s. However, 75% of fatal cases occur among persons 65 and older. Ninety-four percent of persons who contract tetanus in the US are not up-to-date on tetanus vaccination or never received a primary series.[7] While 88% of children in the United States aged 6-11 have measurable tetanus antibodies the figure for those 70 years of age and older is only 28%.[8]

Tetanus toxoid was first produced in 1924. Widespread use began in 1940. It is a formaldehyde-treated toxin. There are two types of toxoid, adsorbed and fluid toxoid. The rates of seroconversion are equivalent but the adsorbed preparation results in higher antibody titers. The tetanus toxoid is combined with diptheria toxoid in the adult vaccine, Td. After a primary series, nearly all recipients achieve anti-toxin levels that are considerably greater than minimal protective levels. Routine boosters are recommended every 10 years as antitoxin levels approach the minimal protective levels by 10 years in many individuals. For those with no history of vaccination, a primary series of three immunizations is recommended (0

months, 1-2 months, and 6-12 months). Boosters every 10 years are recommended for all persons, including those over 65.

The efficacy of the tetanus vaccination has never been studied in a trial. Efficacy is inferred from the protective antitoxin titers that develop to a complete tetanus toxoid series and the fact that tetanus occurring in a fully immunized individual is exceedingly rare.

Local adverse reactions to the vaccination are common but are almost universally self-limited and require no therapy. More serious systemic adverse reactions are very uncommon. An exaggerated local (Arthus-like) reaction can occasionally be seen following Td vaccination. These reactions present as an extensive painful swelling, often from the shoulder to the elbow, and begin a few hours after the injection. They are more likely if the interval between vaccinations is short and are felt to be the result of vaccination in the presence of high serum antitoxin levels. Brachial neuritis and Guillian-Barre¢ syndrome, although very rare, can be associated with Td vaccination. Routine vaccination should be deferred in those individuals who have had a severe allergic reaction to a previous vaccination with Td. Medicare does not currently provide reimbursement for tetanus vaccination.

Pneumococcal Vaccine

Pneumococcal infection causes an estimated 40,000 deaths annually in the United States. Pneumococcal illness results in 3,000 cases of meningitis, 50,000 cases of bacteremia and 500,000 cases of pneumonia annually. Mortality for older persons with bacteremia and meningitis is double that of younger adult populations, approaching 35-40%. With the emergence of drug resistant streptococcus pneumoniae strains, the need for immunoprophylaxis has become more important.

Pneumococcal vaccine was first reported in 1945 as a tetravalent preparation. With the recent availability of antibiotics at that time the vaccine was not adopted for use. A 14-valent vaccine became available in 1977, and in 1983, a 23-valent formulation was produced. The currently available vaccines contained polysaccharide antigens to 23 pneumoccal serotypes and these serotypes account for 85-90 percent of all serious pneumoccal illness.

Pneumocccal capsular polysaccharide antigens induce type-specific antibodies that enhance opsonization, phagocytosis and killing of pneumococci by leukocytes and other phagocytic cells. Greater than 80% of young adults will develop a two-fold rise in serotype-specific antibody within two to three weeks of vaccination. Antibody responses also occur in older populations but antibody concentrations are lower than among healthy young adults. Levels of antibodies to most pneumococcal vaccine antigens remain elevated for at least five years in healthy adults, but in older persons they decline significantly by five to ten years.

The pneumococcal vaccine is generally well tolerated and no neurological disorders (e.g., Guillian-Barre¢ syndrome) have been associated with administration of pneumococcal vaccine.

Most randomized controlled trials looking at non-bacteremic pneumonia in the United States have failed to demonstrate efficacy of the vaccine, in large part because of the lack of specific and sensitive diagnostic tests for non-bacteremic pneumococcal pneumonia. Effectiveness in case control studies looking at invasive [e.g. bacteremic] disease has generally demonstrated 60-80 percent effectiveness in immunocompentent persons aged 65 and older.[10]

The **Centers for Disease Control and Prevention (CDC)** guidelines stipulate that all persons aged 65 and older should receive pneumococcal vaccine, including previously unvaccinated persons and persons who are 65 and previously received the vaccine more than five years earlier. There is no recommendation for re-vaccination of average-risk individuals older than age 65. Pneumoccal vaccine is a covered Medicare benefit.

Other Vaccines

No other vaccines are recommended for the average-risk person aged 65 and older. However, older persons may accrue an indication for other immunizations after age 65. For example, the retiree hospital volunteer who transports laboratory specimens is a candidate for hepatitis B vaccine. In the future, we may see the development of new vaccines for older persons. For example, the varicella vaccine is in trials to test its impact on the development of herpes zoster [Shingles].

Chemoprophylaxis

Aspirin

Vascular disease (including ischemic heart disease, stroke and peripheral vascular disease) is the leading cause of death for older Americans. Clear data recommend aspirin as secondary prevention, i.e., use of aspirin in persons with known coronary heart disease or equivalent (diabetes). However, the data are less clear for primary prevention.

A number of well-conducted, randomized controlled trials have examined aspirin chemoprophylaxis for primary prevention; many have included older men. Most have demonstrated 25-40% reductions in myocardial infarction, but

Immunoprophylaxis	Vaccine Type	Contraindications	Frequency	Medicare Reimbursement
Influenza Vaccine	Inactivated injectible	Egg Allergy	Yearly	Yes
Tetanus Vaccine	Injectible toxoid	Severe reaction to prior vaccine	Routine booster every 10 years. Primary series if not done	No
Pnuemococcal Vaccine	23-valent injectible	None Noted	At 65 if not previously vaccinated or received vaccine more than 5 years earlier. No recommendation for re-vaccination in average risk individuals.	Yes

Table 1. Recommended immunoprophylaxis

Disease State	Medication	Dose Recommendation	Indications
Vascular Disease*	Aspirin	81 or 325 mg po qd	Adults at increased risk for CHD**
Osteoporosis	Calcium	Men: 1000 mg/day Woman: 1500 mg/day	Those at increased risk of fracture***
	Vitamin D	400-800 International Units/day	Vitamin D deficiency
Influenza	N2 Blockers (Type A only) Amantidine Rimantidine	 As directed As directed	At-risk, egg allergic individuals. Un-immunized at-risk individuals during an epidemic. Immunized and un-immunized in nursing home where there is a documented case.
	Neuraminidase Inhibitors Oseltamivir Zanamivi	As directed As directed	

* Ischenmic heart disease, stroke and peripheral vascular disease.
** http://www.ahrq.gov/clinic/cps3dix.htm#heartvasc
*** Low BMD, history of osteoporotic fracture, fracture in 1st degree relative, low body weight, female sex, white race, advanced age.

TABLE 2. RECOMMENDED CHEMOPROPHYLAXIS

concerns about hemorrhagic stroke and other serious bleeding episodes arose in many of the trials. Nested case control studies have demonstrated similar benefits and risks in women. A meta-analysis of 16 trials provides insight into the magnitude of the risks and benefits. This study found that the absolute increase in hemorrhagic stroke over three years was 12 events per 10,000 persons while the reductions in myocardial infarction and ischemic stroke were respectively 137 and 39 events for 10,000 persons over 3 years.[11] These findings have led most groups to recommend aspirin therapy for men (in some cases women) who are at increased risk for CHD. The US Preventive Services Task Force, a conservative evidence-based group, made a definitive recommendation in 2002. They strongly recommend that clinicians discuss aspirin chemoprophyaxis with adults who are at increased risk for CHD and that the balance of benefits and harms favors chemoprophylaxis if the 10-year risk of CHD is greater than or equal to 6%.[12] Using the Framingham point scores, aged 65 alone (with no other risks), imparts a 10-year risk of developing CHD of 8 percent in men, thus justifying aspirin chemoprophylaxis. For women, age alone, without other risks would not justify chemoprophylaxis until age 80, however, age 65 with the addition of the diagnosis of hypertension and a treated systolic blood pressure of 140 would reach the risk benefit threshold.

The optimum dose for aspirin chemoprophylaxis is not known. Primary prevention trials have included dosages ranging from 75 mg per day to 325 mg every other day, with no clearly preferential dose. Furthermore, enteric coated or buffered preparations have not been clearly demonstrated to reduce adverse gastrointestinal effects of aspirin. The choice of 81 mg or 325 mg of aspirin per day would be reasonable alternatives for men aged 65 and older and women aged 65 and older with one or more risk factors.

OSTEOPOROSIS

There are an estimated 1.3 million osteoporosis-related fractures each year in the United States. An estimated one quarter of all women aged 60 and over develop spinal compression fractures and about 15% sustain hip fractures during their lifetime. While most of these fractures occur in older women, up to 30% of osteoporatic fractures will occur in older men. Low bone mineral density is strongly associated with an increased risk of fracture in men and women. A personal history of an osteoporatic fracture, history of a fracture in a first-degree relative, low body weight, female sex, white race, advanced age, inadequate physical activity, heavy alcohol use, lifelong low calcium intake, and estrogen deficiency are all risk factors for osteoporosis. Current cigarette smoking may also increase risk.

Randomized controlled trials have demonstrated the efficacy of calcium supplementation in preserving bone density in men and women. Calcium supplementation in women has also been shown to decreased the risk of fractures, but data on fracture as an end point in men is not yet available.[13, 14] The optimum level of calcium intake has yet to be established, but a generally accepted recommendation is 1000 mg for older men and 1500 mg for older women. This level should be composed of the composite of dietary calcium intake and supplemental intake. A rough guide to estimating dietary calcium intake is to multiply the number of servings of calcium-containing foods per day by 250 mg. Thus, for example, an older woman consuming two servings a day should supplement an additional 1000 mg per day. There are differences in calcium absorption related to the particular calcium substance, the timing of administration and the dose. However, confusion around focusing on which supplement to take, when to take it relative to meals and time of day, and how to break the dose up over th} course of ti

day has been shown to decrease adherence. Thus, it makes more sense to keep the recommendations as simple as possible. Calcium carbonate (the least expensive form available) taken in a dose of 500 mg 1 to 3 times a day to reach the target of 1500 mg for women 1000 mg for men is one standard recommendation.

There are very few dietary sources of vitamin D; the major source in the body is dermal synthesis. Ultraviolet rays in sunlight result in the synthesis of vitamin D3 in the skin from 7-dehydrocholesterol. Dietary vitamin D is absorbed in the small intestine from dietary sources such as fatty fish livers, vitamin D fortified foods and vitamin D supplements. Cholecalciferol, whether absorbed in the small intestine or created by exposure to ultraviolet light in the skin, is transported to the liver and transformed into 25-cholecalciferol. This form is transported to the kidney and is further hydroxalated, producing 1,25 dyhydroxy vitamin D3 which is the most active form

of vitamin D. Its most important biological action is to promote intestinal absorption of calcium. The prevalence of vitamin D deficiency is high in older populations due to a combination of decreased dietary intake, diminished absorption and limited exposure to sunlight. In one study of patients on a general hospital ward, vitamin D deficiency was detected in 57 percent of patients. While overt vitamin D deficiency and osteomalacia or rickets is now uncommon in developed countries, subclinical vitamin D deficiency is common and may contribute to the development of osteoporosis. In temperate climates such as New England, cutaneous production of vitamin D virtually ceases in the winter. Randomized controlled trials have demonstrated that physiological doses of vitamin D diminish the usual decline in bone density and decrease fracture risk in older men and women.[14] The optimum dose of vitamin D supplementation is not clear, but a recommendation of 400-800 IU daily is the generally accepted range.

Influenza Chemoprophylaxis

Neuraminidase inhibitors and N2 blockers have been demonstrated to be effective for the chemoprophylaxis of influenza. The N2 blockers, amantidine and rimantidine, are FDA approved for chemoprophylaxis of influenza A only. However, while they are effective in older populations they have some limitations. Amantidine causes significant CNS and GI toxicities. Rimantidine has less CNS and GI toxicity but like Amantidine, drug resistance has been demonstrated to develop rapidly, thus limiting its use. The neuraminidase inhibitors, Oseltamivir and Zanamivir are effective for chemoprophylaxis of influenza A and B and have a much more favorable side effect profile and less drug resistance. Zanamivir, which is inhaled, is less well tolerated than Oseltamivir. Unfortunately, Oseltamivir is the most expensive of the four drugs.

Situations in which chemoprophylaxis for influenza makes sense include at-risk individuals who are egg-allergic and can't receive vaccine (e.g. nursing home residents) and unimmunized, at-risk individuals during an epidemic, given for two weeks following influenza vaccine administration. A third indication is those immunized and unimmunized residents of a nursing home when there is a documented case of influenza in another nursing home resident. In this setting chemoprophylaxis should continue for one week beyond the onset of the last diagnosed case. For example, if there is a single incident case, chemoprophylaxis should be given for seven days for all residents. If there is a second case at day 5 the chemoprophylaxis should continue through 12 days.

Other Chemoprophyaxis

Many authorities recommend a multivitamin for average-risk older persons. While there is limited randomized trial data to support this recommendation, the cost and the potential risks of a once-a-day multivitamin are probably sufficiently small to support such a recommendation.

Nonsteroidal anti-inflammatory agents, cox-2 inhibitors, vitamin E, estrogen and a number of other agents have been touted for older populations. Many are supported by uncontrolled trials and small studies, but none has been demonstrated in substantial trials to be applicable to average-risk older individuals. Recent data from the Women's Health Initiative has highlighted the need to test the hypotheses generated in non-randomized trials. Thus, at this juncture, we would not recommend any other chemoprophylactic agents for average-risk individuals.

Conclusion

Primary prevention is important in the ongoing care of older persons. While the list of chemoprophylaxis and immunoprophylaxis interventions recommended here is parsimonious, support for additional interventions is lacking. Furthermore, many older persons are not receiving even this short list of interventions. Until additional data emerge, the focus should be on providing all older individuals with the supportable interventions and not expending resources on less clearly helpful interventions.

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Cognitive Screening of Older Adults

Janet Grace, PhD,. and Melissa M. Amick, PhD

The strongest risk factor for dementia is age, and the risk of dementia in the elderly increases with each decade. An estimated 11% of individuals 65 years of age and older, and 25-47% of individuals 85 years of age and older, have dementia.[1] With the advent of pharmacological treatments for dementia, there is an increased need for cognitive screening of older adults. We will review indications to conduct cognitive screening in the elderly, cognitive screening instruments, and web resources for the newly diagnosed patient.

The **US Preventative Services Task Force (USPSTF)** concluded that there is insufficient evidence to recommend for or against routine screening for dementia in *asymptomatic* older adults. However, the USPSTF found "good evidence that some screening tests have good sensitivity but," unfortunately, "only fair specificity in detecting cognitive impairment and dementia." They concluded that there is "fair to good evidence that several drug therapies have a beneficial effect on cognitive function… but the evidence of their beneficial effects on instrumental activities of daily living is mixed, with the benefit being small at best."[1]

The American Academy of Neurology and Canadian Task Force on Preventive Health Care reached similar conclusions. Further, a recent community-based study found that approximately half of the residents living independently in a retirement community were not willing to be screened routinely for memory problems. These results suggest that there is perceived harm associated with memory screening in asymptomatic individuals.[2]

Dementia is a clinical diagnosis which relies upon an experienced clinician's assessment. Clinical methods for diagnosing dementia include assessing the presenting problem, obtaining focused history from a reliable informant, physical exam of the patient, and evaluation of cognitive, behavioral and functional status of the patient. [3] Given the time needed for mental status testing and clinical interview of patient and informant, community physicians understandably fail to diagnosis dementia in over 50% of dementia cases, particularly in the earlier mild to moderate stages.[1] By contrast, use of **National Institute of Neurological and Communicative Disorder and Stroke and the Alzheimer's Disease and Related Disorders Association (NINCDS-ADRDA)** [4] criteria by an experienced clinician for diagnosis of Alzheimer's disease can result in excellent diagnostic accuracy. Studies have found that from 85-93% of individuals diagnosed with AD in dementia specialty clinics have AD confirmed at autopsy. [5]

Routine cognitive screening of all asymptomatic elderly may not be indicated, and this level of care would be costly and impractical. However, as noted by USPSTF,[1] both the American Medical Association and American Academy of Family Physicians recommended that physicians be alert for cognitive and functional decline in elderly patients and for recognition of early stage dementia. Early recognition of cognitive impairment is important for multiple reasons including the high prevalence of mental status changes in the aged, diagnosis of potentially reversible medical conditions, initiation of treatment interventions, and to ensure that the patient is able to adhere to medical recommendations. Early identification of cognitive decline gives patients and caregivers time to prepare for life style changes and plan for the future; e.g;, arranging finances and discussing end-of-life care while the patient is still competent.[1]

Issues around driving an automobile provide a practical example of why early identification of dementia is important. Dementia is a major risk factor for unsafe driving and is associated with an increased rate of fatal accidents. Though specific cut off scores have not been established, impaired performance on brief cognitive screening tools such as the MMSE or clock drawing are related to impaired road test performance or caregiver report of hazardous driving.[6] The presence of dementia does not necessarily preclude driving, but the majority of individuals with dementia will relinquish driving by three years after disease onset. Thus, early identification of dementia highlights the need to monitor key areas in an individual's life (financial management, driving) to prevent harm to self and others.

The potential reversibility of some dementias has been a rationale for cognitive screening and a complete dementia work-up, which could include numerous costly tests, i.e. complete blood count, erythrocyte sedimentation rate, VDRL test for Syphilis, serum folate, serum cobalamin (B12), chemistry panel, thyroid function studies, urinalysis, and brain imaging. Of note, the American Academy of Neurology abbreviated the list of recommended screening tests in the recent practice parameter. They recommended use of DSM-IV dementia criteria or the NINCDS-ADRDA diagnostic criteria, CT or MRI, screening for depression, B12 deficiency, and hypothyroidism.[8] DSM-IV dementia criteria include impairment in memory, associated impairment in abstract thinking, impaired judgment, or other disturbances of higher cortical function or personality change, and gradual onset and continuing cognitive decline, severe enough to interfere significantly with work or usual social activities or relationships.

In a meta-analysis which reviewed the literature from 1972-1987, Clarfield found reversible causes of dementia in 11% of cases (8% partially and 3% fully reversible). Dementias that can be reversed were more likely present in younger individuals and individuals with very recent cognitive decline. Community based studies showed lower rates of reversibility than hospital based research where acute confusion/ delirium more commonly occurs in in-

dividuals. Clarfield[8] subsequently conducted an updated meta-analysis of 50 articles published between 1987-2002. Compared to the previous review, he found that more studies were based in the community or outpatient settings, where the majority of dementia cases are observed. Prevalence of reversible dementia fell from 11% to less than 1%. One explanation for this change was that the individuals were older and irreversible causes of dementia increase with age (i.e. Alzheimer's disease was present in 56% and vascular etiology in 20% of the patients studied). Medication-related causes of dementia fell from 1.5% to 0.1% and depression fell from 4.5% to 0.9%. The findings from both meta-analyses indicate that, reversible dementia was seen predominantly in younger individuals with recent onset of symptoms. This suggests that primary care physicians need to screen and evaluate individuals who recently have complained of mild memory problems. Furthermore, even when a reversible cause such as depression was identified and treated appropriately, half of the individuals manifested progressive intellectual impairment. This finding suggests that primary care physicians should continue to screen for cognitive impairment because multiple diseases can exist concomitantly. For example, in the case of depression co-occurring with AD, treatment of depression may not stop the progressive dementia, but may improve function and quality of life. Further, earlier interventions for the cognitively impaired without dementia (Mild Cognitive Impairment), may offer better prognosis, (e.g. with vitamin B12 treatment).

While routine cognitive screening is not recommended for all older adults, assessing cognitive function is indicated when the individual reports memory complaints, the family or friends raise concerns about the individual's memory or function, or the primary care physician notes a deterioration in the individual's cognition or function.[1] Other risk factors include advanced age (over 80) and a family history of dementia, especially the presence of early onset dementia in first-degree relatives. Some warning signs include problems with learning and retaining new information, repetitive behavior, trouble with complex tasks, impaired reasoning, disorientation, difficulty expressing complex thoughts or following complex conversations, and behavior changes such as apathy, depression, suspiciousness or irritability[9].

Numerous cognitive screening measures are available to assist in detection of dementia. An ideal screening instrument is standardized, reliable, and valid; has good sensitivity, specificity, positive and negative predictive value; and samples a range of cognitive functions. Ideally, scores should not be affected by changes related to normal aging, educational level, or race. The accuracy of screening tests for dementia has been reviewed and updated.[1,9] The most widely used instrument in clinical practice is the Folstein **Mini Mental Status Examination (MMSE).**[10] Its 30-point scale is heavily weighted with language and orientation items. It is most sensitive for Caucasian, high school educated individuals, with a generally recommended cut-off of 24/30, and 26/30 for college-educated individuals. Norms are available for diverse, less educated individuals, generally recommending a lower cut point (18/30).[11] Positive predictive values range from 15-72% and negative predictive value from 95-99%[1.] The **Modified Mini Mental Status Examination (3MS)**[12] allows the clinician to obtain the traditional 30-point MMSE score as well as an expanded 100 point score. A cut-off score of 86/100 has the best sensitivity but 77/100 has been used effectively in community-based screening (88% sensitivity, 90% specificity).[13] The 3MS adds items typically impaired in Alzheimer's disease, such as category fluency and delayed recall memory.

...BOTH THE AMERICAN MEDICAL ASSOCIATION AND AMERICAN ACADEMY OF FAMILY PHYSICIANS RECOMMENDED THAT PHYSICIANS BE ALERT FOR COGNITIVE AND FUNCTIONAL DECLINE IN ELDERLY PATIENTS AND FOR RECOGNITION OF EARLY STAGE DEMENTIA.

The **Blessed Information Memory Concentration Test (BMIC)** and its shortened version, the Short **Blessed or Orientation Memory Concentration test (BOMC)** are highly correlated with MMSE. The BOMC is briefer (6 scored items compared to 19 items on MMSE) and can discriminate among mild, moderate, and severe cognitive impairments.[14] The BOMC assesses similar cognitive functions to the MMSE, and has good test-retest reliability. The BOMC assesses orientation (month, year, and time of day), memory (recall of a name and address), and concentration (counting backwards and reciting months of the year backwards). Each item is weighted for a maximum score of 28, and a score of 10 or greater has been found to be consistent with the presence of dementia. The scoring is somewhat cumbersome, but can be

Table 1

Behavior Disturbance	Informant Rating	Cut off Score
1. Does the patient ask the same questions over and over again?	0=never, 1=rarely, 2=some of the time, 3=often, 4 =most of the time	2 or greater (repetitive questions)
2. Does the patient repeat the same action (e.g. wiping table) over and over?	0=never, 1=rarely, 2=some of the time, 3=often, 4 =most of the time	1 or greater (repetitive behaviors)

mastered if used routinely. An autopsy study of 38 individuals, including individuals with and without dementia, revealed that scores on the BOMC positively correlated with the number of senile plaques. The researchers caution that a diagnosis of dementia should not be based solely on the results of the BOMC, as this task does not thoroughly evaluate all cognitive domains and may not distinguish between individuals with dementia and cognitively intact but depressed elderly adults. However, the brevity of the BOMC suggests that this measure may be a preferable mental status exam for use in the busy primary care setting.

Clock drawing test (CDT) (e.g. ask to draw a clock face, write all the numbers on the clock face and set the clock for 10 past 11), is a quick, simple measure, which can be scored reliably and encompasses multiple domains of cognition including executive and visuospatial functions. Using the CDT, dementia classification accuracy of 85% or more was found when normal older adults are compared to those with mild AD. Unfortunately, in very mild dementia, the CDT has poor sensitivity.[15] The CDT is moderately to highly correlated with the MMSE and combined use of the CDT and MMSE increases detection of AD. The CDT was found to be more significantly related to driving impairment than the MMSE.[6]

Some recent promising screening tools are composites of several tests. For example, the 7-minute Neurocognitive Screening Battery[17] can be administered with little training or clinical judgment so support staff potentially could assist the primary care physician in gathering mental status data. It consists of 4 tests tapping cognitive areas that are typically impaired in AD including an enhanced cued recall task, orientation for time and date, clock drawing test (twenty to four setting), and verbal fluency (i.e. generate animal names in 1 minute). Solomon and colleagues report high sensitivity (92%) and specificity (96%) in very mild, mild and moderate disease as well as high test-retest and inter-rater reliability. However, these results were based on individuals from a memory clinic and have not been replicated in a primary care setting.

By contrast, the **General Practitioner Assessment of Cognition (GPCOG)**[17] is a brief, reliable and valid screening instrument developed for use in a general practice setting. The GPCOG was shown to be easy to administer in an average of 6 minutes, and was strongly correlated with the MMSE. Sensitivity of 85%, specificity of 86%, positive predictive value of 71.4% and negative predictive value of 93% were found. Most importantly, it was acceptable to general practitioners in community practices in Australia where it was developed. It incorporates patient cognitive test data (4 items-time orientation, clock drawing, current event, recall of a name and address), and informant report (6 items-trouble recalling recent events or conversations, word finding, problems managing finances, medications, and transportation). Thus, the informant report addresses the critical inclusion of **activities of daily living (ADLs).** The main disadvantage is that informants may not always be available. Without an informant, the patient cognitive measures have moderate sensitivity and specificity and the informant questions can be answered by telephone if necessary.

Informant data about functional activities and behavior are important for dementia diagnosis. Ready, Ott and Grace[18] found that asking informants two brief questions from the Behavior Disturbance Scale yielded meaningful screening information. (Table 1) Responses above cut off on either question resulted in a sensitivity 97%, specificity 61%, and correctly classified 81% as demented. These simple questions can be easily integrated into an office visit or phone contact with the caregiver.

Conclusions

Consistent alertness to "triggers" or warning signs (e.g. memory complaints, repetitive behavior) and brief cognitive screening with a standardized measure to recognize early dementia

Recommended Web resources:

USPSTF recommendations
http://www.preventiveservices.ahrq.gov
http://www.guideline.gov

AMA Dementia Guide
http://www.ama-assn.org/ama/pub/category/4789.html

Dementia Diagnosis, especially helpful to distinguish normal aging from dementia
http://www.ama-assn.org/ama/pub/category/5031.html

Ten Warning Signs of Alzheimer's Disease
http://www.alz.org/AboutAD/Warning.asp

Managed Care Initiative and Guidelines
http://www.alz.org/Health/managedcare.asp

Support Network for Health Professionals
http://www.alz.org/Health/overview.asp

Information on dementia published by the NIH
http://health.nih.gov/result.asp?disease_id=181

Information on Alzheimer's disease published by the NIH
http://health.nih.gov/result.asp?disease_id=28

Home safety for People with Alzheimer's disease
http://www.alzheimers.org/pubs/homesafety.htm
NIH Publication No. 02-5179

Behavior management strategies
http://www.caregiver.org/caregiver/jsp/print_friendly.jsp?nodeid=391

in at risk elders is recommended in primary care practice. When cognitive impairment is identified on screening, reference to DSM-IV diagnostic criteria for dementia is recommended to guide what additional information (e.g. caregiver interview, neuropsychological testing, functional assessment, medical tests) may be needed for accurate diagnosis. Further, cognitive screening of individuals who show early signs of dementia, with a formal test, is strongly recommended in routine general medical practice. Relying on an informal conversational approach is not recommended since many individuals with dementia have preserved social skills and casual conversation is at an automatic level. The 3MS which includes critical items such as orientation, verbal fluency, visuoconstruction, and verbal abstraction is one recommended screening tool. Adding the CDT to any one of the brief mental status exams such as the 3MS, Folstein MMSE, or Short Blessed/BOMC, will increase detection of cognitive impairment and dementia. Even the use of a subset of tasks from well-known screening measures can be useful for detecting dementia in clinical practice. For example, orientation questions (time, place), production of serial 7s, ability to repeat 7 digit span, ability to recall 3 items, or a clock drawing yield an intermediate probability of detecting dementia.[19] These simple items are specific for dementia but not necessarily sensitive, and should not be the sole basis of diagnosis. The primary care physician can easily integrate these simple cognitive screening items into practice for early detection and further evaluation of cognitive impairment.

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Another Perspective On the Memantine Graphs: Clinical vs. Statistical Significance

Stephen Davis, MD

Recently, there has been a proliferation of advertising about memantine, a new adjunct in the treatment of Alzheimer's Disease. The advertising is primarily in the distribution of two articles, one from the *New England Journal of Medicine*[1], one from the *Journal of the American Medical Association*.[2] Both studies that are reported were reasonably well done, and both produced statistically significant improvements in several measures of behavior and mentation in the study groups.

The graphs of those improvements are impressive. With their presentation of the average scores and standard errors of the study and control groups, the graphs demonstrate the statistical significance of the study data. Two of the graphs from the JAMA study are shown below. (Figure 1. Used by permission.) These graphs represent the data from the Severe Impairment Battery (SIB), and from the 19-item Alzheimer Disease Cooperative Study—Activities of Daily Living Inventory (the ADCS-ADL19). The graphs demonstrate the data for the 24 weeks of the study and include the endpoint scores with the Last Observation Carried Forward, or LOCF, score. (The LOCF score averaged the last score reported from each study subject whether he or she finished the study or not.)

A comment on the graphs is in order: They are misleading in representing the effect of memantine. The SIB graph shows a total span on the vertical axis of eight points. The actual SIB scale is from zero to 100. The ADCS-ADL19 graph in the JAMA article has a total range of six points. The ADCS-ADL19 scale has a total of 54 points. Both graphs have expanded vertical axes and compressed horizontal axes. These factors both exaggerate the differences between the study group and the control group curves. They do make the standard error bars easier to see, but they limit the perspective by abbreviating the actual ranges of the tests.

The SIB score evaluates cognitive dysfunction in the realms of memory, orientation, attention, visuo-spatial ability, and construction. It is based on a scale that goes from 0 to 100. The highest and lowest average scores from the study varied by just about five points. When the data is regraphed on a scale that represents the total 100-point range of the battery, they appear as in Figure 2. (On this graph, the data are normalized so that they can be compared more easily. This is done by giving both the study and control groups a starting score of 80, even though the real starting average scores were 80.0 for the control group and 78.0 for the study group.)

The ADCS-ADL 19 Inventory measures later stages of dementia and independence in everyday tasks. It is

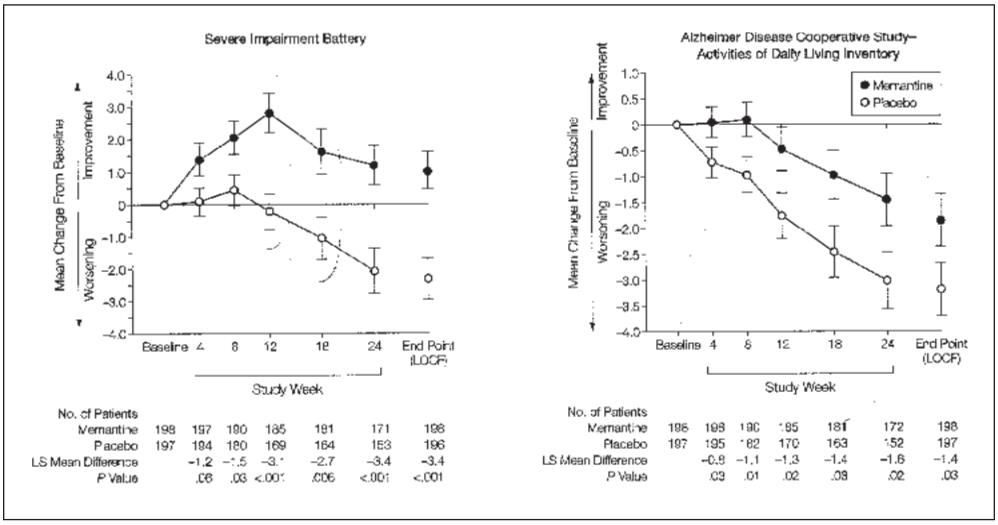

Figure 1. Graphs From JAMA Article

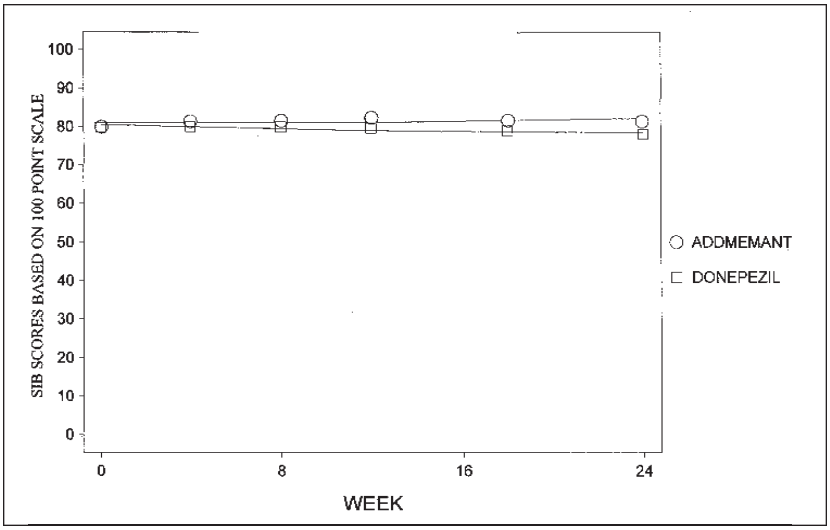

Figure 2. Graph of SIB Scores

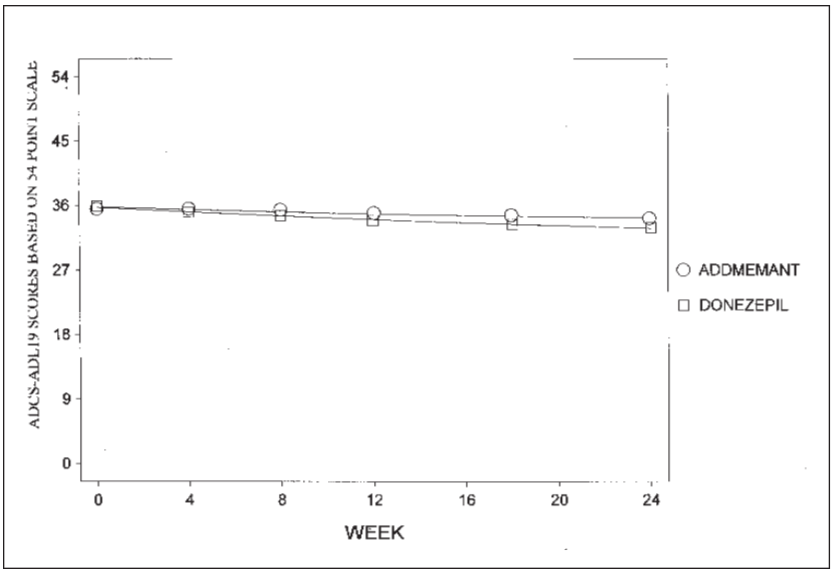

Figure 3. Graph Of ADCS-ADL19 Scores

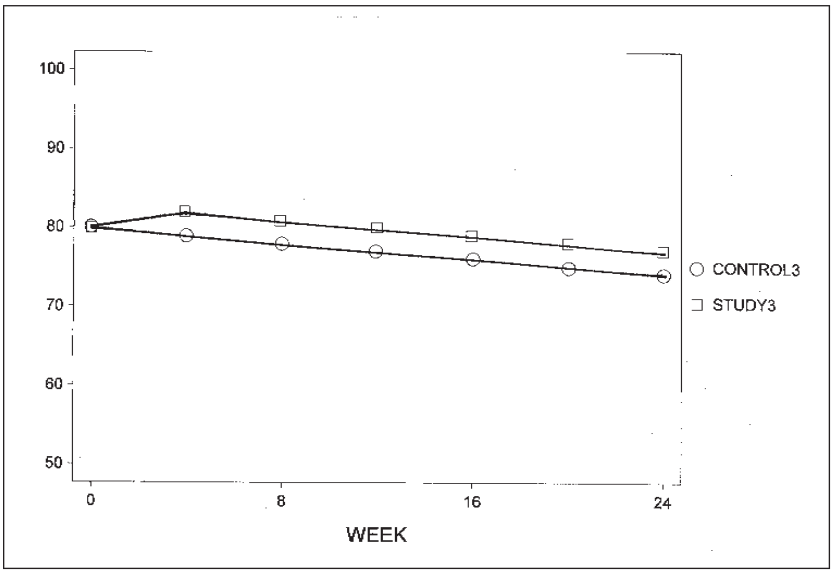

Figure 4. Demonstration of Stable Difference

based on a scale of 0 to 54. The greatest difference between average scores from the study is just about three points. When this data is re-graphed on a 0 to 54 point graph, it appears as in Figure 3.

Although the differences in the study groups and the control groups are indeed statistically significant, this re-graphing of data makes the results much less dramatic and might lead to a different interpretation. Whether these differences are clinically significant, that is, whether treatment is going to make a real improvement in a patient's life, is a much more difficult issue to address. Many other factors need to be considered. For example, what actual improvements are the patients making that are leading to these small improvements in their scores? Are these changes differences that are really improving their lives and the lives of their caretakers? This study was conducted on people who had Mini Mental Status Exam (MMSE) scores from 5 to 14 (out of a total of 30); in short, people who, as the article title suggests, are moderately to severely demented. The researchers did not stratify the improvements made by MMSE scores, so we do not know whether the results were similar in patients throughout the range of MMSE scores or whether the improvements varied as some function of the original MMSE score.

An important clinical difference is a very subjective measure. A two point improvement on the SIB measure might improve one person's quality of life, but a three point difference on the ADCS-ADL 19 measure might not make a difference for another person. The burden on the caretaker should also be considered. In assessing the differences suggested by this study one should consider the pre-study level of function of the subjects as well as their relative changes.

Another crucial issue that the study does not address is whether the difference in functioning between the two groups continues to enlarge over time or whether it stays at the distance measured at the end of the study. A static two-point difference in either of the measures, as shown in Figure 4, might not be of clinical significance; however, if the treated group has an overall slower rate of cognitive and functional decline, a clinical difference might almost certainly be achieved. Figure 5 demonstrates this latter relationship.

The study lasted 24 weeks. One could argue that this is a small time period for the medicine to make a clinical improvement for the patients. However, if a person has a MMSE score of 5 due to Alzheimer's type dementia, then that person has a short life expectancy, and twenty-four weeks may be a large percentage of that person's remaining life expectancy. Neither the JAMA study nor the New England Journal of Medicine study, which compared memantine to placebo without concomitant treatment with donepezil and which lasted 28 weeks, determined the effects of a longer duration.

Also, memantine, as prescribed in the study (two 5-mg tabs BID), would cost $358.36 per month at my local pharmacy, or at least $157.98 per month if more reasonably prescribed (one 10-mg tab BID). Even though we don't like to put monetary values on mental status, the first set of graphs would suggest that this would be money well-spent. Judging by the second and third graphs, the issue is less clear.

This is not to say that memantine should not be used. In interpreting the

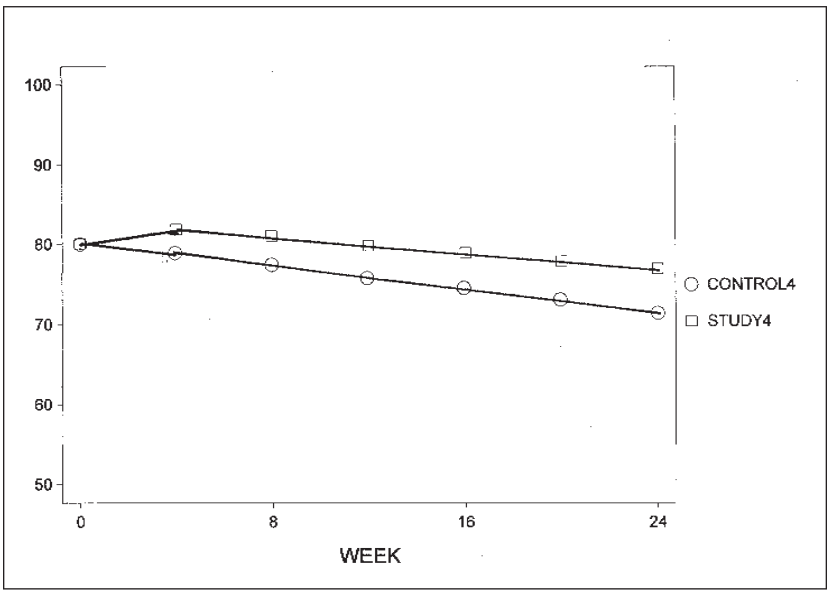

Figure 5. Demonstration of Increasing Difference

data, however, we should be careful in interpreting graphic data which may be exaggerated, and we should also realize that it is clinical improvement that we want our medications to effect not just statistical significance.

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Critique of "Another Perspective On the Memantine Graphs: clinical vs. Statistical Significance"

Joseph H. Friedman, MD

Steve Davis has made us think. He has pointed out aspects of two major papers that he finds troubling, re-graphing for us data that he thinks misleading. His title, obviously provocative, is itself, I believe, a bit misleading. Hence I think his comments require a counter balance. (Unlike the authors of the articles Davis cites, I have not received any money from involved companies.)

Davis is troubled by the magnification of the graphs, showing one point differences as bigger than they'd appear in graphs that detailed the entire scale. However these numbers were not meant to deceive or mislead. The actual numbers and their standard errors were provided. If we look at fever charts a one-point difference can mean a lot, even though a temperature chart could include a y-axis that goes from room temperature to 110°F. Graphs of blood pressure changes that look only at values in the desired range, not starting at 0 would be thought perfectly appropriate by most readers. Graphs of IQ changes, or weight or height changes, routinely focus on the part of the graph that changes, or simply graphs the change itself. There is nothing particularly valuable in including the lower limits of a scale on a graph intended to highlight the results (since the actual numbers are provided). And highlighting the results isn't necessarily sinister. More importantly, Davis overlooks two very important aspects of the New England Journal of Medicine article he cited. The first is the measure of a 48.5 hour reduction in time spent by the caretaker over the course of a month for memantine-treated patients. This is a huge amount of time, 1 1/2 hours each day for caregivers who may have an hour of free time each day or less. In marginal terms, this might represent a doubling of "free" time. Secondly, the article itself worries about the meaning of its findings. "The clinical relevance of treatment effects has been an issue in all trials of medication for Alzheimer's disease. Point differences between drug- and placebo-treated patients in quantitative scales do not necessarily indicate that these effects are clinically meaningful…The treatment effects…seemed to translate into improvements in the patients' behavior (less agitationÖ) and mitigation of the burden on caregivers (fewer hours spent assisting the patient.)"

The clinical relevance of the memantine findings were determined by an expert panel of the Food and Drug Administration. And while it has become clear that the FDA has become tainted by political interests in very recent years, there is no reason to think that the approval of memantine represents a political intervention. I think that these Alzheimer drugs, do, in fact, help people. We certainly can argue about whether their effects are either worth their money, or whether their effects were large enough to justify approval by the FDA (think here about the effects of drugs approved for MS, ALS, stroke and Parkinson's disease), but I don't think it is correct to insinuate that the effect of memantine has been exaggerated by misleading graphs.

Safe and Effective Drug Therapy In Older Adults

Anne L. Hume, Pharm.D., FCCP, BCPS

The 35 million Americans aged 65 or older are a heterogeneous group, ranging from independently living older adults who have "successfully" aged with few comorbid conditions to the 1.6 million frail elderly nursing home residents.[1]

The last two decades have witnessed advances in drug therapy for the treatment and prevention of chronic diseases associated with aging. As death rates have declined, the prevalence of chronic diseases and the need for safe and effective drug therapy in the elderly has increased.

New Drugs and the Elderly

An adage advises that one should not be the first person to prescribe a new drug, nor the last to abandon an old drug. This remains a fundamental principle in safe and effective prescribing in the elderly. Basic questions of safety, efficacy and value continue to challenge clinicians when new medications are introduced. Further, balancing the potential additive benefit against the extra expense compared to established medications can be difficult when an individual and society have limited resources. Also, pharmaceutical promotion traditionally highlights the possible advantages of a new medication while the potential for side effects and drug interactions remains inadequately defined in the elderly.

In 2003, the **Food and Drug Administration** (**FDA**) approved 18 new drug entities.[2] With few exceptions for medications such as memantine that are used primarily in older adults with moderate-to-severe Alzheimer's disease, most new drugs for chronic conditions continue to be studied primarily in middle-aged, relatively healthy populations with few concomitant chronic diseases or medications. Although data are presented on the use of new drugs in "the elderly", these individuals are between the ages of 65 and 74, and rarely have multiple comorbid conditions such as heart failure or chronic renal failure. Despite this, many older adults with multiple coexisting conditions are among the first patients to receive a new drug in clinical practice.

The true safety profile of a new drug in the elderly frequently emerges only after its widespread use and remains dependent on voluntary reports to the FDA's MedWatch program (http://www.fda.gov/medwatch/). Metformin is a classic example. The drug had been used in Europe for many years prior to its US approval. Metformin was considered to be essentially free of lactic acidosis that had prompted the removal of its predecessor, phenformin, from the US market in the 1970s. However, lactic acidosis was quickly recognized to be a significant adverse drug reaction in the elderly who commonly have multiple risk factors for its development including renal insufficiency and heart failure.

One of the best examples of the dilemma in using new drugs in the elderly is with the selective **cyclo-oxygenase** (**COX**)-2 inhibitors such as rofecoxib and celecoxib. **Nonsteroidal anti-inflammatory drugs** (**NSAID**) have been recognized for many years to cause adverse **gastrointestinal** (**GI**) effects ranging from dyspepsia to acute bleeding and death. The risk of serious GI complications from traditional NSAID varies from 7.3 per 1000 patient years in osteoarthritis to 13 per 1000 patient years in rheumatoid arthritis.[3] The mortality rate from NSAID-induced GI complications has been estimated to be 0.22% yearly.[3] Advanced age is consistently identified as a major risk factor, and the risk may increase linearly with age.

Celecoxib and rofecoxib quickly became among the most commonly prescribed medications, generating $3.05 billion and $2.5 billion, respectively, in global sales in 2002.[4] A common perception continues to be that their safety is much better in the elderly. However, the actual overall safety profile of COX-2 inhibitors, compared to traditional NSAIDs, has remained controversial, in part based on two large studies including the **Celecoxib Long-term Arthritis Safety Study** (**CLASS**) and the **Vioxx Gastrointestinal Outcomes Research** (**VIGOR**) trial.[5,6]

The CLASS trial enrolled 8059 patients with osteoarthritis or rheumatoid arthritis, with 39% of participants 65 years of age or older and 12% were 75 years of age or older.[5] Patients received celecoxib 400 mg twice daily, ibuprofen 800 mg 3 times/day, or diclofenac 75 mg twice daily. The use of aspirin in a dose of 325 mg/day or less was permitted for cardiovascular and cerebrovascular prophylaxis with 20% of participants using aspirin. The primary outcome measure was the annualized incidence of ulcer complications including gastric or duodenal perforation, gastric outlet obstruction and upper GI bleeding. The use of celecoxib and the traditional NSAID resulted in the occurrence of the primary outcome measure in 0.76% and 1.45%, respectively, of treated patients. This difference was not statistically significant. When the primary outcome measure was combined with symptomatic ulcers, the use of celecoxib and the traditional NSAID was associated with 2.08% and 3.54%, respectively. Among individuals using low-dose aspirin, celecoxib did not decrease the risk of either the primary or secondary outcome measure compared to ibuprofen or diclofenac.

This study is controversial. Significant concern was raised when it was reported that only 6-month data was released when more information was available and had been reported to the FDA. Although presented as one study, data from CLASS came from two studies including a 12-month comparison between celecoxib and diclofenac and a 16-month comparison to ibuprofen. Based on the CLASS findings, the FDA indicated that celecoxib does not offer major advantages in terms of GI safety, although it stated that the concomitant use of aspirin may have masked the benefit from celecoxib.

The VIGOR trial randomized 8076 patients (mean age, 58 years) with rheumatoid arthritis to receive rofecoxib 50 mg/day or naproxen 500 mg twice daily.[5,6] The

median duration of the study was 9 months. The use of low-dose aspirin was not permitted, and corticosteroids were used by over 50% of individuals enrolled in the study. The primary outcome measure was serious complications including perforation or obstruction, upper GI bleeding and symptomatic gastroduodenal ulcers. The relative risk of developing a perforation, obstruction and severe upper GI bleeding was 0.6 (95% CI 0.2, 0.8) for participants receiving rofecoxib. Although greater GI safety was demonstrated, rofecoxib was associated with a higher risk of myocardial infarction. In individuals for whom prophylaxis with aspirin was indicated, the risk of a myocardial infarction was 0.1% in the naproxen group compared to 0.4% with rofecoxib. In the absence of significant cardiovascular disease, the risk was 0.1% and 0.2% for naproxen and rofecoxib, respectively. Based on the findings of VIGOR, the FDA approved labeling changes to indicate a lower risk of serious GI complications compared to naproxen. New warnings were added concerning the increased risk of cardiovascular events with rofecoxib.

Several points concerning the VIGOR trial deserve particular emphasis. First, individuals for whom cardiovascular prophylaxis with aspirin was indicated were permitted entry into this study and, as a result, did not receive aspirin. Second, the causes of the cardiovascular effects with rofecoxib are unknown. Initially, the finding was explained on the basis that naproxen has antiplatelet effects not possessed by rofecoxib due to its COX-2 selectivity. However, this explanation has been challenged on the basis that the potential benefit reported with naproxen was too great and is inconsistent with the overall benefit from antiplatelet agents such as aspirin reported by the Antithrombotic Trialists' Collaboration. A meta-analysis using data from VIGOR and CLASS, as well as two smaller rofecoxib studies, indicated that the risk of cardiovascular events was 2.38 (95% CI 1.39 – 4.0) for rofecoxib compared to naproxen. The report also indicated that the annualized risk of developing a myocardial infarction with rofecoxib and celecoxib was significantly higher compared to the placebo group in the meta-analysis.[7]

How should these studies be applied in order to provide evidence-based care of older patients? Rofecoxib may offer advantages in terms of a lower risk of upper GI bleeding in individuals not taking aspirin, but this benefit may be overshadowed by a potentially increased risk of cardiovascular events that are under-recognized. Further, it is unknown if the GI benefit of rofecoxib persists in patients who are taking low-dose aspirin. This is critical for older adults many of whom will have concomitant cardiovascular disease or significant risk factors. Finally, the potential benefit of COX-2 inhibitors in older adults at high risk of serious GI complications remains unknown as neither study included these individuals. (Note: Rofecoxib was withdrawn from the market in September 2004 and the safety of the other COX-2 inhibitors is under review by the FDA due to additional new concerns in December 2004.)

INSTEAD OF SIMPLY CONSIDERING THE NUMBER OF DRUGS, A BETTER APPROACH IS TO RECOGNIZE THE "PRESCRIBING CASCADE" IN WHICH A SIDE EFFECT FROM A FIRST DRUG IS MISINTERPRETED AS A NEW ILLNESS.

Medication Costs

Medication costs remain a barrier for many older adults. The interim Medicare prescription cards, introduced in the summer of 2004, have yielded savings to people with low incomes, but its benefits have not been sufficiently worthwhile to encourage a majority of middle-income Medicare enrollees to sign up. Also, enrollees found the plans confusing.

Regardless of the final Medicare prescription drug benefit, at least some medication costs can be controlled by more reflective prescription practices by clinicians. For example, the use of prescription esomeprazole (Nexium) over generic omeprazole (Prilosec) is one example of unnecessary medication costs. Esomeprazole is an optical isomer of omeprazole and provides similar safety and efficacy, yet may cost between $80 and $100 more per month. As always, the most basic issue in controlling costs should always be whether or not a drug is even needed for a problem.

The Prescribing Cascade

Polypharmacy has been defined as "the administration of many drugs at the same time". A discussion of prescribing issues in the elderly traditionally emphasizes the problem of "polypharmacy". However, based on the evidence from major clinical trials, the provision of quality care to a 75 year old patient with hypertension, type 2 diabetes mellitus and a prior myocardial infarction would encourage 2 drugs each for blood pressure and glucose control, a statin and at least aspirin or another antiplatelet agent, easily reaching a total of 6 drugs for a single patient.

Instead of simply considering the number of drugs, a better approach is to recognize the "prescribing cascade" in which a side effect from a first drug is misinterpreted as a new illness.[8] A common example of the prescribing cascade is the iatrogenic increase in blood pressure that occurs with both NSAID and COX-2 inhibitors. Several case-control studies have documented the increased likelihood of initiating antihypertensive therapy after the starting anti-inflammatory therapy. Although not always feasible, a better approach is to reconsider the original need for NSAID or COX-2 inhibitor, trying instead to use acetaminophen, glucosamine or nonpharmacologic therapies for osteoarthritis.

Clinical Trials

Five to seven years ago, a common clinical question would have been to consider the risks and benefits of a therapy such as lovastatin in a 75-year-old man. During the last decade, more older adults have been enrolled in major clinical trials including, for example, studies using statins for the primary and secondary prevention of coronary heart disease. Now, however, the question is increasingly whether or not a therapy such as a statin or other drug has any value in an 85 or 95-year-old person.

In addition to the inclusion of elderly in clinical trials, an increasing number of studies address questions critical in providing evidence-based care to older adults. Landmark studies such as the **Antihypertensive and Lipid-Lowering Treatment to Prevent Heart Attack Trial** (**ALLHAT**) and the **Atrial Fibrillation Follow-up Investigation of Rhythm Management** (**AFFIRM**) study challenge "conventional" practice and assumptions.[9,10] As an example, doxazosin has traditionally been advocated as a preferred therapy for the management of hypertension in elderly men who have concomitant benign prostatic hypertrophy. In ALLHAT, the doxazosin arm of the study was terminated prematurely in February 2000, because the drug was associated with a 25% increase in cardiovascular disease due to a doubling of the risk of heart failure.[9] The excess risk of heart failure was not due to the presence of a higher blood pressure among participants receiving doxazosin compared to chlorthalidone. Fortunately, a recent report has demonstrated that these results have diffused into practice relatively rapidly with the use of alpha-blockers declining by approximately 26%.[11]

In addition, the AFFIRM trial has demonstrated that rhythm control in patients with atrial fibrillation did not improve survival compared with anticoagulation alone and was associated with more hospitalizations and adverse drug reactions in patients who were asymptomatic or had only mild symptoms compared to the rate control group.[10] Most importantly, AFFIRM identified that most strokes occur in patients who had either not received warfarin or who had a sub-therapeutic anticoagulation.

Prevention and the Elderly

The safe and effective use of drug therapy for the management of chronic diseases in older adults begins with prevention. Efforts to prevent complications from chronic diseases using proven pharmacotherapy continue to be underutilized in older adults. The use of aspirin for secondary cardiovascular prevention, angiotensin-converting enzyme inhibitors for heart failure, warfarin for atrial fibrillation, bisphosphonates and other therapies for postmenopausal osteoporosis and corticosteroid-induced osteoporosis and b-blockers following myocardial infarction are often under-prescribed in the elderly. Further, patient and family education can reduce the risk of adverse drug reactions (and interactions) and facilitate earlier recognition of drug-related toxicities.

Computer Drug Information

Hand-held technology including **personal digital assistants** (**PDAs**) provides convenient access to quality drug information to clinicians. However, the primary caution is to remember that the "geriatric dose" in a PDA is based on very healthy older adults in clinical trials. Other major advances in technology such as electronic prescribing particularly when linked to an electronic medical record may reduce the potential for medication-related prescribing errors.

Also, over half of older Americans have used the Internet for health and medical information. (Almost a third of individuals over 65 years have computer access; 15% have an Internet connection in the home). In a national survey from the Pew Commission, 47% indicated that information from the Internet affected personal decisions about their treatment. Unfortunately, little information exists regarding the clinical outcomes associated with older adults using health-related information from the Internet. In the Pew Commission study of older adults, 52% of users who visited health sites thought that "almost all" or "most" of the information on the Internet was credible. Individuals with less formal education indicated greater credibility of health-related website information.[12] In order to help consumers find quality health information on the Internet, the College of Pharmacy at the University of Rhode Island developed a web portal (www.uri.edu/e-health and www.uri.edu/e-salud) with over 400 consumer websites that have been evaluated.

Summary

The last two decades have witnessed the introduction of major advances in drug therapy for many chronic diseases. Despite an increasing number of therapeutic choices, basic challenges remain when using new drugs in the elderly. It is essential: 1) to consider whether a new drug is truly safer, more effective or worth the extra expense compared to established medications, 2) to monitor patients for drug related toxicities, and 3.) to avoid polypharmacy and the prescribing cascade.

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Deep Venous Thrombosis as a Consequence of Benign Prostatic Hypertrophy

Nikhil S. Karanth, MD, MMS, Robert S. Crausman, MD, MMS, FCCP, Vera A. De Palo, MD, FCCP

Benign prostatic hypertrophy (BPH), a common urological syndrome, affects 80% of men age 70 and older. An estimated 90% of men 80 years or older show histologic evidence of BPH and many have BPH-related symptoms (prostatism). [1,2] (Figure 1) BPH is responsible for the majority of urinary symptoms in men over age 50 and symptoms linked to prostatic obstruction coincide with BPH progression. Ten percent present with urinary retention.

Risk factors associated with **deep venous thrombosis (DVT)** are venous stasis, vascular injury, and hypercoagulability, otherwise known as Virchow's triad. DVTs occur frequently, but often are underdiagnosed.[3] The yearly incidence of DVT is estimated to be between 2 to 5 million cases in the United States.[4] Pulmonary thromboembolism and the post-phlebitic syndrome are two of the more significant consequences.[3] Although prostate cancer predisposes patients to venous thromboembolism, BPH has not been identified as a risk factor. Here we describe a potential association.

Case Report

An 88 year-old man presented to the Emergency Department with a two-week history of abdominal pain and distention and the inability to urinate. He had a significant medical history for chronic atrial fibrillation , a **transurethral resection of the prostate (TURP)** in 1990, recurrent prostatism, gait instability and a history of non-adherence to his medical regimen. Current medications included enteric-coated aspirin 325mg daily, atenolol 25 mg daily, magnesium oxide 400mg twice daily and a daily multivitamin.

On examination the patient's abdomen was rigid and tender to palpation. A digital rectal exam demonstrated fecal impaction without palpable nodes or prostate enlargement. An abdominal radiograph revealed a large mass arising out of the pelvis displacing the bowel cephalad and laterally to the right. This finding was consistent with a massively, distended urinary bladder. The patient's abdominal discomfort resolved with the insertion of an indwelling Foley catheter and the draining of six liters of dark concentrated urine.

In addition, the patient had a swollen, erythematous, and tender, right lower extremity. A lower extremity ultrasound revealed an extensive DVT. Treatment with intravenous unfractionated heparin was initiated and followed by insertion of an **inferior vena cava (IVC)** filter; the patient was felt to be a poor candidate for long-term anticoagulation owing to a history of non-adherence and gait instability. A serum **prostate specific antigen (PSA)** level was normal (1.7 ng/ml, normal laboratory range being 0.0-4.0ng/ml) and multiple stool samples were negative for occult blood. The patient declined further colorectal cancer screening

Discussion

BPH

BPH is one of the most common processes affecting elderly men and is responsible for more that 1.7 million office visits each year.[5] Its symptoms are present in 50% of men at 60 years of age and by age 80 more than 81% have BPH-related symptoms.[1,2] The prevalence of BPH at autopsy approaches 90% by the age of 90 years and is present in half of all 60 year old men.[2]

Symptoms linked to prostatic obstruction coincide with BPH progression. Patient complaints are predominately obstructive voiding symptoms and irritative symptoms related to the bladder outlet obstruction that occurs.[6] Prostatism, the symptom complex that is secondary to the bladder outlet obstruction, can be characterized by hesitancy, straining, weak (decreased in size and force) urine stream, or dribbling. Urinary retention, the inability to void voluntarily, distension of the bladder, and increased post-void residual may also be present, resulting in the necessary use of a catheter for bladder evacuation. Patients can complain of irritative symptoms including nocturia (more than once a night), frequency of urination (more than eight times a day during daytime hours), urgency and dysuria.[7] In a survey of men 60 years and older who had no history of prostatic intervention, the prevalence of one or more symptoms of prostatism was 35%.[8] Decreased urinary flow rate is a complaint in about 25% of men at age 55 years and increases to 50% at age 75.[9] The gold standard treatment for men with symptomatic BPH is TURP, with as many as 20-30% requiring this procedure by age 80.. Further, approximately 20% of patients who have undergone the procedure require repeat intervention after 10 years because of recrudescence of urologic symptoms.[10]

DVT

DVT results from the development and propagation of blood clot within the deep veins of the extremities or pelvis, accompanied by inflammation or injury of the vessel wall. Pulmonary embolism is the major complication of DVT and the disease spectrum ranges from unsuspected and clinically unimportant, to massive embolism causing death. Thirty percent to 50% of of untreated proximal DVTs result in pulmonary emboli, of which 10-20% are fatal. Mortality decreases with aggressive therapy, i.e. anticoagulation, Greenfield filter, prophylaxis.[3,4]

The etiology of DVT is best described by Virchow's triad of venous stasis, vessel wall injury, and hypercoagulability. The major risk factors are categorized as patient-related factors, disease states, surgical factors, and hematologic disorders.[11] The patient-related risk factors include age [40 years of age or older], obesity, varicose veins and immobility. Disease states conferring increased risk for thromboembolism include congestive heart

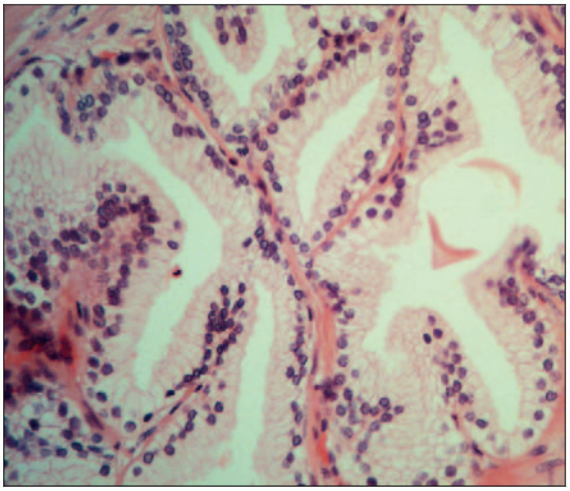

Figure 1. Photomicrograph demonstrating typical histologic findings of BPH in an Elderly Male. (Photomicrograph courtesy of Noubar Kessimian, MD, Chief of Pathology, MHRI).

failure, recent myocardial infarction, malignancy, nephrotic syndrome, inflammatory bowel disease, spinal cord injury with paralysis, and trauma including pelvic, hip or long-bone fracture. Surgical factors depend on the type and duration of the surgical procedure. Pelvic, hip and knee surgery have the highest risk of post-operative DVT due to potential vascular injury during the surgery. The risk for thromboembolic disease is also increased with coronary artery bypass grafting, urologic surgery, and neurosurgery. Hematological disorders such as activated protein C-resistance, factor V deficiency, [Leiden mutation], prothrombin gene mutation, protein C or protein S deficiency, antithrombin III deficiency, lupus anticoagulant, polycythemia vera, paroxysmal nocturnal hemoglobinuria, and dysfibrinogenemia increase the risk for thromboembolism.[12]

BPH and DVT

The difficulty in diagnosing a patient with DVT presenting with acute bladder distension has a historical perspective. In 1960, Carlsson and Garsten first described venous obstruction due to urinary bladder distention in a 3 week-old male infant.[13] The neonate presented with sudden onset of a cold, pale lower extremity and was found to have bladder distention, due to posterior urethral valves, causing venous obstruction. In the last 40 years, there have been about 20 cases reported which describe symptoms of vascular obstruction, mostly lower extremity edema, as a result of urinary bladder distention.[14-19] All patients have been men except for one woman where the cause of bladder distention was thought to be an atonic bladder due to diabetes mellitus.[16] All patients were between 54 and 91 years of age, with the exception of a mentally retarded patient who 48 years old and had a urethral stricture causing bladder distention. Lower extremity edema has been bilateral in most patients but in four patients it was unilateral. The cause of bladder obstruction in most cases was BPH, although other causes included carcinoma of the prostate, bladder neck fibrosis, urethral strictures, and atonic bladder. A distended bladder can compress both the arterial and venous structures in the pelvis. Vascular compression alone may result in lower extremity and scrotal edema; fortunately most do not suffer DVT as a consequence,[19] but it has been previously reported.[20]

Our patient's outlet obstruction led to the development of a massively distended bladder that compressed his pelvic venous system. We speculate that the resultant venous stasis, combined with the mild hypercoagulability of the elderly, predisposed our patient to the development of DVT. This patient's normal PSA and digital rectal examination suggest that prostate cancer is very unlikely and there were no other apparent predisposing factors. Thus, we conclude that this patient's DVT was precipitated by BPH and the resultant urinary bladder distention.

Conclusions

This case highlights the importance of considering the possible differential diagnoses in patients with lower extremity edema and bladder distention. While a severely distended bladder can cause lower extremity edema by venous compression, DVT can also be present even in a patient with no other predisposing risk factor.

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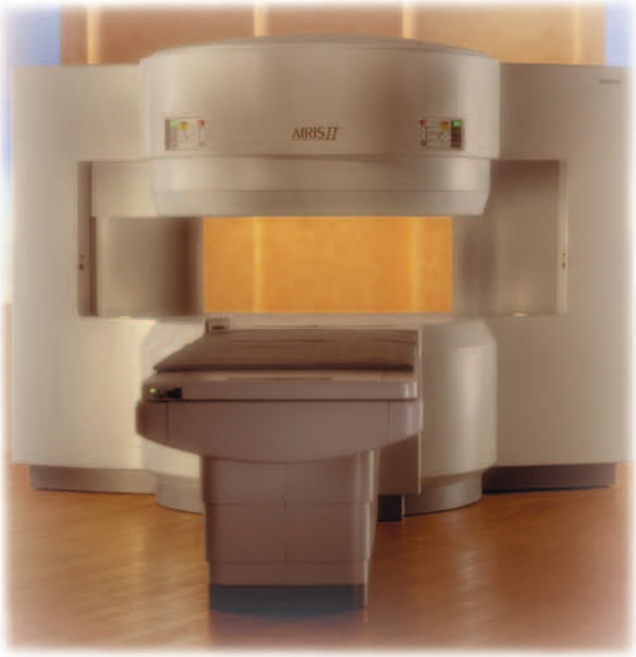

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Prevention of Falls In Older Adults

Alicia J. Curtin, PhD, GNP

Falls are a leading cause of injury-related deaths among older adults in the United States. Falling is associated with morbidity, mortality and functional impairment that often precipitates nursing home placement. Rates of fall-related deaths increase sharply with advancing age.[1] In Rhode Island, falls are the leading cause of accidental death for adults 65 years and over.[2]

Most falls do not result in serious injury. But the consequence of non-injurious falls can be a fear of falling, self-imposed restriction of mobility, and a further decline in functional ability among older adults. Frequently, older adults are not aware of the risks of falling and do not report a fall to their primary care physician unless an injury has occurred. For this reason, primary care physicians need to inquire about falls, and identify patients at greatest risk. Multidisciplinary interventions can reduce and prevent the incidence of falls and fall-related injuries. Here we review the epidemiology of falls in the older adult, risk factors, potential etiologies and fall-prevention strategies.

Epidemiology

The annual incidence of falls in adults living independently in the United States is approximately 30% in adults aged 65 years and over and increases to 50% in older adults aged 80 years and over.[3] Hospitalization rates for hip fracture increase with advancing age for both men and women, but are consistently higher for white women in all age categories. This data may reflect a reporting bias due to the high susceptibility to fracture and injury among older white women, who have the highest prevalence rates of osteoporosis.[4]

The frequency of falls is higher for hospitalized older adults and older adults living in nursing homes compared to those living independently. An estimated 1 in 5 older hospitalized adults will fall during a hospitalization. This results in increase length and cost of hospital stays, use of physical/chemical restraints, and potential litigation and insurance claims.[5]

Incidence rates for falls in the nursing home are approximately three times the rate for older adults living in the community. Further, nursing home residents have a disproportionately high incidence of hip fracture and higher mortality rates associated with hip fracture compared to community-dwelling older adults.[6] This data may reflect the frailty of the nursing home resident, who may fall in situations where healthier adults would not. These statistics may still underestimate the magnitude of the problem due to under-reporting. The need to identify older adults at risk and implement a multidisciplinary, risk-reduction prevention program is therefore a key priority in the management of fall-related injuries and the associated sequelae of non-injurious falls among older adults.

Etiology

A fall is defined as an "event which results in a person coming to rest inadvertently on the ground not as a consequence of the following: sustaining a violent blow, loss of consciousness [i.e. syncope], sudden onset of paralysis as in a stroke, or epileptic seizures."[7] Although some falls may be caused by a single event, the majority are multi-factorial; and studies have demonstrated that the risk of falling increases dramatically as the number of risk factors increases.[3,8,9]

The literature has identified three categories of risk factors: intrinsic, extrinsic and situational/activity-related factors. (Table 1) Intrinsic factors are inherent in the older adult; i.e., advancing age, female gender, chronic illnesses which cause muscle weakness, gait and balance impairments, functional and cognitive impairments, poly-pharmacy, history of falls, visual deficits and depression. [3, 10, 11, 12]

Epidemiologists have demonstrated the association of increased fall-risk with advancing age for both men and women, with the risk being greater for women of all age groups.[1, 2, 3] Age-related changes such as decreased muscle strength and mass, decreased joint flexibility, prolonged reaction times, and impaired vibratory and proprioceptive sensation as well as chronic illnesses associated with aging (i.e. osteoarthritis, osteoporosis) may impair the older adult.

In addition, women and men may have different outcomes after falling. Osteoporosis, while not considered a risk factor for falling, does predispose the person to fall-related injuries, including hip, vertebrae, wrist and pelvic fractures, especially in older white women. The risk of falling, bone density and structure, the direction of the fall, the height of the fall, and the area and force of impact ultimately determine the incidence of osteoporetic fractures associated with falling and account for higher mortality in women across all age groups.[4, 10, 11, 12]

Intrinsic factors

Table 1. Risk Factors for Falls in Older Adults

Risk Factors for Falls

Intrinsic Factors	Extrinsic Factors	Situational/Activity-related Factors
Advancing age	Environmental hazards:	Climbing ladder
Female gender	Inadequate lighting	Ascending/descending stairs
Gait and balance impairments	Slippery floor surfaces	Rising from a chair
Acute & chronic illnesses	Loose rugs	Turning around quickly
Cognitive & functional impairment	Uneven steps/surfaces	Reaching
Depression	Cluttered walkways	Walking on uneven surfaces
Visual deficits	Use of assistive device	
Muscle weakness	Ill-fitting shoes	
Depression		
Polypharmacy		
History of previous falls		

Intrinsic factors that vary with time include acute illnesses such as pneumonia, urinary tract infections, congestive heart failure, blood loss, hypoxemia, syncope, dehydration, and recent hospitalizations. Acute illnesses and exacerbation of chronic illnesses may cause acute confusion and functional changes, thereby increasing the risk of falls.[3, 9, 10, 11]

Abnormalities of gait and balance are also predictive of fall.[3, 10, 11, 12, 13] An abnormal gait is often related to pain, joint stiffness, muscle weakness, spasticity and rigidity, sensory and balance deficits and impaired central processing. These symptoms often result from the accumulated effects of chronic disease, age-related and life-style changes and impairments in sensory, neurological and musculoskeletal functioning. For example, pain or joint limitation related to an arthritic hip results in antalgic gait, assumed to avoid or lessen pain. The person will lean his/her body over the painful side to reduce the weight on the involved hip. Loss of joint mobility may lead to contractures and further impair gait and balance. Spasticity and rigidity related to neurological disorders may affect gait and balance. Rigidity, a common presentation in Parkinson's disease, may result in a flexed posture, shuffling gait with small steps and diminished arm swing.[14, 18]

Postural control is determined by the integration of visual, proprioceptive and vestibular input within the central nervous system. Toxic and metabolic encephalopathies and peripheral neuropathies secondary to diabetes impair balance, as do alcoholism, vasculitis, drug intoxications, and dysfunction in central integration. Depression, dementia, normal pressure hydrocephalus, and frontal lobe syndrome cause impairment in the limbic system and the integration of sensory information from the internal and external environment.[8, 10, 11,13]

Impaired visual acuity, depth perception, and contrast sensitivity have been associated with increase risk of falling and hip fractures in older adults. Visual cues help the walker maintain balance, move and avoid environmental hazards. Age-related visual changes and ocular diseases such as macular degeneration, glaucoma and cataracts increase the risk of falls.[10, 11,12,14]

Foot problems may impair gait and balance. Bunions, callouses, anatomical deformities, pain and neuropathy will alter the older adult's gait and impair proprioception.

Certain medications, changes in dose, and the total number of medications have been associated with increased fall-risk. Centrally acting medications, such as benzodiazepines, neuroleptics, barbituates and antidepressants, decrease alertness, affect judgment, compromise neuromuscular function or cause dizziness and syncope. Narcotic analgesics, antihypertensives, anticonvulsants, nonsteroidal anti-inflammatory, and antiarrhythmics, may impair the sensorium and gait and balance,. Diuretics and antihypertensive medications may cause postural hypotension, fatigue, and electrolyte imbalances.[3, 9, 10,11, 15, 16]

A history of falls is also a predisposing factor for subsequent falls. The number of risk factors and the limitation of function whether due to physical injury or fear of falling may contribute to the increased rate of falling again.[3, 8, 11]

Extrinsic factors

Extrinsic conditions – those circumstantial to the adult who falls - increase the risk of falling. Environmental hazards include inadequate lighting, ill-fitting shoes, loose carpets, lack of non-skid rugs and safety equipment in the bathroom and bedroom, cluttered stairways or walkways, high steps and slippery or icy surfaces.

Activity related factors

The third category is situational or activity-related factors; e.g, climbing on ladders or chairs, walking on uneven surfaces, turning around quickly, reaching up or bending over, and standing or rising from a chair.

Most falls result from a cumulative effect of intrinsic, extrinsic and situational-related activities. The most consistently successful strategy in preventing falls has been a multi-factorial falls risk assessment, followed by interventions targeting the modifiable risk factors.[9, 11, 17]

Assessment

The first step in assessing patients is to identify modifiable risk factors. The American Geriatrics Society, British Geriatrics Society, and the American

Tinetti Assessment Tool: Balance

Patient's Name: Date:
Location: Rater:

Initial Instructions: Subject is seated in a hard, armless chair. The following maneuvers are tested.

Task	Description of Balance	Possible	Score
1. Sitting Balance	Leans or slides in chair Steady, safe	= 0 = 1	
2. Arises	Unable without help Able, uses arms to help Able without using arms	= 0 = 1 = 2	
3. Attempts to arise	Unable without help Able, requires > 1 attempt Able to rise, 1 attempt	= 0 = 1 = 2	
4. Immediate standing balance first 5 seconds	Unsteady (swaggers, moves feet, trunk sway) Steady but uses walker or other support Stead without walker or other support	= 0 = 1 = 2	
5. Standing Balance	Unsteady Steady but wide stance (medial heels > 4 inches apart) and uses cane or other support Narrow stance without support	= 0 = 1 = 2	
6. Nudged (subject at max position with feet as close together as possible, examiner pushes lightly on subject's sternum with palm of hand 3 times.	Begins to fall Staggers, grabs, catches self Steady	= 0 = 1 = 2	
7. Eyes closed (at maximum position #6	Unsteady Stead	= 0 = 1	
8. Turning 360 degrees	Discontinuous steps Continuous steps Unsteady (grabs, swaggers) Stead	= 0 = 1 = 0 = 1	
9. Sitting Down	Unsafe (misjudged distance, falls into chair) Uses arms or not a smooth motion Safe, smooth motion	= 0 = 1 = 2	
		Balance Score:	

Figure 1. Tinetti Assessment Tool: Balance

Tinetti Assessment Tool: Gait

Patient's Name: Date:
Location: Rater:
Initial Instructions: Subject stands with examiner, walks down hallway or across the room, first at "usual" pace, then back at "rapid, but safe" pace (using usual walking aids).

Task	Description of Gait	Possible	Score
10. Initiation of gait (immediately after told to "go")	Any hesitancy or multiple attempts to start No hesitancy	= 0 = 1	
11. Step length and height	a. Right swing foot does not pass left stance foot with step b. Right foot passes left stance foot c. Right foot does not clear floor completely with step d. Right foot completely clears floor e. Left swing foot does not pass right stance foot with step f. Left foot passes right stance foot g. Left foot does not clear floor completely with step h. Left foot completely clears floor	= 0 = 1 = 0 = 1 = 0 = 1 = 0 = 1	
12. Step Symmetry	Right and left step length not equal (estimate) Right and left step appear equal	= 0 = 1	
13. Step Continuity	Stopping or discontinuity between steps Steps appear continuous	= 0 = 1	
14. Path (estimated in relation to floor tiles, 12-inch diameter; observe excursion of 1 foot over about 10 feet of the course).	Marked deviation Mild/moderate deviation or uses walking aid Straight without walking aid	= 0 = 1 = 2	
15. Trunk	Marked sway or uses walking aid No sway but flexion of knees or back, or spreads arms out while walking No sway, no flexion, no use of arms, and no use of walking aid	= 0 = 1 = 2	
16. Walking Stance	Heels apart Heels almost touching while walking	= 0 = 1	
	Gait Score:		
	Balance + Gait Score:		

Figure 2. Tinetti Assessment Tool: Gait

Academy of Orthopedic Surgeons together developed an evidence-based guideline for the prevention of falls. The intensity of assessment depends upon the target population. A primary care physician, as part of the routine evaluation of any healthy older adult living in the community, should elicit a fall history. The physician should perform a more detailed, comprehensive assessment on older adults presenting after a fall, living in a nursing home or having a history of falls.

The comprehensive assessment should include a history of the circumstances of the fall, identification of risk factors of falling, a review of systems for acute medical symptoms, chronic conditions, cognitive, and functional status. A review of the level of activity, nutrition status and alcohol use are important. Screening for depression may reveal vegetative symptoms, poor concentration or apathy. A careful review of prescription and over-the-counter medications should be included. If the older adult is presenting after a fall, a careful description of symptoms, activity at the time of the fall and environmental conditions should be recorded.

The physical exam should focus on the risk factors identified during the history and include an assessment of cardiopulmonary function, neurological function, gait and balance testing, visual screen and an examination of the lower extremities. Cardiopulmonary exam should include assessment of heart rate and rhythm, orthostatic blood pressure, and respiratory status. A basic neurological exam should focus on mental status assessment, muscle strength and tone, reflexes, proprioception, vibratory sense, and tests of cortical, extrapyramidal and cerebellar function. An examination of the lower extremities is especially important including assessment of peripheral nerves, muscle strength, tone, range of motion and any foot deformities, ulcerations, or other painful processes.[9, 11]

In addition, the physician should examine the patient's gait and balance. Many causes of gait and mobility disturbances can be treated. The Tinetti Assessment Tool will detect most gait and balance problems of older adults. (Figure 1 and 2) The screening test is scored from 0 (most impairment) to 2 (independent) on the adults' ability to perform certain tasks. The maximum total score is 28 points, 19 or lower indicates high risk for falls, and 19-24 indicates a risk for falls.[6, 9, 11, 13, 18]

Gait abnormalities may also represent a compensatory mechanism to increase stability and reduce pain. Difficulty rising from a chair may indicate proximal muscle weakness, deconditioning or arthritis. A wide-base stance with shortened strides and en bloc turns may compensate for poor vision or proprioception or a fear of falling.[13, 20]

Additional maneuvers, reaching up, turning the neck while standing, bending over and standing on one leg unsupported for five seconds stress stability and are maneuvers routinely performed in daily activities. The physician should note any movements or gestures that suggest instability, such as staggering or reaching for support. These screening tests assist in identifying functional problems and areas for improvement. Canes and walkers can enhance the adult's balance and ability to bear weight. The gait and balance assessment should include evaluation of the appropriate ambulation device, size, and proper use.[20]

Laboratory and Diagnostic Testing

Laboratory and diagnostic testing may be useful in determining the etiology of the fall. A complete blood count, BUN, creatinine, electrolytes, glucose, and thyroid function test may be indicated to search for electrolyte imbalances, dehydration, anemia, infection or thyroid dysfunction. Drug levels need to be monitor in older adults on anticonvulsants, antiarrhythmics, and tricyclic antidepressants to rule out toxicity.[9, 11]

If an acute illness is suspected, complete blood count, electrolytes, pulse oximetry at rest and with exertion, electrocardiogram, cardiac enzymes, chest radiograph, urine analysis and culture may be useful in determining the etiology of the fall. More extensive testing such as cardiac holter monitoring, may be indicated if the symptoms are intermittant and suggestive of transient arrhythmias or syncopal episodes.

Neuroimaging may be indicated in

adults with focal neurological deficits or abnormal gait and balance assessment. [9, 11] If a patient presents with an abnormal gait, lower extremity spasticity and hyperreflexia, cervical spine films may help rule out cervical spondylosis. Referral to a specialist (podiatrist, neurologist, cardiologist, opthalmologist) may be necessary for further evaluation.

Management and Treatment

The management and treatment of falls is guided by the patient's history and physical examination. The aim of treatment is to reduce the risk of falling while maintaining the patient's functional independence and personal autonomy. For patients who have fallen, treatment is focused on reducing the risk of subsequent falls by modifying and eliminating potential risk factors. Initial treatment of acute illnesses such as infection, metabolic disturbances or adverse drug reactions may improve cognition and gait and balance. However, falls, which tend to be multi-factorial, often require multiple interventions.

The Panel on Falls in Older Adults has proposed strategies for at risk populations.[11] Recommendations include gait training; evaluation of appropriate ambulatory device; review and modification of medications; exercise program which includes strength and balance training; home safety evaluation; treatment of postural hypotension and other cardiac disorders which may contribute to fall risk; and education on fall-prevention for long term care staff.[11]

Exercise programs that focus on gait and balance training, strengthening and endurance help to maintain confidence, flexibility, range of motion, and independent mobility. A referral to physical therapy for gait and balance training and evaluation for an ambulatory device is helpful in increasing gait stability. Physical therapy may have special exercise programs for older adults with osteoarthritis and Parkinson's disease.

Tai Chi has shown promise in increasing postural control and reducing falls. Bed alarms, motion detectors and low beds may be useful in reducing the number of falls in the long term care or acute care settings as a component of the multifactorial approach. Hip protectors in high risk groups may reduce the incidence of hip fractures associated with falling. Physical restraints have not been shown to reduce falls. In fact, vest poseys, wrist restraints and bed rails can lead to injurious falls, immobility, and pressures ulcers.[9, 11]

A home safety assessment by the home care nurse or physical therapist may eliminate environmental hazards, particularly in the bathroom and bedroom. Eliminating loose rugs, clutter in walkways, providing adequate lighting and grab bars and raised toilet seats in bathrooms will reduce extrinsic risk factors.

The treatment of chronic illness, such as cardiovascular disease (postural hypotension, syncope, arrhythmias), osteoporosis, arthritic conditions and neurological disorders, such as Parkinson's disease, spinal stenosis, Alzheimer's disease, is pertinent in the overall fall-prevention. In addition, referral to specialists for evaluation of visual problems, hearing problems, foot problems or focal neurologic deficits may be helpful.

Conclusions

The primary care physician can be instrumental in preventing falls in older adults. S/he should routinely screen for falls. Patients thought to be at higher risk or who have suffered falls should undergo a more detailed assessment. Although it is unclear at what age screening should begin,[9] age 70 seems a reasonable empiric guideline. By identifying patients at risk and implementing a multidisciplinary risk reduction program, the primary care physician can assist them to reduce disability and possibly the need for long term care.

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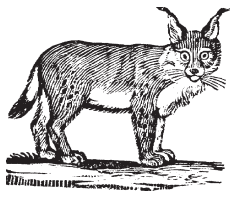

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Influenza 2004–2005

Mark Schwager, MD

The Vaccine Shortage

The Influenza season for 2004-2005 started abruptly on October 5, 2004, before a single case of the disease had been detected, when US health officials learned that 50% of anticipated influenza vaccine would be unavailable for distribution. On that day, Chiron Corporation announced that British authorities had impounded 48 million doses of flu vaccine, intended for the United States, because of bacterial contamination at its manufacturing plant outside Liverpool. [1] At the time of Chiron's announcement, the nation's other remaining injectable vaccine manufacturer, Aventis Pasteur, had shipped 33 million of its expected 58 million doses. The **Centers for Disease Control and Prevention (CDC)**, working with Aventis, took the unprecedented step of applying a reallocation plan to the distribution of the remaining 25 million doses by designating eight priority groups for influenza vaccination (http://www.cdc.gov/flu/). In managing this acute shortage, our public health system has been forced to confront the vulnerability of the nation's vaccine supply.

In the first phase of distributing the remaining vaccine, the CDC and Aventis targeted high-risk patients, shipping 14.2 million doses of vaccine over a six-week period to Veterans Administration facilities, nursing homes, acute care hospitals, and pediatricians. Veterans Administration facilities and pediatricians were allocated at 100%, with hospitals, nursing homes, assisted living facilities and adult day care receiving appropriate amounts of vaccine to immunize residents and direct care staff. Private sector Aventis customers, as well as state and local public health departments, were told they would recieve up to 50% of their initial orders, based on needs.

In phase two of distribution, CDC and Aventis will ship another 10.2 million doses of vaccine to state health departments by the end of January. This will be based on how many high-risk people each state has and the number of doses already received.

Paradoxically, the shortage of influenza vaccine may motivate people at risk who never get the flu vaccine to seek it now because of the publicity surrounding the vaccine shortage. In the most recent National Health Interview Survey, only 43 million doses of vaccine would be required in 2004 to immunize the CDC high-priority groups at the same rates reported for the 2002-2003 season. [2]

The Rhode Island Response

Rationing of vaccine dictates that some high-risk citizens will not be immunized. All local health departments, therefore, are in the difficult role of deciding how to ensure available vaccine is allocated in the fairest way possible.

When the vaccine shortage came to light, the **Rhode Island Department of Health (HEALTH)** published the "State Influenza Outbreak Plan" (http://www.health.state.ri.us/flu/fluoutbreakplan.pdf) and convened a Flu Vaccine Shortage Advisory Group comprised of physicians, healthcare providers, medical ethicists and insurers. Vaccine Updates have been faxed to the physician community and are also available at the HEALTH website, www.healthri.org. The advisory committee, with little evidence-based criteria available, developed expert opinion-based criteria for prioritizing within the CDC high-risk group. On November 16, 2004, HEALTH recommended moving up the age for vaccinating healthy adults in the community from age 65 to age 75 and excluding adults under 50 with intermittent asthma and diabetics managed by diet and exercise alone.

Approximately 185,000 doses of flu vaccine have been shipped to Rhode Island and delivered to pediatricians, hospitals, long-term care facilities, assisted living residences and adult day care facilities. Approximately 50,000 additional doses of vaccine will be distributed by HEALTH through January 2005. HEALTH will maximize the benefit of the vaccine that is available by focusing vaccination efforts on the most vulnerable among the high-risk population. [3]

Vaccine Vulnerability

The vaccine shortage of 2004-2005 and **an Institute of Medicine (IOM)** report, "Financing Vaccines in the 21st Century," have highlighted weaknesses in the structure of the US vaccine supply and distribution system. Our public health system has not addressed the relationship between financing and availability of vaccine while the market-driven purchasing and acquisition system for vaccine has caused a consolidation of vaccine manufacturers. A public-private partnership which involves financial incentives is necessary to protect the existing vaccine supply and to develop new products and vaccines. In addition, the low reimbursement providers receive for vaccine administration exacerbates the problems of distribution. To address vaccine-access disparities and ensure higher overall immunization rates, the IOM recommended government vaccine subsidies be substantially increased, that every health insurance policy include full coverage for vaccination, and that uninsured individuals receive vouchers to obtain recommended vaccines.[4]

Lessons learned from the current shortage on the national and local level will increase preparedness for the next public health emergency such as an influenza pandemic, the emergence of an avian flu mutant that could spread among humans or a bioterrorist attack. The organization, planning, methods of communication, and liaisons developed by HEALTH this year can be applied to other public health emergencies in the future.

On a broader scale, the **World Health Organization (WHO)** convened a meeting of over 50 representatives from drug companies, government, and vaccine-licensing agencies to address patent issues and fears of making huge investments in future vaccines.[4]

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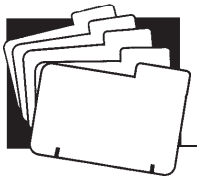

Hospital Case Files

Case Presentations of the Brown University Department of Medicine

Miriam Hospital and Rhode Island Hospital Morbidity and Mortality Conferences, Two Cases of Sudden Weakness

Mary H. Hohenhaus, MD, and Randall S. Pellish, MD

Case 1

Chief Complaint: Leg weakness

History of Present Illness:

A 20-year-old Laotian man with no significant medical history presented after awaking in the middle of the night with profound weakness of his lower extremities. He was unable to bear weight and also noted a milder degree of upper extremity weakness. He had no back pain or sensory changes, nor had he noted any change in bowel or bladder function. On the night prior to admission he reported having a mild frontal headache and an episode of vomiting. He had played soccer the day prior to admission without any difficulty. On further review of his history, the patient recalled a similar episode that occurred approximately one month prior, during which he experienced similar transient lower extremity weakness that resolved spontaneously after 12 hours. He sought evaluation in the Rhode Island Hospital emergency department.

Review of Symptoms:

The patient reported having episodic palpitations, frequent sweats, and tremulousness for many years. He denied diarrhea, fevers, chills, seizures, arthralgias, and myalgias.

Past Medical History: Negative.

Medications: No prescription or over-the-counter medications, no supplements or herbal remedies.

Allergies: No known drug allergies.

Social History: Lives with his family. Student, part-time maintenance worker. No smoking, alcohol consumption, or drug use.

Family History: Father with a heart murmur.

Physical Exam in the ED

Vital Signs: Temp: 98.1F HR: 111 BP: 137/65 RR: 16 SaO2 98% RA

General: Asian male, thin, no apparent distress.
HEENT: PERRL, EOMI, sclerae anicteric, oropharynx clear.
Neck: Supple. Thyroid diffusely enlarged, nontender.
Cardiovascular: Tachycardic with a regular rhythm. II/VI blowing systolic ejection murmur loudest at left upper sternal border.
Lungs: Clear to auscultation bilaterally.
Abdomen: Normal bowel sounds, soft, nontender, nondistended.
Extremities: No edema or cyanosis. Muscles nontender to palpation.
Neurologic: Alert and oriented. Speech

clear, coherent, and fluent. Cranial nerves II-XII intact. Upper extremities displayed a fine tremor with proximal 3/5 strength and distal 4/5 strength. Lower extremities with proximal and distal 2/5 strength with inability to lift legs off the bed. Sensory exam intact. Unable to elicit patellar or Achilles reflexes, biceps and brachioradialis reflexes were 1+. Toes down-going bilaterally.

LABS:
CBC: WBC count: 10,000 per mL
Hemoglobin: 15.4 g/dL
Hematocrit: 46.4%
Platelet cont: 282,000 per mL
WBC Differential: 85% segmented neutrophils, 13% lymphocytes, 1% monocytes, 1% eosinophils

Chem 7:
Sodium: 141 mmol/L
Potassium: 1.6 mmol/L
Chloride: 109 mmol/L
Bicarbonate: 24 mmol/L
BUN: 15 mg/dL
Creatinine: 0.7 mg/dL
Glucose: 138 mg/dL

Chemistry:
Calcium: 9.4 mg/dL
Magnesium: 1.3 mg/dL
Phosphorus: 1.7 mg/dL
CPK 675 IU/L
Troponin I: < 0.15 ng/mL

Chest radiography: Negative
Computed tomography of the brain: Negative
Electrocardiogram: Sinus tachycardia, possible left ventricular hypertrophy with associated ST-T changes, also a question of anterolateral T wave abnormalities.

Emergency Department Course: Potassium repletion was begun in the emergency department with a 60 mEq PO dose and a 10 mEq IV dose.

Hospital Course:

The patient was admitted to the medical step-down unit secondary to his severe electrolyte abnormalities. Potassium repletion was continued, and magnesium was also repleted. Immediate laboratory evaluation for thyroid disease revealed a thyroid-stimulating hormone (TSH) <0.03 μU/mL and a free T_4 of 5.65 ng/dL consistent with thyrotoxicosis. Propranolol therapy was initiated and a thyroid uptake and scan was ordered.

The thyroid uptake and scan revealed 87% uptake in a homogenous pattern consistent with Graves disease. Methimazole therapy was instituted and propranolol was continued. An echocardiogram was performed to evaluate for left ventricular hypertrophy. The study was normal with the exception of mild RV dilation. The patient's weakness quickly resolved with normalization of his potassium level and he was discharged on hospital day 3.

Case 2

Chief Complaint: "My strength is gone."

History Of Present Illness:

A 30-year-old African American male reported a 2-year history of hyperthyroidism. He was in his usual state of health until approximately 10 days prior to admission. At that time, he noted increasing anxiety, irritability, heat intolerance, palpitations, diarrhea, and intermittent blurry vision. He presented to an outside hospital with an episode of severe generalized weakness and was found to have a serum potassium level of 1.8 mmol/L. The weakness resolved with potassium repletion and he was discharged after a brief stay.

A week after discharge, the patient was seen by his primary care physician. The patient complained of persistent mild weakness and intermittent leg cramps. Serum potassium level was 4.7 mmol/L. That night, he awoke with the urge to urinate, but had extreme difficulty lifting his arms and legs. He could not get out of bed without assistance, and subsequently sought evaluation in the Miriam Hospital emergency department.

Review of Symptoms: The patient reported a 30-pound weight loss over a 2-year period. No chest pain or shortness of breath. No melena or bright red blood per rectum.

Past Medical History: Graves disease confirmed by 24-hour thyroid uptake and scan 1 month prior to admission (58.5%, which may have been an underestimate given recent use of propylthiouracil). Had been scheduled for thyroid ablation treatment, but did not keep appointment. Hypertension.

Medications: Atenolol 50 mg daily. Erratic use of propylthiouracil.

Allergies: No known drug allergies.

Social History: Quit smoking 1 month prior. Denies alcohol, occasional marijuana. Lives with mother, receives disability benefits.

Family History: Parents alive, father with diabetes. Siblings healthy, no thyroid disease.

Physical Exam In the ED
Vital Signs:
Temp: 36.8 C HR: 121 BP: 167/90
RR: 18 SaO2: 99% RA
General: Anxious-appearing, diaphoretic young adult male in mild distress.
HEENT: Mild exophthalmos
Neck: Thyroid symmetrically enlarged, nontender, positive bruit.
CVS: Tachycardic, no murmur, rub, or gallop.
Lungs: Clear to auscultation bilaterally.
Abdomen: Positive bowel sounds, soft, nontender, nondistended. Occult blood negative.
Extremities: No cyanosis, clubbing, or edema.
Neurologic: Strength 4/5 upper and lower extremities bilaterally, deep tendon reflexes 3+ with swift relaxation phase, otherwise nonfocal.

LABS:
CBC: WBC count: 4,500 per mL
Hematocrit: 39.4%
Platelet count: 273,000 per mL
WBC differential: 63% segmented neutrophils, 1% bands, 30% lymphocytes, 6% monocytes

Chem 7:

Sodium: 139 mmol/L
Potassium: 2.4 mmol/L
Chloride: 104 mmol/L
Bicarbonate: 26 mmol/L
BUN: 15 mg/dL
Creatinine: 0.6 mg/dL
Glucose: 225 mg/dL

Chemistry:
Calcium: 9.2 mg/dL
Magnesium: 1.4 mg/dL
CPK: 110 IU/L

Thyroid function studies:
3rd generation TSH: 0.084 uU/mL
Free T_3: >2000 pg/dL
Free T_4: 7.63 ng/dL
Hemoglobin A_1C: 6.3%
Electrocardiogram: Sinus tachycardia, rate 125.
Abdominal computed tomography: No adrenal masses.

Emergency Department Course: Potassium repletion was begun in the emergency department, with 40 mEq PO and 40 mEq IV.

He also received labetalol 10 mg IV and 1 liter normal saline IV.

Hospital Course:

The patient was admitted to the medical service for monitoring, repletion of electrolytes, and management of hyperthyroidism. Endocrine and Renal consultation were obtained. He was started on propranolol, propylthiouracil, and intravenous hydrocortisone for his hyperthyroidism. His weakness improved significantly with postassium repletion, but waxed and waned, possibly in association with administration of a sliding-scale insulin regimen.

He was discharged to home on hospital day 8 with weakness resolved and symptoms of hyperthyroidism controlled.

Diagnosis: Thyrotoxic hypokalemic periodic paralysis

Discussion

What is the association between thyrotoxicosis and hypokalemic periodic paralysis?

Hypokalemic periodic paralysis is an uncommon disorder characterized by sudden shifts of potassium into the intracellular compartment. It is most commonly described in young Asian or Hispanic males, as in Case 1, but is not exclusive to those groups. Physicians in Asia and Latin America are well-acquainted with the diagnosis, but physicians in the United States frequently miss it.

Clinically, it is characterized by episodic weakness affecting proximal muscles more so than distal ones; bulbar and respiratory muscles are much less commonly involved, although respiratory failure has been described. Cardiac arrhythmias can occur and may be fatal. Some patients experience a prodrome of muscle pain or cramping. Not surprisingly, creatine phosphokinase levels may be elevated.

Episodes are likely triggered by increased secretion of epinephrine or insulin. They frequently occur after a period of rest after exercise or at night – hence the names "night paralysis" and "night palsy" – and can also be elicited by carbohydrate or sodium loading. Serum potassium levels return to normal between episodes.

The disorder has both inherited and acquired forms. The familial form is inherited in an autosomal dominant pattern, although with incomplete penetrance. A mutation in voltage-sensitive calcium channels in skeletal muscle appears to be involved but is not well understood.

Thyrotoxic periodic paralysis is clinically indistinguishable from the familial form. The hypokalemia appears to be related to the hyperadrenergic state and an exaggerated insulin response, with increased activity of Na-K-ATPase at skeletal muscle ion channels. Asian males with hyperthyroidism appear to be at particular risk, with as many as 10% exhibiting symptoms of periodic paralysis. Hyperthyroidism may not be clinically apparent, consequently thyroid function studies should be considered in any patient presenting with episodic weakness.

What is the management of thyrotoxic periodic paralysis?

Oral potassium repletion has long been the mainstay of treatment for acute episodes of both forms of hypokalemic periodic paralysis. Weakness typically resolves quickly. Potassium should be given cautiously, especially in patients with impaired renal function, as rebound hyperkalemia may occur as potassium shifts extracellularly. It is important to remember that patients with periodic paralysis are not total body potassium depleted.

Phosphate and magnesium levels may also be low, but typically return to normal with normalization of potassium levels. Nonselective beta-adrenergic blockers can help prevent further attacks by attenuating potassium shifts; they are also effective in acute episodes and avoid the risk of rebound hyperkalemia. Correction of the underlying thyroid disorder and reestablishment of the euthyroid state is the definitive treatment.

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Acknowledgement: Papa Kaku Badoe, MD; Elisa Freeman, MD; Olga Lurye, MD; Michael Maher, MD; Ian Wu, MD

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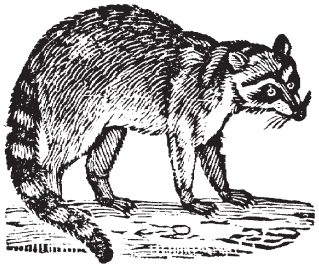

HOSPITAL COMMUNITY BENEFITS IN 2003

BRUCE CRYAN, MBA, MS

The organization of Rhode Island's thirteen community hospitals[1] as 501(c)(3) corporations enables them to carry out their charitable missions without the tax liabilities imposed on for-profit companies. The value of this "not-for-profit" designation is considerable and has fourfold primary significance:

1. Exemption from local property taxes,[2]
2. Exemption from state and federal corporate taxes,
3. Preferential borrowing at tax-exempt rates,[3] and
4. The ability to solicit charitable donations and to invest those monies without tax liability on income earned.

The community, in waiving its right to these revenues, implicitly anticipates that it will benefit from the "public good" of certain hospital benefits, including the provision of healthcare services to all regardless of ability to pay (i.e., uncompensated care).

Rhode Island was one of the first states in the nation to examine hospital community benefits. Since 1989, the **Rhode Island Department of Health (HEALTH)** has analyzed and reported on this issue.[4] In 1997, the General Assembly passed the Hospital Conversions Act (the Act) that further codified the public reporting of these activities.

METHODS

The Act and its regulations broadly define community benefits as:

"…the provision of hospital services that meet the ongoing needs of the community for primary and emergency care …and shall …not be limited to, charity care and uncompensated care. (and)…programs …that meet the needs of the medically indigent; linkages with community partners(,)…non-revenue producing services(,)…public advocacy…(and) scientific, medical research, or educational activities."

In order to quantify the hospitals' efforts in providing community benefits, an annual survey instrument is used to collect descriptive data, and audited financial statements and Medicare Cost Reports are used to provide financial data.

In addition, HEALTH's Minority Health Advisory Committee has advocated that the hospitals' diversity of governance and administration is an important part of their corporate mission. The Committee reasoned that the health of a community is enhanced when it sees itself actively participating in its own healthcare. Therefore, data have also been collected on hospital diversity and benchmarked to the general population in the state.

RESULTS

Notwithstanding the fairly broad regulatory definition of community benefits, charity care and bad debt remain the most fundamental measures of a hospital's community benefits. Both represent an accounting for the uncompensated healthcare provided by a hospital, even though they are technically different from an accounting standpoint and practically different from the patient's standpoint. Uncompensated care as a whole simply means that payment was not received or was waived by the hospital, it does not mean that reimbursement was insufficient to cover expenses. Charity care is the charges recorded for services delivered but never billed because the hospital makes a prospective determination the patient is incapable of payment. Bad debt, on the other hand, is the billing for services rendered but never collected and written off as a business expense.

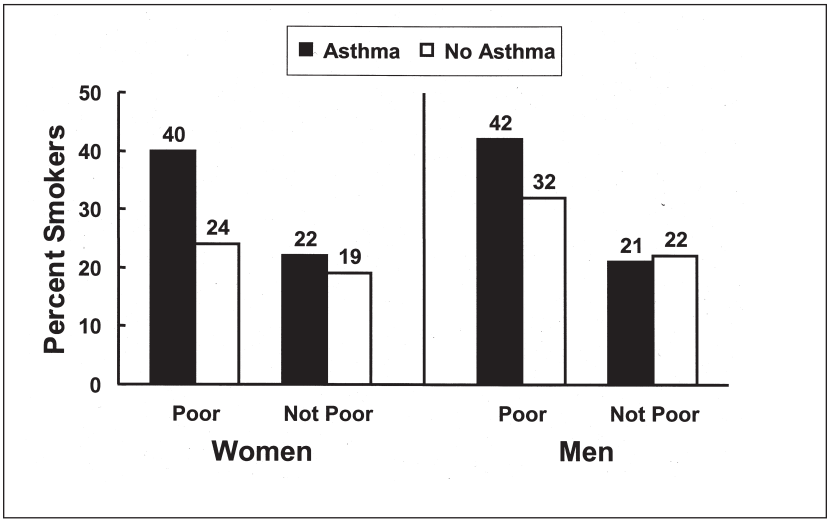

FIGURE 1. UNCOMPENSATED HOSPITAL CARE, BY YEAR AND TYPE, RHODE ISLAND, 1995-2003.

Figure 1 provides the statewide charity care and bad debt amounts from 1995 through 2003. Over this nine-year period, the uncompensated care amounts were fairly consistent, averaging $53.8 million annually. "Fitting" a trend line to these historical data shows uncompensated care increasing by almost $1.3 million per year, on average.

The operational classification of charity care and bad debt has been blurred by

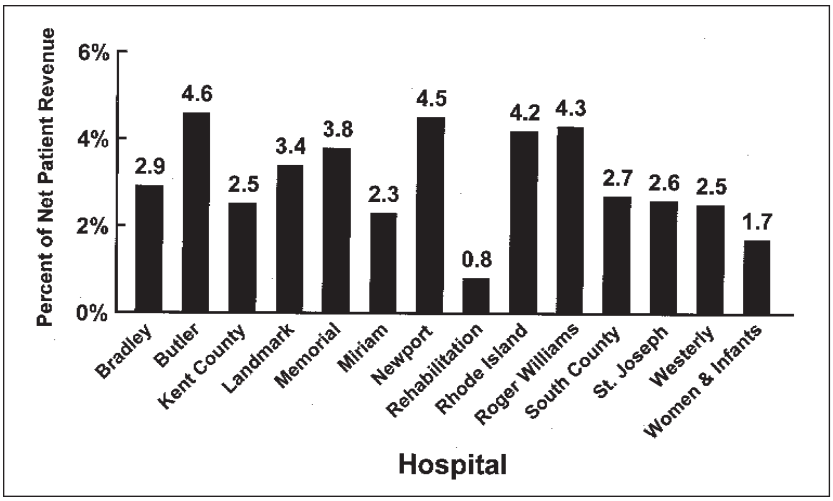

FIGURE 2. UNCOMPENSATED HOSPITAL CARE AS PERCENT OF NET PATIENT REVENUE, BY HOSPITAL, RHODE ISLAND, 2000-2003 (COMBINED).

non-uniform accounting practices and determinations at the time of admission. Often there is reticence on the part of individuals to disclose financial hardship, and so, the eventual non-payment for services falls under bad debt rather than charity care. It is for this reason, the somewhat arbitrary categorization of non-paying patients, that combining both charity care and bad debt is the most precise way to measure a hospital's 'burden' in treating the indigent.

Figure 2 presents each hospital's charity care and bad debt amounts together, on a relative basis for the period 2000-2003. Aggregating four years of data removes any outliers associated with a single year's reporting. Presenting the value as a percentage of the hospital's net patient revenue further standardizes the statistic for comparison purposes. Uncompensated care percentages ranged from highs greater than 4% at Butler, Newport, Roger Williams and Rhode Island Hospital to lows below 2% at Rehabilitation Hospital and Women & Infants Hospital.

Table 1 presents the level of hospital diversity statewide. Hospital governance (i.e., Board members and senior administrators) is not diverse, not reflective of the general population, nor has it changed appreciably since 1998. In 1998, hospital Boards were 27% female, 5% racial minority, and 0% Hispanic. In 2003, that representation was 27% female, 6% racial minority, and 1% Hispanic. The only category that mirrors the general population was the Black representation at 4.5%.

With the exception of gender diversity, senior hospital administrators were even less diverse than the Boards. In 1998, hospital administrators were 35% female, 0% racial minority, and 0% Hispanic compared with 43% female, 2% racial minority, and 0% Hispanic in 2003. Asians were the only minorities represented at all, and Hispanics and Blacks were totally absent.

DISCUSSION

Since 1998, HEALTH has had formal data collection efforts to track and quantify hospital community benefits. As the debate over what constitutes a 'charitable' organization continues, policy makers struggle with the need for more and better information. In response, HEALTH has expanded its public reporting and revised the metrics used to frame and analyze the issues.

In addition, HEALTH also uses this opportunity to align the hospitals' community benefits activities with its own goals of Healthy Rhode Islanders 2010, the blueprint for public health in the state. Understandably, not every hospital addresses all of the state's 2010 objectives, because each hospital's priorities should reflect its own community needs, but it does serve to illustrate where the industry and the state may work together effectively.

Table 1. Demographic Diversity of Hospital Board Members and Senior Administrative Staff, Rhode Island, 2003

DEMOGRAPHIC CHARACTERISTICS		HOSPITAL BOARD MEMBERS (N=287)	HOSPITAL ADMINISTRATIVE STAFF[1] (N=114)	RHODE ISLAND POPULATION[2]
Ethnicity	Hispanic/Latino	*1%*	*0%*	*9%*
	Not Hispanic/Latino	*99%*	*100%*	*91%*
	Totals	*100%*	*100%*	*100%*
Race	American Indian	*0%*	*0%*	*0.5%*
	Asian	*1.4%*	*1.8%*	*2.3%*
	Black/African-American	*4.5%*	*0%*	*4.5%*
	Native Hawaiian/Islander	*0.3%*	*0%*	*0.1%*
	White	*93.7%*	*98.2%*	*84.9%*
	Other or Multiple Races	*0%*	*0%*	*7.7%*
	Totals	*100%*	*100%*	*100%*
Gender	Female	*27%*	*43%*	*52%*
	Male	*73%*	*57%*	*48%*
	Totals	*100%*	*100%*	*100%*

[1]Vice-President level (however titled) and above
[2]2000 U.S. Census data

REFERENCES

1. Rehabilitation Hospital of RI, a Limited Partnership, became a wholly owned subsidiary of Landmark in June 2000.
2. Some hospitals offer payment in lieu of taxes to their host communities.
3. Tax-exempt interest rates are typically less than rates charged to commercial borrowers.
4. Cryan B. *Uncompensated Care Services in RI's Community Hospitals.* Providence RI: Rhode Island Department of Health. 1989; and Cryan B. *Uncompensated Care and Tax Exemption of RI's Hospitals.* Providence RI: Rhode Island Department of Health. 1993.

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COLORECTAL CANCER SCREENING RESOURCES IN RHODE ISLAND

ARVIN S. GLICKSMAN, MD

There is essentially universal agreement that timely and appropriate colorectal screening can detect and remove precancerous polyps and/or detect colon cancer in an early, curative stage. In a previous survey,[1] the Rhode Island Cancer Council found that there was uniform agreement of the gastroenterologists and surgeons who perform endoscopy and the primary care physicians that colonoscopy was the preferred procedure for colorectal cancer screening (the gold standard). Currently, approximately 50% of the population over the age of 50 have not had any test for colorectal cancer whatsoever. Some concern was expressed that if a major educational program were to increase the number of individuals seeking colorectal cancer screening, resources within the State might be overwhelmed. Accordingly the Rhode Island Cancer Council undertook a survey of the endoscopists in the State, which sought information concerning the capacity of current resources and its utilization. A second survey was performed to determine the length of time it would take to schedule an endoscopy appointment.

Sixty-eight questionnaires were sent out to endoscopists we could identify in Rhode Island. Forty-two (62%) were returned. Ninety percent of the respondents have experienced an increase in referrals/requests for colonoscopy in the last year. They reported that 80% of the patients are aware of their status as either a standard risk or being at high risk for colon cancer and they reported that 33% of the procedures resulted in finding some abnormality. (Table 1)

On average, the responding endoscopists reported performing 75 procedures per month. They indicated that they believe their practices could accommodate approximately twice the number that they are performing.

The endoscopists reported that they all performed endoscopy examinations in a hospital endoscopy suite. In addition, a third of the endoscopists also utilized a dedicated freestanding endoscopy suite and only 10% performed endoscopies in their office suites. At no site did they report that the demand exceeded the capacity for performing colonoscopy.

On the basis of these data, an increase in the number of educational programs to improve the number of Rhode Islanders seeking this cancer screening examination can move forward without concern of overwhelming our capacity. In fact, expansion of endoscopy suites is planned at two hospitals, at least, at this particular time. The availability of time that endoscopists can devote to colonoscopy may be a limiting factor in expanding the number of procedures performed. Another limiting factor may be the number of female endoscopists since many women would prefer being examined by a female endoscopist. As in most other disciplines in Rhode Island, recruiting new physicians remains a serious impediment to the delivery of health care. The Rhode Island Cancer Council is investigating other barriers to patient participation in screening colonoscopy.

Since our data would indicate that the State of Rhode Island has adequate facilities for endoscopy, we wished to determine how soon a procedure could be schedule by an individual seeking referral to an endoscopists. We contacted 68 individual endoscopy offices with the following scenarios:

Scenario A

A 63 year old woman with a family history of colon cancer (her father). She has never had any procedure before. She went to the emergency room because she thought she had the flu. The emergency room physician, after taking care of her acute problem, also recommended to her that she should seek an appointment for colonoscopy.

Scenario B

A 55 year old man who, on routine physical examination, was found to have a positive fecal occult blood test. He had never had a colonoscopy before.

Table 1.

Questions	Yes	No
Increase in colonoscopies?	90.5%	9.5%
Are patients aware of risk status?	79.7%	20.3%
Colonoscopies resulting in abnormalities?	32.9%	67.1%

Scenario C

A 70 year old man in good health with no family history of colon cancer. He was convinced by his children that this was an important test that he should have performed.

Scenario A results indicated that a person calling could have a scheduled colonoscopy within 1 month in 52% of the offices, within 2 months in 67% of the offices, and within 3 months for 97% of the offices.

For *Scenario B*, 41% of the offices could schedule an examination within a month and 52% of the offices would schedule him within 6 weeks; 98% of the offices would schedule him within 3 months.

For *Scenario C*, 78% of the offices could schedule an examination within 1 month and 95% of the offices would schedule an examination within 2 months.

On the basis of these surveys, Rhode Island currently has adequate facilities for performing colonoscopy and individuals seeking this screening procedure would not experience an undue delay. (Table 2)

Table 2.

Time to Colonoscopy

	1 month	2 months	3 months
Scenario A	52%	67%	97%
Scenario B	41%	52%	98%
Scenario C	78%	96%	

References

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Judicial Diagnosis

First-Ever HIPAA Conviction Highlights Differing Views of HIPAA's Civil and Criminal Penalties

John Aloysius Cogan, Jr, MA, JD

On November 5, 2004, Richard Gibson, a former cancer clinic employee, was sentenced to sixteen months in federal prison after pleading guilty to violating the privacy provisions of the Health Insurance Portability and Accountability Act of 1996 (HIPAA). Prior to sentencing, Gibson admitted to disclosing the "protected health information" (PHI) of one of the clinic's patients. Gibson confessed to obtaining a cancer patient's PHI, including the patient's name, date of birth, and social security number, and disclosing the information to obtain credit cards in the patient's name. Gibson then used the credit cards to purchase thousands of dollars worth of various items for his personal use.

This conviction, the first ever under the privacy provisions of HIPAA, raises concerns about the diverging HIPAA enforcement theories held by the two federal agencies charged with enforcing HIPAA's privacy provisions. The Gibson conviction also puts physicians (and anyone else who handles confidential patient information) on notice that the field of possible targets for a government enforcement action under HIPAA is much broader than originally thought. That field now includes persons and entities not initially presumed to be covered by HIPAA.

HHS and DOJ Interpret the Term "Person" in HIPAA Differently

To understand the significance of the Gibson conviction, one must look to the source of HIPAA penalties: the statutory provisions that establish the government's ability to punish, either civilly or criminally, violations of HIPAA's privacy requirements. Both the civil and criminal provisions of HIPAA allow for the imposition of penalties against any "person" who violates HIPAA's privacy provisions.[1] While the use of an identical word ("person") in both provisions to define the object of potential penalties would seem to suggest that civil and criminal penalties could only be imposed against the same class or type of violators, this is not the case. The United States Department of Health and Human Services (HHS), the federal agency charged with enforcement of HIPAA's civil penalty provisions, and the Department of Justice (DOJ), the federal agency charged with enforcement of HIPAA's criminal penalty provisions, interpret the term "person" differently.

When the HIPAA Privacy Rule[2] was first published, HHS made clear that it interpreted the term "person" narrowly. HHS stated that it only had authority to impose civil penalties against "covered entities."[3] According to HHS' interpretation, only "covered entities" (CEs) fall within the definition of the term "person" as it appears in the HIPAA civil penalty statute. As a result, only CEs are subject to civil penalties. CEs include health plans (i.e., insurance companies and plans, Medicare and Medicaid contractors, and government agencies that pay for health care), clearinghouses (entities that convert electronic health care data from one format to another for billing or other purposes), and health care providers who electronically transmit

health information in connection with certain transactions, including claims for payment, benefit eligibility inquiries, referral authorization requests, or other transactions for which HHS has established standards under the HIPAA Transactions Rule.[4] Thus, according to the HHS definition of CEs, only certain health care providers are subject to HIPAA's civil penalties. A physician who does not engage in the HIPAA-defined electronic transactions, either directly or indirectly, is not a CE. For example, a physician who is salaried and whose services are not billed to an insurer or a governmental payer, is not a CE. Because that physician is not a CE, he or she is not a "person" under the HIPAA civil penalty statute and is not subject to any civil penalties under HIPAA.[5]

Now, however, DOJ has taken a different position with respect to the meaning of the term "person." For nearly a year now, DOJ attorneys have privately suggested that HIPAA's criminal penalties are applicable to anyone, not just CEs. Under this broad interpretation, non-CEs, employees, business associates, and anyone else who knowingly uses or discloses PHI in a manner prohibited by HIPAA is subject to criminal penalties. DOJ applied this theory of "person" when it indicted and convicted Gibson. Gibson was not a CE. Instead, he was merely an employee of a health care provider.

Implications of the First Privacy Prosecution Under HIPAA

Because Gibson's conviction resulted from a plea agreement, there was little judicial scrutiny of DOJ's theory that HIPAA's criminal provisions apply to anyone. As a result, the Gibson case only confirms publicly what individual DOJ attorneys have been saying privately: DOJ is willing to apply the HIPAA criminal statute in a manner that differs significantly from the way HHS applies the civil penalty statute. In the long run, it will be up to the courts to sort out whether DOJ's position is correct. For the time being, all we can do is assume that further HIPAA convictions of non-CEs are not out of the question. This new development means that any physician who previously thought he or she was exempt from HIPAA should reconsider his or her position. He or she should consider taking some HIPAA training courses and establishing oversight procedures for any of his or her activities involving the use or disclosure of confidential health information.

Criminal Penalties Under the Rhode Island CHCCIA

For Rhode Island physicians, however, the landscape does not appear to have been radically altered by the Gibson case. The Rhode Island Confidentiality of Health Care Communications and Information Act (the CHCCIA)[6] contains its own criminal penalty section, which, by its express terms, applies to anyone who intentionally and knowingly violates the provisions of the CHCCIA.[7] Although little guidance exists on the application of the CHCCIA criminal provision,[8] it would not be unreasonable to assume that this statute, like its HIPAA counterpart, will be interpreted broadly by prosecutors. Yet, there is one thing we can be sure of. Both the Gibson conviction and the now twenty-month old implementation of the HIPAA Privacy Rule have moved the issue of patient confidentiality into the spotlight. Physicians must take care to ensure that confidentiality of patient health information is a central part of their daily practice.

References

1. Compare 42 U.S.C. § 1320d-5 (civil penalty provision) with 42 U.S.C. § 1320d-6 (criminal penalty provision).
2. It is important to remember that HIPAA and the HIPAA Privacy Rule are two separate things. HIPAA is a statute enacted by Congress in 1996 that was designed, among other things, to establish protections for certain health information. HIPAA contains general requirements and penalty provisions related to health information privacy. The HIPAA Privacy Rule, which became effective for covered health care providers in April of 2003, is a set of regulations promulgated by HHS to interpret and apply the health information privacy provisions contained in HIPAA.
3. See 65 Fed. Reg. 82462, 82579 (Dec. 28, 2000).
4. 45 C.F.R. §§ 160.102, 160.103; 42 U.S.C. § 1320d-1(a)(3). The transaction standards are established by the HIPAA Transactions Rule. 45 C.F.R. Part 162. The use of other electronic technologies by a physician, such as e-mail or a fax machine, does not bring a physician within the definition of a "covered entity." However, physicians who do not directly transmit health information electronically in connection with payment or other covered transactions will be considered "covered entities" if they engage in such electronic transmissions of information indirectly. Indirect transmissions would include the use of a billing service or by having a third party submit electronic claims the physician's behalf.
5. Any civil penalties accruing for violations of the HIPAA privacy standards by this physician would be imposed against his or her employer, if the employer were determined to be a CE.
6. R.I. Gen. Laws § 5-37.3-1, et seq.
7. R.I. Gen. Laws § 5-37.3-9(b).
8. No case law currently exists interpreting or applying the CHCCIA's criminal penalties.

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Letters To the Editor

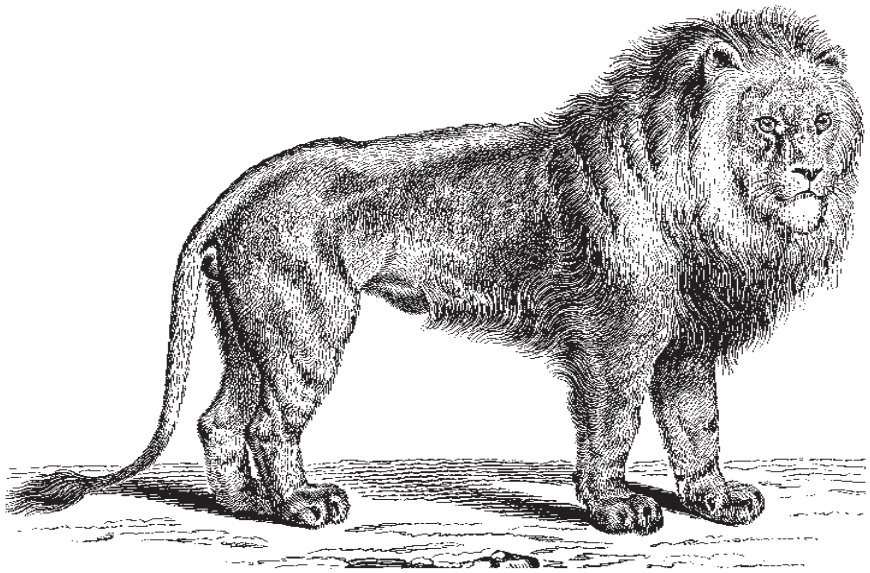
Although I usually agree with your editorials and thoroughly enjoy them as well, I must take issue with one point in the recent one on expert witnesses. You state that " not enough doctors are sued" by way of illustrating that most medical errors never see the light of day. I think you have the cart before the horse. Most medical errors are unreported precisely because the consequences of doing so are too onerous, i.e. sanction, loss of license, lawsuit etc. If we had more of a no-fault system whereby patients received compensation for errors (and here I do not mean frank negligence or malpractice) and less legal ramifications then we all could more safely admit our mistakes. We all make them and most are honest human error. A physician could admit the mistake, learn from it and hopefully establish a system to prevent a similar mishap.

We cannot often reverse patient harm but what is the point in harming the physician if a truly honest error was made and there is no indication of a pattern of malfeasance? I am not defending miscreants here, just the average Joe who tries to do best by his patients but occasionally slips up. We need a quick acting arbitration board to award payments to those hurt and transfer the physician monitoring issues to an arena other than the courts.

– Stephen E. Glinick, MD
Associate Clinical Professor in Dermatology,
Brown Medical School

Compliments on your important essay in Medicine & Health/RI. Perhaps you could do a follow-up article on the problem of Contingency Fee legal actions in the USA. The millions of dollars transferred to lawyers in such cases is without precedent throughout the rest of the world.The USA is the only country which allows Contingency Fee actions,and does not require the losing plaintiff to pay the defendants' fees.It is an outrageous situation, and the public remains mute in the face of it.If doctors were to earn (or be given) such levels of compensation, there would be accusations of greed, duplicity, dishonesty etc etc.; but the lawyers walk off with billions in such fees with impunity. Lawyers claim that without the contingency fee system, the "poor" could not afford legal representation....but what is left for the "poor" after the verdicts?/ Very little in many cases, while the Lawyers walk away with millions. And indeed,why can not the "poor" afford legal representation? Because the structure of hourly rates is beyond the reach of 98% of the population, with charges ranging from $150-700 per hour...devastating!! The needless transfer of wealth to lawyers is a drain on the American economy,and a social evil (in my opinion). You get the message, I am sure. For disclosure reasons,I am a long-time member and financial supporter of Americans For Legal Reform (also known by the acronym HALT, Help Abolish Legal Tyranny).

– Melvin Hershkowitz, MD, and
Leslie Hershkowitz



A Physician's Lexicon

The Passageways of Medicine

Medicine has assembled a bewildering tapestry of words to identify the many preformed passageways within the human body.

The Latin word, *vas*, defines the genital passage, *vas deferens* [the deferens component, also from the Latin, meaning to carry away or to submit]. Vas, meaning a duct or an enclosed pipe, appears in the Latin, *vascularis*, which with time has narrowed its meaning to a duct solely for the passage of blood [ie, vascular]. A variety of English words, including vein, venous and venesection, are all derivative. The word, vase, is similarly an etymological descendant of *vas*, as is the word vessel. Vaseline, a proprietary product, was a word coined by Robert Chesborough in 1877. He had created the *vas-* prefix from the German, *wasser*, meaning water, to form a neologism describing a water-oil chemical.

The word, artery, from the Latin, originally meant a windpipe. In keeping with the rule that Latin is used for anatomic nomenclature and Greek for pathological description, inflammation of veins uses the Greek root, *phlebo-* as in words such as phlebitis. And the lancet used for phlebotomy was the phlebotome which, over the centuries was corrupted, both in spelling and pronunciation, to the English word, fleam.

Duct, from the Latin *ductus*, meaning a passageway, appears in such English words as aqueduct, oviduct and viaduct; and with a con- prefix, in words such as conduction, and conduit. Ductile, meaning malleable, comes from a secondary meaning of *ductus*, namely, that which may be led or commanded. The more flexible, malleable, interpretation of *ductus*, leads to such words as abduct, educate, introduction and even seduction [to lead astray].

The Latin, *ducis*, also meaning to lead, evolved into the Latin *dux*, a military leader and the precursor of the English word, Duke, and the somewhat discredited phrase, *I1 Duce*. Yet another relative of the Latin, *ductus*, is *ducere* which led to the Italian, *doccione*, meaning a jet of water which in turn evolved into the English, douche.

A tube, from the Latin, *tubus*, appears in such medical terms as tubule and intubate. A pipe, from the Latin, *pipare* [meaning to peep] represents one of those words of imitative origin which initially signified a musical instrument before its more generic meaning as a duct. And, of course, *iter*, from the Latin meaning a journey or a roadway, defines some of the interventricular passages within the vertebrate brain. Lastly, there is the word, lumen, meaning the space within a duct or passageway. It is a direct, but puzzling, descendant of the Latin, *lumen*, meaning to lighten as in the word illuminate.

– Stanley M. Aronson, MD, MPH

Rhode Island Department of Health
Patricia A. Nolan, MD, MPH, Director of Health

Vital Statistics

Edited by Roberta A. Chevoya

Rhode Island Monthly Vital Statistics Report

Provisional Occurrence Data from the Division of Vital Records

Underlying Cause of Death	Reporting Period			
	January 2004	12 Months Ending with January 2004		
	Number (a)	Number (a)	Rates (b)	YPLL (c)
Diseases of the Heart	320	3,037	283.9	4,546.0
Malignant Neoplasms	179	2,385	223.0	7,214.0
Cerebrovascular Diseases	54	533	49.8	807.5
Injuries (Accident/Suicide/Homicide)	41	553	51.7	10,403.5
COPD	55	495	46.3	530.0

Vital Events	Reporting Period		
	July 2004	12 Months Ending with July 2004	
	Number	Number	Rates
Live Births	1,418	14,168	13.2*
Deaths	793	10,261	9.6*
Infant Deaths	(6)	(83)	5.9#
Neonatal deaths	(5)	(66)	4.7#
Marriages	988	8,342	7.8*
Divorces	210	3,218	3.0*
Induced Terminations	459	5,481	386.9#
Spontaneous Fetal Deaths	76	1,305	92.1#
Under 20 weeks gestation	(70)	(1,243)	87.7#
20+ weeks gestation	(6)	(62)	4.4#

(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 1,069,725

(c) Years of Potential Life Lost (YPLL)

Note: Totals represent vital events which 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, January 1920

An editorial railed against fashion: "It is, we believe, the duty of the physician to enter a protest against some of the fads and fashions which are a menace to health. This thought is emphasized by a visit to the recent football game on Thanksgiving Day. In spite of the sunshine, the air was cold, raw and penetrating and common sense would prompt one to be all protected against the November chill. In the seat near us we saw a girl of 18 or 20 with her neck exposed to about her 6th rib and with no protection by any wrap, with low shoes, silk stockings and no rubbers. She looked pretty, but she shivered every breath she drew during the entire game…if she did not catch cold and become ill, there is a Providence which overlooks a direct bid for sickness."

Another editorial railed against cosmetics: "Cleanliness is next to godliness, and if one of these 'buds' should be accused of being dirty, she would immediately take offense. The lady who sat close by had so much powder on her face that the pores of her skin were absolutely incapacitated from activity. The fashion of covering up Nature's handiwork with preparations of talcum is not only silly, but is absolutely injurious."

Arthur A. Harrington, MD, contributed "The Present Status of State Care of the Insane in Rhode Island." He gave staff specifics: one superintendent (him), 6 resident assistant physicians, a non-resident sero-pathologist, 4 visiting surgeons (each for 3 months a year), a visiting ophthalmologist, a visiting dentist. The hospital served as a training school for "women nurses." Thanks to a state bond issue, the state would be spending $600,000 to improve hospital care for the 400 patients. Dr. Harrington saw the key problem: "no aftercare."

Harvey S. Bernstein, MD, state pathologist, in "On the Scope and Problem of the Laboratory," discussed the recently-created office in the Department of Health. He urged communication between physicians and the laboratory.

Fifty Years Ago, January 1955

Malcolm A. Winkler, MD, in "Plastic Planing of Post-Acne Scars," praised the Kurtin procedure, after his 16 years of experience with it.

Victor L. Pedorella, Associate Professor of Taxation, Bryant College, and manager of his own accounting and tax offices, described the new tax code in "The Doctor and His Income Tax."

Stanley S. Freedman, MD, in "History Taking in Allergic Diseases (with Special Emphasis on Childhood Asthma)," advised physicians to include family history, past history, onset, frequency and duration of attacks, the season, sensitivities, hobbies, symptoms, home, school, and emotional factors.

Pharmaceutical advertisements supported the journal; e.g., Thesodate, Megandren Linguets and Femandrea, Pyridium, but this issue had a half-page advertisement for Johnnie Walker ("born 1820…still going strong.") [Today *Medicine & Health/Rhode Island* does not accept advertisements for alcohol or tobacco.]

Twenty-Five Years Ago, January 1980

David C. Lewis, MD, in "Diagnosis and Management of the Alcoholic Patient," maintained: "The physician has opportunities for early intervention which improve the prognosis of a major health problem."

Donald C. Williams, from the Rhode Island Department of Health, discussed "Components of Change in Rhode Island's Physician Supply, 1974-99." Using the Physician Re-licensure Survey Data, he tracked the major shift, from primary care to specialties.

John S. Dziob, MD, FACS, contributed "Trauma and Breast Cancer, or The Anatomy of an Insurance Claim." The patient was a passenger in a taxi when it crashed, throwing her forward. She claimed that the accident caused her breast tumor. The investigator concluded: "This pre-existing tumor was providentially revealed, exposed and spotlighted by the trauma, not caused or aggravated by it."

Stanley M. Aronson, MD, in "Dean's Message," reported on "Alcoholism and the Medical Curriculum." He noted David Lewis's appointment to the faculty as Associate Professor of Medicine and Donald G. Millar Distinguished Scholar in Alcoholism Studies, as well as the formation of a faculty committee on Alcoholism and Drug Abuse. "The teaching on alcoholism currently permeates the four years of the medical curriculum."

An Editorial explained that from January 1970-1975, graphic consultant George Patton, Jr, was in charge of choosing Journal covers. Cost constraints ended that relationship. In 1980, thanks to a grant, Mary Frye, an Instructor in the Department of Graphic Design at RISD, would be choosing covers. This January cover featured an etching of a tavern scene, by William Hogarth, from "The Rake's Progress," June 1735, to show the debilitating effects of alcohol, a topic linked to David Lewis's article and the Dean's message.

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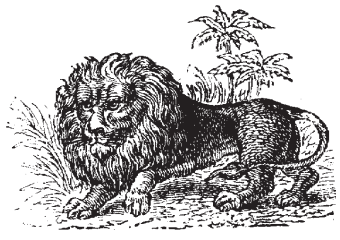

What's in a Name???

GOOD - authentic, honest, just, kind, pleasant, skillful, valid

NEIGHBOR - friend, near

ALLIANCE - affiliation, association, marriage, relationship

CORPORATION - company, business establishment

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