Cover: *"Scratches Among the Flowers,"*
Japanese pictorial quilt, by Joan Smith
McLellan, a Bristol artist. This quilt is
part of an exhibit at the Wickford Art
Association. Ms. McLellan specializes in
the ancient Japanese arts - gyotaku (fish
rubbings), takuga (plant rubbings) and
appliqued quilts.

Medicine & Health
RHODE ISLAND

❧ COMMENTARIES ❧

Really Dead

I was recently invited to participate in a bioethics conference at my hospital with medical students, housestaff and attendings. I was invited because I am a neurologist, not an ethicist. The starting point was a case in which a teenage boy dying from an inoperable brainstem tumor wanted his organs to be used for transplantation. He also expressed fear that his organs might be taken before he was truly dead. The situation was complicated by his desire to die at home, his insurer's desire that he not die in a hospital because of the expense, and the transplant team's need to harvest the organs within a very short time after his heart had ceased to beat (should he die a cardiopulmonary rather than a brain death). Real life, not following the Hollywood script, had the boy dying at home and he was not brought to the hospital in time to allow for use of any body parts other than his corneas. The bioethics discussion centered on the issue of death and the modern concept of "brain death." Also considered was the distinction that most people have between cardiopulmonary death and brain death. The latter is too often an abstract concept which interferes with transplant attempts.

"Brain death" was a consequence following the development and clinical use of mechanical ventilators. With ventilators, people who were unable to breathe could now be artificially ventilated and thus kept alive and, in many cases, recover sufficiently to breathe without mechanical assistance. It rapidly became clear that some patients would never recover the ability to breathe but were otherwise reasonably intact, that some survived in a persistent vegetative state and that others never regained brain function of any type. Since this last group almost invariably suffered a cardiorespiratory death within a short period of time, and since health care economics was not then in a crisis mode, the need for a definition of death was not needed. What forced the need to conceptualize or define death was the success of mechanical ventilation. State attorneys-general demanded a clear and unequivocal definition of death so that limited resources could be better employed and that no legal challenges could be mounted when "the plug was pulled."

Over time the main issue became transplantation. Survival for transplants depends critically on the amount of time the organ is inadequately perfused, i.e. how long the heart stopped before the organ was removed. After a certain critical period of under-perfusion, the organs become useless. So a rigid set of criteria was developed to define death as a brain event rather than as a cardiopulmonary one. This introduced the notion that there were two distinguishable forms of death. In the public's and perhaps physician's mind, was the notion that brain death was a somehow less stringent definition than the cessation of heartbeat. In fact, prior to the development of brain criteria to define death there had been no definition of death. Medical science's definition of death was in a state closely analogous to the "definition" of pornography: "I know it when I see it." And, unfortunately that analogy was applicable to the gray areas as well. Can a transplant team remove a kidney one-minute, two minutes, five minutes, etc after the heart stops? Is the last heart beat a palpable pulse, an auscultated sound or an electrical event recorded in an EKG? Does the timing of the last breath count? Is pCO_2 important? Given the litigious nature of our society and the strongly held ethical beliefs of some people who believe that modern medicine too often crosses ethical lines, the need for a legal definition became crucial.

The history of death then was, as best I can tell, legally uncharted territory until the first state adopted a definition of death. All states in the US now have criteria for the definition of death, which are brain criteria. Most pronouncements of death are based on failure of detectable heartbeat and brain criteria are used uncommonly, primarily for transplant purposes but also to remove needless care. The practice of medicine does not include ministering to the dead.

It is my contention that the phrase "brain dead" should be removed from the medical lexicon, because it introduces a sense of doubt, that "brain dead" is different than "dead", that one might be "brain dead" today and "really dead" tomorrow. One might imagine individuals or groups defining death in idiosyncratic ways, having "liver death" or "pancreas death" or some other seemingly far-fetched notion.

Many years ago a case report was published in which a young adult was "brain dead" in a state that had not yet adopted legal death criteria. The patient had a heartbeat for the next two or three months before it too stopped. At autopsy, when the cranial vault was opened, the liquefied brain content poured out.

Two terms should be used to describe the state of death. Dead or "legally dead." Continued use of "brain death" clouds the public's consciousness and makes the public believe the pronouncement of death is a subjective decision fraught with the possibility of irretrievable error. While a family has no qualms about discontinuing life support on a dead relative, they may on a "brain dead" relative. The law, however, defines death and patients meeting these criteria are no longer patients.

– **Joseph H. Friedman, MD**

Rhode Island's First Hospitals

Seaports were colonial America's first great centers of commerce and industry. But because of their maritime traffic, they were also America's sites of entry for the devastating contagions of the 17th and 18th Centuries. Each new epidemic of smallpox in Boston, for example, begain with a sailing vessel disembarking someone in the acute, communicable phase of smallpox. And thus Boston experienced sustained epidemics of smallpox in 1677, 1689, 1702, 1721, 1751, and 1775.

Newport, in the early years of the 18th Century, was Rhode Island's leading port as well as its commercial center. Smallpox first entered the community in 1716 via an arriving merchant vessel. In addition to the customary quarantine measures for those stricken with smallpox, Newport constructed a small infirmary on an offshore island. This modest undertaking represented Rhode Island's first attempt at providing its very sick with both isolation and rudimentary protection from the elements; this primitive house of contagion was Rhode Island's first hospital.

In 1752 Providence established its own smallpox hospital. And in the next five decades the city at the head of Narragansett Bay built two more so-called fever hospitals consisting of little more than dormitories and attached kitchen.

Yet another epidemic scourge invaded Providence in 1798, a puzzling disorder called yellow fever. Under the mistaken presumption that the disease was directly communicable, the city hastily constructed a two-story house on the western shore of the mouth of the Providence River to isolate victims of the disease. The yellow fever epidemic abated rapidly and the city, left with an empty fever house, designated it as a marine hospital solely for the care and housing of disabled shipboard personnel.

New England's first general hospital, the Massachusetts General Hospital, had been constructed in 1811. New Haven built its own hospital in 1832; and Hartford Hospital was chartered by Connecticut's legislature and opened in 1854. In contrast to the episodic usage of fever houses, these new institutions in Boston, New Haven and Hartford were expressly designed to meet the continuing inpatient needs of the local civilian communities.

In the years immediately preceding the Civil War, Rhode Island relied almost exclusively on the home for the care of its very sick. There also was an institution, built in 1828, called the Dexter Asylum for Paupers. This was an ill-conceived institution which, in the words of one local physician, was an overly crowded dwelling for the city's paupers, the victims of debauchery, the uncontrollably insane, homeless women in labor, and the many malnourished immigrants recently arrived from Europe. There was, in addition, the excellent Butler Hospital, built in 1847, but it confined its admissions to the mentally ill.

Since hospitals are sometimes constructed as adjuncts to medical schools, Rhode Island had an opportunity to establish a general hospital of its own when Brown inaugurated New England's third medical school [Harvard, 1782; Dartmouth, 1798]. It was a modest effort with a faculty of three and a small campus building housing an anatomy amphitheater, a pathology museum, a small library and a few classrooms. The faculty maintained private practices and some of their patients were sometimes used for didactic purposes. But until medical students had access to a hospital ward, their edu-cation would remain a bloodless sequence of blackboard exercises. The Brown medical school accepted its first students in 1811, trained almost one hundred physicians in the next 16 years, but then closed its doors in 1827 because of a dispute between faculty and administration. And thus a possible stimulus for the establishment of a general hosptial in Rhode Island was lost.

The practicing physicians of Rhode Island had repeatedly appealed both to the State Legislature and the philanthropic community for funds to construct and maintain a hospital within the state, but to no avail. During the early decades of the 19th Century Providence citizens identifid the grim Dexter Asylum as its sole inpatient facility, but more in shame than pride.

A Brown University graduate, Thomas Poynton Ives [Class of 1854], was the initiating force which finally accomplished the task of building a fine general hospital for Providence. Ives had been trained as a physician at the College of Physicians and Surgeons in New York and was then apprenticed to Dr. J. Ely, a prominent Providence practitioner.

The economic disaster of 1857, with the closing of many of the local textile factories, and the Civil War of 1861 effectively aborted any efforts to build a local hospital. Prodded by the Ives family, the Rhode Island legislature finally incorporated the Rhode Island Hospital in 1863 and donated the 12 acres of the old marine hospital for its site. The Ives family provided $75,000 for construction of the hospital.

A total of $305,000 was eventually subscribed and construction was begun in December of 1864. This effort represented the largest single charitable drive in the state's history. The architects envisioned a handsome dark brick building, some three stories high in the Italian gothic style with two distinctive and imposing steepled towers. The building consisted of a central unit housing the administration, chapel, auditorium, library, kitchens and central apothecary; and two wings extending in a north-south direction. The wards were spacious 24-bed units with adequate ventilation and sunlight. The Board authorized the opening of about 70 beds to serve the immediate medical needs of the Providence population, then about 70,000. The original hospital had an eventual capacity of about 120 beds.

On the first day of October, 1868, the Rhode Island Hospital opened its doors. The president of the hospital's Board, Robert Ives, said these words in the invocation: "Except the Lord build the house, they labor in vain that build it." And on October 6, 1868, John Sutherland, a local shoemaker, was the first patient to be admitted. He suffered from a deep abscess of his jaw-bone. Surgery was successfully undertaken and within two months he walked out of the hospital with his disease healed.

Rhode Island Hospital has kept its door open, without interruption now, for 134 years. In times of peace it has provided the best of medical care for the Rhode Island community; and in times of war it has recruited its physicians and nurses to form army hospital units which have served with distinction in France, India and Burma. Today it is the state's leading hospital and the premier clinical teaching facility for Brown's new medical school.

– **Stanley M. Aronson, MD, MPH**

Introduction

Barry Stein, MD

The fear of women is the basis of good (men's) health

- Old Spanish Proverb

It is indeed a pleasure to edit this month's issue of *Medicine & Health/Rhode Island*, devoted to men's health. The concept of "women's health" is well ingrained in our consciousness, but men's health concerns are just beginning to surface. It has been my impression, re-enforced by 25 years as a urologist, that often if not for the women in their lives, men might never come forward for health care. Women tend to be more vocal than men about their health care, and lobby more successfully for research dollars. For example, prostate and breast cancer have a similar incidence, yet the **National Cancer Institute (NCI)** devoted $1.145 billion for breast cancer research from 1998-2000, but only $353 million for prostate cancer. In 1997 the NCI spent $1,787 in research on prostate cancer for each prostate cancer death, while the same year it spent $18,800 on breast cancer research per death due to breast cancer. One article in this issue covers osteoporosis in men. With the exception of drug-induced osteoporosis, Medicare does not pay for Dexa scanning in men, while it covers tests for any woman. While osteoporosis occurs more frequently in women, it does affect ~12% of men over the age of 50; and men have a higher mortality rate associated with an osteoporotic hip fracture than women, as high as one out of three in some series.

This issue features four articles on men's health. The first from Drs. Fulton and Marable of the Rhode Island Department of Health gives us a sense of the scope of men's health issues in an aging population. As we baby boomers reach Medicare age the number of older men will increase precipitously. In fact, the largest growing age segment is those over 85. According to Drs. Fulton and Marable, the number of men in Rhode Island over the age of 50 is expected to increase 62% between 1995 and 2025, while the number of men over age 85 is expected to increase 105% over the same time frame. Rhode Island is currently in a virtual tie with Pennsylvania as the second "grayest" state, with 15.6% of our population over age 65. This means that we must prepare for the unique problems of the aging male, including the topics in this Journal, but also including the new concepts such as andropause, and male depression.

The second article discusses the current status in the treatment of the most common disease of the aging male, benign prostatic hyperplasia. New developments in both the medical and surgical treatment of this disease are providing greater benefits with decreasing risks. Medical therapy, particularly with alpha blocker therapy often means that older men failing this treatment present with larger prostate glands. Newer office-based therapies, such as Trans-Uretheral Needle Ablation are springing up to treat the failures of medical treatment.

The third article, written by Dr. Kim, covers the current status of male urinary incontinence, and its treatment options. Unfortunately, the majority of cases in men are iatrogenic, and follow as a complication of radical prostatectomy. Newer techniques in radical prostatectomy are hopefully leading to a decrease in this debilitating complication. The management of the non-iatrogenic urinary incontinence in men, related to the aging process, is also well covered.

The last contribution covers a newly emerging problem on the men's health docket - osteoporosis. A large number of these cases are also iatrogenic, and relate to androgen deprivation therapy associated with the treatment of advanced prostate cancer. Preventative therapy can reverse osteopenia, and prevent osteoporotic fractures.

I hope that dialogues such as this issue of *Medicine & Health/Rhode Island* will promote better recognition of men's health concerns.

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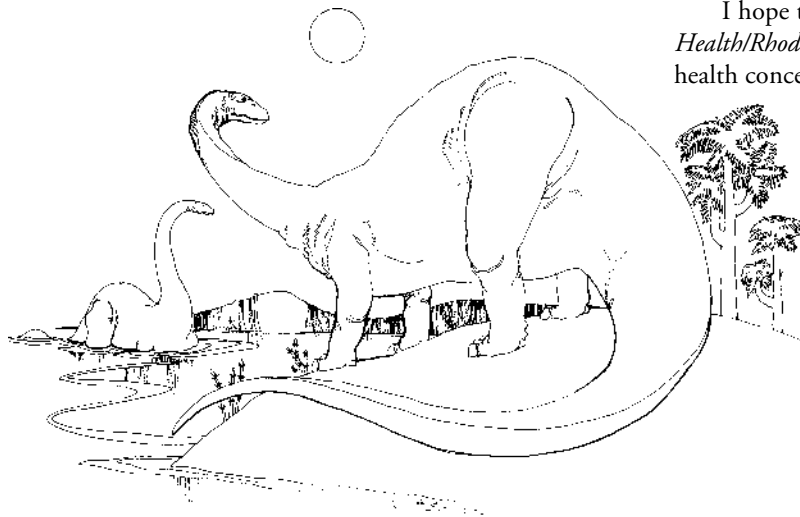
Older Men in Rhode Island, 1995-2025: Population, Life Expectancy, and Men's Health Issues

John P. Fulton, PhD, and Sharon Marable, MD, MPH

To evaluate the need for older men's health care resources over the next 25 years, the **Rhode Island Department of Health (HEALTH)** assembled and constructed projections of population, life expectancy, disease prevalence, and disease incidence through 2025. The evaluation focused on: men ages 50 and over, life expectancy at 65 years of age, the prevalence of benign prostatic hyperplasia, osteopenia/osteoporosis, urinary incontinence, and the incidence of prostate cancer.

METHODS

Sources of Data

Projections of Population

The latest projections of the male population of Rhode Island by age and race or ethnicity (Hispanic status) were obtained from the United States Bureau of the Census for the period 1995-2025.[1] Census projections are constructed in sets of low, preferred, and high, based on differing assumptions about the components of population growth (fertility, mortality, and migration). Preferred projections, which represent the Census Bureau's best estimates of the size and composition of future population, were selected for use in this analysis.

Projections of Mortality

Projections of life expectancy at age 65 for males in the United States, by age and race or ethnicity (Hispanic status), were obtained from the Census for the period 1995-2025.[2] The projections of mortality selected for use in the present analysis were used by the Census to construct preferred projections of the United States population, 1995-2005.

Estimates of Disease Prevalence

Estimates of the current prevalence of **benign prostatic hyperplasia (BPH)**, osteopenia, osteoporosis, and incontinence were derived from the medical literature:

- A variety of clinical studies have reported estimates of the prevalence of BPH at microscopic, macroscopic, and clinical levels. Estimates of BPH, by age, were derived by averaging data from available studies.[3-9] (Table 1)

- Estimates of the prevalence of osteopenia and osteoporosis were obtained from a national study of 3090 U.S. men ages 50 and over.[10] All the men had received bone mineral density tests of the hip. Estimates varied according to that part of the hip studied. Using the trochanter as a reference point, 28% of men ages 50 and over are osteopenic, and 3% are osteoporotic. Using the femur neck as a reference point, 47% are osteopenic and 6% are osteoporotic. The latter estimates were used in this analysis.

- Low and high estimates of the prevalence of urinary incontinence in U.S. men ages 65 and over were derived from the National Institutes of Health Consensus Development Conference Statement, *Urinary Incontinence in Adults* (October 3-5, 1988).[11] The following statement was used to derive estimates:

"Estimates of the occurrence of urinary incontinence depend on the nature of the study population and definition of disorder. Prevalence rates range from 8 to 51%; an estimate of 15 to 30% for community-dwelling older persons seems reasonable, and of these, 20 to 25% may be classified as severe. Prevalence rates are twice as high in women as in men..."[11]

Table 1. Prevalence of benign prostatic hyperplasia per 10 000, as reported in the literature.

Sources	Age Group				
	40-49	50-59	60-69	70-79	80-
Microscopic (Average)	**2233**	**4167**	**6767**	**8033**	**8600**
Lytton 1 (a)	2300	4200	7100	8200	8800
Isaacs and Coffey (b)	2200	4200	6200	7900	9000
Barry (c)	2200	4100	7000	8000	8000
Macroscopic (Average)	**800**	**2230**	**2930**	**4065**	**4605**
Isaacs and Coffey (b)	800	2100	3500	4400	5300
Lytton (d)		2360	3730	3910	
Clinical Evidence (Average)	**1380**	**1712**	**2859**	**3306**	**3010**
Isaacs and Coffey (b)		1800	830	1900	2500
Lytton (d)		2000	3500	4290	
Watanabe (e)		1340	1755	2670	5420
Lytton (f)		1050	3910	3670	1110
Garraway et al. (g)	1380	2370	4300	4000	

Sources:

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f. Lytton B, Emery JM, Harvard BM. The incidence of benign prostatic obstruction. J Urol 1968; 99: 639-45.
g. Garraway WM, Collins GN, Lee RJ. High prevalence of benign prostatic hypertrophy in the community. Lancet 1991; 338 (24 August): 469-71.

Using the following information and assumptions, the prevalence of urinary incontinence among U.S. males ages 65 and over was estimated to range from 9 to 18%:

- The prevalence of urinary incontinence among all community-dwelling persons ages 65 and over in the United States (persons of both sexes and all races) ranges from 15 to 30%.[11]

- The prevalence of urinary incontinence among all community-dwelling persons ages 65 and over in the United States (persons of both sexes and all races) is twice as high in women as in men.[11]

- Among persons of all races ages 65 and over who resided in Rhode Island in 2000, men represented 39% of the population.[1]

Measures of Disease Incidence

The incidence of prostate cancer among Rhode Island males ages 50 and over, by age and race, was calculated from newly diagnosed cases of prostate cancer reported to the Rhode Island

The life expectancy at age 65 of U.S. males is expected to increase 2.5 years between 1995 and 2025, from 15.5 to 18.0.

Cancer Registry (Rhode Island's state-wide central cancer registry) for calender years 1994-1998, inclusive. Incidence as opposed to prevalence was chosen as the best measure of prostate cancer, because the basis of available prevalence estimates for prostate cancer in Rhode Island does not take into account the rapid increase in prostate cancer incidence which occurred in the 1990s.[12]

Methods of Analysis

Estimates of the Rhode Island male population ages 50 and over by age and year were combined with incidence and prevalence rates (assumed to be constant over time) to construct estimated counts of newly diagnosed cases of disease (incidence) or existing cases of disease (prevalence) among Rhode Island men

every 5 years from 1995 to 2025, inclusive. Race-specific statistics were computed for newly diagnosed cases of prostate cancer. Percent change from 1995 to 2025 was computed for the number of resident Rhode Island men ages 50 and over, years of expected life at age 65, and counts of newly diagnosed or existing cases of disease.

RESULTS
Population

Between 1995 and 2025, the number of Rhode Island men ages 50 and over is expected to increase 62%. (Table 2) The number of men ages 85 and over is expected to increase even more (105%). The numbers of older African American, Native American, and Asian men are expected to grow faster than the number of older white men. Similarly, the number of older Hispanic men of any race is expected to grow faster than the number of older non-Hispanic men. These differentials, if correct, will alter the racial and ethnic mix of older Rhode Island men in the next quarter century. For example, African American, Native American, and Asian men, who represented 4.3% of Rhode Island men ages 50 and over in 2000, are expected to represent 7.8% of Rhode Island men ages 50 and over in 2025.

Life Expectancy

The life expectancy at age 65 of U.S. males is expected to increase 2.5 years between 1995 and 2025, from 15.5 to 18.0. (Table 3) This represents a 16% increase for all men ages 65 and over. Men of all races and men who are Hispanic will all benefit from an increase in life expectancy of from 9 to 20%. In 1995 at age 65, Asian men had the longest life expectancy (18.8 years), followed closely by Hispanic men of any race (18.5 years), then Native American men (17.9 years), white men (15.7 years), and Black men (13.6 years). In 2025 at age 65, about the same ordering is expected, with the exception that Hispanic men (22.2 years) are projected to take the lead over Asian men (20.4 years).

Prevalence of BPH, Osteopenia/Osteoporosis, and Urinary Incontinence

Because prevalence rates were assumed to be constant over time, the estimated proportional growth in the

number of men experiencing BPH, osteopenia, osteoporosis, and urinary incontinence between 1995 and 2025 parallels the estimated proportional growth in men ages 50 and over (62 - 63% for BPH, osteopenia, and osteoporosis) or the estimated proportional growth in men ages 65 and over (58% for urinary incontinence; Table 4). By 2025, over 50,000 men are projected to have clinical symptoms of BPH, between 11,000 and 12,000 men are projected to have osteoporosis, and between 8,000 and 17,000 men are projected to be incontinent of urine.

Incidence of Prostate Cancer

According to projections, over 1,200 men will be diagnosed with prostate cancer in 2025. (Table 5) Compared to 1995, the number of prostate cancer cases diagnosed among African American men in Rhode Island in the year 2025 will have tripled (increased 189%), in parallel with the number of African American men ages 50 and over.

DISCUSSION
Caveats

These projections are based on many assumptions. Undoubtedly, some will prove to be better than others, while a few may prove to be totally incorrect. Such is the risk of projections.

Assumptions used by the Census Bureau to construct its estimates of the Rhode Island population by age, sex, and race or ethnicity are based on educated guesses about trends in mortality, fertility, and migration. Although trends in mortality are relatively stable in a modernized country like the U.S., trends in fertility and migration have a number of complex determinants, not the least of which are trends in the economies of the state, nearby New England, the nation, and the world, especially those parts of the world from which Rhode Island draws its immigrants. Rhode Island, because of its small area, small economy, and small population, may gain or lose a significant proportion of its people in response to unique economic events. For example, it has been estimated that the

reassignment of warships from Newport Naval Base to other bases on the Atlantic coast in the early 1970s led to the loss of over 3% of the state's population in 1-2 years. Because the economy of the state is difficult to predict, especially over the course of 25 years, so is its population growth, and all that flows from it, such as the number, type, and severity of illnesses and conditions with which the health care system will have to cope.

The projections of prevalent and incident cases constructed here are based on the simplistic assumption that disease-specific incidence and mortality rates will not change between now and 2025. (Incidence rates affect both incident and prevalent cases. Mortality rates affect prevalent cases.) Incidence rates may be lowered by effective prevention methods. For example, in the next quarter century:

- Will we develop therapies so effective in preventing or reversing BPH in its early stages that symptoms of the condition will arise in a lower proportion of men, or will we extend the length of life far enough beyond present projections that we substantially increase the number of men with clinical BPH?

- Will we screen and treat osteopenia so effectively that we substantially reduce the incidence and prevalence of osteoporosis [See Note], or will we extend the length of life far enough beyond present projections that we increase the number of men with osteoporosis despite downward trends in incidence? Or yet, will the tobacco industry succeed in reversing the downward trend in tobacco use among adult men, thus increasing the prevalence of a potent risk factor for bone loss, and will this trend outstrip our health care system's ability to counter the problem with intensified screening and treatment?

- Will we reduce the natural and iatrogenic causes of urinary incontinence in older men, or will we increase the iatrogenic causes of urinary incontinence with new forms of therapy for other diseases and conditions?[16]

- Will we find a preventive therapy for prostate cancer, or will we extend the length of life far enough beyond present projections that we substantially increase the number of men with diagnosed prostate cancer? Or yet, will we reduce the mortality from prostate cancer sufficiently that the prevalence of disease and the iatrogenic effects of its treatments (such as urinary incontinence) increase?

SUMMARY OF FINDINGS

Assuming stable rates of disease incidence (therefore prevalence) over the next 25 years, and assuming (as does the Census) that the number of older men in the state will grow, Rhode Island will experience a steady increase in the number of prevalent cases of BPH, osteopenia, osteoporosis, urinary incontinence, and prostate cancer. (Even though we have been unable to project prostate cancer prevalence with confidence, the number of prevalent prostate cancer cases will certainly increase over time.) Proportionately, more older men will be African American, Native American, or Asian in 2025 than in 2000, and more will be Hispanic.

COMMENTS

Assuming at least moderate stability in economic processes, demographic trends, and disease incidence rates, prevalent cases may increase by as much as 60% among older Rhode Island men (men ages 50 and over) by the year 2025. The growing population of older men will increase the demand for health care services that specifically address men's health, as well as the many health problems men share with women.

As we plan to meet the increasing demand for men's health services, we must be cognizant of the increasing cultural and linguistic diversity of older men in our state. Older men's varied responses to men's health issues, related as these issues are to personal identity, masculinity, and sexuality, are rooted in culture. As we can deduce from the racial and ethnic backgrounds of older men in Rhode Island, this culture is not monolithic. Rather, it is diverse, and, moreover, is becoming more diverse. Are we prepared for this diversity? Is the health care system ready to serve all men with cultural competence, gender sensitivity, and humility, or will the system soldier on, largely oblivious of culture, leaving many men alienated and thus unserved? The latter problem will grow, unless we build diversity into our thinking, planning, and ultimately, into our service.

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NOTE: We have great potential for osteoporosis prevention right now, were today's screening and treatment techniques to be applied systematically, and new approaches to prevention, such as hormone replacement therapy for men, to bear fruit.[13,14,15]

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The Evaluation and Treatment of BPH, 2002

Barry Stein, MD

Benign prostatic hyperplasia (BPH) is the most common medical problem in men over age 50. At age 40, only 8% of men have histologic characteristics of BPH; however, by age 50, 50% of men will exhibit BPH in their prostate and, by age 80, 90% or more of men will have histologic evidence of BPH.[1] The prostate grows approximately 4% per year, but this increase may occur even more rapidly between the ages of 30 and 50 than between 50 and 70. Approximately 25 - 50% of men will eventually exhibit symptoms of benign prostatic hyperplasia including, but not limited to, decreasing stream, frequency, urgency and nocturia.

The **International Prostate Symptom Score (IPSS)** sheet was validated as a tool useful for following the symptoms of men with BPH. (Figure 1) Although this score sheet does not in any way diagnose BPH as being the etiology of the symptoms, it does provide a means by which you can follow men with BPH from year to year to see if their symptoms are stable or deteriorating. Since a total of seven different symptoms are assessed, and are rated from zero (not at all) to five (almost always), the total symptom score can vary from zero to thirty-five.

The United States Department of Health and Human Services, through the Agency for Healthcare Policy and Research published clinical practice guidelines for BPH in 1994.[2] These are currently in the process of revision. Figure 2 is the algorithm summarizing these guidelines. In practice, one can use these guidelines to try and categorize the patient into one of three general categories. (Figure 3) The first category includes patients with mild symptoms, as determined by reviewing the IPSS, a normal digital rectal exam and, if appropriate, normal PSA level. These patients merely need to be educated that BPH is a part of the normal aging process in men and they should return in one year for a reevaluation. Group 2 includes patients with moderate symptoms on the IPSS sheet, and a normal digital rectal exam and normal PSA level. These patients may be appropriate for further evaluation such as

uroflow testing, measurement of post-void residual or evaluation of upper tracts by ultrasound. Following this, it is appropriate to discuss with these patients four options (watchful waiting, medical therapy, minimally invasive surgical therapy, and surgery). Category 3 includes men with severe symptoms on the IPSS sheet or a complication of BPH, which include: urinary retention, recalcitrant hematuria, recalcitrant urinary tract infections, bladder stones, bladder diverticuli and upper tract obstruction. These patients are usually best treated by a surgical procedure.

Once the decision is made to treat the patient, the options fall into one of the following categories: phytotherapy, medical therapy, including the use of either finasteride and/or beta-blocker therapy, minimally invasive surgical therapy, such as **transurethral needle ablation** (TUNA), or full surgical options.

PHYTOTHERAPY

My first line of medical therapy for patients with mild to moderate symptoms of BPH is herbal therapy, specifically saw palmetto. The phytotherapeutic compounds are characterized as food additives by the FDA, which permits a lack of standardization of the products. The most popular herbal product today is saw palmetto (*Serenoa repens*) which is derived from the berry of the American dwarf palm tree. Phytotherapeutic agents are plant extracts and not the actual plants themselves, a distinction which patients

often miss. The process for extraction of the active ingredient varies greatly from brand to brand and may lead to great differences in potency. In the United States, this big business is worth between $1.5 and $2 billion dollars yearly in the treatment of BPH alone. An interesting article that spoke to the difficulties with phytotherapeutics can be found in "Herbal Therapy for Prostate Probems," Consumer Reports Magazine,[3] an article focused on saw palmetto. Although saw palmetto was considered a medicine for BPH in the early part of the twentieth century and was included in the **United States Pharmacopoeia** (USP) and the National Formulary until the 1950s, it was dropped from both in the 1950s. It was reinstated in 2000 as an acceptable treatment for BPH. The USP monograph concluded that the trials "provide evidence of moderate scientific quality. The commercial extracts of saw palmetto...are more effective than a placebo in relieving lower urinary tract symptoms from BPH."[3] The adverse effects are mild and rare and may include mild stomach upset or diarrhea. Although numerous reports in the literature suggest that saw palmetto is better than placebo, most of these are not long-term, prospective, randomized trials; so the exact effectiveness of saw palmetto remains in question. In addition, the mechanism of action remains in question, every theory having its proponents and detractors. Two large meta-analyses in the literature which
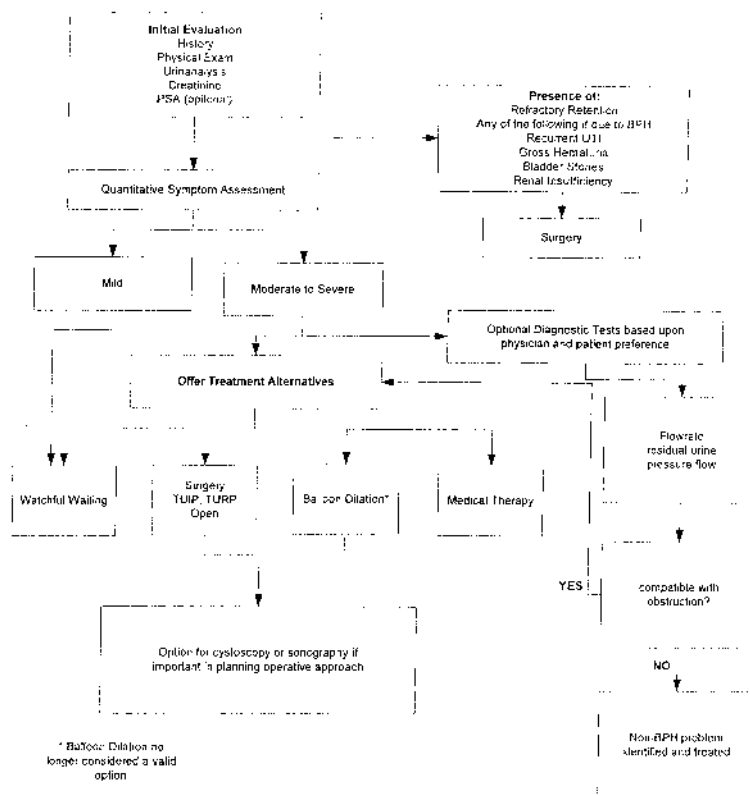

Figure 1

Figure 2.

together total over 5,000 men suggest, but do not prove, that saw palmetto is better than placebo.[4-5] Several studies, however, have shown that, although saw palmetto may improve the symptom score, it does not greatly change the urinary flow rate and has no effect on PSA, suggesting there is no 5-alpha reductase activity in saw palmetto.

MEDICAL THERAPY

Two categories of medical therapy are utilized for BPH. The first includes finasteride (Proscar) and the other includes alpha-blockers (terazosin, doxazosin and tamsulosin). When finasteride first became available, it was thought that this drug would be the panacea for BPH due to its ability to reduce the size of the prostate by up to 25-50%. Unfortunately, in the early studies, the effectiveness on reducing symptom scores or improving uroflow rates was modest. It has recently come to light, however, that one problem with the early studies was that many of the patients had small prostate glands and that finasteride should ideally be used in patients with prostate glands over 40 grams in size. The **Proscar long-term efficacy and safety study (PLESS)** was a 95-center study of over 3,000 patients randomized to either finasteride 5 mg or placebo and followed for over four years. In this study, the patients who were on placebo had a 14% growth in prostate volume over the four years time, whereas the patients on Proscar had an 18% reduction in prostate volume.[6] It has been shown that PSA is a stand-in for prostate size and men with PSA of over 1.6ng/ml at age 50, 2.0ng/ml at age 60, and 2.3ng/ml at age 70 are likely to have a prostate exceeding 40 grams.[7] In fact, it was shown that the larger the prostate volume, the greater the improvement from finasteride in terms of symptom score reduction and flow improvement. In a study published in the *New England Journal of Medicine*, finasteride was shown to reduce the risk of acute urinary retention or need for surgical treatment for BPH in men with large prostates.[8] This was especially true in men with prostates over 60 grams in size, which correlated to a PSA of at least 3.3 mg/ml or greater. In this group, there was at least a 50% reduction in the development of acute urinary retention or need for surgery. For this reason, in men with prostates of 60 grams or more, and especially over 100 grams, I discuss finasteride as an option for chemo prevention. The other area of use for finasteride in men with BPH is

recurrent hematuria.[9] A significant number of men have had a prior TUR of the prostate and are on anticoagulants such as aspirin, warfarin or other blood thinners. A number of studies show that when these men develop recurrent hematuria, finasteride may be useful. There exists a relationship between finasteride treatment and prostate involution and, especially, induction of angiogenesis inhibitor factors such as VEGF. There are also ongoing trials testing finasteride prior to TURP in order to reduce the blood loss, but the results are too preliminary to recommend this routinely.

My medical therapy of last resort is alpha-blocker therapy. The category of men best served by alpha-blockers includes: severely symptomatic men, those with increasing post-void residual, or those who have failed earlier therapy. It has been estimated that, of the pool of approximately 15 million men who have symptoms of BPH, 50% of these men overall will sometime receive an alpha-blocker for therapy. There have been innumerable studies over the past 15-20 years evaluating several different alpha-blockers and all of them have demonstrated efficacy. Taking into account all of the studies, there have been over 2,000 patients studied on alpha-blocker vs. placebo protocols. The mean improvement in symptom score in these men is 49%, with an average drop of 5 to 6.5 points. The average improvement in flow rates is 44%, with an increase in the maximum flow rate of between 2 to 3 ccs per second. Approximately, 93% of patients started on long-acting (alpha-blockers have had symptomatic improvement. In 1996, Lepor and associates published an article on the efficacy of terazosin, finasteride, or both in BPH in the *New England Journal of Medicine*.[10] In this VA multi-center study, the patients were blinded into four groups. One group was placebo only; one group was terazosin only; one group was finasteride only; and one group was terazosin plus finasteride. The conclusions were that the differences between terazosin and finasteride were statistically in favor of terazosin in terms of both symptom score and flow improvements. The combination, however, was shown to be no more effective than terazosin alone, leading to finasteride being labeled a placebo effect. However, having prostate glands that were smaller in size than current guidelines may have blemished this study. The
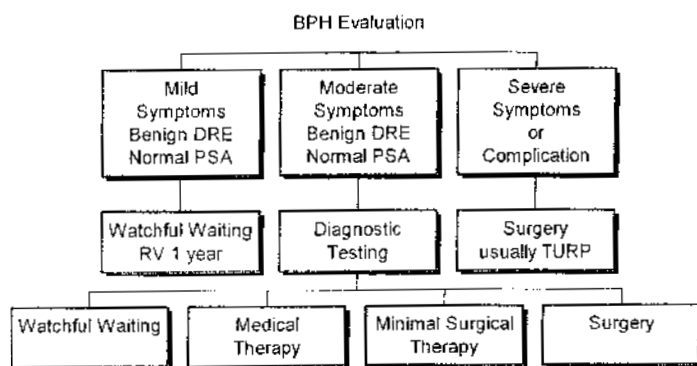
BPH Evaluation

Figure 3.

newest alpha-blocker is tamsulosin which is a super selective alpha1a-blocker which is not indicated, therefore, for hypertension. Due to the lack of antihypertensive effect, this has become a very popular therapy with urologists who do not routinely need to treat hypertension. The ALLHAT trial, which was published in the *Journal of the AMA* in 2000, has been interpreted as a condemnation of the use of doxazosin for BPH, because the patients in the doxazosin arm had an increase in congestive heart failure and cardiovascular disease.[11] This study however may suggest only that alpha-blockers are not ideal treatment of hypertension since this study was not designed to evaluate BPH treatments.

SURGICAL OPTIONS

When patients have failed medical therapy, it becomes necessary to discuss surgical options. My minimally invasive surgical option of choice is **transurethral needle ablation (TUNA)**. This procedure seems particularly suited to both the younger man worried about his potency and ejaculation and the older man who may be a medical risk for anesthesia. We currently have an office-based protocol in which men are given Demerol and Vistaril IM, intravesical lidocaine, and intraurethral lidocaine. The TUNA procedure uses low energy radio frequency ablation, delivered via 2 small needles placed into the substance of the prostate. The actual treatment time has averaged 36 minutes over the cases I performed in 2001.[12] If the patient voids he is immediately discharged catheter-free; if he does not void, then a catheter is inserted for two days. The discomfort rates during treatment have been low, and patients overall have tolerated the office-based procedure well. The improvement in both the patient's symptoms and flow rates exceed that seen in medical therapy including alpha-blockers

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but are less than that seen with traditional surgical therapy such as TUR of the prostate. On the other hand, TUNA is an office-based treatment under local, there is little chance of impotence or ejaculatory disturbance, and no need for anesthesia. The long-term complications of TUNA treatment are indeed minimal. The down side is that the durability is not known and, in studies of up to six years now, the retreatment rates with TUNA do exceed retreatment rates with TURP.

For those patients who have a complication of BPH, or who fail prior minimal surgical therapy, the treatment of choice is TURP, or open prostatectomy for larger prostate glands. The technique has been improved over the years, and the mortality rates now are no more than 1%. The long-term complications include a risk of impotence of approximately 5-10%, retrograde ejaculation of approximately 100%, and possible need for transfusion of 5%. This is an operating room-based procedure, and hospital stays of 1-3 days are routine.

CONCLUSION

BPH is the most common disease entity in the aging male. Newer options now permit a better selection of the treatment to the patient.

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Male Urinary Incontinence

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This article will describe the diagnosis and management of different causes of male urinary incontinence, with a focus on the most common type of male incontinence, post-prostatectomy incontinence.

Urinary incontinence can be broadly divided into **stress urinary incontinence (SUI)**, **urge incontinence (UI)** and overflow incontinence. SUI is loss of urine during physical exertion that increases intra-abdominal pressure, overcoming urethral sphincteric resistance. UI is associated with an overactive bladder, in which an urge to void or involuntary bladder contraction occurs at a volume less than capacity. UI is simply involuntary loss of urine associated with this urge to urinate (sensory UI) or involuntary bladder contraction (motor UI). Motor UI is seen in detrusor instability (caused by bladder outlet obstruction, bladder mucosal inflammation or tumor, or pelvic nerve injury) or detrusor hyperreflexia (caused by an upper motor neuron lesion, such as a brain lesion or spinal cord injury). Sensory UI is associated with increased bladder sensation but no actual detrusor contraction and can be seen in all types of overactive bladder, especially intersititial cystitis. Overflow incontinence is associated with urinary retention secondary to bladder outlet obstruction or detrusor failure and occurs when bladder capacity is exceeded. Mixed incontinence, usually a combination of SUI and UI is a common clinical finding.

Most often, significant male urinary incontinence is iatrogenic SUI directly related to surgery of the prostate and is known as **post-prostatectomy incontinence (PPI)**. By far, the most common surgical procedure associated with PPI is radical removal of a cancerous prostate (**radical retropubic prostatectomy or RRP**). Much less commonly, PPI can occur following surgical relief of prostatic obstruction, such as **transurethral resection of the prostate (TURP)** or open prostatectomy. Non-iatrogenic causes of male incontinence include UI or overflow incontinence as described above. In elderly males, the most common causes of UI include detrusor instability secondary to chronic bladder outlet obstruction, and less commonly detrusor hyperreflexia secondary to stroke or Parkinsonís disease.

POST-PROSTATECTOMY INCONTINENCE

According to physician-reported data, 0.3-87% of men have significant urinary incontinence after RRP.[1-8] Technical advances in the surgery are thought to have dramatically decreased severe incontinence,[3,4] but patient-reported data continue to show high rates of PPI (63%).[9] One per cent of men who have TURP have significant postoperative incontinence.[10]

Common causes of PPI include urethral sphincteric insufficiency, pre-existing bladder dysfunction, anastomotic urethral stricture or **bladder neck contracture (BNC)** or a combination of these factors.[11-14,18]

URETHRAL SPHINCTER INSUFFICIENCY

The most common cause of incontinence from RRP and TURP is direct damage to the male urinary sphincter. The male urinary sphincter has two basic components: voluntary and involuntary. The voluntary component is located predominantly at the urogenital diaphragm but can extend in a limited fashion proximally to the bladder neck. It consists of striated, somatic muscle fibers that contract volitionally for short durations but not continuously. Although it does not contribute significantly to overall urinary continence, the voluntary component may be damaged during RRP and TURP, contributing to incontinence.

The involuntary component extends from the bladder neck distally to the urogenital diaphragm and consists predominantly of autonomic smooth muscle fibers, although some striated muscle is present. This component provides almost all of passive, involuntary, continous urinary continence and is commonly damaged during RRP, resulting in urethral sphincter insufficiency. Possible contributory factors include: 1) excessive operative time or bleeding resulting in ischemia and scarring of the urethral anastomotic site, 2) excessive urinary leakage from the urethral anastomosis resulting in scar, 3) BNC requiring dilation or incision followed by sphincteric insufficiency. The end-result of this sphincteric damage is a shortened functional urethral length with scarred mucosa lacking elasticity and coaptation. Stress incontinence associated with urethral sphincter insufficiency is also known as type III stress incontinence or **intrinsic sphincter deficiency (ISD)**.

Certain risk factors may predispose to PPI following RRP. Younger men undergoing RRP have been reported to achieve continence sooner than older men,[15] possibly because of age-related muscle atrophy and the increased incidence of detrusor instabiity with normal aging. Nerve-sparing RRP has been observed to be associated with higher continence rates that non-nerve sparing RRP.[6] Salvage radical prostatectomy after radiation therapy has higher incontinence rates, which may be attributed to radiation damage to the bladder and urethra as well as the technical difficulty of this operation.

Preexisting neurologic disease is a risk factor for PPI. The risk of PPI after TURP in patients with Parkinson's disease has been reported as high as 20%,[16] because of the high rate of detrusor hyperreflexia and abnormalities of external sphincter control in this disease.

DIAGNOSIS OF URETHRAL SPHINCTER INSUFFICIENCY

Urodynamic (UDs) will rule out detrusor instability as a cause of UI,

detrusor failure as a cause of overflow incontinence and bladder outlet obstruction as a cause of both UI and overflow incontinence. Valsalva leak point pressures is a urodynamic measurement of abdominal or valsalva pressures required to cause SUI. Valsalva leak point pressure <50 cm H2O confirms ISD, although ISD can be demonstrated simply by eliciting severe SUI by coughing or valsalva maneuvers. Fluoroscopy (videourodynamics or voiding cystourethrography) can identify a BNC and evidence of preexisting bladder dysfunction (bladder trabeculation or vesicoureteral reflux).

Cystoscopy may reveal signs of urethral sphincter insufficiency, including a very short posterior urethra with scarred or blanched urethral mucosa. A fixed, damaged external sphincter that opens and closes incompletely may be seen following TURP or open prostatectomy. BNC should be ruled out, especially if an artificial urinary sphincter is contemplated (see below). Severely scarred urethral mucosa would argue against the use of transurethral bulking agents as a treatment for ISD because of the very low success rate. Therefore, cystocopy and UDs with or without fluoroscopy are very helpful in the diagnosis and management of PPI.

TREATMENT OF URETHRAL SPHINCTER INSUFFICIENCY
Conservative Options

PPI can result in tremendous morbidity, including limitation of usual activities, skin rashes, odor, need for anti-incontinence devices and corrective surgery. Usually, non-surgical management is recommended for at least one year because of the possibility for spontaneous improvement. After this, further improvement is unlikely. Prior to surgery, many men try Kegel exercises, empiric medications, bulky pads, external catheters and even penile clamps. Kegel exercises may improve mild PPI but are of minimal benefit in severe SUI because only the voluntary component of the urinary sphincter is exercised.

Bulky pads are inconvenient, uncomfortable, expensive, may carry an odor and facilitate rashes. External catheters can cause frequent **urinary tract infections (UTIs)**, allergic reactions, rash and soft tissue injury of the penis. In addition , they may not stay on the penis, especially if uncircumcized. Penile clamps are uncomfortable and can cause ischemic injury to the penile shaft and urethra.If urodynamics reveal motor UI, anticholinergics may be helpful, but do have common side-effects including dry mouth and constipation. Patients with closed-angle glaucoma may have blurry vision, which is reversible. Anticholinergics are unlikely to improve sensory UI.

The most effective treatment of severe PPI is an artificial urinary sphincter.

Treatment of Urethral Sphincter Insufficiency: Surgical Options

Conservative measures often are of minimal benefit, and surgical intervention is the next step. Surgery for PPI include transurethral injection of bulking agents, artificial urinary sphincter and the male sling. Transurethral bulking agents include bovine collagen, autologous fat, teflon and carbon coated beads. Collagen is injected at the bladder neck in a submucosal location, usually at the 5 and 7:00 or 4 and 8:00 positions. This results in co-aptation of the urethral mucosa. Possible complications include urinary retention, infection, bleeding and allergic reaction to collagen. Urinary retention is almost always temporary and commonly resolves after overnight catheter drainage. A test dose of collagen is injected in the forearm prior to collagen injection to identify allergy to collagen, and the injection site is observed for at least one month. Allergic reactions occur < 1%. Collagen injection for PPI have very poor results, most likely because the injection is usually into scarred mucosa and submucosa. It is not commonly done for PPI after TURP.

Fat as a bulking agent is even less successful than collagen. Teflon is not widely used because of the risk of migration of teflon to distant organs. Carbon-coated beads do not require a test dose and have been reported by its manufacturers to remain in location in a durable fashion. It is unclear whether clinical experience supports this finding. The superficial injection of carbon beads may be even more difficult in a scarred urethra than collagen. A recent report described migration of carbon beads to local and distant lymph nodes,[17] which raises reservations about the use of carbon beads similar to those regarding teflon.

The most effective treatment of severe PPI is an **artificial urinary sphincter (AUS)**. The AUS, made of silicone, consists of a cuff that wraps around the bulbar urethra, a regulating balloon that is placed in a prevesical location, and a scrotal pump. The device is fluid filled and works hydraulically: the pressure of the regulating balloon provides the occlusive force of the cuff. The intraabdominal location of the regulating balloon allows transmission of increased intraabdominal pressure during abdominal straining or stress maneuvers to the cuff to prevent leakage. The cuff is emptied by squeezing the scrotal pump, which transfers fluid to the regulating balloon, allowing voiding. Resistors in the AUS device delay cuff refill for 2-3 minutes, allowing time for voiding.

Preoperative UDs will identify impaired bladder contractility, which may require longer voiding times. The cuff can be emptied more than once. Men with completely acontractile bladders with large-volume urinary retention are poor candidates for AUS placement because of the risk of UTIs, which may infect the AUS. Catheterization is possible with an AUS, especially in children with neurogenic bladder, but this is not an optimal situation and may increase the risk of UTI and urethral trauma, both of which may lead to infection and erosion of the AUS.

UDs will also identify patients with impaired bladder compliance and high-pressure involuntary bladder.

Children with myelodysplasia with abnormal bladder parameters in whom AUS was placed have developed silent upper tract deterioration. Hostile bladder parameters such as impaired compliance or high-pressure contractions will worsen in the face of an inflated cuff, resulting in silent renal deterioration as well as urge incontinence. Therefore, an AUS should not be placed until these hostile parameters are adequately treated, which in itself may reduce incontinence by treating the urge component.

Preoperative cystoscopy will also identify a BNC. This should be treated prior to the AUS and observed for a minimum of 6 months for a recurrent contracture. The urethra around which the cuff is placed may be atrophic or narrowed secondary to ischemic damage during cuff placement. This increases the risk of damage at this site with subsequent passage of a large caliber instrument or urethral dilator. Therefore, a BNC that recurs after AUS placement may be difficult to incise or dilate without an increased risk of urethral injury and subsequent cuff infection or erosion.

Adequate parenteral antibiotics should be given before and after AUS placement. Strict sterile technique should be observed with minimal traffic in and out or the operating room to prevent contamination. Meticulous dissection around the urethra is necessary to prevent ischemic damage to the urethra or urethral injury. If urethral injury occurs, the urethra should be repaired primarily and the AUS should not be placed because of the high risk of infection associated with urinary extravasation. After AUS placement, the cuff should be deactivated in an open position for at least 6 weeks to allow urethral healing.

Three possible problems can occur after AUS placement: infection, erosion and malfunction. In an infection, the patient may have swelling, tenderness or erythema of a component or the entire device. A prolonged course of antibiotics may treat the infection. More commonly, a portion or the entire device is removed, followed by antibiotics and subsequent AUS

replacement several months later.

Signs and symptoms of an erosion include recurrent incontinence, AUS infection, gross hematuria, urethral discharge, perineal pain and edema. Early erosion usually results from urethral trauma at the time of insertion. Delayed erosions can occur because of infection or catheter trauma, especially when a catheter is inserted and kept in without deactivation of the cuff. Erosion can be confirmed by urethroscopy or retrograde urethrography and is treated by removal of the entire AUS and replacement after the erosion heals.

Mechanical malfunction usually occurs because of lack of attention to detail during insertion and usually presents as recurrent incontinence or failure to activate the cuff. Recurrent incontinence is caused by leaks in any component due to injury to the device during insertion or by detachment of tubing due to improper assembly during surgery. This is treated by replacement of the leaking component or by reattachment of the detached tubing. Cuff atrophy and improper positioning of the cuff can also cause incontinence. Cuff atrophy occurs because of ischemic trauma to the cuff during insertion or by pressure atrophy of the urethra from improper sizing. This results in a loose cuff. This is treated by cuff resizing, replacing in a different location, or adding a second cuff, preferably more proximally. If the initial cuff is not placed proximally enough, the patient can sit on the cuff, causing both perineal discomfort and deactivation of the cuff.

Failure to prevent blood or debris from entering the tubing at time of surgery may result in inability to activate the cuff by squeezing the pump. Using improper filling fluid during insertion may also result in clogging of the tubing. This also requires removal or revision of the AUS.

Careful attention to detail, meticulous surgical technique and testing the device for proper function, leaks, secure tubing connections and debris or blood in the tubing at the time of surgery can reduce the likelihood of later revision. However, despite all this, most clinicians are aware of the limita-

tions of this device. The male sling has been proposed as a surgical alternative to the AUS. A strip of cadaveric, autologous or synthetic material is placed underneath the bulbar urethra and tied over the rectus fascia or secured to the ischial rami with bone anchors after enough tension is placed upward to create urethral occlusion pressures as high as 90 cm H2O.[18,19]

Longterm results are not available. There are several additional concerns about this procedure. Unlike the female sling in which as little tension as possible is placed and the mechanism of continence is increased passive support to the bladder neck and proximal urethra, continence appears to be achieved by urethral occlusion. This may result in pathologic obstruction, bladder decompensation, upper tract damage, future urgency and UI. Adverse effects of cadaveric and synthetic materials and bone anchors have yet to be defined. Long-term follow up will hopefully address these concerns.

CONCLUSION

Male urinary incontinence is multifactorial but is most often iatrogenic following prostatectomy. Careful diagnostic evaluation will allow the clinician to determine the most effective treatment. If a surgical option is selected, meticulous surgical technique and attention to detail will optimize results. However, treatment for male incontinence can be suboptimal and frustrating. More effective treatment options are eagerly awaited.

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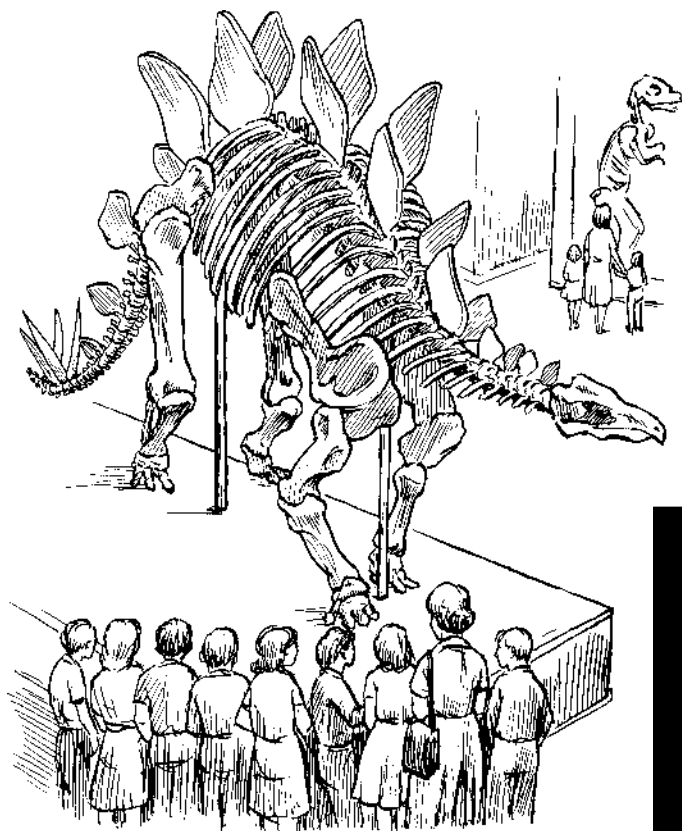
Osteoporosis and the Aging Male

Barry Stein, MD, and Seetharaman Ashok, MD

Osteoporosis in the male is a newly appreciated problem. Long associated with the aging woman, osteoporosis is now known to affect more than 2,000,000 American males over the age of 50, with another pool of 3,100,000 men who are at risk for developing it.[1-5] About one out of every 8 men over the age of 50 will at some point have an osteoporosis related fracture.[6-7] Every year about 100,000 men will suffer an osteoporosis related hip fracture; one third of them will die within the year. In addition, tens of thousands of men will have a fracture of the wrist, spine or rib. Physicians who treat men need to be increasingly sensitive to this problem. Thirty-six percent of the osteoporosis in men is due to low androgen levels, which can occur due to hypogonadism, either congenital, as part of the aging process, or due to acute androgen deprivation, such as the treatment of advanced carcinoma of the prostate.[8] In the latter case, given the usually advanced age of the patient, the acute loss of androgens is occurring in addition to the bone density loss of aging.[7] This can lead to a higher incidence of osteoporosis and its complications. Considering that hormone therapy is initiated earlier today due to the PSA monitoring of prostate cancer patients, a longer duration of androgen deprivation therapy can be anticipated, and the incidence of osteoporosis may yet increase.

At this time, there is no accurate measurement of overall bone strength, so we use the measurement of bone mineral density as its proxy. Bone mineral density accounts for 70-85% of bone strength, and the measurement of bone mineral density correlates with the load-bearing capacity of the skeleton.[5] **DEXA (Dual Energy X-Ray Absorption)** scans, which measure bone density, are our best gauge of bone strength. This technology utilizes X-rays of 2 different energy levels so as to distinguish between bone and the surrounding soft tissue structures. A two-dimensional image is produced, which can be used to measure bone density.[3] A number of prospective studies have been performed, most, but not all, in women, which show that DEXA scans can predict the future risk of fracture. Each decrease in bone mineral density of 1 **standard deviation (SD)** in the hip translates into a 1.5-3.0 fold increased risk of fracture.[5]

In reading DEXA scans, the measurement of the patient's bone mineral density is compared to one of two cohorts of men, whose results are already in the computer's data bank. The z-score compares the patient's DEXA results to an age and ideally race-matched cohort of men, and will compare that man to this cohort by both percent of normal and by SD readings. The cohort is based on the Caucasian male, because insufficient data exist at this time relative to other races. The t-score compares the patient's bone mineral density to young healthy men between the ages of 30-45. This represents the optimum age for peak bone mineral density, and the t-score is the more important measurement for the determination of bone density loss. Thus all results presented in this article will focus on the t-score for determination of the bone density status. In interpreting DEXA scans, a bone mineral density between 1 and 2.5 SD less than the cohort is read as osteopenia, while a bone mineral density loss of >2.5 SD is defined as osteoporosis.[2] When a DEXA scan reading is >2.5 SD less than the cohort of young healthy men, and the patient has a fracture, this is termed severe osteoporosis.

THE SCOPE OF THE PROBLEM IN PROSTATE CANCER

When three of the author's patients suffered hip fractures within the space of a year, we began to evaluate our patients who were being treated with **androgen deprivation therapy (ADT)** with DEXA scans. Of 75 patients studied to date, 70 were on leuprolide therapy, and 5 had undergone bilateral orchiectomy.[9] All patients underwent an in-office screening DEXA scan utilizing the fourth digit of the non-dominant hand. When this proved abnormal, a full table body scan of the hip and spine was performed for comparison. The patients' ages ranged from 46-98, with a mean of 76.4. The patients' duration of ADT ranged from just beginning treatment, to 13 years on therapy, with an average of 3.35 years. Of the entire series, only 34 (45.4%) had a normal bone mineral density. Of the 41 patients with abnormal DEXA scans, 25 (61%) had osteopenia, while 16 (39% or 21.3% of the entire group of 75 men) had osteoporosis.

When we examined the men by age, we found that those over age 70 had a greater risk of loss of bone mineral density than men younger than 70. Overall, 68.4% of the men younger than 70 years of age had a normal bone mineral density, compared to only 37.5% of the men over 70. Of the 25 men with osteopenia, 19 (76%) were older than 70, while all of the 16 men with osteoporosis were over 70. Duration of therapy also appeared to be important. We examined the 34 men who were on treatment for less than 2 years and compared their DEXA results to the men on ADT for more than 2 years. Of the less than 2 year group, 58.8% had a normal DEXA scan, while only 34% of the men on treatment for more than 2 years had a normal DEXA scan. We further examined the 34 men on therapy for less than 2 years, and found that those with normal bone mineral density, had an average age of 72.4 years, while those with abnormal DEXA scans, had an average age of 81.3. We examined the men by the number of years on therapy and as the duration of therapy became longer, the bone mineral density decreased. From our own work we conclude that the older the man is and the longer he is on ADT, the likelier he is to experi-

ence a loss in bone mineral density. Other studies have confirmed our results.

Smith and associates initially reported on 41 men with locally advanced prostate cancer, without metastatic disease, and who had not yet undergone ADT.[10] These men then underwent DEXA scan of the hip and spine. The mean age in this series was 68. Of this group, 66% had normal bone densities, while 29% had osteopenia, and 5% had osteoporosis. This compares very favorably to our findings in men on therapy for less than 2 years.

Stoch et al studied 3 groups of men: group 1 consisted of controls solicited via a newpaper ad, group 2 were men with cancer of the prostate, but not on ADT, and group 3 were men on ADT for at least 6 months, with a mean of 41 months of therapy.[11] The men underwent evaluation of bone mineral density by a variety of techniques including finger, spine and hip DEXA scans. They found that the normal rate of bone loss due to aging is 0.5-1.0% per year, but that LHRH analogue therapy was associated with more than a decade increase in this loss. They also reported an incidence of osteoporotic fractures similar to other groups.

Daniell performed 2 studies on osteoporosis and ADT. In the first paper, he reviewed the records of 235 men with prostate cancer, and from this culled the names of 17 men who had undergone orchiectomy between 1983-1990, and were still alive in 1995.[12] He then performed DEXA scans of the femoral neck and compared the results to 23 controls. He found 10 osteoporotic fractures in the larger group, 8 of which were found in the 17 orchiectomy patients. Of the 16 men who survived for >60 months, 6 had osteoporotic fractures and reduced bone mineral density on DEXA scans. The incidence continued to increase over time. In a follow up study, Daniell evaluated 26 men prior to orchiectomy or LHRH analogue therapy and followed them for 6-42 months, comparing them to 12 controls.[13] They found that bone mineral density in the ADT

patients fell about 4% per year for years 1-2, and 2% per year every year thereafter. The loss continued at a pace of 1.4-2.6% per year from years 3-8. Both orchiectomy and LHRH analogue therapy were likely to cause this loss.

Eriksson et al compared 2 groups of men on hormone therapy for prostate cancer: group 1 (11 pts) were treated with orchiectomy alone, while group 2 (16 pts) underwent orchiectomy plus estrogen therapy (IM or PO).[1] They then measured BMD of the femoral neck, trochanter and ward's triangle. There was a decrease in BMD in the orchiectomy only patients, not seen in the orchiectomy+estrogen patients. Statistical significance was achieved only in the forearm.

Given the high incidence of bone mineral density loss in men with prostate cancer undergoing hormone therapy, we recommend baseline evaluation with a DEXA scan.

RECOMMENDED EVALUATION AND TREATMENT

Given the high incidence of bone mineral density loss in men with prostate cancer undergoing hormone therapy, we recommend baseline evaluation with a DEXA scan. If the baseline scan is normal, no further evaluation is necessary at that time, and a follow up scan should be performed in 1-2 years. If the scan is abnormal, then treatment should be discussed with the patient, explaining the risks and benefits of treatment.

Men with abnormal scans should first be provided with counseling on nutrition and lifestyle issues. They should be instructed to eat a balanced, healthy diet, especially high in calcium content. They should stop smoking, moderate alcohol consumption, and

begin a regimen of physical exercise. Exposure to sunlight is also suggested, providing that they do not have skin cancer.

Our initial medical treatment for osteopenia is bisphosphonate therapy at osteopenic doses, Vitamin D and Calcium. Currently we use Alendronate 35mg once a week; and Vitamin D 400IU or more, and Calcium Carbonate or Citrate 1000mg daily. The Vitamin D and Calcium are often available as a combination marketed specifically for osteoporosis. The initial treatment for osteoporosis is identical except for increases in the dose of bisphosphonate, in this case to Alendronate 70mg weekly.

According to the NIH consensus conference 2000, both alendronate and risendronate are bisphosphonates, and have been shown to reduce the risk of vertebral fractures by 30-50% in randomized, clinical trials, although the majority of such trials involve female osteoporosis.[3] Orwoll et al reported on a randomized, double blind trial in men with osteoporosis, evaluating alendronate 10mg daily (n=146) vs placebo (n=95), with all men receiving calcium carbonate 500mg and vitamin D 400IU daily.[8] All men underwent DEXA scans of the lumbar spine, hip and total body up to 24 months. In the placebo group bone density remained unchanged, while in the alendronate arm the bone density increased, particularly in the lumbar spine. These changes were not related to testosterone or estradiol levels. The incidence of vertebral fractures in the placebo group was 7.1%, while in the alendronate group it was only 0.8% (p=.02). Frediani and associates also performed a placebo controlled study of alendronate 10mg daily (n=30) vs placebo (n=30), with all men receiving calcium 500mg daily. These men were followed up to 24 months with DEXA scans of the hip and spine. The men on placebo in this study had a loss of bone mineral density of 2.8-3.6%, while those on alendronate had a bone mineral density gain of 3.4-6.3%.

Smith et al recently published a new series of 43 men treated either with leuprolide alone (22) or leuprolide plus

IV pamidronate (a bisphosphonate) in an attempt to prevent bone loss.[14] All the men were placed on Vitamin D and Calcium supplements. The patients were evaluated up to 48 weeks with repeat DEXA scans. The authors found that in the patients on leuprolide and supplemental therapy alone the bone mineral density decreased by 3.3% in the spine, 2.1% in the trocanter and 1.8% in the total hip. By contrast, those patients whose treatment included bisphosphonate therapy experienced no change in their bone mineral density.

CONCLUSION

Osteoporosis is a major health threat to the aging male population, but especially to men with prostate cancer on ADT. This can be diagnosed easily with DEXA scans, and can be successfully treated. The men at highest risk are those >70 years of age, and on ADT for >2 years. Successful treatment can be undertaken with bisphosphonates, vitamin D and calcium. This diagnosis and treatment in men is just as important as diagnosing women with osteoporosis.

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❦ Borderland ❧

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Ms. J.D., a 29 year old computer analyst, reported mental fogginess and headache to her primary physician around Christmas 1986. When, in May 1987, I saw her in my office, the studies revealed a large bifrontal tumor. I hoped it would be meningioma. The craniotomy, however, disclosed an extensive, poorly encapsulated mass that was removed mostly by suction. Frozen section pathology showed malignant glioma. The tumor passed through the falx and involved both frontal poles; it was grossly, completely, resected including the falx and anterior sagital sinus. The final diagnosis was Grade III malignant glioma, signaling a poor prognosis.

Judi had a stable post operative course. Because of her youth and the total gross resection of the tumor, the oncology team and I discussed with her and her family very aggressive treatment.

A few weeks after discharge, with radiotherapy already underway, Judi and her mother attended a Catholic Mass during which they received communion. When the other parishioners left, they went to the side altar where they prayed before a visiting statue of Our Lady of Fatima that was on special exhibit. A priest appeared and, without saying anything, opened the tabernacle, the repository for Communion provisions, removed a single host and placed it on Judi's tongue. At that moment, she later told her mother, the Lord revealed to her that she would be healed.

Over the next several months, Judi indicated that her thinking seemed slightly fuzzy but, otherwise, she was functioning well in her daily life. Her gait and balance were good. She completed twenty-five radiation treatments. Since she responded so well to the treatments, she received an additional coned-down, radiation boost to both frontal lobes, for a cumulative tumor dose of 5980 cGy. On her final day of radiotherapy, her treating physician noted that she was "animated and alert" and had a normal examination.

Without telling anyone, Judi attended a Healing Mass where, her family learned later, the Blessed Mother appeared to her on the altar.

After consulting with the oncologists, we treated Judi with a protocol of bone marrow harvesting and storage, followed by intensive chemotherapy and replacement of the marrow. To control the brain edema, she required prolonged significant doses of Decadron which caused Cushingoid side-effects of hirsutism and obesity. She experienced muscle weakness and visual changes. She began having severe hip and shoulder pain from steroid-induced avascular necrosis of the femoral and humeral heads. Judi suffered tremendously during this period but remained stoic. Throughout her treatment, she never asked me or her other doctors, "How long do I have?" (Nor did her family).

By Spring 1989, we succeeded in weaning her off Decadron. She stabilized with only cystic changes appearing on her brain scans. She described her memory and function as normal. Cranioplasty was performed. She resumed her active life style, retraining and working as a medical secretary. She even skied although she had difficulty getting up when she fell.

Followup reports from Judi's radiotherapist and oncologist expressed their increasing amazement at her recovery, as evidenced in this note from 1990: "for all intents and purposes, she has to be considered cured of her primary tumor."

In 1992, Judi consulted an orthopedist hoping for relief from her continual hip pain and difficulty in walking. Considering the expected prognosis for her disease, the orthopedist recommended conservative treatment. Judi, however, insisted on surgery and underwent bilateral total hip replacement. In 1995, her radiotherapist reported that her only problem was pain in the shoulders.

In the summer of 2001, Judi experienced new mental status changes. The scans eventually revealed massive recurrent tumor, a biopsy was performed, and palliative care was instituted.

During her last admission, Judi told her family about the Divine message she received while attending Mass with her mother, so many years ago. She told them about the Healing Mass where the Blessed Mother appeared to her. As she recalled the experience that she had kept secret until now, her eyes filled with tears: "The Blessed Mother was so lovely and She had a beautiful fragrance." (Judi's frontal fossa surgery and radiation had interrupted her sense of smell.)

In her final days, the family heard her arguing, "I don't know why he can't agree with me."

They asked her whom she was talking about.

She replied, "God, but I don't agree with Him."

Her family interpreted this as an argument with God

to cure her, as, she believed, He had before.

Judi's sister, who practiced a charismatic form of Catholicism, also prayed for a miracle. With her Bible in front of her, she received the instruction to randomly open it. On that page would be God's message. The Bible fell open to Isaiah, Chapter 38, verses 1-5, the story of King Hezekiah who was "sick unto death."

> And Isaiah, the prophet . . .came unto [Hezekiah],
> Thus saith the Lord: Set thine house in order:
> for thou shalt die, and not live.
>   Then Hezekiah turned his face toward
> the wall, and prayed unto the Lord,
>   And said, Remember now, O Lord, I beseech
> thee, how I have walked before thee in truth
> and with a perfect heart, and have done that
> which is good in thy sight. And Hezekiah wept sore.
>   Then came the word of the Lord to Isaiah,
> saying,
>   Go, and say to Hezekiah, Thus saith the
> Lord . . . I have heard thy prayer, I have seen
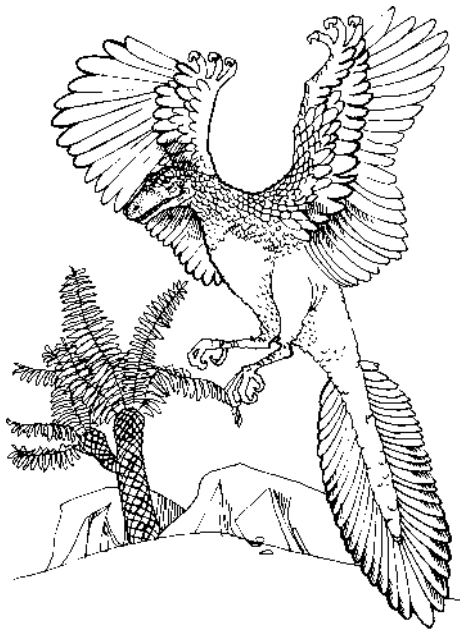> thy tears: behold I will add unto thy days
> fifteen years.*

* King James Version

Her sister realized Judi's fifteen years were up. Judi died soon thereafter.

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Primary and Secondary Prevention of Stroke

Andrew Sucov, MD

Stroke is a leading cause of death and disability in the United States, with an estimated 700,000 events annually.[1] Typically, stroke ranks third in most common causes of death, but a large proportion of victims survive with significant disability, causing economic impact along with the medical disability. Aggressive approaches to prevention and treatment of cardiovascular diseases have led to an increased focus on prevention and intervention in cerebrovascular disease. This review will focus on primary and secondary prevention of stroke, especially regarding some of the newer agents and approaches.

RISK FACTORS[1]

Nonmodifiable risk factors:
- Age: risk of stroke doubles with each decade after age 55.
- Sex: more prevalent in men, but the case-fatality rate is greater in women.
- Ethnicity: blacks and hispanics show approximately twice the risk of stroke of whites.
- Family history: parental history of stroke increases the risk of stroke (fraternal greater than maternal).

Modifiable risk factors:
- Hypertension: risk of stroke rises in direct correlation with both systolic and diastolic blood pressure.
- Smoking: current smoking approximately doubles the risk of stroke.
- Diabetes: increases risk of stroke from 2- to 6-fold, especially when combined with hypertension or smoking.
- Asymptomatic carotid stenosis: roughly 10-fold increase in stroke rate for >60% stenosis.
- Atrial fibrillation: responsible for ~50% of cardioembolic strokes, and 30- to 50-fold increase in risk of stroke if not treated.
- Other cardiac disease: dilated cardiomyopathy, valvular heart disease and intracardiac congenital defects predispose to cardioembolic strokes.
- Sickle cell disease: approximately 10% of patients with SS disease will have strokes by age 20. Patients with evidence of increased cerebral blood flow velocity by transcranial doppler are at particularly high risk.
- Hyperlipidemia: cholesterol > 240 is associated with a doubling of stroke risk.

Treatment recommendations:
- Hypertension: screen for hypertension every two years, with goal of treatment SBP 140, DBP 90. All drug classes appear to have beneficial effect - approximately 40% risk reduction if goal attained.
- Smoking: cessation leads to 50% reduction in risk within one year, and baseline risk within five years.
- Diabetes: tight blood pressure control reduces stroke risk by 45% in diabetics. No apparent benefit for stroke risk from tight glycemic control, but other complications are reduced.
- Asymptomatic carotid stenosis: carotid endarterectomy for patients with > 60% occlusion by surgeon with < 3% morbidity/mortality rate leads to 50% reduction in risk, from 2% to 1%. Because of significant potential complications, may not be appropriate for all patients or with all surgeons.

- Sickle cell disease: screen children with SS disease for increased cerebral blood flow velocity every 6 months, and consider aggressive transfusion (to < 30% Hb SS) - 90% risk reduction. Complications from transfusion may result.
- Atrial fibrillation: antithrombotic treatment with warfarin or aspirin based upon risk assessment (see reference 2 for complete details).
- Hyperlipidemia: use of "statin" agents can reduce stroke risk 20-30% in patients with concomitant cardiac disease. No evidence of stroke reduction in patients without cardiac disease at this time.

Secondary prevention

Once a patient has had a stroke, therapy is focused on preventing further strokes. All the modifiable risk factors should be addressed aggressively. In addition, antithrombotic therapy should be strongly considered. Four different treatment regimens have been studied: aspirin alone; ticlodipine (Ticlid®) alone; clopidogrel (Plavix®) alone; and aspirin and dipyridamole (Persantine®) [Aggrenox®] together.

Aspirin is the prototypical antithrombotic treatment. Its use has been well studied, and has consistently shown benefit. Aspirin's effects are on both platelet aggregation as well as vasodilation, mediated via prostaglandin synthesis in tissues other than platelets. Many studies have reviewed the optimal dosage, and there does not appear to be any difference in effect between doses ranging from 50 mg to 1200 mg daily.[3,4] Formulations in the US are routinely 81 mg and 325 mg/dose - typically once per day. Use of aspirin is associated with a roughly 20% reduction in incidence of second stroke. Side effects are typically mild, but can include gastritis or frank GI bleeding, and risk of bleeding is associated with increasing dosage. Minimizing other antiplatelet agents (alcohol, NSAIDs) will decrease the risk. No current recommendation exists for use of aspirin prior to a first cerebral event as exists with cardiac disease.

Ticlodipine (TC) is a synthetic agent that affects platelet function via cell membranes, not via prostaglandin synthesis. It is generally dosed as 250 mg BID. Use of TC is associated with a roughly 25% decrease in recurrent stroke compared with aspirin alone. Because of rare but serious side effects (predominantly neutropenia (0.9%) and TTP), usage is reserved for those who fail aspirin treatment.

Clopidogrel (CL) is chemically related to TC, and functions by irreversibly binding to the IIb/IIIa receptor on the platelet surface. Again, as prostaglandin synthesis is not affected, the potential for combinations with aspirin are physiologically possible, but have not been established by the literature. CL is generally dosed as 75 mg daily. Use of CL is associated with a roughly 9% reduction in second strokes compared with aspirin alone. While not clearly studied in the literature, there may be additional risk-reduction in patients with peripheral vascular disease. Serious side effects are rare.

Aggrenox® showed a 37% reduction in stroke compared with aspirin usage alone. In the studies, aspirin was dosed at 25 mg BID and DP at 200 mg BID. Combination formulation is available. Adverse events were minor, but hypotension may occur.

To date, few studies have been published that directly compare these regimens to each other. In addition, there is little guidance for the clinician on what to do with treatment failures - increasing doses of previous medication, switching to another medication, or combining medications. Several large studies currently underway should address these issues in the near future. Since little evidence exists to guide the clinician, some have based decisions upon costs of treatment and side effects.[3] Use of TC is generally restricted to patients with aspirin intolerance due to severe side effect profiles. CL has slight advantages over aspirin, but is far more expensive and use should be limited to treatment failures. No literature supports the use of combined CL and aspirin, but they are being used this way every day. Literature does support Aggrenox®, which may be the preferred option after failure on aspirin as both the costs and side effects of DP are low. Finally, patients with atrial fibrillation or structural cardiovascular/valvular disease may require anticoagulation, with or without any additional medication.[2]

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Health by Numbers

Cancer Incidence in Rhode Island Cities and Towns, 1987-2000

John P. Fulton, PhD, and Leanne Chiaverini

Since the recording of its first cancer case reports in October 1986 the **Rhode Island Cancer Registry (RICR)** of the Rhode Island Department of Health has been asked by various sources to produce cancer incidence rates for municipalities. Doing so requires at least ten years of cancer case reports and appropriate population data from censuses of the state's population. With the recent release of detailed demographic information for municipalities from the United States Census of 2000, it has become possible for the first time to produce cancer incidence rates for the 39 cities and towns of Rhode Island.

certained unambiguously in 97%. Another 0.2% included place names and corresponding zip codes that overlap more than one municipality. In these cases, the municipality identified as "primary" for the zip code by the United States

Methods

Counts of malignant neoplasms diagnosed between January 1, 1987, and December 31, 2000, categorized by age, sex, anatomical site, and municipality were prepared from cancer case reports made to the RICR. Municipality of residence at diagnosis was ascertained from three separate data fields: municipality, census tract, and zip code. Of 76,327 cases of malignant neoplasms diagnosed between January 1, 1987, and December 31, 2000, municipality of residence at diagnosis could be as-

Table 1. Age-adjusted, sex-specific statewide and municipal cancer incidence rates per 100,000, Rhode Island, 1987-2000.								
	Males				Females			
Municipality	Colon	Lung	Prostate	All*	Colon	Lung	Breast	All*
State	82.6	105.8	153.0	601.4	56.4	54.3	130.6	435.7
Barrington	81.1	87.9	173.3	581.4	49.8	42.1	146.4	436.5
Bristol	79.3	101.0	143.5	576.6	49.2	36.5	129.7	375.7
Burrillville	89.9	110.9	146.1	556.4	61.2	42.8	113.6	383.8
Central Falls	83.3	147.5	117.4	621.4	55.0	59.0	101.7	398.4
Charlestown	51.9	82.8	224.7	629.9	46.8	75.5	138.9	472.3
Coventry	85.2	124.3	149.7	607.0	61.6	52.9	134.3	445.5
Cranston	86.1	100.6	142.1	592.1	56.5	52.3	135.1	431.0
Cumberland	76.9	81.9	161.1	545.6	66.0	42.5	135.2	429.2
East Greenwich	90.6	105.8	240.4	726.1	68.7	42.3	175.2	508.5
East Providence	82.8	109.1	146.5	591.4	62.1	49.8	127.1	438.6
Exeter	51.0	98.0	125.3	449.0	52.1	51.5	120.7	427.8
Foster	57.2	86.6	175.0	590.6	40.8	71.0	163.5	454.3
Glocester	43.2	73.6	129.1	449.3	44.1	52.6	96.8	354.8
Hopkinton	102.5	100.7	176.9	669.3	61.5	62.6	151.3	512.4
Jamestown	45.8	80.2	196.3	623.6	58.9	66.0	152.4	476.3
Johnston	85.4	113.6	140.0	604.2	58.5	56.0	147.1	468.6
Lincoln	79.2	90.1	150.1	567.5	49.2	36.3	114.9	365.0
Little Compton	65.2	108.1	169.5	587.2	29.2	39.7	141.1	422.0
Middletown	70.6	111.7	236.1	693.8	58.3	70.2	146.4	472.2
Narragansett	77.1	84.9	198.2	579.0	68.7	54.6	130.6	457.6
New Shoreham	20.8	81.9	188.3	636.6	0.0	60.4	174.3	394.0
Newport	76.4	107.3	247.6	716.6	58.3	72.4	139.9	480.9
North Kingstown	96.4	102.2	207.8	706.0	50.9	61.9	139.1	470.7
North Providence	89.4	103.0	124.9	594.2	51.1	56.1	131.8	425.0
North Smithfield	72.7	77.5	118.4	523.7	64.6	45.0	115.8	431.0
Pawtucket	94.5	113.1	130.8	605.0	55.7	52.7	123.2	428.1
Portsmouth	87.4	97.1	202.3	598.5	63.1	62.4	153.1	468.0
Providence	72.2	111.6	136.7	588.7	53.2	56.7	123.2	435.5
Richmond	89.6	102.0	178.5	671.3	47.9	64.4	75.2	331.8
Scituate	66.2	125.2	176.7	602.1	51.6	42.1	167.3	438.1
Smithfield	74.2	93.9	151.8	581.3	54.4	59.5	130.0	435.3
South Kingstown	67.9	99.5	187.0	608.0	56.0	57.0	149.4	447.6
Tiverton	76.1	84.0	149.4	531.3	49.2	46.3	104.1	377.2
Warren	82.1	98.7	137.5	575.7	53.4	59.1	130.6	427.0
Warwick	89.8	114.1	156.8	637.9	57.8	65.1	133.7	460.8
West Greenwich	91.2	137.3	182.0	637.6	46.6	93.6	109.5	475.5
West Warwick	103.8	125.3	144.1	649.9	57.1	59.7	123.3	440.5
Westerly	78.5	89.7	174.0	625.1	61.1	46.8	127.3	433.7
Woonsocket	102.6	121.0	125.5	608.7	59.1	53.0	119.2	425.0

* All cancers combined

Table 2. Statewide cancer incidence rates per 100,000 and standard deviations of municipal rates, Rhode Island, 1987-2000.

Site	Males			Females		
	Statewide Rate	Standard Deviation	SD as % of State Rate	Statewide Rate	Standard Deviation	SD as % of State Rate
Colon	82.6	17.5	21.1%	56.4	11.8	20.8%
Lung	105.8	16.7	15.8%	54.3	11.8	21.8%
Prostate	153.0	34.6	22.6%	--	--	--
Breast	--	--	--	130.6	20.8	15.9%
All*	601.4	59.2	9.8%	435.7	39.8	9.1%

* All cancers combined

Postal Service was selected for use, or absent this information, the largest municipality associated with the place. The remaining cases (slightly less than 3%) contained no useful information on municipality of residence at diagnosis. To avoid underestimating incidence rates, these cases were randomly assigned to municipalities in proportion to the estimated populations of the municipalities for 1994.

Counts of the Rhode Island population by age, sex, and municipality were obtained from publications of the 1990 and 2000 United States Censuses of Population.[1] Analogous counts were estimated for the years 1991-1999 by linear interpolation, and for the years 1987-1989 by linear projection, using data from the two censuses.

Age-adjusted sex-specific statewide and municipal cancer incidence rates were calculated from cancer case reports, actual and estimated counts of the Rhode Island population, and the Year 2000 United States Standard Population.[2] Rates were calculated for all cancers combined and for the four most common malignancies, cancers of the colon-rectum ("colon"), lung-bronchus ("lung"), prostate (males only), and breast (females only).

Results

The statewide age-adjusted cancer incidence rate for all cancers combined is 601.4 per 100,000 among males and 435.7 per 100,000 among females. (Table 1) By municipality, rates among males vary from 449.0 for Exeter to 726.1 for East Greenwich, with a standard deviation of 59.2 over the 39 cities and towns. (Table 1) Municipal cancer incidence rates for all cancers combined among females vary from 331.8 for Richmond to 512.4 for Hopkinton, with a standard deviation of 39.8 over the 39 cities and towns.

Measured relative to statewide incidence rates, the standard deviations of the municipal rates for all cancers combined were 9.8% for males and 9.1% for females. (Table 2) Municipal cancer incidence rates for the four most common site-specific cancers

Table 3. Statewide and municipal cases of diagnosed cancer, Rhode Island, 1987-2000.

Municipality	Males				Females			
	Colon	Lung	Prostate	All*	Colon	Lung	Breast	All*
State	5132	6820	9849	38346	5420	4793	10881	37981
Barrington	88	95	185	623	68	59	196	587
Bristol	122	167	244	936	108	74	231	720
Burrillville	75	94	120	473	80	49	128	454
Central Falls	76	137	106	576	93	84	144	579
Charlestown	24	41	110	305	26	44	75	256
Coventry	151	232	274	1121	163	136	336	1132
Cranston	472	561	814	3291	491	396	979	3280
Cumberland	149	171	322	1086	191	121	353	1157
East Greenwich	67	76	173	535	71	42	177	517
East Providence	296	404	543	2125	376	266	626	2302
Exeter	13	28	33	131	18	18	41	148
Foster	13	19	42	141	12	18	48	129
Glocester	20	41	56	217	23	29	61	208
Hopkinton	37	42	66	262	31	31	75	255
Jamestown	14	30	73	216	26	29	66	205
Johnston	166	229	285	1195	176	149	372	1248
Lincoln	109	131	218	803	102	72	198	672
Little Compton	18	28	48	159	10	14	43	132
Middletown	68	113	236	702	96	101	201	680
Narragansett	66	75	174	517	73	62	139	496
New Shoreham	2	6	14	45	0	6	13	31
Newport	111	158	361	1064	141	161	285	1043
North Kingstown	127	143	281	972	96	116	260	882
North Providence	220	259	317	1458	196	195	429	1440
North Smithfield	56	60	93	406	79	46	114	453
Pawtucket	427	518	607	2760	406	353	761	2777
Portsmouth	83	105	200	611	81	83	199	609
Providence	585	911	1127	4860	717	668	1394	5238
Richmond	23	30	47	177	14	17	27	107
Scituate	42	78	111	382	37	31	122	318
Smithfield	87	112	178	687	105	99	203	728
South Kingstown	90	135	252	820	105	97	253	781
Tiverton	82	89	158	563	64	61	133	481
Warren	66	82	116	474	68	67	138	477
Warwick	515	698	949	3788	518	549	1058	3775
West Greenwich	16	25	32	123	8	19	31	112
West Warwick	170	233	258	1144	145	144	281	1053
Westerly	120	139	281	976	141	96	239	876
Woonsocket	266	325	345	1622	265	191	452	1643

* All cancers combined

vary more widely over 39 cities and towns. Their standard deviations range from 15.8% to 22.6% of the corresponding statewide rates.

Discussion

Cancer is a major cause of morbidity and mortality in Rhode Island, as it is in the United States as a whole. About four out of every 10 people in Rhode Island will develop cancer sometime in the course of their lives, and half of them will die of the disease. Close to 4% of the state's population (nearly 40,000 people) suffer from cancer at any one time.

Cancer is considered a public health problem because some cancers are preventable, and others controllable, through environmental or population-based interventions. For this reason, the United States[3] and Rhode Island[4] both have established clearly articulated cancer control objectives for their populations.

Among the many different forms of cancer that beset humans, cancers of four anatomical sites clearly predominate in the United States: 1) cancer of the colon, 2) cancer of the lung, 3) cancer of the prostate (males), and 4) cancer of the breast (predominantly females). Of these four, the first two are largely preventable, and the last two are more easily controlled if identified as small tumors. For this reason, all four figure prominently in cancer control objectives, using population-based prevention and early detection strategies proven to be effective in research studies.[3,4]

The relative effect of proven cancer control interventions from place to place may be examined by comparing cancer incidence rates computed from cancer registry data. Examining differentials in cancer incidence rates by municipality, for example, may be helpful in targeting local cancer control interventions. For example, municipalities with high lung cancer incidence rates might consider targeting the reduction of tobacco use, while those with high colorectal incidence rates might consider ways of increasing the proportion of eligible persons receiving endoscopic exams of the colon. On the other hand, municipalities with low prostate cancer incidence rates or low breast cancer incidence rates might consider ways of promoting screening tests for these cancers.

A caution that should be observed in comparing rates across geographic entities with small populations is that random factors (factors unrelated to the cause of cancer or their control) are more likely to influence cancer incidence rates in smaller populations, where the numbers of cases are relatively small, than in larger populations. (Table 3) Nonetheless, when interpreted judiciously, municipal cancer rates serve as a good introduction to more comprehensive thinking about the factors that cause and reduce the cancer burden (incidence, prevalence, and mortality) across geographic areas.

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– A Physician's Lexicon –

Diseases Named After Geographic Sites

The names of cities have frequently been used to define such varied things as prepared foods [e.g., hamburger, bologna, wiener schnitzel, consomme madrilene], cooking styles [e.g., lyonaisse, milanese], fabrics [e.g., calico named after Calicut, denims named after de Nims, jeans named Genes, the French spelling of Genoa], and even automobile models [e.g., Monte Carlo, Monterey]. And these cities have taken understandable pride in such namings. But, on the other hand, when diseases are linked with the names of cities, the town fathers vehemently object, feeling that their community has been unjustly stigmatized.

Typically, then, the name of a city [or geographic region] is exploited when some disease, usually infectious, is first encountered within its jurisdiction. For example, in 1946 public health officials observed a neuromuscular disease, clinically simulating acute poliomyelitis, affecting a few children in the Hudson Valley town of Coxsackie. Accordingly, the causative virus and the disease were named after the town. In 1934 a somewhat similar disorder, but with high fever and associated with chest pains, was seen on the Danish Island of Bornholm; and Bornholm disease was eventually shown to be caused by the Coxsackie virus.

In 1968 a small cluster of pneumonia cases was recorded in Pontiac, Michigan. Years later, Pontiac fever was shown to be caused by the *Legionella* organism.

Malta fever, now better known as brucellosis or undulant fever, was first identified on this island by David Bruce. Aleppo [Syria] sore is but one of many names given to the dermatological manifestations of the parasitic disease caused by *Leishmania tropica*. The state of California has had more than its share of geographic names appended to diseases. There is tularemia, caused by *Francisella tularensis* and named after Tulare County; San Joaquin Valley fever, a systemic mycosis caused by *Coccidiodes immitus*; and California encephalitis, an arbor virus infection.

Valleys and rivers also provide names for a number of infectious processes: Murray Valley [Australia] fever, a viral encephalitis; Rift Valley fever; Hantaan fever [named after a Korean river]; Ebola fever [named after a river in northern Congo]; and West Nile fever.

Various forms of arthropod-borne viral infections of the brain still carry their earlier geographic names: Japanese B, St. Louis, Venezuelan and Ilheus [Brazil] encephalitis.

In the year 430 BC, Athens was devastated by a lethal communicable disease which historians now call the Athenian Plague. Fortunately, Athens' self-esteem as home of Western philosophy and early democracy has overshadowed its reputation as the site of Europe's first urban epidemic.

Cities such as Lyme, Connecticut, and St. Louis, Missouri, lament the reality that they are indelibly linked with certain diseases. Yet their burdensome fate is small when compared with the city of Sodeom [cf. Genesis 18,19] identified for all eternity as the site of such practices as bestiality and sodomy.

– Stanley M. Aronson, MD, MPH

Vital Statistics

Rhode Island Monthly Vital Statistics Report

Provisional Occurrence Data from the Division of Vital Records

Underlying Cause of Death	Reporting Period			
	May 2001	12 Months Ending with May 2001		
	Number (a)	Number (a)	Rates (b)	YPLL (c)
Diseases of the Heart	259	3,089	294.7	4,484.5 **
Malignant Neoplasms	205	2,415	230.4	6,584.0
Cerebrovascular Diseases	41	504	48.1	720.0
Injuries (Accident/Suicide/Homicide)	25	375	35.8	6,531.0
COPD	44	517	49.3	472.5

Vital Events	Reporting Period		
	November 2001	12 Months Ending with November 2001	
	Number	Number	Rates
Live Births	832	13,074	12.5*
Deaths	860	10,165	9.7*
Infant Deaths	(15)	(113)	8.6#
Neonatal deaths	(13)	(97)	7.4#
Marriages	570	8,550	8.2*
Divorces	269	3,357	3.2*
Induced Terminations	427	5,449	416.8#
Spontaneous Fetal Deaths	59	991	75.8#
Under 20 weeks gestation	(54)	(915)	70.0#
20+ weeks gestation	(5)	(76)	5.8#

(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 1,048,319

(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
** Excludes two deaths of unknown age.

NINETY YEARS AGO
[APRIL, 1912]

The Journal announced the Program of the 100th Annual Meeting of the Rhode Island Medical Society. The centennial celebration began at 10:30 a.m. at the 136 Dyer St. dock, where the Steamer Squantum ferried celebrants to Rocky Point. The morning was filled with ball games, wrestling matches, round robins, and motion pictures. At 1 pm all feasted on Rhode Island shore dinners, followed at 3 pm by siestas. At 3:30 the steamer returned to Providence. Later that evening, at 8:30, the Society formally opened its new library on Francis St.

In an address before the Providence Medical Association at its 1912 annual meeting, retiring president F.P. Capron, MD, discussed a major concern: "...patients well able to pay a satisfactory fee for an operation, [who] betake themselves to a hospital, hire a private room and [are] operated upon and attended by a surgeon, who happens to be on the visiting-list, thus obtaining the same treatment for nothing which they ought to pay for freely."

Charles Chapin, MD, summarized the "Health of Providence." In February his office recorded 316 deaths (32 fewer than in February 1911), an annual rate of 17.08 in an estimated population of 233,502, the lowest February rate since 1883. In the past two months his office had recorded 98 cases of scarlet fever (4 deaths), 12 of diphtheria (2 deaths), 14 of typhoid (1 death), 179 of measles (24 deaths), and 11 deaths from whooping cough. In addition, Dr. Chapin noted "A good deal of rubella."

FIFTY YEARS AGO
[FEBRUARY, 1952]

Robert Elman, MD, Professor of Clinical Surgery, Washington University, and the author of several surgical texts, delivered the 4th Annual Dr. Isaac Gerber Oration: "The Surgeon as Technician and Physician." Dr. Elman recounted the fluctuating reputation of surgeons, which began on a high note in the time of Hippocrates, then declined with Galen, whom Dr. Elman described as an "intellectual snob." The early Christian church, which considered the body sacred, considered exposing the body for surgery a sinful act.

Malcolm Winkler, MD, contributed "Early Cancer of the Skin," where he described basal, superficial epitheliomatosis, squamous, and melanoma cancers.

In "Extragenital Chorionepithelioma in a Female Arising from a Mediastinal Teratoma," Herbert Fanger, MD, and Raymond MacAndrew, MD, described the first reported case of the condition in a female. (The literature described only 12 other cases, all in males.) Eleven years before admission, this 44 year-old woman had had an inguinal hernia. Five weeks before admission, she had had a cold, with a dry hacking cough, but no chills or fever. The authors found no tumor in the genitalia, but found tuberculosis of hilar lymph nodes and liver.

An Editorial on Fluoridation of Water Supplies urged "continuation of controlled studies," but offered lukewarm support for the practice: The Rhode Island Medical Society..." does not oppose the fluoridation of water supplies...but... is not prepared to urge the adoption of fluoridation at the present time."

A second Editorial on Pollution recounted a meeting of the Providence Medical Society with Mr. Austin Daley, Air Pollution Engineer for the City of Providence. Mr. Daley "is rather pessimistic about improvements in our city streets where large trucks and buses still pour out products of incomplete combustion."

A Eulogy for Isaac Gerber, MD, praised Rhode Island's first full-time radiologist, who first brought radium to Providence for use on patients.

TWENTY FIVE YEARS AGO
[FEBRUARY, 1977]

In a "Message from the Dean," Stanley M. Aronson, MD, reported on the internships at the Brown-affiliated hospitals in 1977-78: the hospitals offered 92 internships; 91 were filled by the Match, drawing students from 45 different American medical schools and 4 overseas ones. Fifteen of the 91 new interns were native Rhode Islanders.

In addition, Stanley M. Aronson, MD, introduced the 61 members of the class of 1977, with brief descriptions and pictures.

Several students contributed essays. Phyllis Ann Margaret Hollenbeck voiced "...little doubt that the sight of any basement room, with blue carpeting, too many chairs, and no windows can cause an acute claustrophobic reaction in most of my classmates."

Herbert Hager, the outgoing Rhode Island Medical Society president, objected to the "Certificate of Need" bill in the General Assembly. The bill was intended to limit the proliferation of computerized radiographic scanners to hospitals. Dr Hager argued: "As a native free-born citizen of this state, which was settled by Roger Williams, and whose capital building is adorned by the statue of the Independent Man, I am appalled at the loss of individual freedom imposed by this bill."

Myra Bergman Ramos, AM, Mary Ellen McCabe, LLB, and Stanley M. Aronson, MD, contributed "A Statistical Profile of Physicians Issued Licenses by the State of Rhode Island, 1967-1976." In that decade the state issued 1273 new licenses. From 1967-70 foreign medical graduates received 24.3% of those new licenses (36 of 148); from 1971-3, 52.5% of new licenses (43 of 177); from 1974-76, 50.8% of new licenses.