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Medicine & Health RHODE ISLAND

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Urogynecology

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COMMENTARIES

CONTRIBUTIONS

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Guest Editor: Deborah L. Myers, MD

COLUMNS

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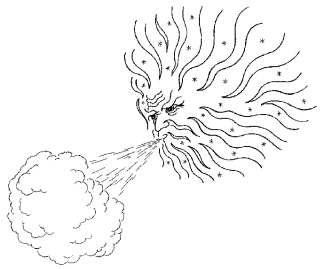
Commentaries

Reimbursement for Experience-Based Medicine



The editor of *Annals of Neurology*, the publication of the American Neurological Association, recently wrote an editorial trying to come to grips with insurance reimbursements being unrelated to experience. As we all know, insurers pay flat rates depending on diagnoses, whether the patient gets good care or not, so long as it is documented care. What I hadn't realized until I read the article was that there is a body of published data that actually tracks "quality" of care in relation to physician's age and duration of practice.

What surprised me was that these data, perhaps not the best epidemiology work extant, indicate that "experience" is *not* associated with improved care, and is often associated with worse outcome.

It's not simply that I'm now an older physician that makes me respond to this like I've heard nails on a chalkboard (a metaphor appropriate for an older person) but rather that I wouldn't have believed that when I was younger, and don't now. When I first started out I used to call my old mentors frequently about troubling cases, refer to the big academic centers for second opinions, and send my EEGs for review. After I got my sea-legs, I reduced my second guessing to a low level, as I learned that when I didn't know something and had an opportunity to research the area, chances were the other guy didn't either.

The literature indicates that younger cardiologists produce better results than older ones, that younger PCPs follow guidelines better than older ones, and that by any criterion of quality or outcome, younger physicians do as well or better than the older ones. On the one hand I can believe this, yet on the other I'm not so sure. Do the older doctors get the more difficult cases? For some of the studies this is clearly not true. Patients were tracked by diagnostic codes in very large numbers using insurance company data.

Obviously I wonder if the older doctors are out of date. We all have to work harder, see more patients for less money than we used to. This means less time for journal review, attending conferences and "keeping up" in general. This time-crunch means that those more recently trained have less keeping up to do. Perhaps their skills in technical areas are better. Or they perform better on the measuring scales because they were trained with the measuring scales in mind. One of the major philosophical debates regarding "No child left behind" is whether teaching to score better on a standardized exam is of any value other than improving test performance. Some, but clearly not all, of these outcome studies may reflect that. But, on the other hand, how can one measure the physician-patient relationship? How can one compare the reassurance a patient feels from a doctor who has helped hundreds of patients cope with the same problem to one whose experience is limited? Is there any way to compare the experience of returning to a doctor who has had a twenty-year experience with the patient and his family to that of a younger doctor? The doctor-patient relationship is sometimes more important than choosing the first line treatment instead of the second. These are intangible; and we are limited, of course, to measuring what we can measure.

The various medical disciplines have tackled the problem of keeping up to date by re-credentialing exams every 10 years. While I am an ardent supporter of this I have not renounced my "grandfather" clause protection that lets me avoid the process. Am I keeping up? How can I tell? In my own narrow subspecialty I'm pretty confident that I do and I have a number of objective measures to support that. In the wider spectrum of neurology am I up to date? Hard to say. In the academic sphere where one interacts with neurology residents it's much easier. They correct you. They quote the expert with whom they rotated the month before, to tell you what is now timely, perhaps work not yet published. Out in the "real world" it is impossible to be sure.

Should doctors be paid differently based on experience or expertise? Do they do a better job? Evidently not by established measures. Are they less expensive, able to rely more on experience than expensive testing? We don't know. In the academic workplace, pay is based on seniority, and collections. In private practice it is not. The Mayo Clinic, an academic-private practice, has a flat payscale that ignores seniority. I don't think a flat reimbursement is right, again perhaps because of my age. Yet that's what insurers pay. One pays more to an experienced famous lawyer than to a newcomer. Yet if I go to a famous doctor or an unknown one, the fee is the same, unless the doctor refuses insurance. Yet psychiatric fees vary enormously in the big cities, with some doctors charging $600/hr, and some $150. They can do this because they refuse insurance. The patient pays out of pocket and the insurance company pays whatever percentage they deem "reasonable." Even when the economy was humming along, this would be impossible in most parts of the country. And if we decide that quality is important, how is that to be determined?

I have thought of abandoning acceptance of insurance, thus reducing overhead enormously and increasing my charges, but then my patients, largely Medicare, almost all insured, would have to pay a lot more; and many of them cannot. Which is why, of course, medicine is so different than law, accounting or other businesses.

If and when our disaster of a healthcare system gets straightened out, this will be another issue that we should confront.

– Joseph H. Friedman, MD

Disclosure of Financial Interests

Joseph Friedman, MD, Consultant: Acadia Pharmacy, Ovation, Transoral; Grant Research Support: Cephalon, Teva, Novartis, Boehringer-Ingelheim, Sepracor, Glaxo; Speakers' Bureau: Astra Zeneca, Teva, Novartis, Boehringer-Ingelheim, GlaxoAcadia, Sepracor, Glaxo Smith Kline, Neurogen, and EMD Serono.

I've Got a Little List...I've Got a Little List

We think of obsessions as emotions so intense as to nullify rectitude or reason. Admittedly, obsessions can be narrowly focused, such as the building of the world's biggest sandcastle or collecting the most diversified display of butterflies, but most are broader and pertain to unrequited human passions. Obsessions may come in all sizes and durations; they may be trivial, short-lived, or enduring and magnificent in their grandeur and sweep. Generally though, they are persistent, phobic, haunting, anxiety-producing and sometimes maniacal.

Then there is the word compulsion or its adjective, compulsive. Dictionaries define this word as the fulfillment of an act, usually initiated by an irresistible impulse which is contrary to the individual's conscious agenda. While most compulsions stem from the psyche of the individual, some compulsions are extracorporeal, established through legislation or societal regulation. Compulsory gymnasium attendance or compulsory military service, for example, representing things required by society but not arising, necessarily, from the inner emotional needs of the individual. Compulsive behaviors, generally, are repetitive, on the surface illogical, and in reaction to obsessive moods. These inciting obsessions may be blasphemous thoughts, unbidden aggressive feelings or, frequently, inappropriate sexual ideation.

And the synergy of obsession and compulsion? Obsessive feelings often initiate unreasonable, compulsive responses. Unreasoned fear of bacterial contamination, for example, may be so distressful that only repetitive hand-washing offers any relief.

Obsessive-compulsive disorder is now a defined psychiatric condition afflicting about two percent of the adult population. Obsessive-compulsive behavior, on the other hand, is far more pervasive, tends to be more narrowly idiosyncratic, episodic rather than continuous and does not overwhelm or paralyze its victims.

It is sometimes stated that Professor X, despite being a victim of Disease Z, nonetheless succeeded in elucidating the cause of Disease Y. The operative word in the preceding sentence, 'despite', suggests that were it not for the oppressive effects of Disease Z, Professor X would have accomplished yet more during his lifetime. Perhaps. But sometimes there are clinical features inherent in Disease X that might enhance rather than restrain the creative impulses of someone with the temperament of Professor X. Consider the lengthy and creative life of Peter Mark Roget, physician, teacher, lexicographer and scientist.

The SoHo district of London, during the 18th Century, was heavily populated with Huguenot refugees from France. Declaring that adherence to the Protestant faith was illegal, Louis XIV expelled the Huguenots [followers of Hugues, a disciple of Calvin] from Catholic France. In 1775, the French Protestant Church in London, in need of a pastor, recruited Reverend Jean Roget of Geneva. In 1778 he married Catherine Romilly, daughter of a prominent British family. On January 18, 1779, their first child, Peter Mark, was born. Later that year Pastor Roget took ill with tuberculosis. He and Catherine fled to Switzerland in hopes of finding a rest cure in the high Alpine altitudes. Peter, still an infant, stayed in London with his mother's family.

These were troubled times for the Roget child: his father was dying in Switzerland, while his surrogate family in England, the Romillys, were distracted by widespread mental disorder within their ranks. Following his father's death, his mother's possessive dependency made his childhood extremely difficult. He learned to cope, however, with a compulsive habit of classifying things; and he maintained a series of notebooks containing all of his revelations on the orderliness of life around him. His great hero was Carolus Linnaeus [1707 – 1778] the great Swedish physician-botanist who organized all living matter, whether plant or animal, in a great binomial classification used, virtually unaltered, to this day. Young Peter marveled at the genius of Linnaeus to reduce the immensity of life, from the smallest to the largest, into a systematized regimen which replaced an unsettling vision of life with an idyllic and structured sense of order: every plant, every creature in proper relation to each other, a tranquil tapestry in accord with God's concept of order.

And so young Peter went through childhood constantly making lists of things as his way of transforming chaos into a serene, symmetrical visage of life. Peter began his University education in Edinburgh in 1793. His medical studies went well and he was awarded his doctoral degree in medicine on June 25, 1798.

Despite intervals of intense anxiety, obsessive ideation regarding cleanliness, and depression, the next few decades saw Dr. Roget practicing in Manchester and finally establishing a commendable practice of medicine in London. He achieved prominence not only in the clinical arts but as the inventor of the log-log slide rule, as Secretary of the Royal Society, as an esteemed lecturer in medical physiology, and as the author of that century's most authoritative text on comparative and human physiology.

These were also difficult years for Roget: he witnessed the suicide of his father-in-law; saw the emotional deterioration of his mother, his sister, and later, even his daughter. His ultimate professional goal in life, however, was fulfilled, namely, the gathering of his many verbal lists into a single, memorable lexicographic text of synonyms. He remembered the words of his professor at Edinburgh: "As it is by language alone that we are rendered capable of general reasoning, one of the most valuable branches of logic is that which relates to the use of words." And Peter's compulsive habit of listing things evolved finally into "Roget's Thesaurus."

– Stanley M. Aronson, MD

Disclosure of Financial Interests

Stanley M. Aronson, MD, has no financial interests to disclose.

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The Role of Urogynecology In Women's Pelvic Floor Disorders

Deborah L. Myers, MD

Pelvic floor disorders (PFD) include urinary incontinence (UI), fecal/ anal incontinence (AI), and **pelvic organ prolapse (POP).** An estimated one third of women will experience at least one of these disorders in her lifetime. A prevalence rate of all PFD combined is not available, but estimates of each of these dysfunctions have been reported in epidemiologic studies. UI is the 8th most prevalent chronic medical condition. It affects approximately 13 million Americans: 50% of nursing home residents, and 15-30% of the community elderly. POP is a common condition amongst women. The exact prevalence rate of the condition is not known, but a study by Henrdrix et al of 16,616 women with a uterus found the rate of uterine prolapse to be 14.2%; the rate of cystocele 34.3%, and the rate of rectocele 18.6%. In the same study 10,727 women who had undergone hysterectomy had similar rates of prolapse, the prevalence of cystocele was 32.9% and of rectocele was 18.3%.[1] Olsen et al found that American women have an 11% lifetime risk of undergoing surgery before the age of 80 for either urinary incontinence or prolapse with 30% of women undergoing repeat surgery.[2] AI may have the most devastating effects on quality of life, self-image, and social functioning of all pelvic floor disorders. It is defined by the International Consultation on Incontinence as the involuntary loss of gas, liquid, or solid stool that causes a social or hygienic problem. Women are twice as likely to report AI as men. Symptoms are highly associated with anal sphincter injury following vaginal delivery. A recent multi-center survey study by Boreham, et al found that up to 28% of women presenting for routine gynecologic care reported AI in the preceding year.[3]

The US Census Bureau projects that by the year 2030, the population over age 65 will double to over 70 million in the US, and over 1 billion worldwide. With the increase in the aging population, the prevalence of pelvic floor disorders will likely increase. Over these next 30 years, growth in demand for services to treat female pelvic floor disorders will increase at *twice* the rate of growth of the same population. These findings have broad implications for those responsible for administering programs that care for women, allocating research funds in women's health and geriatrics, and training physicians to meet this escalating demand.[4]

A urogynecologist is an obstetrician/ gynecologist who has specialized in the care of women with pelvic floor disorders. Urogynecologic training is achieved through three-year fellowship programs in **Female Pelvic Medicine and Reconstructive Surgery (FPM&RS),** under the auspices of both the **American Board of Obstetrics and Gynecology (ABOG)** and the **American Board of Urology (ABU).** There are currently 32 accredited fellowship programs within the United States. Urogynecology fellowships provide comprehensive training in pelvic floor disorders for women; trainees are needed to meet future clinical, research, and educational demands. The urogynecologist provides overall care of the pelvic floor through a complete approach and one that is often multi-disciplinary. The urogynecologist does not work alone since many pelvic floor disorders are affected by other conditions. Sensory and emptying abnormalities of the lower urinary tract and bowel, pelvic and abdominal pain, musculo- skeletal dysfunction of the pelvic muscles/ ligaments, and constipation and diarrheal states all affect PFD. Therefore, urogynecologists work in conjunction with physical therapists, gastroenterologists, urologists and colo- rectal specialists. The urogynecologist is best positioned to diagnose and provide a comprehensive treatment plan for this group of women.

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The author has no financial interests to disclose.

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Pelvic Organ Prolapse

Brittany Star Hampton, MD

The levator ani pelvic muscles and surrounding connective tissues provide support for the pelvic organs. Disruption of this natural anatomic system results in descent or prolapse of one or more of the pelvic structures: bladder and urethra, rectum, uterus and cervix, and small bowel. Patients with prolapse often present with associated urinary, defecatory, and sexual dysfunction, although many women who have prolapse on examination are clinically not affected.[1] The finding of prolapse on exam is not well correlated with symptoms. Thus, pelvic organ prolapse encompasses a wide range of disorders, from asymptomatic altered anatomy to complete eversion of the vagina. The lifetime risk that a woman in the United States will have surgery for prolapse and urinary incontinence is estimated at 11%.[2] The direct cost of prolapse surgery is greater than $1 billion per year.[3]

The pathophysiology of prolapse is multifactorial. Risk factors can be predisposing, inciting, promoting, and decompensating.[4] Risk factors include family history, connective tissue disorders, race, gravidity and parity, prior prolapse surgery, myopathy, neuropathy, advancing age, menopause, and elevated intrabdominal pressure from obesity, constipation, occupational activities, or chronic cough. Nulliparity does not protect against prolapse: one fifth of the nulliparous women in the Women's Health Initiative had some degree of prolapse.[5]

Diagnostic Approach

History

In evaluating a patient with pelvic organ prolapse, it is important to ascertain symptoms and bother, because many patients with prolapse are asymptomatic. Patients who are symptomatic will present with complaints in at least one of four categories: lower urinary tract dysfunction, defecatory dysfunction, sexual dysfunction, or feeling and /or seeing a "bulge". Symptoms of lower urinary tract dysfunction may include hesitancy, slow stream, need for position change to void, or incomplete bladder emptying. Some women with advanced prolapse may recount a history of stress incontinence that has improved over time. This is likely due to the urethral obstruction or "kinking" caused by the advancing prolapse. Symptoms of defecatory dysfunction include incomplete evacuation and the need for application of manual pressure to the perineum or posterior vagina to complete a bowel movement, commonly called "splinting." Pelvic prolapse may interfere with sexual activity secondary to embarrassment, concern, or fear of incontinence. Awareness or palpation of an actual protrusion usually occurs when prolapse is at or below the hymen, but women with prolapse above the hymen may complain of pelvic pressure or heaviness.

The patient interview should address all of the above. Additionally, questions regarding conditions that may contribute to the progression of pelvic prolapse should be asked, such as gravidity and parity, menopausal status, conditions or activities contributing to elevated intra-abdominal pressure, and prior surgery. Again, the interview should assess how much bother the symptoms are creating for the patient. Several validated questionnaires are available to quantify and qualify symptoms. Finally, it is important to ascertain patient treatment goals. Patients will benefit from reassurance and education regarding their condition.

Physical Examination

Physical exam begins with visual inspection of the vulva and vagina. The provider can assess the patient's neurologic function in the pelvis by testing for an anal wink (bulbocavernosus reflex) and the dermatomes of the perineum and upper leg. Efficiency of bladder emptying can be assessed by measuring a voided volume and the post void residual by either catheterization or ultrasound. The extent of

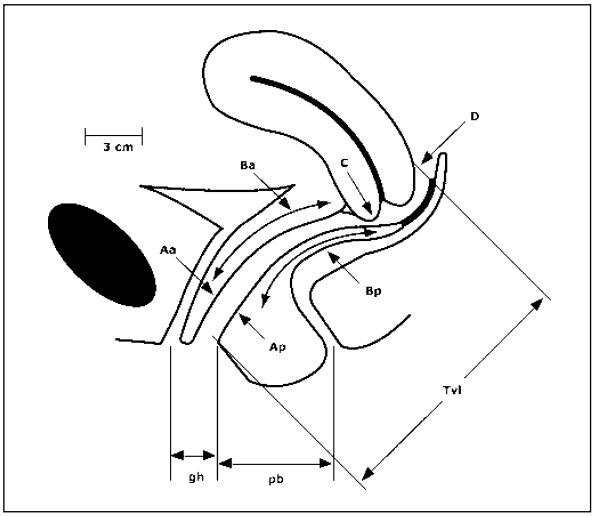

Figure 1. Six sites (points *Aa, Ba, C, D, Bp,* and *Ap*), genital hiatus *(gh),* perineal body *(pb),* and total vaginal length *(tvl)* used for pelvic organ support quantitation. (6)

Table 1. Pelvic Organ Prolapse Quantification Staging (6)

Stage 0	No prolapse is demonstrated.
Stage I	The criteria for stage 0 are not met, but the most distal portion of the prolapse is >1 cm above the level of the hymen
Stage II	The most distal portion of the prolapse is =1 cm proximal to or distal to the plane of the hymen
Stage III	The most distal portion of the prolapse is >1 cm below the plane of the hymen but protrudes no further than 2 cm less than the total vaginal length in centimeters
Stage VI	Essentially, complete eversion of the total length of the lower genital tract is demonstrated. The distal portion of the prolapse protrudes to at least (TVL-2) cm

pelvic organ prolapse should then be systematically evaluated and with Valsalva efforts. If the prolapse is advanced this is often not difficult. However, if prolapse is not obvious, the use of a "split" vaginal speculum is needed to determine which vaginal supports (anterior, apical, posterior) are affected by prolapse. Retraction of the posterior wall of the vagina with the single blade of a speculum will help identify anterior prolapse, and vice versa, retraction of the anterior wall of the vagina with the single blade of a speculum will help identify posterior vaginal prolapse. The supports of either uterus/ cervix or post hysterectomy vaginal cuff of the vagina, that is the apex, are assessed with the bivalve speculum.

The International Continence Society developed a standard system for measuring and staging prolapse known as the **Pelvic Organ Prolapse Quantification (POPQ)** system.[6] This standardized system allows for objective evaluation of prolapse findings, accurate communication between providers, and reliable pre and post-treatment comparison points. The POP-Q system measures nine locations on the vagina and vulva in relation to the hymen. (Figure 1) All measurements, except for the measurement of total vaginal length, are taken with the patient performing a maximal Valsalva maneuver with an empty bladder. Also, if the full extent of prolapse is not appreciated with the patient in the supine or lithotomy position, the patient is examined in the standing position. The POP-Q measurements are then used to assign a stage of prolapse (from 0-IV) for each patient according the most advanced site. (Table 1)

A bimanual examination should then be performed to assess the uterus (cervical length, uterine size and contour, uterine mobility, and the quality of uterine supports) and adnexa. The examiner may also palpate the pelvic levator ani muscles. Muscle tenderness, baseline muscle tone, as well as ability and strength of voluntary contraction that is, the "Kegel squeeze", may be determined at this time. It is important to note if a woman can locate and contract her pelvic muscles. If she cannot, a program of pelvic muscle strength training by a physical therapist would be advised, as an unsupervised course of Kegel exercises will likely not be beneficial. Rectal examination is performed to assess rectal sphincter tone and its voluntary contraction. Rectal prolapse and defects in the rectovaginal septum may be appreciated more fully at this time as well.

Adjunctive testing

Women with prolapse who are seeking treatment should undergo bladder testing to unmask any bladder dysfunction. Occult stress incontinence and voiding dysfunction are often seen in women with prolapse.[7,8] Bladder testing of either simple or complex cystometry, and flow studies with prolapse extended and reduced will reveal such conditions. Reduction will mimic bladder and urethral function once prolapse is repaired or resupported. Instruments such as a single blade speculum, large cotton Q-tips (scopettes), ring forceps, or pessary can be used to reduce the prolapse.

Imaging such as dynamic pelvic floor MRI and defecography are not routinely necessary in women with pelvic organ prolapse, but can clarify etiologies of bowel, bladder or sexual dysfunction; thus they may be useful in formulating management recommendations in a select group of women. Cystoscopy may be needed as well.

Nulliparity does not protect against prolapse...

Treatment

Indications for treatment

Management of pelvic organ prolapse is based upon symptom bother. If the presence of pelvic prolapse is not sufficiently bothersome to the patient to warrant active intervention, watchful waiting is reasonable. Education and reassurance regarding anatomy, symptom progression, and possible treatment options is recommended for these patients. Patients who choose to have no intervention for their prolapse should be encouraged to follow symptom-directed therapy, pelvic floor muscle training, and be monitored for progression of prolapse. As described in the Diagnostic section, a quantitative measurement of prolapse by the POP-Q staging system allows for subsequent comparison of prolapse progression.

When bladder or bowel evacuation becomes compromised or impossible secondary to prolapse, emergent treatment is needed for reduction of the prolapse. If bladder emptying is compromised, resulting in increased post void residuals, patients should be counseled on the possible risk of recurrent urinary tract infections and upper urinary tract damage. In addition, the protruding vaginal epithelium is at risk for erosion and/ or abrasion with advanced prolapse; it can rarely become infected.

If a woman chooses to move forward with active management of her prolapse, she should be counseled about non-surgical and surgical treatment options. Treatment goals should be outlined and patient expectations understood.

Non-surgical treatment

Non-surgical management is ideal for patients who wish to avoid surgery or who present with medical conditions that make them poor surgical candidates. Pessary use is the only specific non-surgical treatment available, but pelvic floor muscle training and symptom directed therapy might reduce the progression of prolapse symptoms.[9]

Symptom directed therapy

Symptom -directed therapy is aimed at altering specific symptoms that are bothersome to the patient and which may contribute to the progression of prolapse. Many practitioners utilize symptom- directed therapy as an adjunct to surgical management in an effort to optimize surgical outcome.

Patients who complain of incomplete evacuation of stool, or the need to splint during bowel movements, should undergo a complete gastrointestinal evaluation, diet and bowel history. If no GI pathology is diagnosed, bowel habits should be regulated to prevent straining and promote regular evacuation. Increasing water and fiber intake should be reinforced. Addition of osmotic laxatives may be done as necessary.

Incomplete bladder emptying, or symptoms of urinary frequency and urgency may be controlled with such methods as timed voids or fluid intake alteration. A voiding diary is helpful for patients to record their daily intake and voiding patterns.

In general, exercise and weight loss

are not proven to decrease prolapse symptom progression, but are encouraged for overall health.

Pelvic floor muscle training

Commonly called Kegel exercises, pelvic floor muscle training is aimed at increasing the strength and endurance of the pelvic muscles. The pelvic muscles, specifically the levator ani muscles (pubococcygeous, ileococcygeous, puborectalis), act in concert with ligaments and connective tissue to support the pelvic organs. Strengthening these muscles therefore theoretically increases the support of these organs. There is no direct evidence that pelvic floor muscle exercises prevent or treat pelvic organ prolapse; however. they are effective for urinary and fecal incontinence and may be beneficial for prolapse.[10] Pelvic floor muscle exercises, like symptom directed therapy, can be used as an adjunct to surgical management. There are virtually no adverse effects of pelvic floor muscle exercises; however, the patient must be willing to invest the time. Few women effectively locate and contract their pelvic muscles when asked to during a vaginal examination. Therefore, independent unsupervised exercise of these muscles may not be as beneficial as supervised exercise with a physical therapist.

Pessary Use

A vaginal pessary is a removable device placed in the vagina to support areas of pelvic organ prolapse. A variety of pessaries are available, made of rubber, plastic, or silicone-based materials. (Figure 2) Like all types of non-surgical management, pessary use is aimed at decreasing symptom frequency and severity. They are a choice of therapy in women who have medical contraindications to surgery or debilitated, and for any woman who desires to avoid surgery. Pessaries may also be used before implementing a surgical plan to assess symptom resolution or to document occult urinary incontinence with reduction of prolapse.

Pessaries are available in a variety of shapes depending on the type of prolapse and vaginal configuration. Broadly, there are support and space-occupying pessaries, and a pessary is fitted to each individual patient. The pessary should be both stable and comfortable, and patients should be able to urinate and defecate without difficulty. Up to 75% of women can be successfully fitted with a pessary; unsuccessful fitting is associated with short vaginal length and a wide introitus.[11] Approximately 90% of women who are successfully fitted with a pessary are satisfied at 2 months.[12]

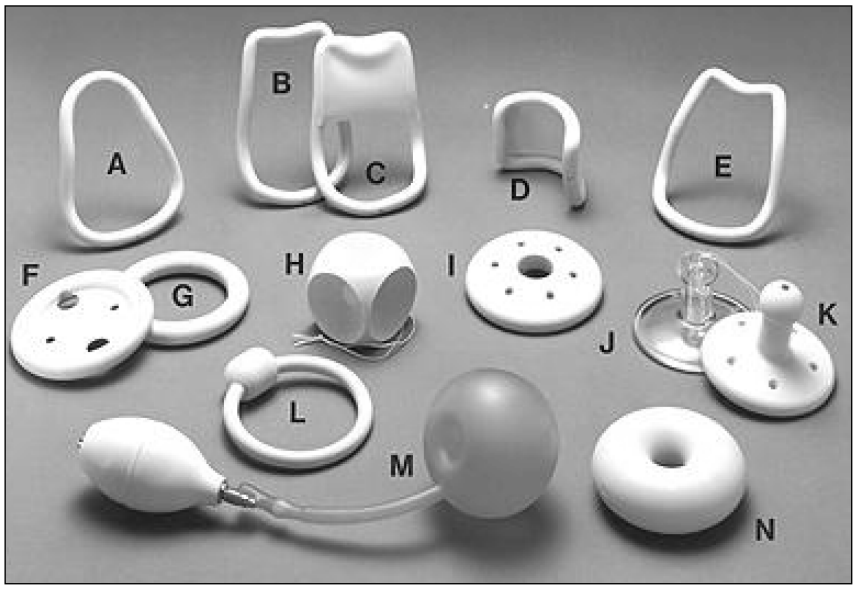

Figure 2: Types Of Pessaries: A) Smith; (B) Hodge; (C) Hodge with support; (D) Gehrung; (E) Risser; (F) Ring with diaphragm; (G) Ring; (H) Cube; (I) Shaatz; (J) Rigid Gellhorn; (K) Flexible Gellhorn; (L) Incontinence ring; (M) Inflatoball; (N) Donut.
(Image from UptoDate)

Patients can insert and remove some types of pessaries on their own, or they may return to their provider for insertion and removal approximately every three months. At the office visit, the provider inspects the vagina for erosion. Maintenance with estrogen cream and/or Trimo-San ® (Milex Inc, Chicago, IL) is recommended to maintain vaginal health during pessary use.

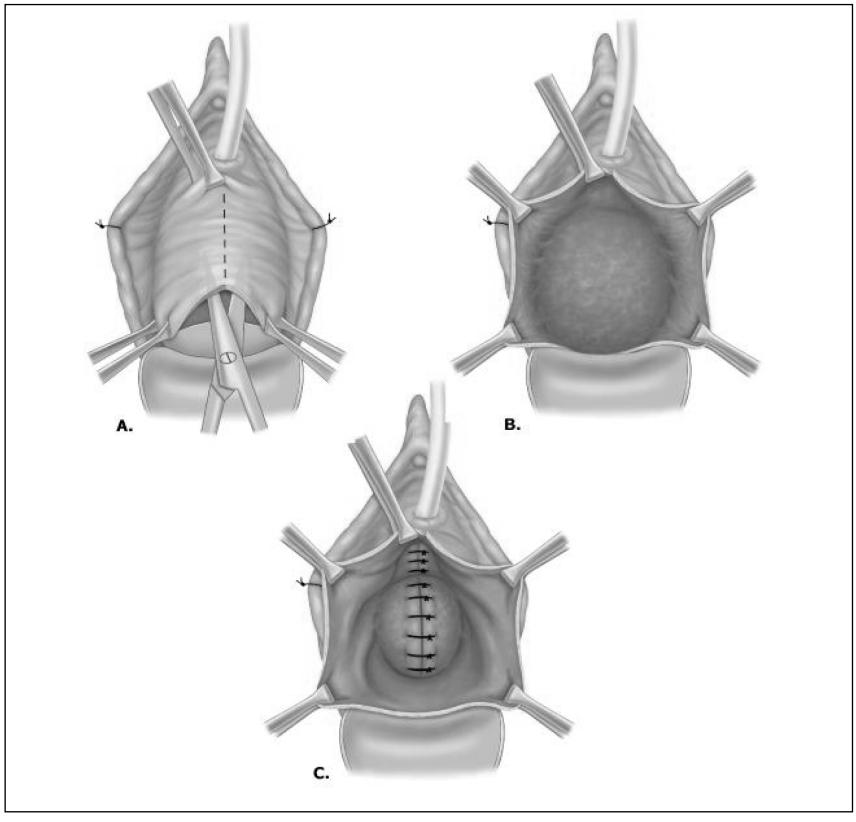

Figure 3. Anterior vaginal colporrhaphy (Image from UpToDate)

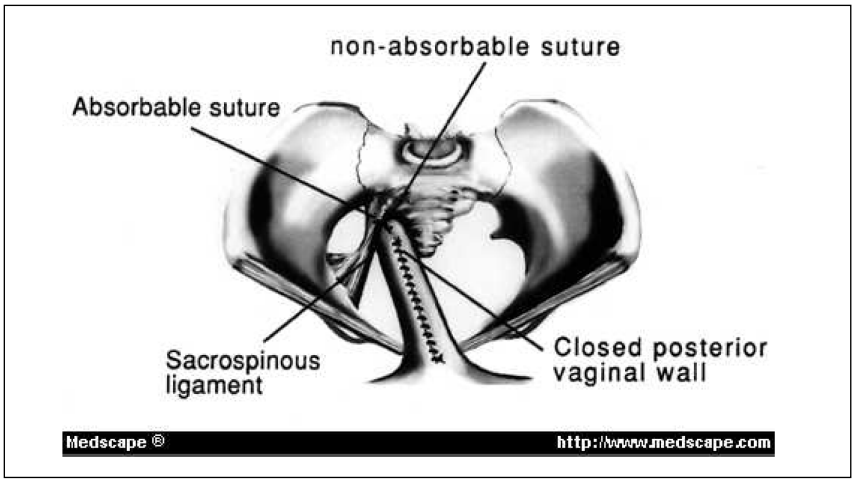

Figure 4. Sacrospinous Ligament Fixation

The main contraindication to pessary use is inability to follow up for treatment monitoring, which would result in pessary neglect and subsequent incarceration and fistula. Relative contraindications to pessary use are severe vaginal atrophy, active vaginitis, and persistent vaginal erosion with pessary use, which may necessitate periodic discontinuation of the pessary. Vaginal neoplasm should be ruled out in these cases of non-healing lesions.

Surgical treatment

The primary aim of prolapse surgery is to improve prolapse symptoms and bowel, bladder or sexual dysfunction associated with the prolapse. Surgery is aimed at either reconstructing the vagina or obliterating the vagina to achieve symptom relief.

Obliterative procedures

For patients who do not desire vaginal function, or who are at high risk for complications during reconstructive procedures, an obliterative procedure, or colpocleisis, may be an appropriate treatment choice. This is performed transvaginally, and can be done with or without a uterus in place. Recurrence rates for colpocleisis are low; however, this may be due to self-selection of a patient population that has a limited life span and activity level.

Reconstructive procedures

Theoretically, prolapse is caused by a disruption and dysfunction of one or both of the natural anatomic supports: connective tissues and muscles. Reconstructive surgery of the vagina repairs or replaces the connective tissue supports, restoring structure and function of the vagina. Reconstructive surgery may use the patient's endogenous support structures, or may attempt to replace deficient support with a graft material. Approaches to pelvic reconstructive surgery for prolapse include vaginal, abdominal, and laparoscopic, or a combination of the above. Depending on the location of prolapse and prolapse symptoms, each compartment of the vagina (anterior, apical, posterior) may be addressed with a specific approach. In addition, concomitant surgery may be planned for the anal sphincter and/or bladder neck. As comparable data for prolapse operations are poor, surgical route is determined based on the type and severity of prolapse, surgeon preference, and desired outcome.

One of the most important complications to remember when counseling a patient regarding reconstructive surgery is anatomic failure, or recurrence. All patients who undergo prolapse surgery must understand that each approach is associated with a recurrence rate, and though lifestyle factors can be modifiable, inherent connective tissue and muscle damage likely contributes to failure.

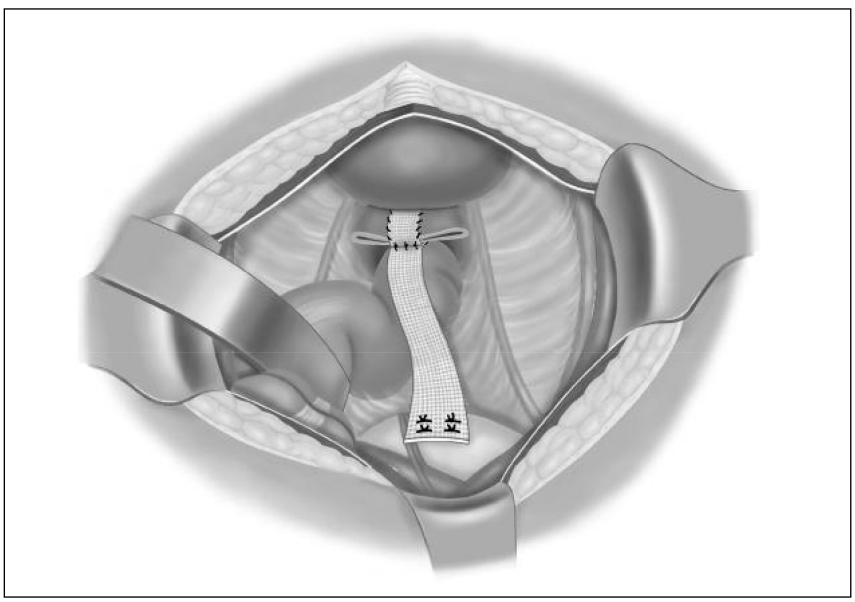

Figure 5: Abdominal Sacrocolpopexy (Image from UpToDate)

Anterior vaginal repair

Anterior vaginal prolapse has traditionally been repaired transvaginally with an anterior colporrhaphy. This entails exposure and plication of the patient's vesicovaginal connective tissue in the midline. (Figure 3) Graft material may be used in addition to or instead of the plication. The aim of graft material in this compartment is to effectively augment the vesicovaginal connective tissue, and therefore theoretically increase anatomic success and outcome. Paravaginal repair of the anterior vaginal wall may be approached either transvaginally, abdominally, or laparoscopically. This is aimed at reattaching the lateral vaginal sulcus to the arcus tendineous fascia pelvis. There are poor data comparing the above surgical approaches. Therefore a surgeon must consider patient presentation, surgeon preference, and concomitant surgeries when choosing an approach to anterior vaginal wall repair.

Posterior vaginal repair

Posterior vaginal prolapse has traditionally been repaired transvaginally with a posterior colporrhaphy. Like an anterior

colporrhaphy, this entails exposure and plication of the connective tissue supports of the rectum, or rectovaginal connective tissue. This can be done with either a site-specific or midline repair, and may be augmented with graft use. Procedures that are commonly combined with a posterior vaginal repair include a levator muscle plication and/or a perineorrhaphy. Perineorrhaphy is usually carried out when there is a separation of the perineal muscles, and is often used to restore the natural posterior deflection of the vagina in the pelvis. Colorectal surgeons will often approach posterior vaginal prolapse transanally.

Vaginal apical repair

Apical prolapse of the vagina includes uterine prolapse with or without small bowel (enterocele) and vault prolapse (when the uterus is absent), which typically includes small bowel. Hysterectomy alone does not repair prolapse of the vaginal apex. It is usually performed by pelvic reconstructive surgeons to gain vaginal access to structures from which to suspend the vagina. Thus, a vaginal vault suspension procedure must be performed with a hysterectomy for apical prolapse.

There are several vaginal approaches to apical prolapse. Each re-suspends the vagina by using strong ligaments or fascia. A sacrospinous ligament fixation is traditionally performed after removal of the uterus, and entails attaching the vaginal apex to the sacrospinous ligament. (Figure 4) Uterosacral ligament suspension is traditionally performed after removal of the uterus when done from a vaginal approach, or may be performed abdominally or laparoscopically with the uterus removed or in place. The surgeon performs uterosacral ligament suspension by attaching the vaginal apex to the uterosacral ligament remnants at the level of the ischial spines bilaterally. Though not the traditional approach, there are reports of sacrospinous ligament fixation and uterosacral suspension being approached vaginally with the uterus in place. Vaginal approaches to apical prolapse are likely similar with respect to anatomic outcome and recurrence rate.[13] Sacrospinous ligament fixation is extraperitoneal, but may have increased risk of vascular and nerve injury, while uterosacral suspension is intraperitoneal and therefore may carry a risk of bowel or ureteral injury, and may be more challenging in post-hysterectomy vault prolapse.

Abdominal and laparoscopic approaches to apical prolapse can be performed with or without the uterus in place. Surgeons who perform abdominal sacral colpopexy use graft material attached to the anterior and posterior vaginal apex to suspend the apex to the anterior longitudinal ligament of the sacrum. (Figure 5) This can be done with the uterus in place, called a hysteropexy.. Uterosacral ligament suspension can also be approached abdominally, with or without the uterus in place. Success rates of the few trials available for comparison of vaginal and abdominal approaches to apical prolapse tend to favor abdominal sacral colpopexy, though complications of abdominal entry and graft use need to be weighed when considering how to approach each patient.

Addressing concomitant symptoms

Symptoms of urinary, bowel, and sexual dysfunction must be discussed with patients before surgery, and resolution of such symptoms may or may not occur with surgical anatomic replacement of the pelvic organs. If a woman demonstrates stress incontinence with pessary support of the pelvic organs preoperatively, she is at a higher risk of having post-operative stress incontinence, and may benefit from an anti-incontinence procedure.[14] Fecal incontinence may be addressed with an anal sphincteroplasty at the time of surgery.

Summary

Pelvic organ prolapse can encompass a range of disorders, from asymptomatic, altered anatomy to complete eversion of the vagina and may present with associated urinary, defecatory, and sexual dysfunction. Patient symptoms are important to elicit, because many patients with prolapse are asymptomatic. Ascertaining patient treatment goals is necessary when discussing options for management, and patients can choose from conservative, noninvasive treatment and prevention to surgical reconstruction. As comparable data for prolapse operations are poor, surgical route is determined based on the type and severity of prolapse, surgeon preference, and desired outcome.

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Physical Therapy for Pelvic Floor Dysfunction

Wendy Baltzer Fox, PT, DPT GCS

Although the field of physical therapy began during the polio epidemic, the sub-discipline of Women's Health Physical Therapy[1] is only approximately 30 years old. Until recently, specialized training in women's health for physical therapists was available only on a post-graduate level. The American Physical Association sanctioned Board certification[2] for the first time in 2009. Those who pass the exam will be deemed to have professional expertise in the management of urinary incontinence, pelvic pain, pregnancy-related pain, lymphedema that occurs following surgery for breast cancer, and pelvic pain.[3]

Pelvic floor muscle dysfunction or chronic pelvic pain are not normal consequences of the aging process. For example, vaginismus may occur in the teen years when girls attempt to use tampons and/or during their initial gynecological examination. Pregnancy may be accompanied by bowel and/or bladder problems as hormonal changes result in support dysfunction or muscle weakness. These changes may also occur along the aging continuum as a consequence of decreased muscle use and decreased activity levels. This article will discuss the physical therapy management of women who present with pelvic floor dysfunction or pelvic pain.

There are two main findings during a physical therapy examination for women with pelvic floor muscle disorders: supportive dysfunction and hypertonus dysfunction. Supportive dysfunctions occur as a result of the loss of nerve, muscle, ligament, or fascial integrity of the pelvic floor muscles resulting in weakness and laxity. Weak supportive dysfunctions could be caused by injury incurred during childbearing or gynecologic surgery, chronic constipation, chronic coughing, obesity, or hormonal changes. A hypertonus dysfunction can cause symptoms of pain in the abdominal area, back, or vulvar region. Patients may report burning, itching, dyspareunia, urinary urgency and leakage, or constipation. Interestingly, both supportive and hypertonus dysfunction contribute to bowel and bladder incontinence, pelvic pain or pressure, and back pain.

Evaluation of Pelvic Floor Muscle Disorders

When a patient is referred to physical therapy, the typical management process includes examination, evaluation, diagnosis of impairments, and determination of prognosis and interventional plan of care.[4] Impairments may include weakness, pain, decreased range of motion, and functional limitations. Interventions may include therapeutic exercises[5] for strengthening, education of behavioral changes, orthotices, biofeedback and electrical stimulation.

A physical therapist will complete a thorough examination before designing an interventional plan. Patient history will include general demographics including primary language and race/ethnicity so that there is no language barrier that can impede treatment[6] and all verbal and written instruction will be appropriate for the patient. An understanding of ethnic beliefs and traditions may alter the treatment approach and dictate the education component. In some cultures, discussion of female pelvic anatomy is limited, even taboo,. The patient's occupation may indicate the need for behavioral modifications. For example, jobs that require prolonged standing or sitting require postural awareness, particularly with patients with chronic pelvic pain. Functional status, activity level, ability and willingness to participate and to be compliant are important to note when setting patient goals. An elder's living environment[7] may be a cause of incontinence if functional mobility or the need for an assistive device such as a walker impedes toileting. Impaired mobility, combined with urinary urgency and frequency, are safety concerns. A bedside commode at night can enhance safety and promote continence.

Determination of variables such as the onset of the current condition, what prompted the patient to seek medical consultation, past interventions or surgeries, as well as the expectations or goals of the patient, will guide the plan of care. A review of all medications is necessary. For example, diuretic use by the patient needs to be considered in the behavioral management of urinary incontinence, particularly in timing activities and overall outcome expectations. A review of all laboratory and diagnostic testing (e.g. urodynamics, cystoscopy, defecogram, MRI) are important as well as bowel, bladder, nutrition and hydration diaries. The patient's medical history, as well as current medical status, are required to understand the connection of pre-existing conditions and outcome. An underlying neurological condition may dictate a course of management rather than a resolution of the urinary concern. A patient's perception of her general health; psychological issues including anxiety, depression, impaired memory; and habits including smoking and exercise all are considered in forming a physical therapy plan of care.

Following history and systems review, additional PT tests and measurements are completed. These may include assessment of the pelvic floor[8] with external observation for anomalies, skin integrity, palpation for tender points or trigger points, pain location, neurological tests, strength grading by manual muscle test of superficial and deep muscles. Examination also includes the evaluation of endurance, relaxation, and contraction speed of the pelvic muscles. **Surface electromyography (EMG)** is used to assess the muscle tone. The patient's breathing pattern at rest and during activity would be observed. Breathing dysfunction is commonly seen with pelvic floor dysfunction; the increased intra-abdominal pressure and straining contribute to the pelvic floor dysfunction. More tests[9] may include musculoskeletal assessment of posture, spinal flexibility, abdominal and back strength/stability, as well as assessment of lower extremity strength, range of motion and length. A relatively new technique, real time ultrasound, is used to observe muscle function during ac-

tivities, as well as a means to provide biofeedback as a treatment.[10] As examination progresses, identification of additional impairments would require referral to other medical practitioners. The physical therapy plan of care will outline a specific physical therapy diagnosis.

Treatment of Pelvic Floor Muscle Disorders

Direct interventions prescribed by physical therapists are evidence-based and include the following elements: coordination of care, communication and documentation, patient education and direct intervention. The primary intervention prescribed by physical therapists has always been therapeutic exercise.[11] These include core strengthening of abdominal muscles, postural and pelvic floor muscles. Breathing and relaxation exercises are typical key components for every patient. Relaxation involves the quieting of the autonomic nervous system and includes visualization,[12] soft tissue mobilization, heat modalities and positioning. Scar management (abdominal or perineal) includes soft tissue mobilization, application of heat or cold, and therapeutic ultrasound. Manual therapy techniques include myofascial release, trigger point release, soft tissue mobilization and massage.[13] Active stretching and specific tissue stretching may be completed with vaginal dilators.

Methods of strengthening may include electrical stimulation, muscle re-education using biofeedback techniques, or instruction in the use of vaginal weights.[14] Biofeedback involves the use of external or internal sensors that record levels of muscle activity that are displayed on a computer as the patient performs exercises. This visual technique can provide motivational support as it increases the awareness of correct muscle contractions in various positions. Electrical stimulation is used to correct incoordination. In the treatment of overactive bladder electrical stimulation is used to inhibit and decrease unstable detrusor contractions. Electrical stimulation is contraindicated for patients for whom there is urinary retention or post void residual volume > 200 cc. Electrical stimulation is also contraindicated for women during pregnancy and may not be effective with patients who are obese.

Summary

This article has summarized the assessment of a woman with pelvic floor muscle dysfunction or pain complaints and has briefly described the interventions used to treat women with these concerns. The American Physical Therapy Association explains: : "As a woman in today's world, you enjoy a life of many choices. The choices we make will determine the way we use our body through the decades. A physical therapist will be there for you as you progress through all stages of your life."

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Minimally Invasive Approaches To Pelvic Reconstructive Surgery

Charles R. Rardin, MD

The term "minimally invasive surgery" generally represents the effort to reduce the impact of surgery on the patient, both in terms of incision size and location, as well patient discomfort and recovery of normal health status. The recognition of the importance of these characteristics has pervaded all aspects of surgery – generally speaking, there is widespread acceptance of the role of minimization of incision size, use of self-retaining retractors, and other measures to improve the patients' overall experience and recovery, and these might be considered efforts to reduce the invasiveness of surgery in general. For the purposes of this article, two main categories of surgery will be considered: laparoscopic (and robotic) surgery, and trocar-based surgical kits and techniques.

The advent of laparoscopy for gynecologic surgery was greeted with much enthusiasm; the potential advantages in decreased postoperative pain[1], rapid recuperation[2], decreased adhesion formation[3] and preferable cosmetic result led to a rapid increase in the rate of tubal ligation. The utility of the laparoscope as a diagnostic tool was quickly realized, and it enjoyed popularity in the diagnosis and treatment of chronic pelvic pain and endometriosis, as well as in surgical sterilization.

As the tool of the laparoscope has been applied to more advanced surgical procedures, these advantages to the patient have remained significant, and other advantages were realized. The microscopic visualization of the laparoscope has improved identification and avoidance of the vascular structures that have complicated retropubic procedures. Additionally, the pneumoperitoneum used during laparoscopy provided some measure of tamponade, again reducing the nuisance of venous oozing during the retropubic dissections. Finally, patient satisfaction with the laparoscopic approach to urogynecologic procedures is favorable.[4]

Laparoscopic Techniques

Laparoscopic Burch colposuspension procedure

The retropubic colposuspension, along with suburethral slings, has become, to many, the gold standard for treatment of urodynamic stress incontinence due to bladder neck hypermobility without intrinsic sphincter deficiency.[5] The Burch procedure, or more accurately, the Tanagho modification of the Burch procedure,[6] was performed laparoscopically and described by Vancaillie in 1991[7], with publication of a case series soon afterwards.[8,9] The laparoscopic advantages of visualization, hemostasis, and quick recovery for the generally healthy population helped to make this a popular procedure for adaptation to the laparoscopic approach.

Technique:

Similar to the open technique, the laparoscopic Burch procedure involves retropubic dissection, clearing of the paraurethral and paravesical fascia, placement of two sutures on each side (one at the urethrovesical junction, the other at the midurethra, at least 2 cm lateral to the urethra itself), which are then suspended from Cooper's ligament ipsilaterally (Figure 1) Suture placement in the paraurethral tissue is facilitated by elevating the tissue with the surgeon's finger in the vagina. Practitioners vary widely in their techniques of assessing the right amount of bladder neck elevation; however, the data have shown that correction of hypermobility is a requirement for successful outcome.[10] Overcorrection, though, can lead to voiding dysfunction; therefore, suture bridges are left to prevent overcorrection. It is also advisable to close any peritoneal incision to prevent incarceration of bowel within these suture bridges.

With the popularity of minimally-invasive slings, the popularity of the Burch procedure has waned; the skillset required and operative time tend to favor the newer generations of slings, and success rates of these slings are at least as high or higher.[11] Interest in the Burch procedure was regenerated, to some degree, by the CARE trial, in which patients undergoing open sacrocolpopexy for vaginal vault prolapse were randomized to receive, or not receive, a concomitant Burch procedure, regardless of preoperative urodynamic findings.[12] Patients who underwent the Burch procedure were half as likely to report postoperative stress incontinence as their counterparts. Many practitioners who have developed

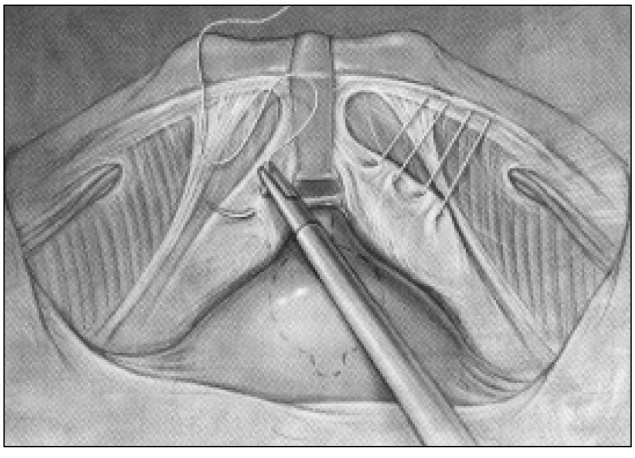

Figure1. Burch Colposuspension

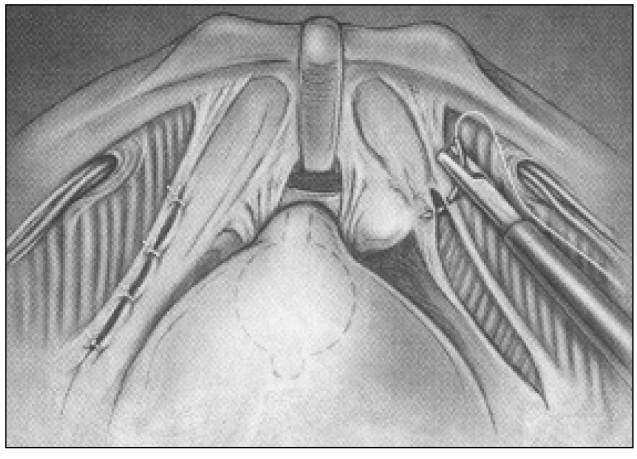

Figure 2. Paravaginal Defect Repair

skills in laparoscopic sacrocolpopexy are adding laparoscopic colpopsuspension in light of these findings.

Laparoscopic repair of a cystocele paravaginal defect

Although the traditional repair of cystocele (colporrhaphy) has involved the central plication of the pubocervical fascia, the idea that anterior compartment defects can be lateral (paravaginal), as well as central, was first published nearly a century ago.[13] (Figure 2) As the surgical reattachment of pubocervical fascia to the arcus tendineus of the fasciae pelvis (or "white line" of the pelvic sidewall) is more challenging than simple colporrhaphy, this idea lay dormant until the 1970s, when Richardson postulated that the majority of cystoceles are a result of this lateral disruption.[14] More recent anatomic studies have confirmed that lateral defects are usually present in cases of anterior compartment prolapse and bladder neck hypermobility.[15] Needless to say, a central repair for a lateral defect may reasonably be expected to yield suboptimal success rates.

Technique:

The laparoscopic paravaginal repair, as with the Burch procedure, starts with retrograde filling of the bladder, supravesical peritoneal incision, and dissection of the retropubic space. As the goal is the reattachment along the full length of arcus tendineus along the pelvic sidewall, the dissection must be carried out more laterally than is required for the Burch. For this reason, the peritoneal incision is usually taken beyond the medial umbilical folds; care must be taken to avoid injury to the inferior epigastric vessels. Similarly, identification and protection of the obturator neurovascular bundles is crucial.

After the paravesical fascia is cleared with gentle dissection, a series of permanent sutures is used to reattach the fascia to the obturator internus muscle on each side. The appearance of the arcus tendineus along the sidewall may be variable; one study showed that the condensation of fibers known as the "white line" are often avulsed and thus attached to the paravaginal fascia, rather than intact along the pelvic sidewall..[15] Thus the surgeon may not always have the clear visual cue along the sidewall. Whether or not the arcus is readily visible, its original location between the ischial spine and the inferior edge of the pubic ramus can be located by palpation under laparoscopic observation. The surgeon will appreciate the fact that, in this approach, the tissues being sutured together are adjacent; in the vaginal paravaginal repair, the vaginal sutures must be placed with the tissue everted, and thus distant from the targeted area of reattachment.

Laparoscopic repair of vault prolapse – Uterosacral ligament suspension

While uterosacral ligament vault suspension has been well described vaginally;[16] the abdominal or laparoscopic approaches are also feasible. However approached, the technique involves identifying the intact remnants of the uterosacral ligaments, at or above the level of the ischial spines, which are then sutured to the ipsilateral aspects of the posterior and anterior fascia of the vaginal vault. It should be noted that, in the case of fascial attenuation, an enterocele sac is likely to be found between these intact anterior and posterior fasciae. The vaginal approach can be made difficult by the challenge of identifying the proximal ligament remnants. In addition, the suture, if permanent (as many advise), must be tied extraluminally, which can be difficult; alternatively, an absorbable suture can be tied within the vaginal lumen. The possibility of ureteral compromise, reported to be as high as 11% with the vaginal approach,[17] necessitates the use of intraoperative cystoscopy. The visualization of the ureters throughout their pelvic course that laparoscopy can provide may be an additional benefit.

Technique:

Laparoscopic uterosacral vault suspension can be performed at the time of hysterectomy, or remotely from hysterectomy, in the case of vaginal vault prolapse. After the ureters and the rectum are identified, the uterosacral ligaments are identified at the level of the ischial spines. Permanent suture is then brought through the ligaments at this level; tensiometry studies have demonstrated that laparoscopically-placed sutures in the uterosacral ligaments have as much tensile strength as vaginally-placed sutures.[18] With the proximal uterosacral ligament thus captured, the sutures are then brought ipsilaterally through the full thickness of the posterior and anterior vaginal walls (excluding epithelium) at the cuff. (Figure 3) The attenuated enterocele sac that may lie at the apex, between anterior and posterior vaginal wall fasciae, can often be visualized with the use of the vaginal probe. Some surgeons advocate the excision of this attenuated tissue sac; whether or not it is removed, the supporting sutures should be placed beyond it, on the intact fasciae. Advocates of this procedure point out that it is restorative of the original anatomic support and vaginal axis.

Sacrocolpopexy

The uterosacral vault suspension procedure described relies on the presence and identification of useful uterosacral remnants; it also depends on vaginal sutures at the vault apex for long-term success. In addition, for the reasons outlined above, the vaginal apex, in the presence of an enterocele, may represent the most attenuated segment of the entire vagina. Vaginal techniques of vault suspension, including the vaginal version of the uterosacral vault suspension, as well as the sacrospinous ligament fixation, may be susceptible to the same vulnerability. For these reasons, many surgeons prefer the sacrocolpopexy using permanent materials. Although not anatomic in the strictest sense, it has been shown to yield a vaginal axis that is closer to normal than that found after vaginal sacrospinous liga-

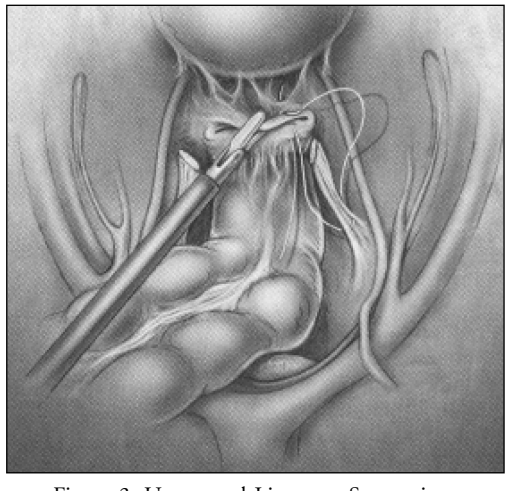

Figure 3. Uterosacral Ligament Suspension.

ment fixation.[19] It also permits the placement of multiple suture points along the anterior and posterior vaginal walls, distributing tension over a wider area and decreasing the likelihood of suture pullout. In its abdominal version, it has been demonstrated to have a remarkably low recurrence rate over the long term.[20]

Technique:

A peritoneal incision over the sacral promontory is made and the underlying anterior longitudinal ligament of the sacrum is visualized. Laparoscopically, the pneumoperitoneum facilitates dissection of the retroperitoneal areolar tissue, and the microscopic visualization allows easier identification of the sacral vessels which, if injured, retract into the sacrum and results in catastrophic bleeding. It should be noted, however, that the left common iliac vein, which lies just below and inferior to its arterial counterpart, can be compressed by the pneumoperitoneum, and therefore inadvertently injured. For this reason, the dissection over the sacral promontory should be kept slightly to the right of the midline. This incision is carried down into the pelvis, remaining slightly to the right of midline (to avoid mesenteric vasculature) but well medial to the right ureter. This incision allows for the retroperitonealization of the mesh after the suspension.

After the vaginal vault is prepared by dissecting peritoneum off of the anterior and posterior aspects (and the development of the vesicovaginal and rectovaginal spaces, respectively), a Y-shaped graft is affixed to both sides of the vaginal vault. At that point, the main arm of the Y is affixed directly to the anterior longitudinal ligament of the sacrum, with a series of permanent sutures. (Figure 4) Care should be taken to avoid the middle sacral vessels, and tools to control for presacral bleeding should always be available. The proximal ends of the graft are then affixed to the sacrum, with care taken to avoid tension. After excess graft is trimmed, the peritoneum is closed over the graft to reduce the likelihood of bowel incarceration or adhesion.

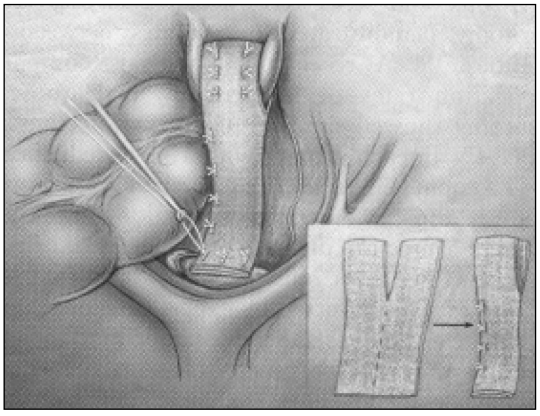

Figure 4. Abdominal Sacrocolpopexy

Although long-term or prospective data regarding the effectiveness of the laparoscopic approach is limited, several reports support the benefits of minimally invasive techniques in the execution of this form of vaginal support. As has been demonstrated in many other arenas, laparoscopic sacrocolpopexy in the hands of trained surgeons yields similar efficacy while enhancing hemostasis and reducing postoperative pain and hospitalization.[21] [22] [23] Here, as before, the principle that laparoscopy is a means of access, and that the steps of the procedures should be identical to that of the open technique, are of utmost importance.

Other Reconstructive Procedures

Rectocele

Variations on the above procedures have been performed and described for the treatment of similar conditions. Laparoscopic rectocele repair has been described, in a procedure which involves the extended dissection of the rectovaginal septum all the way to the perineal body, and either plicating the levator musculature,[24] or suturing mesh material in place.[25] In principle, this approach to mesh-based repair of the posterior wall may enhance outcomes by eliminating vaginal incisions, which are thought to be contributory in the development of problematic mesh erosion.

Uterine Preservation

In addition, several studies have called into question the practice of routine extirpation of prolapsed uteri.[26,27] Patients interested in uterine preservation value the availability of this choice, and the elimination of the hysterectomy decreases blood loss, hospitalization, and other complications[28]. Clearly, patients must understand that future pregnancy and delivery may have deleterious effects on the repair, cervical surveillance remains necessary, and hysterectomy may be needed in the future. This option continues to be valued by some women and some practitioners. Both the uterosacral ligament suspension, and the sacrocolpopexy using mesh, can be performed for the treatment of uterine prolapse among women who desire uterine conservation; the techniques are very similar to those described for vault prolapse above.

Robotics in Pelvic Reconstructive Surgery

The da Vinci robotic surgical platform (Intuitive Surgical, Inc., Sunnyvale, CA) represents a significant technical advancement in the instrumentation for laparoscopic surgery. Sitting at a console, the surgeon uses controls to operate a set of robotic arms fitted with specialized instruments. The main advantages include motion scaling (converting large movements of the surgeon to very fine movements of the instruments), instruments with an additional degree of motion (known as an endo-wrist), and the enhancement of dexterity and psychomotor performance (through tremor-stabilizing algorithms). The da Vinci system also uses binocular, 3-dimensional video, enhancing depth perception. The performance of these systems in the training of residents is in the early stages. One study demonstrated a steeper (that is, more rapid) learning curve, among both experienced and inexperienced surgeons, in the performance of drills using a robotic system.[29] Another study demonstrated that laparoscopic drills were completed more quickly with the robotic system compared to traditional laparoscopy, and that novice surgeons on the robot performed as quickly, and in some cases more quickly, than expert surgeons with traditional laparoscopy.[30] The continued refinement of these systems may redress some of the deficiencies in laparoscopic training by improving skill acquisition. The robotic platform will likely increase the number of surgeons with minimally-invasive skills to treat pelvic floor defects.

Most of the literature regarding the usefulness of the robotic platform addresses its use in Urology, where its application to minimally invasive radical prostatectomy has generated significant interest. In terms of Gynecologic Oncology, the introduction of

robotics resulted in significantly lower blood loss and postoperative hospitalization, while lymph node yield remained similar.[31] Early literature supporting the use of robotics in pelvic reconstructive surgery shows promising results in applications such as sacrocolpopexy[32]

Trocar-Based Mesh Repairs

The trocar - basedTension-free Vaginal Tape suburethral sling procedure heralded the arrival of a new paradigm of reconstructive surgery, and the principles underlying its effectiveness continue to be applied to new pelvic reconstructive techniques. There are three important ways in which the TVT ® (Gynecare Ethicon, Sommerville, NJ) differs from the slings that preceded it: midurethal placement (rather than at the bladder neck), trocar-based delivery, performed blindly and with minimal dissection, and self-retaining mesh that required no anchoring or fixation. A variety of tensioning techniques exist, with the key provision that, at rest, the tape should exert no tension on the underside of the urethra. The blind passage of trocars through the retropubic space requires advanced anatomic understanding and confidence on the part of the surgeon, and is beset with a certain incidence of bladder perforation and, much less commonly, bowel and vascular injury.

Taking several principles of the TVT, de Leval introduced the transobturator sling in 2003. It is similar to the TVT in its trocar-based, midurethral placement, and self-retaining mesh materials. However, this technique passes the trocar through the obturator membrane avoiding entry into the true pelvis. This lateral approach seeks to reduce the likelihood of injury to pelvic organs or vasculature. Indeed, a meta-analysis of randomized trials between the techniques found similar success rates, with an apparent reduction in complications with the obturator approach.[33] The success of the obturator approach in cases of intrinsic sphincter deficiency has yet to be fully described, but several authors have reported lower success rates of this technique among these patients.

Finally, the concept of self-retaining mesh prostheses implanted with trocars performed for incontinence was brought to repair of prolapse. The introduction of trocar-based mesh kits- the intravaginal slingplasty (IVS ™ Tyco Corp.), the Apogee/Perigee™ (American Medical Systems, Minnetonka, MN), and the Avaulta™ (Bard, Covington, GA) and Prolift™ (Ethicon, Cinncinatti, OH) are such systems. These devices, although with some differences, share the fundamental principles of self-retaining, tension-free mesh, introduced vaginally and affixed to a variety of pelvic anatomic landmarks, to support the appropriate compartments of the vagina. These techniques are discussed in greater detail elsewhere in this issue.

Discussion

At its best, laparoscopic pelvic floor defect repair represents an alternative approach to performing established procedures; laparoscopy can offer benefits to the surgeon (improved visualization, access for multiple procedures) and patient (decreased pain, scar formation, recuperation, and improved cosmesis). Many practitioners prefer the term "minimal access surgery" to the more prevalent "minimally-invasive surgery," as, ideally, only the route of access, not the procedure itself, is changed. At its worst, laparoscopy invites surgeons to take these established procedures and to modify them, to eliminate steps and cut corners, to the point where it bears only a tenuous relationship to the original. As such, some experts have challenged laparoscopists that their patients should be consented for "experimental" surgery.[34] Similarly, dialogue and debate about the merits and concerns of trocar-based mesh prolapsed repair kits continue. While Gynecology will benefit from further investigations of outcomes of minimally invasive pelvic reconstruction, there is evidence already that these techniques are feasible and offer options in the treatment of patients with pelvic floor disorders.

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Urinary Incontinence

Vivian W. Sung, MD, MPH

Urinary Incontinence (UI) affects over 13 million people in the United States.[1, 2] Most women suffer in silence and do not seek help. Affected women may feel too embarrassed to discuss this issue with their healthcare provider or may be uninformed about treatment options.

Epidemiology and Impact of Urinary Incontinence

Prevalence estimates of UI in women range from 11%-72%.[3-5] Potential explanations for these variations include varying definitions of UI (i.e. frequency of UI episodes, degree of bother, symptom severity), differing study methods to determine UI (i.e. random survey, self-reporting, personal interview, clinical exam), the dynamic nature of incontinence symptoms, and the study population.[6] In addition, because many women feel embarrassed, it is likely that UI is overall underreported. A recent literature review estimated that 1 out of every 4 women have UI.[7]

The prevalence increases with age, ranging from 20-30% in young adult women, 30-40% in middle-aged women, and 40-50% in older women.[8] It is up to 6 times higher in younger women compared to men; older women are twice as likely to experience UI compared to older men.[1, 8] Despite these differences, women are less likely to seek help compared to men.[2] It is estimated that only 25% of women will seek care.[9]

UI leads to embarrassment, humiliation, a loss of self-esteem, social isolation and depression.[10, 11]

Direct costs are estimated to be over $16 billion (1995 dollars) per year in the US[12, 13] and over $26 billion if other costs of care are included, such as protective garments and treatment of related complications.[14] It is estimated that women with UI pay $750-$900 annually in out-of-pocket for supplies, laundry and dry cleaning. [15, 16]

Risk Factors for Urinary Incontinence

The cause of UI is likely comprised of a variety of risk factors including predisposing, inciting, promoting and decompensating factors. Predisposing factors alone may be significant enough to cause UI or increase the risk of developing UI when other inciting or promoting factors are present. Inciting factors may cause UI due to injury to the continence mechanism. Promoting factors contribute to the development of UI by continuously prolonged deterioration of the continence mechanism. These increase a woman's risk of experiencing UI. Decompensating factors are not sufficient to cause incontinence but may "tip" a woman with other risk factors towards experiencing UI. These factors may be temporary or permanent. The most well studied ones include age, obesity and parity. (Table 1)

Vaginal delivery is generally considered a major cause for the development of UI. However, the exact relationship between UI and vaginal delivery is not well understood and studies in the literature are inconsistent.[8, 17] Borello- France et al reported that in primiparous women, cesarean delivery before labor was not entirely protective against pelvic floor disorders, including UI.[18] Furthermore, in one study, half of women who reported UI had no symptoms at 1 year postpartum, but 19% of women without symptoms reported UI 5 years later.[19] Pelvic floor injury following childbirth is not always associated with UI, UI usually does not occur immediately after vaginal delivery, and women who have not experienced childbirth or have delivered by cesarean delivery may also develop UI—these facts all strongly point to additional causes.

UI can also be caused by several transient or reversible conditions. A useful mnemonic is "DIAPPERS"[20] : 1) **D**elirium or acute confusion; 2) **I**nfection (symptomatic urinary tract infection); 3) **A**trophic urethritis; 4) **P**harmaceutical agents; 5) **P**sychological disorder (depression, behavioral disturbance); 6) **E**xcess urine output (excess fluid intake, diuretics); 7) **R**estricted mobility; and 8) **S**tool impaction. Identifying a woman's modifiable factors may be the greatest opportunity for preventing UI.

Evaluation of Urinary Incontinence

The goals of an evaluation for a woman presenting with UI are to 1) provide a clinical diagnosis of type of UI; 2) determine factors that may contribute to symptoms or that may require further evaluation; 3) assess for

Table 1. Risk factors for urinary incontinence[8]

Predisposing factors	• Familial predisposition • Gender • Race • Anatomic, neurological and muscular abnormalities (i.e. spina bifida, injury to spinal cord)
Inciting factors	• Pregnancy, childbirth, parity • Pelvic surgery • Radiation therapy
Promoting factors	• Obesity • Constipation • Chronic lung disease and smoking • Neurological diseases (i.e. stroke, Parkinson's disease, depression, multiple sclerosis) • Urinary tract infection • Occupational and recreational stresses • Drugs/medications (i.e. alpha-adrenergic blockers, anticholinergic agents, diuretics, psychotropics, sedative-hypnotics, narcotics, sympatholytics, ACE inhibitors, alcohol, caffeine)
Decompensating factors	• Age • Dementia • Physical disability • Comorbidities (i.e. diabetes, vascular insufficiency, congestive heart failure) • Changes in environment (accessibility of restroom) • Drugs/medications

coexisting pelvic floor disorders such as pelvic organ prolapse or fecal incontinence; 4) establish baseline severity to aid in counseling, recommendations, and treatment effects; and 5) determine the impact of UI on quality of life.[21] The initial evaluation should include a history and general assessment, a symptom assessment, a physical examination and baseline tests.

History

Incontinence symptoms: The nature and duration of UI should be detailed, including leakage, frequency, severity and volume of urine loss, activity at the time of urine loss, sensations of urge, and how bothersome these symptoms are to the patient. A woman who leaks large volumes daily may have a greater problem that requires a more complete evaluation compared to a woman who leaks only rarely when she is doing high impact sports. Women are also asked about other pelvic floor symptoms including sensation of vaginal bulging or pressure, nocturia, hematuria, recurrent urinary tract infections, voiding problems, anal incontinence, and defecatory dysfunction.

Medical history: including prior treatments for UI and pelvic floor disorders, medical problems, medications, and mobility issues that may exacerbate UI as well as acute and reversible causes of UI.

Patient functioning: including sexual and bowel function.

Patient goals and expectations of treatment should be assessed at the initial visit. This will help to guide further evaluation.

Examination

The physical examination includes a complete evaluation of the abdomen and pelvis. Abdominal examination is performed to rule out any masses. Detailed pelvic examination is performed to assess for pelvic organ prolapse, pelvic floor muscle function, estrogen status, and to rule out pelvic masses. External genitalia are examined to evaluate for irritative or inflammatory skin conditions. Rectal examination to assess anal tone and pelvic floor function is performed. A neurological examination includes assessment of sensory and motor function of S2-4, which innervate to the pelvic organs. This also includes testing of lower extremity movement and strength.

A cough stress test to evaluate for leakage at the time of cough is helpful to confirm a diagnosis of stress incontinence. To rule out urinary retention, a post-void residual can be assessed using either direct catheterization or by ultrasonography. This should be performed within 10 minutes of voiding to prevent a false positive finding. Although based on limited evidence, the consensus is that a postvoid residual volume less than 50 mL is considered normal and a volume greater than 100-200 mL is considered abnormal.

Baseline tests

A clean catch urinalysis is recommended to exclude urinary tract infections. A negative dipstick urinalysis has a specificity of 97%-99%. In women where there is a clinical suspicion, urine should be sent for culture and sensitivity. It is suggested that for women with a long-standing history of UI who have a positive dipstick urinalysis, a urine culture should also be sent before assuming that their UI is solely due to a urinary tract infection.

A voiding diary can provide further information that can aid in the diagnosis of UI. Patients record information about voiding or UI episode time, volume, frequency, fluid intake, and activity at the time of any UI episode. Three-day diaries are as predictive as 7-day diaries in detecting abnormal voiding patterns. The voiding diary can be particularly helpful for patients who have difficulty describing their voiding or UI patterns.

The pad test is another test less commonly used that can help document the severity of UI. Patients are instructed to wear perineal pads for 24 hours, changing them when necessary. Wet pads are placed in a zip-lock bag and returned to the office within 72 hours to be measured. These weights are compared to the weight of a dry, control pad. An increase greater than 8 grams within 24 hours is considered abnormal.

Urodynamic testing

Urodynamic testing is warranted if the diagnosis is still uncertain such as: if there are discrepencies between the patient's history, voiding diary and examination, surgery is considered, the patient has an elevated postvoid residual, the patient has a neurologic condition that may complicate treatment, significant pelvic organ prolapse, or multiple prior surgical attempts at correction. A urodynamic study is defined as any test that evaluates the function of the lower urinary tract. Often, urodynamic studies incorporate a variety of measures that assess the functional parameters of the bladder, including bladder pressure, capacity, sensation during bladder filling and emptying. They can be helpful in distinguishing between different types of UI.

Briefly, complex urodynamic testing begins with asking the patient to void in a specialized commode that plots the volume of urine passed over time. This provides information on flow time, peak flow rate and time to peak flow increase with the volume voided. Next, the bladder is filled to capacity, usually with fluid at room temperature, and the bladder and urethral pressures are recorded during this filling phase. During the filling phase, the detrusor should not contract, and the pressure within the bladder should stay relatively low in a normally compliant bladder. If a detrusor contraction is noted during the filling phase, this is highly suggestive of detrusor overactivity, which causes urge urinary incontinence (See next section). At various times during the filling phase, the patient is asked to cough to evaluate for stress incontinence. A large capacity bladder with decreased sensation would indicate a hypotonic bladder. Tests of urethral function are also performed to evaluate urethral pressures during filling and emptying phases. Finally, testing usually concludes with the patient voiding while the bladder, abdominal and urethral pressures are measured.

There have been limited large scale trials assessing the utility of urodynamic testing. Recently, a multicenter randomized surgical trial for stress incontinence completed by The Urinary Incontinence Treatment Network (UITN), a National Institutes of Health-sponsored network, suggested that incontinence detection may be highly variable depending on technique.[22] Further research is needed to evaluate if specific populations will benefit from urodynamic testing to improve treatment outcomes.

Types of Urinary Incontinence, pathophysiology and treatments

The **International Continence Society (ICS)** defines UI is defined as "the complaints of any involuntary leakage of urine".[23] This definition may include symptoms (subjective, qualitative patient report), signs (physician observations of urine loss), or urodynamic study observations. The 3 most common types of UI are **stress urinary in-**

Table 2. Three most common types of urinary incontinence[23]

Type of UI	Symptom	Sign	Urodynamic finding
Stress urinary incontinence	Involuntary leakage on effort, exertion, or valsalva	Observation of urine leakage with exertion or valsalva	Involuntary leakage of urine during increased abdominal pressure in the absence of detrusor contraction
Urge urinary incontinence	Involuntary leakage accompanied by urgency	Not defined by ICS	Sudden, compelling desire to void during filling of bladder with fluid
Mixed urinary incontinence	Involuntary leakage associated with both exertion and urgency	Not defined by ICS	Urodynamic observation of both SUI and UUI during the same test

continence (SUI), **urge urinary incontinence (UUI)**, and **mixed incontinence (MUI)** a combination of SUI and UUI. (Table 2) Although there are many extant theories for incontinence, most are based on expert opinion or observational evidence which have not necessarily been proved or disproved by rigorous scientific method.

STRESS URINARY INCONTINENCE

SUI, the most common type, is the complaint of involuntary leakage with effort or exertion. It often occurs during sneezing, laughing, lifting and walking. These activities result in an increase in intra-abdominal pressure, causing the bladder pressure to exceed the maximum urethral pressure ultimately resulting in the loss of urine. This occurs in the absence of a detrusor contraction.

There are multiple theories for how SUI may develop. Two more recent complementary theories include the "integral theory"[24] and the "hammock theory".[25] The "integral theory"[24] describes 3 opposing vaginal muscle forces stretching the vaginal membrane and endopelvic fascia, which helps to secure urethral closure during increased intra-abdominal pressure. The "hammock theory"[25] theorizes that the anterior vaginal wall provides a hammock-like support for the urethra that is critical to maintain urethral closure. Injury to connective tissue supports may cause dysfunction of the continence mechanism.

Any treatment for UI should start with counseling regarding non-surgical interventions. Lifestyle interventions that may decrease SUI include weight loss in overweight and obese women[26] and decreasing caffeine intake. Well designed randomized clinical trials have shown that supervised pelvic floor muscle training ("Kegel exercises") is effective in treating or at least improving any type of UI symptoms (SUI, UUI, MUI).[27] To maximize the effectiveness of pelvic floor muscle exercises, women should be counseled on how to do them correctly, regularly and for an adequate duration. Women may need referral to a physical therapist for evaluation and supervised training sessions.

Vaginal devices (pessaries) and urethral inserts are also non-surgical options for SUI. Specialized pessaries called "continence dishes" provide support for the anterior vaginal wall and urethra to minimize SUI symptoms. Women need to undergo a pessary fitting to find the correct fit and most women (89%) can be fitted succesfully.[28] These may be a good option for women who want to avoid surgery, need to defer surgery, or complete childbearing.

Medications for SUI include alpha-agonists, which increase urethral tone have been shown to have a modest effect in small trials.[29] Serotonin and norepinephrine reuptake inhibitors are being investigated for their role for SUI.

When behavioral and pharmacologic interventions do not improve symptoms, surgery may be offered. The decision to undergo surgery should be a shared decision between the patient and her healthcare provider, as only the individual patient can weigh potential risks and benefits of surgical treatment. Although many surgical procedures have been described to treat SUI, few randomized trials inform treatment. We will review the most common procedures.

The Burch colposuspension and suburethral fascial sling are two well established procedures and for many years, they were considered to be equally effective. A recent trial conducted by the UITN randomized 655 women to undergo either a fascial sling or Burch for SUI and reported that fascial slings were associated with higher cure rates of SUI at 24 months (66% vs 49% P<.001), but were associated with more urinary tract infections, voiding difficulty and postoperative UUI .[30]

The advent of minimally invasive mid-urethral tension free slings introduced first as the "tension-free vaginal tape" or TVT™ (Gynecare, Sommerville, NJ) in 1996 marked a major shift in the surgical treatment of SUI. It is proposed that the mid-urethral sling stabilizes the vagina and urethra during times of increased intra-abdominal pressure, reinforcing the "vaginal hammock". In a randomized trial comparing the TVT to Burch, similar objective cure rates were reported at 2 years between the two procedures: 63% and 51%, respectively, assuming women who were lost to follow up as failures; or 78% and 68% respectively, carrying the last observed result forward at 2 years.[31] The procedure includes placing 2 needles vaginally through 2 paraurethral tunnels, then into the retropubic space, and exiting the retropubic space through 2 small suprapubic skin incisions. No fixation sutures are required and the sling is ultimately held in place by fibrosis. (Figure 1) Minimal dissection is required, and the procedure can be done under local anesthesia as an outpatient with minimal patient morbidity and recovery time. Other approaches to the mid-urethral sling have been developed, but there is limited randomized trial data comparing various approaches. These include

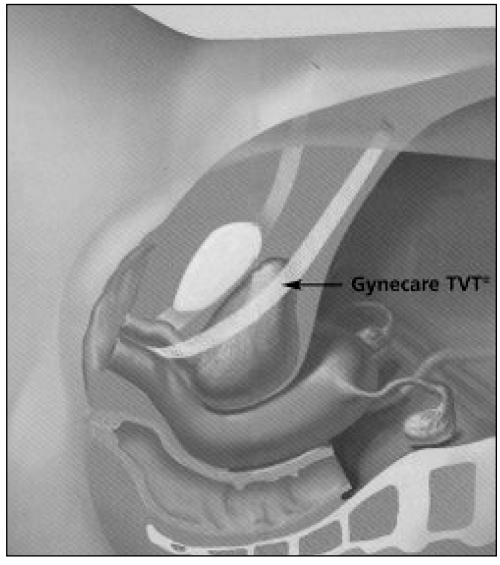

Figure1. Mid- urethral sling TVT ™ (Gynecare, Ethicon Inc, Sommerville, NJ)

the transobturator approach, "needle-less slings", and adjustable slings.

Another surgical option is injection of a urethral bulking agent, such as GAX ™collagen (Bard Inc., Covington, GA) at the bladder neck. Complications are rare and cure rates range from 20%-30%, but 50%-60% report marked improvement. Although improvement is reported to last from 3 months to years, most patients report relief ranging from 3-12 months and will require more than one injection.[32]

Urge urinary incontinence

Micturition involves the interaction of muscular, neurologic, and psychologic systems. During bladder filling, the normal detrusor relaxes to allow filling without resistance. As a person becomes aware of bladder distention, urination is voluntarily delayed through cortical centers in the frontal lobe until one can reach the restroom. The detrusor muscle then contracts in coordination with urethral relaxation in response to cholinergic signals from the pelvic nerves. This results in bladder emptying.

Disruption of these complex interactions results in UUI.

Inappropriate contraction of the bladder causes UUI, often referred to as detrusor overactivity or "overactive bladder". This may be caused by changes at the tissue and cellular levels, injury to the spinal cord, stretch injury to the pudendal nerve during labor, or neurologic problems. Many women may experience bothersome urgency and frequency without actually experiencing UUI.

The treatment includes behavioral therapy, bladder training, pelvic floor muscle therapy, and anti-cholinergic therapy. Behavioral techniques include lifestyle changes, including fluid and dietary modification, scheduled voiding, and pelvic floor reeducation. The overall goal is to train the bladder to store larger volumes of urine and control urgency by using pelvic muscles to inhibit detrusor activity. Fluid management may involve restriction if intake is high. Patients are also asked to restrict intake of known bladder irritants, including caffeinated foods and beverages.

The goal of bladder training and/or scheduled voiding is to increase the bladder's capacity. Patients are asked to schedule voiding if they have problems with frequency. The patient can also delay voiding for as long as possible when a feeling of urgency occurs by using a variety of distraction techniques or quickly contracting the pelvic muscles to inhibit voluntary bladder contraction and reinforce detrusor inhibition. As discussed, pelvic floor muscle training has been shown to improve symptoms for all types of UI.

Acetylcholine is the primary neurotransmitter involved in a detrusor contraction, therefore, anti-cholinergic medications help to reduce detrusor overactivity. There are many brands on the market. The efficacy of anti-cholinergic treatment alone ranges from 9%-56%. Because they all have the same mechanism of action, efficacy is comparable among the different types of anti-cholinergics. Many pharmaceutical companies focus on minimizing side effects by increasing selectivity for the bladder. The most common side effects include dry mouth, constipation and central nervous system effects.

Surgical treatment for refractory UUI includes sacral neuromodulation. In the past, patients with refractory UUI were limited to radical procedures such as urinary diversion or cystectomy. Sacral neuromodulation offers a less invasive alternative. The technique stimulates the sacral nerves to modulate the neural reflexes influencing bladder and pelvic floor function. It is being applied in the treatment of UUI, urinary urgency and frequency, and urinary retention. This implantable system is comprised of a lead with 4 electrodes, an extension cable, and a programmable impulse generator. The lead is usually implanted into the S3 sacral nerve root, and the impulse generator is placed in the upper buttock region. The procedure is usually done in 2 stages. After implantation of the lead, if the patient's symptoms improve (defined as $\geq$ 50% improvement in one bladder parameter), the generator is placed approximately one week later. Both stages are done on an outpatient basis. In one prospective, multi-center study, 68% of patients with refractory UUI and 56% of patients with urgency/frequency reported successful outcomes at 5 years.[33]

Conclusion

UI will affect 30% of all women at some point in their lives. It is associated with distressing psychosocial stigma and substantial medical costs to both the individual and society. Psychological and social effects of UI may prevent women from seeking attention.

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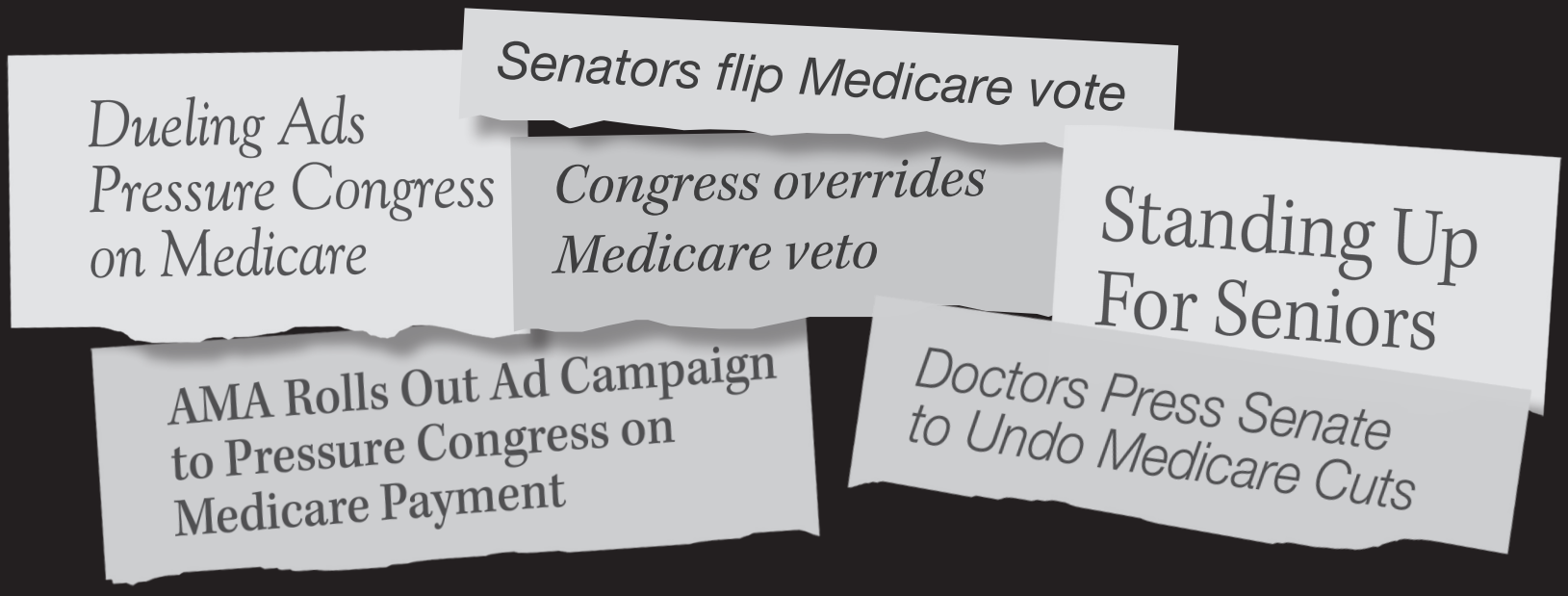
(Actual headlines in support of AMA's efforts)



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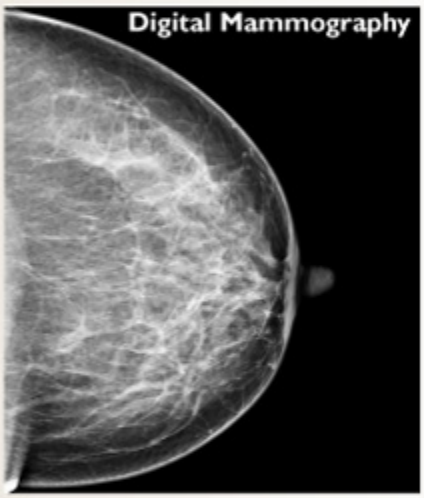

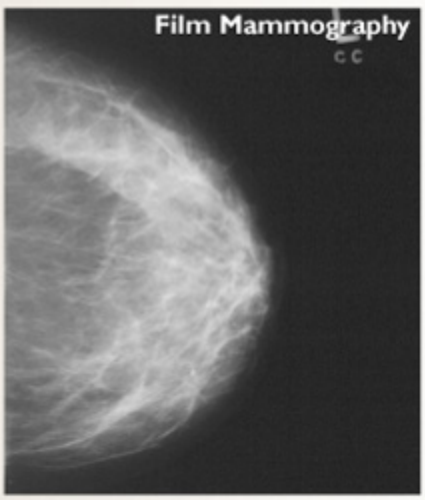

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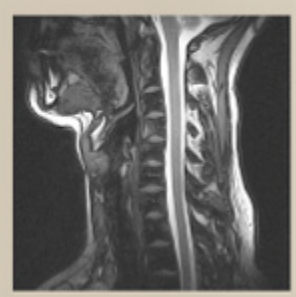

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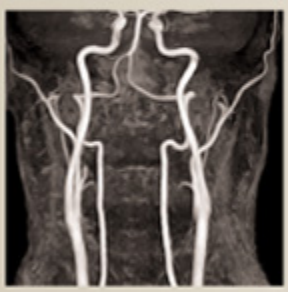

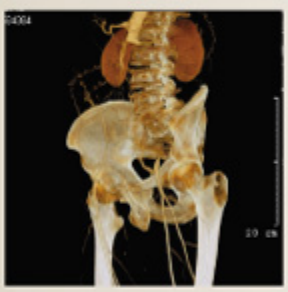

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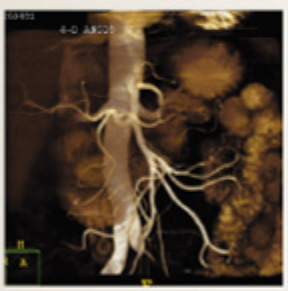

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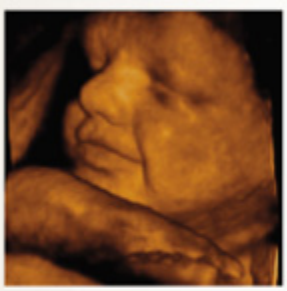

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Interstitial Cystitis

Deborah L. Myers, MD

Interstitial cystitis (IC) is a chronic condition of urinary urgency, frequency, and suprapubic pain in the absence of bacteruria. It is part of the painful bladder syndrome whose known causes are tuberculosis, stones, malignancy, previous chemotherapy of the bladder, and pelvic radiation. Interstitial cystitis is a diagnosis of exclusion when no known cause of painful bladder can be identified. It has classically been diagnosed by the presence of "Hunner's ulcers", a lesion noted on cystoscopy in 1915.[1] The word "ulcer" has proven to be a misnomer; the lesion is actually a coalescence of vessels. IC is a chronic illness for which we do not have a full understanding in terms of etiology or management.

IC occurs predominantly in women between 40-60 years, and in a ratio of 9:1 of women to men. With newer diagnostic techniques and less stringent criteria, the estimated prevalence of IC in the United States is approximately 1.5 million to 25 to 30 million women. Practitioners involved in women's health should know about this condition.[2]

The pathophysiology of IC remains unknown, but two integrated theories, the (1) "leaky epithelium" and (2) "neurogenic up-regulation" are proposed. The bladder uroepithelium has a protective mucous coat layer, the **glycosaminoglycan (GAG)** layer, which, when injured, becomes deficient, or "leaky", thus allowing potassium and toxins in the urine to penetrate into the underlying bladder and causing inflammation and pain.[3] In response to this bladder insult, detrusor mast cells release substance P, histamines and prostaglandins which cause vasodilatation and pain. The sensory C afferent nerve fibers of the bladder can become "up-regulated." (Figure 1) Studies have shown increased nerve fiber density including sympathetic nerves in bladders of patients with IC. IC could be a type of reflex sympathetic dystrophy with abnormal spinal sympathetic activity.[4]

The cause of the "leaky epithelium" still remains unknown. Work by Keay et al has identified proteins in the urine which affect the ability of the uroepithelium to regenerate and repair. Patients with IC have increased levels of **Anti-proliferative factor (APF).** APF inhibits the growth of the bladder lining. IC patients have lower levels of other proteins HB-EGF (heparin binding epidermal growth factor-like), required for epithelial growth.[5] In summary, the damaged epithelium leads to a complex cascade of interactions involving urinary cations, activated mast cells, sensory nerves, detrusor muscle overactivity, and spinal cord sensitization.

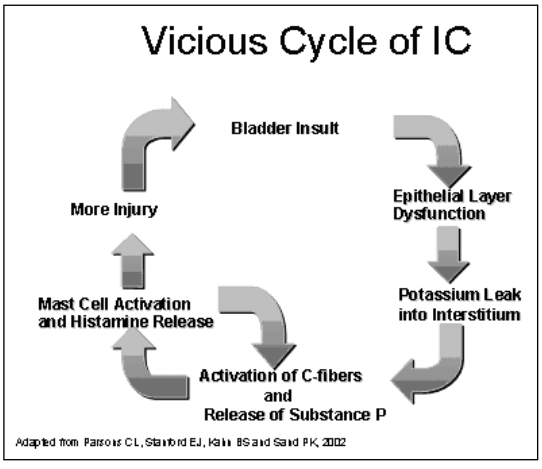


Figure 1. The Integrated Theories of Interstitial Cystitis.

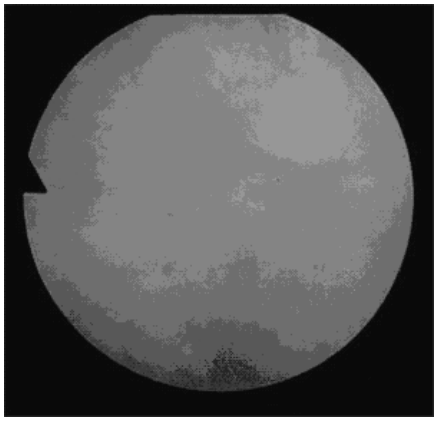


Figure 2. Hunner's Ulcer

Figure 3. The O'Leary- Sant Interstitial Cystitis Symptom and Problem Index (9)

Interstitial Cystitis Symptom Index	Interstitial Cystitis Problem Index During the past month, how much has each of the following been a problem for you?
Q1.During the past month, how often have you felt the strong need to urinate with little or no warning? 0.___not at all 1.___less than 1 time in 5 2.___less than half the time 3.___about half the time 4.___more than half the time 5.___almost always	Q1. Frequent urination during the day? 0.___no problem 1.___very small problem 2.___small problem 3.___medium problem 4.___big problem
Q2. During the past month, have you had to urinate less than 2 hours after you finished urinating? 0.___not at all 1.___less than 1 time in 5 2.___less than half the time 3.___about half the time 4.___more than half the time 5.___almost always	Q2. Getting up at night? 0.___no problem 1.___very small problem 2.___small problem 3.___medium problem 4.___big problem
Q3. During the past month, how often did you most typically get up at night to urinate? 0.___none 1.___once 2.___2 times 3.___3 times 4.___4 times 5.___5 or more times	Q3. Need to urinate with little warning? 0.___no problem 1.___very small problem 2.___small problem 3.___medium problem 4.___big problem
Q4. During the past month, have you experienced pain or burning in your bladder? 0.___not at all 2.___a few times 3.___almost always 4.___fairly often 5.___usually	Q4. Burning pain, discomfort, or pressure in your bladder? 0.___no problem 1.___very small problem 2.___small problem 3.___medium problem 4.___big problem
Add the numerical values of the checked entries; total score:_____	Add the numerical values of the checked entries; total score:_____

(9) O'Leary MP, Sant GR, et al. The interstitial cystitis symptom and problem index. *Urol* 1997;49(suppl 5A).

Figure 4[11]
Pelvic Pain and Urgency/Frequency (PUF) symptom scale
Please circle the answer that best describes how you feel for each question below.

	0	1	2	3	4	SYMPTOM SCORE	BOTHER SCORE
1 How many times do you go to the bathroom during the day?	3–6	7–10	11–14	15–19	20+	____	
2 a. How many times do you go to the bathroom at night?	0	1	2	3	4+	____	
b. If you get up at night to go to the bathroom, does it bother you?	Never	Occasionally		Usually	Always		____
3 Are you currently sexually active? Yes___ No___							
4 a. If you are sexually active, do you now have or have you ever had pain or symptoms during or after sexual activity?	Never	Occasionally		Usually	Always	____	
b. If you have pain, does it make you avoid sexual activity?	Never	Occasionally		Usually	Always		____
5. Do you have pain associated with your bladder or in your pelvis (vagina, labia, lower abdomen,urethra, perineum, penis, testes, or scrotum)?	Never	Occasionally		Usually	Always	____	
6 a. If you have pain, is it usually		Mild		Moderate	Severe	____	
b. Does your pain bother you?	Never	Occasionally		Usually	Never		___
7. Do you still have urgency after you go to the bathroom?	Never	Occasionally		Usually	Always	____	
8 a. If you have urgency, is it usually		Mild		Moderate	Severe	____	
b. Does your urgency bother you?	Never	Occasionally		Usually	Always		____

SYMPTOM SCORE (1, 2a, 4a, 5, 6a, 7, 8a)—SUBTOTAL ____
BOTHER SCORE (2b, 4b, 6b, 8b)—SUBTOTAL ____

TOTAL SCORE (symptom score + bother score) _____

Table 1. NIH-NIDDK Diagnostic Criteria of Interstitial Cystitis[7]

Category A: At least one of the following findings on cystoscopy:
-Diffuse glomerulations (at least 10 per quadrant) in at least three quadrants
-A classic Hunner's ulcer

Category B: At least one of the following symptoms:
-pain associated with the bladder
-urinary urgency

Exclusion criteria:
-age < 18 years
-urinary frequency while awake <8/ per day
-nocturia <2/night
-maximal bladder capacity >350ml while patient awake
-absence of an intense urge to void with bladder filled to 150ml during cystometry
-involuntary bladder contractions on cystometry
-duration of symptoms < 9 months
-symptoms relieved by antimicrobial agents, anticholinergics, or antispasmodics
-urinary tract or prostatic infection in the past 3 months
-active genital herpes
-vaginitis
-uterine, cervical, vaginal, or urethral cancer within the past 5 years
-bladder or ureteral calculi
-urethral diverticulum
-hx of cyclophosphamide or chemical cystitis or tuberculous or radiation cystitis
-benign or malignant bladder tumors

IC can be associated with irritable bowel syndrome, migraines, endometriosis, vestibulitis, vulvodynia, and collagen vascular diseases such as systemic lupus erythematosis. Depression and anxiety are often seen in these women; however, this is likely secondary to the chronic pain of IC. Women with IC score poorly on quality of life questionnaires, but IC should not be considered a psychosomatic disorder.[6]

Symptoms

Patients with IC will complain of urgency, frequency (> 8 voids per day), and bladder pain. Nocturia (>2xs/night) is almost always present. Episodes of incontinence are rare. These women often complain of difficulty voiding or post-void fullness. These patients do not tolerate large volumes of urine in their bladder, thus often sensing fullness. Many patients have been on chronic antibiotic therapy for supposed chronic urinary tract infections. Symptoms of IC overlap with overactive bladder, i.e., urgency and frequency, and thus some patients may have received anti-cholinergic therapy without relief.

Patients with IC can complain of either cyclic or constant pelvic pain. They may also complain of vaginal burning and/ or painful intercourse. Bladder symptoms are often increased with intercourse and near the menses. Symptoms of IC can mimic some gynecologic disorders, particularly endometriosis.

Diagnosis

Traditionally the diagnosis has been made by cystoscopy with other criteria as described by the **National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK)** in 1988. Table 1[7] Inclusion criteria require that the patient complain of urgency/ frequency or pain in the bladder, and have the presence of either glomerulations or Hunner's ulcers (Figure 2) in the bladder at cystoscopy. The exclusion criteria have been shown to be too strict for general clinical use, because approximately 60% of patients judged to have IC by experienced clinicians fail to meet the NIDDK criteria. Clinically, the diagnosis of IC can often be made by history, physical, screening questionnaires, laboratory studies, and office testing.[8]

Figure 5. SAMPLE BLADDER RECORD

Time Interval	Amount voided	Activity	Urge present	Leak / symptoms	Amount/type fluid intake

Instructions:
This is a record of your voiding (urinating) of urine or symptoms. Please complete this according to the following instructions. Choose a 1-2 day period to keep this record when you can conveniently measure every void, and begin with your first voiding upon arising as in the sample below.

Time Interval	Amount voided	Activity	Urge present	Symptoms	Amount/type fluid intake
645 am	550 cc	awakening			
7 00 am		Turned on water	yes		2 cups of coffee, 6 oz juice

1. Record time of all voids, leakage or symptoms, intake of liquids.
2. Measure all intake and output in cc's or oz's. You can measure the amount of urine that you pass by placing a large plastic bowl on the toilet seat for collection. Measure the amount and type of all liquid intake using either cc's or oz's (I cup = 8 oz = 240 cc).
3. If the urge to urinate accompanied (or preceded) the urine leakage write YES. If you felt no urge when the leakage occurred, write NO
4. Use additional sheets as needed.

History taking should query for urinary tract infections, pelvic surgery, known causes of painful bladder syndrome, IC symptoms and the associated conditions described above. O'Leary et al, in 1997, developed two validated self-administered questionnaires to monitor symptoms.[9] (Figure 3) Clemons et al in 2002 found that a score of = 5 on the symptom index was 94% sensitive in diagnosing interstitial cystitis.[10] Parsons CL et al developed the PUF (pelvic pain and urgency/ frequency) questionnaire as another tool to detect IC.[11] (Figure 4)

Physical and pelvic exam will most likely have few findings, but is necessary to rule out other causes of pelvic pain and urgency/ frequency symptoms. Gastrointestinal conditions such as irritable bowel syndrome, neurological conditions of the sacral nerves, musculoskeletal disorders, gynecologic conditions and other urinary causes (bladder stones or malignancy, urethral diverticulum) and urinary tract infection should be considered. In lieu of findings, pelvic exam may only show tenderness of the anterior vaginal wall, bladder and urethra. Spasticity, tenderness, and localized 'trigger points' of the levator ani muscles of the pelvic floor may be elicited. Women with chronic pelvic pain can develop levator spasm which in turn can continue to cause symptoms of pain, urinary urgency, and frequency.[12]

Laboratory

Urinalysis and urine culture are required laboratory studies. Urine cytology would be obtained in patients who have risk factors for bladder cancer. An abnormal urine cytology or microscopic hematuria, will require radiologic studies such as CT nephrogram and referral for cystoscopic evaluation.

Urolog

A 24-48 hour voiding diary (urolog) records the amount and type of fluid intake, the time of each void and the volume voided at each micturition. Patients with IC will usually have frequent voids (>12/ day) and small voided volumes (average of 75-100cc). Nocturia will usually be present. The urolog also allows the clinician to determine if the fluids consumed are potential bladder irritants. (Figure 5)

Assess Post Void Residual

Patients with IC often complain of incomplete emptying and/ or post void fullness, therefore an assessment of post-void residual urine volume is needed, either by bladder ultrasound or by catheterization.

Treatment In the Primary Care Setting

Treatment can be initiated based after careful assessment and exclusion of other causes. Treatment ultimately is multi- modality, but may need to be introduced one at a time, to determine which options will be long term.

Self Help and Patient Education

The chronic nature of the disease, including the possibility of relapses, should be explained to the patient. Several self-help books are available and agencies from which to get additional information on the condition.[13] Both the NKUDIC (3 Information Way Bethesda, MD 20892–3580 Phone: 1–800–891–5390) and the Interstitial Cystitis Association (110 North Washington Street, Suite 340, Rockville, MD, 20850 301-610-5300, 1-800-helpica) have accurate information. The **Interstitial Cystitis Association (ICA)** provides support group information, conferences and medical information. Patients can be referred to the various websites; e.g., www.ichelp.org and www.ic-network.com. Stress reduction techniques (self-visualization, yoga, baths, deep breathing, meditation) can create a sense of well being. Development of coping mechanisms, problem solving, and also sex therapy with the help of a psychologist may also be needed.

Table 2. Dietary irritants to avoid[14]

All alcoholic beverages
Apples
Apple juice
Cantaloupes
Carbonated drinks
Chili,
Spicy foods
Citrus fruits (lemons, limes, oranges, etc.)
Coffee
Cranberries
Grapes
Guava
Lemon juice
Peaches
Pineapple
Plums
Strawberries
Tea
Tomatoes
Vinegar

Dietary recommendations

Avoidance of (1) carbonated, citrus and caffeinated beverages, (2) foods high in potassium content such as citrus fruits and tomatoes or (3) foods with a high acid content, and (4) spicy foods and foods rich in tyrosine and tryptophan can help relieve symptoms in some patients.[14] (Table 2) Increasing water intake is another important dietary recommendation. Patients with IC tend to decrease their fluid intake to limit the frequency of voids; however this concentrates the urine, leading to increased irritation.

Over the counter (OTC) Supplements and alternatives

Glucosamine/ chondroitin sulfate taken 1000mg daily and the amino acid supplement L-arginine taken 500 mg PO TID for 6 months can provide relief of symptoms. Other alternatives Algonot Plus® and CystoProtek ®,(Alaven Pharmaceutical, Marietta GA) and Cysta-Q™ (Farr Laboratories, Westwood, CA) are found at various websites on the Internet. Calcium glycerophosphate (Prelief ®) from AKPharma Inc., Pleasantville, NJ, a tasteless deacidifier, taken before meals can reduce food acidity.

Analgesics and Anti-Spasmodics

Phenazopyridine (Pyridium® Warner Chilcott, Rockaway, NJ) is a bladder analgesic that can relieve symptoms and on an as-needed basis for symptom flares. The anti-cholinergic medications used to treat overactive bladder may improve urinary frequency and urge incontinence if present. However, if used alone, the anti-cholinergics are unlikely to be effective based upon our current understanding of the pathophysiology of IC, since they do not affect the cascade pathway.

Specialized Diagnosis and Management

If initial diagnostic maneuvers are not conclusive and/ or initial treatments have not proven to be effective, then further testing or referral to indicated specialists: urogynecology, urology, physical therapy, psychology, psychiatry or pain clinics for further diagnostic steps and/ or treatment is indicated. The primary care physician should have a working knowledge of the methods used to manage more advanced cases of IC.

Potassium Testing

In 1996, Parsons introduced the potassium sensitivity test as an office test that can detect IC.[15] The KCL test involves instilling two different solutions in to the bladder (sterile H2O vs. a KCl solution) and comparing symptoms. Instilling a solution of potassium chloride into the bladder of a patient with IC with a "leaky epithelium" should cause symptoms of urinary urgency, frequency and pain, but not into the bladder of a normal patient. Although the potassium test may only detect 66% of women with IC, it is still a useful simple office diagnostic test.[16]

Cytoscopy

Cystoscopy with hydrodistension, the traditional method in the diagnosis of IC, is done under either general or regional anesthesia. During cystoscopy the bladder is filled to 70- 80 cm H_20 pressure and held at this capacity for 2-5 minutes. Cystoscopic findings of IC are glomerulations and Hunner's "ulcers" are sought. (Figure 2). Suspicious areas for carcinoma are biopsied. Traditionally, biopsies were routinely taken to look for a high number of mast cells in the bladder muscularis. However, as more research has been done, no characteristic pathologic change has been described for the tissue diagnosis of IC.

Urodynamic Testing

In general full urodynamic studies (cystometrogram, assessment of sphincter function, pressure flow studies, uroflowmetry) are not necessary. However, if after initial screening post void residual volumes are found to be >100cc or if the patient complains primarily of urgency and frequency, then urodynamic testing would be indicated.

Management

Pharmacologic therapy

Pentosan polysulfate

Pentosan polysulfate (Elmiron®, Ortho-McNeil, Raritan, NJ) is the only FDA-approved oral medication for the treatment of IC. Its chemical structure is similar to the GAG layer and it works to rebuild "leaky epithelium". Elmiron has $1/15^{th}$ of heparin's anticoagulant effects and should be used with caution in women with a bleeding diasthesis. Possible side effects include gastrointestinal distress, headache and reversible hair loss. Only 60 % of patients will experience relief of symptoms and relief may not be seen until 4- 6 months of use.[17] Therefore, continued use, despite no change of symptoms at 3 months, is recommended. Other treatment options may be needed during this waiting period as described.

Tricyclic Anti-depressants

Tricyclic anti-depressants such as amitriptyline and nortriptyline are frequently prescribed "off- label" for IC. Tricyclic anti-depressants (1) reduce bladder urgency by their anticholinergic properties, (2) raise the pain threshold, (3) improve sleep by sedation, and (4) elevate mood. Tricyclics can give prompt relief of symptoms in most patients. They should be used with caution in the elderly because they can cause confusion and electrocardiogram changes. Van Ophoven et al, recently demonstrated in a prospective randomized placebo controlled double blind study that amitriptyline can improve symptoms in IC patients.[18]

Central Nervous System Drugs

Medications for neuropathic pain are used off label to manage the pain component of IC. Gabapentin, pregabalin, carbamazepine and duloxetine are used. Prescribing neuroleptics is done as for other pain conditions with escalating

doses until desired effect or until side effects become intolerable. Black box warnings should remain mindful.

Anti-histamines

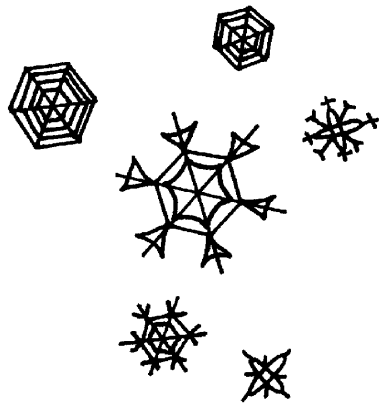
Hydroxyzine is used off- label in the management of IC. Possible mechanisms of action include stabilization of mast cells, anti-cholinergic properties, and a sedative effect. Theoharides and Sant in a 1997 clinical trial demonstrated that hydroxyzine provided an overall 40% reduction of symptoms; in patients with a history of allergies, they found a 55% reduction of symptoms.[19] Hydroxyzine has a sedating effect, thus it can also improve sleep. The allergy/ asthma medication montelukast a leukotriene inhibitor, may prove to be effective as leukotrienes are released from mast cells and thought to play a role in inflammation

Physical therapy and bladder retraining

Referral to a physical therapist who specializes in treatment of pelvic floor dysfunction can help patients re-educate the levator ani muscles. Women with IC often have levator ani muscle spasm. Bladder retraining can be introduced if symptoms are mild or after symptoms are controlled. Monthly provider visits assist with maintaining compliance, providing motivation, and monitoring progress.

Intravesical therapy

Intravesical therapy may be needed if initial oral medications cannot control symptoms or if patients on oral therapy have a flare in symptoms. Intravesical treatments are done by instilling medications into the bladder through a catheter. Dimethylsulfoxidole (Rimso-50 ® Bioniche Pharma USA, Lake Forest,IL) was FDA-approved for treatment of IC in 1978. Heparin, hyaluronic acid (available only in Canada), BCG (Bacillus Calmette Guerin), Elmiron, anesthetic agents, and "cocktails" of combinations of xylocaine, corticosteroid, heparin, antibiotics, and sodium bicarbonate have all been used. Frequency of installations will vary.



Surgical treatments

Cystoscopy with hydrodistention causes epithelial damage by mechanical trauma with regeneration of new epithelium and improvement of symptoms. Sympathetic fiber density has been found to be decreased after bladder distension, thus explaining the relief of symptoms after the procedure.[4] Remission generally lasts for 6 months, with a gradual recurrence of symptoms in most patients. More radical surgical procedures such as enterocystoplasty, cystolysis, urinary diversion alone, and urinary diversion into a continent pouch combined with cystectomy, have been used to treat intractable cases of IC. However, these radical end stage procedures have not shown to be beneficial: patients continue to suffer from sensory urgency/ pain.[20]

Sacral neuromodulation

Sacral neuromodulation (Interstim® Medtronic Corp., Minneapolis, MN) is FDA-approved for patients with urge incontinence, urinary retention and urinary urgency/ frequency, but not yet for IC. It offers a less radical and reversible option than an extirpative procedure and should be considered before an end stage procedure. There are preliminary reports of its use in patients with IC, but the long term success of the sacral stimulation and its management of pain in these patients is still preliminary.[21]

Primary Care Provider's Role

The primary care provider can diagnose IC and initiate several treatment protocols. Simple treatments can be instituted based on symptoms, physical examination, and screening labs. If sufficient relief is not obtained, the provider can initiate further testing or refer to the appropriate specialist for more specialized diagnostics and therapies. The primary care provider should stay involved in the management of these patients as part of a multi-disciplinary team to provide the best overall care for the patient.

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Of-Label Usage of Medications

All medications for IC except DMSO and Elniunrion are off-label.

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Effect of Zoledronic Acid on Bone Pain Secondary To Metastatic Bone Disease

Porpon Rotjanapan, MD

You are in your office. Mr.X, a 64 year-old patient well known to you for over 10 years, comes for persistent severe bone pain despite palliative radiotherapy and recent opioid treatment. Six years ago, he was diagnosed with stage IV prostate cancer with metastasis in the right pubic bone. He was treated with surgery and pelvic irradiation including the right pubis. One year prior to presentation he had an orchiectomy with prompt relief of pain and a decrease in his **prostate specific antigen (PSA)**. He is taking morphine and dexamethasone was added recently with minimal effect. The pain has affected his sleep, appetite, and ability to enjoy life. He does not like the opiates' effect on his ability to make high level decisions.

The patient comes into the office noting that he just saw a commercial about a new medication, zoledronic acid, for treatment of cancer bone pain. He wants to know if this can help him.

What are the current treatment options for metastatic bone disease?

Bone metastases are frequent in patients with advanced cancer. The most common cancer which metastasize to bone are breast, lung, prostate, multiple myeloma, and renal. Skull, spinal column, rib cage, pelvis and femur are the most frequent sites of bone metastases.

Pain from cancer is a major problem. Thirty percent of patients with cancer have pain at the time of diagnosis; 65 to 85% have pain when their disease is advanced. The impact of cancer pain is magnified by its interaction with other common cancer symptoms: fatigue, weakness, dyspnea, nausea, constipation, and impaired cognition.[18] With an integrated program of systemic, pharmacologic, and anticancer therapy, cancer pain can be effectively treated in 85-95% of patients. Many of the remaining patients can be helped by the use of invasive procedures. In the final days of life, pain not controlled by therapies aimed at both comfort and function can be relieved by intentional sedation. No patient with cancer needs to live or die with unrelieved pain. Pain caused by bone metastases lowers the quality of life and performance status of patients, and causes disability occurring at rest or typically during movement.[14]

Treatment of bone metastases is aimed at reducing the risk of pathological fractures and other **skeletal related events (SREs)**, as well as reducing pain to maximize patients' quality of life. The options include surgery, radiation therapy, radiometabolic therapy, chemotherapy, hormone therapy, and other palliative treatment. Bisphosphonate therapy is now well established as a way of reducing morbidity from the lytic skeletal metastases.[19]

BISPHOSPHONATES

Metastatic bone disease is associated with a marked increase in bone resorption and formation rates, which can be evaluated by the measurement of biochemical markers of bone metabolism in the serum or urine.

Bisphosphonates inhibit osteoclastic bone resorption and control bone metabolism via several mechanisms that differ from those of other antiresorptive agents. They contain 2 phosphate groups linked to 1 carbon atom, forming a stable structure (phosphorus-carbon-phosphorus) resistant to the action of osteoclastic hydrolytic enzymes. This backbone and the presence of R1 and R2 chains allow bisphosphonates to bind calcium phosphate and inhibit bone resorption by osteoclasts.[14]

Bisphosphonates inhibit osteoclast maturation and function and ultimately cause osteoclast apoptosis. Initially bisphosphonates were developed to treat predominately osteolytic bone metastases. However histomorphometric and biochemical evidence show that osteoblastic lesions also lead to increased osteolysis and bone turnover and that bone resorption markers are significantly raised in patients with advanced prostate cancer.[14]

There are 3 different classes of bisphosphonates; the first is characterized by the absence of nitrogen atoms; the second contains only 1 nitrogen atom, and the third has 2 nitrogen atoms.[20] **Zoledronic acid (ZA)** is one of the most active nitrogen-containing bisphosphonates, the third generation intravenous bisphosphonate that is at least 100 fold more potent than pamidronate.[7] It inhibits the enzyme farnesyl diphosphonate synthase and has been approved for treatment of bone metastases. Recent studies suggest that ZA also has direct antitumor activity.[16]

We reviewed the literature, using Pubmed (January 1966-2007) and the keywords zoledronic and pain. The initial search yielded 162 articles. After selecting those clinical studies published in English, 25 articles met our review criteria. Thirteen articles were applicable to the current case. The majority of studies were conducted in the United States and Italy (n= 7 and 5 respectively). Others were from Canada, UK, and one study from South Korea. The time frame ranged from 2000-2007. Study populations were Caucasians, and the minority of study populations were black and Asians. Mean age ranged from 57-72 years old. Proportion of male to female was close to 1: 1 overall.

Table 1 shows the types of study and demographic of patients in each study. Table 2 summarizes the methods and results of each study focusing on pain assessment and analgesic use in some clinical trials. A majority of patients received ZA 4

Table 1: Types of study and demographic

Study	Type of study	N	Age (mean age/ range)	Populations
Carteni et al.[1]	Multicenter, open-label study	316 pts screened, 312 pts enrolled	58.6+/- 11.7, 28-86	Breast cancer with newly diagnosed bone metastases
Facchini et al. [2]	Prospective study	60	76, 40-83	Breast and lung cancer
Hong et al. [3]	Prospective, multicenter, open-label trial	19	67.3/ 46-86	Hormone refractory prostate cancer
Ripamonti et al. [4]	Pilot study, prospective observational study	48	66/ 41-84	Breast and prostate cancer (N=34), (N=14)
Weinfurt et al. [5]	Randomized, double-blind, placebo-controlled, parallel-group study	422	71.8/72.2	Prostate cancer
Storto et al. [6]	Retrospective non-randomized trial	49	65(52-78), 70(58-82), 69(57-81)	prostate and breast cancer refractory to conventional treatment
Fulfaro et al. [7]	Prospective study	24	69, 45-75	Prostate cancer
Wardley et al. [8]	Randomized crossover study	101	60(37-87), 59(37-76)	Breast cancer
Berenson et al. [9]	Multicenter, open-label, dose-ranging, safety trial	59	N/A	Breast, MM, lung cancer, renal cell cancer
Berenson et al. [10]	Randomized, double-blind, double dummy, parallel group multicenter study	280	57.6+/-12.9, 56.5+/-13.6, 59.9+/- 11.3, 57.7+/-11.8	Breast cancer, MM
Clemons et al. [11]	Prospective study	31	58, 35-81	Breast cancer
Vogel et al. [12]	Open-label, prospective, multicenter study	638	66.4+/-11, 60+/- 13, 72.6+/- 9	Breast, prostate cancer, and MM
Saad et al. [13]	Randomized placebo controlled trial	458	72/73, 64/65	Prostate, renal cell cancer

mg, the standard dose intravenously every 3-4 weeks for a total of 3 months to 2 years depending on studies except study # 10 that used ZA dose ranged from 0.1-8 mg. pain assessment was evaluated before, while receiving treatment, and at the end of each study.

What is the benefit for this patient?

Of the 13 published studies, 7 focused on patients with metastatic, hormone refractory prostate cancer. Six out of the 7 studies demonstrated significant reduction in pain scores except that Ripamonti et al[4] showed that pain scores showed no statistically significant difference before and after treatment. This could be explained by the very small sample (N= 19) enrolled in this study.

Unlike bone metastases from other types of cancer, most bone lesions in prostate cancer are osteoblastic. However, recent studies showed that osteoblastic lesions not only have upregulated bone growth, but also concomitant increased osteolysis. The new bone created by tumor-stimulated osteoblasts is weak and poorly mineralized, and the osteopenia secondary to the increased osteolysis results in a bone matrix with severely compromised integrity. The risk of developing a skeletal complication is thus increased.[13] Fulfaro et al investigated the use of ZA in patients with bone metastases from prostate cancer and the effect on analgesic response and bone metabolism biomarkers. Besides the impressive pain control from ZA treatment, the bone metabolism biomarkers, C-telopeptide and bone alkaline phosphatase also decreased which confirmed the biochemical mechanism of action of zoledronic acid both on markers of bone formation and resorption.[7]

ZA is the only bisphosphonate confirmed to be effective in reducing skeletal complications associated with bone metastases from advanced prostate cancer.[13] The findings from these studies suggest that patients receiving ZA experienced a higher likelihood of clinically meaningful reductions in pain. Thus, ZA may help to avert the pain experienced by patients with progressing metastatic disease secondary to prostate cancer. The benefit from ZA therapy in terms of pain control and analgesic use from these 7 studies could potentially apply to Mr. X who has been through several modalities of treatment for his bone pain.

What are the risks?

ZA was well tolerated. Reported adverse events ranged from 2-60% of patients. These events were generally mild to moderate in severity and were consistent with known safety profile of i.v. bisphosphonates. From our review, we found that common adverse reactions were pyrexia (22-44%), fatigue (39%), skeletal pain (10-60%), nausea and vomiting (3%), headache (2-19%), hypocalcemia (9-33%), and confusion (7-13%).[1-13] All these events were mild in severity. Renal adverse events were noted, with an increase in serum creatinine levels from screening to final visit of < 0.5 mg/dL in 94.7% of patients from study #4 but all returned to within the normal range during follow-up.

Overall, ZA is the most broadly active i.v. bisphosphonate, and is the only one approved for preventing skeletal complications of malignancy in patients with bone metastases from all solid tumor types.[15] ZA is well tolerated with long-term use. It has an overall safety profile similar to other i.v. bisphosphonates and the renal safety profile is comparable with pamidronate when administered in accordance with treatment guidelines.[17] ZA is associated with a minimal risk of increased serum creatinine in patients with advanced prostate cancer and not influenced substantially by prior bisphosphonate exposure.[13]

Discussion

ZA has demonstrated statistically significant long-term efficacy and has the broadest clinical utility for pain palliation in a variety of tumor types of ZA therapy for bone pain secondary to bone metastases. The number needed to treat for bone pain calculated from available data from our review was 1.92 (indicating that 2 patients need to be treated with ZA to obtain improvement in 1), which strongly confirmed the superior benefit of this new drug treatment. ZA was extremely well tolerated in most clinical trials. Renal toxicity was the only serious safety finding after ZA treatment. Renal toxicity was related to dose (more with 8 mg than 4 mg), infusion duration (more with infusion over 5 minutes than 15 minutes), and total number of infusions. But no long- term complications were observed from the clinical trials.

Hypocalcemia is a side effect common to all bisphosphonates, regardless of administration method. However, it can be controlled with calcium and vitamin D supplements. Other adverse reactions were generally mild to moderate. Some patients reported only a single episode following the first infusion of ZA.

Table 2: Methods and results

Study	Dose/regimen	Parameters	Pain assessment	Results
Carteni et al.[1]	ZA 4 mg q 3-4 weeks* 12 months	BPI/ analgesic use	Every 12 weeks	3.3+/-2.2→2.6+/-2.3 -58% pain decreased -19% pain unchanged -37% decreased analgesic use -46% unchanged analgesic use
Facchini et al. [2]	ZA 4 mg q 3-4 weeks * 12 months	VAS	Before and 1 year after treatment	-Mod pain : 38.6% → 20.4 % -Severe pain: 38.6% → 9.1%
Hong et al. [3]	ZA 4 mg q 3-4 weeks * 6 months	BPI/ analgesic use	Every 4 weeks	1.5+/-0.5→1.7+/-0.5 -no statistically sig different in pain scores -use less strong opioids 6.6%-->0% -use less mild opioids 20→7.1%
Ripamonti et al. [4]	ZA 4 mg q 28 days * 6 months	VRS/analgesic score(six- level)	Every 2-4 weeks	VRS At rest: pain reduction=0.59 On movement: pain reduction= 0.86 Analgesic use decreased: At rest: 31% On movement: 27%
Weinfurt et al. [5]	ZA 4/8 mg q 3 weeks * 15 months	BPI	Every 4 weeks	Decreased pain in 33%
Storto et al. [6]	Gr.A ZA 4 mg q 3-4 weeks Gr.B ZA 4+^{89}Sr-Cl Gr.C^{89}Sr-Cl	VAS	Every week *2 months then 6 months later	Improvement of VAS: Gr. A 96% Gr. B 84% Gr. C 72%
Fulfaro et al. [7]	ZA 4 mg q 3-4 weeks* 1.5 years	VAS	0, 1st, and 3rd month	VAS 7.8+/-0.29→3.0+/-0.4
Wardley et al. [8]	ZA 4 mg q 4 weeks* 9 months	BPI	Every month	No BPI reported but significant pain reduction observed
Berenson et al. [9]	ZA 0.1,0.2,0.4,0.8,1.5,2,4,8 mg q 4 weeks * 3 months	Undefined pain scale/ analgesic score	Every week	No detectable improvement/worsening of pain scores
Berenson et al. [10]	ZA 0.4,2,4 and pamidronate 90 mg q 4 weeks * 10 months	BPI	Every 4 weeks	Decrease in pain scores in both groups, 67% & 50%
Clemons et al. [11]	ZA 4 mg q 4 weeks * 3 months	BPI	Every 4 weeks	Pain reduction 41.9% at 8 weeks
Vogel et al. [12]	ZA 4 mg q 3-4 weeks *6 months	VAS	Every 4 weeks	MM: 33.3+/-27→24.6+/-23 Breast: 32.8+/-24.5→26+/-24 Prostate: 34.7+/-25→33.2+/-29
Saad et al. [13]	ZA 4 mg q 3 weeks *2 years	BPI	Every 3 weeks	No BPI reported but less bone pain in ZA group

These clinical trials have confirmed the favorable benefits of ZA for bone pain secondary to multiple types of malignancy. Moreover, ZA was likely to be associated with clinical reductions in pain not only at rest but also on movement. This supports the consideration of ZA for bone pain reduction in metastatic bone disease due to prostate cancer. Taken altogether, the results of the contemporary randomized controlled trials indicate that ZA decreases the risk of skeletal complications and pain palliation in men with androgen-independent prostate cancer and bone metastases while other bisphosphonates: pamidronate, ibandronate, and clodronate, although tested, seem to be ineffective in this setting.[21,22]

What is this patient's decision for his next step of treatment?

The patient was treated with ZA, with improvement of his pain and reduction in his opiate dose.

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GERIATRICS FOR THE PRACTICING PHYSICIAN

QUALITY PARTNERS OF RHODE ISLAND

Quality Partners of RI
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The Practicing Physicians' Guide To Pressure Ulcers in 2008

Rachel Roach, MSN, ANP, GNP, WCC, and Clarisse Dexter, MSN, FNP, GNP, WCC

MS is a 79-year-old woman admitted to the nursing home following a lengthy hospitalization for pneumonia and COPD. Her hospital course was complicated by *Clostridium difficile colitis* and respiratory failure requiring mechanical ventilation. During her stay, she developed a sacral pressure ulcer. While performing your admission examination, you note that the pressure ulcer is 3 cm x 4 cm wide and 1.2 cm deep, with thick, adherent, yellow slough covering the entire wound bed. The walls of the wound are gray, fibrous tissue; there is undermining from 10 to 2 o'clock. There is minimal wound exudate. The peri-wound tissue is macerated.

Pressure ulcers are, in most cases, preventable injuries.[1] This article will guide the clinician in formulating a reasonable, evidence-based plan to heal pressure ulcers.

ASSESSMENT

A comprehensive assessment of the overall health status of the patient and the characteristics of the ulcer are essential, and form the basis for treatment. Aspects to be assessed and documented include:

- Location and size of the ulcer, documented by anatomical part, and measured as length x width x depth in centimeters.
- Describe the wound from the bottom up:
  - A description of the tissue or necrotic debris in the wound bed, noted as slough or eschar, and reporting the percentage of debris versus granulation tissue.
  - Record the characteristics of any exudate present, documenting odor, color and consistency.
  - Note the presence of tunneling, tracts and undermining, using a clock and head-to-toe direction for documentation reference points.
  - Note and remark on the condition of the surrounding skin.

CLASSIFICATION OF PRESSURE ULCERS

The National Pressure Ulcer Advisory Panel (NPUAP) has developed a specific, standardized rating system to "stage" pressure ulcers.[2]

Stage I is an observable pressure-related alteration of intact skin, as compared with the adjacent skin or opposite area on the body. The ulcer appears as a defined area of persistent redness in lightly pigmented skin, and red, blue or purplish tones in darker skin. This area may be painful, pruritic, and warmer than the surrounding tissue.

Stage II is a partial thickness skin loss involving the epidermis and/or the epidermis. This ulcer appears as an abrasion, blister or a shallow crater.

Stage III is a full thickness loss of the subcutaneous tissue extending to, but not through the underlying fascia. This wound is a deep crater.

Stage IV is also a full-thickness tissue loss, with destruction extending into the muscle, supporting structures or to the bone.

The presence of eschar covering a wound prevents staging. These wounds are documented as "unstageable" until the eschar is removed and the wound bed can be inspected.

Deep tissue injury is the most recent classification of pressure ulcer added by the NPUAP. This type of ulcer often has the appearance of a deep bruise under intact skin and may rapidly progress to a full thickness ulcer.[3]

TREATMENT

The selection of a treatment for a wound should be based on the needs of the patient, the wound, the caregiver and the clinical setting. The dressing should provide moisture balance in the wound bed, manage exudate, prevent infection, not cause pain to the patient and protect the periphery of the wound from damage. The goals of care of the patient and the cost to the payor should also be considered.

The clinician should become familiar with the different categories of dressings and their composition. Knowledge of the facility or institution's protocol and inventory, along with communication with the wound care team will assist in formulating a comprehensive treatment plan.

Table 1 will assist in selection of an appropriate treatment modality.

Table 1:

Wound Care Matrix	Intact Skin	Stage I	Stage II	Stage III	Stage IV
Exudate	None	None	Light	Dry to Moderate	Heavy
Product Category	Skin Care	Barrier Creams or Transparent Film	Hydrocolloid Dressings	Hydrogels to add moisture or Alginates to absorb	Foams Specialty Absorptive Dressings, or Negative Pressure Wound Therapy

Table 2. Assessment of and Intention to Treat Infection in Chronic Wounds[6]

Bacterial Burden	Contaminated	Colonized	Critically Colonized	Local Infection	Systemic Infection
Wound Clinical Symptoms and Signs	Wound progressing, Host stable	+/- early signs of local infection	No/subtle s/s of infection	Local s/s of infection	Constitutional ss/ of infection
Bacterial culture and sensitivity	No	+/- C&S wound	C&S wound	C&S wound	C&S wound and blood culture
Topical antibiotic	No	+/-	Yes	Yes	Yes
Systemic antibiotic	No	No	+/-	+/-	Yes

Wound Bed Preparation

Wound bed preparation provides a conceptual approach to treatment decisions. Wound bed preparation is defined as "the global management of the wound to accelerate endogenous healing or to facilitate the effectiveness of other therapeutic measures."[4] The first step is removal of dead tissue and contaminants in a timely manner.[5] This cleaning of the wound bed can be accomplished by bedside sharp debridement, autolysis (synthetic dressings cover a wound and allow devitalized tissue to self-digest from enzymes normally present in wound fluid), mechanical (wet to dry dressings), chemical removal (collagenase enzymatic debridement), and whirlpool irrigation of the wound. Wounds should be cleansed with low toxicity solutions, preferably normal saline. Topical antiseptics (such as Dakin's solution, Domeboro's solution, Betadine and Acetic Acid solutions) should be reserved for wounds that are not expected to heal or those in which the local bacterial burden is of greater concern than the stimulation of healing.

The second consideration is to control the bacteria in the wound. It is important to differentiate between contamination and the colonization of bacteria in a wound. On the continuum of bacterial burden, contamination is the presence of bacteria on the wound surface. Colonization reflects the presence of replicating bacteria that is not yet causing injury to the host. Critical colonization occurs when the presence of bacteria (or bio-burden) delays or stops healing, but without signs or symptoms of infection. When pain, erythema, warmth, purulent discharge, odor or new breakdown is present, local infection must be suspected. Systemic infection is marked by symptoms which extend beyond the borders of the wound. Symptoms may include erythema, induration, fever, and leukocytosis. These symptoms indicate the need for treatment with a systemic antibiotic.

The swab cultures of contaminated or colonized wounds are of limited diagnostic value. If a wound culture is indicated, a tissue biopsy is preferred.

The third consideration is providing an environment to the wound bed that provides moisture to promote healing and controls exudate. Lastly, optimize the repair process by providing the patient with nutritional support, vitamin supplementation, adequate hydration and by avoiding exposure to cold (vasoconstriction reduces blood flow to the wound). It is also important to provide an appropriate support surface, such as a low air loss or air fluidized mattress for pressure relief. Adjunct therapies should also be considered. This may include negative pressure wound therapy, physical therapy, growth factor modalities and referral to plastic surgery when indicated.

Summary

Let us now revisit the patient MS. The clinician performed sharp debridement of the devitalized tissue with a scalpel. The remaining slough was removed using enzyme therapy - Santyl Collagenase, for example. The debridement revealed a stage III ulcer. The wound was irrigated daily with normal saline and lightly packed with a hydrogel-impregnated dressing (to provide moisture). The macerated skin surrounding the wound was protected from excessive moisture using a topical material, Skin Prep, for example. A nutritionist was consulted, who advised a multivitamin and a protein supplement. Wound closure was achieved in a timely fashion.

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Rhode Island HEALTH Web Data Query System: Death Certificate Module

Annie Gjelsvik, PhD, and Karine Monteiro, MPH

The **Rhode Island HEALTH Web Data Query System** (**RI HEALTH WDQS**) allows health professionals, community agenccies, Rhode Island Health Department personnel, and the general public to access **Behavioral Risk Factor Surveillance System** (**BRFSS**), **Youth Risk Behavior Survey** (**YRBS**) and Death Certificate Data online. This resource can provide health professionals and community organizations with valuable information with which to improve the health of Rhode Islanders through data-driven programs and policies.

The RI HEALTH WDQS is publicly accessible and requires no sign-in or registration. It contains ten years (1998-2007) of RI BRFSS data on over twenty topics, four years (2001, 2003, 2005 and 2007) of RI YRBS data on over eleven topics and two years (2004 and 2005) of Death Certificate data on fifty two underlying causes of death. (Table 1). The BRFSS module has been described elsewhere[1] and the YRBS module has very similar design and functionality. Here we describe the new addition of Death Certificate Data.

Design and Functionality

The RI HEALTH WDQS Death Module allows users to obtain crude and age-adjusted or age-specific rates, select all cause or specific underlying cause of death (with up to four sub categories) and limit tables by demographics. In addition users can combine years in order to obtain stable estimates, define the report content and output. Output can be viewed in a browser window or downloaded to an Excel file. To ensure confidentiality, the RI HEALTH WDQS Death Query module adheres to the same rules as consistent with the Vital Records determination and masks information for causes of death for which there are 5 or fewer deaths. [2]

The RI HEALTH WDQS Death Module includes only data on Rhode Island residents. The deaths of RI residents occurring in other states are included in the data while deaths of non-RI residents occurring in RI are not included. This enables crude, age-adjusted and age-specific rates to be determined using US Census data for Rhode Island as the denominator. Data posted on the RI HEALTH WDQS Death Module are posted initially as provisional data and are updated once to final when the Vital Statistics Annual Report[2] is issued for that year. Provisional data are close to final but subject to changes as additional records are added to the dataset or updated information is obtained. Therefore, before the data are final, deaths of RI residents occurring in other states may not have yet been included.

The Death Module reports on underlying cause of death (not contributing causes). The results found here may not illustrate the full picture of the burden due to deaths of some diseases. For instance an underlying cause of death and contributing cause of death identified 2.5 times more diabetes deaths.[3] Missing, unknown, not stated and not classifiable data are not tabulated in the output tables; however, they are included in the totals. Therefore the individual categories may not sum to the total.

Development process

The Centers for Disease Control and Prevention's (CDC) Assessment Initiative Cooperative Agreement supported the development of this system. Core programming used for the RI HEALTH WDQS Death Module was adapted from the Arkansas Department of Health Center for Health Statistics Query System.[4] The Rhode Island Assessment Initiative will use a similar process to create a Birth Data module, scheduled to go on-line by June 2009.

How to Access the System

You can access each of the modules from the RI HEALTH WDQS homepage (http://www.health.ri.gov/data/webquery.php) or navigate there from the individual database web pages.

Future Plans

The project will incrementally expand access to other databases in phases, beginning with Birth Certificate data and Middle School Youth Risk Behavior. Other databases considered for inclusion in the system within the next five years include **Pregnancy Risk Assessment Monitoring System** (**PRAMS**), Hospital Inpatient and Hospital Outpatient databases and Cancer Registry Data.

The Rhode Island Assessment Initiative will be holding workshops on how to access Rhode Island vital statistics data and how to interpret and present results. This workshop will be modeled on the *Web Access to Rhode Island Public Health Data* workshops developed to provide the Rhode Island public health work force with training in how to access data and interpret and present results of BRFSS and YRBS data.

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Table 1: Causes of Death Currently Available on Rhode Island's HEALTH Web Data Query System – Death Certificate Module

Salmonella infections (A01-A02)
Shigellosis and amebiasis (A03, A06)
Certain other intestinal infections (A04, A07-A09)
*Tuberculosis (A16-A19)
Whooping cough (A37)
Scarlet fever and erysipelas (A38, A46)
Meningococcal infection (A39)
Septicemia (A40-A41)
Syphilis (A50-A53)
Acute poliomyelitis (A80)
Arthropod-borne viral encephalitis (A83-A84, A85.2)
Measles (B05)
Viral hepatitis (B15-B19)
Human immunodeficiency virus (HIV) disease (B20-B24)
Malaria (B50-B54)
Other and unspecified infectious and parasitic diseases and their sequelae (A00, A05, A20-A36, A42-A44, A48-A49, A54-A79, A81-A82, A85.0-A85.1, A85.8, A86-B04, B06-B09, B25-B49, B55-B99)
*Malignant neoplasms (C00-C97)
In situ neoplasms, benign neoplasms and neoplasms of uncertain or unknown behavior (D00-D48)
Anemias (D50-D64)
Diabetes mellitus (E10-E14)
*Nutritional deficiencies (E40-E64)
Meningitis (G00, G03)
Parkinson's disease (G20-G21)
Alzheimer's disease (G30)
*Major cardiovascular disease (I00-I78)
Other diseases of circulatory system (I80-I99)
*Influenza and pneumonia (J10-J18)
*Other acute lower respiratory diseases (J20-J22)
*Chronic lower respiratory diseases (J40-J47)
Pneumonitis due to solids and liquids (J69)
Other diseases of respiratory system (J00-J06, J30-J39, J67, J70-J98)
Peptic ulcer (K25-K28)
Diseases of appendix (K35-K38)
Hernia (K40-K46)
*Chronic liver disease and cirrhosis (K70, K73-K74)
Cholelithiasis and other disorders of gallbladder (K80-K82)
*Nephritis, nephrotic syndrome and nephrosis (N00-N07, N17-N19, N25-N27)
Infections of kidney (N10-N12, N13.6, N15.1)
Hyperplasia of prostate (N40)
Inflammatory disease of female pelvic organs (N70-N76)
*Pregnancy, childbirth and the puerperium (O00 –O99)
Certain conditions originating in the perinatal period (P00-P96)
Congenital malformations, deformations, and chromosomal abnormalities (Q00-Q99)
Symptoms, signs and abnormal clinical and laboratory findings, not elsewhere classified (R00-R99)
All other disorders (Residual)
*Accidentals (unintentional injuries) (V01-X59, Y85-Y86)
*Intentional self-harm (suicide) (X60-X84, Y87.0)
Assault (homicide) (X85-Y09, Y87.1)
Legal intervention (Y35, Y89.0)
*Events of undetermined intent (Y10-Y34, Y87.2, Y89.9)
Operations of war and their sequelae (Y36, Y89.1)
Complications of medical and surgical care (Y40-Y84, Y88)

***Indicates diagnosis category contains subcategories**
ICD 10 codes are in parentheses

Assessment Initiative Cooperative Agreement 5U38HK000051-02.

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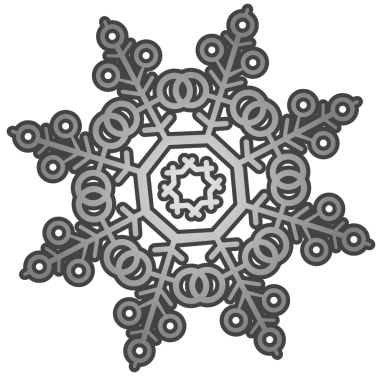

Palliative Care – Evolution of a Vision

Anna Wheat

Dame Cicely Saunders, MD, OM, DBE, established the first *modern* hospice, St Christopher's, in 1967 in London. Prior to that time, terminally ill hospitalized patients did not "fit" the mission of hospitals – to cure. Physicians equated terminal illness with failure; thus terminally ill patients were often "placed… at the end of the hall."[1] Dame Cicely took it upon herself to help these patients, usually suffering from advanced cancer, with the goal of facilitating dignity and comfort as death approached. She founded St. Christopher's Hospice, believing that dying people have the right to an improved quality of life, using pain control and social and emotional support to achieve psychological and spiritual contentment. Dame Cicely's vision for end of life care soon spread to the United States where it became known as "palliative care."

Dame Cicely's apprentice at St. Christopher's, Florence Wald, MN, brought the new vision for end of life care to the United States, establishing the Connecticut Hospice in 1974. Known thereafter as "the mother of the American hospice movement," Ms. Wald not only established an inpatient hospice similar to St. Christopher's, but also developed the first home care program for terminally ill patients in the United States.[2] From these small beginnings the American hospice movement grew rapidly. By the year 2000, hospice organizations in the US were serving an estimated 105,500 patients annually — 18,500 inpatients, and 87,000 home care clients.[3]

Since its articulation at St. Christopher's four decades ago, the basic, comprehensive approach to palliative care has not changed, but the definition of palliative care has evolved – it has broadened significantly – as have unresolved issues of palliative care.

AN EVOLVING CONCEPT

In the 4th century, hospices were initially places for travelers to rest. Then in the 19th century, churches in England and Ireland began to establish places for dying persons called "hospices." When St. Christopher's hospice was established, it differed from existing hospices in the United Kingdom primarily because Dame Saunders' vision of end of life care was broader than the established vision. Dame Cicely's hospice was more than a place to gather up dying people; its goal was *to improve the quality of remaining life* (including spiritual and psychological wellbeing) by providing strategic supports, such as effective pain control.

In 1974, seven years after the founding of St. Christopher's hospice, and shortly after the establishment of the first hospice in the United States, The **International Work Group on Death, Dying and Bereavement (IWG)** was founded. IWG is an organization that seeks to advance and nurture the development of the field of palliative care through thanatology, the study of human death. In 1979, IWG officially adopted a definition of palliative care.

> A service that provides all the essentials of care needed by those who face a terminal illness; allowing palliative and curative treatment, care for the family, and services at home and in inpatient settings. The combined skills of an interdisciplinary team must bring care to the patient and family in the context of their life and values.[4]

In addition to "the care needed by those who face a terminal illness," the IWG's vision squarely addresses "care for the family." Family members of terminally ill patients frequently become caregivers, encountering a gamut of problems—disrupted schedules, fatigue, loss of wages, fear of impending loss, and anxieties related to patient care.[5] Absent financial assistance and psychological counseling, the caregiver's role can be quite overwhelming.[6]

Furthermore, the IWG promotes a *team approach* to the planning and delivery of services. Individual needs – and wants – of terminally ill patients and caregivers are quite diverse, and change over the course of a terminal illness. Therefore, teams designed to support patients and caregivers must incorporate a diverse—and flexible—set of skills. Medicine, nursing, social work, and spiritual counseling are commonly in the mix.

By 1990, Dame Cicely's concept had spread to many nations. In recognition of this *fait accompli*, the **World Health Organization (WHO)** released its own definition of palliative care.

> The active total care of patients whose disease is not responsive to curative treatment. Control of pain, other symptoms, and of psychological, social, and spiritual problems is paramount. The goal of palliative care is achievement of the best possible quality of life for patients and their families. Many aspects of palliative care are also applicable earlier in the course of the illness, in conjunction with anti-cancer treatment.[7]

The WHO defines the goal of palliative care as "the best possible quality of life for patients and their families," expanding the reach of palliative care to "earlier in the course of the illness, and thus, to patients with *potentially* terminal illnesses. The WHO definition recognizes that patients undergoing *curative treatments* may benefit significantly from *palliative care*.

Since 1990, the WHO has been an essential partner in the palliative care movement. In 2002 they revised their official definition of palliative care.

> An approach that improves the quality of life of patients and their families facing the problems associated with life-threatening illness, through the prevention and relief of suffering by means of early identification and impeccable assessment and treatment of pain and other problems, physical, psychosocial and spiritual.[8]

Two important revisions are incorporated in the WHO's new definition. First, "terminal illness" is replaced by "life-threatening illness." Superficially, this modification may appear to be slight, but its implication is nothing short of revolutionary, broadening the reach of palliative care to all people suffering from chronic illnesses. By so doing, the WHO's definition has been brought into line with recent medical advances. Diseases formerly considered to be "death sentences," such as cancer, cardiac disease, and infection with HIV, are now manageable. Second, "relief of suffering by means of early identification" has been added. Thus, palliative care is envisioned as *preemptive*, as well as responsive. In one bold stroke, the WHO affirms that end of life problems have significantly earlier origins, and that treating them early enhances management throughout the course of illness.[9]

Building on the WHO's definitions of palliative care, the **National Consensus Project for Quality Palliative Care (NCP)** developed consensual clinical palliative care guidelines in 2004. The project was launched by 5 organizations committed to end-of-life issues: the American Academy of Hospice and Palliative Medicine, the Center to Advance Palliative Care at the Mount Sinai School of Medicine, the Hospice and Palliative Nurses Association, the Last Acts Partnership, and the National Hospice and Palliative Care Organization. The essence of all previous definitions is incorporated in the NCP's vision of palliative care.

> The goal of palliative care is to prevent and relieve suffering and to support the best possible quality of life for patients and their families, regardless of the stage of the disease or the need for other therapies. Palliative care is both a philosophy of care and an organized, highly structured system for delivering care. Palliative care expands traditional disease-model medical treatments to include the goals of enhancing quality of life for patient and family, optimizing function, helping with decision-making and providing opportunities for personal growth. As such, it can be delivered concurrently with life-prolonging care or as the main focus of care.[10]

This is by far the most comprehensive definition to date. Dame Cicely's original vision of care for the dying is now recognized as nothing less than a *philosophy of care*, operationalized through effective management of pain and other distressing symptoms, while incorporating psychosocial and spiritual care according to the needs, values, beliefs, and cultures of patients and their families.

Palliative Care *vs.* Hospice Care

In 1967, Dame Cicely Saunders redefined hospice care by incorporating an expansive, patient-centered approach to the care of terminally ill patients, focusing on dignity and comfort. In essence, St. Christopher's Hospice *defined* palliative care with clear vision, specific goals, distinct methods, and of course, a successful, functioning model. Since that time, hospice care has become a distinct *subset* of palliative care, as the latter concept has expanded. Palliative care is now broadly valued, not only for terminal illness, but also throughout the trajectory of all serious illness. Hospice care incorporates all of what palliative care (now) is, but continues to focus on the special needs of patients and caregivers *as end of life approaches*.

Issues of Palliative Care Today

The foremost issues of palliative care today are *accessibility* and *utilization*. In Rhode Island, as elsewhere in the United States, palliative care is largely inaccessible and greatly underutilized.[11] Cancer patients, for example, are often unaware of palliative care options, and those who are not considered to be terminally ill do not have access to a palliative care team; minority groups may be distinctly disadvantaged in this regard.[12] The Rhode Island Comprehensive Cancer Control Plan, developed by the Partnership to reduce Cancer in Rhode Island and released in 2007, aims to increase access to palliative care by the year 2012.

As well, issues have always swirled around the use of *palliative sedation*—the use of sedatives to relieve extreme suffering by inducing unconsciousness (deep sleep) while the disease takes its course, eventually leading to death.[13] Advocates of palliative sedation believe it should be used to avoid unnecessary suffering, even if sedation hastens death (by suppressing respiratory function).[14] Opponents argue that palliative sedation is a euphemism for euthanasia.[15] This issue is certainly not new. It is discussed more openly now, however. Let us hope that this new willingness to confront the issue will benefit the patient, as Dame Cicely, who strongly opposed euthanasia,[16] would have insisted. In this regard, perhaps it is fitting to quote the following from Dame Cicely's obituary:

> Many years ago, in response to a question at a symposium about the prospect of death, Saunders declared that she would hope for a sudden demise but would prefer to die—as she has—with a cancer that gave due notice and allowed the time to reflect on life and to put one's practical and spiritual affairs in order.[17]

Dame Cicely died peacefully as a patient of St. Christopher's Hospice in 2005.

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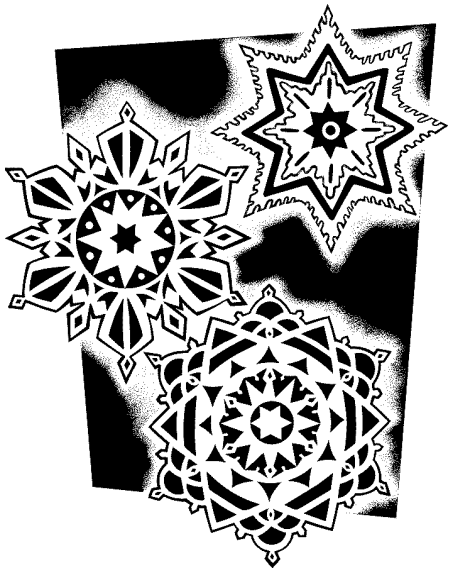
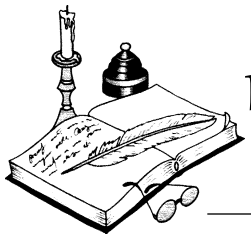
The author has no financial interests to disclose.





Physician's Lexicon

Medical Words in Extremis

The English language is said to embrace a bit over one million words; but since an average citizen can get along readily in urban life with an active knowledge of but 20,000 words, what are the purposes and reasons for survival of the remaining 980,000 terms - beyond burdening the pages of hernia-producing dictionaries?

Lexicographers claim that each learned profession adds its own contrived vocabulary to the general pool of words; in the case of medicine this amounts to an arcane collection of about 85,000 technical words. We are often asked by the lay public: Are all of these polysyllabic words really necessary? With all of your professional commitments to economy and brevity of expression, why do you not have a Committee on Retiring Medical Lexicography, its purpose to prune and discard your older terms which have been replaced by newer and more accurate words? To be precise, the Committee should ruthlessly discard those idle, antiquarian medical words for which even historians can find no use.

In truth, medical dictionaries are suffused with words that only a lexicographer might understand or cherish. Many [particularly psychiatric terms] have long since been supplanted by newer, more accurate terms. Consider, for example, an arbitrarily gathered battery of ancient words culled from a standard medical dictionary:

Innidiation: an archaic term for neoplastic metastasis.

Allotriogeustia: an ancient term for abnormal taste preferences; this word is linked to **allotriophagy**, the eating of bizarre foods [such as the earth-eaters of South Carolina].

Athymia: An old psychiatric diagnosis of an individual displaying an absence of affect; one showing extreme indifference [the word is unrelated to the thymus gland].

Thymergasia: An abnormal psychiatric state, usually mania.

Paralyssa: an acute form of rabies associated with bat-bites. Lyssa, incidentally, is the Greek goddess of rabies, mad dogs and human rage.

Nosophilia: A morbid urge to be ill.

Amaxophobia: A pathologic fear of riding in automobiles.

Anerythroblepsia: Obsolete term for **anerythropsia** which, in turn, is an ancient term for **protanopia** which defines blindness to the color red.

These words, if not dead, are certainly moribund. I doubt that any member of the Rhode Island Medical Society, no matter how erudite, could give accurate meaning to these verbal dinosaurs without retreating to ancient reference books.

– Stanley M. Aronson, MD

Rhode Island Department of Health
David Gifford, MD, MPH
Director of Health

Vital Statistics

Edited by Colleen Fontana, State Registrar

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

Underlying Cause of Death	Reporting Period			
	January 2008	12 Months Ending with January 2008		
	Number (a)	Number (a)	Rates (b)	YPLL (c)
Diseases of the Heart	225	2,679	250.4	3,407.0
Malignant Neoplasms	188	2,277	212.9	5,960.0
Cerebrovascular Diseases	33	384	35.9	592.5
Injuries (Accidents/Suicide/Homicde)	46	523	48.9	8,365.5
COPD	61	444	41.5	342.5

Vital Events	Reporting Period		
	July 2008	12 Months Ending with July 2008	
	Number	Number	Rates
Live Births	1,121	12,775	12.0*
Deaths	737	9,929	9.3*
Infant Deaths	(4)	(77)	6.0#
Neonatal Deaths	(4)	(58)	4.5#
Marriages	710	6,109	5.7*
Divorces	223	2,839	2.7*
Induced Terminations	350	4,897	383.4#
Spontaneous Fetal Deaths	68	829	64.9#
Under 20 weeks gestation	(62)	(757)	59.3#
20+ weeks gestation	(6)	(72)	5.6#

(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 1,067,610

(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

THE RHODE ISLAND MEDICAL JOURNAL

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Issued Monthly under the direction of the Publications Committee

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PROVIDENCE, R.I., JANUARY, 1917

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Ninety Years Ago, January 1919

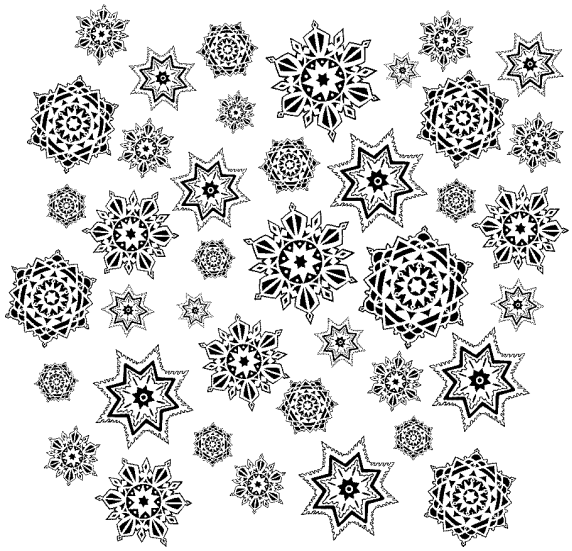
Because key staff at the Rhode Island Medical Society were serving in World War I, the Society suspended publication of its journal throughout 1919.

Fifty Years Ago, January 1959

Leo M. Davidoff, MD, Professor and Chair, Department of Surgery, Albert Einstein College of Medicine, delivered the 11th Dr. Isaac Gerber Oration: "Some Influences of the Intracranial Controls on the Roentgen Appearance of the Skull." The Journal reprinted the oration.

Shields Warren, MD, Professor of Pathology, Harvard Medical School, gave an address at the ceremony marking the cornerstone of the George Memorial Building [named for David E. George] at Rhode Island Hospital. The building was to focus on the treatment of cancer.

An Editorial, "Hospital Sepsis and the Staphylococcus," urged hospitals to adopt changes to the laundry, housekeeping, and engineering departments (for instance, "well-maintained air conditioning systems"). The Editorial urged frequent hand-washing, use of masks during surgery, and "gentle handling of tissues."



Twenty-Five Years, January 1984

Wendy J. Smith, the managing editor, contributed an Editorial, "A New Look at the Fiscal Impact of the Malpractice Premium." The total bill for premiums of physicians, employees and hospitals came to an estimated $3.5 billion in 1983; $1.655 to 1.75 billion of that went for physicians' coverage. Those estimates did not include an estimated $15.1 billion spent yearly for "defensive medicine." In Rhode Island, malpractice claims were pending against one-third of the physicians.

On the President's Page, Charles P. Shoemaker, Jr, MD, noted in "Physician Manpower in Rhode Island" that the Brown University Program in Medicine had decided to create 40 new full-time positions. "The decision….sent shock waves through the entire medical community," especially new physicians, "already struggling to survive in the face of the 'doctor glut.'" Third-party insurers feared an estimated cost for each new position of $300,000 annually. Dr. Shoemaker urged further study, to document evidence of the hypothesized glut.

Henry M. Lichtman, MD, and Stanley D. Simon, MD, in "The Doctor John E. Conley Rehabilitation Center: A Community Resource," urged Rhode Island physicians to "utilize the facilities for the benefit of their patients." The General Assembly had established the center in 1943, expressly to care for injured workers covered by Workers Compensation Insurance – "the first state-operated rehabilitation facility in the country."

Kemi Nakabayashi, Sarah C. Aronson, Michael Siegel, William Q. Sturner, MD, and Stanley M. Aronson, MD, in "Traffic Fatalities in RI: Part I – Descriptive Epidemiology," recounted the basic statistics: traffic fatalities were responsible for 17.6% of all years of life lost before age 65, and 1.4% of all deaths in Rhode Island.

John L. Margolis, MD, Anthony V. Migliaccio, MD, FACS, and Anthony J. Migliaccio, MD, FACS, in "Gastric Gullet Obstruction produced by gallstones in the Duodenal Wall," discussed the case of a 77 year –old man. The authors noted that the condition was generally managed by cholecystectomy and gastrectomy.

2008 Index

Authors

Titles

Column Key

AP: Advances in Pharmacology
CC: Creative Clinician
GPP: Geriatrics for the Practicing Physicina
HBN: Health by Numbers
IM: Image in Medicine
PHB: Public Health Briefing
PL: Physicians Lexicon
POV: Point of View

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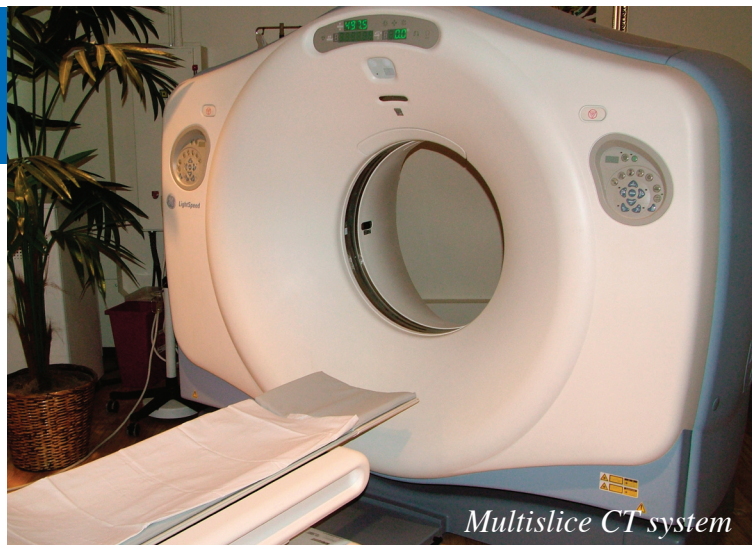
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