Surgical and medical treatment options for urge incontinence

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As the population ages, urinary incontinence is becoming an important health issue. In the United States, urinary incontinence had an estimated annual cost of $26 billion in 1995. The author presents a literature review to inform primary care physicians about the most current medical and surgical treatment of urge incontinence. Developments in medical research have produced oral medications that are increasingly effective in controlling the symptoms of urge incontinence. This advance has resulted in a decreasing need for surgical intervention. Although surgical therapy has not been proven to be as effective as oral medications in the treatment of urge incontinence, it is still considered to be a viable therapy in select patient populations.

(Key words: urge incontinence, pharmacotherapy, biofeedback, electrical stimulation, surgery, bladder augmentation)

Imagine what it would be like to be incontinent of urine and how your life would be different. Urinary incontinence is not only a medical issue, it is also a social issue with an estimated cost of $26 billion in 1995. The majority of cases of urinary incontinence can be treated with minimal medical intervention, including modifications of daily habits, treatment of the underlying medical condition, or oral medication. Surgical options are rarely considered in the treatment of urge incontinence owing to the relative success of nonsurgical intervention. Circumstances exist, however, when surgery is necessary because of the unremitting nature of the symptoms and history of the patient.

Anatomy

The bladder is a hollow organ responsible for storing urine. Three muscle layers compose the detrusor muscle; the external and internal layers are longitudinal, and the middle layer is circular. As these muscle layers descend to the bladder neck, they coalesce and form the involuntary internal sphincter. In males, the internal sphincter is continuous with the prostate. These fibers continue through the urethra in females. The external urinary sphincter is composed of striated muscle. Arterial supply to the bladder is accomplished with branches from the internal iliac artery, superior vesical arteries, and branches from the vaginal arteries in females or the inferior vesical arteries in males.

Neural supply to the pelvic urogenital organs is composed of the pelvic and hypogastric nerves. These nerves supply the bladder and urethra with afferent and efferent parasympathetic and sympathetic innervation. Pelvic parasympathetic nerves come from the gray matter of the sacral spinal cord at S2 through S4. Sympathetic innervation originates from T11 through L2 for the bladder and the urethra. Somatic innervation to the external sphincter emanates from S2 through S4 sacral segments and is carried through the pudendal nerve.

Micturition is mostly a function of the peripheral autonomic nervous system. The peripheral micturition center is located at the S2 through S4 spinal level. Parasympathetic nerves are responsible for contraction of the bladder, and the sympathetic nerves relax the bladder and contract the internal sphincter, promoting storage of urine. Ultimate control resides in the central micturition center and is located in the pontine-mesencephalic gray matter. Input is derived from the cerebellum, basal ganglia, thalamus, hypothalamus, and the cerebral cortex.

Classification of incontinence

Incontinence can be classified into three categories: stress, urge, and mixed incontinence. Stress incontinence is the type of incontinence that describes the type when one leaks urine with a cough, laugh, and sneeze. This type of incontinence usually represents a loss of pelvic floor muscle tone. Urge incontinence is the type described as feeling the sensation to urinate, running to the bathroom, and losing some urine along the way. Urge incontinence is sometimes further differentiated into neurologic and nonneurologic in etiology. If the incontinence is associated with a neurologic lesion, it may be termed detrusor hyperreflexia. If the incontinence is not associated with a neurologic lesion, then it may be termed detrusor instability. Mixed incontinence is a combination of the preceding types. Other variant types of incontinence do exist as well, such as ectopic urethra or bladder fistula. Many different classification schemes do exist regarding incontinence, and this scheme appears to be the most widely accepted.

Etiologies of urge incontinence

As previously stated, urge incontinence may be due to neurologic and nonneurologic conditions (Figure 1). Nonneurologic conditions include urinary tract infections (UTIs), radiation, interstitial cystitis, and chemical-induced cystitis. Those aforementioned conditions in some way irritate the sensory nerves supplying the bladder and stimulate premature contractions. The neurologic conditions listed hereafter will stimulate bladder contractions without an irritative process within the bladder. Neurologic etiologies include multiple sclerosis, Parkinson’s disease, cerebrovascular accidents, spinal cord injuries, nerve tumors, and some congenital neural anomalies. Urge incontinence has many different etiologies that require different modes of analysis. The next section will briefly review the different modalities available to differentiate these etiologies.

Differentiation of incontinence

History and physical examination are of greatest importance in the differentiation of incontinence (Figure 2). In taking the history, it is important to ask about frequency, dysuria, hesitancy, nocturia, and

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urgency. Critical questions that are also important concern the presence of incontinence when coughing, laughing, and sneezing. It is also important to elicit a history of UTIs, perineal instrumentation, pregnancies, neurologic history, and the degree of incontinence the patient reports.

The physical examination should include a thorough neurologic examination with digital rectal evaluation for prostate size and rectal tone. Vaginal examination is also suggested to evaluate for presence of atrophic vaginitis and cystocele. Diagnostic testing may include a kidneys-ureter-bladder x-ray study and urinalysis for UTI. A cystoscopic evaluation may also be done to determine anatomic abnormalities and presence of cancer when indicated. A postvoid residual volume should also be determined with either an ultrasound examination of the bladder or straight catheterization. In the evaluation of the unstable bladder, a very useful modality in the diagnosis is a urodynamic test, which evaluates for bladder capacity and premature bladder contractions. The sine qua non for the unstable bladder is bladder contractions at inappropriately low volumes and when attempting to inhibit a contraction. Flowmetry, videourodynamic studies, and voiding cystometry may also be used to supplement the evaluation for the unstable bladder.

Treatment options

Presently, the first line of therapy for urge incontinence and the overactive bladder is pharmaceutical (Figure 3). Pharmacological therapy is aimed at decreasing the number and intensity of premature bladder contractions. Because of the different etiologies of urge incontinence and the overactive bladder, a number of different medications have been developed. Anticholinergic medications are the most commonly prescribed and act by blocking the cholinergic receptors on the bladder to decrease contractions. Other medications and modes of therapy are used to treat the other varied underlying dysfunctions that are present. In situations when conservative therapy is unsuccessful, surgery may become necessary. The treatment of urge incontinence and the unstable bladder requires a consideration for the patient’s physical condition, social discomfort, and renal damage resulting from high intravesical pressures.

Pharmacotherapy

Advances in pharmacology have made medications increasingly effective in the treatment of incontinence. Many classes of drugs exist for the treatment of urge incontinence and the unstable bladder; they are beyond the scope of this article. Therefore, discussion here is limited to the major medications and their uses.

Micturition is completed by a relaxation of the urinary sphincter and the contraction of the detrusor muscle. Acetylcholine is released to stimulate the contraction of the detrusor. The medications in this class effect their action at different points with one effect; to decrease inappropriate bladder contractions. Anticholinergic medications such as oxybutynin chloride (Ditropan and Ditropan XL) and tolterodine tartrate (Detrol) act by decreasing the muscarinic acetylcholine actions. These medications are particularly effective in treating bladder spasms. Tricyclic antidepressants also have central and peripheral anticholinergic effects. Imipramine hydrochloride (Tofranil), a tricyclic antidepressant, is known to increase urethral pressure and improve bladder capacity. Anticholinergic medications do not treat the underlying condition in urge incontinence and the unstable bladder, but rather control the most obtrusive symptoms.

Interstitial cystitis is a poorly defined disorder that presents with urinary urge symptoms, dyspareunia, or pelvic pain. Different hypotheses have been proposed, with a variety of treatment modalities offered. At one time, treatment options included heparin instillations, dimethyl sulfoxide (DMSO), and long-term antibiotic prophylaxis. Currently, pentosan polysulfate (Elmiron) is prescribed to replace the peptigoglan layer within the bladder, maintaining a protective surface over the urothelium. Capsaicin, L-arginine, and bacille Calmette-Guérin (BCG) vaccine are also being increasingly researched regarding efficacy. Interstitial cystitis continues to be a poorly understood disease with increasingly effective treatment options.

Benign prostatic hypertrophy (BPH) is characterized by increased stromal
hyperplasia and increased smooth muscle tone in the prostate as well as at the bladder neck. To decrease prostatic smooth muscle tone, α-agonists such as tamulosin (Flomax) or doxazosin (Cardura) are used. Benign prostatic hypertrophy is not synonymous with bladder outlet obstruction. Involuntary bladder contractions are noted in more than 50% of men with BPH. For those few patients who continue to have symptoms of urge incontinence, urodynamic testing and an anticholinergic medication may be indicated.

Biofeedback
Biofeedback is a treatment modality that may be used in both stress and urge incontinence with moderate success. Although this type of treatment has been most widely studied in stress incontinence, it has been shown to be beneficial in urge incontinence. This therapy works by helping the patient to retrain the body’s response to bladder contractions and increase the intravesical pressure.

Electrical stimulation
Caldwell was the first to use nerve stimulation in 1963 for urinary incontinence. Nerve-stimulating devices have been used by placing them in the anus, vagina, externally on the perineum, and at the anterior or sacral roots. The results depend on the amount of electricity applied. Different settings are available. Lower frequency modes are adjusted for nerve inhibition, and higher frequencies stimulate nerve impulses. By placing the instruments in the vagina or anus and activating the units, it is possible to inhibit bladder contractions. At this time, though, few standards exist in the application of these units, and results have been mixed.

Surgery
Tizzoni and Foggi, in 1888, were the first to report on augmentation cystoplasty. Many changes have been made since that time, with improvements in the screening of appropriate candidates, technique, and controlling the metabolic and surgical complications of the procedure. Experience has shown that there is no perfect segment of bowel for augmentation. Stomach, ileum, and the large bowel each carries its own risks and benefits. With the many years of experience that has now accrued, our understanding of this procedure has only led to its improvement. Candidates for this procedure are those patients with unremitting symptoms from their incontinence which are unresolved with noninvasive modes of therapy. Preoperative evaluation should be undertaken in a holistic approach to consider the psychosocial as well as the physical well-being of the patient. The physiologic parameters to be evaluated are renal function, bladder dynamics, and other organ function, including heart, lung, and liver. Patients must also be evaluated for the capacity to do intermittent self-catheterization before the procedure. Intermittent catheterization after the procedure is done in 33% to 85% of patients. Patient and family education cannot be emphasized enough in these situations because many of these procedures are done in children with neurologic abnormalities.

Contraindications to the procedure include poor renal function, inability to catheterize oneself, and other significant organ dysfunction. Renal function is very important owing to metabolic abnormalities that are present from the bowel resection leading to renal failure. Inability to self-catheterize is a relative contraindication because many of the patients do perform self-catheterization after the surgery. Other relative contraindications include age greater than 65 years; poor bladder emptying, and low urethral leak-point pressures.

Outcomes of the augmentation cystoplasty vary, depending on the portion of intestinal tract used and underlying renal function. Singh and Thomas reported that if ileum was used, the patient is more likely to void spontaneously; however, the patient is also more likely to continue having symptoms of urge incontinence postoperatively. Signmoid colon when used can result in increased continent as well as increased likelihood of need to perform intermittent self-catheterization.

Postoperative outcome may also be affected by the patient’s underlying diagnosis. Among patients with neurogenic bladders, 93.6% had social continence as opposed to 84.8% of those with non-neurogenic bladders achieving social continence. Postoperative urodynamic studies revealed that bladder capacities were increased and the mean detrusor pressure did decrease at full capacity. Outcomes of these procedures appear to have a favorable result in the short term, with some increased dissatisfaction in the long term. These results also must be weighed against the reality that all these patients prior to the operation had failed conservative therapy and were seeking some form of relief.

Complications related to this procedure are multifactorial secondary to the attachment of intestine to the bladder. Therefore, complications include those related to bowel surgery, bladder surgery, and the combining of both. Patients may have continued incontinence and require self-intermittent catheterization. Recurrent UTIs are found in 37% of patients, and 15% may require long-term antibiotic therapy. Bowel habits are affected in many patients when ileum is used. Ileum is responsible for the reabsorption of water and bile, making its resection more prone...
to loose stools. Nutritional deficiencies do occur, such as vitamin B12, when a significant portion of intestine is removed. Hyperchloremic acidosis is the most common electrolyte abnormality. Normally functioning kidneys correct this acidosis without the need for medication. Complications of the enterocystoplasty vary widely, and preoperative patient education regarding all the risks and benefits is essential.

Alternatives to the enterocystoplasty attempting to improve on its limitations are increasing with time. These alternatives include the continent urinary diversions using cutaneous ileocystoplasty, bladder autoaugmentation, and seromuscular enterocystoplasty. In 1998, Geharz and Woodhouse reported on using a piece of ileum to connect the bladder to the skin as a conduit to self-catheterize. The autoaugmentation removes the detrusor muscle from the bladder urothelium. This procedure is indicated in those patients with high intravesical pressures and not necessarily for those patients with a small bladder. Seromuscular enterocystoplasty combines autoaugmentation with the enterocystoplasty. A vesicomyotomy is done, and then a piece of demucosalized bowel is placed over the urothelium. Procedures like these add to the alternatives that may be done in the place of enterocystoplasty; however, they have not been followed up for long periods like the enterocystoplasty to prove their worth.

References