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EXPERT'S CORNER: A PERSONAL APPROACH

How to address a patient with urinary incontinence

A. Gutiérrez-González*

Urology Service of the ''Dr. José Eleuterio González'' University Hospital of the Autonomous University of Nuevo León, Mexico

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According to the International Continence Society (ICS), urinary incontinence (UI) is defined as the involuntary passing of urine through the urethra. It is estimated that over 200 million people in the world suffer from this condition; a survey conducted in 2002 in different countries found a UI prevalence of 34% in Germany, compared with 15% in Spain. The elderly have a significant increased tendency of this problem. Calculations have shown that 11.8% of people between the ages of 40 and 44 may suffer from this illness, compared to 27.6% of people between 60 and 64, occurring more frequently in females than males at a 5:2 ratio.

In order to better understand the causes of incontinence it is important to comprehend the physiology of the lower urinary tract. The bladder performs two functions: to store and empty urine. In order to store urine it is necessary to have a competent sphincter and a distensible detrusor muscle. This means that both the internal sphincter and external sphincter must have greater pressure in relation to the detrusor. As the bladder is filling up, proprioceptive endings of the vegetative system send impulses toward the brain, specifically the frontal lobe, where the sense of urination becomes conscious, and then the nervous system sends inhibitor signals to the spinal cord segment S2–S4 preventing the exit of urine. When the bladder is full, a disinhibition reflex occurs; this disinhibition produces a decrease in the sphincter's pressure and a detrusor contraction, resulting in urination. Urinary incontinence can be caused by *mechanical* problems like loss of sphincter pressure or reduction in bladder distensibility, or *neurological* effects like those caused by spinal trauma, diabetes mellitus or any other neuropathy which affects the urination mechanism.

There are several types of UI:

Stress incontinence (also known as effort incontinence): When there is a sudden change in intra-abdominal pressure and the sphincter is not capable of controlling bladder pressure.
Urge incontinence (also known as hyperactive bladder): it occurs due to a loss of control of the inhibitor reflex toward the bladder, producing an urge to urinate.
Mixed incontinence: observed in patients with stress incontinence combined with urge incontinence.
Overflow incontinence: Not being able to efficiently empty the bladder, thus having an ''overflow'' with an involuntary loss of urine.
Functional incontinence: in patients with ambulation alterations, and who do not present any abnormality in bladder functioning.

* Corresponding author at: Servicio de Urología del Hospital Universitario''Dr. José Eleuterio González'' de la UANL, Ave. Madero y Gonzalitos, Colonia Mitras Centro, zp 64460 Monterrey, NL, Mexico.
E-mail address: dradriangtz@att.net.mx

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Reflex incontinence: in patients with a neuropathy explaining the loss of urine control.

When examining a UI patient we must ask directed questions, focusing on the causes predisposing the incontinence, differentiate the type of UI, as well as find out the level of incontinence. For example: whether or not the patient uses a protector, and if so, the type and number of protectors, since the extent and severity of the UI will not be the same if the patient uses a pad as a protector, as opposed to the severity of the patient who wears a diaper. After the thorough interrogation documenting the patient's history and the evolution of the urinary pathology, we will proceed to perform physical examinations, corroborating inflammatory alterations on the skin around the genital area because of the constant urination. We must observe if there are any ambulation alterations or the impossibility of walking, as well as examine the anal tone, if there is a voluntary anus contraction and if there is presence of bulbocavernosus reflex, this examination is vital since it will inform us about the integrity of the pudendal nerve.

Among the basic studies there is the urine analysis test and urine culture, in order to rule out the most frequent transitory cause of incontinence, which is urinary infection. Over the years, and having a significant functional bladder alteration, this tends to produce deterioration in renal functions with the possibility of developing hydronephrosis and/or pyelonephritis. We should also consider the possibility of developing lithiasis in the urinary tract. In women, we must consider assessing the level of cystocele or urethrocele, requesting the patient to push and observing whether or not there is a loss of urine in the process. Once these parameters are reviewed, and having diagnosed the type of UI, we ought to offer the proper treatment. If we diagnose stress incontinence, we will assess if the patient is a candidate for pelvic floor rehabilitation or a surgical procedure (urethral suspension or pelvic prolapse repair).

In the case of urge incontinence, by discarding urinary infection and without neurogenic data which explains it, the initial approach must be made with an anticholinergic medication. If the response is positive, continue with the anticholinergic for a period of 3–4 months, after this period we should stop the medication and assess if the vesical function has normalized. In the case that there is no an adequate control a urodynamic study (the golden standard for bladder neurofunctional evaluation) must be performed. This study determines different parameters: intravesical, urethral and abdominal pressure. Moreover, an electromyography must be performed, in the storage period as well as the vesical emptying stage, to try to reproduce the mechanism by which the incontinence occurs as a way to understand and assess the problem.

When we think a neurogenic origin is the cause of incontinence, it is necessary to assess the patient's neurological history which may explain it. If it is not documented it is not possible to rule out this possibility, since between 3% and 12% of the patients with a neurological problem begin with signs of UI. When inspecting the patient with a suspected functional bladder alteration, the urodynamic study will help with the diagnosis of the type of neurogenic bladder, evaluating the possibility of upper urinary tract damage. Subsequent urodynamic studies will reveal whether or not there was a response to treatment.

There are a variety of UI treatments: (a) medical, (b) rehabilitation and (c) surgical.

(a) Medical treatments include the use of anticholinergics like oxybutynin vs tolterodine. Currently, beta-3 adrenoceptors, like Mirabegron (which is not sold in Mexico) are used to reduce the detrusor muscle pressure and increase vesical capacity. Tricyclic antidepressants like imipramine help improve the contraction strength of the external sphincter and reduce stress urinary incontinence periods.
(b) Rehabilitation treatments using Kegel exercises, where the patient is shown how to contract and strengthen the perineum musculature. These are useful in the handling of stress incontinence in women with pelvic floor prolapse, as well as in prostatectomized men. It has been shown that only 40% of the patients perform the exercises correctly, most contract the abdomen instead of the perineum which worsens the symptoms. With proper guidance and training, we can help patients perform these exercises correctly. Perineum electrostimulation can also be used in order to improve muscle tone, or posterior tibial nerve stimulation for a better managing of urge incontinence.
(c) Surgical treatments include the application of botulinum toxin (botox) at the sphincter level to reduce its pressure in patients with extremely high pressures. It is also used with patients who do not respond adequately to anticholinergics. This toxin contributes to the reduction of intravesical pressure and improves storage capabilities. An anesthetic sedation is necessary for the application of botox, and the introduction of a cystoscope to infiltrate the muscular layer of the bladder. In some UI patients who do not respond adequately or have a severe incontinence level, the use of urethral tapes and slings at a urethral level may help control involuntary loss of urine. Patients who do not improve their vesical capacity with any of these interventions can undergo an augmentation cystoplasty, which consists of the use of an intestinal patch to increase distention surface, thus increasing the capacity and reducing vesical pressure, which would be the last line in the management of capacity and intravesical improvement. Those patients with an intrinsic sphincter deficiency can be closed with a sling or tape on the urethra to produce an obstruction, which will have to be managed with intermittent catheterization. Also, there is the artificial sphincter, a surgical procedure with application of a prosthesis with a urethral cuff. When pumped (at scrotum level or in the vulva's labia majora), the cuff empties and the release of urine occurs.

Neuromodulation consists of placing electrodes in the sacral nerve roots, which go to an internal computer system, which regulates and modulates the stimuli which occur in the bladder, improving urinary storage and emptying, as well as controlling the possible pain that the patient may have.

Once we have assessed the patient, made the correct diagnosis and offered the proper treatment, it is important

to give follow-up to prevent complications, improve self-esteem and avoid social stigmatization.

After ruling out correctable causes like urinary infection, vesicoureteral reflux and hydronephrosis, if the patient's symptoms persist, it is important to refer him/her to a specialist with experience in the management and treatment of these type of pathology, considering that the main cause of morbi-mortality in patients with neurogenic bladder is renal failure.

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Conflict of interest

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