gardless of other factors. Patients who are non-ASIA 'A' (not complete) do not necessarily have improved outcomes from the addition of cyclophosphamide to their regimen, and may not need the risk of cyclophosomide. They may get a benefit by just providing steroids and plasma exchange, and not going on to a regimen that includes cyclophosphamide.

We have also identified another group of patients who seem to have a selective response to regimens that include cyclophosphamide; patients with spinal cord inflammation in the setting of an autoimmune disorder. Some patients with diseases, such as Lupus and Sjogren's syndrome have transverse myelitis as a manifestation of their disease. Still other patients with transverse myelitis have evidence of immune system dysfunction without meeting criteria for specific diseases. Thus, when a patient presents with transverse myelitis we will routinely screen for a group of auto-antibodies. If these tests are positive, they are indicators that a patient will have a preferential response to regimens that include cyclophosphamide.

In the absence of these criteria, we routinely use intravenous steroids, PLEX or a combination of the two. While these recommendations are not based on controlled trials, they can be used to help guide therapy. Most important, however, is the institution of some therapeutic intervention as soon as transverse myelitis is recognized - **time is cord.** A spinal syndrome has to be recognized; the diagnosis that it is inflammatory has to occur rapidly. We have to risk-stratify the patient and pick the treatment in a reasonable amount of time in order to protect as much of the spinal cord as possible.

Managing Neurogenic Voiding Dysfunction

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Adapted from a presentation given at the 2004 Rare Neuroimmunologic Symposium

This article will review the kinds of voiding problems that can result from neuropathic conditions, such as TM and MS, as well as the range of available options for the treatment of these problems. These treatment options are focused on accomplishing three important goals. The first goal is to be sure that in issues of voiding dysfunction we are preserving kidney function. Renal preservation is the priority; ensuring that there isn't some silent problem affecting the kidneys somewhere in the background that's going to give us additional trouble. We only get two kidneys; we need to take care of them. Secondly, we want to make sure that one has adequate continence and control of elimination functions. My article is focused on bladder function, but I will also address some of the issues surrounding bowel function. Many people with TM understand that bowel function can sometimes be the more difficult problem to manage. Our third goal in treatment is to maximize one's independence in managing these issues. It takes some creativity, understanding, vigilance and patience to try and work through some of the experiments we will go through to find the most effective treatments for problems associated with TM.

This article will provide a brief review of the physiology of voiding and various strategies for evaluation and treatment planning. It will also address some of the therapy options.

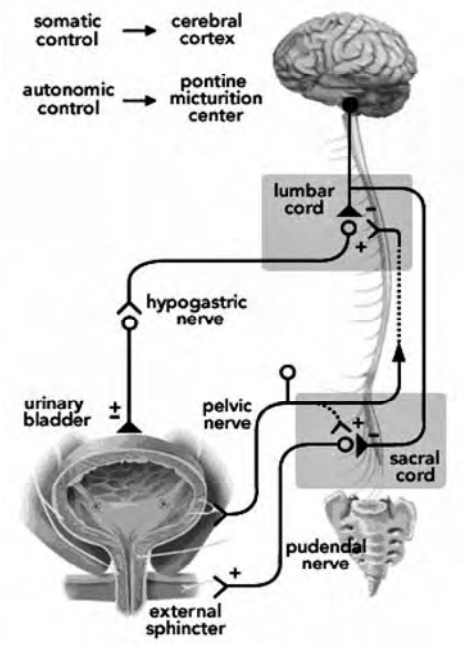
Two functions of the lower bowel and bladder are storage and emptying. It sounds simple, but it is quite complex. Both of these roles are active neurologic processes. The bladder doesn't just sit by passively and fill with urine. It actually has to have neural input in order to do that properly. The same is true for bladder emptying. These functions are driven both by reflexive and conscious control.

Through childhood, we develop or learn behaviors to coordinate this voiding cycle in response to various stimuli. We come into the world with very little conscious control of voiding. Night or day the bladder fills and spontaneously empties in a coordinated fashion. As we age, we gain greater conscious control. Between age two and age seven, most of us have learned to coordinate voiding in response to social cues, time of day, and various afferent stimuli.

This is a simplified schematic of the innervation of the lower urinary tract. The system requires intact circuitry from the brain all the way to the tip of the spinal cord. There is a great deal of interaction and there are many interconnections of the circuits along the spinal cord. The cerebral portion of this system is primarily located in the anterior frontal gyrus. Some people who have experienced a stroke (post CVA) can have difficulties from changes in this area of the brain. Likewise, there can be blood flow changes in the brain, in connection with MS and various neuro-degenerative processes, that can affect voiding function in subtle ways.

These brain functions are orchestrated through the pontine micturition center. This is the coordinating system in the brain. The system is then patched through the sacral reflex arcs through S-3 and S-4. These are located near the base of the spinal cord. As I noted

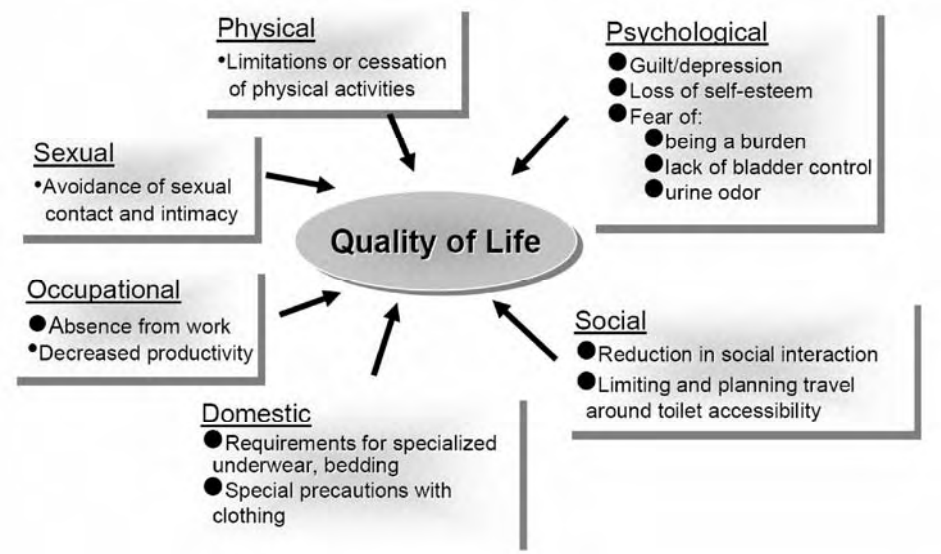
Innervation of the lower urinary tract: control of micturition



previously, these are mediated by the autonomic functions; the autonomic leads to the parasympathetic and the sympathetic nervous systems. The parasympathetic system drives bladder contraction and emptying while the sympathetic side is responsible for relaxation and urine storage. This is in turn wired into an afferent feedback system, to give us some interplay with our environment. Unfortunately, the bladder doesn't have a wide range of sensations to give us. It gives the following messages: I have to go, I really have to go and I have pain. That's about as subtle as it gets. Trying to make sense of those messages in the context of everything else can be a challenge.

The length of these circuits, the complexity of two limbs, and involvement of both conscious and autonomic functions create a complex system with many places for miscue. I am always impressed by the variability of function in patients I see in the TM Center. There can be many subtle or severe changes and the patchy nature of TM can be tricky.

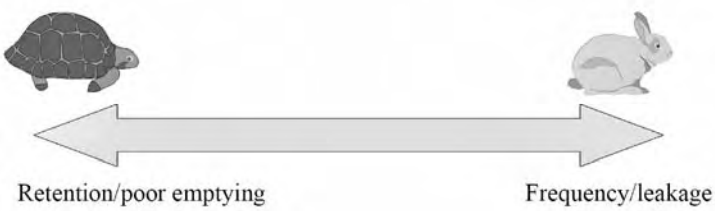
Impact of Bladder Dysfunction on Quality of Life



With voiding, timing is everything. With normal nerve function there is a balance between inhibition and facilitation. The graph represents the two extremes. On the one end, insults in the neural circuitry lead to poor emptying or at the extreme, urinary retention. The system is too inhibited. On the far end there is urinary frequency and reflex voiding without our participation or consent. The system is over facilitated. Our goal is to get back to the normal balance.

The bladder can be pushed in either direction from injury caused by TM or MS. Treatment starts by determining which end of the spectrum the damage has moved the patient.

Voiding dysfunction; timing is everything



Regardless of where the patient is on this continuum of dysfunction, our treatment strategies focus on making the system cooperate in a much more friendly way in order to restore quality of life.

The degree of bladder dysfunction or pelvic dysfunction does not always mirror the other deficits in TM. There can be preservation of large limb motor function and balance with a real discoordination of pelvic function; and the complete reverse can also occur. The degree of bladder dysfunction does not always mirror recovery either. There is a capacity for nerves to regrow and rewire some of the cortical function to try and heal some of the function. I have great hope that we will find ways to push the healing process forward. My role today is to get around the damage that has been caused and to find ways to get past the effects. It would be preferable to be able to cure those effects; to fix the circuitry rather than the manifestations of the impaired circuitry. I think we will get there.

It is important to highlight the impact of bladder dysfunction on quality of life. Bladder dysfunction can affect so many different areas. Physical activity may be limited. There can be occupa-

tional issues regarding adequate toileting facilities or permission for too frequent voiding. There are domestic issues involving the expense of using special underwear, bedding, pads and clothing. Bladder problems can seriously limit social interaction and travel opportunities; people with these issues may withdraw from these social connections. Finally, there are psychological implications including loss of self-esteem and the fear of accidents. These psychological issues are often exacerbated by depression associated with these neural effects.

In order to treat a problem one has to first define it. Then one can begin to think about strategies for improvement. The process begins with finding a urologist who has concern for these conditions. It is important to find a doctor who is willing to collaborate, who is enthusiastic about taking on these issues, who has a focus in their practice on these neurological issues and who has the mindset to find comprehensive solutions. Not every one does, but there are many urologists in the country and it's worth requiring all of these criteria of your physician.

The evaluation process begins with a number of screening questions and taking a medical history. We want to understand certain issues, such as the frequency of voiding, what prompts leakage, what is the experience of voiding, if there is any pain, or if there is night time wetting. We perform a physical examination to determine the neural integrity of the pelvis, focusing primarily in the sacral region, perineum, and peri-anal region. We conduct a urinalysis, looking to exclude such issues as infection, chronic cystitis or stones. It is important to maintain the general or overall urologic health of the patient while we try to get into the subtleties of the neurological problems.

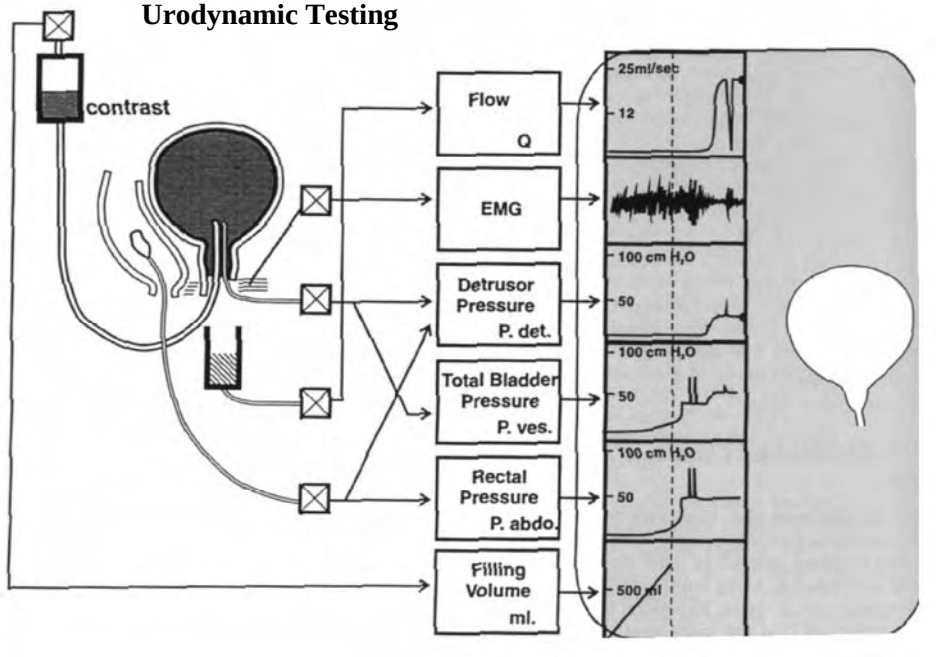
One of the most helpful tools we can use involves keeping a voiding diary. We ask the patient for a period of twenty-four to seventy-two hours to write down their experience of toileting: the date, the time, the amount voided, and some cataloguing of leakage episodes, if these occur. This information provides a picture of where we are starting from and what improvement we need to achieve. This information also helps the patient in thinking about benchmarks. The patient can consciously identify where they are in the process and with diligence, effort and practice, what they might achieve in the way of improvement.

In thinking about this process, it is instructive to consider this in terms of the original toilet training process. This isn't something that is accomplished automatically or overnight. As a young child, we actually have to work at it. If we injure that system later on in life, we have to go back and replicate that phase and relearn the process. We know from experimental work that there is axonal sprouting or some neural plasticity after injury; the system is driven to try and get back to normal function. For any of you who have played a sport or a musical instrument, you appreciate that this learning requires a lot of repetitive action; the learning requires effort. In the background there is a lot of neural change going on, and we can influence and drive this change with hard work and commitment.

There are additional sophisticated tests that can be done. Post-void residual checks provide useful information and can identify how well or how efficiently the system is working. Urodynamic testing is a functional study; the real "get under the hood" type of test for bladder function. For patients with neural injury, it is probably the most important component of the evaluation. Cystoscopy has a limited role in the evaluation. This visual examination of the bladder interior does not tell us much about the function of the system, but it often is done to exclude other potential problems.

This graphic is a depiction of a urodynamics lab. A video urodynamics test basically involves an x-ray unit, a C-arm fluoroscopy unit, a computer or CPU system, a flow meter, and a set-up to measure the pressures inside of the bladder. The test involves catheterization of the bladder and the rectum to measure abdominal pressure and bladder pressure during a recapitu-

Urodynamic Testing



lation of the voiding cycle; the filling of the bladder and the emptying of the bladder. We get a lot of information from this test. We can measure the flow rate; how well and how quickly the bladder empties. Simultaneously, pressure measurements identify if there is bladder weakness or if there is obstruction or discoordination. We can measure an EMG signal to assess whether the neural control of the sphincter or external valve is intact and to determine if it is over active or under active. We then obtain measures of filling to understand bladder capacity. The C-arm unit gives us an actual picture of the system and tells us whether there is reflux into the kidneys or if there is a diverticulum. This test provides us with a range of information that helps us to understand what is going on.

The need and importance for urodynamic testing stems from the fact that the bladder is a poor witness. We are able to see the manifestations of some symptoms, but that doesn't always tell us what's really going on physiologically, and especially in the context of a neuropathic problem. This is really the only way one can get at the heart of the matter. We can define the accommodation or compliance of the bladder; how elastic is the bladder? Does it respond in the relaxation or storage phase the way it should? If it doesn't, this is one of the problems that can cause harm to the kidneys. This is not common, but we need to be on the lookout for this issue. We are trying to assess sensation during the filling process and voiding process. We are determining residual volume, the capacity of the bladder and whether or not there is spontaneous activity. We are looking at the coordination and competence of the sphincter valves. At the end of the study, we are assessing the voiding pressures and flow efficiency. This test allows us to understand the symptoms and their causes.

When it comes to therapy options for bladder dysfunction, there is not a "one size fits all." This is particularly the case with the neuropathic issues associated with TM. We need to match therapy with the individual's needs and abilities. There is a wide range of possible abilities based on the effects of this condition and each situation is unique. Again, we are looking to assemble a therapy strategy that addresses our primary goals: preservation of kidney function, establishment of continence or adequate emptying; and maximizing independence and quality of life. It is important to keep in mind that we are using management strategies at the present time, and not a cure strategy. We don't yet have the ability to cure the neural damage which causes bladder dysfunction. There are things we can do to help the body heal itself, and to implement a management strategy that, at the least, keeps the system functioning as normally as possible. Over the course of time, if there are breakthroughs and discoveries, we want the system primed and ready to accept restorative therapies.

Finally, therapy strategies have to include the caregivers. As many of you know, this is often times a team effort. Management recommendations have to include a clear understanding of what will be required, and who will be required to assist in the care. All of these factors need to be considered and discussed with the whole team in order to arrive at the best and most effective plan. If the physician has not adopted this practice, the patient and caregivers need to advocate for this approach.

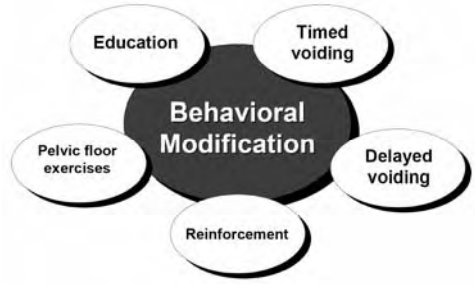
The basic principles for bladder management are the same for pediatric and adult cases. The field of urology has separated the specializations with some practices focused on adults and some focused on pediatric urology. From a neuro-urology standpoint there is a tremendous amount of overlap between the pediatric and adult situations. One of the significant differences is that for young people, there is a much greater degree of neural plasticity; the ability for those systems to rewire and heal. It is for this reason I think we should exercise a degree of conservatism in our treatment decisions knowing that there is this storehouse of potential for change over a long time horizon. We should try to manage problems to create stability for as long as possible, and to see if this potential for improvement is realized as these children develop.

I need to mention that some of the strategies I will be talking about are off-label. This doesn't mean that these strategies are not acceptable. I believe the approaches I am presenting are safe. We have been using them for years and continue to use them.

Referring back to the spectrum of neuropathic voiding dysfunction, most people with problems tend to end up on one side of the fence or the other. People are either on the poor emptying and retention side or they are on the hyperreflexia and overactive side. I am going to describe therapies for each, beginning with the hyperreflexia side. This over activity can range from urinary urgency and frequency to urge incontinence.

The cornerstone and initial approach is behavioral modification and this is true also for people who don't have a neuropathic source for their voiding dysfunction. This behavioral modification is a very diligent and directed effort at trying to coordinate, relearn or retool the toileting processes. This includes pelvic floor retraining and reeducation. I will talk about Kegel's exercises and a number of medications that can be used with oral, transdermal, or intravesical delivery systems.

There is a role for biofeedback, electrical stimulation, magnetic stimulation,



and physical therapy. There are experts in pelvic floor rehabilitation, and there appears to be some benefit from this approach. I will also speak briefly about sacral nerve stimulation.

There is growing enthusiasm for the use of botox injection into the bladder or sphincter in patients with neuropathic voiding dysfunction. This treatment may last 9-12 months and can be re-dosed for continued benefit. Experimental work is ongoing.

Finally, surgical techniques, such as augmentation cystoplasty and urinary diversion, may be used. As a surgeon, I don't want to imply that surgery is where these conditions should end up or that an operation is the answer for everything. There is, however, a definite role for surgery in specific situations, after thoughtful management and lots of discussion. When done well, these operations work and can really make a difference in people's lives.

Behavioral modification does not cost anything and it is fairly simple to do. It is simple but it is not easy and it is time intensive. The behavioral modification platform includes all of the components that are identified in this graphic. It involves a significant amount of education. Unfortunately, the medical system today doesn't provide for a lot of time that is required to perform this education. I am always impressed by how helpful it can be to simply sit down with someone and tell them how the system is supposed to work, where it is not working, and how to try and get back to a place where it does. The capacity within us to consciously bend this system to our will cannot be underestimated.

Behavioral modification includes pelvic floor exercise or the classic Kegel contraction.

This contraction of the pelvic floor muscles was initially developed by Kegel in the fifties as a therapy for stress incontinence. It may or may not be really suitable for stress incontinence, but it does have a role in terms of trying to restore and re-coordinate the circuitry between the brain and pelvic nerves. There is a reflex built into the pelvis; when you activate those muscles, it tries to turn off bladder contraction and lessen the misbehavior.

Timed voiding or delayed voiding can be helpful. This is a conscious effort to stretch out the period of time before spontaneous wetting episodes or the frequency sensation gets the better of us. This process involves a tremendous amount of practice, thus the reference to the reinforcement mode. Part of the behavioral modification process involves having the patience to continue the exercise, while the forces within us try to heal those circuits to the extent that they can. Hopefully, through restorative therapies, such as stem cells, we will find ways to drive this process more quickly and more efficiently. In the meantime, there is a great deal of potential within us that can drive this healing to restore function.

On the medical side, while the medicines have a variable effect, they do have their role in therapy. The muscarinic receptor family (Chapple CR, Urology. 2000; 55:33-46) is the primary target for treating bladder overactivity. The antimuscarinic agents used for the treatment of overactive bladder include Tolterodine (Detrol); it comes in a sustained release form (Andersson KE. BJU Int. 1999; 84:923-947). Oxybutynin comes in a sustained release oral form, a transdermal or patch form, and there is work being done to deliver the medicine intravesically. This was the original of these medications developed in the seventies. It is still around and still effective. Darifenacin, Solifenacin and Trospium are also effective.

I am optimistic and hopeful about medical therapy. This is the next great horizon in urology and voiding dysfunction, both for those with neuropathic conditions and for the general public. They are not always a cure, but in the context of the complete package there is a role for these medications in either modulating or fixing these issues.

Functional electrical stimulation is a behavioral or physical therapy (Resplande J, et al. Neurourol Urodyn. 2003; 22(1)24-8; Smith JJ 3rd. J Urol. 1996 Jan; 155(1): 127-30). This therapy can be done in the doctor's office and at home. There is no exact regimen established, for instance as to the duration of the therapy. The mechanism of benefit is not clearly understood. It can help to stimulate or trigger some of that reflex function by allowing the circuits to awaken. There are objective and subjective success rates of six to eighty percent for people with overactive bladder disorders. The neuropathic group would be considered a subset of these disorders. The follow up on these studies is generally short and the durability of results is somewhat variable and unproven. In considering therapies, this is certainly worth the try; there is no downside and it might help. This therapy is fairly non-invasive and is a reasonable expense.

It is also possible that the success rates may mirror the placebo effect. The placebo effect may actually play a very positive role in therapeutic approaches to treating neuropathic void-

ing dysfunction. It is difficult to control for these effects because of the influence and impact of cortical function in this process. Therapeutic success cannot be measured in a purely objective fashion. The enthusiasm, motivation and commitment of both the practitioner and patient play a critical role in arriving at positive outcomes from these therapies. So, while we might attribute success to a placebo effect, it is the case that the placebo effect in this situation, if that is the right term, is real and positive and powerful. In putting together a treatment package, what we bring in our conscious thoughts about this process is important and has a role to play.

Sacral nerve stimulation is a therapy used to treat urinary urge incontinence; significant symptoms of urgency-frequency. In some situations, it may also be helpful in treating idiopathic urinary retention. Referring back to the spectrum or continuum of neuropathic voiding dysfunction, this is a therapy that, in its greatest utility, can push those two ends back toward the middle. I have great enthusiasm for this therapy, because there are relatively no risks and the potential benefits can be substantial; it may help and won't hurt. When it works it can be a homerun, and when it doesn't, about the worse thing that happens is that people are disappointed.

The therapy I am describing is an off-label use. It is an implantable, programmable, neurostimulation system. The therapy requires two stages. The first stage is a test stimulation procedure; it is a temporary test that can last seven to ten days or longer. If this test proves successful, a device similar to a pacemaker is implanted that can deliver stimulation to the pelvic nerves.

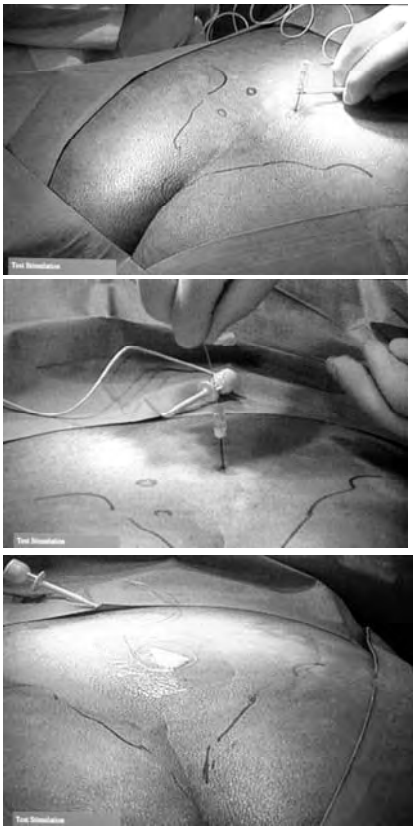
These are a few intra-operative photographs showing how we access these nerves. The patient lies on their stomach, prone. In the area of the lower spine and pelvis is the sacral foramina; an opening below which is found the sacral or pelvic nerves. These nerves drive pelvic function; they go to the bowel, the bladder, and sphincter area. It is possible to get a needle down in that area and a lead that can then stimulate the nerve. It doesn't pierce the nerve; it just has to sit near enough that it can trigger those responses.

This image is a sagittal view of that sacral location. We access the nerves at S3 and at S4. The image shows the tip of the coccyx bone. This area is the pelvis. The spinal cord ends here and the nerves of the cauda equina traverse down in a little sandwich of bone. If one can get those leads close enough, the stimulation can be delivered.

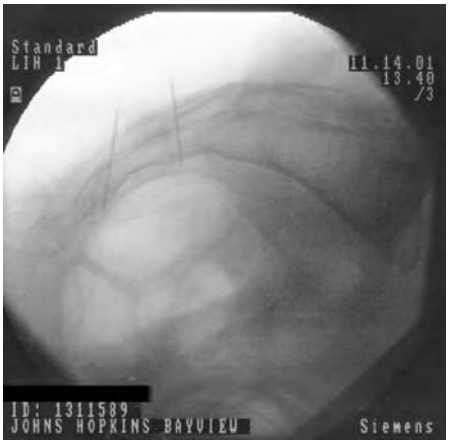
This is a photograph of the device; it is identical to a cardiac pacemaker.

This is what the device looks like when it is implanted. It is placed up near your back pocket or where your wallet would be, in a nice cushioned area so that you are not leaning on it. It basically has no restrictions. If you have this device, about the only thing you can't do is have an MRI or use diathermy; so there is not much in the way of exclusions.

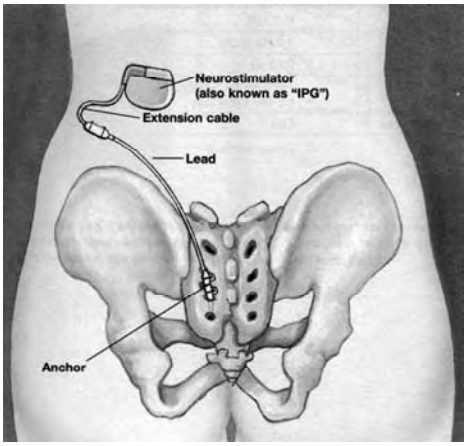
PNE (peripheral nerve evaluation)



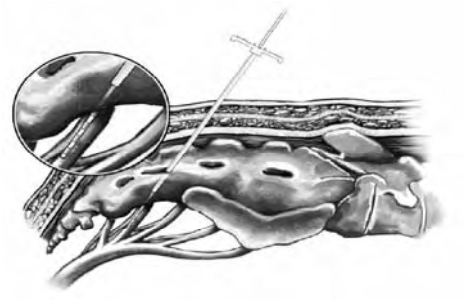
Sacral nerve localization



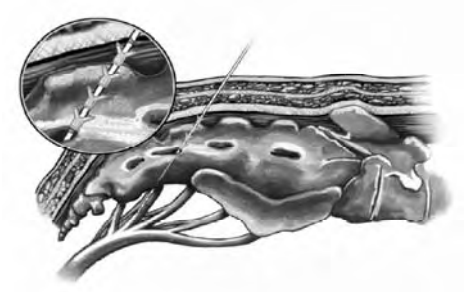
Permanent pulse generator



Tined Lead with Introducer



Tined Lead with Tines Deployed

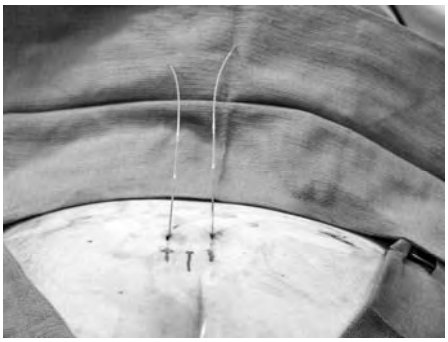


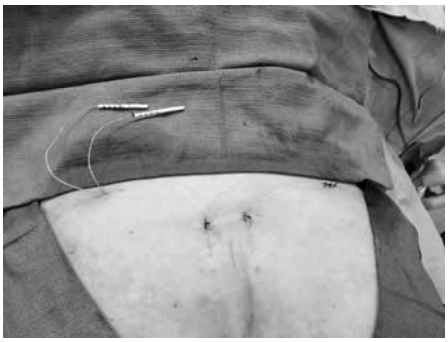
This schematic shows the lead tunneled under the skin; it dwells nearby one of those nerves. The current generation of this lead can be put in with a technique that is about as simple as putting in a central line. It used to involve something that looked a lot like a laminectomy and now it involves only a little centimeter stab incision.

It is placed near to the nerve and the stimulating locations. The lead is held in place by some tying so it moves with the person. It doesn't need any anchorage; it self-anchors and there is no migration.

The photographs are from the test phase; after the procedure is completed. I usually put two leads in because not all nerves respond in the same way. If the therapy doesn't work, you open up these little incisions and pull the leads out; there is no harm done. If it does work, then you usually take one of these out. You open up this little incision a bit more to put that pulse generator up in its location. So, that is about as extensive and as involved as is the entire procedure.

Current generation lead implant





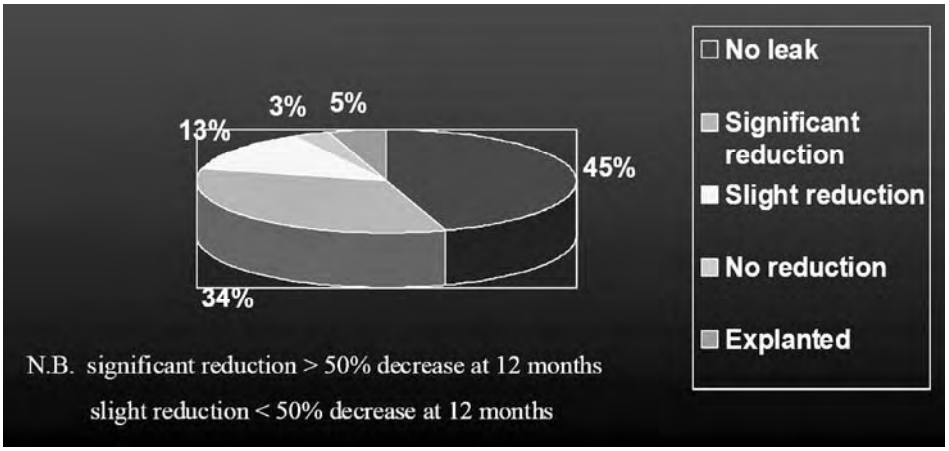
This graphic represents the results we've had from this therapy. The data in this graphic is from patients who did not have neuropathic voiding dysfunction. The caveat for people with neuropathic voiding dysfunction from TM, MS or spinal injury is that there is a greater variability of success. The approach I take in my practice is that if I test fifty people and two respond, it has made a huge difference in those two people and that is my goal. From this group as many as seventy-nine percent of folks had at least a greater than fifty percent improvement. And for those with incontinence, almost half had elimination of the voiding episodes.

What this device does is calm the system. We really don't know the entire mechanism, but it kind of balances bladder behavior. I often tell my patients that it allows the normal signals to get through and it dampens down those spontaneous, reflexive ones that create the over activity.

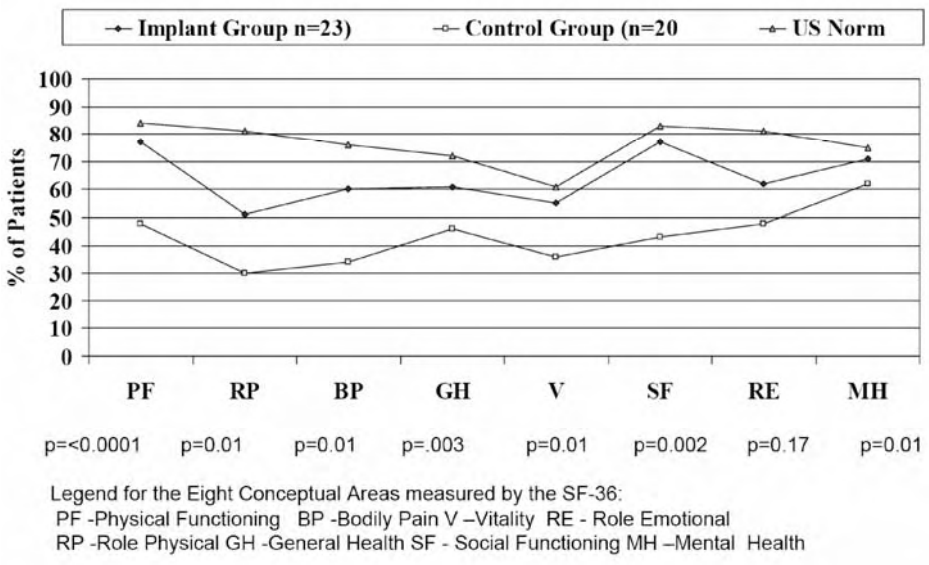
The graphic (next page) shows the results of a quality of life measure for people who have had the sacral nerve stimulation therapy. The SF-36 score is not disease specific, but a general measure of one's sense of well being. In many domains, relative to the control group, the implant group had significant improvement: vitality, social functioning, mental health, wellness, and sense of wellness. This device can make a significant difference in people's lives when it works. If it doesn't work, then it is something that one can check off the list and say, well, it wasn't the right thing for me.

The next therapy I want to talk about is denervation with botox (Botulinum-A toxin for treating detrusor hyperreflexia in spinal cord injured patients: a new alternative to antichoinergic drugs? Preliminary results. Schurch B, Stohrer M, Kramer G, Schmid DM, Gaul G, Hauri D. J Urol. 2000 Sep; 164 (3 Pt1): 692-7). Botox inhibits acetylcholine release at the presynaptic cholinergic junction; it can paralyze

Sacral nerve stimulation: Results

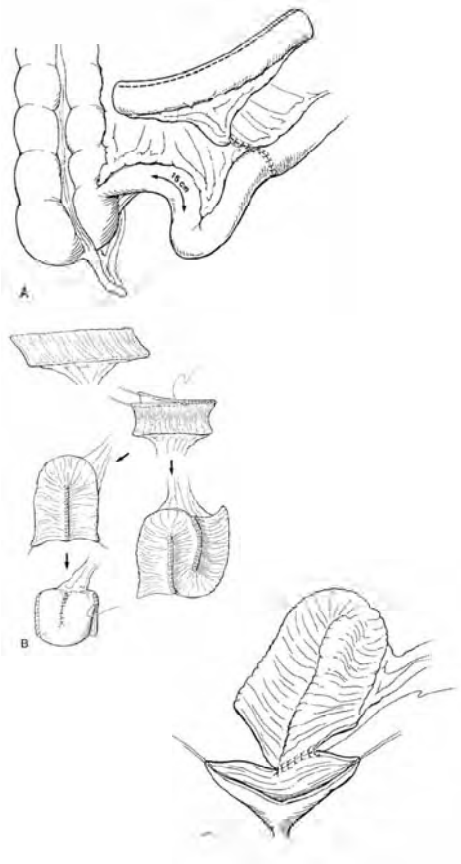


Improvement in Urgency-Frequency 6 Month SF-6 Scores



the system in a durable way. It is eventually absorbed. We have learned that injections of botox into the bladder muscle can calm unstable contractions. The technique is evolving and the protocol is in the process of being worked out; for instance, how many units to use and what sites in the bladder. The effects of this therapy can last three to nine months, and can really calm the system. When looking for something short of irreversible surgery, this may have a role in regard to buying some time.

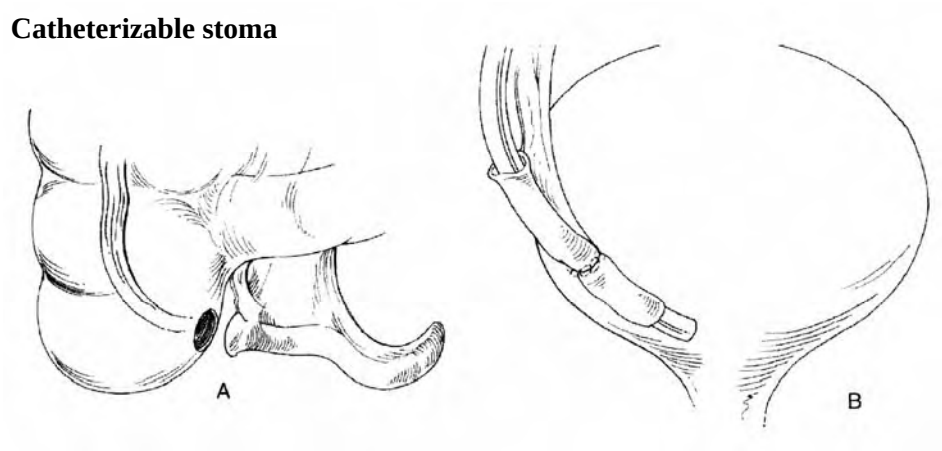
Surgery: Augmentation cystoplasty



This is a schematic of an augmentation cystoplasty. A piece of the intestine is taken out of continuity and is formed into a patch and sutured onto the bladder to make it two or three times its capacity. The result is that it lowers bladder pressures and it can eliminate leakage. There are some potential downsides and it often requires self-intermittent catheterization.

As an alternative to augmentation cystoplasty, there is a catheterizable stoma that can be constructed from the use of the appendix. It can be inserted into the bladder, and one can create a channel on the abdomen. This surgery can facilitate catheterization, rather than having to access one's own urethra. This can be especially helpful for women or if one is in a wheelchair, where it involves having to transfer, get undressed, and find a private place. With a catheterizable stoma, one merely rolls down their waistband and inserts the catheter. When these surgeries are done well, the outcomes can create significant results from the perspective of convenience and quality of life.

Catheterizable stoma



The other side of the neuropathic voiding dysfunction spectrum involves retention. The mainstay therapy for retention and the safest overall therapy is self-intermittent or clean catheterization. Whether it is desirable or not is an entirely different issue. Self-intermittent catheterization is, without question, the safest and most durable form of long- term management until we learn how to turn the bladder back on. Foley catheters, supra-pubic tube drainage have a role, but, if you can avoid these therapies, they are certainly worth avoiding. Sacral nerve stimulation has a limited role. Again, I have no hesitation in testing people, but the results haven't been overwhelming. And then, as I noted, surgery has a limited role. Ileovesicostomy, a so-called bladder chimney

and continent catheterizable stomas also may be considered, but in limited situations.

It is important for me to debunk some of the myths about self-catheterization. Self-catheterization was initiated in the early seventies. Jack Lapides recommended this approach instead of drainage tubes. He was almost drummed out of the urology world for having made the suggestion, and now it is considered the mainstay in therapy. It grew out of the experience with spinal cord injured Vietnam veterans. We began to realize that crede voiding, straining and dyssynergic situations ultimately lead to renal failure, sepsis and a very high death rate in this group. With the advent of intermittent catheterization, those problems essentially disappeared.

Self-intermittent catheterization is very safe over the long term. It is safe to use the same catheter until it disintegrates. There are all sorts of misconceptions about boiling and disinfecting and sterilizing. It's called "clean" intermittent catheterization, not "sterile" intermittent catheterization, and there is a reason for that. It requires no boiling, no sterilizing, no disinfection. There are more bacteria safely colonized in the bladder of those who catheterize than there are bacteria coming out of the U.S. water systems in most of our locations. It's safe to just rinse the catheter, pat it dry and put it in a baggy. One does not increase the risk of infection. So this ought not to be a ritual that adds any time to your day.

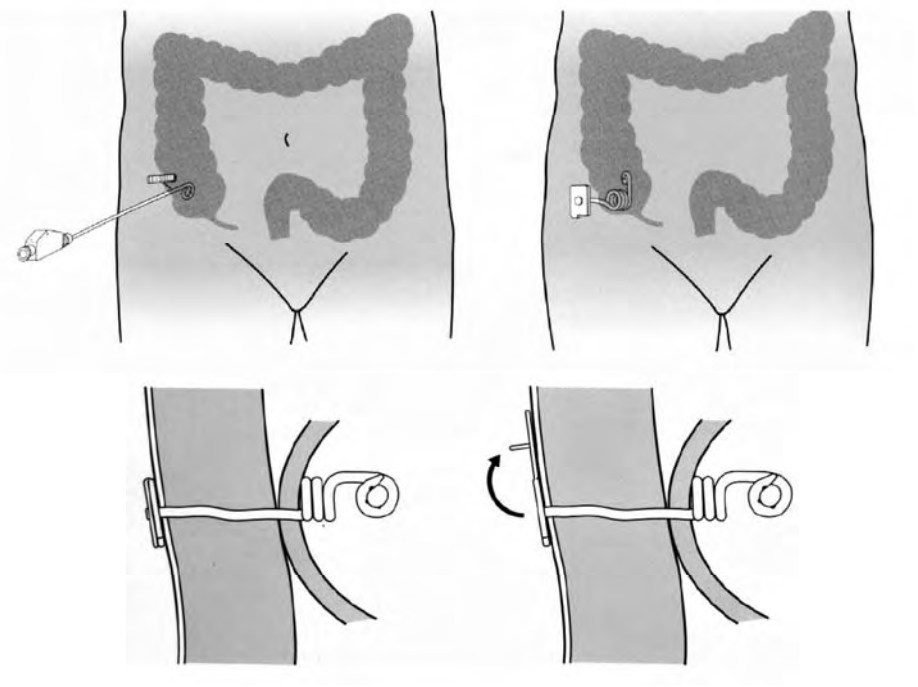
With self catheterization, "more is better." One might tend to think, "Well, I don't want to put that in too many times because I might get an infection." The reality is that the more you do it, the more the bladder is kept empty, and the safer is the system. When it is done more often, the bladder pressure is low, and the bacterial soup is washed away. A person has bacterial colonization, not infection. Often there is some overgrowth and one might get some symptomatic infections in a given year, but it is generally not any more of a problem than what is found in the general population. A short course of antibiotics is usually effective. It does not require a ten to fourteen day course of antibiotics. It is just a question of beating down the numbers. You can't eliminate those bacteria; you really just need to stabilize the situation. It might not be desirable, but if we are trying to buy time, this is definitely the way to go.

Finally, I would like to talk about bowel dysfunction. Fecal incontinence and constipation are often more problematic than voiding dysfunction. Remember, too, the more empty the rectum, the better the bladder will behave. They are wired into the same place, and distention of the colon, poor motility, and constipation really sends confusing messages to the bladder and makes it difficult to sort out. It also has an anatomic obstructive function with big time constipation.

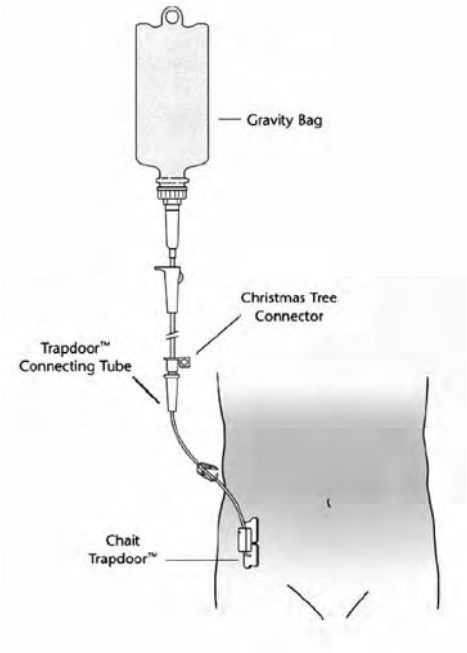
Fiber, fluid, laxatives, and suppositories may all help bowel evacuation. But what happens when these don't work? I want to talk briefly about the ACE procedure. We would consider this for difficult cases. This is called an antegrade continence enema; it is basically a way to give oneself an enema without all the hassle or the need for assistance. It can be created with either the appendix or a special catheter device. Up to 70% of patients with an ACE can establish fecal continence on a reliable schedule. It is placed surgically (I do them laparoscopically) or it can be put in by a radiologist. The best thing about it is that it is reversible. If it is not the full answer, it can be undone with no harm.

This graphic depicts a temporary ACE; this is what it looks like if it is done by a radiologist. It does require a period of time for healing. It is located down in the right lower quadrant. The catheter device is a little coiled catheter, like a phone cord, with a little port at the top that flips open. It doesn't leak, it doesn't smell, it really doesn't impose any kind of impediment. It is self retaining and non-leaking, and it allows one to put fluid in the start of the colon and wash the entire thing out.

Chait trapdoor catheter



Chait/ACE set-up



Most enemas get up about halfway and then fall down. An irrigation schedule can be every one, two or every third day and evacuation occurs within about 15-30 minutes. One can use tap water, saline or phospha-soda. It requires no additional assistance. Folks can sit on the commode and be clean and worry free after 30 minutes and until the next time.

In conclusion, the effects of TM on pelvic function are highly variable. It is possible to diagnose voiding dysfunction and plan appropriate therapy. Our goals in therapy are to preserve the safety of the kidneys and this is ordinarily not hard to do. Continence, independence and maximizing quality of life are the other goals of therapy. Our progress has been incremental, but it is real. We need to be our own best advocates, and that requires that you become educated about your condition and about possible therapy options. One of the goals of the TM Center at Johns Hopkins is to be that place of information transfer. The road is long. I believe that in our lifetime, many of these conditions will be better understood, and we will have medicines and therapies to better restore function.

Interventional Approaches to Neuropathic Pain

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Adapted from a presentation given at the 2006 Rare Neuroimmunologic Symposium

I am a pain medicine specialist, anesthesiologist, and the director of the pain treatment center and pain fellowship program at Johns Hopkins. My article focuses on interventions that we typically use to treat neuropathic pain. As an introduction, I will distinguish acute versus chronic pain and nociceptive versus neuropathic pain and will describe the mechanisms behind the various types of pain. I will then highlight the interventional strategies or injections that we use to help relieve neuropathic pain.

Pain warns of threatened or ongoing tissue damage. The International Association for the Study of Pain defines pain as, “an unpleasant sensory and emotional experience associated with potential or actual tissue damage.” In general, tissue that is injured from surgery, trauma, or even disease processes release inflammatory products such as prostaglandins or histamine. These products trigger pain signals that travel from the body to the spinal cord and then to the brain where the messages are interpreted as painful.

Pain can be classified as both acute and chronic. Subsumed under chronic pain are nociceptive pain, neuropathic pain, visceral pain, and then a mixture of all three types of pain. For instance, some patients who suffer from low back pain experience both nociceptive and neuropathic pain; however, it can be difficult to distinguish the mechanisms that are responsible for particular types of pain.

Acute pain (next page) can be viewed as an unpleasant reaction or sensation due to some type of tissue damage that may be related to surgery or even an injury, such as spraining an ankle. Acute pain is physiologically normal and serves a protective role. The degree of pain a patient experiences typically corresponds to the extent of tissue damage. Acute pain treatment outcomes are good; that is, most acute pain can be successfully treated with medications and patients usually do not suffer from persistent pain. For example, we often use oral or intravenous opioids and/or epidural anesthesia intra-operatively and post-operatively with good pain control and minimal side effects.

Chronic pain poses more of a challenge in understanding the disease and treating the symptoms. The International Association for the Study of Pain classifies or describes chronic pain as, “pain that persists after the

Pain Classification