Management of the urinary tract in spinal cord injury patients

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The urinary tract is a unit and its different components have a direct effect on each other. Management of the upper and lower urinary tracts is paramount for the life expectancy and quality of life of the spinal cord injury (SCI) patient.

Management of the lower urinary tract (LUT) is the most important; control of the LUT is lost after SCI.

Lower urinary tract
When treating SCI patients one must remember that the LUT consists mainly of two components. The bladder stores urine and has to empty on demand. To store urine effectively it needs the sphincter mechanism to control the outflow. To empty effectively the bladder needs an increase in intravesical pressure and a decrease in intra-urethral pressure. The urethral sphincter consists of two different components, which are discussed fully in this article.

Upper urinary tract
The kidneys and ureters are important in the homeostasis of the body. While it is important to prevent damage to the upper tracts, kidney damage in the SCI patient is mostly caused by dysfunction of the LUT. Management is therefore concentrated on the LUT.

Management of the LUT
The level of the injury is important. If the patient still has good hand function, self-intermittent catheterisation should be considered. It is important to create a safe storage function for the bladder and to take over the emptying function by self-catheterisation.

If a patient has no hand function, then free urinary drainage is the only option. This can be achieved by bypassing the sphincter, using an indwelling urethral or suprapubic catheter or creating a stoma. Free drainage using condom catheters in men is feasible as long as the sphincters are inactivated.

SCIs do not usually cause complete anatomical transection of the cord. The level of the injury is particularly important with regard to the effect on the LUT.

Associated treatment includes the prevention of urinary tract infections, bypassing of catheters and bladder stones.

Pathophysiology
SCIs are common, especially in South Africa, where road accidents and violent crimes often occur. SCIs do not usually cause complete anatomical transection of the cord. The level of the injury is particularly important with regard to the effect on the LUT.

The LUT functionally consists of the bladder, the smooth-muscle sphincter and the striated sphincter. These three systems control the storage and emptying functions of the bladder. A dysfunctional LUT unit has a significant impact on the quality of life of patients – not only will it lead to infections and stone formation, but also to deterioration of the upper tracts and therefore reduced life expectancy. Additionally, a dysfunctional LUT has an impact on the social status of the patient and affects his/her quality of life. Urinary incontinence, permanent urethral catheters or even difficulties associated with finding toilet facilities for the disabled contribute to their avoidance of social interaction.

The numbering of the vertebrae does not correlate with the spinal cord level. The sacral spinal cord begins at level T12 - L1 and terminates in the cauda equina of L2. The sympathetic and parasympathetic innervations exit at different levels of the spinal cord. The sympathetic nerves leave the spinal cord at level T7 - 8 which is located at the T6 vertebra. The parasympathetic system correlates with the levels of the somatic system, which is very important in synergy of function. Synergy between detrusor contraction simultaneously with smooth and striated sphincter relaxation is extremely important for a balanced LUT.
Urinary tract in SCI

Dysfunction of the urinary tract in SCI is common and can have a significant impact on the quality of life of patients. Dysynzynergia refers to the simultaneous contraction of both the detrusor and the sphincters, which can obstruct urine flow.

In general, lesions below T6 (spinal cord T7 - 8) give striated sphincter dyssynergia, and above that both striated and smooth-muscle dyssynergia. The significance of dyssynergia is that pressures rise in the bladder when the detrusor contracts, but the sphincter fails to open. An increase in bladder pressure without subsequent urine flow or weak flow leads to complications.

Sexual function must always be considered with LUT function, but is not discussed in this article.

Spinal shock
It is well known that there is muscular paralysis below the level of injury. Normally the striated sphincter is relaxed, but there is still closure of the smooth muscle of the internal sphincter. The bladder is areflexic and relaxed. The precise time of the shock phase is unknown and varies.

Incontinence may be present as overflow incontinence which must be prevented using catheterisation drainage or clean intermittent catheterisation as soon as possible after the injury.

A dysfunctional LUT unit has a significant impact on the quality of life of patients.

Reflex bladder contraction generally returns within 3 months, but may take as long as 2 years. Incomplete lesions will recover earlier than complete suprasacral lesions. Detrusor reflex contraction returns with deep tendon reflexes.

The precise mechanism by which the detrusor regains its contractibility is largely unknown, but may come from any one, or a combination, of the different neuronal, neuromuscular or muscular physiological pathways. Detrusor overactivity is accompanied by dyssynergia with the striated external sphincter mechanism, leading to high intravesical pressure and incomplete bladder emptying.

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What happens to the bladder after SCI?

The level and extent of the SCI are not always evident. The injury to the spinal cord may be complete or incomplete and may be over a longer segment than only the level of injury. It is therefore important to correlate the injury with the clinical picture and the urodynamic findings.

The normal bladder has storage and emptying functions. The detrusor in SCI patients may be overactive, non-compliant or underactive, depending on the level of injury and the patient's response to the denervation.

Suprasacral lesions cause detrusor overactivity and sacral lesions cause detrusor relaxation. The sphincter must be seen as two components. The sympathetic, innervated, smooth-muscle internal sphincter is usually hypertonic and does not relax with a detrusor contraction. The somatic, innervated, striated external sphincter is relaxed initially, but will normally develop reflex spastic activity later.

Management of the urological tract in SCI

The spinal shock phase must be treated by catheter drainage of the paralysed bladder. This can be done by self-intermittent catheterisation or by an indwelling urethral catheter or suprapubic catheter. Reflex detrusor contractions generally return after 3 months and correlate with the return of deep tendon reflexes. This can guide one to when the first urodynamic study must be performed, which is an integral part of the management of the SCI patient.

Urodynamic studies

These are functional studies of the LUT and provide information on detrusor muscle function, capacity and compliance. Important information on the synergy between detrusor contraction and sphincter function can be obtained using urodynamic studies. Internal and external sphincter function can be distinguished on electromyograph (EMG) tracings. Striated muscle, but not smooth muscle, will give EMG activity on contraction.

The first urodynamic study is done at approximately 3 months to obtain a baseline evaluation for comparison in further studies. Thereafter it is necessary to repeat the study annually until the situation is stable. If there are any changes in the clinical picture or the upper tract it is important to repeat the study.

The most important measurement in a urodynamic study in an SCI patient is the detrusor pressure at which the bladder empties. It is widely accepted that if the intravesical pressures rise above 40 cmH2O then catheterisation drainage or clean intermittent catheterisation should be considered.
Urinary tract in SCI

without any urine flow, it will lead to high intravesical pressures and eventually to complications.

Ultrasound
Ultrasound of the urinary tract is the mainstay of follow-up. The upper tracts, bladder and urethra can be visualised by means of ultrasound. It will detect stones, hydronephrosis, bladder diverticula and residual volume.

Urine microscopy and culture
This will normally be done in a patient with symptoms. A positive culture without symptoms is not very significant, particularly if there is an indwelling catheter.

Biochemical analysis
Biochemical analysis of kidney function with S-creatinine or creatinine clearance must be done early.

Treatment options in SCI

Detrusor muscle
The intravesical pressures must be controlled. Co-ordinated voiding is seldom achievable, normally only in incomplete injuries. The main treatment is to lower detrusor contraction to reduce the risk of complications and create a safe storage organ for the urine. To achieve this, anticholinergic medication is used. Currently intravesical botulinum toxin (Botox) is widely being used owing to its efficacy and low side-effect profile.

Anticholinergic treatment
This was the mainstay of treatment before the availability of Botox. Anticholinergic medication will relax the bladder muscle and create a safer storage system. The side-effects are constipation, vision disturbances, dry mouth and cognitive impairment. It is contraindicated in glaucoma.

Intravesical Botox
This is the new wonder drug for the treatment of neurogenic bladders. It is an extremely potent muscle relaxant and is only effective in the organ into which it is injected.

Botox (200 units) is injected into the suburothelium and detrusor muscle every 6 - 9 months. This will greatly relax the bladder, increase its volume and decrease its filling pressure. The side-effect profile in this subgroup of patients is extremely low.

Promising treatments
In the future it will be possible to regenerate muscle with stem cells.

Anterior root stimulators may become an alternative treatment to restore functional voiding. These are still very expensive and have to co-ordinate bladder contraction and sphincter relaxation.

Sacral neuromodulation is not a direct stimulation, but may reduce the development of detrusor overactivity or a non-compliant bladder. This implant has to be done soon after the shock phase to prevent later complications.

Management of sphincter activity
In the SCI patient the sphincter is usually closed. The pressure may be reduced in the urethra owing to striated muscle weakness. In men, because of the prostate and internal sphincter, the pressure is mostly sufficient to keep the patient continent if there is a low-pressure, compliant bladder.

If the bladder is 'unsafe' (high pressure or non-compliant), the sphincter needs to be bypassed or inactivated to prevent high intravesical pressures and its complications.

Pharmacological treatment to increase sphincter function, e.g. alpha-adrenergic stimulants (pseudo-ephedrine), is not very effective and may cause significant side-effects. Obstructive procedures can be done surgically to increase intra-urethral pressure passively or actively, including retropubic, mid-urethral slings, bulking agents or artificial urinary sphincters.

Interventions to reduce sphincter tone include Botox injections, surgical incision of the sphincter muscle or a urethral stent to keep the sphincter open. This will reduce the outflow resistance in a patient with a spastic bladder and prevent complications.

If high bladder pressures are present and the sphincters are inactivated, free drainage with condom catheters is a safe option. The sphincter can also be bypassed by using an indwelling urethral or suprapubic catheter.

It is important to remember that the detrusor and the sphincter in SCI patients are not co-ordinated.
Follow-up
There is no clear consensus as to which tests need to be done as follow-up.

As a guideline:
- Yearly ultrasound and S-creatinine.
- Yearly urodynamic studies until the condition is stable and then only if there are changes in bladder function.
- Urinary microscopy and culture only if the patient is symptomatic or has complications, e.g. stone disease, bladder diverticulae or high residual volume.
- Other more specialised tests only if the clinical picture warrants better visualisation, or function tests.

References are available on request.

IN A NUTSHELL
- Management of the LUT is particularly important in SCI because its deterioration leads to poor quality of life and may affect life expectancy.
- Control of the LUT is lost after SCI.
- An important consideration is the level of injury. If the patient still has good hand function, self-intermittent catheterisation is an option.
- If a patient has no hand function, then free urinary drainage is the only option.
- Associated treatment includes the prevention of urinary tract infections, incontinence or stones.
- SCIs do not usually cause complete anatomical transection of the cord. The level of the injury is particularly important with regard to the effect on the LUT.
- The LUT consists functionally of the bladder, the smooth-muscle sphincter and the striated sphincter.
- These three functional systems control the storage and emptying of the bladder.
- Dysfunction in the unit of the LUT has a significant impact on the quality of life of patients.
- The level of the injury and the extent of the SCI are not always exact.
- The injury to the spinal cord may be complete or incomplete and over a longer segment than only the level of injury.
- A urodynamic study is an integral part of the management of the SCI patient.
- The main treatment is to lower detrusor contraction to reduce the risk of complications and create a safe storage organ for the urine.
- To achieve this, anticholinergic medication is used. Currently intravesical Botox is used widely owing to its efficacy and low side-effect profile.

Single Suture
Clues to aspirin’s anti-cancer effects revealed

One of the world’s oldest medicines may hold the secret to a very contemporary problem: preventing cancer. Exactly why salicylate shows such potential as an anti-cancer treatment remains unclear, but a new study in mice offers clues.

Salicylate, found in willow bark, has been a key ingredient in medicine cabinets for thousands of years – ancient Egyptian manuscripts describe it as a treatment for inflammation. In a modified form – aspirin – it remains a successful anti-inflammatory and analgesic. Recently though, research has revealed a puzzling side-effect of taking aspirin: the drug seems to lower a person’s chances of developing some forms of cancer.

Aspirin is rapidly broken down inside the body into salicylate, so to investigate aspirin’s unexpected side-effects Grahame Hardie at the University of Dundee, UK, applied salicylate to cultured human cells derived from the kidney. He found that the drug activated AMPK, an enzyme involved in cell growth and metabolism that has been found to play a role in cancer and diabetes.

‘This is an ancient herbal remedy which has probably always been part of the human diet,’ says Hardie. ‘But despite that we’re still finding out how it works.’

Co-author Greg Steinberg of McMaster University in Hamilton, Ontario, Canada, then tested high doses of salicylate on various types of mice. He found that those engineered to lack AMPK did not experience the same metabolic effects from salicylate as seen in mice with AMPK.

Salicylate, in a form called salsalate, has also shown promise as a treatment for insulin-resistance and type 2 diabetes. Those effects, however, appear not to be governed by AMPK. When insulin-resistant mice lacking AMPK were given salicylate, they showed the same improvement in blood glucose levels as normal mice.

‘That’s what makes aspirin so scientifically and clinically interesting,’ says Chris Paraskeva at the University of Bristol, UK, who was not involved in the work. ‘It potentially works through a number of different pathways.’

The finding potentially separates aspirin’s pain-relieving effects from its cancer protection, paving the way for new anti-cancer drugs that have fewer side-effects than aspirin. The next step will be to test salicylate directly in mouse models of cancer, and to see whether AMPK remains important in mediating an anti-cancer effect.

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