NEUROLOGICAL INVESTIGATIONS

Investigation of the neurogenic bladder

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Methods of examination which have been used to investigate the neurogenic bladder include tests of bladder function, so-called "urodynamics", and neurophysiological tests of sphincter and pelvic floor innervation. A possible consequence of a neurogenic bladder is damage to the upper urinary tract but the investigation of such complications is essentially urological and is only briefly mentioned in this review.

History of the development of investigations

URODYNAMICS

The term "urodynamics" encompasses any investigation of urinary tract function although it is often used colloquially as a synonym for cystometry. Cystometry, the measurement of bladder pressure, has been the main tool used to show abnormal behaviour of the neurogenic bladder.

The earliest reference to a study measuring bladder pressure is commonly given as the paper by Mosso and Pellacani published in 1882.¹ With a water manometer they showed that bladder pressure rose at the start of micturition and then gradually declined but that during storage the pressure measured within the organ gave little indication of what volume it contained. However, the paper which described a technique for cystometry producing what is regarded as the precursor of modern day urodynamic recordings was published in *Brain* in 1933 by Denny-Brown and Robertson² from the National Hospital for Nervous Diseases, Queen Square. By means of an ingenious system of mirror manometers they recorded intravesical and intraurethral pressure with two transurethral catheters (one inside the other), as well as recording rectal, perineal, and abdominal wall pressures in three neurologically normal men. From their findings they defined the physiological sequence of processes which occur with bladder filling, the initiation of micturition, and voiding to completion.

The introduction of cystometry into clinical practice was gradual and by the 1960s it was being used in only a few specialised urological centres.³ When commercial equipment first became available it consisted of a series of pen recorders which recorded pressure changes as

analogue signals but with the advances in electronics and development of microchip technology the machines have become progressively more complex, more "intelligent", and mostly easier to use. Today measured pressures are digitised allowing on line, real time computer analysis of signals.

During the development of urodynamics an important advance came with the introduction of facilities to record pressure measurements superimposed on the fluoroscopic appearances of the bladder, a "videocystometrogram".⁴ This combination provides a complete picture of the behaviour of the bladder both during filling and emptying although it is expensive and exposes the patient to x rays. Much can be learnt from simple cystometry alone and it is this investigation which is now a standard facility in almost every district general hospital with a urology department.

The hydrodynamic problems of measuring fluid flow are different from measuring pressure within an organ. Introduction of simple and cheap equipment for measuring urinary flow rate⁵ has led to this becoming routine; combined with ultrasound measurement of the postmicturition residual volume, it provides a simple non-invasive means by which much valuable information can be obtained about lower urinary tract function.

NEUROPHYSIOLOGICAL INVESTIGATIONS

Various types of neurophysiological investigation of the pelvic floor and the sphincters have been developed over the years. A neurophysiological method for recording the bulbocavernosus reflex, regarded as clinically valuable in assessing patients with neurogenic bladder disorders, was first reported in 1967.⁶ Neurophysiological recordings of various pelvic floor reflexes were much in vogue in the 1970s but have since lapsed and have been transiently replaced by an enthusiasm for recording the pudendal evoked potential.

Recording from the striated muscle of the urethral sphincter or anal sphincter during cystometry was first recommended as a means of detecting inappropriate sphincter contraction during detrusor contraction, the disorder known as detrusor sphincter dyssynergia.⁷ For several reasons this type of kinesiological EMG is now little used although sphincter EMG performed as a separate neurophysio-

Figure 1 Filling cystometry in a healthy subject. Vinfus = infusion at 50 ml/minute; Pabd = intrabdominal pressure measured by the rectal line; Pves = intravesical pressure; Pdet = Pves-Pabd. Respiratory movements, which were not recorded with the intravesical pressure measurements, were recorded with the rectal pressure line so that these appear as an artefact due to subtraction on Pdet. In the early part of the trace the subject was asked to cough and the subtraction of Pabd from Pves was complete so that no rise in Pdet is recorded.

logical test remains a valuable investigation in some circumstances.

Principle underlying investigations

CYSTOMETRY

Cystometry is the recording of the pressure-volume relation of the bladder. The intravesical pressure is measured and by subtracting the intra-abdominal pressure from this figure an estimate of the true pressure produced by the smooth muscle of the detrusor is obtained. This is best seen by looking at the preparatory stages of cystometric recordings when the patient is asked to cough (fig 1). Coughing raises the intra-abdominal pressure and thus the measured intravesical pressure but under physiological conditions the detrusor does not then contract so that the derived detrusor pressure (Pdet) remains unchanged or becomes slightly negative because the intra-abdominal pressure may rise more than the intravesical pressure. To measure the intraves-

ical and intrabdominal pressures a fine catheter is passed through the urethra into the bladder and another into the rectum. The catheter used to monitor intravesical pressure is passed, together with a somewhat wider diameter catheter through which the bladder is filled. Important information is obtained if detrusor pressure is measured both during filling and while the patient attempts to micturate. In the interests of saving time an unphysiologically rapid rate of filling of 50 ml/min is commonly used in cystometric studies.

Recently, methods have become available for recording bladder pressures over periods of many hours and the bladder is left to fill naturally, so-called "ambulatory urodynamics".⁸

In patients with neurogenic incontinence the commonest finding is of an abrupt rise in detrusor pressure which the patient is unable to suppress and which is usually accompanied by reports of urinary urgency (fig 2). If the patient is recognised as having a neurological

Figure 2 Detrusor hyperreflexia in a woman with multiple sclerosis. After filling to 100 ml (vinfus) there was a detrusor contraction which resulted in a pressure rise of 90 cm H₂O.

Figure 3 Abrams nomogram. Using values for the maximal flow (Q_{max}) and the corresponding voiding detrusor pressure (P_{det}) a point can be plotted on the nomogram that determines whether the bladder outlet is obstructed, unobstructed, or equivocally obstructed.

condition, this is called “detrusor hyper-reflexia”, the condition is otherwise referred to as “detrusor instability”⁹—the cystometric changes in the two conditions being indistinguishable. The cause of detrusor instability is unknown but the weight of opinion is shifting from thinking that it has a psychogenic cause or is due to an occult neurological lesion to the view that it may be due to a disorder of the detrusor muscle itself.¹⁰ Likewise the causes of failure of bladder emptying cannot be identified from cystometry, and urological and neurological disorders can cause indistinguishable cystometric abnormality. Because bladder behaviour cannot always be predicted from the patient’s history it has been argued that urodynamic investigations are important in the investigation of urinary complaints but the failure of urodynamics to provide anything more than a description of bladder dysfunction has not been properly acknowledged.

This has resulted in a large body of medical literature in which patients are classified according to their urodynamic findings rather than by the underlying pathophysiological cause and diagnosis.

In patients with suspected obstruction of outflow, particularly men with prostatic hypertrophy, measurement of detrusor pressure during voiding is important. This, together with urinary flow rate provides information about the outflow tract¹¹ and an estimate of the presence of obstruction can be made (fig 3).

UROFLOWMETRY

Uroflowmetry is the measurement of urinary flow rate. This is a non-invasive investigation. The patient presents with a full bladder and voids into a receptacle in the base of which is a spinning wheel. Urinary flow slows the rate of rotation and from this a graphical output of flow rate can be obtained (fig 4).

Abnormalities of flow can be due to local urological problems such as prostatic hypertrophy, a urethral stricture, or neurogenic disorders of the bladder outlet mechanism. Detrusor sphincter dyssynergia, which occurs with spinal cord disease, is a common example of this and results in interrupted flow (fig 5). Uroflowmetry combined with ultrasound scanning of the postmicturition residual volume is used as a screening test to exclude seri-

Figure 4 Uroflowmetry. The lower trace shows the rate of urinary flow, which reaches a maximum of 40 ml/s in three seconds and then declines. The upper trace is the result of integrating the flow rate and shows that a total of 275 ml was passed.

Figure 5 Uroflowmetry in a patient with multiple sclerosis. He was paraparetic and had frequency and urgency but also symptoms of difficulty voiding and was shown to have incomplete bladder emptying. This interrupted flow pattern is the result of contractions of the striated urethral sphincter occurring during the detrusor contraction—that is, “detrusor sphincter dyssynergia”.

ous outflow obstruction and can also provide information for planning bladder management in patients with neurological disease.

ULTRASOUND SCANNING OF THE URINARY TRACT

The residual volume left after voiding is important, and can readily be measured with a small inexpensive ultrasound scanner. Great precision is not needed—it is simply enough to know whether there is more or less than 100 ml—an obvious abnormality on ultrasound scanning which does not need great expertise to recognise. Most scanners have cursors that can be placed on the black outline of the urine in the bladder and from this, assuming a spherical shape to the bladder, bladder volume can be calculated.

Ultrasound scanning has largely overtaken intravenous urography as the method of choice to examine the upper renal tract to detect dilatation but intravenous urography remains the preferred method to look for ureteric stones. With modern, highly complex three dimensional scanning, details of the structure of the lower urinary tract can be made out.¹² This has exciting possibilities for both urologists and urogynaecologists.

NEUROPHYSIOLOGICAL INVESTIGATIONS OF THE SPHINCTERS AND PELVIC FLOOR

Clinical neurophysiological techniques for examining the pelvic floor have been used for many years. These studies have greatly enhanced our understanding of the physiological and pathophysiological mechanisms of neural control of the lower urinary tract.

The first neurophysiological measurements made were of sacral reflexes starting with the bulbocavernosus reflex.^{6,13} To record this reflex either a surface or a needle electrode was placed over or in the bulbocavernosus muscle and electrical stimuli were applied to the dorsal nerve of the penis. The time taken for the reflex contraction of the muscle to occur after the stimulus was measured. After the introduction of this technique various other pelvic floor reflex contractions were recorded¹⁴ and it was shown that equally useful responses could be obtained by recording from the urethral or anal sphincter or other parts of the striated muscle of the pelvic floor.¹⁵⁻¹⁷ It was argued that abnormalities of the sacral roots, both efferent and afferent, would lead to a delay in this reflex and this did indeed prove to be the case in patients with established cauda equina lesions.^{13,18,19} However, it was found that reflex responses could still be elicited in patients with partial cauda equina lesions²⁰ and more importantly these tests were of little value when applied to patients with uncertain neurological lesions presenting with hypocontractile bladders or impotence. The explanation for this is probably that, as with other reflexes measured using clinical neurophysiological techniques, only the responses mediated by large myelinated fibres are recorded. The small myelinated or unmyelinated fibres which either innervate the smooth muscle or constitute the functionally important afferent nerve supply of

the region are not tested. Possibly tests of the autonomic innervation of the genital region will prove more useful.²¹

Recording the pudendal evoked potential is similar to recording tibial evoked potentials. The same cortical recording electrodes can be used and it is advisable to record the tibial evoked responses first to familiarise the patient with the technique. The patient is then asked to hold the stimulating electrode on the dorsal nerve of the penis or clitoris and a similar number of stimuli as needed for obtaining the tibial evoked potentials are given. Surprisingly the latency of tibial and pudendal responses is similar despite the difference in conduction distance. This is thought to be due to the slower conduction velocity of the pudendal afferents compared with fast conducting muscle afferents which respond when the tibial nerve at the ankle is stimulated.²²

The introduction of a method to record the pudendal evoked potential was initially hailed as promising.²³ It was considered that this would provide a means of testing the afferent innervation from the sacral region and certainly the responses were delayed in patients with conditions such as multiple sclerosis.²⁴⁻²⁶ Like the lower limb somatosensory evoked potentials, the pudendal evoked potential is delayed if there is spinal cord disease but this is also usually apparent on clinical examination. Recent studies have shown that the pudendal evoked potential is very rarely abnormal unless there are other clinical signs of neurological disease²⁷⁻²⁹ and furthermore if the lesion is predominantly unilateral the pudendal evoked potential can be within normal limits.²⁹ It seems that there is little diagnostic gain in recording the pudendal evoked potential although it is sometimes reassuring to show that it is normal in patients in whom neurological problems are suspected.

A technique for measurement of the terminal motor latency of the pudendal and perineal nerves (PTML) was devised at St Mark's Hospital.³⁰ It has been used to show pathophysiological changes in these nerves in women with faecal,³⁰ urinary,³¹ or double incontinence.³² The pudendal nerve is stimulated transrectally near the ischial spine through the wall of the rectum using an electrode mounted on the tip of the examiner's finger. An electrode is mounted at the base of the finger, which records from the anal sphincter, and a ring electrode mounted on a Foley catheter can be used to record from the periurethral striated muscle. The latency of the response has been found to be prolonged in women with urinary stress incontinence after childbirth and women with faecal incontinence due to sphincter weakness. It is thought that stretching of the nerves during parturition and also with straining at defaecation in chronic constipation results in pudendal nerve injury.³³ Although of considerable research value this test is not used in the routine assessment of women with urinary stress incontinence, nor has it proved to be as useful as electromyography of the anal sphincter in the assessment of faecal incontinence.³⁴

Electromyography (EMG) of the striated musculature of the pelvic floor is of value in recognising changes of denervation and chronic reinnervation in patients with cauda equina lesions as well those with suspected multiple system atrophy. A single fibre needle may be used to show changes in fibre density³⁵ or a concentric needle electrode to show changes in configuration of individual motor units which result from reinnervation.³⁶

The striated muscles of the sphincters are innervated by anterior horn cells that lie in Onuf's nucleus in the sacral part of the spinal cord. Neurophysiological studies showed loss of cells in Onuf's nucleus in patients dying with Shy-Drager syndrome.³⁷ These pathological changes may be reflected in abnormalities of sphincter EMG in life. This was first shown by Sakouta *et al*³⁸ and then in a systematic study by Kirby *et al*.³⁹ Sphincter EMG is now used to distinguish between patients with bladder symptoms and multiple system atrophy and atypical parkinsonism and those with idiopathic Parkinson's disease,^{40 41} and a urological disorder as in the first condition changes of reinnervation can be found which are not present in the second. Changes of reinnervation in multiple system atrophy are non-specific and some caution must be exercised in interpreting EMG findings in multiparous women or patients who have had extensive pelvic surgery. However, the changes which occur in the motor units in multiple system atrophy are so extreme as to make the test reliable and robust.

Using a concentric needle electrode 10 different motor units are recorded from either the urethral or anal sphincter, the anal sphincter being more accessible and therefore less uncomfortable for the patient but equally valuable for giving a significant result. The mean duration of the 10 motor units is measured as well as the number of units which exceed 10 ms in duration. In multiple system atrophy, some motor units remain of normal duration, but others become excessively prolonged. By contrast with this—for example, after multiple deliveries—all the units might be mildly prolonged. The values used to define normal are a mean duration of less than 8.5 ms and less than 20% of units having a duration of less than 10 ms.⁴⁰ A mean duration of more than 10 ms is highly abnormal and suggestive of multiple system atrophy but there is inevitably an area of uncertainty when the mean value is less than this.

The other condition in which urethral sphincter EMG has proved to be of particular value is in the investigation of young women with urinary retention.⁴² These patients have no neurological signs on clinical examination and in particular no evidence of spinal cord disease.⁴³ It was previously suggested that they were either presenting with urinary retention as the first symptom of multiple sclerosis or that they had a hysterical disorder. The first is now easy to disprove as imaging and neurophysiological investigation in these women show no appropriate abnormality. Sphincter EMG shows a myotonic-like activity which

has been called "complex repetitive discharges and decelerating bursts".⁴⁴ Detailed EMG analysis of this activity, measuring the jitter of the component potentials, has shown that in common with other complex repetitive discharges, it is due to ephaptic transmission between muscle fibres⁴⁴ and it has been suggested that it is this activity which can be recorded as a continuous phenomenon which prevents the muscle from relaxing. The striated muscle of the urethral sphincter is a circularly placed horseshoe-like structure and it is not difficult to see how a failure of it to relax would result in either obstructed voiding or urinary retention. Why the abnormal activity should develop remains unknown but it is not uncommon for women with urinary retention to have clinical features of polycystic ovaries.⁴⁵ A speculative hypothesis is that the striated muscle of the urethral sphincter, being hormonally sensitive, undergoes a breakdown in membrane stabilisation secondary to the pervading hormonal abnormality of polycystic ovary syndrome allowing ephaptic transmission between muscle fibres to occur. Unfortunately no specific treatment has yet been effective and the women manage best by performing intermittent self catheterisation.

A similar abnormality has not been found in men and extensive neurophysiological testing has failed to show a defect in the less commonly encountered young men with urinary retention without a urological explanation. The role of primary detrusor abnormality in these men needs to be explored as it does in patients with idiopathic detrusor instability. Unfortunately there is as yet no neurophysiological means of investigating detrusor smooth muscle function.

Planning investigations

Investigations of bladder symptoms are carried out for two very different purposes: with bladder symptoms and established neurological disease urodynamic investigations may be performed to try and understand the pathophysiological basis for the patient's symptoms and obtain information on which to base recommendations for management of incontinence.

When the question is being asked "is this a neurogenic bladder?" a different approach is required. In this instance investigations are of a neurological or neurophysiological nature.

URODYNAMIC INVESTIGATIONS IN PATIENTS WITH ESTABLISHED NEUROLOGICAL DISEASE

Poor bladder control is a common and troublesome feature of many types of neurological disease, especially of the spinal cord. The commonest complaints are of urgency, frequency, and urge incontinence and in established neurological disease these can be assumed to reflect detrusor hyperreflexia. However, patients with neurogenic bladder disorders often have a disorder of emptying as well; incomplete emptying is probably due to a combination of detrusor sphincter dyssynergia and poorly sustained detrusor contractions. If the bladder does not empty completely the

persistent postmicturition residual volume acts as a stimulus for repeated detrusor contractions so that efforts to treat detrusor hyperreflexia are unlikely to succeed until effective emptying is achieved. The most effective treatment for detrusor hyperreflexia is an anticholinergic drug (oxybutynin is currently recommended) but there is no oral medication which improves neurogenic voiding disorders and the best management is intermittent catheterisation, performed by the patient or carer.

In summary the presence of detrusor hyperreflexia may be reliably deduced from clinical history, but incomplete emptying, although contributing appreciably to the problem, can be largely asymptomatic. For this reason the single most important investigation when planning the management of patients with neurogenic incontinence is measurement of the postmicturition residual volume. This can either be done by simple ultrasound (see earlier) or by "in-out" catheterisation. The advantage of using catheterisation is that it familiarises the patient with what is involved in intermittent self catheterisation.

Investigation of the postmicturition residual volume is recommended before starting on an anticholinergic drug, as shown in the algorithm in fig 6. A further point about treatment

with anticholinergic drugs is that although they may be effective in lessening detrusor hyperreflexia they can adversely affect bladder emptying. It is advisable therefore if a patient starts on these drugs and fails to respond to recheck the postmicturition residual volume and make sure that this has not significantly accumulated.

If the patient has recurrent urinary tract infections or fails to respond to the regimen outlined in fig 6, it is advisable to refer the patient to a urologist who will carry out investigations to exclude urinary tract stones or some other structural lesion.

Although cystometry is not critical in the routine investigation and management of patients with neurological disability such as multiple sclerosis, there are other neurological conditions in which measurements of bladder pressure are important. This is particularly the case in patients with parkinsonian features and bladder symptoms. There must be a high index of suspicion of multiple system atrophy in such a patient, best investigated by sphincter EMG (see earlier). If, however, sphincter EMG is normal and the disorder seems to be idiopathic Parkinson's disease the question of prostatic obstruction of outflow in men arises and full cystometry with a voiding study is essential.

Cystometry is also of value when investigating patients with an uncertain neurological diagnosis who have among their symptoms complaints of bladder dysfunction. Finding sensory urgency may provide an explanation for bladder symptoms without suggesting a neurogenic basis.

IS THIS A NEUROGENIC BLADDER?

The role of urodynamics in trying to decide if a patient has a neurogenic bladder disorder is limited. In most patients sent by urologists to neurologists, filling cystometry has disclosed bladder overactivity. In this instance the neurologist must try to confirm or refute that there is a neurological basis for the problem. Foremost in the patient's assessment is the clinical neurological examination.

The clinical neurological examination

The neural organisation of control of bladder function is widely distributed throughout the neuraxis. The neural programmes which determine whether the bladder is in storage or voiding mode exist in centres in the dorsal tegmentum of the pons.⁴⁶ For these programmes to be effected there must be intact connections between the sacral part of the spinal cord, which is the level of efferent and afferent neural connections to the lower urinary tract, and the pons. Spinal cord abnormality is therefore a common cause of neurogenic bladder dysfunction. The influence of higher centres and particularly input from the mesial frontal lobes is thought to be important in modulating the activity of the pontine micturition centres and there is probably also input from other supra-pontine regions although these have been less clearly defined. Thus any lesion between

Figure 6 Algorithm for management of patients with neurogenic bladder disorders. By following this both aspects of bladder dysfunction—incomplete emptying and hyperreflexia—are treated.

the frontal lobes and the sacral part of the spinal cord is likely to result in bladder dysfunction.

Because of the relative levels at which the innervation of the lower limbs and the bladder arise, it is unusual to have a lesion between the pons and the sacral part of the cord giving rise to a neurogenic bladder that does not also produce signs of an upper motor neuron lesion in the lower limbs. This is undoubtedly the case in patients with multiple sclerosis⁴⁷ but it also seems to hold for most other instances of spinal abnormality unless the lesion is very small and intramedullary. A predictable exception to this rule might be expected from a conus or cauda equina lesion affecting only S2-S4. It seems, however, that even with such extreme caudal lesions there are usually neurological abnormalities in the lower limbs and foot deformities may be present if the problem has been of long duration.

Brain stem or pontine abnormalities giving rise to bladder dysfunction often cause other neurological deficits but occasionally a lesion can be sufficiently dorsal and discrete to produce predominantly a defect of bladder function.⁴⁸ An internuclear ophthalmoplegia is a frequent additional sign, due presumably to the proximity of the median longitudinal fasciculus.

The contribution of suprapontine disease to neurogenic bladder dysfunction, with the exception of areas in the frontal lobes, is poorly defined. Patients with incontinence due to lesions of the frontal lobe usually have profound neuropsychological impairment including a change of personality but are not indifferent to their incontinence unless there has been extensive frontal lobe damage.⁴⁹ Hydrocephalus probably causes bladder dysfunction by pressure effects from the distended lateral ventricles on the frontal regions.⁵⁰

The neurologist may be asked if a patient's peripheral neuropathy is responsible for bladder dysfunction. Many forms of neuropathy are length dependent, the maximum deficit being evident in the longest fibres whereas the nerve fibres to the bladder are comparatively short. For the innervation of the bladder to have been affected as part of a generalised neuropathy there should be clinical evidence of extensive disease with loss of both knee and ankle jerks and sensory impairment in small fibres to a level well above the ankles. Even if the neuropathy is selective for small fibres symptomatic bladder involvement occurs relatively late and only in patients with other profound neuropathic symptoms.

Imaging of the nervous system

From the preceding section emphasising the value of clinical examination, it is apparent that there are regions of the CNS where a lesion can cause bladder symptoms and yet produce only minor or equivocal physical signs. Imaging is particularly indicated to exclude a suprapontine abnormality or a sub-sacral cauda equina lesion. Magnetic resonance imaging of the lower cord and cauda

equina has replaced myelography as the investigation of choice for imaging this region.⁵¹ It seems that a congenital malformation of the lower spinal cord such as a tethered cord cannot be excluded by a plain radiograph as various forms of dysraphism can occur without spina bifida.

Pelvic floor neurophysiological investigations

There are two conditions in which neurophysiological investigations can disclose an abnormality that might not otherwise be evident.

If there are any other neurological features such as parkinsonism, cerebellar ataxia, postural hypotension, or symptoms suggesting laryngeal stridor, a diagnosis of multiple system atrophy should be considered.⁵² Sphincter EMG has proved a valuable test in detecting this disorder by showing pronounced changes of denervation and reinnervation in the motor units (see earlier).

In young women with bladder disturbance but no other convincing neurological deficit, EMG of the urethral sphincter may show the myotonic-like activity, decelerating bursts, and complex repetitive discharges described earlier.¹² Even if sphincter EMG is not available, in a young woman with urinary retention simply indicating an absence of spinal cord signs on clinical examination makes a diagnosis of multiple sclerosis, a condition otherwise likely to be considered as the cause, highly improbable.

Conclusion

The difficulty that neurologists have had with investigating the neurogenic bladder stems from a combination of factors: the range of presenting symptoms is limited to either incontinence or retention, it is not possible to examine the bladder clinically, and the impression that urodynamics may prove a "diagnosis" has obscured the fact that there are many bladder disorders of unknown cause. Finally there has been the neurologists' reluctance to become involved in the management of incontinence. In bladder dysfunction established neurological disease should be regarded as an essentially neurological symptom and investigated and managed appropriately. Bladder symptoms are among the most treatable of neurological deficits and are an unpleasant and troublesome burden for the patient and their carer.

In patients sent from urologists to neurologists the emphasis should be on excluding spinal cord disease, which can be readily done by clinical examination. A high index of suspicion for multiple system atrophy should be maintained for older patients as this is a neurological disease which can present with bladder dysfunction and the patients do not benefit from urological surgery.

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