What neurologists need to understa

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INTRODUCTION

Urodynamics has been used in the investigation of bladder dysfunction since the 1950s, although the first report describing bladder pressure measurement dates from 1882 (Mosso & Pellacani 1882). In its broadest sense, the term incorporates any test of urinary tract function, although in practice it is applied to the study of the lower urinary tract – and thus allows the two functions of the bladder (storage and voiding) to be measured. The techniques range from simple noninvasive tests such as uroflowmetry to invasive tests such as cystometry, urethral pressure profile, sphincter electromyography (EMG), and more advanced investigations including ambulatory urodynamics and videourodynamics. However, the test with the central role is cystometry and the term 'urodynamics' is sometimes used incorrectly as a synonym for this investigation. In fact, the word 'urodynamics' was first used in the *Journal of Urology* in 1962 by Davis and Zimskind (Davis & Zimskind 1962). Although several investigators had described similar instruments in the late 19th century, it was D.K. Rose from Washington University, St. Louis, who coined the term 'cystometer' in 1927 (Rose 1927). A paper published in 1933 by Denny-Brown and Robertson from the National Hospital, Queen Square, London first described a technique which produced recordings that are regarded as the precursor of modern day cystometry (Denny-Brown & Robertson 1933). They used a special double catheter to measure pressure in the bladder, urethra and rectum, and showed that bladder pressure in humans was independent of intra-abdominal pressure. The development of the uroflowmeter was pioneered in 1948 by William M. Drake Jr., from Jefferson Medical College, prior to which attention had concentrated only on the force and magnitude of the patient's stream. It was in 1962 that Gleason and Lattimer first reported the use of cystometry and uroflowmetry in combination (Gleason & Lattimer 1962).

URODYNAMICS – THE AVAILABLE TECHNIQUES

Cystometry

Cystometry is the recording of the pressure-volume relationship of the bladder. It generally refers to the measurement of detrusor pressure during the filling and voiding phases. The underlying principle is that the detrusor pressure is derived by subtracting the intra-abdominal pressure (measured by a rectal line) from the intravesical pressure (measured by urethral catheter), or:

> Intravesical pressure = intra-abdominal pressure + detrusor pressure.

Urodynamic nomenclature was standardized by the International Continence Society in a series of meetings from 1974 to 1979. These resulted in four separate reports, the fourth of which clarified definitions of detrusor and urethral function and sensation (Bates *et al.* 1981).

Method

The patient is catheterized urethrally. Nowadays there are double-lumen catheters available that allow filling and measurement without the need to manipulate separate lines. A rectal line is also inserted by the investigator, and then both of these lines are fixed by tape. The bladder is filled at a rate controlled by an infusion pump: rapid-fill (greater than 100 mL/min); medium-fill (10–100 mL/min); or slow-fill (2–10 mL/min). Put in context, the physiological rate of bladder filling is approximately 2 mL/min. A medium or slow filling rate should be used in patients with neurological abnormalities, particularly patients with spinal cord trauma, to minimize any risk of increasing artificial detrusor activity. During the filling phase, the patients are asked to report when they first feel a sensation of filling, then a normal desire to void, a strong desire to void, and finally a sense of urgency. The pump is then stopped, the maximum cystometric capacity measured, and the patient encouraged to void with the catheters in-situ; the flow rate is recorded, and the detrusor pressure calculated as before. Conventional cystometry is obviously not a truly physiological representation of bladder function, and the investigator should be aware of artefacts that can adversely affect the readings (such as a displaced rectal line, change in the position of the patient, etc.). It is important to stress here that urodynamics offers descriptive rather than diagnostic information. Correct interpretation of the results depends on being able to spot potentially misleading underlying problems, for example, if the rectal line is not recording, then the detrusor pressure will be artificially high.

Advances in technology, and the development of solid-state transducers (rather than conventional water-filled transducers), led to the introduction of **ambulatory urodynamics** whereby the test is performed with the patient continuing their normal daily routine, enabling their voiding pattern to be recorded less artificially. It is important that the patient keeps an accurate voiding diary, so that this information can be analysed together with the urodynamic traces.
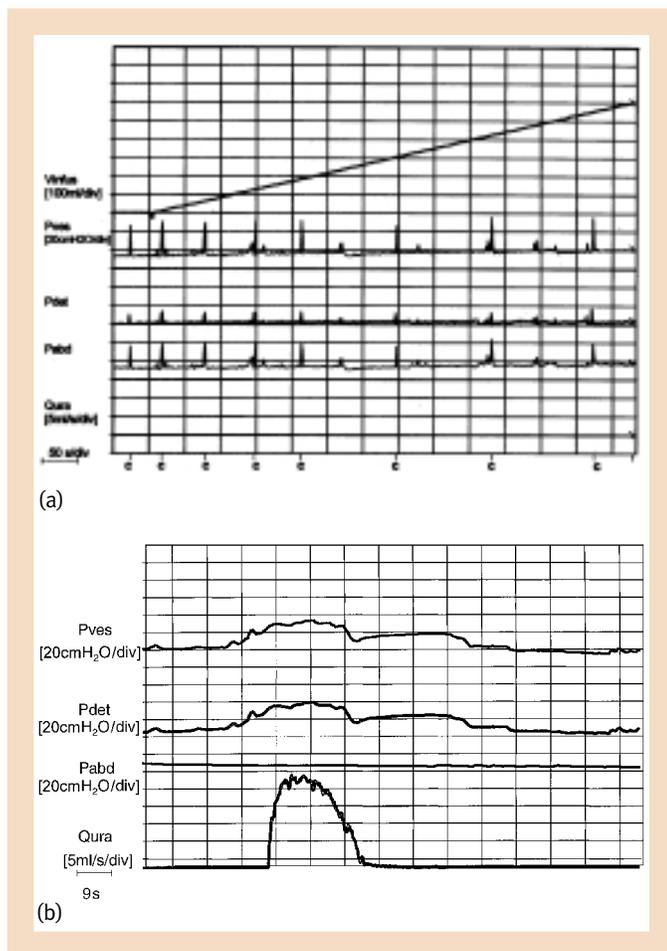
Figure 1 Normal cystometry. (a) filling phase; (b) voiding phase. Vinfus, rate of bladder filling; Pves, intravesical pressure; Pdet, detrusor pressure; Pabd, intra-abdominal pressure; Qura, urine flow; C, cough; FS, first sensation, ND, normal desire to void; SD, strong desire to void.

of the equipment, the extra time and expertise required, and the exposure to radiation during screening. Videourodynamics is not used routinely in patients with neurological disease.

Normal cystometry

This can be considered in two stages: filling and voiding. Figure 1(a) shows the pressures (Pdet = Pves – Pabd) as the bladder is filled at a medium fill rate of 30 mL/min. The trace begins with a test cough to ensure there is no bladder instability and that the transducers are well balanced. The first sensation of bladder filling is recorded (usually at about 150 mL), after which the normal desire to void is noted, when the subject would normally go to the toilet, commonly at about 300 mL. With continued filling, the strong desire to void and urgent desire (at which point the subject is asked to void) are recorded, and the maximum cystometric capacity is noted. In Fig. 1(a), the patient has been asked to cough repeatedly with a full bladder to test for stress incontinence (of which there is no evidence). The pump is then stopped and the patient asked to void at will. The important points to identify are whether the patient has normal sensation of filling, normal compliance (distensibility of the bladder), whether there is any rise in detrusor pressure during filling or bladder instability, and to record the bladder capacity.

The voiding stage is shown in Fig. 1(b). There is a normal shaped flow curve associated with a slight increase in the intravesical pressure (and thereby detrusor pressure) without any change in the intra-abdominal pressure. The normal male voids at 40–60 cmH_2O, whereas the normal female can void at lower pressures because there is less outflow resistance anatomically. A picture of high pressure with low flow would indicate obstruction. In this phase, it is important to identify whether the subject can initiate a detrusor contraction (and if this is voluntary or nonvoluntary) and what pressure is achieved.

Uroflowmetry

This is the simplest urodynamic investigation, and requires the patient to void into a receptacle with a rotating disc at its base. This generates a uroflow curve, which is a graphic representation of the rate at which urine is voided, and from this much information can be gleaned. This includes the maximum flow rate, mean flow rate, voided volume and time to void. The shape of the curve is important, normally being an unbroken bell-
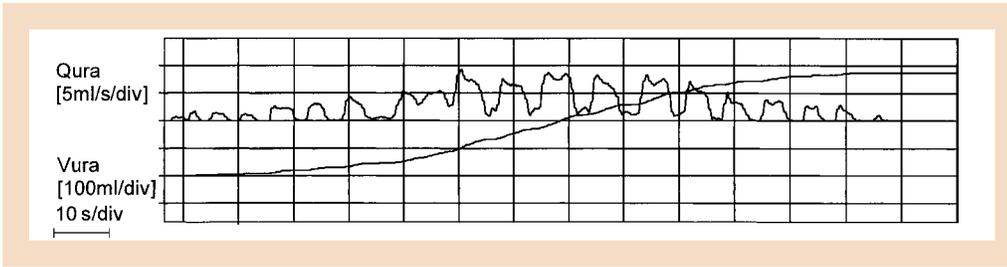
Another addition to the urological armentarium is **videourodynamics**, which requires synchronous recording of urodynamics and cytourethrography. The bladder is filled with radio-opaque material and radiographic screening is used during cystometry. Both structural and functional information is obtained. The bladder can be visualized, and one can assess the presence of detrusor-sphincter dyssynergia, ureteric reflux, the level of outflow obstruction and the degree of bladder base support during coughing. Although this is the 'gold-standard' test for certain conditions such as stress incontinence, it is not routinely undertaken because of the expense

Figure 2 Normal uroflowmetry. Qura, urine flow; vura, volume of voided urine; voided volume, 227 mL; max flow rate, 28 mL/sec.
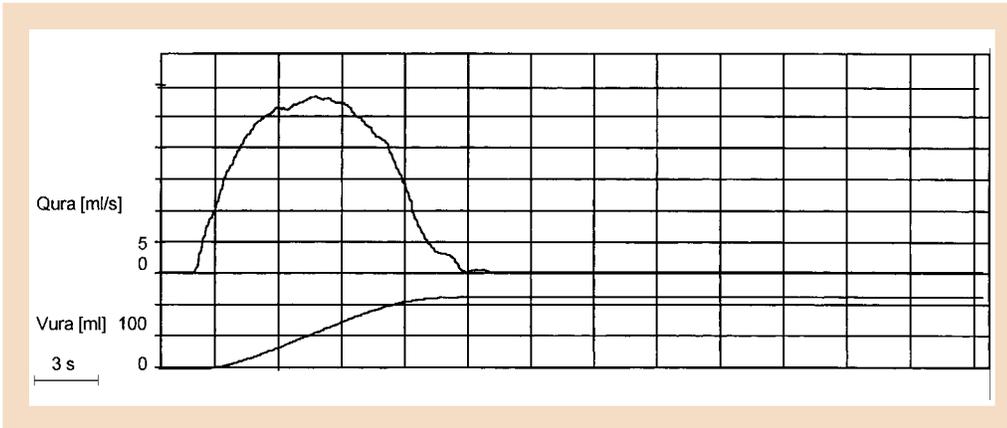

Figure 3 Abnormal uroflowmetry: interrupted voiding. Qura, urine flow; vura, volume of voided urine; voided volume, 370 mL; max flow rate, 9.7 mL/sec.

shape (Fig. 2). An interrupted flow, as seen in Fig. 3, suggests abdominal straining in most cases (remember to check that there is an adequate volume voided, over 150 mL). Other patterns may be seen: obstruction, as in prostatic enlargement, produces an unbroken curve with pronounced asymmetry; constriction (e.g. urethral stricture) produces a long, unbroken, plateau curve; a discontinuous or fractionated curve is seen in detrusor-sphincter dyssynergia (see below). This test is most useful if combined with measurement of **postmicturition residual volume**.

Residual volume

This is measured by either draining off the residual volume with a catheter, or by measuring it with an ultrasound scan. A normal shaped uroflow (with volume voided exceeding 150 mL) with no residual urine is a good screening test for urinary function.

In spinal cord disease, there is commonly a combination of detrusor hyperreflexia and incomplete voiding, and therefore removing the residual volume is important. Generally, a patient with neurogenic incontinence, if the residual volume exceeds 100 mL, would benefit from a procedure for bladder emptying such as clean intermittent self-catheterization. This may help to lessen symptoms of urgency and frequency, and minimize the risk of urinary tract infection or long-term upper tract problems (Fig. 4).

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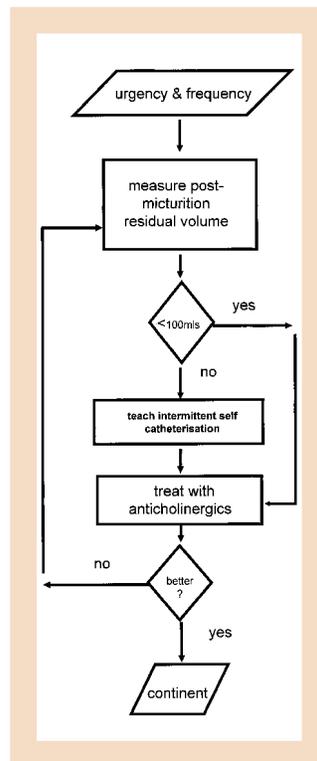

Figure 4 Algorithm for the management of neurogenic incontinence.

Electromyography (EMG)

Sphincter EMG has been used to detect abnormalities of sphincter behaviour during voiding ('kinesiological' studies) and to identify neuromuscular sphincter disorders. Recording from the striated muscle of the urethral sphincter or anal sphincter during cystometry is a means of detecting inappropriate sphincter contraction during detrusor contraction, the disorder known as detrusor-sphincter dyssynergia. However, since the advent of videourodynamics, this type of kinesiological EMG recording is performed less frequently. An example of this recording is seen in Fig. 5(c). Normally, on voiding, EMG activity in the urethral sphincter ceases before detrusor contraction. However, when detrusor-sphincter dyssynergia is present, there is concurrent sphincter activity together with detrusor contraction, as shown in the lower (blue) trace.

> urodynamics offers descriptive rather than diagnostic information

Motor unit analysis may reveal abnormalities in urethral and/or anal sphincter EMGs, which are recorded by needle electrode. Conditions where this test can have a role include cauda equina syndromes and in suspected multiple system atrophy (Vodusek & Fowler 1999). Also, a primary abnormality of the sphincter has been proposed, which may be the underlying problem in women who develop spontaneous unexplained urinary retention, perhaps in association with polycystic ovaries (Fowler *et al.* 1988). The characteristic findings in this sphincter EMG are complex repetitive discharges and decelerating bursts (the latter mimicking the sound of 'whales singing').

CYSTOMETRIC FINDINGS IN NEUROLOGICAL DISEASE

Detrusor hyperreflexia

Bladder overactivity, as shown by cystometry, is the most common abnormality in patients referred by urologists. If there is a known underlying neurological basis for this, then it is termed 'detrusor hyperreflexia', whereas if there is no neurological cause recognized then it is classified as 'detrusor instability'. Note that a range of neurological conditions can cause the same picture of hyperreflexia.

Typical symptoms of detrusor hyperreflexia are urgency, frequency and/or incontinence. There are unstable contractions of the bladder (by definition, a rise in detrusor pressure over 15cmH20) (Fig. 5a). These may or may not be associated with incontinence, as seen by the urine flow trace. The sensation of urgency is accompanied by slight leaking each time. These bladders generally have reduced capacity, and may also show loss of compliance (slowly increasing pressure rise towards the end of bladder filling). It is typically seen in a number of upper motor neurone lesions, which may be suprapontine (either cortical or extrapyramidal) or suprasacral. Interestingly and somewhat surprisingly, detrusor hyperreflexia may also be seen in some patients with cauda equina syndromes.

Cortical lesions producing detrusor hyperreflexia include stroke, tumour and dementia. Sakakibara *et al.* (1996) found that in a group of 72 patients admitted to hospital with stroke, 53% of them had urinary problems; urodynamic studies of 22 symptomatic patients showed detrusor hyperreflexia in 68%. Multiple System Atrophy (MSA) and Parkinson's Disease are examples of extrapyramidal pathology associated with detrusor hyperreflexia (MSA can also produce incomplete emptying, see below).

Examples of suprasacral lesions include multiple sclerosis (MS), spinal cord injury, transverse myelitis or spina bifida (though patients with spina bifida may have a combination of supra- and infra-sacral pathology). Detrusor hyperreflexia is the most common urodynamic finding in patients with MS and urinary symptoms. A correlation has been demonstrated between the presence of frequency/urgency and moderate-severe pyramidal dysfunction in the legs (Kurtzke score above 3) (Betts *et al.* 1993). Consequently there is an argument that urodynamic studies are unnecessary in patients with urgency and moderate disability, because one can assume that the bladder symptoms are due to detrusor hyperreflexia, and empirical treatment with anticholinergics can be started after measuring the residual volume (Fig. 4).

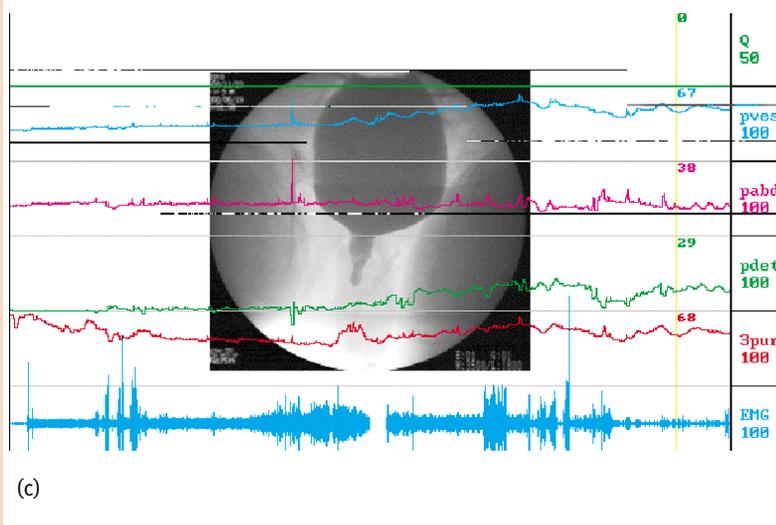### Detrusor–sphincter dyssynergia

The detrusor and external urethral sphincter are normally co-ordinated, such that the detrusor contracts and the sphincter relaxes. This co-ordination is lost in detrusor–sphincter dyssynergia.
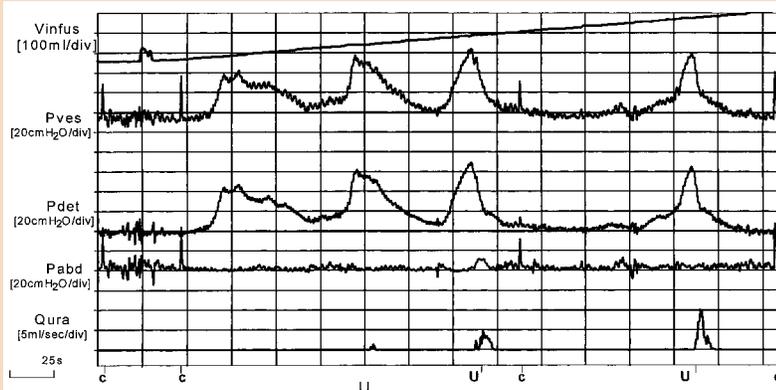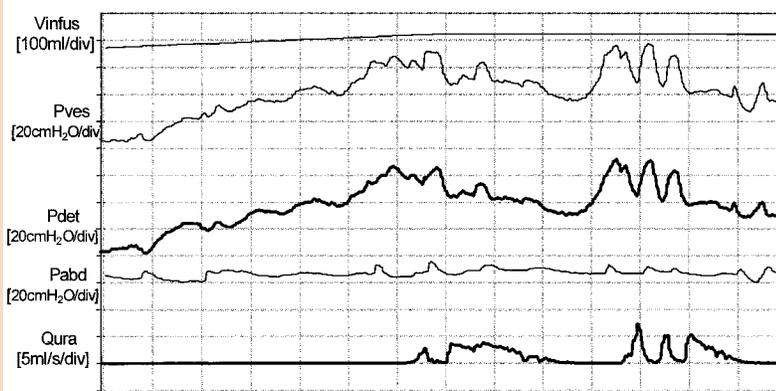
Figure 5 Urodynamic findings in detrusor hyperreflexia and detrusor-sphincter dyssynergia. (a) detrusor hyperreflexia (filling); (b) detrusor hyperreflexia and detrusor-sphincter dyssynergia (filling). (c) Fluoroscopic imaging and EMG trace showing detrusor-shincter dyssynergia (courtesy of Ryuji Sakakibara, unpublished data). The picture shows an open bladder neck with a closed urethral sphincter as a result of dyssynergia. When the patient attempts to void, one can see bursts of EMG activity, as shown by the thickness of the lower blue trace. There follows a rise in the detrusor pressure (lower green line), but no concomitant flow of urine (flat top green line), highlighting the difficulty these patients have in voiding. Vinfus, rate of bladder filling; Pves, intravesical pressure; Pdet, detrusor pressure; Pabd, intra-abdominal pressure; Qura, urine flow; C, cough; U, sensation of urgency.

It is caused by interruption of pathways between the pons and sacral cord. EMG recordings or videourodynamics [both seen in Fig. 5(c)] are required to recognize the condition which would otherwise have to be deduced from cystometry. For example, Fig. 5(b) suggests detrusor–sphincter dyssynergia with detrusor hyperreflexia, as there is delay between bladder contraction and

urethral relaxation, and therefore a delayed flow of urine. Figure 5(c) shows the anatomy of an open bladder neck and prostatic urethra but closed sphincter. There is increased EMG activity at the same time as raised detrusor pressure. Although this would typically produce an interrupted urinary flow, care must be taken not to confuse this with other cases where there is intermittent stream, because the labelling detrusor-sphincter dyssynergia commits one to searching for a neurological cause.

In suprasacral ('spinal') lesions, detrusor hyperreflexia may be seen together with detrusor-sphincter dyssynergia, whereas in suprapontine lesions there is no detrusor-sphincter dyssynergia associated with the detrusor hyperreflexia.

There is a reported frequency of detrusor-sphincter dyssynergia in multiple sclerosis varying from 18% to 66% (Betts 1999).

Detrusor hyporeflexia

This is typically seen in patients with infrasacral lesions such as cauda equina syndromes (for example, caused by trauma, central disc prolapse), peripheral neuropathies (such as in diabetes mellitus) or surgical damage to the pelvic plexus. The bladder has a larger capacity than normal and sensation of filling is diminished. Patients may present with a variety of urinary symptoms, such as frequency and nocturia, due to the large residual volume of urine (this also predisposes to recurrent urinary tract infection), difficulty voiding and even retention. A urinary flow rate and postvoid residual are simple measures that should be performed as first-line investigations.

The typical cystogram findings in a hyporeflexic bladder are shown in Fig. 6(a), with an increased bladder capacity during the filling phase. During voiding, there is considerable abdominal pressure generated (more so than the rise in detrusor pressure), corresponding to the periods of (interrupted) urinary flow [Fig. 6(b)].

Reduced bladder contractility can also occur in Multiple System Atrophy, in which neuronal atrophy can affect the central nervous system at various sites (whereas idiopathic Parkinson's Disease tends to be associated with bladder overactivity). Incomplete bladder emptying and poor flow are caused by cell loss in the intermediolateral cell columns of the spinal cord, producing loss of parasympathetic drive.

Obstructed voiding

Patients may be referred for a neurological opinion after a urologist has excluded anatomical obstruction of the
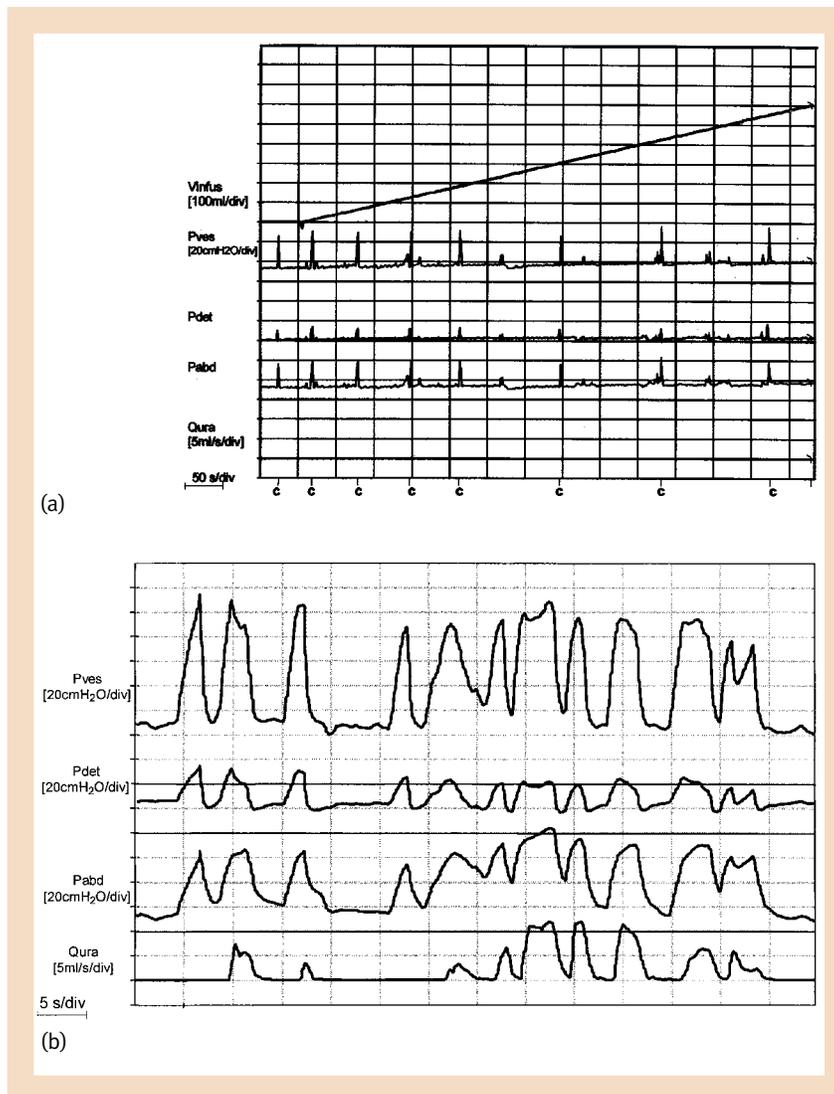

Figure 6 Cystometry in hyporeflexic bladder. (a) filling; (b) abdominal straining during voiding. Vinfus, rate of bladder filling; Pves, intravesical pressure; Pdet, detrusor pressure; Pabd, intra-abdominal pressure; Qura, urine flow; C, cough.
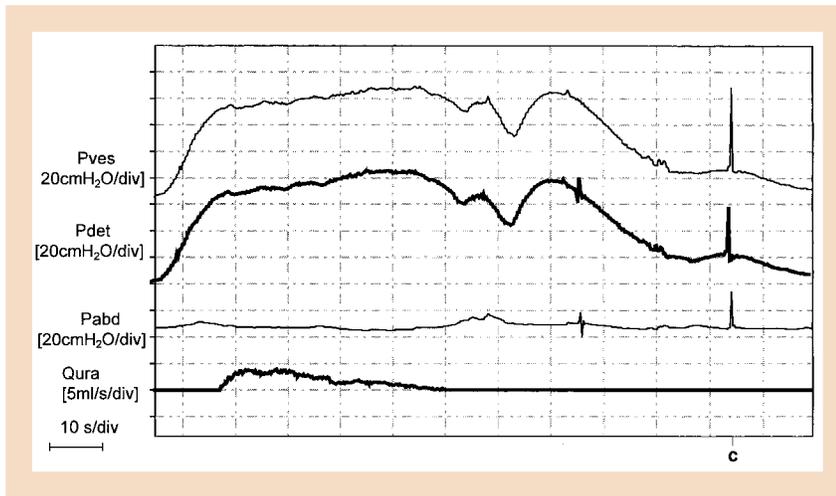
Figure 7 Obstructed voiding. Same conventions as fig. 1.

lower urinary tract by cystoscopy, and yet urodynamics are highly abnormal: these include an obstructive element, which is thought to characterize a 'neurogenic' pattern. The first intimation for this would be from the flow rate, where the height of the curve is much reduced, though it is important to ensure that there is a satisfactory volume voided (one can be misled if the volume of urine voided is too low to produce an adequate stream). A typical cystometrogram in a young female with obstructed voiding is shown in Fig. 7. Despite high detrusor pressure, the flow rate is low and the stream protracted. This pattern is similar to that seen with detrusor-sphincter dyssynergia, and a spinal cord lesion must be excluded. However, if no evidence of this is found on neurological examination, imaging or neurophysiology (lower limb somatosensory evoked potentials), then it is possible that the woman has a primary abnormality of the sphincter (Fowler *et al.* 1988).

CONCLUSION

Urodynamic techniques are useful in investigating the cause of a patient's bladder symptoms. The different patterns described here include the major abnormalities of detrusor-sphincter behaviour that are seen, although numerous variations are described in the urological literature. Neurologists may be asked to see patients because a particular pattern is thought to have a neurogenic basis. However it should be remembered that no urodynamic pattern is diagnostic of a particular underlying pathophysiology, whether it be structural (urological) or neurological, and that these tests are essentially descriptive.

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