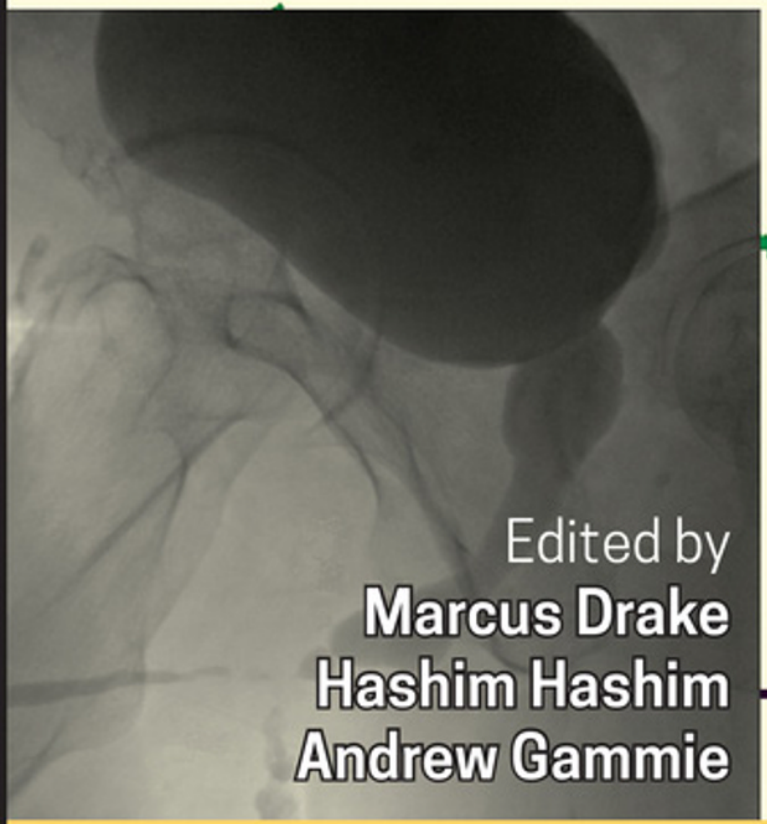
Fourth Edition

Abrams' URODYNAMICS



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WILEY Blackwell

Abrams' Urodynamics

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Contents

Abbreviations

ACh	Acetyl-choline
AD	Autonomic dysreflexia
ADH	Anti-diuretic hormone
AFC	Air-filled catheter
ANP	Atrial natriuretic peptide
AP	Antero-posterior
ARM	Anorectal manometry
ATP	Adenosine Triphosphate
AUDS	Ambulatory urodynamics
AUS	Artificial urinary sphincter
BCI	Bladder contractility index
BMI	Body mass index
BOO	Bladder outlet obstruction
BOOI	Bladder outlet obstruction index
BPE	Benign prostate enlargement
BPH	Benign prostatic hyperplasia
BPS	Bladder pain syndrome
BNI	Bladder neck incision
BNO	Bladder neck obstruction
BNP	Brain natriuretic peptide
BPO	Benign prostatic obstruction
BTX	Onabotulinum toxin-A
BWT	Bladder wall thickness
BVE	Bladder voiding efficiency
CC	Cystometric capacity
CEPNL	Cauda equina and peripheral nerves lesion (infrasacral)
CFS	Clinical Frailty Scale
CKD	Chronic kidney disease
CLPP	Cough leak point pressure
CNS	Central nervous system
CPAP	Continuous positive airway pressure
CSF	Cerebrospinal fluid
CSU	Catheter specimen of urine
CT	Computed tomography
CVA	Cerebro-vascular accident
DLPP	Detrusor leak point pressure
DLPV	Detrusor Leak Point Volume
DO	Detrusor overactivity
DOI	Detrusor overactivity incontinence
DRE	Digital rectal examination
DSD	Detrusor sphincter dyssynergia
DUA	Detrusor underactivity
ED	Erectile dysfunction
EBRT	External beam radiotherapy
EMG	Electromyogram
EUS	External urethral sphincter
FDA	Food and Drug administration
FFR	Free flow rate
Fr	French
FSF	First sensation of filling
FUTURE	Female Urgency, Trial of Urodynamics as Routine Evaluation
FVC	Frequency/volume chart
GI	Gastrointestinal
GUP	Good Urodynamic Practices
HR-ARM	High resolution anorectal manometry
HRM	High resolution manometry
IAS	Internal anal sphincter
IC	Intermittent catheterisation
ICI	International Consultation on Incontinence
ICIQ	International Consultation on Incontinence Questionnaire
ICIQ-B	International Consultation on Incontinence Questionnaire-Bowel symptoms
ICIQ-FLUTS	International Consultation on Incontinence Questionnaire-Female LUTS
ICIQ-MLUTS	International Consultation on Incontinence Questionnaire-Male LUTS
ICCS	International Children's Continence Society
ICS	International Continence Society
IR(ME)R	Ionising Radiation (Medical Exposure) Regulations
ISC	Intermittent self-catheterisation
ISD	Intrinsic sphincter deficiency
IVU	Intravenous urogram

LUTD	Lower Urinary Tract Dysfunction
LUTS	Lower Urinary Tract Symptoms
M	Muscarinic
MCUG	Micturating cystourethrogram
MRI	Magnetic resonance imaging
MS	Multiple sclerosis
MSA	Multiple system atrophy
MSU	Mid-stream urine
MUCP	Maximum urethral closure pressure
MUI	Mixed urinary incontinence
MUP	Maximum urethral pressure
MUT	Midurethral tape
MVV	Maximum voided volume
NIRS	Near infrared spectroscopy
NLUTD	Neurogenic Lower Urinary Tract Dysfunction
NDV	Normal desire to void
NICE	National Institute for Health and Clinical Excellence
NP	Nocturnal polyuria
NPH	Normal pressure hydrocephalus
NPi	Nocturnal polyuria index
NUV	Nocturnal urine volume
OAB	Overactive bladder
OSA	Obstructive sleep apnoea
PA	Postero-anterior
p_{abd}	Abdominal pressure
PAG	Periaqueductal grey
PCR	Penile compression-release
PD	Parkinson's disease
p_{det}	Detrusor pressure
$p_{detQmax}$	Detrusor pressure at maximum flow rate
PFC	Prefrontal cortex
PFME	Pelvic floor muscle exercises
PFS	Pressure-flow studies
PMC	Pontine micturition centre
PMD	Post-micturition dribble
PNS	Parasympathetic nervous system
POP	Pelvic organ prolapse
POP-Q	Pelvic organ prolapse quantification
PPI	Post-prostatectomy incontinence
PRIMUS	PRImary care Management of lower Urinary tract Symptoms
PRO	Patient-reported outcomes
PSA	Prostate-specific antigen
PTNS	Percutaneous tibial nerve stimulation
p_{ura}	Urethral pressure
PUV	Posterior urethral valves
p_{ves}	Vesical pressure
PVR	Post-void residual
Q_{max}	Maximum flow rate
RAIR	Recto Anal Inhibitory Reflex
SBO	Spina bifida occulta
SCI	Spinal cord injury
SDV	Strong desire to void
SPL	Suprapontine lesion
SSCL	Sacral Spinal Cord lesion
SSL	Suprasacral spinal cord/pontine lesion
SNM	Sacral neuromodulation
SNS	Sympathetic nervous system
SUI	Stress urinary incontinence
TURP	Transurethral resection of the prostate
TVT	Transvaginal tape
TWOC	Trial without catheter
UAB	Underactive bladder
UDS	Urodynamics
UPP	Urethral pressure profile
UPSTREAM	Urodynamics for Prostate Surgery: Randomised Evaluation of Assessment Methods
USI	Urodynamic stress incontinence
UTI	Urinary tract infection
UUI	Urgency urinary incontinence
UUT	Upper urinary tract
VLPP	Valsalva leak point pressure
VUDS	Video-urodynamics
VUJ	Vesicoureteric junction
VUR	Vesicoureteric reflux
VV	Voided volume
VVF	Vesico-vaginal fistula
WFC	Water-filled catheter

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Preface

Lower urinary tract dysfunction (LUTD) produces a large burden on sufferers in particular, and on society in general. Lower urinary tract symptoms (LUTS) are very prevalent; 5% of children aged 10 years wet the bed. In all, 15% of women and 7% of men have troublesome incontinence. In elderly men of 75 years, benign prostatic hyperplasia occurs in more than 80% of individuals, with benign prostatic enlargement coexisting in up to half this group and half of these having bladder outlet obstruction. Most people with a neurological disease have some form of LUTD.

Urodynamics is invaluable in assessing people with LUTD. The need to support the clinical assessment with objective measurement is accepted by most clinicians specialising in the care of patients with LUTS. Since the first edition of this book in 1983, urodynamics has become more widely accepted. The number of urodynamic units worldwide has increased to enable access to this important testing modality. Almost every hospital of any significance embraces urodynamic investigations as an essential part of the diagnostic pathway for urology and gynaecology departments. Further, specialists in geriatrics, paediatrics and neurology recognise the importance of urodynamics in the investigation of a significant minority of their patients. The expertise involved in assessing neurogenic LUTD by urodynamicists can help neurologists refine their insights into the neurological deficit in individual patients. However, the take-up is not universal, especially worldwide. This may result from the perceived cost to the healthcare unit, the presumed unpleasantness to the patient, and the varied expertise in functional urology.

The objective of this book is to deliver a definitive manual of practical urodynamics, showing how urodynamic investigation contributes to the management of patients and describing the tests clearly and comprehensively. To do this means not only discussing the tests but also showing in which clinical areas they help management and those where urodynamic tests are largely pointless. It means concentrating on the common clinical problems and on the presenting symptom complexes, while pointing out any limitations and possible artefacts of investigation.

The Bristol Urological Institute (BUI) serves a large patient population in South West England and has developed skills in urodynamics and functional urology over several decades. It runs educational courses (the Basic Urodynamics course, the Certificate of Urodynamics, and the Expert Urodynamics course) which take place in the UK and several places globally, and also online. This makes the BUI one of the world-renowned leading units in female and functional urology generally, and urodynamics specifically, that is visited by healthcare professionals from all over the world. Professor Paul Abrams was not the only individual responsible for this strength, but his contributions to Urology in Bristol and worldwide are truly impressive. They include the development of the Abrams-Griffiths nomogram [1], which was adopted by the International Continence Society (ICS) as the Bladder Outlet Obstruction Index. He was a major promoter of the ICS Standardisations of Terminology in Lower urinary tract function, including being the first author on the paper which has been more widely quoted from urology than any other [2]. He also serves as one of the Chairs of the International Consultations on Incontinence. When he wrote the preceding editions of this book, his aim was to help a clinician with no previous experience in urodynamics to appreciate both the value and limitations of the subject and give the necessary practical advice on the use of the appropriate equipment in the correct situations. This was delivered with characteristic wit and imagination (see figure). One of the principal reasons for producing the 3rd edition was the publication of the ICS terminology report and the 'Good Urodynamic Practices' document [3]. With the updating of Good Urodynamic Practices [4], and

now the 'Fundamentals of Urodynamic Practice' document [5], it is timely to continue Professor Abrams' achievements in this fourth edition, the first to become an eponymous *Abrams' Urodynamics*. In it, we have aimed to stay true to the importance of the practical application of urodynamic tests, we draw on the latest scientific evidence, have sourced an extensive new tranche of illustrations, and have revisited the ICS Standardisations to reflect their revisions in recent years. In doing so, we wish to record our personal appreciation of and debt to Paul Abrams' inspiration, leadership, and support of us and countless others in this field.

Marcus Drake, Hashim Hashim,
Andrew Gammie, 2020
Bristol Urological Institute
and University of Bristol

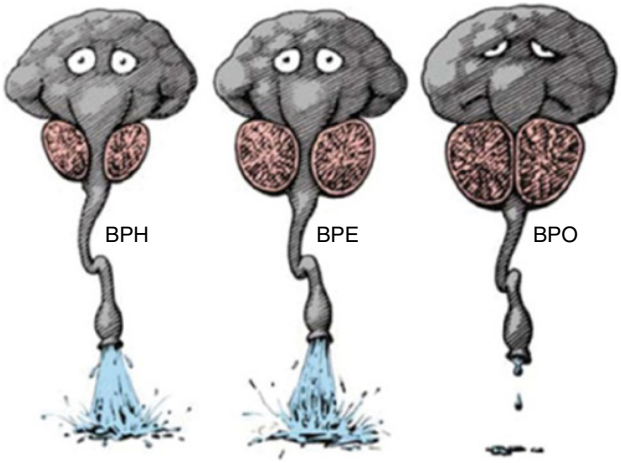

Figure A classic picture of the fundamental insights on the implications of prostate pathology for the male lower urinary tract, showing the relationships between benign prostate hyperplasia (BPH), benign prostate enlargement (BPE), and benign prostate obstruction (BPO). *Source*: Drawn by Alex James from a sketch by Paul Abrams in 1993.

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First Foreword

I feel honoured and humbled to find my name attached to this new edition, but need to set the record straight!

After graduating from Sheffield, I arrived in Bristol in 1972 to begin my surgical training. The surgical registrar (resident) was Michael Torrens, who was a neurosurgical resident, and about to become Roger Feneley's new research fellow in the newly founded urodynamic unit. Mike was going to use urodynamics to evaluate sacral neurectomy in women with intractable detrusor overactivity, then called 'detrusor instability'. After six months of general surgery, I rotated to the urology department and got interested in the older men coming for TURP for their 'prostatism'. Even then I was an annoying and inquisitive individual who constantly asked, 'Why ..... ?'. Mike suggested that I approach Roger to see whether I could start to assess these men, initially by urine flow measurement. I describe these beginnings of my urological life, as they were determined by serendipity. The opportunities I was given, and those I worked with, in that first year in Bristol determined the rest of my professional career. Mike's early encouragement and advice, and Roger's mentorship throughout, have been the bedrock of my professional development. Roger provided the environment where all young, naive but enthusiastic clinicians could speak without fear, knowing that their unanswered questions could be pursued in an academically sound manner according to the null hypothesis.

It has been my privilege and pleasure to work in academic and clinical teams that have been devoted to patient care, and free of rancour and division. The stability of the urodynamic team in Bristol was anchored by Roger initially, then by Angela Shepherd, followed by Lucy Swithinbank and now by Hashim Hashim. The technical side of urodynamics is also of paramount importance to the quality of service. From the beginning, Pat Lewis and then Sue Howell ensured that Bristol Urodynamics adhered to high technical standards, and the clinicians were kept on the 'straight and narrow': any upstart doctor was reminded who were the most important members of staff! We have continued to be most fortunate in having excellent scientific colleagues. Andrew Gammie is our first clinical engineer, and Laura Thomas is our first clinical scientist. With their involvement, not only has our urodynamic quality advanced, but our teaching activity too has been able to improve in quality.

I owe great debts to many others. I met and worked with Derek Griffiths, then in the physics department at Exeter. He was my urodynamic and scientific mentor, and he taught me 'intellectual honesty' – what I knew from what I thought I knew. We collaborated for many years, even after he moved to Holland and then to North America. Early on, I met Alan Wein and Linda Cardozo, who have both been very important in developing functional urology worldwide, and together we have worked closely with the International Continence Society (ICS) and in developing the International Consultation on Incontinence (ICI), and from that the ICI Research Society. Saad Khoury has been another valued mentor who made the ICI possible and has been an important and wise counsel for many years. The camaraderie of these old friends has been very important to me.

Urodynamics remains a controversial subject to the 'non-believers', and there remains much to be achieved in identifying its exact place in the evaluation of lower urinary tract dysfunction (LUTD). What is clear is that the *a priori* argument remains: the bladder, urethra and sphincter are, in engineering terms, a reservoir, outlet and valve, and therefore must be studied by pressure and flow measurements. The United Kingdom Continence Society (UKCS) has led the way in determining how urodynamics can improve and reach the quality standards of other physiological measurement units. Another of the fundamental problems, in managing patients' problems, is that the LUT is connected to the brain. Of course, this is essential, but it leads to enormous problems, as the nervous system is so incompletely understood. In any urodynamic team, there has to be a basic science including neurophysiological input, and Marcus Drake has

added that dimension to our work. Advances will not come until we develop our understanding of the interactions between the nervous system and the LUT. Marcus, like many other members of the urodynamic team, has completed his training in other centres, and this cross-fertilisation by ideas is essential for creative thinking. I am proud that I have had a part in appointing colleagues who will develop a wider range of skills than I have had. This is never a threat, only an opportunity.

We all have the duty to educate, and I hope this book will support that effort. Finally, I must thank my wife Kirsten and my children, the members of 'my crew', who ground me when necessary, and are ever tolerant and supportive. They have certainly become familiar with the basics of lower urinary tract function! So, in addition to developing the science of urodynamics, we have to help all people, as well as patients, to better understand their bladder function so that they can preserve their bladder health and help themselves when it 'plays up'.

Thank you, Marcus, Andrew and Hashim, for your collaboration in science and clinical work and for your friendship.

Professor Paul Abrams, Bristol
December 2020

Figure Marcus Drake, Hashim Hashim, Paul Abrams, and Andrew Gammie holding an extremely long flowmetry printout that exemplifies slow flow and terminal dribble. Bristol, September 2020.

Second Foreword

The name 'Paul Abrams' has been synonymous with expertise in many subjects associated with normal function and lower urinary tract dysfunction (LUTD), but none more so than the science, performance, interpretation and clinical utility of urodynamics of the LUT. I thought that I was a good organiser of subject material and a good and succinct writer when I picked up the first edition of *Urodynamics* in 1983, but I had to tip my hat to Paul. The 229 pages of this text rapidly became the 'gold standard', and the charts, tables and diagrams quickly became a part of my own presentations on the subject (properly referenced, of course!). I found the organisation of the subject, which included the science necessary to understand what happens during filling/storage and emptying/voiding, and how to properly measure and categorise the findings, to 'make sense' and enable understanding of where these straightforwardly explained techniques fit into the overall evaluation of LUTD. The notes on management were a bonus.

Subsequent editions (I am looking at the third now - 331 pages) have expanded the concepts and techniques that have occurred parallel to the advances in the related scientific disciplines, just as the terminology has evolved (please do not ever say 'urge incontinence' as opposed to 'urgency incontinence' in Paul's presence!). It is only fitting that the title of this book, the most complete text on the science and practice of urodynamics, now be preceded by Paul's name and carried on by members of the department that he developed.

Paul, congratulations on having the text renamed *Abrams' Urodynamics*, an honour well deserved!

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Part I

Basic Principles

1

Basic Urodynamics and Fundamental Issues

Marcus Drake[1], Andrew Gammie[2], Laura Thomas[3], Arturo García-Mora[4], and Hashim Hashim[2]

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CONTENTS

Introduction to Urodynamics

Urodynamics has two basic aims:

- **To reproduce the patient's symptomatic complaints while making key observations**
- **To provide a pathophysiological explanation by correlating the patient's symptoms with the urodynamic findings**

These two basic aims are crucial to the purpose of urodynamics – essentially, it is a diagnostic test that will aid in the management of patients. The need to make urodynamic observations reflects the fact that the patient's symptoms are important, but they might be somewhat misleading. Most patients with lower urinary tract dysfunction (LUTD) present to their doctor with symptoms. However, lower urinary tract symptoms (LUTS – Table 1.1) should not simply be taken at face value, since a range of differing mechanisms may result in rather similar symptomatic presentations. The statement 'the bladder is an unreliable witness' [2] reflects how symptoms are the starting point but do not actually identify the ultimate explanation. Since treatment should correct the underlying cause, it is necessary to identify mechanisms, avoiding assumption or prejudice coming from taking symptoms at face value. An excellent example of this is voiding LUTS in men, where the cause on urodynamic testing may prove to be bladder outlet obstruction (BOO) and/or detrusor underactivity (DUA); BOO should respond fully to surgery to relieve obstruction such as transurethral resection of prostate (TURP), while such surgery is potentially not helpful in the second [3]. Voiding LUTS in males are of unreliable diagnostic value, and only slow stream and hesitancy show any correlation with the urodynamic findings of BOO [4–6]. Even with flow rate assessment, one cannot be sure whether BOO is present (Figure 1.1). The difficulty of assessing LUTD by symptoms alone is the uncertainty about establishing truly what is going on in the individual describing them.

For women diagnosed by their symptoms as having stress urinary incontinence (SUI), only 50–68% have urodynamic stress incontinence (USI) [7, 8]. These studies also looked at patients with apparent overactive bladder (OAB) symptoms presumed to be the result of detrusor overactivity (DO), and here, the correspondence was

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Table 1.1 Classification of lower urinary tract symptoms (LUTS) [1].

Storage	Voiding	Post-micturition
Urgency	Slow stream	Post-micturition dribbling
Urinary incontinence	Splitting/spraying	Feeling of incomplete emptying
Increased daytime frequency	Intermittency	
Nocturia	Hesitancy	
Pain	Straining	
	Terminal dribbling	

Note: Do NOT forget to enquire about Pelvic Organ Prolapse in Women and Erectile Dysfunction in Men.*Source*: Modified from Abrams et al. [1].

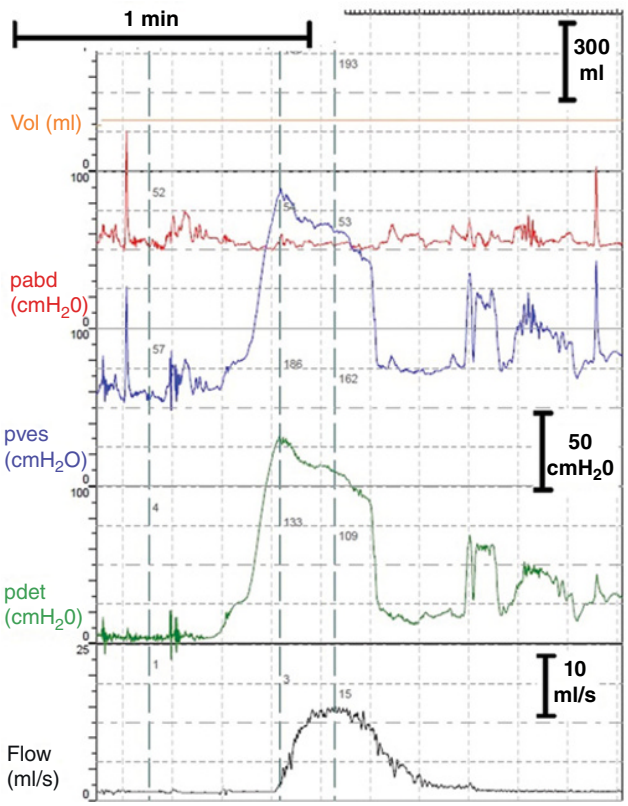

Figure 1.1 Flow rate testing in men gives an uncertain understanding. This man had previously done a free flow rate test which showed a reasonable maximum flow rate of 16 ml/s; taken alone, this might suggest he does not have bladder outlet obstruction (BOO). However, when he attended for urodynamics (see the pressure-flow study illustrated above), his flow rate was 15 ml/s as shown, but the pressure needed to achieve this was high, indicating BOO is present (see Chapter 14 for more details on assessing BOO in men).

33–51%. A key factor is the link to coughing, often used as a question to elicit a history of SUI; if a woman says 'I leak when I cough', it sounds like SUI. However, a cough can be a trigger to set off an overactive detrusor contraction, leading to detrusor overactivity incontinence (DOI) (Figure 1.2). Thus, the history may suggest that SUI treatment is needed, but for some of these women, the urodynamic observation identifies that DO treatment is the appropriate choice.

Accordingly, in both men and women, there is potential mismatch between reported LUTS and the LUTD identified by detailed investigation. This issue is particularly prominent in people with neurological conditions and children. In neurological disease, it is common for sensation to be absent or abnormal, making LUTS even more difficult to interpret. Children may find it difficult to describe their symptoms in any setting and particularly in a healthcare environment. Because symptoms have been shown to lack diagnostic specificity in the key clinical groups, it is not surprising to find that when surgery was based on symptoms alone, the results could be unsatisfactory. Urodynamic studies provide explanations for many symptoms based on mechanism and accordingly provide better support for therapy selection.

There is a well-recognised and substantial placebo effect for therapy in patients with LUTS. The symptoms of men with proven BOO, secondary to benign prostatic enlargement (BPE), can be improved in 40–60% of men in the placebo arm of drug studies. Such an effect can be surmised in men undergoing prostate surgery, but usually it is not sustained and in due course will be counterbalanced by the other effects of surgery, notably impairment of sexual function. Some patients submitted for surgery without objective confirmation of their condition potentially can do badly; this might reflect a poor-quality operation, or it may be that the problem lay in the preceding assessment. Urodynamics in modern practice gives greater insight into each patient's LUTD and hence helps advise patients on potential benefit and risks for intervention, to support their expectations of informed decision-making.

Ultimately, a successful urodynamics test is a clinically relevant investigation which seeks:

- to reproduce the patient's symptoms,
- to define bladder and urethral function,
- to provide precise diagnoses,

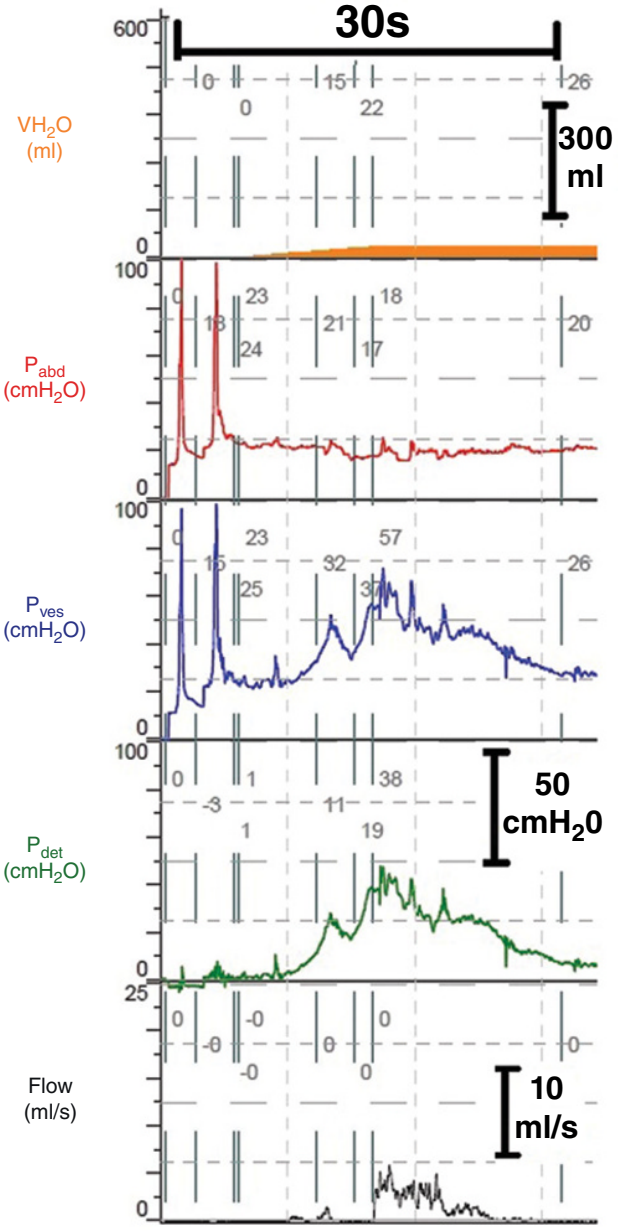

Figure 1.2 A woman who reported leakage with coughing in her history, suggestive of stress urinary incontinence (SUI). Her urodynamic test showed cough-provoked detrusor overactivity (DO) incontinence, and she described this as representative of her presenting complaint. Hence, this is not urodynamic stress incontinence, but effort-provoked detrusor overactivity incontinence; the symptomatic presentation was misleading and could have led to inappropriate surgery for SUI (see Chapter 13 for more details on assessing incontinence in women).

- to define the most significant abnormality,
- to allow selection of most appropriate treatment, and
- to predict post-operative problems.

This book describes how these can be achieved across a wide range of settings, complying with modern practice standards, and how to deal with challenges that may be encountered.

The Urodynamic History and Examination

When meeting a new patient with LUTS, it is important to establish a rapport. The LUTS present must be captured systematically, identifying the severity of individual symptoms and the bother each causes to the patient, preferably by using a symptom score completed before the appointment. There are several developed by the International Consultation on Incontinence Questionnaires group [9] which can suit a wide range of patients. They have the advantage of efficiently capturing both severity and bother for each symptom.

The history needs to cover several influences, for example:

- previous urological treatments,
- urinary tract infections (UTIs) – confirmed or suspected,
- obstetric and gynaecological background (in women),
- bowel function,
- sexual problems, including sexual trauma in the past, or recent emergence of sexual dysfunction,
- medical problems and medications, and
- the possibility of an underlying neurological condition.

Malignancy and neurological disease are key considerations. Undiagnosed cancer, such as bladder, prostate, gynaecological, or pelvic malignancy, must also be considered and is potentially at the back of the patient's mind, even if they don't say so [10]. Most patients with neurological disease have been diagnosed as such before coming to have LUTS assessed. However, some conditions can cause LUTS early on in the disease process. In these patients, it is possible that no one has yet realised the situation. Urological clinics sometimes encounter LUTS which turn out, on investigation, to have been caused by a neurological problem that has not yet been diagnosed – 'occult neurology' [11]; the main conditions which can cause this sort of situation are described in the last part of this chapter.

Patients referred for urodynamics will have been examined in a general way, either in the hospital clinic from which the referral originated, or by the patient's general practitioner (primary care physician). Hence, the urodynamic staff should concentrate efforts on a physical examination relevant to the symptomatic complaints and the possible underlying pathophysiological processes, for example:

- features suggestive of wider problems, such as neurological disease (e.g. slurred speech, altered gait, and tremor),

- abdominal examination to identify scars from previous surgery, or a distended palpable bladder, and
- internal examination to assess pelvic floor tone and contraction, pelvic organ prolapse, or formal prostate evaluation.

Urine examination should be performed in all patients, in the form of a urine dipstick to help rule out obvious causes for the LUTS. Other tests, such as blood tests (e.g. renal function and prostate-specific antigen), radiology, and endoscopy, have their indications and may need to be conducted alongside the ongoing LUTD assessment in accordance with the applicable clinical guidelines.

Invasive urodynamic studies are generally not indicated early in the pathway. They follow on once

1) careful investigations have been performed to exclude other pathologies that might mimic LUTD,
2) a bladder diary has been completed,
3) urinary free flow rate test and post-void residual (PVR) have been done, and
4) conservative treatment, which may include testing out response to medications, has been undertaken for a sufficient duration.

The Aims and Considerations of Urodynamics

A urodynamic test has several aims:

- To reproduce the patient's symptoms
- To define bladder and urethral function
- To provide precise diagnoses
- To define the most significant abnormality
- To allow selection of most appropriate treatment
- To predict potential post-operative problems
- To assess the results of treatment

The prelude to a urodynamic test is to identify the information needed, which can be described as 'formulating the urodynamic question'. The needs of the patient are fundamentally to resolve bothersome symptoms and reduce possible future problems. The history, symptom score, and bladder diary will help specify the situation. It follows that the needs of the clinician are to help suitable therapy selection and ensure avoidance of harm by identifying causative mechanisms. The urodynamicist should be considering 'what do I want to know about this patient?' It can be considered in terms of the micturition cycle ('What is wrong with storage, what is wrong with voiding?') and in terms of the lower urinary tract organs ('What is wrong with the bladder, what is wrong with the bladder outlet?'). In this way, the urodynamicist is in a position to consider 'Which urodynamic investigations need to be performed to define this patient's problems?'

This question will concentrate the clinician's thought processes on undertaking only those investigations which can help to make the diagnosis or indicate the line of management. For example, if a young male patient previously had urethral stricture treatment and voiding LUTS have returned, urine flow measurement will be the principal urodynamic test to identify if stricture recurrence is likely.

Once the questions have been defined, it will become apparent which urodynamic tests to do, as discussed below. The next question should be: 'Is the investigation likely to be of benefit to the patient?' This question reflects how the increased knowledge generated by the test might influence the clinical management. Several aspects are relevant, including:

1) *Individual considerations*: Is there therapy available, and is the patient healthy enough to tolerate the therapy?
2) *Disease knowledge*: In a difficult clinical area without effective treatment, urodynamic insights may facilitate introduction of treatment options in the future. An example is the introduction by the International Continence Society (ICS) of the concept of underactive bladder syndrome [12], which currently has no specific effective treatment but which will stand a better chance of future therapy development now that a terminology framework is in place.
3) *Financial cost and risks of testing*: The incidence of UTI after a UDS test is 2–3%, and some discomfort may be experienced.
4) *Potential harm the tests could do*: In particular, 'Is the urodynamic unit able to make a reliable diagnosis?' with erroneous diagnosis being the greatest concern. Three factors are crucial:

- The urodynamic technique should be free of technical artefacts.
  - The results of investigations should be reproducible.
  - The clinician should be properly trained and able to interpret the results of the urodynamics (Figure 1.3).

From a technical point of view, the tests must be carried out in a careful way, continuously monitoring during the test and eliminating artefacts (see Chapters 18 and 19). The clinician needs to allow for variation in LUTS from day to day and symptomatic progression over longer timescales. At the end of the urodynamic tests, it is pertinent to ask 'Did the urodynamic studies reproduce the patient's complaints and did the complaints correlate with known urodynamic features?' Answers to this question would be yes, no, or partially.

In the Bristol unit, we believe the presence of the clinician, or an experienced practitioner who is aware of the therapeutic possibilities of subsequent treatment, is very beneficial during tests. This individual can then consider

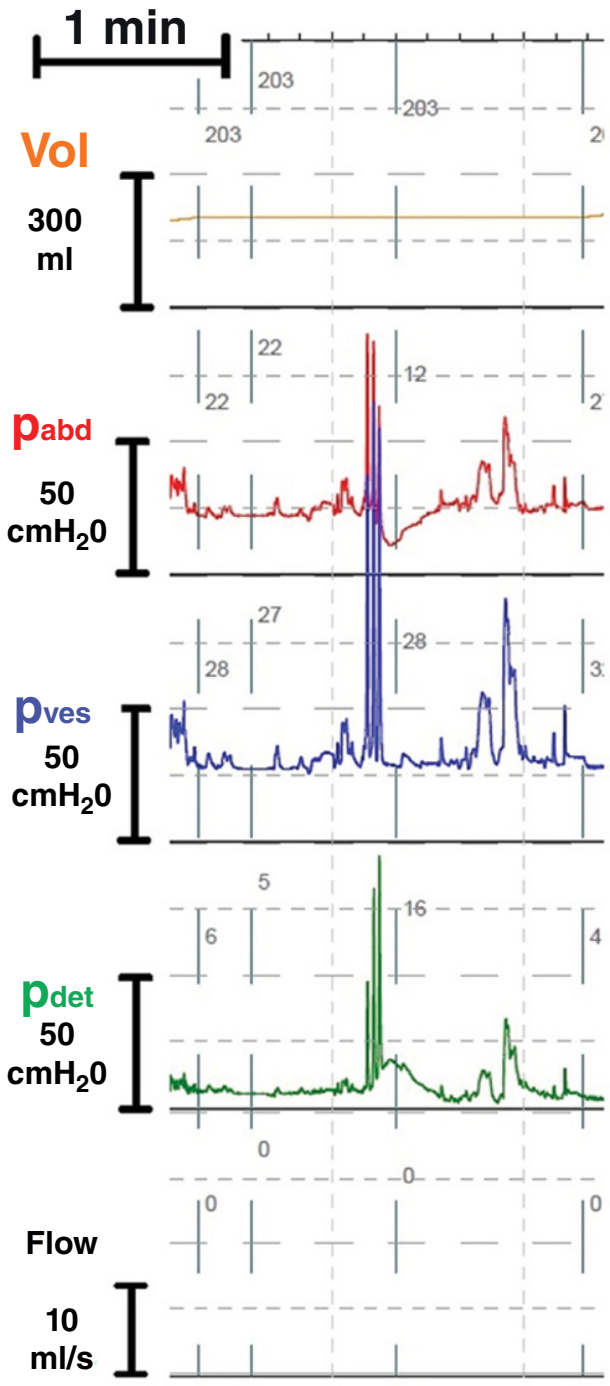

Figure 1.3 The importance of training and interpretation. This trace shows a brief moment from a filling cystometry. It illustrates a transient rise in detrusor pressure after a cough (green tracing), which resembles the detrusor overactivity (DO) seen in Figure 1.2 (but with no associated incontinence). Inspection of the bladder pressure trace (blue tracing) shows there was no bladder contraction associated with the detrusor pressure change, so this is not DO- despite the appearance. The actual explanation is that the cough caused the rectal catheter to shift, causing the recorded drop in abdominal pressure (red tracing) – an entirely different process from an involuntary bladder contraction. Proper training and interpretation will ensure that a mistaken diagnosis of DO is avoided.

whether the sensations felt by the patient during testing fit with the patient's reported everyday experiences and how they may relate to the urodynamic observations. Occasionally, during urodynamic studies, the patient may complain of a symptom they do not generally experience in everyday life, for example, urgency. Alternatively, a urodynamic abnormality may be noted which does not correlate with the patient's symptoms. These discrepancies can be detected and put into perspective if the clinician is present. However, if the urodynamics is delegated to someone with minimal urodynamic experience, the matching of LUTS to observations which underpins therapy selection is less direct. They may likely develop a basic test report which is observational and does not have the clinical interpretations at its heart. This report is of huge importance in therapy decision-making, with potentially life-long implications for the patient. Accordingly, a surgeon making decisions based on a basic report must consider: 'Does the report make sense in the context of the patient's symptoms and preceding tests?' and 'Can the features mentioned in the report be identified on the plotted traces, and is anything visible on the traces not mentioned in the report?'

In some instances, more than one abnormality is detected, so it is important to ask: 'Can urodynamics decide which abnormality is the most significant, if more than one is detected?' Multiple abnormalities are commonly seen in patients with neurogenic LUTD. They also occur in non-neurological patients, such as in women with mixed urinary incontinence. Treatment should be directed to the most significant and/or troublesome abnormality. Hence, the correspondence between the patient's symptomatic complaint and the urodynamic findings is important and needs to be documented in the report.

As well as seeking answers to the above questions, the urodynamicist needs to define the goals of the invasive urodynamic investigation, and these can be listed as follows:

- To **increase diagnostic accuracy** above that which can be achieved by non-urodynamic means.
- To **make a diagnosis on which a management plan can be based**. OAB is usually treated empirically; if a patient fails conservative and medical therapy, urodynamic proof of DO is appropriate prior to invasive surgery.
- If there are coexisting abnormalities, to **provide evidence to determine which abnormality should be treated first**. In a female patient with mixed urinary incontinence, it is usually possible to decide which is the main problem and so establish the treatment priority by careful assessment during urodynamics.
- To **define the current situation as a baseline for future surveillance**. In spinal cord trauma, it is usual to perform urodynamics after spinal shock has resolved. These baseline urodynamics establish whether there is a detrusor contraction in reaction to bladder filling and

whether or not detrusor-sphincter-dyssynergia (DSD) has developed. DSD is a potentially dangerous condition, as discussed in Chapter 16.

- To **predict problems that may follow treatment interventions**. Elderly men with BOO and coexisting DO should be warned that whilst their urine flows and other voiding symptoms should be improved by TURP, OAB symptoms due to DO may persist and in fact leakage due to the DO may occur.
- To **provide evidence that decides the timing of treatment**. In patients with neurological disease (e.g. meningomyelocele) being treated by antimuscarinics, ultrasound may show the development of upper tract dilatation. Urodynamics are vital to confirm whether or not poor bladder compliance is the cause, such that intervention is needed.
- To **exclude abnormalities which might interfere with the management**. For example, in patients with SUI being considered for an artificial urinary sphincter (AUS), demonstration of DO or poor bladder compliance would indicate the need for extra treatment to ensure that the additional problem is resolved (Figure 1.4).
- To **assess the natural history of** LUTD. Our unit, by investigating men and women studied many years ago, provided important evidence as to the natural history of LUT dysfunction [3, 13, 14].
- To **assess the results of treatments**. Simple urodynamics tests, such as urine flow studies, should be used

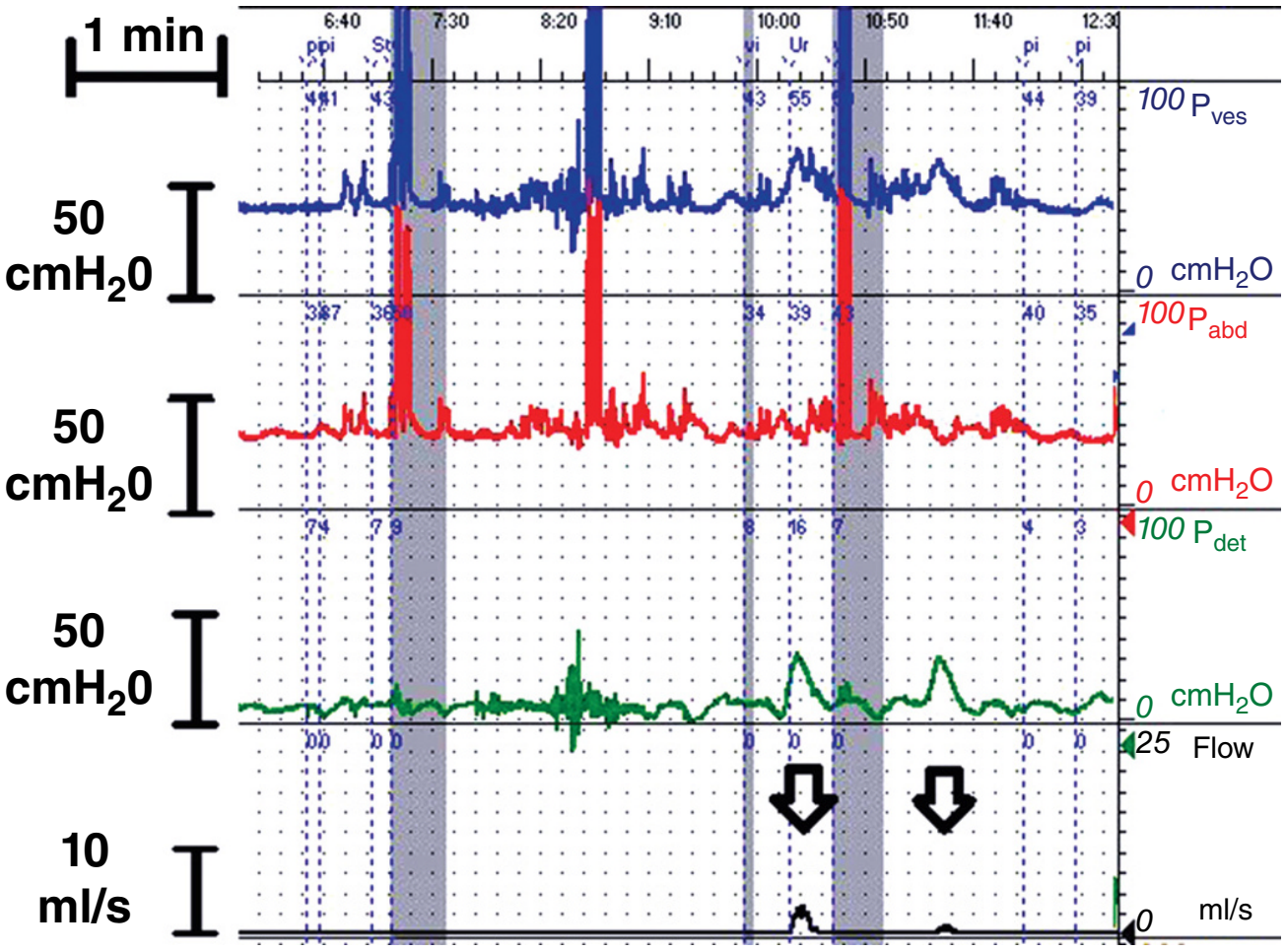

Figure 1.4 A man with a functioning artificial urinary sphincter (AUS) previously placed to treat post-prostatectomy incontinence; he subsequently complained of painful urgency incontinence. This illustration shows a small section of the filling phase, with a series of forceful coughs (stress testing) that did not cause leakage; a fully active AUS can resist 61–70 cmH_2O pressure. While he did not experience stress incontinence despite several forceful coughs, he did experience leakage with low amplitude of detrusor overactivity (DO) (black arrows), and it is hard to explain how forceful coughs did not cause leakage yet low amplitude DO did – we speculate this was due to the sustained nature of the DO pressure change, allowing greater effect on the pressure in the urethra. Indeed, pressure building up in his proximal urethra may have been responsible for the discomfort he described with his urgency incontinence. Note, this trace is not displayed with the correct sequence of traces; the bladder pressure should not be at the top for 'logistical' reasons, notably for the fact that some of the trace may go off the top of the page, as in this case.

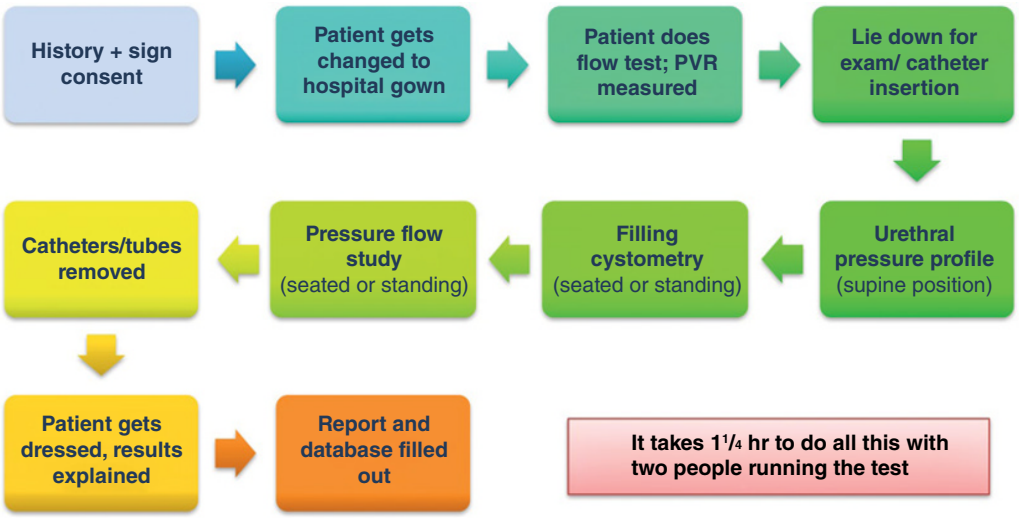

Figure 1.5 Urodynamics pathway in Bristol UDS Unit.

to evaluate outcomes of surgery, for example, after optical urethrotomy for urethral stricture.

This book will help readers understand the scientific basis of LUTD and urodynamic evaluation. It takes a therapy-led approach to framing urodynamic questions in a wide range of contexts. One of the great advantages of the Bristol unit is that adequate time is given for close questioning, the relevant physical examination, a calm urodynamic investigation, and practical advice, usually including the therapeutic recommendation and counselling, generating a report, and filling out the urodynamics database. Generally, we allow 75 minutes for each case, though shorter time is possible in clear-cut straightforward cases (Figure 1.5). This approach is worth emulating in all units, since the insight into each patient's needs and expectations, and ability to help their understanding as they make important decisions, is profoundly beneficial.

The prospect of having this type of clinical investigation causes anxiety for some individuals. Hence, beforehand, it is essential to give proper information, and a leaflet designed in conjunction with patients is probably the most practical and effective way to do this; the one we use in Bristol is included in the Appendix C.2 of this book. Patients should be introduced to the clinicians present and informed of their role, with agreement sought for the involvement of trainees in the procedure [10]. A written consent form needs to be signed. During urodynamic practice, efforts should be made to limit the number of staff present in the assessment room and ensure maximum possible privacy is maintained. After the test, patients should be allowed to get dressed in their normal clothes before the concluding discussions. It is important to discuss side-effects with patients and what to do if they experience any problems. Clinicians and patients will ideally discuss the results of urodynamic testing on the same day as the test or shortly after, with the detail and depth of the explanation in line with the patient's personal preference [10]. This assimilates the patient's medical background, previous therapy, and treatment preferences and goals to guide the therapeutic pathway to a suitable culmination (Figure 1.6).

Basics of Urodynamics

This section describes a starting point for anyone new to urodynamics. It gives a brief summary of what the test is about for starting practice, with directions to help find more details on the key points elsewhere in the book. It should be read alongside the ICS 'Fundamentals' documents for LUTS [15], flow rate testing [16], and urodynamics [17], which are given in the Appendix B.1. Once people have become familiar with basic testing, the extensive experience described in detail in the book will help readers respond to the numerous situations that can arise in real-life practice.

What Is Urodynamics?

Urodynamics is the umbrella term that covers investigations of lower urinary tract function. The term includes uroflowmetry and cystometry, which are the basic tests, along with the advanced tests such as video urodynamics, urethral pressure profilometry, and ambulatory urodynamics. Standard cystometry is the commonest investigation assessing storage function (filling cystometry) and voiding (pressure-flow study [PFS]). Both are normally performed as part of every investigation, unless the patient is unable

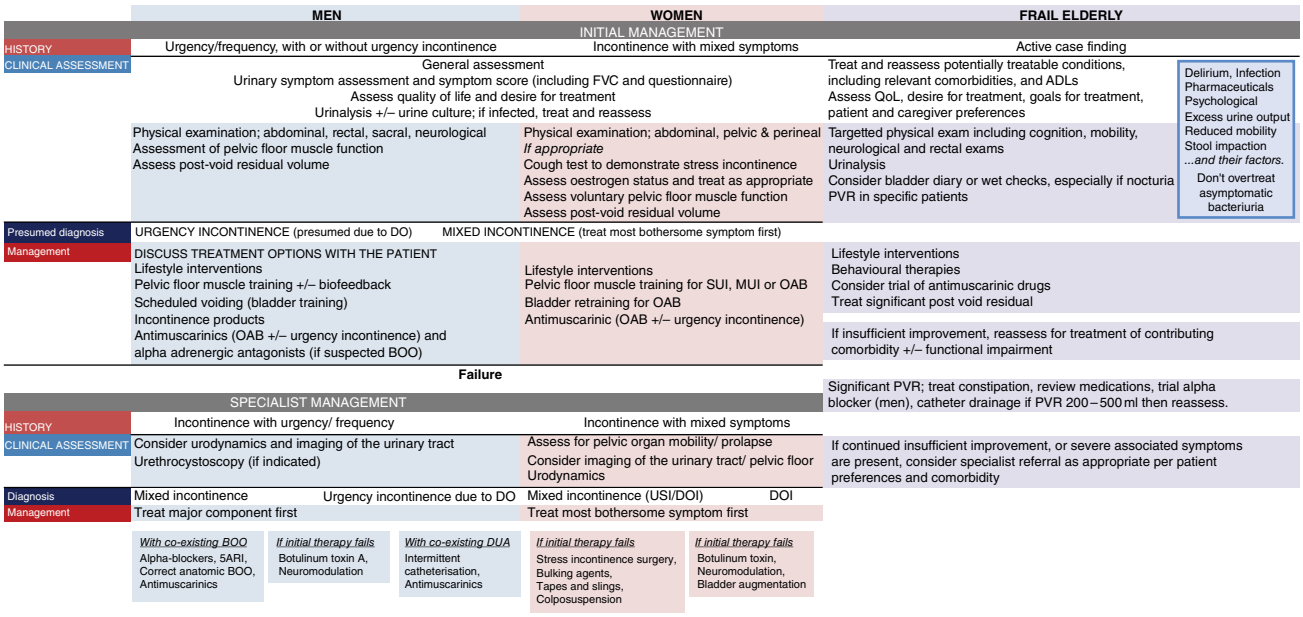

Figure 1.6 The diagnostic and therapeutic pathways for important patient groups seeking treatment of lower urinary tract symptoms (LUTS), summarising key points from the International Consultation on Incontinence algorithms. 5ARI: 5-alpha reductase inhibitor; BOO: bladder outlet obstruction; DO(I): detrusor overactivity (incontinence); DUA: detrusor underactivity; FVC: frequency volume chart; MUI: mixed urinary incontinence; OAB: overactive bladder; PVR: post-void residual; SUI: stress urinary incontinence; ADLs: Activities of daily living

to void (in which case filling cystometry alone would be carried out). Cystometry aims to reproduce a patient's symptoms and, by means of pressure measurements, provide a pathophysiological explanation for them.

During cystometry, there is a constant dialogue between the investigator and the patient so that any symptoms experienced during the test can be related to urodynamic findings. A full report is produced following a urodynamic investigation, which will normally include history, examination, urodynamic findings, and suggestions concerning management. The report should state whether the patient's symptoms were reproduced and whether voiding was felt to be representative.

What Is Measured?

Detrusor pressure is measured indirectly from bladder and abdominal pressures (respectively, referred to as p_{ves} and p_{abd}) using the formula: $p_{ves} - p_{abd} = p_{det}$. Abdominal pressure is measured because the bladder is an abdominal organ, so it is necessary to allow for the effect of increases in abdominal pressure, for example, straining, on vesical pressure. Pressure can be measured as the height of a column of fluid. To describe pressure, you simply need to specify what the fluid is and the height to which it goes. In urodynamics, the unit of pressure has been standardised as if we are measuring the height of a column of water in centimetres (cmH_2O). There are usually two pressure transducers associated with urodynamic equipment. One to measure p_{ves} and one to measure p_{abd}. The pressure generated by the detrusor smooth muscle, p_{det}, is derived by the urodynamic equipment electronically subtracting p_{abd} from p_{ves}.

Pressure transducers are not perfect instruments; therefore, it is important to regularly check their calibration to ensure that accurate pressure measurements are always made. In most urodynamics, the transducers are attached to the urodynamic equipment and are remote from the patient. Pressures inside the patient are transmitted to the pressure transducers via water-filled pressure catheters. To ensure appropriate pressure measurements, there must be:

1) no bubbles of air in the water connection between the patient and the transducer,
2) no water leaks, and
3) a good connection between the transducer dome and the diaphragm of the transducer if using non-disposable transducers.

A simple check of calibration for external pressure transducers (before connection to the patient) is to move the end of the filled pressure line through a known vertical distance (e.g. 20 cm) above the transducer dome and the pressure reading on the urodynamic equipment should change by the same amount (i.e. 20 cmH_2O). For air-filled or catheter tip transducers, calibration can be checked, if necessary, by submerging the catheter tip in a known depth of

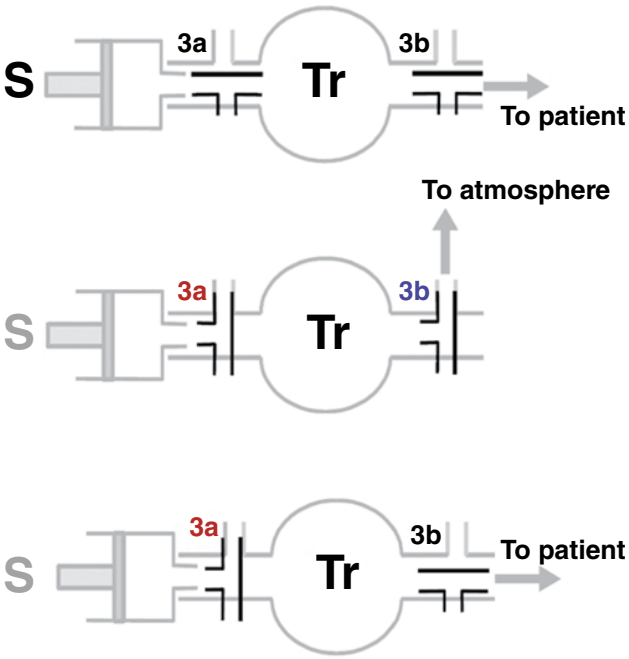

Figure 1.7 A schematic diagram of the set-up of three-way taps to enable key steps in urodynamics for pressure recording from the bladder (the same arrangements are also used for the abdominal pressure recording). The p_{ves} transducer (Tr) has a three-way tap '3a' connecting it to a syringe (S) and another to the patient '3b'. In the top diagram, both 3a and 3b are open, so this is the setting where the syringe can be used to flush air out of the transducer, connection tubing, and catheter. 3a is shut in the other two set-ups, so it is is closed to the transducer (indicated by red colour). In the middle set-up, 3b is open to the air (indicated by blue colour), so this the setting where the zero button can be pressed to set the reference value as atmospheric pressure. The bottom diagram is the set-up used when running the test, with 3b connecting the transducer to the bladder via the connection tubing and catheter.

sterile water. Again, the pressure reading on the equipment should change by the value of that depth.

Good urodynamics is carried out by making pressure measurements relative to atmospheric pressure. This is achieved in a water-filled system by zeroing the equipment with the transducers closed off to the patient and open to the atmosphere. Pressure measurements may also be made in urodynamics by using air-filled catheters. With these, there is a practically weightless connection between the patient and the external transducer. This means that the system is simpler to use compared to the external water-filled devices because there is no need to flush air from the system nor is there any need to place anything at a reference level. However, it is still important to set the baseline pressure of these devices to atmospheric pressure, and these catheters are not yet fully validated (see Chapter 7).

For uroflowmetry and for PFSs, urine flow rate in urodynamics is measured using a flowmeter which can either be mounted on a stand or in a commode. Urine is usually directed into the flow sensor by a funnel. The electronics of the flowmeter converts the changes of volume with time into urine flow rate (Q), which is measured in the units of millilitres per second (ml/s). Flowmeters (and other less common ones) will measure flow rate accurately, but it is important to examine the flow trace after it has been produced in order to correct for any artefacts that have occurred during voiding:

1) Knocking the flowmeter may produce 'spikes' on the trace which need to be ignored.
2) Moving the urinary stream relative to the flowmeter will produce artefactual fluctuations in the flow trace.

Setting Up the Equipment

For external, water-filled transducers, the disposables required are syringes, three-way taps, domes, manometer tubing/catheter to patient, and sterile water or physiological saline. The lines to the patient need to be primed with sterile water to remove air bubbles, and thus create a continuous column of water between patient and transducer. This can be done before the start of the test. The use of two three-way taps either side of the dome makes it easier for troubleshooting (checking zero and flushing) before and during the test (Figure 1.7), without introducing unnecessary air into the system. These allow completion of the important steps of basic urodynamics:

- Prime system: Flush sterile water through the whole system, with both three-way taps open before the domes are attached to the external transducers. A small flush after attachment is also advised.
- Zero to atmosphere: Position the taps so that the transducer is open to the atmosphere and closed to the patient and syringe. The 'zero' or 'balance' option on the urodynamic equipment is then selected. Pressures will now be read relative to atmospheric pressure.
- Set reference height: The pressure transducers need to be placed at the upper edge of the symphysis pubis to avoid artefactual pressure measurements due to the hydrostatic pressure effect. If the patient changes position during the test, the height of the transducers should be changed to the new level of the upper border of the symphysis pubis.
- For recording: The tap to the syringe remains off. The other tap is open to the transducer and the patient, but off to atmosphere. A cough test can now be performed. If the height of one cough peak is less than 70% of the other, the line with the lower value should be flushed with water and the cough test repeated.

If air-filled catheters are used, they need to be connected to their individual pressure transducer units. This can be done with the catheters already inside the patient. The switches on the transducer units are turned to the 'open' position, and the 'zero' or 'balance' option on the urodynamic equipment is then selected. The switches on the transducer units are then moved to the 'charge' position, and the catheters will record pressures inside the patient relative to atmospheric pressure. 'Zero' should not be done when patient pressures are being read, as these pressures are never truly zero.

Running the Test

Before their appointment, an information leaflet (an example is provided in the Appendix C.2) and suitable description should be provided so that the patient knows what to expect. A considerate attitude is needed, since the patient will generally be anxious.

The clinical requirement is to identify the 'urodynamic question', i.e. to clarify what symptoms experienced by this patient we are trying to observe during the urodynamic test. If we are able to reproduce the symptom during the test, the urodynamic features present at that time may provide the explanation for the symptom. Hence, urodynamics is preceded by a full clinical assessment:

1) History
   - Symptoms; best captured with a symptom score
     - Duration
     - Stress/urgency/other incontinence
   - Degree of leakage
     - Pad usage
   - Voiding difficulties
   - Quality of life
   - Past medical history
   - Medication e.g. anticholinergics
   - Allergies (latex)
   - Parity (where relevant!)
2) Frequency/volume chart (bladder diary)
   - Fluid intake – caffeine/alcohol
   - Voided volumes (VVs)
   - Voiding frequency
   - Nocturia?
3) Flow rate test
   - Maximum flow rate (Q_{max})
   - Shape of the flow trace
   - VV
   - PVR

This information is used to make the urodynamic test specific for the patient's clinical need. A decision is needed whether they actually need the test; sometimes, it is clear that no benefit will result for the patient, in which case it may not be appropriate to do the test. Informed consent to run the test is obtained, including a proper description of what the test involves and explanation of risks. The patient is allowed to get changed into a gown in privacy. The patient is asked to empty their bladder into the flowmeter (this is done regardless of how full the patient feels their bladder is; hence, it is not the quite same as a free flow rate test, since the latter should be done when the patient has a normal or strong desire to void [SDV]).

All staff present during the test must be introduced to the patient, and their role should be explained. Physical examination is undertaken. The catheters for recording p_{ves} and p_{abd} are then placed carefully and connected to the relevant transducers using connection tubing.

Before starting the test:

- Check the reference level (transducers level with the pubic symphysis) and zero (pressure lines have atmospheric pressure as zero)
- Check that the vesical and abdominal pressures are in the normal range
- Initial cough to test both lines are picking up rapid pressure changes promptly and equally

If any problems are identified, delay starting the test until the problem has been fully dealt with.

When running the test:

- Run the test in the position the patient usually adopts to pass urine (generally seated for women and standing for men)
- Use annotation marks to record aspects such as sensations reported by the patient, provocation tests, and urodynamic observations
- Monitor recording quality
  - Presence of physiological signals (e.g. if the patient moves, some change in the pressure lines is apparent)
  - Ask for coughs/deep exhalations regularly
- Understand other influences on the pressure recording
  - Drift of one of the baseline pressures may indicate a loose connection
  - Position changes will affect the resting pressure and require the urodynamicist to move the pressure transducers to lie level with the new height of the patient's pubis
  - Rectal contractions will cause a phasic rise in rectal pressure seen in the p_{abd} trace
  - Tube artefacts: knocks on the catheter or connection tubing will cause a clear disturbance in the pressure trace
  - Pump artefact is seen as disturbance in one of the pressure traces (usually p_{ves}) when the pump is running

- Tailor to the individual patient
  - Expected cystometric capacity can be estimated from the largest VV on the bladder diary but should be adjusted according to the patient's actual experience during the test.
  - Adjust filling speed if the patient develops urgency or DO is seen. Sometimes, it is necessary to change to filling in the supine position if overactivity is so prominent that it prevents reaching an adequate volume for reliable assessment of stress incontinence or voiding.
  - Do provocation tests to try to elicit issues, e.g. stress testing with a rapid sequence of coughs, or a Valsalva, to try to see USI. The sound of gently trickling water may help provoke DO.
- Once cystometric capacity is reached:
  - Do a cough to check pressure recording
  - Explain to the patient that urine will pass around the catheter and may land on the floor, but to void as naturally as can be managed in the circumstances
  - Give a clear 'permission to void' to initiate the PFS
  - Do another cough check after voiding has concluded

After the test:

- Remove the catheters and allow the patient to change back to normal clothing and wash their hands
- Explain the preliminary findings and what the next steps will be
- Describe what to look out for as the initial signs of a UTI and how to deal with it

After the patient has departed, clinical waste should be disposed of safely, and the equipment should be cleaned and set up for the next test.

Writing a report:

- Summarise the history, examination, and bladder diary findings
- Summarise the process followed (e.g. type of catheter, patient position, filling rate, and provocation tests) and report any problems encountered
- Describe the urodynamic observations and how they corresponded with sensations described by the patient. Clarify whether the patient's everyday symptoms were reproduced
- Describe any incontinence seen specifically
- Note whether the voiding in the PFS was typical, both from what the patient described and by comparing with the free flow rate test(s) done previously. Was there a PVR?
- Conclude with the summary of urodynamic observations and management suggestions.

Troubleshooting

Troubleshooting is a form of problem solving, defined by Wikipedia as 'the systematic search for the source of a problem so that it can be solved'. Troubleshooting is necessary if there are concerns about the quality of a urodynamic test while it is in progress. There is little that can be done to correct poor traces retrospectively, so quality control checks should be performed both before and during the investigation. Any problems with quality control should be addressed as soon as they are noted; the test can be paused while troubleshooting is performed.

The following information provides only a guide to common problems that are encountered during setting up and running a test, when quality control is not satisfactory. The unexpected can always happen, but problems can be solved if troubleshooting is performed in a systematic manner.

1) Baseline pressure readings outside acceptable ranges: Vesical and abdominal pressure measurements should both be within the range of 5–20 cmH_2O if measured with the patient supine, 15–40 cmH_2O if sitting, and 30–50 cmH_2O if standing. If pressures are outside the acceptable range:
   - If vesical and abdominal pressures are similar, but outside the acceptable range: check the height of the transducers – they should be level with the upper edge of the symphysis pubis
   - If only one pressure is outside the acceptable range:
     - Flush the relevant catheter
     - Check that zero has been set correctly on the relevant transducer
     - Consider re-siting the affected catheter
2) If there is unequal transmission of pressure between vesical and abdominal lines, before or during the test
   - Flush the line which is giving the smaller response
   - Check whether there is any air in the dome over the external transducer
   - Check the three-way taps are in the correct positions
   - Consider re-siting catheter
3) During the test, if there is a fall in pressure of the vesical or the abdominal line during filling
   - Flush the affected line – this may be enough to restore pressure
   - If pressures continue to fall, check for leaks in a systematic manner
     - Check taps and all connections have been adequately tightened
     - Check lines (connection tubes and catheters) – occasionally, there may be a manufacturing fault
4) If lines stop recording and the pressures drop dramatically

This is probably because one of the catheters has fallen out or become compressed
- Reposition or re-site the affected catheter
- If the vesical catheter has fallen out before Q_{max}, consider refilling and repeating the PFS.

5) Troubleshooting with air-filled catheters where a problem arises with quality control:
- Try 'opening' them and 'recharging' the catheters, ensuring that the patient coughs between charges
- While 'open', the zero level can be checked
- Try moving the catheter position, in case the balloon has become trapped or compressed
- If these fail, the catheter will need to be changed.

Fundamentals

Anyone working in urodynamics needs to remain vigilant and ensure high quality of practice. In this section, we describe some issues which are not covered specifically elsewhere in the book, but which nonetheless are important for all practitioners to bear in mind.

'Occult' Neurological Disease

A rare but vital situation a urodynamicist needs to look for in everyday practice is the possibility that someone has been referred for LUTS assessment where a neurological disease is the cause, but it has not yet been recognised by anyone. For some neurological conditions, LUTS can characteristically be an early feature in the disease [11]. LUTS may constitute the sole initial complaint in up to 15% of multiple sclerosis (MS) patients [18], so acute urinary retention of unknown aetiology or an acute onset of urgency and frequency in young adults should be carefully assessed. Every year we pick up some patients in this situation, and the urodynamic unit might be an excellent opportunity to identify these 'occult' cases. Note that the features are subtle (because if they were obvious, the diagnosis would have been made previously). Since urodynamicists may not be particularly confident in neurological examination, it means that they may have to act on suspicion or 'a hunch' that there may be something neurological going on. A vague suspicion and hard-to-explain severe LUTS is sufficient justification to make a neurology referral. No neurologist would object to a referral that turned out not to be neurological after all. On the contrary, failing to refer someone where neurology is later identified and an avoidable complication or progression occurs is a disaster for the affected person.

Occult neurology is especially relevant for people experiencing sudden onset rather severe LUTS, especially in younger age groups. Obviously, straightforward explanations like uncomplicated UTI must be excluded. Accordingly, urodynamicists need to be able to identify particular presentations where additional consideration to exclude an undiagnosed neurological disease is needed. Identifying a neurological mechanism where present is important for several reasons:

- To avoid adverse outcomes of urological intervention
- To minimise progression of the neurological condition by obtaining specialist input to disease management
- To enable patients to adapt their life according to prognosis.

The following conditions may present for LUTS assessment before a neurological condition has been recognised, because LUTS are potentially an early feature in the disease course:

1) MS, which is the most common progressive neurological disease affecting younger people, produces demyelination of the nerve fibres. Because any part of the neuro-axis may be affected, the exact pattern of LUTS and the associated non-urological features is potentially diverse. An important trigger to pick up is a history of transient blindness in one eye (optic neuritis). Suggestive features include weakness or sensory changes in the upper or lower limbs, loss of balance, coordination or falls, or visual symptoms (vision loss and double vision).
2) Parkinson's disease (PD) is a movement disorder with prominent non-motor symptoms, including LUTD. In its early stages, PD can cause storage and voiding LUTS, and motor symptoms may be mild. Important symptoms include a unilateral tremor; rather than the classic 'pill-rolling' tremor, early PD can have a unilateral tremor at a low frequency (approximately 2 Hz). Stiffness, bradykinesia, balance problems (presenting as falls), loss of smell, and urinary incontinence may be relevant.
3) Multiple system atrophy (MSA) is a PD-like problem causing chronic progressive neurodegeneration in the brainstem and cerebellum. This often affects people in their late 40s, with a male predominance. For men, erectile dysfunction (ED) [11] is commonly an earlier feature than LUTS; the reviewing doctor considering this possibility needs to enquire about ED, since men commonly do not volunteer the symptom without prompting. Archetypal symptoms include ED, slow movements, tremor, stiffness, reduced coordination, changes in voice, orthostatic hypotension, and urinary incontinence.
4) Normal pressure hydrocephalus (NPH) is a dilation of the cerebral ventricles, associated with stretching of

white matter tracts in the brain. The classic triad is abnormal gait, urinary incontinence, and dementia.

5) Alzheimer's disease and other forms of dementia are neurodegenerative conditions with wide-ranging effects on memory, cognition, and personality. Although LUTS generally increase in incidence with age, they are also more common in demented than non-demented patients. LUTS can be an early feature of dementia, in particular with Lewy body dementia. The archetypal symptoms are short-term memory loss; difficulty in performing familiar tasks; problems with language; disorientation in time and place, poor or decreased judgement; and changes in mood, behaviour, or personality.
6) Spinal cord conditions. This is a mixed group of potential causes due to direct or indirect effects on the spinal cord. There may be little in the way of indicative or localising symptoms. The archetypal condition is spina bifida occulta (SBO) and tethered cord, in which a developmental abnormality fixes the lower part of the spinal cord, placing it at risk by stretching and distortion as the person grows. Tumours (spinal or vertebral) can have this effect. Spinal stenosis (narrowing of the vertebral spinal canal), leading to claudication (leg pain with exercise) and LUTS, is another potential consideration.

The following symptoms should mandate a more careful neurological assessment in the urodynamic clinic: (i) enuresis, (ii) voiding dysregulation, e.g. voiding in socially inappropriate settings, (iii) involuntary voiding, and (iv) evidence of LUT muscle weakness, e.g. abdominal straining for voiding (bladder weakness), SUI (sphincter weakness), or retrograde ejaculation (bladder neck weakness), or (v) altered perineal sensation. Changes to one or more of the following non-urological features could suggest neurological impairment: gait, speech, cognition, memory, dexterity, vision, balance, or new headache. Some patients may volunteer new and/or sudden focal neurological changes. Furthermore, practitioners should stay alert, and if they feel there is something unusual about the current presentation, they should act on their intuition. On this basis, both urological and neurological examinations should be completed (see

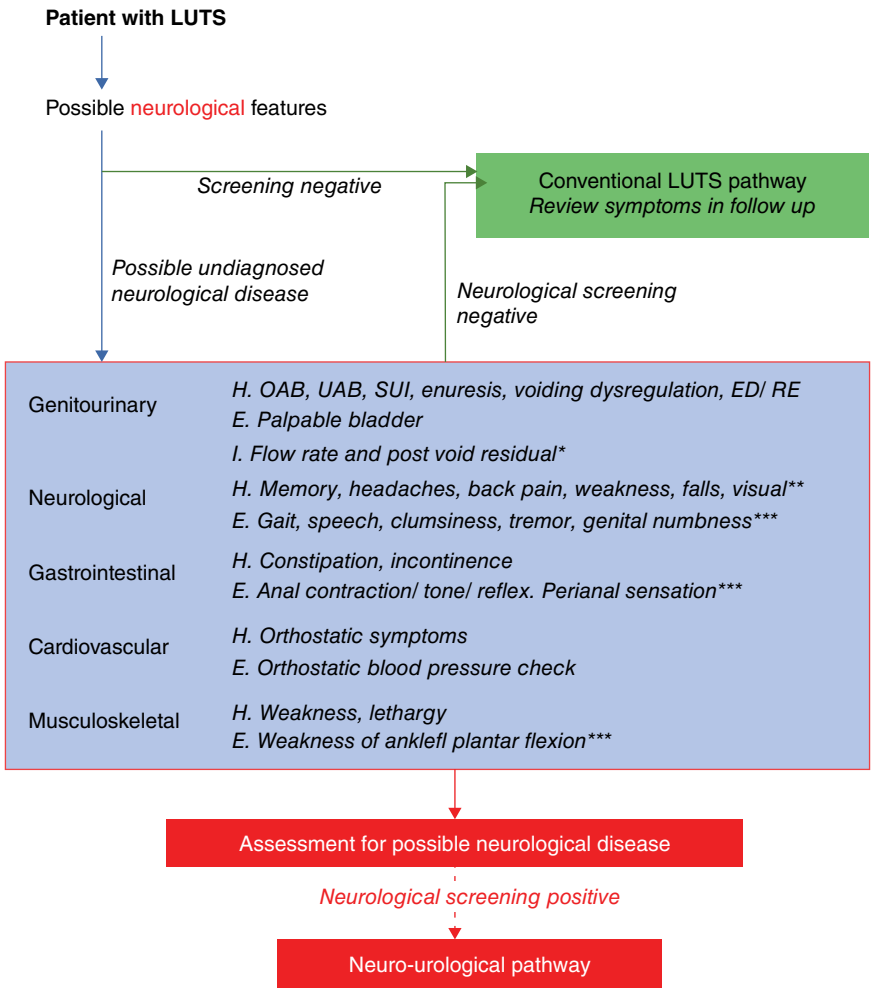

Figure 1.8 Additional evaluation to undertake in the event that a patient has presentation features that might suggest an undiagnosed underlying neurological condition. *Source*: Roy et al. [44].

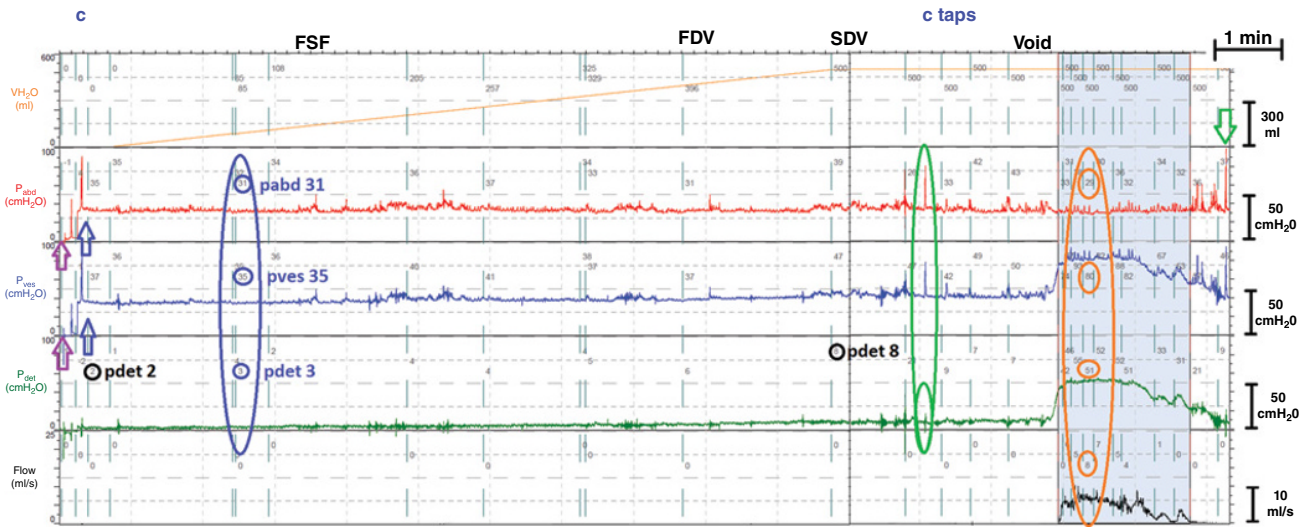

Figure 1.9 Features to look out for when looking at a complete trace done by someone else. The record shows continuous tracings recorded from a male patient aged 55 years, displayed in a manner consistent with International Continence Society recommendations. The abdominal pressure p_{abd} is shown in red and the vesical bladder pressure p_{ves} in blue. These are continuously subtracted ($p_{ves} - p_{abd}$) to give the detrusor p_{det}, in green. Also shown are the volume instilled in orange and flow rate in black. The sequence of the traces is appropriate, with p_{ves}, p_{det}, and flow in the lower half. Axes, units, and timescale are shown. Filling cystometry precedes permission to void (indicated with 'void'), and the pressure-flow study (PFS) follows it. The zero reference point is atmospheric pressure (purple arrows), so when the transducers are connected to the patient (blue arrows), there is an obvious rise in p_{abd} and p_{ves}, referred to as 'resting pressures' – the blue oval indicates the resting pressures for this patient at one timepoint. Coughs (indicated with 'c') are used to check that p_{abd} and p_{ves} detect a short spike of pressure (larger green oval) and that the p_{det} has a deflection which is equal above and below the line, the biphasic artefact (smaller green oval). It is important to check pressure recording with a cough at the start of filling and at other times if there is doubt about recording quality during filling. A cough is also needed before (green oval) and after (green arrow) the PFS to give confidence in the pressures recorded when voiding. Sensations are reported by the patient and annotated on the trace. First sensation of bladder filling (FSF) is the feeling the patient has, during filling cystometry, when he/she first becomes aware of the bladder filling. First desire to void (FDV) is the feeling that would lead the patient to pass urine at the next convenient moment, but voiding can be delayed if necessary. SDV is a persistent desire to void without the fear of leakage. A provocation was applied to try to elicit detrusor overactivity by making the sound of running water 'taps'; no change in p_{ves} or p_{det} was seen, so this patient had a stable detrusor. In the PFS, the key parameters derive from the time of maximum flow rate (Q_{max}), highlighted in orange (calculations derived from this type of data are discussed in Chapter 14). *Source*: Drake [15].

Chapter 16). Early referral to neurology is important, even if no concrete finding is made. Most neurologists do *not* recommend that urologists request imaging without prior discussion with a neurologist. This is due to the additional delay and to ensure that the correct investigation(s) are ordered. A summary algorithm produced by the ICS is shown in Figure 1.8. An example case is described in Chapter 16 (Figure 16.26).

How a Urodynamic Trace Should Be Presented

The ICS has standardised the process of urodynamics [19, 20], and throughout this book, we will repeatedly refer to the crucial importance of these documents and other ICS Standardisations. One of the key requirements is to ensure that traces are displayed in a way that they can be interpreted by someone who was not at the test, without the need for explanation by anyone else. This is equivalent to the presentation of an electrocardiogram (ECG), since any cardiologist or appropriately trained healthcare professional would expect a standardised presentation, enabling them to interpret the traces.

In this book, we try to comply with ICS recommendations throughout. In general, we display traces in a manner consistent with Figure 1.9, though older example traces may not fully comply. Some of the most useful things to check include:

- *What is displayed?* Typically, this will be two recorded pressures, p_{abd} and p_{ves}, a calculated p_{det}, flow and volume instilled. The p_{ves} and p_{det} traces should be plotted below the others (since they are most important, and high pressure might disappear off the top of the page).
- *Are the axes labelled?* Are the units stated and the volume range clear? We try to colour code our traces, so red (rectal) lines correspond to abdominal pressure and blue (bladder) lines correspond to vesical pressure.
- *Was the test set-up with zero set at atmospheric pressure?* This can be deduced if the pressures in p_{ves} and p_{abd} are zero and flat if opened to atmosphere (because atmospheric

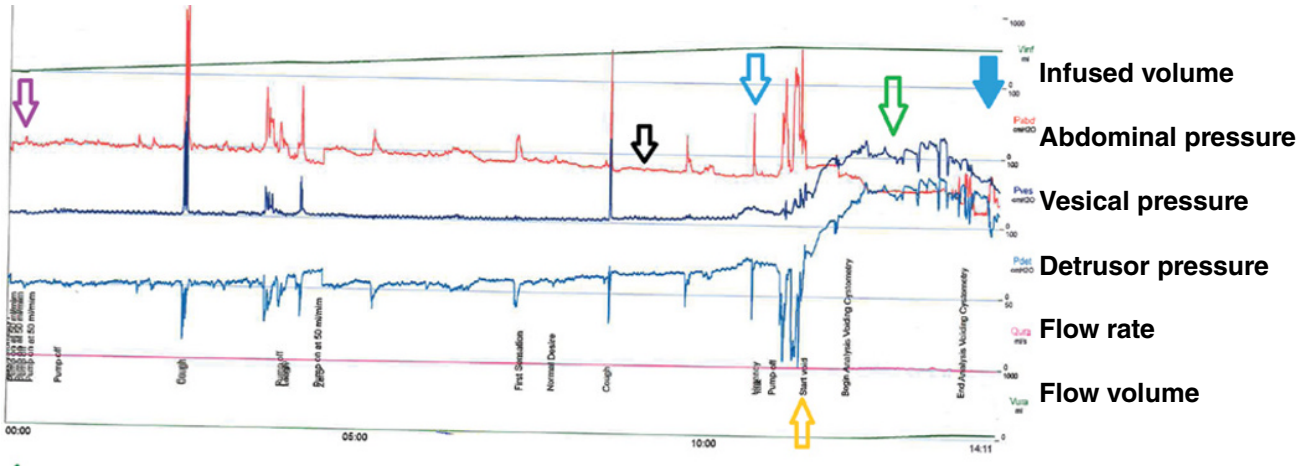

Figure 1.10 Examples of some of the quality control issues that need to be identified during urodynamics. Purple arrow: pressures set to zero while recording from patient, not to atmosphere. Black arrow: downward pressure drift in abdominal pressure. Blue arrows, open arrow: cough before void which is only picked up in the abdominal pressure; closed arrow: lack of cough test after conclusion of the void. Yellow arrow: start of void. Green arrow: drop in abdominal pressure during void. Vesical pressure shows a lack of fine detail and poor cough spikes, without a flush through needed to attempt remedial action. *Source*: Aiello et al. [21].

pressure fluctuates very little). If recording was not started before zeroing, the pressures in p_{ves} and p_{abd} should begin within the normal resting pressure range.

- Once recording from the patient starts, is there movement in the lines as expected, due to the patient breathing, speaking, and moving?
- *Are there annotations?* These are crucial, indicating when events such as provocation tests, sensations, and instructions happened. Without these, the test is uninterpretable. For example, if 'permission to void' is not annotated, a detrusor pressure change accompanied by flow could be either DO incontinence or voiding.
- *Are there checks of recording reliability?* Coughs done during the course of filling, and before and after voiding, help confirm that the recorded pressures can be trusted; the spike associated with a cough should be similar in amplitude in both the p_{ves} and p_{abd} lines, and measures should be taken to resolve them if not (usually a flush through of the transducer and catheter with the smaller spike).
- *Are there provocation tests aimed at reproducing symptoms?* For example, a sequence of high amplitude coughs in quick succession is a means of eliciting USI.

If part of a trace is selectively shown to illustrate a point in this book, we describe the circumstances so that the reader can appreciate what has occurred.

Adhering to High Standards

Disappointingly, the urodynamic community often fails to meet the expectations of the ICS standards [21]. The issues are discussed extensively throughout this book. As described in the preceding section, it is important to ensure that traces are presented in such a way that they can be verified by someone not present at the test. Any major errors must be identified (Figure 1.10). Failure to do this in some centres has led to a deeply unsatisfactory situation that many surgeons simply accept the urodynamic report at face value. This ultimately means that the urodynamic technician can have considerable influence on treatment choice, without necessarily realising it. One particularly problematic issue is failure to identify for an artefact affecting the maximum flow rate (Q_{max}) value, and using this uncorrected value to calculate important information like the BOO index, thus coming up with a wrong value. The urodynamicist *must* look at the trace, and if an artefact is present, the cursor must be moved so that the values (Q_{max}, and hence $p_{detQmax}$) are taken from a spot not affected by spurious influences (see Chapter 6). This is often not done in many established units [21], and the result potentially can mean a wrong diagnosis and consequently a risk of inappropriate surgery.

Many units actually have a very poor understanding of urodynamic techniques, quality control, and interpretation [21]. One extraordinary weakness is a substantial lack of understanding of DUA; the majority of sites get this wrong or fail to comment on it at all (Table 1.2). This gives a risk of misdiagnosis and consequently the potential that some people could undergo surgery inappropriately.

The responsibility placed on the urodynamicist to achieve a meaningful test has a couple of implications for how to practice:

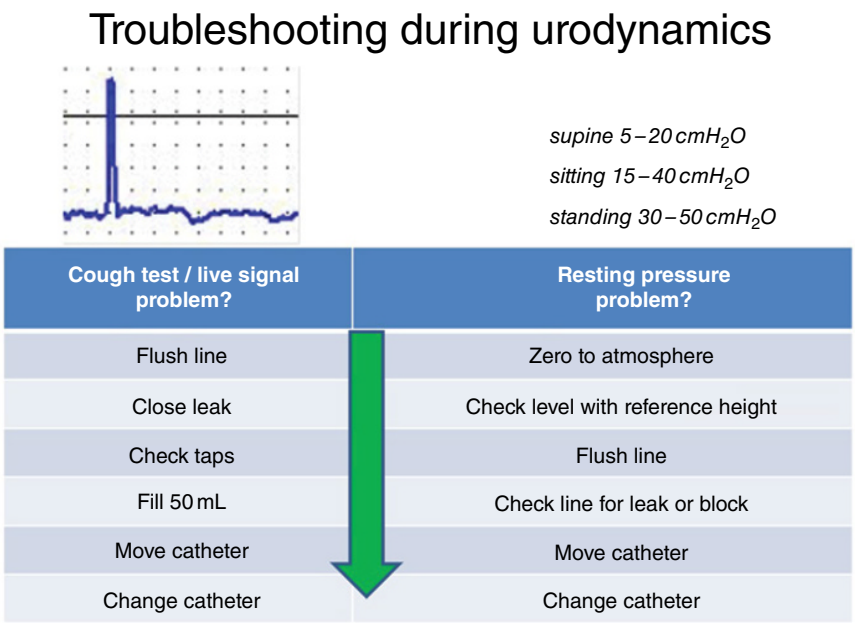

Figure 1.11 Two key parameters are scrutinised routinely throughout the running of a urodynamic study. The first is the resting pressures, which are affected by the position of the patient (supine, seated, or standing). Second, the pressure spikes generated by a cough, which should be of similar amplitude in the vesical and abdominal lines. Spotting when things are not recording properly is crucial, and a sequence of steps taken to deal with issues in either parameter is illustrated. More details are given in Chapter 19.

1) During the test, the traces must constantly be reviewed so that any problems can be identified and dealt with on the spot. We refer to this as 'troubleshooting'. It is described in detail in Chapter 19, and a brief summary is given in Figure 1.11.
2) If, despite best efforts, the test does not proceed smoothly, or the patient's symptoms were not reproduced during the test, then that must be stated clearly in the report. This can happen in any unit, and willingness to concede a test was unsuccessful is a vital protection for the patient.

It is always necessary to stay alert. First, even experienced urodynamics practitioners can make unexpected mistakes from a lapse in concentration, as exemplified in Figure 1.12. Second, unexpected challenges can crop

Table 1.2 Prevalence rates of the urodynamic observation of DO and pressure-flow diagnosis of BOO and DU, comparing categorisations by hospitals ('sites') with central expert review.

Categorisation by central review		Categorisation by sites				
Observation/diagnosis	**Prevalence**	**Correct (*n*)**	**Correct (%)**	**Incorrect (*n*)**	**Uncategorised (*n*)**	**Non-correct (%)**
DO present	46/99 (46.4%)	26/46	57	1/46	19/46	43
DO absent	53/99 (53.6%)	27/53	51	2/53	24/53	49
BOO present (BOOI >40)	55/107 (51.4%)	39/55	71	1/55	15/55	29
BOO equivocal (BOOI 20–40)	14/107 (13.1%)	11/14	79	3/14[a]	0/14	21
Unobstructed (BOOI <40)	19/107 (15.9%)	8/19	42	6/19	5/19	58
Unable to derive BOOI	19/107 (15.9%)	N/A	N/A	15/19[b]	4/19	1
DU present (BCI <100)	27/107 (25.2%)	14/27	52	2/27	11/27	48
DU absent (BCI >100)	59/107 (55.1%)	18/59	31	1/59	40/59	69
Unable to derive BCI	21/107 (19.6%)	N/A	N/A	4/19[c]	15/19	1

Abbreviations: BCI: Bladder Contractility Index; BOO: bladder outlet obstruction; BOOI: BOO Index; DO: detrusor overactivity; DU: detrusor underactivity; N/A: not applicable.

Note: 'Uncategorised' indicates that the site provided the trace but did not make a comment on diagnosis.

The 'non-correct' column indicates what proportion of diagnoses the hospital got wrong ('incorrect') or failed to comment on ('uncategorised').

[a] Categorised as obstructed in two, unobstructed in one.

[b] Categorised as obstructed in 10.

[c] Categorised as DU in two. *Source*: Reproduced with permission from [21].

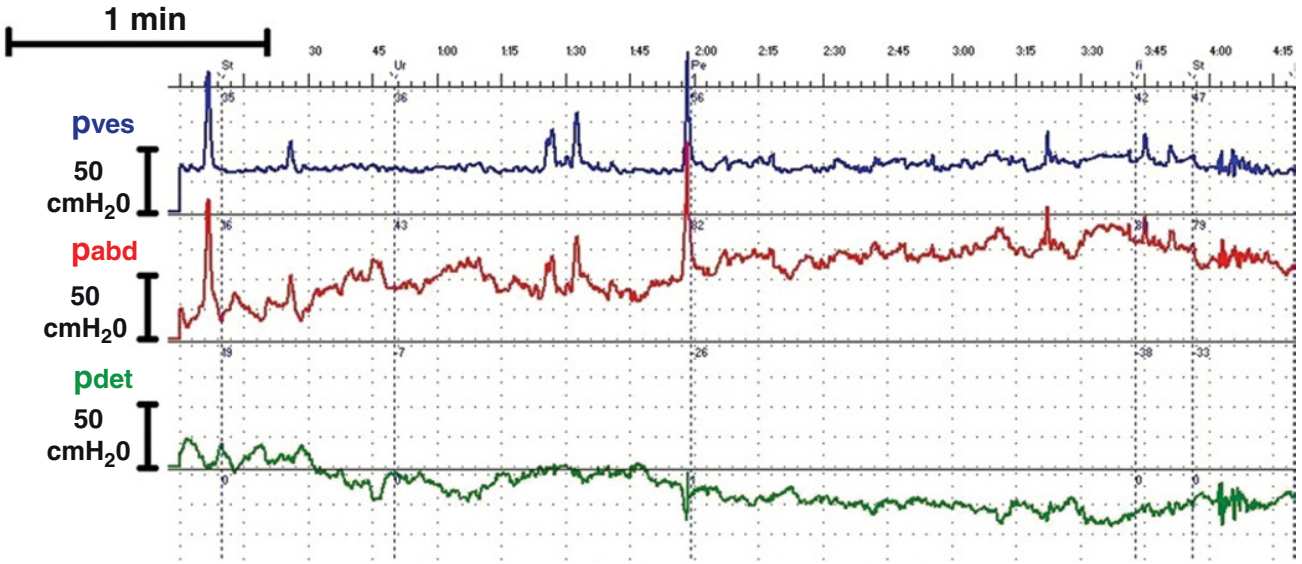

Figure 1.12 An unusual error (fortunately) is to connect the filling pump to the rectal catheter. When the healthcare practitioner asked the patient if she felt her bladder filling, the reply was 'no, but I really want to open my bowels'. The steady climbing p_{abd} is clear, and it is responsible for the falling p_{det} over the time illustrated. This is an old trace; modern traces do not have p_{ves} placed at the top.

up, for which adaptations will need to be made (Figure 1.13).

Safety of the Urodynamics Staff

The Covid-19 pandemic of 2020 highlights a potential for communication of respiratory-transmitted illness from patient to practitioner, or vice versa. Alterations across the entire healthcare system became necessary. Urodynamics is affected, since physical proximity and use of coughs to check pressure recording or provoke symptoms could increase the risk of respiratory pathogen transmission. We have therefore adapted practice to mitigate some of these concerns (Figure 1.14) [22]. Single-use surgical face masks and eye protection are recommended for both patients and staff, in addition to the normal use of single-use gloves and aprons by the urodynamicist. There is no need for patients to wear gloves, but patients will either use hand-gel or wash their hands for 20 seconds before entering and leaving the urodynamics room. Local and national guidelines should be followed with regard to personal protective equipment (PPE). Wherever possible, a distance of 2 m should be maintained between staff and patient. Clearly, for procedures such as catheterisation and examination of the patient, this is impossible. Precautions must, therefore, be taken in the form of PPE as above and adjusting elements of the test to allow observation from a distance of at least 2 m. Where urinary leakage needs to be observed, especially in women, the patient could be asked to stand or squat over a pad on the floor, rather than sit on the flowmeter, in order that leakage can be seen from further away. During video-urodynamics, fluoroscopic screening can provide evidence of urethral leakage and will be sufficient for a diagnosis of USI. Coughing should be kept to an absolute minimum and always with a mask in place. Quality control can be carried out effectively by a Valsalva manoeuvre or even by gentle external pressure on the abdomen by the patient. Valsalva manoeuvre or other physical provocations can be attempted first, and only after that, all other provocations having failed, would the patient be asked to cough,. In that case, the cough must be directed away from others in the room and shielded by an elbow or by a hand-held tissue that is then discarded, since the mask itself must not be touched during use. A period for cleaning the room is needed between each patient.

A Brief History of Urodynamics

Interest in the hydrodynamics of micturition started with the early cystometric studies of the nineteenth century, but it was the advent of electronics that acted as the catalyst for modern urodynamic studies. In 1956, von Garrelts described a simple practical apparatus, using a pressure transducer, to record the volume of urine voided as a function of time. By differentiation, urine flow rates could be calculated. His work stimulated a revival of interest in cystometry because it was then possible to record the bladder pressure and the urine flow rate simultaneously during voiding. As a result, normal and obstructed micturition could be defined in terms of these measurements [23], and a formula was applied to express urethral resistance [24].

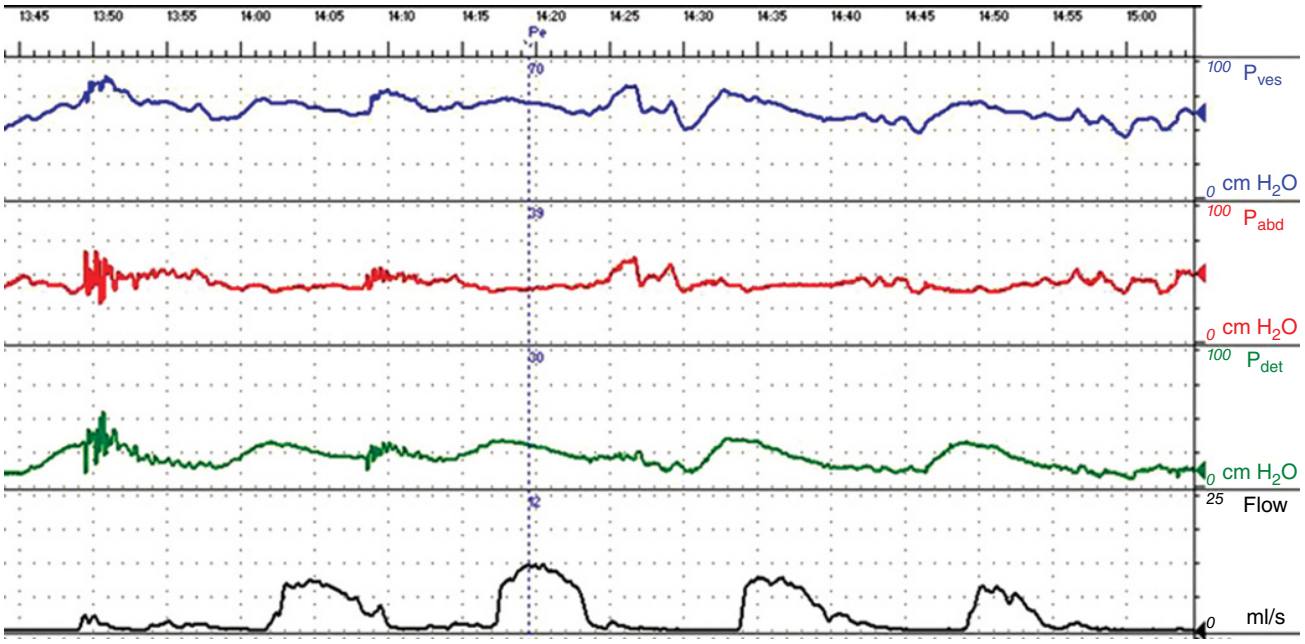

Figure 1.13 This pressure-flow study of a male patient shows a grossly abnormal interrupted flow pattern, yet his pre-urodynamics flow test showed a normal flow pattern and good flow rate. The problem was a consequence of his p_{ves} catheter; as he passed urine, some of it tracked along the catheter so that it was carried beyond the rim of the funnel. When it fell off the catheter, his foot happened to be underneath, which was very distracting for him, causing him to inhibit his voiding contraction. Each time he restarted voiding, the same happened, leading to repeated interruptions. Since this event, we have been careful with routing the p_{ves} catheter. (This is another old trace with p_{ves} placed at the top.)

Enhorning [25] measured bladder and urethral pressures simultaneously with a specially designed catheter, and he termed the pressure difference between them the 'urethral closure pressure'. He demonstrated that a reduction of intraurethral pressure occurred several seconds prior to detrusor contraction at the initiation of voiding. This appeared to relate to the relaxation of the pelvic floor, consistent with the electromyogram (EMG) studies of Franksson and Peterson [26].

Application of urodynamic investigations in the clinical field soon followed. Radiological studies of the lower urinary tract, using the image intensifier and cine or videotape recordings, were already established, and their value in the assessment of micturition disorders had been described [27]. It was a relatively simple step to combine cystourethrography with pressure-flow measurements [2]. Sophisticated techniques followed, for example, using EMG recordings of the pelvic floor, particularly for neurogenic bladder problems [28]. These clinical studies during the 1970s emphasised the need to investigate the function as well as the anatomical structure of the lower urinary tract, when evaluating micturition disorders. Urodynamics was becoming established as a necessary service rather than a research tool.

As these technical developments progressed, there was an increasing awareness of the clinical problem of urinary incontinence. Caldwell [29], working in Exeter in the UK, initiated considerable interest in the subject because he approached the treatment of incontinent patients with electronic implants. In his sphincter research unit, a small receiver was developed that could be placed subcutaneously in the abdominal wall and activated by a small external radio-frequency transmitter. Platinum iridium electrodes led down to the pelvic floor muscles, which could be stimulated. Other new techniques advocated at this time included pelvic floor stimulation, including use of a variety of external electronic devices which could be placed in the anal canal or vagina to stimulate pelvic floor contraction [30, 31].

Both technique and terminology should be standardised, to allow for interpretation of findings and so that others may understand and interpret the results from any urodynamic unit. To facilitate this, the ICS in 1973 set up a Standardisation Committee, which has produced reports on the terminology of lower urinary tract function. The first six reports were collated in 1988 and comprehensively rewritten. Undoubtedly, two key documents published in 2002 which served as a platform in the development of

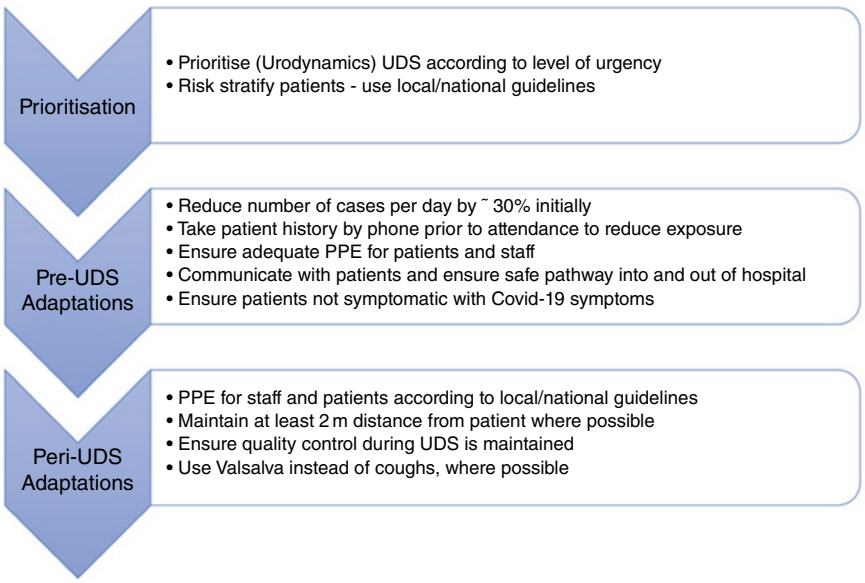

Figure 1.14 Adaptations to urodynamic practice in response to the Coronavirus pandemic. PPE: Personal Protective Equipment. UDS: Urodynamics. *Source*: Hashim et al. [22].

professional terminology and practice were the Standardisation of Lower Urinary Tract Function [32] and Good Urodynamic Practices [19]. The latter document has been extended and updated [20]. In addition, the International Children's Continence Society (ICCS) has published standards for the paediatric population [33–35].

In recent years, the importance of modern-day governance led to the ICS placing the standardisation process under a Steering Committee, which oversees independent working groups and follows clear governance procedures and an evidence-based approach [36]. Subsequently, the Standardisation documents now undergo a process of iterative and ongoing review and development, including documents for chronic pelvic pain [37], neurogenic LUTD [38], female LUTS [39], pelvic organ prolapse [40, 41], nocturia [42], and urodynamic equipment [43].

These ICS and ICCS Standards aim to facilitate comparison of results by investigators who use urodynamic methods. Written publications are expected to acknowledge the use of these standards by stating: 'Methods, definitions and units conform to the standards proposed by the International Continence Society except where specifically noted'. This book applies the ICS Standardisations throughout.

Summary

In the basic assessment, the following are important:

- Establish a rapport with the patient
- Look at the symptom score
- Review previous treatments
- Consider bowel, gynaecological, and sexual function
- Identify medical problems and medications
- Consider possibility of neurological disease or malignancy
- General examination
- Examination features suggestive of wider problems, e.g. occult neurology or malignancy
- Focussed lower urinary tract examination, abdominal and internal

This comprehensive evaluation allows the investigator to formulate their urodynamic questions:

1) 'What do I want to know about this patient?'
   a) 'What is wrong with storage, what is wrong with voiding?'
   b) 'What is wrong with the bladder, what is wrong with the bladder outlet?'
2) 'Which urodynamic investigations need to be performed to define this patient's problems?'
3) 'Is the investigation likely to be of benefit to the patient?'
4) 'Is urodynamics able to make a reliable diagnosis?'

Having run the test, the report should be written in detail, mentioning full aspects of the technical procedure, but also answering the following:

- Did the urodynamic studies reproduce the patient's complaints, and did the complaints correlate with known urodynamic features?
- Does the report make sense in the context of the patient's symptoms and preceding tests?

- Can urodynamics decide which abnormality is the most significant if more than one is detected?
- Can the features mentioned in the report be identified on the plotted traces, and is anything visible on the traces not mentioned in the report?

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2

Applied Anatomy and Physiology

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CONTENTS

Introduction

Urodynamic investigations developed because of dissatisfaction with the assessment of patients and treatment results when management was based on examination of anatomical abnormalities. Urodynamics attempts to relate physiology to anatomy so that both function and structure are considered alongside each other. A sound knowledge of anatomy and physiology form the basis for the effective assessment and treatment of patients. In addition, this knowledge can be used to evaluate the role of urodynamic studies critically in assessing patients with lower urinary tract symptoms (LUTS). The lower urinary tract comprises the urinary structures of the bladder and bladder outlet (Figure 2.1).

In men, the close relationship to genital structures also makes the phrase 'genitourinary tract' appropriate (Figure 2.2). Although the bladder and urethra are described separately below, it should be remembered that they normally act as a functional unit. During urine storage, the bladder is quiescent, and the urethra contracted, and vice versa when voiding. This co-operative function is imposed by the central nervous system (CNS) control, ensuring synergy of the lower urinary tract.

Upper Urinary Tract

The upper urinary tract comprises the kidneys and ureters (Figure 2.3). Kidneys are excretory organs which have a vital role in homeostasis. They ensure appropriate salt and water levels, control acid–base balance, and eliminate water-soluble toxins. This is achieved by varying the composition and volume of urine to reflect overall body requirements when healthy. Water elimination is known as diuresis, and there is typically a production of urine at a rate of at least 0.5 ml/kg body weight every hour. 'Forced diuresis', i.e. absolute maximum rate of urine production to get rid of a large overload of water, is about 900 ml/hour [1]. Elimination of excess salt is known as natriuresis, and this will also increase urine production rate because the salt requires water to dissolve it. The control of how fast urine

Abrams' Urodynamics, Fourth Edition. Edited by Marcus Drake, Hashim Hashim, and Andrew Gammie.

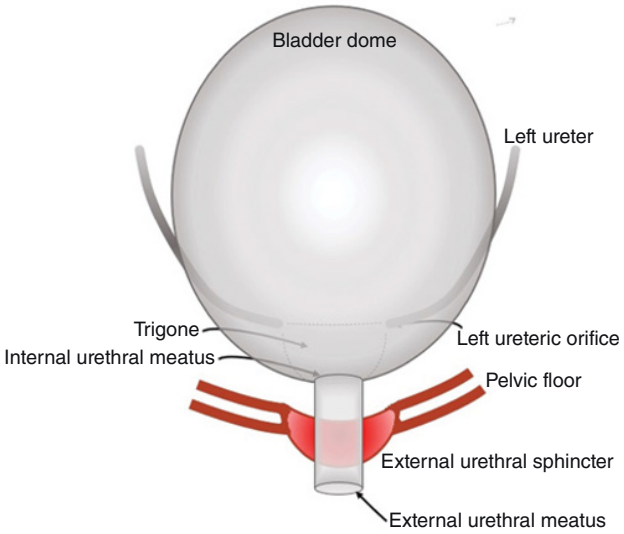

Figure 2.1 The lower urinary tract joins the upper urinary tract at the ureteric orifices. Its main structures are the bladder and the bladder outlet. The trigone sits in the bladder base, demarcated by the ureteric orifices and the internal urethral meatus. The bladder outlet is the urethra, which runs from the internal to the external urethral meatus. The external urethral sphincter in women is a horseshoe-shaped structure, mainly sitting dorsally.

is produced is determined by key hormones, notably antidiuretic hormone (ADH), which is responsible for retaining water if a person is dehydrated. ADH levels are reduced where someone has surplus water to eliminate. Atrial natriuretic peptide (ANP) and brain natriuretic peptide (BNP) are present when there is excess salt to dispose of, a process that increases rate of urine production like diuresis.

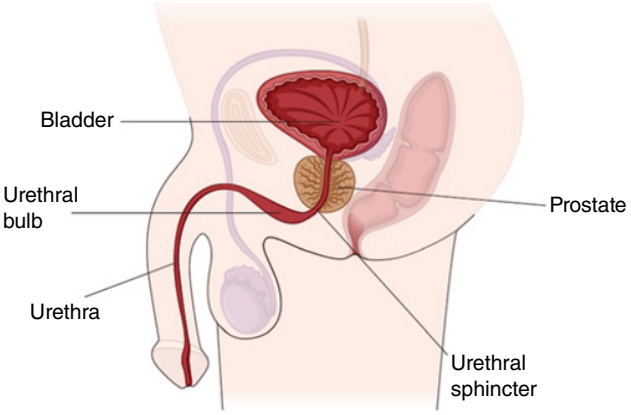

Figure 2.2 The male genitourinary tract. Key differences from the lower urinary tract in women include the entry points of the ejaculatory ducts (see Figure 2.12), the presence of the prostate between the internal meatus and the external urethral sphincter, the circular structure of the sphincter, and the substantially longer urethra.

Where there is kidney disease, rate of production of urine may be altered, depending on which part of the kidney is affected. The glomeruli are the structures in the kidney responsible for filtering the blood, so if they are diseased (reduced glomerular filtration rate), water will be retained (leading to body swelling and increased weight), toxins will accumulate (increased serum creatinine and urea levels), and rate of urine production will be reduced. The kidney tubules reabsorb water and nutrients from filtered urine. In diseases selectively affecting the kidney tubules, failure of reabsorption leads to water loss and hence increased urine production. This can result in dehydration (so the person may compensate by increasing their fluid intake), but serum creatinine is normal (or low). Chronic kidney disease (CKD) generally affects both glomeruli and tubules, so it will have features of both problems. Patients with CKD or tubular disease will have a high rate of urine production (and compensate for this by increasing fluid intake). This can also happen if the tubules become insensitive to ADH (nephrogenic diabetes insipidus), which can be a feature of treatment of psychiatric conditions with lithium. Central diabetes insipidus is another cause of water loss, in this case, due to impaired production of ADH.

Natriuresis can be severe when asleep in patients with obstructive sleep apnoea (OSA), due to high production of ANP; this is a major cause of nocturia. If someone is losing water or salt and presents for treatment of increased voiding frequency, it is not appropriate to advise fluid restriction, since they could become dangerously dehydrated. Drinking due to a constant feeling of thirst is the key point to ascertain in the history. Other notable medical factors include fluid and salt retention, leading to ankle swelling, as is seen in heart failure in particular, as this retained fluid may be excreted when the patient lies down.

Osmotic diuresis is a process of water loss as a result of high amounts of solutes in the urine. An important example is poorly controlled diabetes mellitus, in which very high sugar levels spill over into the urine and, consequently, increase the volume passed. This is rarely a cause of nocturia, but it could happen if someone is on a treatment schedule which is insufficient to control their overnight sugar levels. A spot test of daytime sugar levels would not necessarily identify that, but a glycosylated haemoglobin blood test would probably be abnormal.

These influences on urine production are fundamental factors determining voiding frequency, along with increased awareness (see below) and reduced bladder storage capacity (discussed throughout this book). A schematic summary is given in Figure 2.4.

Most people have two kidneys (1% are born with one kidney), which drain into the urinary bladder along the ureters

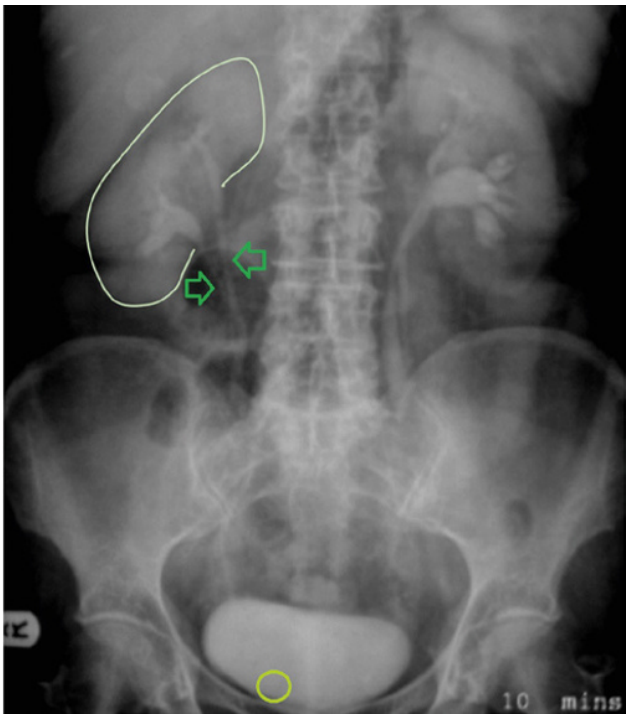

Figure 2.3 The upper urinary tract comprises the kidneys and ureters. In this intravenous urogram (IVU), the right kidney is outlined in white. On both sides, the ureter is partially duplicated, and the two duplex parts for the right ureter are indicated by green arrows. The majority of people have one ureter on each side, but duplication can happen to one or both sides and ranges from partial to complete. The lower limit of the upper urinary tract is the vesicoureteric junction (VUJ). In this IVU, the VUJs cannot be seen as they are hidden by contrast in the bladder, but the approximate location of the right VUJ is indicated by the green circle. This film was taken 10 minutes after contrast injection and shows just how quickly the bladder can fill in a well-hydrated patient receiving a diuretic injection prior to administration of the contrast. *Source*: Marcus Drake.

(usually one on each side). The ureters enter the wall of the urinary bladder obliquely for about 2 cm and open by slit-like apertures at the lateral angles of the trigone. This oblique course through the bladder musculature helps the terminal ureters to occlude, thus acting like a one-way valve, the vesicoureteric junction (VUJ). The VUJ allows urine to enter the bladder and prevents backwards return of urine (vesicoureteric reflux, or VUR). VUR can be a result of various pathologies, notably embryological problems, surgical damage, and high bladder pressures. When VUR is severe, it prevents efficient drainage of the kidney, which can lead to CKD.

Alterations in Fluid Excretion

Commonly, 24-hour urine output lies between 1 and 3 l, and 1.5 l (i.e. 1500 ml) is a perfectly healthy value for most people. This equates to an average output of about 1 ml per minute (since there are 1440 minutes per 24 hours). The normal daily fluid output from the kidneys reflects homeostatic processes, comprising diuresis (disposing of excess water), natriuresis (disposing of excess salt), and excretion (getting rid of toxic metabolic products) to achieve overall balance. These basic processes are hugely influenced by several further factors:

1) The dietary sources of water are not only in the drinks but also in the water content of food consumed. The need for consuming drinks is determined by osmoreceptors, which trigger a sensation of thirst where additional water is needed. On top of that, people often drink more than their thirst-guided requirements, notably with cups of tea or coffee, fizzy drinks, or alcohol. Alterations in the quantity of fluid imbibed may occur at times of stress and during periods of social change, for example, at times of redundancy or retirement. Many people follow spurious trends like 'drinking more is healthier', but some drink more specifically because of advice to do so (notably those with a history of kidney stones or urinary tract infections [UTIs]).
2) Allowance is also needed for the loss of water elsewhere from the body, notably sweating, in the faeces, and water vapour in breath.
3) Overnight, there is normally a reduction in the rate of urine output due to circadian hormonal control (increased ADH secretion overnight). Approximately 80% of the 24 hour volume is excreted during the waking hours, and this nocturnal reduction therefore means it is not necessary to empty the bladder at night in the normal condition. Abnormalities of the normal circadian rhythm may be induced primarily by relevant diseases, such as renal failure or heart failure, or be secondary to drugs used in the treatment of such conditions, for example, diuretic therapy.
4) There is a lot of capacity in the human body to store water and salt, and the processes affecting their balance may be slow and unpredictable. Consequently, it is rather common to find the bladder diary suggests the urine output exceeds the intake for a certain period. Provided the patient looks appropriately hydrated, it is generally safe to assume that this would balance out if everything was accurately measured for sufficient duration.
5) Any disease of the kidney will influence urine output. Renal tubule dysfunction may hinder the reabsorption of filtered urine and hence increase urine output. If this occurs alongside renal glomerular dysfunction, then the renal disease will show up in standard renal blood tests. However, some problems affecting the tubules are not associated with glomerular disease, notably diabetes insipidus. Here, the renal fluid handling may be abnormal, yet with normal renal function blood tests.

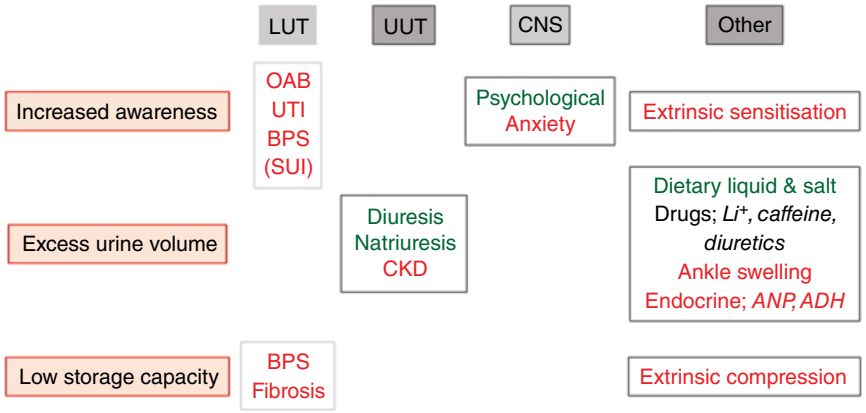

Figure 2.4 Influences on voiding frequency; how often someone passes urine is determined by amount of urine, storage capacity, and awareness, which are affected by normal functions (green text) and medical causes (red text), in a wide range of organ systems. Overactive bladder (OAB) and urinary tract infection (UTI) increase urinary tract sensation via bladder afferents (A-delta) and stress urinary incontinence (SUI) via urethral afferents. The bladder afferents can be sensitised by extrinsic inflammation, such as diverticular disease or gynaecological inflammation. Bladder pain syndrome (BPS) can do so via bladder afferents (gamma) and by reducing storage capacity. Chronic kidney disease (CKD) affects the ability of renal tubules to reabsorb filtered urine. Several hormones are relevant, notably atrial natriuretic peptide (ANP) which triggers salt loss (natriuresis) and anti-diuretic hormone (ADH) which controls water loss. Li^+: Lithium. Extrinsic compression relates to the space-occupying effect of nearby organs; in the constrained space of the pelvis, this means less room for the bladder (see Figure 2.5). CNS: central nervous system; UUT: upper urinary tract.

6) Many medical conditions away from the urinary tract affect urine output due to effects on water or salt balance or endocrine function. Nocturnal polyuria in elderly men might reflect subclinical cardiac failure, leading to increased ANP released at night. ANP is also released in OSA, again causing severe nocturnal polyuria, and reversed by treating the patient's OSA with a continuous positive airway pressure (CPAP) machine.
7) Medications can be relevant, notably anything with a diuretic effect, e.g. furosemide or bumetanide. Lithium can cause nephrogenic diabetes insipidus.
8) The bladder may be a 'mirror of the mind', and psychological influences occasionally manifest themselves initially as urological symptoms. Such voiding patterns are often 'diagnoses of exclusion' following persistently negative urological studies. The frequency/volume chart (FVC) may show a high voiding frequency and sometimes nocturia, occurring at times of social and mental stress. Sometimes, nocturia is absent, despite high frequency during the day. Completing the bladder diary may lead the patient to recognise such influences.

The Urinary Bladder

The urinary bladder is a musculomembranous organ located in the bony pelvis (Figure 2.5) and acts as a reservoir of urine. The fundus of the empty bladder, which is entirely located in the front part of the pelvis, is separated from the rectum (posteriorly in the pelvis) by the rectovesical fascia in men or the vagina, cervix, and uterus in women. The superior surface of the bladder is covered with peritoneum. The inferior surface is in contact with the fascia covering the levator ani and obturator internus muscles. When distended, the upper part of the bladder rises up above the pubis (Figures 2.5 and 2.6) and lies between the peritoneum and the back wall of the rectus abdominis muscles (meaning that a suprapubic catheter can enter into the bladder without traversing the peritoneum). The bladder in the newborn infant is largely abdominal, and it gradually moves to the adult pelvic position by about nine years.

Within the bladder, there is an area of the base referred to as the trigone (Figure 2.1), which is the space between the two ureteric orifices and the internal urethral meatus. Here, the trigonal epithelium is firmly attached to the muscular layer. Two bands of oblique muscle fibres originating from the two VUJs, converging to the back of the prostate, are thought to help retain the oblique direction of the ureters with voiding, thus preventing VUR.

Elsewhere, in the greater part of the bladder, the urothelium is rather loosely attached with a submucosal layer comprising loose areolar tissue. This can make the urothelium sufficiently mobile that it can drift into the eyehole of a catheter and block it, interfering with drainage (urinary catheters) and pressure recording (urodynamic catheters). The urothelium is a non-glandular transitional epithelium,

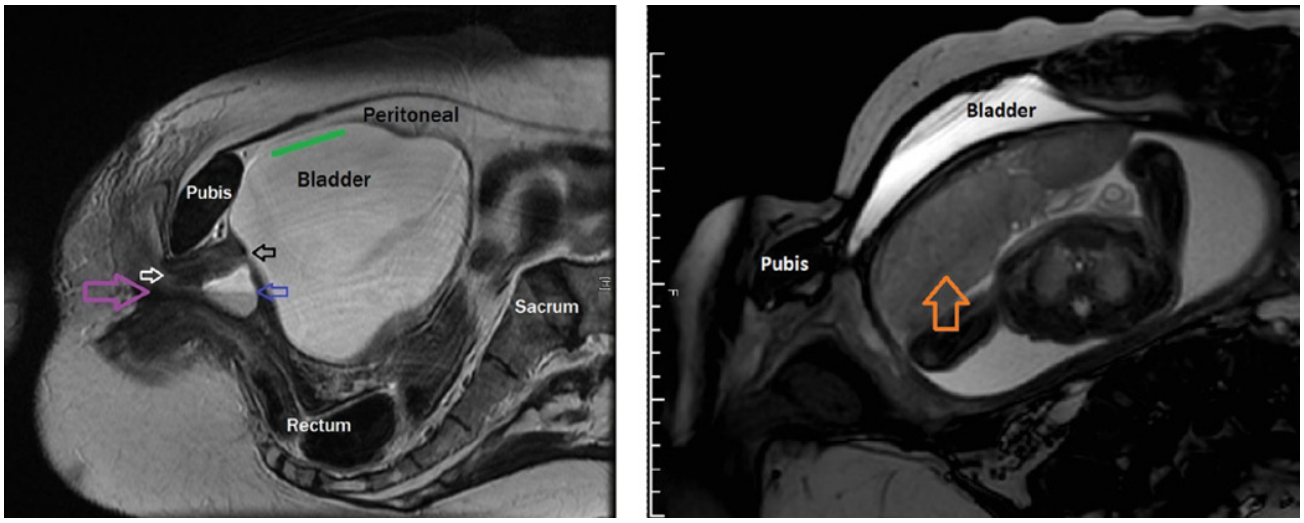

Figure 2.5 Anatomical relationships of the bladder scanned with magnetic resonance imaging (MRI) in the supine position in two women, one with a urethral diverticulum (left) and another with an augmented bladder during pregnancy. The pubis is in front of the lower part of the bladder and the urethra. When full, the bladder dome rises into the abdomen, with the top part (indicated by 'peritoneal') related to the peritoneal cavity. The non-peritoneal part is where a suprapubic catheter would normally be targeted (indicated by a green line). Black arrow: internal urethral meatus; white arrow: external urethral meatus, located in the vaginal vestibule (purple arrow). The blue arrow indicates the urethral diverticulum (containing a fluid level). Behind the urethra and bladder lies the vagina, which has the cervix and uterus superiorly. Space in the lesser pelvis, between pubis and sacrum, is constrained. Hence, any bulky tissue (fibroids, pregnancy) or fluid collection will compress and distort the bladder and could reduce capacity, as illustrated on the right. This woman has a bladder augmentation, so the bladder is tethered by the previous surgery. She will need a caesarean section, and the bladder lies directly in line with the access to the uterine lower segment, so specialist expertise will be needed to avoid catastrophic bladder damage. The placenta is indicated by the orange arrow. *Source*: Marcus Drake.

configured to be able to expand sufficiently to maintain waterproof lining properties so that it can protect the inside of the bladder over the wide range of volumes it routinely encounters. When empty, the loose attachment of the urothelium permits it to fold. The luminal aspect of the bladder is protected by a glycosaminoglycan layer attached to the superficial umbrella cells of the urothelium. This helps with the barrier properties and also gives some resistance to bacterial adhesion. Alongside its barrier role preventing water and toxins from reaching inner layers, the urothelium is metabolically active and able to release a range of active molecules, notably adenosine triphosphate (ATP) [2]. Since there are numerous afferent nerves just under the urothelium, the compounds released from it may influence sensory reporting from the bladder.

The muscular layer comprises smooth muscle, known as the detrusor (short for detrusor vesicae). In humans, it forms an interlacing meshwork of bundles, with fine nerve fibres interspersed throughout (Figure 2.7). These nerve fibres express acetylcholine (ACh) as their main transmitter, but they also contain other possible transmitters, notably ATP and peptides [4]. The role of these other transmitters is not fully understood, but the number of nerve fibres and the range of transmitters they express are markedly reduced in neurological disease (Figure 2.7) [4]. The detrusor and suburothelial layer have large numbers of a metabolically active cell type known as the 'interstitial cells'. These may mediate some aspects of neuromuscular transmission, sensory transduction (i.e. the process of turning a stimulus into nerve impulses), and muscle excitation [5].

The nerve fibres branch out within the connective tissue planes between the muscle bundles, from which they enter the bundles (Figure 2.8) in order to gain close access to the detrusor, which ensures the entire bundle can contract effectively in response to command from the spinal cord.

Notably, the role of the innervation is not solely for making the detrusor contract for voiding. It is also needed for suppressing spontaneous muscle activity. Once the nerve supply is removed, there is considerable movement evident in detrusor. This is very clear in the laboratory setting, where strips of muscle and isolated whole bladders show intriguing micromotions – localised contractions and elongations [6]. The micromotion activity is an important property for normal bladder function [7] and also provides a measure of compensation which can compensate for a small amount of denervation [8]. It may also explain why some people can get substantial urgency even if they do not have an associated change in bladder pressure (Figure 2.9). Overall, denervation may explain why some people experience both

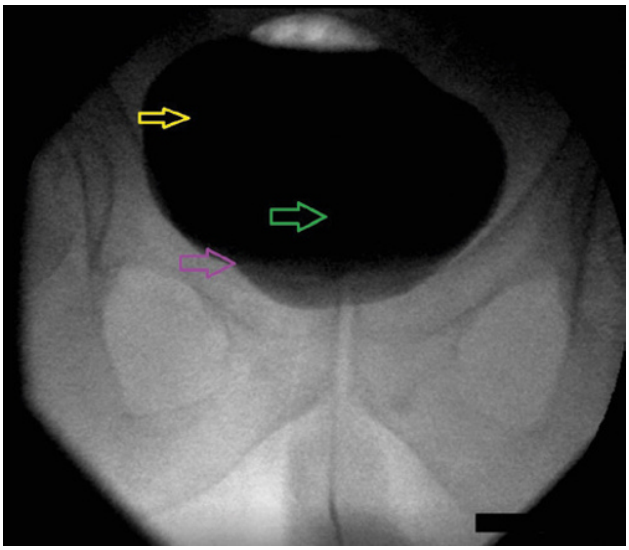

Figure 2.6 Relationship of the bladder to the pubis; X-ray taken during video urodynamics in a man in the standing position. The purple arrow indicates the top of the pubis. The green arrow is where a suprapubic catheter could be placed safely in someone slim who has not previously had abdominal surgery (ultrasound confirmation to ensure no bowel is in the way can be used to check safety of this location in other patients). The yellow arrow indicates an estimation of where the peritoneum crosses from the top of the bladder to the back of the rectus muscles; there is no indicator on an X-ray image to indicate exactly where this occurs. *Source*: Marcus Drake.

overactive bladder (OAB) (urgency during the storage phase due to increased micromotions) and underactive bladder (weak voiding, as the reduced innervation is insufficient to generate adequate overall bladder contraction) [8].

The Male Urethra

The male urethra averages 16–20 cm in length (Table 2.1) and can be considered in four portions (Figure 2.10):

1) Prostatic urethra: from bladder neck to prostate apex. The bladder neck constricts the internal urethral meatus and stays shut at all times except when voiding. This

Table 2.1 Comparison of male and female lower urinary tract.

Female	Male
3–4 cm straight urethra	15–20 cm 'S'-shaped urethra
Wide diameter outlet	Narrow diameter outlet
Sphincter 'horseshoe'	Sphincter 'circular'
Laminar flow	Turbulent flow
Voiding 'low pressure'	Voiding 'high pressure'

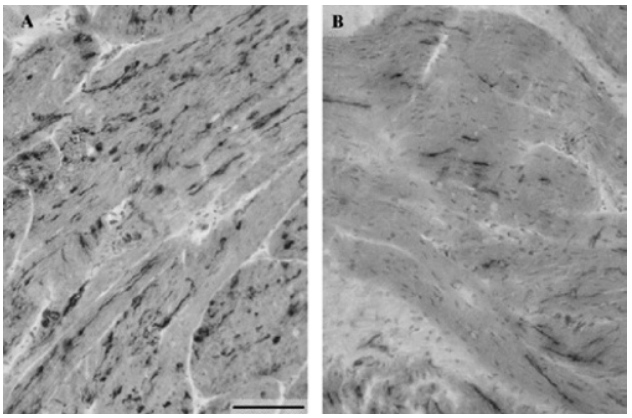

Figure 2.7 Thick muscle of the detrusor (light grey). On the left, the muscle has normal cholinergic innervation density, shown by the numerous black fibres (nerves viewed from the side) and black dots (nerves cut transversely). On the right, a person with spinal cord injury, shown to have partial denervation (far fewer black fibres and dots). *Source*: Reproduced with permission from [3].

well-defined muscle represents the 'genital sphincter' designed to prevent reflux of ejaculate at the time of orgasm. Below the bladder neck, the prostatic urethra is surrounded by the prostate gland and hence is at risk of any nodular enlargement of the gland intruding into the lumen, leading to benign prostate obstruction. The posterior aspect of the prostate is palpable from the rectum (Figure 2.11). This part of the urethra is about 3 cm long and it receives the ejaculatory ducts (Figure 2.12). It is horseshoe shaped on transverse section, with the convexity upwards. The smooth muscle of the prostatic urethra in males is histochemically distinct from that of the detrusor and from urethral muscle in females. This muscle also forms the prostatic capsule. It is richly provided with noradrenergic (norepinephric) nerve terminals, and little acetylcholinesterase has been found. Changes in pressure can occur in this part of the urethra during penile erection, and these changes do not seem to occur during any part of the micturition cycle unless there is erection.

2) Membranous urethra: the part which traverses the pelvic floor, closed by the urinary sphincter in the storage phase. The membranous portion is the shortest portion of the urethra at 2 cm and is the least dilatable. It perforates the pelvic diaphragm behind the bottom end of the pubic symphysis (viewed with the man standing) (Figure 2.10). The male external urethral sphincter is circular, and it works by concentric constriction of the lumen. It applies a constant sustained (tonic) squeeze, supplemented by an enhanced squeeze (phasic) which is reflexly applied prior to and during physical exertion. Additional enhancement of the sphincter squeeze can be exerted deliberately by the person. Voluntary contraction of the

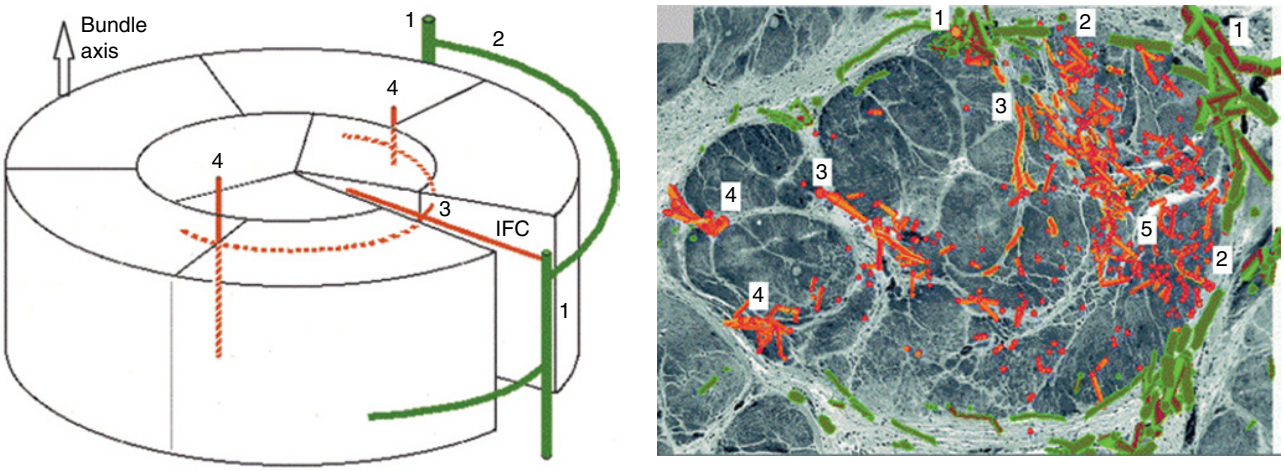

Figure 2.8 Left: a schematic representation of the midpoint of a muscle bundle, made up of eight fascicles separated by connective tissue planes. 1: longitudinal peribundle nerve trunk (green); 2: circumferential peribundle nerve branch; 3: transverse interfascicular branch (orange); 4: axial interfascicular branch, source of the terminal innervation [3]. IFC: interfascicular cleft. Right: the patchy nature of denervation in spinal cord injury. *Source*: Reproduced with permission from [3].

relaxed sphincter is probably the way that most people interrupt the urinary stream, should they choose to do so when voiding. The sphincter stays shut at all times, except when voiding and during ejaculation. The opening of the sphincter during ejaculation does not usually lead to any urinary incontinence due to the maintained closure of the bladder neck. However, previous damage to the bladder neck (e.g. prostatic resection) or loss of its nerve supply (e.g. retroperitoneal lymph node dissection to treat testicular cancer metastases, or neurological disease of the thoracolumbar spinal cord) may lead to reporting of incontinence with ejaculation ('climacturia') and/or dry orgasm (retrograde ejaculation).

3) Bulbar urethra: from below the sphincter to the penoscrotal junction. This is the fleshy part of the male urethra palpable from outside just in front of the anus, an area which can pool urine after voiding in men and hence be responsible for post-micturition dribble (Figure 2.13). It lies close to the attachments (crura) of the corpora cavernosa on the ischiopubic rami. This part receives ducts of urethral glands. The proximal part of the bulb has some skeletal muscle on its ventral side, the bulbospongiosus; this muscle contributes to expelling semen during ejaculation.
4) Penile urethra: extends to the external meatus. This section may be responsible for the spiral nature of the urine stream when it leaves the external meatus. This effect may help prevent spraying and allow accurate direction of the stream. It is perhaps equivalent to the spin of a rifle bullet introduced by spiral 'rifling' grooves inside the barrel, which keeps the bullet stable in flight. If this effect is lost, due to scarring or damage anywhere along the urethra, the resulting turbulence is experienced as a splitting and spraying stream.

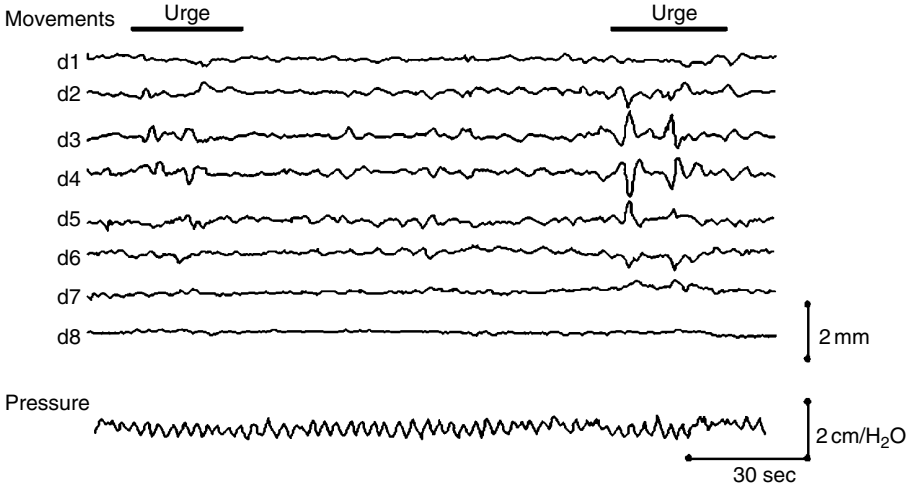

Figure 2.9 Detrusor muscle can show localised contractions which distort the bladder sufficiently to cause urgency sensations but do not cause sufficient pressure change to be detectable by urodynamic equipment recording at conventional sensitivity settings. Pressure is shown at the bottom, while d1–d8 plots the separation of points in different parts of the bladder. Picture reproduced with permission from [9]. 'Urge' indicates the moments at which the patient experienced urinary urgency. *Source*: Drake et al. [9].

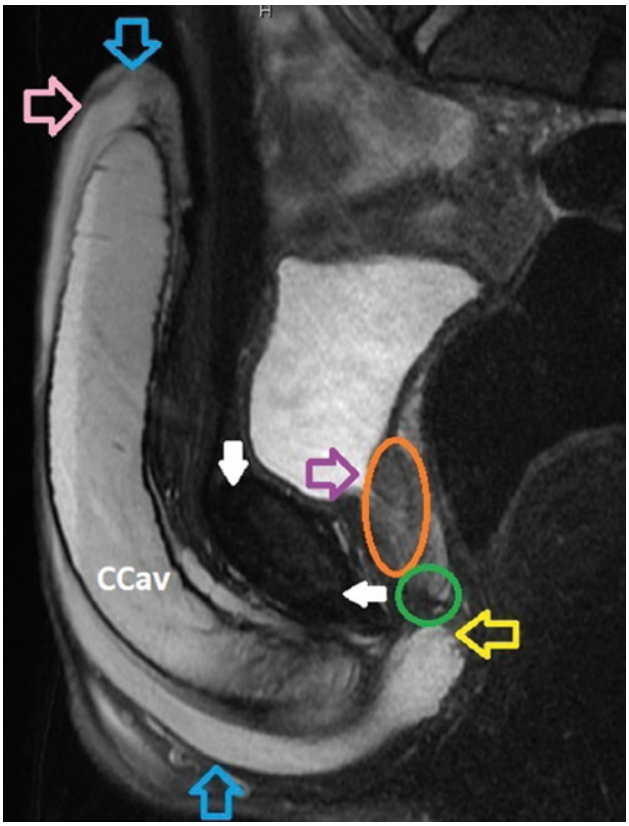

Figure 2.10 Anatomy of the male urethra shown with a magnetic resonance imaging (MRI) scan (sagittal view). White arrows indicate the top and bottom of the pubic bone (which shows up dark in these MRI settings). Purple arrow: bladder neck. Orange circle: the prostate – the prostatic urethra cannot be seen as it is empty during the storage phase of the micturition cycle. Green circle: membranous urethra, lying level with the bottom of the pubis. The membranous urethra is also not actually visible on MRI at these settings during the storage phase. Yellow arrow: start of the bulbar urethra. Blue arrows: penile urethra running from the bulbar urethra alongside the penile crura (anchor point of the erectile mechanism) to the external urethral meatus just beyond the right corpus cavernosum (CCav; erectile mechanism).The pink arrow indicates a dilation in the distal urethra known as the navicular fossa, which can sometimes cause awkwardness when trying to catheterise. *Source*: Marcus Drake.

The male urethra is well supported due to its relationships to the prostate, pelvic floor, and crura of the corpora cavernosa. The curvatures of the male urethra are worth considering, since they have to be negotiated for placing vesical catheters. Holding the penis vertically when the man is lying supine means that the main curve is where the bulb joins the membranous urethra just in front of the anus (Figure 2.10). This is approximately a right angle. Once past the membranous urethra, the prostatic urethra represents a slight upwards curve, which basically continues the preceding bulbar-membranous curve so that the overall effect is greater than a right angle – almost a hairpin bend. At the proximal end of the prostatic urethra, the bladder neck can provide a lip against which the tip of the catheter can bump, preventing entry into the bladder when virtually there. This can very easily be overcome with a catheter possessing a slight angulation at the end (Coudé or Tiemann-tipped catheter [Figure 2.14]); should the catheter tip bump against an obstruction at the bladder neck (or indeed anywhere along the urethra), rotating the catheter while trying to advance it will usually allow the angle to find the lumen very easily. This is occasionally necessary at the distal end of the penile urethra, level with the rim of the glans, as this can have a bit of a lip at the start of the navicular fossa (Figure 2.9), which can cause a bit of awkwardness to find the lumen. In this area, there can sometimes be a blind pit which looks like the urethra (Figure 15.10). It is then necessary to inspect carefully with a good light available and try passing the catheter at various angles in the anterior-posterior plane until the lumen becomes evident.

The Female Urethra

The female urethra is membranous canal, approximately 4 cm long, which extends from the internal to the external urethral meatus (Table 2.1). It is placed behind the pubic symphysis (Figure 2.5) and obliquely traverses the anterior wall of the vagina. When a woman is lying supine, the urethra is roughly horizontal.

The smooth muscle of the female urethra is arranged longitudinally so that when it contracts, the urethra becomes a bit shorter and wider, which facilitates voiding. When passing urine, the urinary stream for a woman does not spiral (as it does for a man); the flow is described as 'laminar', with the inner part of the stream moving quickest, and the outer part slowest, due to the slight friction drag against the epithelium. Another big difference from the male is the configuration of the sphincter, which is asymmetric, being much thicker on the dorsal side (on top of the urethra when the woman is lying supine) [11]. Consequently, the sphincter in women does not constrict, it presses in to cause a 'kink'. This is an efficient way of occluding flow, and a similar approach is used by gardeners to stop flow along a hosepipe by bending it, as opposed to constricting it. The other muscles of the pelvic floor also contribute to this effect.

The female urethra could be considered in segments: intramural, midurethra, membranous (in the urogenital diaphragm), and distal [12]. The maximum urethral pressure (MUP) at rest is in the region of the 40th and 54th percentile along the total urethral length. This peak occurs within the area of the rhabdosphincter and the circular

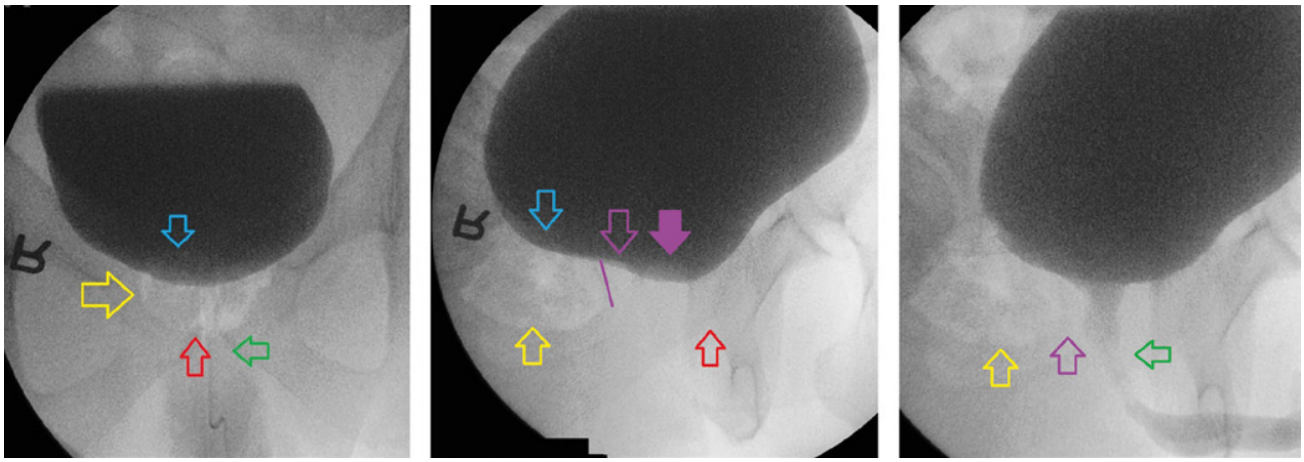

Figure 2.11 In the postero-anterior (PA) view (left), an object resembling the prostate is indicated by the yellow arrow (compare with Figure 8.19). This object extends distally to the level of the external urethral sphincter (green arrows), as expected of the prostate. However, the oblique views during filling cystometry (middle) and pressure flow study (right) show the object is too posterior to be prostate, and it is in fact a hard bolus of faeces in the rectum, consistent with a reported history of constipation. The prostate is indicated by purple arrows (open for posterior, closed for anterior). The red arrows are included to show the bottom of the pubic arch, as this is the only indicator of the likely level of the external urethral sphincter during filling cystometry, and its appearance on oblique view needs familiarisation. The blue arrows are included to indicate the absence of any intrusion of the faecal mass into the bladder, despite the presence of a reasonably large amount of firm faeces. This rather suggests that a widely held assumption that constipation reduces bladder capacity by taking up excessive space in the pelvis is hard to support in reality, in contrast to the potentially substantial effect of uterine anatomy illustrated in Figure 2.5. *Source*: Marcus Drake.

smooth muscle of the urethra. This high-pressure zone is located proximal to the urogenital diaphragm [13]. Voluntary contraction of the rhabdosphincter and the pelvic floor muscles increases the MUP in this area and can be demonstrated by asking the patient to contract their pelvic floor, with the urethral catheter at this area. This can also be used as a biofeedback tool in educating patients whilst performing urethral pressure profile studies. Pathologies affecting these muscles affect the MUP.

The muscle is of a 'slow twitch' type, adapted to maintain contraction over a relatively long period of time. The pelvic floor is separated from the urethra by a layer of connective tissue and is histochemically and histologically different from the intraurethral striated sphincter. It is important in providing an effective platform for sphincter function, including reinforcing contraction when undertaking physical exertion. This solid platform is particularly important for function reliant on an asymmetric sphincter, as contraction of the sphincter could otherwise simply push the urethra away without generating the necessary kink. Hence, healthy pelvic floor muscles, and the associated ligamentous supports, are particularly important for urinary continence in women.

The epithelial lining of the urethra is organised into longitudinal folds that give the urethral lumen a stellate appearance when closed. When opened (as in voiding), this arrangement ensures considerable distensibility so that the urethra does not restrict the channel for urine flow. Oestrogen receptors are noted in the urethra to a similar extent as the vaginal epithelium in women [14]. The submucosal layer below the epithelium is a vascular plexus. Zinner et al. [15] discussed the role of this layer in relation to inner urethral wall softness. He suggested that the submucosa acts in a passive plastic way to 'fill in' between the folds of mucosa as the urethra closes and hence improves the efficiency of the closure of the urethral lumen. The contribution of this vascular closure may explain the presence of urethral pressure changes synchronous with the arterial pulse (Figure 2.15). Following menopause, oestrogen deficiency may reduce turgor of the vascular plexus and could be one factor in the increased prevalence of LUTS.

Overall, maintenance of high intraurethral pressure, and hence continence, can be considered to have three contributors [16]. Striated muscle (rhabdosphincter) of the urethra and pelvic floor is responsible for some of the total intraurethral pressure. The vascular plexus of the urethra contributes as well, and the remaining part could be attributed to the smooth muscle and connective tissues in the urethra. The integral theory is an anatomical framework for understanding pelvic function and dysfunction. 'Restoration of the form (structure) leads to restoration of function' is the main principle of the integral theory [17]. According to the theory, the pelvic muscles, connective tissue (endopelvic fascia and its condensations), and nerve components are crucial in the support and functioning of the pelvic organs like the lower urinary tract, vagina, and

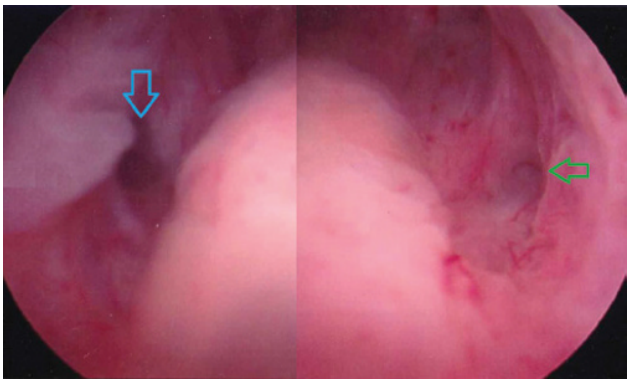

Figure 2.12 Urethroscopy showing the prostatic urethra, to illustrate the entry points of the ejaculatory ducts on the right (blue arrow) and left (green). The pink mound between the arrows is known as the verumontanum, an anatomical landmark for surgeons, as just below it is the external sphincter (not shown). *Source*: Marcus Drake.

the anorectal canal. The suspension bridge analogy explains the pelvic structure, and the trampoline analogy explains the function. This theory formed the basis for the development of the midurethral sling operation in managing stress urinary incontinence [17].

In women with stress urinary incontinence, imaging can find changes related to the urethral sphincter deficiency and defects of the urethral support ligaments and urethral hypermobility. These include a small urethral muscle volume or a short urethra, defects in the urethral sphincter, funnelling at the bladder neck, distortion of the urethral support ligaments, cystocele, an asymmetric pubococcygeus muscle, abnormal shape of the vagina, enlargement of the retropubic space, and an increased vesicourethral angle [18].

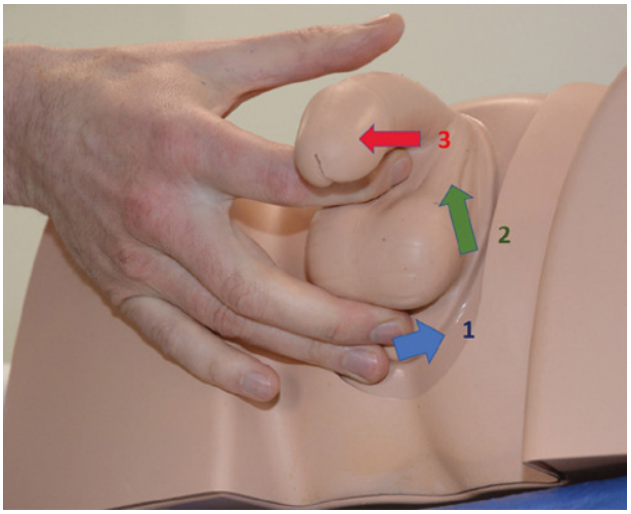

Figure 2.13 The location of the urethral bulb, and how knowledge of this area is important when teaching men how to get rid of drops of urine caught here after voiding, which later on dribble out into the underwear (post-micturition dribble). Instructions given to patients: '1. Press upwards gently but firmly, between the anus and scrotum. 2. Gently keeping the pressure on, ease your fingers forwards, so the urine can be eased forwards towards the penis. 3. You can shake the last drops from the penis'. Instruction 1 relates to compressing the urethral bulb. Figure reproduced with permission from the TRIUMPH (TReatIng Urinary symptoms in Men in Primary Healthcare) study (protocol [10]). *Source*: Marcus Drake.

The Pelvis

The Bones

The pelvis forms embryologically from the pubis (in front), the ischium (below), and ilium (behind) and is completed by the sacral part of the spinal column. This provides a solid framework for the lower urinary tract and nearby structures and, being radio-opaque, shows up clearly on X-ray. Interpreting the 3D framework in 2D images is an important step for understanding video urodynamics (the subject of Chapter 8) (Figure 2.16).

The Pelvic Floor

The levator ani (pubococcygeus, puborectalis, and the iliococcygeus) and the coccygeus muscles run in the anterior-posterior plane. In the midline, they diverge around the anus and urethra, and vagina in women, as they leave the abdominal cavity on to the perineum. This muscular diaphragm thus supports the pelvic viscera and hence is referred to as the pelvic floor. It curves downwards slightly when relaxed, and contraction of the muscles flattens the curvature, lifting up the pelvic organs. This also compresses the medial edges of the levator muscles together. The elevation and compression can be felt by internal examination during a voluntary pelvic floor squeeze. Elongation of the muscle or damage to its ligamentous support makes both the elevation and the compression more difficult to achieve so that organs are less well supported (prolapse risk) and the asymmetric sphincter contraction is less able to compress the urethra (in women). Some of the predisposing factors that can contribute to this include:

- myopathy,
- denervation,
- ligamentous damage (notably caused by childbirth),
- chronic straining,
- postural problems, and
- bony malformations, e.g. pubic diastasis or sacral anomalies.

Older people are at greater risk of accumulating some of these problems.

Figure 2.14 Coudé catheter. Gently rotating the catheter during the insertion often facilitates placement, overcoming awkward curves in the male urethra. It can sometimes be helpful for catheterising women as well. *Source*: Marcus Drake.

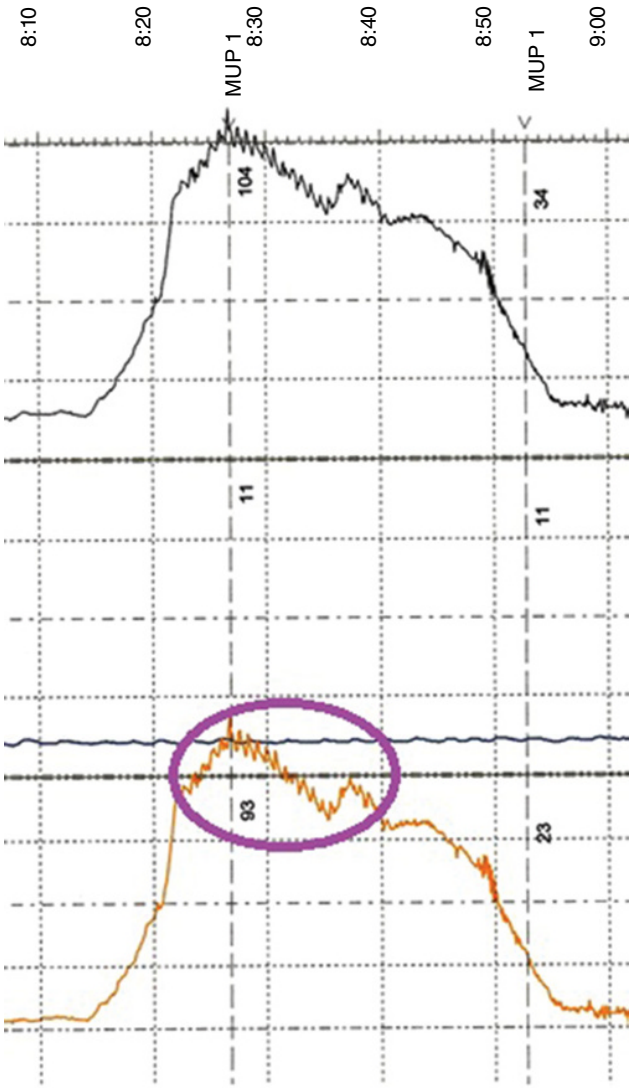

Figure 2.15 Urethral pressure profile in a woman showing the small pulsations on top of the high-pressure part of the profile (purple oval), synchronous with the pulse, known as vascular pulsations. The vascular plexus under the epithelium of the female urethra is prominent and may be a contributor to maintaining continence.

Endopelvic Fascia

The connective tissue (collagen, elastin, and smooth muscles) covering the pelvic organs and sidewalls is referred to as the endopelvic fascia and has an important role in supporting and functioning of the pelvic organs. A distinctive band of connective tissues extends from the lower pubic symphysis to the ischial spine on either side of the pelvic side wall, over the upper fascia (perineal membrane), and the levator ani. This condensation of the endopelvic fascia is referred to as the arcus tendineus fascia pelvis or 'the white line'. There are lateral attachments to the white line which support the pelvic organs. Some of the attachments can be distinctively observed and have a role in maintaining continence and support to the pelvic organs. The thickened pelvic fascia between the pubic bone and the anterior vaginal wall (prostatic tissue in males) is called the pubourethral ligament. This supports the urethra and bladder and has a role in continence. Any damage to the pubourethral ligament in women is clinically observed as urethral hypermobility and can be demonstrated on speculum examination and by asking the woman to strain or bear down. There are lateral (Mackenrodt's or cardinal ligaments) and posterior (uterosacral ligaments) fascial attachments to the cervix and the upper vagina, which provide important supports. Any trauma to these structures may result in pelvic organ prolapse. It is also important to recognise that the endopelvic fascia (loose connective tissue) carries much of the vascular, lymphatic, and nervous supply to the pelvic viscera and could be damaged during pelvic surgery.

Nervous System Control of the Lower Urinary Tract

The Peripheral Motor Nerves; 'The Efferents'

Excitatory efferents are the nerve fibres that go to the muscle and cause it to contract. Damage to these nerves will lead to paralysis, meaning that the muscle will not contract

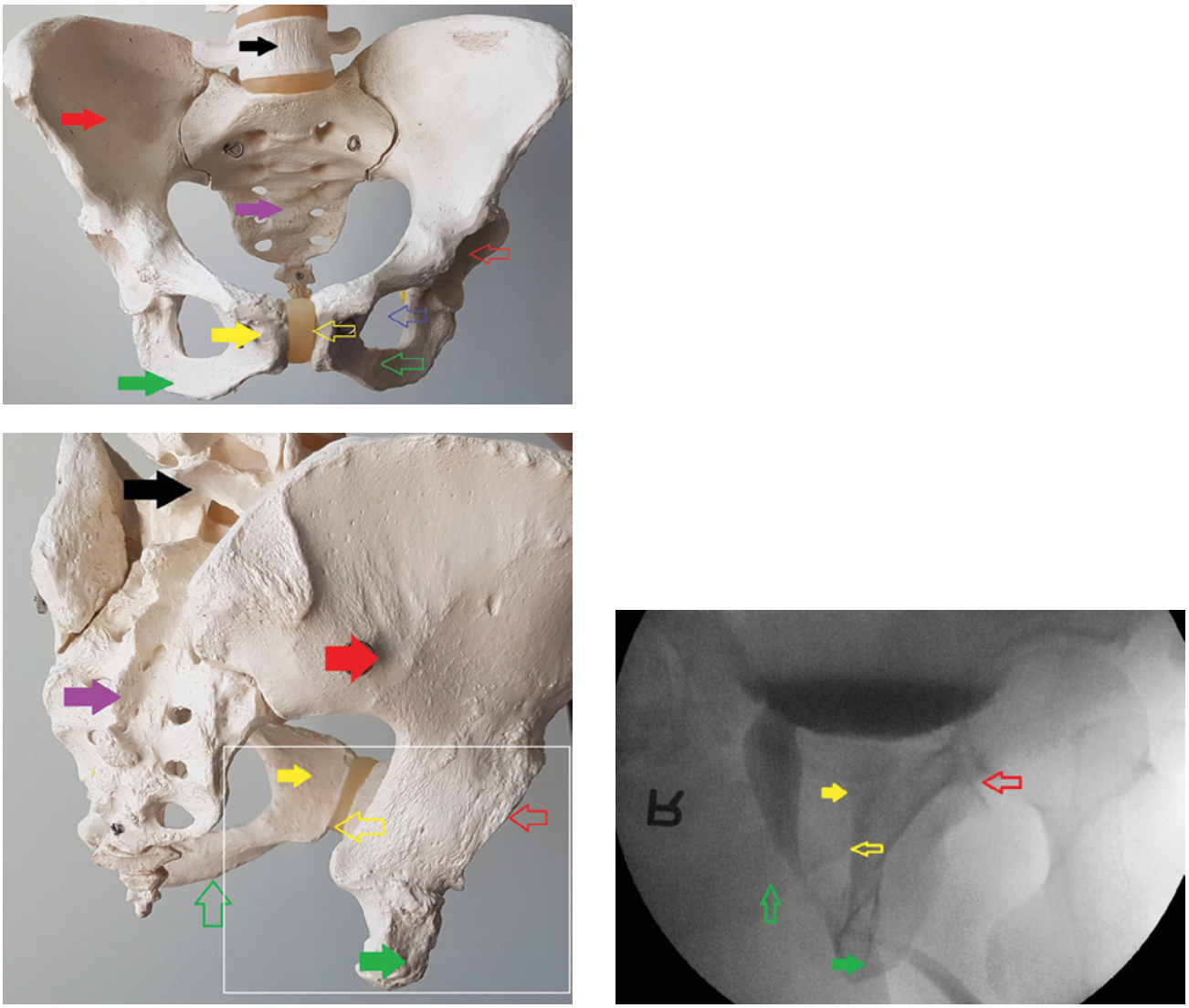

Figure 2.16 The bones of the pelvis, seen straight on (top) and obliquely; the pubis (solid yellow arrow), ischium (solid green arrow), and ilium (solid red arrow). Open yellow arrow indicates the pubic symphysis; open green arrow is the ischiopubic ramus; open blue arrow is the obturator foramen; open red arrow is the acetabulum (the hip socket). Bones of the spinal column; the sacrum (solid purple arrow) and fifth lumbar vertebra (solid black arrow). In the oblique view, the white rectangle gives the approximate area of an X-ray image taking during voiding in a man undergoing video-urodynamics. Illustrated on the right. *Source*: Marcus Drake.

when the person wishes, or a reflex relying on the muscle is triggered. For the bladder, the excitatory efferents are a network ('plexus') of fine fibres running over the other pelvic organs. As a consequence, they may be affected in diseases or surgery to the nearby organs, most notably gynaecological and colorectal operations. In the past, radical surgery (as needed in some cancer operations) to either of these organs could damage the plexus of bladder nerves so much that the bladder became acontractile. Modern-day surgical techniques have been designed to reduce the potential impact on the bladder. Nonetheless, there is likely to be some damage to a few of the nerves; the effect will usually be subsymptomatic, but a patient with extensive past background of pelvic operations could be at risk of impaired voiding function as a result of the accumulation of multiple low-level damage events.

Inhibitory efferents are also present in the plexus of nerves running to the bladder. These suppress spontaneous muscle 'non-voiding microcontractions', which are an innate feature of uninhibited detrusor muscle. The presence of an inhibitory innervation is needed to ensure the quiescence of the detrusor generally applying during filling. Nonetheless, microcontractions can sometimes emerge in normal function, indicating that the detrusor inhibition by inhibitory efferents is probably varied physiologically; allowing some microcontraction activity may be

relevant to maintaining tone [8, 19]. Likewise, the localised movements would potentially stimulate afferents even in the absence of any associated effect on intravesical pressure. Consequently, such a mechanism could help report state of bladder filling or may explain 'latchkey urgency' (the strong desire to void when returning home, signifying a safe environment) (Figure 2.9). Excessive or widespread emergence of microcontractions, perhaps because of dysfunction of the inhibitory efferents, would be a factor in detrusor overactivity [8], and the consequent activation of afferents would generate urgency (OAB) [8, 9].

The sphincter is supplied by the pudendal nerve, a clearly identified structure (unlike the plexus supplying the bladder), which runs on the inner side of the ischium. Here, there is a ligamentous/bone channel known as 'Alcock's canal' (Figure 2.17). The nerve can get compressed in the canal, for example, during labour, or from fibrous narrowing, leading to pain affecting the perineum and genitalia. This location can be targeted by interventional radiologists, injecting local anaesthetic and steroid under computed tomography (CT) guidance (Figure 2.17). The relevant fibres then run up the ischiopubic ramus, so they can be damaged when a person fractures their pelvis. The pudendal nerve contains nerve fibres which the person can control voluntarily ('somatic'), enabling deliberate tightening of the sphincter. In addition, the nerve contains autonomic nervous system input comprising involuntary 'visceral' fibres, ensuring the sphincter is held shut without the person having to think about it. This direct combination of somatic and visceral components is highly unusual in the human body. Note that the bladder is a visceral structure; people cannot directly make the bladder contract, they can only elicit a bladder contraction indirectly by setting off the voiding reflex.

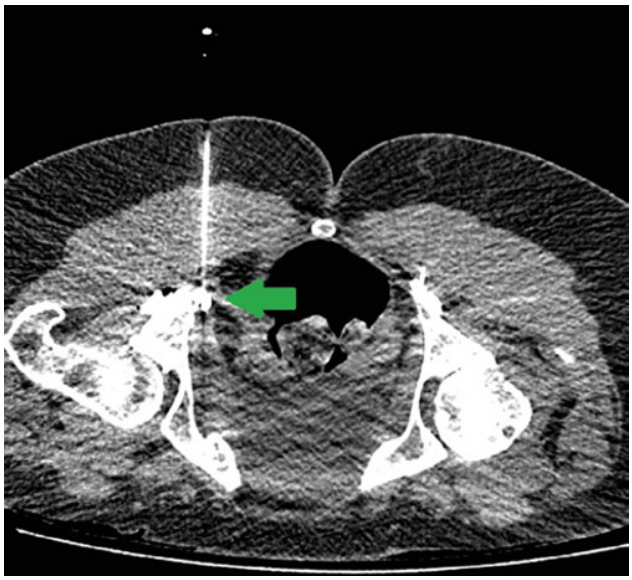

Figure 2.17 Computed tomography-guided placement of a needle for injection of local anaesthetic and steroid adjacent to the pudendal nerve in Alcock's canal (green arrow) in a woman with pudendal neuralgia. She is lying in the prone position. *Source*: Marcus Drake.

The bladder neck in men is supplied by the sympathetic nerves (excitatory efferents), which are partly plexiform, but they have some thicker nerve bundles, referred to as hypogastric nerves. If there is a problem with these nerves, paralysis of the bladder neck means it is open during the storage phase – a readily observed feature in video urodynamics (Figure 2.18).

Neuromuscular Transmitters and Their Receptors

Much recent effort has been directed towards the analysis of receptors in the urinary tract, and the following are fairly well-established observations:

- Alpha-adrenergic receptors, causing smooth muscle contraction when stimulated, are present in the bladder neck and the proximal urethra principally in men. Alpha-adrenergic antagonists ('alpha-blockers'), such as tamsulosin, are used to treat voiding LUTS in men.
- Beta-adrenergic receptors are present in the detrusor. The beta-3 subtype has the effect of eliciting detrusor relaxation, and this is exploited using beta-3 agonists to treat OAB and detrusor overactivity.
- ACh is the major efferent neuromuscular transmitter (cholinergic transmission) generating the detrusor contraction for voiding, acting via muscarinic receptors (M2 and M3). The bladder contains acetylcholinesterase enzymes which break down ACh and shorten its duration of effect. Deficiency in these enzymes might be a factor in detrusor overactivity. Even during the storage phase, there is a very low level of ACh release within the bladder, partly from the nerves, partly from the urothelium. While this may seem counterintuitive, in fact the bladder keeps some 'tone' during filling, which is probably why people can generally void however much (or little) is in their bladder. It is probably this low-level release that is blocked by the comparatively small doses of anticholinergic medications used to treat OAB and detrusor overactivity. These small doses are easily overcome by the substantial surge of ACh seen during voiding, which is why these drugs generally do not cause urinary retention.
- ATP is often released as a co-transmitter (purinergic transmission), and this is especially significant at low-frequency nerve firing and possibly in pathophysiology, such as detrusor overactivity. No medication has been developed to exploit this clinically as yet.

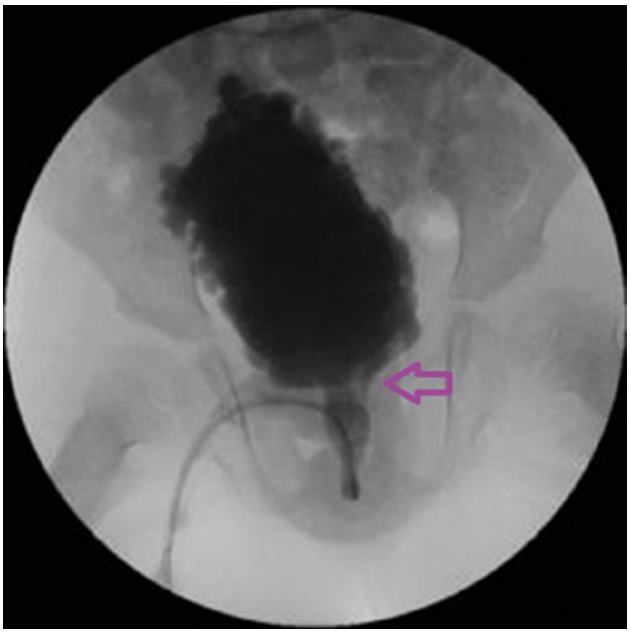

Figure 2.18 Open bladder neck in the storage phase (purple arrow) caused by neurological lower urinary tract dysfunction in a young male. The roughened bladder wall is caused by thickened muscle (trabeculation) and little pockets of urothelium (diverticulation) pushed out by high bladder pressures. *Source*: Marcus Drake.

Several other active compounds are present in nerves, such as nitric oxide and peptides. Their roles are not properly understood, but they do reflect potential interest for physiological understanding and therapeutic potential. For example, fewer of these compounds are present in neurological disease [4]. Nitric oxide is interesting as it may not influence the muscle directly, but instead alter function of the interstitial cells.

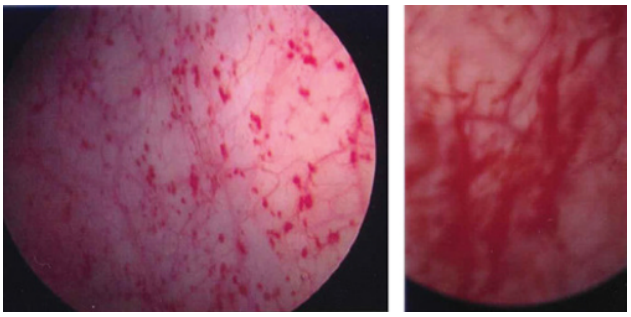

Figure 2.19 Cystoscopic appearance of inflammation in bladder pain syndrome. On the left, the blood spots referred to as 'glomerulations', seen when filling the bladder under general anaesthetic. These are generally seen only during general anaesthetic cystoscopy, as the patient demands the test be discontinued if still awake due to the significant level of discomfort. On the right, a different patient where the inflammatory glomerulations are so superficial they lead to bleeding. *Source*: Marcus Drake.

Peripheral Sensory Nerves; 'The Afferents'

Afferents translate a stimulus into a nerve firing pattern which reports to the spinal cord. From the bladder, there are two main classes of afferents. Some report bladder wall distension and distortion, so they become more active if the bladder is fuller. The sensory reporting from the bladder is delivered by afferents located under the urothelium or in the detrusor layer, and the traffic is probably carried along a similar peripheral pathway to the parasympathetic motor nerves. They report continuously, providing a graded nerve traffic proportionate to how full the bladder is. Note that people are not normally aware of this sensory information most of the time, i.e. it is reporting subconsciously. The conscious sensations derived from this information (sensation of filling or desire to void) are only experienced sporadically (see below). The nerve fibres carrying this type of information are mainly myelinated (A-) and small diameter (A-δ).

Another bladder afferent group becomes active only at very high volumes, beyond the point at which the person would have a very strong desire to pass urine. Consequently, they are rarely active, since the person will take steps to pass urine before the threshold volume for these afferents is reached. If activated, they give rise to an unpleasant/painful sensation ('nociception'). These afferents run with the sympathetic nerves. There are some patients who have lost function of the usual A-δ bladder sensory nerves and describe low abdominal pain and a large mass due to overdistension becoming sufficient to reach the nociceptive afferent threshold volume. These fibres are unmyelinated (γ), so they transmit slowly.

The amount of information coming from the urinary tract can be sensitised, i.e. more afferent nerve 'traffic' is carried for the same bladder volume. The increased sensory information due to bladder inflammation is an important sensitiser in the clinical setting. UTI or other forms of bladder inflammation lead to a stimulation of the afferents in the bladder wall (affecting both the usual sensory nerves and the noxious painful nerves). As a result, people have both increased sensation and an unpleasant 'noxious' feeling, leading to increased urinary frequency, and discomfort which persists after voiding. Characteristically, there will be leukocytes in the urine, and the bladder will appear inflamed if viewed cystoscopically; this might be a factor in bladder pain syndrome (Figure 2.19).

Sensitisation can also result from inflammation present anywhere along the track of the peripheral nerves. This might be a result of gynaecological inflammation, such as pelvic inflammatory disease or endometriosis. Alternatively, there might be an inflamed colonic diverticulum close to the bladder. The result is increased sensory

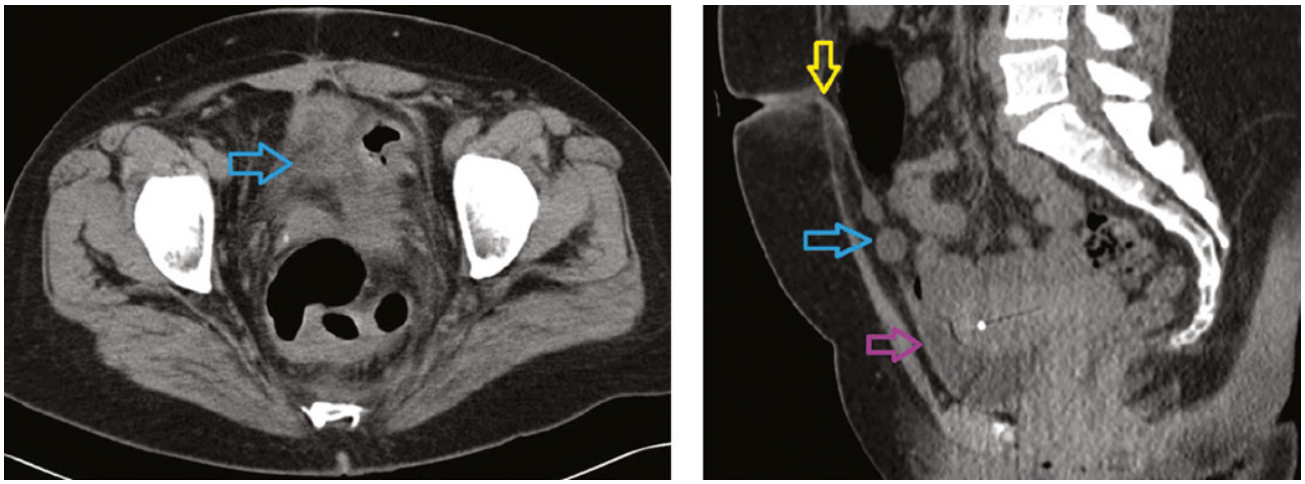

Figure 2.20 Computed tomography scans in people with extrinsic inflammation of bladder afferents, which the affected patients described in such a way that a UTI was suspected, but urinalysis showed no leukocytes. Imaging revealed the cause in each case. Left: inflamed colonic diverticular mass stuck to the bladder dome (blue arrow). Right: urachal cysts (blue arrow) which had become infected. The urachus is a structure joining the bladder (purple arrow) to the umbilicus (yellow arrow) in the foetus, which can persist into childhood giving rise to cysts and sometimes even allowing urine to escape from the umbilicus. *Source*: Marcus Drake.

information, both normal and noxious (as for the inflamed bladder), but without leucocytes in the urine. In these cases, history of gynaecological or colonic symptoms, and CT imaging to look for an inflammatory mass, may be informative (Figure 2.20). Should colonic diverticular disease progress, it can eventually lead to a colovesical fistula, where the urinalysis findings are strongly suggestive of UTI. However, this is not an ordinary case of cystitis, but actually a consequence of the bladder communicating with the colon. For this reason, it is vital to understand the possible significance if a patient describes gas in the urine or 'bubbly urine' (properly known as pneumaturia), faecal matter in the urine, or urinary diarrhoea (Figure 2.21).

Urethral receptors are an important group of afferents, which serve to detect flow. They are thus only occasionally perceived by the person (since there should be no flow during storage). However, they are extremely important in voiding. When urine is flowing, the resulting stimulation of the urethral afferents reports to the pontine micturition centre (PMC; see below). The presence of urine in the urethra indicates to the CNS that bladder emptying is not yet complete, and this sustains the drive maintaining detrusor contraction. Once the bladder has emptied, the urethral afferents stop reporting urine flow; at this point, the voiding reflex terminates. Thus, the reduced urethral afferent traffic is a key contributor to concluding the voiding contraction.

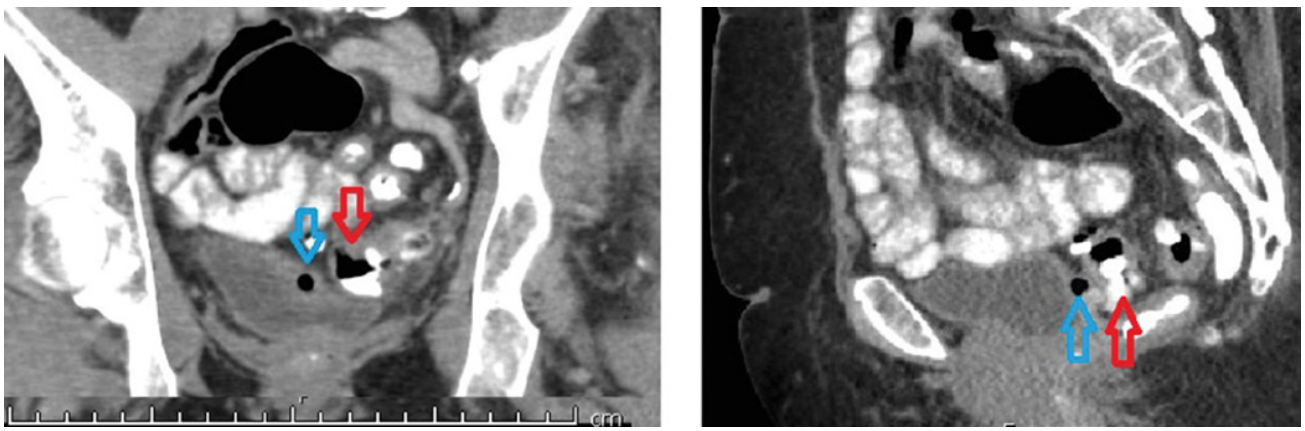

Figure 2.21 Abdominal computed tomography scan (left: antero-posterior view; right: lateral view) of a woman with diverticular disease which has stuck to the bladder and penetrated the bladder wall. It has caused a colovesical fistula so that gas is present not only in the colon (red arrow) but also in the bladder (blue arrow), leading to pneumaturia. *Source*: Marcus Drake.

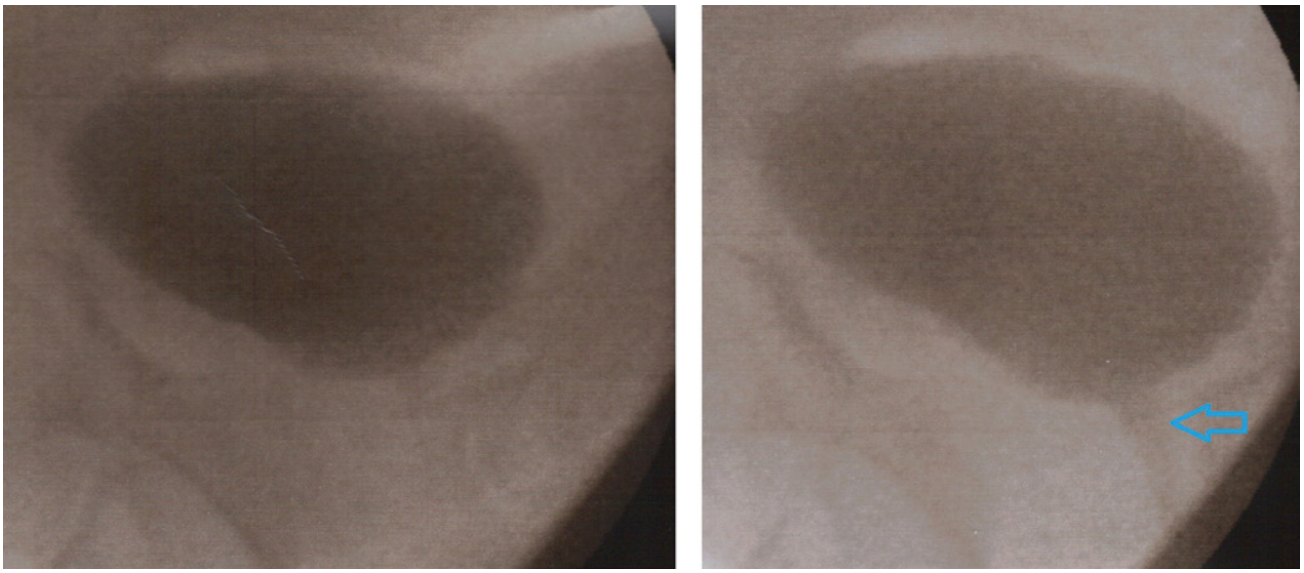

Figure 2.22 Urethral receptors can cause urgency. In this man undergoing video-urodynamics, urgency was reported when his prostatic urethra was stimulated by entry of liquid from the bladder into the prostatic urethra (blue arrow, right-hand image). There was no detrusor overactivity. He previously had had a bladder neck incision. *Source*: Marcus Drake.

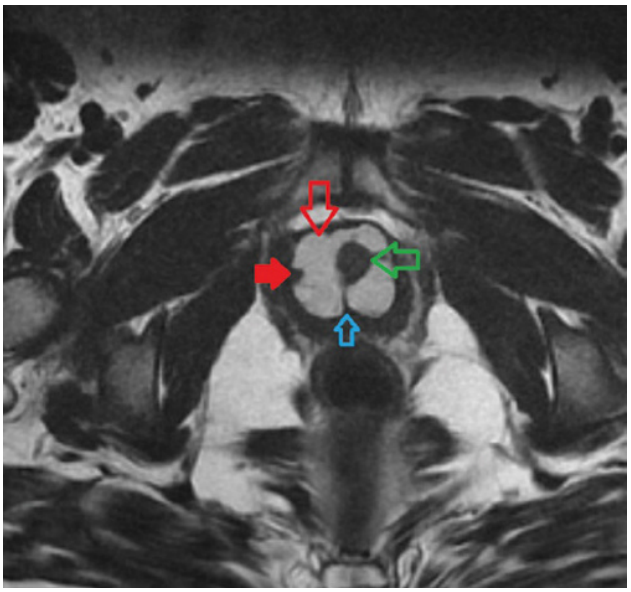

Figure 2.23 Structural changes related to the urethra can lead to lower urinary tract symptoms (LUTS). In this case, a large urethral diverticulum (UD) in a woman was associated with both storage and voiding symptoms. The storage symptoms could result from inflammation in the trapped contents of the diverticulum, while voiding LUTS could be a direct compressive effect on the urethra. The image is from magnetic resonance imaging, with a UD (open red arrow) fully surrounding the urethra (green arrow). The closed red arrow indicates a small lump in the UD; this will need interval scanning, as malignant change in a UD is occasionally reported. The blue arrow indicates the meeting point where the two arms of the UD make contact with each other behind the urethra. *Source*: Marcus Drake.

A couple of interesting clinical observations can be understood by appreciating the contribution of these receptors:

1) If by chance they get stimulated during the storage phase, they give an extremely strong sensation, perceived by the patient as powerful urgency and concern that leakage is imminent (Figure 2.22). This is a factor in the urgency experienced by some people with stress incontinence. Inflammation close to the urethra (e.g. a urethral diverticulum [Figure 2.23]) can also cause increased sensation.
2) Men who have had bladder outlet surgery usually lose bladder neck function. They still have external sphincter function, so they are not incontinent. However, the fact that urine can now stimulate the prostatic urethral receptors means they get a strong urgency feeling and worry that they are about to leak (Figure 2.22).
3) People who have lost urethral afferents do not know when flow is happening and so can only detect it indirectly. For example, when reported by skin afferents of the thigh/perineum, auditory (sound of water hitting the toilet bowl), or by looking at the flow emerging from the external urethral meatus.

In summary, the peripheral innervation of the LUT has several components with distinct functional roles. Selective loss of the fibres or the associated spinal centres has associated effects that can be derived logically and sometimes can be discerned clinically (Table 2.2).

Table 2.2 Matching specific LUT functions to subpopulations of LUT nerves.

Problem	Implication	Clinical manifestation
Loss of bladder excitatory efferents	Impaired voiding contraction	Underactive bladder/detrusor, retention
Loss of bladder inhibitory efferents	Storage microcontractions	Overactive bladder, detrusor overactivity
Loss of bladder afferents	No normal sensation. Overdistention	Person palpates full bladder. Noxious sensation if very full
Bladder afferent sensitisation (intrinsic/ extrinsic)	Increased afferent traffic	Increased filling sensation
Loss of urethral afferents	No feedback of urine flow	Person listens or visualises to detect when flow happens
Loss of pudendal nerve (sphincter efferents and perineal afferents)	Sphincter weakness and lack of genital sensation	Stress/continuous incontinence. Difficulty achieving orgasm
Loss of bladder sympathetic innervation	Storage microcontractions	Overactive bladder, detrusor overactivity
Loss of bladder neck sympathetic nerves (male)	Bladder neck paralysis	Retrograde ejaculation (if sexual function preserved). Feeling of leakage (if urethral afferents preserved)

Spinal Centres

The CNS plays a major part in lower urinary tract and genitourinary function at all levels (Figure 2.24). The sacral spinal cord (segments S2–4) is crucial, as it contains the cell bodies giving rise to the nerve fibres that head to the detrusor and the external urethral sphincter, gathered together in groups known as 'nuclei'. The parasympathetic nucleus is in the intermediolateral (autonomic) horn, which is the source of the plexiform nerves of the bladder; hence, in the urodynamic context, it is sometimes referred to as the 'detrusor nucleus', even though it controls more than just the bladder. Onuf's nucleus is also in S2–4, in the anterior horn, and is the source of the nerves supplying the urinary and anal sphinc-

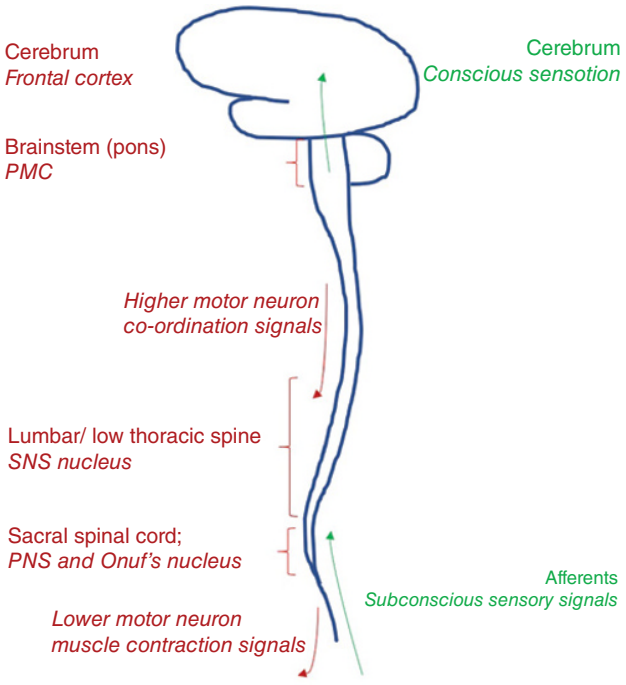

Figure 2.24 Key parts of the central nervous system responsible for lower urinary tract function. The sensory pathway is shown in green, and the motor pathway is shown in red. PMC: pontine micturition centre; PNS: parasympathetic nervous system; SNS: sympathetic nervous system.

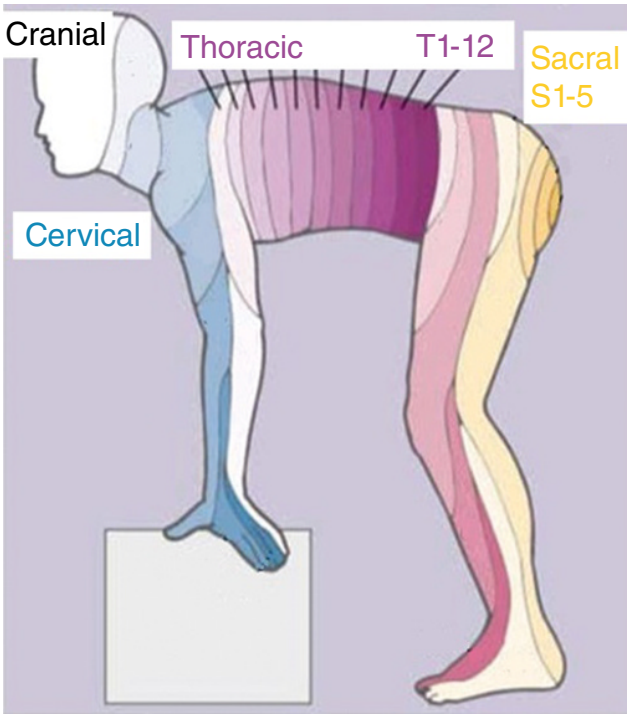

Figure 2.25 The dermatomes are the areas of skin supplied by the various spinal cord segments. These are easiest to appreciate when viewed from the evolutionary perspective of originating from a four-legged configuration.

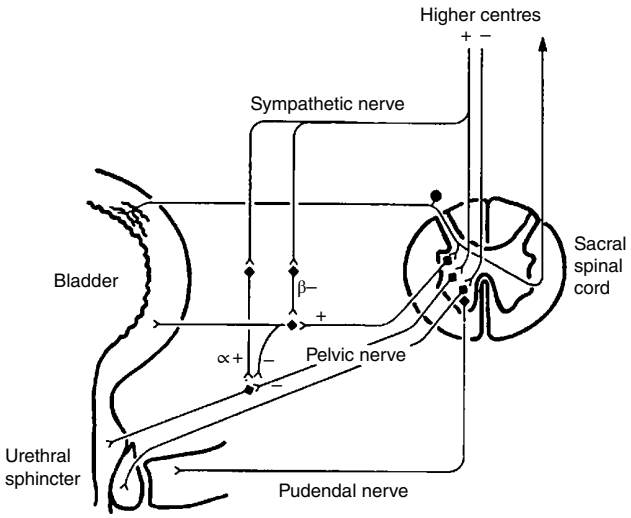

Figure 2.26 A wiring diagram summarising sacral spinal centres and their interactions with the lower urinary tract organs.

ters, carried in the pudendal nerve. Onuf's nucleus has somatic inputs, enabling people to squeeze their sphincters voluntarily. It also has visceral inputs underpinning reflex sphincter contraction (e.g. the guarding reflex, which is an anticipatory contraction just before lifting a heavy object).

Most of the sensory nerves of the lower urinary tract enter the spinal cord at this level, including the bladder afferents reporting filling state. The urethral afferents and the dermatomal (skin) sensation of the perineum and genitals also enter at this part of the spinal cord (Figure 2.245). However, the nociceptive afferents enter the spinal cord in the lumbar spinal cord. This anatomical variation in the afferent nerve course means that selective nerve loss, such as trauma or surgical damage, can occur. Hence, some people never experience normal bladder or urethral sensation but may report unpleasant/painful sensations once their bladder overdistends, indicating that the main bladder afferents have been damaged but the nociceptive nerves have not.

In the thoracolumbar spinal cord, the intermediolateral (autonomic) horn is the sympathetic nucleus. This structure controls the bladder neck and is also responsible in part for keeping the bladder quiescent during the storage phase. Furthermore, This nucleus controls the blood vessels and hence is crucial for maintaining a healthy blood pressure through regulation of vascular tone. Damage to sympathetic nucleus results in:

- low blood pressure at rest (due to loss of resting vascular tone) and postural hypotension (due to loss of reflex vascular tone adjustment);
- a paralysed bladder neck in men, visible as an open bladder neck in the filling phase of video-urodynamics (Figure 2.18); and
- overactive detrusor, due to loss of sympathetic inhibitory influence.

If the sympathetic nucleus is preserved but the inputs from the PMC are damaged, as in spinal cord injury (SCI), there is a risk of autonomic dysreflexia. This is a rapid elevation of blood pressure due to marked vasoconstriction of blood vessels of the gut and lower limbs, which is triggered by traffic in the nociceptive afferents coming up to this level of the spinal cord. Urethral catheterisation is a classic trigger for this, so urodynamics units need to be aware of this risk of testing people with SCI. It applies in patients with SCI at the level of T6 or higher, since this allows enough of the sympathetic nucleus to be preserved to result in dangerous potential for hypertension. These interactions from the sacral spinal cord to the periphery and back again are summarised in Figure 2.26.

The cervical spinal cord does not contain any specific centres responsible for lower urinary tract function. Nonetheless, it is traversed by the ascending sensory pathway in the white matter and the descending motor pathways. The entire spinal cord is the route by which afferent information ascends to the brain, and motor instructions come down from higher centres. Significant damage to the spinal cord at any point means the person may not experience conscious sensation derived from the sensory information carried in those afferents. Nonetheless, the afferent traffic is still present in the spinal cord (subconsciously), hence the potential to trigger autonomic dysreflexia. In addition, these people will have uncoordinated muscle function, notably detrusor sphincter dyssynergia (DSD; see below) since the higher centres cannot communicate the co-ordination instructions to the motor nuclei in the spinal cord.

Dysreflexia and DSD reflect failure of co-ordination of spinal cord centres, but they do not happen if considerable lengths of the spinal cord have been killed, meaning that the nuclei are actually dead. This can happen in major trauma, or a blockage of the anterior spinal artery, which is the dominant blood supply of the lower spinal cord. The resulting 'spinal stroke' leads to flaccid paralysis of all the muscle groups in the lower urinary tract, along with the other organs and the lower limbs.

An important anatomical point to bear in mind is that the segments of the spinal cord are numbered according to the vertebrae which they develop adjacent to in the embryo. By the time a person reaches adulthood, there has been considerable difference in growth, with the vertebrae growing substantially more than the spinal cord. As a consequence, whilst the cervical spinal cord segments are close by the original vertebrae, they develop next to, this does not apply in the sacral segments, which actually lie adjacent to, the upper lumbar vertebrae (Figure 2.27). Hence, if some-

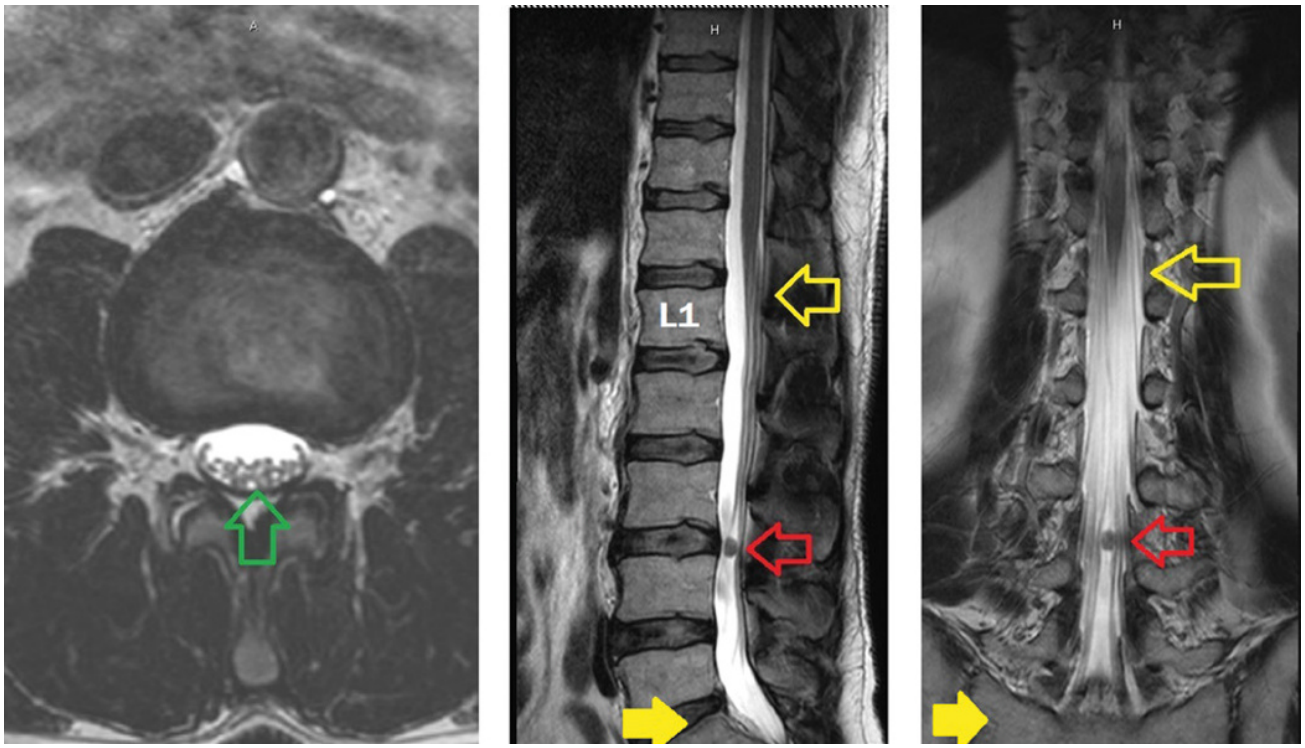

Figure 2.27 Magnetic resonance imaging of the lower spine and spinal cord. The top of the sacrum (closed yellow arrow) is anatomically at a considerable distance from the lowest sacral segment of the spinal cord (open yellow arrow), which is level with the first lumbar vertebra. In order to reach the foramina in the sacrum, the sacral nerve roots travel alongside each other (green arrow); this structure is the cauda equina. Hence, a lumbar prolapsed intervertebral disc, lumbar infection, or metastasis causes sacral nerve root dysfunction. This patient had a resolving abscess in the spinal canal (red arrow), which was responsible for cauda equina syndrome. *Source*: Marcus Drake.

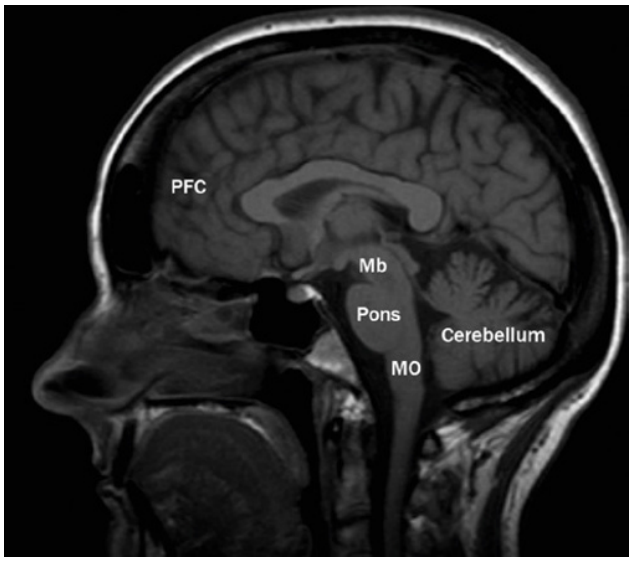

Figure 2.28 Key centres in the higher regions of the central nervous system. The hindbrain includes the pons, near to the location of the pontine micturition centre, and the medulla oblongata (MO). The midbrain (Mb) is the location of the periaqueductal grey. The front part of the cerebral hemispheres is the prefrontal cortex (PFC). *Source*: Marcus Drake.

body is unlucky enough to develop a central prolapsed intervertebral disc, the disc affected will be numbered according to its vertebrae, but the effect of the disc as it enters the spinal canal will be to compress a different spinal cord segment. L1 is the lowest vertebral segment at which the spinal canal contains spinal cord (Figure 2.27), so a disc prolapse between vertebrae L2 and L3 will affect the cauda equina (the nerves spreading out from the sacral spinal cord into the sacrum).

Brain Centres

The brain is divided into three parts anatomically: the forebrain, the midbrain, and the hindbrain. Each of these has direct relevance to LUT function (Figure 2.28).

The hindbrain comprises the pons and the medulla oblongata and is particularly important in the motor control of the LUT. The pons contains the PMC, which regulates many vegetative functions including storage and voiding. For men, the urethra is also part of the genital tract, so the PMC additionally co-ordinates semen emission and ejaculation. By default, the LUT is held in the storage phase, and this is generated by PMC signals sustaining inhibition of the detrusor