CHAPTER 11

Committee 7

Dynamic Testing

Chair

D. GRIFFITHS (USA)

Co-chair

A. KONDO (JAPAN)

Members

S. BAUER (USA),

N. DIAMANT (CANADA),

LIMIN LIAO (CHINA),

G. LOSE (DENMARK),

W. SCHÄFER (USA),

N. YOSHIMURA (USA)

Consultant

H. PALMTAG (GERMANY)

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ABBREVIATIONS

BOO	bladder outlet obstruction
BOOI	bladder outlet obstruction index, previously known as AG number
BPE	benign prostatic enlargement
BPO	benign prostatic obstruction
DHIC	detrusor hyperactivity with impaired contractile function
DO	detrusor overactivity
DOI	detrusor overactivity incontinence
EMG	electromyogram/electromyography
FDV	first desire to void
FSF	first sensation of filling
ICS	International Continence Society
IPSS	International Prostate Symptom Score
ISD	intrinsic sphincter deficiency
LPP	leak point pressure
LUT	lower urinary tract
LUTS	lower urinary tract symptoms
Max Cap	maximum cystometric capacity
MRI	magnetic resonance imaging
MUP	maximum urethral closure pressure
NDV	normal desire to void
NPV	negative predictive value (see section C.IV.1)
OAB	overactive bladder syndrome/symptoms
POP	pelvic organ prolapse
PPV	positive predictive value (see section C.IV.1)
PVR	post-void residual urine (volume)
SD	standard deviation
SDV	strong desire to void
SEM	standard error of the mean
SPECT	single photon emission computed tomography
SPT	specificity (see section C.IV.1)
STV	sensitivity (see section C.IV.1)
TURP	transurethral resection of the prostate
TVT	tension-free vaginal tape
USI	urodynamic stress incontinence
VLPP	Valsalva leak point pressure

Dynamic Testing

D. Griffiths, A. Kondo

S. Bauer, N. Diamant, Limin Liao, G. Lose, W. Schäfer, N. Yoshimura

H. Palmtag

A. INTRODUCTION

I. GENERAL REMARKS

This chapter on "Dynamic Testing" is the successor to the chapters on "Urodynamic Testing" in the two previous consultations. [1,2] The name change reflects the inclusion of a new topic, faecal (or anal) incontinence. In the section on faecal incontinence, the available functional tests, their applications in various groups of patients and types of faecal incontinence, and the evidence for their clinical utility, are reviewed in as much detail as is practicable. For urinary incontinence, reviews of this sort appeared in the two previous consultations. [1,2] In this 3rd consultation we have updated the evidence for the reproducibility and reliability of urodynamic measurements, and the evidence for their clinical utility. The primary aim of the chapter however is to reassess the functional and anatomic mechanisms of urinary continence, to discuss what urodynamic tests ought to be performed to elucidate these mechanisms; and to make recommendations for what tests should be performed and when. Thus we have tried to present a critical view of the role of urodynamics, the current state of urodynamic assessment, and recommendations for the future.

In this chapter, therefore, faecal (anal) and urinary incontinence are addressed rather differently. Yet in fact the 2 topics clearly have much in common, in regard to both pathophysiological mechanisms and clinical application. In future consultations we expect a more integrated approach to emerge and yield fruitful insights.

Following this introduction, and an explanation of the definitions of the various types of urinary incontinence and their corresponding urodynamic observations, the chapter contains a fundamental discussion of the two main types of urinary incontinence (stress and urge), a review of data about normal values and reliability of urodynamic parameters, reviews of the literature regarding clinical urodynamic evaluation of different patient groups with urinary incontinence (women, men, children, neurogenic dysfunction, and the frail elderly), a section on dynamic testing in faecal incontinence, and finally the committee's recommendations regarding dynamic testing.

II. HISTORICAL BACKGROUND

1. What is urodynamics?

The conventional view – implicitly adopted in the previous consultations – is that urodynamics is a series of more or less agreed-upon clinical tests, such as filling cystometry and pressure-flow studies, and that all that remains to be done is to establish which tests have clinical utility and in what circumstances. Urodynamics is defined by the International Continence Society (ICS) however as the study of the function and dysfunction of the urinary tract ***by any appropriate method*** [emphasis added]. [3] According to this definition, urodynamics is the only way of understanding why people are continent or incontinent, because the attempt to gain that understanding is what constitutes urodynamics. Therefore urodynamics occupies a central place in this consultation. It is the pivotal link between basic science on the one hand and clinical reality on the other. It can answer questions such as: How does the normal person stay

continent? What really goes wrong when they become incontinent? These questions can be posed as part of clinical or animal research, but they are important in individual patients too. Conventional urodynamic testing may not be necessary in every patient, but making the appropriate measurements remains the only way of knowing what urinary tract function is. Treatment that is not carried out blindfolded but is based on knowledge requires urodynamics.

2. What has urodynamics accomplished?

Since urodynamics entered clinical consciousness 40 years ago, its main achievement has been to focus attention on neuromuscular function and dysfunction, especially as embodied in the concept of detrusor overactivity. To a considerable extent this has been useful. For example, it is now universally recognized that stress incontinence is not the only possible reason for urine loss in an otherwise healthy middle-aged female, and that surgery is not the only treatment. Drug and behavioral treatments, aimed at correction of function, have been greatly enhanced, as demonstrated in the chapters on pharmacotherapy and conservative management in this book. A less desirable consequence has been that attention has been directed away from gross anatomy and structural issues, so that understanding of the anatomical and mechanical basis of stress incontinence has advanced very slowly. In spite of the deeper understanding of dysfunction provided by urodynamics, numerous studies have demonstrated only weak correlations between symptoms and urodynamic findings, and there has been little objective evidence that doing urodynamics – and selecting treatment options on this basis – improves clinical outcomes. In the previous consultation, accordingly, the evidence for clinical utility was judged not particularly cogent, and a rather limited clinical place was suggested for urodynamic testing, especially in women. [1]

Main recommendations for urodynamics in clinical practice according to the previous Consultation (ICI 2000) [1]:

- Investigation should only be performed in women if voiding difficulty or neuropathy is suspected, if previous surgical or non-surgical treatments have failed, or if invasive or surgical treatments are considered.
- In men and children, detailed urodynamic investigation should be undertaken.

3. What should be the role of urodynamics in clinical practice?

There is general agreement among experts that the immediate aim of urodynamic testing is to reproduce the symptom(s) of the patient under controlled and measurable conditions, so that the cause of the symptoms can be determined. Certainly, if the symptom is not reproduced the test can be regarded as a failure (although failure to understand the symptoms may also contribute). Conceptually, reproduction of the symptom allows diagnosis, helps inform treatment choice, and improves treatment outcome.

Urodynamic studies provide an objective description of lower urinary tract function and dysfunction in terms of qualitative and quantitative variables. Thus ideally they should provide objective information useful for the clinician:

a) to identify or to rule out factors contributing to the incontinence and assess their relative importance

b) to obtain information about other aspects of lower urinary tract dysfunction

c) to predict the consequences of lower urinary tract dysfunction for the upper urinary tract

d) to predict the outcome, including undesirable side effects, of a contemplated treatment

e) to confirm the effects of intervention or understand the mode of action of a particular type of treatment, especially a new one

f) to understand the reasons for failure of previous treatments for incontinence

Urodynamic studies should be performed and reported in accordance with the standards of the International Continence Society [4] to optimize interpretation and facilitate comparison between different studies.

The conventional view is that urodynamic investigation ought to provide a gold standard for assessing incontinence. However there is still a pressing need scientifically to conceptualize continence function in terms which can be measured urodynamically. Ideally the chosen variables should fulfill several requirements: 1) measurements should be standardized; 2) results should be reproducible; 3) parameters for health and disease should be specific and sensitive with clear cut-off levels and without too much overlap, so as to give clinical useable predictive values; 4) parameters should contribute to choice of therapy; and 5) parameters should be correlated with the out-

come of therapy for the disease. [5] To what extent these ideal requirements can in fact be fulfilled is considered in this chapter.

Evidence-based medicine is founded on the assessment of evidence for and against the efficacy of particular types of therapeutic intervention. Obviously there is a temptation to try to apply the same rules of evidence to clinical tests such as urodynamics, because evidence that testing improves outcome (through treatment choice and patient selection) would provide a strong basis for its use. However, testing and therapeutic intervention are different concepts, and urodynamic testing has another important objective, which is not applicable to interventions and lies outside the scope of evidence-based medicine. It is to generate knowledge about the condition(s) to be treated in a given patient, so that the practitioner can formulate rational treatment options based on knowledge rather than work blindfold; that is, he or she can practice "knowledge-based medicine".

To judge the importance of this second objective different criteria are needed. Indeed, reproduction of the symptom is too narrow and too limited an aim for urodynamic testing if this objective is to be fulfilled. Particularly in the referral setting, the physician is confronted with complicated cases in whom the underlying pathophysiology is uncertain, and what is required is not just to reproduce the symptom, but to identify all the factors that may be contributing. Thus a comprehensive evaluation of the function and dysfunction of the lower urinary tract is needed. Urodynamics in children with congenital urinary tract problems provides a good example: pathophysiology is variable and unpredictable, and some type of treatment is mandatory, yet often irreversible. Any available information about the baseline dysfunction before treatment decisions are made is helpful, and it would be unethical to neglect to gather it. Urodynamics is necessary because it contributes to "knowledge-based medicine", whether or not there is narrowly-defined "evidence" that it improves outcomes.

Of course, it remains true that we should seek evidence of the conventional kind for and against testing. The co-sponsor of this consultation (the ICUD) recommends that, as a minimum, any test should be subjected to three questions:

1. Does the test have good technical performance, for example, do three aliquots of the same urine sample give the same result when subjected to 'stix' testing?
2. Does the test have good diagnostic performance, ideally against a "gold standard" measure?
3. Does the test have good therapeutic performance, that is, does the use of the test alter clinical management, does the use of the test improve outcome?

It is revealing that the example chosen for question 1 is a particularly simple one where the result can be checked against other methods of measuring the same thing. Question 2 begs a deeper question: In measuring urethral/vesical function, how can there be any gold standard other than the measurements themselves? Question 3 is relevant in the context of evidence-based medicine, and in this chapter we shall address it.

III. THIS CHAPTER

A recent Cochrane review [6] attempted to test the hypotheses that:

- Urodynamic investigations improve the clinical outcomes of incontinence management
- Urodynamic investigations alter clinical decision-making
- One type of urodynamic test is better than another in improving the outcomes of incontinence management and/or influencing clinical decisions.

The authors found only 2 relatively small randomized trials (both in women) that tested one or more of these hypotheses. [7,8] They concluded that there was not enough evidence to show whether women with incontinence who underwent urodynamics were less likely to be incontinent after treatment than women who did not undergo urodynamic testing. They recommended further randomized trials in all types of patients whose incontinence might be investigated with urodynamics, estimating that such trials would need about 400 patients in each arm to provide 80% power to detect a 10% difference in incontinence rates at a significance level of 5%.

At present therefore there is limited objective evidence for the clinical utility of urodynamics, and a pressing need to examine this question. If future studies confirm that urodynamics has limited effect on outcomes or decision-making, however, this would imply that it is not important to understand how the lower urinary tract works in order to treat it with current methods. Such a surprising conclusion could have only a few possible explanations:

a. All the patients in any given symptom group (e.g. women with stress incontinence) have similar underlying pathophysiology requiring similar treatment, and so urodynamics cannot classify them any better than the symptoms alone.

b. Conventional urodynamics does not address the important pathophysiological differences that affect treatment success – i.e., we are doing the wrong tests.

c. We are doing the right tests, but we are doing them so poorly (technically) that the results are unreliable.

d. Current treatments are so non-specific and non-quantitative that the underlying dysfunction is unimportant: treatment works equally well or poorly in any case.

Comments:

- The studies that show that symptoms and urodynamic findings do not correspond very well [1] make possibility "a" unlikely, because they show that similar symptoms have different underlying pathophysiology as revealed by urodynamics. Moreover, we think we know that detrusor overactivity and urge and stress incontinence each come in multiple types.
- There is strong suspicion, based on expert opinion, that possibility "c" is correct – that urodynamics is often done poorly; the International Continence Society and other bodies are improving this situation by establishing standards for proper training and certificate programs in urodynamics. [9]
- Possibility "b" is a likely explanation: the conventional urodynamic testing that we do is inadequate by itself to unravel the pathophysiological behavior we are interested in.
- Possibility "d" is also likely to be correct: because of our lack of understanding, current treatments are not aimed accurately at the right target.

Some of the mismatch between symptoms and urodynamic findings is due to inherent physiological variability in the function of the lower urinary tract, which is reflected in the urodynamics (as well as in the variability of symptoms). For example, post-void residual urine in the elderly [10] shows a diurnal variation that implies that there are systematic changes in some unknown controlling parameter. Similarly, analysis of the variability of voiding studies in adult males reveals an unknown source of variation that affects detrusor contractility and urethral resistance simultaneously. [11] Even more revealingly, normal subjects and near-normal patients often find it difficult to reproduce their usual behavior – for example to void, or demonstrate incontinence – under the artificial circumstances of a urodynamic test, or they require a particular type of provocation such as hearing running water, to elicit it. This physiological variability of urodynamic results shows that – in neurologically intact subjects – they are not just measurements of the fixed properties of a mechanical system. Physiological variability is not an indication of unreliability; it is a promise that there are neurological or cortical sources of control that we have hardly begun to imagine. Identification of these sources offers the prospect of entirely new therapies.

Possibilities "b" and "d" both imply that the mismatch between urodynamic findings on the one hand and symptoms, treatment selection, and outcomes on the other reflects gaps in our understanding of what is wrong and how it should be corrected. In fact, there are obvious omissions in conventional urodynamics. Attention is focused on the accessible end organs (the bladder and urethra) and the easy measurements (pressure, volume and flow rate), leaving large gaps in what is studied:

a. The urethra and pelvic floor form a complicated structure whose mechanics is critical to continence under stress conditions, yet it is currently understood in only a most rudimentary way. [12]

b. The bladder and urethra are just the tip of the iceberg of a complicated nervous control system that can go wrong at any level. Any abnormality, from cerebral cortex to brainstem, to spinal cord, right down to the end organs, may lead to symptoms such as urgency, frequency or incontinence. Impairment of cortical control is obviously important but almost entirely ignored.

Filling in these gaps will deepen our understanding of the pathophysiology of urinary incontinence, improve our understanding of patients' symptoms, and possibly lead to new treatments. However, it will also require new or improved types of urodynamic study. One of the aims of this chapter is to discuss what types of study ought to be done in this new urodynamics.

Finally, evidence-based medicine as currently conceived is only one aspect of good medicine. Another aspect is understanding of function and dysfunction – knowledge-based medicine – and it depends on urodynamics. In each patient group the relative importance of evidence-based and knowledge-based medicine differs and must be established.

IV. DEFINITIONS AND TERMINOLOGY

One of the main aims of a urodynamic test – although not the only aim – is to reproduce the symptom. Standards have been set out to define symptoms and to define urodynamic observations, but it not always clear what urodynamic observation reproduces what symptom. Here we attempt to clarify the situation.

1. DEFINITIONS

The following definitions are taken from the standardized terminology of the International Continence Society. [4] Comments are bulleted. The standardisation reports of the International Continence Society should be consulted for definitions not included here. [3,4]

Urinary incontinence is the complaint of any involuntary leakage of urine.

- In each specific circumstance, urinary incontinence should be further described by specifying relevant factors such as type, frequency, severity, precipitating factors, social impact, effect on hygiene and quality of life, the measures used to contain the leakage and whether or not the individual seeks or desires help because of urinary incontinence. For the purpose of urodynamics, the type of incontinence is the most relevant factor.
- Each type of incontinence may be described as a symptom, a sign, or a urodynamic observation.

Detrusor overactivity is a urodynamic observation characterised by involuntary detrusor contractions during the filling phase which may be spontaneous or provoked.

- Although we will use this term, we believe it is misleading to call detrusor activity observed during the filling phase "overactivity", since it is observed also in healthy volunteers (see section B.II, urge incontinence).

"Urethral instability": Fluctuations in urethral pressure have been defined as the "unstable urethra". However, the significance of the fluctuations and the term itself lack clarity and the term is not now recommended by the International Continence Society

- If symptoms are seen in association with a decrease in urethral pressure a full description should be given.
- It is misleading to term urethral pressure changes observed during the filling phase "urethral instability", since the term seems to imply dysfunction when the behavior may be entirely normal.

Stress urinary incontinence denotes a symptom : the complaint of involuntary leakage of urine on effort or exertion, or on sneezing or coughing.

The corresponding sign of **stress urinary incontinence** is the observation of involuntary leakage from the urethra, synchronous with exertion/effort, or sneezing or coughing, i.e. a positive stress test.

The corresponding urodynamic observation is **urodynamic stress incontinence**, which is the involuntary leakage of urine during increased abdominal pressure, in the absence of a detrusor contraction, noted during filling cystometry.

- This means the demonstration of leakage on abdominal pressure increase due to moving, straining or coughing, without any effect on the detrusor pressure signal. This requires careful control of signal quality.

Urge urinary incontinence is the complaint of involuntary leakage accompanied by or immediately preceded by urgency.

The corresponding sign has not been clearly defined.

- This is partly because urge incontinence can present in different symptomatic forms; for example, frequent small losses between micturitions or catastrophic, complete bladder emptying; and partly because urge incontinence is unpredictable and difficult to elicit in a physician's office.

The corresponding urodynamic observation is **detrusor overactivity incontinence**: incontinence due to an involuntary detrusor contraction.

Urethral relaxation incontinence, defined as leakage due to urethral relaxation in the absence of raised abdominal pressure or detrusor overactivity, might also correspond to the symptom urge incontinence.

- The sensation accompanying detrusor overactivity incontinence or urethral relaxation incontinence should be noted, as if there is none, this could not strictly speaking be said to reproduce the symptom ***urge*** incontinence.

Urgency is both a symptom and a urodynamic observation.

The symptom of **urgency** is the complaint of a sudden compelling desire to pass urine which is difficult to defer.

- It may accompany urge incontinence (see above), or may occur with other overactive bladder symptoms (see below) or in isolation.

Urgency as a urodynamic observation is a sudden compelling desire to void**,** noted during filling cystometry.

- Urgency may be associated with leakage (detrusor overactivity incontinence), or with detrusor overactivity, or with neither. It is frequently observed without any demonstrable detrusor overactivity.
- One might expect a relation between urethral pressure changes and the sensation of urgency. However, there seem to be no publications showing such a relation.
- Urgency, with or without urge incontinence, is often associated with frequency and/or nocturia. The terms **overactive bladder syndrome**, **urge syndrome** or **urgency-frequency syndrome** describe symptom combinations that are suggestive of urodynamically demonstrable detrusor overactivity but may be due to other forms of urethral/vesical dysfunction. These terms are used if there is no proven infection or other obvious pathology.

Mixed urinary incontinence denotes a symptom : the complaint of involuntary leakage associated with urgency and also with exertion, effort, sneezing or coughing.

- This ICS definition leaves it open as to whether both forms of incontinence occur (a) simultaneously or (b) at different times. Possibility (b) seems to be more common.
- In publications it sometimes appears that mixed incontinence is assumed to include the symptom of stress incontinence, together with complaints of urgency and frequency not related to the incontinence episodes. This is not mixed incontinence as defined by the International Continence Society and use of the term in this way should be avoided.

The sign corresponding to mixed incontinence is not clear.

The corresponding urodynamic observations include separate demonstration of urodynamic stress incontinence and detrusor overactivity incontinence in a single urodynamic testing session.

- The picture becomes complicated if detrusor overactivity is provoked, e.g. by coughing, and leakage ensues. Nevertheless it may reproduce possibility (b).
- It is sometimes assumed that the observation of urodynamic stress incontinence and (separately) detrusor overactivity reproduces the symptom of mixed incontinence. This is not in conformity with the definition.
- Clinical reports show that standard stress incontinence surgery can cure “mixed incontinence” but also can lead to new symptoms of urgency. However, as neither the symptom definitions used nor their urodynamic correlates are very clear, urodynamic objectification of symptomatic mixed urinary incontinence is difficult, and these reports remain difficult to interpret.

Increased daytime frequency is the complaint by the patient who considers that he/she voids too often by day.

This term is equivalent to the term pollakisuria, used in many countries.

The corresponding sign is not clear.

The corresponding urodynamic observation might be detrusor overactivity; alternatively a small bladder capacity, with or without detrusor overactivity, might account for the symptom. (Note however that bladder capacity is a variable measurement, see section B.II, urge incontinence.) Increased diuresis, as shown by a bladder diary or voiding record, might also account for increased frequency.

2. Further comments

Differentiation between urodynamic stress incontinence and detrusor overactivity incontinence is simple if clear detrusor overactivity can be demonstrated without physical activity and with the sensation of urgency; or if leakage occurs on stress with no hint of any detrusor overactivity. However, in many cases the correlation between symptoms and urodynamic observations is less clear.

Table 1 sets out for the main symptoms the principal corresponding urodynamic observations. Note that this table does not imply that the cited urodynamic observation is always made with the given symptom; urodynamic tests often reveal unexpected subtleties. For definitions and commentary see main text.

Table 1. Symptoms and corresponding urodynamic observations

Symptom	Urodynamic observation
Stress incontinence	Urodynamic stress incontinence
Urge incontinence	Detrusor overactivity incontinence with urgency *or* Urethral relaxation incontinence with urgency
Mixed incontinence	Urodynamic stress incontinence (USI) + detrusor overactivity incontinence (separately) *Not recommended:* USI + detrusor overactivity
Urgency	Detrusor overactivity with sensation *or* other relevant urethral and/or vesical dysfunction (e.g. increased sensation)
Frequency	Detrusor overactivity *or* other relevant urethral or vesical dysfunction (e.g. small bladder capacity or increased diuresis)

B. MECHANISMS OF URINARY CONTINENCE AND INCONTINENCE

I. STRESS INCONTINENCE

1. Stress Urinary Incontinence

According to the standardised terminology of the International Continence Society, the symptom of stress urinary incontinence is the complaint of involuntary leakage of urine on effort or exertion, or on sneezing or coughing. [4] Its relation to signs and urodynamic observations is considered in the introductory section A.IV, Definitions and terminology. In this chapter we will also use the following urodynamically based definition of stress incontinence: Stress incontinence occurs when the bladder closure mechanism is competent (= continent) at rest, but becomes incompetent under stress.

The key to the pathophysiology of stress incontinence lies in understanding the impact of abdominal pressure increase ("stress") on the closure mechanism of the bladder outlet. Urodynamic measurements of the function of the bladder closure mechanism during stress are difficult, and most existing data are reported with inadequate control of data quality, or none at all. In considering any changes observed in the bladder closure mechanism and pelvic floor, or the impact of stress on these structures, the inherent limitations on measurement quality should be borne in mind. In general it is possible to verify stress incontinence urodynamically, but it is difficult to exclude it urodynamically.

• Mixed urinary incontinence

This is discussed in section A.IV, Definitions and terminology.

2. Normal continence mechanisms

The simple urodynamic rule for continence is that as long as the urethral pressure is higher than the intravesical pressure, i.e. the urethral closure pressure is positive, incontinence cannot occur. Active and passive mechanisms play a role in maintaining continence. Sustained tone of the circularly-arranged smooth and striated urogenital sphincter muscles provides the resting closure pressure. The striated muscle within the proximal and mid-urethral regions is generally referred to as the striated sphincter, the external urethral sphincter, or the rhabdosphincter. In cooperation with the circular smooth muscle, the external urethral sphincter mechanism as a whole comprises nearly the proximal two-thirds of the urethra. [13] The requirement for equilibrium of forces suggests that the longitudinal smooth muscle is also active during urine storage (not during voiding, as often suggested), so as to prevent tissue from being squeezed out from the high-pressure zone under the striated sphincter; and that it forms an integral part of the closure mechanism, although it contributes to the closure pressure only indirectly. [12] Any pressure signal recorded in the striated sphincter is influenced by the surrounding abdominal contents and the pelvic floor muscle. The fact that the striated sphincter and the periurethral skeletal muscle of the pelvic floor differ histomorphologically and are physically separate does not mean that that they do not work together mechanically; in fact, the periurethral skeletal muscle of the pelvic floor seems to correspond to the zone where urethral closure pressure is highest (see however Figure 1 for a different point of view). Thus one must assume a mechanical continuity between sphincter and surrounding pelvic floor so that it is not possible to distinguish by urodynamic pres-

sure measurements between the individual contributions of the sphincter and the pelvic floor muscle. [12]

Urinary continence is thought to be maintained during elevation of abdominal pressure by multiple mechanisms: the same sustained closure mechanism that also acts during resting conditions; passive pressure transmission within the abdominal pressure region;[13] and extra closure forces generated by active contraction of the sphincter and the pelvic floor. Because urethral pressure recording cannot identify the origin of any pressure increase, and because published human data appear not to be of high enough quality to exclude an artifactual "pressure" increase due to catheter movement within the urethra, or to the forces associated with bending or acceleration of the catheter, quantification of any active increase in urethral pressure is difficult.

The transmission of abdominal pressure to the proximal urethra is considered to be an important mechanism of continence. However, the term "transmission" should not be taken literally in the sense that the pressure is generated somewhere else (e.g. in the upper abdomen) and transmitted to the proximal urethra. Much more realistic is the concept of an abdominal pressure region in all parts of which the abdominal pressure increases or decreases simultaneously. For an increase in abdominal pressure to occur, the muscles which bound this region (the abdominal and pelvic muscle sheets and the diaphragm) must develop tension. To generate this tension, muscle contraction must start before the abdominal pressure increases measurably.

Simple mechanical considerations show that as long the abdominal pressure increase affects the bladder and urethra similarly, the pressure balance maintaining continence is not altered and stress incontinence cannot occur. Although this simplistic concept is valid in principle, it is obviously not sufficient to explain the mechanics of stress incontinence, as is clear from the failure of a variety of surgical concepts, which have attempted to elevate the bladder neck back into the abdominal pressure region, so as to fully expose it to "abdominal pressure transmission". [14]

Another possible mechanism for maintaining continence is neurally-mediated urethral closure under stress conditions. This concept is based on various clinical observations combined with urodynamic measurements, including: (i) the fact that the urethral pressure rises prior to cough transmission, [15,16] (ii) the fact that the increase in urethral pressure during coughing exceeds the increase in bladder pressure, [15,17-19] and (iii) the fact that bilateral pudendal nerve blockade reduces urethral closure forces during coughing. [20] However, recording of local urethral pressures under stress is subject to artifacts caused by transducer movement along the urethral axis, or by interaction with the urethral wall due to deformation or acceleration. In fact, Constantinou and coworkers [18] emphasized the directionality of the recorded signals, so making it clear that they were not recording a closure pressure; therefore the interpretation of the reported urodynamic observations is questionable.

Nevertheless, despite these criticisms, the concept of an active, neurally-mediated urethral closure mechanism operating under stress conditions may still be correct. A recent study in rats using microtip transducer catheters and nerve transection techniques has shown that urethral contractile responses during sneezing, which did not disappear after abdominal opening (and therefore cannot have been caused by "abdominal pressure transmission") were suppressed by transection of somatic nerves innervating the striated urethral sphincter and pelvic floor muscles. [21] These apparently active contractile responses were most prominent in the middle third of the urethra, at the location of the striated sphincter and pelvic floor muscles. At the bladder neck, on the contrary, the urethral pressure changes on sneezing were very similar to those in the bladder, and were abolished on opening the abdomen, suggesting that "abdominal pressure transmission" does play an important role in bladder neck closure. This work supports the view of Turner-Warwick that there are 2 urethral closure mechanisms, a proximal and a distal one (**Figure 1**). [22,23] According to this view, the proximal mechanism relies on abdominal pressure transmission to the bladder neck, which is maintained closed by sustained smooth muscle contraction. The distal mechanism relies on changes in the pelvic floor and striated sphincter, which have active as well as passive components. Only if both mechanisms are faulty does the patient become stress-incontinent (Figure 1).

The "pressure" increase recorded in the urethra prior to any abdominal pressure increase has been termed the "guarding reflex". [18] However, contraction of the muscles surrounding the abdominal pressure region is an essential part of any activity which is associated with an abdominal pressure increase, irrespective of continence function. Muscle contraction, as recorded for example in the striated sphincter zone, may therefore precede the abdominal pressure increase.

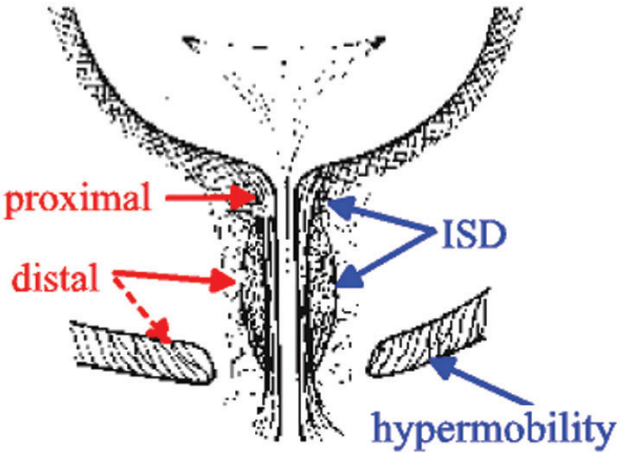

Figure 1. Concepts of female (in)continence mechanisms. On left in red, according to Turner-Warwick [22] the proximal continence mechanism is formed by the smooth muscle of the bladder neck; the distal mechanism is formed by the striated and smooth muscle of the mid-urethra, together with (less certainly) the muscles of the pelvic floor (levator ani). On the right in blue, intrinsic sphincter deficiency (ISD) implies weakness of the smooth and striated muscles of the proximal and mid-urethra; hypermobility implies weakness of the striated muscles of the pelvic floor.

3. What goes wrong? How abnormality leads to incontinence

Differences in anatomy, direct physical insult from childbirthing events, and hormonal changes associated with pregnancy, aging and menopause, leading to alterations in the distribution of neuronal receptors, have all been suggested as contributors to the prevalence of incontinence in an aging female population. [24,25] DeLancey et al have also shown that approximately 60% of patients with prolapse but only 30- 40% of incontinent females have a significant levator damage. [26]

Stress urinary incontinence is conventionally subdivided into two categories; (1) anatomic incontinence (type II incontinence or urethral hypermobility) and (2) intrinsic sphincter deficiency (type III incontinence, or a poorly functioning but better supported urethral sphincter). [27,28] From the point of view of our definition - that stress incontinence overcomes a closure mechanism which is competent at rest - then this distinction seems to be either trivial or artificial. Obviously it takes less stress to overcome a weak urethral closure mechanism, and it is also obvious that greater mobility indicates a weaker pelvic floor which will allow more deformation. The data show - as is to be expected - that sphincter function, as represented by urethral closure pressure, is widely overlapping between continent and incontinent patients. [5] There are no convincing data about mobility in normal or incontinent females, but it seems clear that stress urinary incontinence will always be caused by some combination of these factors. [29]

The dichotomy between intrinsic sphincter deficiency and urethral hypermobility is a modern reinterpretation of the 2 continence mechanisms suggested by Turner-Warwick [22] (see Figure 1). Impairment of the proximal mechanism means that the bladder neck is open at rest, and thus is related to intrinsic sphincter deficiency (albeit in the proximal urethra only). Impairment of the distal mechanism means that there is striated-sphincter/pelvic-floor weakness or lack of muscle activity. Pelvic floor weakness implies hypermobility (at least in women). Impairment of both mechanisms seems to be a requirement for incontinence. In women this implies a combination of intrinsic sphincter deficiency and hypermobility.

In spite of the preceding criticism, a low closure pressure may have prognostic value for surgery, as surgery primarily modifies the mobility by providing additional suspension, but does not improve sphincter function. It seems clear that in cases with a very weak sphincter, - i.e. where it needs less “stress” and mobility to cause stress incontinence - surgery will be less successful. Usually these cases show clinically more severe stress incontinence. [30]

As stress incontinence appears to be caused by mechanical changes in active and passive tissues, and most urodynamic techniques measure mechanical parameters, the remainder of this section will focus on the mechanics of continence and stress incontinence. Indeed, stress incontinence can be treated successfully by modifying the mechanics of the bladder closure, either by improving active function through muscle training or electrostimulation, or else by adding a passive suspending structure such as TVT (tension-free vaginal tape), which alters mobility and deformation of the bladder closure mechanism but does not normalize the physiological mechanics.

4. Urodynamics and stress incontinence

The preceding discussion shows that our current understanding of the pathophysiology and urodynamics of stress incontinence is limited. Therefore it appears reasonable to accept for the moment that the

diagnosis and treatment of stress urinary incontinence should follow a pragmatic path based primarily on clinical experience rather than urodynamic measurements. Nevertheless, we should view this current practice critically, even though it seems to be reasonably successful. Diagnoses of stress incontinence that rely on symptoms and signs alone, and the indications for surgery drawn from them, are not based on *knowledge* of the details of the pathophysiology. Correspondingly, treatment is not aimed at restoration of physiological function. Diagnostic imaging such as sonography offers some insight into the changes that are introduced by surgery, e.g. by TVT. [31,32] The functional interpretation of these observations is more speculative and descriptive than systematic and scientific. However, the fact that these functional interpretations are not supported by consistent urodynamic data does not cast doubt on the observations but rather emphasizes the problems with current urodynamic measurement during stress. It is difficult to imagine that a catheter fixed outside the body could follow the remarkable deformations observed during Valsalva, [32] without the point of measurement shifting or the urethra kinking and influencing the measurement.

Taking into account the preceding discussion, the following questions will be addressed:

Question 1.	What is required for current routine practice?
Question 2.	What should be done urodynamically to improve current clinical practice in diagnosing and treating stress incontinence?
Question 3.	What should be done urodynamically to further our understanding of the pathophysiology of stress incontinence and allow us to distinguish different defects and combinations of defects that may lead to different forms of stress incontinence?
Question 4.	What could be done urodynamically to develop methods of repairing specific defects and restoring physiological continence function?

a) What is required for current routine practice? *(Question 1)*

All urodynamics should follow the principles of good urodynamic practice. However, the idealistic aim of reproducing symptoms while making precise measurements in order to identify the underlying causes and to quantify the related pathophysiological processes, [33] is not yet fully applicable in stress incontinence. It is not difficult to support a symptomatic diagnosis of stress incontinence urodynamically, but it is not clear that such a confirmation is needed. The additional capability of urodynamics to quantify parameters such as urethral pressure is also not of any clinical advantage as current treatment is not quantitative, and does not need quantitative information. Thus, invasive urodynamics is probably not needed in the diagnosis of uncomplicated stress incontinence in a routine practice of high standard which includes, in addition to a careful dedicated history and physical examination:

- a positive stress test;
- a voiding diary showing normal bladder capacity > 300 ml and no symptoms of urgency or urge incontinence;
- a normal voiding function by diary and uroflowmetry, as documented by maximum flow rate, together with voided volume and post-void residual urine volume. [33]

Urodynamics may however be helpful for prognosis and patient counselling. The evidence for these views is discussed in section D.I, Evaluation of the female patient.

b) What should be done urodynamically to improve current clinical practice in diagnosing and treating stress incontinence? *(Question 2)*

As the success rate in the treatment of ***uncomplicated*** stress incontinence seems to approach 100% [34] the focus of urodynamics should be on making sure that the stress incontinence is indeed uncomplicated, or on identifying the complicating factors. This is important for patients with previous surgery; with voiding problems; with suspected obstruction or weak detrusor contraction; with reduced bladder capacity; without a positive stress test; with urge or mixed urinary incontinence, particularly with suspected detrusor overactivity incontinence; or with severe forms of stress incontinence. Carefully performed urodynamics can give information relevant to all of these aspects. Because these potential complicating factors, as well as the quality of urodynamics, differ greatly among patients and among centres, it is not surprising that no sound basis of evidence for the use of pre-operative urodynamics has

yet been established. Nevertheless, it is the committee's opinion that the knowledge provided by carefully performed urodynamics is of advantage to an experienced surgeon in all forms of ***complicated*** stress incontinence.

c) What should be done urodynamically to further our understanding of the pathophysiology of stress incontinence, and allow us to distinguish different defects and combinations of defects, which may lead to different forms of stress incontinence? *(Question 3)*

According to our urodynamic definition, stress incontinence occurs when the bladder closure mechanism is competent (= continent) at rest, but becomes incompetent under stress. ***At rest,*** we can measure the urethral closure function quite accurately in terms of urethral closure pressure, keeping in mind that it depends on location within the urethra (among other factors) and can vary with time. But the measurements are not sufficient to characterize incontinence ***under stress.*** Thus we would not expect any single urodynamic parameter measured at rest to be specific or diagnostic for stress incontinence, i.e. to be clearly different in women with and without stress incontinence. Clearly, as stress incontinence only occurs during stress, we should make measurements under stress, but this is difficult with current techniques.

1. Resting conditions

At rest, the biomechanics of the closure function of a tubular structure such as the urethra is quite simple. It requires folding of the soft tissue tube and pressing of the inner lining by a concentric contraction. [12] (The term "pressing" is preferable to the usual term "compressing," which misleadingly may suggest that tissue is compressed to a smaller volume, while tissue in fact is incompressible. [12]) The best parameter to measure closure function is the pressure in the lumen as it just closes or opens. [35] Although technically difficult, this is possible and there is no other meaningful parameter available.

Urethral pressure recording requires a catheter to be placed in the urethra, inevitably distending the urethra to the catheter's diameter. Therefore the catheter should be as thin as possible. Any catheter will have some stiffness and weight which will deform the curved, soft urethra. Therefore the catheter should be as flexible and light as possible. As the urethra has significant pressure gradients along its length, the location of the recorded pressure must be known.

For a ***urethral pressure profile*** (UPP), the pressure along the urethra is recorded at rest during a given period of time, typically about 40 seconds with a withdrawal speed of 1 mm/s, and thus combines information about pressure, location and time variation in a single graph. [1] From this graph the total (anatomic) urethral length, the functional length or continence zone, and the urethral closure pressures can be obtained. One specific aspect of the UPP is the maximum urethral closure pressure (MUCP), which is the highest pressure (relative to bladder pressure) generated along the functional length of the urethra. It usually corresponds to the striated sphincter in the mid-urethra. [1] However, the profile depends on multiple factors[36] such as age, position of the patient, and filling volume. [37] Moreover, urethral pressure may vary with time in all locations, independently, synchronously, or even organized as peristaltic motility. Catheter movement and the direction of movement influence the result, and they depend on the catheter size, stiffness and surface friction. There are multiple other influences which depend on the method of pressure measurement. The perfusion technique appears to mimic best the condition for which urethral pressure is defined: a fluid pressure just opening the urethra. For such measurements it is possible to use small-diameter flexible catheters and low perfusion rates (about 1 ml/min under resting conditions).

The commonly used catheter-mounted microtransducers record a signal produced by interaction with the urethral wall. The sensing surface area of the transducer is small and only a minimal force is needed to yield a significant apparent pressure (1 gram weight acting on 1 mm^2 results in an apparent pressure of 100 cm H_2O). It is well documented that signals recorded with catheter-mounted microtransducers are directionally different, and thus cannot represent just urethral pressure. [38] They depend on the urethral deformation caused by the catheter's lack of flexibility and weight. For the urethra (and also for the anal canal) it is misleading to interpret such directional differences in terms of local "directional pressure" or any contribution to closure function. The directional differences exist only with the specific catheter in the urethra. In fact it is probably easiest to interpret the directional interactions if a rigid, thick catheter is used, [12,35,39,40] the exact opposite of the conditions best suited to making urethral pressure measurements.

The maximum urethral closure pressure alone does

not provide any information about the integrity of the bladder neck or proximal urethra (i.e. the proximal continence mechanism), and it can be highly variable as a result of involuntary contractions of the smooth and striated muscles of the urethral sphincter, perhaps provoked by the irritative nature of the catheter itself. The size, stiffness and type of catheter, rate of perfusion, patient position, and bladder volume, all have an effect on the pressure readings. A study by Wang and coworkers showed that the pressure measured by the perfusion method was 24.5 cm H_2O higher on average than that measured with a microtip catheter. However, pressure profiles measured with the two types of catheter had similar reproducibility. [41] Culligan and coworkers compared an 8 F microtip catheter with a 10 F fibre-optic catheter and found that the urethral pressures measured with the larger catheter were significantly greater, by 14 cm H_2O on average, but they found no differences between the values of the leak point pressures (see below) measured with the two catheters. [42]

Balloon catheters, either water-filled as used in early studies [43,44], or air-filled, as recently redeveloped under the name T-doc, have a limited spatial resolution along the length of the urethra, determined by the size of the balloon. The resolution appears however to be better than 5 mm for T-docs. These balloons should not have any directionality and record a true pressure (which may not be equal to the urethral pressure because of their finite diameter and potential for overinflation). Air- and water-filled balloons measure pressure from different reference levels, and this has to be allowed for. A further possibility is to measure urethral pressure with a microtransducer enclosed in a balloon. [45]

To summarise, urethral pressure recording in a resting patient is possible but quite difficult. Even with standardized methodology there is a variety of potential artifacts which should be considered critically when interpreting the results.

2. What happens to the urethral closure mechanism during stress?

The perfusion technique has a limited temporal resolution and is reliable only at rest, not during conditions of rapidly varying stress. Other techniques have better temporal resolution, but that does not mean that they show fewer artifacts, only different artifacts. In practice, it seems to be impossible to record a precise local pressure in a mobile urethra during stress, using a catheter. All aspects of urethra/catheter interaction increase under stress, and the site of pressure recording is difficult to determine because of the potential for uncontrollable shifting of the catheter during stress. Shifts of up to 8 mm have been reported at the bladder neck during coughing. [46] In theory, it should be possible to determine this shift by using multiple transducers along the catheter over the whole profile length, [47] but multiple-transducer catheters are of rather large diameter and quite rigid, and therefore yield directional force signals rather than urethral pressures.

• Urethral stress profile

A large number of publications deal with a great variety of methods for measuring and analyzing the so-called stress profile, i.e. a recording of a urethral pressure profile while the patient coughs at regular intervals.

The profile is a sequential presentation of time-varying local pressures, subject to the same limitations as discussed above, which are aggravated by additional problems. A popular form of quantification of the stress profile is the "pressure transmission ratio." (Note: as discussed above, pressure transmission is a misleading term.) Most published stress profiles show obvious artifacts. For example, one would expect a gradient in the response to cough in the midurethra, where both the abdominal pressure and the cough response fall to zero outside the abdominal pressure region. Nevertheless, many stress profiles show pressure spikes in the most distal urethra, even outside the functional urethral length (**Figure 2**)

Usually the response to cough is measured in the urethra and in the bladder and the transmission ratio is calculated by dividing the amplitude of the urethral pressure increase by the intravesical increase, expressing the result as a percentage. The typical transmission profile, which is the transmission ratio plotted against position in the urethra (expressed as a percentage of the urethral length), shows a decline from 100% pressure transmission at the bladder neck to 70% in the proximal urethra, followed by an increase to 85% or higher in the mid- or distal urethra. [48] This is often interpreted as evidence for an active contraction of sphincter and/or pelvic floor contraction during stress, but it can also be explained just by shifting of the catheter (**Figure 3**). [49] In practice it seems to be impossible to identify the origin of a higher or lower transmission ratio, and sophisticated interpretations are therefore questionable.

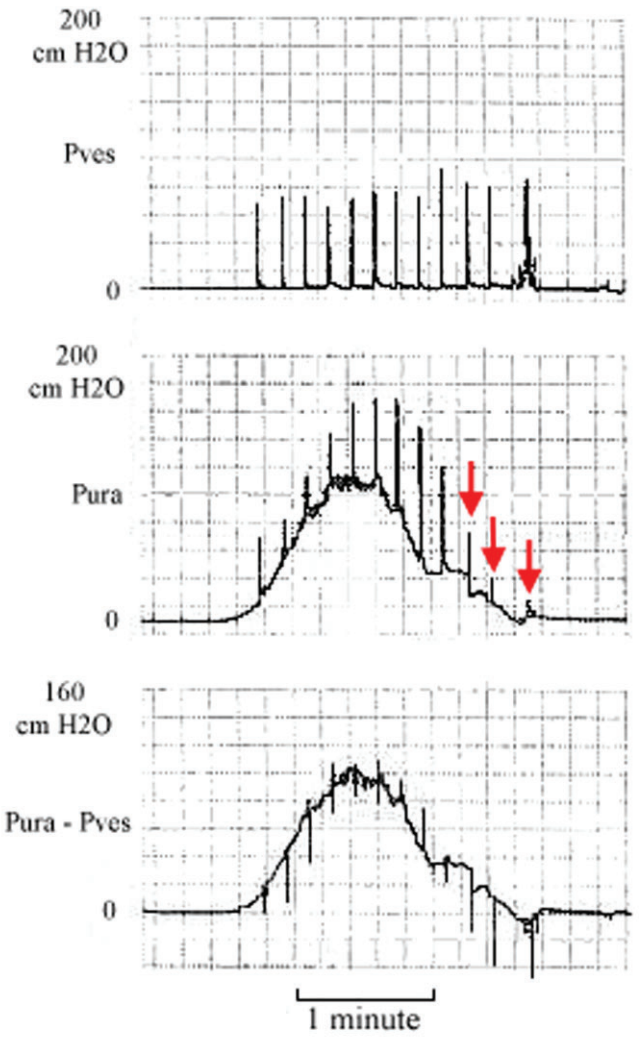

Figure 2. Example of a stress urethral pressure profile. The top trace shows the bladder responses to a series of coughs (spikes). The middle trace shows the corresponding urethral responses, recorded while the measuring catheter is slowly withdrawn out of the bladder, through the urethra, and into the atmosphere. Responses are seen even in the most distal part of the urethra (red arrows). The bottom trace shows the difference between the middle and top traces.

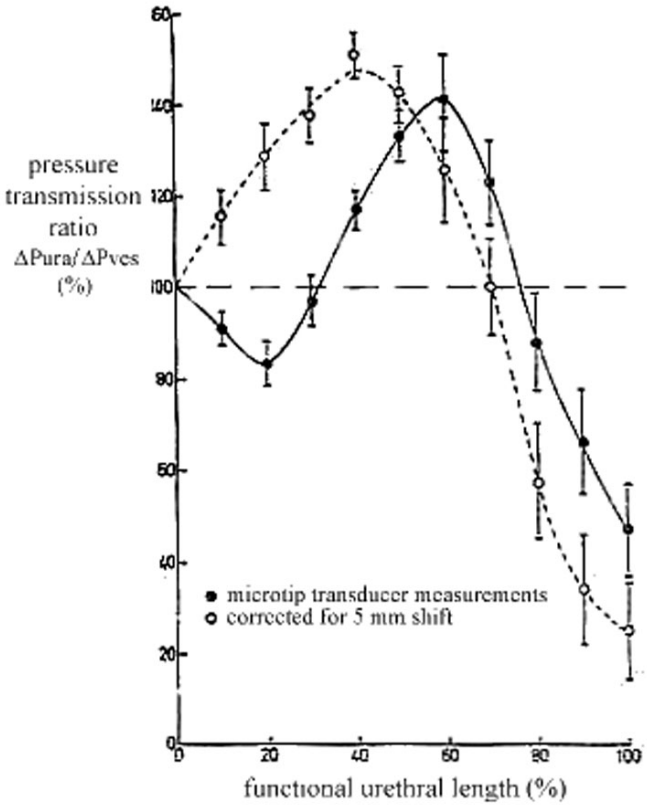

Figure 3. Schematic diagram of measured values of pressure transmission ratio (full circles) along the length of the urethra, based on traces similar to those in Figure 2. [49] Part of the urethral response may be due to a shift of the measuring catheter within the urethra on each cough. Correction for an assumed shift of 5 mm alters the values to those shown by open circles, revealing that the dip in pressure transmission ratio at around 20% of functional length may be an artifact of the catheter shift.

In summary: In practice it is impossible to use a catheter to measure a strictly local, artifact-free urethral pressure under stress.

• Can we measure the "stress"?

Leak-point pressures: The 'abdominal' or 'Valsalva' leak-point pressure (LPP), as defined by International Continence Society, is the intravesical pressure at which urine leakage occurs due to increased abdominal pressure in the absence of a detrusor contraction. [4] The procedure is a dynamic evaluation of the severity of sphincteric dysfunction, and as such has been considered by many to reflect the pathophysiological mechanism associated with stress incontinence. Further, it avoids the complex artifacts of urethral pressure recordings. A low pressure reading may identify a subgroup of patients with a severely deficient sphincteric mechanism who may benefit from a videourodynamic evaluation. [50] This procedure requires only basic urodynamic equipment: one intravesical pressure catheter and one abdominal pressure catheter (placed in either the vagina or rec-

tum). As the patient performs a progressively stronger Valsalva manoeuvre, the leak point pressure corresponds to the lowest intravesical pressure that results in a loss of urine. However, the Valsalva leak point pressure has a significant percentage of false negative results, up to 40% as reported by Peschers et al. [51]

Accurate and reproducible leak point pressures are difficult to obtain because of the many independent variables affecting the test results: catheter size, bladder volume and patient position. Another limiting factor among older patients may be that they are unable to generate adequate abdominal pressures to cause a leakage of urine under test circumstances. If this occurs, greater abdominal pressures can be generated with a cough leak point pressure. Kuo has recently compared the Valsalva and cough leak point pressures, using videourodynamic examinations. He concluded that the value of the leak point pressure required to make a diagnosis of intrinsic sphincter deficiency (ISD) was lower for Valsalva than for cough. [52]

A cough, in contrast to a Valsalva manoeuvre, may cause greater muscular contraction within the pelvic floor, resulting in decreased vesical neck mobility. For this and other reasons, cough-induced leak point pressures are usually higher than those obtained with a Valsalva manoeuvre.

Since there is no standardized protocol for the test, discrepancies are inevitable. Madjar et al. retrospectively investigated aspects of the Valsalva leak point pressure in 264 female patients who afterwards underwent anti-incontinence surgery. [53] They looked at the relationship between "VLPP(tot)" (measured from true zero of pressure, see **Figure 4**), and "ΔVLPP" (the increase in pves over the pre-Valsalva baseline). As is obvious from the figure, if a fixed value of 60 cm H_2O is used as a cutoff to differentiate intrinsic sphincter deficiency from urethral hypermobility, ΔVLPP is much more likely to suggest intrinsic sphincter deficiency than VLPP(tot) (in 67% versus 40% of patients). However, neither parameter strongly predicted the outcome of anti-incontinence surgery. (See section I.3 for further discussion.) (Figure 4)

The commonly quoted reference of 60 cm H_2O or less for the diagnosis of intrinsic sphincter deficiency has not been consistently utilized either in study protocols or in prospective studies proving that this pressure can accurately make the diagnosis. [54] Patients with a leak point pressure greater than 90 cm

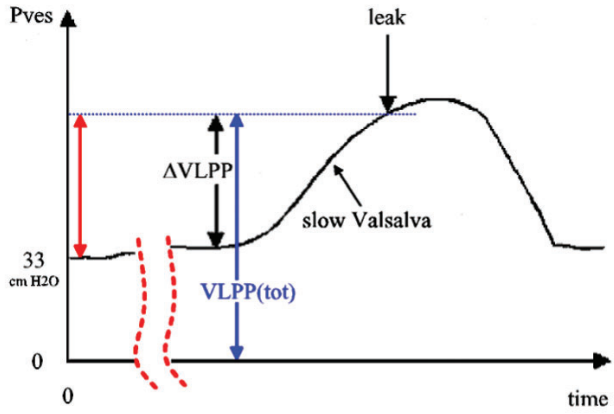

Figure 4. Measurement of Valsalva leak point pressure (VLPP). The VLPP is the intravesical pressure at which leakage occurs on Valsalva; it may be measured from the true zero of pressure (VLPP(tot), blue arrow), or from the baseline intravesical pressure just before the Valsalva (ΔVLPP, black arrow). In fact neither corresponds to the original description,[143] which used the value of intravesical pressure prior to bladder filling as the baseline (red arrow). For women in the standing position, ΔVLPP is much smaller than VLPP(tot), by about 33 cm H_2O on average. [53]

H_2O are conventionally not considered to have significant intrinsic sphincter deficiency, as such high pressures are usually associated with urethral hypermobility. It has been reported that, in clinical practice, leak point pressure values in the range of 60–90 cm H_2O are usually judged individually according to the severity and duration of the patient's symptoms, as well as accompanying co-factors. [55] As discussed above, however, all stress-incontinent patients have some combination of hypermobility and intrinsic sphincter deficiency.

Other factors that can affect leak point pressure readings include the presence of the catheter itself as well as its diameter. Maniam and Goldman examined patients with a positive cough stress test and a negative Valsalva leak point pressure for leakage of urine, and found that more than 50% of these women lost urine on repeating the Valsalva leak point pressure after removing the catheter. [56] This suggests but does not prove that the catheter could mask urine loss. Studies of *voiding* flow rates with and without a catheter are sometimes taken to indicate the effect of the catheter itself. For example, Baseman et al determined in a study of 21 continent women that flow rates with a small (6 F) catheter in the urethra were significantly lower than those in free-flow studies, by 6.4–7.4 ml/s. [57] However, there are other

possible reasons for such a difference, and it is not clear that a catheter will necessarily inhibit stress urine leakage, or falsely elevate the pressure required for leakage to occur. Faerber and Vashi [58] reported that Valsalva leak point pressure measurement made at a bladder volume of 250-300 ml correlated best with the fluoroscopic observation of type III stress incontinence with an open bladder neck at rest (i.e. a failure of the proximal continence mechanism).

Höfner and coworkers [59] have suggested performing a cough leak point pressure combined with measurement of the actual volume of urine loss, using a standard uroflowmeter with a modified funnel. They report significant differences in the time delay measured between the cough pressure spike and the urine loss. They interpret this by assuming that immediate leakage indicates stress incontinence and delayed leakage potentially indicates urge incontinence, or detrusor overactivity incontinence triggered by the cough. Although not yet confirmed, these results show clearly the difference between observing leakage with the naked eye and measuring leakage objectively. Furthermore, the authors found that the leakage volume, but not the measured leak point pressures, were associated with the clinical degree of stress incontinence.

In summary, despite problems with standardization, with current equipment it should be possible to measure the intravesical and abdominal pressure at the time of leakage under stress, and to identify and hence correct artifacts. However, we do not know of any systematic research into the various aspects of the leak point pressure. In addition, it is important to ask what potential information could be gleaned from this parameter. It is obvious that any leak point pressure depends on a multiplicity of factors, and that it is a measure of SUI severity, as it reflects the critical level of "stress" needed to produce leakage. It is unclear however whether a quantification of the severity of incontinence by a leak point pressure is more useful than a traditional clinical grading. It can be expected that, when it has been adequately evaluated, the recently developed urethral retro-pressure URP [60] will perform at best similarly to the leak point pressure and UPP.

d)What could be done urodynamically to develop methods of repairing specific defects and restoring physiological continence function? *(Question 4)*

We need the abdominal pressure to understand detrusor contraction, i.e. to determine detrusor pressure. The current technique is well suited to this purpose and provides good results if the established standards are followed. [33] The situation is different when we try to understand the impact of stress on the bladder closure mechanism. "Stress" is always associated with an increase in ***abdominal pressure*** and its effect on the pelvic floor and bladder outlet, but the abdominal pressure is not *specifically* related to the stress which causes incontinence.

The ***pelvic floor*** is the anatomical structure which provides closure to the abdominal region between the pelvic bones. It is a three-dimensional structure, i.e. it has a considerable thickness, which is penetrated by the urethral, vaginal, and anal canals. We can expect a gradient in the abdominal pressure, from its full value to zero across the thickness of the pelvic floor. This gradient must affect measurements such as the urethral pressure profile, which are performed across the thickness of the pelvic floor.

To summarise, the current urodynamic parameters and the currently available techniques do not offer the clinically relevant information needed for a knowledge-based (i.e. detailed and specific) approach, to the diagnosis and treatment of individual pathophysiology in a patient with stress incontinence.

5. What urodynamic tests should be done in future?

a) Imaging

Imaging, although not usually considered part of urodynamics, is developing so rapidly that it will soon have adequate spatial and dynamic resolution to provide quantitative mechanical information complementary to that given by pressure measurements. Sonography and MRI are particularly promising. Investigation of local pelvic-floor deformations and mobility in three dimensions would be very interesting, because the strength of the pelvic floor determines not only its own mobility and deformation, but that of all structures resting on it. Without any deformation of the pelvic floor under stress nothing else can move.

The speed of deformation occurring under stress is likely to be higher than can be observed with the naked eye. In practice the eye is supplemented by an imaging system with its own time constant, which slows down observation. The usual X-ray image intensifier systems are obviously slower than the

naked eye. Thus, it is likely that the true extent of deformation is not yet known. Sonography should provide a better dynamic resolution.

The combination of a defined pressure in an intra-urethral balloon with imaging of how the urethra is distended by that pressure can provide additional information about bladder outlet structure and function. [61,62]

b) Urethral pressure

As the urethral pressure is the key to understanding what happens to the closure mechanism under stress we must develop new methods to measure it that avoid the artifacts we have described, or at least allow us to identify and correct the artifacts. There are in principle two possibilities, either to detect and measure the movement of the catheter, or else to make the catheter so thin and flexible that it does not move or interfere with the measurement. To avoid movement, some form of intravesical or intra-urethral fixation may be unavoidable. On the other hand, a thin, flexible catheter could incorporate the very small transducers that are available through nanotechnology. In any case, it will be necessary to make specifically *local* pressure recordings in order to understand closure function under stress. Global measurements of compound parameters such as a leak point pressure cannot provide the information that is needed.

c) Measurement of stress

In principle we could try to measure the "stress" itself, but not by such a crude and unspecific measure as abdominal pressure. It is not possible to even begin such a program without a detailed and realistic biomechanical model of the function of the urethral sphincter and the pelvic floor, so as to identify and define the pathophysiologically relevant parameters that should be measured, such as (physical) stress and strain, deformation and acceleration. It is difficult to develop such a biomechanical model of continence function and the specific biomechanical changes which lead to stress incontinence. Yet only with a proper model will we be able to understand the limits and potential of mechanical urodynamic parameters, define better parameters, develop new ways and methods of measurement, and test the new parameters.

d) Vaginal pressure profile and abdominal pressure region

The ***abdominal pressure region*** is a suitable urodynamic term for the region which includes all of the lower urinary tract except the distal urethra, together with the surrounding intra-abdominal tissues, all of which are subject to the same abdominal pressure. Across the thickness of the pelvic floor, the pressure falls from the abdominal pressure inside to zero (atmospheric) outside. In the urethra and the rectum the sphincters provide a clear demarcation of the abdominal pressure region, but as there is no sphincter in the vagina the demarcation is less clear. The vaginal pressure profile should provide information about it.

e) Elements of a biomechanical model and measurements to test it

Some essential elements of a biomechanical model and the urodynamics of stress incontinence have been outlined by Ashton-Miller and DeLancey. [26] Increasing tension and stretch of the pelvic floor during an abdominal pressure increase affect the urethral sphincter also. The anatomical/biomechanical details of the penetration of the urethra through the pelvic floor are key to understanding how continence is maintained as abdominal pressure increases, how stress incontinence occurs, and how it can be cured surgically. Such hypotheses as the vaginal hammock theory [63] could be tested by making vaginal as well as urethral pressure profile recordings. Since there is necessarily a pressure gradient across an effectively supporting hammock, there must be a difference, during stress and perhaps also at rest, between the abdominal and urethral and vaginal pressures measured at the same anatomical level, with the vaginal pressure being the lowest.

Similar recordings could be used to test other concepts, such as that of independent proximal and distal continence mechanisms, one relatively passive and one clearly active, which react differently to changes in abdominal pressure.

Of course, in making such measurements all the limitations of pressure recordings during stress apply, but if we have a "catheter-free" pressure recording method for the urethra we can use the same technique in the vagina. Simultaneous recording of intravesical and abdominal pressures together with multiple local pressures in the urethra and in the vagina should be possible - without interference from catheter-shift and directional artifacts - and this will be a very important step in the understanding of the behavior of the urinary tract under stress.

6. Stress incontinence: Conclusion and recommendations

A plausible biomechanical model, based on current knowledge is a prerequisite for deeper understanding of the pathophysiology of stress incontinence. New types of measurements will be required to test and refine it. Only after that, when the pathophysiology of stress incontinence is understood in much more detail, will new therapies and new surgical approaches, aimed at re-establishing normal physiological conditions, suggest themselves.

• ***Recommendations for research***

1 Development of new methods of measuring urethral, vaginal and rectal pressure or force, free of artifacts due to distortion by and shift of the catheter.

2 Integration of new pressure or force measurements with advanced (high-speed) imaging techniques, so as to develop a new and more complete biomechanical model of normal and abnormal pelvic and bladder outlet mechanics.

3 Integrated approaches to the study of urinary and faecal (anal) incontinence.

II. URGE INCONTINENCE

1. Introduction

a) Urodynamic tests conventionally performed

Urge incontinence is one of the symptoms of the overactive bladder symptom complex (see section A.IV, definitions and terminology). [64] To identify it a history is needed, but to objectively establish the underlying mechanism of incontinence requires testing. This is particularly important because symptoms are not specific for the underlying dysfunction (see **Tables 6-9**, section C.IV). To reproduce the symptom during urodynamic testing, it is often necessary to perform manoeuvres to provoke detrusor overactivity and leakage, since only about 50% of detrusor overactivity is revealed during slow, supine filling cystometry. [65] Although the urodynamic observation corresponding to urge incontinence is "detrusor overactivity incontinence" [64] (see Figure 5) in fact, however, when examining patients with urge incontinence, many investigators have been satisfied with the observation of involuntary contractions (detrusor overactivity) only, rather than actual leakage. This has led to some confusion (**Figure 5**).

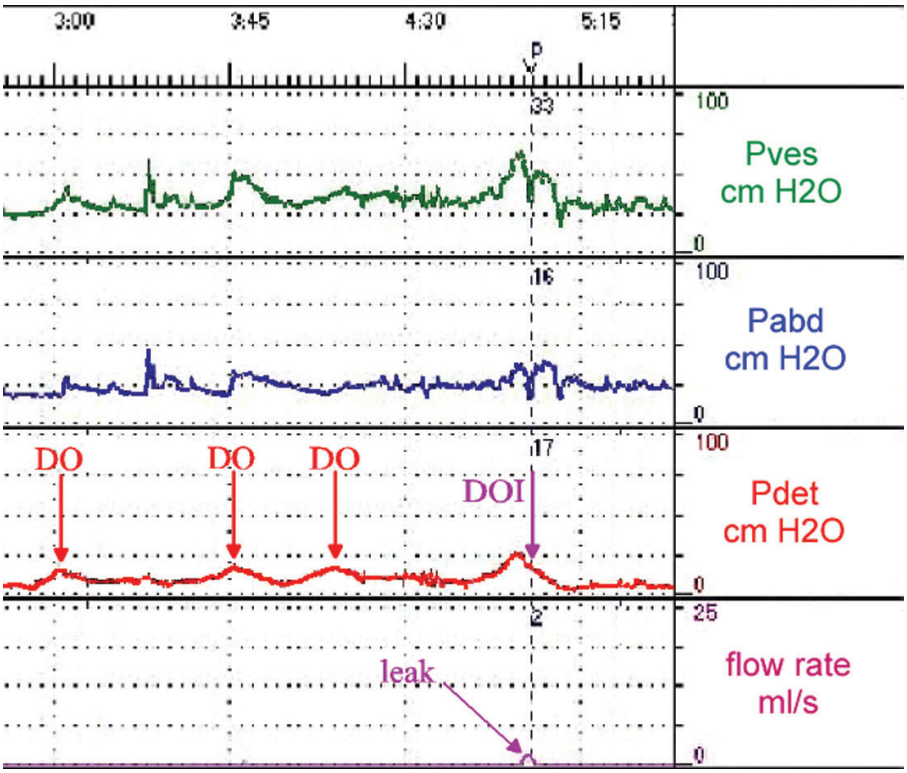

Figure 5. Detrusor overactivity (DO). In this example there is phasic detrusor overactivity (red arrows), which ultimately leads to leakage; this is detrusor overactivity incontinence (DOI, purple arrow).

Filling cystometry with various ***provocative manoeuvres*** is the basic test in urge incontinence and other manifestations of overactive bladder. Conventionally, it is intended to identify or rule out detrusor overactivity and/or detrusor overactivity incontinence, as well as to test subjects' bladder-filling sensations. In addition it is desirable to rule out urodynamic stress incontinence as an explanation of the subject's symptoms.

Ambulatory urodynamics may be performed in an effort to capture more realistic or more physiological observations, especially of incontinence episodes. Monitoring is usually continued for a period of about 3-4 hours.

Other supplementary tests may be done to check for possible alternative or coexisting pathology:

1. *Coughing and Valsalva manoeuvres*, sometimes including abdominal leak point pressure measurement or video observations, are usually done to reveal or rule out possible urodynamic stress incontinence. They may also provoke detrusor overactivity.
2. *Urethral pressure measurements* may be performed to identify a poorly functioning urethral sphincter (intrinsic sphincter deficiency), a possible contributor to both urodynamic stress incontinence [1] and urethral relaxation incontinence. [66]
3. *Voiding studies* may be done to confirm or rule out bladder outlet obstruction, a possible contributor to detrusor overactivity, especially in men.

Many technical aspects of these tests were discussed in detail at the previous consultation;[1] others are dealt with in sections B.I (Stress incontinence) and D.I (Evaluation of the female patient) of this current chapter.

b) Mechanisms of continence and (urge) incontinence

1. Detrusor overactivity

It seems clear that, if there is never any involuntary detrusor contraction, there can be no detrusor overactivity incontinence or urge incontinence (with the caveat that urethral sphincter relaxation without detectable detrusor contraction may sometimes be responsible [4,67]). However, the converse is not necessarily true: some individuals show involuntary contractions (detrusor overactivity) during urodynamic testing but do not suffer from urge incontinence (see Figure 6 and Table 3 in section C.II, Normal values). (Similarly, sphincter relaxation can occur in normal volunteers also. [68]) Detrusor overactivity is a urodynamic observation, not a condition and not a diagnosis. It may be associated with any of the overactive bladder complex of symptoms, with or without urge incontinence, or with no symptoms at all (see Table 8 in section C.IV and Figure 6). Therefore the mechanism of continence is not just the absence of detrusor overactivity. There is some evidence (a) that good sphincter function helps to prevent urge incontinence in the face of detrusor overactivity, [66,69] (b) that abnormality of bladder sensation may play a role, [66,70,71], (c) that the characteristics of the detrusor contraction itself may differ in those who suffer more or less severely from urge incontinence, [72] and (d) more generally that many different factors interact to determine whether or not a given subject will suffer from urge incontinence, and how severe it will be (**Figure 6**).

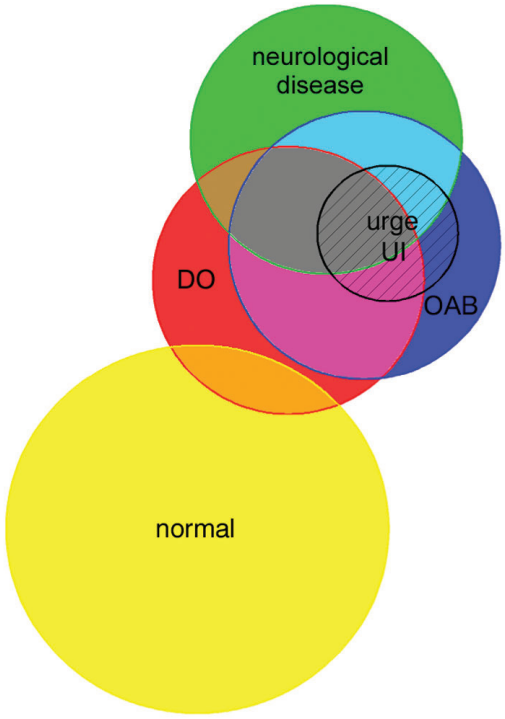

Figure 6. Relations among the overactive bladder complex of symptoms (OAB), urge incontinence (urge UI), detrusor overactivity (DO), and neurological disease. Detrusor overactivity (red circle) may be neurogenic (overlap with green circle) or non-neurogenic. It is observed in some healthy, symptom-free subjects (overlap with yellow circle). Many individuals with DO have overactive bladder symptoms (overlap with blue circle). Urge incontinence (hatched circle) is one of the overactive bladder symptoms and so falls entirely within the blue circle. As shown by its overlaps, urge incontinence may occur with or without detrusor overactivity, and with or without neurological disease, but it does not occur in normal subjects (yellow circle). Not shown on this diagram are some subjects who present with OAB symptoms that have a behavioural basis (e.g. excessive fluid intake) and are urologically normal.

Nevertheless, detrusor overactivity is at the heart of urge incontinence, and it is essential to define it and to try to identify its causes. Detrusor overactivity is the observation of an involuntary increase in detrusor pressure during urodynamic testing. To generate a measurable pressure increase requires a coordinated contraction of the greater part of the bladder since, if part of the bladder is non-contracting (and therefore relaxed and extensible), the contracting part can shorten without a detectable increase in pressure. In fact, local contractions of the bladder wall are ubiquitous, in bladder strips, excised bladders and intact bladders *in situ.* [73,74] Thus small-scale detrusor overactivity is normal, and it must result in small pressure fluctuations (which however are not usually measurable in awake, moving and breathing humans). Therefore, to place any particular numerical limit on the amplitude of pressure changes that might be regarded as normal or abnormal (e.g. 15 cm H_2O [75]) would be entirely arbitrary.

There is no reason to believe that detrusor overactivity has a single cause. The lower urinary tract is a deceptively simple organ system that is controlled by very extensive tracts of the central nervous system. Dysfunction at any level from the cerebral cortex to the end organs may lead to detrusor overactivity and urge incontinence. [76,77] Detrusor overactivity is common in neurological conditions such as multiple sclerosis, spinal cord injury, and stroke. [76] Urge incontinence becomes more common in old age, [78] where it is associated with cognitive impairment and regionally impaired cerebral perfusion. [71] Thus urge incontinence and its underlying mechanism detrusor overactivity may sometimes have a neuropathic origin. The types of neuropathy in question are associated with suprasacral lesions, which are believed to interfere with voluntary control of the micturition reflex by the cerebral cortex. Detrusor overactivity of this sort often involves abnormality of urethral function (e.g. detrusor-sphincter dyssynergia) as well as detrusor function. Of course, patients with a neurological lesion may sometimes show detrusor overactivity that is non-neurogenic.

2. Causes of detrusor overactivity

In spite of the important role of neurological lesions, many patients with detrusor overactivity and urge incontinence have no detectable neurological abnormality. Some have co-existent urethral obstruction, and it is often claimed that the detrusor overactivity is a consequence of this mechanical outflow obstruction, or of changes in the bladder secondary to it. However, this may not necessarily be so:[79] another possibility is that prostatic enlargement by itself may alter afferent signals to the brain, precipitating detrusor overactivity. Other patients may have subtle abnormalities of the neurological control system that cannot be recognized with current urodynamic techniques. Even when there is no evident neurological basis for this condition, it can often be triggered by factors that apparently act at the cortical level, such as the proximity of a toilet, the sound of running water, or hand-washing. [80] Thus there is evidence that the brain is involved in urge incontinence. Yet the afferent signals reaching the brain, cortical processing of these signals, and the influence of the emotions on bladder function remain almost entirely uninvestigated. Bladder sensation – one manifestation of the afferent signals – is studied in a most rudimentary way. It is unlikely that any abnormalities involving these factors, if they existed at all, would be recognized by current techniques.

Detrusor overactivity is a fairly frequent observation in apparently healthy, symptom-free volunteers. Thirty years ago, idiopathic detrusor overactivity was regarded as being like red hair, unusual (prevalence 10%) but not abnormal, and the original term "unstable bladder" was invented precisely because it did *not* suggest any particular underlying abnormality. [22] Two decades later it was claimed that "Any bladder contraction during filling is abnormal."[81] In fact, the average proportion of healthy subjects with detrusor overactivity on filling cystometry is 11%, and it is much greater – about 45% - when observations are made during ambulatory monitoring (see Tables 3 and 4 in section C.II, Normal values). Thus detrusor overactivity may sometimes be normal. For this reason it would be helpful to use descriptive terminology that does not imply pathology, for example "phasic detrusor contraction(s)" instead of "detrusor instability" or "detrusor overactivity". [82]

The observation of detrusor overactivity in apparently normal, symptom-free subjects has been referred to as "overdiagnosis." In contrast, detrusor overactivity is quite frequently *not* observed during urodynamic examination of patients who indubitably suffer from urge incontinence [83] (see discussion of sensitivity and specificity in section C.IV). This has been referred to as "underdiagnosis". Ambulatory testing, like other aggressive provocative manoeuvres was intended to reduce "underdiagnosis," but it also increases "overdiagnosis." In fact the terms "over-" and "underdiagnosis" are inappropriate because

detrusor overactivity is not a diagnosable condition, but a urodynamic observation that may or may not be made in any particular case, [84] depending in part on the method of measurement. [79] If it is related to neuropathy or to symptoms such as urge incontinence, urgency, frequency, or nocturia, the observation is suggestive of abnormality. If not, it presumably reflects normal behavior.

An essential characteristic of detrusor overactivity is that it is involuntary. Even in humans it is sometimes difficult to establish whether a particular detrusor contraction or voiding episode is voluntary or involuntary, for example if there is a language barrier or a communication disorder. In animals it is particularly difficult to construct reliable models of detrusor overactivity and urge incontinence.

Even if detrusor overactivity *begins* involuntarily, it may be possible to voluntarily suppress it, or at least to contract the urethral sphincter voluntarily so as to prevent leakage until the contraction subsides. Such characteristics of patient behavior may be clinically just as important as the presence or absence of detrusor overactivity. [85-87]

3. Sensations and cystometric volumes

Sensations are an important part of the observations made during cystometry. The normal sensations of bladder filling form a continuum from first sensation of bladder filling, through first desire to void, to strong desire to void. During cystometry most investigators note the bladder volumes at which the normal sensations occur (or they note the absence of such sensations). It is usually believed that the volumes tend to be smaller – that is, sensation is increased - in the overactive bladder syndrome.

The sensation of urgency – the sudden and compelling desire to void that is difficult to inhibit [4] – is abnormal. It seems to be dichotomous – either present or absent – in contrast to the continuum of normal cystometric sensations. It is part of the definition of urge incontinence, but may also be an independent symptom or an independent urodynamic observation. The sensation of urgency during cystometry is often but not always associated with the presence of detrusor overactivity. Ideally the sensation of urgency, if it is reported as a symptom, should be reproduced during urodynamic testing.

When detrusor overactivity is observed, the quality of the accompanying sensation (if any) may be an important observation. [69,72,83,88]

Maximum cystometric capacity is nearly always noted. Clearly a very large capacity suggests at the least reduced sensation, since, with normal sensation, discomfort limits the volume that can be infused into the bladder. The meaning of the maximum cystometric capacity is not always the same, because the criteria for stopping filling are different in different situations. In near-normal individuals, strong sensation (discomfort) is usually the limiting factor. If detrusor overactivity, or overt detrusor overactivity incontinence, occurs, then this will usually limit further bladder filling, particularly if accompanied by urgency. If compliance is low (see section "compliance" below), then the constantly rising detrusor pressure may persuade the investigator not to continue filling; alternatively overflow incontinence may occur and filling be discontinued for that reason. If sensation is reduced and compliance is normal, and there is little or no detrusor overactivity, then the bladder may be filled almost indefinitely and the patience of the investigator and the subject may be the limiting factor.

During filling cystometry, the volumes corresponding to the various sensations and to maximum capacity should in principle represent the volume of liquid actually in the bladder. [4] Frequently however the volume infused into the bladder is recorded. The difference between these two volumes is the volume of urine produced by the subject It may be substantial if low filling rates are employed.

2. Reproducibility and fundamental reasons for variability of filling cystometry

Data on the variability of cystometric parameters are given in section C.I, Reproducibility of filling cystometry and ambulatory monitoring.

• Reproducibility and voluntary control

The lower urinary tract is controlled by numerous layers of the nervous system. The lower layers (peripheral, spinal and supraspinal up to and including the periaqueductal gray), constitute the voiding reflex and can work together automatically, but the topmost layers – the emotional or limbic nervous system [89] and the cortex – integrate reflex behavior into the social life of the individual. Their purpose is to prevent the automatic operation of the reflex and allow voiding only when it is both safe and appropriate to do so. They are able both to advance and delay the moment of reflex voiding. By design, therefore, the upper layers of the control system introduce variability into the function of the

lower tract, and this variability depends on conscious decision-making (to void or not to void) and on the unconscious operation of the emotional nervous system (safe to void or not safe?) (**Figure 7**).

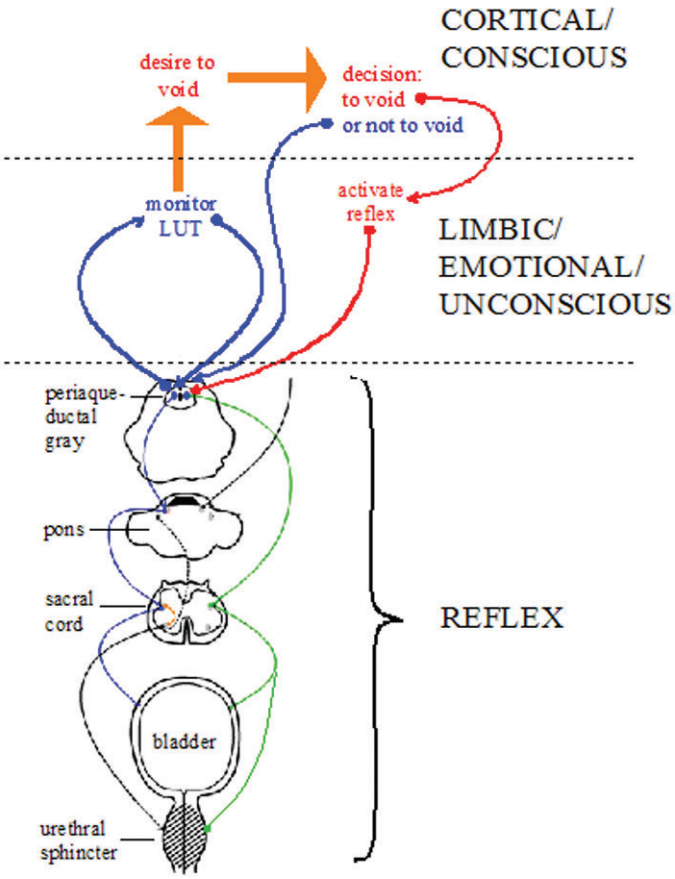

Figure 7. Schematic concept of the neural control of the lower urinary tract. The voiding reflex extends from the bladder and urethra up to the brainstem (pons and periaqueductal gray, PAG), but does not control the bladder directly. Via afferent signals diverted from the PAG, the state of the lower tract is monitored by the limbic parts of the brain. As the bladder fills, a sensation of desire to void is passed to the cortical part of the brain, where a decision is made whether or not to do so. If the decision is to void, and if the limbic (emotional) part of the brain permits, the reflex is activated and the bladder empties; otherwise the bladder continues to fill. Thus cortical and emotional control introduces variability into the operation of the voiding reflex.

The variability of urodynamic measurements reflects the operation of the control system, mainly from the upper layers. The automatic operation of the deeper layers is presumably less variable. For example, spinal cord injured patients are relatively easy to examine, because their bladder behavior is reasonably reproducible. Thus, abnormal findings in patients with severe neurological diseases may be reliable and reproducible (unless the disease itself is variable), while similar findings in subjects with an intact nervous system may be merely the result of the variable operation of that system, and require independent confirmation. Variability originating in the topmost layers of the nervous system appears able to mimic the effect of any neurological lesion: e.g., detrusor acontractility or underactivity, or detrusor overactivity, or detrusor-sphincter dyssynergia.

Reproducibility has to be considered in the context of neuropathy. Provided technical problems have been ruled out, substantial variability is not a sign of poor urodynamics but a sign that the upper layers of the nervous control system are intact. Reproducible but abnormal function on the other hand may suggest automatic operation associated with neuropathy. Clearly, the reproducibility of urodynamic measurements is dependent on the patient population studied, and this is a topic for research that should be considered in any discussion.

Although variability may be a sign of normality, we may nevertheless prefer to reduce it. The prime source of uncontrolled variability appears to be the emotional nervous system, and the optimum way of reducing its impact is to influence the patients' surroundings so as to reduce anxiety and distract attention from bladder behavior. The typical urodynamics laboratory appears ill-designed for this purpose. Ambulatory monitoring is one way to attempt improvement, but the most important aspect is probably not that it is ambulatory but that it is conducted at a leisurely pace, in a series of natural postures, and in non-threatening surroundings. These aspects should be mimicked in more conventional urodynamic examinations as well.

3. What urodynamic tests should be done in urge incontinence and detrusor overactivity?

a) The role of urodynamics

What should be the role of urodynamics? It is unrealistic to expect that the limited and crude measurements currently employed should be essential in selecting patients for our current treatments or in improving outcomes. It is clear that we have a very incomplete understanding of lower urinary tract function, and that present methods do not address many of the issues. Urodynamics is pivotal here: it is

the only way of understanding what is going on. For the near future therefore the real role of urodynamics has to be to keep advancing the boundaries of what is known about function and dysfunction of the lower urinary tract, in the expectation of developing *new* and *more effective* treatments. In the long run, whether advanced urodynamic testing will prove important in selecting patients for treatment is uncertain. It is possible that lower tract behavior is influenced by so many uncontrollable factors, and hence is so variable in individual patients both in daily life and during testing, that urodynamics will never be able to provide reliable individual diagnoses. Nevertheless, urodynamics will always remain the key to understanding the behavior of bladder and urethra in human subjects.

b) Understanding of abnormal behavior

Better understanding of LUT behavior is no guarantee of improved therapeutic outcomes, but improved outcomes are unlikely without understanding, and to gain understanding urodynamic measurement is crucial. However, these statements do not tell us what should be measured. In suspected urge incontinence, should we measure what the bladder does (cystometry)? Is it necessary also to measure the urethral contribution? Should we also examine the nervous control system? Clearly the answer to all 3 questions is *yes*, because all are involved in urge incontinence: the bladder because detrusor contraction is the primary cause of urge incontinence; the urethral sphincter because inadequate resistance to leakage is a causal factor; and the nervous control system because inadequate control of the LUT is synonymous with urge incontinence.

To measure all these things is difficult, because the more that is measured, the more complicated the urodynamic testing becomes, and the more likely it is that the patient will be inhibited by the study and find it difficult to reproduce the symptoms.

Conventionally, cystometry is always performed, but urethral behavior is often omitted, partly because the urethra is a much more complex mechanical system than the bladder and current instrumentation is inadequate to perform the required measurements (see section B.I, Stress incontinence).

So little is known clinically about the behavior of the neural control system that any extra information at all would be useful. The pelvic-floor surface (kinesiological) EMG is assumed to provide information about motor control of the striated urethral sphincter, although it does not always do this reliably, [90] and moreover is often badly performed technically. Measurements have demonstrated the reliability of intra-anal and intra-urethral EMG electrodes, but current instruments are too thick, too stiff, or too uncomfortable to be useful. An intra-urethral EMG probe, flexible and thin like the urethral pressure probe described in section B.I on stress incontinence, is desirable.

Afferent signals from the lower urinary tract are a crucial part of neural control. Yet the usual way of assessing them is through subjective reports of sensation, which often are elicited by patient prompting during cystometry. Sensation is therefore poorly measured and reported (see discussion of normal values and reproducibility above).

Cerebral control of the bladder is another crucial element, clearly implicated in urge incontinence, yet we usually make no attempt at all to examine it. Recent developments in functional brain scanning by PET and fMRI promise at least to shed some light on characteristics of different patient groups [91-94] and may one day be used to objectify the brain behavior of individual patients

c) Attempts at a new urodynamics

1. Improvements of existing techniques

• Cystometry

There have been several attempts to refine or quantify the observation of detrusor overactivity. In most cases the amplitude of the contractions has played a major role – the bigger the contraction the more severe the overactivity is judged to be. [95]

Groen et al [96] attempted to diagnose and grade detrusor overactivity by means of a computerized algorithm, comparing the results with expert opinion. They reported that a panel of experts, asked to judge the severity of detrusor overactivity from a set of traces, appeared to base their judgment on the highest contraction amplitude together with the percentage of the filling phase during which overactivity occurred.

The computerized overactivity parameter developed by these authors depended on the area under the curve describing involuntary detrusor contractions, after subtraction of any passive component of pressure increase during filling. (Overt low compliance was excluded from the study.) Thus the overactivity parameter depended on the frequency, duration and amplitude of the contractions. It provided a different classification of overactivity severity than the expert panel, although the contraction amplitude played an

important role. This parameter was used to assess changes in detrusor overactivity on treatment by sacral neuromodulation, [97] and may have some clinical utility for this type of assessment.

Miller et al [66] examined, in a group of 95 older women, what urodynamic factors were associated with the severity of detrusor overactivity and urge incontinence. They hoped to find parameters or combinations of parameters that might best represent the real clinical problem. They found that the functional bladder capacity was one such factor: a smaller functional capacity was associated with more severe incontinence. Interestingly, the amplitude of the overactive contractions was not positively associated with severity. In fact the opposite was the case, and they interpreted this to mean that the strength and control of the urinary sphincter was one of the most important determinants of incontinence severity, since a high amplitude of contraction can only occur in the face of a strongly contracting sphincter.

Another group [88] has suggested that different patterns of detrusor overactivity, perhaps representing different pathologies, should be distinguished,. They distinguished phasic detrusor overactivity (see Figure 5) from terminal detrusor overactivity (**Figure 8**). Terminal detrusor overactivity, especially if associated with reduced sensation of bladder filling prior to the terminal contraction that leads to leakage, suggests that a cortical abnormality is responsible for the detrusor overactivity. This deduction is strengthened if there is also a positive response to the bladder cooling (ice water) test (see section C.IV.1, subsection Provocative manoeuvres). Thus these authors have suggested that the bladder cooling test should become a standard part of urodynamic investigation [98].

One study suggests that several of the distinctions made above might have some predictive value for the outcome of treatment. [99] The authors investigated whether detrusor overactivity persisting after transurethral prostatectomy for benign prostatic hyperplasia could be predicted. In this small study of 19 male patients aged 69-83 years with presumed neurogenic detrusor overactivity together with evidence of benign prostatic obstruction, terminal detrusor overactivity that occurred at greater bladder volumes (>160ml) disappeared post-operatively in 6/6 patients. Phasic detrusor overactivity occurring at small volumes, on the other hand, persisted in 4/4 patients. SPECT brain scans confirmed that persisting detrusor overactivity was associated with reduced perfusion of the frontal brain region, suggesting a cortical abnormality.

As far as we know, apart from the above, there is no further evidence that these refinements of cystometry may be useful in predicting or improving treatment outcomes.

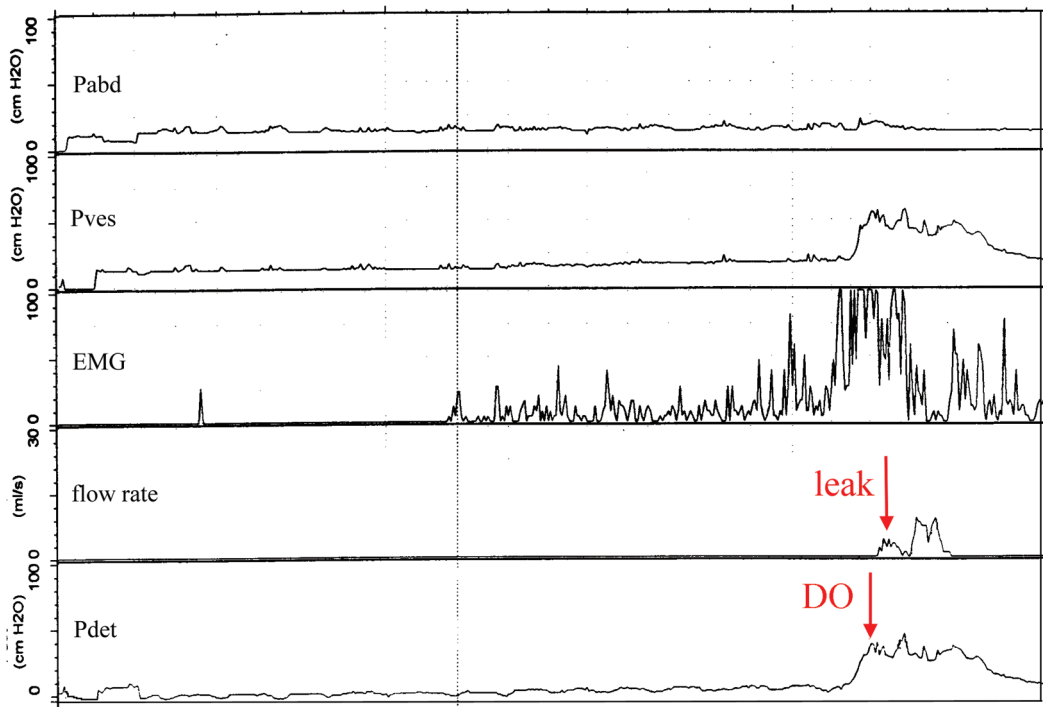

Figure 8. Terminal detrusor overactivity. There is no evidence of involuntary detrusor contraction until a single such contraction occurs (DO) and leads to leakage (detrusor overactivity incontinence). In this case leakage occurs in spite of the voluntary sphincter contraction revealed by pelvic-floor EMG.

• Urethral measurements

We have drawn attention to the need for technical improvements in the measurement of urethral pressures in section B.I. Stress incontinence. One new and promising technique is urethral reflectometry, a modification of acoustic rhinometry, which enables simultaneous measurements of pressure and cross-sectional area. [62]

2. New types of study

There are obvious gaps in current urodynamic practice, which is focused on end-organ behavior. Afferent signals, central (spinal cord and cerebral) processing, and influences at cortical levels, such as the effect of the emotions on LUT function, remain almost uninvestigated in clinical practice, and are difficult to study even in animal models. We should aim to monitor afferent and efferent signals, and signal processing at sacral and cerebral levels.

• Afferent signals

In the past, sensation was poorly and subjectively measured and recorded, although more attention has been paid to it recently. [100] Usually the patient has to be prompted to report sensations. There is great variation both from centre to centre and within subjects (see sections C.I and C.II). Oliver et al [101] have shown that a patient-activated keypad device can yield an objective and reasonably reproducible measure of sensations of desire to void, without prompting, and can reveal responses to therapy. At least in research studies such methods should be introduced.

Sacral neuromodulation has been introduced as treatment, but its potential as a research tool has not been fully realized. It seems clear that neuromodulation and other types of electrical stimulation can suppress bladder filling sensations to some extent. Thus they can and should be used to study central processing of afferent signals. A start has been made by studying the effect of neuromodulation on brain activity. [102]

• Signal processing

As pointed out above, a reliable but preferably non-invasive kinesiological EMG of the striated urethral sphincter would improve our knowledge of how the nervous system responds to lower urinary tract events. The technique and difficulties of the measurement were considered in the previous Consultation. [1] Little progress has been made since then.

Cortical processing of afferent signals – and their relation to efferents driving the bladder and urethra – can be studied by functional scanning of brain activity. [89,91-94] At present this is strictly a research tool, as intelligible results can only be obtained by averaging within groups of subjects. In the longer term however it will become possible to relate changes in brain activity to bladder and urethral events and sensations in individual patients. Certainly this will yield new diagnoses, and perhaps even new treatments. For example, 'neurofeedback' based on showing abnormal patterns of brain activity to the subject, and asking him/her to correct them, has already been demonstrated to be possible. [103]

4. Mixed incontinence

Some of the difficulties surrounding the concept of mixed incontinence are described in section A.IV, Definitions and terminology. For example, according to the authors of a recent study "Mixed incontinence is a term that can apply both to a combination of incontinence symptoms (stress urinary incontinence and urge urinary incontinence) and to a combination of urodynamic conditions (urodynamic stress incontinence and detrusor overactivity)."[104] While this statement may represent general opinion, in fact the ICS definition of mixed incontinence is a symptomatic definition only. [4] The corresponding urodynamic observations which may be taken to reproduce the symptom are described in the above-mentioned section. Nevertheless, an important goal of urodynamic testing in patients presenting with mixed incontinence is to determine whether the incontinence is associated with stress, with detrusor overactivity, or with both. To this end it is important to try to demonstrate actual detrusor overactivity incontinence, not just detrusor overactivity.

Bump et al [104] showed that a pharmacological treatment (duloxetine) presumed to be for stress incontinence was equally efficacious in mixed incontinence (with a predominant stress component). Reduction of incontinence frequency was approximately 60% in all treatment groups. The urge component of the symptom complex appeared to be more closely related to the severity of the leakage than to urodynamically demonstrable detrusor overactivity.

In a study of patients with mixed incontinence symptoms with a predominant urge component, pharmacological treatment (with tolterodine) aimed at detrusor overactivity was equally efficacious in mixed and in urge incontinence. Reduction of incontinence frequency was 67-75%. [105]

Osman [106] performed sling and colposuspension

surgery in a group of patients presenting with mixed incontinence and a normal cystometrogram. A subgroup was treated with anticholinergics. A second group with pure stress incontinence symptoms was treated with surgery. He suggested that the surgery patients did significantly better than the pharmacologically treated ones. Thus a treatment aimed at stress incontinence (surgery) was equally successful in patients with mixed incontinence symptoms (albeit with no urodynamic evidence of detrusor overactivity).

Taken at face value, these results seem to imply that "mixed incontinence" (defined in various ways) appears to respond equally well to pharmacological treatment aimed at detrusor overactivity, pharmacotherapy aimed at urethral resistance, or to surgery. If true, this would imply that not just urodynamics, but even a good history, was superfluous: any treatment will produce good results. In fact, mixed incontinence is a good example of a situation where there is a need for knowledge-based medicine, but the physiological understanding needed to provide that knowledge is absent.

5. Urge incontinence: Recommendations for research

1 Investigation of novel sources of variability of urodynamic parameters

2 Definitive studies of the reproducibility of urodynamic parameters in normal subjects, and also in specific patient groups, especially those with various types of neurological lesion.

3 Study of the pathophysiology of mixed incontinence

4 Study of cerebral and emotional control of lower urinary tract function

5 Study of the effect of neuromodulation on cerebral control of the lower tract

6 Improved recording of afferent signals during cystometry

7 Study of the effect of ambient surroundings, laboratory atmosphere, and investigator behavior on urodynamic observations and findings, with a view to optimizing and standardizing the performance of urodynamics and reducing variability.

8 Efforts to improve equipment for measurement of kinesiological EMG during urodynamic studies

C. URODYNAMICS: NORMAL VALUES, RELIABILITY, AND DIAGNOSTIC AND THERAPEUTIC PERFORMANCE

I. REPRODUCIBILITY OF FILLING CYSTOMETRY AND AMBULATORY URODYNAMICS

1. Inter-observer reproducibility

Inter-observer variability is usually assessed by asking different urodynamic practitioners to interpret the same traces. This method is flawed because a principle of good urodynamic practice is that the person doing the test should interpret it. Nevertheless, it is interesting that 4 experienced practitioners, evaluating 17 pediatric urodynamic datasets, failed to agree on aspects of detrusor function (including detrusor overactivity) in a quarter of the cases. [107] This result is reminiscent of a similar study of the interpretation of urodynamic recordings of male voiding function. [108]

2. Short-term (within-session) reproducibility

Since the previous Consultation, a number of authors have investigated the within-session reproducibility of cystometric measurements. Because such measurements are conducted within a short period of time, the possibility that the first measurement influences the second through a direct effect on the mechanical properties of the bladder (hysteresis and/or viscoelasticity) has to be considered.

Brostrom et al [109] examined 30 healthy women with a mean age of 52 years, performing 2 consecutive medium fill rate (50 ml/min) cystometries in a single session. The volumes at first desire to void and normal desire to void increased significantly from the first to the second measurement, by 34 and 51 ml respectively. The maximum bladder capacity showed no significant change. The proportion of these healthy subjects who showed detrusor overactivity decreased from 4/30 (13%) in the first cystometry to 1/30 (3%) in the second. This change is not statistically significant but suggestive. Since these patients had an intact nervous system, some of the observed

changes may be due to variability introduced by the upper layers of the control system.

In a similar study [110] of short-term repeatability in 31 female patients aged 14-74 y, all but 10 had at least some overactive bladder symptoms: 5 urge incontinence, 11 mixed incontinence, 1 enuresis nocturna, and 4 urgency and/or frequency. 2 consecutive cystometries were performed with body temperature liquid at a rate of 50 ml/min. On the first cystometry first desire to void occurred at a median volume of 112 ml (range 26-503 ml). The cystometric capacity had a median value of 150 ml, with a range from 39-633 ml. (These values may be compared with the normal values in **Table 3**, below.) The volume at first desire to void increased by 46 ml from the first to the second cystometrogram, while the cystometric capacity increased by 35 ml. These changes are similar to those seen in normal volunteers, but, as the authors point out, they are not clinically important because they are much smaller than the random variability within subjects, as measured by the 95% confidence limits of ±130 and ±106 ml respectively. The random variability presumably reflects the intact operation of a normal nervous control system in these women without evident neuropathy. The systematic changes from the first to the second cystometry might originate from the mechanical properties of the lower urinary tract itself (hysteresis), from the operation of the control system, or from both.

Chin-Peuckert et al [111] examined the variability between two consecutive cystometries in 32 male and 34 female children with a mean age of 7. Most suffered from spinal dysraphism. A smaller number showed detrusor overactivity on the second study than on the first ($p< 0.05$), and similarly the volume at which detrusor overactivity was first observed was larger on the second study ($p <0.05$). Interestingly, these results are similar to those obtained in children without overt neuropathy, [112] suggesting some element of mechanical hysteresis originating in the lower urinary tract itself. However, a contribution from cortical control cannot be ruled out, even in these children with profound neurological abnormalities.

Hess et al [113] did not perform repeated cystometries, but first measured the bladder pressure "as is" at whatever volume was in the bladder initially, in 21 men and 1 woman with "neurogenic bladder." They then drained the bladder and refilled to the same volume and again measured the pressure. The second "cystometric" pressures were higher than the initial "physiological" ones by approx 6 cm H_2O ($p = 0.01$), although there was a strong correlation between them. This systematic change may represent the regular physical viscoelastic properties of the bladder, since the cerebral parts of the nervous control system presumably exert little control in these patients with marked neuropathy. We shall return to this topic in section C.II.2, Compliance.

3. Intermediate-term reproducibility

Changes occurring in the intermediate term (several weeks) can hardly be due to a direct mechanical effect of one test on the next. Almost certainly therefore they do not originate in the lower urinary tract itself but represent habituation to the circumstances of the urodynamic examination: i.e., learned changes in the control system.

Homma et al [114] found that, in 30 patients with detrusor overactivity, repeat cystometry carried out 2-4 weeks after initial testing showed a systematic shift toward normal. Bladder volumes increased by 10-13% ($p<0.01$), while detrusor overactivity disappeared in 10% of subjects and decreased in amplitude by an average of 18% in the remaining cases. The random variability of cystometric capacity was ± 57 ml (95% CI). In this group of patients without overt neuropathy, it appears that these observations indicate changes in the way in which the emotional nervous system influences the behavior of the lower urinary tract, associated with habituation to the test.

4. Long-term reproducibility

Long-term changes in cystometric variables may represent progression of disease, but it is unlikely that learned behavior persists over periods as long as a few years, and even less likely that one test can have a direct mechanical effect on another performed some years later.

Sørensen et al [115] investigated 10 healthy females (mean age 34 years), twice at an interval of 2 years. They made measurements in both supine and sitting positions. First sensation of filling occurred at a mean volume of 378 ml supine and 354 ml sitting, with intra-subject variability (over two years) of 76 and 100 ml (SD) respectively. Maximum capacity was 512 ml supine and 502 ml seated, with intra-subject SD of approximately 75 ml in both cases. Inter-subject SD's were a little larger: 76-144 ml. No significant systematic changes in volumes over 2 years could be demonstrated. There was no significant change in bladder compliance (see section C.II.2, Compliance) (**Table 2**).

Table 2. Intra-subject variability of cystometric parameters from one test to the next, within-session, intermediate-term (2-4 weeks), and long-term (2 years)

Authors	Population	Period between tests	Systematic change (test 2- test 1)				Within-subject random variation from test 1 to test 2	
Brostrom [109]	Healthy ♀	Same session	ΔFDV =+34	ΔNDV = +51	ΔMaxCap = ns	ΔDO = -3/30		
Mortensen [110]	Patients ♀	Same session		+46	ΔMaxCap = +35		NDV: ±130 (95% CI)	MaxCap: ±106 (95% CI)
Chin-Peuckert [111]	Children, spinal dysraphism	Same session	ΔVDO= + (P<0.05)			ΔDO= - (P<0.05)		
Hess [113]	Neurogenic bladder	Same session	ΔP = + 6 cm H_2O					
Homma [114]	Patients ♀	2-4 weeks	ΔV = +10% -13%			ΔD =-10%		MaxCap: ±57 ml (95% CI)
Sørensen [115]	Healthy ♀	2 years	ΔFSF: ns		ΔMaxCap:ns		FSF:± 76-100 ml (SD)	MaxCap: ±75 ml (SD)

Key to symbols: FSF = first sensation of filling; FDV = first desire to void; NDV = normal desire to void; MaxCap = maximum cystometric capacity; P = bladder pressure; V = bladder volume; VDO = volume at which detrusor overactivity occurs; DO = proportion of subjects with detrusor overactivity; Δ = increase from test 1 to test 2.

5. Summary

In patients and in healthy volunteers, if cystometry is repeated either during the same session or within about 4 weeks, the bladder volumes at which the various sensations are felt and the bladder capacity tend to increase by 30-50 ml, while the proportion of traces showing detrusor overactivity tends to fall. These systematic changes are fairly small in comparison with the random within-subject variability, which has a standard deviation of about 50-60 ml. If cystometry is repeated after about 2 years, the intra-subject variability is a little larger, and systematic changes are no longer evident.

Although a direct mechanical bladder effect such as hysteresis probably accounts partly for the increased volumes and reduced detrusor overactivity seen in a second cystometry during the same urodynamic session, it is unlikely that it would still be present after 4 weeks. A more likely explanation for the persistent effect of the first test is that subjects accommodate larger bladder volumes and show less detrusor overactivity when they have become accustomed to urodynamic testing (a cortical effect). After 2 years, any such effect appears to have worn off.

6. Reproducibility of ambulatory urodynamics

There appear to be no data on the reproducibility of ambulatory urodynamic monitoring.

II. CYSTOMETRY: NORMAL VALUES

1. Normal values: filling cystometry and ambulatory urodynamics

Table 3 shows normal values for cystometric variables reported by a number of authors, both long ago and more recently. A striking observation is that there is great inter-centre variability, even for nominally similar patients. There is no evidence from the table that these variations are associated with differences in filling rate, infusate temperature, or patient position. It appears that the sensations themselves are ill-defined and inadequately standardized (**Table 3**).

In a recent study, [109] referred to above, 30 healthy women, mean age 52 years, were examined with 2 consecutive medium fill cystometries at a filling rate

Table 3. Normal values (mean or median) of filling cystometry variables

Authors	population	parameters	FSF	FDV	NDV	SDV	MaxCap	DO
Brostrom [109]	Healthy ♀	Seated, 50 ml/min		171 43-508	284 182- 576		572 338-1016	4/30
Wyndaele [100]	Healthy ♂	Body temp 30 ml/min	222 ±151	325 ± 140		453 ± 94	453 ± 94	7/50
	Healthy ♀	Body temp 30 ml/min	176 ± 96	272 ± 106		429 ± 153	429 ± 153	
Van Waalwijk [116]	Healthy ♂ and ♀	Seated 35 ml/min	104 ± 57	172 ± 66	263 ± 93	294 ± 96		3/17
Robertson [117]	Healthy ♂	Room temp 50 ml/min					342 (269-471)	2/12
	Healthy ♀	Room temp 50 ml/min					500 (345-562)	
	Healthy _	Room temp 100 ml/min					475 (400-600)	0/12
	Healthy ♀	Room temp 100 ml/min					500 (390-790)	
Sørensen [118,119]	Healthy ♀	Supine, body temp 60 ml/min	347 ± 101				482 ± 103	0/10
	Healthy ♀	Seated 60 ml/min	357 ± 126				491 ± 147	
	Post-meno ♀	Supine 60 ml/min	396 ± 163				551 ± 223	0/12
	Post-meno ♀	Seated 60 ml/min	331 ± 168				489 ± 196	
Heslington [120]	Healthy ♀	Supine 100 ml/min					420 175-810 (range)	4/22
Walter [121]	Healthy ♀	Supine, body temp 30 ml/min	225 (150-300)				425 (400-490)	0/15
						Total DO 20/180 = 11%		

Key to symbols: FSF = first sensation of filling; FDV = first desire to void; NDV = normal desire to void; SDV = strong desire to void; MaxCap = maximum cystometric capacity; DO = proportion of subjects with detrusor overactivity; ± = SD; (xxx-yyy) = interquartile range; xxx-yyy = 95% confidence interval. All volumes are in ml

of 50 ml/min, in the sitting position. On the first cystometry, first desire to void occurred at a mean volume of 171 ml (range 43-508 ml). Normal desire to void occurred at 284 ml (182-576 ml). Maximum bladder capacity was 572 ml (338-1016 ml). Detrusor overactivity was observed in 4/30 subjects (13%).

Wyndaele et al [100] examined 18 men and 32 women. The bladder was filled at 30 ml/min with body temperature saline. For males (mean age 22 ± 3 years (SD)), first sensation of filling occurred at 222 ml ± 151 ml (SD); first desire to void occurred at 325 ml ± 140 ml; strong desire to void occurred at 453 ml ± 94 ml, and this was taken as maximum capacity. For females (mean age 21 ± 2 years (SD)) first sensation of filling at 176 ± 96 ml; first desire to void at 272 ml ± 106 ml; strong desire to void at 429 ml ± 153 ml. Detrusor overactivity was observed in 7/50 (14%) of these normal volunteers.

Van Waalwijk van Doorn et al [116] report similar figures. In 17 healthy subjects with a mean age of 24 years, but a wide age range, first sensation of filling occurred at a mean volume of 104 ± 57 ml (SD); first desire to void occurred at 172 ± 66 ml; normal desire to void occurred at 263 ± 93 ml; strong desire occurred at 294 ± 96 ml. Detrusor overactivity was observed in 3 of 17 subjects (18%) on conventional cystometry in the seated position. The filling rate was 35 ml/min. On ambulatory monitoring the proportion showing detrusor overactivity rose to 69% (**Table 4**).

Robertson [123] reported on 11 male and 6 female healthy asymptomatic volunteers, age range 22-72 years, examined by conventional filling cystometry (using room temperature saline) and by ambulatory monitoring. In females: the "filling volume" (presumably equivalent to maximum capacity) was: 342 ml median (269-471 ml interquartile range) at 50 ml/min; and 475 ml median (400-600 ml interquartile range) at 100 ml/min. In males it was: 500 ml median (345-562 ml interquartile range) at 50 ml/min; and 500 ml median (390-790 ml interquartile range) at 100 ml/min. Detrusor overactivity was observed in 2/12 volunteers (17%) during filling cystometry at 50 ml/min; there was none at a filling rate of 100 ml/min (which preceded the 50 ml/min filling). During ambulatory monitoring detrusor overactivity was seen in 6/16 subjects (38%). The median volume voided during ambulatory monitoring was markedly smaller than the filling volume on conventional cystometry (compare Tables 3 and 4 above).

Table 4. Normal values – ambulatory monitoring

Authors	population	Mean/median voided volume	DO
Van Waalwijk [116]	Healthy ♂ and ♀	200 ± 78 ml (mean ± SD)	11/16
Robertson [117]	Healthy ♂ and ♀	263 ml (201-346) (interquartile range)	6/16
Heslington [120]	Healthy ♀	212 ml 100-550 (range)	15/22
Salvatore [122]	Healthy ♀		2/21

Total DO 34/75 = 45%

Sørensen et al [119] examined 10 younger women (mean age 34 years, range 29-46 years) at 3 points of the menstrual cycle, filling the bladder with body temperature fluid at 60 ml/min. Because the variables showed little systematic variation during the cycle, average values are given here. First sensation of filling occurred at 347 ml ± 101 ml (SD) supine and 357 ml ± 126 ml seated. Maximum bladder capacity was 482 ml ± 103 ml, supine and 491 ml ± 147 ml, seated. No detrusor overactivity was observed.

The same authors [118] investigated 12 healthy postmenopausal females, mean age 59 years, using similar parameters. The means of 2 studies showed: First sensation of filling, supine, at 396 ml ± 163 ml (SD); and sitting at 331 ml ± 168 ml. Maximum bladder capacity, supine, at 551 ml ± 223 ml; and sitting at 489 ml ± 196 ml. They reported no detrusor overactivity.

Heslington and Hilton [120] examined 22 asymptomatic healthy female volunteers, performing conventional cystometry and ambulatory study in a random order. Detrusor overactivity was observed in 18% on conventional cystometry and 68% on ambulatory monitoring.

As indicated above, most studies of ambulatory monitoring in healthy subjects have revealed high percentages with detrusor overactivity (typically of modest amplitude [116,124]). However, Salvatore et al, [122] by using 2 bladder catheters simultaneously and only 'counting' detrusor overactivity shown by both, suggested that some of this apparent overactivity was a measurement artifact, and that 90% of a group of 26 healthy women showed no detrusor overactivity on ambulatory monitoring. This conclusion remains to be confirmed by others.

2. Compliance

Compliance can be judged in 2 ways: either from the raw detrusor pressure rise on filling the bladder, or from the ratio of the filled volume to this pressure rise (**Figure 9**). Normal (high) compliance implies only a small pressure rise, or a high value of the volume/pressure-rise ratio.

In 17 healthy subjects, using a filling rate of 35 ml/min, [116] the range of compliance values was very wide (range = 11-150 ml/cm H_2O; mean ± SD = 46 ± 40 ml/cm H_2O). Such a mean value implies only a small rise in pressure on filling (a few cm H_2O), consistent with a high compliance. Sørensen et al [115] reported similar mean values for compliance in a group of healthy females: 62 ml/cm H_2O initially and 58 ml/cm H_2O on re-examination 2 years later.

Similarly, Robertson [123] showed that in 17 healthy male and female volunteers the median detrusor pressure rise on conventional filling cystometry was 5 or 6 cm H_2O at filling rates of 50 and 100 ml/min respectively. On ambulatory monitoring the median pressure rise was 0 cm H_2O, however, a significantly smaller value.

Consistently, among 22 healthy female volunteers, conventional cystometry and ambulatory study performed in random order showed a significantly larger detrusor pressure rise on filling in conventional as compared with ambulatory studies. [120]

Although it is clear from these figures that conventional filling cystometry provokes higher pressures than ambulatory monitoring with natural filling of the bladder, this is not necessarily a disadvantage. Robertson [123] reported cystometric variables for 6 patients with "low-compliance bladders" due to neuropathy. Conventional cystometry showed pressure rises of 18 (± 7) and 46 (± 13) cm H_2O during bladder filling at rates of 20 and 100 ml/min respectively, but the pressure rise during natural (ambulatory) filling was much smaller (in fact, hardly distingui-

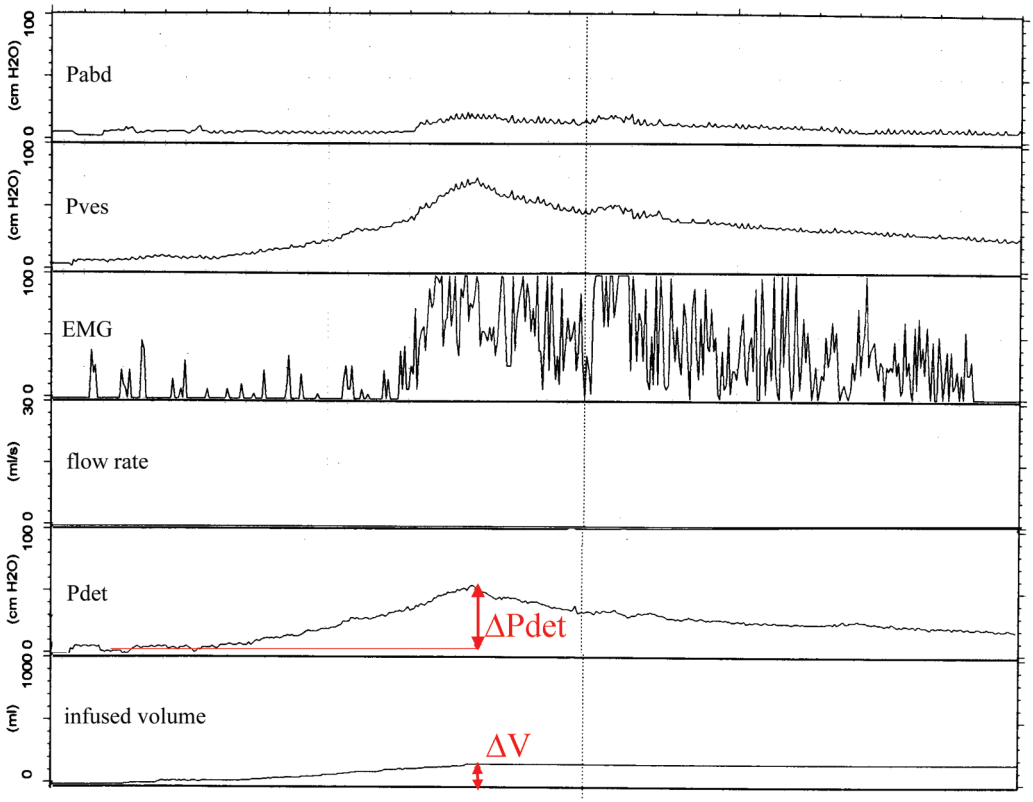

Figure 9. Example of a bladder with reduced compliance. As the bladder is filled the detrusor pressure rises by $\Delta p_{det} \approx 50$ cm H_2O. The corresponding increase in bladder volume $\Delta V \approx 150$ ml. The compliance $\Delta V / \Delta p_{det} \approx 3$ ml/cm H_2O, a value much smaller than the normal range of about 30 ml/cm H_2O upward. Reduced compliance is a urodynamic observation, not a diagnosis.The causes can range from fibrosis to elevated muscle tone.

shable from normal): 6 (± 4) cm H_2O. The corresponding compliance values were 8 (± 2) and 6 (± 3) ml/cm H_2O for conventional cystometry (abnormally low), but 114 (± 32) ml/cm H_2O for natural filling (a normal value). Thus, in fact, conventional filling cystometry exaggerates the abnormality – a diagnostic advantage. It seems clear that in some patients low compliance observed during fast filling becomes detrusor overactivity when natural bladder filling is used. [124]

3. Summary: Normal sensations and bladder capacity

Mean or median values, and SD's or ranges, are shown in Table 3 (normal values) above. It does not appear that the temperature of the filling fluid, the rate of filling, or the position of the patient, have much effect on the volumes at which various sensations are felt, or on bladder capacity. Much more striking is the variability from centre to centre: first sensation of filling for example occurs at about 100 ml in one centre but at about 350 ml in another, a value large enough to provoke a strong desire to void in the first. Thus it is clear that the various sensations during bladder filling are not precisely enough defined to be applied uniformly in all centres. It is likely that the patients' emotional state differs in different laboratories, and affects the reported bladder sensations.

The bladder capacity is somewhat less variable from centre to centre, its mean varying from 340 to 570 ml. Apart from this inter-centre variability, the inter-subject variability of all the parameters is also substantial, with a standard deviation of about 100 ml in order of magnitude. Some of this variation represents genuine differences between subjects, but it should be noted that the within-subject variability is also quite large, in order of magnitude 50 ml (Table 2).

To try to abstract normal values from the above is difficult but, as a guideline in healthy subjects, and omitting some of the more inconsistent values, first sensation of filling typically occurs at about 170 ml, first or normal desire to void (the International Continence Society makes no distinction [4]) at 250 ml, and strong desire to void at about 400 ml; maximum cystometric bladder capacity is about 480 ml. It is interesting that the mean voided volume on ambulatory monitoring falls between first sensation of filling (on cystometry) and first/normal desire to void. Thus, in daily life, the bladder is usually emptied long before a strong or even a "normal" desire to void would be felt on cystometry, although much more can be held if voiding has to be postponed. Correspondingly, there is a discrepancy in bladder capacity especially in patients with detrusor overactivity, depending on the method of measurement (uroflowmetry, voiding diary or cystometry). [125]

4. Summary: Detrusor overactivity in normal subjects

Detrusor overactivity is shown during conventional cystometry by up to 17% of normal subjects with a mean percentage of about 11% (Table 3). The percentage is much higher (up to 69%, see Table 4) on ambulatory studies, although one group disputes this. In any case, detrusor overactivity is not an all-or-nothing condition, but an observation that may be made, on some occasions and under some circumstances, in healthy subjects without neuropathy and without the symptoms typical of detrusor overactivity. In some cases, of course, apparently idiopathic detrusor overactivity may in fact be associated with abnormality of the neural control system that remains unrecognized because of the inadequacy of our testing.

III REPRODUCIBILITY, RELIABILITY AND NORMAL VALUES OF URETHRAL AND LEAK POINT PRESSURE MEASUREMENTS

1. Urethral pressure profilometry

a) Normative and comparative data for maximum urethral closure pressure (MUCP)

Table 5 shows values of maximum urethral closure pressure that have been obtained by different authors in normal (or, at least, without urodynamic stress incontinence) and abnormal female populations. It has several striking features. The first is the between-centre variability in the values reported, with mean maximum urethral closure pressure varying from 36 to 101 cm H_2O in non-stress-incontinent subjects. The second is the large between-subject standard deviation reported in most studies (from 10 to 52 cm H_2O). The third is that, in spite of this variability, in every study the mean maximum urethral closure pressure was smaller in stress-incontinent patients than in non-stress-incontinent women, sometimes significantly so and sometimes not.

Table 5. Maximum urethral closure pressure in stress-incontinent women and women without proven urodynamic stress incontinence, from Weber [126]

First author	Stress incontinence	No stress incontinence	P value
Awad [127]	35.9 ± 16.3 n = 20	101 ± 52.0 n = 10	< 0.001
Bunne [128]	21.2 ± 20.9 mm Hg n = 11	44.4 ± 15.2 mm Hg n = 10	< 0.01
Hendriksson [129] 30-39	41.1 ± 12.0 mm Hg n = 10	54.4 ± 15.1 mm Hg n = 10	< 0.05
Hendriksson [129] ages 40-49	36.5 ± 11.2 mm Hg n = 33	49.2 ± 13.4 mm Hg n = 12	< 0.01
Hendriksson [129] ages 50-59	32.4 ± 9.6 mm Hg n = 29	40.7 ± 12.8 mm Hg n = 10	< 0.05
Hendriksson [129] ages 60-69	29.4 ± 14.6 mm Hg n = 13	36.2 ± 10.2 mm Hg n = 10 ns	
Kaufman [130]	35.9 ± 1.2 (SEM) mm Hg n = 86	46.4 ± 2.5 (SEM) mm Hg n = 32	< 0.001
Godec [131]	42.0 ± 27.0 n = 66	56.0 ± 27.0 n = 31	< 0.05
Rud [132]	37 n = 24	38 n = 6	ns
De Jonge [133] supine, empty	45.4 ± 17.6 n = 38	61.9 ± 21.0 n = 28	
De Jonge [133] standing, full	48.8 ± 20.3 n = 38	60.6 ± 19.3 n = 28	
Kujansuu [134]	46.3 ± 11.8 mm Hg n = 15	49.5 ± 9.6 mm Hg n = 14 ns	
Kach [135]	57.9 ± 20.5 n = 78	93.9 ± 16.9 n = 44 ns	
Richardson [136] abnormal	46 ± 30 n = 30	65 ± 35 n = 5	ns
Richardson [136] normal	59 ± 28 n = 43	67 ± 35 n = 59	ns
Versi [137]	28.1 ± 5.3 (SEM) n = 95	49.2 ± 6.0 (SEM) n = 114	< 0.01
Cadogan [138]	34.1 ± 16 n = 40	49.7 ± 17 n = 16	= 0.0036
Versi [139]	25.4 ± 40.8 n = 70	42.2 ± 24.6 n = 102	< 0.01
Thind [19]	20	45	

Mean values of MUCP are in cm H_2O except where noted. ± = SD, except where noted. SEM = standard error of mean

Although, clearly, some of the variations shown in the table are the result of different patient populations, and others are the result of technical errors (see critique in section B.I, Stress incontinence), a weighted averaging of the mean values suggests that a normal maximum urethral closure pressure is about 54 ± 25 cm H_2O (SD). In stress-incontinent women the corresponding figures are 39 ± 24 cm H_2O. Clearly there is so much overlap that this test can hardly be useful for diagnosis [126] (see section D.I.2, subsection Severity of stress incontinence).

Studies providing normative data for men are scant, but there are some data on the urethral pressure profile in normal males. [140,141]

b) Reliability of urethral pressure variables

1. Within-patient reproducibility of maximum urethral closure pressure

In clinical practice, use of a fluid-perfusion technique to measure resting urethral pressure profile parameters such as maximum urethral closure pressure yields a standard deviation varying from 3.3 to 8.1 cm H_2O. On average the standard deviation is approximately 5 cm H_2O (95% confidence limits ± 10 cm H_2O) or ± 5%. With a microtip transducer technique the standard deviation varies between 3.3 and 16.5 cm of water, which means that the 95% confidence limits may be as high as ± 33 cm H_2O. The coefficient of variation when using the microtip transducer technique has been reported to be 17% (95% confidence limits ± 34%).

Inter-observer variability has been scarcely investigated. In one study using fluid perfusion the coefficient of variation of maximum urethral closure pressure varied from 3% to 11% (95 % confidence limits 6% - 23%). [142]

Reproducibility depends on the position of the patient (sitting position seems to have a higher SD compared with the supine position) and also whether other manoeuvres are performed during testing, such as straining or coughing (see next paragraph).

Stress urethral pressure profile. In the measurement of stress urethral pressure profiles the "stress" may be provided either by repeated coughs or by Valsalva manoeuvres. Stress profile variables show greater variability than static profile variables. The pressure transmission ration is the ratio between the urethral and intravesical pressure changes, on Valsalva or coughing. It varies from point to point along the urethra (see Figure 3). The *within-subject standard deviation* for the pressure transmission ratio varies between 13 and 18.5 % (95% confidence limits up to ± 37 %). The coefficient of variation has been estimated to be 20% (95% confidence limits ± 39%).

2. Normal aging

It is well established that in women the urethral pressure profile changes with age. A recent study of 255 women, ages 20-77 years, without detrusor overactivity, overt neuropathy or pelvic or incontinence surgery, confirmed that the maximum urethral closure pressure is negatively associated with age ($r = -0.489$, $P < 0.0001$). [37] This study was not a randomized trial but one that used a convenience sample of patients.

c) Summary: urethral pressure variables

Reproducibility of urethral pressure profilometry parameters is poor. The lack of reproducibility is due to large within-patient and between-patient variability, secondary to both biological variation and variation within the test procedure itself (which is related in part to lack of standardization).

2. Leak point pressure

The bladder pressure (p_{det} or p_{ves}) at which involuntary expulsion of urine from the urethral meatus is observed is the leak point pressure (LPP). The rise in bladder pressure causing leakage may originate either from the detrusor (caused for example by the filling of a low-compliance bladder, see Figure 9) or from an increase in the abdominal pressure (Figure 4). Thus there are two different leak point pressures – the detrusor leak point pressure and the abdominal leak point pressure. In stress incontinence we are interested only in the latter. The abdominal pressure increased during the test is produced voluntarily by coughing (cough leak point pressure) or by Valsalva (Valsalva leak point pressure, VLPP). The baseline that is used to judge the Valsalva leak point pressure is not definitively standardized, and this can lead to gross discrepancies between values reported by different laboratories (see Figure 4 in section B.I, Stress incontinence).

a) Conclusion

Leak point pressure is not consistently defined and techniques are not standardized. This renders any

comparison of findings between studies difficult. The elements which contribute to the confusion include: 1) the basic definition of leak point pressure (baseline value of pressure; route of measurement – urethral or rectal), 2) whether Valsalva or cough is used to produce leakage, 3) technique to confirm urine loss, 4) location of catheter, and shift in location on cough or strain, 5) caliber of catheter (if transurethral) 6) type of pressure sensor, 7) volume in bladder, 8) rate of prior bladder filling and 9) patient position. [5]

b) Reliability

There are very few studies in which the reproducibility of the leak point pressure has been investigated.

c) Within-patient variability

McGuire and coworkers [143] found a SD of 5.4 cm H_2O yielding a true 95% confidence interval of 89-111 cm H_2O. In another study where the microtip transducer technique was used the true value was found to vary between 72 and 128 cm H_2O in the standing position and between 61 and 139 cm H_2O when semirecumbent.

There are no reports on ***inter-observer*** variability.

d) Conclusion: leak point pressure

Reproducibility of leak point pressure is poor, due to both biological variation and variation within the test procedure itself (related in part to lack of standardization).

IV DIAGNOSTIC PERFORMANCE OF FILLING CYSTOMETRY AND AMBULATORY MONITORING

1. Sensitivity and specificity of filling cystometry in urge incontinence

a) Sensitivity, specificity, and positive and negative predictive values

The performance of a diagnostic test is usually described by its sensitivity, specificity, positive predictive value and negative predictive value. Classically, it is the sensitivity (etc) of the test for the condition that is reported. The presence or absence of the condition is assumed to have been established by some other "gold standard" (e.g. symptoms plus signs). The definitions are based on the following table:

		Condition = "gold standard"	
		present	**absent**
Test	positive	a	b
	negative	c	d

a, b, c, and d are the numbers of patients in each category.

The sensitivity (etc) of this test for the condition is:

sensitivity = a /(a + c)

specificity = d /(b + d)

positive predictive value (PPV) = a /(a + b)

negative predictive value (NPV) = d /(c + d)

In urodynamics however (for example in urodynamic stress incontinence) the test is assumed to define the condition. Therefore the test is apparently the gold standard and the diagnostic performance of the symptoms is judged against it. The situation is reversed and the table is flipped:

		Condition = "gold standard"	
		present	**absent**
Symptom	present	a	c
	absent	b	d

The sensitivity (etc) of the symptoms for the test result (the condition) are:

sensitivity = a /(a + b)

specificity = d /(c + d)

positive predictive value (PPV) = a /(a + c)

negative predictive value (NPV) = d /(b + d)

Comparing the 2 tables, the sensitivity of the test for the symptoms is the same as the positive predictive value of the symptoms for the test result, and similarly for the other parameters.

In the case of urge incontinence and detrusor overactivity the situation is even more confusing, because it is not clear what the condition in question is: urge incontinence, detrusor overactivity, detrusor overactivity incontinence, or none of these? This has to be made clear in each report of sensitivity and specificity.

In reality, neither a test result nor symptoms and signs can be taken as a "gold standard". Each pro-

vides independent but imperfect evidence about the probability of an underlying abnormality, and both should be taken into account. Sensitivity and specificity calculations simply provide information about the degree of concordance of the test result and the symptoms and signs.

b) Detrusor overactivity incontinence

Many authors have tested the sensitivity and specificity of the symptomatic presentation (urge incontinence) for *detrusor overactivity*. There are 2 objections to this: (a) *detrusor overactivity* may be a normal variant; therefore, from the point of view of this consultation, the sensitivity and specificity for *detrusor overactivity incontinence* should be tested; (b) detrusor overactivity or detrusor overactivity incontinence is a urodynamic observation which is inherently variable and does not define a condition.

Table 6, reproduced from the previous Consultation, [1] shows that some authors have found quite high positive sensitivity, specificity and predictive value of symptoms for urodynamic stress incontinence, but they have had more difficulty with detrusor overactivity incontinence and still more with mixed incontinence.

Van Waalwijk van Doorn et al [152] made careful tests of the sensitivity and specificity of non-ambulatory urodynamic observations (a) for any incontinence and (b) for urge incontinence. Among 348 women of mean age 41 years with symptomatic incontinence, urodynamics demonstrated incontinence in only 164, a sensitivity of only 164/348 = 47%. For a similar group of men of mean age 44 years the corresponding figures were 31/83 = 37%. Thus a principal reason for the discrepancy between urodynamics and symptoms was not that urodynamics revealed an unsuspected abnormality, but that it did not reproduce the symptom at all. In a similar group of 102 female patients with voiding complaints but without symptoms of incontinence, urodynamics revealed no incontinence in 96/102, a specificity of 94%. For men the corresponding figures were 71/75 = 95%. Thus uro-

Table 6. Value of patient history for predicting urodynamic findings, evaluated by various methods and in various female patient populations, from Homma et al [1]

Author	Year	Sample size	Urodynamic stress incontinence			Detrusor overactivity incontinence		Mixed incontinence	
			STV	SPT	PPV	STV	SPT	STV	SPT
Jensen [144]	1994	Review	0.91	0.51	0.75	0.74	0.55	0.48	0.66
Handa* [145]	1995	101	0.77	0.44	0.52				
Handa* [145]	1995	101	0.82	0.59	0.70				
Haeusler [146]	1995	1938	0.56	0.45	0.88	0.62	0.56		
Cundiff [147]	1997	535	0.44	0.87	0.87	0.71	0.41	0.68	0.48
Videla [148]	1998	72			0.82				
Diokno* [149]	1999	76	0.83	1.0	1.0				
James [150]	1999	555			0.81				
Lemack* [151]	2000	174			0.92				

STV= sensitivity, SPT = specificity, PPV = positive predictive value

* = predictive values are for type II stress urinary incontinence (i.e. with hypermobility)

dynamics is highly unlikely to demonstrate incontinence if there is no complaint of incontinence.

Among 154 female patients (in the same study [152]) in whom urodynamics reproduced incontinence, the observation of pure detrusor overactivity incontinence had a sensitivity of 25/28 = 89% for symptomatic pure urge incontinence; its specificity was 103/126 = 82%; and its negative predictive value was 97%. Although these figures may sound encouraging, its positive predictive value was only 25/48 = 52%. In large part this low value was due to a group with mixed symptoms who, not unreasonably, revealed detrusor overactivity incontinence on urodynamics. Taking account of the patients with mixed symptoms does not however improve the overall agreement between symptoms and urodynamic findings, because nearly half of them proved to have isolated urodynamic stress incontinence on urodynamics. Similar findings for stress incontinence are discussed below, in section D.I.2.

Griffiths et al [83] examined 100 older men and women, median age 79.5 years, with incontinence proven on 24-hour monitoring. The type of incontinence was believed to be urge in the majority. During filling cystometry (room temperature fluid, filling rate 60 ml/min, supine and seated) incontinence was not demonstrated in 32%; i.e. the sensitivity of conventional urodynamics for (urge) incontinence was 67%.

Thus urodynamic testing is not very sensitive for symptomatic incontinence in general, but it is highly specific. Among those in whom leakage is demonstrated, detrusor overactivity incontinence strongly suggests either urge or mixed incontinence, while observation of leakage not due to detrusor overactivity pleads strongly against pure urge incontinence.

c) Detrusor overactivity alone

The sensitivity, specificity and predictive value of *symptoms* for *detrusor overactivity*, given in a recent review of papers, [153] are shown in **Table 7**. (Note that this sensitivity and specificity are equal to the positive and negative predictive values of *detrusor overactivity* for corresponding *symptoms.*)

In spite of the wide variations shown in Table 7, all authors agree that the correspondence between

Table 7. Sensitivity, specificity and predictive value of symptoms obtained on patient history for the urodynamic observation of detrusor overactivity

First author	# patients	Sensitivity	Specificity	Predictive value	
				Positive	Negative
Awad [154]	108	0.96	0.25	0.82	0.67
Bent [155]	81	0.83	0.49	0.32	0.91
Cantor [156]	214	0.91	0.45	0.80	0.79
De Muylder [157]	408	0.62.	0.47	0.62	0.48
Glezerman [158]	128	0.40	0.86	0.27	0.92
Hilton [159]	100	0.77	0.38	0.44	0.72
Jarvis [160]	100	0.91	0.45	0.54	0.87
Korda [161]	537	0.47	0.63	0.44	0.66
Lagro-Janssen [162]	103	0.84	0.77	0.67	0.90
Ouslander [163]	135	0.89	0.21	0.49	0.68
Phua [164]	84	0.84	0.31	0.82	0.84
Sand [165]	218	0.78	0.39	0.80	0.78
Summitt [166]	79	0.46	0.76	0.57	0.46
Thiede [167]	196	0.88	0.39	0.86	0.88
Valente [168]	102	0.74	0.97	0.88	0.74
Walters [169]	106	0.35	0.91	0.67	0.35
Sandvik [170]	40	0.56	0.96	-	-
Cundiff [147]	102	0.71	0.87	0.41	-
Haeusler [146]	130	0.62	0.56	0.64	0.39
Fantl [171]	17	-	0.64	0.57	-

symptoms and detrusor overactivity is far from perfect.

Van Brummen et al [172] examined the sensitivity of *detrusor overactivity,* observed during conventional cystometry, for urge incontinence. They examined 95 women, with symptomatic overactive bladder, symptomatic stress incontinence, and/or prolapse. Symptoms were assessed by a bladder diary and conventional filling cystometry was performed (sitting, fill rate 60 ml/min). Urinary frequency, urgency and urge incontinence had similar associations with the cystometric observation of detrusor overactivity (**Table 8**). Among patients with one of these symptoms, detrusor overactivity was not observed in 77-81%: i.e., there were large numbers of 'false negatives'.

Looking just at the association between detrusor overactivity and urge incontinence in Table 8 [109,172], there was again a substantial number of 'false negatives' – subjects with urge incontinence but no detrusor overactivity. Correspondingly the specificity and negative predictive value of detrusor overactivity were low (15/67 = 22%; 27/79 = 34%). It thus appears again that one of the principal problems is failure to reproduce the symptom – in this case to evoke detrusor overactivity, even without leakage. This must be viewed mainly, although not exclusively, as a consequence of poor urodynamic technique.

Table 8. Detrusor overactivity and symptoms [172]

	Detrusor overactivity		
	Yes	No	*P*-value
Frequency			
Yes	16 (23)	54 (77)	0.01
No	0 (0)	25 (100)	
Urgency			
Yes	12 (19)	50 (81)	0.56
No	4 (12)	29 (88)	
Urge incontinence			
Yes	15 (22)	52 (78)	0.03
No	1 (4)	27 (96)	
Nocturia			
Yes	15 (23)	51 (77)	0.02
No	1 (4)	28 (96)	
Frequency and urgency and nocturia			
Yes	11 (23)	36 (77)	0.11
No	5 (10)	43 (90)	

Values are numbers (%).

d) Detrusor overactivity and overactive bladder syndrome (OAB)

Other recent papers report investigations of detrusor overactivity in patients with the symptomatic overactive bladder syndrome (not necessarily urge incontinence). The relevance of this work to the problem of urge incontinence is difficult to ascertain, since it compares a urodynamic observation that is not necessarily related to incontinence with a set of symptoms that also may not include incontinence.

Digesu et al [173] reported a retrospective review of 4500 women aged 22-73 years. Neurological disorders were excluded. As shown in **Table 9**, not only was there a substantial proportion of 'false negatives', but also a large number of 'false positives' – patients with detrusor overactivity but without OAB symptoms. The sensitivity and specificity of detrusor overactivity for overactive bladder were 457/843 (54%) and 2473/3657 (68%) respectively, while its positive and negative predictive values were 28% and 86% respectively.

Table 9. Overactive bladder symptoms and detrusor overactivity, from reference [173]

	Detrusor overactivity	No detrusor overactivity	Totals
OAB symptoms	457	386	843
No OAB symptoms	1184	2473	3657
Totals	1641	2859	4500

Hyman et al [174] examined 160 men, mean age 61 ± 15 years, without neuropathy but with symptoms "suggestive of detrusor overactivity." They observed detrusor overactivity in 68, suggesting a sensitivity of 43% for overactive bladder symptoms. Detrusor overactivity was seen more often with urge incontinence than with symptoms of frequency, urgency, nocturia, suggesting a rather higher sensitivity for urge incontinence.

These papers suggest that in non-neurogenic dysfunction failure to reproduce the symptom (i.e. to demonstrate detrusor overactivity in subjects with overactive bladder symptoms) is common, while on the other hand detrusor overactivity is also observed in symptom-free subjects, consistent with its status as a normal variant.

e) Distinguishing or defining characteristics of detrusor overactivity

Several groups have attempted to find characteristics of detrusor overactivity that may distinguish incontinence with different aetiologies, or incontinence of different severity.

One group [69,86] examined 132 patients with overactive bladder symptoms (with and without neurological disease). Based on the characteristics of their involuntary contractions, patients were divided into 4 categories: *type 1*- no evidence of involuntary detrusor contractions on videourodynamics; *type 2* - involuntary detrusor contractions present, and patient aware and able to abort them; *type 3* - contractions present, patient aware and able to contract the sphincter but not to abort contractions; and *type 4* - contractions present and patient unaware but unable to contract the sphincter or abort contractions. There was no significant relationship between category and severity of symptoms as judged by voiding frequency, functional bladder capacity, or pad test. The authors concluded that the characteristics of the involuntary contractions were not distinct enough to aid in differential diagnosis, but that the ability to abort detrusor overactivity and stop incontinent flow might have prognostic implications, especially for the response to behavior modification, biofeedback training, and pelvic floor exercise. Thus, although interesting, this classification does not appear very promising as a diagnostic tool.

Defreitas and coworkers [175] examined three groups of patients: group 1, men with lower urinary tract symptoms (LUTS) and no known neurological condition with DO (n = 22); group 2, men with Parkinson's disease and LUTS (n = 39); and group 3, women with Parkinson's disease and LUTS (n = 18). Patients with Parkinson's disease had a significantly lower median volume at first detrusor contraction than those with non-neurogenic DO. The percentage of group-1 patients with urge incontinence was significantly lower than that found in the other two groups (9% versus 54% and 56%, P <0.001 and 0.002, respectively). No statistically significant correlation between the duration or severity of Parkinson's disease and urodynamic parameters was found. The distinction between Parkinson's disease proper and multiple system atrophy, which appears to be important with regard to bladder dysfunction, [176] was unfortunately not made in this study.

In the study cited above, [174] 160 older men without neuropathy but with symptoms "suggestive of detrusor overactivity" were examined. Detrusor overactivity was seen more often with urge incontinence than with symptoms of frequency, urgency, nocturia. The bladder volume at which detrusor overactivity was observed tended to be lower in those with urge incontinence and frequency and/or urgency than in the rest (P = 0.07). The prevalence of detrusor overactivity was similar in men with and without bladder outlet obstruction.

Miller et al [66] suggested that functional bladder capacity was smaller in those with more severe incontinence as judged from voiding diary (see further discussion below).

To summarize, it appears that there may be slight characteristic differences between various types or severity of detrusor overactivity/urge incontinence, but it has not yet been shown that they have diagnostic or prognostic value.

f) Provocative manoeuvres

Because of the low sensitivity of urodynamics for urge incontinence, provocative manoeuvres are nearly always used in an attempt to increase it. In publications, the effect of provocation has nearly always been related to the observation of detrusor overactivity only, not detrusor overactivity incontinence.

It has long been known that only 50% of DO occurs during supine cystometry without provocation. The remaining 50% is revealed only by posture change, standing cystometry, on provocation by cough, or on catheter removal. [22,23,65]

Awad and McGinis [154] observed detrusor overactivity in 30% of female patients in the supine position versus 61% in the standing position.

Webster et al [177] found that in 52% of women with detrusor overactivity, provocation by fast filling in the standing position, and with exercises such as coughing, was required to reveal it.

Investigative technique, in particular the inflation of a balloon in the proximal urethra, [178] or the instructions given to the patient, [179] affects the frequency of the observation of detrusor overactivity.

Choe et al [80] systematically examined which manoeuvres were most provocative of detrusor overactivity. In 134 women with symptomatic urge incontinence they performed gas (CO_2) cystometry.

Six provocative manoeuvres were performed consecutively to evoke detrusor overactivity, including lying supine, rising to a seated position, walking toward the bathroom, handwashing, coughing and

sitting on the toilet with instructions not to void. By filling to maximum capacity and performing these manoeuvres in 2 different orders, they were able to demonstrate detrusor overactivity in 76/134 subjects (67%). Sitting on a toilet with full bladder and with instruction not to void was the most provocative manoeuvre, responsible for revealing detrusor overactivity in 52 of the 76 (68%). Handwashing was a distant second, revealing overactivity in 15 of the 76 (20%). Other manoeuvres revealed very little detrusor overactivity. Note that these authors unfortunately used gas cystometry, which is not a currently recommended method. Even with these provocations, detrusor overactivity could not be demonstrated in one-third of subjects with symptomatic urge incontinence; thus the sensitivity is poor.

An extreme provocative manoeuvre is the bladder cooling (ice water) test advocated by Geirsson et al. [98] The empty bladder is filled with water at a temperature of less than 10 ^{o}C. This stimulates C-fibers that normally carry afferents from receptors sensitive to temperature and pain. In infants stimulation of these receptors can initiate a detrusor contraction, but this response is normally lost at ages over 5 years. It is believed that in some types of neurogenic bladder these afferent signals again become involved in the voiding reflex and can stimulate detrusor contraction. Consequently, in adults, involuntary expulsion of the cold water within a period of 1 minute suggests neurogenic detrusor overactivity. The bladder cooling test stimulated detrusor contraction (neurogenic detrusor overactivity) in 91-97% of patients with traumatic upper motor neuron lesion, but in only 47% of those with presumed idiopathic detrusor overactivity. Detrusor contraction was not observed in any patient with a lower motor neuron lesion or pure (urodynamic) stress incontinence. Thus the bladder cooling test is highly sensitive for neurogenic detrusor overactivity, and highly specific for detrusor overactivity in general. However, its sensitivity and specificity for ***idiopathic*** detrusor overactivity are not particularly high.

2. Ambulatory urodynamics: sensitivity and specificity

Ambulatory urodynamics is performed in an effort to capture more realistic or more physiological observations, especially of incontinence episodes. Thus, similar to provocative manoeuvres, it is an attempt to increase sensitivity (by providing a longer time for overactivity to manifest itself). Because ambulatory monitoring usually demonstrates detrusor overactivity in a large proportion of symptom-free subjects, this observation has low specificity for urge incontinence. If used to detect detrusor overactivity *incontinence*, however, ambulatory monitoring may be valuable, since it is difficult to see how actual leakage could be observed in patients without symptomatic incontinence. The authors of a review article [180] concluded that ambulatory monitoring detects more actual incontinence than conventional cystometry; i.e., it is more sensitive in this respect.

Radley et al. [181] found that ambulatory monitoring revealed detrusor overactivity in 70/106 women with symptoms suggestive of bladder overactivity (twice as many as conventional cystometry with provocation by handwashing), and that it detected detrusor overactivity incontinence in 40 of the 70. The observation of detrusor overactivity incontinence was correlated with symptom severity, but it was not clear how many women complaining of urge incontinence showed detrusor overactivity incontinence. Therefore the sensitivity is unknown.

3. Summary: Diagnostic performance of filling cystometry and ambulatory monitoring, in the setting of the overactive bladder symptom complex

The phrase “diagnostic performance,” suggested by the ICUD, reflects a narrow and simplistic view of the goals of (uro)dynamic testing. It suggests that there is a standard against which performance can be judged. In fact, neither symptoms nor the results of testing provide such a standard. Symptoms may be associated with different abnormalities as revealed by dynamic testing. The results of testing on the other hand have to be interpreted with knowledge of their variability, of their systematic changes from one test to the next, and of the overlap in the values measured in normal subjects and in patients. Even the crucial observation of detrusor overactivity can be made in healthy volunteers or missed in patients with clear symptoms of urge incontinence. Thus dynamic testing, by itself, does not provide a diagnosis but provides a basis of understanding of lower tract (dys)function which, together with the symptoms, will help to establish a diagnosis and choose therapy. Such understanding is particularly important when the condition investigated is complex and uncertain.

There is a natural tendency to regard the variability of urodynamic observations and their loose

association with symptoms as a disadvantage. However, these characteristics are a manifestation of the normal operation of the nervous control system, which has to accommodate the need for periodic bladder emptying on the one hand to the social and emotional life of the individual on the other. Variability of response, depending on the emotional state and social circumstances of the individual, is an essential part of this system; in fact, it is the point of the system. Fortunately, if there is serious LUT dysfunction due to subpontine neurological disease, the influence of the emotional nervous system is reduced, and the results of urodynamic testing are more reproducible. In patients without such disease, variability of response to testing may be taken as evidence of an intact neural control system.

Future research should be directed at understanding the sources of variability of urodynamic testing (and symptoms), with a view (1) to improving diagnosis by controlling variability; and (2) to utilizing the variability to develop new therapies, since physiological variation implies the possibility of therapeutic manipulation.

V THERAPEUTIC PERFORMANCE OF FILLING CYSTOMETRY AND AMBULATORY MONITORING

1. Prediction of treatment response

a) *Filling cystometry*

The authors of a recent review of papers from 1980-2000 [153] (see **Table 10**), concluded that "it is not possible to correlate the results of urodynamic tests with the effects of non-invasive therapy."

Consistent with the table, Malone-Lee et al [84] reported on 356 female patients with overactive bladder symptoms. On urodynamics, 266 showed detrusor overactivity. There was no significant difference (between those with and without detrusor overactivity) in treatment outcome after 6-8 weeks of oxybutynin and bladder retraining.

On the other hand, previous reviews have concluded that women with incontinence and detrusor overactivity respond less well to *surgery* for stress incontinence than those without detrusor overactivity. [1]

Friis et al [185] conducted a blinded prospective study to evaluate the usefulness of urodynamic examination compared to clinical diagnosis. The study showed that when urodynamic examination was added to the preoperative planning of treatment for female urinary incontinence a more beneficial cure rate was found if the patient was treated in accordance with the urodynamic findings. However, the patient material is small and the power of the study is weak.

A retrospective study has shown that a careful minimal evaluation may be adequate to identify intrinsic sphincter deficiency (ISD), predict postoperative voiding difficulties and maximize surgical outcomes. [186]

A recent Cochrane review concluded that current evidence is insufficient to demonstrate a clear improvement in clinical outcomes as a result of performing urodynamic studies. [6]

In men, fewer studies have been done. Golomb et al [187] examined whether preoperative urodynamic examination allows us to predict the risk of incontinence after radical prostatectomy. A small group of 20 patients underwent radical retropubic prostatectomy for prostate cancer. Urodynamics showed detrusor overactivity in 12/20 pre-operatively. 5 of these 12 suffered from urge incontinence post-operatively. The positive predictive value of preoperative detrusor overactivity for post-operative incontinence was thus only 42%.

Table 10. Response to medical treatment for urinary incontinence in subjects with negative or positive results of urodynamic tests, from reference [153]

First author	# patients	Treatment	Urodynamics	Results
Wagg [182]	290	Oxybutynin and retraining	Cystometry	No relationship between urodynamic variables and response to treatment
Hashimoto [183]	77	Oxybutynin 6 mg/day for 4 weeks	Cystometry	No difference in effect of oxybutynin in motor and sensory urge
Holtedahl [184]	87	Estriol and pelvic floor exercise and bladder training + electrical stimulation if requested	Cystometry + urethral pressure profile	Outcome similar in subjects with/without urodynamically confirmed diagnosis

b) Ambulatory monitoring

Brown and Hilton [188] used conventional and ambulatory urodynamic monitoring to study the incidence of detrusor overactivity before and after colposuspension. They showed that preoperative ambulatory monitoring was unable significantly to predict which patients would suffer from urgency postoperatively, nor even which women would demonstrate detrusor overactivity post-surgery.

Another paper addressed specifically the effect on clinical management of doing ambulatory urodynamics. [189] In this retrospective chart review of 71 women there were technical difficulties in 30/71 ambulatory studies although only 2 were not interpretable. 32/71 women showed detrusor overactivity and were nearly all treated with medication. Among the remainder without detrusor overactivity fewer received medication. However, fewer than half of those who received medication improved. The authors concluded that ambulatory urodynamics was not very helpful in deciding on management.

2. Can urodynamic testing improve clinical outcome?

The ultimate goal of clinical urodynamics is to improve the basis for choosing the correct therapy and hence improve outcome. However there is a remarkable paucity of high quality prospective randomized controlled studies to elucidate this issue. Similarly there are few good studies of whether urodynamics can predict the outcome of incontinence treatment. The studies that have been performed provide no evidence that it might do so to any helpful extent.

However, before instituting irreversible treatment, especially in complex cases, comprehensive urodynamic testing to provide the knowledge on which treatment choice should be based, and to establish the baseline situation pre-treatment, should be considered mandatory.

The committee has no doubt that urodynamics plays an important role in clinical work, and this is not so much because it directly predicts or improves clinical outcomes as because it illuminates the pathophysiological situation, enabling a rational choice of treatment. However the central problem is that urodynamics often has to be introduced into the clinical work-up without any standardisation, without a sound physiological/pathophysiological basis and without sufficient documentation of its utility.

D. CLINICAL APPLICATIONS OF URODYNAMIC STUDIES

I. EVALUATION OF THE FEMALE PATIENT

1. Introduction

The clinical evaluation of an incontinent woman is based on the combination of the medical history and physical examination, complemented as appropriate with selected urodynamic tests. The purpose and manner of testing are discussed more fully in the introductory section of this chapter.

2. What is usually evaluated?

a) Stress incontinence

1. Type of incontinence

Stress incontinence is a clearly defined symptom, which often can be objectively demonstrated (i.e. by a sign – see section A.IV, Definitions and terminology) without any urodynamic measurement. Traditionally, provocative cystometry has been the core test for the urodynamic diagnosis of urodynamic (genuine) stress incontinence (USI). Videourodynamics, stress urethral pressure profilometry and ambulatory urodynamics have also been used.

Previous studies have found significant variation of the predictive value of symptoms in identifying 3 urodynamic observations i.e. urodynamic stress incontinence, detrusor overactivity and mixed incontinence. [190,191] This variation can at least partly be explained by inhomogeneous patient materials and lack of consistency in the clinical and the urodynamic diagnosis of stress incontinence. A carefully performed study [152] showed that, in a group of 154 females with both complaints and urodynamic demonstration of incontinence, the observation of pure urodynamic stress incontinence had a sensitivity and specificity of 90% and 65% respectively for the isolated symptom of stress incontinence. The positive and negative predictive values were 60% and 91%. The relatively low values were due to a substantial number of patients with mixed symptoms who showed pure urodynamic stress incontinence.

The figures given in a recent review [191] are slightly different: the isolated symptom of stress incontinence had a positive predictive value (PPV) of 56%

for the observation of pure urodynamic stress incontinence (USI) and 79% for USI with additional urodynamic abnormalities. The corresponding negative predictive values were 66% and 42% respectively. The PPV of stress incontinence in association with other symptoms was 77% in detecting USI (with or without additional urodynamic abnormalities). A positive cough stress test had a PPV of 55% for detecting pure USI and 91% for the "mixed condition" (USI and additional abnormalities). These predictive values were based on an average prevalence of pure USI of 41%.

In view of these discrepancies between symptoms and urodynamic findings, the soundest basis for the clinical diagnosis and evaluation of stress incontinence and urge incontinence remains to be established, as does the significance of the corresponding urodynamic observations such as urodynamic stress incontinence, detrusor overactivity incontinence and detrusor overactivity.

Besides their diagnostic potential, urodynamic investigations can contribute qualitative and quantitative information about the underlying or coexisting pathophysiology.

2. Pathophysiology of stress incontinence

There is a clear need to understand better the pathophysiology of stress incontinence (see section B.I, Stress incontinence). Neither the clinical nor the pathophysiological value of classification into types 0-III, [27] or of distinguishing between hypermobility and intrinsic sphincter deficiency (ISD), has ever been documented in prospective studies. The term intrinsic sphincter deficiency has gained much attention over the last two decades. Clinically, intrinsic sphincter deficiency has been defined as "... sphincter weakness..." because "...the urethral sphincter is unable to coapt and generate enough resistance to retain urine..." . [192] This clinical term has never been conceptualized into sound urodynamic parameters. Conventional static urethral parameters such as maximum urethral pressure or maximum urethral closure pressure (MUCP), or cough profile parameters such as pressure transmission ratio and leak point pressure cannot be used to characterize the extent and the type of urethral dysfunction [5,54] (See critique in section B.I, Stress incontinence). Thus conventional urethral pressure profile parameters do not provide reliable pathophysiological information. Possibly urethral elastance may turn out to be a more useful variable for characterizing intrinsic sphincter deficiency. [62]

3. Severity of stress incontinence

There is no consensus on how to measure severity of stress incontinence clinically or urodynamically. Severity can be expressed on the basis of simple clinical measures such as questionnaires, a voiding and incontinence diary or pad weighing tests. Some studies have shown correlation between the clinical severity of stress incontinence and urethral pressure profile parameters. However, a recent critical review concludes that static urethral pressure profilometry parameters such as maximum urethral closure pressure or cough profile parameters such as pressure transmission ratio cannot be used to characterize the severity of incontinence. [5] Nevertheless, abdominal leak point pressure (LPP) measurement shows promise as a useful tool to quantify urethral dysfunction associated with stress incontinence. [54]

4. Aspects of urodynamic studies relevant to therapy for stress incontinence

Most retrospective studies show higher failure rate after surgery in women with low maximum urethral closure pressure (often defined as MUCP $\leq$ 20 cm H_2O [5]). However, other investigators have shown that a low maximum urethral closure pressure is not an efficient predictor of surgical failure. [190] The value of leak point pressure in predicting outcome of surgery remains to be established. Nevertheless, there are data to suggest that leak point pressure may be a sensitive indicator of changes in incontinence status, reflecting treatment effect. [54]

5. Voiding difficulties after surgery

Surgery for stress incontinence may lead to voiding difficulties. [193] At the moment risk factors (including urodynamic risk factors) for delayed resumption of voiding are not well defined. The type of surgery clearly plays a role: TVT is less obstructive than Burch colposuspension. [194] One major problem is that at the moment there is no clear urodynamic definition of obstruction or detrusor underactivity in women. It has been reported that low maximum flow rates (< 20 ml/s), [195] or "inadequate contraction strength" defined as p_{det} <15 cm H_2O during voiding (but note that this signifies a low urethral resistance, not necessarily a weak detrusor contraction), or significant use of the Valsalva manoeuvre when voiding, are associated with postoperative voiding difficulties. [196-198] The possibility exists that, in women who do not use their detrusor during voiding, detrusor contractility may be assessed preoperatively by mechanically interrupting the flow. [199,200]

5. Postoperative urgency

The preoperative symptoms of urge incontinence and urgency, and the urodynamic observation of detrusor overactivity, have each consistently been shown to be associated with poorer surgical outcome in patients with mixed incontinence. Several studies where the amplitude of detrusor overactivity was graded have shown that the risk of persistent urgency was more closely associated with high-pressure detrusor overactivity ($p_{det} \geq 25$ cm H_2O) than low-pressure overactivity. [95,201,202] Consequently cystometry may allow a more precise selection of patients who respond well to surgery despite concurrent urge symptoms. The success rate after anti-incontinence surgery in patients with low-pressure contractions seems to be similar to the success rate in those without detrusor overactivity, [95,201-203] but the success rate in women with high-pressure contraction is less than 50%. [95,201-203]

On the other hand preoperative urge symptoms resolve in a substantial proportion of patients (50-65%). [204-206] One untested hypothesis is that surgical correction of the bladder outlet may prevent ingress of urine into the proximal urethra (which if it occurs, may induce detrusor overactivity in some patients). [207-210]

De novo urge incontinence has been reported to occur in 10-20% of patients after surgery. [193] There is scant information on clinical or urodynamic risk factors; possibly the type of surgery plays a role. It should be remembered however that de novo ***detrusor overactivity*** may merely represent detrusor overactivity that was missed preoperatively.

6. The role of urodynamic studies in predicting occult stress urinary incontinence in women due to be treated for pelvic organ prolapse (POP).

Because 11 to 22% of continent women undergoing vaginal repair for a large cystocoele develop stress incontinence following surgical repair, [211,212] it would be helpful to devise methods to evaluate patients who are at risk for this complication. [213] Many women with severe pelvic organ prolapse do not have incontinence symptoms in daily life, but may develop it when the prolapse is reduced, so-called "occult" stress incontinence. Voiding difficulty and bladder outlet obstruction may coexist with occult stress incontinence; all may be associated with pelvic organ prolapse, and all may be altered if the prolapse is reduced during urodynamic testing. Based on the literature, advanced age, incontinence before development of pelvic organ prolapse, and extensive dissection at the time of the repair, seem to increase the risk of postoperative incontinence (level of evidence 3).

The overall incidence of occult stress incontinence was 25% when videourodynamic testing was performed with and without pessary support of the bladder base during stress manoeuvres. [214] In a small group of patients with severe genitourinary prolapse occult incontinence was found in 59%, and about 20% (4/22) were reported to demonstrate stress pressure profiles suspect for stress incontinence after pessary placement. [215] However, it should be remembered that stress profiles are not particularly reliable measurements (see section B.I, Stress incontinence). A special technique (Scopette, Birchwood Lab) for reducing prolapse during multichannel urodynamics revealed a 56% incidence of low-pressure urethra (possibly related to intrinsic sphincter deficiency, but see caveats regarding urethral pressure profiles above) and an overall incidence of occult stress incontinence in 83% of patients with massive pelvic organ prolapse but without clinical urinary incontinence. [216] As expected, urethral hypermobility is correlated with the degree of prolapse. [217] Surprisingly, so too is detrusor overactivity (revealed by prolapse reduction), although impaired detrusor contractility and intrinsic sphincter deficiency were not significantly associated with prolapse in this one study.

The literature thus emphasizes the importance of urodynamic assessment with prolapse reduction to assess potential occult stress incontinence and possibly detrusor overactivity. [213,214,218,219] However, although occult stress incontinence is revealed in a high proportion of cases with severe prolapse, there seem to be no studies assessing reproducibility, a particularly important point since the technique of reduction is variable and non-standardized. Similarly there seem to be no prospective studies that test whether occult incontinence revealed by prolapse reduction in fact manifests itself after surgery, if no particular precautions are taken to avoid it, or whether urodynamic measurements made during prolapse reduction in fact lead to improved clinical outcomes.

Many different tests (e.g. pessary test, speculum test) have been used to document "occult" incontinence. However all of them seem to overestimate the risk since they are positive in 50-77% of patients. In various papers the Pereyra and Kelly procedures have been used prophylactically but there is no docu-

mentation that they reduce the risk of postoperative incontinence (level of evidence 1 B). [220-222] A prospective controlled study [223]showed that the pessary test was positive only in 50% of incontinent women and was falsely positive in 72% (level of evidence 1 B). Another study [219] concluded that urodynamic testing before a pelvic organ prolapse operation was not cost-effective. Thus there is no reliable test that can show who is at risk (level of evidence 1 B).

7. Urge incontinence

Urgency and urge incontinence are poor predictors of the urodynamic observation of detrusor overactivity, as discussed in section C.IV on that topic. The positive predictive value of the symptom complex overactive bladder (frequency, urgency and/or urge incontinence) for urodynamically determined detrusor overactivity is only around 50%. [173] On the other hand detrusor overactivity is reported in 10-69% of asymptomatic female volunteers, depending on the definition and type of cystometry. [22,116,120,123,190,224] (See further discussion in section C.II, Cystometry: Normal values.)

• Pathophysiology and severity of urge incontinence

As discussed in section B.II (Urge incontinence), it remains a challenge whether detrusor overactivity can be classified into different pathophysiological groups. For example it might be categorized into "true" (idiopathic?) detrusor overactivity and "secondary" detrusor overactivity due to ingress of urine into the proximal urethra and subsequent induction of the micturition reflex. It remains unclear if the chronological sequence of bladder and urethral pressure changes may distinguish between those categories. [225]

No statistically significant relationship between the various cystometric variables and reported symptom severity has been established.

3. Recommendations for urodynamic studies in women with incontinence

a) Recommendations for clinical practice:

1 Non-invasive urodynamics (voiding and incontinence diary, post-void residual, and possibly uroflowmetry) is recommended for all incontinent patients.

2 The committee finds that invasive urodynamic studies are not necessary prior to treatment in situations where the type of incontinence is clear and there are no complicating factors, particularly if planned treatment is reversible . These include:

- uncomplicated stress incontinence (symptomatic pure stress incontinence with no symptoms or signs of voiding difficulties)
- uncomplicated urge incontinence (symptomatic pure urge incontinence with no symptoms or signs of voiding difficulties)

3 It is recommended that, whenever there is doubt about the pathophysiology, or about whether the incontinence is uncomplicated or not, then invasive urodynamics should be performed in order to provide the knowledge on which rational treatment decisions or prognosis can be based. The investigation should be tailored to the individual patient; typically this means that it will be a comprehensive examination of multiple aspects of storage and voiding function, and not just of the incontinence itself.

4 The committee further recommends action:

- to promote new or existing urodynamic tests and parameters which have a sound technical and physiological basis
- to discourage the use of tests and procedures which are not soundly based
  - e.g. stress urethral pressure profile as currently performed

5 An important dimension of urodynamics is to provide information which may be of value for prognosis and patient counselling. It is recommended:

- in patients with mixed incontinence where surgical intervention is considered
- in incontinent patients who have symptoms or signs of voiding difficulty as well

b) Recommendations for research:

1 The committee recommends research programs:

- to more clearly establish the technical and physiological basis of the urodynamic observations that are made in women with incontinence
- to design and conduct randomized studies that may provide objective documentation of the utility of soundly based tests
- to conduct studies that may provide objective evidence of the utility of performing urodynamics in

near-normal subjects and in defined patient groups

(note that the more complex and morbid the pathophysiological situation, the greater the differences between patients, and thus the more important it is to do urodynamics in order to obtain knowledge of what is to be treated)

- to develop new tests, for example tests of urethral properties, which have a sound technical and physiological basis

2 The committee recommends that no new therapy should be introduced without extensive urodynamic testing of all accessible aspects of its effect on lower urinary tract function and dysfunction.

II. PATIENT EVALUATION: MEN

1. Introduction

Although the incidence of urinary incontinence in men is generally regarded as much lower than in women, this is not necessarily true for all patient groups, especially when dealing with the elderly (See section D.V, Patient evaluation: Frail elderly). In both sexes those who lose urine, whatever the cause may be, increase in number with advancing age. [226] In this section the characteristic pathologies that provoke incontinence in men are discussed from the point of view of urodynamic testing. Although urinary incontinence related to bladder outlet obstruction or prostatic surgery is most frequently encountered, other important pathological conditions such as bed wetting and post-micturition dribbling are also clinically relevant. The information in this section is based mainly on references from 2000 or later. Older references can be found in the reports of the previous Consultation. [1,227]

This section is organized according to the suspected origin or cause of incontinence or related lower-urinary-tract symptoms, reflecting the very varied aetiologies responsible for the condition in men.

2. What is usually evaluated?

a) Lower urinary tract symptoms (LUTS) related to bladder dysfunction

LUTS are fairly common among men of 50 years and over. Incontinence is not usually prominent but, if it is present, urodynamics can be helpful to establish the underlying vesical/urethral dysfunction, in particular, bladder outflow obstruction (BOO) or detrusor overactivity.

Non-invasive investigations such as uroflowmetry and measurement of post-void residual urine (PVR) are easy to perform, but even in simple situations can only give clues to the underlying pathology.

Moreover, since elderly people often suffer from comorbidities – e.g. insidious neurogenic ailments such as Parkinson's disease, multiple sclerosis, cerebrovascular disease or diabetes mellitus – invasive urodynamic tests are frequently necessary to finalize the diagnosis and plan any intervention, especially if surgical treatment is considered.

Urodynamics may be useful for selecting candidates for unusual treatments. For example, Vandoninck et al. [228] used urodynamics to evaluate patients with overactive bladder (OAB) symptoms who were to be treated with percutaneous tibial nerve stimulation. Overall, the objective and subjective success rates of stimulation were 56% and 64%.

The treatment did not abolish detrusor overactivity, but subjects without detrusor overactivity at baseline were 1.7 times more likely to preferably respond to percutaneous tibial nerve stimulation than those with detrusor overactivity.

• Recommendations for urodynamic investigation in those with LUTS related to bladder dysfunction

Patients who suffer from non-complicated bladder outflow obstruction (BOO), without incontinence, need urinary flow study, PVR measurements, IPSS (International Prostate Symptom Score) and frequency-volume charts prior to surgical intervention, but probably do not need to be investigated with invasive urodynamic study[229,230] (Grade of recommendation B).

However, men with ***incontinence*** and a suspicion of urethral/vesical dysfunction form a much more restricted group with complicated, disparate, and uncertain diagnoses. In some the incontinence may be an initial symptom; in others it may follow treatment (see the following section on post-prostatectomy incontinence).

Evidence that urodynamics improves outcome is limited, but nevertheless the committee recommends that all such patients should receive a complete urodynamic evaluation in order to understand the problem that is to be treated and that surgeons should plan surgical interventions only after scrutinizing the lower urinary tract by urodynamics. (Grade of recommendation C).

b) Post-prostatectomy incontinence

1. General

After prostatectomy a considerable number of patients suffer from urinary incontinence. The incidence following this surgery varies widely depending on patient age, bladder function, definition or degree of urinary incontinence, benign or malignant diseases and the type of surgery. Understanding of prostato-vesical anatomy and the pelvic floor and meticulous surgical technique are of prime importance in preventing these distressing symptoms. Urodynamic studies are important to establish the aetiology and provide a rational basis for treatment.

Kondo et al [227] have analyzed the aetiology of urinary incontinence following surgery for benign prostatic hyperplasia or prostatic cancer using accumulated data from 573 patients reported in 8 articles from 1978 to 1997. Urodynamic studies suggested that the most common aetiology was sphincter weakness (urodynamic stress incontinence), present in 34% of patients, followed by sphincter weakness plus detrusor overactivity in 33%, and detrusor overactivity or detrusor overactivity incontinence alone in 26%. Other aetiologies including low compliance and urethral stricture were responsible for the remaining 7%. Thus, although sphincter weakness was present in two-thirds of patients, a blanket assumption that it is the only cause of incontinence would be wrong in two-thirds. Clearly urodynamic testing is needed for diagnosis, and perhaps to choose treatment.

2. TURP, Open Prostatectomy and Thermal Treatments

Approximately 1% of patients who have undergone TURP suffer from post-prostatectomy incontinence. [231] Is urodynamics required prior to treatment? Das et al. [230] performed holmium laser resection of the prostate in 100 men patients for management of LUTS without performing any urodynamic studies except for a urinary flow rate. Their follow-up study 2 years later revealed that persistent incontinence remained in only 1, who required pads, and that the International Prostate Symptom Score (IPSS) and maximum flow rates improved from 21.6 to 6.8 points and from 7.5 to 21.6 ml/s, respectively. This report does suggest that if patients are appropriately selected, post-operative urinary control is quite satisfactory, leaving only about 1% of cases with urinary incontinence. This implies that the role of urodynamic investigation before laser resection of the prostate may be marginal.

After failed treatment, however, there may be an important place for urodynamics. Kuo [232] urodynamically evaluated 185 men aged from 55 to 91 with a mean of 75 years who had had variable LUTS after TURP and had been refractory to conventional treatments. He found that urinary incontinence was present in 74 patients (40%) and that bladder outlet obstruction and detrusor hyperactivity with impaired contractility (DHIC) were the most common aetiologies for post-prostatectomy incontinence, followed by detrusor overactivity. Since these diagnoses imply quite different treatments, urodynamic investigation has an important role.

3. Radical Prostatectomy and Radiotherapy (RT)

• Radical retropubic prostatectomy

Radical retropubic prostatectomy for prostatic cancer results in much higher incidence of post-prostatectomy incontinence than TURP. In physician-reported studies, the incidence of total incontinence is a few % and the incidence of stress incontinence requiring some degree of protection is about 10%. [233] In studies based on patient self-report, however, the incidence of any degree of incontinence is 66% and the incidence of pad use is 33%. [234] One of the main determinants of prevalence is the time following surgery, since continence is regained after radical prostatectomy over the first year. [235] Continence after radical prostatectomy depends on minimizing the injury to the striated urethral sphincter and the use of well-designed surgical techniques. [236] Patients who undergo a nerve-sparing radical prostatectomy appear to have a better chance of achieving continence than those undergoing standard radical prostatectomy. [237] Recent enhancements to the nerve-sparing prostatectomy may preserve external sphincter function and shorten the time to achieve post-operative continence. [237]

Given the above, is invasive urodynamic investigation of this type of incontinence necessary?

Groutz et al [238] evaluated 83 men of mean age 68 years, who were consecutively referred for persistent urinary incontinence following radical retropubic prostatectomy. They reported that urodynamic stress incontinence was the most common urodynamic finding (73 patients, 88%), followed by detrusor overactivity in 6, bladder outlet obstruction in 1, impaired detrusor contractility in 1, and normal findings in 2. Of 73 men diagnosed as having urodynamic stress incontinence, 27 suffered from pure urodynamic stress incontinence, and the remaining 46 had conco-

mitant bladder disorders such as impaired detrusor contractility (22 men), bladder outlet obstruction (14 men) or detrusor overactivity (10 men). The authors reported that 25 of the 83 men had what they called "low urethral compliance". This non-standard term expressed the fact that there was a difference of more than 10 ml/s between maximum free uroflow and maximum invasive (pressure-flow) uroflow. This term is not recommended, however, as it implies a cause for these observations that may not be correct.

McCallum et al [239] reported that 21 of 180 men who had been treated for incontinence following radical prostatectomy still remained incontinent 2 years later. 16 of the 21 were evaluated, and half had urodynamic stress incontinence together with urge incontinence or decreased bladder compliance. The authors emphasized that stress incontinence was one of the predominant symptoms but that comorbid detrusor dysfunction had to be taken into consideration, as well as intrinsic sphincter deficiency (ISD), in order to properly treat persistent post-prostatectomy incontinence.

From the above, whether urodynamics is required depends on the treatment contemplated. Before implanting an artificial urethral sphincter one would certainly want to rule out significant detrusor overactivity, but before conservative therapy by pelvic floor muscle exercises there is no obvious need for urodynamics.

• Radiotherapy

There are few manuscripts reporting the effects of radiotherapy in those with prostatic cancer, and even fewer discussing the place of urodynamics. [240, 241]

Henderson et al. [242] assessed the clinical role of urodynamics in the selection of prostate cancer patients for brachytherapy with a minimum dose of 145 Gy. 100 consecutive patients were assessed after implantation. Prior to the treatment an unselected group of 57 of the 100 patients had been evaluated urodynamically: normal detrusor function (no detrusor overactivity) was found in 48 and detrusor overactivity in 9. No patients had permanent urinary incontinence and 2 required surgery for bladder outlet obstruction. Acute urinary retention developed in 7 patients, clean intermittent catheterization was utilized by 27, and 89% of patients had a deterioration in their LUTS with the worst symptoms 6 weeks after implantation. They found that those who postoperatively had acute retention or preferred to utilize clean intermittent catheterization had either larger prostatic volumes (> 35 ml) or were urodynamically obstructed. Consequently they concluded that urodynamics might have an important role in the selection of treatment for men with early prostate cancer, in particular to improve the outcome of brachytherapy. However, the evidence for this conclusion is weak.

Gomha and Boone [243] treated 86 patients who were incontinent following prostatectomy with implantation of an artificial urinary sphincter and assessed whether or not prior radiation affected surgical outcomes in those who had or had not had radiotherapy. The aetiology of the urinary incontinence in group I (without radiation) was radical prostatectomy in 55 patients, TURP in 2, orthotopic ileal reservoir in 1; in group II (with radiation) the aetiology was radiation with salvage prostatectomy in 5, adjuvant radiotherapy after radical prostatectomy in 20, and radiotherapy and TURP in 3. Urodynamic study prior to artificial sphincter implantation revealed that detrusor overactivity was much more prevalent in group II (radiation, 26%) than in group I (no radiation, 5%), a significant difference ($P = 0.04$). In spite of this, post-operative urgency with or without urge incontinence was found in similar proportions of the 2 groups (47% of group I and 43% of group II), and similar proportions wore 0 to 1 pad a day to protect against incontinence (60% and 64% respectively). Thus pre-operative urodynamics did not predict outcome.

Castille et al [244] prospectively assessed 229 men who were scheduled to undergo radical retropubic prostatectomy with preoperative urodynamics in an attempt to help physiotherapists predict postoperative incontinence. They observed that all men diagnosed as having detrusor overactivity or bladder outlet obstruction were incontinent 6 weeks after operation but had improved 4 months later. They stressed that detrusor overactivity and bladder outlet obstruction in those undergoing radical retropubic prostatectomy are significant risk factors for postoperative incontinence, although only for a short period of time. Thus the role of pre-operative urodynamics is limited.

4. Recommendations for Urodynamics in those having Post-Prostatectomy Incontinence

Sphincter weakness, bladder outlet obstruction, detrusor overactivity and mixed incontinence are significant aetiological factors contributing to post-prostatectomy incontinence. [227,232,239] These parameters can be only identified by urodynamics, which is considered by most, [243,245,246] but not

all, [247] to be one of the main tools for investigating this type of incontinence (Level of Evidence 3). In brachytherapy for prostate cancer urodynamics may have some value for predicting which men might develop acute urinary retention or might require intermittent catheterisation after treatment. [242]

To summarize, it is urodynamics that clearly identifies the real aetiology of urinary incontinence following prostatectomy or following radiation, in the clinical setting. [232,239] It is recommended in every case of this sort (Grade of recommendation C).

c) Nocturnal enuresis and Parkinson's disease related to male incontinence

Several ailments or pathological conditions have been reported to be closely associated with male incontinence, e.g., neurological diseases, prior radiotherapy, neurogenic detrusor overactivity, diminished bladder compliance, nocturnal enuresis, postmicturition dribble and terminal dribbling. [227]

1. Nocturnal enuresis

Nocturnal enuresis in adult males is rather rare. Sakamoto and Blaivas reported important and interesting observations based on data of over 3000 patients referred for the evaluation of LUTS. [246] They found that 8 of 3277 patients (0.02%) had adult onset nocturnal enuresis without daytime enuresis. All these patients were male, with a mean age of 63 years (48 to 80 years) and all suffered from bladder outlet obstruction with mean maximum urinary flow rate 8.5 ml/s, mean International Prostate Symptom Score 12.6, and mean post-void residual urine 350 ml (50 to 489 ml). The authors identified hydronephrosis in 5 of the 8 patients, a bladder diverticulum in 3/8, vesico-ureteral reflux in 4/8 and low bladder compliance in 4/8. Five of the 8 underwent TURP resulting in improved symptoms. Thus bladder outlet obstruction is one of the distinct pathologies that can provoke nocturnal enuresis with variable LUTS, but it is certainly not the only pathological factor. For example, Hunsballe [248] found increased delta activity in electroencephalography among adult primary enuretics compared to normal controls. Invasive urodynamics may be justified in such patients, because it is the only way of reliably identifying bladder outlet obstruction.

Another factor contributing to nocturnal enuresis is the presence of a neobladder. Nocturnal enuresis plagues nearly 28% of such patients. Indeed, 25 of 30 patients (83%) who underwent the Stanford pouch ileal neobladder had nocturnal enuresis 1 year later. [249] Patients older than 65 years are at greater risk because of the physiological increase in nocturnal diuresis associated with aging. An orthotopic neobladder produces variable lower urinary tract dysfunction, including both failure to empty the bladder and failure to store urine. The urodynamic behavior depends on the type, length, and configuration of the bowel segment used. [250] There may be overdistension, elevated residual urine volume, lack of sensation, reduced maximum urethral closure pressure, or more frequent and higher-pressure detrusor overactivity. [251] Urodynamic investigation is important to establish the diagnosis and select treatment, although one group suggested that bed wetting could be simply alleviated by waking up at least twice per night to void. [249]

Stroke is a common and well-known cause of lower urinary tract dysfunction in the older population. The predominant symptoms are urinary frequency, urgency and urge incontinence (including night-time incontinence) while detrusor overactivity is the most common finding on urodynamics. [252] Detrusor-sphincter dyssynergia is uncommon. Consequently, conservative or pharmacologic treatment is often instituted without prior urodynamic investigation, even though the size and the site of the stroke do have an influence on urological findings. [253] Simple tests such as uroflowmetry and – especially – post-void residual urine may be useful if poor bladder emptying is suspected. Cystometry has limited value: the prevalence of detrusor overactivity in older people is quite high even in the absence of stroke (perhaps 10% in women and 25 to 35% in men). Therefore the observation of detrusor overactivity alone is not definitive, while the observation of detrusor overactivity incontinence merely confirms what one would suspect in any case.

2. Parkinson's disease

Parkinsonian diseases are known to significantly influence bladder function (see section D.V.6). Detrusor overactivity incontinence is common, [175] but urodynamic findings differ in different diseases of this group. [254] In multiple system atrophy for example – as opposed to Parkinson 's disease – post-void residual urine volume >100ml, detrusor-sphincter dyssynergia, or EMG evidence of internal or striated sphincter denervation are common. Such findings, especially that of elevated residual urine, may influence choice of treatment. Particularly if there is residual urine, invasive pressure-flow studies may be indicated to diagnose or rule out bladder outlet obstruction.

3. Recommendations for urodynamic investigation for men suffering from nocturnal enuresis or Parkinson's disease

Nocturnal enuresis in adult males is rare but problematic, and it is associated with many possible aetiologies. The committee recommends that urodynamic evaluation should be conducted in all cases (Grade of recommendation C).

When a male patient complains of LUTS and suffers from coexisting diseases such as Parkinson's disease, multiple sclerosis or other neurological deficit, he should be thoroughly investigated by means of urodynamics (Grade of recommendation C).

There are no recent manuscripts on post-micturition dribble or terminal dribbling. When a complaint of terminal dribbling is objectively identified in the urinary flow curve, urodynamic studies may be indicated to verify or rule out the presence of bladder outlet obstruction or urethral pathology (Grade of recommendation C).

III NEUROGENIC LOWER URINARY TRACT DYSFUNCTION

1. Introduction

In the previous Consultation [1] it was pointed out that neurogenic incontinence may express itself as urge incontinence, reflex incontinence, overflow incontinence or stress incontinence. Urge and stress incontinence have already been reviewed exhaustively in this present chapter; reflex and overflow incontinence are terms not currently recommended by the International Continence Society. They will be discussed below, together with other relevant information updated since 2001.

2. What is usually evaluated?

Because not all patients with neurogenic conditions develop typical urinary symptoms or urodynamic findings, a specific understanding of the dysfunction in each individual is an absolute prerequisite for the correct choice of therapy. [255-257] The aim is to describe the (dys)function of the bladder, the urethra and the pelvic floor, their coordination during filling and voiding, and their influence on other pathological conditions (e.g. autonomic dysreflexia) or organ systems (e.g. renal function). Except in a few diseases where empirical, conservative therapy can safely be instituted, or where lower urinary tract dysfunction is predictable (e.g. post-stroke), urodynamic investigation is required to provide the understanding of the situation on which rational treatment must be based. Even if empirical therapy is instituted without urodynamics, the progress of the patient must be carefully reviewed to determine whether urodynamics is needed after all.

Because many patients with neurological conditions show anatomical abnormalities that involve the lower urinary tract, or detrusor-sphincter dyssynergia (lack of coordination) that can be demonstrated easily by imaging, comprehensive videourodynamics is the test of choice. [255-259]

a) Special tests

The ***ice water (bladder cooling) test*** is sometimes used in an attempt to identify neurogenic detrusor overactivity (see section C.IV.1, subsection Provocative manoeuvres).

The ***carbachol test*** is intended to reveal supersensitivity to muscarinic agents following neurological decentralization, typically in bladders in which a voiding reflex cannot be demonstrated. [260] A subcutaneous injection of a muscarinic agonist (0.25 mg carbachol or bethanechol) is given and the detrusor pressure is monitored for 30 min or until it rises to over 20 cm H_2O. The test is considered positive if the detrusor pressure increases above 20 cm H_2O. [261] Results published more than 20 years ago report detrusor decentralisation in a variable proportion of patients with a positive carbachol test (from as few as 50% up to as many as 98%). [260,262,263] Thus in some hands (though not in all) it does not have a very good diagnostic performance and as a result it has fallen out of favour. A recent attempt to use it to help predict the results of sacral neuromodulation proved unsuccessful. [264]

b) Neurogenic detrusor overactivity incontinence ("Reflex incontinence")

Detrusor overactivity of neurogenic origin is frequently observation in association with neurological disease, and often leads to actual leakage, i.e. incontinence. Observed on urodynamics, this type of incontinence should be termed *neurogenic detrusor overactivity incontinence*. The corresponding symptom is variable: if the detrusor overactivity is accompanied by sensation (desire to void) it might be termed urge incontinence; frequently however any sensation is absent and so the term *urge* incontinence is misleading. For this reason the term *reflex inconti-*

nence was introduced, [3] implying automatic filling and emptying of the bladder without sensation or control. This term is no longer recommended. [4]

c) Detrusor-sphincter dyssynergia

Neurogenic detrusor overactivity is often accompanied by detrusor-sphincter dyssynergia: a neurogenically determined failure of coordination of detrusor and urethra. The failure of the urethral sphincter to relax, when the detrusor contracts, causes a functional urethral obstruction which may not only hinder bladder emptying, but may also permit the development of high detrusor pressures. If high pressures are present for prolonged periods in daily life, renal function may be endangered (see the following subsection on "overflow incontinence").

d) "Overflow incontinence"

Overflow incontinence is another term that is no longer recommended. [3] It means continual leakage from a constantly overdistended bladder. [3] The presenting symptom is usually characterized by continual small amounts of incontinence, exacerbated by stress, together with an inability to empty the bladder by voiding.

On urodynamics the usual corresponding observation is a bladder with low compliance and little or no detrusor activity; as the bladder is filled the detrusor pressure rises because of the poor compliance, until it reaches a value sufficient to open the urethral sphincter. Dribbling leakage then ensues. Clinically, the most important variable is believed to be the detrusor pressure at which leakage occurs, the detrusor leak point pressure. If this pressure is elevated, and if similar pressures are attained during continual leakage in daily life, then renal function is endangered because the constantly high detrusor pressure hinders outflow from the ureters. Conventionally, detrusor leak point pressures of 40 cm H_2O or more are believed to be unacceptable. There is level 3 evidence (mostly from pediatric studies) that upper urinary-tract deterioration is more probable when detrusor leak point pressure is elevated. [265-267] However, the evidence for a cut-off at 40 cm H_2O seems less clear.

3. Reproducibility and reliability of tests

Since many patients with neurogenic dysfunction of the lower urinary tract have severe subpontine neuropathy, the influence of the emotional (limbic) nervous system on lower-tract function is reduced or eliminated; thus, urodynamic observations may be less variable and more reliable than those made in individuals with an intact nervous system. Nevertheless, the test conditions (e.g. the rate of filling the bladder) do influence the results, and should be chosen carefully. [1]

4. Does urodynamic testing improve clinical outcome?

The aims of therapy for neurogenic lower urinary tract dysfunction are to achieve the most nearly physiological filling and voiding conditions [255-257] as well as a management situation acceptable to the patient in daily life. Long periods of elevated detrusor pressure during bladder filling or (abnormally prolonged) voiding put the upper urinary tract at risk [265-267] (Level of evidence 3). The primary aim of therapy in patients with such problems is conversion to a low pressure bladder during filling, [255,257] even if this leads to incomplete emptying. Adequate therapy depends on whether the detrusor is overactive or has reduced compliance, and only urodynamics can answer those questions unequivocally. Timely and adequate diagnosis is of paramount importance for the patient's quality of life. [256,257,268,269] Urodynamic investigation is essential for checking the efficacy of treatment and in following up any sequelae of the disease and its management.

To summarize, there is level 3 evidence that urodynamic testing improves clinical outcome in patients with continually elevated detrusor pressures. In many other types of neurogenic dysfunction, whether or not there is evidence that clinical outcome is improved, rational treatment is impossible without the knowledge that invasive urodynamic testing provides.

5. Recommendations for urodynamic studies in incontinence due to neurogenic lower urinary tract dysfunction

a) Recommendations for clinical practice:

The committee recommends that:

1 Every patient with suspected neurogenic dysfunction of the lower urinary tract should receive comprehensive urodynamic evaluation, to establish the state and function of the lower tract (Grade of recommendation B/C).

2 If possible, videourodynamic testing should be employed (Grade of recommendation C).

3 Anorectal function should be evaluated at the same time as urinary function (see section E on faecal incontinence) (Grade of recommendation C).

4 Urodynamic testing should be done in specialized centres by trained and certified personnel (Grade of recommendation C).

b) Recommendations for research:

1 Comprehensive urodynamic testing should form an essential part of the evaluation of new therapies such as botulinum toxin injection or intravesical instillation of capsaicin analogs.

2 New types of urodynamic study need to be developed to delineate more precisely the types of neurogenic dysfunction that arise from supraspinal abnormalities of the lower urinary-tract control system, which up to the present have been neglected.

IV PATIENT EVALUATION: CHILDREN

1. Introduction

The indications for urodynamic evaluation in children have been set out on neurological, anatomical and functional lines, with the types of studies to be performed being based on the underlying pathological conditions rather than on the presenting symptoms. The findings, efficacy and reliability of urodynamic studies for each of these conditions will be discussed.

Many of the conditions for which urodynamics is employed in children involve profound anatomical and neurological abnormalities, in which lower urinary tract dysfunction is variable, complicated, and unpredictable. Urodynamics is used to establish as clearly as possible the baseline situation, so that (1) changes as a result of treatment and/or growth can be assessed, and (2) some guidance is obtained in the choice of treatment (although urodynamics may not necessarily be the deciding factor). If urodynamics were not performed, treatment would be instituted in a blindfolded manner, without any understanding of the pathophysiology of the condition that was to be treated. In this situation, it is an ethical imperative that all possible information about the patient's problem should be gathered before a treatment approach – frequently irreversible – is decided upon. The existence of evidence that performing urodynamics improves treatment outcomes is of secondary importance, and unlikely ever to be fully obtainable. Here, therefore, perhaps more clearly than in any other patient group, the aim of urodynamics is to provide knowledge and understanding: i.e., knowledge-based medicine.

Of course, it is still important that the tests used should be reliable and reproducible in the patient population considered, and the evidence for this will be discussed.

2. Neurogenic bladder dysfunction

a) Myelodysplasia

For the last 20 years initial urodynamic studies very early in the neonatal period have been recommended for children with myelodysplasia, the basis being that they help identify children at risk for subsequent urinary tract deterioration or a changing neurological picture. [270] Detrusor overactivity on cystometry, detrusor underactivity during voiding, dyssynergia between the detrusor and sphincter (usually on electromyography (EMG) of the striated urethral sphincter), detrusor leak point pressure, and residual urine measurement are the key elements of a detailed urodynamic study that need to be measured. [271,272]

In an exhaustive review of the efficacy and reliability of urodynamic studies in newborns with myelodysplasia, [273] of 24 studies analyzed, 13 focused on EMG activity of the striated urethral sphincter or pelvic floor, 7 on bladder compliance and 2 on cystometric technique. Twenty-one studies were at level of evidence 4, 2 were at level 3 and 1 was at level 1. Nine of the 24 studies were performed at international sites and the remainder within the United States.

The urodynamic patterns of normal detrusor function (66%), acontractile detrusor (33%), overactive detrusor function (57%), and detrusor compliance, as well as detrusor-sphincter synergia (21%), dyssynergia (37%) and sphincter denervation (60%) were similar, with little variability across comparable studies. This suggests that urodynamic testing is valid and reproducible in infants and children with spina bifida. Bladder capacity was measured in only 1 study of 506 myelodysplastic children [274] and found to conform to the formula: capacity in ml = 24.5 x (age in years) + 62. This formula for the increase in bladder size with age is 25% less steep than published age-related bladder capacities in neurologically normal children [275,276] (e.g. 30 x age + 30). It pro-

bably reflects a truer picture of the functional bladder capacity for this condition. Whether or not it influences treatment regimens and the need for augmentation cystoplasty has not been addressed; thus, the clinical significance of this formula is still a matter of conjecture. However, those children who did not have detrusor overactivity had a bladder capacity similar to normals. Because neurological impairment affects detrusor elasticity (bladder compliance), cystometric fill rates also influence measured capacity and compliance as noted in 3 studies that addressed this issue. [274,277,278] The lower the fill rate the greater compliance and the larger the capacity.

Six studies evaluated the relationship between level of the neurological lesion (on clinical examination) and lower urinary tract function but none could predict a specific urodynamic pattern based on the level of the lesion. Sacral level lesions can be associated with an upper motor neuron lesion just as readily as the expected lower motor neuron findings. [279] Similar findings have been noted for children with thoracic or high lumbar level lesions where the incidence of sacral sparing is 45%.Pontari, 1995 #237}

Approximately 90% of children born with spina bifida will have a normal upper urinary tract at birth. Over time many children who have not received proactive urological care develop upper and/or lower urinary tract deterioration. The deterioration is an acquired phenomenon secondary to the development or progression of various lower urinary tract hostility factors such as neurogenic detrusor overactivity, poor bladder compliance, detrusor-sphincter dyssynergia and/or increased leak point pressures from denervation fibrosis. [270-272] Despite the fact that only one study was at level 1, all urodynamic studies corroborated their reliability by predicting upper urinary tract deterioration with 90% accuracy.

Not all authors consider that prophylactic treatment is beneficial, but all recommend periodic cystometry when new onset hydronephrosis, reflux or incontinence develops, the latter in children on a continence program already (i.e. clean intermittent catheterization and/or drug therapy). [280,281] When new onset incontinence not related to urinary infection nor easily treated by increasing current treatment regimens occurs, only 1 study has shown that repetition of cystometry, urethral pressure profilometry, and EMG measurements, is helpful in management. [282]

Monitoring the striated urethral sphincter EMG periodically during the first 3 to 6 years of life and then periodically throughout puberty when (1) incontinence develops despite strict adherence to bladder and bowel continence programs, (2) changes occur in leg function, or (3) the child experiences back pain or increasing scoliosis, is believed to be the most accurate way to detect a change in neurological impairment. Four studies noted that a considerable number of myelodysplastic children (40% to 61%) regardless of their neurological level have progressive neurological deficits as they grow up and reach puberty. [279,283-285] Two of these studies noted changes particularly early in infancy, while a third study noted changes throughout childhood. Thus, most clinicians agree that myelodysplasia is a dynamic disease process that changes as the child grows and that warrants constant vigilance over the neurological picture. Although its efficacy has not been proven, it is recommended that a cystometrogram/EMG be repeated 3 months following any neurosurgical intervention to correct a tethered cord, or spinal surgery to repair increasing scoliosis, for this provides a new baseline for comparison should further spinal cord tethering take place after corrective surgery.

b) Occult spinal dysraphism

Several series characterizing the preoperative urodynamic evaluation of children with occult spinal dysraphisms have documented abnormalities in striated urethral sphincter function (denervation and/or detrusor-sphincter dyssynergia) in 20 to 35% of babies under 2 years of age with normal neurological examinations, thus emphasizing the need for preoperative urodynamic testing in these children. [286-289] Four reports in older children revealed a greater correlation (70 – 90%) between an abnormal neurological examination and the likelihood of finding an abnormality on urodynamics. [286,290-292] A few studies demonstrated that between 10% and 20% of patients experience a loss in function from their surgery directly (most of whom had abnormal lower urinary tract function preoperatively), and a variable number, usually inversely related to age, have improved sacral cord function on postoperative assessment 3 or more months after surgery. [286,288,290-292] Two studies, both retrospective, revealed an efficacious response in EMG activity, with stabilization or improvement in up to 60%, on postoperative urodynamic assessment when the dysraphic state was corrected before 2 years of age. [286,290] When children were first operated on after 2 years of age, 2 urodynamic reports documented an additional 25 to 35% with progressive changes in urethral sphincter

function with axial growth, very few of whom had a detectable change on physical examination. [287, 290]

c) Sacral agenesis

Sacral agenesis, absence of the lowermost vertebral bony segments, is a lesion that is often missed in infancy because of its subtle clinical manifestations, with generally no loss of lower extremity motor and sensory function, and the non-progressive nature of its pathophysiology. [293,294] Urinary and/or faecal incontinence usually manifest themselves at an older age when the child fails to toilet train on time. A careful physical examination noting flattened buttocks and a short gluteal crease is pathognomonic for the diagnosis. In 8 reports that provided enough data, urodynamic studies had been 90% accurate in delineating the neurological deficit, which cannot be predicted by the level of absent vertebrae, [289,295-301] and in managing the incontinence and/or upper urinary tract abnormalities (i.e., hydronephrosis, reflux). These studies reveal that between 30 and 40% of these patients have an upper motor neuron type lesion with an overactive detrusor and an intact but dyssynergic sphincter, while 25 to 50% have signs of a lower motor neuron deficit with acontractile detrusor and denervation in the sphincter, and 15 to 20% have normal lower urinary tract function. [293,295,296] It is presumed that the neurological deficit associated with this entity is fixed because no study showed any progression of the neurological disorder with increasing age.

d) Spinal cord injury

The rarity and variability of spinal cord injuries in children make it difficult to propose any one treatment program unless the specific type of lower urinary tract function is known. [302] Even if the individual regains the ability to void spontaneously and empty his/her bladder, it is imperative to know the detrusor filling and emptying pressures. Even if the child is continent on clean intermittent catheterization, it is important to measure detrusor compliance in order to determine the potential risk for hydroureteronephrosis and reflux. [303] A poorly compliant bladder with or without elevated voiding pressures from detrusor-sphincter dyssynergia often leads to the development of hydroureteronephrosis and vesicoureteral reflux; when combined with urinary tract infection progressive renal failure is often the result. [304] Four studies extol the need for urodynamic studies once the spinal shock from the initial injury wears off, to determine the presence of low filling and voiding pressures and the ability for complete emptying, for the reasons cited above. [305-308] There are no published studies that deal with specific urodynamic findings for various levels of spinal vertebral lesions in children – all provide generalities but it is possible to glean some information from observations in adults. All studies are retrospective and use historical controls for comparison.

A thoracic or cervical level injury may produce an upper motor neuron deficit leading to poor compliance, high voiding pressures and incomplete emptying over time, secondary to detrusor-sphincter dyssynergia. [309-311] In the presence of elevated filling and voiding pressures, there is a 30% incidence of upper urinary tract deterioration. [266] Balanced voiding with pressures below 40 cm H_2O in the absence of detrusor-sphincter dyssynergia ensures a stable upper urinary tract. [307]

A cauda equina injury often leads to a lower motor neuron deficit of the striated sphincter that may not require any treatment whatsoever because the bladder empties readily at low pressure, but it probably necessitates medical and/or surgical therapy to achieve continence. Urodynamic studies are considered invaluable in describing lower urinary tract function and in efficaciously managing any dysfunction to maintain a healthy upper urinary tract and long-term survival with minimal morbidity. [304,308,312]

e) Cerebral palsy

Only a few published studies describe the urodynamic findings in children with cerebral palsy. [313-316] The vast majority of the children with cerebral palsy tend to toilet train completely, but often at an age that is later than expected for normal individuals. [313] Any incontinence is usually secondary to urgency from detrusor overactivity associated with an inability to be toiletted on time. [315] A meta-analysis of urodynamic studies performed in children with either persistent incontinence despite frequent toileting, or urinary tract infection, revealed either normal function (15%) or detrusor overactivity (73%; 85 of 117 patients);[313-315] very rarely is dyssynergia between the detrusor and urethral sphincter noted during voiding (5%; 12 of 249 patients). [279,314,316] Therefore, it has been suggested but not proven that cystometry and sphincter EMG are obligatory only when frequent toileting or anticholinergic therapy fails to control incontinent episodes, the child develops urinary infection from an inability to empty the bladder completely during voiding, or ultrasonography reveals hydronephrosis.

3. Imperforate anus

Imperforate anus is classified as high, intermediate or low depending on whether or not the rectum ends above, at or below the levator ani muscle. In the past, imperforate anus repair for high lesions frequently resulted in urinary incontinence due to a pudendal nerve injury that often occurs from a perineal approach to bringing the rectum down to the anal verge. [317] With the advent of the posterior sagittal anoplasty this complication has been eliminated as a cause for subsequent urinary incontinence, although bladder neck incompetence may be a consequence of extensive mobilization of the sigmoid colon which helps transfer the rectum to its final location. However, recent reports of spinal MRIs reveal a 35% incidence of distal spinal cord abnormalities in children with an imperforate anus. [318-320]

The VATER or VACTERL association is a group of diverse abnormalities that include Vertebral bony, Anal atresia, Cardiac, Tracheo-Esophageal fistula, Renal and Limb anomalies. [321] Imperforate anus may occur as an isolated lesion or in conjunction with this association. Spinal cord pathology occurs in 38% of cases producing a picture of upper and/or lower motor neuron deficits to the lower urinary tract. [300,319,322-324] By combining the incidences in 3 studies it was found that the presence of an abnormal sacrum increases the likelihood of neurogenic dysfunction to as high as 76% (38 of 50 children). [300,322,323] When the rectum ends above the levator ani muscle there is a much greater chance of neurogenic bladder dysfunction than when it ends below the pelvic floor [322] and the older the child is at the time of urodynamic assessment the more likely he/she is to have abnormal lower urinary tract function. [324,325] The reliability and reproducibility of these findings among the various studies analyzed confers an important role on videourodynamic studies as an integral part of the evaluation and management of these children: it has diagnostic accuracy; it provides a reason to explore any intraspinal abnormality to improve the child's chances of achieving urinary and faecal continence; and, it is useful as a basis for future comparison if incontinence should subsequently become a problem in children not undergoing early spinal cord exploration.

4. Anatomic abnormalities

The use of urodynamics for evaluating anatomic lesions that affect the lower and, consequently, the upper urinary tract in children is still somewhat controversial, although many clinicians now feel its usefulness is beyond question.

a) Posterior urethral valves

Nowhere is the above statement truer than in boys with posterior urethral valves. [326-328] Prior to the ready availability of urodynamics, persistent upper urinary tract dilation was managed with bladder neck resection and/or striated urethral sphincter resection. By demonstrating the presence of detrusor under- and/or overactivity, urodynamic studies have helped explain the radiological findings of hydroureteronephrosis that many of the children exhibited over time despite adequate valve ablation. These studies changed the focus from increased bladder outlet resistance to altered bladder function as the aetiology. In the only urodynamic report prior to valve ablation detrusor overactivity was seen in 60%, poor compliance in 10% and normal function in 30%. [329] In a series of urodynamic studies after valve ablation, the type of bladder function found correlates with the time elapsed from surgery; detrusor overactivity is the predominant pattern initially [326] but improvement is noted in both overactivity and compliance over time. [329-334] Myogenic failure in conjunction with increasing capacity and poor emptying are primarily a later phenomenon, most likely secondary to increased urine production and decreased frequency of voiding with advancing age. [331] Vesicoureteral reflux is most commonly noted in boys with detrusor overactivity whereas hydronephrosis is most frequently seen with a poorly compliant bladder. [329,335] The persistence of upper urinary tract changes is related to the bladder's unresponsiveness to medical therapy for the detrusor overactivity and/or underactivity. Several studies have shown the predictability of the development of renal failure based on specific detrusor patterns seen on urodynamic evaluation; persistent poor compliance and myogenic failure with increased residual urine are the most likely causes of this progressive deterioration. [326,327,330,331,335,336]

b) Bladder exstrophy

Once the exstrophied bladder is closed it may be difficult to determine how best to manage persistent incontinence, upper urinary tract dilation or vesicoureteral reflux, whether to further strengthen bladder outlet resistance or whether to perform augmentation cystoplasty for a small capacity, poorly compliant bladder. In addition, as more children undergo complete primary repair of the exstrophic bladder in the neonatal period the most concrete assessment of

bladder function is via urodynamics. Only 2 studies characterize the change in function following bladder neck reconstruction in patients with persistent incontinence; 20% show detrusor overactivity preoperatively versus 37% postoperatively, and compliance worsens in up to 50% after surgery. [337-339] No studies are extant that have correlated incontinence with bladder capacity, compliance, instability and leak point pressure.

c) Ectopic ureterocele

Urodynamic studies in babies with an ectopic ureterocele have shown that bladder function may be altered in many as half the affected patients; 2 reports revealed that between 55 and 70% had a larger than normal capacity bladder for age with high compliance, and poor bladder emptying due to detrusor underactivity. [340,341] In another multicentre analysis of 616 children with a variety of ureterocele types, investigators found only a 6% incidence of bladder dysfunction (all in children with an ectopic ureterocele) consisting of primarily detrusor overactivity; less than 1% had poor bladder emptying whereas the remainder had stable bladders (normal bladder function) and complete emptying. [342] Urinary incontinence and/or infection following surgical incision or excision of a ureterocele is likely to be secondary to the obstructive effects of the ureterocele directly on the bladder outlet and not to any surgical complication. [340-342] Additional preoperative urodynamic assessments are warranted to further characterize and define the cause of this abnormality.

d) Vesicoureteral reflux

Recent evidence has confirmed that vesicoureteral reflux may be a secondary phenomenon resulting from detrusor overactivity and not a primary anatomic abnormality at the ureterovesical junction. [343-348] There is growing evidence that an overactive detrusor may lead to reflux in a marginally competent ureterovesical junction mechanism. [323,349,350] This overactivity may be a natural phenomenon in the infant bladder, especially males (due to the presence of high voiding pressures), [351-355] and/or a learned dysfunction in older children who try to withhold voiding throughout the day. [346] Several investigators have shown that detrusor overactivity tends to resolve with increasing age. [349,353,356] Despite this finding, there is ample evidence in 4 studies to show that treating the overactivity and/or voiding dysfunction with anticholinergic agents leads to a faster rate of resolution of the reflux (63 - 92% within 1 year) [357-359] than it might when the child is treated with antibiotics alone to prevent recurrent infection (25 - 54%). [360]

In this setting, history-taking about voiding habits, [361] and urodynamics with cystometry and uroflowmetry to confirm the abnormal bladder and possibly sphincter function, become paramount to just treating the child with antibiotics and getting yearly voiding or nuclear cystograms. Urodynamic studies have confirmed the presence of detrusor overactivity and/or high voiding pressures in at least half the babies studied with high grades of reflux, whereas only 38% had totally normal function. [351,355,362-364] Upper urinary tract damage is more apt to occur in children with abnormal bladder function as reported in 3 retrospective reviews. [356,358,364] Many clinicians treating patients with reflux advocate urodynamics to assess the lower urinary tract in children with high grade reflux, especially those who have incontinence, renal damage, or who are about to undergo antireflux surgery. [358,365]

e) Urethral stricture

Urethral stricture disease in boys is rare, usually arising from a previously unsuspected straddle injury. Uroflowmetry can accurately predict the presence of a urethral stricture in 88% of affected males. [366] Because recurrence of a stricture is often both frequent and insidious, periodic urinary flow rates, analyzing the maximal flow rate in relation to volume voided, may alert the clinician to early signs of renarrowing but efficacy of periodic flow rates has not been corroborated. [367]

5. FUNCTIONAL DISORDERS OF THE LOWER URINARY TRACT

When assessing functional disorders involving the lower urinary tract in children, one must take into account the dynamics of the maturing nervous system, learned habits of elimination for bladder and bowel function and social influences that might modulate the child's behavior. [368,369]

a) Diurnal incontinence

Urodynamics has a limited place in diurnal (day and night) incontinence. This condition is not considered worrisome before age 5 or 6. Then, most children without an unsuspected anatomic or neurological lesion should be dry. [370] If they are not, a thorough physical examination and careful questioning about when the incontinence occurs is usually adequate to help to define and treat the problem. When a girl is continuously wet during both the day and the night

without ever having had a dry period, radiological investigation (renal ultrasound or computerized tomography) to detect an ectopic ureter is mandatory, but not urodynamics. In girls who wet only after voiding, vaginal trapping of urine, with subsequent postmicturition leakage on standing, is the most likely cause. Urge incontinence is often due to unrecognized urinary infection in girls and/or to an overactive detrusor in either sex. Urinary infection is very common in girls; it can lead to low compliance, and to detrusor overactivity or large swings in urethral pressure. It is often caused, as well as aggravated by dysfunctional elimination "syndromes". [371,372] Treating these elimination disorders with behavioral modification, biofeedback training, drug therapy or intermittent catheterization (CIC) [373] and/or antibiotics to prevent further urinary infection is necessary before considering urodynamics. [374] Uroflowmetry with a post-void residual urine determination and cystometry are indicated if the incontinence persists despite medical therapy. In one study of girls with recurrent infection without reflux two distinct patterns of dysfunction emerged in 80% of those studied, either an overactive detrusor with a normal urinary flow pattern and complete emptying, or a normal detrusor with an intermittent flow pattern and incomplete emptying. [375]

In boys with persistent day and night-time incontinence, voiding cystourethrography is warranted to determine the presence of posterior urethral valves or other forms of bladder outlet obstruction that may be contributing to the detrusor overactivity. [376] Faecal incontinence in the absence of any anatomical or neurological deficit often affects lower urinary tract function and contributes to urinary incontinence in a number of ways. Constipation and faecal impaction have been shown to cause detrusor overactivity and a reduced functional bladder capacity. [377] Understanding and eliminating this possible aetiology can normalize lower urinary tract function without the need for urodynamics. [371] Persistent daytime and night-time incontinence in the absence of urinary infection and a normal bladder and bowel emptying regimen warrants cystometry, pressure-flow studies and a urinary flow rate. A meta-analysis of 460 children with daytime incontinence evaluated with urodynamic studies during the last 20 years reveals detrusor overactivity in 57% (261 of 460), dysfunctional voiding (failure to relax the sphincter mechanism) in 22% (34 of 152) and normal findings in only 14% (64 of 460). [351,378-382] These findings are not gender-specific but are age-dependent, with most children outgrowing the abnormal findings by puberty. [379] Presumably normal children without day or night-time wetting do not have a pronounced degree of detrusor overactivity or dyssynergia but the evidence for this is lacking due to the paucity of studies in normal children.

Urodynamic testing has clearly improved our understanding of the aetiology of diurnal incontinence but no study has shown that urodynamic characterization of any abnormality has improved the efficiency of treatment for these children.

b) Enuresis (nocturnal)

Night-time wetting (enuresis) is a condition that is common in children aged 5 years but which improves with time, so that less than 15% of pubertal boys and 5% of pubertal girls continue to be affected. [368-370,383] Multiple causes for the persistent wetting, ranging from genetic factors, to maturational delays, to sleep disturbances, to social causes, to attention deficit disorders, to bladder and urethral dysfunction, to excess fluid intake, to abnormal vasopressin secretion and/or to constipation, have been implicated. [371,384-390]

Although in various cultures there may be social and familial pressures to resolve the condition before puberty, in western societies it is generally not necessary to conduct urodynamics until adolescence, to determine why the wetting has not abated. Urodynamic testing in 615 enuretic children with and without daytime symptoms has identified detrusor overactivity in 61%. [391-395] When the children are divided into those with day and night-time incontinence (polysymptomatic) versus those with just nocturnal wetting (monosymptomatic), the incidence of detrusor overactivity decreases from 64% to 35% in the latter group. [393,394] In another prospective study comparing enuretics to age-matched nonenuretic controls, bladder capacity at night (enuretic capacity) was significantly less in those who wet versus those who did not. [396]

Although the authors did not speculate on aetiology they felt that enuretics were less able to hold their urination than nonenuretics. Management should be directed at improving the child's ability to withhold urination. Treating the polysymptomatic child using antimuscarinic agents can be very effective (as high as 77% cure) with low recidivism rates when based on the findings of urodynamic testing. [386,390,397-401]

6. Technical concerns: Reliability and reproducibility of tests

A reduced rate of filling, e.g. 10% of the expected bladder volume per minute, has been recommended in children to accurately determine detrusor compliance and functional bladder volume. [402] Some investigators advocate that infants should be assessed with much lower rates of filling or with natural filling cystometry. [403,404] Several studies do show lower detrusor pressures under natural filling versus even slow filling rates during cystometry. [277,278,405] Even though the practicality of time management plays a role in a busy urodynamic laboratory, it is essential to perform urodynamic testing in a way that reveals what one considers clinically important. One study in particular [277,278,405] demonstrated lower intravesical pressure when it was measured initially along with the volume of urine at the time of catheterization to empty the bladder and then compared to the pressure at the same volume during the subsequent cystometrogram. Except for determining bladder volumes at specific pressures, [278] no study has shown that these differences are crucial in the management of children with lower urinary tract dysfunction. One study looked at the effect of the temperature of the instillate (25º versus 37.5º C) on measured detrusor pressures and found no significant differences in compliance. [111]

The smallest dual-channel urethral catheter available should be used in children for the same reasons as specified for adults, although the measuring lumen must be large enough to measure pressures in a technically adequate manner. Although urethral catheters of moderate size do not always obstruct the urethra, [406,407] or produce higher than normal voiding pressures measured with suprapubic tubes, it is prudent to employ the smallest caliber catheters that are practical when doing a cystometrogram that measures filling as well as voiding pressures. For very young infants it may be better to insert a suprapubic catheter placed under anaesthesia the day before the test to make the subsequent investigations more accurate [408,409] but this has not been assessed with any precision. Most children can undergo urodynamic studies without pre-medication; only the most agitated may require some degree of sedation. Even then, children should not be so heavily sedated that they cannot void around the catheter. However, there are no studies that show a difference in bladder filling pressure (whether related to compliance or to detrusor contractions) in awake versus anaesthetized children.

7. Recommendations for urodynamic studies in children

a) Recommendations for clinical practice:

The committee recommends that:

1 Children with neurological or anatomical abnormalities affecting the urinary tract should be investigated with comprehensive urodynamics (Grade of recommendation C)

2 Children with such neurological or anatomical abnormalities should have an evaluation of anorectal function as well (see section E on faecal incontinence) (Grade of recommendation C)

3 Children without overt neurological disease, but with abnormalities of lower urinary-tract function that are believed to be the result of learned behavior, should receive a trial of conservative (behavioral) therapy before full urodynamic investigation (Grade of recommendation C).

4 If conservative therapy fails and if invasive therapy is being considered then they should receive comprehensive urodynamic investigation in a urodynamic laboratory specializing in pediatric testing with appropriately trained personnel (Grade of recommendation C).

5 If imaging is desired, initial investigation should be by ultrasound. If it is abnormal videourodynamics may be considered (Grade of recommendation C).

b) Recommendations for research:

1 Establishment of normative values and reproducibility of urodynamic data is required both in normal children and in well-defined groups of pediatric patients.

2 Integrated approaches to the management of anorectal and urinary dysfunction should be explored.

V PATIENT EVALUATION: FRAIL ELDERLY

1. Introduction

Elderly patients should not be considered differently from younger subjects simply because of their chronological age. Lower urinary tract symptoms, especially storage symptoms, showed age-related alterations in the two sexes in the absence of any overt underlying disease, and bladder function in both sexes may be subject to a gender-independent aging

process. [410] Urodynamic findings in the elderly tend to demonstrate an overactive detrusor, [411,412] and a reduction in bladder capacity, urinary flow rate and detrusor contractility. [413] Because of these changes the utility of urodynamics has to be judged against a different background in the elderly.

Firstly, the invasive nature of conventional urodynamics becomes a more important factor in the old-old or frail elderly, who may be more vulnerable to any insult than younger people. For example, one study [414] showed that there was a significant association between age and the presence of asymptomatic bacteriuria before cystometry and between this bacteriuria and urgency (without detrusor overactivity) on cystometry. These results do not however support a policy of universal screening for bacteriuria before urodynamic investigation. Asymptomatic bacteriuria did not influence the urodynamic outcome except in patients with urgency (without detrusor overactivity); and the authors recommended that screening and treatment be considered individually in older women who are being investigated for storage symptoms. About 20% of this group of patients developed UTI (mostly asymptomatic) after the urodynamic investigation. This information should be included in the counselling before urodynamic investigation and should be incorporated into the patient information leaflet as part of good clinical practice. Unfortunately, there are few adequately powered studies (and none that we are aware of in this patient group) of the efficacy of antibacterial prophylaxis.

Secondly, given the multifactorial nature of incontinence in the elderly, [66] and the fact that there may be easily reversible causes or contributory factors, conservative therapies are indicated initially. [415] Urodynamic examination is reserved for patients in whom conservative management has failed or has proved inadequate, who desire further attempts to correct or manage the incontinence, and who therefore need a detailed and objective diagnosis.

The place of urodynamics in the elderly with incontinence is therefore quite limited even in principle and, given that the number of studies seeking to establish its clinical utility is even smaller than in younger adults, there is very little objective evidence for or against clinical urodynamics in this population group.

On the other hand, because of the changes in function that occur with age not only in the lower urinary tract, but in the neural system that controls it (or fails to control it), and also in other organ systems that may have an impact on urological problems, research urodynamics is essential to establish what these changes are, how they are related to other aging-associated changes, and how they may be reversed, so that there is a chance of developing new, more suitable therapies. Therefore, in academic centres at least, many geriatric patients should be examined so as to generate a steady stream of high-quality clinical urodynamic research, addressing mechanisms of disease and deterioration in all the main geriatric patient groups (including 'normal aging'), and in the less common patient groups as well.

2. What is usually evaluated

Among the old-old, incontinence is the paramount troublesome symptom in both men and women, with a steeply rising incidence after age 80. [78] The type of incontinence appears to be predominantly urge. [83,412] Stress incontinence is relatively uncommon in men of any age (except post radical prostatectomy) and it seems to become gradually less common in women after the age of about 50, [78] for reasons that are incompletely understood. One recent paper based on a retrospective review of referred patients finds that the decrease in incontinence after age 50 is due to a decrease in the prevalence of detrusor overactivity. [416] This surprising result is presumably a reflection of age-related referral patterns in that practice.

a) Urge incontinence

Urge incontinence is usually the result of detrusor overactivity. (It is believed that occasionally it may be due to involuntary relaxation of the urethral sphincter mechanism, without a measurable detrusor contraction. [4]) Thus one possible reason to perform urodynamics might be to identify detrusor overactivity. The relevant test would be filling cystometry. There are reasons to question whether this is the best approach:

In straightforward urge incontinence in the elderly, detrusor overactivity is highly probable, and it is not necessary to perform urodynamics to prove this before trying pharmacological or behavioral therapy. This is one of the reasons for the limited clinical role of urodynamics referred to above.

Because detrusor overactivity is only one contributor to urge incontinence, [10,66] not all individuals with detrusor overactivity are incontinent. In fact, detrusor overactivity is believed to be quite common in healthy older people who are apparently free of blad-

der symptoms of any kind. Thus it is important to look not just for detrusor overactivity, but for actual leakage caused by detrusor contraction - detrusor overactivity ***incontinence.***

Because only the more difficult or intractable cases are investigated with urodynamics, it is important to look for other coexisting LUT dysfunction, beyond simple detrusor overactivity.

Among the elderly, the most common type of detrusor overactivity is the 'terminal' pattern (Figure 8, Section B.II.3), in which a single involuntary detrusor contraction occurs at the end of filling and leads directly to leakage (incontinence). [72] Quite frequently there is reduced bladder sensation also, so that the subject does not feel any sensation of bladder filling or the need to void until the contraction is about to take place, [70] and thus has very little warning of impending leakage. These characteristics are believed to have a neurological (cerebral) origin. [70,72]

In elderly people, urge incontinence frequently coexists with incomplete bladder emptying. Among men who have not had prostate surgery, urethral obstruction is a possible contributor to incomplete emptying. If there is no obstruction, and particularly in women in whom obstruction is rare, incomplete emptying is a sign of impaired bladder contractility. The urodynamic abnormality underlying urge incontinence with incomplete emptying (assuming no obstruction) has been named DHIC (detrusor hyperactivity with impaired contractile function). [417] Its significance is that the standard pharmacological treatment of urge incontinence – with antimuscarinics – may worsen bladder emptying and possibly cause urinary tract infection or even make the incontinence worse.

Thus the principal urodynamic tests that are done are:

1. Free uroflowmetry:

a. may be useful as a screening test for obstruction or diminished detrusor contractility

   • A slow, prolonged or intermittent flow curve may indicate either urethral obstruction or diminished detrusor contractility; pressure-flow studies are required to distinguish between them

2. Measurement of post-void residual urine:

a. to check whether anticholinergic (antimuscarinic) therapy is contraindicated because of a large residual

b. to help identify DHIC

Observation of consistently elevated post-void residual thus has therapeutic consequences and so, among urodynamic investigations, measurement of post-void residual urine is important. If it is small, significant infravesical obstruction or detrusor underactivity or acontractility is less likely, and a small dose of anticholinergic medication may be tried. If a large amount of residual urine is found then incontinence associated with chronic overdistension or infection may be suspected, and intermittent catheterization may be indicated. The measurement is included in the Resident Assessment Protocol (mandated for nursing homes by the US Congress) along with the stress test. [418]

3. Filling cystometry:

a. to demonstrate or rule out detrusor overactivity or – more importantly – detrusor overactivity incontinence

b. to identify the pattern of detrusor overactivity – terminal or phasic

c. to identify reduced or normal bladder sensation

Cystometry is sometimes said to be an essential part of the diagnostic evaluation, both in defining underlying pathophysiology and directing treatment. [69] However, none of these aims (a.-c. above) have particular therapeutic importance except in difficult cases where initial therapy has failed and it is uncertain what the underlying problem is.

4. Pressure-flow studies of voiding:

a. to identify or rule out prostatic obstruction (in men)

b. to identify or rule out impaired contractility (reduced detrusor contraction strength)

• Simple cystometry

It is sometimes recommended that urodynamics in the elderly should be done by "simple cystometry" if cystometry is indicated and no equipment or referral is available. [419] The procedure needs only an open syringe attached to a single-lumen catheter, steri1e water or saline and a tape measure. Fluid is infused by gravity at a pressure head of l5-20 cm H_2O. Bladder capacity, sensation of filling and presence of a detrusor contraction or overactivity can be semi-quantified. Pressure is measured by observing the height of the column of water. These simple measures can be carried out at the bedside and may be useful for disabled patients. [420-425] Simple cysto-

metry, as compared with multichannel cystometry, has a specificity of 75-79% and a sensitivity of 75-88% for the observation of detrusor overactivity. [420,421] The accuracy can be improved by combining it with even simpler tests [421,422] such as a stress test to exclude stress incontinence. [426]

However the clinical significance of these findings is limited. Detrusor overactivity is found in up to 50% of symptom-free elderly (and so it is not pathognomonic), while detrusor overactivity incontinence is the most likely finding in incontinent frail elderly in any case. [83,412,417] Thus the test is performed only to rule out detrusor overactivity in a small subset of patients.

Furthermore, most of the studies recommending simple cystometry were conducted before the high prevalence of detrusor hyperactivity with impaired contractility (DHIC) was recognized. [412] This dysfunction is the most common abnormality observed in the frail elderly population with incontinence. [412,417] A total of 185 patients who had persistent LUTS after TURP were enrolled in one study, and the results revealed that a normal videourodynamic tracing was found in 9%, pure detrusor overactivity in 10%, low detrusor contractility in 19%, DHIC in 14%, poor relaxation of the urethral sphincter in 19%, and bladder outlet obstruction in 28%. [232] DHIC is easily misdiagnosed as a stable detrusor on simple cystometry, [426] because single-channel cystometry is less sensitive for detecting low-pressure detrusor contractions than multichannel recording. If a detrusor contraction coincides with a cough, the leakage may be regarded as the sign of a positive stress test.

By design, simple cystometry can at best recognize only detrusor overactivity, or detrusor overactivity incontinence if actual leakage is recorded. There is no possibility of studying voiding dynamics. Furthermore, the checks on measurement quality that are part of conventional urodynamics are not available. Hence, because recognition of detrusor overactivity by itself has little therapeutic importance, and urodynamics in the elderly is reserved for difficult or intractable cases, it is more reasonable in such cases to conduct a full urodynamic examination of both filling and voiding phases, in which quality control can be maintained to eliminate artifacts, and the more relevant aspects of lower urinary tract behavior can be assessed, such as obstruction and reduced detrusor contractility.

If there is already a urethral or suprapubic catheter in place, it is possible and may be worthwhile to conduct simple cystometry and measurement of post-void residual urine without recatheterisation.

b) Stress incontinence

Among older ***men,*** stress incontinence is almost entirely confined to post radical prostatectomy patients (see section D.II, Patient evaluation: Men). Among elderly women, pure stress incontinence seems to be rare. Urodynamic testing usually follows the methods used in younger women. Frequently, it is difficult to perform an adequate examination because the patient is not able to produce a strong enough cough or Valsalva manoeuvre to cause leakage during testing, and cannot easily be examined in the upright position, which is the most likely posture to produce incontinence. On the other hand, it may be just such factors that make stress incontinence uncommon in this population in the first place.

A weak urethral sphincter (intrinsic sphincter deficiency, ISD) is a contributory factor to stress incontinence. There is some evidence that a weak sphincter or inadequate sphincter control may contribute to the severity of urge incontinence as well. [66,69] Methods of assessing the sphincter include measurement of Valsalva leak point pressure (difficult for the reasons stated above) and urethral pressure measurements. Unfortunately, urethral pressures diminish with age, whether or not there is stress incontinence, and are not diagnostic.

3. Evidence for reproducibility and reliability of urodynamic tests in the geriatric or frail elderly population

There is little published evidence about reproducibility and reliability in this patient population. Two groups have recently examined specific aspects of geriatric urodynamics which have some bearing on this topic. There is a little older evidence on the reproducibility of some parameters.

a) Filling cystometry

One group [427] sought urodynamic changes associated with behavioral and drug treatment of urge incontinence in 105 ambulatory, nondemented, community-dwelling women, of mean age 67 years (range 55-91). They especially sought changes which might mediate the positive effects of treatment on the frequency of incontinence. Positive evidence that urodynamic results are related to treatment outcomes would tend to confirm the reliability and relevance of urodynamic measurements.

This was a randomized, double-blinded, placebo-controlled, clinical trial of subjects who were stratified according to type of incontinence (urge only vs mixed urge and stress) and severity of incontinence (frequency of accidents as documented in a bladder diary). Participants received 8 weeks of biofeedback-assisted behavioral training, or drug treatment with individually-titrated oxybutynin chloride, or placebo control. Two-channel cystometry was performed to determine threshold volumes for first desire to void, strong desire to void, bladder capacity, and the volume at which detrusor overactivity or leakage occurred. These measurements were made both before randomization and after completion of treatment. Two-week bladder diaries were used to document episodes of incontinence and voiding frequency before and after treatment.

Treatment significantly improved incontinence: behavioral training resulted in a mean 82% reduction in frequency of accidents, oxybutynin resulted in a mean 78% reduction, and the control group had a mean 52% reduction ($P = 0.002$). Treatment also changed urodynamic variables significantly: bladder capacity increased by 69 ml in the oxybutynin group ($P < 0.001$) and by 17 ml in the behavior group; it decreased by 6 ml in the control group. Volume at strong desire to void increased by 70 ml in the oxybutynin group ($P < 0.001$), 40 ml in the behavior group ($P < 0.05$), and 8 ml in the control group. Volume at first desire to void increased by 44 ml in the oxybutynin group ($P < 0.001$), 19 ml in the behavior group, and 9 ml in the control group. There was some reduction in the frequency of detrusor overactivity in all 3 groups, but the differences were not significant.

Although oxybutynin and behavioral treatment were both effective, and although oxybutynin increased cystometric bladder volume at strong desire to void and bladder capacity, the authors were unable to demonstrate that the improvement in incontinence was related to the urodynamic changes observed – that is, that the changes mediated the improvement. One possible explanation for this negative result is that the urodynamic parameters measured show considerable variability, as in other patient groups (see sections C.I and C.II).

b) Post-void residual urine

Residual urine is believed to depend on the presence of bladder outlet obstruction (in men) as well as on detrusor underactivity (i.e. impaired contractility). [428] Thus, in a man, the presence of substantial residual urine (> 100 ml) in the absence of severe bladder outlet obstruction suggests that the increased residual urine is mainly due to a reduction in detrusor contractility, with bladder outlet obstruction making only a minor contribution. There may be an age-dependent decrease in contractility in both sexes.

Residual urine varies in a given individual for no known cause: recorded values wax and wane over time [62]. Significant daily variations have been observed in elderly patients of both sexes, with larger residuals (up to 40% greater) being measured in the early morning. Similar changes have been described in patients with bladder outlet obstruction or detrusor underactivity [13,124]. No clear predictor of deterioration of residual urine volume or of complete urinary retention has been identified.

c) Pressure-flow studies

Another group [200] compared consistency, reproducibility, and responsiveness of various methods of estimating detrusor contraction strength from pressure-flow studies. They retrospectively analyzed urodynamic data on 84 females 53 years old or older, with urge incontinence, who received either a titrated dose of antimuscarinic medication or placebo in a controlled trial. Data were gathered before and at the end of treatment. Three different variations of the stop test were compared. In a stop test, flow is prevented and the isovolumetric detrusor pressure attained is taken to be a measure of detrusor contraction strength. Flow may be stopped by a voluntary contraction of the urethral sphincter midway through voiding (voluntary stop test: often impossible in stress incontinent women); by blockage of the outlet by a balloon midway through voiding (mechanical stop test); or by attempted voiding against an outlet that is already blocked by a balloon (continuous occlusion).

The voluntary stop test yielded isovolumetric detrusor pressure values inconsistent with the other 2 tests (a mean and SD of 31 ± 16 cm H_2O as opposed to 47 $\pm$ 26 and 49 $\pm$ 24 cm H_2O). The mechanical and continuous occlusion tests gave very similar results that were highly correlated with one another ($r = 0.87$). Measurements pre- and post-treatment in the 20 women who received a placebo showed that the continuous occlusion test had the highest reproducibility ($r = 0.9$, $p < 0.01$), followed by the mechanical ($r = 0.7$, $p = 0.01$) and voluntary ($r = 0.7$, $p < 0.01$) stop tests. Treatment with oxybutynin decreased isovolumetric detrusor pressure by up to 6 cm H_2O, but

the decrease was statistically significant only for the continuous occlusion test.

The authors concluded that to assess detrusor contraction strength in elderly females with urge incontinence either a mechanical stop test or a continuous occlusion test is acceptable but the continuous occlusion test has better reliability and more sensitively detects slight drug-induced changes.

This work demonstrates that some urodynamic measurements in a geriatric population are quite reproducible, reliable, and responsive to the effects of treatment. This means that they may be useful for research, but are not necessarily clinically relevant. For example, there is no evidence that a weak detrusor contraction strength predicts poorer response to treatment with anticholinergic medication or surgery.

4. Evidence that performing urodynamic testing improves clinical outcomes in the geriatric population

Few relevant studies have been published in this population. One recent publication [429] assessed the results of tension-free vaginal tape (TVT) for the treatment of stress urinary incontinence in 76 consecutive women more than 70 years old (median age 76 years). 31% (24/76) of the patients had overactive bladder symptoms and 4 (3%) had proven detrusor overactivity controlled by anticholinergic therapy. All patients had preoperative multichannel urodynamic evaluation. At a mean follow up of 25 months, 67% of the patients were cured. Preoperative urgency symptoms were cured in 46% of the group. Among the failures, 14 (18%) had urge incontinence, while "de novo" urgency without incontinence was noted in 21%.

Thus, this paper provides no clear evidence that preoperative urodynamics was able to predict the outcome of a popular stress incontinence procedure in older women. It does show that post-operative difficulties may be due to urgency and urge incontinence, which however could not be predicted from the tests performed preoperatively.

To summarize, there is no clear evidence that urodynamic testing improves clinical outcomes in the geriatric population (Level of evidence 3).

5. The practical indications for urodynamic studies and which tests are needed

a) Post-void residual urine

Based on the above, there is general agreement that post-void residual urine measurement is indicated before treatment of incontinence either with anticholinergic medication or by stress incontinence surgery. A consistently large residual urine certainly is a reason for caution and careful monitoring of bladder emptying, and may be a relative contraindication to such treatment.

b) Uroflowmetry

Uroflowmetry is a simple and noninvasive test. A normal uroflow without much residual urine probably rules out significant urethral obstruction or impaired contractility, but this finding is unusual in the elderly. Conversely, a poor uroflow is common in the elderly irrespective of sex, and although it cannot distinguish between obstruction and poor contractility, in either case there is a relative contraindication to anticholinergic therapy. Consequently, uroflowmetry (with residual urine measurement) may be a useful screening tool prior to instituting therapy.

c) Pressure-flow studies

A frequently asked referral question, in an older man who is incontinent, has an enlarged prostate, and is cognitively impaired or has a disease such as Parkinson's disease or multiple system atrophy, is whether the incontinence is due to prostatic obstruction or to cerebral changes. If the former condition is present, then prostate surgery might be considered. As outlined above, prostatic obstruction is not usually the "cause" of urge incontinence, which is typically multifactorial. However, there is weak evidence to suggest that, if the obstruction is urodynamically severe, then surgery may improve the incontinence. [430]

If obstruction is equivocal or absent, then there is little point in performing surgery in an attempt to eliminate it. After screening with uroflowmetry and residual urine measurement, pressure-flow studies may be indicated in older men in whom obstruction cannot be ruled out and surgery is at least contemplated. See also discussion of Parkinson's disease below.

6. The urodynamic parameters important in various geriatric conditions

Recent papers have examined the urodynamics of Parkinson's disease and related diseases. It is important to know the characteristic urodynamic features of these diseases, because a frequently asked clinical question is whether lower urinary tract dysfunction observed in an elderly male patient is due to such a disease or to BPE/BPO. Similar questions may sometimes arise in women.

• Parkinson's Disease

One group [175] found that men with presumed obstruction-related lower urinary-tract symptoms were less likely to have urge incontinence (detrusor overactivity incontinence) on urodynamics than men or women with Parkinson's disease. Detrusor overactivity owing to Parkinson's disease occurred at smaller bladder volumes than that in obstruction-related detrusor overactivity, although this finding was more pronounced in women than in men. The duration and severity of Parkinson's disease were not related to the nature or severity of urodynamic abnormalities.

Another group [254] found that the majority of patients with Parkinson's disease (72%) or multiple system atrophy (100%) had symptoms of urinary tract dysfunction. Neurogenic detrusor overactivity was more common in Parkinson's disease (81% vs 56% in multiple system atrophy). Detrusor-external sphincter dyssynergia was seen only in multiple system atrophy (in 47%). Urethral obstruction (AG number or BOOI > 40) was more common in Parkinson's disease than in multiple system atrophy. A weak detrusor (impaired contractility) was less common in Parkinson's disease (66% of women and 40% of men) than in multiple system atrophy (71% of women and 63% of men). Postvoid residual urine volume > 100 ml was not observed in patients with Parkinson's disease but was present in 47% of patients with multiple system atrophy.

Thus urinary tract dysfunction was prominent in both diseases but patients with Parkinson's disease had less severe dysfunction: primarily detrusor overactivity starting at small volumes and urethral obstruction. (Unfortunately these findings may make it difficult to distinguish from dysfunction associated with benign prostatic obstruction). However, a postvoid residual urine volume > 100 ml, detrusor-striated sphincter dyssynergia, or an open bladder neck at the start of bladder filling are suggestive of multiple system atrophy.

7. Recommendations

a) Recommendations for clinical practice

1 Post-void residual urine measurement by a noninvasive method is recommended before institution of pharmacological or surgical treatment of incontinence. It should be repeated to monitor the effect of such treatment (Grade of recommendation C).

2 Uroflowmetry may be used to screen for voiding abnormalities prior to such treatment (Grade of recommendation C).

3 Filling cystometry alone has limited value in this patient population. "Simple cystometry" is not recommended unless a urethral or suprapubic catheter is already present for management and must be interpreted with care (Grade of recommendation C).

4 Comprehensive urodynamics including, at a minimum, filling cystometry and pressure-flow study of voiding, is recommended in difficult and intractable cases that have not responded to behavioral or pharmacological therapy, and in whom further therapy is desired; and in complicated cases or cases with complicated comorbidity, where treatment is desired but the nature and aetiology of the urinary tract problems are unclear (Grade of recommendation C).

5 If stress incontinence is suspected, extra tests of urethral function and/or pelvic floor mobility may be useful, although stress incontinence is not only less common than in younger patients but may be difficult to prove or rule out in this population (Grade of recommendation C).

6 The committee recommends that comprehensive urodynamic testing be performed in specialized centres with a special interest in incontinence, by trained and certified staff who routinely perform urodynamic testing of any patients referred with suspected lower urinary tract dysfunction (Grade of recommendation C). To maintain adequate urodynamic expertise in this difficult-to-examine patient population, to provide a background of 'regular' patients against whom specific patients can be judged, it is essential that such centres examine substantial numbers of frail elderly patients (Grade of recommendation C).

b) Recommendations for research

1 Study of biological mechanisms of continence and incontinence in the frail elderly, especially those related to supraspinal control or lack thereof.

2 Development and testing of treatments specific to the frail elderly.

3 Establishment of the reproducibility and reliability of urodynamic measurements in the frail elderly.

E. DYNAMIC TESTING FOR FAECAL INCONTINENCE

I. INTRODUCTION

In contrast to testing for urinary incontinence, "dynamic" testing for faecal incontinence is not yet well defined. The causes of faecal incontinence fall into two broad interactive categories: factors that are associated with the nature of stool and the frequency of bowel movements; and anatomical or functional disturbances of the anorectal area. The stool features commonly associated with incontinence are loose and frequent bowel movements, that is, diarrhoea. However severe constipation and packing of the rectum with stool can lead to "overflow incontinence". The local anorectal mechanisms that maintain continence include the specialized anatomy and innervation of the region, the sensory-motor function of the rectum, and the resting, voluntary and reflex function of the musculature (both smooth and striated) of the anal sphincter apparatus. [431] Other factors such as cognitive ability, mobility, and drugs can impact the anorectal continence mechanisms and/or the patient's ability to defaecate normally. Testing of the anorectal continence mechanisms has included many approaches directed at different anatomical and physiological sensory-motor aspects, and functional behaviours. [431-433]

Although urodynamics can be considered the study of (lower) urinary tract function and dysfunction by any relevant method, it has developed primarily into assessment of pressure, volume and flow, electromyogram activity and responses of the urethral sphincter, and bladder sensation. These measurements are made in relation to dynamic activities such as bladder filling, micturition, and contraction of the urethral sphincter and pelvic floor musculature. It is reasonable at this stage to approach dynamic testing for faecal incontinence in the same way. That is, to assess those tests that measure pressure, volume and/or flow, electromyogram activity and responses of the external anal sphincter, and rectal sensation – these in relation to dynamic activities such as rectal distension and contraction, defaecation, and contraction of the anal sphincter and pelvic floor musculature. Perhaps a term such as "faecodynamics" will arise.

Anorectal testing techniques are directed primarily at four aspects of function and dysfunction: 1) the anatomical integrity and function of the anal sphincter(s); 2) the sensory-motor activity of the rectum; 3) puborectalis and pelvic floor muscle function; and 4) integrity and function of external innervation from the periphery to the brain. [431-433] In the context of faecal incontinence, there are few large scale studies that validate anorectal tests against other techniques, or properly controlled treatment trials with placebo components, adequate control populations, and careful outcome assessment. In particular, there is little experience with or information about the external sphincter innervation and its functional behaviour during defaecation and rectal activation. Therefore, this review documents some of the relevant information available about testing modalities that address the dynamic aspects of faecal incontinence, with attention to their reproducibility and reliability, and the active physiological mechanisms they monitor. Further assessment of the literature and future research is required to fully understand the practical use of these tests in the investigation and management of different clinical conditions causing faecal incontinence. A major confounding factor in their use is the knowledge that up to 80% of patients with faecal incontinence have more than one contributing pathogenesis.

II. WHAT IS USUALLY INVESTIGATED?

The testing modalities that will be discussed are anorectal manometry and rectal pressure-volume relationships, pudendal nerve terminal latency, external anal sphincter electromyography, rectal sensory-motor and sensory activity, anal-sphincter electromyography, and dynamic imaging of the anorectal area.

1. Anorectal manometry

A recent AGA (American Gastroenterological Association) technical review of anorectal testing techniques has noted that in patients with faecal incontinence, anorectal manometry can be useful in two main ways: to define functional weakness of one or both sphincter muscles; and to perform and predict responses to biofeedback training. [431] Both of these aspects relate to dynamic activity. These conclusions are based on clinical experience and uncontrolled studies. Alone or in combination with other tests of anorectal function, manometry has been assessed for its ability to characterize different

features that may contribute to faecal incontinence and/or aid in its management. These aspects include: localization of structural damage to the sphincters by vector manometry;[434-437] assessment of rectal sensation;[438-441] assessment of rectal compliance and irritability;[438,439] demonstration of spontaneous relaxations of the sphincters and their relationships to rectal events;[439,440] and assessment of electromechanical dissociation. [442] However, there is little or no well-documented evidence of the clinical value of the information provided about these aspects by anorectal manometry, over and above a good history and physical examination, and beyond other more sensitive and specific tests. This lack of evidence reveals the need for adequate research and proper evaluation of these and other aspects of anorectal dysfunction.

a) Sphincter pressures

Seemingly satisfactory measurements of anal canal pressures and anal sphincter responses can be obtained with open-tipped or side-opening water-perfused catheters, catheter-mounted solid-state microtransducers, or air- or water-filled balloons of various sizes and configurations. As with urethral pressure measurements, however, catheter-mounted microtransducers do not measure pressure but record a different property of the anal canal (see section B.I on stress urinary incontinence). Early studies indicated that the recorded pressure was a measure of sphincter strength. [443] Normal anal canal pressures vary according to gender, age and technique employed. [431] In general, pressures are higher in males and in younger persons but there is a considerable overlap of recorded values. Males have longer anal canals than do females, albeit with considerable overlap. Sphincter pressures decrease with age, especially after age 50-55, the decrease being most marked in females. [431]

There have been studies of whether simple physical rectal examination at rest and/or during voluntary contraction of the anal sphincter can assess sphincter integrity and activity just as well as manometry. In a study of 66 patients, digital assessment was significantly correlated ($r=0.56$, $p<0.001$) with anal canal resting pressure, and with maximum squeeze pressure ($r=0.72$, $p<0.001$), although in individual patients the digital assessment was often inaccurate. [444] Similar observations were made in two studies of 280 patients [445] and 191 patients. [446] In another study of 64 patients, the agreement between digital assessment and resting anal canal pressure was found to be only moderate (contingency coefficient 0.41) and the agreement between digital exam and squeeze pressure was only slightly better (contingency coefficient 0.52). [447] These correlations with objective measurements are too low to support the use of digital examination alone in the assessment of anal sphincter function.

A number of studies have examined the reproducibility of anorectal manometry. Rogers and colleagues [448] evaluated 16 patients on 2 occasions 20 days apart, and found that the average difference between studies was 10 cm H_2O (95% confidence interval, -25 to 4.7 cm H_2O) for resting pressure, and 9 cm H_2O (95% CI, -32 to 14 cm H_2O) for squeeze pressure. In a study of only 14 subjects, Hallan and colleagues [444] found that independent resting pressure measurements were well correlated ($r=0.77$, $p<0.001$), and independent squeeze pressure measurements also were correlated ($r=0.66$, $p<0.01$). Another study in 11 subjects demonstrated that individual variation of resting pressures measured on two separate days was less than 12%. [449] Read et al, [450] in a study of 27 subjects, reported that maximum pressures measured on two separate days were significantly correlated ($r=0.78$ and $r=0.88$ for basal and squeeze pressures respectively). In a more recent study of 19 subjects assessed on two separate days, Bharucha et al reported that resting pressures, maximum ($r=0.8$) and average ($r=0.73$), and squeeze pressures, maximum ($r=0.75$) and average ($r=0.9$), were significantly correlated between the separate days. Values measured on initial and subsequent study days did not differ significantly. [451] Although it is difficult to draw conclusions about reproducibility from correlation coefficients, it appears from these figures that anorectal functional measurements may be more reproducible than their urinary counterparts.

b) Resting sphincter pressure

Resting pressures reflect the tonic activities of both the internal and external anal sphincters, the internal anal sphincter accounting for up to 75-85% of the measured pressure. Incontinence of liquid stool is reported to be associated primarily with low internal anal sphincter pressures; patients incontinent of both liquid and solid stool have abnormally low squeeze pressures (external anal sphincter) in addition. [452] In patients with faecal incontinence, resting anal-sphincter pressure is decreased but the sensitivity of this decrease is poor in discriminating between incontinent and continent patients, as reported in older [449,450,453,454] as well as more recent studies. [455] In one study of 143 incontinent subjects, 39% of females and 44% of males had both resting

sphincter pressures and maximum squeeze pressures within the normal range. [449] A more recent study taking into account the 95th percentile reported that in 45 incontinent patients, 23% and 27% had normal resting anal-sphincter pressure and maximum squeeze pressures respectively. [456]

The concept that the internal anal sphincter just acts as a fine-tuning mechanism and provides control of flatus and liquids has been challenged. [457] It may have a more significant role in continence. This alternative view is that low resting pressures (poor internal anal sphincter function) are associated with *passive* incontinence of solid, liquid or gas throughout the day. [458] Low squeeze pressures (poor external anal sphincter function) are associated with leakage of solid, liquid or gas at times when the normal continence mechanism is challenged. For example, when the sampling reflex allows faeces into the anal canal and there is a desire to defaecate, a normal person will contract the external anal sphincter (if defaecation is inappropriate) and this will aid propulsion of stool back into the rectum. If external anal sphincter function is compromised, ability to contract it is impaired; once the desire to defaecate is felt, there is limited ability to defer it and it may be impossible to get to the toilet in time. [459]

It is usual to take the maximum (or average) resting pressure along the anal canal as a measure of internal sphincter function. Some measure these parameters relative to atmospheric pressure and some measure it relative to the rectal pressure. There is no consensus about which is more valuable. There is no consistency in the terms used for these parameters: maximum resting pressure and maximum basal pressure are commonly used, but neither defines what is being measured.

c) Maximum squeeze pressure

A decrease in maximum squeeze pressure has the greatest sensitivity and specificity for identifying faecal incontinence, the efficiency of this measurement depending on the cutoff pressure considered as normal and the sex of the patient. [439,460] Felt-Bersma et al [461] studied 178 patients who were faecally incontinent by history, and 80 normal controls. Maximum squeeze pressure had the greatest sensitivity and specificity; at a cut-off of less than 60 mm Hg in females, sensitivity was 60% and specificity was 78%. At a cut-off of 120 mm Hg in males, sensitivity was 67% and specificity was 67%. Sun et al [439] compared anorectal physiological tests in 302 faecally incontinent subjects and 65 healthy controls. Taking the mean minus 2 standard deviations for controls as the cutoff, the sensitivity for maximum squeeze pressure was 92% and specificity was 97%.

It is usual to quantify external anal sphincter function by the maximum pressure achieved in the anal canal on voluntary contraction. Some measure this relative to atmospheric pressure, some relative to rectal pressure, some relative to the resting pressure at the same point, and some relative to the maximum resting pressure (even if this is found at a different point along the anal canal). There is no consensus on which of these is the most valuable and none on terminology. Maximal or maximum squeeze pressure, maximal voluntary squeeze and maximum voluntary contraction pressure are all commonly used terms, but do not define what is measured. Presumably maximum squeeze pressure should refer to the incremental increase relative to resting pressure, while maximum contraction pressure should refer to the total pressure generated, relative either to rectal or to atmospheric pressure. However, these terms require standardisation.

The maximum duration of squeeze (e.g. > 5mm Hg in lower anal canal) is about 2 minutes; this is often decreased in patients with incontinence. [462] The rate of decrease in pressure can also be greater in incontinent patients. [463] However, the value of these measurements in distinguishing such patients from normal subjects is unknown and requires study. In incontinent patients with diarrhoea, the duration was not different from controls. [450]

In 1998, Marcello et al [464] studied fatigue of the external anal sphincter by manometry and proposed a fatigue rate index based on a 40 second squeeze. They showed that the index was lower in subjects with faecal incontinence than in controls and that it was highly correlated with the degree of incontinence. In 2004, Telford et al [463] modified the index by reducing the time of the squeeze to the more practical value of 20 seconds. They showed that the index was lower in subjects with faecal incontinence than in controls even though there was no significant difference in resting pressure or squeeze pressure. The study of fatiguability demands continued investigation.

***d)* Vector manometry**

This approach assesses the "pressure" profile as measured in a number of radial directions along the length of the anal sphincter. It is recorded both at rest and with voluntary contraction, and is used to locali-

ze any area of anatomical sphincter damage. [434-437,465] A few studies of small numbers of incontinent patients have compared its effectiveness with more objective measures of sphincter damage such as ultrasound, electromyography or MRI. If 8 radially oriented recording tips are used the test can probably detect major damage, especially of the external anal sphincter, but it is not as accurate as intra-anal or intravaginal ultrasound. [435-437,466] or MRI [467,468] If 4 recording tips are used, the test is useless. [434] The reproducibility of vector-manometric studies has not been reported, nor are there adequate studies to properly assess sensitivity and specificity. Furthermore, there are technical objections to such studies. They do not measure sphincter pressure, but directionally dependent forces that vary unpredictably with the stiffness and position of the catheter (section B.I on stress urinary incontinence).

• Transient internal sphincter relaxations

Transient spontaneous relaxations of the internal anal sphincter occur and are considered part of the sampling mechanism whereby proximal anal sensory receptors can distinguish the nature of rectal contents. [469] Patients with faecal incontinence can exhibit an increase in spontaneous episodes of internal sphincter relaxation which are not associated with compensatory increases in external sphincter activity. These episodes last about 45 seconds and in the incontinent patient are more frequently associated with increases in rectal pressure. [439,440] These relaxations have not been adequately evaluated for distinguishing incontinent patients and their diverse aetiologies. For example, they have been reported as increased [470] or decreased [471] in diabetic patients with faecal incontinence. Furthermore, their contribution to incontinence can be viewed from two perspectives: if increased in frequency they may lead to incontinence through excessive relaxation of the anal sphincter, [440] while if decreased they may lead to incontinence through defective anorectal sampling. [469]

e) Anorectal pressure gradient

The gradient (difference) in pressure between rectum and resting anal-sphincter pressure and maximum squeeze pressure has been assessed in patients with faecal incontinence, and found not to discriminate these patients from control subjects. [472]

f) Rectoanal reflexes

Normally, the internal anal sphincter relaxes in response to rectal distension and during attempted defaecation. During assessment, this recto-anal inhibitory reflex may be elicited by distending a rectal balloon with different volumes of air or water. The resting pressure within the anal canal is monitored and, if the reflex is present, a drop in resting pressure is seen. [473-475] During a defaecation manoeuvre the external anal sphincter should also relax. This can be assessed by simultaneous EMG recording of external anal sphincter activity during attempted defaecation, to establish that decreased pressures are indeed associated with inhibition of external anal sphincter activity. Absence of the rectoanal inhibitory reflex is suggestive of Hirschsprung's disease, for which it is the first line screening test, or Chaga's disease. However, absence of rectoanal reflex when the resting anal pressure is low may be an artifact, because relaxation is hard to detect if the pressure is already low.

g) Internal anal sphincter relaxation

In addition to increased spontaneous transient relaxations, incontinent patients have low thresholds for relaxation induced by inflation of a rectal balloon and show post-squeeze or post-strain relaxations. The latter are not usually seen in normal controls. [440] When sphincter pressures are very low, the balloon-induced relaxation may be absent, indicating markedly impaired internal anal sphincter function. [476] These findings require further study to identify their value.

h) External anal sphincter contraction

Rectal balloon distension also induces a transient external anal sphincter contraction that precedes the internal anal sphincter relaxation. [477,478] These contractions, which are considered to be protective, are reported to be decreased in faecal incontinence. [479] These findings require further study to identify their value.

i) Ambulatory anorectal manometry

A number of studies have used micro-transducers or multilumen perfused sleeve catheters, with or without EMG recording of the external anal sphincter, to monitor anorectal motor activity over time. [480-482] Although transient spontaneous anal sphincter relaxation has been seen in patients with pruritis ani, [483] the technology has not been applied to patients with faecal incontinence.

j) Clinical application

In addition to discriminating faecally incontinent patients from controls, anorectal manometry is used

in combination with other techniques to a) attempt to identify some of the dynamic features of the internal anal sphincter and external anal sphincter associated with the incontinence of various aetiologies, and b) monitor the effectiveness of any prescribed treatment. It may help to distinguish and classify the various types of anal incontinence, just as it does for urinary incontinence.

As noted above, the measurement of pressures at rest, with voluntary contraction and in response to rectal distension, can to some extent differentiate faecally incontinent patients from controls, assess regions of sphincter damage, and partially separate internal anal sphincter from external anal sphincter function. However, it should not be surprising that anal canal pressures are not perfectly correlated with continence. There is a wide range of normal pressures, and there are other factors associated with faecal incontinence even in the absence of decreased anal canal pressures. The relationship between these other factors and anal canal pressures requires future study in various clinical conditions causing faecal incontinence.

In certain conditions causing incontinence, there is some consistency in manometric findings. Obstetric injury is associated primarily with external anal sphincter damage and reduced maximum sphincter pressure, but the pressure abnormality may not relate well to the presence or severity of incontinence, and additional abnormality such as decreased rectal sensation is often present. [484-487] In diabetes, similar findings are consistently noted, [470, 471,474] while in addition spontaneous transient internal anal sphincter relaxations are reported as being either increased or decreased. In other conditions such as rectal prolapse, a sphincter pressure abnormality is often found, but its relationship to incontinence has not been determined. [488,489] A similar lack of adequate study is also seen in the descending perineum syndrome, [490] Parkinson's disease, [491] and central and peripheral nerve injury. [492-494]

Anorectal manometry has been one of many techniques used to monitor the effect of numerous medical, surgical and radiation therapies. [495-513] However, the direct relationships of the manometric findings to faecal incontinence and the mechanisms of incontinence are not established in these reports. Research directed to this end is urgently required.

Anal manometry is a tool universally used by gastrointestinal physiologists to quantify closure of the anal canal. These measurements appear to have good inter-operator reproducibility. [514] However, the many variations in devices, technique and terminology limit their universal usefulness and make it difficult or impossible to compare or combine results between centres. Even units of pressure vary from centre to centre. Some use mm Hg, some cm H_2O. A working party in 1989 [515] recommended the use of kPa but this attempt at standardisation failed. Standardisation of terms and units is necessary to understand the place of anal manometry. It would facilitate communication and help any accreditation process. When some standardisation has been achieved it will be possible to begin to determine which measurements are most useful and perhaps establish *Good Anal Manometry Practices*, similar to the *Good Urodynamic Practices* recommended by the International Continence Society. [33]

2. Pudendal nerve terminal motor latency

The external anal sphincter is innervated by the pudendal nerve, arising from the S2-S4 spinal cord region. Terminal latency is measured using a glove-like device with electrodes to stimulate the nerve intrarectally at the ischial spine and to record the response at the external sphincter. Prolonged latencies seen in patients with idiopathic faecal incontinence [516] demonstrate that a neuropathic process may be involved.

In the clinical setting, latency measurement has been found by some [517] but not others [518] to be a useful prognostic indicator for certain anorectal procedures. The diversity of opinion may be partly due to the operator-dependence of the measurement. Even the upper limit of normal (2.2 – 2.6 ms) is debated. [519,520] To obtain reliable results it is essential to deliver a supramaximal stimulus in the correct location, but even if this is done the measurement is inherently insensitive to small amounts of nerve damage and at best distinguishes the clearly normal from the obviously abnormal. Moreover, prolonged latency does not necessarily mean that there is abnormal squeeze pressure or abnormal muscle function. [521] There is a clear need to develop more sensitive and specific tests of muscle and nerve in the lower gastro-intestinal tract that are patient-acceptable and more useful in the research or the clinical setting.

3. External anal sphincter electromyography (EMG)

From the dynamic point of view, electromyography is performed to determine whether the external anal

sphincter contracts or relaxes (i.e. it is a kinesiological EMG). Electromyography can be performed using a needle electrode, [522] [523] a fine wire electrode, [442] a surface electrode on the perianal skin, [524] an anal plug, or an assembly of multiple surface EMG electrodes placed in the anal canal. [525,526]

Surface EMG has been used for biofeedback training [527,528] and in the evaluation of anal sphincter function. The firing pattern of the motor unit potentials is reduced in incontinent patients in conjunction with the lower resting anal-sphincter pressure and maximum squeeze pressure. [524] The quantitative aspect of this EMG information relative to sphincter integrity and function has not been assessed, and it is unlikely that obtaining this data will supplant the easy and well-established use of manometry. As noted above, simultaneous recording of the external anal sphincter activity with EMG helps to ensure that decreased pressures are associated with inhibition of external anal sphincter activity. The multichannel surface EMG can also serve to identify the pattern of innervation and/or denervation of the external anal sphincter in patients with faecal incontinence. [529] The role for this information is still not clear and further testing is required to elucidate it.

To identify areas of both internal anal sphincter and external anal sphincter injury, mapping of the external anal sphincter by needle EMG has been supplanted by ultrasound and MRI imaging and will not be discussed. However, needle EMG is useful for assessing the severity, progression and mechanism of neural injury. [530] EMG of the smooth muscle internal anal sphincter has been performed, [442,531,532] but its clinical value has not been assessed.

4. Rectal sensory-motor and sensory activity

From the dynamic point of view the rectum is of major importance in the maintenance of continence and the production of faecal incontinence. As noted above, sensory-motor reflexes regulating external anal sphincter and internal anal sphincter function and anal sampling are present and active, and can be monitored by anorectal manometry. Two other interrelated aspects of rectal function are also important: a) the cognitive sensations such as the desire to have a bowel movement that motivate voluntary actions such as external anal sphincter contraction or defaecation; and b) the sensory-motor activities that regulate contractile activity of the rectum. These two aspects are inter-related because muscle activity regulating rectal wall tone and contractions also impacts on the mechanisms for cognitive sensation. This discussion will deal primarily with the sensory-motor aspects.

Measurement of the motor activity of the rectum and its sensory-motor relationships is generally performed with two main techniques: simple catheter manometry as in the anal canal; or rectal balloons. In the case of catheter manometry, the rectum can be challenged with infusion of a substance such as water while the rectal pressure is measured. With a balloon, the volume–pressure relationship of the rectum can be assessed also.

a) Irritability of the rectum

Irritability of the rectum that is associated with an early and vigorous rectal contraction and the sensation of urgency has been considered a factor contributing to faecal incontinence. The threshold volume of distension or fluid for induction of a motor response has been used to assess this irritability in conditions such as irritable bowel syndrome and inflammatory bowel disease. [533-537] Another approach has been to infuse saline [450] or a substance with stool-like consistency such as porridge [538] into the rectum, and then monitor the time (and volume) of first leak, and the total volume leaked. This latter testing will not only be subject to the rectal motor response, but also the anal sphincters' ability to resist stress. The clinical value of these assessments is unknown, although the responses may relate to the sensory thresholds and/or to rectal compliance.

b) Rectal tone and compliance

Increased rectal tone and decreased rectal compliance, that is a "tight rectum", can be perceived as contributing to incontinence in a number of ways, such as reducing the reservoir capacity of the rectum and/or serving to accentuate the rectal motor response to sensory stimulation such as distension. Neither of these mechanisms has been carefully assessed or applied in detail to the dynamic motor aspects of faecal incontinence resulting from various aetiologies. More attention has been directed to the relationship of these mechanisms to cognitive sensation, [455, 461, 539,540] especially in patients with irritable bowel syndrome (IBS) where faecal incontinence is common. [541,542]

For measurement of rectal tone and compliance, the rectum can be distended using an isometric or an iso-

tonic (isobaric) technique. With the isometric technique, the inflation occurs at a fixed flow rate and the pressure is measured at specified volumes or times. However, there is no agreement as to where on the curve to select endpoints. Moreover, the balloon is blind, and it is uncertain when it engages the rectal wall or how far it extends up the lumen. Consequently the results are variable and difficult to compare. With isobaric distensions, a constant pressure is maintained by altering the volume in the organ in response to rectal tonic or phasic contraction, and to changes associated with the resistance to flow of the balloon catheter. The volume is measured and related to the pressure maintained in the system.

Regardless of which technique is used, the rectum is a dynamic organ and its physical and reflex responses, as well as its fundamental physiological principles and physical properties, must be considered. [543,544] Constant-rate infusion while measuring pressure with the isometric technique, and continuous ramp or ramp-step increases in pressure while measuring volume with the isobaric technique, both produce an s-shaped curve. Such a curve has a sound physiological basis. For example, inflation at a constant rate of filling results in an initial rapid rise in pressure, followed by a slower rise, before another more rapid rise ensues, [545] while a steadily increasing distension pressure results initially in little increase in volume, followed by a rapid increase in volume, which then slows down again. [544,546,547] These findings are attributed to an initial resistance to distension due to resting tone (less compliant), followed by a decrease in tone due to receptive reflex-mediated relaxation (more compliant), and finally increased resistance to stretch as further lengthening of the rectal wall is limited by less compliant passive elastic components. It has been suggested that compliance should only be measured after conditioning stimuli have been applied to stabilize the reflex accommodation. [447,463,544,548] However, this approach may effectively remove important physiological components normally contributing to compliance. [543] Furthermore, the relationship of compliance to sensation may be subject to a similar distortion since the sensory receptors responsible for symptom reporting appear to be behave as if in series with the contractile elements. Finally, the calculation and expression of compliance as either “dynamic” or “static” introduces other methodological differences. [543] Independent of the aetiology of the faecal incontinence, the methodology used and whether or not conditioning stimuli have been introduced can also contribute to the variability of the findings that have been reported.

In general, rectal compliance in patients with faecal incontinence has been reported as decreased in many conditions. [461,470,472,489,494,496,539] However one study reports normal compliance. [455] Since the mechanisms resulting in decreased compliance will vary depending on the clinical disorder, further research is necessary to establish the role of compliance and its changes in faecal incontinence.

It is common to use a simple balloon distension technique (without manometry) to measure the threshold (volume) for sensation, sensation of desire to defecate, and maximum tolerated capacity. However, this technique has not been validated.

5. Imaging of the anorectal area

From the dynamic point of view, primarily two imaging techniques are employed, defaecography and magnetic resonance imaging (MRI). Neither is normally combined with simultaneous pressure measurement. Endoanal ultrasonography is used primarily to identify damage to the anal sphincters, and is especially useful for the internal anal sphincter, while MRI may be superior for the external anal sphincter, including assessment of muscle atrophy. [549-552] Endoanal ultrasound can be carried out at the same time and by the same personnel as anorectal manometry. MRI is an important research tool but is only used in selected cases in the clinical setting.

Defaecography is an X-ray study of the anal canal and rectum. It involved inserting a thick barium paste into the rectum, usually until the patient has some desire to defecate. Images are taken (and recorded on videotape) at rest, on voluntary contraction of the sphincters, on straining without defaecation, and on evacuation. This procedure can identify various anatomical features such as rectal prolapse, perineal descent, rectocoele, enterocoele, inappropriate contraction of the anal sphincters and/or puborectalis, and abnormal anorectal angle. It is mainly used in cases of difficulty with defaecation (which may paradoxically coexist with incontinence). However, there is no evidence that the information obtained in this way is as good as or better than a clinical examination or other imaging techniques, nor that it is of significant value in patients with faecal incontinence uncomplicated by prolapse or difficulty with defaecation. [431,432,553,554]

Dynamic MRI imaging has also been used to assess

the pelvic floor musculature in real time and during procedures such as straining and defaecation. This technique can be performed using a phased-array coil placed around the pelvis. [555] The procedure can assess dynamic aspects such as contraction and relaxation of pelvic floor musculature, pelvic organ prolapse, and pelvic floor descent. [556-558] However, this procedure is still evolving, the optimal technique is still not established, and its use and value in faecal incontinence are still to be assessed by further research. [553,554]

6. Other dynamic testing techniques

One other technique, that of measuring the force and travel of the levator ani musculature, has been proposed and reported on for patients with faecal incontinence. [443,456] This test appears to have a good correlation with the severity of incontinence, as well as being a predictor of response to biofeedback therapy. [456] Further research is necessary to establish its value.

III CONCLUSIONS: FAECAL INCONTINENCE

1. Recommendations for clinical practice

Anorectal manometry is an optional test that may be used in difficult-to-evaluate cases of faecal (anal) incontinence.

2. Recommendations for research

1 Establishment of normal values for parameters obtained by anorectal testing

2 Determination of the reliability, reproducibility and association with symptoms of anorectal testing parameters, using appropriate statistical methods

3 Determination of the prognostic value of anorectal testing parameters.

F. CONCLUSION

I. GENERAL

1. Knowledge-based medicine

In this chapter we have argued that:

a Urodynamics (and by extension, dynamic testing for faecal problems) is the only way of obtaining quantitative knowledge of function and dysfunction in the field of incontinence

b Any intervention for incontinence that is based on knowledge of the (dys)function to be treated must rely on urodynamic assessment

c Whenever pathophysiology is uncertain or unpredictable, and especially if irreversible treatment is necessary or contemplated, it is an ethical requirement to gather as much quantitative knowledge of the dysfunction as possible in order to make a rational treatment choice

d In such situations, the aim of urodynamic evaluation is to identify all factors contributing to the dysfunction, expected or unexpected; therefore the evaluation must be comprehensive

e In most patient groups, for example children or patients with neurogenic incontinence, the pathophysiology is unpredictable and comprehensive urodynamic evaluation is essential in order to practice knowledge-based medicine (see recommendations below)

f There are a few well-defined, uncomplicated patient groups in whom treatment without urodynamics may be justified. Women with uncomplicated stress incontinence, confirmed by history and physical examination (stress test) and without voiding difficulties, are an example. Complicated or unclear cases however require urodynamics

g Within such well-defined patient groups, the principles of evidence-based medicine can be applied. There is limited current evidence (and randomized trials to increase the evidence base are required), but such information as there is may suggest that quantitative knowledge of the dysfunction does not greatly affect treatment choice or outcome

h Reasons why treatment within these patient groups can be carried out without attempting to obtain such knowledge by urodynamics include:

Non-specific, non-quantitative therapies that apply equally well or poorly in all circumstances (due to lack of understanding of aetiology and pathophysiology)

Poor choice of urodynamic variables (resulting from a lack of understanding of biological mechanisms)

Poorly carried out urodynamics

i Reasons why the attempt to obtain knowledge may confuse rather than enlighten the practitioner include:

Intrinsic variability of urodynamic parameters

Consequent weak associations with symptoms

Poorly carried out urodynamics

Poor interpretive skills

j Some of the intrinsic variability of urodynamic parameters is not due to poor measurement but reflects the operation of neural control, especially cerebral control. The origin of this variability is not understood, but deserves intense study, since what can vary physiologically can be controlled, for diagnostic or therapeutic purposes.

2. Training and certification

Urodynamic evaluation is required in complicated cases in which the underlying pathophysiology is uncertain. In such cases, the aim is not just to "reproduce the symptom" but to identify all the factors that may be contributing. Thus, a comprehensive and reliable evaluation of the function and dysfunction of the lower urinary tract is required. In those strictly defined, straightforward patient groups where invasive urodynamic evaluation is not important it can be omitted. There is therefore no clear place for simplified urodynamic testing performed and interpreted by questionably qualified personnel.

Because good urodynamics is not easy to perform, it is important to examine sufficient numbers so that abnormalities can be judged against a background of more nearly normal cases. Therefore urodynamics should be performed in specialized centres that are accredited to do so, and that handle large volumes of patients. Accreditation criteria should be based on staff training (certification and/or the following of recognized training procedures), evidence of practice in accordance with guidelines, and an adequate volume of patient examinations.

At the primary or secondary levels of care it would be unreasonable to require that all patients should receive comprehensive urodynamics. Thus complicated cases, or cases in whom incontinence remains bothersome in spite of appropriate conservative therapy, would be referred to a specialized laboratory. Whether the remaining straightforward cases require urodynamics at all, especially if they are to receive conservative treatment, is questionable. Nevertheless, *if* urodynamics is to be performed at a secondary or even primary level, it should be simple (for example, it might aim just to "reproduce the symptom") and it should be certified and reimbursed appropriately.

II RECOMMENDATIONS FOR CLINICAL PRACTICE

The following recommendations are at Grade C except where noted.

1) Invasive urodynamic studies should be performed in accredited urodynamic laboratories, by trained and certified staff, with formal control of the quality of the results. To this end the **establishment of national accreditation, training, certification and quality-control programs** is highly recommended.

2) A simple assessment of possible **anorectal dysfunction** by history and physical examination should be performed whenever lower urinary tract function is evaluated.

3) **Non-invasive urodynamic testing**, including a voiding/incontinence record or diary, determination of post-void residual urine (preferably by ultrasound), and uroflowmetry, should be performed prior to treatment in every patient.

4) It is recommended that **invasive urodynamic evaluation** should be tailored to each individual and should include at least:

- Filling cystometry (with provocation)
- Pressure-flow study of voiding

together with one or more of the following, as indicated for the individual patient:

- Abdominal leak-point pressure measurement
- Urethral pressure measurement (made by a technically sound method)

- Simple neurological assessment
- Simple assessment of anorectal abnormalities

5) **Invasive urodynamic evaluation** is recommended:

- Prior to invasive or irreversible treatment or re-treatment of all types of incontinence except for the following:
  - Uncomplicated female stress incontinence
  - Uncomplicated female urge incontinence
  - Post-void dribbling in males
  - Male urge incontinence, associated with other typical LUTS, for which surgical therapy has not been performed nor is planned
  - Functional disorders in children that respond to a trial of behavioral therapy
  - Incontinence in frail elderly patients in whom conservative management has not been attempted
- Whenever there is any doubt about the underlying pathophysiology, or doubt whether the incontinence is truly uncomplicated
- In neurogenic bladder dysfunction, as an initial assessment and as part of long-term surveillance (Grade of recommendation B)

6) For **children** it is recommended that:

- Those with neurological or anatomical abnormalities affecting the urinary tract should be investigated with invasive urodynamics if invasive treatment is considered or if there is any risk of irreversible deterioration if treatment is omitted
- Such children should have an evaluation of bowel function as well
- If conservative therapy fails in children with functional disorders then they should receive comprehensive urodynamic investigation in a urodynamic laboratory specializing in pediatric testing

7) In **frail elderly** patients with incontinence, measurement of post-void residual urine by a noninvasive method is recommended before instituting pharmacological or surgical treatment for the incontinence. It should be repeated to monitor the effect of such treatment at an appropriate time.

8) For patients with **faecal (anal) incontinence**, it is recommended that dynamic anorectal testing should be considered if therapy based on simpler assessments fails to yield the desired improvement.

III RECOMMENDATIONS FOR RESEARCH

1 The committee recommends that no new therapy should be introduced without extensive urodynamic testing of all accessible aspects of its effect on lower urinary tract function and dysfunction. Such urodynamic testing should form an essential part of the evaluation of new therapies such as botulinum toxin injection.

2 Research is recommended in many areas in which current knowledge is inadequate, including:

- Design and performance of randomized controlled trials that may provide objective evidence of the utility of performing urodynamics in well-defined patient groups, including those with and without overt neurogenic dysfunction
- Development of new and more complete biomechanical models of normal and abnormal pelvic and bladder outlet mechanics, and of new methods of measuring urethral, vaginal and rectal pressure
- Development of new urodynamic tests, for example tests of urethral properties, with a sound technical and physiological basis
- Integrated approaches to the study and management of urinary and faecal (anal) incontinence
- Definitive studies of the reproducibility and physiological variability of urodynamic parameters in normal subjects, as well as in specific patient groups, such as women, men, children, frail elderly, and patients with various types of neurogenic dysfunction
- Study of cerebral and emotional control of lower urinary tract function
- New methods of recording afferent signals during cystometry
- Establishment of normal values for parameters obtained by anorectal testing
- For anorectal testing parameters, determination of reliability, reproducibility, association with symptoms, and prognostic value
- Establishment of standardisation for terminology and techniques in anorectal testing

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