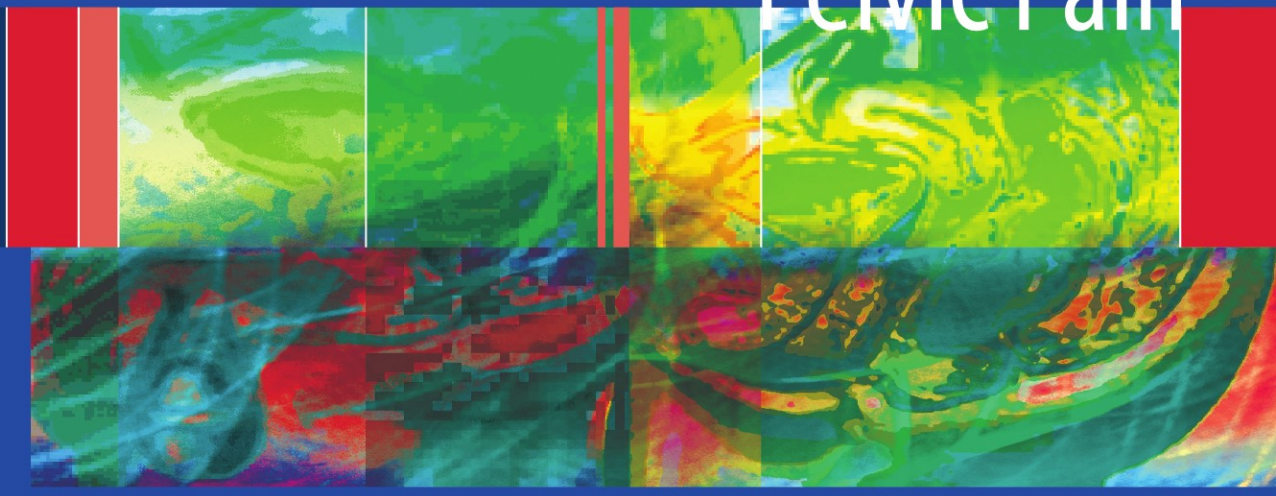
Jeanette Haslam
Jo Laycock *Editors*

Therapeutic Management of Incontinence and Pelvic Pain



Pelvic Organ Disorders
Second Edition

Springer

J. Haslam and J. Laycock (Eds)

Therapeutic Management of Incontinence and Pelvic Pain

Pelvic Organ Disorders

2nd Edition

Springer

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This book is dedicated to our husbands, Bob and David, for their continued love and support, and to our children who never fail to amaze us with their achievements.

J. Haslam and J. Laycock
Editors

Foreword to the Ist Edition

As medical knowledge advances we tend to compartmentalise our specialties into smaller units; but, hand in hand with this, there is a growing understanding between the different disciplines within the caring professions. Thus we are able to share our special skills to the benefit of patients. This book is an excellent example of the advantage of interdisciplinary communication and demonstrates a refreshing holistic approach to the problems of incontinence and pelvic pain. Written with physiotherapists in mind, the editors have invited contributions from many distinguished experts in their own field. These have been compiled into a comprehensive book, which will appeal to many healthcare professionals.

I have had great pleasure in reading this book. During the time that I have been involved with 'pelvic dysfunction' there have been many exciting advances. These are all included in a most readable sequence, some presented with a refreshing new twist. In particular, I would like to bring to your attention the section on 'pelvic pain'. Because of our lack of understanding it has been a problem that is too often ignored and here at last are some practical ideas for therapeutic management.

There is still much progress to be made in the field of incontinence and pelvic pain and as yet, no editors can be expected to produce a definitive work. However, I would like to recommend this book most strongly. It has a new approach to this topic, which is still a major problem for many people.

Angela Shepherd, MD, FRCOG, MCSP
Southmead Hospital, Bristol

Foreword to the 2nd Edition

It has been a pleasure to follow the development of the second edition of 'Therapeutic Management of Incontinence and Pelvic Pain'. This builds on the success and popularity of the first with updates and new sections on all aspects of pelvic floor dysfunction, including pelvic organ prolapse and faecal incontinence. It is most pleasing that the emphasis remains with therapy and evidence-based recommendations.

The Editors' extensive knowledge and experience in pelvic floor muscle training through exercise physiology and research has resulted in many organisations, including the National Institute for Clinical Excellence (NICE) in England, and the International Consultation on Incontinence, recommending pelvic floor muscle training as first line therapy, as well as prevention of urinary incontinence

This and other conservative measures can produce good outcomes and the new chapters on training techniques plus recommendations for future research such as improving compliance are welcome.

Where these treatments are unsuccessful surgery is an option and this too is included, further enhancing the quality of the book.

Children's problems, bowel disorders, pelvic pain, quality of life and ethical issues are often forgotten aspects of pelvic floor dysfunction and will be of great help to those new to this 'disease area' as well as experienced clinicians.

The Editors emphasise the importance of collaboration within a multi-disciplinary team and it is particularly pleasing to see a chapter on the role of the occupational therapist. As clinicians committed to the management of patients with pelvic floor dysfunction this collaboration can only improve our understanding of the conditions and our treatments.

We are indebted to the Editors for gathering such a wide range of international experts to write about so many relevant and important aspects of the pelvic floor and making this such a valuable reference; it is a 'must' for the library!

The first edition has been extremely popular and I have no doubt the second will continue to help clinicians in their everyday practice providing further benefit for patients with these distressing conditions.

Bob Freeman
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Preface to the 1st Edition

In times past incontinence was a taboo subject, never to be mentioned in public, to family or friends. Going to a doctor with a complaint of either urinary or faecal incontinence usually resulted either in advice to use pads or being referred for surgery. Times have now changed, men and women demand ever more of the healthcare system, and research evidence has shown that incontinence can be well managed and in many cases cured by the use of appropriate therapies. Likewise, other pelvic floor disorders, leading to constipation, pelvic pain and prolapse can be treated by a variety of healthcare professionals, each bringing different skills to ameliorate their patients' symptoms. There is a growing body of research evidence and consensus on which to base clinical practice, and there are also several "alternative" therapies, which need scientific evaluation.

The purpose of this book is for many different specialists including doctors, nurses, physiotherapists and researchers within various fields of work to present their work with a critical review of the literature, and to identify and give recommendations for both practice and possible further research.

The book provides a clear, concise and practical clinical introduction to the assessment and conservative treatment of pelvic floor disorders in both men and women.

Section I looks at the prevalence, anatomy and aetiology of urinary incontinence, including the neurological patient and the elderly. Clear guidance is given regarding assessment and the investigations available. Section II contains chapters that look at specific therapeutic management strategies by exercise, biofeedback, vaginal cones and electrical stimulation. The importance of patient compliance to therapy is discussed and the use of plugs and devices considered, as are the appropriate types of surgery available. The male patient receives particular consideration as does bladder training, voiding problems and drugs acting on the lower urinary tract. Section III considers colorectal disorders, including an overview of the possible problems and the therapies available. Section IV is concerned with all aspects of pelvic pain and its therapeutic treatment and management, and section V looks at the various aspects of pelvic organ prolapse. Section VI considers osteopathy and the diversity of alternative therapies that are at present practised by clinicians with specialist knowledge and training: these include acupuncture, reflex therapy and homeopathy relating to pelvic disorders. The final section looks at the issues that need to concern health professionals including research trials, audit and infection control issues.

The book is written primarily for physiotherapists working in this specialist field. However, it is hoped that anyone working with patients suffering with incontinence or other pelvic disorders, such as nurse continence specialists, students of 'alternative' therapies, osteopaths, urologists, gynaecologists and general practitioners will increase their knowledge and all find something of use within its covers.

We are most grateful to Angela Shepherd for agreeing to write the foreword. Dr Shepherd initially qualified as a physiotherapist before medical training, becoming renowned internationally as a specialist urogynaecologist; she is the ideal person to give words of wisdom on the subject.

Jo Laycock and Jeanette Haslam

Preface to the 2nd Edition

Due to the World Health Organisation, International Continence Society, many other societies, and several government initiatives, patients with bowel and bladder disorders world wide should now expect improved standards of care. These organisations have pooled research and clinical expertise contributing to a multi-professional approach in treating many pelvic floor disorders. Consequently, many of the contributors to this book are international experts from various professions, who have helped to set the standards for evidence-based ethical practice, to improve the quality of life for the millions of sufferers world-wide.

Divided into sections, this well referenced book will complement under-graduate and post-graduate formal lectures, demonstrating the value of Integrated Continence Services. In this way, all professions working in this specialist field of medicine will better understand their role in promoting continence and ameliorating pelvic floor disorders in men, women and children.

In this second edition there are updated chapters from the first edition - some with new authors - and also the introduction of several new topics. In section 1, there are new chapters on children's continence problems, and also on quality of life issues.

Section 2 incorporates new chapters on pelvic floor muscle training using real-time ultrasound biofeedback, manual therapy, exercise balls, the effects of childbirth and the menopause on the pelvic floor muscles and related disorders. Further chapters are presented on the impact of urinary incontinence on athletic women, the value of having an occupational therapist in the service, and the use, and misuse, of catheters in managing urinary incontinence.

Sections 3, 4 and 5 have all been updated and include the assessment and conservative treatment of faecal incontinence and constipation, pelvic pain management, and the evaluation, treatment and prevention of pelvic organ prolapse. Finally, Section 6 also has new chapters on ethical issues relating to research, investigations and treatment, and a new chapter on UK Government Guidelines. The opinions expressed in each chapter are those of the individual authors.

We are very grateful to Bob Freeman for writing the foreward. Mr Freeman is an internationally renowned urogynaecologist with a wealth of experience in both the surgical and conservative management of incontinence; who better to appreciate the multi-professional concept of this book.

Jeanette Haslam and Jo Laycock

Acknowledgments

We would like to thank all the authors and co-authors who have contributed to this book; we think they have made a remarkable contribution to the understanding of pelvic floor disorders. Also, our thanks to all at Springer for their patience and assistance.

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I Urinary Incontinence in Adults and Children with Bladder and Bowel Disorders

1
Prevalence of Urinary Incontinence

S Elneil

1.1 Introduction

The prevalence of urinary incontinence (UI) in men and women has been shown to vary widely from 2 to 60% in subjects living at home[1–3] (see Table 1.1) and 23 to 93% in institutionalised patients.[4,5]

Exactly how common incontinence is depends on which study you read and the definitions and methods used to determine the numbers. In general, the main conclusions are that it is much higher than you might expect, and that prevalence increases with age; childbearing being the most significant risk factor in women.[6,7] Although the prevalence of male UI is less common, it is important to note that the association of incontinence with disabilities, such as reduced mobility and cerebrovascular and neurological disease,[8] affects both genders equally (see Table 1.2).

Other contributory factors in prevalence studies on UI are the countries and populations studied[9–13] (see Table 1.1), anatomical and physiological differences[14–18] between genders and races, as well as socioeconomic and cultural factors of the communities studied.[19,20] However, the extent of the influence of these factors on women and men's health remains relatively unknown.

Table 1.1. A summary of selected studies of the prevalence of urinary incontinence in subjects living at home

Authors	Study Group	Method	Prevalence
Brocklehurst (1993)[1]	4,007 Adults over 30 years old	Postal questionnaire	6.6% Men 14% Women
Ko et al. (2005)[42]	141,815 Adults over 65 years old	Postal questionnaire	20.9% Men 27.5% Women
Milsom et al. (1993)[43]	10,000 Swedish women selected randomly from 7 birth cohorts	Postal questionnaire	24.6% 1900 Cohort 13.9% 1920 Cohort 12.1 % 1940 Cohort
Hunskaar et al. (2004)[44]	29,500 Women over 18 years old in Spain, France, Germany, United Kingdom	Postal questionnaire	23% Spain, 44% France, 41% Germany, 42% United Kingdom
Wong et al. (2006)[45]	540 Chinese women aged 17–77 years old	Telephone questionnaire	40.8% Urinary stress incontinence 20.4 Urge incontinence 15.9% Mixed incontinence
Biri et al. (2006)[46]	2,601 Turkish women over 15 years old	Structured interview	16.1% Urinary stress incontinence
Song et al. (2006)[47]	3,371 Korean women aged 30–89 years	Structured interview	47.5% Urinary stress incontinence 0.4% Urge incontinence 15.5% Mixed incontinence
Teunissen et al. (2004)[48]	5,278 Adults over 60 years old	Postal questionnaires	19% Urinary incontinence 3% Urinary and faecal incontinence
Stenzelius et al. (2004)[49]	4,277 Adults over 75 years old	Postal questionnaires	39% Urinary incontinence 14.5% Urinary and faecal incontinence

Table 1.2. A summary of selected studies of the prevalence of urinary incontinence in subjects living in institutions and other groups

Authors	Study Group	Method	Demographics	Prevalence
Damian et al. (2004)[50]	800 Adults in 45 nursing homes	Structured interview of staff and patients	Mean age 83.4 years 44% Cognitive impairment 33% Dementia 14% Alzheimer's disease	53% Overall prevalence
Aggazotti et al. (2000)[4]	839 Adults in 14 nursing homes	Clinical record review	Mean age 82 years	59.8% Women 39.2% Men
Tivlis et al. (1995)[51]	649 Adults in nursing homes	Structured interview	3 age cohorts with baseline prevalence rates of urinary incontinence as below: 75 year cohort 10.5% 80 year cohort 20.4% 85 year cohort 28.6%	51.8% in those with severe depression 37.6% in those with previous stroke 48.8% in those with dementia
Hennessey et al. (1999)[52]	221 Patients with multiplesclerosis	Postal questionnaire	Mean age 39.2 years	14% Permanent indwelling catheter 44% Bladder dysfunction
Sakakibara et al.(2001)[53]	115 Adults with Parkinson's disease	Postal questionnaire	Average age 59 years old	42% Urinary urgency in women 54% Urinary urgency in men

1.2 Prevalence of Urinary Incontinence and Age

In an extensive study by the Royal College of Physicians in 1995, the prevalence of incontinence in the adult population increased with age and was more common in women than in men.[21] In women (15–44 years) living at home, the prevalence was 5–7%, whereas in women aged 45–64 years it was 8–15%, and in those over 60 years it increased to 10–20%. In men aged 15–64 years living at home, the prevalence of UI was 3%, whereas in those over 65 years it was 7–10%. In institutionalised adults, the rates were significantly higher (see Table 1.2). Other studies have mirrored these findings.[22–24] In a review of 21 studies that compared the prevalence of incontinence based on frequency, age, and gender, Thom found that incontinence was 1.3- to 2 times more common in older men and women.[25] Overall reported rates were higher if in-person interviews were used for datacollection.

In the United States, more than 13 million adults and children experience incontinence. This accounts for 1 in 20 of the population, with over half of all cases occurring in women. In a study of American women the prevalence of UI in those over the age of 65 years was 35%, while in those under 65 years it was 10%.[19] In a similar study of men, the prevalence of UI was 22% in those over 65 years and 1.5% in those under 65 years.[25] Although the prevalence of incontinence was generally higher in the elderly and the institutionalized (30–50% of all adults), it affects many young people, too, but it usually disappears naturally over time, which suggests that incontinence, for some people, may be a normal part of growing up.

1.3 Prevalence of Urinary Incontinence in Women

Urinary incontinence is more common in women, but few seek medical advice, because they consider their symptoms to be slight and not worth reporting.[26,27] Some recent epidemiological studies described below give an indication of the prevalence of incontinence in women and what factors may be involved.

A study of 2,900 Italian women aged between 18 and 50 years found that the overall prevalence of UI was 20% and it increased steadily with age.[28] In women between 25 and 30 years, the incidence of UI was 10%, but that increased to 23% for women between 35 and 40 years and 35% for women

between 45 and 50 years. Of the 20% of women who experienced UI, 83% reported symptoms of stress incontinence, 44% symptoms of urge incontinence, and 47% noticed that the start of their incontinence problem coincided with the delivery of a baby.[28] Surprisingly, only 20% of these women had seen a doctor about their problem. A much larger study of 28,000 women over the age of 20 years conducted in Norway yielded similar figures.[29] The authors found the overall prevalence of UI to be 25%. Even in younger women, aged 20 to 24 years, 10% reported minor symptoms of incontinence. In women over 50 years old, the prevalence was 30%. Most of these women reported moderate or severe symptoms.[29] A similar age-related prevalence of UI (35% in women over 50 years old) was reported in an Austrian study of 1,262 women.[30] Of the women who suffered from UI, 66% stated that the quality of their life was impaired because of their incontinence.[30]

1.3.1 Parity

Parity is another important factor in the prevalence of incontinence.[18] A recent study in the United Kingdom looked at the incidence of UI before, during, and after pregnancy in 492 primiparous women.[31] The prevalence of UI pre pregnancy was 3.5%. During pregnancy, it rose staggeringly to 36%, but within the first 3–5 days after birth, it dropped to 14%, a figure that was sustained at 3 months post delivery. Two features of this study were of great interest: the high prevalence of incontinence during pregnancy and the persistence of UI symptoms for at least 3 months if women experienced incontinence in the first few days of the postnatal period. Yet, the impact of incontinence was small in this study, as most women reported that it only had "a little" effect on their lives.[31]

In a longitudinal study of 4,214 parous women carried out over 6 years in the United Kingdom and New Zealand, the impact on women's lives was greater.[32] The prevalence of persistent UI (at 3 months and 6 years after index birth) was 24% overall, with caesarean births being a protective though not a preventative factor, as 14% of women who had exclusive caesarean births had persistent UI. There were no significant associations between persistent UI and operative vaginal delivery, but there were significant associations with increasing number of births and older maternal age.[32]

1.4 Prevalence of UI and Other Factors

Other anecdotal accounts suggest that the prevalence of UI may be adversely affected by strict toilet training practices in childhood,[33,34] obesity, and a previous history of hysterectomy.[35] It may occur less in certain races such as African-Americans and Asian-Americans when compared to white Americans[36,37] and may be affected by socioeconomic factors.

In a study of 863 Andorran women (prevalence of UI was 37%), UI was perceived as a far more bothersome and disabling condition by working, middle-class women than in other socioeconomic groups.[38] Women in this particular group are more limited by UI and less likely to seek medical advice. Socioeconomic status also was implicated when trying to explain racial differences in UI knowledge.[37] In a study of Caucasian and ethnic minority women in California, UI knowledge was statistically higher among the Caucasian group until the scores were adjusted for socioeconomic status; this showed no statistically significantly differences among the different racial groups,[20] which may be an important consideration when developing UI educational programs. Cultural norms in certain societies, such as different sports undertaken by female athletes, also affect UI prevalence.[12,39] A series of studies found that a large number of athletes suffered from urine leakage during exercise, ranging from 6 to 67%, depending on the study and on the sport. The highest rate of urine leakage observed was among gymnasts.[12] Interestingly, these women only experienced urinary leakage during intense physical exercise but not when coughing or sneezing.[39]

1.5 Conclusion

All UI prevalence studies give an indication of the number of people who are managing this condition at any given time and all show how large a problem UI can be, particularly in the elderly.

Due to increasing longevity, it is anticipated that there would be a greater demand for continence services. The cost of these services, which can account for over 2% on the national health budget,[40,41] need to be contained. The only way to achieve this is by raising awareness and improving our knowledge of UI, understanding the risk factors and instituting preventative strategies, and improving our service provision by acquiring accurate epidemiological data by using validated questionnaires. By using these comprehensive methods, we can meet this economically challenging problem.

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2
Anatomy

UM Peschers and JOL DeLancey

The pelvic floor is a complex structure, having not only to allow the passage of urine or stool at appropriate times but also to preserve continence daily. It is necessary for sexual activity, conception and fertility and is also the birth passage at the time of vaginal delivery.

The following descriptions offer a brief overview of some clinically relevant aspects of pelvic floor structure that help us to understand the normal and abnormal behaviour of this system.

Table 2.1. Topography of urethral and paraurethral structures

Approximate location	Region of the urethra	Paraurethral structures
0–20	Intramural urethra	Urethral lumen traverses the bladder wall
20–60	Midurethra	Sphincter urethrae muscle Pubovesical muscle Vaginolevator attachment
60–80	Perineal membrane	Compressor urethrae muscle Urethrovaginal sphincter muscle
80–100	Distal urethra	Bulbocavernosus muscle

2.1 Female Pelvic Anatomy

2.1.1 Pelvic Viscera

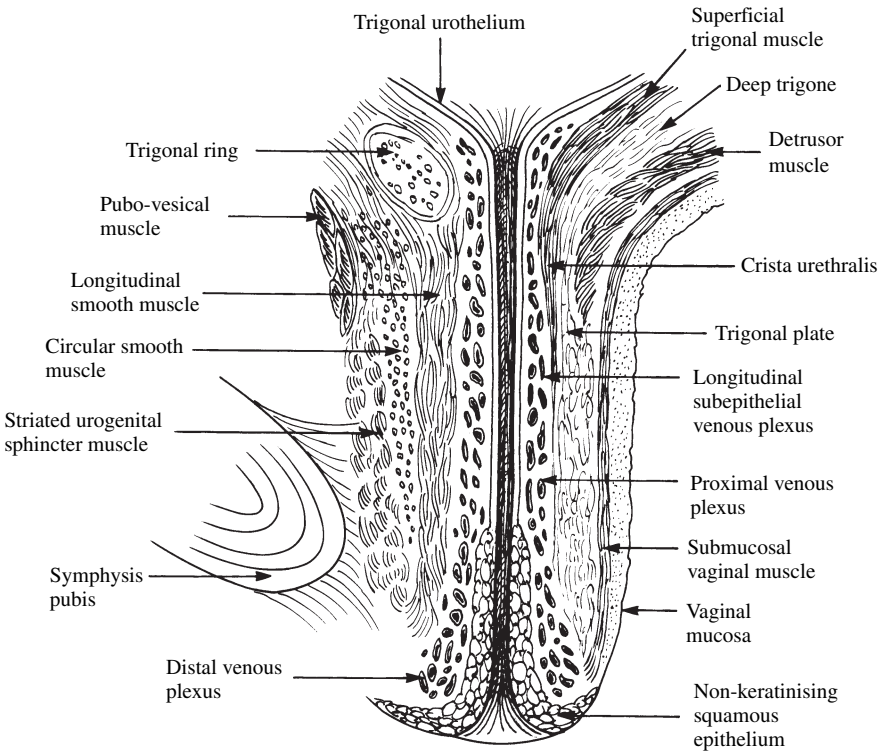
The lower urinary tract can be divided into the bladder and urethra. At the junction of these two continuous yet discrete structures lies the vesical neck. The bladder consists of the detrusor muscle, which is covered by a serosa layer at its dome and lined by a submucosa and transitional epithelium. Two prominent bands on the dorsal aspect of the bladder that are derived from the outer longitudinal layer pass beside the urethra to form a loop on its anterior aspect, called the detrusor loop.[1] On the anterior aspect of this loop, some detrusor fibres leave the region of the vesical neck and attach to the pubic bones and pelvic walls; these are called the pubovesical muscles.

The trigone is a structure at the bladder neck whose visible apices are formed by the ureteric orifices and the internal urinary meatus. The urethra is a complex tubular viscus with its upper third clearly separate from the vagina and the lower two-thirds intimately connected to the lower anterior vaginal wall. Embedded within its substance are a number of elements that are important to lower urinary tract dysfunction,[2] summarised in Table 2.1.

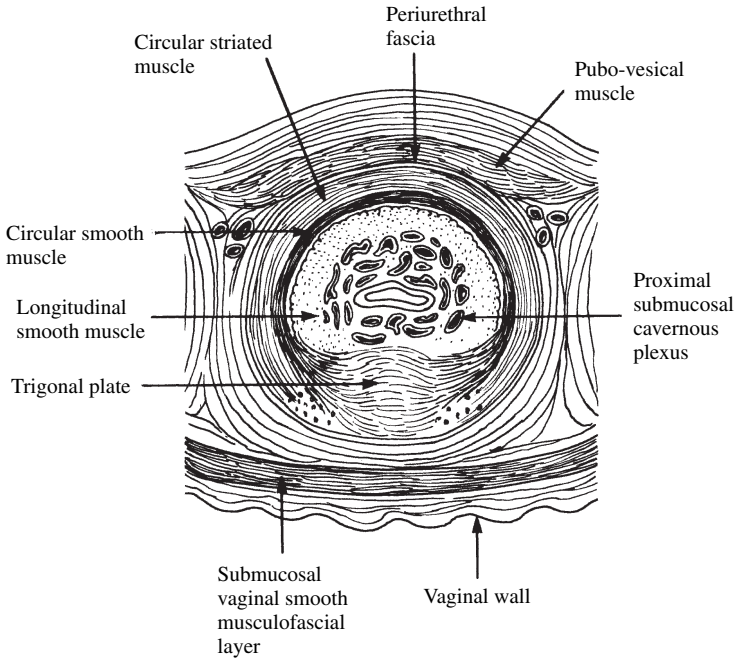
2.1.2 Striated Urogenital Sphincter

The outer layer of the urethra is formed by the muscle of the striated urogenital sphincter (Fig. 2.1). In its upper two-thirds, the sphincter fibres lie in a primarily circular orientation; distally, they leave the confines of the urethra and either encircle the vaginal wall as the urethrovaginal sphincter, or extend along the inferior pubic ramus above the perineal membrane (urogenital diaphragm) as the compressor urethrae.

The sphincter is composed largely of slow-twitch muscle fibres,[3] which are well suited to maintaining the constant tone as well as allowing voluntary increases in urethral constriction when increased closure pressure is needed. In the distal



a



b

Figure 2.1. Sagittal section (**a**) and axial section (**b**) of a female urethra. [Strohbehn K, DeLancey JOL. The Anatomy of Stress Incontinence. Operative Techniques in Gynecologic Surgery 1997; 2: 5–16.]

urethra, this striated muscle compresses the urethra from above, and proximally, it constricts the lumen. It has been suggested that it is responsible for approximately one-third of resting urethral closure pressure.[4]

2.1.3 Urethral Smooth Muscle

The smooth muscle of the urethra is contiguous with that of the trigone and detrusor. The fibres of the inner layer are directed longitudinally and are surrounded by a thinner circular layer. The smooth muscles lie inside the striated urogenital sphincter muscle, and are present throughout the upper four-fifths of the urethra. The configuration of the circular muscle suggests a role in constricting the lumen, and the longitudinal muscle may help shorten and funnel the urethra during voiding although its exact function is not known.

2.1.4 Submucosal Vasculature

Lying within the urethra is a well-developed vascular plexus which is more prominent than expected for the ordinary demands of so small an organ.[5] The flow of blood into the large venules can be controlled, assisting in forming a watertight closure of the mucosal surfaces.

2.1.5 Mucosa

The mucosal lining of the urethra is continuous with the transitional epithelium of the bladder and with the squamous epithelium of the vestibule below. The mucosa is hormonally sensitive and under-goes significant change depending on its state of stimulation.

2.1.6 Vesical Neck

The vesical neck is the area at the base of the bladder, where the urethral lumen passes through the thickened musculature of the bladder base. Therefore, it is sometimes considered as part of the bladder musculature, but also contains the urethral lumen studied during urethral pressure profilometry. It is a region where the detrusor musculature, including the detrusor loop, surrounds the trigonal ring and the urethral meatus. The vesical neck has come to be considered separately from the bladder and urethra because of its unique functional characteristics.

2.2 Vagina and Uterus

The distal third of the vagina is fused with the urethra anteriorly, to the perineal body posteriorly and the perineal membrane and levator ani muscles laterally; it is lined with stratefied nonkeratininzing squamous epithelium. The support of the vagina varies at different levels. In the upper third of the vagina, it is suspended on its lateral wall to the pelvic sidewall by the paracolpium, an ill-defined body of connective tissue that also contains smooth muscle, elastic fibres, nerves and vessels. The cervix is directly connected to the vaginal wall. Posterior to the cervix, the vaginal wall is covered on its dorsal wall with the perineum of the cul-de-ac. As the vagina is directed posteriorly in a normal woman, the upper third of the vagina and the cul-de-sac are closed like a flap-valve by intraabdominal pressure by being compressed against the rectum and levator plate. The middle of the vagina is connected laterally to the pelvic walls by the pubocervical and rectovaginal fascias.

2.2.1 Rectum and Anal Sphincters

The rectum is located posteriorly in the pelvis and is composed of a mucosa, a submucosa, a circular and a longitudinal layer of smooth muscle and a serosa. The anterior wall of the rectum is located directly adjacent to the posterior vaginal wall.

The anal sphincters are a complex system for faecal continence, with the anal canal having a pressure level to secure a waterproof closure. The innermost layer is the mucocutaneous lining of the anal sphincter complex. The mucosa is surrounded by the internal anal sphincter muscle, a thickened prolongation of the circular smooth muscle layer of the bowel. The function of the internal anal sphincter has been discussed for decades, just as the detailed anatomy of the anal canal was a subject of debate. Currently the internal sphincter is thought to be responsible for 75% of resting pressure of the anal sphincter system.[6,7] The smooth muscle of the internal sphincter is well suited to its job of generating a constant tension over long periods of time. Between the internal and the external anal sphincter are the fused longitudinal fibres of the intestinal wall and levator ani muscles that continue into the superficial part of the external anal sphincter

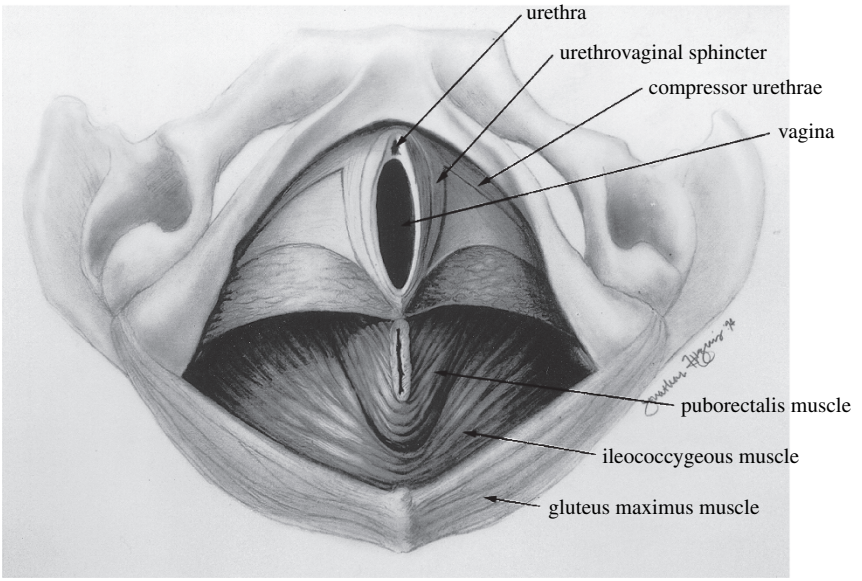


Figure 2.2. Levator ani muscles seen from below. That portion of the pubovisceral muscle which inserts into the rectum and forms a "U" behind it is called the puborectalis.

muscle. The external anal sphincter composition has been discussed for a long time, and several anatomic models have been proposed.[8–10] In our opinion, supported by studies of sectional anatomic specimens, the external sphincter is mainly composed of two parts: a deep part and a superficial part, separated by a connective tissue layer. The superficial part forms a round structure at the edge and below the caudal end of the internal sphincter. Both parts send connective tissue fibres to the tip of the coccyx. In the female the external anal sphincter is shorter anteriorly than posteriorly.[11]

The third part of the anal continence mechanism is the puborectalis muscle that forms a sling around the rectum. The puborectalis muscle and the deep part of the external sphincter muscle form the characteristic double bump in a sagittal image (Fig. 2.2).

The internal anal sphincter is responsible for the maintenance of resting pressure, but both the external anal sphincter and the puborectalis muscle actively contract and thereby increase anal pressure in situations of faecal urgency. It is well accepted that childbirth may lead to neurogenic damage to the innervation of the external anal sphincter[12,13] and to disruption of the internal and external sphincters.[14] However, little is known about the impact of vaginal delivery on internal sphincter innervation and function. Resting anal sphincter tonus is associated with internal sphincter function and is known to be lower in women after vaginal birth complicated by a third-degree perineal tear compared to controls without rupture of the sphincters.[15]

2.3 Pelvic Floor Muscles

2.3.1 Levator Ani Muscle

The levator ani muscle provides the main muscular support for the pelvic organs and is of crucial importance for pelvic floor re-education. It can be divided into two parts: the pubovisceral muscle and the ileococcygeal muscle.[16] On its inferior and superior surfaces the levator ani muscle is covered by connective tissue, which forms respectively the inferior and superior fascia of the levator ani fascia. The layer formed by the muscle and its fascial layers (superior and inferior) is referred to as the "pelvic diaphragm".

The pubovisceral muscle is a thick U-shaped muscle, which arises from the pubic bones on either side of the midline. It passes behind the rectum forming a sling-like arrangement and also attaches to the walls of the vagina, perineal body and anal sphincter complex (Fig. 2.2).

Laterally, the iliococcygeus arises from a fibrous band on the pelvic wall (arcus tendineus levator ani) and forms a relatively horizontal sheet that spans the opening within the pelvis and provides a shelf on which the organs may rest (Fig. 2.3).

The pubovisceral muscle has several components. Clinicians have often referred to the entire pubovisceral muscle by the term pubococcygeus. This term is somewhat misleading because it signifies a muscle that would connect two relatively immovable structures (pubis and coccyx). Functionally it therefore cannot contribute to any lifting of the pelvic organs, but might contribute to pelvic support by isotonic contraction. The puborectalis portion of the pubovisceral muscle passes beside the vagina, with some attachment to the lateral vaginal walls. The muscle then continues dorsally where some fibres insert into the rectum between the internal and external sphincter while others pass behind the anorectal junction. The vagina attaches to the medial portion of the pubovisceral muscle and the fibres between the vagina and pubic bone are referred to as the pubovaginalis muscle. These muscle fibres are responsible for elevating the urethra during pelvic floor muscle (PFM) contraction; this occurs because the muscle is connected to the fascia supporting the urethra, rather than because the muscle goes to the urethra itself (Fig. 2.4).

The iliococcygeal muscle is a sheet of muscle that spans the pelvis from the arcus tendineus levator ani on one side to the other, passing dorsal to the anorectum and anal sphincter. The pubococcygeus is the most cephalic portion of the levator and passes from the pubic bones to insert on the inner surface of the coccyx and comprises only a small portion of the overall levator complex. Some fibres also pass to the anal canal and behind the rectum. The term pubococcygeus is often used by gynaecologists to refer to the entire pubovisceral muscle and pubococcygeus muscle.

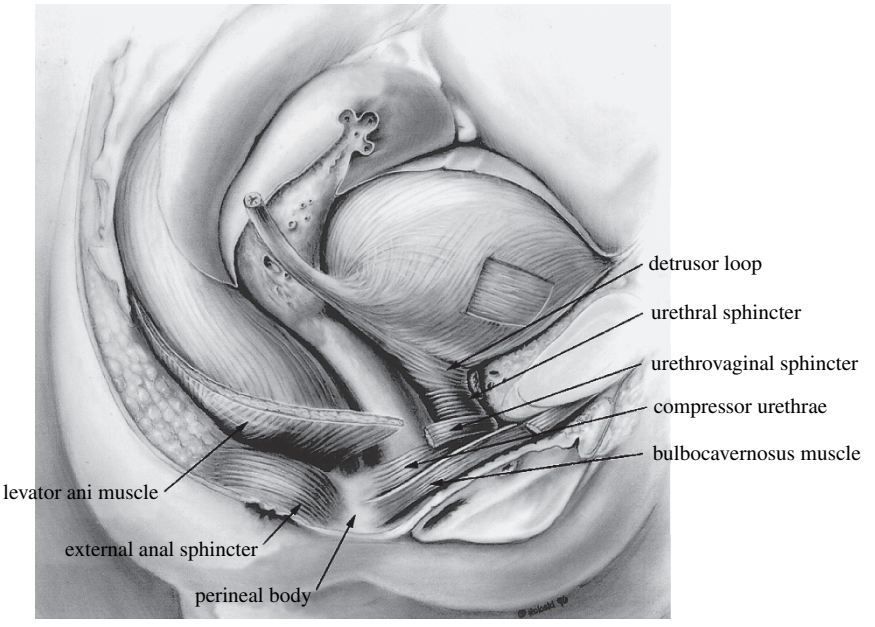


Figure 2.3. Lateral view of the pelvic organs and their relation to the pelvic floor.

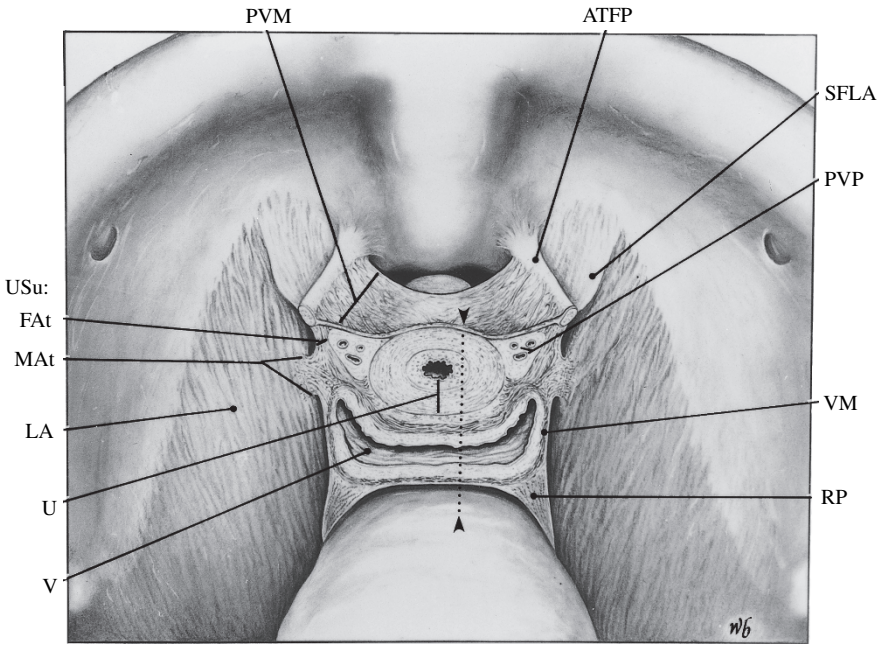


Figure 2.4. Drawing of the urethra (U), vagina (V) and levator ani muscles (LA) just below the vesical neck. The pubovesical muscles (PVM) can be seen anterior to the urethra and the periurethral vascular plexus (PVP) and attach to the arcus tendineus fasciae pelvis (ATFP). Urethral supports (USu) run underneath (dorsal to) the urethra and vessels. Some of its fibres (MAt) attach to the muscle of the levator ani (LA), while others (FAt) are derived from the vaginal wall and vaginal surface of the urethra (U) and attach to the superior fascia of the levator ani (SFLA). [DeLancey JOL. Pubovesical Ligament: A separate structure from urethral supports. Neurourol Urodyn 1989; 8: 53–61.]

The levator ani muscles are composed of striated muscle. It is known that two-thirds of the fibres are type I (slow-twitch) muscle fibres[3] responsible for the resting tonus of the levator ani muscle. One-third of the fibres are type II (fast-twitch) fibres which are thought to act when a quick, powerful contraction is needed.

2.3.2 Perineal Membrane

The perineal membrane (urogenital diaphragm) is a relatively thin layer underneath the pelvic diaphragm. Triangular, with the tip directed anteriorly, it lies at the level of the hymenal ring, and attaches the urethra, vagina and perineal body to the ischiopubic rami. It mainly consists of a connective tissue membrane, with muscle lying immediately above.[17] The striated muscle fibres associated with the perineal membrane (compressor urethrae and urethrovaginal sphincter) act to constrict the distal urethra and have been discussed with that organ.

2.3.3 Bulbocavernous Muscles

The bulbocavernous muscles, the most distal muscular layer of the pelvic floor, merely have sexual function and do not provide pelvic floor support.

2.4 Anatomic Basis of Urinary Continence

The two factors important to urinary continence during increases in abdominal pressure are urethral constriction by the muscles of the urethral wall and urethral stabilisation by the PFM during stressful events. The urinary continence mechanism is complex, still not completely understood and based on the functional co-ordination of several parts of the pelvic floor.

The primary basis for continence is the urethra whose wall contains small muscles (striated urogenital sphincter and urethral smooth muscles)

that are able to generate intraurethral pressure. The support of the urethra is determined by the surrounding suspending structures and by the state of relaxation or contraction of the levator ani muscle. Voluntary contraction of the levator ani muscle causes bladder neck elevation and voluntary relaxation causes it to be lowered – this is easily visualised on perineal ultrasound. The urethra and the levator ani muscle are connected to each other by the arcus tendineus levator ani.

The role of the urethral supportive tissue's connection to the levator ani is important for the following reasons:

- The resting position of the proximal urethra is high within the pelvis, some 3 cm above the inferior aspect of the pubic bones.[18] Maintenance of this position would best be explained by the constant muscular activity of the levator ani.[19]
- In addition, the upper two-thirds of the urethra is mobile[20–22] and under voluntary control.
- At the onset of micturition, relaxation of the levator ani muscles allows the urethra to descend, which obliterates the posterior urethrovesical angle. Resumption of the normal tonic contraction of the muscle at the end of micturition returns the vesical neck to its normal position. Some of the control of the proximal vesical neck's position and mobility, therefore, must come from activity of the levator ani muscles and connections of the periurethral tissues to the fibrous elements of the pelvic wall (ligaments and fasciae).

From these observations, it seems that the support of the urethra involves both voluntary muscle and inert elements.[23,24] Observations that patients with stress incontinence have neurological damage to the pelvic floor support the importance of the levator ani to urinary continence.[25] Functionally it has been shown that when the PFM are paralysed there is a decrease in continence.[26] There does not appear to be a one-to-one relationship between denervation and stress incontinence, and so the relative role of the muscular and fascial tissues in urethral support remains incompletely defined. The anterior vaginal wall and urethra arise from the urogenital sinus, and are intimately connected. The support of the urethra depends not on attachments of the urethra itself to adjacent structures, but on the connection of the vagina and periurethral tissues to the muscles and fascia of the pelvic wall. On either side of the pelvis, the arcus tendineus fascia pelvis is a band of connective tissue attached at one end to the lower one-sixth of the pubic bone, 1 cm from the midline, and at the other end to the ischial spine. In its anterior portion this band lies on the inner surface of the levator ani muscle which arises some 3 cm above the arcus tendineus fasciae pelvis. Posteriorly, the levator ani arises from a second arcus, the arcus tendineus fasciae levatoris ani, which fuses with the arcus tendineus fasciae pelvis near the spine.

It is the urethral supportive structures rather than urethral position that is crucial for the maintenance of urinary continence. Simulated increases in abdominal pressure in anatomic specimens reveal that the urethra lies in a position where it can be compressed against the supporting hammock consisting of the connective tissue layer of the anterior vaginal wall and the lateral suspension structures connecting it to the levator ani muscle (Fig. 2.5).[24] In this model, it is the stability of this supporting layer rather than the height of the urethra that determines stress continence. In an individual with a firm supportive layer the urethra is compressed between abdominal pressure and pelvic fascia in much the same way that you can stop the flow of water through a garden hose by stepping on it and compressing it against an underlying concrete path. If, however, the layer under the urethra becomes unstable, the opposing force that causes closure is lost and the occlusive action is diminished. This is analogous to trying to stop the flow of water through a garden hose resting on soft soil.

2.5 Pelvic Organ Support

Pelvic floor function is understandable only through knowledge of the fascial connection between muscles and viscera. The ligaments and fascia of the pelvic floor are composed primarily of loose connective tissue, smooth muscle, elastic fibres, blood vessels and nerves. They resemble a mesentery more than a skeletal ligament. This is an important concept to keep in mind when the function of these structures is discussed.

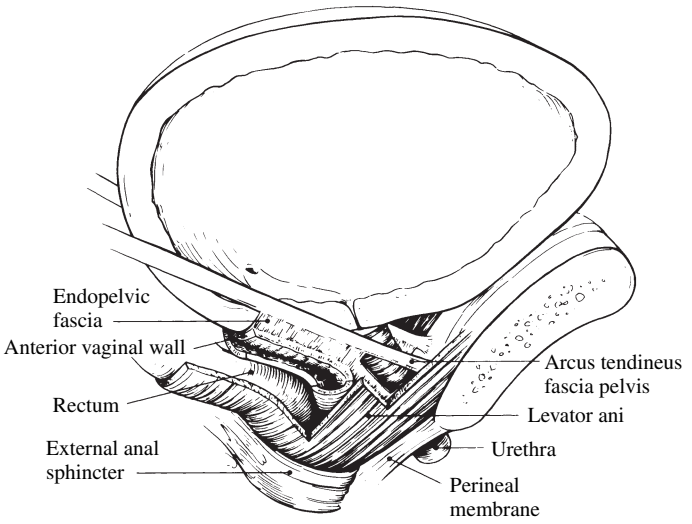


Figure 2.5. Lateral view of the pelvic floor structures related to urethral support seen from the side in the standing position, cut just lateral to the midline. [DeLancey JOL. Structural support of the urethra as it relates to stress urinary incontinence: the hammock hypothesis. Am J Obstet Gynecol 1994; 170: 1713–23.]

The top layer of the pelvic floor is provided by the endopelvic fascia that attaches the pelvic organs to the pelvic walls thereby suspending the pelvic organs,[27–29] it will be referred to as the viscerofascial layer. The endopelvic fascia is a continuous mass of tissue with different thickened parts attaching the uterus and the vagina to the pelvic sidewall.

Withstanding constant forces is not the main function of ligaments. Connective tissue is not suited to withstanding gravitational forces over a long period of time. If the ligaments and fasciae within the pelvis were subjected to the continuous stress imposed on the pelvic floor by the great weight of abdominal pressure, they would stretch. It is therefore illogical to believe that pelvic floor ligaments are the primary factors that prevent the uterus and the vagina from prolapsing. The fact that these ligaments do not limit the downward movement of the uterus in normal healthy women is attested to by the observation that the cervix may be drawn down to the level of the hymen with little difficulty.[30] As in other parts of the body, it is the muscular tonus that holds the organs in place. The opening within the levator ani muscle through which the urethra and vagina pass (and through which prolapse occurs) is called the urogenital hiatus of the levator ani. The rectum also passes through this opening, but because the levator ani muscles attach directly to the anus and external anal sphincter it is not included in the name of the hiatus. The hiatus is therefore bounded ventrally (anteriorly) by the pubic bones, laterally by the levator ani muscles and dorsally (posteriorly) by the perineal body and external anal sphincter. The normal tonic activity of the levator ani muscle keeps the urogenital hiatus closed. On contraction, the levator ani squeeze the vagina, urethra and rectum closed by compressing them against the pubic bone, and lift the floor and organs in a cephalic direction.

The levator ani muscles constantly contract[19] and close the lumen of the vagina. This constant action eliminates any opening within the pelvic floor through which prolapse could occur, and forms a relatively horizontal shelf on which the pelvic organs are supported.[31,32] In the healthy female the vagina is directed posteriorly; the upper part of the vagina is therefore located above and on the levator ani muscle plate. Girls born with neurogenic deficits of pelvic floor innervation such as spina bifida are known to develop pelvic organ prolapse at a young age.[33]

This support of the uterus has been likened to a ship in its berth floating on the water attached by ropes on either side to a dock.[34] The ship is analogous to the uterus, the ropes to the

ligaments, and the water to the supportive layer formed by the pelvic floor muscles. The ropes function to hold the ship (uterus) in the centre of its berth as it rests on the water (PFM). If, however, the water level were to fall far enough that the ropes would be required to hold the ship without the support of the water, the ropes would all break. The analogous situation in the pelvic floor involves the PFM supporting the uterus and vagina, which are stabilised in position by the ligaments and fascias. Once the PFM become damaged and no longer support the organs, the connective tissue fails because of overload.

2.6 Male Pelvic Anatomy

Urinary and faecal incontinence are frequent in females, but less common in males. Pelvic organ prolapse is virtually unknown in men. This might be explained by two basic anatomic differences. Men do not have the genital hiatus of the vagina through which pelvic organ prolapse occurs, and the male urethra is significantly longer and thereby better able to provide continence. Male stress urinary incontinence mainly occurs when the urethral sphincter is damaged during prostatic surgery.

The male urethra extends from the bladder neck to the external meatus at the end of the penis and is divided into three portions: the prostatic, the membranous and the spongy. The prostatic portion is widest and passes through the prostate gland; the membranous part is the shortest and narrowest extending between the apex of the prostate and the bulb of the corpus spongiosum; the spongy portion is the longest section and is contained in the corpus spongiosum. The male urethra is composed of a continuous mucous membrane lined with transitional epithelium. The submucous tissue consists of a vascular erectile layer surrounded by a layer of circular smooth muscle separating the mucous membrane and submucous tissue from the tissue of the corpus spongiosum.

In terms of urinary incontinence the prostate gland plays an important role. Its enlargement (benign prostatic hypertrophy) causes voiding difficulties (obstructed voiding with poor flow, dribbling and overflow incontinence). Radical surgical excision of the prostate for prostate cancer can lead to stress urinary incontinence. The prostate gland is a firm, partly glandular, partly muscular body located immediately below the vesical neck and around the commencement of the urethra. In shape and size it resembles a chestnut. It is located in the pelvic cavity behind the lower part of the pubic symphysis and rests on the rectum. It is easily palpated through the rectum, especially when enlarged. The prostate is immediately enveloped by a thin but firm fibrous capsule. It is separated from the rectovesical fascia by a venous plexus. The prostate gland is perforated by the urethra and the ejaculatory ducts that open into the prostatic part of the urethra.

The two Cowper's glands are located behind the fore part of the membranous portion of the urethra: they are small, rounded bodies the size of a pea. The excretory duct of each gland opens into a minute orifice on the floor of the bulbous part of the urethra.

The penis consists of a root, body and extremity (the glans penis). The root is connected to the os pubis and ischium by two strong fibrous processes, the crura. The suspensory ligament is a strong fibrous band that passes from the front of the pubic symphysis to the upper surface of the root of the penis. The penis is composed of a mass of erectile tissue enclosed in three cylindrical compartments: the two corpora cavernosa side by side along the upper side of the organ, and the corpus spongiosum surrounding the urethra.

2.7 Neurogenic Basis of Continence and Micturition

Continence and micturition are naturally contrary conditions. Because of the bladder's dual function of storage and elimination of urine, many of the neural circuits controlling micturition demonstrate phasic or switch-like patterns of activity, unlike other viscera.[35] Their correct functioning reflects the summary of inhibitory and facilitative neural mechanisms which comprise highly complex central and peripheral, afferent (sensory) and efferent (motor) autonomic pathways that are

integrated and coordinated by cephalic control centres, spinal cord nuclei and infraspinal relay stations in peripheral ganglia.[36]

The following summary in mainly based on the work of Mahony et al., whose papers are highly recommended for further reading.[37–39]

The sacral micturition reflex centre is the important "steering centre" for the regulation of micturition and continence. If it is underfacilitated, voiding can be difficult to initiate or maintain. If it works properly, the patient can maintain continence and is able to void. If the sacral micturition reflex centre is overactive, this will result in detrusor overactivity and urgency. The ability to initiate micturition voluntarily, to empty the bladder completely and to maintain continence in situations when the pelvic floor is stressed is critically dependent on correct function of storage and voiding reflexes. The storage reflexes are responsible for the maintenance of continence, and the voiding reflexes initiate and support complete bladder emptying.

The neural reflex system is based on receptors that are located in the detrusor muscle, in the trigone, in the bladder neck, in the urethra and in the levator ani muscles. These receptors send afferent signals to the sacral micturition reflex centre or to the pontine micturition reflex centre (PMC) which is located in the brain stem. Cortical effects on these two centres are mainly facilitating.

Mahony and co-authors described 12 main reflexes, 4 of which are storage reflexes, 7 are micturition reflexes and 1 stops voiding and reinstalls the storage phase.

2.7.1 Storage Reflexes

Increasing mural tension of the bladder wall activates the sympathetic detrusor-inhibiting reflex and also the sympathetic sphincter constrictor reflex, leading to an increase in urethral pressure. The perineo-detrusor inhibiting reflex is activated by an increase of tension in the PFM, so a contraction of the levator ani muscles can induce an inhibition of the detrusor. Tension of the trigone or entrance of urine into the proximal urethra can activate the urethrosphincteric guarding reflex, resulting in an increase in tone of the external urethral sphincter.

2.7.2 Initation of Micturition and Continued Micturition

The perineobulbar detrusor facilitative reflex is activated by voluntary increase of intra-abdominal pressure through contraction of the diaphragm and the rectus abdominis muscles, and simultaneous relaxation of the pelvic floor muscles. This facilitates the sacral micturition reflex centre. Mural tension with increasing bladder volume activates the detruso-detrusor facilitative reflex, which has a strong facilitating impact on the pontine micturition reflex centre, which in turn facilitates the sacral micturition reflex centre.

2.7.3 Maintenance of Detrusor Pressure During Micturition

To secure complete bladder emptying, detrusor pressure has to be strong throughout micturition and the urethral sphincters have to remain relaxed. The latter is maintained by the detruso-urethral inhibitory reflex, which results in inhibitory impulses to the bladder neck and the proximal urethra. This is supported by the detruso-sphincteric inhibitory reflex inhibiting the striated sphincter. Both reflexes are activated by mural tension of the bladder wall. Receptors located in the urethra also contribute to complete bladder emptying. The urethro-detrusor facilitative reflex increases the excitability of the micturition reflexes. The urethro-sphincteric inhibition reflex is activated by urine passage through the urethra and results in further relaxation of the urethral sphincters.

2.7.4 Cessation of Micturition

When the voiding process is terminated, contraction of the PFM activates the perineobulbar detrusor inhibitory reflex.

In summary, the function of the lower urinary tract is regulated by a complex neural control system located in the brain and spinal cord. This control system performs a simple switching circuit to maintain a reciprocal relation between the reservoir (bladder) and outlet (urethra and urethral sphincter).[40]

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3
Epidemiology of Urinary Incontinence

M Spiteri and V Khullar

3.1 Introduction

Epidemiology is the study of the distribution and determinants of a condition. Prevalence is the probability of having the condition within a defined population at a defined point in time, whereas incidence is the probability of developing the condition during a defined period of observation. For further information on the prevalence of urinary incontinence, See Chapter 1.

Epidemiological studies have attempted to determine the prevalence of incontinence in the general population and to isolate the specific risk factors common to incontinence sufferers. Unfortunately, such studies are limited by the reluctance of women to admit to incontinence, and consequently the true extent of this distressing problem probably is underestimated.

Although urinary incontinence is considered to be a normal consequence of childbirth and ageing, many other factors are important in the development of this condition. Sex, age, ethnicity, smoking, constipation, obesity, connective tissue weakness, genital prolapse, and gynaecological surgery all have been studied as potential aetiological factors.

Urinary incontinence may be the end result of many different pathological processes and considerable research interest has focused on its accurate diagnosis and subsequent management. Central to all of these studies is the common classification of lower urinary tract dysfunction provided by the standardisation committee of the International Continence Society (ICS). The ICS, a multidisciplinary body of scientists and clinicians, first established a committee for the standardisation of terminology in 1976. These standards have been revised and extended (see ICS web site: www.icsoffice.org) to keep pace with improvements in urodynamic techniques and have been adopted worldwide to provide a common framework for clinical and research work in the field of urinary incontinence. Unless otherwise stated all definitions referred to in this chapter conform to the standards recommended by the ICS. One must note however, that in all studies the terms used to define the condition, the time period of the study, the methods used to obtain the data, and the population as well as the location being surveyed must be carefully noted. Thus a community-based population would be different from that seeking medical attention. Such factors make comparison among studies difficult.

The complex symptomatology of lower urinary tract dysfunction and the common presentation of predominantly mixed symptoms mean that diagnosis based on urinary symptoms alone often correlate poorly with urodynamic findings. Versi et al. analysed the value of urinary symptoms for the prediction of stress incontinence (SI) in 252 patients and correctly diagnosed 81% of cases with a false-positive rate of 16%.[1] These findings have been confirmed by other investigators.[2,3]

3.2 Incidence and Remission Rates

Most epidemiological studies are cross-sectional but only longitudinal observations will determine the true natural history of incontinence. The most comprehensive longitudinal survey is limited to a population over 60 years of age.[4] The one-year incidence of incontinence for women was 22.4% and at the second-year follow-up the incidence

was 20.2%. The one-year remission rate was 11.2% and for the second year of follow-up for women who reported incontinence during the first year, it was 13.3%.[9] In another series, the one-year incidence for young and middle-aged women was 3% and 6%, respectively.

3.3 Risk Factors

3.3.1 Age and Gender

Urinary incontinence is more common among women than men and its prevalence increases with age.[5,6] Of significance, the prevalence of SI decreased with age, whereas urge incontinence increased in a population of 3,110 Danish women aged between 30 and 59 years.[7] A problem encountered by prevalence studies among older women is that many consider their urinary symptoms to be a normal part of the ageing process rather than a manifestation of disease. In a study by Gjorup et al., over 50% of patients over the age of 75 considered their urinary symptoms to be normal.[8]

Bladder function changes with age, with a fall in maximum bladder capacity in the eighth and ninth decades secondary to a decrease in bladder sensation.[9] Additionally, nocturia becomes more common with advancing age.

Fluctuations in oestrogen levels throughout the life of a healthy female have been shown to result in changes in lower urinary tract function.[10,11] Menopause is associated with an increased incidence of urinary symptoms.[12] In addition, urinary tract infections become more frequent.[13] At present the importance of oestrogen deficiency compared to that of the ageing process per se is not clear. In a review of the literature published from 1966 to 1997,[14] there was no difference in incontinence among pre- and postmenopausal women in some studies, whereas in others there was a reduced prevalence following menopause. The effect of oral oestrogen on urinary incontinence also is controversial but has been reported to increase the prevalence of SI in some epidemiological studies.[15,16] These reports should be interpreted with caution and prospective randomised studies designed to investigate urinary incontinence are required.

Physical conditions such as dementia, faecal impaction, decreased mobility, confusional state, drugs such as diuretics as well as hypnotics, heart failure, renal problems, diabetes as well as other metabolic and endocrine derangements result in additional causes of incontinence not frequently encountered by younger women. Many of these conditions are reversible and should be elicited by a thorough clinical history and physical examination. Improved life expectancy has resulted in a steady increase in the size of the elderly population and more research is required to alleviate urinary symptoms in this group of patients (See Chapter 5) as well as allow for the increase in financial cost of continence care.

3.3.2 Ethnicity

Inevitably, there are enormous biases in studies investigating the differences of medical conditions, not least of which are the differences in perception and reporting of urinary symptoms by women of various ethnic origins. Consequently, very few reliable epidemiological data are available regarding the racial differences of incontinence.

Bump et al. compared clinical and urodynamic parameters of 54 black and 146 white women referred for evaluation of incontinence and severe prolapse.[17] The proportion of black and white patients with severe prolapse was similar, but there was a significantly lower prevalence of urodynamic SI (27% vs. 61%) and an increased prevalence of mixed incontinence (17% vs. 11%) as well as detrusor overactivity (DO) (56% vs. 28%) in black women compared to white women.

In 1977, Zacharin reported a low prevalence of uterine prolapse and SI in Chinese women. Dissections on cadavers showed differences in the pelvic supporting tissue in Chinese compared with Caucasian females.[18] The general anatomic relationship of the levator ani muscles and urethra was similar to that found in Western women. The levator ani muscle bundles of the Chinese cadavers, however, were judged to be thicker and extended more laterally on the arcus tendineus than in white female cadavers. In addition, the fascia of the pelvic diaphragm extending from the levator ani muscles to the posterior pubourethral ligaments was particularly dense. In 1996, Brieger et al. reported that an audit of major

gynaecological surgery for benign conditions in five major public teaching hospitals in Hong Kong revealed that genital prolapse and/or urinary problems were not uncommon.[19] These authors concluded that there appeared to have been a significant change in the prevalence of these two conditions in Hong Kong in the previous 20 years. This apparent increase may represent a legacy of Western diet or practices, as well as the consequent increase in longevity and in the standard of living in Hong Kong Chinese. Previous underreporting of the condition by women might play a significant role.

3.3.3 Pregnancy and Childbirth

Several studies reported a significant association between incontinence and parity.[7,20] Incontinence was less common among nulliparous women of all ages, but no difference in the prevalence of incontinence was noted for women who had one, two, or three vaginal births.[5] Women who had four or more vaginal deliveries were more likely to report regular incontinence. The association of parity and incontinence mainly is due to an increase in the incidence of SI and postpartum DO and appears to be little affected by pregnancy or childbirth. Cardozo and Cutner showed an increased incidence of DO during pregnancy.[21] Most symptoms, however, resolved postpartum. Many of the irritative bladder symptoms of pregnancy do not appear to correlate with objective urodynamic diagnoses.[22] When women who had three elective caesarean sections were compared to age-matched women having three vaginal births, caesarean section was associated with a lower risk of urinary incontinence. Women in this study were selected from a London teaching hospital database of 40,000 women delivering between 1977 and 1998. The first vaginal delivery also was strongly associated with later surgery for SI in another study, but the effect of parity disappeared with age.[23] In this same study involving 27,900 women, the strongest association of incontinence was found in women aged 20 to 34 years (RR of 2.2; CI 1.2 ,2.6) in primiparous and multiparous (RR 3.3; CI 2.4, 4.4) women. The association was weaker in women over 35 and under 64 years with no association among women over 65 years.[23]

Childbirth injury has been implicated as the major aetiologic factor in SI, and there is histological[24] and electromyographic (EMG) evidence of injury to the pelvic floor postpartum.[25] Concentric needle EMG, pudendal nerve conduction tests, and assessment of pelvic floor muscle (PFM) contraction using a perineometer were used by Allen et al.[26] Re-innervation in the PFMs occurred in 80% of those studied. Women who had a long active second stage of labour and had heavier babies (over 3.4 kg) showed the most EMG evidence of nerve damage. Forceps delivery and perineal tears did not affect the degree of nerve damage seen. Data on the effects of specific obstetric factors such as instrumental vaginal deliveries, foetal weight, smoking or episiotomy are inconsistent. The mechanism by which denervation and other damage of the pelvic floor translates into development of urinary incontinence or other pelvic floor disorders are not clear.

3.3.4 Connective Tissue Factors

Research has suggested that the collagen of women with prolapse and SI may be abnormal and predispose them to develop these conditions. In one study, abdominal wall collagen was stiffer in women with SI and collagen of the pubocervical fascia from SI women was weaker than that of continent controls.[24] Type I collagen forms thick strong fibre units, whereas type III collagen forms thin, weak, and isolated fibres. Keane et al. performed periurethral biopsies on 30 nulliparous women with urodynamically proven genuine stress incontinence (GSI) and found a decrease in type I collagen compared to type III collagen, as well as a reduction in the total amount of collagen in GSI sufferers compared to continent controls.[27] Other researchers reported similar findings among parous women with genital prolapse and GSI.[28]

3.3.5 Smoking, Obesity, and Constipation

Any condition resulting in a chronically elevated intra-abdominal pressure is likely to increase the risk of developing or exacerbating SI. Obesity, smoking, chronic coughing, and chronic constipation have been suggested as important predisposing factors in the causation of urodynamic stress incontinence, although

insufficient data exist to refute or confirm these assumptions. In addition, surgery for urodynamic SI may be technically more difficult in obese patients with increased postoperative complications. Such predisposing factors also may increase the risk of recurrent incontinence, although again this is unproven and further research is required.

Bump et al. studied 13 morbidly obese women before and after surgically induced weight loss. Weight loss resulted in a significant improvement in incontinence obviating the need for treatment of incontinence in the majority of women.[29] In a large case-controlled study the same researchers noted a two- to threefold increase in the incidence of all forms of urinary incontinence among cigarette smokers.[30] Although the increased incidence of chronic chest complaints is likely to exacerbate SI, the effects of nicotine on the bladder require further evaluation.

Spence-Jones et al investigated 23 women with SI, 23 with uterovaginal prolapse, and 27 control women. Straining at stool and an increased bowel frequency were significantly more common among women with SI and prolapse than among controls.[31]

3.3.6 Hysterectomy and Urinary Incontinence

Incontinence may be a complication of hysterectomy but reports are inconsistent. There are many potential mechanisms of incontinence following hysterectomy, namely derangement of the bladder supports, pelvic nerve damage, as well as oestrogen deficiency resulting from concomitant oophorectomy. In addition, postoperative catheter care may be suboptimal and prolonged overdistension of the bladder resulting from urinary retention can lead to chronic bladder hypotonicity. Although cross-sectional studies showed an increase in urinary incontinence many years following hysterectomy, one prospective study reported no increased incidence of incontinence.[14]

3.3.7 Genital Prolapse and Urinary Incontinence

Genital prolapse and urinary incontinence are common conditions, and therefore it is not surprising to find both in some women. Anterior vaginal prolapse may be the cause of voiding difficulties, and in one study 60% of women with severe genital prolapse but no urinary incontinence had underlying urinary incontinence revealed by urodynamic testing.[32] Forty-one percent of their patients had urodynamic SI following reduction of the prolapse with a ring pessary. In the same study, 30% of women with either small or large cystoceles had DO and a higher percentage had symptoms of urge incontinence. Similarly, in another study, among 97 women, 63% of patients with SI had genital prolapse while 62% of those with prolapse had urinary incontinence.[33] In contrast, the severity of the prolapse only showed a weak correlation with the degree of urine incontinence or any other symptoms of pelvic floor dysfunction.[34] A decrease in urethral closure pressure with SI following prolapse surgery has been reported in other studies.[35]

3.3.8 Radiotherapy and Urinary Incontinence

Radiotherapy is still frequently used for the treatment of locally invasive bladder or cervical cancer.[36,37] Subsequent frequency and urgency of micturition affect many patients, many years after treatment.[38] This is partly attributable to fibrotic bladder damage and partly to denervation supersensitivity. The characteristic urodynamic findings are a reduced bladder capacity and low compliance.

3.4 Conclusion

Urinary incontinence has a high prevalence in the community and it impairs women's quality of life. Few randomised trials attempted to prevent incontinence, namely during and after pregnancy. Pelvic floor physiotherapy was effective at reducing incontinence.[39,40] The results reveal that women with a greater awareness of their pelvic floor have greater voluntary contraction of the pelvic muscles during physical exertion, thus improving continence. More studies are required in this field.

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4
Physiotherapy for Children with Bladder and Bowel Disorders

WF Bower

4.1. Introduction

Until recent times bladder and bowel problems in children were considered to be behavioural in origin and managed without consideration of underlying pathophysiology. With the growth of the field of paediatric urology a greater understanding of the developing bladder has allowed targeted intervention. Physiotherapists are skilled in motor reeducation and the interaction between the autonomic and musculoskeletal systems, and with the addition of a thorough understanding of paediatric urinary and faecal disorders, are well placed to facilitate education and treatment of affected children and their families.

There are two important differences between evaluating and managing bladder disorders in children versus adults. First, effective screening to identify underlying structural, urological, or neurological abnormalities is vital. Significant morbidity is seen in children with undetected anomalies and may range from vesicoureteric reflux through to structural bladder changes and ultimately renal failure.[1] Second, the urinary tract in children is dynamic and one dysfunction can merge into another disorder as the child grows and develops. Without adequate surveillance the therapist may miss such changes. For these reasons physiotherapy intervention as part of a multidisciplinary team is advised.

4.2. Assessment

Children presenting to physiotherapy require prior evaluation to exclude structural changes, current urinary tract infection (UTI), upper tract pathology, and neurological findings. This routinely includes physical examination (spine, abdomen, gait, reflexes, genitalia); kidney, ureter, bladder wall, spine, and gut ultrasound; uroflowmetry; and urine analysis. Investigations to clarify detrusor contractility and level of activity (ie, urodynamic evaluation), bowel transit time, and voiding and defaecation dyanamics are helpful but may be available only in tertiary referral centers.

Most centres have devised a history proforma to facilitate the assessment procedure for paediatric incontinence. These therapist-administered questionnaires identify coexisting symptoms that may not be initially reported by the child or family but are implicated in the underlying pathology. Where possible recently established standardised terminology for paediatric lower urinary tract dysfunction[2] and bowel symptoms[3] should be utilised.

4.2.1. Bladder and Bowel Diary

The bladder and bowel diary, symptom score, quality of life measure, and Child Behaviour Checklist are easily administered clinic-based tools that provide the physiotherapist with baseline data. The ideal bladder diary covers a 48-hour period

and indicates fluid intake pattern, voiding frequency and amount, incontinence, 24-hour urine output, time spent asleep, nocturia episodes and amount voided, nocturnal enuresis and diaper weight, plus amount of first morning void.[2] In order to accurately evaluate bowel dysfunction, which commonly includes infrequent passage of stool, a bowel diary of 14 days is required. Stool passage, size, shape, call to stool versus scheduled toilet visit, and soiling episodes are documented.

4.2.2. Symptom Scores

Symptom scores have recently been advocated to identify lower urinary tract dysfunction, classify severity, and document response to treatment.[4,5] While two such measures have been proposed and aspects of validity demonstrated, neither has yet demonstrated test–retest reliability or sensitivity to intervention. Urinary symptom scores to date do not include adequate evaluation of coexisting bowel dysfunction, a difficult task given that constipation alone must meet a 3 to 5 point criteria and defaecation difficulty carries a measure of subjectivity. Clearly, symptom scores will evolve and in the near future will provide a useful evaluation tool.

4.2.3. Quality of Life

Quality of life (QOL) measures are a validated way of measuring a patient's perspective of their life situation[6] and can be used as potent outcome measures of symptom control following interventions, at times discriminating among treatment approaches. Recently a cross-cultural continence-specific paediatric quality of life measurement tool (PinQ) has been devised, tested psychometrically, and shown to be reliable under test–retest conditions when completed by children from age 6 years.[7,8] This tool is currently undergoing sensitivity testing.

4.2.4. Behaviour

Since psychological disorders likely to limit intervention success are present in up to 40% of children with lower urinary tract symptoms, a screening questionnaire for problematic behaviours should be included in the physiotherapy assessment.[9] The most common tool is the Achenbach Child Behaviour Checklist,[10] which includes both parent and child components. Children with clinically relevant or genuine psychiatric comorbid behaviours require preliminary and often concurrent counseling/psychotherapy to facilitate adherence to and efficacy of continence intervention.

4.3. Symptom Sets

Table 4.1 outlines the major symptom sets commonly seen in children seeking help for bladder and/or bowel dysfunction. Children may present with more than one disorder or develop additional symptoms, although frequently these secondary symptoms have been present but not elicited during evaluation. For a thorough description of current understanding of the associated underlying pathophysiology the reader is referred to Nijman.[9]

The various forms of nonstructural dysfunction may have been acquired during the toilet training years, be a sequel to constipation and associated anal trauma, have developed during a period of dysuria, or be a reaction to unpleasant toilets that lack either privacy or hygienic conditions. Currently there is no evidence to suggest that the overactive bladder is normative in neonates or children, although incomplete emptying and voiding in discrete amounts may be a transient but normal physiological finding in infants. Less commonly lower urinary tract/bowel symptoms may occur secondary to sexual abuse.

4.4. Treatment Goals

Physiotherapy aims to resolve the symptoms of childhood incontinence, urinary urgency, constipation, and faecal soiling and positively impact the grade of vesicoureteric reflux and frequency of urinary tract infections. Objectively, treatment aims to normalize voiding and defaecation dynamics by teaching pelvic floor and sphincter quiescence during elimination. A secondary aim is to regulate volume-appropriate bladder and bowel storage and train spontaneous emptying at this volume.

Table 4.1. Common symptoms sets in children with lower urinary tract symptom +/− bowel dysfunction

Classification	Parent/child complaint	Findings (any or all)
Simple overactive bladder	Wet episodes, urge posturing, frequent toileting, nocturnal enuresis (or nocturia)	Urgency, urge incontinence, small voided volumes, frequent voiding, high flow rates
Nocturnal enuresis	Wet bed (generally > 1 in 14 days)	Incontinence while asleep +/− bladder symptoms during the day
Voiding dysfunction	Wet episodes, urgency, UTI	Incontinence, urgency, recurrent UTI, constipation, alterations of flow, incomplete bladder emptying
Dysfunctional elimination	Wet episodes, urgency, infrequent voiding, faecal soiling, difficult or infrequent bowel actions	Incomplete/dysfunctional bladder emptying, sphincter/pelvic floor activity during void +/− defaecation, constipation
Giggle incontinence	Wet when laughing	Insensible or urge leakage during laughter
Vaginal entrapment or mechanical obstruction	Wet clothes immediately or soon after toileting	Toilet position, labial compression/adhesion or incomplete removal of clothing induces symptoms
Voiding postponement	Child refuses to toilet when he/she obviously needs to	Acquired/behavioural postponement of micturition until incontinence occurs; co-exisiting clinically relevant behavioural symptoms
Faecal soiling	More than a smear of stool in the underwear on a regular basis	Poor rectal sensation, disordered defaecation, incomplete emptying +/− constipation
Constipation	Opens bowel < 3 times per week, strains, often painful, +/− blood on toilet paper	Slow gut transit or dysfunctional defaecation dynamics

4.5. Intervention Strategies

Therapy begins with education of the child and family, exploring each person's understanding of the lower urinary tract and explaining the child's specific dysfunction. Visual aids that are age appropriate, backed up by written information for parents, are vital. Equally important is time spent debunking assumptions of an association between incontinence and low intelligence, naughtiness, dirtiness, or laziness. When the problem can be attributed to factors external to the child's control, the child is a guilt-free and equal party able to collaborate in the treatment. Motivation is an integral part of intervention success, and it has been suggested that this can be explored by asking the child to identify three positive and three negative aspects of having lower urinary tract dysfunction.[11] A child who cannot conceive of any downside to his or her dysfunction may not be ready to commence therapy.

Physiotherapy intervention is multimodal, with initial strategies described as standard therapy versus full-spectrum therapy where all treatment options are offered. The distinction between approaches is immaterial and mentioned only to clarify the terms as they appear in relevant publications. The basic premise of any intervention for lower urinary tract dysfunction is to facilitate routine bladder filling and unopposed emptying.

4.5.1. Fluids

It has long been held that drinking relatively large volumes of water will normalize bladder storage capacity that has been impaired by an underlying overactive bladder. However, there is no evidence to support a curative association between high fluid intake and resolution of bladder symptoms in childhood. Increased drinking will not alter voiding mechanics per se nor normalize gut transit or defaecation dynamics. Regular hydration, however, will reverse voluntary dehydration and dilute concentrated urine, thereby relieving some symptoms of irritative urgency and help reestablish a cycle of routine filling and emptying.

4.5.2. Voiding Interval

Standardising a child's voiding interval is fundamental to any intervention. Voiding every 2–3 hours prevents pelvic floor holding maneuvers implicated in dysfunctional voiding, routine detrusor overdistension associated with incomplete bladder emptying, and evolution of an overactive bladder into an underactive system. Routine voiding may also reestablish sensory awareness for age-appropriate volumes of urine. The crucial factor is that voiding should be unopposed by pelvic floor or urethral muscular activity.

4.5.3. Motor Control

Physiotherapists are skilled at facilitating or retraining motor control. Children with urgency, dysfunctional voiding, or altered defaecation dynamics have often learned to alter the normative pattern of pelvic floor and sphincter muscle relaxation during elimination. Initially a response to urgency induced by repeated detrusor pressure rises associated with an overactive bladder or as a voluntary withholding behaviour; in anticipation of pain, selective pelvic floor contraction becomes routine. Successful retraining of elimination (emptying both bladder and bowels) begins with teaching pelvic floor muscle awareness, then effective contraction and relaxation, followed by task specificity. Dysfunctional voiding is not an indication for pelvic floor muscle strength training. However, incontinent children with long-standing lung disease, obesity, or overactivity of upper abdominal muscles, such as in elite athletes, may have true pelvic floor muscle weakness and benefit from strength retraining.

4.5.4. Muscle Awareness

Pelvic floor muscle awareness can be taught via a whole body approach, using techniques such as progressive relaxation, seated ball work, and known synergistic patterning. Once the child achieves some proprioception of pelvic girdle muscles, activity of the abdominal and corset muscles can be explained. A full-length mirror is helpful to demonstrate rectus and oblique activity and active relaxation (often perceived as a lengthening) of the lower abdominal muscles. It has been suggested that pelvic floor contraction cannot occur during transverse abdominal relaxation and that the latter muscle can be monitored by palpation medial to the anterior superior iliac spines.[12] Thus, pelvic floor muscle relaxation with simultaneous abdominal relaxation may be trained using visual feedback or self-palpation of the lower abdominal muscles that share a common fascia with the PF. A hand–held mirror may provide visual feedback to a child who has difficulty understanding the concept of pelvic floor/perineal elevation and descent associated with contraction and relaxation.

4.5.5. Biofeedback

More commonly, bladder clinics utilise electromyographic (EMG) biofeedback systems with electrodes placed on the perineum, around the anal sphincter, inside the anal canal, and on the abdominal wall.[13, 14] Children learn proprioception of the pelvic floor structures by observing the visual and auditory feedback when they attempt pelvic floor activity.[15] More recent biofeedback units engage the child in motor control training by interfacing games with muscle activity.[16] While positive gains have been reported from EMG biofeedback, it is often possible to use accessory muscles or other manouevres to generate the required pressures. A biofeedback system should always be used in conjunction with thorough instruction and supervision of motor activity during muscle training.

4.5.6. Defaecation and Voiding Training

Motor training is of little value if it is not accompanied by application to the life situation, in this case, to voiding and defaecation. Since the sitting posture can be engineered to promote maximal pelvic muscle relaxation, all children may benefit from initial voiding training in the sitting position. Optimal positioning ensures adequate leg support, either by feet on the ground or the use of a footstool. Legs should be abducted to shoulder width, hips flexed, trunk extended but inclined forward, and elbows resting on knees. Active abdominal relaxation without valsalva precede the voiding effort. Real-time uroflowmetery can be used during the

training voids to demonstrate the resultant flow curve and to identify any significant volumes of postvoid residual urine. Caution is needed when using voiding curves to identify voiding dysfunction or its resolution as detrusor pressure during simple uroflowmetry is unknown.

The adjunctive use of pelvic floor or abdominal EMG during uroflow retraining displays the timing of relaxation efforts with respect to voiding. Abdominal or perineal ultrasound also may have a biofeedback role in voiding retraining, since the position of the bladder base can be viewed during initiation of void and the early phase of flow. Obviously the limitation with this tool is that a reasonable volume of urine is required to allow visualisation.

Home training of optimal voiding is currently limited, although use of both portable uroflow units and handheld EMG machines during weekends has been proposed. Voiding technique should be reviewed frequently and the child offered more intense training if either voiding coordination or symptoms do not improve. Strategies proposed include half-day training with another age- and gender-matched patient followed by regular clinic reviews, a series of biofeedback sessions, hospitalisation for a week of intensive training, or attendance at a live-in bladder rehabilitation camp with other sufferers.

4.5.7. Electrotherapy

Electrotherapeutic physiotherapy intervention is common in adults with urgency, urge incontinence, and stress leakage. Less frequently intravesical stimulation is offered for an underactive bladder. The underlying rationale is that therapy has a direct effect on the central nervous system by artificially activating neural structures, thereby facilitating both neural plasticity and normative afferent and efferent activity of the lower urinary tract. The application of current is also known to change the availability of neurotransmitters and in the case of bladder overactivity to both reduce cholinergic and increase beta adrenergic activity. In children with an overactive bladder, transcutaneous and percutaneous neuromodulation delivered over either the sacral outflow or peroneal region of the ankle at a frequency between 10–25 Hz has proven a useful adjunctive treatment.[17–19] As yet there are no randomised controlled trials of paediatric neuromodulation nor any predictors of efficacy; however, clinical improvements in the number of wet episodes and intensity of urgency have been reported.

4.5.8. Clean Intermittent Catheterisation

Where a child presents with infrequent voiding (1/2 urinary tract infections) and on evaluation is at risk of developing a decompensating bladder, it is useful to consider the inclusion of clean intermittent catheterisation.[20] Usually taught by the team nurse, this technique prevents bladder overdistension and allows the detrusor muscle to begin a voiding contraction at a more appropriate length–tension ratio. The child is instructed to void at regular intervals and the residual volume of urine drained with catheterisation. Even quite young children can be taught to empty their bladder with clean intermittent catherisation (see Chapter 23). Over time both sensation of bladder fullness and contractile ability may improve.

4.5.9. Bowel Dysfunction

Bowel dysfunction is known to be associated with urinary incontinence, voiding dysfunction, and urinary tract infection,[21,22] although the precise mechanism of interaction remains unclear. All children should be questioned closely with respect to possible constipation or defaecation difficulty. Therapy aims to produce a second daily or daily pain-free bowel action with a clearly perceived call to stool that the child answers without withholding. Management will include training of unopposed defaecation with an optimal posture and effective abdominal activity. A child may require dietary changes, the addition of a bulking agent, stool softener, osmotic laxative, or enema.

4.6. Summary

Aside from purely therapeutic intervention the physiotherapist addresses specific issues raised by the child or family. This varies from finding solutions to situation-specific episodes of incontinence, containment advice, contact with the

school/teacher through to support of parents and caregivers. All empathic clinicians can foster a child's psychological well-being, and through problem-solving and persistent effort actively enhance their quality of life.

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5
The Ageing Lower Urinary Tract

AS Wagg

5.1. Introduction

The major problem defining the changes in lower urinary tract function associated with age, especially in humans, has been that of ascribing causation. Studies in aged humans have been typified by a lack of age-matched controls and it is only recently that to an extent this has been rectified. Since the first edition of this book there has been considerable advance in the understanding of observed changes and this chapter will illustrate what is currently understood about age-related changes in lower urinary tract function, drawing upon human data.

5.2. Basic Architecture

The bladder muscle (the detrusor) is made up of smooth muscle cells in bundles surrounded by a matrix of collagen and elastin. The smooth muscle cells are not arranged in any particular orientation; each is sheathed by a collagen sheet. These loosely join to unite the muscle bundles. Each individual myocyte is enclosed within its own collagenous matrix. This linkage enables the tension generated by myocytes to be transmitted across the entire muscle. Elastin fibres appear to be arranged in a loose network around these muscle fascicles.[1]

Most studies concerned with changes associated with ageing in the bladder are derived from cross-sectional analysis of human bladder specimens, and thus ascribing the reported changes as being due to ageing is difficult. Most have noted an increase in bladder weight,[2,3] and an increase in the collagen to smooth muscle ratio in the order of 20–30% in the elderly when compared to younger controls. The absolute increase in collagen appears to be greater for females.[4] The reason that the ratio does not fall in men may be due to the effect of bladder outflow tract obstruction causing associated detrusor muscle hypertrophy. There appears to be a change in the predominant form of collagen from type I to type III. This type forms more cross-linkages and contributes to an increased stiffness, resulting in the decreased compliance of the elderly bladder. These findings, once thought to be due to the influence of prostatic outflow tract obstruction, have been described both in the presence and absence of such obstruction and are described in age-matched females.[5] Outflow tract obstruction is rare in women.[6]

A reduction in the number of acetylcholinesterase-containing nerves in association with increasing age has been seen using microscopy and confirmed using polymerase chain reaction techniques. There was a negative correlation of receptor number with age in detrusor muscle from men and a selective age-related decrease in mRNA for muscarinic M3 but not M2 receptors in both male and female bladders. These findings correspond with reports of decreased detrusor contractility with ageing.[5,7,8] In addition, there are data that show a reduction in the amount of acetylcholine released from the nerves of older bladders in response to muscle stimulation. Results from physiological and microdialysis experiments indicate that purinergic transmission increases with age, whereas cholinergic transmission decreases; this change appears to be implicated in the pathophysiology of detrusor overactivity (DO), a condition much more common in later life.[9–11]

Activation of the adrenergic, beta-three receptor in maintaining relaxation of the human bladder during filling has been further elucidated. The population of these receptors decreases in association with increasing age, perhaps accounting for the reduction in bladder compliance in addition to the observed changes in the constitution of the bladder matrix.[12]

The appearance of bladder wall trabeculation, initially thought to be associated with increasing age and the development of prostatic hypertrophy and outflow tract obstruction, is now almost invariably associated with DO.[13,14] An increase in muscle cell size in association with outflow tract obstruction is generally described.[15] Electron microscopy studies of bladder from the elderly have reported both a tight association between structural abnormality, detrusor contractile function, and urodynamic diagnosis.[16] These abnormalities are common to both elderly men and women and are reflected in the detrusor muscle, its interstitium, and in the surrounding nerves.[17,18] However, other studies using a similar method have reported a random occurrence of these same ultrastructural changes throughout the elderly population, regardless of underlying diagnosis.[19] It is difficult to reach a firm conclusion regarding the relevance of such described changes and there is clearly room for more work in this area. However, given that disease is likely to occur along a continuum, it is unlikely that such a clear distinction between ultrastructural abnormality and pathological diagnosis can be made.

5.3. Functional Studies

Much of the data regarding changes in lower urinary tract function are derived from studies of individuals with lower urinary tract symptoms who have undergone urodynamic studies. Data from community-dwelling continent individuals are sparse, but where they do exist, they tend to confirm the associations identified by other means. There is, as ever, a paucity of the definition of normal in later life and when compared to norms from younger individuals only 18% of normal community-dwelling elderly men and women have been reported as having normal lower urinary tract function.[20] There is an increase in the prevalence of people with urinary frequency in later life.[21] Significant features are a reduced bladder capacity, an increased incidence of DO, an increased incidence of outflow tract obstruction, and alterations in renal function and water and solute excretion. The study of Pfisterer et al., however, while agreeing with the findings of the majority of previous investigators, suggests that the commonly observed reduction in bladder capacity in later life may be due to the influence of patients with DO being included in many studies.[22] Factors unrelated to the function of the lower urinary tract include concomitant cardiovascular, neurological, and musculoskeletal disease and the effects of drug therapy and environmental change, all of which promote an increased frequency of micturition in an attempt to avoid incontinence.

Detrusor contractile function as measured in vivo by an index of maximal speed of muscle shortening (Figure 5.1), bladder capacity (Figure 5.2), and urinary flow rates (Figure 5.3) all appear to decline in association with greater age in both sexes, although the decline in contractile function is not so apparent in men.[23] Studies of contractile function of the bladder using urodynamic variables rely on measures using bladder pressures.[24,25] There is a dependence of the relationship between pressure and flow upon bladder volume and variation during voiding due to the interaction of flow with the properties of the outflow tract. There are data that suggest that Q*, a measure of isotonic contractile function,[26] is able to classify "fast" from "slow" contracting bladders and may accurately predict the occurrence of voiding difficulty following colposuspension for stress incontinence.[27] However, the relationship between urodynamic measures and contractility of the bladder has not been established.

There is also an increase in the prevalence of incomplete emptying as demonstrated by the existence of a significant postmicturition residual volume of urine[28] (Figure 5.4). In men, the progressive enlargement of the prostate with age tends to dominate the behaviour of the urinary

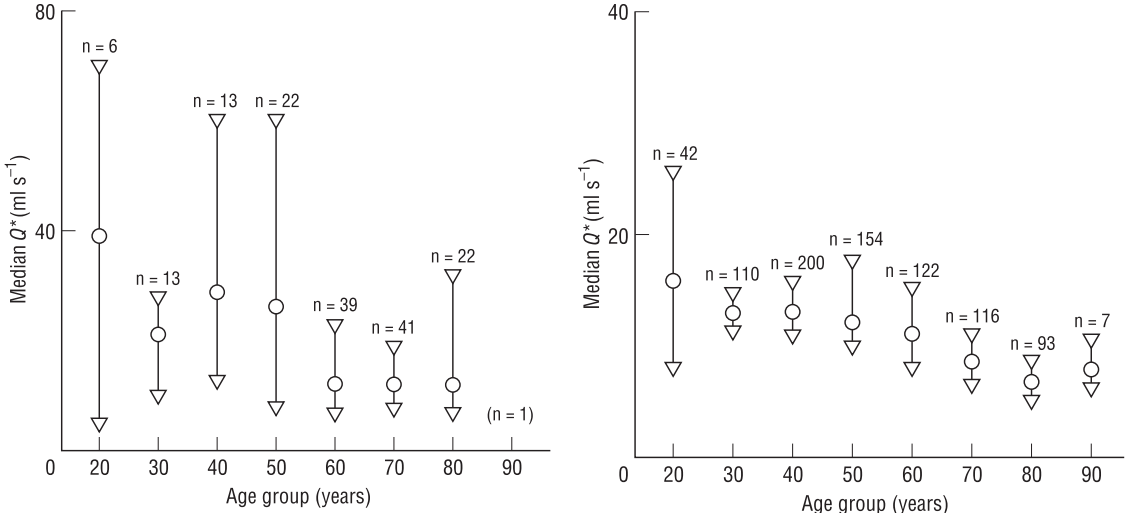

Figure 5.1. Contractile function as measured by Q* in association with greater age in (a) men and (b) women with lower urinary tract symptoms, n = 157.

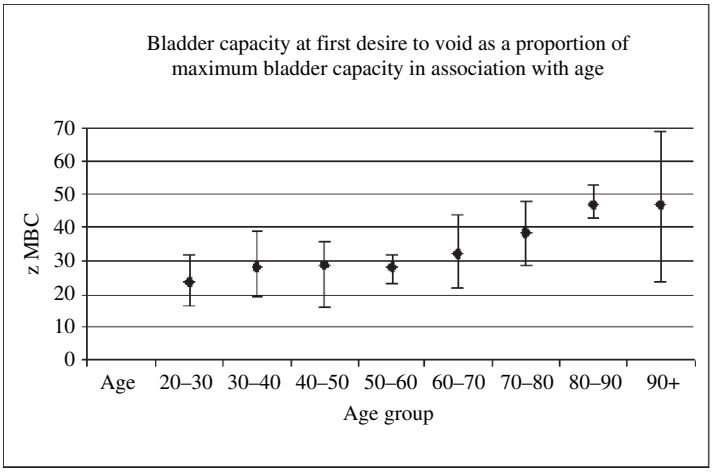

Figure 5.2. Increased age is associated with a reduction in bladder capacity.

outflow tract, which accounts for up to half of all men suffering from outflow tract obstruction. [20] As obstruction increases, the bladder requires a greater contractile effort to overcome the effects of the obstruction; this eventually leads to a chronically overdistended bladder, which fails to empty effectively. In others acute urinary retention may develop. In women, the prevalence of ineffective voiding is much lower, although in one community study the finding of a residual volume was commonplace, however the significance of this is questionable.[29,30] Detrusor overactivity is

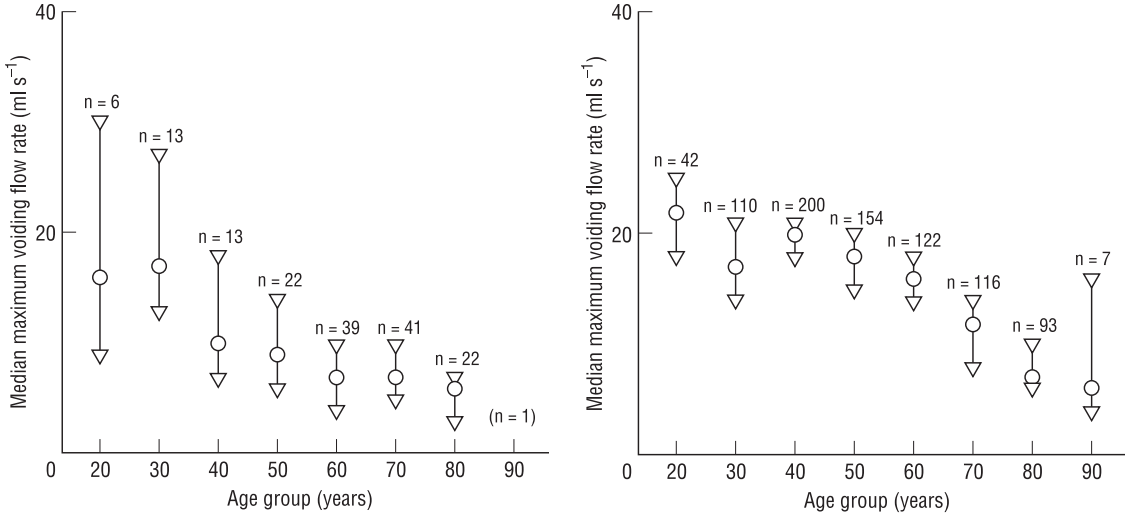

Figure 5.3. Median (95% CI) maximum flow rate for (a) men and (b) women with lower urinary tract symptoms in relation to greater age.

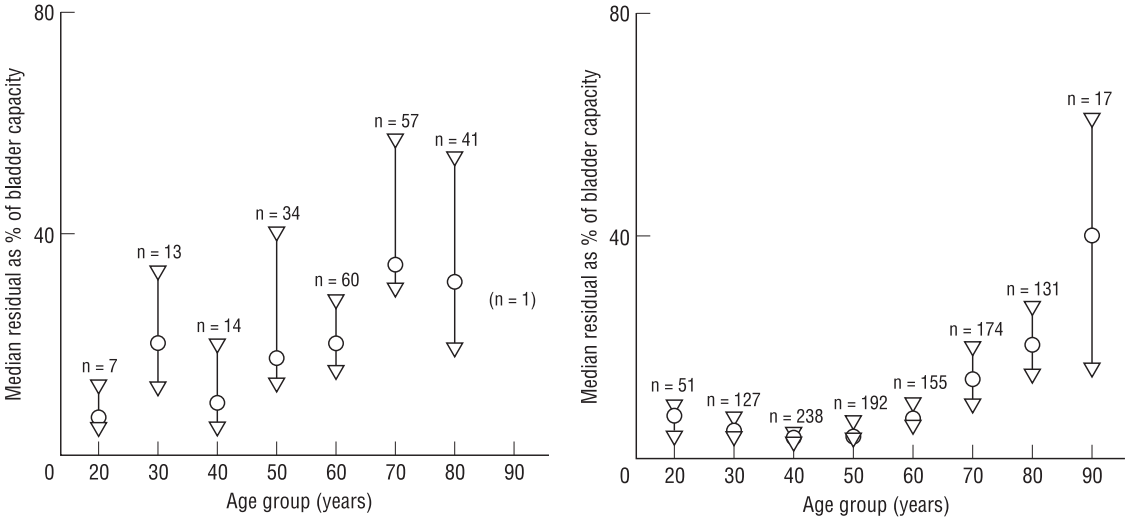

Figure 5.4. Postvoid residual volume of urine related to increasing age in (a) men and (b) women with lower urinary tract symptoms.

conventionally thought to be associated with the development of outflow tract obstruction and is present in 43–86% of patients.[31] This viewpoint has been reinforced by the fact that relief of the obstruction leads to bladder stability in a significant proportion of the population.[32] The incidence of overactive bladder (OAB) syndrome increases in association with age per se and is similar in females, except in the oldest-old when the symptoms predominate in men.[33]

In men with lower urinary tract symptoms, the likelihood of DO being the cause of these reaches 85% in the eighth decade, regardless of outflow tract obstruction. The true incidence of symptomatic DO is unknown due to the inherent difficulty of underreporting and the unreliability for cystometry to diagnose the condition, but it is estimated to be between 10 and 15% of men and women between 10 and 50 years, rising to 35% of those aged $>$ 75 years.[34]

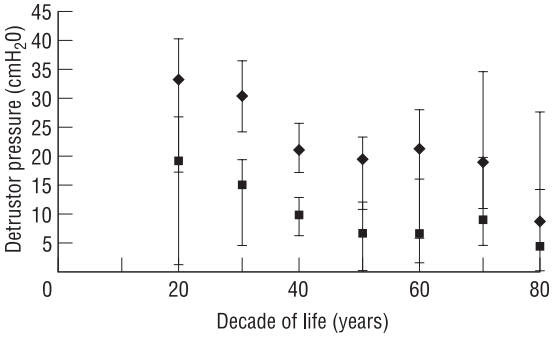

Figure 5.5. Detrusor pressures at urethral opening and closure for women with stable bladders in association with greater age. Black diamonds (♦), detrusor pressure at urethral opening; black squares (■), detrusor pressure at urethral closure.

There are also age-associated changes in the sensory function of the bladder. The bladder capacity at first desire to void, as a proportion of bladder capacity, rises significantly in association with increasing age in women.[35] This suggests a decreasing sensitivity of the bladder to filling, and coupled with the reduced maximal bladder capacity tends to reduce the time that an individual has available to successfully and appropriately void. There may be an influence of higher centres accounting for this observation[36] (Figure 5.5).

5.4. Urethral Function

Studies using the urethral pressure profile[37,38] or detrusor closure pressures[39] have both described a reduction in urethral function in later life. There is evidence of an increased collagen content in the urethra of elderly females.[40] There is also a loss of striated muscle cells in the aged female urethral sphincter and around the bladder neck.[41,42]

5.5. Nocturia

Nocturia is an increasingly prevalent and bothersome urinary symptom associated with considerable impact and morbidity in later life.[43–46] This is due to a combination of factors. In later life the majority of urine production occurs nocturnally.[47] In older individuals, renal concentrating ability falls[48,49] and glomerular filtration rate increases in the supine position. There is a redistribution of fluid at night, particularly if the individual has venous insufficiency or is on medication that predisposes to the development of peripheral oedema, such as calcium channel blockers or nonsteroidal anti-inflammatory agents. In addition, some older adults have a delayed diuresis in response to a fluid load and lose their diurnal rhythm of ADH secretion,[50] although there are studies in other groups showing no such loss[51]. When taken together, this means that the kidneys are working harder overnight to produce greater quantities of more dilute urine, the amount of which may be in excess of a smaller functional bladder capacity. The presence of nocturnal frequency of two or more has been reported as 13–29%, the latter figure from an older age group.[52,53] There is evidence for the efficacy of DDAVP[54] and early evening diuretic,[55] and limited evidence for daytime recumbence[56] in treating nocturia, but these are not well tolerated by all. In particular, the usefulness of DDAVP may be limited to those individuals with true nocturnal polyuria rather than urinary frequency,[57] and its use may be hampered by drug–drug interactions predisposing to hyponatraemia which occurs in approximately 7% of individuals.

Primary nocturnal enuresis may persist into adulthood. This affects approximately 0.6–1% of all adults; 50% of sufferers had never sought help for the problem.[58] The majority of men with persistent nocturnal enuresis, up to the age of 65 years, appear to have underlying DO.[59–61] Treatment with imipramine has shown a marked antidiuretic effect on patients with nocturnal polyuria.[62] A study comparing the use of DDAVP with the antimuscarinic medication oxybutynin showed no difference in improvement between the DDAVP group and combination treatment. The incidence of nocturnal incontinence also increases linearly in association with greater age. This incidence is increased in the presence of concomitant urinary storage disorders.[63] Once the relative contributions of associated conditions can be established, better planning of intervention studies may be conducted.

5.6. Conclusion

The changes in urinary tract structure and function identified in association with greater age enable the generation of hypotheses, which will

enable prospective, longitudinal studies to be undertaken. The interrelationship of urinary tract symptoms, coexistent diseases, and their treatment make the conclusions from studies conducted in elderly individuals more difficult to interpret and there is a continued need for studies utilising younger controls. The lack of normal data in the elderly makes the identification of abnormal function more problematic. However, it is becoming clearer that the changes in the bladder once thought to be associated with prostatic outflow tract obstruction are apparently just as common in women. The incidence of detrusor overactivity also follows a similar pattern, regardless of the presence or absence of outflow tract obstruction.

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6
Assessment and Treatment of Urinary Incontinence in Neurologically Impaired Patients

ML Kujawa and CD Betts

6.1. Introduction

This chapter is mainly concerned with the neurogenic bladder dysfunction in multiple sclerosis (MS), following a cerebrovascular accident (CVA), in idiopathic Parkinsons' disease (IPD), and in multiple system atrophy (MSA). Urinary retention in young women is not uncommon and the management is often difficult. There is now a much improved understanding of bladder retention problems in premenopausal women and a section on this has been included.

For many patients with neurologic disease the bladder symptoms can be the most distressing feature of the disorder. In a patient with neurological impairment, the combination of urinary urgency and frequency is likely to have a highly adverse effect upon their well-being. In MS, MSA, CVA, and IPD it is extremely uncommon for the neurogenic bladder disorder to result in serious upper urinary tract problems and kidney failure. However, patients with spinal cord injuries or spina bifida are at a significant risk of developing renal impairment, and because their management is particularly specialised, these conditions have not been included in this chapter.

6.2. Prevalence

The central neural pathways, which are important in bladder control, extend from the frontal lobes to the sacral spinal cord; lesions at many levels of the neuraxis may cause neurogenic bladder dysfunction. The coordinated action of the somatic and the autonomic nervous systems are necessary for normal bladder storage and emptying. Central nervous system disease involving the pathways for bladder function may result in abnormal storage or emptying, and especially when there is disease in the spinal cord both phases of micturition may be disordered.

6.2.1. Multiple Sclerosis

The high incidence of bladder symptoms in MS has been recognised for more than 100 years. Oppenheim[1] found urinary symptoms in 80% of patients with MS, and most investigators since have reported that approximately 75% of all patients with MS develop urinary symptoms. A few studies have reported a lower incidence of bladder problems in MS,[2] but this is almost certainly because the patients included in these studies had mild degrees of disability. Miller et al.[3] found bladder symptoms in 75% of patients with MS.

In the past, urinary symptoms alone were thought to be the first symptoms of MS in a small number of patients. Miller et al.[3] and Goldstein et al.[4] stated that bladder symptoms were the sole presenting feature of MS in 2 and 2.3% of patients, respectively. A later study of 170 patients with MS and urinary symptoms none of the patients had first presented with urinary symptoms alone.[5] Urinary retention in

women was often said to be due to MS and this could account for the reports that patients with MS had first presented with bladder problems. An alternative cause for retention in young women now is recognised and retention without other neurological features should not be regarded as a symptom of MS.[5–8]

In 1 to 14% of patients, urinary symptoms in conjunction with other neurological features may be part of the presenting symptom complex of MS.

6.2.2. Idiopathic Parkinson's Disease

Parkinsonism is a disturbance of motor function named after James Parkinson, who first described a case of paralysis agitans in 1817.[9] There are several causes of parkinsonism and the commonest is idiopathic Parkinson's disease (IPD) followed by multiple system atrophy (MSA), the Steele–Richardson–Olskowski syndrome, and certain drugs. The clinical features are muscular rigidity, tremor, and slowing of movement. The diagnosis of IPD is largely based on clinical features. Recent postmortem studies suggest that the other causes of Parkinsonism, especially MSA, have been underdiagnosed.[10,11]

In IPD the main site of pathological change is within the substantia nigra, although other parts of the basal ganglia may be abnormal. The pathological hallmark of IPD is spherical intracytoplasmic hyaline inclusion bodies, called Lewy bodies.

The neurodegeneration in IPD involves the neural mechanisms that are important in the central control of bladder function; frequency and urgency of micturition are common urinary symptoms.[12] Urge incontinence may occur especially if mobility is poor. In elderly men with IPD, it is often difficult to distinguish between the bladder symptoms due to the neurological disorder and the urinary problems resulting from benign prostatic hypertrophy (BPH).[13] In many it is likely that the urinary symptoms arise from a combination of BPH and the neurogenic bladder dysfunction.

The prevalence of urinary symptoms in patients diagnosed as having IPD has been reported to be between 37 and 71%.[12, 14–16] Figures for the incidence of urinary symptoms in IPD need to be interpreted with some caution, since there may be uncertainty about the neurological diagnosis and in men there is the possibility of prostate disease.

6.2.3. Cerebrovascular Accident

Bladder dysfunction is an important complication of a stroke; in addition to affecting morale it can adversely affect rehabilitation, discharge from hospital, and the long-term outcome from the stroke.[17]

Brocklehurst et al.[18] reported urinary continence problems in 52 of 135 consecutive stoke patients during the first 2 weeks after the CVA. The authors concluded that incontinence after a CVA was largely a result of immobility and usually was a transient problem.[18] In a prospective study of 151 patients following a stroke, 60%, 42%, and 29% of those surviving the CVA had problems with urinary incontinence at 1 week, 4 weeks, and 12 weeks, respectively. At 4 weeks, the factors that were significantly associated with urinary incontinence were moderate or severe motor deficit, impaired mobility, and mental impairment; 66% of patients who were said to have mild incontinence at 4 weeks were continent by 12 weeks. In several studies of urinary incontinence after stroke, the investigators concluded that bladder incontinence occurring shortly after a CVA is a specific indicator of a poor prognosis.[19–22] The long-term prevalence of bladder dysfunction in patients who have had a CVA is uncertain since the population at risk from strokes also are likely to have symptoms from prostatic disease, bladder stones, bladder cancer, and idiopathic detrusor overactivity (IDO).

6.2.4. Aetiology and Pathophysiology of the Neural Control of Bladder Function

The neural pathways, which are important in bladder function, traverse the entire length of the spinal cord between the pons and the sacral spinal cord and are particularly likely to be affected by diseases involving the spinal cord. Knowledge of the areas in the central nervous system that are important in bladder control may be helpful in predicting the bladder dysfunction in central neurologic disease. In humans, the exact location of the central neural pathways for bladder control remains uncertain.

6.2.5. The External Urethral Sphincter and the Nucleus of Onuf

The external urethral sphincter—the rhabdosphincter—is made up of circularly orientated, predominately type 1 (slow twitch) striated muscle fibers.[23] In men the external sphincter is just distal to the prostate gland and in women it surrounds much of the shorter urethra. The striated muscle of the urethral sphincter is the only part of the urinary tract to receive a somatic innervation. Onufrowicz in 1899[24] described a nucleus in the ventral horn of the spinal cord at S2, S3, and S4. Onuf's nucleus contains the cell bodies of the motor neurons that innervate the urethral and anal sphincters. Motor fibres from Onufs' nucleus pass into the pudendal nerves giving branches to the anal and urethral sphincters.

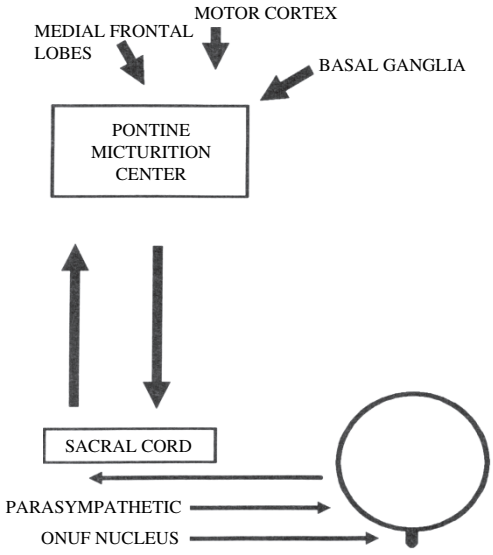

Figure 6.1. Schematic representation of the neural control of the bladder.

6.2.6. Detrusor Muscle Innervation

The main motor supply of the detrusor muscle of the bladder comes from parasympathetic preganglionic neurons with cell bodies in the intermediolateral columns of the cord between S2 and S4. The preganglionic parasympathetic nerve fibers pass by way of the pelvic nerves to the pelvic plexus and then by short postganglionic nerves to innervate the detrusor muscle. The role of the sympathetic nervous system in human micturition remains unclear. The bladder neck in males has a rich noradrenergic innervation; sympathetic activity during ejaculation causes closure of the bladder neck and antegrade ejaculation.

6.2.7. The Pons and the Frontal Regions in Bladder Function

The importance of the pons for the control of micturition was first recognised by Barrington, working on decerebrate cats at University College London.[25] A nucleus in the dorsal tegmentum of the pons has been identified as a centre important in coordinating micturition. This nucleus in the pons has been referred to as **Barrington's nucleus**[26] and it is also known as the pontine micturition centre (PMC). In 1964, Andrew and Nathan[27] first drew attention to the importance of the frontal regions of the brain in bladder control.[28] Furthermore, de Groat has proposed that higher centres control the PMC, which acts as "a switch" on a long reflex, alternating the bladder's storage and voiding phases.[29,30]

In recent years there have been some interesting positron emission tomography (PET) studies in humans, which have shown that the neural control in humans is similar to that described in cats and other animals.[31,32] For a detailed description of these studies the reader is referred to an editorial.[33] The evidence supports a "bimodal control" of the bladder; suprapontine centres, and particularly areas in the frontal lobes which are involved in the decision as to when to "switch" the PMC between the storage and voiding states.

Higher centres, particularly the medial frontal lobes, act on the PMC in a mainly inhibitory manner. When the bladder is full and voiding is socially acceptable, higher centres, particularly the medial frontal lobes, facilitate the micturition reflex through the PMC. Pelvic nerve activity increases, resulting in contraction of the detrusor muscle. At the same time the nervous innervation of the external urethral sphincter muscle is inhibited and this relaxes to allow voiding. In normal circumstances the detrusor muscle contracts only when a person wishes to empty the bladder. The external urethral sphincter

normally relaxes at the same time as bladder contraction and the PMC is known to play a key role in this coordinated action.

6.2.8. Bladder Dysfunction in Multiple Sclerosis

6.2.8.1. Urinary Symptoms in Multiple Sclerosis

The most common urinary symptom in MS is urgency of micturition, followed by frequency and urge incontinence. In the literature concerning MS and the bladder, the term "retention" has been used to describe a complete inability to void and also hesitancy; this means that the true incidence of complete retention is unclear. A complete inability to void from a failure of detrusor muscle contraction is thought to be unusual.

6.2.8.2. Urodynamic Studies in Multiple Sclerosis

There have been a large number of papers concerned with urodynamic studies in MS and the data can be confusing. Bladder overactivity [neurogenic detrusor overactivity (NDO)] has been the most common urodynamic finding in patients with MS and urinary symptoms.[34] In this disorder the bladder contracts in an abnormal involuntary manner often when the urinary volume is low.

At the onset of one of these bladder contractions the patient usually will experience the sensation that they are about to empty their bladder. The person may be able to avoid an episode of incontinence by tightly contracting their pelvic floor muscles (PFM) until they reach a toilet or sometimes the involuntary bladder contraction will abate and the sensation will fade away. Often the pressure within the bladder during one of these involuntary detrusor contractions can be so great that urine escapes from the bladder and incontinence occurs (urge incontinence).

In our study[5] the urodynamic finding of detrusor overactivity (DO) correlated well with the symptoms of urgency, frequency, and urge incontinence. All of the patients who had moderate or severe pyramidal dysfunction in their legs (Kurtzke pyramidal scores 3 or higher) and complained of frequency, urgency, or urge incontinence were found to have DO on urodynamic testing.

In MS the spinal cord lesions interrupt the neural pathways from the pons to the sacral cord,

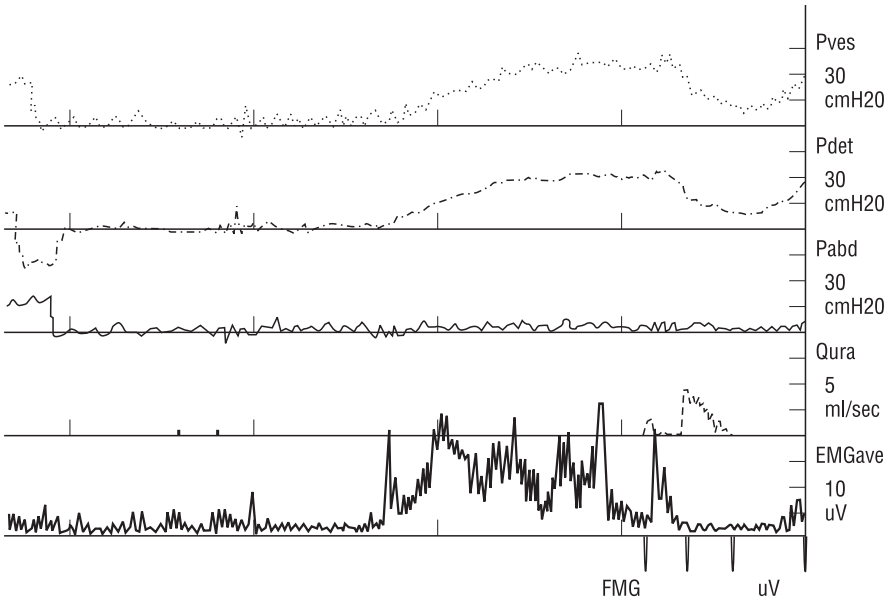

Figure 6.2. Urodynamic study during voiding in a patient with MS and complaining of hesitancy and an interrupted flow. The trace shows DSD. Sphincter activity is recorded by an EMG needle in the external sphincter. Sphincter activity increases when the detrusor contracts and voiding (Qura) takes place only when the sphincter activity reduces.

causing DO. The spinal lesions also may result in a loss of the coordinated action of the detrusor muscle and the external urethral sphincter, and this is known as detrusor sphincter dyssynergia (DSD). Bladder overactivity and DSD often occur in the same patient.[35–37]

Detrusor sphincter dyssynergia occurs when the external sphincter contacts in an involuntary manner at the same time as contraction of the detrusor muscle. In a patient with spinal cord disease, the symptoms suggestive of DSD include hesitancy of micturition, an interrupted urinary flow, and a failure to completely empty the bladder. The demonstration of DSD is difficult because it involves the simultaneous measurement of bladder pressure, urinary flow, and urethral sphincter electromyography. Detrusor sphincter dyssynergia is likely to be present in a patient with spinal cord disease who has a significant postmicturition residual.

The occurrence of bladder areflexia[34] in MS remains a controversial issue. It has been suggested that early studies of the bladder function in MS may have included patients who did not actually have MS.[5,38] There is little doubt that bladder overactivity with or without DSD is the most common urodynamic finding in MS patients with urinary symptoms.

6.2.9. Genitourinary Dysfunction and the Neurological Features of Multiple Sclerosis

Several authors have reported a correlation between bladder symptoms and the presence of pyramidal tract signs in the patients' lower limbs.[3,5,39] Also, the severity of the urinary symptoms has been shown to be related to the degree of pyramidal tract dysfunction in the lower limbs.[3,5,39] In our study the majority of patients with MS and moderately severe paraparesis experienced urge incontinence, at least to some extent[5] (see Figure 6.3). In general, the more severe the neurological dysfunction in the lower limbs, the more marked are the bladder symptoms.[5]

In men with MS it is known that there is a strong association between urinary symptoms, male erectile dysfunction (MED), and neurological impairment in the lower limbs.[3,40–42] In women with MS, little is known about the relationships between abnormalities of the bladder and sexual function.[42]

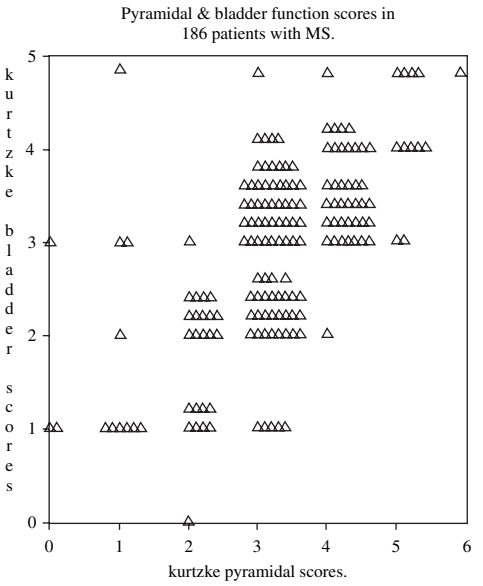

Figure 6.3. The Kurtzke bladder and pyramidal scores for 186 patients with multiple sclerosis.

6.3. Bladder Dysfunction in Idiopathic Parkinson's Disease

6.3.1. Urinary Symptoms in Idiopathic Parkinson's Disease

Frequency and urgency of micturition are common in patients with idiopathic Parkinson's disease (IPD).[12] The problem of distinguishing neurogenic bladder symptoms from prostatic symptoms in elderly men with IPD has been discussed earlier.

6.3.2. Urodynamic Studies in Idiopathic Parkinson's Disease

Urodynamic studies in patients with IPD and urinary symptoms have shown a high incidence of DO.[43,44] In neurologically intact humans, the basal ganglia are thought to exert an inhibitory influence on the PMC and the micturition reflex. In IPD it is often proposed that with loss of cells in the substantia nigra the inhibitory effect of the basal ganglia on the micturition reflex is reduced and DO is the result.[45,46]

Investigators have undertaken urodynamic tests in patients with IPD and studied the effect

of administering L-dopa or apomorphine.[47–50] The overall results have been inconclusive; in some patients with IPD there has been a lessening of the overactivity after L-dopa and in others a worsening of the bladder disorder.

In addition to DO some authors have suggested that there may be a slowness of external sphincter relaxation or bradykinesia in IPD and this may result in some obstruction to the urinary flow from the bladder.[50,54,56] This impairment of sphincter relaxation may be reversible by a subcutaneous injection of apomorphine,[54] and it may be possible to distinguish between bladder outflow obstruction due to the prostate gland and that resulting from abnormal sphincter relaxation in Parkinsons' disease.[54]

6.4. Multiple System Atrophy

Graham and Oppenheimer[51] introduced the term **multiple system atrophy** (MSA) to cover several related conditions. The condition is probably more common than previously believed and in recent postmortem studies between 10–15% of patients diagnosed in life as having idiopathic Parkinson's disease actually had MSA on pathological examination of the nervous system. It is beyond the scope of this chapter to give a detailed account of this neurological disease and the reader is referred to Quinn,[10,11] Wenning,[52] and Chandiramani and Fowler.[53]

Clinically there are various subtypes of MSA with varying degrees of extrapyramidal, pyramidal, cerebellar, and autonomic dysfunction. It is possible to consider MSA in two main forms: an atypical form of Parkinson's disease which is often poorly responsive to the usual dopaminergic medication and a brain stem and a cerebellar form with loss of coordination, gait ataxia, and dysarthria. Postural hypotension is an important feature of MSA and well described in the neurological texts.

In MSA pathological studies show cell loss and gliosis in several "at risk" central nervous system structures.[54] Many of these sites are important in the nervous control of the bladder; therefore, disturbances of micturition and sexual function are common in MSA.[55]

In MSA, there is often cell loss in Onuf's nucleus resulting in denervation of the striated muscle of the urethral and anal sphincters.[56,57] Also, there is degeneration of cells in the midbrain and this may result in DO. As the disease progresses many patients develop a failure of the detrusor muscle, and this probably results from cell loss in areas of the sacral cord containing the parasympathetic neurons to the bladder. In MSA the degree of urinary incontinence often becomes very marked and this probably is due to the combination of a denervated urethral sphincter and abnormal bladder function, either overactivity or an underactive detrusor muscle.

6.4.1. Urinary Symptoms in Multiple System Atrophy

Studies have shown that about 60% of patients with MSA develop urinary symptoms before or at the same time as they present with parkinsonism.[55,58] Many patients with MSA are referred to a urologist with urinary symptoms or erectile dysfunction at an early stage in the disease, very often before the neurological disorder is fully apparent.[55] Patients presenting to urologists in this way are frequently thought to have prostatic disease or women to have uncomplicated stress urinary incontinence. Unfortunately, urinary control in patients with MSA often is made worse by surgical intervention. In one study, all of the men were incontinent either immediately after prostatic surgery or within one year of the operation.[55]

In the early stages of the disease, urgency, frequency, and nocturia are the most common urinary symptoms and in women stress incontinence may be a feature. The majority of men develop erectile failure. As the neurological disorder progresses, urinary incontinence and nocturia become very prominent and disturbing symptoms.

6.4.2. Urodynamic Studies in Multiple System Atrophy

Detrusor overactivity is common in the earlier stages of the disease, but as the neurological disorder advances, detrusor failure develops with incomplete bladder emptying.[55,59] Many patients develop large postmicturition residual volumes. In one study of 57 patients with suspected MSA, the average residual was 275 ml[55] and only 10 patients

had residual volumes less than 100 ml. It probably is this tendency to high residual volumes that results in a significant number of men undergoing prostatic surgery, before the neurological diagnosis is realised. As described earlier, the results of surgery are invariably poor.

6.4.3. Sphincter Electromyography in Multiple System Atrophy

In MSA, the degeneration in Onuf's nucleus results in denervation of the striated muscle in the anal and urethral sphincters. The changes of chronic reinnervation have been well demonstrated by electromyography (EMG) in the sphincter muscles of patients with probable MSA.[55–57,60] In Parkinson's disease, Onuf's nucleus is not affected and there is no denervation in the sphincter muscles. Sphincter EMG can be helpful in distinguishing between MSA and Parkinsons's disease.[61]

In sphincter electromyography, the most important measurement for detecting chronic reinnervation is the duration of individual motor units. The mean motor unit duration for the anal and urethral sphincters is normally less than 10 ms.[61] In a study, 82% of patients who were eventually diagnosed as having MSA had an abnormal sphincter EMG.[60] EMG may test the anal or urethral sphincters and the duration of at least ten individual motor units are measured.

Urinary and male sexual dysfunction criteria for suspecting a diagnosis of MSA have been proposed.[60] Impotence is frequently the first symptom of MSA and may occur several years before the onset of any other neurological symptom.

The diagnosis of MSA may not have been considered by the doctor managing a patient with parkinsonism and the clinician investigating the genitourinary symptoms may need to question the neurological diagnosis.

6.5. Cerebrovascular Accident

This section is concerned with the bladder function in patients who have had a cerebrovascular accident (CVA). Bladder problems occurring after brain stem strokes or vascular accidents involving the spinal cord have not been included. In the literature, there is information about urodynamic tests in patients following a stroke and other investigators have concentrated on the prevalence of urinary incontinence after a CVA.

6.5.1. Urodynamic Studies in Cerebrovascular Accident

Khan et al. undertook two studies of bladder dysfunction after stroke and correlated the computer tomography (CT) findings with the urodynamic results.[62,63] In the first study 20 patients following CVA were studied of which 19 had DO. The second study demonstrated again that DO was the most common urodynamic abnormality present in 26 of 33 patients who had urinary symptoms after a stroke.[63] The authors concluded that it was not possible to correlate the area of brain injury with bladder dysfunction. The findings were similar in another study: urge incontinence and DO being the most common problem.[64] In both studies it was noted that a few patients have urinary retention after a stroke.

In a further investigation,[65] 53% of patients had significant urinary symptoms at 3 months after a stroke: 36% had nocturnal frequency, 29% had urge incontinence, and 25% difficulty in voiding. In 6% urinary retention occurred in the acute phase after the stroke. CT and MRI studies were undertaken and there was a positive correlation between urinary symptoms and hemiparesis.[65] Those patients with an anterior brain injury site tended to have urinary symptoms, whereas those without disturbance of micturition had more posterior lesions. The findings from the studies of stroke patients with urinary symptoms indicate that lesions in the anteromedial frontal lobe and its descending pathway are most important in the aetiology of bladder dysfunction after stroke. Frequency, urgency, and urge incontinence are the most common urinary symptoms after a stroke and bladder overactivity is the most frequent urodynamic finding.

A stroke may result in urinary symptoms and incontinence by causing a true neurogenic dysfunction of the bladder, immobility and/or impaired cognitive function (see Chapter 22).

6.6. Urinary Retention in Premenopausal Women

Retention of urine in young women is not an uncommon clinical problem for the urologist; neurological disease is part of the differential diagnosis. In the past, the problem was often said to be due to demyelination in the sacral cord, but since the introduction of magnetic resonance scanning it is relatively easy to exclude MS as a diagnosis. In the absence of finding an obvious urological or neurological cause for the inability to void, some clinicians have regarded the problem as "hysterical" or psychogenic.[66–69]

Fowler and Kirby[7] described an abnormality of the striated muscle of the urethral sphincter in young women with retention and this disorder is thought to impair relaxation of the sphincter. The abnormality described by Fowler and Kirby has a very characteristic EMG signal[7] and consists of complex repetitive discharges (CRDs) and decelerating bursts (DBs).[61,70,71] The work undertaken by Fowler and Kirby has been supported by reports from other centres.[72,73] Approximately, one half of the patients with the abnormal EMG finding have polycystic ovaries and it has been suggested that there may be an underlying hormonal basis for the abnormal EMG activity and voiding dysfunction.[70] Based on detailed analysis of the abnormal EMG activity it has been proposed that there is abnormal direct muscle-to-muscle transmission of impulses within the sphincter, which results in an impairment of sphincter relaxation.[79] The urethral sphincter EMG abnormality also has been reported in women presenting with urgency and frequency with detrusor overactivity, low urinary flow rates, and sometimes incomplete bladder emptying.[76] Further work is required to understand the relationship between the sphincter activity described by Fowler and Kirby and urinary symptoms in premenopausal women.

6.7. Evaluation of Urinary Symptoms in Patients with Neurological Disease

In patients with neurological disease and urinary symptoms it is necessary to establish whether there is neurogenic bladder dysfunction or urological pathology in addition to the nervous system disorder. In some patients there may be urological disease and neurogenic bladder dysfunction. The likelihood of coexisting conditions at least in part will be determined by the age and sex of the patient. In a young woman with MS and urge incontinence it is extremely likely the symptom will be due to neurogenic detrusor overactivity (NDO), whereas in an elderly man with IPD, a combination of benign prostatic hypertrophy and DO is quite probable.[13]

6.7.1. History and Examination

There are many factors to take into account before deciding the most appropriate management of the bladder dysfunction in a patient with a neurological disease. It is necessary to learn about the patient's disabilities, their home circumstances, work, and daily routine. With the patients' consent their carers and family members should be involved in the evaluation process.

It is important to obtain accurate information about the patient's urinary symptoms (urgency, frequency, nocturia, urge incontinence, stress leakage, enuresis, hesitancy, strength or weakness of the urinary flow, interruption of the flow, sensation of incomplete bladder emptying), the severity of the complaints, and the effect on their lives. The completion of a chart recording the urinary frequency and the number of incontinent episodes is most valuable. Haematuria may indicate serious disease and the patient should be investigated to exclude urological malignancy. In patients with spinal cord disease, bladder and sexual dysfunction often coexist; when asking about urinary symptoms it may be an appropriate time to enquire about problems with sexual function.[74,75]

General examination should include an assessment of hand function, vision, and neurological impairment in the lower limbs. If there are fixed deformities or marked adductor spasm, then intermittent catheterisation may be difficult. The lower limbs should be examined for pyramidal tract signs particularly in patients with MS where there is a strong correlation between pyramidal dysfunction and NDO.[5,42]

The bladder may be palpable on abdominal examination due to incomplete emptying and

inspection of the external genitalia may be helpful in assessing the degree of soiling, in detecting skin problems, and in determining if there is a rectal or uterine prolapse. The position and condition of the external urethral meatus is important if intermittent catheterisation is being considered.

6.7.2. Bladder Emptying

Measurement of the residual volume in the bladder after micturition is the single most important test in a patient with a neurological disease and urinary symptoms.[76,77] An ultrasound scan of the bladder immediately after voiding, or passing a catheter are the usual means of measuring the residual urine (see Figure 6.4). A postmicturition volume of 100 ml or more is generally considered significant.

6.7.3. Urinary Flow

This is one of the simplest urodynamic studies and can be undertaken in combination with the measurement of the postmicturition residual volume. In some very disabled patients it may be impossible to record the urinary flow. However, measurement of the residual volume is nearly always possible.

A very prolonged tracing with a low maximum flow rate suggests obstruction and in men this may be due to prostatic disease or a urethral stricture (see Figures 6.5 and 6.6).

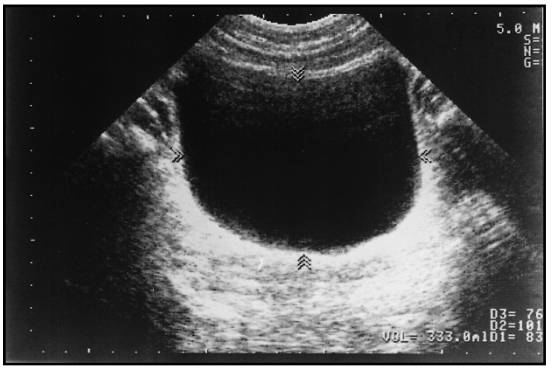

Figure 6.4. Postmicturition ultrasound scan of the bladder in a woman with MS. The residual was calculated to be 333 ml.

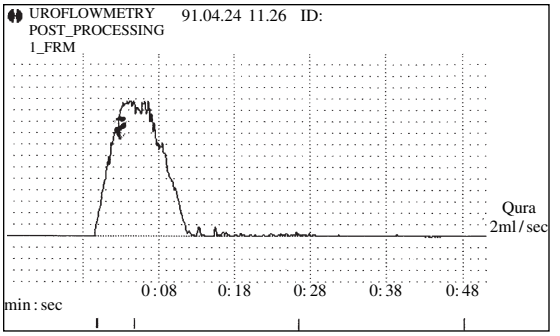

Figure 6.5. Normal urine flow (Uroflowmetry). The maximum flow rate was 22 ml.

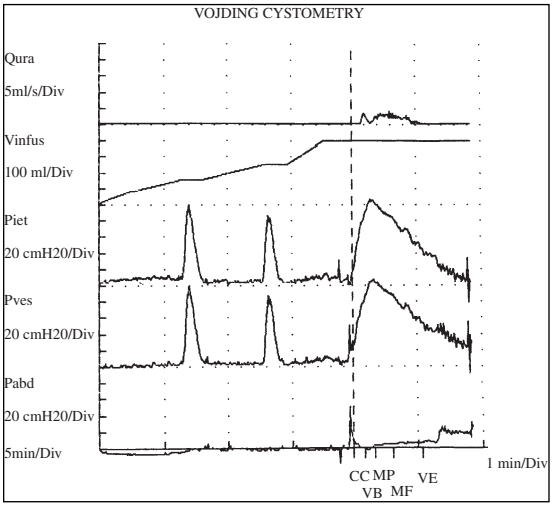

Figure 6.6. A filling and voiding urodynamic study in a man with IPD. During filling there was marked DO as shown by the rises in Pdet and Pves. At the dotted line the patient was asked to void; there was a strong detrusor contraction, resulting in an intravesical pressure 100 cm H_2O but the urinary flow rate was poor (Qura); hence, indicating some form of bladder outflow obstruction. The man gained considerable improvement in his symptoms following resection of the prostate gland and anticholinergic medication to suppress the DO.

An interrupted flow pattern in a patient with spinal cord disease is likely to be due to detrusor sphincter dyssynergia.

6.7.4. Cystometry

A very detailed description of bladder cystometry is beyond the scope of this chapter and the reader is referred to Chapter 9a.

There has been considerable debate about the indications for cystometry in patients with central nervous system disease and urinary symptoms.[5,42,78] Some authors recommend cystometry in every patient with urinary symptoms while others suggest that treatment may be commenced on the basis of symptoms and measurement of the residual volume.[79] In MS, DO will invariably be present in patients with irritative urinary symptoms (frequency, urgency, urge incontinence) and pyramidal tract signs in their legs.[5] For patients with MS and bladder dysfunction, treatment can often be instituted on the basis of the symptoms and the residual volume measurement. Detailed urodynamic studies are indicated in those patients who respond poorly to initial therapy.

In IPD and cerebrovascular disease, DO is the most common finding during filling CMG. Patients with IPD or cerebrovascular disease are often at an age when urological disease is common and this should be considered as a possible cause for the symptoms. Voiding CMG may help establish if there is bladder outflow obstruction due to prostatic enlargement in men with neurological disease and urinary symptoms (see Figure 6.7).

6.7.5. Sphincter EMG

In the literature, the term "sphincter EMG" is often applied to very different tests. A concentric needle electrode (CNE) can be positioned in the external sphincter and analysis of individual motor units undertaken.[61,80] This form of detailed EMG study enables changes within the muscle due to denervation and reinnervation to be investigated.[61] EMG is also used to detect abnormal spontaneous activity in the sphincter muscle, which may be observed in some women with urinary retention. Fowler concluded that "*EMG (detailed) is the most important investigation in diagnostic uroneurology.*" [61]

Sphincter EMG can also be performed to examine the pattern of activity of the sphincter muscle. This type of EMG test is performed in conjunction with urodynamic studies to examine the activity of the urethral sphincter in relation to detrusor function (see Figure 6.2).

6.7.6. "Urological" Investigations

Neurogenic bladder dysfunction may result in urinary tract complications including infection, stones, dilatation of the upper urinary tracts, and renal impairment. See Chapter 9a for urological investigations.

6.8. Treatment of the Bladder Dysfunction in Neurological Disease

This section deals with the conservative treatment of the bladder dysfunction in neurological disease. Treatment must be modified according

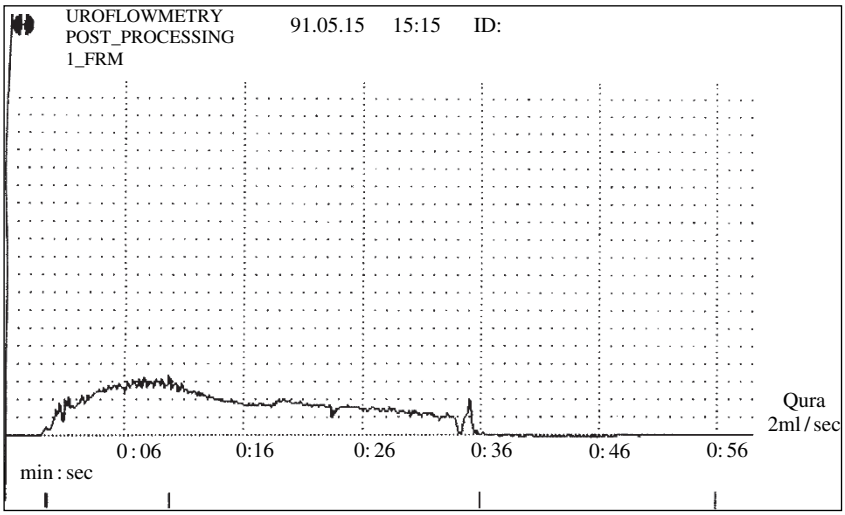

Figure 6.7. An abnormal urine flow test as a result of prostatic hypertrophy. The maximum flow rate was 6 ml.

to the patient's disabilities and wishes, cognitive function, and nature of the underlying neurological condition. Conditions such as MS and MSA are very likely to progress and this must be taken into account when deciding upon treatment. In patients with neurological disease, the inconvenience and occasional complications of treatment should be measured against the possible benefits.

For many patients with neurological disease and urinary symptoms conservative treatment is directed toward controlling the DO with anticholinergic medication and instituting intermittent catheterisation if there is incomplete emptying. Renal failure due to neurogenic bladder dysfunction is unusual in patients with neurological disease but it is an important problem after spinal cord injury and in those with congenital spinal cord abnormalities.

6.8.1. Intermittent Catheterisation

Intermittent catheterisation (IC) has transformed the management of patients with neurogenic bladder disorders; for further information, please see Chapter 23. In general only patients with residual volumes of more than 100 ml are likely to benefit from IC. If DO is present, then the full benefit from IC will be obtained only if the overactivity is managed by anticholinergic medication. Intermitten catheterisation is undertaken between one and six times a day and it is often helpful if a patient talks to another patient who has benefited from this management technique.

Symptomatic urinary tract infection is a surprisingly infrequent complication of clean intermittent catheterisation (CIC), although many patients undertaking the technique have asymptomatic bacteruria. Patients with significant postmicturition residual volumes and recurrent infections often experience a reduction in symptomatic infections after commencing CIC. Patients undertaking CIC who have recurrent infections may benefit from single-use catheters, increasing their fluid intake, and low-dose antibiotic prophylaxis.

6.8.2. Therapy for Detrusor Overactivity

6.8.2.1. Anticholinergic Agents

Drug therapy is the main treatment for DO (see Chapter 25). The residual volume should be measured before commencing therapy and if it is more than 100 ml, then IC should be instituted. If the residual is less than 100 ml, then the patient should be prepared for the possibility of needing IC; this will depend upon the effect of the anticholinergic on bladder emptying (Figure 6.8).

6.8.2.2. Capsaicin

Capsaicin is the pungent ingredient in red-hot chilli peppers and the first use of intravesical capsaicin in human bladders was reported in 1989.[81] Capsaicin blocks the C-fibre-mediated long-latency micturition reflex in cats.[82] In humans, intravesical capsaicin can increase bladder capacity and decrease overactive contractions; the beneficial effect may last for up to 3 months.[83–85] Further research in this field is required, but intravesical therapy with capsaicin or more potent capsinoidlike substances could have a major impact upon the management of DO.

6.8.2.3. Desmopressin

In neurogenic bladder dysfunction, night-time urinary frequency may be a problem and some patients may benefit from desmopressin, a synthetic antidiuretic hormone (DDAVP).[86,87] Desmopressin is available as a nasal spray or tablets and when taken before going to bed reduces urine output for 6–8 hr. It increases the reabsorption of water in the collecting tubule of the kidney, and because of the risk of hyponatraemia it should be used with caution in patients over the age of 65 years. It is reasonable for some patients to use desmopressin on a very occasional basis in the daytime for an important event such as attending a wedding.[88]

6.8.2.4. Botulinum Neurotoxin A

Until recently, there have been limited options available for patients with DO who fail to respond to anticholinergic medication. Before botulinum neurotoxin A (BoNT/A) the choice for patients who did not benefit from anticholinergics with or without CIC was either to accept their urinary incontinence or undergo major surgery usually in the form of an augmentation cystoplasy or urinary diversion (see Chapter 24). Intradetrusor BoNT/A has provided an exciting

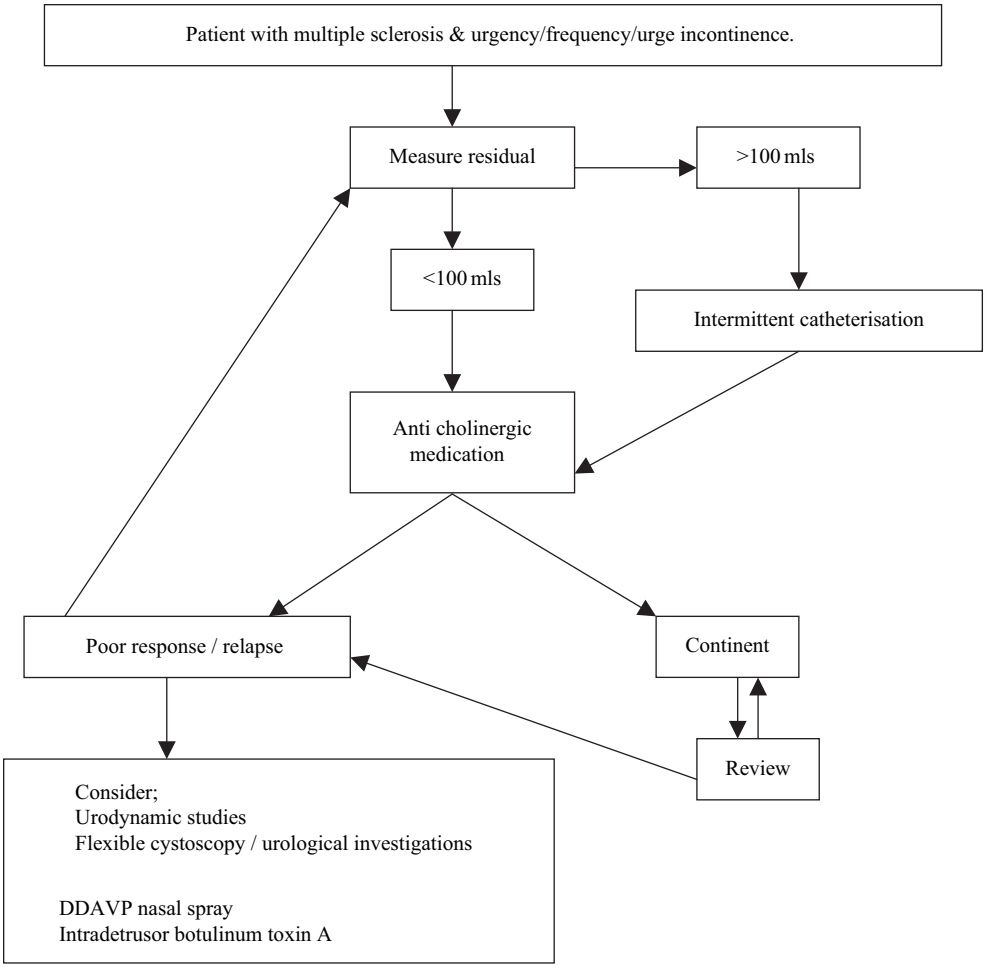

Figure 6.8. Algorithm for the management of neurogenic urinary incontinence in MS.

new option for patients with intractable DO. The action of BoNT/A is to bind and enter the peripheral cholinergic terminals, resulting in a prolonged blockade of acetylcholine release, causing a flaccid paralysis (see Chapters 24 and 25).

6.8.2.5. Sacral Neuromodulation

Caldwell used electrical stimulation in the management of voiding dysfunction as early as 1963.[89–91] In 1982, the first sacral neuromodulation (SNM) procedure was performed at the University of California.[92] In 1990, Interstim® Therapy (Medtronic) was approved by the US Food and Drug Administration for refractory urge incontinence and urinary retention (see Figure 6.9). SNM has been used mainly in patients with idiopathic bladder overactivity and urinary retention. There is little evidence for its use in patients with established neurological disease; further information regarding the technique can be found in Chapter 24.

SNM is mainly indicated for those difficult patients with intractable urge incontinence and women with urinary retention. A great advantage of SNM is that it is relatively noninvasive and does not involve intra-abdominal surgery with its potentially serious long-term effects. The results are encouraging in this difficult group of patients.[93,94]

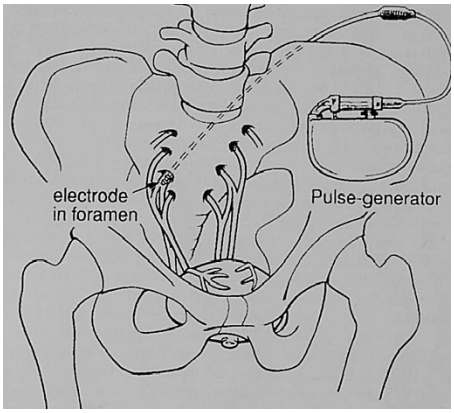

Figure 6.9. Interstim® therapy (see text).

6.8.2.6. Chronic Indwelling Catheter

In some circumstances an indwelling catheter may be the best option to ensure personal hygiene and adequate bladder drainage. Despite the disadvantages of a long-term indwelling catheter there are times when the insertion of a catheter can provide the patient with a welcome relief from incontinence (see Chapter 23).

Bypassing around a urethral catheter is a common problem, resulting usually from uninhibited detrusor contractions; it may be resolved by anticholinergic medication or intradetrusor BoNT/A. Treatment of symptomatic urinary infections is important but prolonged antibiotic therapy is likely to lead to the emergence of resistant organisms.

Suprapubic catheterisation has several benefits over long-term urethral catheterisation including avoidance of urethral destruction, ease of changing, improved catheter hygiene, and facilitation of sexual activity.[95,96]

6.8.2.7. Appliances

The most commonly used appliances for containing urinary leakage are penile sheaths and absorbent pads. In women, there is no widely used external appliance for the containment of urinary leakage. A variety of penile sheaths are available but it may still prove impossible to fit a sheath satisfactorily often because the penis is retracted. Also, some patients develop skin reactions to the material of the sheath and others with impaired mental function may pull at the appliance.

6.9. Summary

In neurological disease, the urinary symptoms can be the most distressing aspect of the disorder for the patient, the carers, and the family. Explanation of the cause of the bladder dysfunction in relation to the neurological disease process is likely to be of great benefit to the patient and those concerned with providing care. The main forms of conservative treatment are pharmacotherapy for control of detrusor overactivity and intermittent catheterisation to ensure satisfactory bladder emptying.

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7
Patient Assessment

J Laycock, MM Whelan and C Dumoulin

7.1 Introduction

Bladder and bowel symptoms rarely occur in isolation and many are due to adaptive strategies. To fully understand the pathophysiology of lower urinary tract (LUT) disorders, information is required about problems during bladder filling, storage and emptying, and the character, onset and duration of symptoms. All past and present obstetric, gynaecological, urological, medical and relevant surgical history should be documented, along with current drug therapy, including over-the-counter (OTC) medication. Finally, a physical examination is carried out, examining the abdomen, external genitalia and pelvic floor muscles (PFM). This information is best collected on a pro-forma (see Appendix 1).

7.2 Bladder Diary/ Frequency Volume Charts

The normal adult fluid output from the kidneys varies between 1 and 3 litres per 24 hours, with approximately 80% excreted during waking hours, negating the need to empty the bladder at night; the average adult bladder capacity is in the range 300 to 600 mL[1].

Probably the most useful assessment tool is the bladder diary; it is a means of self-recording the time and quantity of drinks and urine output and the time of incontinent episodes; a three day diary is recommended to confirm a pattern of day to day practice[2]. A frequency/ volume (F/V) chart is used to determine a voiding pattern and the voided volumes (see Appendix 2). The chart can be mailed to the patient prior to the first appointment, and completed by the patient or carer; consequently, the instructions should be easily understood.

Further questioning will be necessary to identify the types of drinks and the severity of incontinence; this can be measured by recording how often incontinence generally occurs, how much urine is lost at each incontinent episode, and the size and number of incontinence pads used per day or week. See appendix 2. It is also important to identify what caused the incontinence; for example, was urine leakage caused by an increase in abdominal pressure e.g. coughing, or by not getting to the toilet in time.

From the chart, information can be determined regarding frequency [normal frequency is 6 - 8 voids in 24 hours], incontinent episodes and nocturia (waking at night to void, sleeping before and after the void[3]). Maximum and minimum voided volumes and total volume voided, over 24 hours, give an indication of fluid intake, functional bladder capacity and bladder habits. The first void of the day is generally the largest, and physical activity and heat reduce urinary output.

7.3 Assessment Forms

An assessment form enables recording of findings in an orderly and systematic way. The form shown in Appendix 2 is designed to facilitate an audit of clinical outcomes, by recording at the initial visit and on discharge, the presence and severity of LUT symptoms, in addition to information gained from the frequency/volume chart and other investigations. Various items on the form are discussed below.

7.4 Bladder Symptoms

Life-style, occupation and hobbies affecting incontinence should be discussed. Symptoms of urinary loss and urgency noted, and thought given to possible causes. If urgency is associated with frequency and nocturia it is indicative of overactive bladder (OAB) syndrome[1]. Urge incontinence can be 'triggered' in a variety of ways; running water, coughing, 'key-in-the-door' syndrome, or simply entering the bathroom. Patients should be questioned about hesitancy, or any change in their flow rate or time taken to complete voiding. It may not be abnormal to have hesitancy when trying to void less than 100mL or very large amounts (>700mL).

The bladder is intended to be compliant during filling and contract during the voiding phase, emptying to completion. Failure to empty completely may be due to outflow obstruction, inability to relax the PFM and/or external urethral sphincter, or detrusor hypotonia/atonia.

Post micturition dribble refers to an involuntary loss of urine after finishing voiding. This is usually after a woman arises from the toilet or a man leaves the toilet[3] (see Chapters 9 and 21).

Bladder pain during the storage phase, or dysuria (pain on voiding), should always be investigated. It is important that pain is distinguished from urgency or bladder fullness. Any blood in the urine should be investigated; possible causes include kidney damage due to trauma, glomerulonephritis, pyelonephritis, strenuous activity, cystitis, urethritis, calculi and tumours.

7.5 History

A complete history is taken[4] (see Box 7.1)

7.5.1 Quality of Life

Quality of life issues are seen to be increasingly important. Please see Chapter 8 for full details.

7.5.2 Investigations

The results of urodynamics and other upper and lower tract studies should be recorded (see Chapter 9a). Urinalysis is essential especially for those patients with irritative symptoms: see Chapter 9b.

Box 7.1

Information required for a complete assessment

- Urological history
- Medical history
- Surgical history
- Childhood problems and relevant family history[4]
- Gynaecologic history[5]
- Obstetric history[6]
- Weight[7]/BMI
- Bowel history
- Medication: all present and relevant past medication

7.5.2.1 Stop Test

Historically, patients have been told to exercise their PFM by repeatedly stopping and starting the flow of urine. However, many respected clinicians believe that this is inappropriate[5] due to:- possible reflux of urine to the kidneys, interference with the micturition reflexes, possible inability to restart the urine flow and disillusionment of those unable to stop the flow. The stop test should be discouraged for all patients.

7.6 Physical Examination

Each department should have an agreed policy on infection control and consent procedures prior to any intimate examination. It is essential to obtain and record valid consent (some institutions require written consent) with any refusal recorded. The General Medical Council (GMC) has published guidance for intimate examinations[6]. Furthermore, each professional must take heed of the governing body of their own profession.

7.6.1 Neurological Examination

Activation of dermatomes and myotomes supplied by S 2.3.4 is thought to have an inhibitory effect on the sacral micturition reflex centre[7]. Testing of the appropriate dermatomes and myotomes will give information on the integrity of the nerve roots.

Myotomes supplied by S 2.3.4 include the PFM, external urethral and anal sphincters, hip extensors and lateral rotators. In addition, the plantarflexors, lumbricals and interossei all derive some innervation from S2. If abnormalities are noted, or neurological problems suspected, further assessment of the lumbo-sacral dermatomes, myotomes and reflexes should be carried out. If the history and subsequent simple tests are suspicious of a neurological problem, the patient should be referred appropriately[2].

7.6.2 Abdominal Examination

The abdomen should be evaluated for skin condition and surgical incisions, and palpated to identify any pain, abnormal pelvic mass, hernias or full bladder.

7.6.3 Vaginal Examination

The patient should be examined in supine, with knees bent, feet apart and in good light[8]. An agreed infection control policy (dated and signed) should be in place that includes examination and any instruments used during the assessment or treatment (see Chapter 36). Findings will be described under general observations and PFM assessment. Many of these findings can be recorded on 'The Rings of Continence' (ROC)[4] as depicted in Figures 7.1 and 7.3. This scheme previously combined assessment findings in both a vertical and a horizontal plane[4]; these planes have now been separated. See Figure 7.2. The vertical clock is shown in Figure 7.1. The large ROC represents the vagina, with 12 o'clock denoting the anterior and 6 o'clock the posterior segment; 9 o'clock represents the patient's right lateral wall and 3 o'clock the left. The smaller anterior ROC represents the urethra, and the smaller posterior ROC represents the anal canal.

7.7 Observations

Relevant observations[4] are detailed in Box 7.2

In addition, the ROC (see Figure 7.3) can be used to show the performance, during a MVC, of the urethral and pubovisceralis muscles. Both the modified Oxford scale (0 to 5) and the 4-point ICS scale (absent, weak, normal, strong)[8] (see Figure 7.3) are shown.

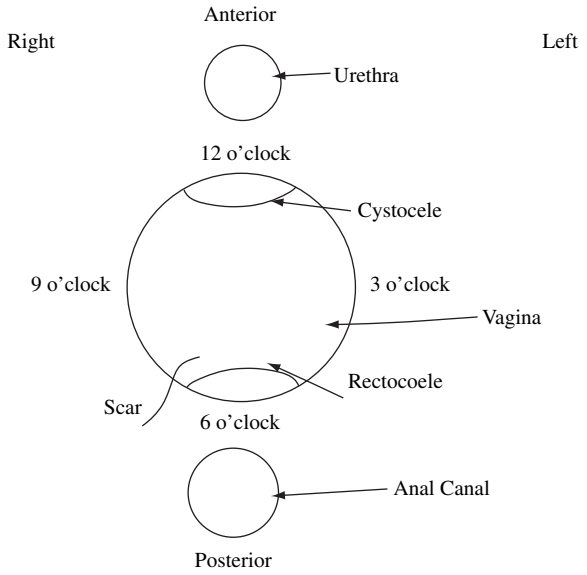

Figure 7.1. Rings of continence; vertical clock showing orientation of urethra, vagina and anal canal. Also shown: cystocoele, rectocoele and scar

Box 7.2

Observations on perineal inspection:

- Red, excoriated skin on the upper thighs and vulva
- Vaginal oestrogenisation status[9,10].
- Scars following childbirth or surgery. See Figure 7.1.
- Prolapse, described in greater detail in Section 5, Chapters 31 to 33. Prolapse can be recorded on the ROC (Figures 7.1 and 7.3).

7.8 Pelvic Floor Muscle Assessment

The patient is instructed to squeeze and lift the PFM as if preventing the escape of flatus and/or urine. If a vaginal digital assessment is inappropriate, it is useful to be able to observe the perineum and assess the muscle action.

During a maximum voluntary contraction (MVC) of the PFM[8], the following may be observed: the anus retracts, perineum is drawn inwards, posterior vaginal wall moves anteriorly and the urethral meatus 'winks'.

There is growing evidence to support the synergistic action of the transversus abdominis (TrA) with the PFM (for details, see Chapters 11c and 12b) inferring that co-contraction of the TrA may take place during a MVC of the PFM.

7.8.1 Procedure

The basic procedure[4] for vaginal assessment is shown in box 7.3. Valid consent must be obtained prior to any procedure[6].

Box 7.3

Procedure for vaginal examination

- Separate the labia with the thumb and index finger
- Introduce the lubricated index finger of the examining hand into the vagina
- Assess for pain, sensation and vaginal capacity
- PFM[11] and urethral sphincter evaluation (see below)

7.9. Vaginal Palpation (M. Whelan)

7.9.1 Planes of Examination

The PFM can be examined and described on two different planes, a vertical clock perpendicular to a horizontal clock.

The vertical clock has already been described. The pubic symphysis lies at 12 o'clock and the perineal body lies at 6 o'clock. Pubococcygeus also known as puboviscerali lies at 4 o'clock and 8 o'clock; contraction of pubococcygeus produces a squeeze at the base of the palpating finger.

The horizontal clock shares a base with the vertical clock at the perineal body (6 o'clock) the coccyx now represents 12 o'clock. The pad of the palpating finger at 10 o'clock and 2 o'clock will palpate the iliococcygeus with the base of the palpating finger in contact with the pubococcygeus (Figure 7.3); the finger must be fully extended in order to reach the depth required. On this plane the contraction of the muscles will produce a cranioventral lift of the pad of the examining finger. Puboviscerali will lie in the plane of the vertical clock at its anterior attachment; it will then become part of the horizontal clock at the posterior attachment. Through differentiation of activation at the pad and base of the palpating finger on the horizontal clock the therapist can assess quality of contraction of the PFM (see Chapter 11b; Figure 11b.1).

7.9.2 Muscle Activation

The resting tone at the anterior and the posterior PFM may vary. Commonly when palpating the horizontal clock with the finger-pad at 10 o'clock and 2 o'clock, an inhibited posterolateral vaginal wall yields a fibrous almost tendinous type structure that does not lift the finger-pad cranio-ventrally. This tendinous structure will be painful on deep palpation. The more proximal puboviscerali may squeeze and lift the base of the palpating finger but the finger-pad loses contact with the posterolateral wall as the puboviscerali contracts, indicating inhibition of iliococcygeus. This is in contrast with a

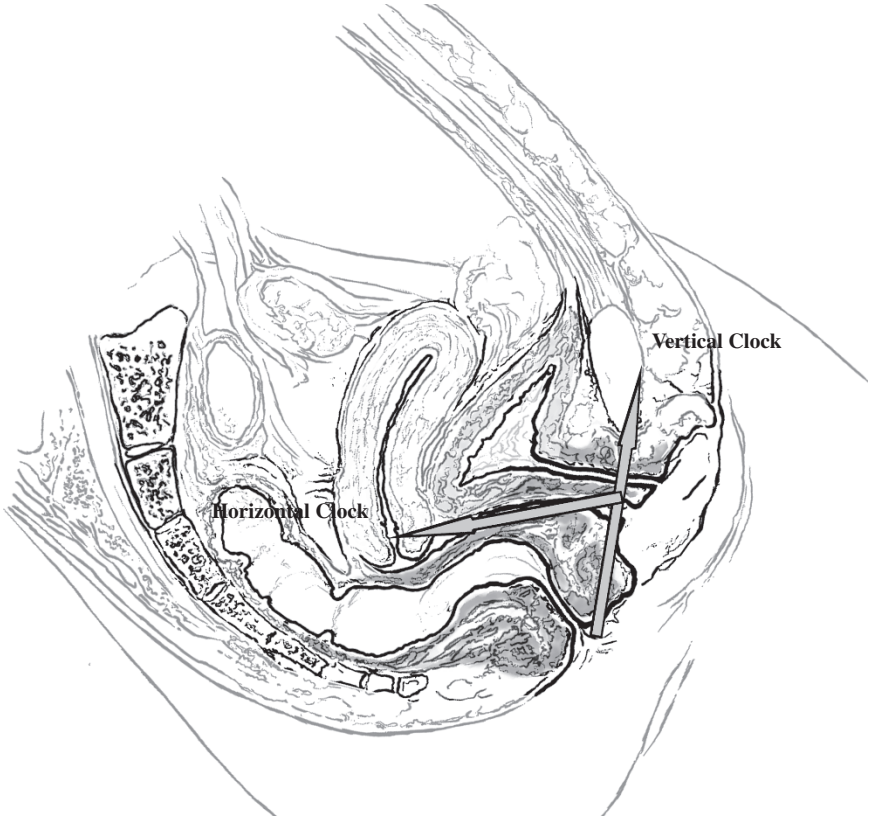

Figure 7.2. Planes of Examination

more balanced contraction where the entire ventral surface of the palpating finger is lifted as a unit and contact with the finger-pad is maintained (see Chapter 11b; Figure 11b.2).

7.9.3 Maximal Relaxation

If the PFM feels taut on palpation then muscle release is not maximal; it is equally important to assess the degree of relaxation when assessing a contraction. Maximal relaxation is assessed at the anterior attachments of the puboviscerali at the base of the palpating finger and at the iliococcygeus under the finger-pad. The degree of relaxation can be graded as a percentage of the degree of contraction. When maximal relaxation on both planes is reached, then the muscle will feel soft on palpation. This can be challenging with a dysfunctional muscle, and techniques to achieve this are described in Chapter 11b.

7.10. PFM Assessment (J Laycock)

7.10.1 Muscle Contractility. The New PERFECT Scheme

This acronym (see Table 7.1) was developed and validated[12] to assess pelvic floor muscle contractility and enable the planning of patient specific muscle training regimens[4] (see Chapter 11a); it has now been further developed.

Table 7.1 The New PERFECT assessment scheme

P	**Performance**	A measure of the strength of an MVC
E	**Endurance**	The time (in seconds) that the MVC can be held before strength is reduced by 50% or more
R	**Repetitions**	Number of times the MVC can be repeated
E	**Elevation**	Lifting of the posterior vaginal wall during an MVC
C	**Co-contraction**	Co-contraction of the lower abdominal muscles during an MVC
T	**Timing**	Synchronous involuntary contraction of the PFM on coughing (see below).

7.10.1.1 Performance [P]

Having established that a correct PFM contraction is being performed, the examiner grades the performance (P) of the contraction, using a modified Oxford scale (see Box 7.4) by assessing the strength of the maximum voluntary contraction, using one finger[8]. Both sides are graded, and the stronger side assessed for endurance, repetitions and fast contractions (see below). The non-examining hand palpates the abdominal muscles to detect appropriate activity.

Careful palpation can identify weakness in different portions of pubovisceralis. The original modified Oxford grading study[12] demonstrated reliability and reproducibility between 18 trained physiotherapists, however a later study has reported high inter-observer variability[13]. This suggests that training is essential to ensure all participants in a study are grading equally. The International Continence Society (ICS) now recommends a 4-point scale: absent, weak, normal or strong[8]. However this scale has not yet been validated, and in clinical practice is less sensitive. Furthermore the author feels that 'normal' is a difficult grade to quantify, and suggests that, if this revised scale is used, 'normal' should be considered moderate. The 4-point scale for an MVC reads:- A-absent, W- weak, N- normal, S- strong. See Figure 7.3 for application of these scales and Box 7.5 for a comparison between the scales.

The external urethral sphincter performance is evaluated by palpating with the pad of the index finger through the anterior vaginal wall during a MVC, and graded absent, weak, normal or strong. Using the same scale, the urethrovaginal sphincter is assessed on the left and the right. See Figure 7.3.

Box 7.4.

The modified Oxford scale

Grade 0: no discernible PFM contraction.

Grade 1: a flicker, or pulsing under the examining finger- a very weak contraction.

Grade 2: a weak contraction - an increase in tension in the muscle without any discernible lift or squeeze.

Grade 3: a moderate contraction- characterised by a degree of lifting of the posterior vaginal wall and squeezing on the base of the finger (pubovisceralis) with in-drawing of the perineum. A grade 3 or higher grade contraction are generally discernible on visual perineal inspection.

Grade 4: a good PFM contraction producing elevation of the posterior vaginal wall against resistance and in-drawing of the perineum. If two fingers (index and middle) are placed laterally and/or vertically in the vagina and separated, a grade 4 contraction can squeeze them together against resistance.

Grade 5: a strong contraction of the PFM; strong resistance can be given against elevation of the posterior vaginal wall and approximation of the index and middle fingers as above.

Box 7.5

Oxford Scale	ICS Scale
0	Absent
1 and 2	Weak
3 and 4	Normal (moderate)
5	Strong

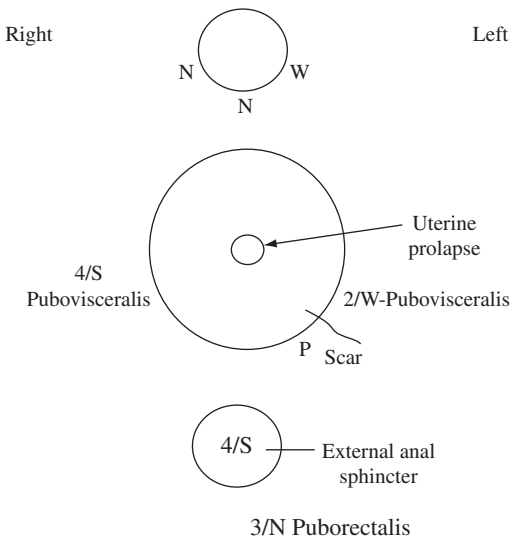

Figure 7.3. Rings of Continence (ROC); vertical clock showing from above: Right side urethrovaginal sphincter – normal, Left side urethrovaginal sphincter – weak, External urethral sphincter - normal, Right pubovisceralis – grade 4 / strong, Left pubovisceralis – grade 2 / weak, Uterine prolapse, scar, P – pain. External anal sphincter – grade 4 / strong, Puborectalis – grade 3 / normal

7.10.1.2 Endurance [E]

This is the time up to 10 seconds that the maximum contraction is held, before a reduction in power of 50% or more is detected.

7.10.1.3 Repetitions [R]

The number of repetitions (up to 10) of the MVC is recorded, allowing 4 seconds rest between contractions.

7.10.1.4 Fast [F]

After a short rest of at least one minute, the number of fast (up to 10), one second MVC's is assessed, with the patient instructed to 'contract – relax' as quickly and strongly as possible, in her own time, until the muscles fatigue.

7.10.1.5 Elevation, Co-contraction and Timing – [E C T]

See Table 7.1

Below are examples of PERFECT assessments. Performance (P) is graded using the modified Oxford scale and the revised ICS scale.

Example 1

P	E	R	F	E	C	T
3/N	5	4	7	√	√	0

This assessment describes a patient with a 'normal' (grade 3) MVC, held for 5 seconds and repeated 4 times, followed, after a rest, by 7 fast contractions. In addition, elevation of the posterior vaginal wall and co-contraction of the lower abdominals was detected, but involuntary contraction of the PFM on coughing was not demonstrated. The aims of treatment include increasing strength and endurance (and repetitions) but, most importantly, to teach 'The Knack'[14] (see Chapter 11.)

Example 2

P	E	R	F	E	C	T
2/W	2	3	/	0	√	0

This patient has weak fatiguable muscles: a grade 2 /weak contraction held for 2 seconds and repeated 3 times. In this case, it is inappropriate to assess the fast muscle contractions. This example also shows there is no elevation of the posterior vaginal wall on MVC of the PFM, there is co-contraction of the lower abdominals, but no discernible involuntary contraction of the PFM on coughing. Treatment will aim at increasing strength and endurance, and making the patient more aware of a PFM contraction; this will be followed by teaching 'The Knack'[14] (see Chapter 11a).

7.11 Self Assessment

Some women may wish to examine themselves by observation of a perineal lift with a mirror or by self-palpation. However this should not be assumed as it has been shown that only 30% of older women are quite comfortable with the concept of touching their own genitalia[15]. If appropriate, self vaginal examination can be explained as a method of proprioception and self assessment. It can be explained as introducing a clean thumb vaginally, 'hooked over' the postero-lateral PF, perhaps when in a bath or shower. If appropriate, it may also be suggested that they get feedback from their sexual partner. One of the side effects of increased PFM activity is increased sexual satisfaction[16]; this may in itself encourage effort and compliance to exercise.

7.12 Measurement of PFM Function by Dynamometry (C. Dumoulin)

7.12.1 Introduction

Dynamometers have been widely used by physiotherapists for evaluating trunk and limb muscle function for more than 40 years[17]. The Montreal dynamometer, comprising a computerised central unit and a dynamometric speculum (Figure 7.4), has been designed specifically to assess the PFM. The central unit consists of a laptop computer and a data acquisition card; the

Figure 7.4. Montreal PFM dynamometer

dynamometric speculum is made of two aluminium branches; an upper fixed branch and a lower adjustable one.

7.12.2 PFM Assessment

Direct measurements of the PFM strength are taken at different openings of the speculum, thereby allowing measurement at different muscle lengths. The resultant forces exerted by the PFM on the speculum are measured on the cantilever principle using two pairs of strain gauges glued on each side of the moveable lower branch of the speculum. Voltage values from the strain gauge are amplified, digitised and converted into units of force (N) and then presented in graphic form (Figure 7.5).

The dynamometer has shown the ability to measure the resultant PFM force independently of its point of application on the branch of the speculum implying that the voltage difference remains constant wherever the same force is applied on the branch of the speculum[18].

7.12.3 Psychometric Evaluation

Test-retest reliability studies with young and middle-aged women suffering from persistent postpartum stress urinary incontinence have indicated good to very good reliability of strength measurements[19,20]. Women's reaction to the measurement procedure, assessed in the reliability study, showed that this new instrument and the measuring procedure are acceptable[19]. In a study comparing the new dynamometer with the modified Oxford grading system[12, 21], significant correlations with coefficients of r = 0.727, r = 0.450 and r = 0.564 for

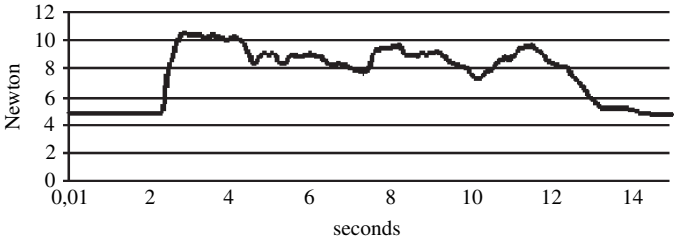

Figure 7.5. Recording of PF passive strength (baseline) and maximal strength

continent, incontinent and all women, were found[22]. These studies support some of the various aspects of the construct validity of the dynamometric speculum[22]. Finally, the capability of the dynamometer to discriminate between incontinent and continent postpartum women[23] in addition to pre- and post-treatment postpartum women[24] confirms further aspects of the construct validity.

Although this new dynamometer seems a highly promising tool for assessing PFM function, the effect of intra-abdominal pressure, coughing and straining on dynamometric measurements needs to be investigated. More psychometric evaluation is required before the dynamometer is available commercially.

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8
Quality of Life Assessment and Questionnaires

KNL Avery and JL Donovan

8.1 Health and Quality of Life

> *Health is a state of complete physical, mental and social well-being, and not merely the absence of disease or infirmity*
>
> World Health Organization, 1946[1]

Health is cited in the general population as an important determinant of an individual's quality of life (QOL)[2]. Illness may not just impinge upon physical functioning but also on other aspects of well-being, including psychological, emotional, and social well-being, subsequently affecting QOL[3]. The World Health Organisation's definition conceptualised health as a broad, multidimensional concept, emphasising the intrinsic relationship that exists between health and QOL and thereby highlighted the relevance of including QOL as an outcome measure in this field.[2]

8.2 Health-Related Quality of Life

For the requirements of clinical medicine and clinical trials, it is more relevant to focus on those aspects of QOL that may be influenced by disease or its treatment.[4] "Health-related quality of life" includes "attributes valued by patients including their resultant comfort or sense of well-being; the extent to which they are able to maintain reasonable physical, emotional, and intellectual function; the degree to which they retain their ability to participate in valued activities within the family, in the workplace and in the community" (Wenger and Furberg, quoted in ref. 5). This emphasises the multidimensional nature of QOL and the importance of considering individuals' perceptions of their own situation in the context of nonhealth-related aspects.

8.3 Impact of Incontinence on Quality of Life

Urinary incontinence (UI) interferes with everyday life, causes practical inconvenience and financial hardship, and often requires elaborate planning to conceal or prepare for incontinent episodes.[6] Feelings of embarrassment or negative self-perception are common.[7]

8.4 Measuring Quality of Life

8.4.1 Clinical Measures of Incontinence

Treatments for UI largely aim to reduce the occurrence of episodes or limit the impact of symptoms. Diagnosis and classification of symptom severity is commonly achieved by pad testing and urodynamic studies. Overall, there is only a weak relationship between symptomatic, QOL, and objective clinical or urodynamic measures of UI. The type, severity, or number of symptoms or the results of urodynamic or clinical investigations cannot predict impairment.[7–9] While UI is prevalent among the general population, it is not always bothersome.[8] It is not

surprising that what is demonstrated clinically is distinct from what is perceived by patients to be troublesome: clinical measures and questionnaires probably measure different but related aspects of UI. Consequently, it is now widely accepted that measurement of UI should incorporate both self-reported symptoms and their perceived impact on QOL. The importance of assessing QOL in health outcomes research has been acknowledged by many groups, including the UK Medical Research Council (MRC) and the European Organization for Research and Treatment of Cancer (EORTC), who have outlined policies stipulating that QOL should be considered as an endpoint in all new trials.[10] Traditionally, the clinical history has been used to summarise symptoms and their impact on QOL. Clinicians' assessments of impact have been shown, however, to underestimate the degree of interference perceived by patients, and to focus on issues of lesser importance to patients.[11,12]

8.4.2 Self-Completion Questionnaires

Increasingly, patient-completed methods of measuring UI are being used, including voiding diaries (frequency-volume charts or urinary diaries) and questionnaires. Voiding diaries assess a limited number of UI symptoms. For a full assessment of symptoms and their impact, questionnaires that have been shown to be valid and reliable are recommended. Questionnaires provide a method for standardised data collection from patients.[13] They measure "subjective" phenomena in an objective way and increasingly assess symptoms, bothersomeness, and impact on multiple QOL dimensions.

8.4.3 Suitability/Feasibility

Importantly, a questionnaire should be suitable for its purpose and feasible to be used. There is a tension between encompassing all aspects of the condition and avoiding respondent burden and making the instrument simple to use. Increasingly, short and long versions of questionnaires are being produced to allow detailed research as well as rapid evaluation for research and clinical practice.

8.4.4 International Implementation

Increasingly, questionnaires are required to be used in different populations and settings but psychometric properties are not necessarily transferable. There are particular problems with the interpretation of symptoms and aspects of QOL in different populations and cultures.[14] Steps can be taken to ensure that questionnaires may be used by different cultural groups.[15,16]

8.5 Questionnaire Grading

Questionnaire design and utilisation is not simple. To have confidence that the questionnaire is measuring what it is supposed to measure, that it does this reliably, and is appropriate for use in the patient/population group under investigation, a number of studies must be conducted. Empirical evidence is required to demonstrate the precision and accuracy of measures, more commonly referred to as psychometric properties, including validity, reliability, and responsiveness to change. When designed and tested thoroughly, questionnaires can have levels of precision that equal or exceed clinical measures.

The Symptom and QOL Assessment Committee of the International Consultation on Incontinence has published regular literature reviews of the assessment of UI and vaginal and pelvic floor problems.[17–19] The Committee has developed standardised grades of recommendation for questionnaires based on the Oxford Centre for Evidence Based Medicine's Levels of Evidence and applied them to categorise questionnaires in these areas.[19] For UI/lower urinary tract symptoms (LUTS), there are two grades of recommendation: Grade A (highly recommended) is for established measures with documented, rigorous validity, reliability, and responsiveness in several clinical studies; Grade A^{new} (highly recommended) is for newly developed measures with published reports of acceptable validity, reliability, and responsiveness indicated with rigour in at least one study. Grade B (recommended) is for measures with some validity, reliability, and responsiveness indicated, or for which only validity and reliability (but not responsiveness) have been established with rigour.

Table 8.1. Highly recommended (Grade A) qestionnaires for the assessment of QOL impact of UI alone or in the presence of LUTS, including overactive bladder (OAB).

Questionnaire	Gender	Validation	Source-language	Type of UI	UI symptoms	UI QOL	OAB symptoms	OAB QOL	Translations available
International Consultation on Incontinence Questionnaire (ICIQ-UI SF)	Both	Grade A [new]	UK-English	Any	✓	✓			✓
Incontinence Quality of Life (I-QOL)	Both	Grade A	US-English	Any		✓			✓
SEAPI-QMM incontinence classification system	Both	Grade A[new]	US-English	Any		✓			✓
Bristol Female LUTS Short Form (BFLUTS-SF)	Women	Grade A	UK-English	Any	✓	✓	✓	✓	
ICS*male*SF	Men	Grade A	UK-English	Any	✓	✓	✓		✓
Kings Health Questionnaire (KHQ)	Women	Grade A	UK-English	Any		✓	✓		✓
Incontinence Impact Questionnaire (IIQ) & IIQ short form (IIQ-7)	Women	Grade A	US-English	Any					
SUIQQ	Women	Grade A[new]	Norwegian	Stress, urge	✓	✓			
Urinary Incontinence Severity Score (UISS)	Women	Grade A[new]	Finnish	Any		✓			✓
CONTILIFE	Women	Grade A[new]	French	Any		✓			✓
Overactive Bladder questionnaire (OAB-q)	Both	Grade A[new]	US-English	Urge			✓	✓	✓

8.6 Questionnaires to Assess Symptoms and QOL Impact of UI and/or LUTS

Many UI questionnaires have been developed for men or women separately and several include assessment of LUTS. Table 8.1 shows highly recommended (Grade A) questionnaires for the assessment of QOL impact of UI alone or in the presence of LUTS, including overactive bladder (OAB). Characteristics that may be important for choosing an instrument for a study or patient population are indicated. Given the plethora of questionnaires now available, only those reaching the highest level of rigour (Grade A) are reported. Ten assess QOL related to UI and one QOL related to OAB. Five of these simultaneously assess symptoms. Only one (the International Consultation on Incontinence Questionnaire[20]) assesses both symptoms and QOL impact and encompasses all aspects of UI in men *and* women.

Established UI questionnaires often are modified for use in selected patient groups (eg, males, different cultural/language groups) and a number of those reported here now have published modified versions, such as the Urge Incontinence Impact Questionnaire for patients with urge UI. Although it may be appropriate to modify questionnaires, it is advisable to keep modifications to a minimum and to use original versions whenever possible. Modifications of established questionnaires may result in changes (sometimes substantial) in the instrument's psychometric performance, and thus modified instruments should be subjected to the same psychometric testing as that employed in developing a new instrument.

Numerous other questionnaires have been developed to assess QOL impact of LUTS and UI. They did not have, by the time of the ICI Committee's 2004 review,[19] the full complement of psychometric evaluation or robust data, or are less relevant for UI than the questionnaires reported above. They include the Leicester Impact Scale (men and women) and the Male Urinary Symptom Impact Questionnaire, King's Health Questionnaire, and Nocturia Quality of Life questionnaire (men).

8.7 Questionnaires to Assess Symptoms and QOL Impact of Vaginal and Pelvic Floor Problems

Many women present with vaginal symptoms and pelvic organ prolapse (POP) is frequently implicated. A clinical history is usually used to assess

Table 8.2. Recommended (Grade B) questionnaires and those with potential (Grade C).[a]

Questionnaire	Validation	Source-language	Symptoms	POP bother/QOL	Translations available	Comment
Pelvic Floor Impact Questionnaire (PFIQ)	Grade B	US-English		✓	✓	Adaptation of IIQ
P-QOL/St. Mary's Questionnaire	Grade C	UK-English		✓	✓	
Pelvic Floor Dysfunction Questionnaire	Grade C	US-English		✓		
e-PAQ Pelvic Floor Symptoms Questionnaire	Grade C	UK-English	✓	✓		Electronic format available
Danish Prolapse Questionnaire	Grade C	Danish	✓	✓		
ICIQ-Vaginal Symptoms Questionnaire (ICIQ-VS)	Grade C	UK-English	✓	✓		

[a]Notably, none currently meet the criteria to be highly recommended (Grade A).

symptoms but, as with UI, symptoms often do not correlate with objective findings. Clinician-based history is inconsistent, impact may not be assessed, leading questions can be asked and patients may be unwilling to volunteer symptoms. Unlike UI, the ICI Committee noted that the assessment of vaginal and pelvic floor problems is a developing area with few questionnaires currently reaching the highest levels of evidence. Consequently, despite the prevalent nature of these conditions we have little idea how intervention alters symptoms and QOL. A third grade of recommendation (Grade C) therefore was developed to indicate questionnaires in early development, where further validation and application is required.[19] Table 8.2 details recommended (Grade B) questionnaires and those with potential (Grade C). Notably, none currently meet the criteria to be highly recommended (Grade A).

8.8 Generic Health Measures for UI

These questionnaires aim to measure the multidimensional nature of health status (Table 8.3) and cover the major QOL domains and, with the exception of the EQ-5D, other well-being issues. All are suitable for a range of illnesses and populations.

Table 8.3. Generic health measures for UI.

Questionnaire	Gender	Validation	Source-language	Use in relevant samples	Domains	Translations available
MOS Short Form – 36 items (SF-36)	Both	Grade A	US-English	UI, benign prostatic hyperplasia (BPH), LUTS, OAB	Physical function, social function, emotions, personal productivity, pain, general health perception/ status, other well- being issues	✓
MOS Short Form – 12 items (SF-12)	Both	Grade A	US-English	Prostatectomy patients	Physical health, mental healht	✓
Rand-36	Both	Grade A	US-English	UI, prostate cancer	Retains the same items and subscales as the SF-36 but uses a slightly different scoring algorithm	✓
EuroQoL EQ-5D	Both	Grade A	UK-English, Dutch Swedish, Norwegian, Finnish	UI	Physical function, social function, emotions,personal productivity, pain, general health perception/status	✓
Nottingham Health Profile (NHP)	Both	Grade A	UK-English	UI, LUTS, prostate cancer	Physical function, social function emotions, personal productivity, pain, other well-being issues, sexual function	✓
Sickness Impact Profile (SIP)	Both	Grade A	US/English	UI, LUTS, BPH	Physical function, socialfunction, emotions, personal productivity, other well-being issues, neuropsychological/ cognitive function	✓

While they do not contain specific questions on UI, they have been widely used to assess the QOL of incontinent adults on the assumption that UI impacts on general well-being. During the past five years, the SF-36 has been the most widely used generic health-related QOL measure to assess UI. Several other instruments, such as the SIP, the NHP, and the EQ-5D, have had limited use in recent years. All have achieved acceptable reliability and validity in incontinent individuals. They can, however, be lengthy and relatively insensitive to UI. Overall, these generic instruments are useful in comparing across conditions or in describing the general health status of incontinent adults.

8.9 Questionnaires to Assess Sexual Function/Satisfaction

Sexual function may be regarded as a dimension of overall QOL for which a wide choice of specific measures has been validated. A number have been specifically developed to examine sexual function in patients with pelvic floor disorders such as UI. Established measures that have been shown to demonstrate adequate psychometric properties are desirable but the appropriateness of using a particular instrument also must be considered. Clinicians often prefer to use unstructured interviews in clinical practice to allow the tailoring of questions to the individual or couple being assessed. They allow vocabulary, level of assertiveness, and depth of questioning to be modified and enable the clinician to support patients who feel vulnerable and encourage discussion. This flexibility is not readily achievable with questionnaires. Some patients, however, find discussing intimate issues very difficult and questionnaires may allow issues to be measured more effectively in private before exploring questionnaire responses in the clinical interview.

Highly recommended (Grade A) questionnaires for evaluating sexual matters in patients with UI include the Golombok–Rust Inventory of Sexual Satisfaction (men and women) and the International Index of Erectile Function, ICS*sex*, and BPHQOL9 (men). Recommended (Grade B) questionnaires include the Psychosocial Adjustment to Illness Scale (men and women), Pelvic Organ Prolapse/Urinary Incontinence Sexual Questionnaire and Brief Index of Sexual Function for Women (women), and the Brief Sexual Function Inventory (men).

Many of the questionnaires assessing the psychosocial impact of UI/LUTS contain some sexual function questions. Those containing just one question tend to focus on general overall impact on sexual functioning. The KHQ, IIQ, and I-QOL are recommended for UI/LUTS.

8.10 Questionnaires for Specific Patient Groups

Most questionnaires have been developed for use in the general population or patients with UI or vaginal and pelvic floor problems. However, some patient groups may experience particular problems with UI (eg, children, frail elderly, spinal cord or neurological damage, prostate/bladder cancer, benign prostate disease), which may require more specific measures or addition of items to existing instruments. Researchers should use existing recommended questionnaires, if possible, to aid comparison and reduce the proliferation of questionnaires.

8.11 Conclusions

It is encouraging that the use of high-quality validated questionnaires in research and clinical practice with regard to UI and vaginal and pelvic floor problems has increased markedly in recent years, offering potential benefits to patient care due to the more accurate measurement of symptoms and QOL.[14] This chapter provides a general introduction to the assessment of QOL in this field. Available questionnaires recommended for use and the characteristics, benefits, and shortcomings associated with them have been discussed. The selection of instruments for clinical or research purposes will depend on the objectives of the assessment, the patient/population being assessed, the nature of the intervention (where applicable), and the specific research questions being addressed.

An increasing number and variety of questionnaires to assess UI have been identified that reach the highest level of scientific rigour. Many others, however, lack sufficient evidence of psychometric properties or significant use in relevant populations. Researchers and health professionals therefore are encouraged to use established questionnaires with published evidence of psychometric properties where possible. The development of questionnaires to assess vaginal and pelvic floor problems, however, is at a much earlier stage and further work to develop and validate these questionnaires is encouraged.

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9a
Investigations for Lower Urinary Tract Dysfunction

C Chaliha and V Khullar

9a.1 Introduction

The evaluation of women with lower urinary tract (LUT) dysfunction should include a detailed history, physical examination, urinalysis, and if possible assessment of postvoid residual. However, the basic evaluation often performed in the primary care setting and the basis for which empirical therapy is instituted may lead to inappropriate treatment due to inaccurate diagnosis especially as history often has a poor relationship to underlying pathology.[1,2] Further investigations for secondary care usually are reserved for those with complicating factors or comorbidities. They also should be considered if a trial of empirical treatment fails or symptoms worsen. It is imperative that urodynamic studies are performed prior to irreversible or surgical procedures.

There are a variety of investigations for LUT dysfunction as shown in Table 9a.1. Not all patients require such detailed investigation and assessment techniques should be tailored to the individual patient.

Table 9a.1. Various investigations for LUT dysfunction.

Non-specialist investigations	History
	Examination
	Midstream urine
	Frequency–volume chart
	Postvoid residual
Specialist investigations	Uroflowmetry
	Cystometry
	Videocystometry
	Ambulatory urodynamics
	Urethral pressure profilometry
	Intravenous urography
	Micturating cystography
	Ultrasonography
	Magnetic resonance imaging

9a.2 Specialist Investigations

9a.2.1 Pad Tests

These are simple, noninvasive investigations that enable objective measurement of urine loss under standard and reproducible conditions.

The pad tests can be divided into those < 1hour, 1 hour, 24 hours, and 48 hours. The short pad tests (<1 hour and 1 hour) have not been found to be good in assessing the severity of urinary incontinence[3,4] and a significant proportion of incontinent patients will not leak during the 1-hour pad test. The reproducibility of the 1-hour pad test is improved by using a standardised bladder volume and a provocative exercise regime. The 24- and 48-hour pad tests have better sensitivity and reproducibility [5,6] with similar sensitivity such that overall the 24-hour pad test is sufficient. The amount of urine lost during the test is determined by the change of weight of the pad. A weight gain of greater that 1gm signifies incontinence. Although the pad test cannot distinguish between detrusor overactivity (DO) and urodynamic stress incontinence (USI), it can indicate severity of incontinence and is a relatively simple and inexpensive test. If pads are going to be used, the pad test protocol should be standardised for all patients with clear instructions.

9a.3 Urodynamics

Urodynamics assess the function of the LUT. These investigations should be planned prior to continence surgery, for those with recurrent symptoms after previous continence surgery, those with mixed symptoms, those with voiding dysfunction, and after failure of empirical therapy.

9a.3.1 Uroflowmetry

Uroflowmetry is an essential part of the urodynamic investigation as a screening test for voiding difficulties. It enables the measurement of maximum flow rate, average flow rate, flow time, and time to maximum flow. Its major advantage is that it can identify women with poor peak flow; however, it cannot distinguish between outflow obstruction and detrusor hypotonia. A flow rate is best assessed if the patient voids more than 150mL and if the patient feels that the void was representative of how he or she normally voids. A flow rate of less than 15mL/s indicates impaired voiding. Flow rates are of particular value in identifying women at high risk of voiding difficulty if continence surgery is contemplated.

The patient should be asked to attend with a full bladder and the flowmeter placed where voiding can occur in privacy. There are several commercial flowmeters of which those that use the principles of weight transduction, a rotating disk, and capacitance transducer are the best known and most well validated. All three types are accurate enough for clinical use though an independent assessment of flowmeters found that the rotating disk device is most accurate.[7]

9a.3.2 Cystometry

Cystometry involves the measurement of intravesical pressure during bladder filling and emptying. It describes the pressure–volume relationship of the bladder, detrusor contractility, bladder sensation, and compliance. Ideally all patients with LUT symptoms should undergo cystometry as the bladder is an unreliable witness and treatment based on symptoms alone may not be accurate.[2,8]

Cystometry should be performed in the upright position as the majority of patients complain of symptoms when standing. The filling medium should be water or 0.9% saline at room or body temperature. The bladder pressure can be measured with either a fluid catheter connected to an external pressure transducer or a solid microtip transducer mounted onto a solid 7 F catheter. Abdominal pressure can be measured with either a rectal or vaginal catheter. Three filling rates have been described by the International Continence Society (ICS)[9]: a slow fill at 10 ml/min, a medium fill of 10–100ml/min, and a fast fill when the rate is greater than 100 ml/min.

Fast filling is provocative for patients with DO although it will not "provoke" a stable bladder to contract.[10,11] During the test the patient should stand and provocations such as heel bouncing and coughing observed.[12] Any rises in detrusor pressure may be noted if associated with urgency. At the end of filling, the filling catheter is removed and the patient is asked to void into a flowmeter to record the maximum voiding pressure and flow rate. This can distinguish a patient with detrusor hypotonia (low pressure and low flow) from one with obstructed flow (high pressure and low flow). After voiding any postvoid residual can be measured. Evaluation of pressure flow parameters has high reproducibility and consistency in evaluating LUT function during voiding[13] and can provide prognostic information regarding the likelihood of developing voiding dysfunction postoperatively.[14–16]

9a.3.3 Videocystometry

This can be considered the gold standard for LUT investigation.[17] A radio-opaque medium is used to allow visualisation during cystometry with X-ray screening and is said to be more sensitive in the diagnosis of USI than simple cystometry. The LUT is visualised while there is synchronous recording of bladder function. The diagnostic criteria for intrinsic sphincter deficiency is the presence of funnelling (an open bladder neck and proximal urethra) with subsequent leakage of urine/contrast with rises in abdominal pressure without a detrusor pressure rise. However, it should be noted that observation of contrast medium leaking into

the proximal urethra without associated bladder contraction has been seen in 50% of continent women.[18]

If the history does not fit the diagnosis, where there has been previous surgical intervention or a history of neurological abnormality and suspicion of detrusor sphincter dyssynergia, videocystometry can be useful and the cost justified.[19,20] Videocystourethrography also can visualise bladder or urethral diverticulae, urethral stenosis, or vesicoureteric reflux which may be present in up to 7% of incontinent patients.[17,21]

9a.3.4 Ambulatory Urodynamic Monitoring

This aims to investigate the bladder under more physiological conditions and when laboratory urodynamics do not match symptoms. The recording system should be portable and battery operated and can be connected to an electronic nappy to allow urine leakage to be quantified and timed. A normal protocol for ambulatory urodynamics lasts 4 hours and during this time the patient is asked to drink a prescribed volume of fluid usually every half an hour and keep a detailed diary of symptoms and events. The patient voids prior to the test, and following calibration, microtip transducers are inserted into the bladder and rectum. Every half hour provocative manoeuvres are carried out. All traces should be interpreted with the patient present so that more information can be obtained about particular recorded events.

There is conflicting evidence regarding the usefulness of ambulatory testing as, although it may be thought to be more sensitive and physiological than laboratory urodynamics,[22,23] it may overdiagnose DO. This has been demonstrated in studies of asymptomatic subjects that show that 38–69% have DO on ambulatory testing but only 18% on laboratory urodynamics.[24–26] Thus it may be that ambulatory urodynamics has a high false-positive rate for DO such that applying standard ICS diagnostic criteria may not be appropriate.[9] The use of a symptom diary and more than one transducer in the bladder can decrease by almost two thirds the diagnosis of pathological DO on ambulatory urodynamics.[27]

9a.3.5 Urethral Pressure Profilometry (UPP)

Continence is maintained as long as the intraurethral pressure exceeds the intravesical pressure. UPP allows the simultaneous measurement of intravesical and intraurethral pressure. A soft silicone catheter with two transducers 6 cm apart is gradually withdrawn along the urethra allowing simultaneous recording of urethral and vesical pressure. Electronic subtraction of these recordings gives a measure of urethral function at rest and during stress conditions. UPP is not required in all patients but should be considered in those with voiding difficulties and those with previous failed surgery as a low maximum urethral closure pressure (<20 cm H_20) has been considered predictive of poor outcome of conventional surgery and is a strong indicator of intrinsic sphincter deficiency.[28]

The maximum urethral pressure and maximum urethral closure pressure are reduced in women with urodynamic stress incontinence compared to controls[29,30]; however, there is a wide overlap in values making this of limited value. A high maximum urethral closure pressure indicates obstruction and in these cases a urethrotomy may be considered. It also may be of use in diagnosing urethral diverticula.[31]

9a.4 Radiology of the LUT

Imaging of the LUT is increasingly used as an adjunct to urodynamic studies and has the advantage that it allows evaluation of the LUT and its surrounding structures.

9a.4.1 X-ray

A plain film of the abdomen can be used to screen for urinary calculi or as the first film in an intravenous urogram. Anteroposterior and lateral images of the spinal cord are used to identify vertebral defects in patients suspected of having neurogenic incontinence, especially if there are associated abnormalities on neurogenic examination. Increased visualisation of the LUT is possible with the use of contrast agents, such as videocystourethrography as described. Contrast instilled at the time of urodynamics may allow visualisation of diverticula, fistula, stones, tumours, and reflux.

9a.4.2 Ultrasound

Ultrasound can be used to visualise fluid-filled structures without the use of contrast medium, so it has the advantage of avoiding ionising radiation. It allows the visualisation of soft tissues such as the kidney, urethra, urethral sphincter, and bladder wall. The bony enclosure of the pelvis limits ultrasound imaging to the transabdominal, transvaginal, transrectal, and transperineal approaches. Each approach has its advantages and limitations. The type of probe and frequency of the ultrasound wave emitted (1–10 mHz) determines which tissues are seen and the quality of the image obtained. The higher the ultrasound frequency, the better the resolution of the image, although an increased frequency has reduced depth of penetration due to increased attenuation.

Vaginal and abdominal ultrasound is routinely used for measuring residual bladder volumes. A portable bladder scanner is useful for assessing residual volume and reduces the risk of urinary tract infection from catheterisation. Ultrasound also can image diverticulae, associated transitional cell carcinoma, and calculi.[32] Transabdominal imaging does not allow visualisation of the bladder neck as this is obstructed by the pubic symphysis. Transvaginal ultrasound allows imaging of the bladder and periurethral structures as the probe is closer to relevant structures. The urethral sphincter can be seen as an ovoid structure around the urethra. However, as the probe distorts the urethra, it is not good for assessing bladder neck movement during rises in intra-abdominal pressure.[33] Translabial and transrectal imaging has been suggested as an alternative as they do not distort vaginal and urethral anatomy. In neurological patients with decreased or absent anal sensation transrectal imaging may be of particular use, although with complete spinal cord transection insertion of the probe into the anal canal may alter sacral cord reflexes. The bladder neck and proximal urethra can be visualised with transrectal ultrasound during voiding, with images similar to those obtained with videocystourethrography[34].

Bladder wall thickness can be estimated in a reproducible manner using a perineal or transvaginal probe. This method has good sensitivity as a screening test for DO.[35]

Three-dimensional ultrasound can be used to assess a specific area allowing images to be obtained in unlimited number. The urethral sphincter can be defined by this approach and these images correlate well with cadaveric studies.[36]

Ultrasound imaging is not only limited to the LUT, but good quality images of the pelvic floor can be obtained (see also Chapter 12b). The levator ani hiatus and surface area are altered in women after vaginal delivery and in those with prolapse.[37–39]

9a.4.3 Magnetic Resonance Imaging (MRI)

Static, dynamic, and three-dimensional imaging MRI studies have enhanced the understanding of pelvic floor anatomy.[40–42] The advantages of MRI include no requirement for urethral catheterisation and detailed views of the pelvis at rest and during straining. However, it is expensive and at present limited to research use.

9a.4.4 Electromyography (EMG) and Nerve Conduction Studies

These specialised tests may be useful for investigation of the neurological patient and are often part of the routine diagnostic investigation as well as a research tool; it does not have a role in routine clinical practice.

There are two main techniques: surface EMG and single-fibre EMG. More myogenic damage and denervation is seen in patients with intrinsic sphincter deficiency compared to those with stress incontinence due to urethral hypermobility.[43]

9a.5 Conclusions

Specialist investigations in secondary care allow thorough evaluation of the incontinent patient to refine diagnosis and treatment. These investigations should be tailored appropriately to the individual patient and their limitations as well as potential values understood.

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9b Urinalysis

KJ Wilkinson

9b.1 Introduction

Urinalysis with test strips is a key component of a continence assessment[1,2] providing valuable information that can lead to a diagnosis or disprove a suspected condition. However, it is a procedure that frequently is omitted during continence assessment.[3] Laboratory testing for culture and sensitivity should be done only if abnormalities are detected.[4]

Ideally the urine sample to be tested should be the first voided sample of the morning, as this will be concentrated. The patient's privacy and cultural beliefs should be considered if assistance is required.[5] The sample should be collected in a sterile or clean container and tested immediately if possible or within 4 hours. False results can result from testing stale urine.[6]

It is recommended that a "multiproperty" test strip be used.[7] This must be in date and must have been correctly stored in an airtight container. The manufacturer's instructions should be followed and any electronic analysing equipment maintained and used correctly.

A variety of test strips are available on the market, each testing for a different combination of substances. When recording the result, the type of test strips used should be documented, as a "normal" or "negative" result is meaningless to another health professional reading the patient's notes.

9b.2 Interpreting the Results

Normal urine is straw coloured and clear.[5] Observation of the urine for cloudiness or debris or a strong odour can indicate presence of disease or infection. Please see Table 9b.1 for

Table 9b.1.

Substance	Normal reading	Common causes of positive result
Glucose	Negative	In patients with raised blood glucose, eg, new or poorly controlled diabetes, abnormal renal absorption. In pregnancy, glycosuria may be found in the absence of raised blood glucose.
Bilirubin	Negative	Liver injury or disease, viral or drug-induced hepatitis, biliary tract obstruction due to gallstones, or carcinoma of head of pancreas.
Ketones	Negative	Breakdown of fatty acids due to fasting, vomiting, low-carbohydrate diet, diabetic ketoacidosis.
Specific gravity	1.002–1.035	High value reading: dehydration, chronic renal failure
		Low value reading: high fluid intake, diabetes insipidus, chronic renal failure, hypercalcaemia, hyperkalaemia
Blood	Negative	Urinary tract disorders, eg, infection, trauma, stones, tumour, prostatic disease.
		Kidney disorders, eg, glomerulonephritis; polycystic kidneys, tumour. Trauma, eg, fractured pelvis.
Ph	4.5–8.0	Low value reading: acidaemia, eg, diabetic ketoacidosis, lactic acidaemia. Starvation, potassium depletion.
		High value reading: alkalaemia, eg, vomiting.
Protein (albumin)	Negative	Urinary tract infection, glomerulonephritis, pre- eclampsia in pregnancy, heart failure, fever.
Urobilirubin	Negative	Hepatitis, biliary tract obstruction, thalassaemias, sterilisation of colon, eg, due to Neomycin.
Nitrite	Negative	Urinary tract infection due to nitrite-producing organisms. A negative result does not exclude infection as some organisms are unable to convert nitrate to nitrite.
Leucocytes	Negative	Urinary tract inflammation, most commonly due to infection.

further details. Having followed the correct procedure for dip testing the urine, abnormal results must be reported and appropriate treatment or further investigations initiated.

9b.3 Conclusion

Simple urinalysis is a quick, inexpensive procedure that can exclude or diagnose a wide range of conditions and is an essential component of a continence assessment. Positive results always should be followed by appropriate further investigations.

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II Treatment and Management of Urinary Incontinence

10
Historical Perspective of Pelvic Floor Muscle Training

J Haslam

10.1 Introduction

Pelvic floor muscle training (PFMT) is recommended by noteworthy authorities as the basis for conservative management of stress urinary incontinence.[1–4] The American gynaecologist Arthur Kegel is often wrongly reported as the instigator of PFMT. He may have been the first to write at length about PFMT[5] associated with perineometry, but there were many before him who espoused the power of PFMT. Over the years there has been a pursuit of further knowledge, but often a "reinventing of the wheel"; the search continues for the "holy grail" of the optimum PFME regimen.

10.2 The Origins of Physiotherapy

Hippocrates, Galen and others, devised exercise regimens in the baths and gymnasia of ancient Greece and Rome.[6] However it was later in the 18th and 19th centuries with the development of new medical techniques and training, that exercises became the cornerstones of what is now known as *physiotherapy*.

In the early 19th century Per Henrik Ling, a Swedish fencing teacher, developed a system of gymnastics and massage that were further promoted and developed by his son Hjalmar Ling and others. These became known as Swedish remedial gymnastics and massage. In the 1860s they treated women with "gynaecological gymnastics," but unfortunately no written accounts have survived.

10.3 The Foundation of UK Physiotherapy

In 1886 Dame Rosalind Paget, a nursing sister/midwife at the London Hospital, became interested in Swedish massage, and after her own training, began to train others. She became a founding member of the Society of Masseuses, and in 1895 she became the first Chairman of Council. A Swedish remedial exercise examination was held in 1909; by 1913, practical experience had become a part of this training. Over the following decade massage and medical gymnastics associations were formed in Australia, Canada, and South Africa. In 1944, the organisation eventually became the Chartered Society of Physiotherapy.[6]

10.4 Early Childbirth Education

In 1912, Miss Minnie Randell, a nursing sister/midwife at St. Thomas' Hospital, became interested in the work of Ling. As Principal of the School of Massage and Medical Gymnastics, she instigated a system of exercises for women in the maternity ward.[7] Her exercises, which unfortunately were not specified, were developed in agreement with the thoughts of Dr. Kathleen Vaughan and "those used in well-known Swedish, American and German systems."[7]

Many matters relating to continence were first described by Miss Randell. She encouraged a squat position by using a "hygienic footstool" to resemble native closets, aiming for

full micturition and defaecation without strain.[7] The pictures of the positions (see Figures 10.1 and 10.2) for defaecation and the recommended footstools makes one realise that the defaecation techniques of the 1990s are based on advice given in the 1940s.

In 1948, she further wrote of using a crouching position to assist evacuation and the use of a low stool or upturned box acting as a footrest; perhaps she had evolved this thought after talking with a Dr. Vaughan who had worked in Kashmir and noticed the posture positions of the local women in childbirth and other activities. Miss Randell and Doctor Vaughan evolved a system of exercise in pregnancy including squatting exercises and pelvic and lumbar spine-mobilising exercises. They maintained that squatting with the knees apart with a hollowed and then rounded back would assist in stretching the pelvic floor and relieve constipation. Stretching exercises and the use of a lubricant such as lanoline or pure olive oil for massage of the perineum were taught and squatting introduced into activities of daily living to make constipation less likely.[7]

Meanwhile Margaret Morris, an ex-ballet dancer and pupil of Miss Randell, had also developed an exercise system aimed at pregnant women. In 1936, she wrote of the importance of the teaching of the conscious control of tension and relaxation of the pelvic floor muscles (PFM) together with the tightening and relaxing of the sphincters in positions that alternately facilitate tension and relaxation.[8] She also taught women to tighten the lower abdominal wall and brace the PFM on expiration. This early work seems to be a precursor to the pelvic stability work of more recent years.

Similarly, Minnie Randell wrote of PFMT associated with abdominal, gluteal, and adductor contractions.[7] More than 50 years later, Bø & Stien[9]

Figure 10.1. Posture hygienic stool. Reprinted from Randell M. In: Training for Childbirth, 2nd ed. p 40. Copyright 1941, with permission from Elsevier.

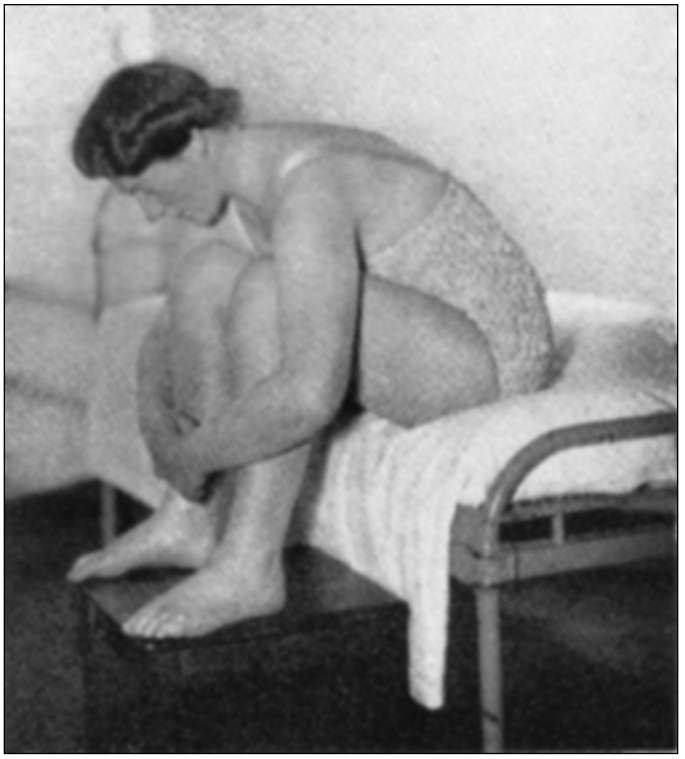

Figure 10.2. A useful position for micturition and defaecation. Reprinted from Randell M. In: Training for childbirth, 2nd ed. p 125. Copyright 1941, with permission from Elsevier.

have shown the interaction between these muscles and the PFM. Randell also mentioned using the PFM functionally by squeezing the front and back passages, drawing up inside the body, immediately before coughing, sneezing, or lifting heavy weights, to prevent the escape of urine.[10] This advice now has been validated by Miller et al.[11] and is known as "The Knack."

In1950, Grantly Dick Read, an obstetrician, proposed that women should be prepared for childbirth in both mind and body.[12] He worked in association with the physiotherapist Helen Heardman to improve the education and preparation of women for childbirth. She was instrumental in the formation of The Obstetric Physiotherapists Association in 1948, which went through several name changes to finally become the Association of Chartered Physiotherapists in Women's Health in 1994. She and Grantly Dick Read embarked on an American tour in 1947, teaching natural childbirth education to physicians, nurses, and obstetric childbirth nurses. In their visit to Yale she taught the exercises that she had devised. She described PFM in association with gluteal, low abdominal, and hip adductor contractions, suggesting daily exercise progressing to more upright positions eventually to standing with the toes turned out.[13] In the 1950s, physiotherapists increasingly used more direct language. Instructions to practice PFME both ante- and postnatally spoke of the need to "draw up as though trying not to pass water or have a bowel action."[14]

10.5 The 21ST Century

The use of PFME during the childbearing years has been substantiated.[15–17] But over time many eminent physiotherapists have proposed different exercise regimens[18]; as yet the literature does not support the use of any specific regimen.

Could it be that our skills of empathy, motivation, and listening, which are impossible to quantify, are equally important? The equipment at our behest now ranges from ever-more complex

biofeedback to "fun" equipment such as exercise balls and mini-trampolines. What is essential, what is frippery, and what do we really need? Specialist continence nurses and physiotherapists have a responsibility to continue to validate, audit, research and document their clinical techniques; this is of particular importance in the present economic climate. Research must continue to search for truths, but we have the responsibility to acknowledge the influence of the past and ensure that future research is of relevance and high quality.

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11a
Pelvic Floor Muscle Exercise in the Treatment of Urinary Incontinence

J Haslam

11a.1 Introduction

Pelvic floor muscle (PFM) reeducation is an effective, lowrisk intervention that can significantly reduce incontinence in varied populations and should be considered prior to other interventions.[1–4] The effectiveness of pelvic floor muscle exercises (PFME) has been demonstrated [5–7] with follow up at 5 years[8,9] and 6 years.[10] However at 15 years the results are disappointing.[11] Systematic reviews of randomised controlled trials (RCT) on the conservative treatment of stress urinary incontinence (SUI) support PFME as being effective in reducing the symptoms of SUI[12,13] and also urge and mixed incontinence.[13] This corresponds well with the findings that vaginal and urethral pressures correlate significantly with pelvic floor muscle contractions[14] and that PFME are useful in both the management of SUI and symptoms of urgency.

For PFME to be effective, a person must be able to contract the correct muscles and comply with a specific exercise regimen. Age, clinical severity, urethral support, or urethral profilometry do not determine whether or not a woman will be able to perform an adequate PFM contraction,[14] vaginal assessment of a women is necessary to evaluate this[15,16] (see Chapter 7). Male asessment is considered in Chapter 21.

11a.2 Exercise Physiology

A knowledge of both muscle physiology and exercise theory is necessary to gain understanding and ability to prescribe appropriate PFME. The number of motor units recruited depends on the effort exerted; the greater the effort, the greater the frequency of excitation and the greater the number of motor units recruited. In asymptomatic women the PFM are composed of approximately 33% fast fibres and 67% slow fibres.[17] The muscle fibre type is determined by the nerve supplying it. Slow oxidative muscle fibres (tonic, type 1) sustain activity, whereas fast glycolytic muscle fibres (phasic, type 11) are involved in bursts of activity. According to Henneman's size principle of recruitment, when a striated muscle contracts, slow motor units are initially recruited; as greater effort and load are placed on the muscle, the fast motor units are recruited.[18]

11a.3 Principles of Training

The main factors to be considered in any muscle training include overload, specificity, maintenance, and reversibility.[19] However, there are many differences in application between training an athlete and a person with SUI.

Table 11a.1. Muscle training considerations.

Strength	The maximum force or tension that a muscle can generate
Power	The rate at which the work can be done. The ratio betwee the maximum force and the time taken to achieve that force
Endurance	The length of time over which a contraction can be main tained or repeated
Repetitions	The number of times that contractions of equal force can be repeated
Fatigue	Failure to maintain the required or expected force

11a.3.1 Overload

Overload refers to muscle performance including both strength and endurance. To achieve this it is necessary to work the muscles harder than usual with concentrated effort. The initial training period must ensure that the correct pattern of movement is being used, only then to be followed by encouragement of maximum voluntary contractions (MVC) for overload. Endurance can be further increased by repeating the MVC as many times as possible with a short rest (approximately 4 seconds) between each MVC. When able to perform ten MVC each lasting 10 seconds, the rest time can be reduced. Biofeedback (see Chapter 12a) can help a patient to see the results of their effort and encourage an MVC.

An initial assessment is essential to establish the starting point in the exercise programme; this is the only way to determine an appropriate "exercise prescription" to overload the muscles. In order to maintain the overload, there must be ongoing assessment and the exercise programme adjusted apppropriately.

Skeletal muscle has been known over a period of 6 months training to improve 25–50% in strength[21], therefore a minimum period of 15–20 weeks PFM exercise has been recommended.[20] It is believed that effects of the first 6–8 weeks are due to neural adaptation[22] (increased frequency of excitation and more effective motor units); muscle hypertrophy takes longer and continues over many months.[22]

11a.3.2 Specificity

Muscle specificity training of the PFM, requires exercises "specific" to functional activities. Although muscles work in patterns of movement, it is essential to ensure that the pattern of movement is neither detrimental nor masks activity in the target group. When aiming to strengthen the PFM, it has been shown in a small study[23] that contraction of the transversus abdominis (TrA) results in a PFM contraction in healthy subjects. However, gross contraction of the abdominals, especially the rectus abdominus, results in raised intra-abdominal pressure and is inappropriate when re-educating the PFM. Pelvic floor muscle stability and trunk muscle co-activation is considered in greater depth in Chapter 11c.

Miller et al. have shown that by teaching women to contract the PFM before and during a cough (the knack), stress urinary loss can be reduced by an average of 73.3% after 1 week of practice.[24] This can be taught and monitored during a digital examination. The need to contract the PFM during any activity that puts the PFM under any additional strain that causes urine loss should be emphasised. The aim is to acquire learned reflex activity; therefore this must be practised many times.

11a.3.3 Maintenance and Reversibility

Muscle training can succeed in improving strength, power, and endurance. But in order to maintain the improvement, the exercise needs to be continued on a regular basis.[21] A reduced exercise programme can maintain moderate levels of strength and endurance[25]; but if training ceases, the oxidative capacity of the muscle diminishes in 4–6 weeks,[26] the endurance ability declining more quickly than the ability to exert maximal power.[27] It is assumed, but not proven, that the same applies to the PFM. It may be that if sufficient PFM strength is gained to relieve SUI, then long-term incorporation of PFM contractions into activities of daily living may be enough to maintain continence.

11a.4 Exercise Regimen

It has been suggested that the recommended exercise regimen to develop strength in skeletal muscle is to practise three to four sets of 8–12 high resistance, slow velocity contractions three times a week[21]; however, it is difficult to give high resistance to the PFM. Nevertheless, regard should be paid to enough effort, of suitable duration, and adequate frequency of exercise over a sufficient length of time. At present the minimum successsful regimen reported in any RCT of PFME is eight PFM contractions repeated three times per day.[5] This recently has been recommended by NICE as the minimum number of contractions to aim for.[4] However, it must be noted that the women in this 6-month study also attended a 45-minute PFM exercise class each week.[5]

A review of 16 papers[28] concerned with strength training of the PFM found variability in

frequency of exercise, length of contractions, and the total period of exercise. However, all studies agreed that subjects should receive individual instructions, vaginal palpation, feedback, and close follow up. The self-reported cure and success rates varied between 17 and 84%.[28]

The PFM not only should be able to generate force, but also to work in a coordinated fashion at the time of need. There is a greater chance of success by teaching PFM awareness, an appropriate exercise regimen, and the ability to contract the PFM when needed. An exercise diary can be utilised both as a motivator and to provide accurate information as to when the patient has found the best times for exercise (see Appendix 3).

11a.4.1 Response to PFME

Many studies have shown the effectiveness of PFME related to childbirth.[29–31] Although bladder function and striated muscle alter with age, this does not mean that the elderly are incapable of improving their PFM function. A holistic assessment should be made including mobility, any polypharmacy, and behavioural considerations.

Researchers have attempted to identify those patients most likely to succeed with treatment. It has been shown that the best reponders to intensive PFME were significantly older, had a longer history of SUI, higher body mass index, stronger PFM, and more motivated to therapy at the commencement of the study.[32] It also has been shown that support by the physiotherapist, together with patient motivation and compliance, are necessary for good results.[33] However, there is little evidence to identify which PFME regimen is the most effective as studies have used different parameters.[12] Until there is a RCT comparing different exercise programmes, no conclusions can be made regarding the most effective exercise protocol; meanwhile, the NICE recommendations should be considered.[4]

11a.4.2 Teaching PFME

Patient education on the anatomy and working of the PFM, using diagrams and models, is necessary before embarking on PFM reeducation. If vaginal assessment is not possible, observation of an inward movement of the perineal body on attempted PFM contraction denotes at least a Grade 3 modified Oxford scale contraction (see Chapter 7). Any bulging indicates an increase in intra-abdominal pressure with insufficient PFM activity. If a PFM contraction is not observed, pressure on the perineal body with the instruction to "try and lift away" may be helpful. During digital assessment extra encouragement can be given by using such phrases as "squeeze and pull in" or "squeeze and lift" or "try and pull your tail bone forward" or "try and stop me from removing my examining finger." The aim is to be able to concentrate on a ventrocephalic movement to increase pressure at the bladder neck.

11a.4.3 Practical Application of PFME

11a.4.3.1 Grade 0–2

If there is little or no discernible contraction, the examiner may be able to elicit a better contraction by hooking the index finger over the PFM and stretching it in a posterolateral direction. If the vagina is capacious enough for two digits to be introduced in an '"eastwest" direction, then both sides can be put on the stretch simultaneously to stimulate the stretch receptors in the muscle. If this together with verbal encouragement results in an improved contraction, the patient is assessed for muscle strength, duration of contraction, ability to repeat that contraction, and the number of fast contractions possible. If the contraction is still Grade 2 or less, neuromuscular electrical stimulation (NMES) or biofeedback should be considered.

For muscles of a low grade the rest phase should be 4 sec or the same as the length of the contraction, whichever is the greater. People with less ability may find it easier to practise their PFME while lying down, thus eliminating the effects of gravity; others find it easier to exercise with a pillow under their hips, thus having a gravity-assisted exercise. As ability increases, the aim must be to assume more functional positions.

11a.4.3.2 Grades 3 and Over

A baseline assessment will determine the specific exercise programme for the individual (see Chapter 7). Although initially PFME may be easiest in sitting, with a neutral lumbar spine and

Figure 11a.1. A 50% contraction with intermittent fast contractions.

forearms resting on knees (stimulating the dermatome of S4), PFME must be developed functionally and suitable for daily living activities. Objectives need to be given involving increased length and number of contractions. Sustained contractions will increase tonic activity of the PFM and fast contractions are necessary to gain a quick response to utilise "the knack." Submaximal contractions at 50% of maximal effort, sustained for longer periods of time accompanying daily activities, should also be practised. Functional activity such as holding a submaximal contraction for a certain number of steps while walking may be practised. Another useful technique is to attempt a submaximal contraction and superimpose fast, strong, short contractions within the main contraction (see Figure 11a.1). This could be thought to further harness the recruitment of slow to fast fibres as described by Henneman.[18] Any regimens of PFME are patient specific according to ability.

11a.4.4 Pelvic Floor Classes

Many therapists now find it beneficial to introduce class work into PFM training. The session can be used not only for specific PFM training and trunk muscle coactivation (see Chapter 11c), but also to impart information, introduce more "fun" activities such as exercise balls (see Chapter 13) and for comradeship to develop among the women. It has been shown that exercising intensively in groups once per week for 6 months is more beneficial than exercising alone.[34] If classes can be incorporated as well as individual assessment, the patient will receive increased motivational input, peer support, and an opportunity to participate in more generalised pelvic and trunk muscle training.

11a.5 Summary

At present there is no way of predicting who will succeed in reducing symptoms of SUI; even those patients who may not at first seem to be ideal candidates for conservative management should be given the opportunity of PFM reeducation, provided that their motivation is high and compliance with treatment likely. If the patient is prepared to practise assiduously for 3 to 6 months, it can be considered to be a fair trial of conservative management. Continuing research including biomechanical analyses of the overall mechanism with targeted research into the interaction between the relevant muscles, nerves, and ligaments should further the knowledge base in effective therapy.[35]

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11b
Advanced Manual Therapy for the Pelvic Floor

MM Whelan

11b.1 Introduction

On vaginal examination, areas of reduced/nil PFM activity are often detected on palpation during a voluntary contraction. This used to be thought to be due to denervation, but may be due to muscle inhibition. If a muscle (or part of a muscle) is inhibited (not contracting) – due to pain or injury – it will not be recruited during voluntary or involuntary contractions. In addition, some muscles respond to pain by being 'overactive' and non-relaxing. Consequently, these inhibited/overactive muscle fibres, along with surrounding fascia, need to be 'released and facilitated' prior to muscle training. See below.

The terms anterior and superficial pelvic floor are often used interchangeably but they do not always refer to the same structures. This is also the case for posterior and deep pelvic floor. The anterior/superficial pelvic floor is treated in the 'vertical plane' and the deep or posterior pelvic floor is treated in the 'horizontal plane'. These planes of examination are further described in Chapter 7. The author utilizes the following techniques to release and facilitate the PFM before strengthening.

11b.2 Manual Therapy Techniques

11b.2.1 Pubovisceralis Stretch on the Vertical Plane

The therapist's flexed index finger is inserted into the vagina and rests with the finger pad on the posterior vaginal wall. The therapist stretches in a downward motion repeating the action from 8 o'clock posteriorly to 4 o'clock . The repetitions and length of treatment is decided by the resistance of the tissue; the author has found that a duration of 20 -30 seconds per stretch is effective in feeling the tissue release and that the patient will tolerate this treatment well for 5–10 minutes. The indications for this technique are primarily overactive pelvic floor (OAPF) and where there is a stronger anterior/superficial pelvic floor in contrast to the inhibited posterior/deep pelvic floor

11b.2.2 Pubovisceralis Stretch on the Horizontal Plane

This stretch starts at the anterior attachments of the pubovisceralis extending to the posterior attachments, it starts in the vertical plane and extends to the horizontal plane. The finger position is initially as in the technique above but the stretch is now posterior in direction; the finger will gradually become more extended as it follows the pubococcygeus and puborectalis posteriorly (see Figure 11b.1). The patient may report pain where trigger points are present and may often get the urge to defecate, which should be explained before treatment. This technique is indicated for defecation dysfunction, OAPF and in urinary incontinence where a muscle imbalance exists.

11b.2.3 Facilitation of Iliococcygeus and the Posterolateral Wall

The posterolateral wall is commonly inhibited. The posterolateral wall is on the horizontal plane of examination; it consists of the lateral fibres of pubococcygeus and the iliococcygeus muscle. The iliococcygeus muscle is important for the shelf support of the pelvic organs[1,2], therefore inhibition may mean

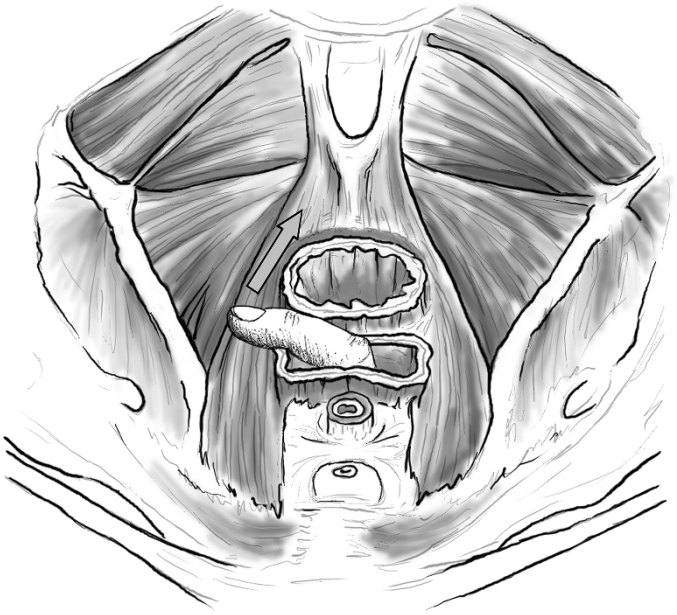

Figure 11b.1. Pubovisceralis stretch technique.

the pelvic organs have a lower resting position. In addition, if a muscle is inhibited the surrounding muscles may also be inhibited[3,4], therefore facilitation will help rehabilitation. Perineal realtime ultrasound has demonstrated that the direction of movement of the contracting PFM may change after facilitation of this muscle from more vaginal origin towards the base of the bladder to a movement from the anorectal junction towards the urethrovesical junction.

The therapist's finger must be fully extended on to the posterolateral wall at 10 o'clock and

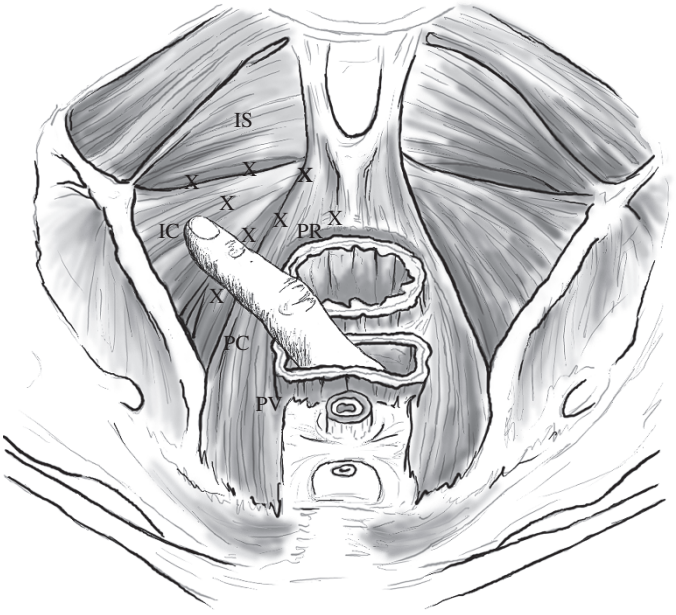

Figure 11b.2. Facilitation of iliococcygeus and posterolateral wall.

2 o'clock on the horizontal clock (see Figure 11b.2). If treatment is required the muscle will feel fibrous and almost tendinous to touch. Trigger points are identified by downward pressure of the palpating finger pad; once a painful point is identified this downward pressure can be held for 6–12 seconds[4] until the pain gradually eases, then repeated along the muscle until all the painful points have been treated and the muscle feels more flexible. It may be helpful to apply the same technique to the ischiococcygeus muscle where injury to the coccyx has occurred.

11b.3 Self Help Techniques

11b.3.1 The Release : 'Sniff , Flop and Drop'

Manual therapy alone is insufficient; the patient is also required to exercise to maintain the resting length of the muscle. The following technique once mastered has been found effective in achieving and maintaining muscle length. There are three components, which the patient must learn to coordinate (see Box 11b.1).

It can be very difficult to co-ordinate all three movements and it may be a good idea to combine two of the three components at first. Common mistakes are: over sniffing therefore making the breathing too dominant, using the ribs and upper abdomen to force an abdominal release, bearing down causing descent of the organs.

Box 11b.1

Instructions for 'sniff flop and drop'

The patient *sniffs in through the nose* and is instructed to bypass the chest on the in breath so that no chest lifting occurs.

The patient *flops or swells out the lower stomach* on the in breath into the palpating hand without forcing, bracing or using the upper abdomen to push out. The patient's hand should be resting on or just above the pubic bone.

The patient *simultaneously drops or releases the pelvic floor* by opening up at the back passage (see Figures 11b.3 and 11b.4).

11b.3.2 Maximal Activation to Maximal Release

The patient only progresses to activation of the PFM once they have learned how to maximally release. The patient contracts transversus abdominis (TrA) , then contracts the PFM from the back passage lifting upward and forward as if to

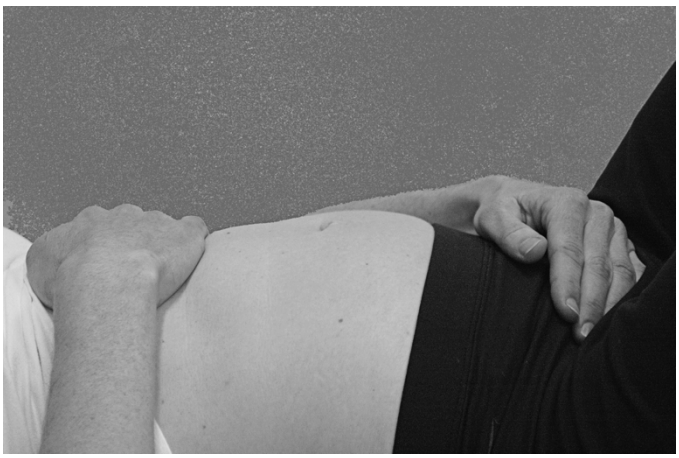

Figure 11b.3. Correct release technique.

Figure 11b.4 Incorrect release, use of ribs to force.

meet the contracting TrA. The patient then breathes in and all the way out while still holding the TrA and back passage in. At end of the out breath an extra squeeze helps to make sure that the contraction has not been lost before the release. To release as described above sniff in, flop out the lower stomach and drop the back passage.

11b.4 Home Exercise Programme

Exercising twice a day is sufficient. The patient is instructed to do 15 repetitions of the 'Sniff, Flop and Drop' and 15 repetitions of the maximal activation to maximal release. However these must be 'quality exercises' they take approximately 10 minutes to ensure concentration and coordination. Initially the exercises are done in crook or side lying to maximize the awareness by eliminating gravity, organ descent and postural control. The patient is told that their exercise session is dependant on performance and that 100% concentration is required in order to get the desired results.

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11c
Pelvic Floor Stability and Trunk Muscle Coactivation

RC Jones

11c.1 Introduction

The pelvic floor muscles (PFM) form the base of the abdominal cavity and as such contract not only to maintain continence, but to augment intra-abdominal pressure (IAP).[1] Co-coordinated co-activation of the PFM is therefore necessary in order to balance the functional demands of continence and lumbo-pelvic stability. The PFM contribute to lumbo-pelvic stability by stiffening the sacro-iliac joints[2] and indirectly the lumbar spine through contributing to rises in intra-abdominal pressure (IAP).[1–3]

11c.2 Lumbo Pelvic Cylinder

The PFM form the base of a multi-structural unit with the respiratory diaphragm forming the top and Transversus abdominis (TrA) the sides. The spinal column is part of this lumbo-pelvic cylinder (LPC) and runs through the middle, supported posteriorly by segmental attachments of lumbar multifidus and anteriorly by segmental attachments of psoas. Muscles of the LPC work at low levels at all times and increase their activity when the central nervous system can predict timing of increased load, such as occurs in coughing, lifting, or limb movements.[4–6] It is known that in the presence of postural changes, respiratory demands, and pain, trunk muscle activity is altered.[6,7] Yet in all tasks that increase IAP, the PFM must contribute to ensure urethral and anal closure before the rise in IAP if continence is to be maintained and consequently, in normals there is an increase in urethral pressure prior to an increase in intra-abdominal pressure[5].

11c.3 Stress Urinary Incontinence

Clearly then, attempting to activate the PFM in isolation is non physiological.[8–11] Yet it appears that augmenting a PFM rehabilitation programme with an abdominal muscle training programme does not improve the treatment outcome in women with stress urinary incontinence (SUI).[12] Therefore the usefulness of our current knowledge regarding co-activation of the PFM with, in particular the TrA muscle, is yet to be elucidated under research conditions. However, it is a clinical and researched observation that women can facilitate a PFM contraction by use of the TrA (see Figure 11c.1)[9] and other muscles of the LPC or in close connection to it.[13] It is suggested by the author that facilitation strategies may be of particular clinical value when a patient is unable to voluntarily activate a PFM contraction, or if the PFM contraction is unable to visibly support the urethra, or increase the intra-urethral pressure.[14]

In a sample size of 31 women, 22 continent and 9 with SUI, all produced a reflex co-activation of the PFM during a voluntary TrA contraction, verified by 2D dynamic ultrasound and palpation. Women with SUI produced statistically significantly less displacement of the ano-rectal junction than the healthy controls ($P<10.01$)9 (see Figure 11c.1).

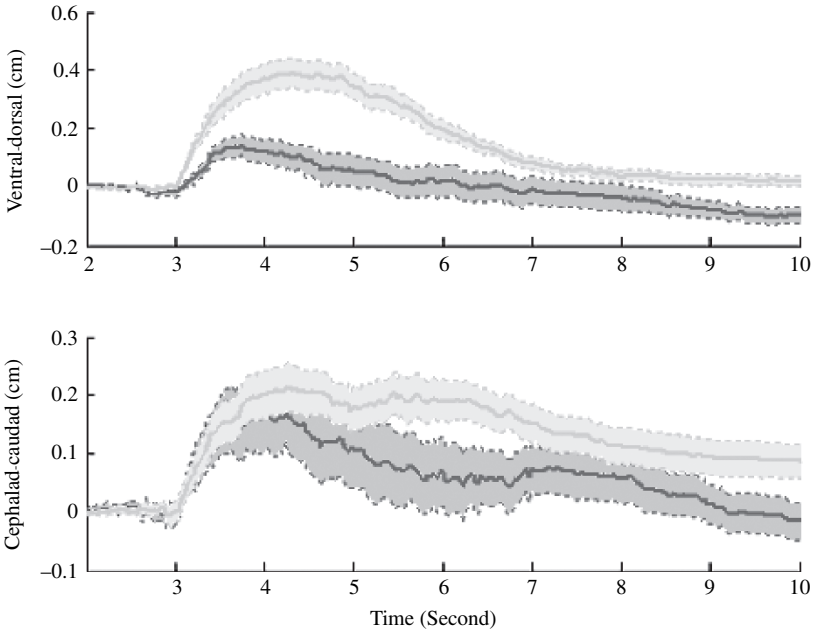


Figure 11c.1. Two-dimensional dynamic ultrasound showing the mean and standard error (STE) of the movement of anorectal junction during a voluntary TrA contraction in a ventral-dorsal and cephalad-caudad direction, indicating PFM co-contraction. Upper = continent, lower = incontinent women, STE is marked by the shaded area.

Box 11c.1

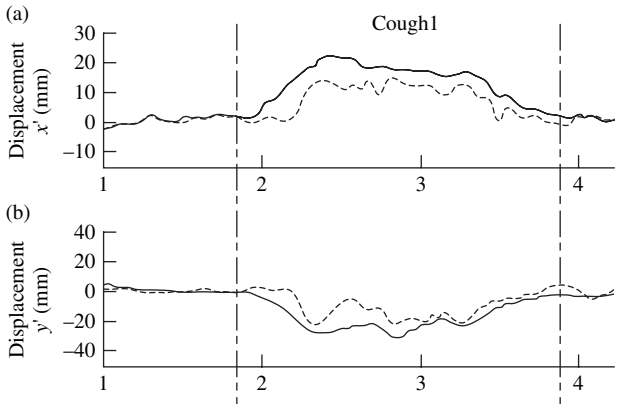
Case Study

A 34 year old woman, 32 weeks pregnant with twins was complaining of urinary leakage during coughing, sneezing and any sudden movement. She had had symptoms of stress urinary incontinence (SUI) following the birth of her first child; however, during her second pregnancy, her symptoms were rapidly deteriorating. PFM strengthening and the knack had been of little benefit. On examination she had anterior vaginal wall laxity and a flicker of a PFM contraction. Using perineal ultrasound it was clear that a voluntary contraction of her PFM was unable to support the urethra, and performing the knack did not limit the degree of urethral rotation or descent during a cough. However, supplementing a PFM with a TrA contraction during a cough visibly reduced bladder neck descent by over 1cm (see Figures 11c.2a, 11c.2b) and was sufficient to control urinary leakage.

11c.4 Technique

In order to easily facilitate a TrA contraction, it appears important for the patient to:

1. Adopt a starting position that is pain free, comfortable and relaxed for them. Suitable positions are 4 point kneeling (see Figure 11c.3), supine crook, side or prone lying, standing or sitting, often with support, but ideally in a neutral position. (A neutral position is one where the patient is somewhere in *their* mid range position of lumbar spinal flexion/extension)
2. Visualise their deep abdominal muscles as a corset that wraps around their abdomen.
3. Place one hand above their umbilicus and one below, and SLOWLY draw in their lower abdomen, as if they were pulling in their stomach away from their knicker elastic.
4. Hold the contraction, whilst breathing normally for as long as they are able. Frequently I will ask that they aim to hold the contraction for 10 seconds,



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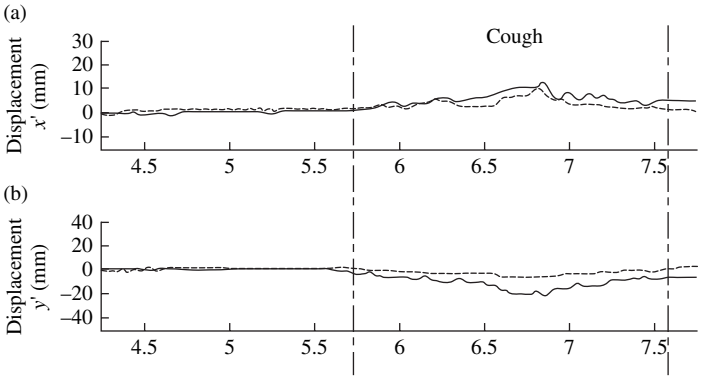

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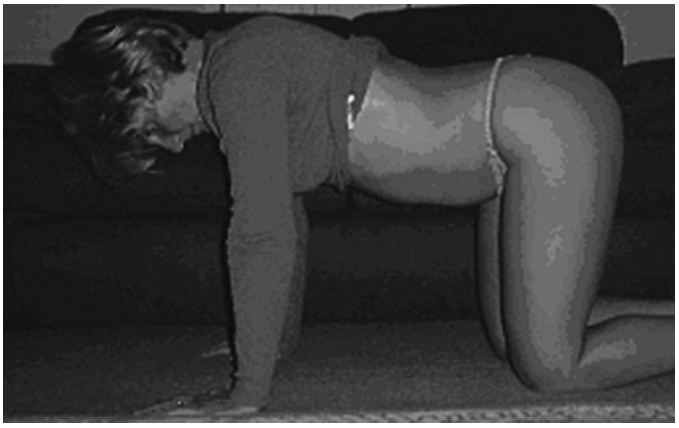
Figure 11c.2. (a) Displacement of the urethra (solid line) and ano-rectal junction (dashed line) (in mm) during a cough in a woman with SUI (b) Reduced displacement of the urethra and ano-rectal junction of the same woman during a cough with PFM and TrA pre contraction.

repeating it 10 times, watching for substitution strategies (see Table 11c.1).

The therapist should encourage the patient to work at much less than their maximum perceived effort of contraction. It should be of just sufficient magnitude that the patient can feel their deep abdominal muscles work and can breathe normally. Many patients find it difficult to coordinate breathing with a voluntary contraction of their TrA. It is the author's clinical opinion that the ability to sustain a contraction and breathe normally is an essential part of successful rehabilitation. It is not crucial for a patient to feel a simultaneous contraction in their PFM, but many women report that they do. If a woman has been previously unsuccessful in becoming continent by contracting their PFM, focusing on the TrA can also be of positive psychological benefit.[15]

11c.5 Real Time Ultrasound

In order to verify a TrA contraction, the therapist can either confirm by palpation or real time ultrasound (see Chapter 12b). Using ultrasound the transducer head is placed transversely, midway between the iliac crest and ribcage, approximately 10cm either side of the umbilicus. Using her fingers, the therapist palpates immediately inferior and slightly medial to the anterior superior iliac crests. As the patient draws her lower abdomen in, palpate for a symmetrical tensioning, without excessive bulging, underneath the palpating fingertips.



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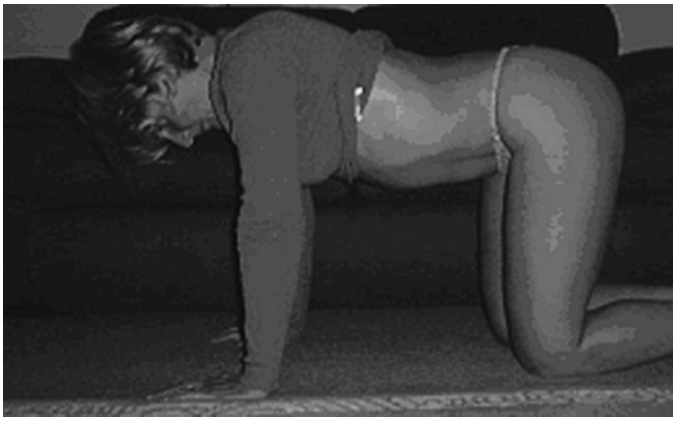

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Figure 11c.3. (a) Abdomen relaxed in 4 point kneeling. (b) Correct activation of TrA in 4 point kneeling.

Table 11c.1. Substitution strategies with possible solutions.

Common Substitution Strategies	Possible Solutions
Movement of the trunk or pelvis out of a neutral position	Encourage the patient to work less hard and place a small, rolled-up towel or pressure biofeedback unit (Chatenooga, Australia) in the small of their back
Asymmetry of contraction	Encourage the patient to concentrate and visualise drawing the one side that may not be working into the midline
Breath holding, apical breathing	Teach diaphragmatic breathing, placing a hand above the umbilicus, feeling the rise of the abdomen as they breathe in
Drawing in of the upper abdominal wall	Encourage the patient to draw their lower abdomen in by concentrating on or feeling the area below their umbilicus
Lateral flaring or bulging of the waist	Encourage the patient to try less hard!
Excessive superior, inferior, or lateral movement of the umbilicus	This often is suggestive of excessive oblique abdominal muscle activity; once again, encourage the patient to reduce their effort and stop as soon as they feel a slight tensioning of their lower abdominal muscles

11c.6 Clinical Practice

It is a clinical observation that those women who have little proprioceptive awareness of their PFM can start to 'feel' their PFM after a short time (less than a week) of repeated (2–3 times a day) TrA contractions. As soon as the therapist is confident that the patient can activate their TrA and PFM, they should be encouraged to integrate this contraction into their daily activities. The timing of contraction is probably of greatest importance[16] and it is suggested that alongside improving the endurance of co-activation, the patient learns to co-activate prior to any activity that raises IAP. It is suggested that this is a progressive programme, learning to activate in lower load activities, before progressing to tasks with greater load or speed, such as heavy lifting, jumping or running.

It is known that there are different patterns of muscle activation in different postures[17] and certainly if a patient tends to adopt extreme sway or kyphotic lordotic postures, then correcting this will automatically facilitate the TrA (and consequentially the PFM) see Figures 11c. 4a, 11c.4b.

Patients (and therapists!) often complain that compliance with an exercise programme can be very challenging. The more ways that the therapist can encourage the patient to exercise, whilst not seeing it as exercise, the more likely the programme is able to help, such as standing tall and breathing normally whilst waiting in a shopping queue, or sitting upright instead of slouching.

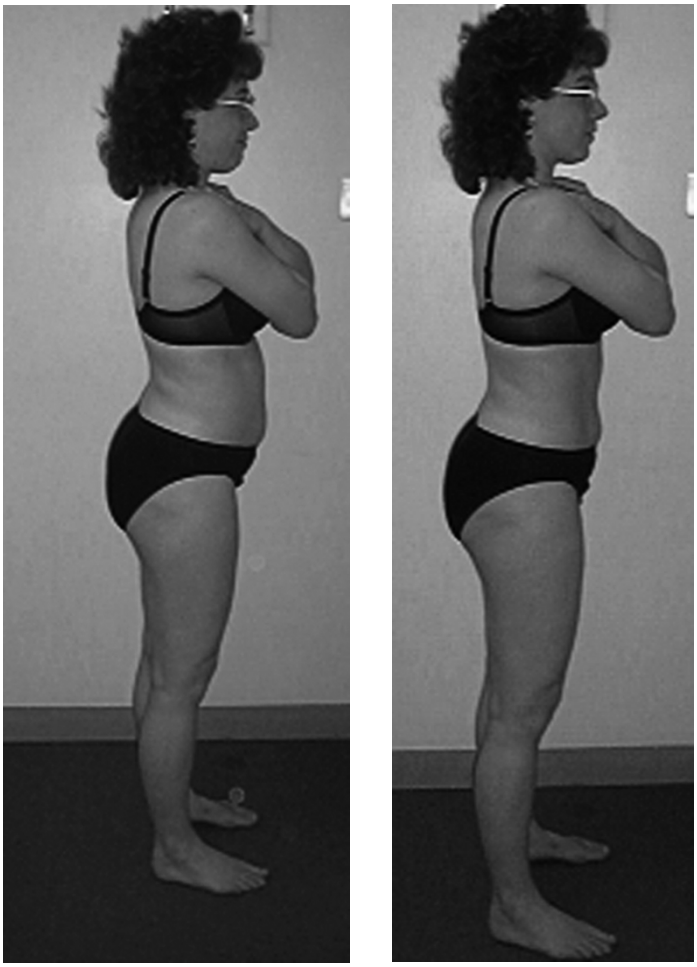

Figure 11c.4 (a) Relaxed standing lordotic posture with little automatic contraction of the TrA muscle. (b) When the standing posture is corrected, there is automatic activation of the TrA and consequentially the PFM.

It is very important to remember that a TrA contraction in conservative rehabilitation of SUI is used to facilitate or enhance a PFM contraction. It is not meant as a substitution for PFM exercise, just another tool that the therapist can use for some women with SUI, especially those who have little awareness of their PFM.

11c.7 Lumbo Pelvic Pain

There is emerging evidence that links lumbopelvic pain to PFM dysfunction[18,19] suggesting that there maybe a common physiological mechanism between these disorders that warrants closer inspection. There is still much to learn regarding normal muscle function and physiology and so much more regarding the most effective way to rehabilitate dysfunction. The illustrations provided are far from supplying a fully exhaustive list.

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12a Biofeedback

J Haslam

12a.1 Introduction

Biofeedback was first used in the mid-20th century by psychologists interested in operant conditioning. They included any techniques used to enable conscious regulation of a body function by mental control. Originally it was thought to be purely a reward system but later was thought to be more important for skills acquisition. About the same time, Kegel described his use of pneumatic apparatus used intravaginally to exercise the "birth canal muscles"[1]; he may be considered the first person to use pressure biofeedback to rehabilitate the pelvic floor muscles (PFM).

Physiotherapists in the UK became interested in the use of perineometers in the 1970s and then in more sophisticated equipment in the late 1980s. A range of "biofeedback tools" is shown in Box 12a.1.

Box 12a.1

Biofeedback tools

- Digital vaginal palpation. Therapist gives 'verbal biofeedback' on status of PFM contraction
- Digital self-palpation
- Digital vaginal palpation with pressure to increase proprioception to identify PFM
- Hand-held mirror
- Educator™
- Vaginal cones
- Manometry
- Electromyography (EMG)
- Real-time ultrasound (see Chapter 12b)
- Dynamometry (see Chapter 7)

12a.2 Definition and Use

The International Continence Society (ICS) has defined biofeedback as "the technique by which information about a normally unconscious physiologic process is presented to the patient and/or therapist as a visual, auditory or tactile signal."[2] In other words, biofeedback aims to consciously adjust the control of an activity by increasing knowledge of that activity.

Biofeedback not only improves awareness of PFM activity, but also provides more interest and challenge in what may be considered quite tedious exercise. However, many studies of stress urinary incontinence comparing biofeedback with PFM training (PFMT) versus PFMT alone have demonstrated that biofeedback offers no additional improvement.[3–6] In view of this the National Institute for Health and Clinical Excellence (NICE) has stated that perineometry or EMG should **not be routinely** used as part of PFM training.[7]

However, clinical practice takes "all comers," not just those according to a research study protocol. The well-trained therapist will find that biofeedback may not be perceived as always being essential, but there is no doubt that it is a useful method of enthusing and gaining compliance in a patient, and hence having successful outcomes.

12a.2.1 Digital Biofeedback

Many women are unsure how to contract their PFM using verbal and/or written instructions alone.[8,9] PFM palpation and appropriate pressure

and stretch with vocal correction and encouragement can all heighten the patient's awareness.

12a.2.2 Theoretical Considerations

Specific PFM biofeedback equipment using either manometry or EMG with a vaginal/anal sensor (probe) may be used to monitor activity of the PFM during a voluntary contraction, by visual or auditory feedback. However, the position of the patient, probe location, and sensitivity of the equipment can all affect the readings; consequently, care must be taken to ensure that any subsequent biofeedback sessions reproduce these factors. Only then can the results demonstrate actual change and increase patient motivation in their performance. Some considerations before using any apparatus for patient assessment and treatment are shown in Box 12a.2.

Biofeedback monitors/screens should be adjustable in relation to scale, duration, and goal lines. This will enable even weak PFM contractions to be monitored and encourage patients to maximise their efforts. Untrained muscle may have a sluggish response to both the instruction to contract and relax (see Figure 12a.1), whereas well-trained muscle can recruit motor units in an efficient manner as shown by manometry in Figure 12a.2. The use of predetermined templates for either contraction or relaxation (see Section 3 on pelvic pain) can assist in gaining patient interest and compliance.

Other extraneous factors that may affect performance include the time of day, general fatigue, and hormonal status. The aim of reeducation is not only to have a greater ability to generate force with the PFM, but also to have better functional ability to counterbrace whenever there is need. Using EMG, patients with stress urinary incontinence (SUI) can practice a pretimed contraction prior to coughing while observing PFM activity on the screen.

Box 12a.2

Practical Considerations

- Therapy must be explained and valid consent documented
- Appropriate vaginal/anorectal assessment must take place prior to the insertion of any probe
- Patient should be comfortable and able to observe the monitor
- Pressure probes should be positioned deflated, then inflated to patient comfort, and maintained/held in position during any pressure recordings
- Position of the patient and probe is documented
- Breath holding is monitored and corrected if necessary
- Activity of other muscle groups is monitored; further information regarding muscle coactivation can be seen in Chapters 11c and 12b
- Future biofeedback sessions should be carried out under the same conditions to allow comparison with previous results
- Pressure probe design limits the positions that can be used for biofeedback
- Many surface EMG vaginal/anal sensors can be used in functional positions, for example, walking
- All equipment must be CE marked and therapists must comply with manufacturer's instructions concerning single use, single-patient use or multiple-patient use (and disinfection)
- An agreed dated infection control policy must be in place (see Chapter 36)

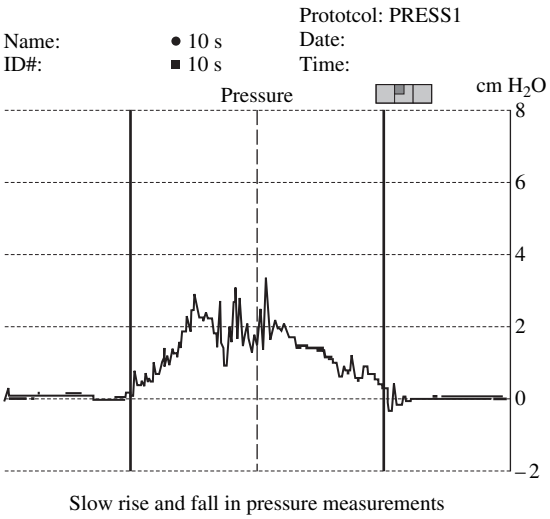

Figure 12a.1. The sluggish response of untrained muscle.

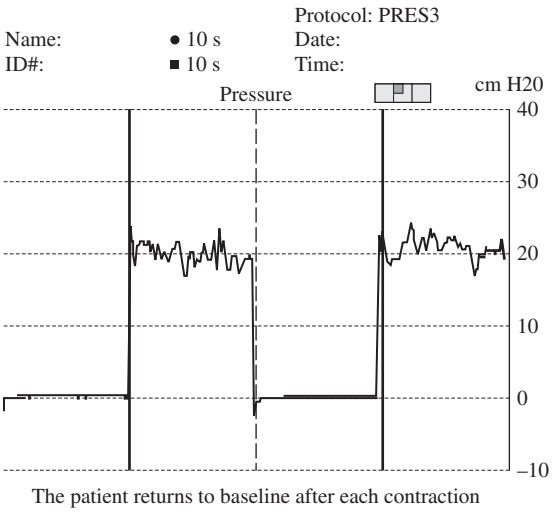

Figure 12a.2. The efficient recruitment of motor units in well trained muscle.

12a.3 Electromyography

Electromyography (EMG) utilizes feedback from the electrical potentials generated by the depolarization of muscle, which are then amplified, rectified, and smoothed, giving the picture visible on screen (See Figures 12a.3 and 12a.4).

EMG is defined as the recording of muscle bioelectrical activity,[10] measured in microvolts, which correlate to motor unit activity. It does not measure the muscle contractility itself but the electrical correlate of the muscle contraction. It is an electrical and not a kinetic measurement; in other words, EMG is an indicator of the physiological activity and not the actual activity itself. PFM training aims to improve the synchronicity of motor unit activity. A patient will not necessarily increase their microvolt output, but should aim to improve functionality by gaining better control of the coordination and timing of contractions. Biofeedback training should not just be concerned with a maximum voluntary contraction (MVC) but also working at a lower level (50%) with longer sustained contractions (see Chapter 11). The EMG feedback will enable the patient to visualise the improvement in their PFM function.

Internal EMG electrodes (sensors) have been developed to produce light-weight comfortable devices that stay in place allowing monitoring of PFM activity during functional activities, for example, walking, coughing. However, no such development has advanced the design of pressure sensors, and so manometry is usually carried out in supine position.

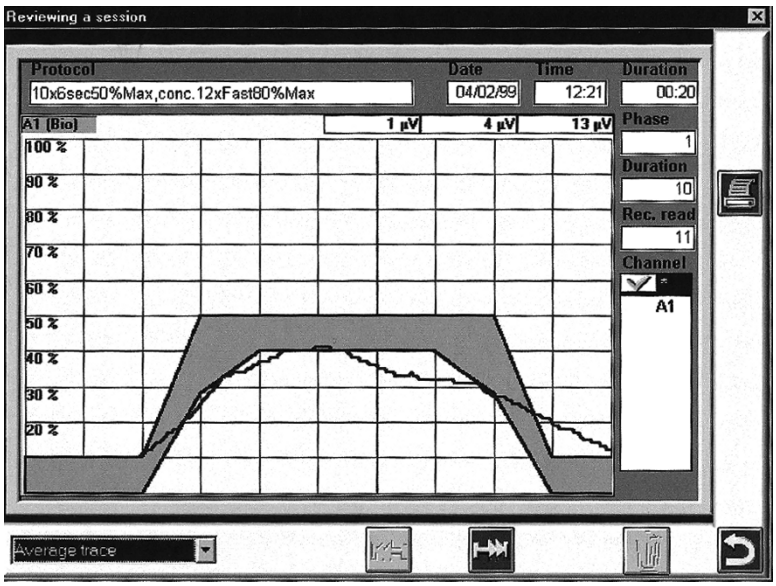

Figure 12a.3. An attempt to follow a predetermined template.

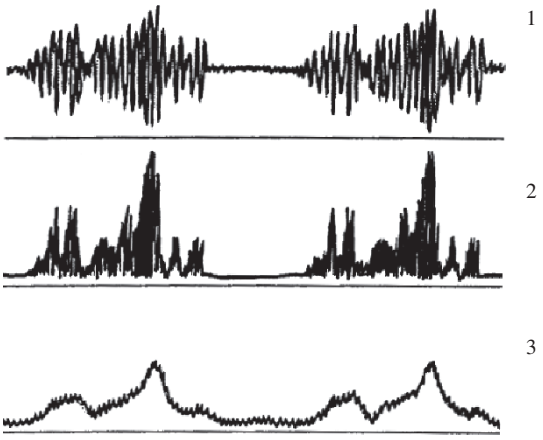

Figure 12a.4 **a** Raw EMG. **b** Rectified. **c** Smoothed.

However, surface EMG (sEMG) (as opposed to needle EMG) can detect only an overall pattern of concurrent activity of motor units within the field of the surface electrode. The sEMG may be contaminated by cross talk from other muscles in the area. This lack of selectivity may lead to erroneous conclusions of "coactivation."[11]

There are also interpatient variables that can affect sEMG values, including muscle volume, intervening adipose tissues, and the motivation of the subjects.[12] Other variables include the EMG equipment, electrodes, and the cabling between the two. As surface electrodes measure action potentials from a number of motor units, the active electrodes and the reference electrode should be as close together (without touching) as possible; this will reduce cross talk and interference from other adjacent striated muscle. There are advantages and disadvantages in both EMG and manometric biofeedback devices[13] and appropriate training is essential for all types of equipment.

12a.3.1 EMG Equipment

The purpose of EMG is to pick up bioelectrical signals from the muscle fibres under investigation without picking up any electrical energy transmitted from elsewhere, using two "active" electrodes and one "reference" electrode. The two active electrodes detect electrical activity each with respect to the reference electrode. The two signals are then fed into a differential amplifier; this amplifies the difference between the two signals, while eliminating any signals common to both. The common mode rejection ratio is a measure of how able the differential amplifier is in eliminating the common mode signal; this is important in areas of extraneous environmental electromagnetic radiation, for example, other electrical equipment in the room. Erroneously high readings may occur if there is any deterioration of the electrode, poor skin contact, battery failure, or faults within the cables or the machinery itself. Artifacts of cardiac activity (ECG) also may be picked up and recognised by regular interference patterns corresponding with cardiac activity. Instruments often have a notch filter to reduce the background noise of the main frequency (50 Hz in the United Kingdom and 60 Hz in the United States).

The normal frequency distribution of EMG signals resembles that of a bell curve. The lower signals between 0 and 20 Hz often are considered unstable; therefore, manufacturers may filter the lower and higher signals by band pass filters. Clearly, any difference in bandwidth filters will lead to different readings, especially at the lower end of the spectrum, if examining very weak PFM.

12a.3.2 Electrodes

Single fibre and concentric needle electrodes are used mainly in neurophysiological studies and research purposes. They are used to detect activity in a single or very few motor units and are beyond the remit of this chapter.

Surface patch electrodes (generally self-adhesive) can be used by therapists for the EMG assessment and treatment of many muscles. To ensure good conductivity, the skin should be cleansed with an alcohol-based cleanser or soap and water.[14] The electrodes should be placed close together on the muscle belly in the direction of the muscle fibres. Close positioning ensures greater accuracy and specificity; wider spacing of electrodes results in a monitoring of bioelectrical activity from a greater area. A reference electrode is placed equidistant from the active electrodes; the nearest bony point is recommended by some manufacturers as being a point of minimal bioelectrical activity. Even though great care is taken regarding accurate electrode placement, it is

unlikely that surface EMG is totally reproducible and accurate.

A vaginal or anal electrode (for monitoring PFM activity) incorporates the two "active" electrodes in the design; the reference electrode (generally a self-adhesive patch) is usually placed on a bony point such as the iliac crest or knee. Internal sensors generally are designed for single patient use; they should demonstrate good electrical conductivity, ease of insertion and comfort, resistance to movement when in situ, be simple to clean, and lightweight in order to be used in functional positions. Vaginal secretions and/or aqueous lubricating gel may affect conductivity. Written and verbal instructions must be provided if patients are to be responsible for probe care.

12a.3.3 The Use of EMG Biofeedback

EMG may be used in assessing resting muscle activity and increasing patient awareness during a voluntary contraction; it can be used to enhance recruitment of motor units and improve relaxation. EMG helps both to isolate individual muscle activity and identify unwanted muscle recruitment. However, each particular manufacturer or even different models made by the same manufacturer may have different parameters regarding filters, bandwidth, and amplification. Each patient is unique and may demonstrate different conductivity levels according to the placement of the electrode. Subcutaneous fatty tissue, skin resistance, and different electrodes can all produce differing results. The electrodes used are also variable in conductivity and design, and therefore the same type should be used on each occasion. Any couplant gel used must be specified and also used on subsequent occasions.

12a.4 Other Methods of Biofeedback

12a.4.1 Vaginal Cones

Biofeedback using weighted vaginal cones has been described.[15] The feeling of loosing the cone alerts the patient to contract the PFM. She is told that the same muscles that retain the vaginal cone will help to retain urine in the bladder and reduce incontinence.

12a.4.2 The Pelvic Floor Educator™

The Pelvic Floor Educator™ (Neen Healthcare, UK) is a cost-effective method of home biofeedback. Although there is no research-based evidence to underpin its use, it is based on the "Q-tip test" developed to demonstrate urethral hypermobility[16] and has been found to be clinically useful. The body of the device (with the "indicator" attached; see Figure 12a.5) is introduced into the vagina. The indicator should move downward when the PFM contract; an upwards-deflection indicates inappropriate bearing down. This cost-effective device therefore indicates when the PFM are working correctly.

Figure 12a.5. The Pelvic Floor Educator™ (Neen Healthcare UK).

12a.4.3 Other Uses of Biofeedback

Biofeedback in many forms may be used for patients presenting with hesitancy and urethral spasm associated with urethral syndrome and/or pelvic pain, and for teaching PFM awareness and relaxation. This is termed "down-training." Simple biofeedback techniques, for example, observing uroflowmetry, listening to urination, can reinforce PFM relaxation during voiding. Biofeedback is also useful following ileoanal pouch surgery to ensure minimal loss of pelvic floor awareness during any temporary stoma period, and is extensively used in the treatment of constipation and other bowel dysfunctions (see Chapters 27 and 28a). Biofeedback (down-training) also may be helpful in teaching relaxation to patients with vaginismus and other sexual disorders (see Chapter 30) and has also been used in the treatment of detrusor instability during cystometry.[17]

12a.5 Summary

Whatever type of biofeedback is used, once the patient has learned the appropriate activity, it should be functionally harnessed. If a patient is using weighted vaginal cones, they should be used during increased activity, going up and down stairs, coughing, jumping, and so forth. If using EMG , the patient may commence biofeedback in a lying position but should proceed to treatment in sitting, standing, and other functional positions and activities. Biofeedback is a most exciting modality of treatment for PFM disorders, and with appropriate training and care therapists can have a formidable weapon in their armoury.

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12b
Ultrasound

RC Jones

12b.1 Introduction

Two-dimensional ultrasound imaging is gaining in popularity within the clinical setting due to its apparent ease of use and limited cost; consequently there are increasing numbers of clinicians utilising this technology, without adequate training. The following chapter is a synopsis of its uses and limitations in the management of incontinence.

12b.2 Transperineal/Translabial Ultrasound

Transperineal or translabial ultrasound places the ultrasound probe on the perineum or labia in a mid- sagittal direction, orientating the probe so that the clearest images of the bladder, urethra, rectum, and pubic symphysis are viewed (see Figures 12b.1.a,b). The X and Y coordinate system most commonly used to measure displacement of the urethra on two-dimensional perineal dynamic imagining has a low specificity for patients with stress urinary incontinence (SUI); however, it is clinically useful and has good inter- and intratester reliability.[1–3] The degree of rotation and movement of the urethrovesical junction (UVJ) has been shown to be a specific measure for detecting SUI, but because it has been difficult to accurately measure the changes after particular manoeuvres, the intertester reliability of this particular measure is only fair and the sensitivities and negative prediction values have suffered.[2,5,6]

12b.3 Transabdominal Imaging

Transabdominal imaging (see Figure 12b.2) places the ultrasound probe suprapubically in a sagittal or transverse direction, viewing the bladder and levator plate on which it rests. Transabdominal ultrasound views have more recently been used by physiotherapists to measure the "lifting" aspect of the bladder base during various manoeuvres,[7–10] since it is less invasive than a transperineal ultrasound view. Transabdominal imaging provides less information than transperineal imaging; and although the reliability[7,10] and face validity[10] of this measure have been reported to be good during PFM contraction and Valsalva, the authors have been unable to account for apparent motion of the probe relative to the structures being examined, so the results need to be interpreted with caution.

12b.3.1 Two-Dimensional Imaging

Dynamic ultrasound imaging contains a considerable amount of useful data that can be obtained with the minimum of invasion to the patient. Current quantitative measurements tell us about the resting position of the urethra and the displacement at the end of events such as valsalva, voluntary pelvic floor muscle (PFM) contraction, and coughing.[11–13] The practical difficulties with accurately determining the finishing point of any manoeuvre, particularly those that are fast, such as coughing, are numerous and are a potential source of error.[14] The operator has either had to make multiple on-screen

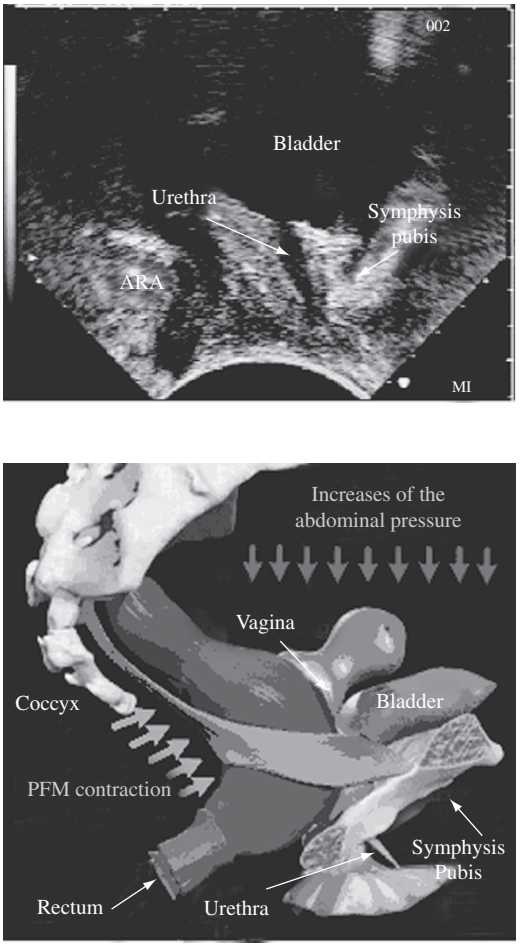

Figure 12b.1. (a) Typical still image of perineal ultrasound frame at rest (anorectal angle, ARA). (b) This demonstrates how a PFM contraction will help prevent urinary incontinence. The continent PFM contracts in preparatory response to increased intra-abdominal pressure. (Copyright Primal Pictures Ltd. www.primalpictures.com)

measurements or has had to determine the exact peak moment, or end position of the manoeuvre, freeze it, and then measure the change in position manually on screen or with in-built electronic callipers. Without correcting for probe movement relative to the pubis symphysis the percentage errors range from 18 to 87%.[15] In addition, measuring only the repositioning of urogenital structures appears to be of limited value because intermediary anatomical changes are not registered. There are limitations with two-dimensional ultrasound imaging in general, as it is unable to analyze out-of-plane structures and the accuracy of the image analysis will be affected.

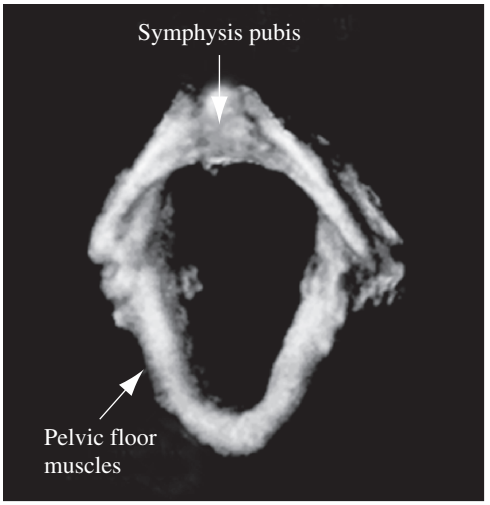

Figure 12b.2. The pilot study of three-dimensional ultrasound visualisation. Ultrasound images of the pelvic floor in a normal 31-year-old woman were acquired using GE Voluson 730 Ultrasound System. The three-dimensional reconstruction and the segmentation of anatomic structures were performed using GE 4D View (V 4.1).

12b.3.2 Three-Dimensional Imaging

More definitive anatomical identification of the mechanisms involved in response to manoeuvres potentially could be made using three-dimensional ultrasound imaging. However, current three-dimensional imaging techniques are not fast enough to record the timing of the movement of urogenital structures in manoeuvres such as coughing, but they can be used to assess PFM morphology (see Figure 12b.2), and the method holds considerable promise in the investigation of pelvic floor disorders in the future.

12b.4 Stress Urinary Incontinence

In healthy women, there is an increase in intraurethral pressure[16] (see Figures 12b.3.a,b) and recruitment of PFM motor units[17] prior to an increase in intra-abdominal pressure (IAP). There are also altered PFM activation patterns during a cough, measured by EMG in women with SUI compared to healthy volunteers.[18] with shorter activation periods, lack of response, or paradoxical inhibition. Data by the author indicate that there is a feed-forward activation of PFM during a cough in continent women, which is absent in women with SUI.[19,20]

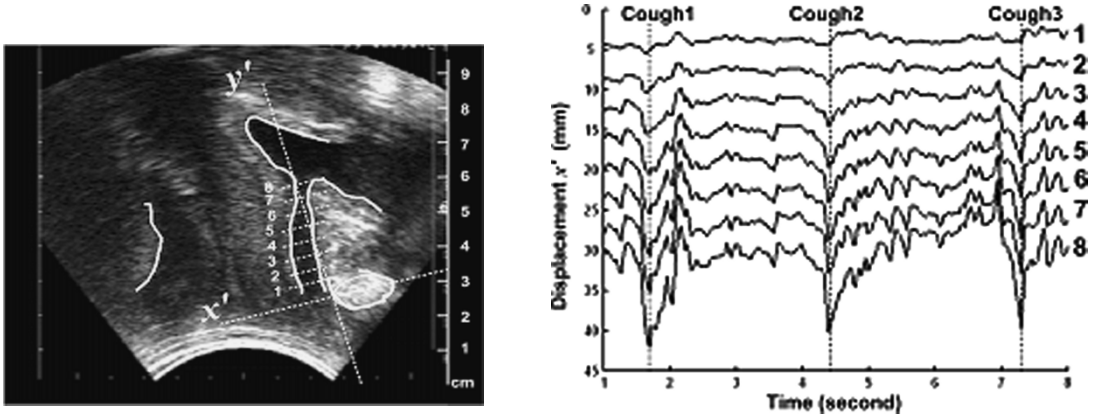

Figure 12b.3. a,b Ultrasound image and segmental analysis showing displacement of the urethra during a cough as viewed by two-dimensional ultrasound imaging [Ann Biomed Engineer 34(3):2006: Figures 5, 16b]. Two-dimensional ultrasound image processing in identifying responses of urogenital structures to pelvic floor muscle activity (from reference 22: Peng Q, Jones RC, Constantinou CE. With kind permission of Springer Science and Business Media).

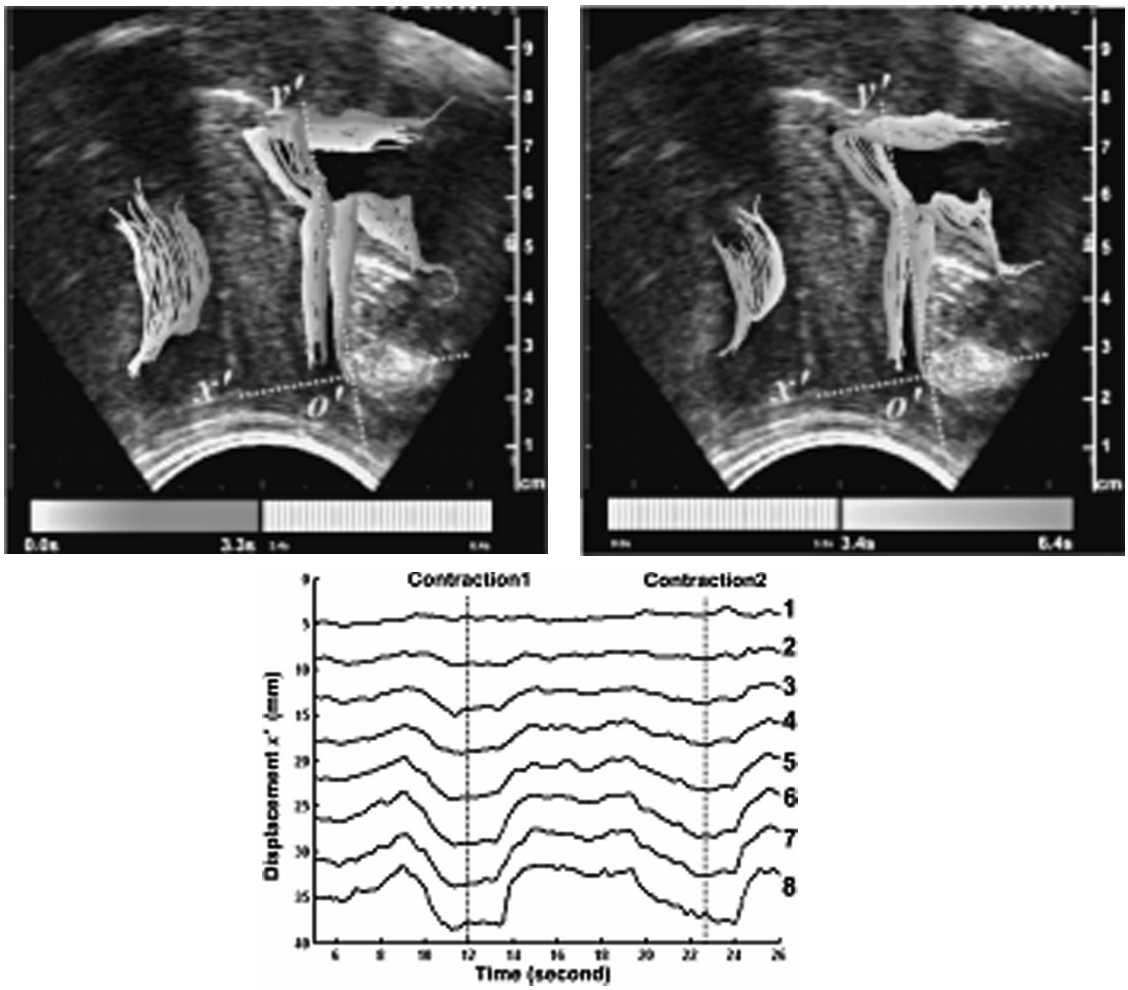

Figure 12b.4. a-c Shows displacement of the anorectal angle (ARA) and urethra during a PFM contraction as viewed by two-dimensional ultrasound imaging and segmental analysis [Ann Biomed Engineer 34(3):2006: Figures 7a, 7b, and 16c. Two-dimensional ultrasound image processing in identifying responses of urogenital structures to pelvic floor muscle activity (from reference 22: Peng Q, Jones RC, Constantinou CE. With kind permission of Springer Science and Business Media).

Not all women with SUI are able to displace the urethra sufficiently to increase the intraurethral pressure with a voluntary pelvic floor contraction,[21] and clinical (see Chapter 11) and research observations by the author,[22] using two-dimensional dynamic ultrasound imaging, indicate that there are subtle differences in the direction of displacement of the urogenital structures of some women when asked to contract their PFM. The direction of voluntary PFM contraction is not always in a ventral–cranial direction, nor does it always directly support or occlude the urethra. However, with manual therapy and facilitation techniques to the PFM, the direction and magnitude of voluntary contractions can appear to change rapidly, within minutes of treatment intervention. Clinically, ultrasound serves as a very useful biofeedback device, as patients are able to see the effect of a PFM or transversus abdominis (TrA) contraction (see Figure 12b.4a, b, c). Ultrasound can also detect displacement of urogenital structures during a voluntary muscle contraction which occasionally cannot be palpated manually.

12b.4.1 Current Research

Current research by the author using two-dimensional ultrasound, which tracks the urogenital structures throughout five different manoeuvres,[19,20,22] indicates that not only do the PFM in continent women have a preparatory contraction, which is most likely an intrinsic mechanism of a cough under cortical control rather than a local stretch response, but that the healthy PFM automatically functions to resist rises in IAP and decelerate momentum caused by rises in IAP by increasing the stiffness of the PF. These functions are lost in women with SUI. The differences of PFM responses between healthy subjects and SUI patients are statistically more significant in cough, knack, Valsalva, and TrA contraction than in PFM contraction.

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13
The Use of Exercise Balls

B Carrière

13.1 General Information

Exercise balls, also known as Swiss balls, can be used for patients with incontinence as well as pelvic pain. The ball sizes most often used are 45, 55, or 65 cm diameter.

13.1.1 Safety Precautions

- Use ABS balls (anti-burst-system).
- Manufacturers' instructions should be followed when inflating the ball.
- Users should check the weight allowed on the ball, which could vary between 400 and 1000 lbs.
- For hygiene, keep the ball clean (wash with soap and water).
- Patients with reduced balance can sit on a flat ball cushion or start exercising on a double ball. Close supervision may be required during exercises until they can be done safely alone.
- Exercise in front of a corner, in a ball-dish, or between two chairs to prevent the ball from running away and also to enable stabilisation with both hands while exercising.

13.1.2 Advantage of Using the Ball

- Ability to exercise in the direction of muscle fibers of the pelvic floor muscles (PFM).[1,2]
- Coordination of the PFM with abdominal muscles and pulmonary diaphragm.
- Practice of function such as sit to stand, leaning on the ball to sitting, fast/slow muscle fiber training.[2,3]
- Cocontraction with other synergistic muscles (hip extensors, abdominal muscles, pulmonary diaphragm, back muscles).
- Exercises can be done with two people sitting back to back on the same ball for resistive, isometric concentric or eccentric exercises.[4]
- Changing the load of the viscera on the pelvic floor by altering the position of the pelvis in space [having the hips and legs higher than the trunk (supine) or sit and bounce on the ball].
- To improve sensory awareness when sitting on the ball utilising its texture and buoyancy.
- To combine relaxation or tightening of the muscles with ball movements.
- Mobilisation of the autonomic nervous system by lying on the ball supine, side lying, prone, or leaning against it.[5]
- Ability to apply motor learning principles, for example, specific, global, and functional activities.[3,6]
- Exercising on the ball is fun and motivating.
- Very comfortable to sit on for patients with hypertonic or painful pelvic floor.

13.1.3 Treatment Considerations:

In sitting, the pressure should be firm to allow for body/ball contact between the ischial tuberosities, coccyx, and symphysis pubis. The size of the ball depends on the body proportions, mobility of the patient, and the exercises intended. During exercises the chest and the knees remain stable in space; the "sitting bones" initiate the movements of the ball backward–forward, side-to-side, or diagonal direction. The patient should sit on the correct sized ball to allow for good upright posture: approximately 90 degrees hip and knee and ankle flexion.[5] Possibilities in ball therapy are many and varied[5] (see Figures 13.1 and 13.2).

Figure 13.1. Patient lying on a knobbed ball for increased sensory input. The pelvic floor is not loaded, the viscera are not pressing in the pelvic floor.

13.1.4 Treatment of Incontinence

Unloaded slow and fast fiber exercises can be practiced in supine or prone with the feet on the ball, then sitting; progress to bouncing. Aim to coordinate PFM activity, correct breathing, and strengthening of surrounding muscles.

13.1.5 Treatment of Pelvic Pain

Emphasis on sensory awareness training and relaxation of the PFM in coordination with breathing can assist in the treatment of pelvic pain. Exercise the spine in all planes to mobilise the sympathetic trunk of the autonomic nervous system.

Figure 13.2. Isometric training: The patient moves the left sitting ball toward the right stable knee and gives herself resistance by pushing the ball backward with her left hand. For increased sensory awareness this ball is knobbed.

13.2 Summary

Exercising with a ball is fun and motivating. It can be done as a home exercise programme while holding a baby, back-to-back with a partner, or while exercising back, trunk, and leg muscles in combination with the PFM. The versatility of the ball makes it a great tool for motor learning. It can help improve balance and general strength and posture while restoring the pelvic floor functions.

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14
Therapeutic Electrical Stimulation

DB Vodušek and J Laycock

14.1 Introduction

In our opinion, nonimplanted electrical stimulation (ES) treatment (as opposed to surgically implanted devices) is a neglected treatment modality in lower urinary tract (LUT) dysfunction, both in terms of research and clinical use. This chapter is a brief review on the subject, referring the reader to more comprehensive texts, and is mainly aimed at clarifying the basic physiology of nonimplanted ES. It furthermore provides data as to the present position of nonimplanted ES therapy.

14.2 History of Concepts and Terminology of Methods

Electrical stimulation has been introduced to treat LUT dysfunction with the idea of providing a substitute for the lack of functional integrity of the system. Early workers have drawn parallels to ES as used in physical medicine, where functional (electrical) prostheses have been developed to improve walking in hemiparetic patients (correcting foot drop on walking). Since then the literature has been stuck with the term "functional electrical stimulation" when referring to ES in LUT disorders. We suggest that only those ES applications should be called "functional" that directly substitute electrical stimuli for the lacking neural control. Functional ES thus would apply to the implanted bladder pacemaker, which in effect is an electronic bladder prosthesis[1]; the function of bladder emptying is achieved by stimulation in real time of sacral parasympathetic fibres in anterior sacral roots.

The development of ES therapy led to many variations of parameters and applications. In terms of application of nonimplanted ES we suggest that the stress should be on unification of approach and research, oriented primarily toward selection of patients.

We suggest the term "electrostimulation" (ES) therapy be used for any method that depolarises nerves and does not rely on direct substitution of absent neural control in real time. Under the heading ES therapy we would include quite different methods described in the past: electric pessary,[2] maximum perineal electrical stimulation,[3] acute electrical stimulation,[4] maximal electrical stimulation,[5], and others,[6–10] as well as all modern derivations and modifications.

14.3 Basic Physiological Principles of Electrical Stimulation

Nowadays it is accepted that the effects of ES are mediated through excitation of nerve fibres in the particular region, using a variety of electrodes. Furthermore, it is accepted that it is best to depolarise as many of the relevant nerve fibres as possible, therefore stimulating them where they are concentrated—in peripheral nerves. It is possible to selectively depolarise afferent (sensory) nerve fibres (when stimulating sensory nerves, such as the dorsal nerve of penis/clitoris), but it is impossible to *selectively* depolarise motor nerve fibres without using an invasive technique.

14.3.1 Depolarisation of Motor Nerve Fibres

Excitation of motor nerve fibres leads to activation of muscle(s) supplied by the nerve. We can call this the direct effect. A single stimulus depolarising the motor nerve will lead to a single muscle contraction.[11] If the stimulation is performed as a continuous delivery of stimuli at a certain rate, a so-called tetanic muscle contraction will be achieved, which is (mechanically) much stronger than the individual contraction. To achieve such a contraction, frequencies from 20 to 50 Hz are usually applied. One has to consider, however, that muscle is fatigable and that higher frequencies lead to fatigue much faster. Using an intermittent type of stimulation (with trains of stimuli interrupted by pauses: the "duty cycle"), the muscle fatigue can be limited to a certain extent.[12] Furthermore, due to the reliance of muscle fibres' metabolism on rate of activation, muscle fibres can be changed by ES from one type to another. Therefore, prolonged ES of a muscle not only causes hypertrophy (and increase in strength) of muscle fibres, but also a change in motor unit properties,[13] but lasting only so long as the stimulation programme is upheld.

We propose that achieving a pelvic floor muscle (PFM) contraction may be a helpful strategy when muscle training is the basic and relevant therapeutic approach. (The validity of this concept has not been proven so far.) Muscle contraction, of course, is a powerful source of sensory input in the central nervous system. We do not believe ES is a good substitute for muscle training as such.

14.3.2 Depolarisation of Sensory Nerve Fibres

Depolarisation of sensory fibres (in sacral roots, pudendal nerves, dorsal clitoral nerves, dermatomes, etc.) leads not only to various reflex responses, but to a widespread invasion into the central nervous system. This means, among other things, perception of stimulation and also many potential changes due to the potentiated afferent input from particular body areas, leading to changes in the central nervous system (due to its plasticity).

The easily measurable effects of stimulation of sacral afferents are reflex contractions of the PFM (the so-called sacral reflexes, which have many synonyms).[14–16] On the other hand, a decrease in PFM spasticity on ES has also been described.[17,18] There is no contradiction in these results, since both effects are physiologically interpretable. In addition, stimulation of sacral afferents has been shown to interfere with sensory input from visceral afferents.[19]

Therapeutically, the vesicoinhibitory influence of perineal ES[20] seems to be of greatest clinical relevance. Inhibitory postsynaptic potentials and inhibition of discharges of parasympathetic neurons due to a spinal reflex with a somatic (pudendal) afferent limb have been demonstrated. Stimulation of these somatic afferents may have both an excitatory as well as an inhibitory influence on the bladder, depending on whether intravesical pressure is low or high respectively. Stimulation of pudendal afferents has been observed to induce bladder inhibition due to pudendal-to-hypogastric[21] as well as to a pudendal-to-pelvic spinal reflex.[22] The latter is assumed to be of main importance for detrusor inhibition in humans. Intravaginal electrical stimulation activated the same reflex mechanisms in cats.[23] The belief that primary activation of perineal (sphincter) muscles is the triggering factor of (reciprocal) bladder inhibition seems to be widespread. However, it has been shown in animals that sphincter muscle contraction is not necessary to achieve detrusor inhibition by afferent stimuli.[20,24]

Clinical experience has accumulated positive results of ES therapy particularly in patients with urge incontinence. Unfortunately, the immediate physiological effects of ES therapy cannot really explain long-term therapeutic results; some additional memorising in the CNS of the vesicoinhibitory input is postulated.[29]

14.3.3 Characteristics of Electrical Stimulation Methods

The authors do not discuss all the various details of different modifications of electrodes, electrode placement, and differences of ES parameters used, but propose as relevant only the issue of intensity of ES, the frequency of electrical pulses,

and the type of ES application (continuous, intermittent, short- or long-term, etc.).

Bladder inhibition can be obtained in humans by ES of nonmuscular pudendal afferents, that is, the dorsal penile nerve, even with a stimulus that is threshold for the bulbocavernosus reflex response.[25] Most of the authors who used vaginal or anal electrodes for vesicoinhibitory ES in their clinical studies used strong stimuli.[26] On the other hand, it was shown that when electrodes are placed in close proximity to the pudendal nerve, substantially weaker stimuli, that is, stimuli strong enough to depolarise pudendal afferents (A-alpha-beta, and possibly at least some A-delta) is all that is required for bladder inhibition.[25,27] Thus the proposed importance of high-amplitude stimulation may be necessary particularly because of higher electrodes-to-nerve distances that occur in the case of anal or vaginal stimulation. On the other hand, it is well known that a stronger stimulus leads to a stronger reflex response. Thus anatomy of placement, the type of electrodes, pulse shape, and duration all may be described in terms of optimisation of depolarising a certain nervous structure, which is thought to be relevant to achieve a certain physiological effect. For example, inhibition of the activated detrusor has been achieved optimally with frequencies between 5 and 10 Hz.[24] The necessary quantity of stimulation necessary to achieve therapeutic effects is an important unknown and so far has been solved pragmatically by increasing the sessions if deemed justified in an individual patient. In future research, a dose-finding approach has to be adopted.

14.3.4 The Effects of Electrical Stimulation

Correctly applied ES of motor and sensory sacral nerve fibres (in the pudendal nerves) leads both to a PFM contraction and inhibition of the active detrusor.[25,27] All these effects have been demonstrated during actual (ongoing) stimulation but continuous nonimplanted ES is not practical/feasible.

Interestingly, patients were observed to remain improved after cessation of the period of stimulation. Thus delayed or carryover effects of stimulation have been claimed. Short-term carryover detrusor inhibition has been demonstrated,[25] and might be due to the activation of the spinal transmitter system such as the opioid.[28] Long-term effects[26] as yet are not clarified. A change in the bladder receptor properties has been postulated[5] but not confirmed in an experimental study with intravesical ES.[29]

The postulated central effects—interferences of electrostimulation with neural mechanisms—seem to be integrative because quite opposite effects can be observed by the same type of ES therapy in different patient groups. Whereas hyperreflexive bladders are inhibited (ie, to improve storage,[45]) the hypo- or areflexive bladders in another group of patients became activated, that is, to improve emptying.[30] It is as if the disrupted neural control is nonspecifically rearranged by electrostimulation, tuning the control mechanism to a more functional status.

To conclude the discussion of physiological effects of ES, it is the basic tenet of the authors that insofar as ES is effective in LUT dysfunction it is through modification of dysfunctional neural control of LUT, not due to some enduring changes in end organs. Thus it is neither surprising nor contradictory that therapeutic effects in lower urinary tract dysfunction have been claimed by vaginal,[31,32] anal,[33] penile and clitoral,[25] and even tibial nerve ES[34] by various types of electrodes, periods of stimulation, frequencies, duration, and shape of pulses and both in patients with incontinence and those in retention.

14.4 Therapeutic Electrical Stimulation in Practice

The different methods of therapeutic (perineal) ES may be most practically distinguished by the different time course of treatment. Thus all proposed nonimplantable ES methods may be classified as either *long-term* or *short-term stimulation*.

Long-term (also called chronic) ES is characterised by prolonged application of low-intensity stimuli for many hours a day and/or night for a period of several months. It seems no longer to be useful in the way it was introduced due to its impracticality. But the implanted varieties of neuromodulation indeed are heirs to these methods first introduced as nonimplantable systems. Short-term ES is characterised by pulses of strong

intensities, which are given for short periods of time (15–20 min), applied in a daily regimen for a certain time period, either as an in-clinic or home treatment.

14.4.1 Short-Term Electrical Stimulation

Faradic stimulation (or faradism)[3] and interferential therapy (IFT)[9,35] have been described in the early days of ES, but now are seldom used and are only of historic interest.[3] Short-term therapeutic application of high-intensity, low-frequency ES in the perineal area was first used as an in-clinic procedure[36] and then developed for home treatment of incontinence.[8] This type of ES has been used for over 20 years in Slovenia (and elsewhere) with encouraging therapeutic results and without any serious side effects. The treatment programme consists of repeated (every day) application of high-intensity, vaginal, anal, perineal, penile, or clitoral ES (maximum current up to 90 mA, maximum voltage 38 V, pulse duration 0.75 ms, frequency 20 Hz) lasting 20 minutes per session for 1 month. Treatment sessions are patient-controlled: the patient switches the stimulation on and is encouraged to continuously adjust the stimulation strength to just below the level of discomfort, that is, to the level of the feeling of strong (but not painful) ES[26].

14.5 Effectiveness of Nonimplanted ES

A critical review of published data on efficacy of ES therapy is beyond the aim of the present text, since comprehensive recent reviews are available.[37] We just mention examples to illustrate the frustrating fact that there is no strong case for evidence-based routine application of ES.

Sand et al.[38] have reported on a multicentre prospective randomized double-blind placebo-controlled trial of ES in treatment of stress incontinent patients. Success rates of 48% (subjective) and 62% (objective assessment), as opposed to 13 and 19% in the placebo-treated group were obtained, respectively. This difference was significant, but the dropout rate in the ES-treated group was much larger (20%) than in the placebo treated group (6%). No significant differences between the group obtaining ES and the one obtaining sham ES for women with stress incontinence was reported by Luber and Wolde-Tsadik[39] and Brubaker et al.[40]

In urge-incontinent patients Brubaker et al.[41] reported a 50% success rate in ES-treated versus 13% in the placebo treated group, the difference being significant. Using short-term strong ES as an in-clinic procedure Abel et al.[42] reported a placebo-controlled trial in urge incontinent patients with a success rate of 91% versus 45% (subjective assessment) and 54% versus 27% (objective assessment), the difference being significant only for the subjective assessment. Their dropout rate was 21% in the ES-treated group.

Due to the paucity of RCT trials, the small numbers of included subjects, and the variation in the stimulation protocols, it is difficult to interpret findings of trials comparing ES to placebo stimulation (which is a difficult issue in itself). Drop out of patients also seems to be a problem. In the in-depth and authoritative review of ES therapy in both genders, Wilson et al.[37] conclude, after critically assessing results and quality of research, that they can see only a trend in favour of ES over placebo in patients with detrusor overactivity and no conclusive evidence for stress or mixed incontinence. In fact, in their recommendation they state that there is insufficient evidence to judge whether (nonimplanted) ES is better than no treatment. They do recommend, however, further RCTs with large samples and long-term follow up to investigate the added value of ES as compared to other treatments in urinary incontinence.[37]

The general impression from pooled data from trials comparing PFM training and ES in stress-incontinent women is that training is better, although in most single trials no statistically significant differences could be demonstrated.[37] In a comparative study of oxybutynin and short-term strong ES, Wise et al.[43] concluded that both oxybutinin and ES reduce the severity of urinary symptoms in women with detrusor instability and that ES is less effective but more acceptable.

14.6 Concluding Comments

Nonimplanted ES continues to be used,[44] despite the discouraging results of the few RCTs, and in our opinion continues to be useful, but only randomised controlled trials will finally solve the issues, and we believe that further research is very worthwhile.

For implanted sacral nerve root neuromodulation more robust data have been gathered (due to strong industry support of research). Thus, at present, there is the recommendation (based on Grade B of evidence) that implanted neuromodulation may be offered for women with urinary incontinence due to refractory detrusor overactivity and refractory irritative symptoms,[45] while no such recommendation was given for nonimplantable ES. Women with urinary incontinence due to detrusor overactivity and irritative symptoms thus logically would represent subjects for a large controlled multicenter study of nonimplanted ES to gather the necessary data. We propose that a significant percentage of patients who would profit from implanted ES would respond to nonimplanted ES therapy.

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15
Bladder Training and Behavioural Training

KL Burgio and PS Goode

15.1 Introduction

Behavioural treatments are a diverse group of therapies that are now well established for the treatment of urinary incontinence and other disorders of the lower urinary tract and pelvic floor. Some interventions, such as bladder training and behavioural training, focus on altering bladder function. Others focus on improving pelvic floor function or lifestyle changes. What they all have in common is that they improve bladder control by changing the patient's behaviour or teaching new skills. In actual practice, most behavioural programmes are composed of several components tailored to the needs of the individual patient. Components of behavioural treatment can include voiding schedules, bladder control strategies, urge suppression techniques, urethral occlusion, biofeedback, self-monitoring with a bladder diary, and behavioural lifestyle changes such as weight loss and fluid and diet management.

15.2 Changing Bladder Habits

Patients who experience frequent urges often respond with frequent voiding. This relieves the immediate sensation of urge but sets the stage for more frequent urgency. Once frequent voiding becomes a habit, it can be difficult to break and may lead to reduced functional bladder capacity, diminished ability to control urges, bladder overactivity, and urge incontinence. One approach to breaking this cycle is to place the patient on a voiding schedule using bladder training.

15.2.1 Bladder Training

The goal of bladder training is to break the cycle of urgency and frequency using consistent, incremental voiding schedules. One of the most important features of bladder training is that it dissociates voiding from urgency. Voiding by the clock rather than in response to urgency weakens the urge–void response.

The patient first completes a voiding diary, which shows the clinician when and how often the patient is voiding. After reviewing the diary with the patient, a voiding interval is selected based on the longest time interval between voids that is comfortable for the patient. Patients are given instructions to void first thing in the morning, every time the interval passes, and before going to bed at night. Over time, the voiding interval is gradually increased. An example of a bladder-training program starts with a voiding interval of every hour, increases by 15 minutes every 3–5 days as the patient feels comfortable, to a goal of voiding every 3–4 hours. Guidelines for bladder training appear in Table 15.1.

Several clinical series have demonstrated efficacy of bladder training.[1–8] The most definitive study is a randomised clinical trial that showed a mean 57% reduction in frequency of incontinence in older women.[9] In this trial, bladder training not only reduced incontinence associated with detrusor overactivity, but also incontinence associated with sphincter insufficiency, possibly because patients acquired a greater awareness of bladder function or that having to postpone urination increased pelvic floor muscle (PFM) activity.

Table 15.1. Instructions for bladder training.

Step 1	Review voiding diary with the patient. Note the voiding intervals and their variability.
Step 2	With the patient, select the longest voiding interval with which he/she finds comfortable.
Step 3	Instruct the patient to Empty your bladder: • First thing in morning • Every time your voiding interval passes during the day • Just before you go to bed
Step 4	Teach patient techniques for coping with the urge to void that occur before the interval has passed: • Distraction to another task that is mentally but not physically engaging (ie, reading, calling a friend, writing a letter, balancing a cheque book) • Deep breathing and relaxation • Self-statements (eg, "I can wait until it is time to go" "I am in control") • Urge suppression strategy: using pelvic floor muscle contraction to keep bladder relaxed
Step 5	Gradually increase the voiding interval • When the patient is comfortable on the voiding schedule for at least 3 days • By 15- to 30-minute intervals as determined by the patient's confidence and clinical judgment

15.2.2 Delayed Voiding

Delayed voiding is another approach to teaching patients to control the bladder but without placing them on a voiding schedule. Initially, patients are encouraged, whenever they have an urge to void, to wait 5 minutes before voiding. For patients who have experience with incontinence, even a small sensation of urge heralds possible leakage and triggers a degree of anxiety or tension. They tend to respond to the urge to void as soon as possible and neglect to exercise any control they may have. Even the most urgent patient can usually be convinced to try a 5-minute delay. Patients are often surprised to find that after a brief wait the urge subsides or disappears altogether, which enhances their sense of control. Once confidence has been established, the delay interval can gradually be increased and a normal voiding interval can be attained.

15.2.3 Urge Control Techniques

In bladder training as well as delayed voiding, the patient is asked to postpone urination, which for most patients involves coping with the sensation of urge while they wait. The traditional approach to urge management has been to suggest various techniques for relaxation or distraction to another activity.[9,10] Patients are encouraged to get their minds off the bladder by engaging in a task that requires mental but not physical effort. Examples include reading, calling a friend, or making a to-do list. Distracting their attention from the bladder can reduce anxiety and allows time for the urge to subside. Also used are affirming self-statements such as "I am in control of my bladder," or "I can wait."

More recently, traditional bladder training has been enhanced by incorporating techniques from behavioural training. Repeated contractions of the PFMs are used to control urgency and detrusor contractions while the patient postpones urination.

15.3 Behavioural Training and the Role of the Pelvic Floor

Behavioural training is an intervention that combines the principles of physical therapy, behavioural therapy, and motor learning into an increasingly accepted conservative treatment for incontinence. The goal of behavioural training is to improve bladder control by teaching the patient how to voluntarily suppress detrusor contractions. This involves adopting a new response to urgency and learned use of the PFMs to control urgency and detrusor contraction. Historically, PFM exercises were viewed primarily as a treatment for stress incontinence. However, PFM exercise is now a central element of behavioural training for urge incontinence because it has been shown that the PFMs play a role in overactive bladder and urge incontinence in women as well as men. Voluntary contraction of the PFMs not only can occlude the urethra, but also can inhibit or abort detrusor contractions.[11,12] This is a skill that can be accomplished by most patients and provides significant reduction of incontinence.

The first step in behavioural training is to help patients to identify their PFMs and to contract and relax them selectively without increasing pressure on the bladder or pelvic floor. Home exercise then helps patients increase muscle

strength as well as motor control. PFM exercise is described in detail in Chapter 11a.

In addition to scheduled exercises, patients with urge incontinence can often benefit from practice in interrupting or even slowing the urinary stream during voiding, once per day. Not only does this provide needed practice in occluding the urethra and interrupting detrusor contraction, it does so in the presence of the urge sensation, when patients with urge incontinence need the skill most. Some clinicians have expressed concerns that repeated interruption of the stream could lead to incomplete bladder emptying in certain groups of patients. Therefore, caution should be used when recommending this technique for patients who may be susceptible to voiding dysfunction.

15.4 Changing the Response to Urgency: Urge Suppression Strategy

An essential component of behavioural training is teaching patients a new way to respond to urgency: the urge suppression strategy.[11–13] In behavioural training, they are taught how the seemingly normal response of rushing to the nearest bathroom is actually counterproductive. Rushing enhances the sensation of fullness, exacerbates urgency, and triggers detrusor contraction. Furthermore, as the patient approaches the toilet, visual cues can trigger urgency and incontinence. To avoid this conditioned response, it is best to stay away from the bathroom during episodes of urgency. This new response may seem counterintuitive at first, but patients can learn to stop what they are doing, sit down if possible, and contract their PFMs repeatedly to diminish urgency, suppress the detrusor contraction, and prevent urine loss. They concentrate on voluntarily inhibiting the urge sensation and wait for the urge to subside before they walk at a normal pace to the toilet. Step-by-step instructions for the urge suppression strategy appear in Table 15.2.

Most patients are firm in the attitude that, "When you gotta go, you gotta go." To even attempt delaying voiding using the urge suppression strategy, patients must suspend this belief. When a patient is hesitant to attempt the urge strategy, they can be encouraged to begin in a safe place and to wear absorbent protection to avoid embarrassment. Any positive results including partial success should be regarded as progress. Once patients have the experience of control, that experience can be used to debunk their "gotta go" belief. "When you gotta go, you DON'T gotta go" is a useful mantra for the patient to use in the face of urgency.

The effectiveness of behavioural training has been established in several studies.[11,12,14,15] In the first randomized controlled trial,[12] behavioural training reduced incontinence episodes significantly more than drug treatment for women with urge incontinence. Behavioural training is also useful for managing nocturia.[16] Patients are instructed to use the urge suppression strategy when they wake up at night. If the technique is successful and the urge subsides, they are encouraged to go back to sleep. If after a minute or two the urge to void has not remitted, they may get up and void so as not to interfere unnecessarily with sleep. The skills learned through behavioural training are also useful for treatment of stress incontinence in women as well as for men with postprostatectomy incontinence.[17,18]

15.5 Behavioural Lifestyle Changes

15.5.1 Fluid Management

Changes in the type or volume of fluids are recommended by many clinicians either as a primary or as an adjunctive strategy to optimize incontinence outcomes and have been shown to improve both stress and urge incontinence.[19]

Table 15.2. Patient instructions for the urge suppression strategy.[a]

	When you experience a strong urge to urinate
Step 1	Stop and stay still. Sit down if you can.
Step 2	Squeeze your pelvic floor muscles quickly three to five times and repeat as needed.
Step 3	Relax the rest of your body. Take several deep breaths.
Step 4	Concentrate on suppressing the urge feeling.
Step 5	Wait until the urge subsides.
Step 6	Walk to the bathroom at a normal pace.
Step 7	If the urge returns on the way to the bathroom, return to Step 1.

[a] From reference 13, p 81. © 1989 Kathryn L. Burgio, K.Lynette Pearce, and Angelo J. Lucco. Reprinted with permission of the Johns Hopkins University Press.

Many patients with incontinence attempt to manage their symptoms by restricting fluid intake at particular times of day when they anticipate not having access to a restroom or they believe they are at risk of having an incontinence episode. This can be a very effective strategy. However, some patients restrict their overall fluid intake, which places them at risk of dehydration. Although it may seem counterintuitive, it usually is good advice to encourage patients to consume at least six 8-ounce glasses (approx. 1500 ml) of fluid each day.[20] A useful strategy to encourage adherence to this recommendation is to point out that concentrated urine has a stronger odour than more dilute urine. Patients with incontinence often are quite sensitive about urinary odour and welcome a suggestion to decrease this.

When patients consume an abnormally high volume of fluid (eg, resulting in >2000 ml of output per 24 hours), fluid restriction often is an appropriate measure and can be helpful for reducing urine loss.[19] Some people have increased their fluid intake deliberately in an effort to "flush" their kidneys or lose weight. In others it simply is a habit. In these cases, reducing excess fluids can relieve problems with sudden bladder fullness and help to reduce urgency, frequency, and urge incontinence.

Elimination of caffeine from the diet has been shown to improve both stress and urge incontinence.[19,21–23] Many patients are reluctant initially to forgo their caffeinated beverages, but they may be convinced to try it for a short period of time, such as 3 to 5 days. If they experience relief from their symptoms, they often are more willing to reduce or eliminate caffeinated beverages from their diet. For patients who routinely drink more than one caffeinated beverage per day, caffeine reduction should be approached gradually and may include mixing or alternating caffeinated and decaffeinated beverages to avoid withdrawal symptoms (see Chapter 17). Although there are few data on the role of sugar substitutes and other bladder irritants, there are clinical cases in which these substances appear to aggravate incontinence and reduction has provided clinical improvement.

15.5.2 Weight Loss

Obesity is an increasingly common health problem that also is an established risk factor for urinary incontinence.[24] There is a small literature that demonstrates significant improvement in continence status with as little as 5% weight reduction in women.[25] Because this small amount of weight loss is achievable for many overweight women, more clinicians are recommending weight loss as a component of behavioural treatment.

15.6 Summary

Bladder training and behavioural therapy are widely used interventions that improve bladder control by altering bladder habits and teaching new skills. They are useful for both urge and stress incontinence and are supported by an abundance of evidence demonstrating their efficacy. Although the majority of patients are not cured with these treatments, most can achieve significant improvements in continence. This makes them very reasonable first-line approaches to the treatment of men and women of all ages.

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16
Voiding Problems

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16.1 Introduction

Voiding difficulties can result from a myogenic, psychogenic, or neurogenic problem or a bladder outlet obstruction. Any of these can result in difficulty in initiating voiding, a low flow rate, or a postvoid residual (PVR) of urine. Urinary retention can be defined as the inability to void.[1] Uroflowmetry and other urodynamic investigations may be warranted to investigate the cause of PVR (see Chapter 9). This chapter considers the causes and dangers of PVR and techniques to assist voiding.

16.2 Contributory Factors

Careful history taking (see Chapter 7) and investigations (see Chapter 9) should identify the causes and the severity of the problem. Some contributory factors to voiding difficulties are shown in Table 16.1.

16.3 Dangers of Incomplete Emptying

If untreated, incomplete emptying or a high-pressure bladder may lead to damage of the upper urinary tract. If there is any suspicion of an incomplete void, the patient should have an ultrasound bladder scan or an "in–out" catheter (if a scanner is not available). If there is significant residual urine, it should be considered in relation to the amount voided, any known pathology, and any other possible causes of the symptoms; voiding in unfamiliar surroundings may lead to unrepresentative results. Less than 50 ml PVR are considered adequate voiding and more than 200 ml abnormal.[6] Any patient with a PVR without obvious cause should be given a full neurological assessment (see Chapter 6). They may require teaching clean intermittent self-catheterisation (CISC) and regular monitoring may be required (see Chapter 23).

Chronic residual urine can lead to an ever-increasing likelihood of recurrent urinary infections requiring an escalating provision of medication. A chronic obstruction ultimately can lead to high intravesical pressure resulting in hydronephrosis and kidney damage.[6] Therefore, an obstruction of any kind should be investigated and medical referral made as appropriate. More information regarding urine flow is shown in Table 16.2.

16.4 Conditions

16.4.1 Neurogenic Bladder

In patients with a neurogenic bladder the aims must be to keep bladder pressure low, to regularly empty the bladder to completion, and to avoid reflux at all times (see Chapter 6). Patients respond to a variety of triggers, but there must be extreme caution in using any of them (see Table 16.3).

16.4.2 Postmicturition Dribble (see Chapter 21)

For many years men have been taught how to massage the bulbar urethra in order to effect urethral emptying and avoid postmicturition dribble. It now has been found that a contraction of the pelvic floor muscles (PFM) is equally effective[14] (see Chapter 21).

Table 16.1. Possible causes contributing to voiding problems.

Neurological conditions	• Any condition affecting the neurological control of the bladder at higher centres or spinal cord eg Parkinson's disease, MS, stroke
Endocrine	• Diabetes[2]: neuropathy of either the autonomic supply to the bladder and/or somatic nerve supply to the pelvic floor muscles (PFM) • Hypothyroidism[2]
Obstetric	• An overdistended bladder during or after labour.[1] Factors may include vacuum extraction,[3] prolonged 1st and 2nd stage,[3] birth weight ≥380 gm, [3] perineal pain after a tear, or episiotomy.[1]
Gynaecological	• Painful herpetic lesions[1] • Postgynaecological surgery,[1] eg, midurethral sling procedures, colposuspension • Acute urethritis, cystitis, vulvovaginitis[2]
Obstructive	• Males: prostatic enlargement, immediate postprostate surgery • Females: cystocoele, uterine prolapse[2] • Either sex: urethral stenosis, pelvic mass,[2] constipation
Elderly	• Detrusor hyperactivity with impaired contractility known as DHIC[4]
Psychogenic	• Anxiety or depressive illness[2] • Hysteria[2]
Habit	• Women crouching over rather than sitting down on a toilet seat to void[5]
Pharmacological	• Tricyclic antidepressants[2] • Anticholinergics[2] • Adrenergic agents[2] • Ganglion blocking agents[2]
Others	• Hypersensitive urethra[2] • Detrusor mypathy[2]

Table 16.2. Factors contributing to abnormal urine flow.

Hesitancy	• Voiding in public toilets • First large void of the day • Immediately postdelivery • Postgynaecological surgery
Slow stream	• Obstruction • Low detrusor pressure • Consequence of a neuropathy of the pelvic nerves supplying the bladder • Any combination of the above
Interrupted stream	• Lack of a sustained detrusor contraction • Incoordinated activity between the detrusor and sphincters • Obstruction • Unsustained straining.

16.4.3 Childbirth

Postpartum urinary retention makes a woman susceptible to bladder dysfunction in the future, it is more likely to follow prolonged labour, epidural analgesia, and operative deliveries.[15] It also has been shown that 85% of women having a vaginal birth sustain some form of perineal trauma,[16] which may inhibit micturition. As it may take up to 8 hours for bladder sensation to return after epidural anaesthesia, there is a danger of urinary retention[17] if catheterisation is not continued after delivery. Advice should be given to attempt voiding once voluntary control of the lower limbs has returned, regardless of a lack of bladder sensation. Each obstetric unit should have an evidence-based protocol of care in place regarding bladder care in labour and immediately postpartum.

Antenatal education is a useful way to inform women of normal bladder function, how to recognise potential bladder problems, and to advise them to void at regular intervals. Postnatal symptoms may include inability to spontaneously void, voiding frequent small volumes, bladder distension, increasing pain in the lower abdomen, or involuntary loss of small volumes of urine. It is essential to check for a PVR, ideally by a bladder scan, after any traumatic delivery, for those patients with a long period without bladder emptying, postepidural anaesthesia, or those that report any urinary symptoms.[15]

16.4.4 Postsurgery Outflow Obstruction in Women

Obstruction can be a result of urethral stricture or surgically induced by bladder neck suspension surgery. Women undergoing such procedures should be assessed prior to their surgery to determine if they might be at risk of postoperative voiding dysfunction. If this is thought likely, the patient should be appropriately counselled and taught CISC prior to surgery. Both constipation and prolapse can contribute to outflow obstruction. Appropriate catheterisation protocols and procedures should be in place for patients with PVR following gynaecological surgery.

Table 16.3. Activities that may assist in voiding.

Privacy	Comfort and privacy are necessary to void easily. Particular attention should be paid to this especially in long-stay accommodation.
Sitting	Women should always sit down properly on the toilet with the feet supported, relaxing the PFM, allowing sufficient time to void to completion.
Double voiding	After the initial void, stand up, move around, then sit down and attempt to void again.
Leaning forwards	It has been found that leaning forward can promote micturition.[7]
Stroking/tickling	Stroking or tickling the lower back to stimulate the micturition reflexes has also been reported as helpful in some patients.
Whistling	Whistling provides a sustained outward breath with a gentle increase in intra-abdominal pressure.
Pubic hair	Some people can initiate a detrusor contraction by pulling pubic hair; this is thought to have a reflex action on the bladder.
Running water	It is known that the sound of running water can promote a detrusor contraction[8]; however, care must be taken not to promote detrusor instability by overuse of this technique.
Warm water	In those with a traumatised perineum and pelvic floor, the action of pouring warm water over the perineum can aid in promoting relaxation, and therefore assist in voiding.
Relaxation	General relaxation techniques should be taught to those patients who are generally tense and anxious about their condition. The method chosen should be one that is most appropriate for the individual.
Prolapse	If a women has a large cystocele, she may need to be taught to manually reduce the prolapse prior to voiding while awaiting surgery.
Tapping	Can result in a gross reflex of both the detrusor and urethral sphincter. If appropriate use at least seven to eight percussions with intervals of a few seconds.[9] Ceasing tapping at the initiation of the void should result in relaxation of the sphincter while the slower-reacting detrusor continues to contract.[9] Tapping then can be repeated if necessary. Only used in neurological patients if they are shown to be urodyna mically safe and stable.[11]
Credé manoevre	The hand gives gentle downward pressure suprapubically. This is a potentially hazardous manoevre and should be used only in those with an underactive detrusor and underactive/incompetent sphincter mechanism causing low urethral resistance. This also may assist in stimulating the detrusodetrusor reflex.[12] Sphincter–hyperreflexia and detrusor–sphincter dyssynergia are contra-indications.[11]
Valsalva manoeuvre	The act of holding the breath with a closed glottis while increasing abdominal pressure. As this action has the potential to increase genital prolapse, rectal prolapse, and haemorrhoids, it should be used only with great caution. The same cautions must be taken as when using a credé manoevre.
Squeezing the glans penis and the scrotal skin	A form of bladder reflex triggering; must have integrity of the sacral reflexes.[8]
Anorectal manipulation	Found helpful in some patients with spinal cord injury.[10] Only used in neurological patients if they are shown to be urodynamically safe and stable.[11]
The "queen square bladder stimulator"	This suprapubic vibration device has also been found useful in neurogenic patients with detrusor hyperreflexia who are not severely disabled.[13]

16.5 Prevention of Postvoid Residual Urine

Women should always sit down comfortably and allow time to void to completion without hurrying. Children should be taught good habits during their early toilet training experiences; girls should sit with their feet supported.[18] Voluntary stopping and starting of urine flow should be strongly discouraged, as this goes against normal voiding reflexes.[12,19] Constipation should be avoided and a healthy diet encouraged. Any pregnant women wishing to undergo epidural anaesthesia for childbirth should be counselled regarding appropriate bladder care both during labour and after delivery.

16.6 Treatment

In nonneurogenic patients some simple measures may assist in bladder emptying (see Table 16.3). If these are insufficient, further action may be necessary (see Box 16.1).

Box 16.1.

Treatment of Postvoid Residual

- Neuromuscular stimulation may be indicated to assist in normalising reflex activity (see Chapter 14).
- Those unable to relax their PFM may respond to biofeedback.
- Clean intermittent catheterisation (see Chapter 23).
- Medication including cholinergic agents and intravesical prostaglandins have been suggested, but there has been inadequate evidence of clinical benefit.[2] Diazepam may help in postsurgery voiding difficulties.[2]
- Urethral dilatation may be necessary in the case of urethral stricture causing difficulty in voiding. Stricture therapy using CISC may need to be continued.
- Patients with a neurogenic bladder and PVR have to be treated with extreme caution. Triggered reflex voiding, CISC, medication, electrostimulation, use of appliances, surgery, or an indwelling catheter all may be considered. However, triggered reflex voiding is recommended only if urodynamically safe.[11] It also must be remembered that autonomic dysreflexia with symptoms of paroxysmal hypertension, anxiety, sweating, headache, and bradycardia[9] may be triggered by reflex voiding (see also Chapter 6).

16.7 Summary

There are many possible causes of PVR. Each person needs to be assessed and treated individually and the simple measures described in Table 16.3 may be of help. Patients with voiding difficulties should be monitored and appropriate investigations and referrals made if there is a lack of improvement by the use of these simple measures.

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17
The Importance of Fluids

GR Stephens

17.1 Introduction

Fluid intake plays a minor role in the pathogenesis of incontinence[1]; however, there are some fluids that have the potential to exacerbate lower urinary tract symptoms and some fluids that are thought to be therapeutic.

17.2 Recommended Fluid Intake

There is a general consensus within the medical community that adults should aim to drink sufficient fluid to void 1.5 litres (3 pints) per 24-hour period.[1] This equates to 6–8 cups of fluid per day or between 1800 and 2400ml,[2] but is variable according to environmental factors such as health status, temperature, and exercise. Enough fluid should be consumed to produce urine that is clear, odourless, and of pale yellow colouring. If insufficient fluid is consumed, there is an increase in urine concentration and constipation, which are known to exacerbate lower urinary tract symptoms.[1]

17.3 Cranberry Juice

Cranberry juice has been used for many years in the prevention and treatment of urinary tract infections (UTI). There is an abundance of anecdotal evidence and clinical evidence is gathering to support the recommendation of cranberry juice in the prevention of UTI.[3]

There have been two hypotheses postulated to explain the mechanism of action of cranberry juice. The first hypothesis pertains to cranberry juice reducing the urinary pH, achieving bacteriostasis. This has been refuted since clinical studies have produced contradictory results, suggesting this theory is unsubstantiated.[3]

The current hypothesis is that cranberry juice works by preventing bacteria, specifically *Escherichia coli* from adhering to the uroepithelium. This theory has received microbiological justification and suggests that proanthocyanidins present in cranberry juice possess the ability to prevent *E. coli* from expressing fimbriae, specifically type-P –fimbriae.[4] This therefore inhibits its adherence to the uroepithelium and *E. coli* are voided from the bladder. These proanthocyanidins have been identified and are present only in the vaccinium species, that is, Cranberries and blueberries.[5]

Clinical studies investigating the effectiveness of cranberry products, in the prevention and management of UTI in susceptible populations, have been published. In the elderly population, studies suggest that cranberry juice is capable of reducing the level of bacteriuria, and thus prevent UTIs[6]; however, there are some concerns over the validity of this study. In sexually active women, clinical trials have been positive in their findings, relying on UTI rates and antibiotic use rather than bacteriuria as used in previous research.[3]

A systematic review concluded that there is no reliable evidence to recommend cranberry juice for the prevention of UTI and recommended further research.[7] Trials included in the review were criticised for their small sample sizes, poor design, and poor analysis.[7] However, since this publication, several well-designed clinical studies, which have been analysed on an intention-to-treat basis, have been published. These studies

supported the role of cranberry juice in the management of recurrent UTI in certain patient groups. There is insufficient evidence to support the use of cranberry juice in the treatment of UTI,[3] the prevention of UTI in neurogenic bladders or in-dwelling catheters. It has been demonstrated that cranberry capsules, that have been manufactured to contain the active proanthocyanidin, are the most cost-effective means of preventing UTI compared to cranberry juice or placebo,[8] At present there is no regulation of these active ingredients in juice or capsules, and clarification regarding optimal preparation and dosage is necessary.[3]

Cranberry juice is safe and has relatively good tolerability and currently is being investigated for additional therapeutic properties.[3] Caution, however, is recommended in the following groups: patients prescribed drugs with potential interaction with cranberry juice, for example, warfarin (increased potency with cranberry juice), patients with a history of oxalate calculi, and diabetics who should use capsules owing to the sugar content of the juice.[9]

17.4 Caffeine

Caffeine (methylxanthine) is the most widely consumed psychoactive stimulant drug in the world, reaching peak plasma level at 30–60 minutes after ingestion.[2] It is the active constituent of common beverages such as tea, coffee, cola, chocolate-based products, energy drinks, and over-the-counter medications. As well as its stimulant properties, caffeine is renowned for its diuretic effect. Therefore, recommendation to reduce caffeine intake has been an accepted practice internationally among clinicians treating patients with urinary dysfunction. Despite the apparent consensus, there is little evidence to support this association. There are only a few published studies that explore caffeine and urine symptomatology and these are conflicting in their conclusions.[1]

It is postulated that caffeine has an excitatory effect on the detrusor muscle comparable to that observed in skeletal muscle.[10] This relationship has been shown using cystometry, where there was a statistically significant rise in intravesical pressure in women with detrusor instability compared to those with normal bladder function.[10] This has been corroborated by an observational study that showed a statistically significant association between women with detrusor instability and high caffeine intake of more than 400 mg daily.[11]

The few published clinical trials suggest that reducing caffeine intake should reduce urinary symptoms.[2,12,13] It is suggested that reducing caffeine to an intake of less than 100 mg caffeine per day is likely to reduce episodes of urinary incontinence in the short term, but urgency and frequency are unaffected and this dosage level is unsubstantiated.[14]

It has also been suggested that caffeine may reduce urethral smooth muscle pressure, and therefore exacerbate symptoms in those patients with a low urethral pressure profile.[15] To date no further research has been published regarding this effect.

Further clinical trials are required to provide more rigorous scientific evidence to provide a clear relationship between caffeine and urinary symptoms. Until such time, a trial of reducing caffeine is worthwhile particularly in those patients who have identified caffeine intake as being associated with their symptoms.[14] Trials of caffeine reduction such as this should be carried out by the "caffeine-fading" method over 7 to 10 days to avoid caffeine withdrawal syndrome and the response evaluated. Patients will require an individual management programme to yield the best caffeine reduction plan for optimum continence management.[14]

17.5 Carbonated Drinks

Ingestion of carbonated drinks has been associated with an increased risk of developing overactive bladder and stress incontinence.[16] It is at present unclear why this effect occurs and may be due to diuresis or chemicals found within carbonated drinks, such as artificial sweeteners, which is supported by anecdotal evidence.[1]

17.6 Fruit Juice

Anecdotally, some fruit juices, especially citrus juices, may provoke urgency and frequency; however, the mechanism for this is unknown.

17.7 Alcohol

There is no association identified between urinary incontinence and alcohol,[1] although alcohol is renowned for its diuretic and sedative effects.

17.8 Conclusion

Health care professionals have the power to inform and educate appropriately selected clients of the physiological action and potential therapeutic benefits of consuming certain fluids. Clinicians must use their clinical reasoning skills, being cautious in their recommendations that reflect current evidence thus enabling patients to make an informed choice.[3]

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18a
Pregnancy, Childbirth and Urinary Incontinence

DS Kapoor and RM Freeman

18a.1 Introduction

The prevalence of female urinary incontinence increases with age probably due to the effects of menopause with atrophic change and muscle and connective tissue weakness. However, evidence suggests that such weakness takes its origins in pregnancy and after vaginal childbirth.[1] This chapter assesses the contributions of pregnancy and childbirth to the pathogenesis of pelvic floor dysfunction, in particular stress urinary incontinence (SUI).

18a.2 Pregnancy and Childbirth

It long has been known that vaginal delivery is associated with pelvic floor defects, lower urinary tract symptoms (LUTS), pelvic organ prolapse (POP), and faecal incontinence. There are, however, few long-term follow-up studies assessing the natural history of these defects to confirm if they are the reason for long-term urinary incontinence and POP. However, follow-up of patients with obstetric anal sphincter injuries (OASI)/"3rd degree tears," has suggested that faecal incontinence is a common long-term result,[2] and so it is possible that damage to other parts of the pelvic floor might also result in symptoms such as urinary incontinence and POP.

18a.2.1 Anatomical Changes in Pregnancy

In pregnancy, the gravid uterus and fetus are capable of producing anatomical changes through pressure effects. Imaging studies have shown changes to the bladder neck and urethra, for example, alteration of angle, bladder neck opening, and mobility. In addition, hormonal effects can alter urethral sphincter function and connective tissue strength resulting in reduced urethral resistance. With the increase in intra-abdominal pressure due to the gravid uterus, this combination is likely to increase the risk of stress urinary incontinence in pregnancy. Evidence suggests that this occurs in up to 64% (mean approximately 40%) of women,[1] and in 20% this can be bothersome as judged by demonstrable SUI, daily leakage, and pad test.[3,4]

Postnatally the incidence of SUI is much less (6% to 32%; mean 15%[1]) Interestingly, it usually is reported as being "less severe,"[3] with only 3% reporting daily leakage and 11% seeking help.[5] Fewer patients develop SUI for the first time after delivery with incidences varying from 0 to 27% (mean 9%),[1,6] and so it would appear that the majority of patients with postnatal SUI probably had it *antenatally*.

Why antenatal SUI should persist into the postpartum period is unclear. It no longer can be due to the pressure effects of the fetus or hormonal changes of pregnancy. Therefore, it either is due to as yet unknown irreversible changes in pregnancy or it is likely due to trauma from labour and vaginal delivery. While the debate exists as to whether pregnancy or vaginal delivery is responsible, it is inconceivable that trauma to the pelvic floor during childbirth does not have some effect on the pathogenesis of postnatal SUI, itself a known risk factor for long-term SUI.[7]

18a.2.2 Risk Factors

Antenatal SUI is common and usually self-limiting but not in all cases. If such women could

be identified before delivery, prevention might be possible.

There are a large number of reports suggesting that antenatal SUI is a high-risk factor for postnatal and long-term SUI.[8,9] In which patients is this likely to persist after delivery? In one study of primigravidae women with antenatal bladder neck mobility (BNM) of more than 5 mm on perineal ultrasound (a possible marker for a weak pelvic floor) and who reported symptoms of SUI, they had a higher risk of postnatal leakage.[10] This suggests that these primigravidae women might have an underlying weakness of the pelvic floor, for example, connective tissue/collagen (the BNM was unlikely to be a pregnancy effect as it was seen in only 30% of the primigravidae women).

These women might be "constitutionally" at risk of SUI following vaginal delivery. Prevention with pelvic floor training in such women has shown positive results (see below). Other risk groups need to be identified so that similar preventative measures can be employed.

18a.2.3 Pathophysiology

It is not clear how labour and delivery affect the anatomical supports of the pelvic organs. However, it is likely that tears of the endopelvic fascia and the levator ani muscles and stretching of the pudendal nerve are important.

Fascial tears like any other connective tissue injury repair with in-growth of new connective tissue. However, this will not be as strong as the original, and with the weakening of connective tissue and fascia that occurs with ageing and menopause, these tears are likely to result in weakness of pelvic floor support, resulting in SUI and POP.

These conditions might occur even earlier in women with constitutionally weakened collagen/connective tissue. For example, Keane et al.[11] have shown that premenopausal nulliparous women with SUI have weaker pelvic floor collagen than controls, suggesting that there are individuals with congenitally weak connective tissue and fascia who might be at risk of SUI and POP as a result of pregnancy. If they could be identified before or early in a first pregnancy,[10] then prevention might be possible.

Levator ani trauma can also occur and defects have been identified. For example, on MRI scanning after a first vaginal birth.[12] Recovery has been noted between 2 and 6 months, but long-term follow-up is required to assess the clinical importance of these defects.

Pudendal nerve trauma has been demonstrated following vaginal delivery[13] and caesarean section late in labour (ie, at $>$ 8 cm cervical dilatation)[14] but not following elective caesarean section. Again, recovery appears to occur within 2 months of a first delivery[13]; but following subsequent deliveries, the nerve function does not return to normal, resulting in pelvic floor denervation.

18a.2.4 Obstetric Factors

What happens in labour to cause trauma to the fascia, muscle, and nerve? Forceps delivery[13] has been implicated, but this is not a consistent finding in relation to the onset of SUI. There are few data on the effect of Ventouse/vacuum delivery. It is likely, therefore, that instrumental delivery is not the cause but the reason. For example, primigravidae women with a larger baby in an occipito posterior (OP) position are known to be at higher risk of levator ani injury.[15] These and other risk factors, for example, short stature, long second stage of labour, or epidural anaesthetic might contribute to the indication for instrumental delivery. Episiotomy, while arguably having a protective effect on obstetric anal sphincter injury, has not been shown to affect pelvic floor strength or reduce the incidence of postnatal SUI.[16]

18a.2.5 Other Risk Groups

The obstetric risk factors mentioned above, for example, first baby, OP position, long labour, large birth weight, epidural, and so forth, are likely to be important particularly in women at risk of postnatal SUI, for example, those with antenatal and prepregnancy SUI, as mentioned above, and in those with a family history of incontinence in pregnancy. Obesity also is thought to be a risk factor for postnatal SUI.[17]

It might be that women at risk should avoid these obstetric risk factors. However, whether this should be by elective caesarean section is a matter of considerable debate.

18a.2.6 Prevention

While caesarean section might prevent pudendal nerve injury and postpartum SUI,[13,18] this finding is not consistent with studies showing up to 16% of patients still having SUI despite caesarean section.[19,20] This lends further support to the view that many women are at risk of urinary incontinence possibly due to an underlying weakness of the pelvic floor[11] and irrespective of parity.[21] Caesarean section might merely delay the onset rather than being preventative. However, no prospective randomised controlled trials of elective caesarean section versus vaginal delivery have been performed to see if caesarean is preventative, but secondary outcomes of the "term breech delivery" trial showed no difference[22] and the forthcoming "twins" trial might provide valuable data.

Pelvic floor muscle training (PFMT) is a less invasive and arguably more appropriate form of prevention. In at-risk primigravidae (eg, those with *antenatal* bladder neck mobility), antenatal PFMT has produced a reduced incidence of *postnatal* SUI compared with untreated controls.[23] Similar results have been seen in unselected primigravidae.[24] While supervised postnatal PFMT seems to be preventative in the short-term,[25] the long-term effects are not good, with a high relapse rate at 6 years almost certainly due to poor compliance.[26] Caesarean section is not completely protective and so PFMT should be considered initially until there are more data on the protective effects (or otherwise) of caesarean section. Motivation and supervision are necessary to help women comply with PFMT.

In summary, pregnancy and vaginal delivery are likely to affect the pelvic floor and result in long-term symptoms. However, follow-up studies are required. Nonetheless, it is likely that the defects noted in the endopelvic fascia, levator ani, and pudendal nerve are likely to become more obvious with the ageing process and the effects of menopause.

18a.3 Conclusion

The hypothesis is that urinary incontinence (in particular SUI) is the end result of pregnancy-related pelvic floor dysfunction. This might affect only those at risk, for example, those with inherently weak pelvic floor collagen/connective tissue. Also in women with an intact external anal sphincter and with pelvic floor damage, the puborectalis serves as an efficient compensatory closure mechanism preventing faecal incontinence. It is likely that the cumulative effects of multiple deliveries, progressive neuropathy/denervation, ageing, and the menopause overcome these compensatory mechanisms to produce symptoms. Identification of individuals at risk is required for prevention to be possible.

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18b
Childbirth and Anal Incontinence

JH Herbert

18b.1 Introduction

When a woman discovers that she is pregnant, it is highly unlikely that she will begin to think about whether she will have difficulty controlling wind or even getting to the toilet in time to open her bowels without soiling herself after she has had her baby. Even the slightly less taboo topic of leaking urine is not commonly discussed among pregnant women.

In today's world of computer technology, information about health and well-being is more accessible than ever. While there is a plethora of information available on the internet regarding a wide range of subjects related to pregnancy and childbirth, examining the top three websites found by a reputable search engine, one is unable to easily find information regarding pelvic floor muscle (PFM) injuries and the resulting symptoms of urinary and anal incontinence.

Even in a section entitled "pregnancy, labour, and birth complications"[1] incontinence is not mentioned and one can find information regarding this topic only by typing "incontinence" into the search engine within the site. So it is not surprising that some women who have the misfortune of developing anal incontinence after their delivery are shocked, unprepared, and sometimes angry at what has happened. It has been demonstrated that physical health problems, including urinary incontinence and bowel problems, have a significant effect on the development of postnatal depression.[2]

18b.2 Terminology

There is some difficulty in reviewing the literature because of a variation in the terminology used. Earlier articles about this subject tend to use the definition described by the Royal College of Physicians,[3] that of "faecal incontinence" referring to the loss of liquid or solid stool; however, more recent articles use the wider definition of "anal incontinence" to include the addition of the embarrassing symptom of inability to control flatus.[4] The inability to control flatus may be just as detrimental to a woman's quality of life as actually leaking stool depending on her activities; for example, a female solicitor whose job involves appearing in court may not be able to continue working because of the embarrassment of not being able to control flatus. It therefore is important that this symptom is included when discussing anal incontinence.

As a consequence of this difference in terminology it is difficult to compare prevalence rates. A systematic review of studies examining the prevalence of both faecal and anal incontinence in community living adults[5] quotes an estimated prevalence of anal incontinence of 2 to 24%, compared to a lower figure for just faecal incontinence of 0.4–15%. This systematic review commented on the general poor quality of study design with significant bias and therefore was unable to pool any of the results.

More specifically, looking at the prevalence of symptoms postdelivery, these have been quoted as ranging from 4%[6] when using the definition "faecal" incontinence to 8.6% when including incontinence of flatus.[4]

18b.3 Obstetric Trauma

Obstetric injuries to the anal sphincters have also been subject to a variety of definitions. The most comprehensive to date is the Royal College of

Obstetricians and Gynaecologists (RCOG).[7] This guideline document on the management of third- and fourth-degree tears following vaginal delivery divides obstetric tears into second-, third-, and fourth-degree. It defines these tears as follows:

- Second degree: injury to the perineum involving perineal muscles but not involving the anal sphincter.
- Third degree: injury to the perineum involving the anal sphincter complex [external anal sphincter (EAS) and internal anal sphincter (IAS)]
  - 3a less than 50% of EAS thickness torn
  - 3b more than 50% of EAS thickness torn
  - 3c IAS torn
- Fourth degree: injury to perineum involving the anal sphincter complex (EAS and IAS) and rectal mucosa.

Despite now having clear definitions of the degrees of injury to the pelvic floor complex at delivery there is still a problem of diagnosis. Immediately following a vaginal delivery, the perineum is likely to be oedematous and there usually will be a degree of haematoma. Under these circumstances it is often difficult to see the extent of the damage and to identify the external and internal anal sphincters.

18b.4 Prevalence of Obstetric Tears

There is also some discrepancy in the literature about the incidence of third- and fourth-degree tears. This could be due in part to the difficulties in diagnosis. The use of ultrasound, whether performed endoanally or using a transperineal technique, may increase significantly the detection rate and enable appropriate postdelivery referral for treatment.

Most papers appear to quote a prevalence of obstetric tears between 0.5% and 2.5%; however, some quote much higher figures. For example, in a case review of 2,078 deliveries, the incidence is reported as 4.4%.[8] The RCOG Guideline 29[7] suggests that the incidence may be as high as 9% in association with midline episiotomy, a practice until recently mostly associated with North America. If episiotomy is indicated, the approach now recommended is a mediolateral approach.[9]

Kalis et al.,[10] in a retrospective case-control study of 9,600 vaginal deliveries in their unit between January 1997 and March 2004, report an incidence of 0.84%; however, they stress the importance of a detailed knowledge of the structure and anatomy of the pelvic floor in those diagnosing these injuries. They suggest that the best evaluation of the perineal injury occurred when a urogynaecologist carried this out.

18b.5 Risk Factors for Obstetric Tears

If a woman has a vaginal delivery, there are a host of factors that over the years have been suggested to be a risk to sustaining an obstetric injury. Some of the factors are well documented and are quoted as significant in a number of studies[6–8,11,12]:

- Birth weight $>$ 4kg
- Persistent occipitoposterior position
- Primiparity
- Forceps delivery

However, even apparently well-established risks such as forceps deliveries have been challenged by some. A French team recently published a paper[13] that specifically looked at the prevalence of anal sphincter injury after forceps delivery. They concluded that due to the low incidence of tears in their population (13%), forceps delivery was not a significant risk factor and that the only factor with significant predictive value for anal sphincter injury was perineal tear. Other factors highlighted but not thought to be as significant[8,10,14–16] are shown in Box 18b.1:

Box 18b.1

Other factors

Epidural anaesthesia
Induction of labour
Second stage longer than 1 hour
Episiotomy
Ventouse delivery
Shoulder dystocia
Foetal macrosomia
Postdates
Maternal cooperation
Ethnic background

18b.6 Pregnancy or Delivery

There continues to be great debate among health care professionals as to whether it is just factors associated with delivery that contribute to the development of anal incontinence or whether pregnancy itself has a role to play. An interesting study of 184 primipara delivered by caesarean section and 100 delivered vaginally[17] demonstrated that there was no significant difference in the prevalence of symptoms of incontinence 10 months postpartum. These findings confirmed those of the Australian group[18] who reported the results of a survey of 4,400 households randomly selected and questioned about pelvic floor disorders. This study concluded that the prevalence of incontinence was not significantly reduced by caesarean section. It is suggested that during pregnancy, changes occur to the properties of collagen and other connective tissues. This, combined with possible inherited susceptibilities, may be responsible for the development of anal incontinence as a result of the pregnancy alone, regardless of the mode of delivery.[17]

Consideration should also be given to the morbidity associated with caesarean section, such as increased risks of postpartum haemorrhage, infection, pulmonary embolism, and ileus, with the risks of hysterectomy secondary to haemorrhage ten times higher than with vaginal delivery. There is also the serious risk of maternal mortality, which is thought to be considerably lower following vaginal delivery compared to caesarean section.[19]

18b.7 Implications for Practice

It is clear that the distressing symptoms of anal incontinence will affect a significant number of the female population, in particular those who have experienced childbirth, whether or not they have a vaginal delivery. Undoubtedly these symptoms will have a dramatic impact on their quality of life, affecting all aspects of their life from work, personal and family relationships, to social activities. It is essential that treatment is easily made available to these women. In the United Kingdom, the RCOG Guideline 29[7] states that all women who have had a third- or fourth-degree tear should be offered a follow-up appointment at 6–12 months by a gynaecologist with an interest in anorectal dysfunction or a colorectal surgeon. If symptomatic, they then should be investigated by endoanal ultrasound and anorectal manometry and should be referred to a colorectal surgeon for consideration of a secondary sphincter repair. It is of note that no conservative therapy is mentioned in this document; also by implication it is likely that the follow-up appointments will be in the acute care setting at the hospital, which may be a reason that many of these clinics report a high "did not attend" rate. This guidance also does not include services for women with second-degree tears or those who may have a "missed" third-degree tear.

There is a distinct gap in the literature of qualitative research looking at women's experiences following obstetric perineal trauma. It is essential that this is addressed and the information gathered used to influence the development of future obstetric and continence service provision to women in the childbearing year.

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19
Incontinence and the Menopause

DS Kapoor and RM Freeman

19.1 Introduction

While it is believed that menopause increases the risk of stress urinary incontinence (SUI), the causative mechanism is not fully understood. In a 7-year follow-up study menopause per se was not associated with an increased incidence of urinary incontinence; it was associated with other factors such as parity, obesity, prior gynaecologic surgery, and constipation.[1] In contrast, a study in a cohort of nuns found that the prevalence of urinary incontinence in nulliparous postmenopausal nuns was the same as parous postmenopausal women,[2] suggesting that oestrogen deficiency and muscle weakness might affect the pelvic floor irrespective of parity. Collagen weakness has been seen in menopausal women not on any hormone replacement therapy (HRT),[3] and other studies have shown a loss of mature collagen and an increase in synthesis of new collagen.[4]

19.2 Is HRT Preventative?

The evidence for HRT in treatment of SUI remains equivocal. A Cochrane review of the role of oestrogens for SUI found it to be useful only when combined with another agent (phenylpropanolamine). However, in terms of subjective perceptions, 50% of women in the oestrogen group felt their symptoms had improved compared to 25% in the placebo group; however, this was more evident in those with overactive bladder symptoms than SUI.[5] Some studies have shown that oestrogen and progesterone (combined) HRT[6] and oestrogen therapy in oral and transdermal forms *increased* the risk of urinary incontinence in postmenopausal women,[6] while another review found no improvement in stress incontinence in women on HRT.[7] Similarly, a multicentre trial failed to show any improvement with raloxifene (a newer form of HRT) over a 3-year follow-up.[8] In conclusion, the role of HRT for the treatment of SUI is doubtful, and any potential benefits must be balanced against the risks of long-term use of HRT.

19.3 Overactive Bladder

The role of pregnancy and the menopause in causing or worsening the symptoms of overactive bladder (OAB) remains unclear. Few studies exist on the incidence of OAB in pregnancy, but a recent study has shown a high incidence of frequency and urgency at 12 weeks' gestation with spontaneous remission in 12–22% in late pregnancy.[9] With ageing and oestrogen deficiency some symptoms can worsen; for example, nocturia, which has been shown to be more strongly related to ageing than to the transition from pre- to postmenopausal status.[10] However, nocturia can be related to nocturnal polyuria due to a relative deficiency of antidiuretic hormone (ADH).

A recent systematic review concluded that local oestrogen therapy was beneficial in improving all symptoms of OAB, that is, frequency, nocturia, urgency, and urge incontinence.[11] However, this may be due to reversal of urogenital atrophy rather than a direct effect.

19.4 Faecal Incontinence

Studies performed in identical twin sets found the menopause to increase the risk of faecal incontinence by more than 50%. Incidentally, the study also found that SUI in a woman doubled the risk of faecal incontinence.[12] These symptoms might be the end result of trauma during childbirth.

Oestrogen receptors have been identified in the external anal sphincter,[13] and improvement in faecal incontinence has been shown with HRT in one study.[14] Further studies are required.

19.5 Conclusion

The hypothesis is that urinary incontinence (in particular SUI) is the end result of pregnancy-related pelvic floor dysfunction. This might affect only those at risk, for example, those with inherently weak pelvic floor collagen/connective tissue. Also, in women with an intact external anal sphincter and with pelvic floor damage the puborectalis serves as an efficient compensatory closure mechanism preventing faecal incontinence. It is likely that the cumulative effects of multiple deliveries, progressive neuropathy/denervation, ageing, and the menopause overcome these compensatory mechanisms to produce symptoms. Identification of individuals at risk is required for prevention to be possible.

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20
The Athletic Woman / Women and Exercise

LM Southon

20.1 Introduction

Incontinence is not a widely reported problem for female athletes, and yet epidemiological research has shown it to be prevalent, if somewhat variable, in certain sporting activities. Clinically, continence therapists may not see many elite athletes but often see women who complain of incontinence after joining an aerobics class or bouncing on their child's trampoline. In some cases this then can become a barrier to further exercise.[1]

Some of the research in this field was completed over a decade ago and used relatively small cohorts of elite athletic women. The clinician, therefore, should take into account the methodologies used and the paper's relevance to current practice should be considered.[2] Clearly the question arises as to what makes some sports women more prone to incontinence than others. Is it merely the strength of the forces produced during a particular sporting manoeuvre or is it also a question of the individual's body makeup?

20.2 The Continence Mechanism and Continence Threshold

The continence mechanism (see Figure 20.1) is a term used to encompass the many factors contributing to a woman's continence. During exercise an increase in intra-abdominal pressure is generally produced. This in turn affects the fascia surrounding the bladder neck and urethra, support of which is critical in maintaining continence. The concept of a continence threshold was described as being a point at which a normally continent woman will be incontinent if only transiently because of increased biomechanical forces.[3] Trampolining is an example of a sport where the continence threshold is often breached due to the great increase in intra-abdominal pressure. Such are the forces involved that even nulliparous teenagers are not exempt from leaking.[4]

20.3 Factors Affecting the Continence Mechanism

20.3.1 Collagen

Connective tissues are made up of specialised cells and a matrix that is composed of protein fibres and a ground substance. It is the proportion and types of these components that dictate the properties of specific connective tissues. The composition and structure of connective tissues supporting the urinary system are thought by some to be important for an effective continence mechanism, but present research is conflicting as to the role that the various collagen fibres play in this process.[7,8]

Collagen is one of the body's most important fibrous proteins providing both strength and elasticity and is common in connective tissues. There are many different types of collagen fibres, although mainly types I and III are present in the body with type I predominating. Type III fibres are less rigid and more flexible and are more plentiful in those individuals who are more flexible. It may be that those people with greater flexibility are more likely to be encouraged into athletics and gymnastics because of their ability to perform certain manoeuvres, but this may predispose them to continence problems.[3]

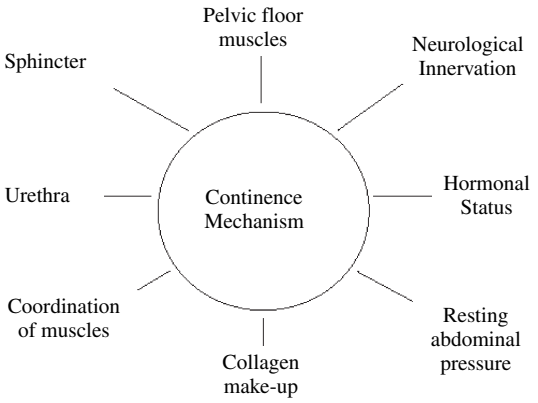

Figure 20.1. Factors influencing the continence mechanism (compiled from references 3, 5, and 6).

The function of connective tissue in the pelvic floor is varied; it supports the pelvic organs, helps dissipate forces through the pelvic floor, and transmits muscular forces onto the urethra. Between collagen fibres there are cross-linkages that act like glue to hold the whole structure together more rigidly, thus reinforcing the strength of the tissue. With maturity more cross-linkages are formed.[9]

If large forces are transmitted through the tissue, microtrauma may occur. Repair will ensue, but the remodelled tissue may be malformed and dysfunctional, making it less effective in conveying muscular forces to the urethra; hence promoting failure of the continence mechanism.[4,9] The process of regeneration of the connective tissue matrix is dependent on numerous factors; low oestrogen and low alpha-1 antitrypsin-inhibiting enzymes have been established in patients complaining of stress urinary incontinence (SUI).

20.3.2 Hormonal Influences

Many hormonal influences affect the urinary system and collagen metabolism. The effect of oestrogen is probably best known, although the pathophysiology remains unclear.[10,11] In women, higher basal levels of relaxin are found, which regulates the regeneration of the connective tissue matrix. During the adolescent period, a time when girls are often selected for intensive gym training, even higher levels of relaxin are found.[12]

In some studies female athletes have reported an incidence of hypothalamic amenorrhea (80% in some sports), possibly as a result of intense exercise, eating disorders, or other extrinsic factors.[11] Oestrogen levels may become low, and this then has an effect on the connective tissue and urinary system as a whole. Eating disorders also appear to lead to a higher incidence of urinary incontinence.[10]

20.4 Training versus Competition

The continence status of athletes has been reported to differ between training and competition. One study showed that 95.2% of subjects leaked in training, whereas only 51.2% leaked during competition.[13] A possible mechanism for this is through catecholamine, a neurotransmitter secreted by the adrenal gland and affecting the sympathetic system. It stimulates alpha receptors present throughout the body, which has an effect by increasing urethral tone and pressure, thus keeping the urethra closed.[13]

20.5 Forces

Large forces can be generated during sporting activity, for example, long jumpers can generate up to 16 times their own body weight on heel landings.[3] The pelvic floor muscles (PFM) therefore must be able to withstand the constant, repetitive downward force of the abdominal viscera such as that caused by running and jumping.[4] It appears that the lower extremities absorb forces as they are transmitted from the feet to the pelvic floor. Nygaard et al.[14] postulated that shock-absorbing footwear and load-dampening orthotics may help to decrease force transmission to the pelvic floor and thus decrease incontinence; however, there has been little new research to support this.

As mentioned previously some sporting activities appear to cause more incontinence than others. Some data from Nygaard et al[3] are shown in Figure 20.2 and raise some interesting questions. Why do tennis players leak more in daily life than when playing tennis and why does basketball appear to produce more incontinence than

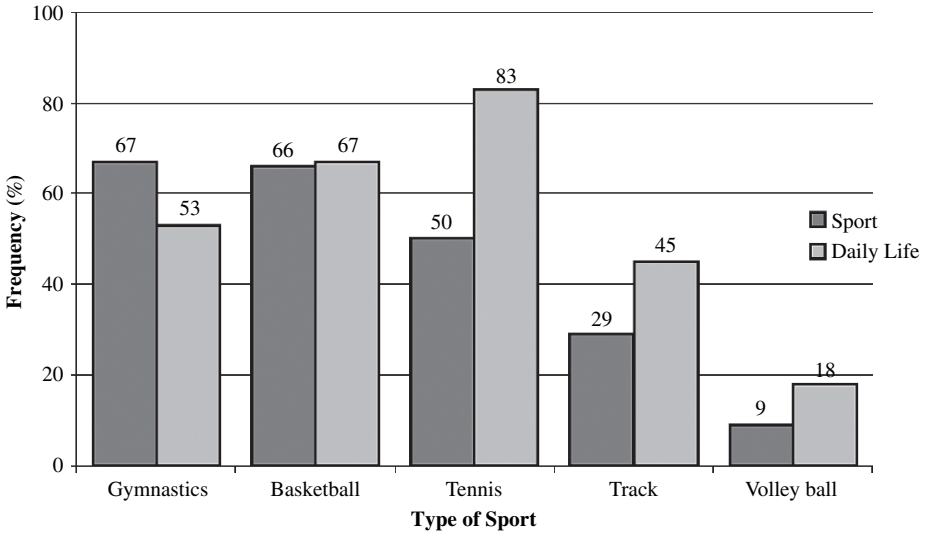

Figure 20.2. Frequency of reported urine loss during sporting activity and in daily life (modified from reference 3).

volleyball and yet both appear to be fast-moving games? Thyssen et al.[13] reported somewhat differing statistics, but the cohorts differed in composition (age and parity) and this highlights the difficulty of comparison between such research statistics.

20.6 The Role of the Physiotherapist

20.6.1 Pelvic Floor Muscle Exercises

The pelvic floor muscles counteract downward forces that occur with increased abdominal pressure and bladder compression and also aim to improve the urethral closure mechanism. Power, endurance, and timing are all important for this to be effective and many assume that athletic women will have a strong pelvic floor. However, Bo et al.[15] found this not to be the case with a small cohort of physical education students. Although the effectiveness of pelvic floor muscle exercises (PFME) has been established, little research has been done to assess the effects in athletes.[16]

20.6.2 Core Stability Exercises

Richardson et al.[17] described a capsule, made up of muscles of the pelvic floor, multifidus, transversus abdominis, and the respiratory, diaphragm, together with the lumbar vertebrae. Together they combine to help maintain the trunk posture in an ideal position and all act synergistically to give optimal stability and function. Sporting activity may encourage incoordination, and therefore the physiotherapist must consider a programme of muscle rehabilitation that incorporates more than just the PFM in isolation (see Chapter 11a).

20.6.3 Biofeedback and Electrical Stimulation

Both may be used as important adjuncts to PFM re-education (see Chapters 12a and 14).

20.6.4 Prosthetic Devices

Several intravaginal devices are available which act by producing an upward pressure in the area of the bladder neck. Studies have not looked specifically at use by athletic women, and there would seem to be a need to research these devices further. Anecdotally, sanitary tampons and the contraceptive cap also are said to be effective, but caution must be used as manufacturers do not recommend this usage and state that tampons should only be used during menstruation.[18]

20.7 Faecal Incontinence

Gastrointestinal symptoms can occur in runners and a few may experience faecal incontinence.[19] The symptoms can include the urge to defecate, increased bowel frequency, and diarrhoea during or following a run. However, in some cases it is not true incontinence as some runners would rather soil themselves than stop during a race.

Methods to manage and reduce these problems include:

- Never trying a new supplement or drink for the first time before a race
- Increase training volume and intensity steadily
- Keep well hydrated
- Consider dietary changes, for example, eating a low-fibre diet. Improved fitness through training reduces the blood shift away from the gastrointestinal tract; therefore, highly fit athletes tend to suffer fewer disturbances.[19]

20.8 Liaison with Coaches and Fitness Instructors

Some coaches and fitness instructors seemingly are ignorant of incontinence problems. It would seem obvious that there is a need to educate them with a view to perhaps modifying training regimens by shortening sessions to avoid fatigue of the PFM and only introducing highly provocative jumps slowly. Would this help the PFM to adapt? Further research is clearly needed.

20.9 Current Thoughts

At present there is no evidence to suggest that participation in regular, strenuous, high-impact exercise predisposes women to significant urinary incontinence or connective tissue damage later in life,[11,20] but strenuous activity may unmask incontinence in otherwise asymptomatic women.[3,11] Repeated perineal descent may lead to secondary changes in the anatomy of the urethrovesical neck.[4] Not all women who practise high-impact sports are incontinent, implying that absolute continence at high levels of intra-abdominal pressure is possible.[21]

20.10 Summary

Physiotherapists have an important role in assisting athletes to understand the mechanism behind the causes of incontinence and possibly to find a treatment programme to help them overcome it. They also can use this knowledge to help the woman who wants to exercise but finds that incontinence is a hindrance.

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21
The Male Patient

G Dorey

21.1 Introduction

Men may experience several types of urinary incontinence and lower urinary tract (LUT) symptoms, which include stress urinary incontinence, overactive bladder symptoms (urgency and urge urinary incontinence), mixed stress and urge incontinence, postmicturition dribble, extraurethral incontinence, continual incontinence, and functional incontinence. Stress urinary incontinence may occur after transurethral resection of prostate (TURP) for benign prostatic hyperplasia (BPH) or after radical prostatectomy or radiotherapy for prostate cancer, due to bladder neck sphincter damage. Urge urinary incontinence is associated with urinary frequency and nocturia. It may be due to neurogenic detrusor overactivity.

Lower urinary tract symptoms in men may be divided into storage, voiding and post micturition symptoms.[1] Storage symptoms include frequency, urgency and nocturia, urge urinary incontinence, stress urinary incontinence, mixed incontinence, and enuresis. Voiding symptoms are due to obstruction of the urethra by an enlarged prostate and include slow stream, intermittent stream, hesitancy, straining, and terminal dribble. Postmicturition symptoms occur immediately after micturition and include the feeling of incomplete emptying and postmicturition dribble.

Investigations including urinalysis, uroflowmetry, and urodynamics may identify the pathophysiology of the dysfunction (see Chapter 9a).

21.2 Assessment

Patient details should include age, occupation, hobbies, physical activities, dates and outcomes of TURP, repeat TURPs, radical prostatectomy, radiotherapy, urethral stricture division and abdominal or bowel surgery and LUT symptoms as shown in Box 21.1.

The duration of each symptom needs to be noted, plus the improvement or deterioration to

Box 21.1

LUT Symptoms

- Is there leakage on exertion during coughing, sneezing, shouting, rising from sitting, and bending?
- Are there symptoms of urgency, urge incontinence, frequency, nocturia, and nocturnal enuresis?
- Does he have difficulty starting and maintaining the stream and is the stream weak?
- At the end of the stream is there a terminal dribble?
- Does the bladder feel empty after micturition or does he perform a Credé manoeuvre or double void?
- Has he the sensation of both voiding and leaking?
- Is there postmicturition dribble?
- Is it painful to pass urine?
- Is the urine dark, smelly, cloudy, or blood stained?

Box 21.2

Further Questions

- How is the problem being managed at present [pads (type and number), appliance, leg bag, intermittent catheterisation, indwelling catheter]?
- What triggers the leakage?
- Any constipation or straining to defaecate? How many times a week does defaecation occur? Is there faecal urgency, faecal incontinence, or use of laxatives?
- Symptoms suggestive of prostatitis or cystitis?
- Known allergies to latex.
- Any respiratory problems; smoker?
- Medication: anticholinergics, alpha blockers, 5 alpha inhibitors, antiandrogen treatment or any other?
- Is there a neurological problem?
- Any erectile dysfunction? Can he maintain his erection? Does he wake with an erection? Does he have premature ejaculation?
- Position for urination?
- Mobility, dexterity, and access to the toilet?

date and the severity marked on a visual analogue scale (0–10) indicating from 0 = no problem to 10 = severe problem. Further questions may then be asked (see Box 21.2).

21.3 Examination

The patient should be given the opportunity to be chaperoned. The need for a digital anal examination is explained. If the patient declines, he may allow a perineal examination. Valid consent or refusal must be recorded. The patient should be lying on his back with two pillows under his head with knees bent and feet on the plinth with a paper sheet over his pelvis.

21.3.1 Perineal Examination

The skin condition is examined for redness, rashes, and infection in the penile, perineal, scrotal, and anal areas. Congenital abnormalities such as hypospadias, epispadias, enlarged testis, warts, haemorrhoids, or tumours are noted.

The patient is asked to tighten the anus as if to prevent wind escaping and feel a testicular lift and the base of the penis move slightly into his abdomen while the anal wink is observed. He is asked to cough which may produce leakage and then asked to cough while he is tightening his pelvic floor muscles (PFM) to prevent leakage. The S2, 3, 4 dermatomes and myotomes are tested if neurological impairment is suspected.

Table 21.1. Assessment of male pelvic floor muscle strength.[2]

Description	Grade	
Nil	0	No muscle contraction
Flicker	1	Muscle flickers
Weak	2	Weak contraction with no movement
Moderate	3	Moderate contraction with movement
Good	4	Good contraction against resistance
Strong	5	Strong contraction against strong resistance
Very strong	6	Very strong squeeze gripping finger tightly

21.3.2 Digital Anal Examination

The external anal sphincter is digitally assessed at rest and during a maximum voluntary contraction using a well-lubricated gloved index finger, which is gently introduced into the anal canal while the patient is instructed to bear down as if releasing flatus. The patient is asked to contract the anus and hold for 5 seconds, while the therapist grades the strength of the contraction and notes the duration of the hold in seconds. While the patient is relaxing, the examining finger is introduced to 3–4 cm from the meatus and the anterior pull of puborectalis gently felt at the anorectal angle and graded 0–6 for muscle strength, for the duration of the hold, and for the ability to perform a fast contraction[2] (see Table 21.1).

21.4 Treatment

21.4.1 Patient Education

All treatment commences with an explanation of the problem and the treatment options available. Basic anatomy and physiology can be demonstrated using a model showing the male PFM.

21.4.2 Stress Urinary Incontinence

If the bladder neck sphincter is damaged, continence relies on an effective external urethral sphincter and PFM. The amount and progression of exercise is determined by digital anal examination. Men should tighten and lift the PFM strongly as in the control of flatus and feel a slight movement of the penis into the body and a testicular lift and hold the contraction for up to 10 seconds (see Figure 21.1).

Initially PFM exercises are taught and practised in a lying position. They are then progressed into functional positions and activities including sitting, standing, and rising from sitting.

"The knack" of tightening the PFM before and during activities that increase the intra-abdominal pressure such as coughing, sneezing, rising from sitting, or lifting[3] should also be taught. Further encouragement should be given to contract the PFM up to 50% of maximum while walking to enhance postural support and endurance (see Chapter 11a). Research has also shown that a small number of maximal PFM contractions twice a day are effective in increasing PFM strength in men with erectile dysfunction.[4]

Biofeedback such as digital, manometric, electromyographic (EMG), and real-time ultrasound can encourage greater muscular effort (see Chapter 12a & 12b). The patient is placed in supine lying with knees bent and apart and a paper sheet over his pelvis and able to view the monitor screen. The anal pressure probe is covered with a condom, lubricated with gel, and approximated to the patient's anus. The patient is asked to bear down as if releasing flatus while the probe is gently inserted and held.

The anal EMG probe is for single-patient use and applied using lubricating gel while asking the patient to bear down. An anal probe such as the Anuform™ (Neen Health Care, UK) allows ambulatory use. Alternatively, two small self-adhesive surface electrodes may be placed longitudinally over the pubococcygeus and ischiococcygeus muscles 1cm lateral to the midline. A third reference sensor is placed over a bony point such as the sacrum or coccyx.

Electrical stimulation may benefit patients with weak PFM. Suitable electrical parameters should be used (see Chapter 14), using a single-patient use anal electrode or by surface electrodes on the coccyx and perineum or either side of the perineal body. Electrical stimulation is contraindicated for patients who have cancer or after radical prostatectomy.[2]

Men need a full continence assessment before receiving pads or appliances. The penile clamp may cause pressure necrosis to the penis and is not recommended.

21.4.3 Urge Urinary Incontinence

Men with urge urinary incontinence are advised to perform urge suppression techniques. These include keeping calm, sitting down or standing still, and waiting 1 minute until the urge disappears. Once the urge has abated, patients are advised to walk calmly to the bathroom or continue their activities. They are advised not to rush to the toilet midurge, as they may leak urine.

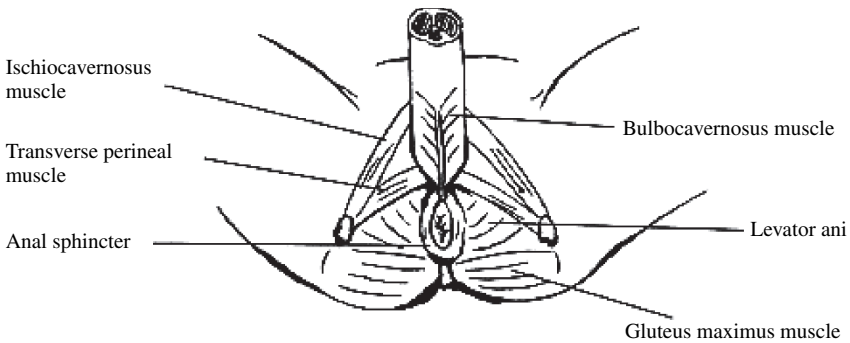

Figure 21.1. Male superficial pelvic floor muscles.

Urge urinary incontinence may also be treated with biphasic electrical stimulation at a frequency of 5–10 Hz and 200 microseconds pulse width for 20 minutes, although there is no good research to support this in men.

Lifestyle changes include weight reduction, fluid adjustment, bowel management, and smoking reduction[5] if necessary. Both prescribed and over the counter (OTC) medication should be reviewed; anticholinergics may be prescribed for men with severe urge urinary incontinence (see Chapter 25).

21.4.4 Postprostatectomy Incontinence

Postprostatectomy incontinence should be treated according to the symptoms of stress urinary incontinence, urge urinary incontinence, or mixed incontinence. Pelvic floor muscles should be strengthened prior to prostatectomy.

21.4.5 Postmicturition Dribble

A strong postvoid PFM contraction replaces or reinforces the normal postvoid reflex bulbocavernosus muscle contraction, which empties urine from the bulbar urethra and supersedes bulbar urethral milking.[6]

21.4.6 Chronic Retention of Urine

Clean intermittent self-catheterisation may be necessary for patients with incomplete emptying (see Chapter 23). A suprapubic catheter may be more acceptable for permanent catheterisation.

21.4.7 Functional Incontinence

Functional incontinence is caused by mobility and dexterity difficulties and is treated by social care, environmental, lifestyle and clothing adaptations (see Chapter 22).

21.5 Conclusion

PFM exercises and advice can improve storage and postmicturition symptoms and erectile dysfunction in men. Men with stress urinary incontinence are advised to perform "the knack" of tightening during exertion. Urge suppression techniques are advised for men with urge urinary incontinence.

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22
The Occupational Therapist's Approach to the Management of Incontinence

JM Vickerman

22.1 Introduction

Occupational therapists (OTs) have not traditionally been viewed as having an active role to play in the management and treatment of incontinence. While it has become increasingly accepted that the future lies with the inter/multidisciplinary approach, the practice of OTs specifically working in continence promotion remains a new but developing concept.[1]

Recent national guidelines[2,3] have reinforced the need for integrated services in many aspects of health care, including continence promotion. They have highlighted the importance of a more rehabilitative approach to continence problems and a move away from the compensatory model of "pads and pants."[4] It has become increasingly clear that an individual's functional ability can impact on the management of their toileting needs, and that improving function generally often can improve the situation for these individuals.[5,6]

22.2 What Can OTs Offer?

Occupational therapy assists people of all ages to achieve health and life satisfaction by improving their ability to carry out the activities that they need to do or choose to do in their daily lives[7]. Therapists use their skills of breaking down activities into their individual components to assess the individual's abilities. These components may include cognition, physical dexterity, balance, perception, sensory needs, upper limb function, emotional demands, and proprioception. Therapists will then work with the individual to acquire skills, adapt activities or the environment, or use compensation with the use of equipment to enable them to function to their maximum potential.[8]

OTs, therefore, focus on enabling people to perform everyday activities. Maintaining one's own toileting needs is one of the most basic of everyday activities, and therefore should be an integral aspect of a continence or functional assessment.[1]

In reporting the results of a joint survey of continence advisors and OT managers, Pomfret[9] concluded that OTs have a strong role to play "given that many problems relating to continence care are not physical but relate to mental state, environment, clothing, functional ability and carers difficulties."

22.3 Why Assess Function?

An individual's functional ability is an important consideration when assessing their continence needs. This is true whether the problem is caused by dysfunction or a physiological cause is identified. Where a physiological cause is identified, consideration must be given to functional factors, as these can have a significant impact on the success or otherwise of any treatment. It is also important to consider that where continence products are used, the individual has the functional ability to

Box 22.1

- Transfer ability: from bed to chair, standing to sitting (independent or with assistance).
- Mobility: walking with or without aids or support.
- Balance: standing without help and ability to wipe self and adjust clothing.
- Arm strength and body flexibility.
- Manual dexterity: ability to adjust clothing and fasteners.
- Eyesight: adequate vision to locate the toilet unaided.
- Toileting ability: ability to wipe self or manage any continence products.

manage them. This assessment will aim to identify the problem areas, develop solutions, implement changes, and evaluate progress.

Functional assessment related to continence should include consideration of both the physical and cognitive function of the individual. This information then should be put into the context of the individual's environment and social support network. According to Williams and Gaylord,[10] a functional assessment should include the factors shown in Box 22.1.

22.4 Specific Aspects of Assessment

Developing on from the Williams and Gaylord[10] model, it may be useful to consider specific areas where an OTs expertise could be of particular value in promoting continence.

22.4.1 Toilet

22.4.1.1 Height

Assess whether the height allows for safe and easy sitting and standing. Toilet heights can be increased using raised toilet seats. Care must be taken here to ensure that the individual is still able to adopt an effective defaecation position. Consider rails both around the toilet and / or on the adjacent wall if necessary. Alternatively, a height adjustable, combined toilet raise and frame could be used to assist with rising.

22.4.2 Location

Assess the accessibility of all toilets within the environment. Rails may be needed beside any outside steps. Assess the general condition and safety of steps or paths. It is important to consider the distance of the toilet from the main living areas. Note if there are any day or night variances that could influence the individual's ability to reach the toilet.

22.4.3 Space

Ensure that there is sufficient space to turn around in the toilet with any mobility aids, wheelchair, or personal assistance. If space is limited or likely to compromise safety, a commode may be a useful alternative.

22.4.4 Environment

Ensure that all toilets offer privacy and protect the individual's dignity. This is particularly important in hospital or care setting. Ensure that doors can be securely closed and that the room is well-lit and warm.

22.4.5 Bedroom

- The bed should be of a correct height to allow safe mobility when getting in and out of it. If the individual uses a wheelchair, the bed height should equal the wheelchair height. Nighttime commodes should be the same height as the bed. A bed-attached commode is an alternative option, especially where the individual is unsteady or has previously experienced stumbles.
- Urinals for both men and women are valuable where mobility is severely restricted, safety is compromised, or assistance is needed but is not available.
- Equipment to assist the individual to sit up in bed can be useful if this presents a problem. Various styles of rails, pillow lifters, and mattress elevators are available.
- The position of the bed within the room should be noted to see if alternative positioning could make transfers easier and safer or shorten the distance to the toilet.
- Bedding style sometimes can contribute towards difficult nighttime transfers. The use

of lightweight bedding will allow for easier movement.
- Lighting: The use of "touch lights" or low-wattage bulbs in the bedroom and route to the toilet will reduce the chance of nighttime falls or stumbles.

22.4.6 Living Area

Care should be taken to ensure that the individual is using a chair that suits their overall height and ability. The effort of rising from a low chair when experiencing urgency or stress incontinence often can exacerbate the problem. Many different styles of chair raisers are available to reduce the effort involved in this activity. The use of additional cushions should be discouraged, as this impairs the effective use of chair arms to assist standing. Specialist "riser chairs" also are available for individuals who find this movement difficult.

22.4.7 General Issues

22.4.7.1 Access to the Toilet

Ensure that the route to the toilet is easy to negotiate. If mobility is impaired, consider the provision of support rails on walls or stairs to aid safety. While stair lifts are a valuable resource when mobility is difficult, consideration should be given if the individual has any degree of urgency, as they are slow to travel.

22.4.7.2 Flooring

It is important to check the condition of the flooring in all rooms used by the individual. Any tears or loose mats can be hazardous. Walking on cold surfaces such as tiling with bare feet has been known to produce spontaneous bladder voiding in some individuals.

22.4.7.3 Footwear

Firm, well-fitting shoes with nonslip soles promote safe mobility and increase confidence.

22.4.7.4 Clothing

Clothing that is loose, easy to unfasten, or manoeuvre promotes continence. Changing the style of clothing or adapting existing clothing and underwear could make using the toilet or a urinal much easier. Difficult fastenings can be replaced with strips of Velcro without altering the outward appearance of the garment.

22.4.7.5 Fluid Intake

While a general continence assessment will always consider the amount and type of fluids an individual is consuming, it is important to assess whether these are being limited because of functional problems such as poor dexterity. Alternative ways of providing drinks may include having a flask of water or cordial prepared and left to drink throughout the day.

22.4.7.6 Medication

When reviewing the medication that the individual takes, it is important to assess whether they actually can access the medication. Some modern packaging can be difficult to open. Alternative styles are available if discussed with the supplying pharmacist. Where an individual has difficulty remembering to take their medication, prompt charts, tablet organisers, or alarm reminders can help.

22.4.7.7 Eyesight

It is essential to check that the individual is able to see well enough to take their medication, adjust clothes, walk to the toilet, avoid hazards, and so forth.

22.4.7.8 Lighting

Light switches in the principal rooms should be easy to find and operate. Heights of switches can be positioned to suit individual needs. Many different styles of switches are also available. For example, rocker switch, automatic sensors.

22.4.7.9 Hygiene

The ability to clean oneself after using the toilet is an important aspect of assessment. Long-handled wipers are available or a portable bidet may be required. Alternative types of toilets could be considered for people who have a long-standing problem with cleansing.

22.4.7.10 Cognitive Function

It is important to assess whether the individual has the capacity to interpret body signals for when they need the toilet, whether they can understand verbal or written instructions, and whether they are sufficiently motivated to engage in recommended treatment strategies and are orientated enough to locate the toilet or ask for assistance. Where an individual is disorientated, clear visual signs on toilet doors (eg, photographs) often can help.

22.4.7.11 Social Support

The support network of the individual always should be taken into account. Do they live alone or with family members or have carer support? Are there day- and nighttime variances that may exacerbate a problem, for example, someone who lives alone, is able use the toilet independently during the day, may be unable to get in and out of bed during the night to use the toilet without assistance.

22.5 Conclusion

For OTs to practice holistically, it is essential that they consider all aspects of their client's functional ability to carry out their activities of daily living. Maintaining continence is an activity fundamental to the independence and quality of life of the individual. It is essential that this be considered an integral part of the OTs' role.[1,4]

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23
Catheter Care

AR Winder

23.1 Introduction

Indwelling urinary catheters often have a reputation of being an easy way to manage urinary problems.[1] In certain circumstances this form of management is adequate, providing the only safe way to ensure bladder drainage.[2] Unfortunately, in reality, this form of bladder management frequently has associated problems, specifically in the long term. It is an invasive procedure that carries a high risk of infection, trauma, and other associated catheter problems.[3]

Problems include catheter blockage, leakage, urethral erosion, infections, and stone formation.[4] Septicaemia can occur, even resulting in death.[3] Therefore, this form of management must be treated with respect and only used as a last resort.

23.2 Urethra

Prior to catheterisation it is important to consider the anatomy of the urethra to avoid unnecessary trauma. In particular, the urothelium lining the urethra is composed of unequal and changing folds. Underlying this mucosal layer is a vascular layer that is easily damaged during invasive procedures. Therefore, it is important to use a lubricating gel when catheterising to reduce trauma.[5]

In postmenopausal women with atrophic vaginitis topical oestrogen therapy can alleviate the sensory symptoms of urgency and also can reduce the re-occurrence of infection.[6] It is worth considering such treatment in those women with a urethral catheter that is causing discomfort.

23.3 Assessment

An essential part of any catheter care is to engage the patient/relative at an early stage. First for consent, but also to give them the opportunity to discuss all aspects of their management. Key elements that often are overlooked are the patient's cultural, religious, and sexual beliefs[7,8]; in certain cultures food and drink is forbidden during certain religious festivals. The emptying of drainage bags and the technique of self-catheterisation could hold additional complications.[9] It is everyone's responsibility that a thorough holistic assessment (see Box 23.1) is carried out before any form of catheterisation is performed.[10]

Many urinary tract infections (UTI) are linked to the insertion of an indwelling urethral catheter.[11] The reasons for catheterisation should be documented. Unfortunately in some cases community staff are either not informed or there

Box 23.1

Checklist prior to Catheterisation

- Patient's micturition problem, plus reasons for catheterisation.
- Cultural and religious beliefs
- Sex, sexuality
- Support (relatives/carers)
- Medical history (medication/constipation)
- Fluid intake precatheter insertion
- Patient knowledge and valid consent

is a delay in information being received, thus placing the patient at risk.

A multiprofessional approach is often overlooked in this type of assessment. Occupational therapists, physiotherapists, and adult care services should be involved in appropriate care planning.

23.4 Infections

In a study of patients with chronic infection, 14% had infections with bacteria significally less sensitive to the common antibiotic.[12] Bacteremic UTI in the older age group was associated with a 33% mortality rate.[12] Most catheter-associated UTIs (CAUTI) are asymptomatic.

Infectious organisms utilise both extra- and intraluminal routes, most commonly via the patient's own colonic and perineal flora, or from the hands of health professionals.[13] Prolonged catheterisation $>$ 6 days increases the risk by nearly sevenfold and by 30 days infection is universal.

Scrupulous hand washing is a key factor and there is no evidence that meatal cleansing or the use of antiseptic agents added to drainage bags prevents this risk. Neither is the use of catheters impregnated with antimicrobial agents beneficial; in fact, this may increase the potential risk of resistant bacteria.[14]

23.5 Methods of Urinary Catheterisation

There are three main ways to drain a bladder.

- Urethral catheterisation
- Suprapubic catheterisation.
- Intermittent catheterisation

The advantages and disadvantages of urethral and suprapubic catheterisation are shown in Table 23.1.

23.5.1 Suprapubic Site Dressings

Dressings need only be applied to the incision site around the catheter tube for 24–48 hr after the initial insertion. It has been shown that long-term use of a prophylactic dressing can result in infection of the site and is not recommended.

23.6 Intermittent Catheterisation

Intermittent catheterisation (IC) reduces infection hazards and greatly improves the quality of life of patients with micturition disorders.[15] It is now recognised as the safest method in managing patients, especially those with neurogenic bladder problems.[16] Professionals who teach this technique must be competent and understand

Table 23.1. The advantages and disadvantages of indwelling catheterisation.

	Advantages	Disadvantages
Urethral	• Nonsurgical procedure • Patients/carers can be trained to self-care • Ability to use with various pathologies, eg, tumours/haematuria • Immediate drainage in an acute situation (CVA/trauma) • May be used intermittently with other management	• Urethral damage (stricture) • Discomfort (infections/atrophic vaginitis) • 50% indwelling catheters are problematic • Infection and associated risks high • Bladder calculi • Problems for patients who have poor physical or cognitive abilities
Supra-pubic	• Protects the integrity of the urethra and the bladder neck • Greater patient comfort • Freedom to have a sexual relationship • Catheters can be clamped off to enable voiding patterns and residuals to be measured • Frequent "blockers" can use larger- gauge catheters • May use in those who persistently expel their urethral catheters	• Minor surgical procedure, with a risk of bowel perforation • Patient must have a bladder capacity of 300 ml to enable the procedure to be viable • Altered body image • Infections, blockage, and calculi as prevalent as with urethral catheters • Occasional infection and over- granulation of the insertion site • Not recommended in those with various pathologies, eg, haematuria, tumours, obesity

Box 23.2

Possible Uses for IC

- A voiding dysfunction leading to significant postvoid residual greater than 30% of the total bladder capacity.[18]
- Bladder outflow obstruction with or without detrusor hyper activity.[19]
- Retention in premenopausal women.[20]
- An enlarged prostate, either benign or malignant.
- Previous surgery involving the bladder neck (eg, colposuspension).[21,22]
- Urethral strictures.[23]
- Neurological disorders (multiple sclerosis/diabetes).[24]

the complexities of this practice, which often can have hidden psychological and physical issues. For example, hidden sexual abuse can often be raised during this intimate teaching process. Patients must be motivated with sufficient dexterity and capability to understand the principles of the technique. It is important to remember that this can be either a complete or partial way to manage an individual's bladder.[17] Additional micturition problems for which intermittent catheterisation could be beneficial are shown in Box 23.2.

23.7 Catheters, Valves, and Bags

When selecting any urethral catheter, the smallest diameter should be used that allows adequate drainage. The routine sizing recommended for an adult is 12 to 18 Ch.[25] However, when the route is via a suprapubic incision, a larger gauge can be used, up to a 20 Ch.[26] The retaining balloon of catheters should be between 5 and10 ml.

Despite the years of research there is no one type of catheter that is impervious to blockage. The consensus of professional opinion is that the best way to manage the "blockers" is by observing when they block and routinely changing the catheter prior to this time.

The "natural" flushing of the catheter is the most beneficial way to prolong the "life" of a catheter, but this depends on the patient having an adequate fluid intake. The use of a catheter valve in some patients has contributed toward the flushing effect,[27] with an advantage of not having the problems associated with body-worn urine drainage bags.

There are many types of drainage bags, drainage taps, support systems, valves, and gadgets to assist with the patient's independence. Nurses, continence specialists, and occupational therapists all can help with appropriate selection.

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24
Surgery for Urinary Incontinence

MG Lucas and S Bjornssen

24.1 Introduction

When conservative treatment of incontinence has failed, it is usual to consider surgical therapy. The objective of all surgery for incontinence should be to restore the patient as closely as possible to normal function with the minimum short- and long-term morbidity and for this improvement to be durable. Patients usually expect that an operation will restore normal bladder function, but in reality this is unusual and they should be aware that surgery also carries risks and potential complications that may be lasting. Any procedure may cause voiding difficulty and sometimes thought should be given to preoperative teaching of intermittent self-catheterisation.

This chapter will describe the variety of surgical approaches to urinary incontinence, which range widely in their efficacy and morbidity. All operations for incontinence aim either to reduce bladder-filling pressures or to augment urethral closure. It makes no difference what the underlying aetiology is and the procedures can be applied, in principle, to neuropathic and nonneuropathic patients alike. However, it is vital to see the bladder problem in the context of the whole patient and their underlying disorder. The wheelchair-bound patients with degenerative disease cannot be treated the same as fully ambulant patients who are planning for 30 or 40 years of improved function.

Many procedures have been described, although most are consigned to history. Nevertheless, patients will still be seen who have undergone these operations, so some will be described later in the chapter in Table 24.1.

24.2 Surgery for Stress Incontinence

The reported outcomes of procedures are difficult to interpret because both populations and outcome measures are usually poorly defined and surgical technique varies widely from one surgeon to another. There have been very few high-quality trials with long enough follow up for conclusions to be drawn about the "best" procedure. The operations commonly used fall into two broad categories: those that aim to support the bladder neck or urethra and those that aim to occlude the urethra.

24.2.1 Supportive Operations

The Burch colposuspension aims to support the urethra by suturing the anterior vaginal wall upward to the back of the pubic arch. Until recently, it was the commonest surgical treatment due to its high success rates (85% at 5 years) and longer reported follow up than other procedures. It is, however, a major operation with a hospital stay of 5 days and at least 6 weeks recovery time. Complications include voiding difficulties (10.3%), de novo detrusor overactivity (17%), and posterior pelvic organ prolapse (14%).[1,2]

Laparoscopic Burch colposuspension avoided a large incision, offering shorter hospital stay, and quicker recovery and return to normal activity. The laparoscopic approach is more difficult and in an assessment of eight trials there was evidence of poorer outcome on long-term follow up and objective testing.

Slings of autologous fascia have been placed under the urethra for many years and reported

Table 24.1. Obsolete operations.

Stress incontinence operations	Principle of the operation	Reason for obsolescence
Marshall–Marchetti–Krantz Needle suspensions (Raz, Peyreyra, Stamey, Gittes) Aldridge sling	Paraurethral support	Risk of osteomyelitis of pubis Poor long-term outcomes
Anterior colporhaphy (anterior repair)	Correction of cystocoele	Operative morbidity Poor long-term outcomes
Teflon /marlex /silastic slings	Paraurethral support	High erosion rates
Periurethral Teflon injection	Urethral occlusion	Teflon embolism
Operations for detrusor overactivity	**Principle of the operation**	**Reason it has been abandoned**
Sacral root rhizotomy	Central division of all motor roots to bladder	Poor long-term outcomes for all these
Indlemann–Sundberg operation Bladder transection (open or endoscopic) Prolonged bladder distension Perivesical phenol injection	Peripheral interruption of motor innervation	

long-term outcomes have been similar to colposuspension, although with wider confidence limits.[1] In the past they were placed under tension, and voiding difficulty was common. Recently these have been placed with little tension and as shortened suspended fascial strips to minimize postoperative morbidity.

The newer suburethral tension-free vaginal slings [tension-free vaginal tape (TVT)], introduced and typified by Gynecare TVT in 1996,[3] now are widely used and have been recommended by the National Institute for Clinical Excellence[4] as first-line treatment alongside colposuspension in women for whom conservative management has failed.

TVT is inserted vaginally through a 1-cm incision, the needle is inserted on either side of the urethra and advanced into the retropubic space with an exit point through the skin suprapubically (see Figure 24.1). It has been suggested that the mode of action for TVT is to stabilize the mid-portion of the urethra at the time of increased abdominal pressure without modifying cervicourethral mobility. Insertion of TVT can be performed as a day case under local anaesthesia.

TVT has a success rate of 85%, 5% urgency, and 2% voiding disorders requiring release of tape. In a large randomised trial on TVT versus open Burch colposuspension, a similar success rate was achieved for both procedures. TVT was performed under local anaesthesia, colposuspension was performed under general anaesthesia. The Burch colposuspension group had a longer stay in hospital, a higher rate of self-catheterisation, and requirement for prolapse surgery. The TVT group had a higher rate of bladder perforation.[5]

The blind passage of the TVT needle through the retropubic space can result in damage to organs such as bladder, urethra, vessels, and bowel. To avoid these complications, alternative approaches have been developed and the one that stands out is the transobturator approach. Delorme and colleagues[6] described their technique where the tape is inserted through the obturator foramen from the thigh fold toward the mid-urethra.[6] A modification of this technique, that is, starting the passage of the needle from the vaginal incision and out through the obturator foramen introduced by de Leval,[7] may further reduce the risk of damage to the urethra. The passage of the needle is below the level of the levator muscles and enters neither the retropubic space nor the pelvic cavity (see Figure 24.2). Success rate with the trans-obturator technique is similar to the retropubic insertion of TVT with apparently lower risk of bladder perforation. Although preliminary results are promising for the transobturator approach, further evaluation is required and the technique needs to be compared with the retropubic approach in well-controlled clinical trials.

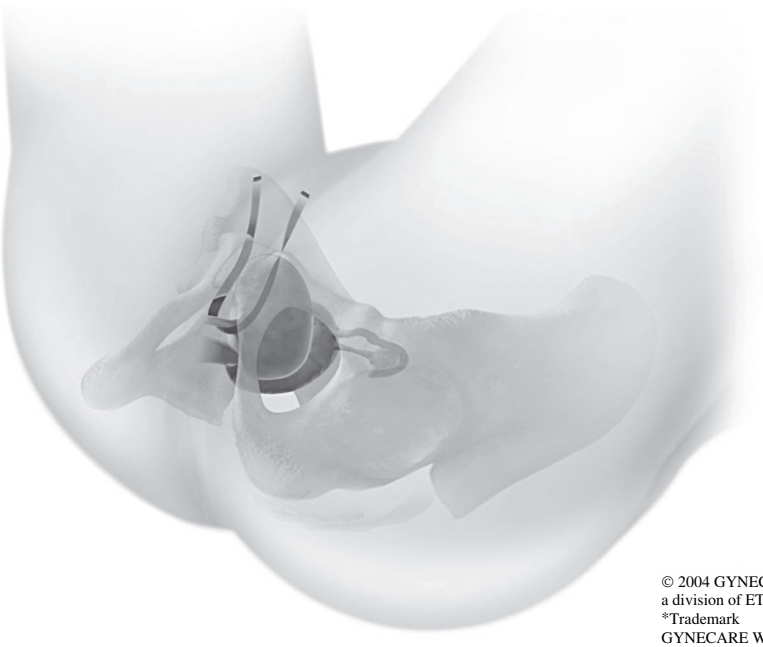

Figure 24.1. Drawing showing the tension-free vaginal tape placed around the female urethra via a suprapubic route (TVT). Image reproduced with the permission of Ethicon Womens Health & Urology, a division of Johnson & Johnson Medical Limited.

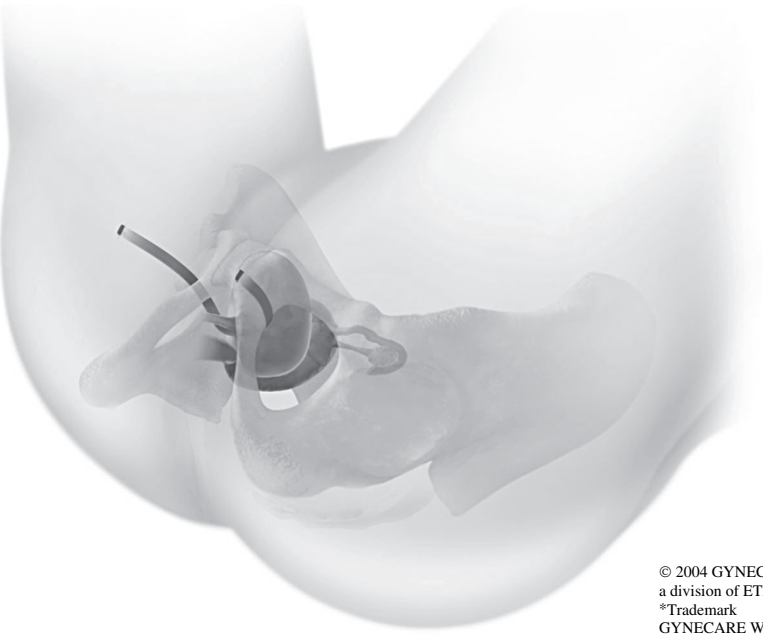

Figure 24.2. Drawing showing the tension-free vaginal tape placed around the female urethra through the obturator foramina (TVTO). Image reproduced with the permission of Ethicon Womens Health & Urology, a division of Johnson & Johnson Medical Limited.

Since the development of Gynecare TVT, the market has been flooded with similar devices using different applicators and different mesh types. Each mesh type should be evaluated and its use backed up by research into its biomechanical properties and incorporation into human tissue. Most of the new urethral sling procedures have little scientific support, but they need to be compared with standard procedures such as the Burch colposuspension and Gynecare TVT to establish their role in the surgical treatment of urinary stress incontinence.

24.2.2 Occlusive Procedures

24.2.2.1 Bulking Agents

A variety of bulking agents can be injected endoscopically into the urethral submucosa distal to the bladder neck to create artificial urethral coaptation and restore continence. Reported success rates vary with material used, but overall there is a high recurrence rate with a fall down to 50% cure rate at 2-year follow up.[8] The procedure can be performed under local anaesthesia and has a low morbidity. It may have a role in elderly women and for those who are unfit for surgery.

The artificial urinary sphincter is a device that features a circumferential periurethral/bladder neck cuff that is maintained constantly inflated by a pressure- regulating balloon, and a control pump inserted in the scrotum or labia allows complete cuff deflation for voiding (see Figure 24.3). Used primarily for men with postprostatectomy incontinence, the device is also implanted for patients with neurogenic bladder dysfunction, women after failed surgery for stress incontinence, and in conjunction with major reconstructive bladder surgery.[9,10]

Unfortunately the device does not respond well to stress, so if a patient can exert a high enough abdominal pressure, they will still leak; not difficult for obese patients. Complications include implant infection, erosion of the cuff, leakage of the device or pump failure, and atrophy of the urethra within the cuff, which results in progressive incontinence. Device failure requires further surgery to correct the problem. It is expensive and repeat operations to correct problems are required in about 50% of patients.

24.2.3 Urinary Incontinence and Concomitant Genital Prolapse

Many women who present to a urogynaecology clinic may have concomitant prolapse and/or failed previous continence and prolapse operation. Over 40% of women with stress incontinence have a coexisting cystocele. Colposuspension traditionally is performed to correct both but has been shown to have a high failure rate of up to one third for the correction of anterior vaginal wall prolapse. It is not clear what type of surgery is best for this group of women. They need careful assessment and investigations, and it would be sensible to refer them to surgeons with a special interest in reconstruction as the failure rate with secondary procedures is high.

24.3 Surgery for the Overactive Bladder

Only a very small percentage of patients with overactive bladder syndrome will prove to be so refractory to noninvasive treatments that surgical options have to be considered. It follows that one would never consider surgery unless all conservative options have been tried. The principles of surgical treatment have included:

- Paralysing the bladder
- Augmenting its capacity
- Modulating its activity
- Diversion of the urine

24.3.1 Paralysis

Paralysis was achieved through attempted denervation of the bladder. Devervation techniques enjoyed a zenith of popularity in the 1980s but soon were found to be disappointing in long-term outcomes. The rationale was to interrupt the sacral reflex on which the propagation and continuation of a detrusor contraction depends. This can be achieved theoretically either in the sensory or the motor side of the reflex and both have been tried, but the long-term results were universally poor. The range of obsolete procedures is shown in Table 24.1.

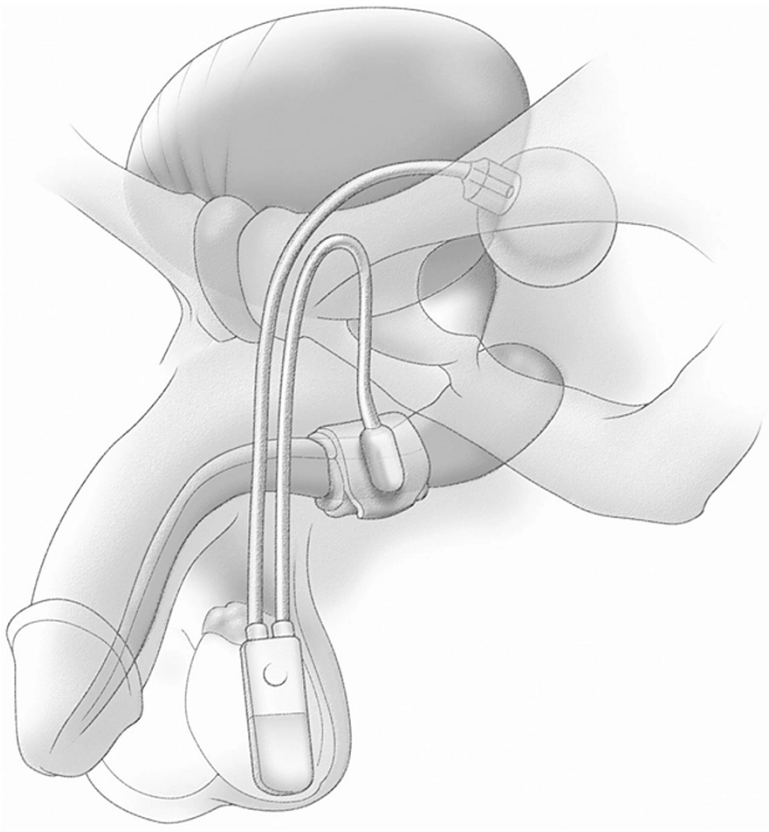

Figure 24.3. Photograph of an artificial urinary sphincter device comprising an occlusive cuff that is inserted around the urethra, a pressure-regulating balloon, and the control pump that is implanted into the scrotum or labium. Picture courtesy of American Medical Systems Inc, Minnetonka, MN, USA.

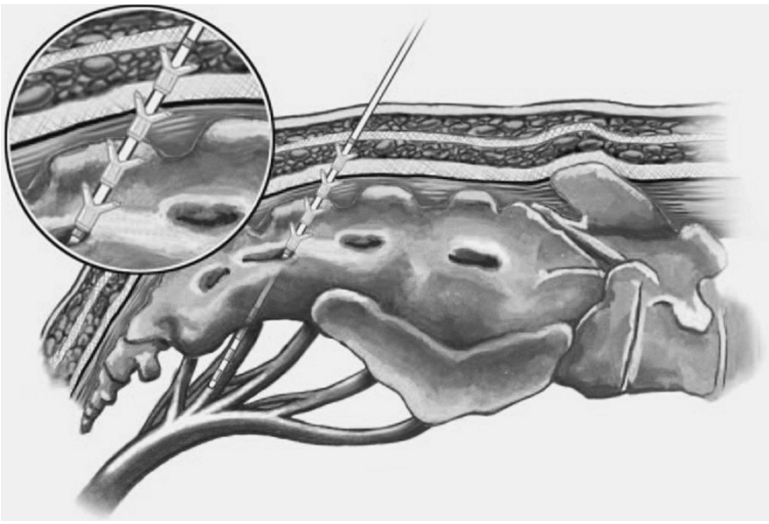

Figure 24.4. Drawing showing the inserted "tined lead" of an implanted sacral nerve root stimulator passing through a sacral foramen to lie in contact with S3 sacral nerve root. Picture reprinted with kind permission of Medtronic Limited, Watford, UK.

24.3.2 Bladder Augmentation

A more pragmatic approach for dealing with the overactive bladder has been to attempt surgically to convert a high-pressure storage system into a low-pressure one by incorporating into it a "gusset" constructed out of a segment of bowel. The technique that achieved popularity in the UK is the "clam cystoplasty."

Bramble[11] described this technique where the bladder is opened widely like a clam and a length of small bowel opened and laid as a patch into the resultant defect. This appears to act largely by increasing functional capacity and allowing overactive contractions to occur at larger volumes with a lower pressure rise. However, there are risks including early postoperative complications and longer-term problems that are all consequent on incorporating bowel into the lower urinary tract and are summarised in Table 24.2.[12] In addition, long-term satisfaction rates are worse than initially thought (53–78%) and up to one in five remain incontinent.[1]

"Detrusor myectomy" is a procedure where the detrusor muscle above the bladder "equator" is removed from the underlying mucosa while the bladder is kept partly filled to assist in the dissection. The effect is to create a broad opening bladder diverticulum with no intrinsic contractile capacity in the diverticulum. Myectomy is a lesser operation than cystoplasty and does not have the long-term complications of infection, mucus production, voiding dysfunction, and malignancy. In neuropathic patients the procedure can be extremely difficult and cystoplasty still represents a better option for this group. Reported outcomes are very limited and have focused on urodynamic results, but the symptomatic outcomes are not reported.[14]

Modifications to cystoplasty that will increase functional capacity of the bladder but without the same complications include gastrocystoplasty (sewing a segment of stomach in to the bladder defect) and seromuscular cystoplasty (covering a myectomy defect with a bowel segment denuded of epithelium). However, the technical difficulty of the surgery and postoperative complications have prevented more widespread use.

Future developments include the potential for using tissue engineering. Harvested urothelium cells and muscle cells can be grown in vitro and replanted onto an acellular matrix, which in turn is reimplanted into the bivalved bladder. Initial results in dogs have been encouraging but there is as yet no clinical application.

24.3.3 Modulation of Activity

The principle of neuromodulation is that by introducing electrical stimulation into the sacral reflex pathway, the normal reflex behaviour of the bladder can be modulated to the extent that uninhibited bladder contractions are inhibited. Dermatome stimulation with surface electrodes is covered elsewhere in this book. Permanently implantable sacral root stimulators have been developed to provide chronic stimulation directly to the S3 roots (see Figure 24.4). This has been shown dramatically to reduce detrusor hyperreflexia and instability.

Table 24.2. Complications caused by Bowel incorporation into urinary tract.

Bowel disturbance	Most patients Caused by decreased bile acid absorption
Voiding difficulty/poor emptying Leads to: • UTIs/excessive use of antibiotics • Stone formation	Most patients need to self- catheterise
Metabolic disturbance	Acidosis and risk of impaired renal function and altered bone growth in children; need for long-term alkali to be taken Vitamin B12 deficiency and pernicious anaemia
Risk of malignant transformation	Rare incidence of malignancy reported in cystoplasties; theoretical increased risk
Mucus production	Mucus smells and is difficult to drain from the bladder, especially through catheters; regular washouts often needed

Patients must first undergo a percutaneuous test stimulation in which needles are implanted directly through the sacral foramina under local anaesthetic. Only a proportion (50% or more) will respond and these patients are eligible for a permanent implant. Excellent outcomes have been reported from three randomised controlled trials showing between 56–85% improvement in symptoms compared to 4–5% in controls.[15,16] Two of the three Randomised Controlled Trials (RCTs) showed improvements in quality of life.

The technique is expensive both in terms of implant cost and the use of resources such as theatre and medical physics. However, the evidence for neuromodulation now represents the only level 1 evidence available for any surgical therapy of the overactive bladder and clearly merits serious consideration. It is essential for us to understand the cost effectiveness of treatments more clearly so that rational choices can be made by health commissioners.

24.3.4 Botulinum Toxin

The surgical approach to treating the overactive bladder has transformed in the last 3 years with the introduction of "botulinum toxin" (BTX) treatment. BTX is a potent neurotoxin produced by the bacterium *Clostridium botulinum* and has been used therapeutically in many ways. Of the seven toxin types available types A and B have been used therapeutically. Uses include paralysis of somatic muscles in orthopaedic and neurological practice as well as cosmetic use through paralysis of facial muscles.

When injected into the bladder of patients with neuropathic detrusor overactivity (DO), the treatment reduces both symptoms and urodynamic variables as well as improving quality of life.[17–19] The effect wears off after a number of months and repeated injections appear to work as well, although up to only four injections have been reported. The injections can be delivered through a flexible cystoscope to a conscious patient. However, the optimum site, dose dilution, and concentration of BTX to use in each clinical situation has not been clarified, nor has the long-term outcome from repeated injections. Antibodies are known to develop to BTX and this may ultimately prove to be a limitation in its clinical use. The logistics and long-term cost-effectiveness have yet to be established.

Ultimately the choice of therapy for individual patients with refractory DO will depend on careful discussion and assessment of the risks and benefits of each therapeutic approach. Patients must be made aware of the risks involved as they are considerable.

24.3.5 Urinary Diversion

Urinary diversion should be considered in patients for whom all other treatments have failed or when such failure is predictable, as is the case with some patients with degenerative neurological disease. One of the simplest methods of urinary diversion is a urethral closure and suprapubic vesicostomy, commonly employed in women with multiple sclerosis (MS). Provided that the urethral tissues are fairly healthy and the patient not too obese, then the operation is quicker and easier than any other form of diversion. However, the urethra is often badly eroded and this will preclude adequate closure. Also, most patients with MS will have severe DO, and this will result in incontinence through the vesicostomy instead of the urethra. Its usefulness therefore is limited.

"Ileal conduit" was described in 1952, and the technique has evolved little since that time. Both ureters are joined to an isolated segment of ileum, which in turn is used to create an incontinent stoma on the abdominal wall. The abdomen must be opened, but the operation technically is fairly simple and offers reliability and a low likelihood of reoperation. However, there are significant complications including parastomal hernia, stomal prolapse and fistula, infection and stone formation, and apparent upper tract deterioration over several years[20].

24.3.6 Continent Diversion

Continent urinary diversion involves the creation of a compliant reservoir for storage of urine and creating a continent catheterisable outlet through which the bladder can be emptied. The reservoir is created through augmentation of an existing bladder. If the bladder is thickened or diseased, it may be easier to create from a bowel segment a

pouch into which the ureters then will have to be reimplanted.

The catheterisable continent outlet is usually made from appendix or a tapered small bowel segment. There are many varieties of reconfiguration that can be used and little evidence that any one is better. However, they all represent major surgery and suffer from a wide variety and high incidence of complications, many of which will require reoperation to sort them out.

24.3.7 Rectal Bladder

New techniques of ureteric implantation, antibiotics, better suture materials, and development of pouches with low-pressure urodynamics have led to a resurgence of implantation of the ureters into the large bowel, particularly in parts of the world where stoma bags are either unavailable or culturally unacceptable. The sigmoid rectal pouch uses a side-to-side linking of the sigmoid colon to make a pouch and implantation of the ureters to prevent reflux. It is essential that the anal sphincter complex is normal beforehand and authors have presented high continence rates. Repeated surveillance with colonoscopy and barium examinations will be essential for these patients.

24.3.8 The Choice of Surgery

With such a wide repertoire of operations available some of the skill in surgery comes from careful choice of procedure for the most appropriate patient and nowhere is this more true than in patients with mixed incontinence. The judgment is in whether to offer patients less invasive treatments first, regardless of symptoms, or whether to treat the most bothersome symptom first, regardless of the magnitude of surgery involved. The urodynamic purist would choose to treat DO first because creation of a compliant reservoir can render minimal degrees of stress incontinence irrelevant. The pragmatist will usually perform less invasive treatments first on the basis that if they work, more major procedures have been avoided. In the absence of any good evidence to support one approach over another, patients must at least be made fully aware of the uncertainties of treating complex incontinence and that the outcomes are at best uncertain.

The subject of intermittent catheterisation (IC) has been covered comprehensively elsewhere in this book (see Chapter 23). Following operations for incontinence, voiding difficulty may occur because of outlet obstruction or inadequate detrusor contraction or both. Incontinence in such patients will usually be reduced by improving their bladder emptying. For some patients, surprisingly, having to self-catheterise is more odious than the original incontinence and this presents a major management problem. Simple sling procedures and injection therapies can be fairly easily reversed by urethrolysis, but more major operations are less easily reversed. When there is any doubt about the acceptability of IC patients should be taught before surgery is contemplated.

The clinical problem can be very complex; for instance, combined anorectal dysfunction and urinary incontinence, accompanying urogenital prolapse, multiple previous operations for incontinence, congenital and degenerative neuropathy, bladder disease such as interstitial cystitis, tuberculosis, or postradiotherapy cystitis which cause incontinence. These complex cases should be managed only by surgeons with a high level of specialist training, working in multidisciplinary teams so that all of the complexities will be covered by the available knowledge and expertise.

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25
Drugs Acting on the Lower Urinary Tract

PM Latthe and PM Toozs-Hobson

25.1 Introduction

Urinary incontinence broadly falls into two groups: those with failure of the continence mechanism (stress incontinence) and those with overactivity of the bladder (overactive bladder). This chapter discusses the classes of drugs used to treat these conditions.

25.2 Pharmacological Management of Stress Urinary Incontinence

25.2.1 Estrogens

The recent WHI[1] study has cast doubt over the use of estrogens in incontinence. This study, however, used oral preparations. Historically there are several studies that are summarised in the HUT committee[2] report suggesting a good subjective effect for vaginal estrogens. Whether this effect is through modulating atrophic vaginitis or through an effect on the urethra has not been established.

25.2.2 α-Adrenergic Agents

The bladder and proximal urethra contain α-adrenergic receptors. Stimulation results in an increase in the maximum urethral closure pressure. Phenylpropanoline is an α-adrenergic agonist with properties similar to those of ephedrine without causing significant central sedation. A Cochrane systematic review has summarized that there is weak evidence to suggest that use of an adrenergic agonist is better than placebo treatment. There is insufficient evidence to assess the effects of adrenergic agonists when compared to or combined with other treatments.[3] The number of α-adrenergic receptors in the urethra has been found to increase two- to threefold in animal experiments following estrogen therapy.[4,5]

25.2.3 Duloxetine

Duloxetine hydrochloride is a potent and selective inhibitor of serotonin (5-HT) and norepinephrine reuptake. It works via Onuf's nucleus in the spinal cord.[6] The medication increases serotonin and norepinephrine concentrations in the synapse, which enhance the effect of the key neurotransmitter (glutamate) in maintaining the pudendal nerve stimulation to the urethral sphincter during the storage phase. During voiding, glutamate is switched off and duloxetine has no effect (hence, the medication does not cause urinary retention). In trials it has been found to be as effective as physiotherapy but has had problems with compliance due to the side effects of nausea and sleep disturbance. To overcome this problem, commencing at the initial dosage to 20 mg twice daily has proved useful. Currently the role of duloxetine is as a adjunct to pelvic floor exercises (immediate effect)[7] or for women unwilling, awaiting, or unfit for surgery.[8]

25.3 Pharmacological Management of Detrusor Overactivity

The mainstay of pharmacotherapy is anticholinergics, which abolish or try to reduce the severity of detrusor muscle contraction. Side effects occur because of stimulation of muscarinic receptors in the parasympathetic system, resulting in dry mouth

or eyes, constipation, and more rarely headache or nausea. The mode of action probably is working at a cellular level blocking the effect of local release of acetylcholine rather than blocking the synaptic transmission. Hence, there is a relatively low risk of retention on the therapeutic concentrations of anticholinergics.[9]

One meta-analysis concluded that anticholinergics had little clinical effect.[10] One systematic review highlighted that all the "modern" anticholinergics appeared to offer fewer side effects than immediate release oxybutinin.[11] Either way it must be remembered that pharmacotherapy should be offered as part of a treatment package including lifestyle modification and bladder re-education.

25.4 Anticholinergic Drugs

25.4.1 Oxybutynin Hydrochloride

Dose

- 2.5–5 mg once to three times daily
- Extended release 5–15 mg once daily
- Patches 3.9 mg twice weekly

Oxybutinin has been the gold standard medication in clinical use for more than quarter of a century. Besides its anticholinergic activity, oxybutinin has some anesthetic effects as well. It acts predominantly on the muscarinic-3 (M3) subtype receptors. These receptors are responsible for the contractile properties of the bladder. While oxybutinin is effective, its use is restricted by poor compliance due to side effects which probably are related to metabolite N-desethyloxybutinin (found as a 4:1 concentration to oxybutinin). A once-a-day controlled release formulation showed similar efficacy between the long-acting and short-acting formulations.[12] The extended release system uses osmotic pressure to deliver oxybutinin at a controlled rate over approximately 24 hours smoothing out the plasma peaks and troughs. The introduction of patches using a parenteral administration route has reduced dramatically the incidence of side effects as the ratio of NDEO is reduced to 1:1 and this may be helpful in patients who were unable to take oral oxybutinin.[13] Antimuscarinic activity resides predominantly in the R-isomer and in the future it may be that the L isomer is removed to further reduce side effects.

25.4.2 Tolterodine

Dose

- 1–2 mg twice daily
- Extended release 4 mg XL

Tolterodine is a balanced (nonmuscarinic receptor subtype-specific) competitive muscarinic receptor antagonist. In experimental models, tolterodine shows selectivity for the urinary bladder over the salivary glands, and hence, the incidence of dry mouth is significantly lower than otherwise expected.

Tolterodine is metabolized in the liver, resulting in the 5–hydroxymethyl derivative, a major pharmacologically active metabolite, which exhibits antimuscarinic activity similar to that of tolterodine. Both tolterodine and its metabolite exhibit a high specificity for muscarinic receptors.[14] The long-acting formulation of tolterodine has fewer side effects and similar or improved efficacy than the twice a day formulation.[14,15] Generally the extended release preparations are preferred to immediate release preparations because there is a lower incidence of dry mouth.[16]

In a multicentred prospective randomised double-blind active-control study[17] in about 800 women, extended release oxybutinin at 10 mg/day or tolterodine at 4 mg/day were given to women with urge incontinence. Improvements in weekly UUI episodes were similar for the women who received extended release formulation. Oxybutinin was significantly more effective than tolterodine in reducing micturition frequency ($P = 0.005$). Dry mouth, although mild, was more common with oxybutinin; however, both groups showed similar rates of discontinuation of treatment due to adverse events.

25.4.3 Trospium Chloride

Dose

- 20 mg two–three times daily

Trospium chloride is a broad-spectrum atropine derivative with a quaternary ammonium group and acts as a parasympatholytic agent with

antispasmodic activity and ganglion blocking activities. These properties produce an additive lowering effect on parasympathetic tone, mainly in the urinary tract.

The main elimination of trospium chloride occurs via the kidneys. As 80% of the substances excreted in urine consist of the active parent compound, the intravesical presence of the drug also may contribute to the pharmacological activity in vivo and may explain the higher effect on the bladder compared with the effect on salivary glands. Oral bioavailability in humans is around 10%, with maximum serum levels reached after 5–6 hours of application. There is age- or gender-dependent change in absorption and elimination of the substance. Ingestion after food is reduced and the drug should be taken before meals. It is noted to be hydrophilic and as such does not significantly cross the blood brain barrier[18] unlike other anticholinergics and hence often favoured in the elderly patients.

25.4.4 Solifenacin

Dose

- Solifenacin 5–10 mg once daily

Solifenacin is a newer anticholinergic with a similar M3 specificity to oxybutinin. In the recent STAR study[19] Solifenacin was shown to be as effective as Detrusitol XL in reducing micturition frequency (primary end point) but superior in reducing leakage, urgency, and in the percentage of patients remaining dry.

25.5 Tricyclic Antidepressants

These agents act both centrally and peripherally to block the reuptake of the neurotransmitters norepinephrine and serotonin. Although the exact mechanism of action on the lower urinary tract is unclear, tricyclic antidepressants exert anticholinergic as well as musculotropic effects and possibly α-adrenergic effects, increasing bladder outlet resistance. It is this combination of anticholinergic effect and increased bladder outlet resistance that allows better storage of urine and has been found to be a useful combination in patients with overactive bladder as well as incontinence. Imipramine remains the most commonly used tricyclic antidepressant. The usual starting dose is 25 mg/day, lower than the dosage needed to achieve a therapeutic effect in depression.[20]

Cardiac toxicity is recognised, but in the doses used this is not a problem and an ECG is seldom necessary. Other side effects include dry mucous membranes, sleep disturbances, personality changes, weakness, fatigue, anxiety, and appetite changes. These are more common but definitely more worrisome. Although amitriptyline is often useful in patients whose symptoms are more at the pain end of the spectrum, imipramine is also helpful at night due to its longer mode of action and arguably greater somnolence effect.

25.6 Other Drugs

25.6.1 Desmopressin (DDAVP)

Desmopressin is a synthetic analogue of vasopressin and acts as a antidiuretic. It traditionally has been used in children with nocturnal enuresis. The mechanism of action is by decreasing urine output, which allows the patients to sleep without disruptive trips to the toilet.[21]

Fluid overload and hyponatremia results from excessive fluid intake and is one of the major side effects, although probably not a problem in children. Patients with abnormalities of electrolytes and fluid balance such as congestive cardiac failure and cystic fibrosis should be advised to avoid this medication or be monitored very carefully if they are started on the medication.

25.6.2 Intravesical Botulium toxin A

Botulium toxin A is emerging as a significant alternative to anticholinergic medication in refractory patients especially in neurogenic detrusor activity.[22] Large randomized controlled trials are underway to determine its effectiveness in idiopathic detrusor overactivity. The toxin appears to work at several levels rather than just blocking acetyl choline receptors and as such may have a much extended function compared to its use elsewhere in medicine (see Chapter 24). The main side effect of this treatment is retention and the need to self-catheterise.

25.7 Conclusions

Medical management of incontinence and in particular drug treatment of incontinence have started to undergo a dramatic revolution. For the first time, we are now able to be confident that there is a drug suitable for the majority of patients with an acceptable side-effect profile. For the first time there is a realistic prospect of a drug for stress incontinence that may augment the physical therapy in a similar way to the anticholinergics with bladder drill.

It is important to remember, however, that drug treatment is part of a behavioural package and that fluid management, bladder drill, and pelvic floor re-education remain the cornerstones of conservative management. Despite the improvements in drug therapy there still needs to be a change in the attitude of both the patient and physician in accepting that this is management of a chronic condition.

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III Colorectal Disorders

26
An Anatomical Overview

D Kumar

26.1 Anatomy of the Anorectum

26.1.1 Anal Canal

The anal canal is approximately 3–4 cm in length; it extends from the anal skin margin to the level of the pelvic diaphragm (the pelvic floor). The anal canal is surrounded by the two sphincter muscles (Fig. 26.1). The inner circular smooth muscle area of the rectum becomes thickened and forms the internal anal sphincter, which is easily palpated on digital examination. The internal sphincter is involuntary but provides a resting tone that helps to keep the anal canal closed at all times. The external anal sphincter consists of striated muscle and has three distinctive parts: the subcutaneous, superficial and deep. The deep part of the external anal sphincter fuses with the puborectalis muscle at the top of the anal canal and helps to maintain continence.

The puborectalis muscle maintains the anorectal angle by pulling the rectum forward at the anorectal junction. The puborectalis forms part of the levator ani muscle and is its most medial component. Lateral to the puborectalis lies the pubococcygeus, then the iliococcygeus and the most lateral is the ischiococcygeus. Together

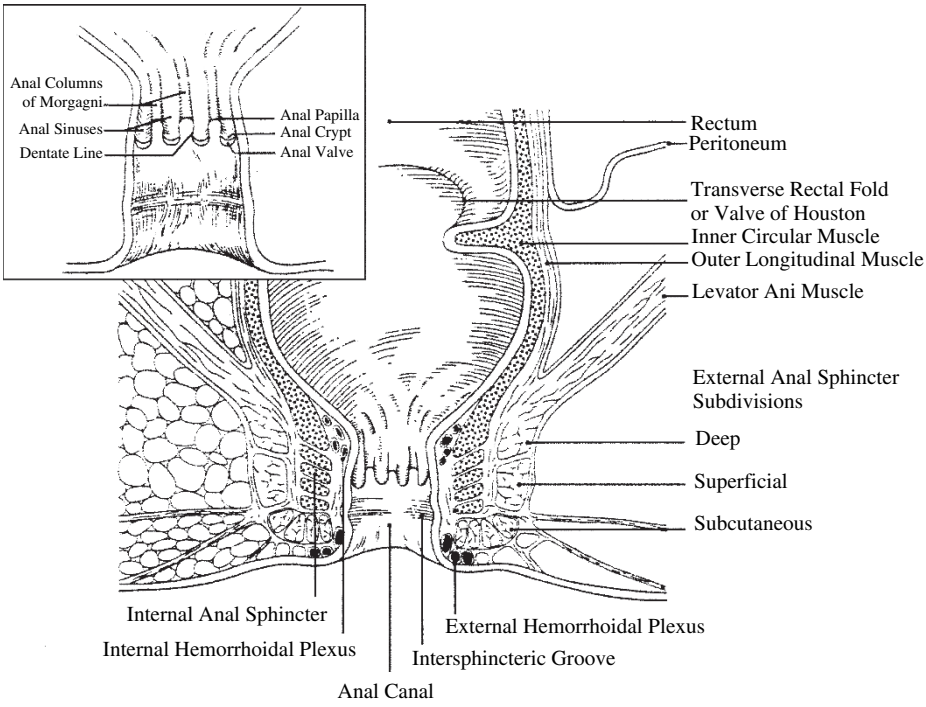

Figure 26.1. Section of the rectum and anal canal, demonstrating the internal and external anal sphincters and the valves of Houston.

these muscles form the pelvic diaphragm or the pelvic floor, and support the pelvic viscera.

The anal canal is very richly supplied by nerves; the upper half is supplied by both sympathetic and parasympathetic nerve fibres. The preganglionic sympathetic nerve fibres originate from the second to the fourth lumbar splanchnic nerves and the post ganglionic fibres travel to the sigmoid colon, rectum and upper anal canal via the pelvic plexus. The preganglionic parasympathetic nerves originate in the sacral cord (S2–4). These emerge as the nervi errigentes and become the pelvic splanchnic nerves. The lower half of the anal canal is supplied by branches of the pudendal nerve called the inferior rectal nerves. The external anal sphincter is also supplied by branches of the pudendal nerves.

26.1.2 Rectum

The rectum is approximately 15 cm long and lies in the sacral hollow. The beginning of the rectum is identified by the disappearance of tenia coli and appendices epiploicae which are present in the colon. The rectum has a complete coat of longitudinal muscle.

The anterior and posterior muscles are slightly shorter than the lateral muscle and this configuration produces three lateral shelves called the superior, middle and inferior rectal valves or valves of Houston. The rectum is supplied by both sympathetic and parasympathetic nerve fibres similar to those of the upper anal canal. The sensory fibres of the rectum are derived from the autonomic nervous system and enter the chord at the L1, L2, S2, S3 and S4 levels. The rectum is relatively insensitive to painful stimuli but does respond to distension and stretch.

26.1.3 Mechanism of Continence

A number of theories have been put forward to explain anorectal continence. The flutter valve theory argues that intra-abdominal pressure forces applied to a high-pressure zone in the lower rectum produces occlusion of this area of the rectum, thus preventing passage of rectal contents into the anal canal. However, Duthie observed a high-pressure zone within the anal canal and therefore argued against the flutter valve theory.[1] He proposed that for a flutter valve to produce a high-pressure segment the intra-abdominal forces would have to be applied below the levator muscles. Parks put forward the flap valve theory and stressed the importance of an acute anorectal angle which helped in the application of intra-abdominal forces to the anterior rectal wall.[2] During a rise in intra-abdominal pressure, the top of the anal canal is occluded by anterior rectal mucosa which plugs it and prevents rectal contents reaching the anus. According to Parks, incontinence occurs when there is excessive perineal descent and the anorectal angle becomes obtuse. He developed the postanal repair operation to recreate the anorectal angle.

Rectal filling results in lowering of resting anal canal pressures which is due to inhibition of internal anal sphincter activity. This reflex activity is called the recto-anal inhibitory reflex. Local intrinsic nerve pathways then lead to an increase in internal sphincter pressure thus maintaining continence. The recto-anal inhibitory reflex allows the rectal contents to come into contact with the specialised anal sensory epithelium which then initiates the anal sampling reflex.

Two other factors, rectal compliance and gastrointestinal transit, also help in maintaining continence. Rectal filling is appreciated from volumes of 10 ml or more and the rectum can tolerate 300 ml before a feeling of fullness is noticed. This is achieved by the accommodation reflex, and once the maximum tolerable volume is achieved, there is an urgent desire to defecate. Similarly, rapid colonic or gastrointestinal transit results in rapid filling of the rectum, resulting in urgency or urge incontinence. This can occur in the presence of a normal and competent sphincter complex.

26.1.4 Mechanism of Defecation

Defecation is a complex act which is under the influence of the central nervous system. Rectal filling, distension and stimulation of the anorectal mechano receptors provide the stimulus for the initiation of the act of defecation. It has two main components:

- the rise in intra-abdominal pressure which is achieved by the contraction of the abdominal wall muscles and contraction of the pelvic floor muscles.
- the evacuation of the rectum achieved by relaxation of the anal canal and contraction of the rectal wall.

Defecation can occur without abdominal wall contraction, however, especially in circumstances when the stools are loose. In addition to the central cortical control, there is a spinal centre in the lumbo-sacral region which can maintain reflex defecation following transection of the spinal cord at this level. The onset of defecation in this case is marked by descent of the pelvic floor, followed by propulsive contractile activity in the sigmoid colon which propels faeces to the rectum.

Rectal filling induces the conscious urge to defecate and causes relaxation of the internal sphincter muscle. This sequence is completed by the decision to defecate. The external anal sphincter relaxes, and faeces are expelled by alternating contraction and relaxation of the levator muscles. This is aided by a rise in intra-abdominal pressure transmitted through the relaxed pelvic floor Defecation is not possible when the pelvic floor is raised, presumably because intra-abdominal pressure cannot then be transmitted to the recto anal contents. The colon also contributes to the mechanism of defecation by producing high-amplitude contraction waves in the proximal colon immediately prior to defecation.

26.2 Aetiology and Pathophysiology of Anorectal Disorders

26.2.1 Aetiology and Pathophysiology of Faecal Incontinence

Continence is maintained by an interaction of several factors including normal transit of a normal-consistency stool, normal capacity rectum to provide an adequate reservoir, and a normal voluntary control and reflex function provided by the anal sphincter complex. The most common cause of faecal incontinence in adult healthy women is obstetric trauma. Other causes include pudendal nerve neuropathy and iatrogenic causes such as damage to the sphincter muscles during surgical operations. Neurological causes of faecal incontinence include upper motor neurone lesions and lower motor neurone lesion or peripheral nerve lesions from the sacral outflow or the pudendal nerve. In patients with diabetes, mixed motor and sensory loss may result in faecal incontinence. Direct trauma to the sphincters can also cause faecal incontinence. It may also be the presenting symptom in patients with faecal impaction and idiopathic constipation. Similarly, patients with rectal prolapse and those with proctitis may also have associated incontinence. In children Hirschsprung's disease and anorectal malformation can also result in faecal incontinence.

26.2.1.1 Obstetric Trauma

It is now well established that in healthy adult women the most common cause of faecal incontinence is sphincter damage during childbirth. Often there is a history of a difficult vaginal delivery, forceps-assisted delivery or a perineal tear.[3]

Sultan et al in a study of 128 women found that pudendal nerve latencies were significantly prolonged after vaginal deliveries.[4] Only a third of those who had a prolonged pudendal nerve latency were still affected after 6 months, suggesting that most women who sustained sphincter trauma during childbirth appeared to recover normal nerve function. With the availability of better imaging technology we now know that sphincter disruption is the most common form of obstetric damage.[5] Approximately a third of all primiparous women develop a sphincter defect involving one or both muscles following a vaginal delivery.[6] Patients who sustain external sphincter damage also have impaired sensation which appears to persist in the upper anal canal at 6 months.[7]

26.2.1.2 Iatrogenic Sphincter Trauma

This is usually the result of operation on the anal canal for fissures, fistula-in-ano and haemorrhoids. It is now well established that anal manual dilatation also results in damage to the anal sphincter complex.

26.2.1.3 Faecal Impaction

Faecal impaction is common in geriatric patients, and is easily seen as a cause of faecal incontinence.[8] In younger patients it is often

associated with a megarectum or congenital anorectal malformation.

26.2.1.4 Internal Anal Sphincter Dysfunction

Swash et al. found ultrastructural changes in internal anal sphincter biopsies in patients with neurogenic anorectal incontinence.[9] Pharmacological studies have demonstrated a poor response to adrenaline stimulation and lack of response to electrical field stimulation of internal anal sphincter biopsies from patients with neurogenic incontinence.[10,11]

26.2.2 Aetiology and Pathophysiology of Constipation (see also Chapter 28a)

Constipation is a symptom, and its causes can be organic or functional. Before making a diagnosis of chronic idiopathic constipation, specific causes such as metabolic and endocrine abnormalities, neurological lesions, drugs and muscular lesions, as well as gastrointestinal tract disorders, need to be excluded.

An anatomical and morphological variation in the size of the colon and rectum and consistency and size of the stool can have a significant impact on the mechanism and the frequency of defecation. Small hard stools are more difficult to expel than soft stools of the same shape and volume. Stool form and consistency also correlate with transit time; hard stools are passed when the transit time is long and a soft mushy stool is associated with rapid transit through the colon. The consistency of stool depends on its water content; longer residue time in the colon results in a harder stool. In some patients with constiption the length of the colon is abnormal (this is often referred to as a redundant colon on contrast radiology). Using a continuous colonic profusion and a dye dilution technique, colonic volumes were found to be about 50% greater in constipated patients than in control subjects.

In patients with a megarectum and a megacolon there is thickening of the muscle layers in the rectum and the colon; this may be in response to a zone of functional obstruction. The megabowel most commonly involves the rectum and the rectosigmoid. In children there is often a history of faecal soiling. Some patients have normal and others delayed transit times.[13]

26.2.2.1 Idiopathic Constipation: Motility Disorder

Perfused tube manometry over a period of 24 h has shown that colonic motility consists of low-amplitude contractions most of the time but high-amplitude propagating contractions were noted early in the morning, after waking and before defecation.

During defecation, the peristaltic wave progresses to the distal bowel and causes internal sphincter relaxation. In patients with constipation the frequency and duration of the high-amplitude pressure waves is reduced.

26.2.2.2 Abnormalities of Sensation

Rectal volume sensation can be recorded by distending a balloon in the rectum and noting the volumes at which first sensation, urgency and pain are experienced. Patients with constipation require a greater volume to produce their initial sensation and urgency but have a normal maximum tolerated volume when compared with controls.[13] Rectal sensation can also be tested by applying a slowly increasing current to the rectal mucosa.

Constipated patients demonstrate an elevated mean sensory threshold compared with a control group. However, there is considerable overlap between constipated patients and the control population.

26.2.2.3 Abnormality of the Pelvic Floor

Some patients with chronic idiopathic constipation exhibit abnormalities of pelvic floor function and may be unable to expel a water-filled balloon from the rectum. Rectal function may be abnormal in patients with slow colonic transit, but also in patients who have a normal colonic transit time. The latter abnormality is more commonly seen in patients with increased pelvic floor descent and those who have a history of chronic straining resulting in pelvic nerve damage.

Electromyography (EMG) studies of the striated external anal sphincter muscles have shown

that many patients with severe constipation have paradoxical contraction of the muscle during straining.[14] It has been suggested that inappropriate contraction of the puborectalis and external sphincter muscles is responsible for blocking rectal emptying. This phenomenon has been termed "anismus". However not all patients with constipation exhibit this EMG abnormality, and moreover some normal subjects and patients with other anorectal conditions may have this abnormality as well. It is conceivable that the phenomenon of paradoxical pelvic floor contraction may be an abnormal learned response rather than an inherent abnormality of the neuromuscular mechanisms.

26.3 Assessment and Investigations

26.3.1 Evaluation of a Patient with Faecal Incontinence

All patients presenting with faecal incontinence should have a careful history taken. Special attention should be paid to the stool consistency, history of straining and a previous history of difficult vaginal delivery with or without the use of forceps.

A physical examination of the abdomen and anorectum should be carried out (see Chapters 27 and 28a). Neurological symptoms should also be recorded. Attention should be paid to symptoms suggestive of colonic or rectal disease such as the presence of diarrhoea and the leakage of mucus or blood in patients with inflammatory bowel disease. Any history of previous pelvic or anal surgery, especially anal dilatation, fistula surgery and prolapse repairs, should be documented.

26.3.2 Anorectal Investigations

26.3.2.1 Anal Manometry

A variety of pressure measurement equipment is commercially available. Using these devices, the function of both internal and external anal sphincters is assessed by measuring anal canal pressures. The manometry assembly is inserted into the rectum and then gradually withdrawn. When the high-pressure zone is reached, the distance from the anal verge is recorded and the pressure in the anal canal measured at 1 cm intervals as the catheter is withdrawn further. This records the resting pressure produced by the internal anal sphincter as well as the resting sphincter length. Manometry is the only method of measuring resting tone in the anal canal.

The internal anal sphincter is responsible for approximately 70–80% of the resting tone in the anal canal. Measurement of the resting tone therefore provides an assessment of the internal anal sphincter function. The procedure is repeated but this time the patient is asked to voluntarily squeeze the external anal sphincter so that the maximum squeeze pressure produced by the external sphincter can be recorded. Voluntary squeeze pressure is the greatest pressure achieved above resting pressure during a maximal voluntary contraction. It is mainly an expression of the external anal sphincter function. The patient should be instructed not to use the gluteal muscles during voluntary squeeze as this will result in an erroneous recording of the squeeze pressure. In the majority of patients with incontinence the resting and squeeze pressures are significantly lower than in normal subjects. Patients who are incontinent to liquid and solid stool have lower squeeze pressures than those who are incontinent to liquid stool alone, and the length of the high-pressure zone is shorter in patients with incontinence than in normal subjects.

26.3.2.2 Rectal Volume Sensation

This can be measured by using a latex balloon tied to a rubber tube, which is attached to a three-way tap and a syringe (Fig. 26.2). The balloon assembly is inserted into the rectum and known aliquots of air are injected into the balloon. The volume of air at first perception is the threshold volume. The volume at which the patient can perceive the presence of an inflated balloon inside the rectum determines the volume at constant sensation and volume at which the patient has an uncontrollable desire to defecate is the maximum tolerated volume.

In patients with faecal incontinence due to rectal factors, the volume at constant sensation is often below normal. The maximum tolerable volume may

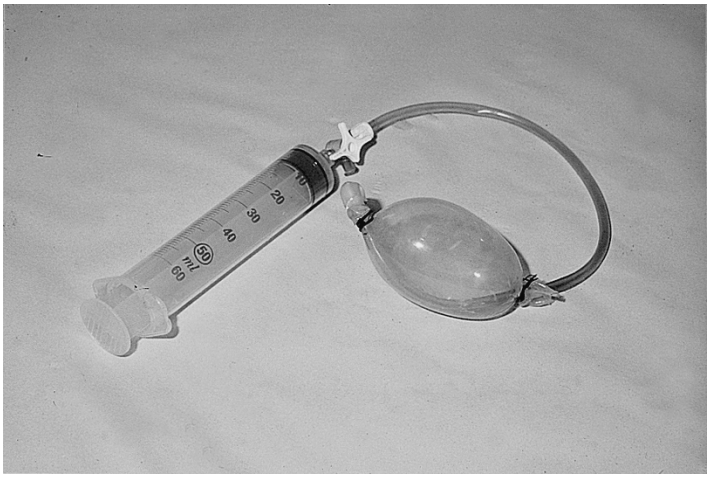

Figure 26.2 Latex balloon for rectal sensitivity evaluation.

also be impaired in patients with faecal incontinence. In some patients constant sensation or maximum tolerability at a low volume may be the only abnormality. These patients often have symptoms of severe urgency associated with faecal incontinence.

26.3.3 Neurophysiological Studies

26.3.3.1 Electromyography

Electromyography is used to assess denervation or reinnervation in patients with faecal incontinence. It is commonly performed by using a concentric or single fibre needle electrode (see Chapter 9). The concentric needle electrode has a relatively large uptake area and records the activity of several motor units; the EMG activity will be reduced where the number of functioning fibres is reduced. Patients with neuropathic faecal incontinence have reduced activity in both puborectalis and external anal sphincter muscles. By contrast, the single fibre EMG allows analysis of changes in single muscle fibres. It demonstrates reinnervation of previously denervated muscle fibres by surrounding neurons and is expressed as fibre density. It is increased in patients with neurogenic faecal incontinence and is said to be a sensitive and specific marker of neuropathy.[15]

26.3.3.2 Pudendal Nerve Terminal Motor Latency

Pudendal nerve terminal motor latency measures conduction in the terminal part of the pudendal nerve. The pudendal nerve is stimulated as it crosses the ischial spine whilst recording the evoked potential in the external anal sphincter. This is studied using a specifically designed device mounted on a glove.[10] Recordings are made from both sides of the pelvis, as pudendal nerve damage may be asymmetrical in some patients. Pudendal nerve terminal motor latency is prolonged in patients with idiopathic faecal incontinence. However, it must be remembered that pudendal nerve terminal motor latency and fibre density increase with age and this should be considered when interpreting the data.

26.3.4 Endoanal Ultrasound

Endoanal ultrasound provides high-resolution images of both the internal and external anal sphincter and the puborectalis muscles. The examination is performed with the patient in the left lateral position and serial images are obtained at rest and during squeeze in the lower, mid and upper anal canal. The equipment used to perform endoanal ultrasonography is shown in Fig. 26.3. The normal ultrasound (Fig. 26.4) consists of a compete ring of internal sphincter muscle surrounded by the mixed echogenic uninterrupted external sphincter. In patients with a direct sphincter injury or obstetric trauma, a sphincter defect is seen in the internal and/or external anal sphincter (Fig. 26.5). It also provides a dynamic assessment of the sphincter muscles on voluntary contraction.

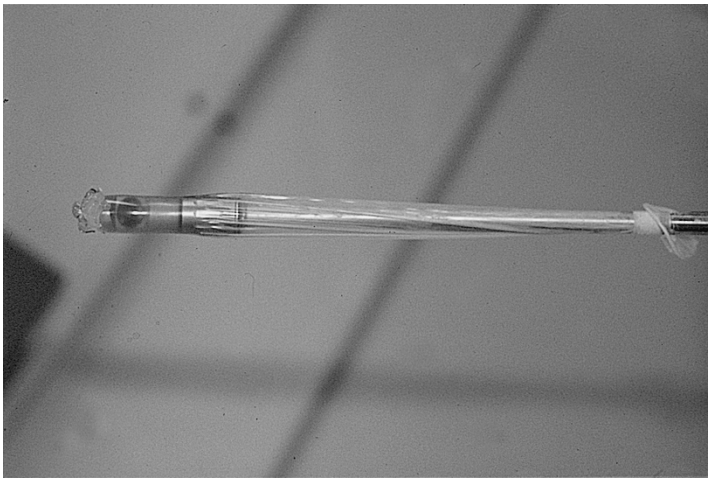

Figure 26.3 Anal ultrasonography equipment.

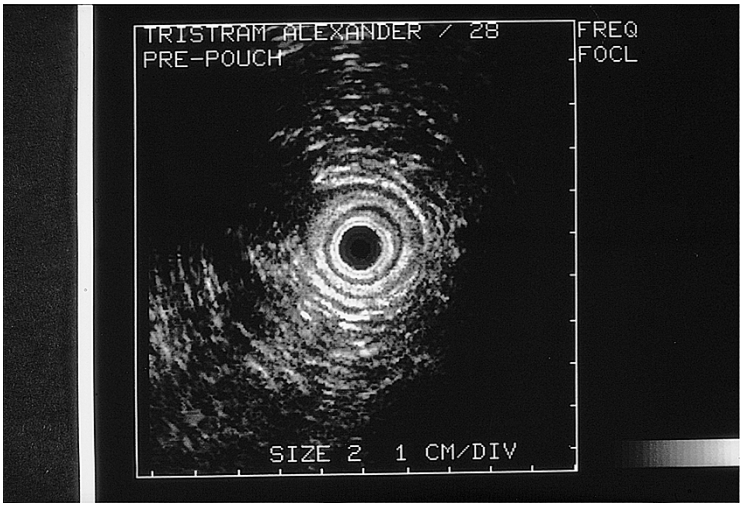

Figure 26.4 Normal ultrasound of the anal canal.

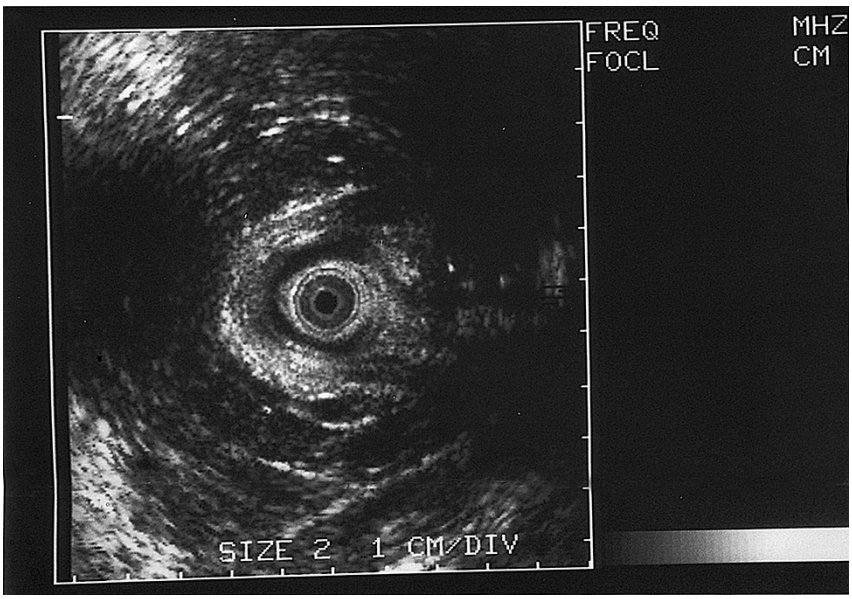

Figure 26.5 Internal and external anal sphincter defect.

26.3.5 Evaluation of a Patient with Constipation (see also Chapter 28a)

The patient should have a thorough general physical and systemic examination to elicit signs of hypothyroidism and other associated medical conditions. Haematological and biochemical abnormalities should be excluded by a full blood count, urea and electrolyte estimation, thyroid function tests and liver function tests. Serum calcium should also be checked in all patients. A barium enema examination should be performed to exclude a mechanical reason for constipation. After all possibilities of an organic cause for constipation have been excluded, a complete functional evaluation of the patient with chronic idiopathic constipation should be made. This should include an assessment of colonic transit, rectal transit and pelvic floor function. In some cases additional assessment with colonic motility or small bowel motility measurement may be necessary.

26.3.5.1 Colonic Transit

Radiological techniques involving ingestion of radio-opaque markers of different shapes and sizes on different days followed by several abdominal radiographs over the subsequent 5–7 days are commonly used but are time consuming and subject the patients to excessive radiation.

Colonic scintigraphy is the method of choice in the author's unit for the assessment of colonic transit. It is performed in outpatients without bowel preparation. The subject ingests a capsule which is methacrylate coated and contains 2 MBq of indium-111 as the radionuclide. Imaging starts within 4 h. Radioactive (cobalt-57) markers are applied to bony landmarks to permit correct alignment of images. Imaging is continued hourly between 9 a.m. and 5 p.m. on the first day, and the subject returns home overnight. Imaging is continued for 2 further days between 9 a.m. and 5 p.m. On days 2 and 3 images are acquired at 4-hourly intervals. Subjects are encouraged to maintain as normal a lifestyle as possible. They are asked to collect faecal samples and record the time of evacuation. These activities permit subsequent measurement of evacuated radioactivity.

From the computerised data, dynamic images are generated and the position of the colon determined. To evaluate segmental colonic transit, regions of interest (ROI) are defined on scans and transit through each region is separately determined. Four regions of interest are used:

- ROI 1 is the caecum, ascending colon and hepatic flexure.
- ROI 2 is the transverse colon
- ROI 3 is the splenic flexure
- ROI 4 is the descending colon, sigmoid colon and rectum.

The proportion of the scintographic counts in each region on each image is calculated and the distribution of scintigraphic activity throughout the colon with time is determined. Scintigraphy has advantages over other methods of measuring colonic transit in that a repeated or continuous observations can be obtained without additional radiation hazards. The estimated dose equivalent is less than one abdominal radiograph. The measurement of radiation counts in stool samples within closed containers makes the process of faecal examination convenient and aesthetically acceptable. Scintigraphic transit measurement also has the advantage over other methods in that segmental colonic transit can be quantified. The main problem with scintographic transit measurement is poor image resolution which can sometimes lead to difficulty in differentiating the colon from overlapping small bowel.

26.3.5.2 Rectal Transit

Chronic idiopathic constipation may be due to impaired rectal evacuation, slow colonic transit or a combination of both functional abnormalities.

Assessment of rectal evacuation is therefore an important component of the evaluation of the constipated patient. The variety of different methods of assessing rectal evacuation testify to the difficulties and the lack of consensus about the optimum method.

26.3.5.3 Barium Proctography

Conventional assessment of anorectal function with barium proctography involves evacuation of a barium-labelled artificial stool. Barium proctography is widely used for assessment of anorectal angle, pelvic floor descent, rectoceles and mucosal prolapse. However, it is debatable how important it is to evaluate these parameters. The anorectal angles in constipated patients and in controls are similar and there is no relationship between symptoms and the anorectal angle. Pelvic floor descent, rectoceles and mucosal prolapses are probably the result rather than the cause of impaired rectal evacuation. Another drawback with barium proctography is safety. A typical investigation involves 1–2 min of fluoroscopy and 2–3 radiographs

26.3.5.4 Isotope Defecography

The drawback of conventional proctography led to the development of a quantitative scintigraphic method of assessing defecation. Scintigraphic defecography involves evacuation of a radiolabelled artificial stool.

Radioactive markers, each containing 1 MBq activity are placed over the subjects pubis, lumbosacral junction and coccyx to promote alignment of images. With the subject in the left lateral position, 260 ml of an oat porridge and water mixture containing 100 MBq technetium is introduced intrarectally. The subject is imaged whilst seated on a commode with a gamma camera head against the left hip. Dynamic digital images are acquired during evacuation for up to 10 min and stored on computer.

The images are replayed and a rectal region of interest is drawn around the bolus of scintigraphic activity in the rectum. Rectal emptying curves are obtained. Rectal percentage of evacuation time and evacuation rate are easily calculated. Ano-rectal angle, pelvic floor descent and rectocele can all be measured with reference to the markers and the anal canal. The advantage of scintigraphic defecography is that it provides quantitative and dynamic information on rectal evacuation. The other main advantage is the minimal radiation dose administered to the patient.

26.4 Conservative and Medical Management of Faecal Incontinence

The principles governing conservative management of faecal incontinence are:

- keep the stool formed
- keep the rectum empty.

See also Chapter 27. The roles of dietary measures and pelvic floor exercises and re-education are also discussed in subsequent chapters.

26.4.1 Keep the Stool Formed

Keeping the stool formed is extremely important, as a compromised anal sphincter finds it difficult to control liquid stool and maintain continence. Stool consistency also influences rectal function in that liquid stool makes urge incontinence worse than does solid stool.

Stool consistency can be altered either by dietary manipulation or by use of constipation agents or both. It is important to recognise that the introduction of a high-fibre diet or fibre supplements in the diet can be used to soften the stool as well as to make it formed. This can be achieved by regulating the amount of oral fluids. If the stool is already liquid, then the introduction of fibre supplements with limited oral fluids makes the stool firm as the fibre draws fluid from the stool itself.

Constipating agents that work by slowing intestinal and colonic motility are also beneficial. The agents most commonly used are codeine phosphate and loperamide. These agents increase the residue time for the stool in the colon and therefore provide a better opportunity for absorption of water from the stool. Loperamide has an added beneficial effect in that it also increases the resting tone in the internal sphincter function, which may be beneficial in patients who have internal sphincter weakness.

Occasionally, a combination of fibre supplements and constipating agents can work better than either of these used in isolation. This also has the advantage that a very small dose of the motility reducing agent is required.

26.4.2 Keep the Rectum Empty

Keeping the rectum empty is an important aspect of the management of faecal incontinence. It is

particularly important in the elderly patient who often has faecal loading or impaction. In these patients faecal incontinence is secondary to the faecal impaction, and often the treatment of faecal impaction results in complete resolution of the symptom of faecal incontinence.

The simplest way of keeping the rectum empty is by regular use of glycerine suppositories, which can be brought over the counter. Two suppositories a day will give an adequate and satisfactory bowel action. In some patients suppositories do not provide a satisfactory answer and in those circumstances daily enemas or washouts may be necessary.

In a small number of patients, particularly young children, antegrade continence enemas may be necessary. This procedure involves bringing the appendix to the surface as a small opening providing access to the colon. The patient irrigates the colon with half a litre of water and this clears the colon completely, leaving an empty bowel.

26.5 Conservative and Medical Management of Constipation

Most patients with constipation can be managed successfully by conservative means. Less than 10% will require surgical intervention. The conservative measures include dietary manipulation, judicious use of laxatives and specific drug therapy. Once a mechanical cause for constipation has been excluded, all patients should be given advice regarding a high-fibre diet, fibre supplementation and increased oral fluid intake prior to rectal and colonic transit studies. Any electrolyte abnormalities or impairment of thyroid function should be corrected. More than half of all patients presenting with the symptom of constipation will respond to such measures. Dietary manipulation is discussed in more detail in Chapter 27.

A precise definition of the extent of the problem and the segments of the colon or rectum involved helps in deciding whether the treatment should consist of oral laxatives, rectal suppositories or enemas, or a combination of both. Approximately 26% of patients will have only rectal problems, a third will have a combination of rectal and colonic problems, another third will have colonic problems alone and the remainder will have normal transit constipation. In patients with colonic inertia only, judicious use of laxatives provides the most satisfactory results. This may be combined with dietary measures or fibre supplementation. Such patients will not respond to treatment with rectal suppositories or enemas. In contrast, patients with delayed rectal transit will respond extremely satisfactorily to regular use of suppositories or enemas. Oral laxatives are of no benefit to such patients. Patients who have a combination of rectal and colonic transit abnormality need a combination of oral laxatives and suppositories or enemas.

There is a resurgence of interest in the development of more targeted drug therapy for chronic idiopathic constipation, but at present no satisfactory targeted treatment is available.

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27
Faecal Incontinence

C Norton

27.1 Prevalence of Faecal Incontinence

Faecal incontinence (FI) has been defined in several different ways. For the purposes of this chapter, "faecal incontinence is the involuntary loss of flatus, liquid or solid stool that is a social or hygienic problem" will be adopted.[1] The term "anal incontinence" is usually used to denote any involuntary leakage, whether of solid, liquid, or gas.

FI is much more common in the general population than is often realised. Although the problem does increase with advancing age and disability, there are also large numbers of young otherwise healthy adults with this distressing symptom. Studies have found widely varying prevalence rates of faecal incontinence, depending on the definition of incontinence used and the population studied. The most recent community study included over 10,000 community-dwelling respondents in the UK.[2] Overall, 6.2% of men and 5.7% of women reported experiencing some FI in the past year; 0.7% of men and women reported daily episodes that limited their quality of life. Other studies have found even higher reported prevalence, with many finding rates over 10%.[3] There is a large overlap between people reporting FI and those complaining of difficulties with rectal evacuation.[4]

FI becomes more common with advancing age, especially in the very elderly, with 16.9% of women over 85 years reporting occasional involuntary loss of faeces.[5] For most people in institutional care faecal incontinence is compounded by urinary incontinence. Among the frail elderly in nursing homes prevalence of over 20% is common,[6] but rates of over 90% are also reported,[7] suggesting that variation may be due to factors other than the ageing bowel (such as institutional regimens and policies). Many people are reluctant to mention symptoms to health professionals,[8] even when directly questioned.[9]

People with neurological disease or injury are especially vulnerable to loss of bowel control. It should be noted that in the colorectal literature "neurogenic incontinence" usually refers to incontinence caused by damage to the pudendal nerve rather than patients with neurological problems. This can cause confusion. Over one third of people who have a stroke have faecal incontinence on admission to hospital. This falls to 10% at 6 months, with older patients, women, and those with the most severe strokes, diabetes, or another disabling disease being most at risk.[10] Spinal cord injury commonly causes major bowel dysfunction, with 11% reporting faecal incontinence weekly or more and only 39% having reliable bowel continence.[11] Multiple sclerosis is associated with constipation in over 40% and faecal incontinence in over 50%; those with greatest disability being most affected by both.[12] However, FI may be more a product of immobility and dependency than neurological damage, and patients with neurological problems also can have other risk factors such as obstetric trauma.

27.2 Assessment of Faecal Incontinence

Incontinence of faeces is a devastating symptom in our hygiene- and odour-conscious society. To lose bowel control in adult life often causes the

individual to feel extremely embarrassed and a social outcast. This means that unless people are given the opportunity to discuss these symptoms, they will often not be disclosed, even when being treated for a related problem such as urinary incontinence. There are many opportunities for physiotherapists to become aware of possible problems with bowel control: during ante- or postnatal classes; on the maternity ward; during a consultation for bladder problems; during a consultation for an unrelated problem; and working in rehabilitation settings.

As with any problem, investigation starts with talking to the patient. It is essential that, whichever member of the multidisciplinary team the incontinent or constipated person presents to, she or he is met by an empathetic and positive response. It will be important to build a relationship of trust with the patient if the whole picture is to be openly and frankly discussed. Some patients will not be aware of medical terms such as "defaecation" or even "stool," but are reluctant to use their usual slang terms to a professional. Many people find it very difficult to talk publicly about their bowels and often do not have a vocabulary of words they consider socially acceptable to describe what is happening.

Investigation must include a detailed history of bowel function, symptoms, and the effect on lifestyle. The present author's own bowel assessment checklist is given in Table 27.1.[13] The following areas should be considered.

27.2.1 Usual Bowel Pattern

- *How often do you open your bowels?*

"Normal" bowel function varies between one and three times a day to once in 3 days.[14] It probably is a minority of the total population (40% of men and 33% of women) who have their bowels open once each day.[15] Most are irregular in bowel habit, with young women being the most irregular. One third of young women open their bowels less than once per day and 1% do so less than once per week.[15]

Table 27.1. Assessment of faecal incontinence checklist.

Main complaint:	Evacuation difficulties?
Duration of symptoms/trigger for onset	Straining?
Usual bowel pattern	Incomplete evacuation?
Usual stool consistency (Bristol stool chart)	Need to digitate anally, vaginally, or to support the perineum?
	Painful defaecation?
Faecal incontinence:	
How often? How much?	Bloating?
Urgency? Time can defer for Urge incontinence:	Sensation of prolapse?
Never/seldom/sometimes/frequently	Pads/pants?
Difficulty wiping: Yes ☐ No ☐	Bowel medication?
Sometimes ☐	Other current medication
Postdefaecation soiling: Yes ☐ No ☐	PMH (include psychological).
Sometimes ☐	History of depression/antidepressants?
	Previous bowel treatments & results
Passive soiling: Yes ☐ No ☐ Sometimes ☐	Obstetric history: para:
Events causing?	Difficult deliveries or heavy babies?
Amount of flatus: Control of flatus:	Dietary influences:
Good ☐ Variable ☐ Poor ☐	Weight/Height/BMI
	Fluids (caffeine)
Ability to distinguish stool/flatus?	
Yes ☐ No ☐	Smoker?
Abdominal pain relieved by defaecation?	Skin problems
Rectal bleeding?	
Mucus?	Bladder problems
Nocturnal bowel problems?	Effect on lifestyle/relationships/Emotional/psychological effect

"Constipation" means different things to different people. Infrequent bowel motions, if the stool is easy to pass and not hard, are not a cause for concern or intervention. Conversely, some very constipated people can produce a stool several times per day, but only at the expense of long hours of straining on the toilet. In the general population, there is no age-related decrease in the actual frequency of defaecation, although there seems to be an increase with age in self-report of constipation and an increase in laxative use.[16] Just because someone is taking laxatives, it does not necessarily mean that they actually are constipated.

It is important to record if the patient reports a recent change in the frequency of bowel motions as this may indicate underlying disease or malignancy. Any recent unexplained change in bowel habit in a patient over 40 years should be investigated by barium enema X-ray or by colonoscopy. Asking the patient to keep a diary or chart of bowel actions for a week often provides useful baseline information. This chart can also record food and drinks taken.

27.2.2 Usual Stool Consistency

- *What is your stool (bowel motion) like; is it loose, soft but formed, hard or hard pellets?*
- *Does this vary?*

It is important to record if stool consistency has altered and if the patient reports a change in stool colour. Patients with the irritable bowel syndrome are particularly prone to a very variable stool consistency. Where the patient has difficulty in describing the stool, a visual prompt may be helpful (Figure 27.1). Type 3 or 4 is the most usual consistency, but in women only 56% of stool are these "normal" types; 61% of stool are these types in men.[15] If the stool is loose, this makes both passive and urge faecal incontinence far more likely. Pellet stool is common in slow transit constipation.

27.2.3 Faecal Incontinence

This often will need sensitive enquiry as many people are reluctant to admit FI. It also is quite difficult to gauge severity as many people restrict their lifestyle to limit the possibility of urge incontinence, so it may actually happen very infrequently yet still be a major problem, and many find it difficult to estimate the amount lost. It is helpful to question separately about the two major symptom types of urge or passive incontinence.

27.2.4 Urgency and Ability to Defer Defaecation

- *When you need to open your bowels do you need to rush to get to the toilet?*
- *How long can you hold on for?*

Normally defaecation can be deferred for long periods of time as the urge to defaecate is opposed by voluntary contraction of the external sphincter,[17] which should generate sufficient pressure to prevent immediate stool expulsion and to return the stool to the rectum (see Figure 27.2). If the external sphincter is not functioning well, the squeeze may be insufficient to suppress the urge to defaecate, even when the stool is a normal consistency. A reduced squeeze pressure and an inability to sustain a submaximal contraction has been found to correlate with the symptom of urgency.[18] Patients generating high bowel pressures seen in inflammatory bowel disease or irritable bowel syndrome may report severe urgency. Some patients with faecal incontinence generate pressure waves of very high amplitude (up to 500 cm H_2O pressure). Without adequate sphincter response to oppose this, urge incontinence is very likely.[19] In instances where a patient is suffering from diarrhoea this is especially difficult, due both to increased bowel pressures opposing sphincter function and to loose stools.

27.2.5 Urge Incontinence

- *Do you ever not get to the toilet in time and have a bowel accident?*

This often highlights a weakness or defect in the external anal sphincter, which is unable to oppose rectal contractions and so allow delay in defaecation.[20,21] Urgency may become a persistent problem for an individual who has suffered an episode of faecal incontinence, as

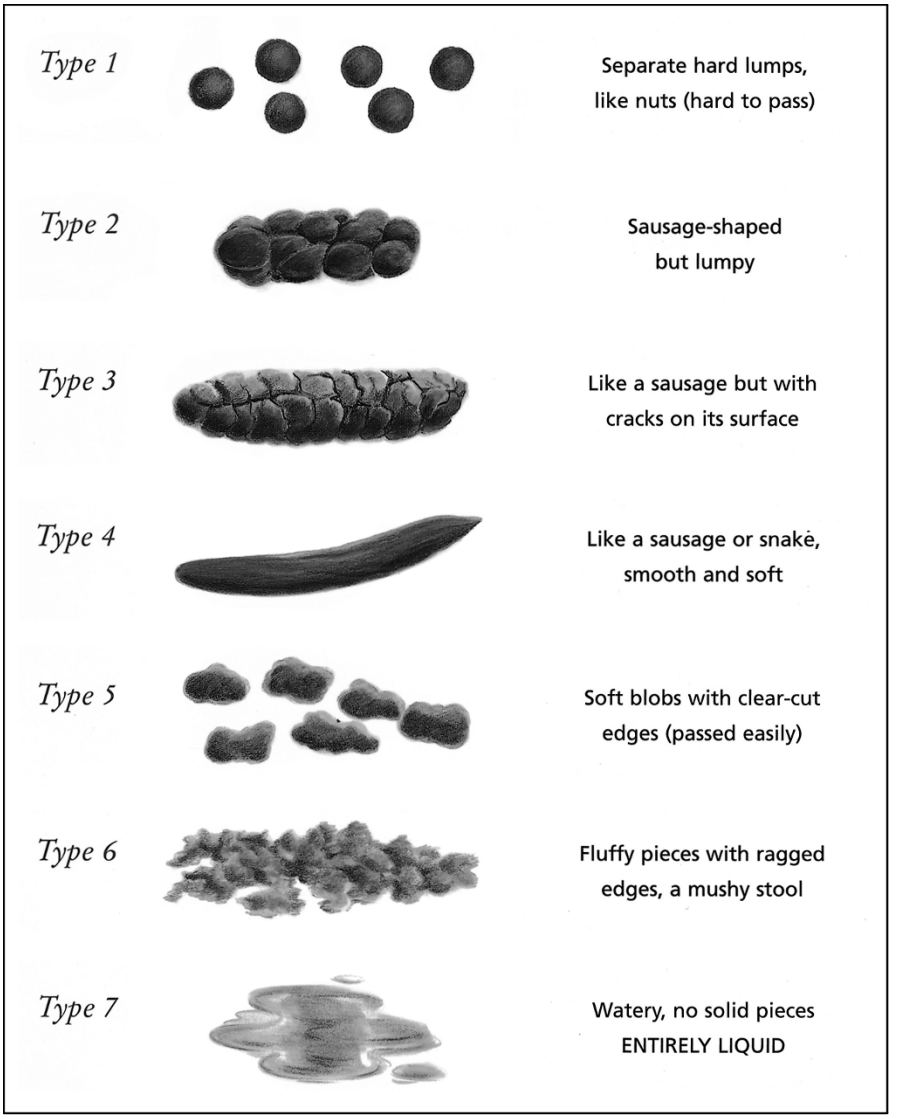

Figure 27.1. Bristol Stool Form Scale, with permission from Dr K Heaton.

the catastrophic effect leads to the fear of future accidents and so an immediate rush to the toilet to prevent this. It is possible that a vicious circle develops—any bowel sensation is interpreted as urgent and likely to lead to incontinence—this causes great anxiety, even panic, which in turn exacerbates the sense of urgency. It is important to establish how frequently urge incontinence actually occurs and whether restrictions in activities are due to actual or feared incontinence. Some people have very infrequent episodes, but self-impose major lifestyle restrictions and never venture far from a toilet "just in case."

27.2.6 Passive Soiling

- *Do you have any leakage from your back passage, of which you are unaware?*
- *Is this liquid or solid?*
- *Does this occur at any time or only after you have opened your bowels?*

The smooth muscle internal anal sphincter is responsible for resting tone in the anal canal. A weak or disrupted sphincter will not close the anal canal completely, and therefore, if the stool is loose or soft, some will remain in the anal canal and will ooze out after defaecation. Passive soiling is associated with internal anal sphincter

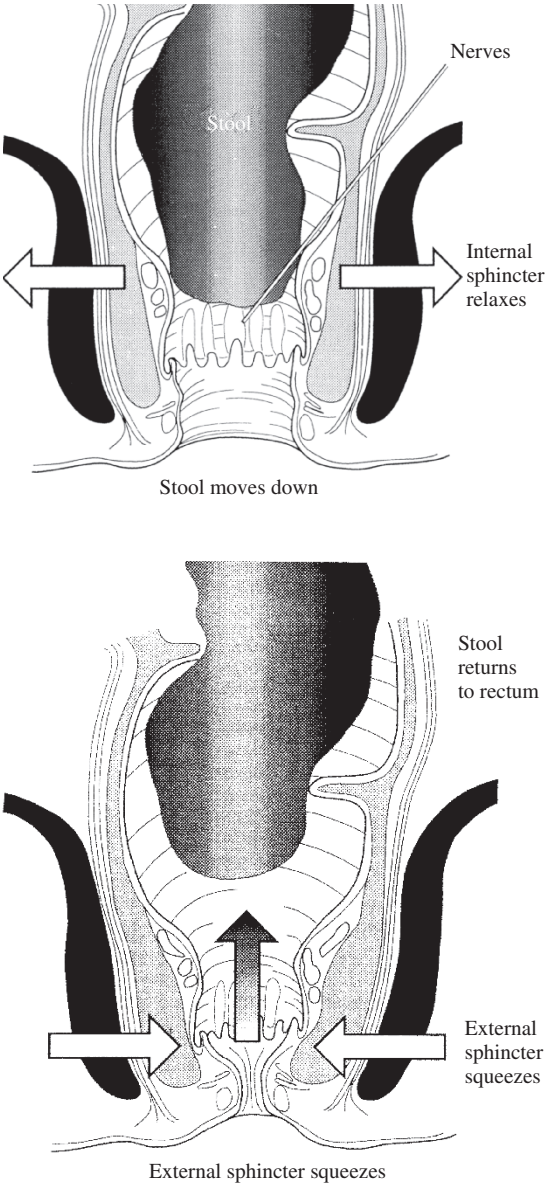

Figure 27.2. Section of the rectum and anal canal. **a** Stool moves down. **b** External sphincter squeezes.

damage on ultrasound.[20,21] These patients complain of great difficulty in cleaning the anus after defaecation and subsequent soiling, possibly for several hours. Others experience passive soiling at any time without being aware of it or loss of stool exacerbated by physical exertion such as walking or playing sports. Passive loss of pellet stool or loss of copious amounts of mucus may result from a rectal prolapse (see below).

27.2.7 Flatus

- *Can you control wind?*
- *Are you able to tell the difference between wind and the need to empty your bowels?*

This also examines the function of the sphincters and whether they are able to maintain an airtight seal. Some patients report incontinence of liquid or solid stool occurring on passing flatus because they are unable to distinguish between flatus and stool.

27.2.8 Presence of Blood and Mucus

- *Do you pass any blood or mucus when you have your bowels open?*

Fresh blood may be passed if a patient has haemorrhoids or an anal fissure. Darker blood may indicate underlying disease or malignancy. The presence of blood and mucus in an individual with a history of straining may indicate the presence of a solitary rectal ulcer. Copious mucus with normal bowel function may indicate a villious adenoma; with disturbed bowel function, mucus is a frequent accompaniment to the irritable bowel syndrome. When a patient reports passing blood and/or mucus further investigation is warranted.

27.2.9 Pain

- *Do you have pain associated with opening your bowels?*
- *Is this before opening your bowels and relieved by opening your bowels or is it a pain as you actually pass a stool?*

Pain with an urge to defaecate may be reported by patients with inflammatory bowel disease or the irritable bowel syndrome, making it difficult to defer defaecation. They may refer to a colicky or spasmodic 'stabbing' pain. Pain with defaecation is often caused by haemorrhoids or an anal fissure. Chronically constipated people often report abdominal discomfort, especially accompanied by bloating.

27.2.10 Evacuation Difficulties

- *Do you have difficulty opening your bowels?*
- *Do you need to strain?*

- *If so, for how long?*
- *Do you ever need to insert a finger into your back passage or vagina to help stool out?*
- *Do you need to push on the area by your back passage?*
- *Does it feel as if you have not completely emptied your bowels?*

These questions examine whether the patient suffers from an evacuation difficulty. See Chapter 28a for details of constipation assessment.

27.2.11 Sensation of Prolapse?

Some people may report a dragging feeling, or even that the rectum protrudes from the anus, particularly during or after straining. Most prolapses will reduce spontaneously after defaecation, but occasionally the patient may find it necessary to manually replace the rectum.

27.2.12 Pads or Pants

- *Do you need to wear a pad due to problems with leakage from your bowels?*
- *If so, what type of pad?*
- *Do you need to change your underwear during the day due to leakage?*
- *If yes, how often?*

Patients may wear pads due to urinary incontinence and so it is important to identify if pads or underwear are changed due to FI. Also, patients who describe urgency may always wear a pad due to fear of incontinence rather than actual accidents.

27.2.13 Medication

Many medications can influence bowel function, either to constipate or to loosen the stool (see Chapter 28c).

27.2.14 Past Medical History

The history may give important clues as to causation. Major neurological disease, abdominal surgery, diabetes, thyroid disease, psychological disturbances, and many other disorders may have an influence on bowel function. Any anal trauma may be relevant, as may unwanted anal intercourse, which has been found to be associated with internal anal sphincter damage.[22] Many women date the onset of bowel symptoms to gynaecological surgery, especially hysterectomy.[23]

27.2.15 Obstetric History

- *How many babies have you had?*
- *Were forceps used for any of these deliveries?*
- *Did you tear or did you have stitches?*
- *How heavy were the babies?*
- *Was there any problem with bowel control following the deliveries?*

Women who have had a difficult delivery, particularly assisted by forceps or involving a third-degree tear, are especially likely to report faecal incontinence.[24] Prolonged labour (particularly the second stage) and heavy babies can cause trauma and damage to the anal sphincters. Postnatally women are frequently asked about bladder function and taught the importance of pelvic floor exercises. Bowel function is much less frequently considered or mentioned. Women with pre-existing irritable bowel syndrome are known to be more likely than others to develop faecal urgency post-partum (64% vs.10%) and poor control of flatus (35% vs. 13%), at least in the short–term.[25]

27.2.16 Diet, Smoking, Weight, and Fluid Intake

Many people find that what they eat influences their bowel function. Nicotine is thought to slow upper gut motility and increase total transit time, but it seems that it can speed rectosigmoid transit,[26] and this fits with many people reporting clinically that smoking a cigarette facilitates initiation of defaecation. It is not known whether obesity has an adverse effect on bowel control. Some people with anorexia become very constipated; others abuse laxatives; a few seem to experience pelvic floor problems, possibly secondary to excessive exercise regimens or muscle wasting. Caffeine is a known gut stimulant[27] and will exacerbate urgency in many patients.

27.2.17 Skin Problems

Some patients with faecal incontinence seem to have few problems with skin excoriation. Others

suffer greatly from soreness and itching. Certainly, if there is diarrhoea, there is the possibility of small bowel digestive enzymes in contact with the skin. If there is both urinary and faecal leakage, this makes soreness worse.[28] Some postmenopausal women may have skin problems due to hormone deficiency (and oestrogens have been suggested to have some beneficial effect on symptoms of faecal incontinence[29]). Where skin problems prove resistant to simple skin care and barrier creams, it is worth seeking a dermatological opinion as there may be secondary infection or a treatable skin condition.

27.2.18 Bladder Control

- *Do you have any problems with leakage from your bladder?*
- *Does urine leakage occur if you cough, sneeze, or laugh?*
- *Do you need to rush to the toilet to pass water?*

This helps to identify any other continence symptoms. Referral for further investigations may be indicated. Up to a quarter of women attending a urodynamic clinic for investigation of urinary incontinence will admit to FI on a postal questionnaire, but only 15% do so on direct questioning, emphasising the difficulty many women have in admitting this symptom.[9] FI more often seems to be associated with an overactive bladder than with stress incontinence, possibly reflecting the overlap of irritable bowel syndrome with an unstable bladder.[30]

27.2.19 Effect on Lifestyle/Relationships/ Psychological Factors

If a patient reports symptoms of faecal incontinence, it is important to ascertain how this has affected his or her lifestyle. Some patients report feeling very restricted, planning their journeys around toilet facilities; others may become housebound. Obviously such behaviour will have a profound effect on the individual and any partner and family. Patients often report avoiding a sexual relationship due to feeling dirty or a fear that an episode of incontinence will occur. Although intuitively and clinically it is often felt that faecal incontinence has a major negative impact on pscyhosocial well-being, there have been remarkably few formal studies of this. A condition-specific quality of life index has been developed.[31]

27.3 Examination

It has been suggested that a good history and physical examination can predict findings of anorectal physiology studies in many cases. A low resting tone in the anal canal on digital examination is associated with passive leakage and there is often gaping of a "funnel-shaped" anal introitus if gentle traction is applied away from the anal verge.[22] Reduced strength and duration of voluntary contraction has been found to correlate with the symptom of urgency.[21,22] Urge incontinence is also associated with reduced puborectalis squeeze and a reduced anorectal angle, and many patients also report urge and stress urinary incontinence.[21]

However, estimation of resting and squeeze sphincter tone has not been found to correlate well with any objective measure of sphincter function. Also, there is as yet no validated scale for assessing the strength or endurance of anal squeeze. Possibly, adaptation of the Oxford grading scale would be useful, but it is not known whether this would be reliable or reproducible or have any clinical relevance. In men digital examination has been found to be reliable except for assessment of fatigue.[32] It is increasingly recognised that fatigue of voluntary squeeze is an important parameter of assessment, and it is possible to work out a fatigue rate index using manometry.[33] It is not known whether digital examination can accurately assess the rate of fatigue of an anal squeeze. It is also very important to remember that there is a lot more to anal continence than just the sphincter; control involves a complex interplay between sphincter function, gut motility, stool consistency, and the physical and mental ability to respond appropriately to the call to stool.[13]

Aspects of physical examination other than the resting and squeeze tone of the anal canal should include:

- Inspection of the perianal skin for skin excoriation, presence of soiling, any congenital abnormalities, and any haemorrhoids or skin tags.

- Inspection of the perineum for scarring from episiotomy or tears (however, perineal inspection has been shown not to correlate well with the presence or absence of occult anal sphincter damage as seen on anal ultrasound, and an apparently intact perineum does not preclude underlying sphincter damage[34]).
- Inspection of the posterior wall of the vagina for any rectocele at rest and on straining.
- Where there is any suspicion from the history of rectal prolapse, this seldom will be apparent if the patient is examined lying down. The best way to check for prolapse is to sit the patient on a commode or toilet, sitting forward on the toilet seat and leaning forward and ask her to strain as hard as she can. A prolapsing rectum will be visible or can be felt at the anal verge or below.
- Perineal descent of greater than 2 cm on straining is considered abnormal.
- The contours of the lower back may suggest previously unsuspected spina bifida occulta.
- The presence of a loaded rectum may suggest constipation or faecal impaction, particularly in a frail or immobile person. Digital rectal examination provides an unreliable indicator of colonic loading, particularly where stools are soft and puttylike rather than hard.[35] Plain abdominal X-ray may be helpful.

Table 27.2. Physical examination results.[a]

Inspection	Palpation	Endoscopy
Perineal soiling	Resting tone	Intussusception
Scars	Squeeze tone	Solitary rectal ulcer
Anal closure		
Muscular defect	Sphincter defects	Scarring
Loss of perineal body	Anal canal length	Mucosal defects
Rectal prolapse	Puborectalis tone and motion	Neoplasm
Muscular contraction		
Perineal descent	Rectal content	Inflammation
Anal skin reflex to pinprick	Soft tissue scarring	Inflammatory bowel disease
Anatomic anorectal pathology:	Rectocele	Infectious colitis
Haemorrhoids	Intussusception	Others
Skin tags		
Fistula	Rectovaginal fistula	Fistula
Mucosal ectropion		
Fissure		
Others		

[a]From Jorge and Wexner.[36]

- A general assessment of physical abilities and any disabilities that might impair the individual's coping with independent toileting.

Ideally, every person should have had a full medical examination to check for any rectal masses. Jorge and Wexner[36] have proposed a format for recording the findings from physical examination (Table 27.2).

27.4 Rating the Severity of Incontinence

It is important for both clinical practice and research that a reproducible and valid estimation of the severity of the incontinence is made. Only thus can the effectiveness of therapy be gauged. There have been several rating scales for faecal incontinence proposed. All have been found to correlate reasonably well with a careful clinical evaluation of the severity of incontinence,[37] but none as yet has been subject to evaluation with large patient groups to examine the ability to reflect changes in severity.

27.5 Investigations

As well as a detailed history, patients should have a digital rectal examination to check for local pathology. Patients with a recent unexplained change in bowel habit need at minimum a sigmoidoscopy and preferably a barium enema or colonoscopy. It has been found that people with colorectal carcinoma are much more likely to report faecal incontinence than age-matched controls, with about a quarter reporting this symptom. [38]

Where the cause of impaired bowel control is unclear or when first-line management has failed, the patient will need further investigations. Anorectal physiology tests and anal ultrasound provide assessment of the function and the structure of the internal and external anal sphincters (see Chapter 26). However, there is some debate as to the value of these tests for routine assessment, with information from tests only changing diagnosis in 19% of cases and resulting in a changed management plan for 16% in one series.[39]

Symptoms, especially their severity, often do not correlate well with objective findings. It seems that as continence is a complex interplay of sphincter integrity and function, as well as stool consistency, gut motility, and many other factors, there is often residual capacity even in damaged muscle.

27.6 Summary

Assessment of bowel control requires appropriate sensitive questioning, which will facilitate discussion and allow patients to voice any symptoms or anxieties. Physiotherapists and nurses will often have access to people at risk of faecal incontinence, and it is important to use every opportunity to allow discussion of this sensitive issue. Patients may express a sense of relief that they have been asked about their bowel control and provided with an opportunity to ask questions and seek help.

27.7 Conservative Management

27.7.1 Bowel Habit and Timing

Some patients find that if they can achieve a regular complete rectal evacuation at a predictable time, then episodes of faecal incontinence can be controlled. Sometimes patients get into a chaotic bowel pattern and all attempts at control feel helpless. Trying to impose a regular pattern, if successful, can greatly enhance the individuals sense of control. The bowel usually is inactive during sleep. Maximal motility occurs on waking for most people. This can be enhanced by eating, drinking, and physical activity. Therefore, the patient is advised to eat breakfast, take a couple of warm drinks, and then attempt to defaecate about 20–30 minutes later (sooner if the urge is felt), using the evacuation techniques outlined below.

Unlike urinary incontinence, where there has been quite a lot of work on bladder retraining for urgency, there is no good evidence that instruction to defer the urge to defaecate for as long as possible can “retrain” the bowel, although clinically it is recommended by expert opinion.[40]

27.7.2 Dietary Management of Faecal Incontinence or Urgency

The bowel is designed for processing food and so diet can affect FI. However, it is not easy to offer advice on this as it seems to vary from person to person and there is very little research on which foods can make incontinence better or worse. What will make life a nightmare for one person seems to have no effect at all on someone else.

It is worth experimenting a little to see if each individual can find anything that upsets bowel control. Fibre supplements have been found to contribute to faecal incontinence in frail immobile people,[35,41] but supplements of soluble fibre can be helpful in those with loose stool.[42] Softer stool is more difficult to hold during an urge to defaecate and also is more likely to passively leak. There are no trials on the effect of fibre reduction on faecal continence, but clinically a lot of patients derive benefit from moderating their fibre intake.[13]

Very spicy or hot food can upset some people. Other foods to consider include milk products and chocolate, which some people find make their stools looser. A few people find that artificial sweeteners (in many low-calorie foods, drinks, and chewing gum) have a tendency to make their stools looser. If the patient thinks that there may be a link with what she or he eats, keeping a diary may reveal if there is a pattern. A course of antibiotics can upset bowel function. Probiotics are an area of increasing interest. These “beneficial bacteria” are found by some patients to regulate bowel function, particularly if it has become disturbed after a bout of gastroenteritis or a course of antibiotics. Research is as yet limited.

Conversely, some foods help to make stools firmer, and therefore easier to control for some people. Arrowroot biscuits, marshmallow sweets, and very ripe bananas each help some people.

Incontinence of flatus is difficult to treat. Dietary changes, such as reduction in bran products, vegetables, and caffeine can reduce the amount of flatus produced for some people. Products such as probiotics and aloe vera are reported as helpful in reducing flatus by some patients.[43]

27.7.3 Drinks

What is drunk can make a difference to some people, and again this is individual and it is worth experimenting. Alcohol seems to cause the bowels to be loose and urgent for some people, but less so for others. Different types of alcoholic drink can affect people in different ways. Some find that beer is better than wine or that white wine is better than red, or vice-versa.

Some people have a bowel that seems to be very sensitive to caffeine, which is in coffee, tea, cola drinks, and expensive chocolate. Caffeine stimulates the colon and so makes the stools move through faster. This means that less fluid is taken from the stools, which are then looser and more urgent. For the patient with urgency, frequency, or loose stool, it is worth trying to spend at least a week without any caffeine (decaffeinated tea, coffee, and cola are available at most supermarkets) to see if this helps. If things are better without caffeine, then the patient has a choice of whether or not to drink it. If someone drinks a lot of caffeine, do not stop it suddenly as headaches may result: advise people to gradually cut down.

27.7.4 Medication

The aim is to achieve a formed stool without actually constipating the patient. Firm stool is easier to hold during urgency and has less tendency to leak. There is some evidence for using loperamide[44] and this is the drug of first choice.[40] If tablets constipate, a liquid formulation makes the exact dose easier to adjust. Loperamide can either be taken regularly or on an "as-needed" basis. As the only significant side effect is constipation, it can be taken in doses up to 16 mg daily on an ongoing basis. Codeine phosphate is an alternative, but has more side effects.

Some patients benefit from using suppositories or a microenema to achieve complete rectal emptying at a time they can control. This can be combined with use of loperamide. There is some initial experimental work on drugs that enhance anal sphincter closure,[44] but none is commercially available.

27.7.5 Exercises

There have been remarkably few studies of pelvic floor muscle training alone for faecal incontinence. One controlled study has suggested that there may be no advantage over patient education and "urge resistance."[45]

27.7.6 Biofeedback

The colorectal literature abounds with case series of biofeedback used for faecal incontinence. This may include the use of manometric pressure or EMG to give the patient feedback about anal squeeze performance during exercises, but more usually it involves more complex two- or three-balloon systems that attempt to teach the patient to alter rectal sensitivity and to respond to the normal decrease in anal pressure when the rectum is distended (rectoanal inhibitory reflex) by a voluntary squeeze to avoid incontinence.[46] Controlled studies are few, and those that exist have not found that one method is superior to any other or that biofeedback is better than exercises alone.[45,47,48]

27.7.7 Electrical Stimulation

Intra-anal electrical stimulation has been used in a few studies as an adjunct to exercises or biofeedback, with some suggestion of improved effect.[49] However, one controlled study has suggested that any effect may be more enhanced sensation than actual motor strengthening,[50] but much more work is needed to confirm this.

27.7.8 Conservative Management of Constipation

Myths abound in the management of constipation.[51] Simple recent-onset mild constipation may respond to increasing fibre, fluids, and exercise levels, but these patients are seldom referred for specialist assessment and care. Many patients have been given the mistaken impression that if only they took more care of themselves the constipation would resolve. This can lead to great frustration and at times counter-productive excessive fibre, fluid intake, and obsessional exercising. Sedentary

patients with constipation may benefit from increased exercise, but those with normal activity levels probably will see no benefit from an increase.

27.7.9 Environmental Factors

People who are unwell or generally weak may be unable to generate enough abdominal effort to stimulate a defaecation reflex. Poor toilet facilities or lack of privacy may exacerbate this or worsen symptoms of urge FI. Confused people may fail to realise what social behaviour is required for toileting and ignore the call to stool. For those in institutional settings, attention to adequate privacy is important.

27.7.10 Defaecation Technique

Adopting a semisquat position by raising the feet on a footstool, breathing normally, and using the abdominal muscles to "brace and bulge,"[52] rather than holding the breath and straining seems to benefit many patients clinically. These techniques can be taught using biofeedback (EMG and/or expulsion of a rectal balloon) to demonstrate good technique and any paradoxical tightening. However, no study has yet shown that biofeedback enhances the effect of simple instruction and patient teaching.

27.7.11 Exercises

Patients with a descending perineum may benefit from pelvic muscle training in order to "stabilise" the anal canal during defaecation. There have been no studies evaluating the effectiveness of this.

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28a Constipation

PE Chiarelli

28a.1 Prevalence of Constipation and Associated Factors

Constipation is a subjective term used to describe difficulty in defaecation, due to straining or the infrequent passage of small hard stools, or both. Bowel patterns in individuals change over time and prevalence studies have used different definitions of constipation; therefore, it is difficult to obtain an accurate estimate of the prevalence of constipation in adult populations. The Rome criteria suggest functional constipation includes a group of disorders with symptoms that include difficult, infrequent passage of stool, sometimes with feelings of incomplete emptying, which have persisted for at least 12 weeks (not necessarily consecutively).[1] In the United States a study found an overall prevalence of constipation of 14.7%; 4.6% functional, 2.1%, irritable bowel syndrome (IBS), 4.6%, outlet obstruction (OO), and 3.4 % reported mixed symptoms of IBS and OO,[2] with a higher prevalence of OO in women and functional constipation in men.

Both the American study and a Swedish study[3] found lower overall prevalence of constipation in women than an Australian study of 41,857 women.[4] This study reported that common to all groups from age 18 to74 was an association of constipation with haemorrhoids and "other bowel symptoms." Also:

- In the *middle cohort (45–49 years)* prevalence of constipation was estimated to be 26.6%; other factors included going through the menopause in the last year, currently taking hormone replacement therapy, having had gynaecological surgery, and taking drugs for sleep or "nerves."
- In the *older cohort* (70–74 years) the prevalence of constipation was estimated to be 27.7%; other factors included having had gynaecological surgery and taking drugs for "sleep" or for "nerves."

Constipation is commonly experienced by women during pregnancy; the overall prevalence is estimated to be about 40%, with 24% of these women reporting constipation occasionally and 16% reporting constipation often or almost always.[5] While studies suggest that these disturbances in gastrointestinal motility might be largely attributed to hormonal alterations,[6] there are many other factors that contribute to constipation during pregnancy.

Constipation is a commonplace symptom, largely self-managed,[4] the impact of which might not be realised by health care professionals. There is some evidence to suggest that other sequelae of chronic straining at stool might include urinary incontinence,[7] pelvic organ prolapse, and anal incontinence.[8] Although constipation is not associated with mortality, a significant proportion of women experience it and the financial costs of its sequelae are great.

28a.2 Aetiology and Pathophysiology

Defaecation has been defined as "a coordinated process of storage and expulsion of faeces that depends on sensorimotor activity of voluntary

and involuntary sphincters. Integration of somatic and autonomic mechanisms, intrinsic gut neural and endocrine systems, cortical and conditioned reflex mechanisms and local and spinal reflex responses are required with voluntary contraction and relaxation of the striated muscles of the pelvic floor and anal sphincters, abdominal wall, and respiratory muscles of the diaphragm and rib cage."[9]

Many of the factors identified as being associated with constipation are also associated with dysfunction of the pelvic floor muscles (PFMs). Damage to the PFMs and their innervation, which can occur during childbirth or gynaecological surgery, may contribute to constipation in women.[10–14] Repeated straining at stool is thought to exacerbate the damage and can result in weakness of the pelvic floor (PF), perineal descent during straining, and secondary anatomical changes. These changes may lead to anorectal dysfunction, difficulties in defaecation, and at their worst, faecal incontinence.[11,15–17] Constant straining at stool also has been implicated in the development of uterovaginal prolapse in the presence of defective uterine supports.[16] About 5% of Australian women aged 45 to 49 years and 21% of older Australian women (70–74 years) report having undergone surgery for repair of a prolapsed bladder, uterus, or bowel.[18]

Constipation is a symptom of many diseases and disorders, both physical and psychological. As well as these myriad extracolonic factors, constipation also may have underlying congenital or pharmacological causes. Colonic causes of constipation are either structurally or functionally based. If conventional investigations do not reveal any organic cause, then constipation is considered to be functional.

28a.3 Functional Constipation

Functional constipation can be subdivided into constipation caused by colonic inertia (slow transit constipation) or obstructed defaecation, which sometimes is referred to as disordered defaecation, anismus, paradoxical puborectalis contraction, or pelvic outlet obstruction.

28a.3.1 Slow Transit Constipation

These patients have no organic cause for their symptoms and have delayed transit through the colon and rectum. It is considered to be a neuromuscular disorder, classified as a disorder of the mesenteric plexus. Patients with slow transit constipation have a normal-sized colon.[19] The upper level of normal transit time might be considered to be 72 hours.[20]

28a.3.2 Obstructed Defaecation

Functional constipation but not outlet obstruction is associated with advancing age, and outlet obstruction is clearly more common in women than men.[21] Obstructed defaecation includes such syndromes as descending perineum (with or without prolapse), dyssynergia (anismus), and less commonly anterior rectal wall prolapse, intussusception, and enteric prolapses.

28a.4 Syndromes Associated with Obstructed Defaecation

28a.4.1 Intussusception

Intussusception is a circular rectal wall infolding of more than 4 mm of the rectal mucosa during straining.[22]

28a.4.2 Enteric Prolapses

Sigmoidocele and enterocele are prolapses of the colon into the rectovaginal fossa. Rectal prolapse is herniation (procidentia) of the full thickness of the rectum through the anal canal.[22]

28a.4.3 Anterior Rectal Wall Prolapse (Rectocele)

This is a herniation of the anterior rectal wall through the rectovaginal septum and posterior vaginal wall into the lumen of the vagina. Found in 20–80% of women, but most are asymptomatic and caused by primary laxity of the rectovaginal septum or by septal damage due to chronic or

excessive straining.[23] Some constipated women assist defaecation by placing one or two fingers into the vagina during a bowel motion and pressing backward toward the coccyx or by applying firm pressure on the perineum. This has the effect of flattening out the "pouch" of the rectocele, which allows emptying to proceed.

28a.4.4 Descending Perineum Syndrome

Usually only seen in women, this syndrome is clinically defined as occurring when the plane of the perineum (led by the anal verge) extends beyond the ischial tuberosities during valsalva; normally the perineum moves 2–3 cm during a bearing down effort. The puborectalis muscle plays an important role in supporting the anal sphincter during defaecation.[27] It helps to "funnel" the contents as the EAS relaxes and allows for faecal expulsion. When the muscles are overstretched as in the descending perineum syndrome, this support is lost.

28a.4.5 Muscle Dyssynergia

The normal coordinated action of the muscles of the PF often is taken for granted. Two types of impaired muscle coordination can affect the act of defaecation.

- Some patients contract rather than relax the muscles around the anal canal during expulsive efforts. During a rectal examination, it may found that during attempts at expulsion (simulated defaecation), the anorectal angle becomes more acute rather than being felt to widen and/or no decrease in anal sphincter tone.
- Some patients are unable to generate an adequate propulsive force within the pelvis.

28a.5 Assessment of the Constipated Patient

The comprehensive assessment of a patient complaining of constipation should include a close examination of the symptoms experienced by the patient, examination of the abdomen by palpation, and a physical examination of the PFMs and their function. This necessitates a rectal examination in males and a vaginal/anal examination in females including assessment of the patient's straining pattern. Diagnostic criteria for chronic functional adult constipation, the Rome II criteria,[24] are shown in Box 28a.1.

Box 28a.1

Rome 11 Criteria [24]

The patient should report *at least two* of the following symptoms *for at least 12 weeks* (not necessarily consecutively) within the previous 12 months

- Straining in ≥ 25% of bowel movements
- Lumpy or hard stools in ≥ 25% of bowel movements
- Sensation of incomplete emptying in ≥ 25% of bowel movements
- Fewer than three bowel movements per week

Symptoms of constipation often manifest themselves as part of IBS. It is important that clinicians try to differentiate between slow transit constipation and disordered defaecation while realising that both can be experienced simultaneously.[20]

Clinicians use the term "constipation" to describe infrequent, incomplete, difficult, or prolonged evacuation or to describe stools that are too small, too hard, or too difficult to pass; patients tend to dwell on such symptoms as bloating, abdominal and pelvic pain, and nausea. Investigators in the US have developed a well-validated constipation scoring system (CSS) aimed at obtaining a universally objective definition to assist in the clinical diagnosis and treatment of functionally constipated patients[22] (see Table 28a.1).

Using the CSS, patients scoring more than 15 (out of a possible 30) are considered to have constipation. Careful evaluation of the pattern of symptoms within the CSS provides information that might allow for clearer differentiation

Table 28a.1. The constipation scoring system.[a]

Symptom	Score
Frequency of bowel movements	
1–2 times per 1–2 days	0
2 times per week	1
Once per week	2
Less than once per week	3
Less than once per month	4
Difficulty: Painful evacuation effort	
Never	0
Rarely	1
Sometimes	2
Usually	3
Always	4
Completeness: Feeling incomplete evacuation	
Never	0
Rarely	1
Sometimes	2
Usually	3
Always	4
Pain: abdominal pain	
Never	0
Rarely	1
Sometimes	2
Usually	3
Always	4
Time: Minutes in lavatory per attempt	
Less than 5	0
5–10	1
10–20	2
20–30	3
More than 30	4
Assistance	
Without assistance	0
Stimulative laxatives	1
Digital assistance or enema	2
Failure: Unsuccessful attempts for evacuation per 24 hrs	
Never	0
1–3	1
3–6	2
6–9	3
More than 9	4
History: Duration of constipation (yr)	
0	0
1–5	2
5–10	3
10–20	3
More than 20	4

[a]From Agachan et al.[22] Reprinted with permission from Dr S. Wexner.

between those patients with slow transit constipation and those experiencing disordered defaecation (see Table 28a.2).

28a.5.1 Physical Assessment of the Constipated Patient

Examination of normal sequences of bowel function shows that the important physical components from a therapist's point of view are PFM dysfunction (weakness, imbalance, or incoordination) as well as a normal straining pattern. A full physical examination of patients complaining of constipation should include a visual assessment of the perineum and the anus at rest, during contraction, and during straining (see Table 28a.3). Following this a digital assessment PV and/or PR of the vagina and the anorectum and PFM function (at rest during contraction and during straining) should be carried out (see Chapter 7 and Table 28a.4).

28a.5.2 Vaginal Assessment of the Pelvic Floor Muscles

PFM function should be tested according to guidelines suggested by the International Continence Society[26] and the results recorded (see Chapter 7).

28a.5.2.1 Anorectal Assessment

A full explanation of why the examination is being carried out should be given at every stage; also a warning should be given that the patient might feel as though she might lose control of her bowels. She should be reassured that this is a normal response and that any "accidents" are highly unlikely to happen.

While visual inspection of the anal area can be made at the same time visual assessment of the perineum is taking place, a more efficient visual anal assessment can be made with the patient in a side-lying position with a pillow supporting the upper thigh, a towel draped over the buttocks to provide a sense of modesty.

The examiner uses a clean, gloved, well-lubricated index finger; the patient is told when the rectal examination is about to begin and reassured that no instruments will be used. Gentle pressure is first applied on the posterior anal verge until the sphincter yields and the *relaxed* finger glides gently into the anus, the fingertip aiming forward initially and then posteriorly in

Table 28a.2. Comparison between patient profiles of slow transit constipation and disordered defaecation.[a]

Constipation group	General comment	Main symptoms	Method used to effect bowel emptying
Slow transit constipation	Predominantly female Many failed treatments	Abdominal pain and bloating No spontaneous bowel movements	Best results with laxatives
Disordered defaecation		Feelings of incomplete emptying Often requires digitation	Best results with enemas, suppositories, and digitation

[a]Table adapted from studies by Pemberton[20] and Agachan.[22]

the direction of the coccyx. It is sometimes helpful to ask the patient to bear down to facilitate relaxation of EAS. If resistance is still felt, ask the woman to gently bulge the lower part of her abdomen, that part just above the pubic hairline. The relaxed finger should slide gently in past the anal sphincter and will naturally come to rest upon the anorectal angle with the tip of the finger pointing toward the coccyx (see Figure 28a.1).

The examining finger can sweep across the levators from anterior to posterior and back again on either side of the rectal canal to feel for muscular deficiencies in the puborectalis. The patient's proprioception can be assessed by gently applying downward pressure onto the levators on each side and asking the patient to describe what they can feel. In constipated patients, there may be little sensation in the presence of overstretched muscles, especially in cases where descending perineum syndrome is evident. On removal of the examining finger any traces of blood should be noted and reported appropriately.

28a.5.2.2 Assessment of Defaecation Pattern

For this assessment, the patient should sit, clothed, on a firm chair without back support. She is then asked to adopt her usual defaecation position. The therapist places one hand on the woman's lateral abdominal wall at waist level while the other hand is placed suprapubically over the lower abdomen. The woman is asked to simulate defaecation and inappropriate responses are noted. This pattern is then corrected, if necessary, according to the guidelines in Figure 28a.2 and Table 28a.4.

28a.6 Treatment Programmes for the Patient with Constipation

Dietary advice and advice regarding fluid intake are routinely given to patients complaining of constipation (see Chapters 17 and 27). PFM rehabilitation will be necessary where muscle weakness, imbalance, or incoordination (dyssynergia)

Table 28a.3. Outline of the physical assessment of the constipated woman.

Assessment	At rest	During a contraction	During straining
Anorectum (digitally)	Pull the anus apart gently at the 9 o'clock and 3 o'clock positions to see if it gapes at rest Constant resistance during finger insertion, gradually relaxing. Palpate circumferentially for areas of thinning or absent tissues. Anorectal angle should be acute. Faecal bolus present? Feel puborectalis as a sling posteriorly. Sweep across the bodies of puborectalis, pubococcygeus, and iliococcygeus	Muscle deficiencies in EAS usually on anterior wall. Use a sweeping motion to assess the levator ani and check for areas of: Atrophy Hypertrophy Unilateral or bilateral elevation Assess proprioception	Anorectal angle should open and become more obtuse. Puborectalis should be felt to relax The EAS should relax

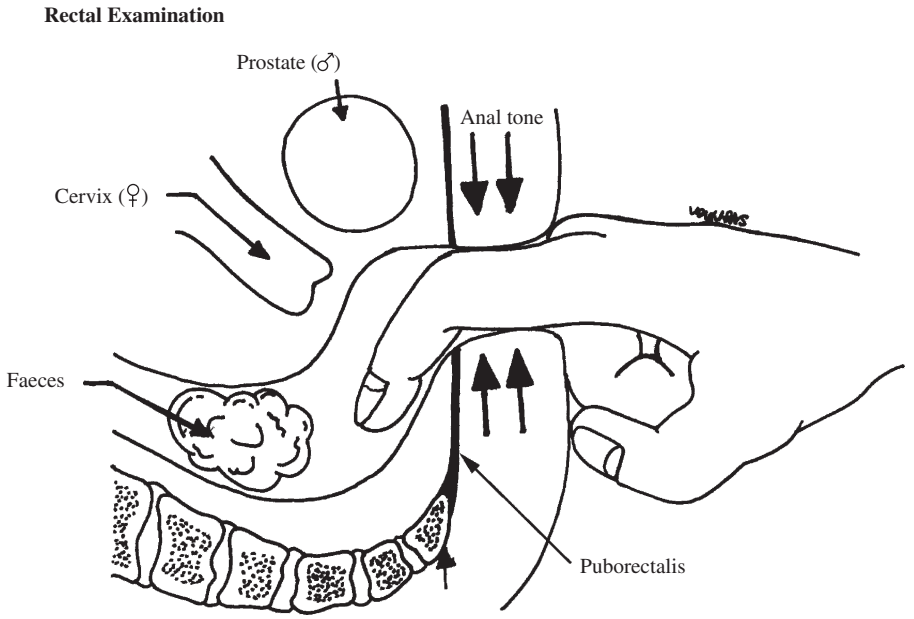

Figure 28a.1. Rectal examination.

have been found and should be tailored to suit each patient. If incorrect defaecation patterns are detected, they will require retraining. Biofeedback programmes of various types are used to manage pelvic floor dyssynergia, but the evidence supporting the efficacy of such protocols is weak.[28]

28a.6.1 Pelvic Floor Muscle Rehabilitation

Various methods for PFM rehabilitation have been addressed comprehensively in Chapters 11a–c, 12a,b, 13, and14.

Many studies have shown that the response to isometric resistance training is very specific to the exercise both in terms of the muscle itself, the muscle length in isometric exercise, the speed of the contractions, as well as the postural specificity of the exercise.[29,30] It has also been suggested that rather than using conventional exercises to strengthen individual muscle groups, it might be more effective to identify particular functional deficits and then repeatedly practice these, with or without added resistance.[29,31] There seems little point in restoring muscle strength if this cannot be appropriately utilised in activities of daily life. It is not yet certain whether strength training provided soon after a period of disuse in the form of voluntary muscle contractions can induce a faster or a more complete recovery of muscle strength than that provided by the correct performance of everyday activities alone.[29]

Since the transversus abdominis (TrA) plays an important role in the development of abdominal pressure (AP) as well as enhancing PFM contractions,[32] careful attention should be paid to the cocontraction of TrA during PFM contractions. This is achieved by asking the woman to pull in and hollow her lower abdomen each time she performs any PFM contraction.

28a.6.2 Improving Perineal Elevation

Teaching patients to precede a cough by PFM "bracing" has been called "the knack" and has been shown to be important in reducing stress urinary incontinence in women.[33] Since studies show the urethral and anal sphincters both work maximally during a cough,[34] it seems rational to use this bracing manoeuvre as part of any strengthening programme, as well as to provide specificity to PFM exercise during simulated coughing, nose blowing, and when lifting objects.

Figure 28a.2. The body position and functional pattern for effective defaecation.

Table 28a.4. Assessment of the defaecation pattern.

The patient's body	Correct response	Incorrect response
Position:		
• Arms	• Forearms resting on the thighs	• Any other position
• Legs	• Hips at more than 90°, heels raised	• Hips at $<90°$, feet not touching the floor, or, feet flat on the floor.
• Spine	• Forward lean with a normal spinal curve	• Posterior pelvic tilt, slumping
Muscle actions:		
• At the waist	• The waist widens	• No change or the waist narrows
• Lower abdomen	• The stomach bulges	• No change or is pulled inward
• Anus	• Opens	• Tightens, moves up, or closes
The breathing	• Deep, basal, diaphragmatic • The intercostal angle increases • Breath held with diaphragm held down low	• No basal expansion • Diaphragm held high

28a.6.3 Retraining Altered Defaecation Patterns

In retraining patients with disordered defaecation, the goal is to increase abdominal pressure (AP) without increasing the anal sphincter pressure. Relaxation of the internal anal sphincter (IAS), the external anal sphincter (EAS), and the puborectalis is facilitated by the squatting position and by increased AP which accompanies straining. An increase in AP during heavy lifting does not normally produce the same response. There is a functional interrelationship between the visceral and somatic elements of the sphincter mechanism but these are not clearly understood.[9]

Sensory training uses simulated defaecation by means of air- or water-filled balloons within the rectum. Patients are encouraged to concentrate on relaxation of EAS to facilitate "defaecation" as the balloon is slowly removed.

Using *EMG and manometry,* intravaginal or intra-anal pressure probes can be used to provide feedback. Alternatively, intraluminal or surface EAS electrodes show the patient how to relax their PFM during attempts to defaecate. Patients are asked to gradually increase their strain effort while maintaining relaxation of the PFM.

28a.6.3.1 Defaecation Dynamics

Other studies have favourably compared the outcome of muscle training with and without the aid of biofeedback.[35] This supports the use of defaecation retraining as proposed by Markwell.[36] Such retraining should be tailored to individual dysfunctional straining patterns (see Table 28a.4).

- *Correct position.* This should be enhanced by the use of a small stool under the feet.
- *Lower abdominal bulging.* Leaning forward, forearms supported on knees, hands in front of the abdomen, the patient is asked to relax the belly and let it bulge out in front. It should be allowed to "fall into the hands." In this position, the lower abdomen should be made to "bulge and harden." This is brought about by eccentric rectus abdominus activity in the lengthened position, which in turn inhibits activity in the EAS and puborectalis.
- *Facilitate defaecation.* Intra-abdominal pressure is increased (by contraction of external obliques, internal obliques and transversus abdominus) against a closed glottis. Contraction of the EAS during defaecation (anismus) may be caused by contraction of the TrA with rectus abdominis in a shortened position (ie, belly pulled in) and is common in patients with extra strong recti.
- *Anal sphincter opening.* Relaxation can be taught only following contraction. This can be facilitated using EMG or manometric biofeedback or the use of a simple hand mirror.

When each of the component parts of the defaecation pattern has been mastered, the pattern can be practiced as a whole. Thereafter, this sequence should be used only on the toilet.

28a.7 Prevention of Constipation

The consequences of constipation can be seen to be significant. In view of the association between urinary incontinence (UI) and constipation,[16,37] it would seem prudent that health care professionals routinely include the careful assessment of the bowel habits of all women presenting themselves for the treatment of UI.

28a.7.1 During Pregnancy

Antenatal education should include information about the impact of constipation on women's health as well as pointing out the causative factors of constipation during pregnancy.[25]
These factors include:

- Stool dehydration resulting from decreased fluid intake which is secondary to nausea, vomiting, and peripheral oedema.
- Changes in dietary patterns together with iron supplements.
- Psychological problems.
- Delayed colonic transit. This can result from hormonal changes, including increases in plasma progesterone levels and decreased levels of oestrogen and motilin.
- Pseudo-obstruction. Outlet obstruction caused by haemorrhoids, anal fissures, increased pressure arising from the gravid uterus, and adhesions from previous deliveries.[38]

28a.7.2 Postpartum Period

Focus groups held among postpartum women revealed wide disparity in the levels of information and assistance given to them during their hospital stay. They also revealed that women in the postpartum period do not seek help for constipation from health care professionals but feel confident with self-management of their symptoms.[39] The postpartum continence promotion programme designed using a customer focus includes perineal descent-minimising techniques such as perineal support during defaecation, information about the long-term consequences of constipation, and checking the straining patterns of women who admit that they experience constipation.

28a.7.3 Postoperative Urogenital Surgery

In view of the significant association between constipation and urogenital surgery, it seems rational to propose that women routinely be given instructions about avoiding constipation. These might include instruction in correct straining patterns for defaecation along with functional PFM exercises and information about diet, laxatives, and fluid intake before discharge from hospital.

28a.7.4 The Elderly

The prevalence of constipation in elderly people is much higher than in the rest of the population. Decreases in levels of mobility, poor teeth, concomitant medical conditions, and polypharmacy all contribute to constipation in the elderly.[40]

28a.8 Conclusions

The promotion of good bowel habits should be seen as an important contribution to a holistic approach in the management of patients who are experiencing any form of PF dysfunction.

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28b
Abdominal Massage for Constipation

MC Emly

28b.1 Introduction

The usual treatment for chronic constipation is laxatives and in many cases laxative polypharmacy.[1] Long-term use leads to further constipation and impaction.[2] Abdominal massage has no known adverse side effects and a treatment programme may re-educate normal bowel activity.[3,4]The massage, by altering intra-abdominal pressure, has a mechanical and reflex effect on the gut, thus encouraging peristalsis.[5] The effectiveness of abdominal massage may be further enhanced by utilising the mass movement of the gut, so increasing the strength of the contraction, and therefore its propulsive force. A massage programme may reduce gastrointestinal transit time, soften stool and load the rectum.[6]

Abdominal massage has been found to be effective in people with chronic constipation and/or faecal incontinence, altered abdominal muscle tone, abdominal pain due to cramps or flatulence, and problems with defaecation. It is not recommended where there is a history of malignant bowel obstruction or abdominal tumours, inflammatory disease of the intestine, spastic colon with irritable bowel syndrome, unstable spinal column, or pregnancy. Hollis[7] lists general contraindications to massage. Abdominal massage should never be used in isolation but only as part of a holistic bowel management strategy.[8]

28b.2 Abdominal Massage Technique

The following paragraphs describe the technique as developed in a primary care setting in Leeds, UK,[4,9] based on similar methods described in older physiotherapy textbooks.[3,5]

The abdominal massage is applied in a clockwise direction around the presumed course of the large intestine on a relaxed abdominal wall. The treatment takes 15 to 20 minutes.

There are four basic strokes in abdominal massage for constipation: stroking, effleurage, kneading and vibration. Before commencing the treatment the abdomen is assessed for flatus, pain, and faecal matter in the gut.

The massage begins with a gentle relaxing stroke up the abdominal wall. People with hypertonicity may experience an initial tightening of the abdomen in response to touch. In the case of hiatus hernia or reflux, the direction of the stroke is reversed.

28b.2.1 Stroking

Stroking then continues from the small of the back, over the dermatome of the vagus nerve, over the iliac crests, and down both sides of the pelvis toward the groin. This is repeated several times and followed by effleurage.

28b.2.2 Effleurage

Effleurage strokes follow the direction of the ascending colon across the transverse colon and down the descending colon as shown in Figure 28b.1. This is repeated several times with increased pressure to stimulate the haustral and segmental contractions of the large intestine. This will assist in propelling the faecal matter along the gut.

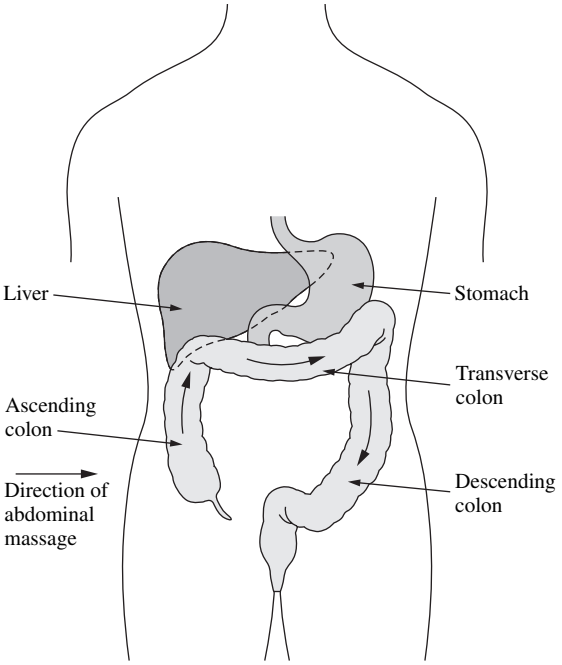

Figure 28b.1. The anatomical basis for abdominal massage.

28b.2.3 Kneading

Palmar kneading down the descending colon, up the ascending colon, and down the descending colon once again is the heart of the massage. Kneading assists in propelling the faecal matter along the gut to load the rectum. Finger kneading may be required to break up faecal mass. This part of the massage may sometimes be uncomfortable because of the deep compression required.

There is then a return to effleurage following kneading, continuing wth a relaxing transverse stroke over the abdomen. The massage concludes with vibration over the abdominal wall to relieve flatus.

28b.2.4 The Research Evidence Base

Ernst[10] conducted a systematic review of four clinical trials and one single case study looking at the efficacy of massage to treat constipation. All the studies were small and only two had controls. Comments regarding the five studies are as follows:

1. Merlo treated 14 constipated, physically disabled children for 8–10 weeks with five massage sessions per week. Results showed a 42% increase in stool frequency for half the group.
2. Twelve weeks of massage and exercise applied to 12 elderly, immobile, long-stay patients resulted in a significant increase in stool frequency with a decrease in faecal incontinence.
3. Seven healthy volunteers and nine elderly constipated patients each acting as their own control were measured for colonic transit time, stool frequency, weight, and consistency during a control phase and a massage treatment phase of nine sessions. The results showed no significant changes in outcome measures overall, although stool frequency and weight were greater during the massage phase for healthy volunteers.
4. A randomised controlled crossover trial measured gastrointestinal transit times for 32 profoundly disabled adults with a history of laxative use over a 7- week massage phase and a 7-week laxative phase. Stool frequency, size, consistency, the requirement for enemas, and patient well-being also were compared. There were no statistically significant differences between either treatment.[6]
5. A single case study using abdominal massage and connective tissue manipulation found that both treatments improved baseline measures for a constipated adult.

Ernst concluded that although none of the trials were free from methodological flaws, the results imply that massage could be a promising treatment for chronic constipation.[10] Therefore, there is a need for more rigorous research. Randomised control trials of larger population groups need to be studied over longer periods of time.

28b.2.5 Strategy for Implementation

The health professional deciding to embark on a massage programme and having obtained patient consent needs to consider the following:

- Contraindications to treatment. If in doubt, consult the patient's general practitioner or referring consultant.
- An individualised bowel management plan that reviews exercise, diet and fluid intake, toileting posture, and medication use, especially laxatives.[8]

- A baseline assessment of stool frequency, size, and consistency and the requirement of enemas/laxatives.
- Regular treatment. Recommended regime is five sessions per week.[6]
- Recording of all treatment outcomes to compare with baseline measures.
- The timing of the massage to coincide with the usual bowel pattern to utilise the mass movement. This can be triggered by a hot drink before the session.
- Review of laxative use following increased stool frequency associated with massage.
- Review of the number of weekly massage sessions if there is a return to normal bowel habit.

28b.3 Conclusions

Since there will be an ongoing need for treatment in most cases where massage is considered appropriate, careful consideration must be given as to how this can be sustained. The training of care staff/carers in the technique and guidance on application therefore is necessary. Self-massage also can be taught. A training module has been developed in Leeds, UK for this purpose.[4]

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28c
Laxatives

K Irwin

28c.1 Introduction

Laxatives are used to stimulate or increase the frequency of bowel evacuation, or to encourage a softer or bulkier stool. It is not always necessary to resort to the use laxatives, as some causes of bowel dysfunction can be simply resolved.[1] With the implementation of a simple stepped approach, practitioners can provide patients with the appropriate information on dietary and fluid intake, along with lifestyle and general advice with regard to their symptoms and treatment. Other causes of constipation may be more complex, and therefore require further investigation and treatment.[2] The cost to the National Health Service for treating constipation is significant,[3] with prescriptions for laxatives rising dramatically over the last 5 years. Over 14 million prescriptions have been written annually for laxatives, with the overall cost to the National Health Service being suggested as £47 million.[4] This does not take into account the nonprescribed laxative items being purchased over the counter.

There is limited clinical evidence available to objectively measure the efficacy and effectiveness of laxatives, which unfortunately can lead to the inappropriate choice of laxative preparation.[5] The National Prescribing Centre, however, does suggest that in the absence of randomised controlled trials, the choice of laxative should be based on the presenting symptoms, patient preferences, side effects, and cost of medication.[1]

28c.2 Assessing the Problem

Laxatives should not be introduced as first-line intervention to treat constipation.[1] A detailed history should be taken[8,9] to establish the nature and underlying cause of the problem. It is important to ensure that the constipation is not secondary to an underlying undiagnosed complaint that warrants further investigation. It also may be necessary to undertake a physical examination, which may include inspection of the anal/perianal area and digital rectal examination. Guidance now is available regarding digital rectal examination[10] and the health care practitioner should consult local guidelines and protocols regarding these procedures.

Constipation may be alleviated simply. Diet, in particular fibre intake, should be assessed along with review of fluid intake. The recommended intake of fibre is between 18 and 30 g per day. However, in some patients, increasing fibre can worsen their symptoms. Caution also should be exercised in patients with obstructive symptoms, such as faecal impaction, slow transit constipation, and defaecatory disorders.[1] Fluid intake should be 1.5–2 litre/day for healthy adults. Review of lifestyle, physical activity, environment, toilet habits, and routines is also essential. It is only once this approach has been implemented that other methods of treatment and management should be considered.

28c.3 Prescribing Laxatives

Choice of agent will depend largely on presenting symptoms, nature of complaint, patient acceptability, and compliance. Health care practitioners should refer to the BNF/NPF for information on doses, indications, cautions, contraindications, and side effects prior to prescribing. Table 28c.1 outlines some of the commonly used laxatives.

Table 28c.1. Some common causes of constipation.

- A diet that is insufficient or lacks adequate fibre/fluid intake
- Delay in colonic transit time
- Nerve injury or disease that may include patients with spinal injury, multiple sclerosis, Parkinson's disease, and cerebrovasucular accident
- Hormonal changes, eg, menopause, pregnancy, hypothyroidism, and diabetes
- Medications, eg, opiate analgesics, anticholinergics, antidepressants and iron preparations,
- Evacuation difficulties caused by hard impacted stools or nerve damage
- Anorectal conditions, eg, haemorrhoids or anal fissure, rectal prolapse, rectocele, anismus (contraction of anal sphincter instead of relaxation), megacolon or megarectum
- Bowel disorders such as Inflammatory bowel disease, inflammatory bowel syndrome, and carcinoma
- Cognitive impairment, eg, senile dementia, Alzheimer's, and learning disability
- Psychiatric disorders, eg, depression, eating disorders
- Surgical/diagnostic procedures, eg, barium enema/meal, post-operative constipation
- Environmental factors may be indicated.
- Habit/routine, eg, ignoring the call to stool
- Immobility due to frailty or illness
- Underlying medical conditions

The use of laxatives long term should not be necessary. There are circumstances, however, where repeated or prolonged use may be required, for example, progressive neurological conditions and where opioids are used in palliative care.[1]

28c.4 Bulking Agents

These preparations can be used for patients who are unable to modify and increase their dietary fibre intake and in those who need to increase the bulk of their stool. It is important to advise the patient that due to their mode of action, these preparations may take a number of days to work. It also is important that patients are advised to drink adequate amounts of fluid. They can be used long term in those patients with normal gut motility and uncomplicated constipation see Box 28c.1.[1,11]

28c.5 Stimulant Preparations

It is thought that these preparations act on the myenteric nerve plexus, thus stimulating the nerves that produce colonic contractions. Stimulant laxatives should be used with caution especially in elderly people and those with faecal impaction. These preparations are commonly taken at bedtime,[1] and should be used as a short- term measure or for intermittent use see Box 28c.2.

Box 28c.1

Bulking Agents Key Points

- Can take several days to have an effect.
- Not suitable for acute relief, but can be used long term in those with normal gut motility.
- Not to be used in elderly patients with an atonic bowel. Also the thirst reflex may be diminished in the elderly.
- They may cause abdominal discomfort, bloating, and flatulence.
- Ensure good fluid intake.
- Avoid use in faecal impaction, colonic atony, and severe dehydration.

Box 28c.2

Stimulant Laxatives Key Points

- Rapid action 8–12 hr
- Stimulant laxatives should be used short term, for simple constipation, however, intermittent use may be justifiable
- Side effects of abdominal cramps may lead to poor compliance
- Avoid these preparations in intestinal obstruction
- Danthron is potentially carcinogenic and is therefore only prescribed in the terminally ill[12].

28c.6 Osmotic Preparations

Osmotic preparations work by increasing the amount of fluid in the large bowel. This is achieved by reducing the absorbency of fluid from the bowel and by drawing fluid from the body to the bowel.[11] Magragol preparations are also used in the treatment of constipation see box 28c.3.

Box 28c.3

Osmotic Laxatives Key Points

- Often prescribed as first line treatment for constipation, yet should not be recommended as such.
- May be used both short or longer term.
- Can be used when stimulants/bulking agents failed.
- Lactulose may take up to 48 hour to work and needs to be taken regularly for maximum efficacy.
- Movicol can be considered for the treatment of faecal impaction.

28c.7 Faecal Softeners

These preparations are used where defaecation is difficult and hard stools require softening.[1,11] They act by decreasing surface tension and increasing penetration of intestinal fluids into the faeces see Box 28c.4.

Box 28c.4

Faecal Softeners Key Points

- Liquid paraffin is no longer recommended for use[1]
- Docusate sodium acts as both a stimulating and softening action. This can be of particular use where defaecation is painful; for example anal fissures and haemorrhoids.

28c.8 Rectal Intervention

It is important to mention rectal preparations. If oral laxatives have been considered or prove ineffective or the patient requires an immediate evacuation to relieve the rectal loading/impaction, it may be necessary to administer a suppository or enema. If left untreated, constipation may lead to faecal impaction, and as a result faecal incontinence may also occur secondary to the impacted colon. Patients also may present with urinary retention as a result of the rectal loading.

Table 28c.2. Commonly prescribed laxatives and rectal preparations.

Bulk-forming laxatives

- Ispaghula (eg, Fybogel, Isogel, Regulan)
- Sterculia (eg, Normacol)
- Methylcellulose, which also acts as faecal softener (eg, Celevac)
- Natural bran (eg, generic or Trifibre)

Stimulant laxatives

- Bisacodyl (eg, dulco lax)
- Senna (eg, senakot, manevac + Isphaghula)
- Docusate Sodium (eg, dioctil, docusol), also faecal softener
- Danthron, restricted use (eg, codanthrumer/codanthrusate)

Osmotics

- Lactitol
- Lactulose
- Magragols
- Idrolax
- Movicol, also used for faecal impaction

Rectal preparations

- Osmotics
  Sodium citrate (eg, Microlax, Relaxit, Microlette, Fleet Micro enemas)
  Phosphates (Fletchers phosphate enema)
- Faecal softener
  Arachis oil* check for nut allergy
- Stimulant suppositories
  Glycerol
  Bisacodyl

Choice of preparation will very much depend on the patients presenting symptoms. A rectal faecal softener or phosphate enema may be used in severe cases of hard impacted stool; however, one should exercise caution when using phosphate enemas[13] and exclude nut allergy in the case of arachis oil enemas. Stimulant rectal preparations may be used to evacuate the lower rectum of either hard or soft stools.

28c.9 Conclusions

When laxatives or enemas are prescribed, it is essential that the health care practitioner reviews and considers laxative withdrawal once the constipation has been cleared. If a laxative regimen has been prescribed for use over a longer period of time, for example, in the case of some patients with neurogenic and spinal cord injury bowel management, regular reviews should be undertaken.

Health care practitioners play a key role in improving the management of constipation. By implementing simple steps and providing patients with the appropriate advice and information in regard to their symptoms and treatment, constipation can be successfully treated and managed. It also is vital that practitioners take an active role in the prevention and recurrence of constipation.

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IV Pelvic Pain

29
Prevalence, Definitions, and Aetiology of Pelvic Pain

SJM Knight and ER Shelly

29.1 Introduction

Pelvic pain is a poorly defined condition, affecting women and men, which often becomes chronic in nature. It is difficult to diagnose and may have a multifactorial origin.[1]

The nature and severity of the pain may bear little or no relationship to the actual physical findings. In many cases extensive investigation still reveals no obvious pathology.[2] Lack of a specific diagnosis and continuation of symptoms has a severe effect on the quality of life for the sufferer.

Chronic pelvic pain (CPP) may be defined as "noncyclic pain of 6 months or more duration that localizes to the anatomic pelvis, anterior abdominal wall at or below the umbilicus, the lumbosacral back or the buttocks, and is sufficient to cause functional disability or lead to medical care."[3] Chronic pain is pain that has been present for 6 months or longer, due to persistent nociceptive input. Acute pain indicates actual or potential tissue damage and serves as a useful warning signal. When pain persists, even after healing has taken place, the nerve cell membranes undergo a change in conformation, establishing new receptor sites and the formation of new neural pathways that become embedded into the central nervous system. Once established, the chronic pain pathways are difficult to eradicate. Chronic pain no longer serves a useful purpose, but leads to physical, behavioural, and psychological changes that combine to produce the pain experience.[1]

Management of CPP is variable. Patients may be referred to gynaecologists, urologists, colorectal specialists, and pain specialists and undergo multiple tests and invasive procedures only to be told "nothing is wrong." Others may be given a specific medical diagnosis or clustered into "syndromes." The musculoskeletal system is frequently overlooked during medical evaluation of pelvic pain. A significant amount of literature exists to support the role of the physiotherapist on the CPP team.

29.2 Prevalence

The prevalence of pelvic pain is not clearly known due to inconsistency in definitions and differential diagnoses. The prevalence of female CPP is estimated to be 14–24%.[4,5] An extensive community-based study in the UK[6] reported a prevalence of CPP of 21 in 1,000, slightly less than that of asthma (37 in 1,000).

In a community-based study in the US,[5] 61% of the CPP group ($n = 773$) had pain of unknown aetiology. Those with a definite diagnosis had a variety of conditions, including endometriosis, pelvic inflammatory disease, and irritable bowel syndrome. Musculoskeletal problems were not cited among the diagnostic findings. The total annual direct costs were estimated to be 2.8 billion dollars in the US. Respondents had significantly lower general health scores and higher scores for depression and other quality of life measures. Eighty-two ($n = 548$, 15%) of women in full- or part-time employment reported one or more lost work hours per month because of pelvic pain.

A controlled questionnaire study of demographic and other variables was administered to women with idiopathic pelvic pain of at least 6 months duration.[2] In this study the CCP group

took three times more prescription medications than the pain-free control group. Additionally, the total number of minor and major surgical procedures performed on the study group was significantly higher, indicating a large financial cost to health-care resources.

Women with CPP tend to have a higher lifetime prevalence of sexual abuse.[7,8] Collett[7] compared women with CPP, pain not related to the pelvic area, and a pain-free control group. The CPP group had a significantly higher history of sexual abuse than the comparison pain group or the control group.

29.3 Aetiology of Pelvic Floor Pain Syndromes

A significant population of urologic, gynecologic, and colorectal patients present with pain of unknown etiology. The most common pain diagnoses related to the PFM are outlined in Table 29.1. Factors that may cause or perpetuate pelvic pain are discussed in the following sections.

Table 29.1. Terminology associated with pelvic foor pain syndromes.[a]

Condition	Signs/Symptoms
Levator ani syndrome[10] Pelvic floor tension myalgia[11]	Poorly localised pain in the perivaginal, perirectal, suprapubic, or coccyx re gions, possibly down the posterior thigh; PFM spasm
Nonrelaxing puborectalis, puborectalis syndrome[9]	Bowel movement dysfunction, constipation
Paradoxical puborectalis contraction[12]	
Proctalgia fugax[13]	Sharp, fleeting rectal pain (seconds to 20–30 minutes)
Coccygodynia[14,15]	Coccygeal pain and tenderness noted primarily when seated
Vaginismus[16]	Spasm or active holding of introital muscles, dyspareunia
Urethral syndrome[17]	Urinary urgency, frequency, and suprapubic pelvic or perineal pres sure; urethral pain, burning, or sen sitivity; pain after voiding
Vulvodynia[18]	Chronic genital burning, pain, and rawness
Pudendal neuralgia[19]	Constant localized burning, itching sensations

29.3.1 Musculoskeletal Factors

King et al.[20] examined 132 patients with CPP, most of whom had undergone unsuccessful gynaecological and psychological treatment. Of those patients, 75% were identified as having a typical pattern of faulty posture. This was described as having an exaggerated lordotic curve in the lumbar spine, an anterior tilt of the pelvis, and associated problems of reduced internal rotation of the hips, decreased iliopsoas length, and shortened hypertonic pelvic floor muscles. The musculoskeletal components relating to hypertonus dysfunction include joint, muscle or movement dysfunctions.

29.3.2 Joint Dysfunction

Mense[21] describes how changes in normal joint input may influence the process of reflex inhibition and atrophy of muscles. He acknowledges that there is no generally accepted concept that explains why muscles react with spasm to one lesion and inhibition to another. Anecdotal evidence suggests that postural muscles tend to become hypertonic and phasic muscles demonstrate inhibition. Trauma to the coccyx or pelvis at childbirth or from direct falls can create pelvic floor muscle changes, or hypertonus, in response to ligament injury or joint malalignment.[22] Coccygodynia is a common diagnosis where the coccyx joint can cause pain or spasm in the pelvic floor, specifically the coccygeus muscles.[14,15] The length and strength of the PFM also can be influenced by asymmetries of the sacroiliac, symphysis pubis, or hip joints.

29.3.3 Muscle Dysfunction

Myofascial pain is recognized as one of the etiologies of chronic pelvic pain.[23] Falls, sexual abuse, vaginal delivery, episiotomy, or pelvic surgery can cause soft tissue and muscle injury to the pelvic floor region, with resultant pain. Myofascial pain syndromes may be due to trigger points or soft tissue dysfunction.[24]

Pelvic floor hypertonus is a muscle impairment in which an increase in PFM tension or muscle spasms cause musculoskeletal pain or dysfunction of the urogenital and/or

colorectal systems. Muscle pain differs from cutaneous and visceral pain in that it is difficult to localize, refers pain to other deep somatic tissues, and usually is perceived as aching and cramping.[21]

29.3.4 Trigger Points

A myofascial trigger point (TP) is defined as a hyperirritable spot in skeletal muscle that is associated with a hypersensitive, palpable nodule in a taut band.[24] The TP is painful on palpation and can give rise to characteristic referred pain, referred tenderness, motor dysfunction, and autonomic phenomena. An active TP is always tender, prevents full lengthening of the muscles, weakens the muscle, and mediates a local twitch response of muscle fiber when adequately stimulated.

The onset of symptoms is related to some degree of muscle overload which may be acute, sustained, and/or repetitive. Causes of TPs parallel those of muscle hypertonus as they may stem from musculoskeletal, psychogenic, visceral or systemic, iatrogenic, and neurological origin. Hypertonic muscles do not always contain trigger points.

Travell and Simons[25] identified TPs in specific muscles of the pelvic region (see Figure 29.1). Slocumb[26] found trigger points in 131 (74%) of 177 patients referred for evaluation of CPP. In addition, Reiter and Milburn[27] reported abdominal trigger points in approximately 55% of women referred for CPP following negative laparoscopy.

29.4 Movement and Muscle Recruitment Dysfunction

Movement dysfunction can occur with abnormal use patterns of the PFM. An example of abnormal use is chronic holding of the muscles due to a "falling out feeling" due to prolapse or fear of bladder leakage. Dysfunctional use patterns such as contraction of a muscle rather than relaxation in cases of paradoxical puborectalis contraction is a cause of some types of constipation.

29.4.1 Psychogenic Factors

Lifestyle and stress issues and past sexual abuse can increase muscle tension held in the PFM.[28]

29.4.2 Visceral or Systemic Factors

Pelvic organ pathology such as pelvic inflammation or infection can trigger PFM hypertonus in response to pain or lead to a chronic "holding" pattern of the PFM. In addition, systemic influences of the endocrine and metabolic function such as oestrogen or thyroid hormone deficiency can activate latent trigger points.[25]

29.4.3 Iatrogenic Factors

Urogenital or rectal surgery may result in painful inhibition associated with guarding of the PFM. Adhesions or scar tissue can create pain or muscle tenderness in the PFM. The position on the operating table may cause joint dysfunctions resulting in subsequent pelvic pain.

29.4.4 Neurological Factors

Neurological factors can contribute to painful urogynaecologic or colorectal symptoms. The ilioinguinal nerve (L1) and the genitofemoral nerve have pain distribution patterns in the region of the medial groin, mons, labia, and inner thigh (Figure 29.2).

The character of this pain varies from constant burning to sharp or stabbing and can be aggravated by lifting movements of the ipsilateral leg, walking, or coughing. Nerve entrapment is an often misdiagnosed postoperative complication. A Pfannenstiel's incision can cause postoperative adhesions restricting the iliohypogastric and genitofemoral nerves and referring pain to the genital region.[29]

There are three potential sites for entrapment of the pudendal nerve. These sites include a constriction between the sacrotuberous and sacrospinous ligament, in the pudendal canal of Alcock, and as the pudendal nerve straddles the falciform process of the sacrotuberous ligament. Pudendal nerve entrapment is a recognised cause of chronic perineal pain and may be caused by a variety of factors, including childbirth and long- distance cycling.[30]

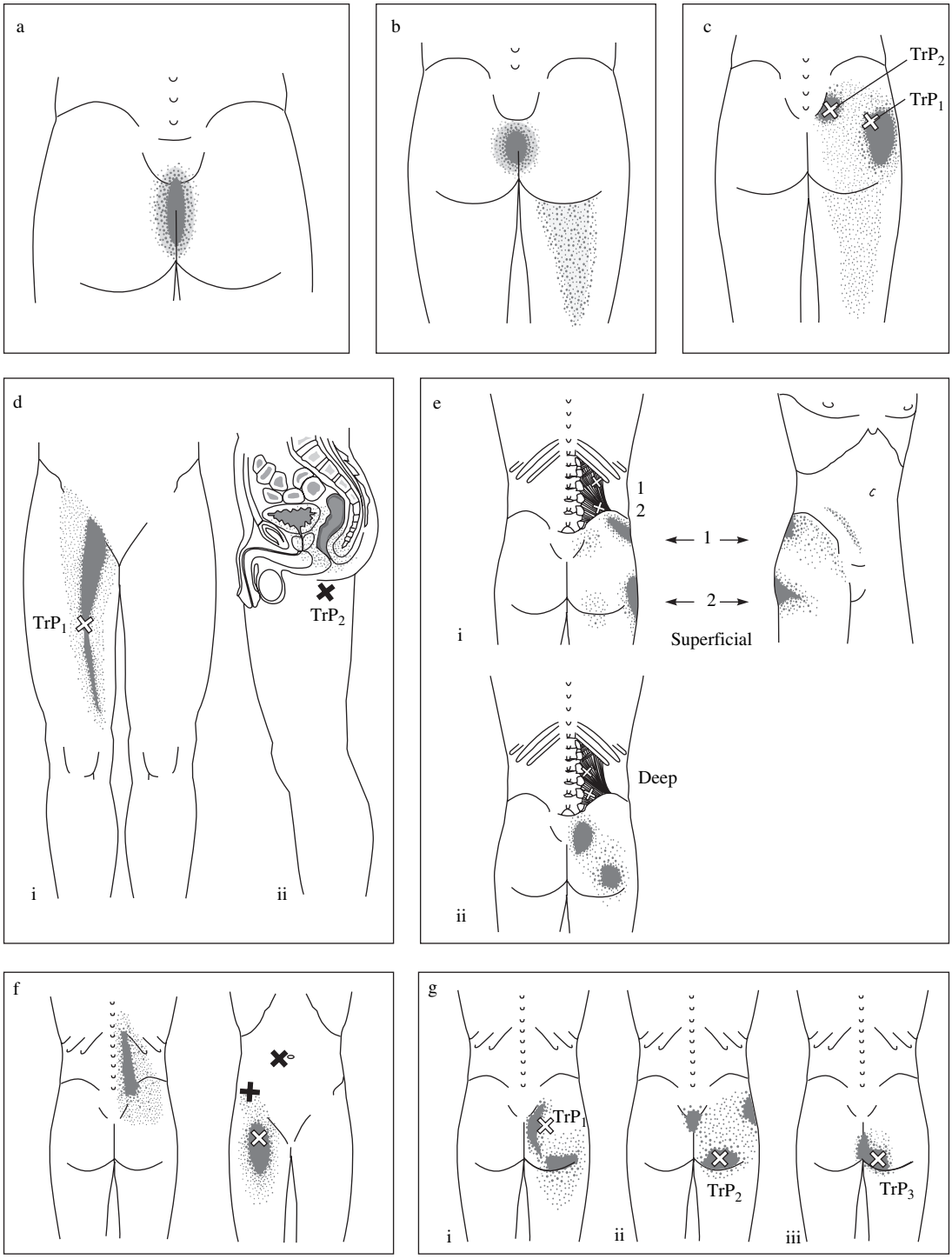

Figure 29.1. Trigger point referral patterns.[25] **a** Levators ani. **b** Obturator internus. **c** Piriformis. **d** Adductors. **e** Quadratus lumborum. **f** Iliopsoas. **g** Gluteus maximus.

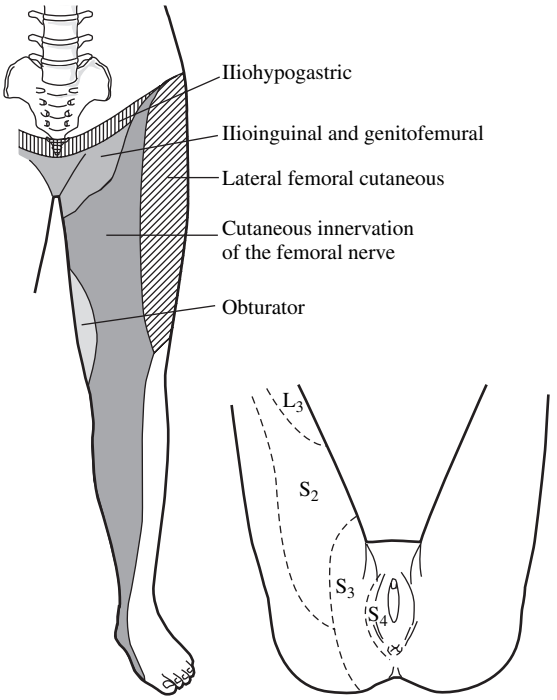

Figure 29.2. Sensory nerve innervation of the lower limb and perineum.

The upper lumbar levels of disk herniation and protrusion also can refer pain to the pelvic region.

In summary, pelvic floor pain syndromes may be influenced by musculoskeletal conditions, psychogenic or iatrogenic causes, neurological pain, or systemic diseases, or the PFM may be the primary souce of pain.

29.5 Vulvodynia

29.5.1 Prevalence and Definitions

The prevalence of vulval pain in women attending gynaecology clinics is estimated to be around 14%.[31,32] The community prevalence is unknown. The vulva is the term used for the female external genitalia. It includes the skin around the vaginal opening, the labia minora and majora, the clitoris, and the perineum. There are many reasons for a painful vulva, and women with vulval pain require investigation by a dermatologist or gynaecologist to identify any treatable pathology.

The term "vulvodynia" was a term established by the International Society for the Study of Vulval Diseases (ISSVD) in 1984 to describe a condition associated with vulval discomfort or burning.[33] Subsequently, the ISSVD divided vulvodynia into subcategories to include a variety of vulval skin conditions, vulvar vestibulitis, and vulvodynia. Vulvar vestibulitis (VVS) was described by Friedrich in 1987,[34] as vulval pain associated with vaginal penetration, localised to the vestibule, with variable findings of erythema. Vulvodynia is a burning or knifelike pain, which is constant and more diffuse in nature. The pain may extend to the anus and inner thighs, and often coexists with interstitial cystitis.

The most recent ISSVD classification abandons the terms vulvodynia and vulvar vestibulitis in favour of referring to all vulval pain of unknown origin as a chronic vulval pain syndrome, which may be localised or generalised, and provoked or unprovoked, or a combination of these categories. It is widely agreed that most vulval pain disorders are multifactorial problems, aetiologically and psychologically.[35]

29.5.2 Aetiology

Vulvodynia is often idiopathic with many possible theories as to the cause. Onset of symptoms may occur after yeast infection, sexually transmitted diseases, vulvar or pelvic surgery, a stressful period of time, or pregnancy. The pelvic floor muscles may develop increased tension as a secondary response to the original irritation. White et al.[36] have proposed a theory based on instability of the PFM, diagnosed by surface EMG, which leads to increased irritability of nerve endings supplying the vulva and the development of a chronic pain syndrome.

29.6 Male Pelvic Pain

Male chronic pelvic pain syndrome is included in the classification, as the prostate gland is assumed to be the source of pain. The condition is currently classified as category III chronic abacterial prostatitis/chronic pelvic pain syndrome/CPPS,[37] after tests for bladder or prostate pathology prove negative (Table 29.2). In men under 50 years of

Table 29.2. Classification of prostatitis.[a]

Category	Classification
I	Acute bacterial prostatitis
II	Chronic bacterial prostatitis
III	Chronic prostatitis/CPP syndrome
IIIA	Inflammatory
IIIB	Noninflammatory
IV	Asymptomatic inflammatory prostatitis

age this is a common urological diagnosis, with a prevalence of around 16%.[38] The main symptoms of chronic prostatitis are pain in the perineum, lower abdomen, testicles, or penis, with variable lower urinary tract symptoms. Medical treatments are based on the assumption that the source of the problem is the prostate gland, even though investigations are negative. It has been recognised that structures other than the prostate may be responsible for the pain and may include involvement of the musculoskeletal system, in particular the PFM.

The PFM are closely associated with the prostate gland and urethra. If these muscles become overactive or "hypertonic," they may develop trigger points referring pain to the perineum, lower abdomen, and genitalia and symptoms that mimic those of prostatitis. Hetrick et al.[39] discovered increased pelvic muscle tone, tenderness, and PFM spasm in a group of men with CPPS ($n = 30$) compared to an asymptomatic control group ($n = 51$). Biofeedback may be a useful tool in reducing pelvic floor muscle hypertonus.[40]

Anderson et al.[41] studied a case series of 138 men with CPP syndrome who had failed prior medical therapy. The protocol combined myofascial release with so-called paradoxical relaxation training. The authors report that 69% of the patients had clinical improvement, associated with a 25% reduction in pain scores. Further research is necessary to establish pelvic floor physiotherapy as a clinically significant treatment for male CPP.

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30
Assessment and Treatment of Pelvic Pain

SJM Knight and ER Shelly

30.1 Introduction

The role of the physical therapist in the evaluation of pelvic pain is principally to identify and treat neuromusculoskeletal factors contributing to this multifactoral condition. However, it is important that therapists adopt a holistic approach to the examination of these complicated patients.[1]

This section will describe the subjective and objective evaluation of a patient with chronic pelvic pain (CPP). These components are in addition to a standard musculoskeletal evaluation of the lower quadrant. The urogenital and gastrointestinal systems also require extensive evaluation to provide a differential diagnosis and eliminate any red flags.

30.2 Subjective Assessment

The examination usually begins with the therapist – patient interview. At least 1 hour should be allowed for the initial interview to gather information and also to establish a therapeutic relationship with the patient.[2] It is important to allow women with pelvic pain to "tell their story," as much as possible without interruption.

30.2.1 Subjective Reports of Pain

Location, duration, nature, and intensity of symptoms should be recorded. Whole body diagrams and detailed diagrams of the perineum are needed to document exact pain locations. Pain intensity may be measured by visual analogue or numeric ranking (0–10) scales.[3] There are many different outcome measures for functional ability and quality of life scores.

30.2.2 Dyspareunia

Questions about dyspareunia can help identify the location of the dysfunction. Frequency of sexual interaction varies widely among the general population and there is no "normal" frequency of intercourse. Current dysfunction should be compared to the frequency of intercourse before the dysfunction occurred. Women with primary dyspareunia have difficulty judging symptoms as they have never experienced pain-free intercourse. General questions such as "Can you have intercourse whenever you like?" may be used to assess function (see Box 30.1). Lack of desire and limited arousal are common problems with pelvic pain patients and may require intervention from a psychosexual counsellor.

30.2.3 Depression

Rosefeld[5] reports chronic pain and sexual dysfunction as two risk factors for depression. Referral to appropriate professionals is indicated if depression appears to be affecting treatment.

30.2.4 Sexual Abuse

Sexual abuse is a common finding in pelvic pain patients. If a patient discloses a history of sexual abuse, treatment should be modified accordingly.[6,7] Survivors of sexual abuse often need to progress more slowly and may take longer to achieve therapy goals. Referral to a psychosexual counsellor may be considered and discussed with the patient.

Box 30.1

Specific Questions about Dyspareunia with Interpretation

1. Do you have pain on penetration or deeply? Penetration pain is usually associated with adhered introital scars (episiotomy, vaginal surgery) or dermatological lesions. Vulvodynia also can result in penetration pain. Deep pain can be related to PFM spasm or organ dysfunction/adhesions.
2. How much pain do you have and how long does it last? Pain is usually rated on a visual analogue or numeric scale. Length of pain after intercourse can give some indication of the irritability of the tissues
3. Complex scales can be used to determine sexual function. However, physical therapists usually find simple scales, like the one listed below, to be adequate.[4]
   - 0–No pain or limitation of intercourse
   - 1–Some pain with intercourse but no limitation in frequency of sexual activity
   - 2–Significant pain with intercourse and limitation in frequency of intercourse
   - 3–Severe pain and inability to tolerate any penetration

   Level two may be further clarified with percent of successful intercourse.
4. Can you achieve orgasm? Does it increase pain? Orgasm is a complex process and is seldom easily interpreted. Pain with orgasm may indicate PFM spasm. Lack of orgasm (anorgasmia) may indicate PFM weakness. However, there are many other reasons for pain with orgasm and anorgasmia.

30.3 Objective Assessment

Objective assessment for the pelvic pain patient includes posture assessment, joint integrity, muscle length and strength, and palpation.

30.3.1 Postural Assessment

The initial examination begins with an observation of the patient's habitual posture. Note the position the individual chooses for standing, is weight bearing equal on both lower extremities? Is the weight shifted to one side in relationship to pain or habit? A standard musculoskeletal evaluation of the pelvis and lower extremity should be completed including lumbar, sacroiliac, pubic symphysis, hip, knee, and ankle joints.

The position of the coccyx, pubis, and sacrum can affect the length of the PFM, and thus contribute to painful dysfunctions in that muscle group. Dysfunction of the lumbar spine, hips, and other lower extremity joints also can affect length/tension relationships in the PFM. For example, an anatomically short lower extremity often results in unilateral standing habits that place torsion or rotational stresses on the sacroiliac joints and pubic symphysis.

30.3.2 Muscle Strength and Length Tests

Normal length and strength of all hip and trunk muscles is necessary for pain free function of the pelvis. All pelvic and trunk muscles require testing in the usual manner (see Box 30.2).

30.3.3 Vulvar Assessment

The vulvar region should be checked visually as well as physically for erythema, oedema, discharge, and for sensitivity to pressure or to light touch. Sensory testing should be performed in the saddle area. This can be accomplished using a cotton tipped swab applied to dermatomal regions

Box 30.2

Important Muscles to be Tested Include

Gastrocnemius
Soleus
Iliopsoas
Quadratus lumborum
Abdominal muscles
Piriformis
Obturator internus
Lumbar paraspinals
Adductors
Gluteus maximus and medius

and compared bilaterally. Reflex testing also should be completed by brief stroking of the clitoris (bulbocavernosus reflex) and of the perineal body (anal wink). A normal response to both would be a reflex contraction of the superficial perineal muscles or anus, respectively. A cotton swab test, using a moistened cotton tip swab, is used to assess pain in the vestibule. Pressure is exerted at the introitus in three areas: three, six, and nine o'clock (12:00 = clitoris). A positive test is recorded when the patient complains of pain with gentle inward pressure in all these areas.

30.4 Palpation

Palpation may be the most important part of the evaluation in some patients and great time and care is taken in careful evaluation of all soft tissue structures. Palpation of joint structures for oedema, tenderness, and temperature change is important to complete the examination of these structures. Soft tissue palpation also includes muscles and their associated tendons, fascia including skin and scars, of the trunk, and lower extremities. All the muscles and tendons of the pelvic area, including the pelvic floor, are assessed for length, strength, stiffness, trigger points, and scar mobility.

30.4.1 Abdominal Palpation

Abdominal palpation requires knowledge of visceral anatomy. With training, a therapist can develop the ability to assess organ position, mobility, and motility. Carnett's test is used to differentiate abdominal wall pain from visceral pain, by palpation of the abdomen with and without head and shoulder raising. If the abdominal wall is the source of pain, pain may increase when a head and shoulder lift is performed. If the pain decreases when the abdominal muscles are tensed, this indicates that the viscera are the source of pain, as the abdominal wall is now protecting the organs from the examining pressure.[8] The psoas muscle can be palpated through the relaxed abdomen with care taken not to compress organ structures. Any suspicious mass in the abdomen should always be reported to the physician.

30.4.2 External Palpation of the Pelvic Floor Muscles

If the patient does not consent to vaginal or rectal palpation of the intrapelvic structures, it is possible to perform external palpation of some of the deeper pelvic structures. This is done with the patient in side-lying with the top leg approximately in 60 to 80 degrees of hip flexion with the knee flexed comfortably. Two or three pillows are placed under the top leg for support. Myofascial release of muscle or connective tissue and external palpation of the sacrotuberous ligament can also be carried out in this position.

It is also important to fully assess the external perineal structures at the pubic arch, lateral to the clitoris, around the ischial tuberosity and coccyx.

30.4.3 Internal Palpation of the Obturator Internus and Piriformis

The obturator internus can be palpated through the vagina and rectum by palpating slightly more cephalad and lateral than the PFMs. The piriformis can be reached through the vagina, but is more easily palpated through the rectal canal slightly lateral to the sacrum and cephalad to the coccygeus. The sacrospinous ligament is located just behind the coccygeus muscle.

30.5 Treatment of Pelvic Pain

The key to successful outcomes from physiotherapy intervention is a therapeutic plan with specific goals based on examination findings. Therapists should remember that treating a PFM dysfunction in isolation without also addressing other musculoskeletal imbalances will probably be ineffective.

Patient education, stress management, therapeutic exercise treatments, modalities, and manual treatments are all utilized in the management of the pelvic pain patient. Adherence or compliance to a therapeutic program is influenced by several factors, one of which is the patient's understanding of the purpose of the prescribed interventions. The possible cause of pain and potential interventions should be explained at the initial appointment. Therapy will be less successful if the patient's expectations are unrealistic.

30.5.1 Posture and Body Mechanics

Interventions aimed at posture impairments in pelvic pain patients have been reported to be successful by many clinicians.[9–11] The primary goal is to have spinal curves in neutral whenever possible to reduce strain on lengthened tissues and joints.

30.5.1.1 Sitting Postures

Prolonged sitting often increases coccyx pain. Proper sitting instruction is the most important posture education for patients with PFM spasms, levator ani syndrome, and coccygodynia in order to relieve undue pressure on the coccyx (see Box 30.3). Sinaki et al.[9] reported marked improvement or complete resolution in 19 out of 35 patients receiving posture instruction for tension myalgia of the PFM.

30.5.2 Relaxation and Stress Management

Linton[12] and others have emphasised the importance of relaxation training in the management of chronic pain. Excessive muscle tension in the PFM, low back, and hips are known to be associated with pelvic pain. Diaphragmatic breathing and progressive relaxation are useful methods of relaxation training, while biofeedback is helpful in achieving relaxation of specific muscle groups.

Box 30.3

Chairs and Cushions

- Avoid chairs with excessive cushion and poor support
- Avoid hard chairs without a cushion
- Doughnut cushions increase pressure on the coccyx and should be avoided by most patients, but may relieve perineal wound pain
- Special coccyx cushions increase anterior pelvic tilt and relieve pressure on the coccyx area.
- Vulvodynia patients typically do not benefit from any special type of cushion.
- All patients are instructed to change positions frequently.

30.5.3 Therapeutic Exercise

Three categories of exercise are important in pelvic pain patients: general aerobic capacity, trunk stability, and muscle balance around the hip. Patients with chronic pain often have adopted a sedentary lifestyle and become generally deconditioned. General aerobic exercise helps to enhance overall body health and cardiovascular condition, increase circulation, decrease daily stress and depression, aids in pain relief (due to endorphin release), and can prevent reoccurrence of myofascial pain.[13] Many patients with PFM spasm and vulvodynia find walking to be very helpful in decreasing pain. Jogging and other high-impact activities may aggravate pelvic floor muscle pain. Swimming is suitable exercise for most patients, except those with vulvodynia who may be irritated by chlorine in the water.

Trunk stability is necessary for pelvic pain patients. Richardson et al.[14] presented EMG studies of the interaction of the PFM with the tranversus abdominis (TA). Poor functioning of the TA could disrupt this interaction and result in dysfunction of the PFM.

30.5.4 Manual Therapies

Manual therapy has become a highly specialised skill with many diverse modes. There are many manual therapy techniques useful for pelvic pain patients, some of which are listed in Box 30.4

30.5.5 Connective Tissue

The connective tissue system includes ground substance, elastin, collagen, bone, cartilage, and adipose tissue.[15] Connective tissue houses blood and lymphatic vessels and nerve bundles.

Box 30.4

Manual Techniques Useful for Pelvic Pain

- Soft tissue mobilisation
- Myofascial release/trigger point release
- Visceral mobilisation
- Proprioceptive neuromuscular facilitation
- Strain–counterstrain
- Muscle energy and joint mobilisation

Loose connective tissue or fascia spreads in a three-dimensional web throughout the body. Every muscle, bone, organ, nerve, and vessel is wrapped in fascia. The purpose of fascia is to separate, support, bind, nourish, connect, and defend.

Treatment of fascial restrictions is essential to restoring pain free function.

30.5.6 Soft Tissue Mobilisation for Pelvic Floor Dysfunction

Thiele[16] introduced massage of the levator ani and coccygeus muscles via the rectum. This technique involved rubbing the muscle fibres from origin to insertion with a stripping motion 10 to 15 times on each side. This technique can be modified according to the location of specific restrictions.

The concept of soft tissue mobilisation (STM) is to treat according to the specific layer of tissue restriction and direction of motion barrier. The goal is to achieve the desired results while using the least amount of force. The tissue is compressed to evaluate for depth of dysfunction. Direction of dysfunction is assessed by directing force across the fibres as if touching the numbers on a clock. Dysfunction is noted by restriction of fascial movement; this may or may not be the most painful area. To release a myofascial trigger point, sufficient force must be exerted on the restriction, in a precise depth, direction, and angle to take the dysfunctional tissues to their end range. As the tissue "lets go," the therapist follows the pathway of the release. The patient can assist the treatment with conscious relaxation, contract/relax of the PFM, and verbal feedback.

Patients with adhered episiotomies and abdominal scars may be taught STM techniques to improve scar mobility.

30.5.7 Modalities for Pelvic Floor Muscle Spasms

The use of modalities to relieve pelvic floor pain has two main objectives: to directly treat and heal muscle spasm and to decrease pain so the patient can participate in activities that will help in the healing of the spasm. Modalities also may be used before or after manual therapy or exercise. Use before treatment helps to prepare the tissue for stretching. Application after manual treatment or exercise may help to reduce posttreatment soreness. Modalities that may be applied include heat, cold, ultrasound, and TENS/neuromuscular electrical stimulation (NMES).

30.5.7.1 The Application of NMES and TENS

The positioning of electrodes bilaterally on the sacrum over the S3 sacral nerve roots may be an effective placement for perineal pain. Stimulation of the S3–S4 nerve roots may decrease abnormal activity in the PFM, thus causing a decrease in pain and restoring normal voiding patterns.[17] A crossover four-electrode pattern on the lower abdomen is often effective for abdominal pain. Some iliopsoas spasm and trigger points can be treated by placing electrodes unilaterally on the abdomen and back.

Rectal or vaginal electrodes also may be used to treat pelvic floor and perineal pain. Using an anal electrode at 20 Hz stimulation has been found to be effective in reducing pain in male patients with CPP syndrome.[18] In another study women with levator ani spasm were treated successfully with vaginal electrical stimulation, although there was no long-term follow-up.[19] In cases of pelvic pain refractory to all other treatment, sacral nerve root stimulation may provide effective pain relief.[20]

30.5.8 Biofeedback

Early surface electromyography (SEMG) research[21] clearly showed that muscle tension may be present even when the patient or therapists cannot feel it. This is especially true in the PFM of women, as nerve input is often disrupted following childbirth. High resting baseline SEMG with high variability and occasional spasms sometimes are seen in pelvic pain patients. Ideally, the patient will gain the ability to recognize the difference between relaxation and contraction. The use of SEMG biofeedback to enhance muscle relaxation is referred to as "down-training." (see Box 30.5) Teaching a muscle to relax is often more difficult than teaching it how to contract, as the feeling of relaxation is small. Good body awareness and persistence is necessary; but even then some patients never gain the conscious ability to recognise relaxation.

Box 30.5

Down-Training Methods

1. Practice 20 to 30 minutes twice per day. Build relaxation into daily activities. Many patients need home SEMG units to perform relaxation effectively.

   Experiment with several different techniques
2. Diaphragmatic breathing: Contraction and relaxation of the respiratory diaphragm can encourage relaxation of the PFM
3. Visualisation: A relaxing place, the ischial tuberosities separating, heavy warmth, a hole getting larger
4. Perineal bulging: Have patient place a hand over the anal cleft with the middle finger in the cleft. Gently push out as if expelling gas. The patient should feel the tissues bulge into the finger. Remind patients to be gentle, not to bear down.
5. Quiet environment, soft music, relaxation audiotapes, low lights, comfortable position, warmth.
6. A picture of the muscle may help visually oriented patients to visualise the sagging of a hammock
7. Total body relaxation, progressive relaxation
8. Body scanning: patients learn to scan (or bring attention to) the PFM often during the day for stress and tension. When tension is recognized, the patient performs a brief relaxation exercise.
9. Advanced training: Progresses to more stressful environmental situations. Visualise a stressful event, keep the bright lights on in the treatment room, distract your patient during SEMG practice, relaxation in standing or sitting.
10. Dilator practice: "Practise" intercourse. External SEMG anal electrodes allow the patient to insert the vaginal dilator while monitoring the PFM. This gives the woman control over the situation; she can stop without worrying about her partner's reaction. Start by inserting the dilator slowly, then progress to moving the dilator, simulating intercourse. Visualising her partner during this exercise also helps in the transition to actual intercourse.

Not all vaginal pain is related to PFM hypertonus, and it is useful to perform an SEMG evaluation to establish the presence of PFM hypertonus or instability. The EMG equipment with a bandwidth of 25 or lower is needed to accurately measure microvolt output of the PFM. If high resting tone is not seen on initial measurement, it may be necessary to provoke the muscle pain with functional activities.

Surface EMG biofeedback has been successfully used in the treatment of vulvar vestibulitis syndrome. Glazer et al.[22] instruct the patient in twice daily exercises that improve the strength, stability, and resting tone of the PFM. Biofeedback with the aim of improving pelvic floor relaxation may also have potential benefit in the treatment of male pelvic pain.[23]

30.6 Summary

Chronic pelvic pain has a variety of causes and is always multifactorial. Physiotherapy is useful in a multidisciplinary treatment approach to improve the quality of life for these complex patients.

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V Pelvic Organ Prolapse

31
Background to Pelvic Organ Prolapse

FG Khan and LL Wall

31.1 Introduction

Pelvic floor (PF) dysfunction encompasses the problems of genital prolapse, urinary and faecal incontinence, voiding dysfunction, and discoordination–spasm of the muscles of the PF. These conditions may occur alone or in combination with one another. Unfortunately, the medical community itself does not fully understand the etiology of these conditions and how best to treat them. Many current therapies have high failure and recurrence rates.

The prevalence of PF disorders will mirror changing demographics. In the United States alone, the number of women between the ages of 65 and 84 will double by 2050 and those over 85 will quadruple the current number.[1] By one estimate, the need for PF- related health services will increase at twice the rate of the population.[2] Better understanding of aetiology and treatment of PF dysfunction, specifically pelvic organ prolapse (POP), is vital for all health care practitioners treating women.

31.2 Definitions

Genital prolapse or *POP* refers to a loss of fibromuscular support of the pelvic viscera resulting in a vaginal protrusion. The prolapse is usually described according to the area of the vagina in which it occurs. Assumptions are often made about which organ is behind the vaginal wall that is prolapsing; hence terms such as cystocele, rectocele, and enterocele. Studies have shown, however, that vaginal topography does not necessarily correlate with specific organs.[3] Instead, prolapse should be documented based on which compartments of the vaginal canal—anterior, posterior, and apical—are involved.

An *anterior vaginal prolapse* generally involves the bladder (*cystocele*), and often involves hypermobility of the urethrovesical junction as well (*cystourethrocele*). The small bowel also may displace the anterior vaginal wall.[4]

A *posterior vaginal prolapse* can involve the rectum *(rectocele)*, small bowel *(enterocele),* or the sigmoid colon *(sigmoidocele)*. The term *pseudorectocele* was introduced to describe an inadequate or defective perineum resulting in exposure of the midportion of the posterior vaginal wall. It mimics the appearance of a rectocele, but does not involve creation of a rectal pouch that incorporates both the rectal and vaginal walls with loss of vaginal rugation.[5]

Apical prolapse describes loss of the support at the apex of the vagina. The term *vaginal vault prolapse*, which is less descriptive, refers to a complete or partial inversion of the vaginal apex, usually found in patients who have had a hysterectomy. Most commonly, apical prolapse is due to a herniation of a peritoneal sac (usually filled with small bowel) through the vaginal apex. This is typically referred to as an *enterocele*. An enterocele may be classified further as a *traction enterocele* or a *pulsion enterocele.* A "traction" enterocele is a protrusion of the posterior cul-de-sac that is pulled down by the prolapsing cervix or vaginal cuff. The "pulsion" enterocele is a protrusion of the cul-de-sac through the vagina resulting from chronically increased intra-abdominal pressure. Pulsion

enteroceles are frequently large and always contain small bowel.

Procidentia refers to a complete protrusion of the uterus and vagina.

31.3 Prevalence

Estimates suggest that 50% of parous women have some degree of genital prolapse, but only 10 to 20% seek evaluation and treatment for their condition.[6] Most women with prolapse are parous; nulliparous women account for only 2% of prolapse cases in North America.[5] The incidence and prevalence of POP increases with age. American women have an 11.1% lifetime risk of undergoing surgery for POP and urinary incontinence, and 29% of women who undergo repair subsequently will undergo another operation.[7] Similar to changing demographics, the need for health care for PF disorders will increase. Currently, the direct cost of prolapse surgery is over $1 billion per year in the US.[8] Factors that predispose women to the development of prolapse include vaginal delivery, chronic increases in intra-abdominal pressure, obesity, cigarette smoking, advancing age, and estrogen deficiency.[9–12] There is little good epidemiological information about the incidence and prevalence of prolapse in different ethnic groups, but it is clear that women of all races may develop this condition.[5,13]

31.4 Risk Factors

The aetiology of POP may be multifactorial. While some risk factors predispose a woman to prolapse and are out of her control, such as family history or ageing, others are developed and controllable such as weight or parity. Individual risk factors will be discussed briefly below.

Risk factors that predispose to the development of POP include race, gender, and genetics. Caucasian women are more likely than African–American women to develop prolapse and Caucasian women undergo surgery for prolapse at a rate three times that of African–American women.[14]

A strong family history of genital prolapse also is a risk factor, and this is probably related to differences in collagen structure in different subpopulations.[10] Connective tissue disorders and corticosteroid use may also affect collagen strength. Further weakening of tissues may result from other chronic medical problems such as diabetes mellitus or chronic obstructive pulmonary disease.

Increasing age is also related to the development of prolapse, for a variety of reasons.[15] Older women tend to be of higher parity, have frequently undergone hysterectomy, and are often postmenopausal. Prolapse among older women is often more severe and more complex, involving multiple sites. Progressive denervation of the pelvic musculature that occurs with ageing may also contribute to prolapse. Recent studies using magnetic resonance show progressive changes in PF structures such as levator plate angle, levator hiatus height, and perineal descent in women with PF dysfunction such as prolapse as compared to normal women.[16] Women under the age of 35 more commonly have uterine prolapse only. It is possible that the increased complexity of prolapse among older women reflects delays in treatment and therefore may represent the natural progression of this pathology. Younger patients with prolapse may have medical conditions that predispose them to prolapse, such as congenital anomalies, neurological abnormalities, or rheumatologic diseases.[15]

Pelvic trauma and pelvic surgery may damage the neurovascular structures, connective tissue, and muscles of the PF. A vaginal delivery birth leads to stretching, dislocation, tearing, and avulsion of pelvic tissues.[15] Neurological injury to the pudendal nerve may also occur, as has been demonstrated in women with stress incontinence and POP.[17,18] There is some evidence that women who had an episiotomy or a perineal laceration at delivery were less likely to develop urinary stress incontinence or POP, perhaps by directing obstetric trauma in other directions. Other studies have shown, however, that episiotomies do not prevent incontinence or prolapse.[19,20] Chronic straining also may damage the pudendal nerve and lead to subsequent PF dysfunction by compromising neuromusular function.[21]

Posthysterectomy vaginal vault prolapse is a distressing and increasingly common problem. It may occur following vaginal or abdominal hysterectomy and often results from inattention to the proper reconstruction of the vaginal apex support following removal of the uterus.[22–25]

Patients with obvious congenital defects involving the PF, such as bladder exstrophy or spina bifida, are predisposed to the development of prolapse. However, so are women with subclinical, neurological defects such as spina bifida occulta, which may lead to subtle pelvic muscle dysfunction.[21,26,27]

31.5 Symptomatology

Shifting of vaginal anatomy is common in women, especially parous women. While some with advanced prolapse clearly describe discomfort with protrusion of tissue outside the body, most are either asymptomatic or describe a variety of nonspecific symptoms. POP can present with many symptoms, depending on the organs involved. The most frequent symptom of advanced prolapse is the complaint of a protrusion or "bulge" from the vagina, which worsens with prolonged standing or walking. In some cases the prolapse may be large enough to impair ambulation. Other common symptoms include low back pain, urinary incontinence, voiding difficulty, and difficulty emptying the rectum. Changes in the vaginal epithelium are frequently present in women with prolapse. In younger women the vaginal skin may be hypertrophic, while in older women it will be atrophic, particularly if they are not receiving estrogen replacement therapy.[28] Prolonged protrusion may lead to irritation or ulceration of the prolapse with the development of vaginal bleeding.

Approximately one third of women with POP report urinary tract symptoms, including voiding difficulty, urinary incontinence, and irritative symptoms such as frequency and urgency.[29] Some women report stress urinary incontinence due to urethral hypermobility and incompetence, while others report paradoxical improvement in symptoms as the prolapse progresses. Anatomically, the progression of anterior wall prolapse kinks the urethra and causes obstruction. As the prolapse progresses, some women will splint to void. Similarly, gastrointestinal symptoms may also occur. Patients may report impaired faecal evacuation, faecal incontinence, or a need to reduce or manually splint the prolapse or perineum in order to empty the rectum.[30–33] Women with perineal descent and damage to the rectovaginal septum often report defaecatory dysfunction, which in many cases can be debilitating. Damage to the pelvic muscles, connective tissue, and the pudendal nerve form a common aetiological mechanism for both POP and faecal incontinence.[34,35]

Sexual dysfunction may also be present in women with prolapse due to alterations in vaginal anatomy and pelvic organ function. The patient's age and the presence of relationship conflicts are the most important factors in predicting sexual functioning. Although most sexually active women with prolapse have sexual function that is comparable to women of similar age without prolapse, advanced forms of prolapse may interfere with coitus on a purely mechanical basis. Embarrassment, fear of worsening the prolapse, or fear of bladder and bowel incontinence may prevent women from being sexually active. Surgery that is successful in correcting the prolapse often results in improved sexual functioning in such women.[36,37]

31.6 Anatomy and Physiology

The PF is a supportive layer of fibrous connective tissue and muscle at the base of the abdominal cavity. It closes off the bony canal of the pelvis. The PF contains an aperture (the levator hiatus) which is pierced by the urethra, the vagina, and the rectum.[6] The bladder, uterus, vagina, and rectum are attached to the pelvic sidewalls by the endopelvic fascia[23] (see Chapter 2).

The endopelvic fascia is a fibrous connective tissue layer extending diffusely throughout the PF to form a continuous sheetlike mesentery. The ligaments and fascia of the pelvis are not dense connective tissue structures like the ligaments of the knee or thick sheets of tissue like the fascia lata that covers the lateral thigh. Rather, these structures are areolar folds of connective tissue and smooth muscle that contain the nerves and vessels of the pelvic viscera.

The vagina has three main levels of support[23] (see Figure 31.1):

- Level I support includes the vaginal apex and the paracervical vagina, which is suspended by the long connective tissue fibres of the superior paracolpium.

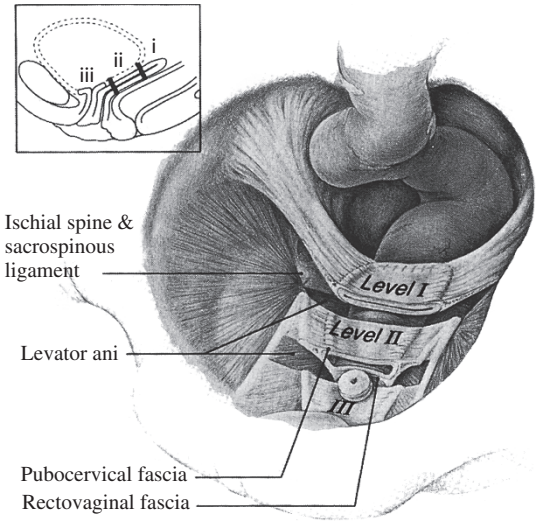

Figure 31.1. The three main levels of vaginal support. [From Delancey JOL Anatomic aspects of vaginal eversion. Am J Obstet Gynecol 166; 1992: 1719.]

- The midportion of the vagina (level II) is attached laterally, stretching between the bladder and the rectum and supported by the inferior portion of the paracolpium. At this level the anterior vaginal wall and the endopelvic fascia merge to form the pubocervical fascia, which underlies the bladder. Posteriorly, the endopelvic fascia merges with the posterior vaginal wall to form the rectovaginal fascia. This layer prevents the rectum from protruding through the posterior vaginal wall.
- The lowest portion of the vagina (level III) is found at the vaginal introitus and has no intervening paracolpium to suspend it. At this level the vagina fuses directly with the levator ani muscles laterally, the urethra anteriorly, and the perineum posteriorly.

Injury to the suspensory fibers at level I may result in vaginal and uterine prolapse and enterocoele formation. Damage to the pubocervical fascia or rectovaginal fascia (the supportive fibers of level II) leads to the development of anterior and posterior prolapse, respectively. Injury often occurs at both levels and results in a combination of defects. Another important component of the PF is the levator ani muscles, which may be subdivided into a pubococcygeal or "pubovisceral" portion and an iliococcygeal portion (see Chapter 2).

The iliococcygeus muscle forms the diaphragmatic portion of the levator ani.[38] It is a thin, horizontal sheet of muscle arising from a thick band of fibrous tissue along the pelvic sidewall called the "arcus tendineus levator ani." The iliococcygeus muscle inserts into a midline raphe behind the rectum and forms a shelflike diaphragm on which the pelvic viscera rest.[21] The pubovisceral or puborectalis muscle is a thick U-shaped muscle that arises from the pubic bones and attaches to the lateral walls of the vagina and rectum and then passes behind the rectum to create a supporting sling. When the pubovisceral muscle contracts, it pulls the rectum, vagina, and urethra/bladder neck forward toward the pubic symphysis, constricting their lumens. This is extremely important for the maintenance of both continence and normal pelvic support. The vaginal axis in the normal-standing female is almost horizontal, and the urethra, vagina, and uterus lie on top of the levator ani muscles, away from the urogenital hiatus.[39, 40]

31.7 Aetiology and Pathophysiology

Pelvic floor dysfunction and genital prolapse result from the interaction of many different aetiological factors. Bonney[41] suggested that prolapse originates from a process similar to that used by a surgical scrub nurse who must evert the invaginated finger of a surgical glove. By compressing the air within the glove, the invaginated finger is forced outward. In a similar way, increases in intra-abdominal pressure may force the uterus and vagina to prolapse if their supports are defective.[41]

DeLancey,[42] inspired by Bonney, suggests that the uterus and vagina are maintained in their normal position by three mechanical principles:

- First, the endopelvic fascia suspends the uterus and vagina through its attachments to the pelvic sidewalls.
- Second, the levator ani forms an occlusive and supportive layer on which the pelvic organs rest.
- Third, this anatomic structure creates a flap–valve effect, which is the third mechanical force at work in maintaining normal pelvic support.

Suspended by the endopelvic fascia, the uterus and vagina rest against the adjacent supporting wall. Increases in abdominal pressure force the pelvic organs against the wall, pinning them in place.

These principles can be visualized at work by thinking of the uterus and vagina as analogous to a "boat in dry dock" (Figure 31.2). The uterus and vagina (the boat) are anchored in place by the ligaments and endopelvic fascia, which serve as a mooring to keep them from drifting out of place, but the real support is provided by the musculature, analogous to the water under the boat. If the moorings are cut or stretched, the boat will be displaced. If the water level drops, the moorings are strained past their capacity to support the boat. Although gynecological surgeons tend to think of ligaments as the main factors important in pelvic organ support, the real bulk of the work is done by the muscles of the pelvic floor. Injury to these structures predisposes women to the development of pelvic organ prolapse.[43]

The levator ani muscles are critical in PF support. These muscles maintain a constant basal tone that maintains the uterus and vagina in place. Above the levator ani, the ligaments and fascia stabilise the organs in position. Constant adjustments in muscular activity prevent the stretching of the pelvic ligaments. Contraction of the puboviscerel muscle pulls the rectum toward the pubic bone, closing the urogenital hiatus and compressing

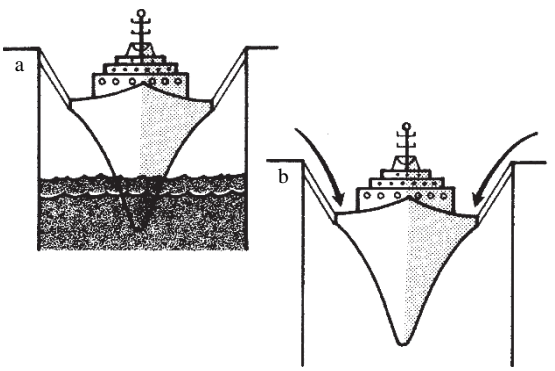

Figure 31.2. **a** The uterus and vagina (the boat) anchored in place by the pelvic ligaments and endopelvic fascia (the mooring), supported by the pelvic musculature (the water). **b** Defects in or weakening of the pelvic floor musculature may strian the ligments beyond thier capacity to support the uterus and vagina. [From Norton PA (1993) Pelvic Floor Disorders: The Role of Fascia and Ligamnets. JB Lippincott, Philadelphia, p 927.]

the urethra, vagina, and uterus. Rather than a static slab, the PF should be seen as a dynamic trampoline that constantly is expanding and contracting in response to changing stimuli.[44]

Connective tissue holds the body together and forms the tendons, fascia, and ligaments that allow muscles to exert traction through their points of attachment. Connective tissue is composed primarily of collagen and elastin.[43] Collagen is the most common protein in the human body, accounting for 30% of the total protein. Over 12 different types of collagen have been identified. Type I collagen is the most common. It forms large, strong fibres and is found in large amounts in ligaments, fascia, and tendons. Type II collagen is more prone to degradation by collagenase and is found in higher proportions in flexible tissues such as skin, blood vessels, the uterus, fascia, and ligaments. Many collagen fibres contain a mixture of collagen types, which produces variation in tissue strength and flexibility.[45]

Visceral connective tissue provides a capsule for the pelvic organs, which nonetheless allows for displacement and changes in volume. The visceral connective tissue contains important neurovascular structures, smooth muscle cells, elastin, and collagen. The connective tissue of the supportive ligaments of the uterus (the cardinal and uterosacral) is similar to that of vessel walls. Ligaments such as the arcus tendineus fascia pelvis or the iliopectineal (Cooper's) ligament are composed of stronger, more fibrinous collagen.

The collagen components of connective tissue undergo remodeling in response to stress and injury. Injured ligaments heal by scarring (collagen deposition), but never recover their full strength.[46] In pregnancy cervical "ripening" in preparation for labour causes the breakdown of the collagen and elastin and leads to loss of mechanical strength. There may be a similar breakdown in adjacent supportive tissues of the vagina and uterus.[43] Oestrogen promotes collagen maintenance.[47] Collagen abnormalities have been found to be more common among patients with PF dysfunction and genital prolapse than among women without these problems.[48, 49] This strongly suggests that intrinsic collagen abnormalities ("poor tissue") are involved as one factor in the development of disorders of pelvic support.

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32
Evaluation of Prolapse

FG Khan and LL Wall

The evaluation and classification of pelvic organ prolapse is a crucial component in the initial assessment of the patient. An accurate initial description of pelvic anatomy allows observers to assess the stability or progression of prolapse over time and also allows an accurate assessment of treatment outcomes.[1]

32.1 Physical Examination

The examination commences with inspection of the external genitalia for evidence of erosions, ulcerations, bleeding, or other lesions. If identified, such lesions are biopsied. Visual inspection may also reveal a widened genital hiatus commonly seen in prolapse patients. Visible soiling at the anus or loss of anal folds over the anal canal (dovetail sign) is indicative of anorectal pathology. A shortened perineum or alternatively abnormally small introitus heightens awareness of prior perineal trauma or surgery. A speculum, bimanual, and rectovaginal exam are then performed to assess the vagina, cervix, uterus, and adenexa. During the bimanual exam, additional information regarding pelvic muscle health and function can be assessed by asking the patient to tighten the pelvic floor (PF) around the examiner's hand. This simple test gives the practitioner information about the strength and the patient's awareness of her PF muscles.

The cough stress test may be performed to assess the presence of urinary incontinence. This is commonly done in the lithotomy and standing position. If the patient has significant anterior wall prolapse, the prolapse can be manually reduced (with caution taken not to overcorrect the anterior wall or obstruct the urethra) before instructing the patient to cough or use the Valsalva movement. If positive, the patient may have occult stress urinary incontinence, which further should be evaluated by urodynamics.

Accurate grading of prolapse depends on the patient's ability to strain vigorously during clinical examination. Prolapse tends to worsen as the day progresses, particularly if the patient stands upright for long periods. Patients should be examined in either the supine or Sims position, as well as in the standing position, in order to accurately assess the degree of prolapse that is present. Occasionally, a patient may be instructed to use the Valsalva movement on the toilet prior to the exam to try and reproduce the prolapse in its most severe form. Patients with prolonged pessary use may even require a second visit and examination after the pessary has been removed for a few days. An accurate assessment of the severity of the prolapse is essential in planning surgical management; thus every attempt should be made to reproduce the maximal degree of prolapse experienced by the patient.

Measurements for the following grading systems can be made using a Sims speculum with the patient in either the dorsal lithotomy or the left lateral decubitus position. Maximum descent with straining can also be assessed in standing; with genital hiatus, perineal body, and total vaginal length measured in lithotomy.

32.2 Grading

The International Continence Society (ICS) has developed a standardised system for describing the anatomic position of the pelvic organs to allow

reproducibility and comparison of physical examinations and establishment of the clinical significance of different grades of prolapse.[2] It utilises clear anatomic reference points defined in terms of vaginal wall segments rather than the organs lying behind the vagina. The hymenal ring provides an easily identifiable fixed landmark, even though the actual plane of the hymen may vary according to the degree of levator ani dysfunction. Six defined points in the anterior, superior, and posterior vagina are located in reference to the hymen and measured in centimetres (see Figure 32.1). A negative number denotes a point proximal to the hymenal ring. For example, "–2" indicates a point 2 cm above the hymenal ring. A positive number indicates a point protruding beyond the hymen. Measurements are made with a ruler or an appropriately marked measurement device such as a cotton-tipped swab.

- The first two points are located on the anterior vaginal wall. Point Aa is 3 cm proximal to the external urethral meatus in the midline. It corresponds approximately to the "urethrovesical crease," which is often obliterated in postmenopausal patients. Its measurement ranges between –3 and +3. Point Ba, the second anterior vaginal wall point, represents the most distal or dependent portion of the upper vaginal wall from the anterior vaginal fornix or the posthysterectomy vaginal cuff. In the absence of vaginal prolapse, it is approximately 3 cm proximal to point Aa.

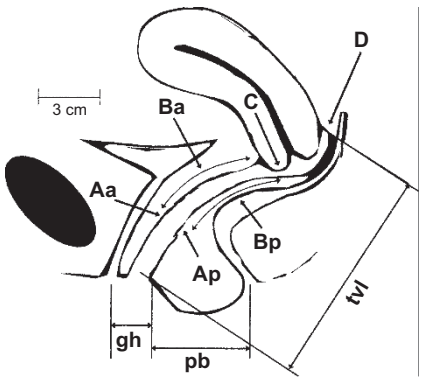

Figure 32.1. The six points (Aa, Ba, C, D, Ap, Bp), total vaginal length (tvl), genital hiatus (gh) and perineal body (pb) utilised in quantification of pelvic organ prolapse. From Bump RC, Mattiasson A, Bø K et al. The standardization of terminology of female pelvic organ prolapse and pelvic floor dysfunction. Am J Obstet Gynecol 1996;175: 12.

- Two additional points are identified on the superior vagina. Point C represents the most distal edge of the cervix or the vaginal cuff in the posthysterectomy patient. In a woman with a cervix, point D indicates the position of the posterior fornix or pouch of Douglas at the level of the uterosacral ligaments. Comparison between points C and D allows differentiation between cervical elongation and suspensory failure of the uterosacral–cardinal ligament complex (possible enterocoele). Elongation of point C in comparison with point D indicates cervical elongation.
- The points along the posterior vaginal wall are analogous to those of the anterior vaginal wall. Point Ap is in the midline 3 cm proximal to the hymenal ring. As with point Aa, it ranges from –3 cm to +3 cm relative to the hymen. The most distal or dependent portion of the upper posterior vaginal wall is point Bp. In the nonprolapsed vagina, point Bp is approximately 3 cm proximal to point Ap.
- Additional measurements, also in centimetres, include the genital hiatus, the perineal body, and the total vaginal length. The distance between the middle of the external urethral meatus to the posterior midline hymen is the genital hiatus. The firm, palpable tissue of the perineal body is used as a reference point if the location of the hymenal ring is unclear. The length of the perineal body is measured from the posterior midline hymenal ring (the posterior margin of the genital hiatus) to the midanal opening. The total vaginal length is the greatest depth of the vaginal canal in its normal position.

Although this system appears complicated upon initial examination, it is easy to use in practice. Understanding the system is greatly aided by an instructional videotape, available at nominal charge from the American Urogynecology Society (2025 M Street, NW, Suite 800, Washington, DC 20036–3309). Once the quantitative description of the prolapse has been completed, a stage may be assigned according to ICS criteria. Allowance is made for the distensibility of the vagina and the inherent imprecision in the measurement of vaginal length by allowing a 2-cm buffer. The ICS defines five stages of prolapse, as follows:

- Stage 0: No prolapse is demonstrated. Points Aa, Ba, Ap, and Bp are all at –3 cm. Point C or D

is between the total vaginal length or the total vaginal length minus 2 cm.
- Stage I: The most distal portion of the prolapse is >1 cm above the hymeneal plane.
- Stage II: The most distal portion of the prolapse is ≤1 cm proximal or distal to the hymen.
- Stage III: The most distal portion of the prolapse is >1 cm below the level of the hymen, protruding no greater than 2 cm less than the total vaginal length.
- Stage IV: Complete eversion of the total vaginal length, protruding at least ≥2 cm less than the total vaginal length (TVL-2). Generally, the leading edge of the prolapse will be the cervix or the scar left at the vaginal cuff by prior hysterectomy.

To measure the anterior vaginal wall and the superior vagina, the speculum is placed against the posterior wall of the vagina. Traction on the speculum exposes the anterior and superior vagina. Upon identification of a point of reference the patient is asked to bear down. The extent of the descent is measured with a ruler or other calibrated measuring device. Assessment of the posterior vagina and the posterior fornix is performed in similar fashion by retracting the anterior vaginal wall superiorly. Nine separate measurements (eight if posthysterectomy) are made of the six reference points, total vaginal length, genital hiatus, and perineal body. Once all measurements are recorded, a diagrammatic vaginal profile may be drawn (Figures 32.2).

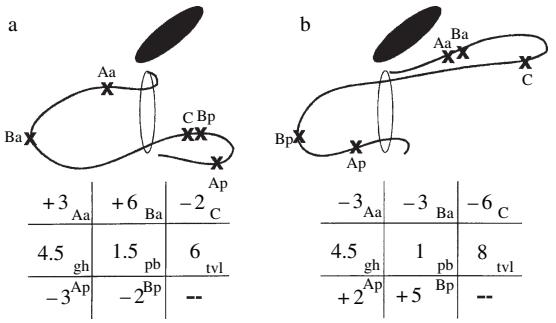

Figure 32.2. Predominant defect of anterior vaginal support denoted in grid and line diagrams. Stage III prolapse with the leading edge of the defect at point Ba. Predominant defect of posterior vaginal support, with leading edge of defect at point Bp. There is minimal descent of the vaginal cuff. (−8 to −6). This is also a stage III defect. [From Bump RC, Mattiasson A, Bo K et al. The standardization of terminology of female pelvic organ prolapse and pelvic floor dysfunction. Am J Obstet Gynecol 1996; 175: 12.]

The system developed by the ICS provides an objective site-specific means for description, quantification, and staging of pelvic organ prolapse. It has been demonstrated to be reliable and reproducible, but is not yet in universal use.[3] A more common, but less precise, classification system has been recommended by the American College of Obstetrics and Gynecology.[4] This examination is also performed with a Sims speculum as previously described, also without the use of traction, attempting to duplicate the patients' daily symptoms. The presence of prolapse is classified into four degrees of severity:

- First degree: On depression of the perineum, the prolapse extends to the midvagina.
- Second degree: The prolapse approaches the hymenal ring.
- Third degree: The prolapse is at the hymenal ring.
- Fourth degree: The prolapse extends beyond the hymenal ring.

The examination is divided into inspection for rectocele, cystocele, enterocoele, and uterine (cervix) or vaginal apex prolapse.

32.3 Testing

Voiding function in women with prolapse can easily be tested by measuring a voided volume and postvoid residual. Postvoid residuals are considered normal if less than 100 cc or less than 20% of the total voided volume (void + postvoid residual). Women with large postvoid residuals have evidence of voiding dysfunction which may be due to obstruction from a large prolapse. If the prolapse is not large enough to explain the elevated residual or reduction of the prolapse does not alleviate the urinary retention, other causes of urinary retention should be sought.

Further evaluation of urinary tract symptoms with multichannel subtracted cystometry is often indicated (see Chapter 9a). A history of fecal incontinence warrants further investigation by sigmoidoscopy, anorectal manometry and endoanal ultrasonography to assess the integrity of the sphincter (see Chapter 26).

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33
Treatment and Prevention of Prolapse

FG Khan and LL Wall

33.1 Therapeutic Options for the Patient with Prolapse

Unless the patient is symptomatic, prolapse may not require treatment. An exception to this is if the patient has developed urinary retention as a sequela of their prolapse. Symptomatic prolapse may be managed conservatively or surgically. Nonsurgical therapies that may benefit these patients include oestrogen replacement therapy, pelvic muscle exercises ("Kegels") with or without biofeedback, and support of the prolapse with a pessary. Surgical options vary widely from newer "minimally invasive" vaginal procedures to major abdominal reconstructive procedures. Details of surgical procedures are beyond the scope of this chapter.

Many women with pelvic organ prolapse are perimenopausal or postmenopausal; therefore, evaluation of the patient's oestrogen status is essential. Hypoestrogenism results in thin, atrophic vaginal and perineal skin that is easily traumatized. Patients often note vaginal dryness and decreased mucosal rugation is notable on examination. In addition to changes in the epithelium, low oestrogen levels also lead to a diminished blood supply to pelvic organs, and this may also compromise pelvic support. Oestrogen replacement therapy will limit these changes and may improve patient tolerance of conservative therapy (such as the use of a pessary). It will also improve the quality of tissues with which the surgeon has to work if surgical treatment is undertaken.[1] The use of hormone replacement therapy, however, has not been found to reverse the presence or severity of pelvic organ prolapse (POP).

Pelvic floor (PF) muscle exercises may help prevent the development of prolapse in normal women as well as delay its progression in women with early symptoms.[2] Pelvic floor reeducation is discussed in Chapters 10–14.

33.2 Pessaries

Patients with mild symptoms associated with an early-stage prolapse will often get temporary relief by using a tampon or diaphragm in specific situations that are associated with their symptoms, such as exercising or standing for long periods of time. With more severe degrees of prolapse and more significant symptomatology, patients may obtain relief by using a pessary. A pessary is a prosthetic device that is inserted into the vagina to support the prolapse. Most pessaries are now made of soft, flexible silicone that allows them to be inserted and removed easily, without irritation. Some pessaries permit drainage, minimising the development of vaginal discharge. Pessaries are useful devices for patients who are poor candidates for surgery or for those who do not wish to undergo an operation for their condition. Pessaries also offer temporary relief of symptoms in cases where definitive surgical correction of the prolapse must be delayed.

Pessaries may also be used as a diagnostic tool for the evaluation of pelvic floor dysfunction. Urinary incontinence, defaecatory dysfunction, and pelvic pain that are attributed to the POP can be reassessed with the pessary in place. Women with POP may have occult stress incontinence. These patients will often develop a "kinking" of the urethra and bladder neck as

the prolapse descends to or beyond the hymen. Women note a prior history of urinary incontinence, which actually improved as the prolapse worsened. During the pessary trial, reduction of the prolapse reveals previously undetected ("occult") stress incontinence. This finding should prompt further investigation of the patient's continence status using complex urodynamics. Alternatively, care must be taken not to occlude the urethra and mask stress incontinence.

In those with defaecatory dysfunction or pelvic pain, the use of the pessary can help determine whether these multifactorial symptoms are a result of POP. Such knowledge is helpful when counseling women seeking operative correction of prolapse for these specific symptoms.

A wide variety of pessaries are available. Identifying the best type for a particular patient is a process of trial and error. Ring pessaries, with or without platform support, and doughnut pessaries are readily accepted by patients due to their similarity to contraceptive diaphragms (see Figures 33.1 and 33.2). These pessaries are most useful in the treatment of uterine prolapse, particularly stage I or II prolapse.[3] The platform type of pessary avoids the rare complication of a uterus being incarcerated by protruding through an opening in a pessary. Platform pessaries are folded in half and inserted into the vagina, where it then springs back into its previous shape. When in position, these pessaries rest below the cervix and above the pubic symphysis in a position similar to that occupied by a contraceptive diaphragm. Sexual intercourse is not recommended while these pessaries are in place. Sexually active women are asked to remove the pessary prior to intercourse.

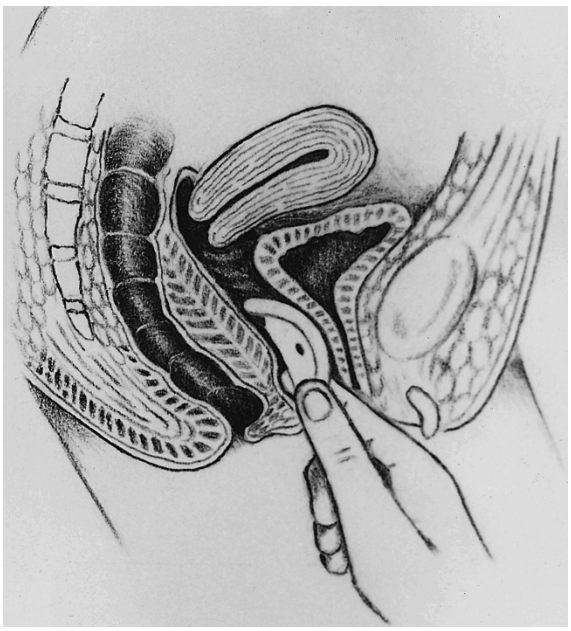

Figure 33.1. Insertion of ring with support.

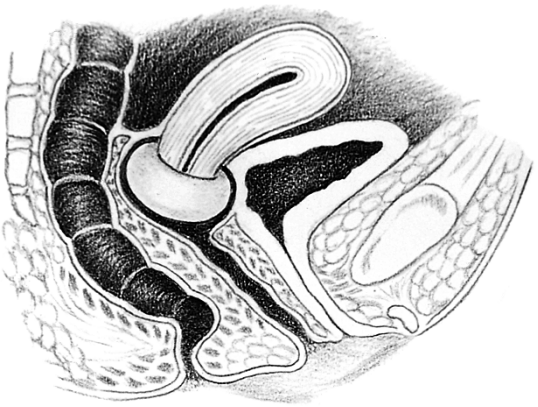

Figure 33.2. Donut pessary in position. Courtesy of Milex Products, Inc., Chicago, IL.

The *Gellhorn pessary* provides more support for patients with severe degrees of uterine or vaginal prolapse. Gellhorn pessaries are especially useful in cases where there is a large prolapse of the anterior vaginal wall. They hold their position better if the patient has an intact perineal body.[4] The flared base of the pessary is inserted along the base of the vagina and is tipped into position, maintaining the prolapse in position by obstructing the vaginal outlet (Figure 33.3). A Gellhorn pessary should be removed prior to attempting intercourse.

Inflatable pessaries are doughnut-shaped devices with an attached stem. The stem protrudes through the vagina, where it is accessible for inflating and deflating the device (Figure 33.4). Use of this device requires manual dexterity, so it

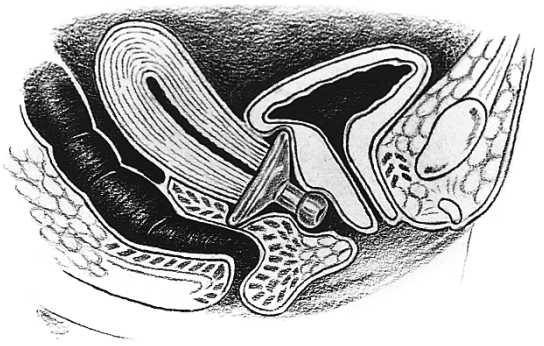

Figure 33.3. Gellhorn pessary in position. Courtesy of Milex Products, Inc., Chicago, IL.

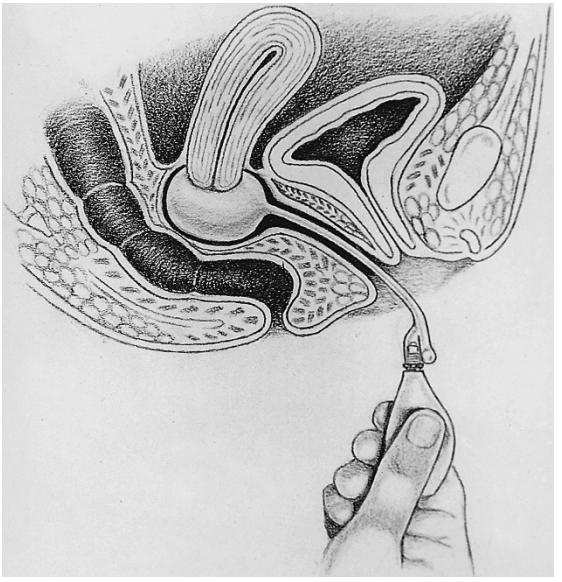

Figure 33.4. Inflatable pessary being inflated. Courtesy of Milex Products, Inc., Chicago, IL.

is not suitable for all women. Protrusion of the stem beyond the vaginal introitus may create discomforting vulvar irritation.

Cube pessaries are constructed with six concave surfaces that create a suction effect with the vaginal walls and hold the pessary in place (Figure 33.5). Pessaries of this type are particularly useful in patients with vaginal eversion or complete uterine prolapse. Unless the suction is broken periodically, however, ulceration of the vagina may occur. Ideally these pessaries should be

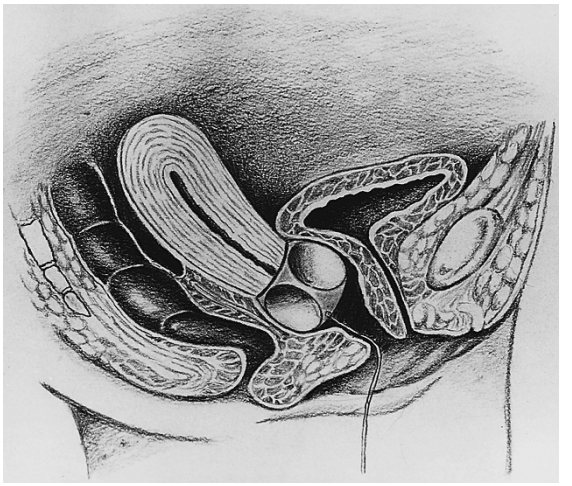

Figure 33.5. Cube pessary in position. Courtesy of Milex Products, Inc., Chicago, IL.

removed, cleaned, and reinserted on a daily basis. Unless patients are able to do this themselves, they require careful monitoring and frequent pessary checks by a knowledgeable clinician.

Smith–Hodge pessaries elevate the bladder neck into a retropubic position and provide support similar to that obtained by bladder neck suspension surgery (Figure 33.6). This type of pessary may be useful in helping predict the likelihood of incontinence after surgery for the correction of prolapse, as well as in helping predict whether reconstructive surgery will help restore normal urinary continence.[5]

Proper fitting of the pessary will minimise the development of complications. A pessary should not interfere with normal voiding. Three or four

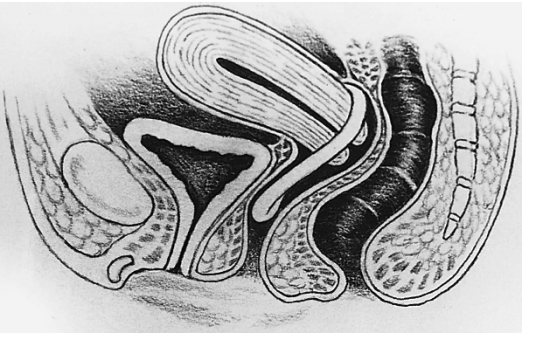

Figure 33.6. Hodge pessary in position. Courtesy of Milex Products, Inc., Chicago, IL.

sizes of a variety of types of pessary should be available during a pessary-fitting session. If the pessary is too large, it may create a sensation of pelvic fullness and possibly pelvic pain. Pessaries that are fitted improperly may cause urinary retention and vaginal irritation, ulceration, or bleeding. Neglected or forgotten pessaries, most commonly seen with elderly women, can become entrapped or embedded in the vagina or erode through the vagina. This can create vesicovaginal or rectovaginal fistulas, or form a focus of chronic irritation that contributes to the development of a vaginal or cervical cancer. Neglected pessaries also commonly lead to an abnormal or excessive vaginal discharge. If the pessary is too small, it may simply fall out or be expelled by the patient with a cough or during defaecation or urination.

When a pessary is placed for the first time, the patient should be encouraged to wear it around the office or clinic for a brief period of time to make sure that it is comfortable and provides adequate support. If this is satisfactory, the patient may go home with the pessary in place, but should return in a few days to have it checked and to review instructions for its insertion, removal, and care. Under ideal conditions, a patient with good manual dexterity should be encouraged to remove her pessary twice a week, wash it with soap and water, and reinsert it using a water-soluble lubricant. Some pessaries, such as Gellhorn or doughnut pessaries, can be left in place safely for 3 months or longer. Local oestrogen administration either in the form of half an applicator of vaginal oestrogen cream or an oestrogen-impregnated vaginal ring is extremely useful in maintaining optimal condition of the vaginal epithelium. Patients with pessaries should have a pelvic examination after 3 months and at periodic intervals thereafter.[6] Patients who have poor manual dexterity should have the pessary removed, washed, and reinserted during a clinic visit every 3 to 4 months, depending on the type of pessary and the patient's clinical history. Pessary use should be discontinued if erosion or ulceration of the vagina occurs. Patients who develop a heavy vaginal discharge while using a pessary may find periodic vaginal douching with a dilute solution of vinegar and water helpful.

33.3 Prevention

Prevention of prolapse is difficult to do because the aetiology is not completely understood and many different factors seem to influence its development.

Pregnancy and delivery are known risk factors for the development of prolapse. The chronic strain on the nerves of the pelvis from the pregnancy as well as from the delivery can compromise pelvic organ support. Although some advocate the use of elective caesarean section for the prevention of prolapse due to delivery, this has not been definitely shown to reduce incidence. Obstetrical trauma can be limited by reducing the use of operative delivery, episiotomy, and shortening the second stage of labour.

Chronically increased intra-abdominal pressures can also contribute to muscle and nerve deterioration. Obesity, smoking (which leads to chronic cough and poor tissue integrity), chronic straining with constipation, and heavy lifting can all stress the pelvic musculature and nerve function leading to prolapse. Minimising these risk factors may help in prevention.

Pelvic floor muscle exercises have also proven to be useful in maintaining and even restoring the integrity of the musculature. Most commonly used in mild to moderate anatomic defects, exercise can prolong the symptom-free period. Pelvic floor exercises can be used in the antenatal and postpartum period.

Attention should also be given to the potential for prolapse inherent to certain surgical procedures.

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VI Professional Issues

34
Ethical Principles in Pelvic Floor Physiotherapy

LL Wall

34.1 Introduction

Many writers on ethics make the subject seem esoteric and confusing, when in reality it is quite straightforward. At its most fundamental level, ethics is simply an attempt to answer the question "How should we live?"[1,2] Within the health care setting, medical ethics ("bioethics") is the attempt to answer the more limited question of how doctors, nurses, physiotherapists, and other providers of health-related services should interact with patients and with one another. The question "How should we live?" becomes "How should we act with respect to those who need health care and with respect to those who help us provide it?"

Although there are many different approaches to moral philosophy in general,[3] a broad (but not quite universal) consensus has arisen among writers on bioethics that the provision of health care services should be governed by four fundamental ethical principles. These principles are nonmaleficence, beneficence, autonomy, and justice.[4] The first step in resolving clinical ethical dilemmas is to understand what questions may arise with respect to each of these principles in any particular case.

34.2 Ethical Principles

34.2.1 Nonmaleficence

Nonmaleficence is probably the oldest principle in medical ethics, traceable in the Western medical tradition to the ancient aphorism of Hippocrates, *primum non nocere*; that is, "First of all, do no harm." It means that in providing care, one's first obligation is to avoid inflicting foreseeable, preventable harm. This principle is one of the moral foundations on which professional licensure rests, since it is through the mechanism of licensure that societies hope to ensure that doctors, surgeons, physiotherapists, pharmacists, nurses, and other providers of health care services are competent in what they do. While the duty of nonmaleficence appears most obvious in cases where patients are undergoing surgical operations (perhaps for urinary stress incontinence, pelvic organ prolapse, or associated conditions), the obligation to avoid harm also includes the duty to avoid causing mental, emotional, social, or financial harm to the patient where this can be avoided. One of the most important corollary obligations arising from this principle is the expectation of patients that their privacy and confidentiality will be respected with regard to their medical problems and the treatments they receive.

34.2.2 Beneficence

Beneficence refers to the obligation to act always in the patient's best interests, to do good for the patient, and to promote her welfare. In this, beneficence clearly is inextricably intertwined with the principle of nonmaleficence and the licensure requirement to be technically competent. Corollaries of the principle of beneficence include not only the duty to promote good, but also the obligations to prevent harm when it may occur and to remove harm where it presently exists. Physicians, surgeons, physiotherapists, and nurses are usually trained to believe that in

doing their jobs and carrying out the day-to-day tasks that this involves, they are working for the patient's good, but this is not possible unless "what is in the patient's best interests" is clear to everyone involved. Unfortunately, the goals of treatment are not always obvious to all parties, and the resolution of ethical dilemmas often hinges on an appropriate understanding of what the phrase "the patient's best interests" means in any given case and how that determination has been made. For example, in cases in which a programme of pelvic floor physiotherapy has been ordered by the treating doctor, it is crucial for the doctor, the patient, and the physiotherapist to understand what the intended goals of treatment are, whether or not these goals are realistic, and what will be required in order to achieve them. This requires that all parties communicate closely with one another as treatment progresses.

The principle of beneficence requires doctors and other health care providers to act as fiduciaries or trustees on behalf of the patient.[5] Patients place themselves under care as a matter of trust, rightly expecting that doctors, nurses, and physiotherapists, by virtue of their special training, enhanced knowledge, and clinical experience, will place patient welfare foremost in their professional concerns. Ethical problems arise if the welfare of the patient no longer comes first but is subordinated to other concerns, such as recruiting her to participate in a research project, generating income by performing unwarranted or unnecessary procedures, or by abusing her trust in order to profit personally from financial incentives offered by pharmaceutical companies or device manufacturers that bear no direct relationship to the patient's welfare.[6]

34.2.3 Autonomy

Autonomy refers to the obligation to respect the individual's right of self-determination and bodily integrity, to respect patients as persons entitled to make their own judgments about what is in their best interests, and to develop a plan of care for them that takes this into consideration. The word "autonomy" is derived from the Greek words *autos* ("self") and *nomos* ("rule"). In health care, the exercise of autonomy implies that decisions are made by the patient free from control or undue influence by others. It also implies that the patient has the inherent capacity to make the kind of carefully reasoned decisions that are often necessary in deciding on whether or not to undertake, continue, or abandon particular forms of therapy.

The principle of respect for autonomy is the foundation for one of the most important concepts in modern bioethics: the necessity for obtaining informed consent before treating patients or carrying out research involving patients.[7,8] The requirement to obtain informed consent is not just a bureaucratic hurdle to be overcome by getting the patient to sign some kind of "permission slip"; rather, it refers to the moral requirement to make sure that the patient understands the reasons for the proposed therapy, understands the risks that may be involved as well as alternative means of treating the problem, has had the opportunity to make an informed decision, and voluntarily consents to undergo treatment. At its most fundamental level, informed consent is the bedrock on which all truly therapeutic health care relationships are built.

34.2.4 Justice

Justice, as philosopher John Rawls has pointed out, basically is a matter of fairness.[9] Everyone wishes to be treated fairly. The principle of justice implies that health care should be delivered to patients fairly. The concept of fairness or justice in health care delivery can usefully be broken down further into two subcategories: *distributive justice* and *procedural justice*.

The concept of distributive justice implies that health care services should be spread throughout the population in a just and equitable manner. Patients should be able to gain access to needed care. Distributive justice obviously is a central issue in the planning, administration, and funding of health care systems at the national, state, and local levels. Most industrialized countries have national systems of health insurance, designed to provide their citizens with access to needed health care services (within the constraints imposed by the economic and political conditions of each country). Some countries (notably the United States) have systems that provide incomplete, inadequate,

and piecemeal coverage for their citizens. The lack of access to basic medical services by a large and growing segment of the American population represents a major moral, economic, and political problem that will soon require a solution. But lack of access to specific kinds of needed services also plagues many other health care systems as well. If, as I believe, access to competent physiotherapy is a fundamental prerequisite for managing complex pelvic health problems (problems that disproportionately affect women in all countries), then there are very pressing problems of distributive justice even in those countries that have national health insurance systems.

Similarly, there are potential problems with procedural justice in all health care systems. Procedural justice refers to the requirement that people with similar conditions be treated similarly and that they are treated justly within the operation of the system. Favoritism or arbitrary exclusion should not be tolerated. People should not be able to "jump to the head of the queue" unless there is a pressing medical reason for this to occur. Issues of procedural fairness may exist in all health care systems and they are particularly troublesome in cases in which the suspicion exists that unfairness is present due to racial or cultural discrimination or if it disproportionately affects the poor or the powerless (notably women and children).

Table 34.1. Principles of medical ethics in clinical practice. Modified from Jonsen, Siegler, and Winslade.[10]

Medical indications	Patient preferences
• The principles of beneficence and nonmaleficence 1. What is the patient's medical problem? What is its history, diagnosis, and prognosis? 2. Is the problem acute, chronic, critical, an emergency? Is it reversible? 3. What are the goals of treatment? 4. What are the probabilities of success? 5. What plans are there if treatment fails? 6. In summary, how can this patient be benefited by medical and nursing care and physiotherapy and how can harm be avoided?	• The principle of respect for autonomy 1. Is the patient mentally capable and legally competent? Is there any evidence of incapacity? 2. Is the patient competent; what has the patient stated about her preferences for treatment? 3. Has the patient been informed of the risks and benefits of the proposed treatment? Has she understood this information and given her consent? 4. If she is incapacitated, who is the appropriate surrogate for decision-making? If a surrogate is used, is the surrogate using appropriate criteria for making decisions? 5. Has the patient previously expressed preferences about treatment (such as in the form of a written "advance directive" concerning health care?). 6. Is the patient unwilling or uncooperative with medical treatment? If so, why? 7. In summary, is the patient's right to choose being respected as far as possible?
Quality of life	**Contextual features**
• The principles of beneficence, nonmaleficence, and respect for autonomy 1. What are the prospects for a return to normal life? 2. What physical, mental, and social deficits is the patient likely to have even if treatment succeeds? 3. Are there biases that might prejudice the evaluation of the patient's quality of life? 4. Is the patient's present or future condition such that her continued life might be judged undesirable? 5. Is there any plan or rationale to forgo treatment? 6. Are there plans for comfort care and palliative treatment?	• The principle of justice 1. Are there family issues that might influence treatment decisions? 2. Are their issues with doctors, nurses, or physiotherapists that might influence treatment decisions? 3. Are there financial and economic factors to consider? 4. Are there religious or cultural factors? 5. Are there limits on confidentiality? 6. Are there problems with the appropriate allocation of resources? 7. How does the law affect the treatment decision? 8. Is clinical teaching or research involved? 9. Is there any conflict of interest on the part of the healthcare team or the institution involved?

34.3 Resolving Ethical Problems in Clinical Practice

Principles are all well and good, but how are they put into practice in everyday clinical situations? A huge literature and several specific journals on clinical ethics have attempted to address this issue. It is impossible in this short space to do more than offer some general guidance. One useful approach has been advocated by Jonson, Siegler, and Windslade[10] in their book *Clinical Ethics: A Practical Approach to Ethical Decisions in Clinical Medicine*. These authors suggest that the most practical way to begin solving any given ethical problem in clinical practice is by breaking down the relevant aspects of the case into four categories, which they define as: (1) the medical indications; (2) patient preferences; (3) quality of life issues; and (4) the social, legal, economic, and administrative context in which the case occurs. By analyzing the salient features of each case with respect to the four fundamental principles of ethics described here, the clinician can work toward a determination of what the most proper course of action is under any set of circumstances (See Table 34.1).

As the ancient Roman philosopher Seneca once said, "The greater part of goodness is the *will* to become good." By this he meant that the first step in acting ethically was the conscious determination that this is how one should act. Thoughtful consideration of the importance of these four principles of clinical ethics when dealing with patient problems will go a long way toward improving the quality of care that women with pelvic floor disorders receive in clinical practice.

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35
Research Trials in Incontinence

RP Assassa and F Mensah

35.1 Introduction

Current health care provided in most specialities has evolved by trial and error over many years. This is true for incontinence care as much as any other speciality. Observations of improvement in health after various interventions have been noted and incorporated into practice. However, many of these interventions are not able to stand up to close scrutiny in terms of their effectiveness in providing health benefits to patients. There is a movement toward providing a high standard of care based on scientific evidence. Where new treatments are being introduced, these should undergo scientific investigation before being incorporated into widespread practice.

35.2 The Randomised Controlled Trial

The most powerful methodology for investigating the effectiveness of treatments is the randomised controlled trial (RCT). This is a true prospective experiment in which investigators randomly assign an eligible sample of patients to one or more treatment groups and follow patients' outcomes. The fundamental feature of an RCT is that the response of the patients receiving the intervention under investigation is compared to the response of a control group receiving a standard intervention or placebo. Confounding variables that are likely to influence the results of the trial such as age, sex, marital status, or social class will be distributed randomly between the groups. However, other less obvious and less easily definable variables (for example, patients' willingness to comply with treatment or their anxiety about their condition), which might influence treatment response, will also be distributed randomly.

Uncontrolled trials are not advisable in incontinence as the condition is prone to spontaneous variation over time. It has been shown that incontinent people may spontaneously remit at a rate of about 10% per year.[1,2] Thus an uncontrolled trial investigating the use of a new drug for incontinence over 6 months might show an improvement rate of 5–10 %, which is unrelated to any action of the drug itself.

The starting point of a good RCT is a clear hypothesis stating the study population, the condition to be treated, the intervention under investigation, and the outcome or response to treatment to be measured.

35.2.1 Study Population

It is important to use a suitable case definition when selecting patients to take part in a trial. For example, if we were interested in a drug treatment for patients with detrusor overactivity, we would need to make a diagnosis based on accepted guidelines such as those put forward by the International Continence Society[3] (all cases therefore would need to be recruited following urodynamics). However, if we were interested in a primary care continence service, we would want to include all patients within the community with significant urinary leakage. This may be defined by reported symptoms, for example, leakage several times per month[4] or clinical assessment, for example, a urine pad loss of more than 10 gm.[5]

The definition of a case should include patients in whom the intervention is likely to produce

benefit and preferably should include all groups who are likely to receive the intervention in the future. It is advisable to exclude as few patients from a trial as possible. However, some trials have excluded elderly and frail patients. Within these groups there is a high prevalence of incontinence and a great need for effective treatment. Extrapolation of the results of trials that have excluded such groups may be misleading if applied to them. In general, if a group is likely to receive treatment in the future on the basis of the trial results, they should be included as cases within the trial. The source of cases needs to be considered carefully. Patients are probably best recruited from the place where the treatment under investigation is likely to be offered in the future. This will depend on the condition and type of intervention being studied. The prevalence of the condition to be treated should be known. If a relatively rare condition is to be investigated there is likely to be a need for multicentre collaboration to recruit enough patients.

35.2.2 Size of Trial

The sample size must be large enough to be able to give reliable estimates of the response to treatment of each of the treatment groups, and thus powerful enough to detect a difference between the treatments, if there is one. A sample size calculation should be carried out during the planning phase of the trial to ensure that it is feasible to recruit an adequately sized sample. This statistical calculation will be based on the expected response to the standard or control treatment, the minimum difference between the treatments you wish to be able to detect, and the ability of the trial (power) to detect this difference.

35.2.3 Randomisation and Blinding

To randomise, a list of allocations is drawn up before recruitment starts, using a simple method such as random tables or a computerised statistical package. When a patient consents to enter the trial, allocation to a treatment can then be achieved by referring to this list, using whichever method is most convenient. Such methods include opening a sealed envelope containing the treatment to be allocated or a hospital pharmacy dispensing pre-randomised numbered packages of drug treatments. If feasible, the trial should be double blind; this is achieved by ensuring that both the patient and anyone involved in assessment or treatment are unaware of which treatment group the patient has been allocated to. The purpose of this is to remove any influences, which may be present due to knowledge about which treatment a patient is receiving, for example, better morale in the patient group receiving a new treatment or clinicians' judgment of which treatment is likely to have the better outcome.

35.2.4 The Intervention and Control

Wide ranges of interventions are available for continence care. These may be very specific such as drug, surgical therapies, and physical therapies, or may be more general such as service packages or health promotion. All of these interventions may be evaluated through carefully designed RCTs. A literature search should be performed to describe this and the key elements of the intervention. The intervention should be formally described and follow predefined protocols. It is important that the intervention follows accepted best practice, for example, using the right dose of drug or correct pelvic floor exercise regimen, so that it is not open to criticism later. An accurate description will help others repeat the trial or transfer it into general clinical practice. The length of the intervention should be long enough for any treatment effects to be apparent but an overlong intervention period may result in excessive withdrawals from the trial.

Selection of the control is important. In comparative studies an accepted treatment may be compared to a new treatment. The result of such a study will show if the new treatment is better, worse, or comparable to accepted treatment. To truly measure efficacy, a placebo should be used; however, the use of placebo treatments is restricted by the Helsinki Declaration. A placebo-controlled trial should be conducted only generally in the absence of existing proven therapy. Most previous studies in incontinence have found a large degree of improvement in people receiving placebo.[6] As far as possible, apart from the experimental intervention, the treatment and care the groups receive should be identical.

It is feasible to make most drug trials double blind. For many others, such as alternative forms of physiotherapy for stress incontinence, it is not always possible to blind the patient or treatment provider.

35.2.5 Assessment

It is important to perform a thorough assessment of each patient at the beginning of the trial. This will measure the baseline condition of each patient and repeating the assessment at the end of the study will show any change.

Questioning the patient using a structured questionnaire is usually easily performed. However, it is important that the questions have been validated. Such questionnaires are available.[7,8] Incontinence is a socially disabling and hygienic problem to patients and so the outcome of an intervention should be measured from a patients' perspective. The questions should ask about the severity of the condition but should also explore the effect of the condition on the patient's quality of life in terms of psychosocial morbidity.[9] As far as possible, an independent observer who is unaware of which intervention the patient has received should perform questionnaires used as outcome measures, for example, surgeons should not question patients about the outcome of their own surgery.

Many clinicians believe that objective assessment of bladder function is the best form of outcome assessment. The use of a well-structured, easily completed urinary diary can be achieved in the majority of patients, provided detailed verbal and written instructions are given (see Chapter 7). Another objective measurement often used in incontinence is the pad test (see Chapter 9a). Preweighed pads can be worn by a patient and then reweighed to calculate the amount of urine lost. The pad can be worn while a standard set of exercises are performed, such as during the ICS 1 hour pad test,[3] or can be worn and collected over a period of time in the patient's normal environment. The former allows comparison between patients under standard conditions. The latter allows the severity of a patient's condition to be measured in their normal environment.

Objective measurements used as outcome measures should be performed under as similar conditions as possible and so should be performed on the same day of the week, in the same place, and so forth. Repeat urodynamic investigations have been used in some studies. Other outcomes that may be important are compliance and side effects of the interventions used, as well as how satisfied each patient was with the intervention. These will help to show how easily the intervention might be used outside of the study. In modern health care it also is important to perform some economic evaluation within most studies.

35.2.6 Ethical Considerations

All trials in incontinence in the UK are subject to research ethics committee approval. Most journals will not accept papers about experimental studies for publication without proof of approval being granted. Research ethics committees are bound by the principles of the Helsinki Declaration, of which the fundamental principal is that the researcher must protect the life, health, privacy, and dignity of the human subject at all times (see also Chapter 34).

Before commencing a study a complete protocol should be assembled and externally reviewed. This must state the research question and how it will be answered, together with a literature review that outlines what is already known. Informed consent from each subject is paramount and patient information material should be produced that states the study aims, the nature of the research, and the voluntary nature of participation. This should be in language easily understood by the layperson. All protocols, information material, and consent forms must be submitted to the relevant medical research ethical committee for approval before commencing recruitment. Within the UK the central office for research ethics committees (COREC) supplies standardised application forms and advice on informed consent, which can be completed and submitted online.

35.2.7 Analysis

The aim of analysis of an RCT is to compare the response of each of the treatment groups, to try to estimate whether there are differences between the treatments, and the size of any difference. Various analysis techniques are available to do this, ranging from a simple *t*-test to more

complicated modelling and survival analysis.[10,11] The involvement of a medical statistician early in planning the trial is to be encouraged as the type of analytical techniques that can be used will depend on the trial design, the hypothesis to be tested, and the outcome measures used.

35.2.8 Systematic Reviews

It is important to remember that most studies will address a specific question in a very controlled manner often with limited numbers of subjects. To get a broader, more generalisable and accurate picture, it is often better to combine studies. Great care is needed in performing the initial literature search to identify all studies addressing the specific question. Not all studies will be published (often negative trials are less likely to be published) and there is a risk of publication bias. A well-conducted systematic review will try to identify even unpublished trials by searching other areas such as conference abstracts. Sometimes translations from other languages will be needed. Many studies will not use similar outcome measures, making combination of their results difficult. Contact with the original researchers may be needed for access to the original data. Statistical analysis will usually take into account both the size and quality of the trials. The conclusions may be clear enough to prevent the need for further large, randomised, controlled trials needing to be conducted.

35.3 Conclusion

Properly conducted clinical trials require a large amount of planning and are often difficult to perform. It is the results of such trials, however, that will have the greatest ability to determine future organisation and delivery of health care provision.

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36
Infection Control Issues

L Barkess-Jones

36.1 Introduction

The following chapter refers to the UK; however, the principles should be transferable to other countries.

Following the development of the Clinical Governance Framework within the NHS[1] and the publication of "Winning Ways,"[2] Infection control is now at the heart of the government's strategy for health care improvement.[3,4] The release of "Saving Lives"[5,6] requires trusts to reduce health care-associated infections (HCAIs), including methicillin-resistant *Staphylococcus aureus* (MRSA) bacteraemias by implementing infection control practices and procedures in order to provide a clean environment and safe and auditable practice.

The Controls Assurance Standard[7] has largely been subsumed within the Health care Commission's core standards.[8] Separate core standards exist for primary care trusts and acute NHS trusts, each covering seven domains of activity, including safety, care of the environment and governance. Each NHS trust is expected to have defined written standards in order to prevent HCAIs. The Health care Commission for 2006 has implemented the first annual health check, which aims to verify individual NHS trust performance against the seven domains of activity; these results will be made public. Other aspects that are central to the annual health care checks are "Saving Lives," the "Clean Your Hands Campaign,"[9] decontamination of equipment, and working toward cleaner hospitals and lower rates of infection."[10]

All professional staff are now required to be conversant with their equipment and be fully competent, knowledgeable, and experienced to implement the associated operating processes incorporating infection control, and that these processes should also be research-based, auditable, and develop a framework/culture within which HCAIs are reduced. The "Clean Your Hands Campaign"[9] is designed to ensure that staff have a sound understanding that hands should be decontaminated near to the point of delivery of patient care. The campaign endorses the use of alcohol handrubs on clinically clean hands.

Toward cleaner hospitals[10] builds on existing guidance[11–13] and emphasises that patients have a right to expect a clean environment that provides the right setting for good patient care practice incorporating infection control.

36.2 Background

The following background information should be considered when formulating departmental infection control policies.

36.2.1 The Environment

The environment must be free from dust to ensure that microorganisms are kept to a minimum.[5,6] The majority of dust is made up from skin scales and within it some microorganisms can survive for long periods of time.[14] Working in a dusty environment helps to facilitate contamination of equipment and hands; therefore an acceptable standard of cleanliness for the working environment should be agreed with the domestic manager.[15]

The patient should be examined on a couch covered with either disposable paper sheeting or a clean linen sheet that is changed between patients. Any surfaces that have been contaminated must be cleaned adequately using detergents and water and disinfected if required.[16,17] Each NHS trust infection control department should have a decontamination policy and a list of recommended disinfectants.

Tubes of lubricants do become contaminated, so it is preferable to utilise individual lubricant sachets for each patient. However, some NHS trusts still recommend discarding the first 2.5 cm of lubricant from the tube at the beginning of each day and the tube is discarded after 1 week's usage; however, this practice is not research based.

Within the room there should be appropriately allocated separate waste bins for domestic waste (black bin liner) and for clinical waste products (yellow bin liner). Clinical waste includes anything that has come into contact with the patient and could be contaminated with the patient's body secretions or fluids. Domestic waste covers any other waste that can be disposed of in a landfill site.[16]

There should also be a washbasin for general cleaning and a clinical hand-hygiene basin to prevent cross-contamination from the dirty aerosols caused by water splashing back onto the hands during the washing process along with air back draft from the overflow recess.[18] Where an examination/treatment room does not provide a clinical hand washbasin, some infection control departments will accept alcohol hand gels, providing staff do wash their hands prior to patient consultation. Staff should be aware of the social perception that this may develop. Storage space (cupboards or drawers) should be used to prevent environmental contamination of gloves, aprons, and other equipment.[14]

36.2.2 Risk Assessment

Risk assessments should be an integral part of patient care in order to help staff prioritise and manage any professional risks and should be undertaken on each patient in order to identify any infections. An appropriate decision[14] can then be made either to postpone a required procedure and make an appropriate medical referral or to adopt suitable protective clothing and ensure the decontamination of equipment.

The management of risk assessment is the responsibility of the individual carrying out the procedure and is undertaken to protect both the clinician and the patient from the possibility of acquiring infection from one another. The use of pressure probes, vaginal or anal electrodes, or cone therapy should not proceed if there is any cause for concern or where a risk assessment identifies a possible communicable disease; guidance from the local infection control team should be sought. Risk assessment allows staff to choose appropriate protective clothing.

36.2.3 Standard Precautions

The term "standard precautions" is the generic term for the infection control precautions required while interfacing clinically with a patient. Standard precautions encompasses contact precautions and what was previously known as universal precautions or standard body fluid precautions or blood and body fluid precautions. These standard precautions should be observed when there is an anticipated clinical contact with the patient's skin, blood or body fluids.[19] These precautions, in addition to appropriate protective clothing, are normally adequate to protect both the clinician and the patient.

36.2.4 Protective Clothing

Protective clothing is a two-way barrier to protect the patient as well as the clinician and should be worn. Gloves and apron should be worn to prevent clothing and hands from becoming contaminated with bacteria or body fluids where there is a possibility of contact with a patient or their bodily fluids.[5,6,14] Where there is a risk of aerosol spray or splashing with body fluids, goggles also should be worn[5,6,14] by the clinician. For routine vaginal assessment, examination gloves (bulk boxed) should be worn. For any catheterisation procedure or examination of broken vaginal or vulval membranes, then sterile gloves and an apron must be worn and the clinician must follow principles of aseptic techniques.[5,6]

All gloves must be checked before the examination of a patient to ensure that they are not damaged. When gloves are removed, care must be taken to ensure that there is no possibility of self-contamination with the patient's body fluids and be discarded into a yellow clinical waste bag. The clinician must always wash their hands using an appropriate product before putting on and after removing gloves.

36.2.5 Probe Sheaths and Condoms

The condom or probe sheath may be of latex or some other material and is applied to cover the full length of the pressure device extending along any extension tube or other equipment as appropriate. Condoms are deemed to be adequate protection for perineometers, as perineometry does not use as vigorous a force as sexual activity (for which condoms have been shown to be impermeable to viruses with a 90–95% success rate[20,21]).

Hands must always be washed using an appropriate technique before placing a new sheath or condom on equipment; after use, the condom must be discarded into a yellow clinical waste bag. NB. Condoms that have a spermicide applied to them should not be used on patients having fertility investigations or problems because of the residual effects of the spermicide.

36.2.6 Cautionary Note on Latex

Standard precautions still require the use of latex products, as research shows that it provides proven protection against blood-borne viruses,[20,22] but research is ongoing into polyurethane and nitrile products as future alternatives. However, during the risk assessment, it should be ascertained whether the patient has any known latex problem or cross-protein-related allergies, for example, banana, kiwi fruit, and so forth. If there is a suspected latex irritation, sensitivity, or allergy reported, then no procedure using latex gloves or condoms should take place without prior medical consultation. Clinicians must follow their NHS trust policy on latex, which should incorporate specific action plans, procedures, and products in order to maintain a latex-free environment. Latex-free gloves, for example, Dermaprene and Allegard, and latex-free condoms/sheaths (eg, Femidom, Avanti, Trojan Supra) can be readily purchased.

36.2.7 Hand Hygiene

Thorough hand-washing technique should be practised at all times to reduce the number of surface skin microorganisms on the clinician's hands,[9] along with any transient environmental or patient microorganisms, which may have been picked up inadvertently. Thorough hand washing is normally sufficient to remove surface and transient bacteria.[16]

The hand hygiene technique is more important in removing bacteria than the time spent washing hands. Clinicians can augment hand washing by the use of 60–70% alcohol hand rub/gel, which will destroy transient and resident microorganisms. Where rapid skin disinfection on the same patient is required, an alcohol hand-rub solution can be used[5,6,11,14–16] if the hands are free from soil and clinically clean. This use of alcohol hand rub is supplementary to hand washing, not a substitute for it. The technique for distributing alcohol gel across the surface of the skin is the same as that for hand washing.[5,6,15]

A waterproof, bacterial proof, and viral proof dressing/cover must be applied by the clinician to any cuts, abrasions, or eczema that they may have in order to protect from environmental microorganisms and blood-borne pathogens.[5,6,16,19] Any skin disorder such as eczema or psoriasis should be reported to occupational health for treatment/advice.

36.2.8 Inoculation Injuries

All NHS Trusts are required to have an inoculation injury policy that incorporates the reporting of any inoculation injury or near-miss incident that places either patient or staff at risk. Any incidence is to be reported in accordance with local policy and should include the index case's and recipient's risk assessment, especially if there is any history of blood-borne or any sexually transmitted diseases. This will help to formulate the inoculation injury risk assessment and enable the clinician or patient to receive appropriate prophylactic treatment as required.[19] Incidents should be evaluated and audited to modify clinical procedures as appropriate.

36.2.9 Equipment

It is imperative under the controls assurance for infection control decontamination of medical devices[5–8] and the medical device agency guidance[23] that equipment is used in line with the manufacturers' stipulation with respect to its use. It may be considered single use (used once then discarded), single patient use (used for one patient's treatment programme and decontaminated between appointments), or multiple use (used on any patient provided that it is sterilised between each patient using appropriate infection control measures).

In view of the presence of normal skin and genital bacterial flora and other potential microorganisms in the genital area, equipment must be protected and decontaminated after use.[24] Each individual piece of equipment requires risk assessment for the level of decontamination required. This advice must be incorporated into written departmental protocols.

Items that have contact with mucous membranes are considered medium risk and should be sterilised by an autoclave or appropriate chemical sterilant solution between patients.[25] Where this cannot take place, the equipment must be cleansed, disinfected, and covered with a sterile sheath (agreement with the local infection control team should be sought). Items considered of lower risk are those in contact only with intact skin. In this case cleansing is sufficient.

When choosing any decontamination process, the issue of consumer protection[26] and medical devices regulations[27] must be borne in mind at all times. An individual clinician must comply with a manufacturer's guidance or instructions on decontamination and reprocessing, otherwise any product liability will transfer to the clinician or NHS Trust.

Collaboration between the users and companies can lead to an improved liaison between health care decontamination facilities and product design. This enables vaginal equipment to be sterilised in accordance with the requirements of Department of Health Safety Action Bulletin 108[24] and the controls assurance for infection control: decontamination of medical devices.[5–8] The issue of infection control not only encompasses the decontamination of equipment prior to and after use, but also includes consideration of the environment and risk assessment of the patient and staff and incorporates any product or company guidance as appropriate. This information should form the basis of any infection control policy to be agreed between the professional user-groups and the infection control department and then approved by the local infection control committee. Each policy should include names of the authors, names of those who have agreed it, the date on which it was agreed, and a review date, so that any clinician who reads or implements the policy can clarify any points or changes in practice required.

36.2.10 Decontamination

Decontamination is a three-stage process consisting of cleansing, disinfection, and sterilization, as described in Table 36.1.

Table 36.1. Possible decontamination processes.

Decontamination stage	Process	Method
Cleansing	The physical removal of contamination and many microorganisms	Detergent and water at 40–55°C. Rinse
	Cleansing always precedes disinfection or sterilisation	
Disinfection	The physical reduction of the number of microorganisms to a level at which they are not harmful. The process removes the majority of bacteria but spores will not usually be destroyed	Cleanse first as above. Temperatures of 90°C or chemical disinfectants
Sterilisation	The removal or destruction of all micro-organisms including spores	Cleanse first as above. Steam sterilisation at 121°C or 136°C, chemical sterilisation

It is imperative that any clinicians engaged in the hand washing of equipment must be suitably protected to guard against splashing of the eyes and the contamination of hands and clothing, ensuring that the clinician is protected from both microbiological contamination and chemical exposure. When choosing chemical disinfectants and sterilants, the local health and safety officer should be consulted to ensure that the environment is suitably ventilated or recommend appropriate extraction equipment.

Equipment washing should be performed only in a sink dedicated to that purpose; separate washing and rinsing facilities are required.[7] The purpose of washing equipment, prior to decontamination or sterilisation, is to remove body fluid and secretions and to reduce the level of microorganisms present in order to allow contact and/or penetration of a chemical disinfectant or steam.[17] When making up a solution of detergent it is imperative to follow the dilution instructions recommended. Insufficient detergent will not break down grease and proteinous material, while excessive detergent may not be rinsed off, which could result in the neutralisation of some disinfectants and sterilants.[18] Most detergents are activated between 40–55°C; as this is at the upper limit of being "hand-hot," it reinforces the need for hand protection, that is, the wearing of gloves.

The difference between disinfection and sterilisation is that the latter eradicates all microbial contamination including spores, whereas the former may not necessarily remove or inactivate all microorganisms from the surface of the equipment.[7,16]

It is preferable that any decontamination process for equipment is undertaken by the hospital sterilisation and disinfection units (HSDU) or a central sterilization services department (CSSD), as they are required to comply with the controls assurance in infection control: decontamination of medical devices,[7] and to implement the guidance on washer disinfectors (HTM2030)[28] and autoclaves (HTM2010).[29] Thereby, HSDUs and CSSDs are able to provide the required validation and documentation to enable batch tracing of sterilized items in the event of manufacturing problems or potential litigation.

36.2.11 Benchtop Washer–Disinfectors and Steam Sterilizers

In many NHS Trusts, decontamination of equipment has moved away from hand washing and local sterilization to automated processes under the control of HSDU/CSSD. However, where local decontamination is to be performed in a benchtop steam sterilizer, it is necessary that the decontamination be undertaken by automated procedures that are documented and can be validated in accordance with HTM2030.[28] Equipment must first be cleaned in a washer–disinfector prior to a secondary disinfection or sterilization process in a benchtop steam sterilizer. The department must have guidance and checklists relating to the decontamination process used on their equipment. These checklists must contain the validation and testing results that are undertaken daily, weekly, monthly, quarterly, and annually as shared functions between the clinician and the sterilizing engineer to ensure that the washer–disinfector performs consistently within set parameters. Hand washing of equipment does not meet this expectation as it introduces the potential for human error, for example, inappropriate water temperature and detergent and washing technique. Therefore, departments should have a protocol that specifies the frequency for selecting items of equipment at random for protein testing prior to further decontamination. Failure to remove all protein will result in failed decontamination.

Where benchtop steam sterilizers are being used for the sterilization of vaginal equipment, it is now expected that there is full compliance with sterilization guidance TM2010,[29] along with either benchtop steam sterilizers guidance[30] (for equipment for immediate use) or benchtop vacuum steam sterilizers[31] (for bagged equipment prior to autoclaving). This will protect equipment from environmental contamination for up to 6 months if stored in dry, nonhumid conditions.

It is imperative that anyone using these washer–disinfectors and sterilizers reads the MDA documentation in order to evaluate whether local or centralised sterilization is the most effective in terms of overall service provision. A transition period up to 2008 has been allowed under the

controls assurance for infection control: decontamination of medical devices[7] for NHS Trusts to achieve compliance.

36.2.12 Use of Chemical Disinfectants and Sterilants

During the transition period allowed under the controls assurance for infection control: decontamination of medical devices[7] to comply with automated decontamination and sterilisation, chemical disinfectants, or sterilants may continue to be used. The types of chemical disinfectants and sterilants that may be used are shown in Table 36.2.

The concentration of disinfectant or sterilant normally used should be of sufficient strength to kill viruses (refer to manufacturer's guidance). It should be noted that in the UK there is a move away from using aldehyde products due to the control of substances hazardous to health (COSHH) regulations on potential exposure to fumes.[32] Compatibility with the equipment manufacturer's specifications must be ensured before choosing any disinfectant or sterilant.

After use the disinfectant or chemical sterilant must be rinsed off using either sterile or distilled water to avoid environmental contamination and to remove chemical residue. It is recommended, where appropriate, that a condom or sheath be used on vaginal/anal devices prior to clinical procedure, ensuring full compliance with Safety Action Bulletin 108.[24] However, after the use of a nonchemical sterilant, for example, Sterilox, the equipment must be rinsed with sterile water only in order to avoid desterilising the equipment. Providing this is done effectively, then a condom/sheath should not be required prior to clinical procedure.

The inability to validate hand decontamination processes is the main reason for the introduction of validated and verified automated processes in the NHS. The added benefit of this automation is that condoms/sheaths will not be required, minimising the risk of latex sensitisation in future.

Table 36.2. Types of chemical disinfectants and sterilants.

	Chemical	Trade names
Disinfectants	Hypochlorite solution	Milton
	Sodium dichloriosocyanurate	Sanichlor
		Haztab
	Chlorine dioxides	Tristel
	Hydrogen peroxide	Virkon
Sterilants	Paracetic acid	Nu-Cidex
		Steris
		Peraclean
		Perasafe

36.2.13 Decontamination of Specific Equipment

Wherever possible all equipment not designated for single-use should be cleansed and disinfected or sterilised in accordance with Table 36.1. There may be instances where equipment cannot be disassembled for automated immersion decontamination, for example, ultrasound bladder scanner head. In such cases, where the bladder is scanned through the abdominal wall, the scan head should be cleansed using 5mL (one teaspoon) of detergent to 5L of hand-hot water, rinsed, dried, and wiped over with a 60–80% ethanol or isopropanol alcohol wipe between patient examinations.[14] If a translabial approach is used, in addition to the above decontamination technique, the scanner head should be covered with aqueous gel, followed by an appropriate clean disposable cover.

36.2.14 Decontamination of Equipment for Home Use

Each patient should be given full written instructions regarding the use and decontamination of equipment for home usage. This includes vaginal cones or any electrodes used for home stimulation or biofeedback. For example, in the case of vaginal cones these instructions should include information that the vaginal cones are intended for personal use only. Instructions should state that the hands must be washed thoroughly before using the cones and prior to first usage the cones must be disassembled, washed in warm, soapy water, rinsed, and dried thoroughly (this requires 5 ml of detergent to 5 liter of hand-hot water). After use the cones again should be disassembled, the same procedure followed, and then stored in a dry container. It should also be stated that it is not recommended to use cones during menstruation, or if there is thought to be any vaginal infection present,

and finally that the use of the cones should be discontinued if there is any discharge or discomfort. A contact number should be provided.

36.3 Summary

Infection control procedures will alter in light of future research and legislation. It is imperative for the clinician to receive current updates by liaison with their infection control specialist.

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37
UK Government Guidelines

F Booth

37.1 Introduction

Continence and incontinence were almost nonexistent words in political terms before 1991 at which time the UK government started to consider the problems of patients with incontinence.

37.2 1991: Agenda for Action on Continence Services[1]

When the agenda for action on continence services[1] was introduced, the whole issue of improving the quality of life of people who have any level of bladder or bowel dysfunction was instigated.

37.3 1999: Audit Commission[2]

In 1999, an audit commission review, "First Assessment. A Review of District Nursing Services in England and Wales,"[2] made it abundantly clear that standards of continence care fell well below that considered to be acceptable. The report highlighted that district nursing services were under pressure with often a mismatch between resources, skills, and demand.

37.4 2000: Good Practice in Continence Services[3]

When "Good Practice in Continence Services"[3] was commissioned, it was hoped that it would be a report that would be prescriptive and with which NHS and other organisations would be required to comply. However, the final report only included clinical guidelines. Although based on research and/or clinical evidence, it recommended, but could not enforce, improved standards of care.

For many years and arguably still today, incontinence pads were the mainstay of many continence services. Outcomes were measured dependent on the satisfaction of clients between one pad or another, or whether the pads were fit for the purpose. There was little consideration as to cure; this seems to have been a concept that has taken a very long time to be developed in the mindset of individual clients and health professionals, many assuming wrongly that nothing can be done to ameliorate incontinence symptoms.

Many continence services were established with the added title of "advisory" so the "continence advisory service" was born. However, many services have dropped the title of "advisory," as advice is now only a small part of what many services offer.

37.5 2000: NHS Plan[4]

In our present day, the concept of cure or treatment should form the mainstay of all continence care provision whether this is in hospital, primary care, or the independent sector. Previously, the domain of the nursing profession[4] acknowledged that other health care professionals, especially physiotherapists and occupational therapists, should have a significant role to play either as part of the cure or treatment programme and most certainly as a partner within the care pathway.

Table 37.1. Useful websites for government standards and best practice statements.

http://www.dh.gov.uk/PolicyAndGuidance/ResearchAndDevelopment/ResearchAndDevelopmentAZ/PhysicalAndComplexDisabilities/PhysicalAndComplexDisabilitiesArticle/fs/en?CONTENT_ID = 4015932&chk = VWKapn
http://www.nice.org.uk/home.aspx?c31333
http://www.prodigy.nhs.uk/guidance.asp?gt = Constipation
http://www.seekwellness.com/incontinence/references.htm
http://search.library.nhs.uk/nhs_sse/zengine?VDXaction = ZSearchResults
http://continenceaudit.rcplondon.ac.uk/modules/page/page.aspx?pc = welcome
http://www.parliament.the-stationery-office.co.uk/pa/cm/cmhansrd.htm

37.6 Care Pathways

To ensure that all patients receive the optimum treatment, they should be involved, where possible, in decisions regarding their care. Consequently, care pathways have been developed (see Table 37.1). Care pathways should be patient-led and driven accessible as part of the single assessment process and have a unified single point of access.

This will fulfil the government's agenda initiated as part of the NHS plan in 2000,[4] be provable through the essence of care[5] (benchmarking process), and take into consideration the national service frameworks that are on going, for example, for older persons[6] and children (see Table 37.2 for Department of Health and other websites).

37.7 National Institute for Health and Clinical Excellence

The National Institute of Health and Clinical Excellence (NICE) offers guidance that is specific to a clinical condition and not as a generic statement. There is an ongoing process of commissioning NICE guidelines. Multiprofessional committees review quality research on specific topics; the guidelines are then accessible on the NICE website (see Table 37.2).

Table 37.2. Care pathway

Single assessment process begins through single point of access
Incontinence issues identified
Treatment/cure or management programme is identified and actioned
Review process to evaluate programmes of care
Programme changed or continued for an agreed upon period
Initial contact
If programme changed, new programme agreed to
If no changes, agree to review periods

37.8 2001: The Expert Patient[9]

All health care professionals are bound by their professional body's codes of professional conduct, for example, for nurses, this is the nurses and midwifery council codes of professional conduct.[8] These standards may cause certain conceptual problems with the need to listen to and act on the decisions of the expert patient[9] and practice in ways that promote choice.

37.9 2003: DoH Policy for Choice[10]

Giving patients more choice about how, when, and where they receive treatment is one cornerstone of the government's health strategy.[10] The greatest concerns may well be that patients will not be able to make valid choices about their own health conditions especially if their condition is serious or life threatening or they are in pain. It is up to all health professionals to ensure that information is delivered in a clear and understandable way, at a level that patients can understand.

Incontinence (bladder and bowel dysfunction) remains to this day a taboo subject; there is stigma among health and social care professionals as well as individual clients and their carers, even though the Leicester MRC Study[11] of more than 10,000 adults identified some 15% of respondents admitting to levels of incontinence.

Although campaigning has been vigorous by numerous organisations, many questions have been asked within both the House of Lords and the House of Commons. There is all government party support, yet there is little progress further than recommendations that may be followed as best practice.

37.10 2005: Royal College of Physicians National Audit[12]

A Royal College of Physicians National Audit[12] reported that trusts still do not comply with some guidelines that were issued as part of the *National*

Service Framework for Older People.[6] This included key requirements that there would be an integrated continence service in place by April 2004. More than 2 years after the deadline date there has been little progress. Perhaps the government should be more prescriptive.

None of these concepts or attitude changes are new. Lewin,[13] as long ago as 1947, recognised the need for change and the processes that it would incur. In 1982, Smith[14] reported that "much has changed, but much remains the same."

Effective health care provision for continence needs requires not only knowledge of the evidence for best practice, but also any governmental changes in policy and provision.

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Appendix 1
Fluid Intake and Bladder Diary

Start your diary on . . . Id Number . . .

Time	Record drinks taken type and amount	Record Volume	Tick when you changed a pad/panty liner	Each time you leak urine, circle whether you were:
6am				Almost Dry Damp Wet Soaked
7 am				Almost Dry Damp Wet Soaked
8am				Almost Dry Damp Wet Soaked
9am				Almost Dry Damp Wet Soaked
10am				Almost Dry Damp Wet Soaked
11am				Almost Dry Damp Wet Soaked
Midday				Almost Dry Damp Wet Soaked
1pm				Almost Dry Damp Wet Soaked
2pm				Almost Dry Damp Wet Soaked
3pm				Almost Dry Damp Wet Soaked
4pm				Almost Dry Damp Wet Soaked
5pm				Almost Dry Damp Wet Soaked
6pm				Almost Dry Damp Wet Soaked
7pm				Almost Dry Damp Wet Soaked
8pm				Almost Dry Damp Wet Soaked
9pm				Almost Dry Damp Wet Soaked
10pm				Almost Dry Damp Wet Soaked
11pm				Almost Dry Damp Wet Soaked
Midnight				Almost Dry Damp Wet Soaked
1am				Almost Dry Damp Wet Soaked
2am				Almost Dry Damp Wet Soaked
3am				Almost Dry Damp Wet Soaked
4am				Almost Dry Damp Wet Soaked
5am				Almost Dry Damp Wet Soaked

One day of 3 days' charts provided to patients. (Courtesy of Leicestershire MRC Incontinence Study).

Reminders for Completing the Charts for the Leicestershire MRC Incontinence Study:

- Don't forget to record the time you woke up in the morning and the time you went to sleep.
- Don't forget to record what happened overnight when you get up in the morning.
- Try and make a record of things just after they happen in case you forget them later on.
- Record things to the nearest hour.
- Record type and amount of drinks taken (e.g. 2 cups of tea, 1 mug of coffee, 1 can of coke, 1 glass of water/wine/juice, 2 pints of beer).
- Start a new sheet for each new day.

Appendix 2
Urinary Continence Assessment Form

URINARY CONTINENCE ASSESSMENT FORM

Name ..... DOB ..... Age .....
Address .....
Tel. No: Home ..... Work .....
Occupation/Hobbies ..... Referral .....
Problem .....
Duration ..... GP .....

Symptoms	Initial	Disch	Severity	Initial	Disch
Stress			Daily		
Urgency			>1/week		
Urge incontinence			<1/week		
Frequency			>1/month		
Nocturia			<1/month		
Nocturnal enuresis					
Incomplete emptying			Few drops		
Pain			Wets underwear		
Hesitancy			Wets outerwear		
Family history			Runs down legs		
Childhood problems					
Other: e.g. dyspareunia			No. of pads per day		
Coital incontinence			Size of pads		
Frequency Volume Chart Freq. of void/24h					
Freq. of incont/24h					
Max. voided volume					
Min. voided volume					
No. of drinks/24h					
Caffeine/24h			Stop test – not permitted		

History

Parity .....
Wt. heaviest Baby .....
Types of delivery .....
Menopausal state .....
Pregnant Yes/No/Planning
HRT Yes/No
When commenced HRT .....
Smear test .....

Surgical History

.....
.....
.....
Bowels: B/O .....week
Faecal incontinence .....
Faecal urgency .....
Constipation .....
Stool consistency .....

Medical History

Cystitis .....
Smoking .....
Respiratory Problems .....
Allergies .....
Cardiac .....
Diabetes .....
Back/Neck Problems .....
Other .....

Height .....
Weight .....

Investigations:
MSU .....
Urodynamics .....
Other .....

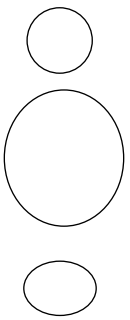
Current Medication .....
.....
Previous treatment (including medication) .....
.....

On Examination **Informed consent given Y/N**

Dermatomes .....
Myotomes .....
Reflexes .....
Abdominal examination .....

Pelvic floor – digital assessment

	Initial	Discharge
Oxford Grading 012345		
Endurance		
Repetitions		
Fast contractions		
Perineometer reading		
Hold with cough		



If you were to spend the rest of your life with your urinary condition just the way it is now, how would you feel about that?

Quality of life due to urinary symptoms	**Delighted**	**Pleased**	**Mostly satisfied**	**Mixed: equally satisfied and dissatisfied**	**Mostly dissatisfied**	**Unhappy**	**Terrible**
Initial							
Discharge							

Signature ..... Date: .....

Comments

Appendix 3
Useful Websites

International

- International Continence Society : *www.icsoffice.org*
- International Urogynecological Association: *www.iuga.org*

UK Organisations

- Association for Continence Advice (ACA): *www.aca.uk.com*
- Association of Chartered Physiotherapists in Women's Health: *www.acpwh.org.uk*
- Continence Foundation: *www.continence-foundation.org.uk*
- Chartered Physiotherapists Promoting Continenece: *www.cppc.org.uk*
- Education and Resources for Improving Childhood Continence: *www.eric.org.uk*
- InconTact: *www.incontact.org*
- Promocon: *www.promocon.co.uk*

Subject Index