Principles and Practice of Urogynaecology

A Tamilselvi
Ajay Rane
Editors

Springer

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and Women's Health
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ISBN 978-81-322-1691-9 ISBN 978-81-322-1692-6 (eBook)
DOI 10.1007/978-81-322-1692-6
Springer New Delhi Heidelberg New York Dordrecht London

Library of Congress Control Number: 2014960233

Dedicated to my family and the quietly suffering women the world over.

Ajay Rane

This book is dedicated to my parents – my father, whose only dream has been to see me as a doctor, and my mother, supporting me all the way through – and to all my teachers.

A Tamilselvi

Foreword

Urogynaecology as a subspecialty is a relatively new field in India. Approximately one-third of gynaecological practice involves women presenting with complaints relating to pelvic floor problems. Urogynaecology involves not only vaginal surgery but conservative and medical management of pelvic organ dysfunction as well. An understanding of both the anatomy and physiology of pelvic organs is essential for optimal management of patients presenting with these problems.

There has been a plethora of textbooks relating to other subspecialty topics in Gynaecology such as Reproductive Medicine, Laparoscopy and Gynaecological Oncology. Books in Urology cover the topics of Bladder Physiology, Neurourology and Urodynamics in detail, with minor emphasis on complex features of pelvic organ prolapse and anal incontinence. Similarly, most textbooks on Gynaecology give minimal details on subjects such as Neurourology and Urodynamics. There is an immediate need for comprehensive textbooks on urogynaecology, which spans the entire spectrum of female pelvic floor dysfunction.

In this regard, this textbook, *Principles and Practice of Urogynaecology*, is a timely and welcome step by the authors. This book not only provides basic anatomical and physiological concepts of female pelvic floor disorders but also covers evidence-based and up-to-date clinical and surgical principles. I believe that this book will help both postgraduates and practising Gynaecologists and Urologists understand the fundamental concepts of female pelvic floor disorders and apply them in clinical practice.

Each chapter in this book is written by authors with extensive experience in their particular field. The arrangement of chapters is logically designed to start from basics and take the readers to complex topics, which can be easily understood.

I congratulate the authors for this monumental work and wish them the best in their endeavours and contribution to the field of Urogynaecology.

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Preface

The faction of general gynaecologists is gradually reducing in numbers, with various sub-specializations in Gynaecology. The truth however is that most gynaecologists encounter women with pelvic floor problems irrespective of their sub-specialization. It therefore becomes vital to be armed with knowledge on important aspects of Urogynaecology in caring for these women. Acquisition of knowledge usually starts with understanding basic principles and moving on to evidence-based assessment and management.

This textbook, *Principles and Practice of Urogynaecology*, is designed to meet this need in Urogynaecology. The textbook attempts to achieve a level where everybody – students, trainees, specialists and sub-specialists – will benefit from the collective wisdom of the authors. The book initially deals with both the structural and neurological anatomy of the pelvic floor to provide a better understanding of basic principles. The subsequent divisions on pelvic organ dysfunctions elaborate on the evidence-based management of these problems.

The highlight of the book is an almost 'East meets West' flavour, with a blend of contributing authors from around the globe who are most respected in their fields. We would sincerely like to thank the contributing authors for their professionalism, clarity in their chapters and patience with us.

We hope that readers of this textbook will find it useful in their clinical practice – both in the outpatient setting as well as in operating theatres. We also trust that students will benefit from the illustrations employed to make the concepts easier to assimilate.

We would like to thank our colleagues for providing their feedback, proofreading the chapters with patience and providing any assistance as deemed necessary. Sincere thanks to our publishers Springer (India) for their conviction in bringing out the textbook in Urogynaecology. Particular thanks are expressed to Ryan Bishal Faruque, sixth-year medical student at James Cook University, Australia, and Mr. Rajan Babu, Chennai for the illustrations.

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Part I

Anatomy and Physiology

Current Concepts in Pelvic Anatomy

1

Arjunan Tamilselvi

Introduction

Female pelvic anatomy can be a conceptual challenge. Understanding the anatomy helps in identification, assessment, and management of pelvic floor pathology. To those dealing with disorders of the pelvic floor and to the reconstructive pelvic surgeon, knowledge of the functional anatomy is crucial. Apart from the anatomical dissections, various imaging modalities particularly magnetic resonance imaging (MRI) have helped in appreciating the relationships and supports of the pelvic organs better. Improved understanding of the pelvic anatomy should make it possible to align anatomy with clinical concepts seamlessly.

The pelvic viscera have an integrated anatomical support with each other due to their orientation within the pelvis, and at the same time, they largely function as individual units. The pelvic floor musculature and the pelvic connective tissue (including fascia and ligaments) form the primary support for the pelvic organs with the bony pelvis providing additional support. In general, the normal anatomical supports of the female pelvic viscera play a vital role in the reproductive, storage, and elimination functions of the pelvic organs, apart from trying to maintain them in their normal position.

The aim of this chapter is to describe the topographic or spatial pelvic anatomy, in a practical aspect to help in optimizing the surgical techniques in pelvic surgery. Three different anatomic systems contribute to the support of the pelvic organs – the bony, muscular, and fascial support. The clinically relevant bony, muscular, and fascial supports and the surgically important spaces in the pelvis are described below along with a brief mention of their clinical/surgical relevance. For a more exhaustive anatomy, the reader is referred to anatomy textbooks cited in the reference.

Bony Support

The female bony pelvis provides the essential framework for muscular and fascial supports. It is important in reconstructive pelvic surgery as certain bony landmarks are the surgical reference points for suture placement and for graft anchoring in reconstructive surgery. The (innominate) hip bones (composed of the ilium, ischium, and pubis) articulate with the sacrum at the sacroiliac joint posteriorly and with each other anteriorly at the pubic symphysis. Within the bony pelvis, there are several important landmarks, openings, and spaces which are of clinical relevance.

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A. Tamilselvi, A. Rane (eds.), *Principles and Practice of Urogynaecology*,
DOI 10.1007/978-81-322-1692-6_1, © Springer India 2015

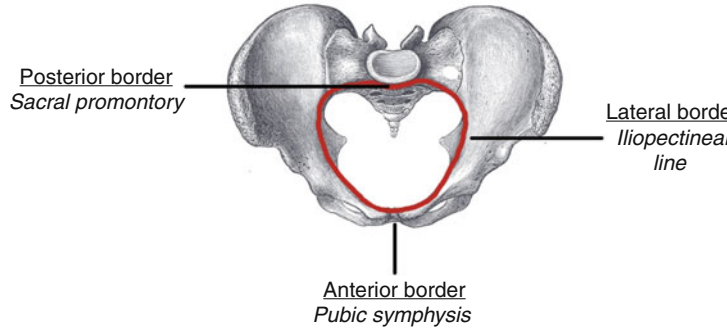

Fig. 1.1 Borders of the pelvic brim (Reproduced from: www.teachmeanatomy.com; with permission)

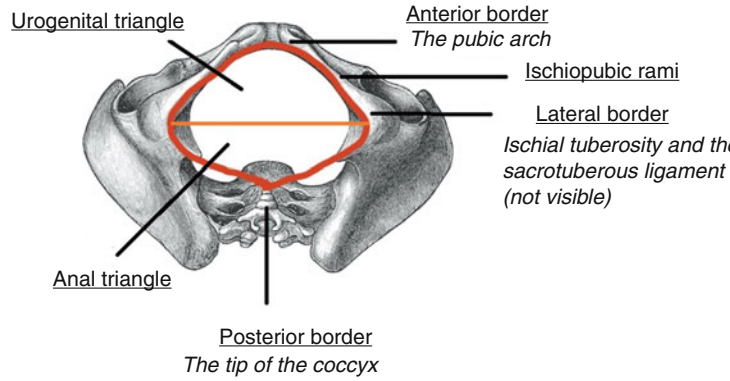

Fig. 1.2 Pelvic outlet borders (Reproduced from: www.teachmeanatomy.com; with permission)

The anterior projection of the first sacral vertebra, Sacral Promontory, forms the posterior boundary of the plane of the pelvic brim, while the superior aspect of the symphysis pubis and the iliopectineal lines form the anterior and lateral boundaries respectively (Fig. 1.1). Above the plane of the pelvic brim, the prominent projection of the ilium is the anterior superior iliac spine, from which the inguinal ligament stretches up to the pubic tubercle medially. The large femoral neurovascular bundle passes from the abdomen to the thigh midway underneath the inguinal ligament.

The pelvic outlet is diamond shaped with the apices defined by bony landmarks – inferior aspect of the symphysis pubis anteriorly, ischial tuberosities laterally, and tip of the coccyx posteriorly. The outlet can be subdivided into the anterior urogenital triangle and the posterior anal triangle. The lateral edges of the anterior triangle are the ischiopubic rami – which have assumed significance in delineating the obturator foramen for graft anchorage (Fig. 1.2).

Obturator Foramen

The obturator foramen is a large oval window, bounded by the superior and inferior pubic ramus, body of the pubis, ischial ramus, and body of the ischium. The obturator membrane covers this opening almost completely except for a small opening in the superolateral portion of the foramen, the obturator canal through which the obturator neurovascular bundle passes from the pelvis to the medial compartment of the thigh. The obturator internus muscle on the inner side of the obturator membrane originates from the bony margin of the obturator foramen and partly from the pelvic side of the obturator membrane. A curvilinear thickening of the parietal fascia overlying the belly of the obturator internus known as the arcus tendineus levator ani (ATLA) extends from the posterior pubic symphysis to the ischial spine. The obturator externus arises from the outer surface of the obturator membrane and from the pubic and ischial rami and from the medial two-third of the obturator membrane,

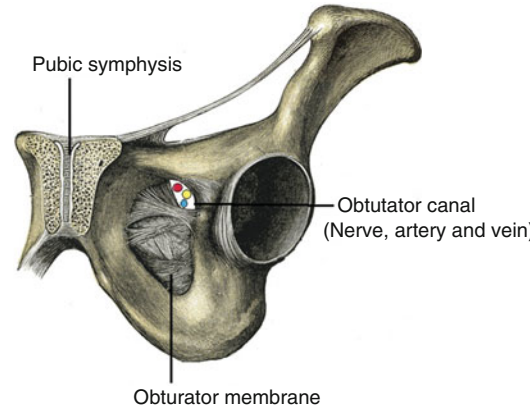

Fig. 1.3 Obturator foramen covered with obturator membrane and the location of obturator canal (Reproduced from: www.teachmeanatomy.com; with permission)

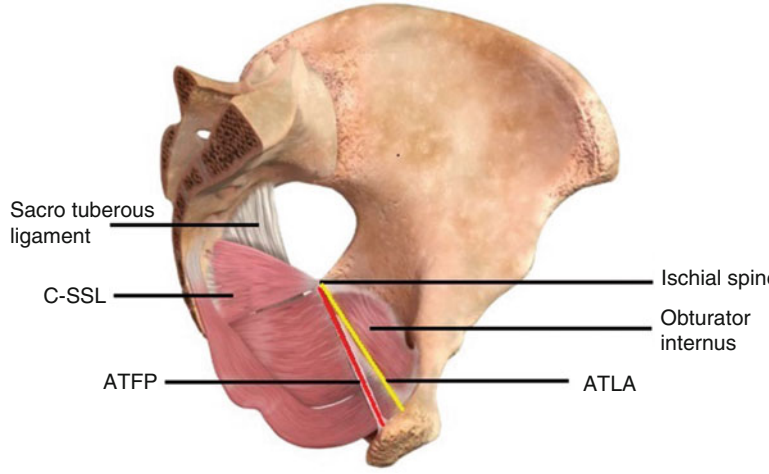

Fig. 1.4 Sagittal section of the bony pelvis. *ATLA* arcus tendineus levator ani, *ATFP* arcus tendineus fascia pelvis, *C-SSL* coccygeus–sacrospinous ligament complex

attaching itself to the greater trochanteric fossa of the femur (Fig. 1.3).

Applied Anatomy

The potential safety of the obturator foramen with the neurovascular bundle occupying only its superolateral portion, has made it a safe zone for insertion of the trocars in the transobturator mesh anchoring techniques.

The Ischial Spine

The ischial spines are bony prominences projecting from the medial surface of ischium at the anterior border of greater sciatic notch. The sacrospinous ligament (SSL) passes medially and posteriorly from the ischial spine to the lateral aspect of lower portion of the sacrum and coccyx. The sacrotuberous ligament extends from the posterior surface of sacrum to the ischial tuberosity, and this along with the SSL separates the greater sciatic foramen from the lesser sciatic foramen.

Applied Anatomy

The ischial spines are important surgical reference points for several pelvic structures (Fig. 1.4):

- The pelvic ureter usually leaves the pelvic sidewall about 1–2 cm from the ischial spine to pass medially on the pubocervical fascia before entering the bladder.
- The pudendal nerve and vessel exit the pelvis through the greater sciatic foramen and course beneath the ischial spine and sacrospinous ligament before reentering the lesser sciatic

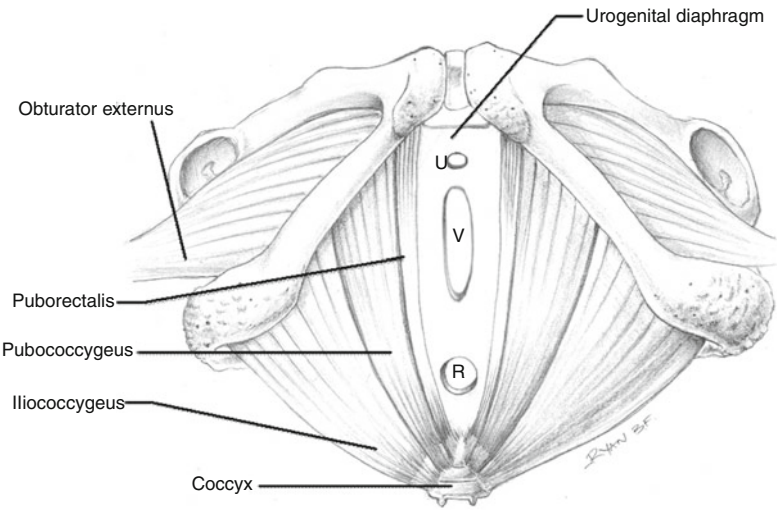

Fig. 1.5 Levator ani muscle – pubococcygeus, puborectalis, and Iliococcygeus. *U* urethral opening, *V* vaginal opening, *R* rectum

foramen. Identification of ischial spine is essential when performing sacrospinous fixation and planning suture placement to avoid injury to the pudendal neurovascular bundle.

- The arcus tendineus fascia pelvis (ATFP) and arcus tendineus levator ani (ATLA) both extend from the posterior surface of the pubic bone and end at the ischial spine.

Muscular Support

The levator ani (LA) muscle constitutes the primary muscular support to the pelvic organs. The LA muscle consists of three components – the pubococcygeus, puborectalis, and iliococcygeus, nomenclatures based on the origin and insertion of the muscle components (Fig. 1.5).

The anterior division of levator ani, the pubococcygeus, sweeps downward from the inner surface of the pubic bone along the sides of the urethra, vagina, rectum, and perineal body. This is further subdivided into the pubovaginalis, puboperinealis, and puboanalis. The pubovaginalis portion attaches to the lateral wall of vagina, the puboperinealis attaches to the perineal body, and the puboanalis portion attaches to the anus at intersphincteric groove.

The puborectalis, most caudal part of levator ani, originates from the inner surface of pubic bone and forms a U-shaped sling behind the anorectal junction and contributes to the anorectal angle.

The iliococcygeus portion arises from the ATLA and ischial spine on both sides and joins with each other at the iliococcygeal raphe and coccyx. The iliococcygeal raphe between the anus and coccyx is referred to as the levator plate and provides support to the rectum, upper vagina, and uterus. The openings between the levator ani muscles through which the urethra, vagina, and rectum pass is known as the urogenital hiatus. The whole expansion of the levator ani along with the coccygeus muscle, perineal membrane, and perineal body is the pelvic diaphragm.

Applied Anatomy

- The normal resting levators maintain a constant state of contraction and relaxation occurs only during the elimination process (micturition, defecation, and parturition). Contraction of the pubococcygeus elevates the urethra and the anterior vaginal wall helping in urinary continence. Pubococcygeus and puborectalis contraction also elevates the anus and keeps the urogenital hiatus closed. Contraction of the levator ani can be assessed on rectovaginal examination while instructing the patient to squeeze the muscles as if holding bowels. The "U-shaped" muscle is felt along the side and posterior vaginal wall.
- Neuromuscular injury to the levators such as during childbirth can lead to widening of the urogenital hiatus and lead to vertical inclination

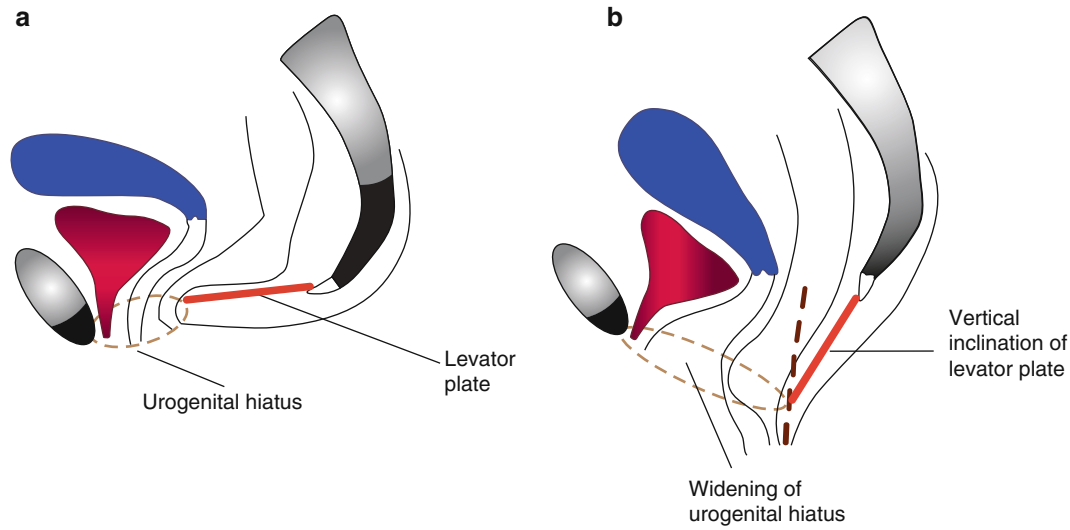

Fig. 1.6 Levator plate in horizontal orientation (**a**) and levator plate with vertical inclination (**b**) (From: Beco [2]; with permission)

of the levator plate leading to dysfunction or prolapse of the pelvic organs (Fig. 1.6a, b).

- Levator avulsion, a documented injury of childbirth, was first reported as early as 1907. Using ultrasound imaging Dietz identified that levator avulsion involves detachment of the puborectalis portion from pelvic sidewall and it occurs in about 36 % after vaginal delivery. Avulsion can be diagnosed digitally by palpating the inferior pubic ramus and feeling for insertion of the puborectalis portion. In the presence of levator avulsion, 2–3 cm lateral to the urethra, bony surface of the pubic ramus can be palpated devoid of the muscle.

Perineal Membrane

A thick fibromuscular sheet that stretches across the anterior urogenital triangle of pelvic outlet, caudal to levator ani, is the perineal membrane (formerly known as the urogenital diaphragm). It attaches laterally to the ischiopubic rami and has a free posterior margin with anchorage at the perineal body. The urethra and vagina pass through the hiatus in perineal membrane (Fig. 1.7). The perineal membrane hence fixes distal urethra, distal vagina, and the perineal body to bony pelvis at the ischiopubic rami. The superficial perineal space lies external to the perineal membrane and contains the superficial perineal muscles, ischiocavernosus, bulbospongiosus and superficial transverse perineal muscles. The deep perineal pouch lies between the perineal membrane and levator ani and contains the external urethral sphincter, compressor urethrae, urethrovaginalis, and the deep transverse perineal muscles (Fig. 1.8).

Perineal Body

The perineal body situated between distal vagina and anus is a point of convergence for different structures. The superficial perineal muscles, bulbospongiosus and superficial transverse perineal muscles, external anal sphincter, perineal membrane, deep transverse perineal muscles, distal part of the rectovaginal fascia, pubococcygeus and puborectalis portion of the levator ani all insert into this mass of connective tissue (Fig. 1.9). The perineal body plays an important role in the support of distal vagina and maintaining normal rectal function. In reconstructive surgeries, it is therefore important to restore the perineal body anatomy by proper re-approximation.

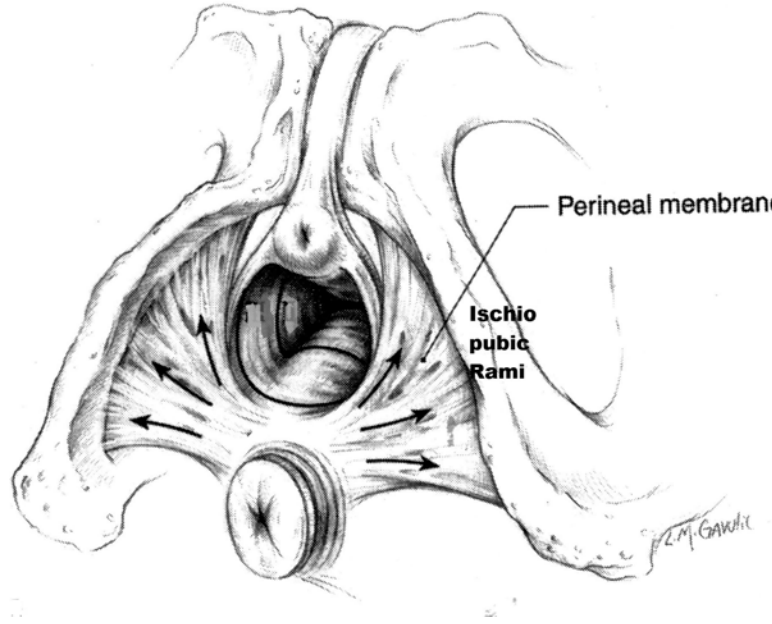

Fig. 1.7 Perineal membrane, direction of the fibers and its attachment (From DeLancey [6]; with permission)

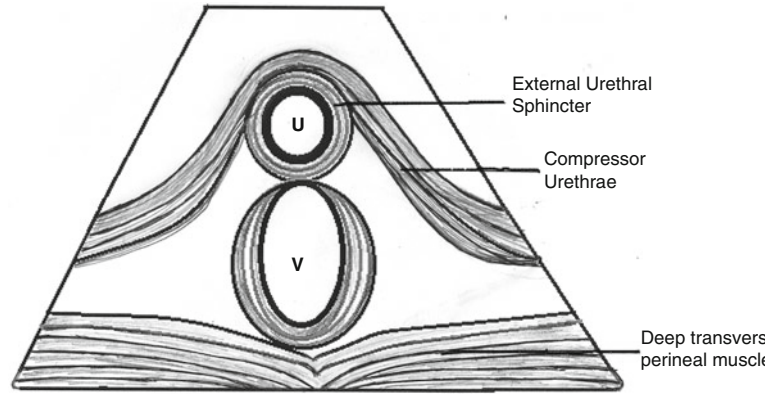

Fig. 1.8 Muscles of the deep perineal pouch

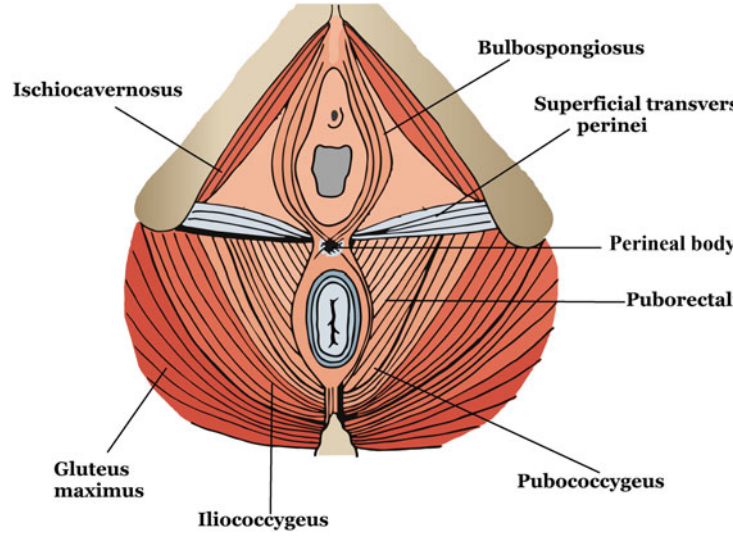

Fig. 1.9 Perineal body with its muscular attachments

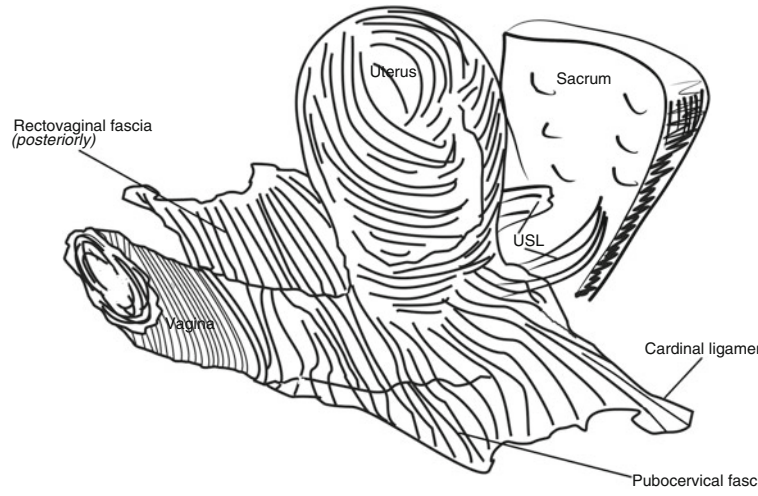

Fig. 1.10 Schematic representation of the endopelvic fascia extent. *USL* uterosacral ligament

Applied Anatomy

Orientation of the superficial perineal muscles in relation to vaginal outlet is important in reconstruction of the perineum (perineorrhaphy) and in episiotomy repair. In an ideal reconstruction, the widened genital hiatus is narrowed, and perineal body length is maintained.

Connective Tissue Support: Pelvic Fascia and Ligaments

The pelvic fascia has two components: parietal and visceral fascia. The parietal fascia covers the muscles along lateral pelvic wall and on the superior surface of pelvic diaphragm. The fascia covering the obturator internus muscle, obturator fascia, has two thickened portions: arcus tendineus levator ani (ATLA) and arcus tendineus fascia pelvis (ATFP), extending from the ischial spines to posterior surface of pubic bone. Portions of levator ani originate from ATLA, while the ATFP provides the lateral point of endopelvic fascial attachment (*see* Fig. 1.4).

The existence of the visceral fascia, in certain areas of pelvis and its various nomenclatures, has been an area of controversy in pelvic anatomy. The bladder and vagina are not enclosed in their own fascial layer, and the vagina appears to be separated from bladder anteriorly, only by the adventitial layer of vagina. The existence of a separate fascial layer between anterior vaginal wall and bladder has been of dispute on histological studies, and the nomenclature of pubocervical/pubovaginal fascia is being questioned. On the other hand condensation of the visceral fascia between rectum and vagina, the rectovaginal fascia, is an identifiable separate layer. This extends from the perineal body proximally to about 2–3 cm above hymenal ring. Above this level there is no separate fascial layer, and the endopelvic fascia attaches posterior vaginal wall laterally to pelvic side wall.

The endopelvic fascia is not a true fascia on histology and correct terminology would be endopelvic connective tissue. The term endopelvic fascia, however, is commonly used. Condensations of these fascia termed as ligaments include the uterosacral, cardinal, pubovesical, and pubourethral ligaments. The endopelvic tissue is a continuous layer extending from the uterosacral ligaments proximally to the pelvic portion of levator ani muscle distally, up to the level of urethra (Fig. 1.10).

The endopelvic fascia also extends laterally across the pelvis, with its medial attachments to the lateral wall of cervix and vagina and lateral attachments to pelvic sidewall along the ATFP. This attachment stretches the vagina transversely between bladder and rectum and divides pelvis into an anterior and posterior compartment. The bladder and urethra occupy the anterior compartment

the rectum and anal canal, posterior compartment and the uterus and cervix, the middle or apical compartment.

The three integrated levels of pelvic support projected by DeLancey to explain pelvic organ prolapse are defined by the endopelvic connective tissue attachments.

Level I Support

The cervix and upper vagina are suspended by the endopelvic fascia (parametria, paracolpium) and condensations of the connective tissue, uterosacral and cardinal ligaments. Uterosacral ligaments pass posteriorly from cervix and upper vagina, form lateral boundaries of pouch of Douglas, and attach to the front surface of sacrum from sacrococcygeal joint up to the level of S3. The cardinal ligaments (transverse cervical) extends from the cervix and lateral vaginal fornix to the posterolateral pelvic wall. These attachments are referred to as the level I or suspensory support. Failure of level I support leads to uterine or vaginal vault prolapse (apical prolapse).

Level II Support

The fascial attachment in mid-vagina extends from lateral vaginal walls to the ATFP and medial surface of levator ani. It prevents descent of the anterior and posterior vaginal walls with increase in abdominal pressure. This is referred as level II support or attachment axis. The differentiation between a "central cystocele" and a "paravaginal defect" in anterior compartment prolapse is based on the type of endopelvic fascia deficiency at this level. In central cystocele (distension cystocele), there is weakening of the connective tissue in midline, resulting in loss of midline rugosity of the vaginal wall. Lateral cystocele or paravaginal defect results from lateral detachment of fascia from the ATFP, and central rugosity are preserved in these. Prior to surgical intervention, it would be helpful to identify the type of anterior wall prolapse, lateral detachment or central failure to plan the optimal surgical technique.

The endopelvic connective tissue also extends as pubourethral ligaments, from the urethra to posterior surface of pubic bone, providing urethral support and maintenance of bladder neck closure during Valsalva maneuvers. The bladder neck in addition, through its relation to the anterior vaginal wall is indirectly supported by the attachment axis. Hence failure of level II support results not only in anterior and posterior vaginal wall prolapse but also to stress urinary incontinence.

Level III Support

The lower one-third of vagina is fused with the surrounding structures; through the endopelvic fascia anteriorly to distal urethra, posteriorly to perineal body, and laterally to pubovaginalis muscle and perineal membrane. This is referred to as the level III support or fusion axis. Level III disruption anteriorly can result in stress urinary incontinence from urethral hypermobility and posterior disruption can result in distal rectocele or perineal descent.

Ligaments Used in Reconstructive Surgery

Sacrospinous Ligaments (SSL)

The sacrospinous ligament with its lateral attachment at ischial spines has a close relationship with pudendal neurovascular bundle and to sciatic nerve (Fig. 1.11). The anterior surface of the SSL is muscular and forms the coccygeus muscle and is referred to as the coccygeus–sacrospinous ligament complex (C-SSL). The average length of the SSL is 5.43 cm and the position of the pudendal complex is about 0.9–3 cm medial to ischial spine behind the SSL. Suture placement for SSL fixation ideally should be 1.5–2 cm medial to ischial spine. It should never be across the entire thickness of the ligament to prevent damage to underlying structures. The sciatic nerve lies 2.5 +/− 0.4 cm lateral to the ischial spine posterior to SSL. On cadaver dissections, either the third sacral nerve or the pudendal nerve is found to course on the superior border of C-SSL complex at its midpoint in a significant proportion. The diligence in trying not to enclose the whole width of the ligament at SSL fixation is important for this reason.

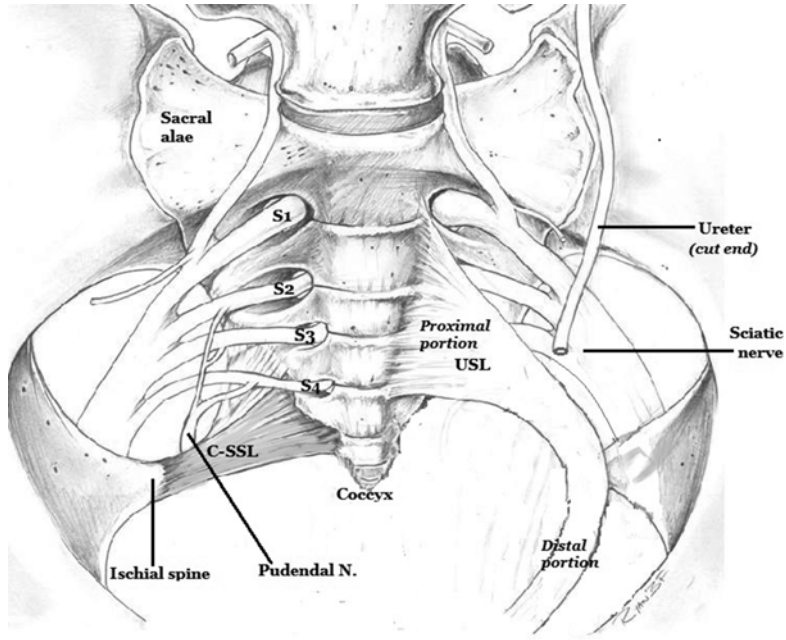

Fig. 1.11 Coccygeus–sacrospinous ligament complex (*C-SSL*) with the pudendal nerve posterior to the ligament. Uterosacral ligament (*USL*) and relation to the sacral plexus, ureter, and Sciatic nerve

Uterosacral Ligaments

The uterosacral ligaments (USL) are attached distally to the posterior aspect of cervix and upper vagina and laterally merge with the cardinal ligaments. Proximally it has a diffuse sacral attachment to the pelvic surface of sacrum, vertically from sacrococcygeal joint to S3 vertebra and transversely from sacral foramina medially to 5 cm lateral to sacroiliac joint. USL is 12–14 cm long and can be subdivided into three sections: distal (2–3 cm), intermediate (5 cm), and proximal (5–6 cm). The distal portion is commonly used to anchor the vaginal apex in McCall's culdoplasty. The proximal portion is diffuse in attachment and generally thinner. The intermediate portion is thick and well defined and is at least 2.5 cm away from the ureter and hence suitable for suspension procedures.

Interaction between Muscular and Connective Tissue Supports

The dynamic interaction between the muscular and connective tissue supports is critical for pelvic organ support. The pelvic organs are suspended over the levator plate by fascial support. The resting tone of levator ani muscle reduces the tension on the connective tissues due to their lateral muscular site attachments. With increase in intra-abdominal pressure, apart from the resting tone of levator ani and the closure of urogenital hiatus, there is further reflex contraction of pelvic floor musculature moving the pubococcygeus and puborectalis towards pubic bone. This further narrows the urogenital hiatus and prevents pelvic organ descent. All these reduces the strain on fascial support at times of increased abdominal pressure. It therefore follows that when levator muscles function normally, pelvic organ support is maintained. The interaction between the muscular and connective tissue support has been explained using the analogy of a ship floating in water attached by ropes on either side to a dock. The ropes represent endopelvic fascia, water pelvic floor muscles, and the ship, pelvic organs. When the pelvic floor muscles are intact structurally and neurologically, the endopelvic fascia is under less tension (akin to the ropes when water level is maintained). When the pelvic floor muscle weakens, fascia becomes the primary support mechanism in this scenario and overtime can

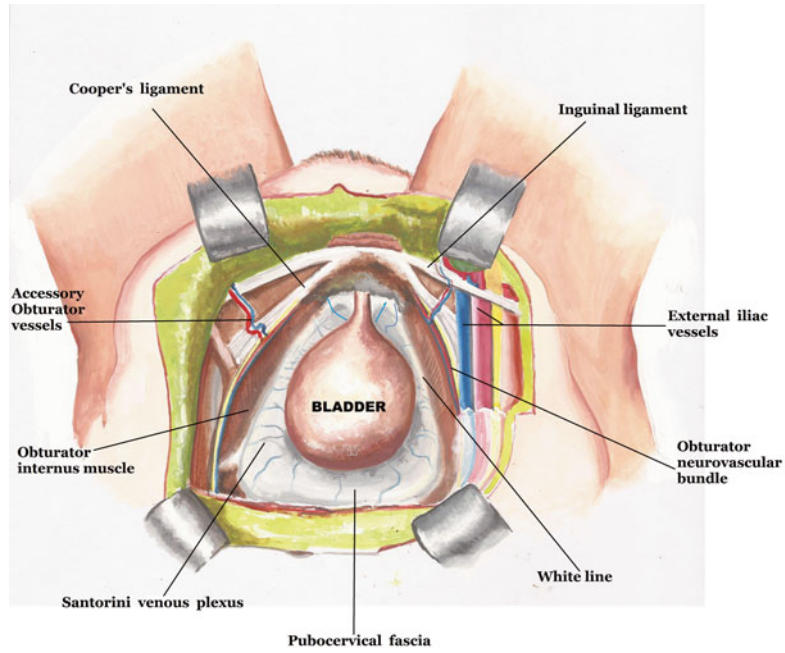

Fig. 1.12 Retropubic space – base of the space, boundaries and the vessels in area shown, as viewed from above (Reproduced with permission from Baggish [1])

weaken or break leading to prolapse or other pelvic dysfunction, like the ropes put under tension when water level is reduced.

Spaces in the Pelvis

Retropubic Space

The retropubic space also known as the space of Retzius is bounded anteriorly by symphysis pubis with the transversalis fascia inserting on the posterior surface of pubic symphysis. The urethra, urethrovesical junction, and anterior surface of bladder form the floor of retropubic space. The endopelvic connective tissue, extending from the lateral vaginal wall to pelvic side wall, partly contributes to the floor laterally (Fig. 1.12).

The space assumes importance in pelvic surgery due to some important structures, nerves and vessels being related to it. The pectineal or Cooper's ligament lies on the superior dorsal surface of pubic ramus, and a flat triangular extension of Cooper's ligament, the lacunar ligament, widens as it travels medially and joins the inguinal ligament at pubic tubercle. The obturator canal lies approximately 1–2 cm below the upper margin of Coopers' ligament.

Lateral to bladder and bladder neck, within the endopelvic connective tissue, lies the deep venous plexus (venous plexus of Santorini). The dorsal vein of clitoris drains into this venous plexus. Accessory obturator vessels from inferior epigastric or external iliac have also been noted in this space crossing the Cooper's ligament in about 20–30 % of population. The venous plexus and the vessels coursing this area can be a source of bleeding during retropubic bladder neck suspension procedures, paravaginal defect repairs, pelvic lymph node dissections and with passage of needles and trocars through this space in midurethral sling procedures and mesh placements. Instruments traversing this area should ideally follow the medial portion of the space away from the urethra close to the bony structure. While medial deviation can lead to urethral injury, lateral deviation can lead to obturator or accessory obturator vessel injury.

Presacral Space

Presacral space extends from the aortic bifurcation above to pelvic floor below, with internal iliac vessels and ureters forming the lateral boundary (Fig. 1.13). The space lies between rectum anteriorly

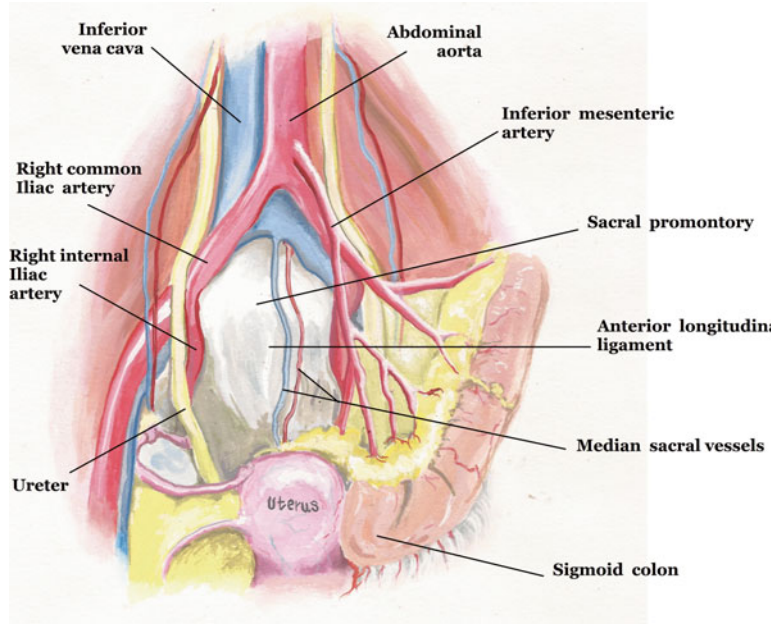

Fig. 1.13 Presacral space – boundaries and relations shown (Reproduced with permission from Baggish [1])

and the sacrum and coccyx posteriorly. There are several neural and vascular plexuses within this space. The median (middle) sacral artery arising from the abdominal aorta, descends in midline in front of the sacrum and coccyx in this space. The middle sacral artery anastomoses to the lateral sacral arteries, and sends offsets into the anterior sacral foramina. An extensive sacral venous plexus also occupies this space with anastomosis of medial and lateral sacral veins with contributions from lumbar veins. The neural plexus includes the superior hypogastric plexus with contributions from the inferior hypogastric plexus. The sacral artery and the venous plexus lie superficial to the anterior longitudinal ligament of sacrum, while the endopelvic fascia envelops the neural plexus in this area.

Awareness of this relationship is important in abdominal sacrocolpopexy (ASCP) procedure. The mesh in ASCP is secured to the anterior longitudinal ligament over S1 or S2 vertebrae. Identification of median sacral vessels intra-operatively is important to avoid vascular injury, since control of bleeding in this area can be challenging. Entry into the presacral space for ASCP and presacral neurectomies also requires knowledge of the proximity of iliac vessels, ureters and sigmoid colon in this region.

Anatomy of Lower Urinary Tract

In maintaining urinary continence, the structural anatomy of bladder and urethra play a role along with the neuronal control. The bladder epithelium is surrounded by smooth muscle layer, the detrusor muscle. The detrusor muscle has an important function in both storage and voiding. During storage, detrusor muscle is relaxed with muscle fibers stretching, to facilitate filling without significant increase in bladder pressure. This is referred to as the compliance of bladder. Conversely during voiding, detrusor muscle contracts with relaxation of the proximal urethra to initiate voiding. The position of bladder neck in relation to the pelvic diaphragm appears to be important both in maintaining continence during storage and for efficient voiding. The neuroanatomy and physiology of bladder control is detailed in the next two chapters.

The female urethra is about 4 cm long and from the bladder neck to external urethral meatus it lies on the anterior vaginal wall, being closely related to it by the endopelvic connective tissue. The urethra has a mucosal, submucosal and muscular layer (Fig. 1.14). The urethral muscle layer consists of inner longitudinal and outer circular fibers. In female urethra, the internal urethral sphincter is

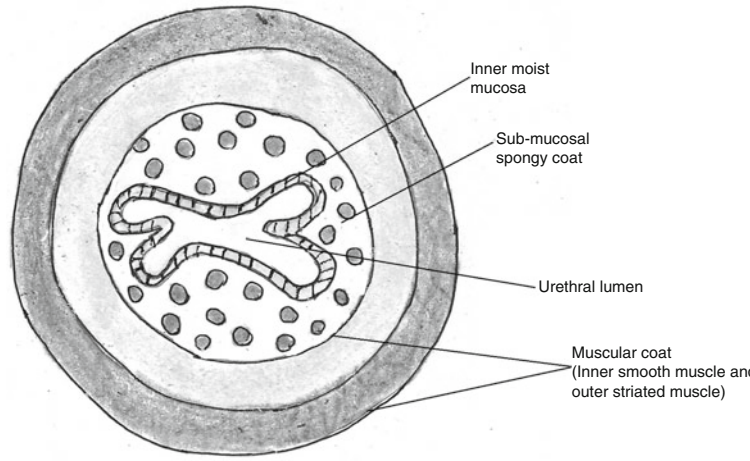

Fig. 1.14 The female urethra – inner mucosal lining keeps the urothelium moist; vascular spongy submucosal coat is important in the mucosal seal mechanism, and muscular coat helps to maintain the resting urethral closure mechanism

a functional rather than an anatomical concept, bladder neck and proximal urethra forming the internal sphincter. At the midportion of urethra, thickened striated muscle fibers form the external urethral sphincter, ventral side of which is thick and the dorsal side, thin. In addition to this, just above the perineal membrane, the urethra is surrounded by the compressor urethrae and urethrovaginal sphincter muscles, in the deep perineal space (see Fig. 1.8). The intraurethral pressure is maintained higher than vesical pressure during storage by contribution of these muscles.

The urethral mucosa is thrown into longitudinal folds, epithelium being composed of stratified squamous epithelium, which becomes transitional near bladder neck. Many small mucous glands open at the base of these folds, which helps to keep the urethra moist. These can give rise to urethral diverticula. Near the lower end of urethra, mucous glands known as Skene's glands, open into the paraurethral duct and blockage of these ducts can give rise to cysts presenting as vulval or lower vaginal cysts. The urethral submucosa is a spongy layer with rich vascular plexus, with a loose lamina propria consisting of collagen fibrils and elastic fibers arranged both circumferentially and longitudinally. The spongy submucosa keeps urethral mucosa opposed and plays a role in maintaining the urethral closure pressure, augmented further by urethral sphincters (Fig. 1.14).

The posterior wall of urethra is embedded in and supported by the endopelvic connective

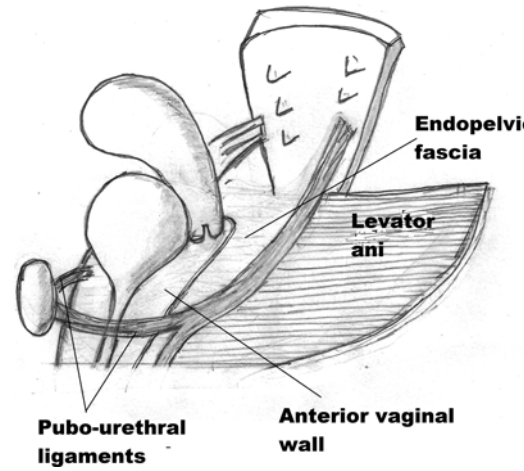

Fig. 1.15 Pubourethral ligament, urethra, and its relation to the anterior vaginal wall

tissue and anterior vagina. This supportive tissue is compared to a hammock beneath the urethra and bladder neck, acting as a backboard support. The distal wall of urethra is attached anteriorly to pubic bone by the pubourethral ligaments, extensions of pelvic fascia (Fig. 1.15). With increase in intra-abdominal pressure, urethra is compressed against the firm support of connective tissue and anterior vaginal wall. At the same time pubourethral ligaments pull the urethra towards pubis, promoting the rotational movement of urethra around pubic bone. When these connective tissue supports are intact, the urethra is supported as a

whole by its passive support to anterior vaginal wall and its active support through the urethral sphincters and ligaments.

Conclusion

Our understanding of anatomy is evolving with use of imaging techniques and use of biomechanics evaluating the anatomy and its related function. With alteration in anatomy, the function seems to be compromised and in pelvis, this can result in pelvic floor dysfunction involving urinary, bowel, sexual, and reproductive functions. Hence understanding of pelvic anatomy not only reduces the risk of complications during surgery but also ensures preservation or restoration of normal anatomy and thereby the function. Review of relevant anatomy prior to any surgical procedure can be a significant factor in reducing surgical morbidity.

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Neuroanatomy of the Female Pelvis

2

Stephanie Pickett and S. Abbas Shobeiri

The neuroanatomy of the pelvis is a complex system with many interactive pathways that is not completely understood yet. The advancement of technology in the basic sciences and imaging continues to allow improved understanding in this area. This chapter serves as an overview of basic neurology as well as a look into the innervation of the musculature related to pelvic floor support and continence mechanisms.

Basic Neurology

The basic, functional cellular unit of the nervous system is a nerve cell or neuron. The neuron is composed of a bilayered membrane that is strongly negative on the inside compared to the outside at a resting state. It is the difference in electrical potential across the membrane that creates a resting state known as a resting potential. The nerve cell creates an action potential by changing the ionic flow across its membrane when stimulated. Dendrites receive information, from other neurons. Axons conduct information encoded in action potentials. The axon of one neuron is connected to the dendrites and cell body of another neuron (Fig. 2.1). Communication from one neuron to another occurs at synapses. The neuron sending the information is the presynaptic neuron and the one receiving information is called the postsynaptic neuron. The synapse is composed of a presynaptic terminal, a synaptic cleft, and the receptive membrane (Fig. 2.2).

In order to communicate, a presynaptic neuron releases a neurotransmitter, previously stored within vesicles, into the synaptic cleft. The neurotransmitters diffuse across the synaptic cleft and bind to receptors in the postsynaptic membrane. The neurotransmitter changes the polarization of postsynaptic neuron by exciting or inhibiting the neuron. Different neurotransmitters have different effects on the neuron – excitatory (depolarizing) or inhibitory (hyperpolarizing). The most abundant neurotransmitter in the body is acetylcholine (ACh) which is also the predominant neurotransmitter in motor neurons [1]. Glutamate and ACh typically are excitatory neurotransmitters, γ and-yaminobutyric acid (GABA) and glycine are inhibitory [2]. Additional neurotransmitters include dopamine and serotonin, which have different effects on different neurons.

Additionally, there are neuroglial cells or glia that provide structural and metabolic support for neurons. In the central nervous system (CNS), the axons are typically supported by glial cells and myelinated by oligodendrocytes. In the peripheral nervous system (PNS), axons are myelinated by Schwann cells. The myelin sheath

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A. Tamilselvi, A. Rane (eds.), *Principles and Practice of Urogynaecology*,
DOI 10.1007/978-81-322-1692-6_2, © Springer India 2015

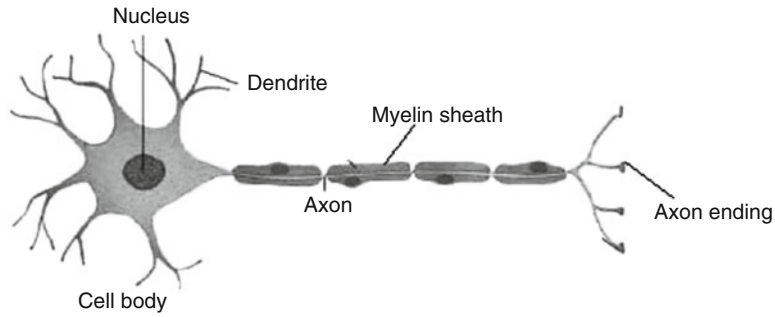

Fig. 2.1 Neuron – the basic nerve unit

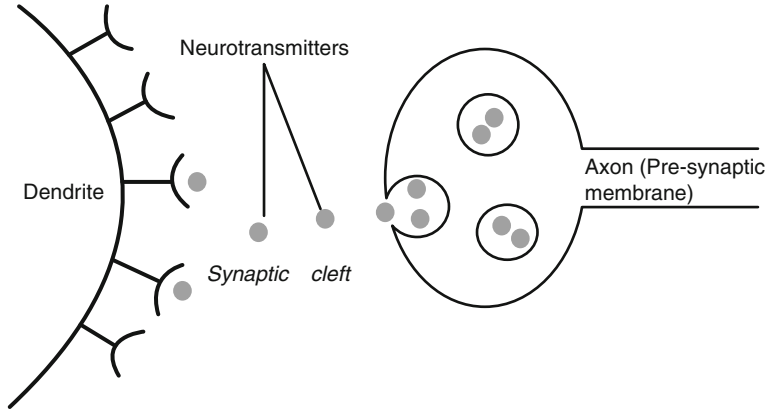

Fig. 2.2 Neuronal synapse – release of neurotransmitters at the synaptic cleft

increases the rate of action potential and leads to faster transmission. Schwann cells also are involved in organizing the formation of connective tissue sheaths around peripheral nerves during development and regeneration after damage.

The Nervous System

The CNS is composed of the spinal cord and brain. There are twelve paired cranial nerves and 31 paired spinal nerves that innervate the skeletal muscle. The spinal cord processes all the sensory information, controls body movements, and regulates visceral functions. It serves as a passageway for sensory information to the brain.

The PNS is divided into the somatic and autonomic divisions, each containing both afferent and efferent elements. The somatic division provides sensory neurons to the skin, muscles, and joints and handles voluntary control of body movements. The motor nerves control skeletal muscle movement and the sensory nerves return information to the CNS involving touch, proprioception, pain, and temperature. The autonomic division components are involved in the involuntary control system, and in the pelvis this relates to the function of the pelvic viscera.

In the somatic division, the primary motor neurons are α, β, and γ. The α neurons innervate the extrafusal muscle fibers (muscles that contract) and the γ neurons innervate the intrafusal muscle (proprioception) fibers [1]. The somatic efferent fibers originate from motor neurons in the anterior cell column of the spinal cord and exit the spinal cord at the dorsal root ganglion to form the spinal nerve [3]. In the pelvis, these fibers make up the sacral plexus, which is located on the posterior wall of the pelvis anterior to the piriformis muscle (Fig. 2.3). The nerves are formed by the ventral rami of the first through fourth sacral nerve roots (S1–S4) and the fourth

and fifth lumbar nerve roots (L4–L5). Somatic afferent fibers supply the peritoneum, urethra, vagina, and rectum. The dorsal horn of the spinal cord and the dorsal column nuclei are the places where the information from the body surface and tissue travel to reach the brain [4].

The pudendal nerve is composed of the ventral roots of the second, third, and fourth sacral nerve roots and originates from the ventral portion of the anterior horn. It carries somatic motor, general sensory, and visceral sympathetic motor nerve fibers, contributed from the sacral plexus and pelvic plexus, correspondingly [5]. The pudendal nerve exits the pelvic cavity below the piriformis through the greater sciatic foramen. It reenters the pelvic cavity by curving around the ischial spine and passing through the lesser sciatic foramen. It continues down the lateral wall of the ischiorectal fossa into Alcock's canal, which is caudal to the levator ani muscles. The pudendal nerve separates into three branches: the dorsal clitoral nerve, the inferior rectal nerve, and the perineal nerve which innervate the clitoris, the external anal sphincter, and the perineal musculature and inner perineal skin, respectively [6].

Several other somatic nerves are encountered in the pelvis during pelvic surgery. The ilioinguinal nerve departs from the first lumbar nerve root and is responsible for sensation of the skin covering the upper and medial part of the thigh, the mons pubis, and labia majora. The genitofemoral nerve originates from the first and second lumbar nerve roots and also innervates the skin of the mons pubis and labia majora.

The autonomic nervous system in the pelvis is composed of the thoracolumbar outflow which is sympathetic and the sacral outflow which is parasympathetic. The efferent fibers carry information from the CNS to the organs and afferent fibers carry sensory information away from the internal organs back to the CNS. This system is distinctive in that the preganglionic and postganglionic neurons are in the PNS [1]. The sympathetic system of the autonomic nerves controls the "fight or flight" response, while the parasympathetic system controls the "rest response." The nerves generally travel in large bundles referred to as plexuses that contain both sympathetic and parasympathetic nerves fibers (Fig. 2.3). For example, the pelvic plexus or inferior hypogastric plexus is primarily located on the pelvic sidewall but stretches out over the rectum and vaginal sidewalls to the base of the bladder. The plexus lies internal to the internal iliac arteries and travels with the arteries to the visceral organs. It is composed of both sympathetic and parasympathetic fibers from the hypogastric nerves, sacral splanchnic nerves, and the pelvic splanchnic nerves. The pelvic plexus can be further subdivided into the rectal plexus, uterovaginal plexus, and the vesical plexus.

The parasympathetic nerves in the pelvis arise from S2–S4 and are also referred to as the pelvic splanchnic nerves. Their cell bodies are in the lateral gray horn of the spinal cord and the axons extend within the pelvic nerve to the ganglia adjacent to the end organ [7]. The afferent fibers control primarily unconscious visceral motor reflex sensations, like bladder distension, rectal fullness, and sexual sensation. The spinothalamic and spinoreticular tracts receive afferent fibers from the vagina and clitoris and are linked to arousal and pleasure sensation [4]. Efferent fibers originate in the intermediolateral cell column at S2–S4 [3]. The preganglionic and postganglionic synapses primarily use ACh as the neurotransmitter.

The pelvic sympathetic nerves composed of the thoracic and lumbar autonomic nerves that originate as preganglionic neurons in the intermediolateral cell column of the spinal cord or as preganglionic splanchnic nerves [3]. The hypogastric nerve carries sympathetic nerve roots from the tenth through twelfth thoracic nerve (T10 to T12) up to the second lumbar (L1 to L2). The superior hypogastric plexus is located just below the bifurcation of the aorta and contains only visceral afferent fibers. The inferior hypogastric joins the hypogastric beneath the lateral margins of the rectouterine pouch [8]. For the sympathetic nerves in the pelvis, the preganglionic neurotransmitter is typically ACh and the postganglionic neurotransmitter is typically norepinephrine. Sensory information from the vagina, uterus, and cervix is received via sympathetic afferent fibers.

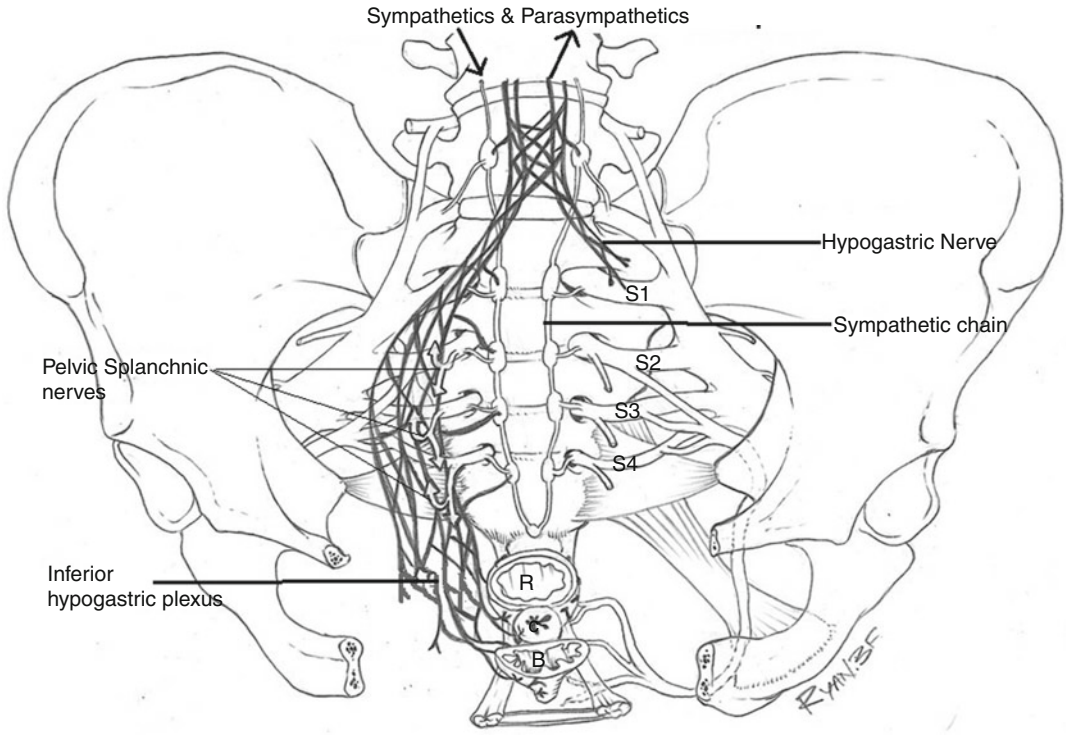

Fig. 2.3 Sympathetic and parasympathetic nerve plexus pathway into pelvis. Inferior hypogastric plexus and pelvic splanchnic nerves origin and course seen on right side. *R* rectum, *C* cervix, *B* bladder

Pelvic Musculature Innervation

The cell bodies of the somatic neurons that innervate the pelvic floor are known as Onuf's nucleus, and they extend peripherally from the gray matter to innervate striated muscle [7]. The levator ani and coccygeus muscles are innervated by the third and fourth sacral motor nerve [9]. The nerve originating from S3–S4 nerve root, referred to as the levator ani nerve, runs on the cranial surface of levator ani muscle (Fig. 2.4). It was believed that the levator ani nerve and the pudendal nerve together innervate the levator muscle, the latter running on the perineal side of the muscle and innervating the puborectalis portion. The concept of dual somatic innervations of levator ani has been questioned by recent anatomical studies [6, 10]. After exiting the sacral foramina, the levator ani nerve travels 2–3 cm medial to the ischial spine and arcus tendineus levator ani across the coccygeus, iliococcygeus, pubococcygeus, and puborectalis and innervates all the subdivisions of the muscle.

The remainder of the pelvic floor is however innervated by the pudendal nerve including parts of striated urethral sphincter and external anal sphincter through separate branches [11]. Most of the pelvic muscles are not easily accessible; hence a crude assessment of the muscle function can be obtained using reflexes such as bulbocavernosus reflex and anal wink.

Autonomic innervations of the pelvic viscera prove to be more complex than the somatic innervations. Autonomic nerves can vary greatly from patient to patient and can have a diversity of nerve roots and terminal projections.

Bladder Innervation

The micturition cycle revolves around the storage and socially appropriate elimination of urine. On a basic level, urine storage is controlled by the

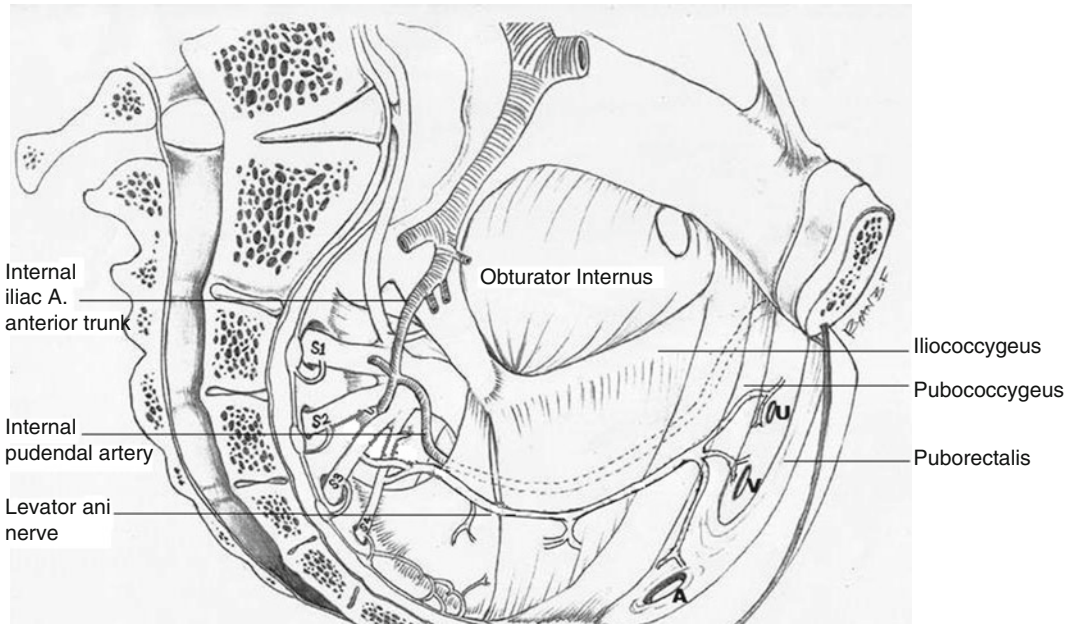

Fig. 2.4 Pelvic musculature innervation – origin and course of levator ani nerve on cranial surface of muscle. Internal pudendal artery coursing on caudal surface of levator ani (*accompanied by pudendal nerve not depicted in the diagram*). *U* urethral hiatus, *V* vaginal hiatus, *A* anal opening

sympathetic nervous system, while elimination is controlled by the parasympathetic nervous system. The bladder is composed of the smooth muscle – detrusor muscle. The bladder dome is rich in β-adrenergic receptors that are stimulated by the sympathetic component of autonomic nervous system. The bladder outlet consists of the trigone and proximal urethra and differs embryologically from bladder dome [1]. The bladder outlet is rich in α fibers. During storage, the smooth muscle remains relaxed and this allows the bladder to fill under low pressures [1]. The CNS controls the learned inhibition of detrusor contraction. Sympathetic innervation of the bladder originates in the thoracolumbar region, passes through the sympathetic trunk, and travels as the inferior hypogastric (pelvic) and sacral splanchnic nerves.

Parasympathetic innervation to the detrusor muscle is from the second, third, and fourth sacral roots. Parasympathetic stimulation leads to detrusor contraction and voiding mediated by ACh. Additionally, it is important to note that among the two main types of cholinergic receptors, nicotinic and muscarinic, the bladder has only muscarinic receptors. Three types of muscarinic receptors are found in the bladder – M1, M2, and M3. While M2 receptors are the most plentiful in the detrusor, it is the M3 receptors that predominantly mediate the detrusor contraction [7].

Urethral innervation is a bit more complex. The proximal urethra is controlled by the autonomic system, through the α receptors in this region. When urine storage is desired, the α receptors are activated by the sympathetic, and the proximal urethral sphincter is closed. This activity is controlled by fibers in the hypogastric nerve. The external urethral sphincter is composed of striated and smooth muscle layers and the sphincter is innervated by the perineal branch of the pudendal nerve and is under voluntary control. Parasympathetic stimulation leads to detrusor contractions and the relaxation of the internal sphincter urethrae to allow voiding. Somatic innervations through the pudendal nerve allow for the relaxation of the external sphincter of the bladder when convenient.

Anus Innervation

There is a paucity of data related to the autonomic neurological function of the anus. Discussion of the neurologic events leading up to the arrival of stool in the rectum is beyond the scope of this chapter. The rectum receives its sympathetic innervation from the lumbar splanchnic nerves and its parasympathetic innervation from the pelvic splanchnic nerves. The parasympathetic system senses rectal distention. Anal canal sensation is received from the inferior rectal branch of the pudendal nerve.

The external anal sphincter is innervated by the pudendal nerve. The internal anal sphincter is innervated by the parasympathetics from the first, second, and third sacral nerve root as part of the pelvic plexus. Alpha-adrenergic activity gives rise to the constant tone of the internal anal sphincter. Inferior branches from the pelvic plexus form the inferior rectal plexus and serve as efferent nerve fibers. These fibers innervate the posterolateral rectal wall and the internal anal sphincter [9]. The sympathetic nerves are from L5 and contract the internal anal sphincter to provide continence. With parasympathetic stimulation, the internal anal sphincter relaxes and allows defecation.

The rectoanal inhibitory reflex is not under CNS control and is triggered by stool in the rectum. During this reflex, there is a transient relaxation of the internal anal sphincter and an increase in tone of the external anal sphincter. This allows for sensory receptors to sample the fecal matter and assess whether the contents are solid, liquid, or gas [9]. The rectum relaxes when volume increases, in a response known as accommodation. When there is increased distention in the rectum, the sensation to defecate occurs. The urge to defecate can be suppressed via cortical control mechanisms and is initiated under cognitive control. In order to defecate, there are coordinated efforts of the pelvic floor muscles to relax and an increase in intra-abdominal pressure via Valsalva. This allows the anorectal angle to widen, shortening the anal canal [10]. After defecation is complete, a closing reflex causes a contraction of the pelvic floor muscles and reactivation of the continence mechanism.

Conclusion

A thorough understanding of the nerves supplying the pelvis is essential for the pelvic surgeon. It not only helps to avoid nerve injury during surgery but also helps to understand the pathophysiology of pelvic dysfunction.

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Physiology of Urinary Continence

3

Maya Basu and Jonathan Duckett

Introduction

Maintenance of bladder continence involves a multitude of interactions between neural pathways in the brain and spinal cord, local neural pathways in the bladder and urethra, the smooth and striated muscles of the bladder and urethra and also local mediators in the urothelium and suburothelium. Disruption at any level of this system can result in urinary incontinence. Hence, a good understanding of the normal physiology of the micturition cycle is necessary to provide insight into the mechanism of disease. The anatomical basis of continence has been discussed in the chapter on Current Concepts of Pelvic Anatomy. In this chapter, the function of the nervous system, smooth muscle and the role of urothelium and suburothelium in the maintenance of continence will be discussed.

Neuronal Control of Continence

The micturition cycle has both an autonomic control mechanism and a voluntary control mechanism which develops as a result of learned behavior with increasing age. The bladder has only two functions: storage and voiding. Therefore, many of the neuronal systems that control these functions have a binary on or off switch-like mechanism, unlike the autonomic functions of other organ systems which function with varying degrees of tone [1].

Micturition is regulated by areas in the brain and tracts in the spinal cord which function as part of both the autonomic and somatic systems, meaning that the autonomic functions are under voluntary control via the influence of higher centers. Neuronal control of micturition involves an extensive and complex network of neurons both at the central nervous system and spinal cord. The chapter on Neuroanatomy detailed the bladder innervation, receptors, and the neurotransmitters. In this chapter, the physiology in relation to the neural pathways, correlation with higher centers, and role of the detrusor along with urothelium in maintaining to urinary continence will be discussed.

Parasympathetic and sympathetic preganglionic neurons controlling bladder function are found in the intermediate gray matter of the lumbar and sacral spinal cord segments [2]. The somatic motor neurons involved in the voluntary control of external urethral sphincter are located in the ventral horn of Onuf's nucleus [3]. There is also a population of "interneurons" in the lumbosacral segments of the spinal cord, which are involved in lower urinary tract function. These send projections to the brain and participate in local segmental spinal reflexes [4, 5]. These

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A. Tamilselvi, A. Rane (eds.), *Principles and Practice of Urogynaecology*,
DOI 10.1007/978-81-322-1692-6_3,

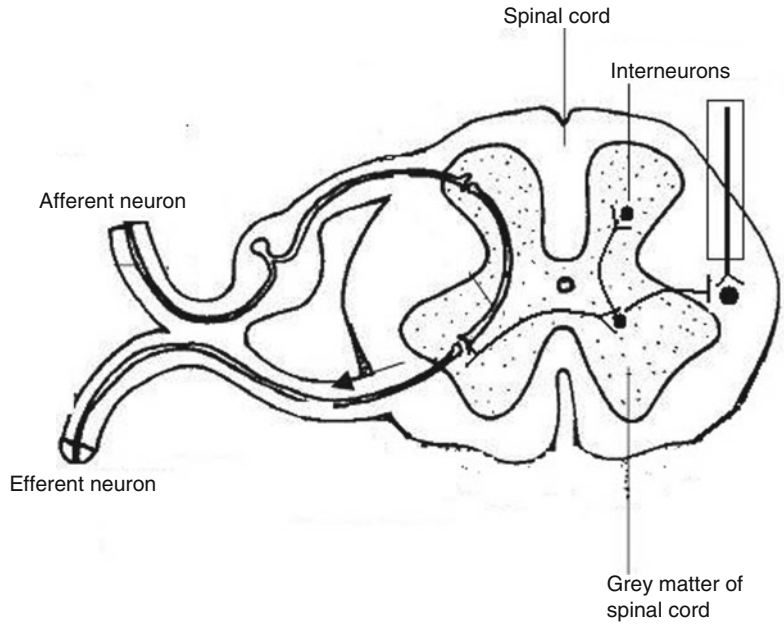

Fig. 3.1 Spinal cord neurons. The afferent and efferent neurons of the spinal cord and relation with interneurons

interneurons seem to be important sites of viscerosomatic integration since both bladder afferents transmitting sensory information from the bladder and afferent neurons from the urethra and urethral sphincter project to these regions [2] (Fig. 3.1).

Specific areas in the brain which are involved in control of micturition include the pontine micturition center, the periaqueductal gray, and also several parts of the cerebral cortex which influence voluntary control. All these areas are interconnected with each other, as well as to relevant regions in the spinal cord. The interneurons from the spinal cord are known to project to the pontine micturition center and periaqueductal gray and projections from the periaqueductal gray and hypothalamus are known to pass to the pontine micturition center [2, 6]. At the peripheral nervous system pathway, the parasympathetic outflow from the sacral segments S2, S3, and S4 involved in the voiding function travels via the pelvic nerves, while the sympathetic from T10, T11, T12, L1, and L2 segments involved in the storage phase of the bladder travels via the hypogastric nerves.

Bladder Filling

Bladder filling is associated with storage reflexes, which are organized primarily at the spinal cord level. Filling is associated with inhibition of parasympathetic stimulation to the detrusor muscle, plus activation of the smooth and striated muscles of the urethral sphincter. This coordinated activity is thought to be mediated by urethral reflexes collectively known as the "guarding reflex," which is activated by afferent feedback from bladder stretch receptors conveyed via the pelvic nerves [2]. Studies in feline models have demonstrated input of afferent neurons into the periaqueductal gray, which in turn provides inputs into the pontine micturition center [7]. The periaqueductal gray is likely to act as a relay between higher brain centers and the pontine micturition center, allowing modulation of the excitatory input into the pontine micturition center. Functional brain imaging studies in humans have demonstrated that activity in the periaqueductal gray increases during bladder filling and when bladder afferent activity increases [8, 9]. During bladder filling, the activity of parasympathetic efferent neurons to the bladder is downregulated, i.e., turned off [10]. In

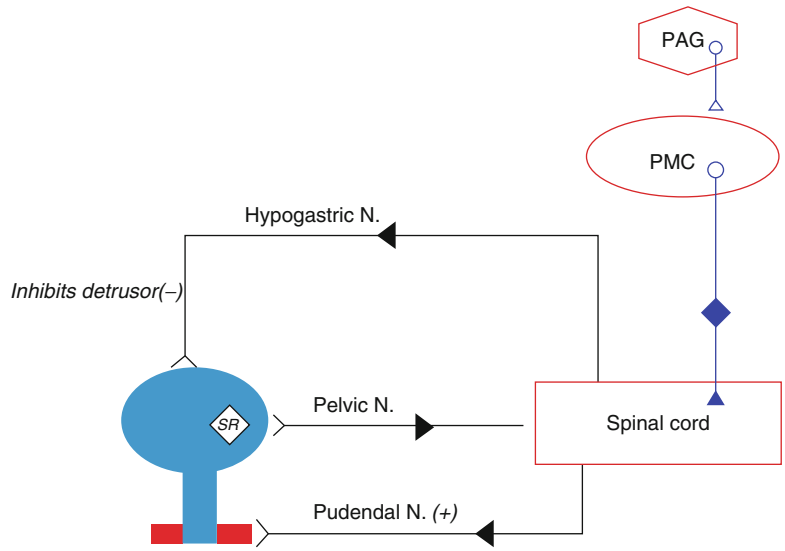

Fig. 3.2 Bladder storage reflexes – simplified schematic representation. Bladder filling with stimulation of bladder stretch receptors (*SR*) associated with inhibition of detrusor and activation of urethral outlet (*PAG* periaqueductal gray, *PMC* pontine micturition center)

short, with bladder filling sympathetic activity is increased, parasympathetic activity is inhibited, and the somatic neurons are activated (Fig. 3.2). At a critical level of distension, afferents from tension receptors in the bladder stimulate the parasympathetic pathway to be "switched on" to maximal activity [11]. Despite these involuntary changes in activity, activation of micturition is under strict voluntary control.

Voiding

The periaqueductal gray (PAG) integrates the voiding process from bladder afferents and spinal pathways to the pontine micturition center and frontal cortex. PAG is involved in the registration and conscious awareness of bladder filling sensations and in the manipulation of firing of the voiding reflex at the appropriate time [6]. The PAG also receives projections from higher brain centers and modulates primary input into the pontine micturition center, allowing excitation only when voiding is consciously desired.

Meticulous PET functional brain imaging studies have confirmed the activation of a well-defined area in the dorsal pons, which becomes active during the initiation of voluntary voiding [8]. Clinical and functional imaging studies suggest that in humans, the frontal lobes play an important part in assessing the appropriateness of micturition [2]. The prefrontal cortex is thought to be an area central to planning complex behaviors and appropriate social behavior, as well as response selection mechanisms [12]. This region is directly connected to the periaqueductal gray, which suggests that it may be responsible for inhibition of micturition until an individual is in a situation where voiding is wanted and socially appropriate [13] (Fig. 3.3).

There are certain prerequisites which are necessary in order for a void to take place, such as an appropriate degree of bladder distension (i.e., afferent signals from the bladder of sufficient strength to trigger output from the periaqueductal gray) and a socially appropriate and safe situation in which to void (influenced by signaling from the cerebral cortex and hypothalamus) [14]. Once these prerequisites are met, neuronal input from the periaqueductal gray leads to excitation of the pontine micturition center. Voluntary voiding seems to be associated with interruption of the tonic suppression of the PAG input into the pontine micturition center. This in turn activates descending pathways leading to urethral relaxation (via a

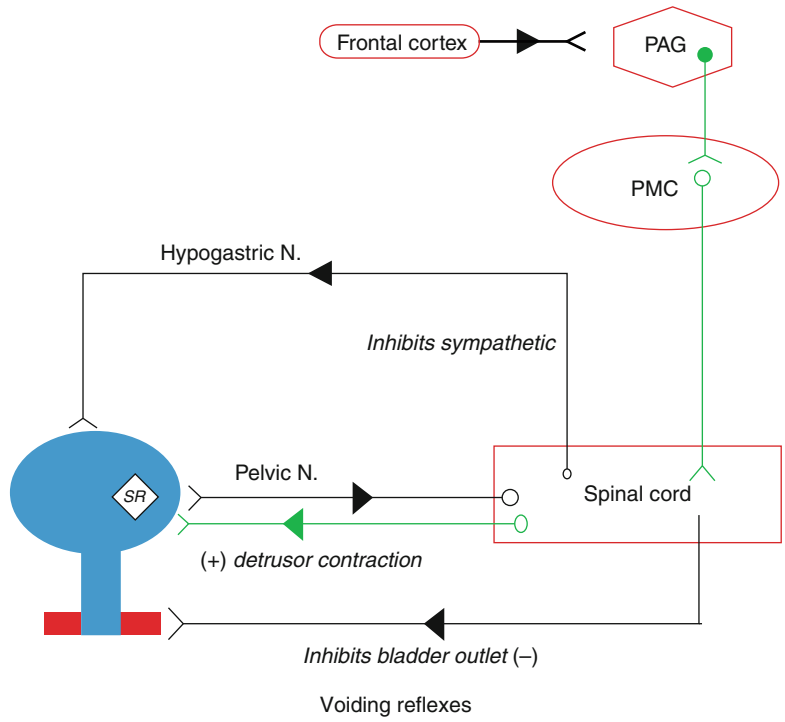

Fig. 3.3 Voiding reflexes – simplified schematic representation. After appropriate bladder distension, inhibition of bladder outlet and the sympathetic pathway with activation of parasympathetic and detrusor contraction

parasympathetic-mediated release of nitric oxide, which decreases adrenergic and somatic cholinergic and excitatory signaling to the urethra), followed by activation of parasympathetic outflow from the sacral nerves. Functional brain imaging studies have indicated that voiding is associated with activation of a variety of structures including the prefrontal cortex, the insula, the hypothalamus, and the periaqueductal gray [15, 16].

Role of Detrusor Muscle

The detrusor smooth muscle is comprised of small spindle-shaped cells with a central nucleus [17]. Although isolated detrusor strips can show spontaneous mechanical activity, spontaneous fused tetanic contractions (such as those seen in the uterus) are generally not seen in normal bladders. These, together with experiments which have measured the tissue impedance of detrusor muscle, suggest that the detrusor is characterized by poor electrical coupling between the smooth muscle cells [18]. This feature is likely to be necessary in order to prevent synchronous activation of detrusor smooth muscle cells during bladder filling [19]. Indeed, ultrastructural studies of human detrusor muscle have shown that although coupling of smooth muscle cells does occur in asymptomatic volunteers, it is upregulated in patients with urgency symptoms to form limited functional syncytia, suggesting increased intracellular signaling in the detrusor smooth muscle of individuals with detrusor overactivity (DO) [20–22]. Other histological studies have additionally found patchy denervation of the detrusor muscle together with potassium supersensitivity in women with DO compared to healthy controls [23]. There is also evidence that calcium handling in detrusor smooth muscle cells in those with DO is altered, leading to a higher level of intracellular calcium [24, 25].

Localized distortions of the bladder wall due to autonomous activity may contribute to

increased bladder sensations and urgency in women with overactive bladder by stimulating sensitive tension receptors in the detrusor smooth muscle. These localized distortions are known as "micromotions." Physiological experiments in women with increased bladder sensation have shown a higher prevalence of micromotions in the detrusor smooth muscle compared to women with normal bladder sensation [26]. Micromotions can be multifocal with separate areas of the bladder wall contracting independently as modules [27]. This localized activity is thought to stimulate afferent activity in ascending neurons and may therefore lead to urgency, frequency, and reduced functional bladder capacity. A population of cells in the detrusor known as interstitial cells (or myofibroblasts) have been suggested to act as "pacemaker" in the bladder, with respect to the regulation of this spontaneous activity [28]. Interstitial cells in the detrusor are known to be spontaneously active and also stimulate calcium influx in response to muscarinic receptor activation. Interstitial cells are also found in the suburothelium, where they are closely related to C-fiber nerve endings in the submucosal layer of the bladder. It is likely that bladder distension during filling leads to release of cytokines such as adenosine triphosphate (ATP) and acetylcholine (Ach), which activate afferent neurons in the suburothelium via the action of the suburothelial interstitial cells [19].

Role of the Urothelium

The central nervous system reflexes have an overall controlling influence on bladder function. In addition, the afferent nerve output, local mediators and cells found within the urothelium and suburothelium also have a profound influence on the bladder.

Afferent neurons returning from the bladder travel in the pudendal, pelvic, and hypogastric nerves and are comprised of thin myelinated Aδ fibers and nonmyelinated C fibers. The primary afferent neurons of the pelvic and pudendal nerves are contained in the sacral dorsal root ganglia, with afferent innervation of the hypogastric nerves arising in the lumbar dorsal root ganglia. The central axons of the dorsal root ganglia in turn convey sensory information from the lower urinary tract to second order neurons within the spinal cord [29, 30].

The Aδ fiber endings are in the smooth muscle and are the most sensitive nerve endings in the bladder, being activated by increasing bladder wall tension secondary to intravesical filling. The C fibers have their endings in the lamina propria and urothelium and are only activated at greater degrees of bladder distension than Aδ endings (Fig. 3.4). C fibers are activated also by neurotransmitters and mediators in pathological situations such as inflammation and high osmolality [31]. Thus, C fibers may be of more importance in women with pathological urgency and OAB. C-fiber receptors such as the transient receptor potential vanilloid 1 (TRPV1) and P2X3 (ATP receptor) are modulated by mediators such as nerve growth factor (NGF) and are believed to be of importance in afferent activation in the bladder in women with sensory urgency, although there is conflicting evidence on their role in women with DO [32, 33].

The source of the initiating stimulus to the afferent fibers is thought to be the urothelium [34]. The cells comprising the urothelium are in close proximity to the afferent nerves and activity in this layer is likely to be intimately involved in signal transmission and modulation, apart from its established barrier function. The urothelium is known to express a variety of ion channels and receptors which respond to chemical as well as mechanical stimuli, by releasing mediators which then stimulate afferent nerve fibers. A variety of urothelial mediators, thought to be of importance in afferent function and dysfunction, have been identified. Often the best way of understanding the normal role of these mediators is to understand their role in patients with abnormal bladder function. Patients with OAB are known to have higher levels of ATP, NGF, and prostaglandin E2 (PGE2), when compared to normal controls [35–37]. Some of the more important and better understood signaling pathways are discussed below.

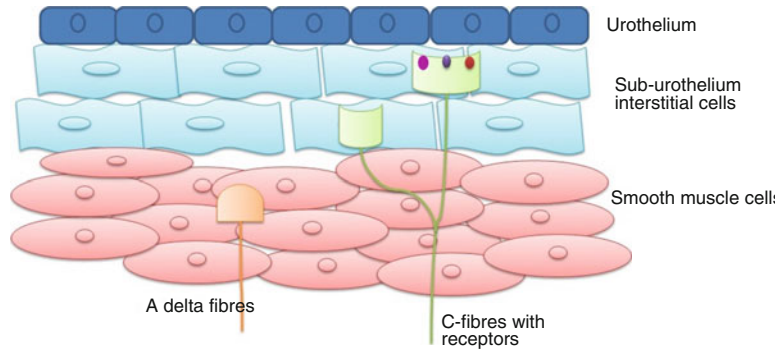

Fig. 3.4 Schematic representation of bladder urothelium, interstitial and smooth muscle cells. Myelinated A delta fibres and unmyelinated C-fibre endings depicted

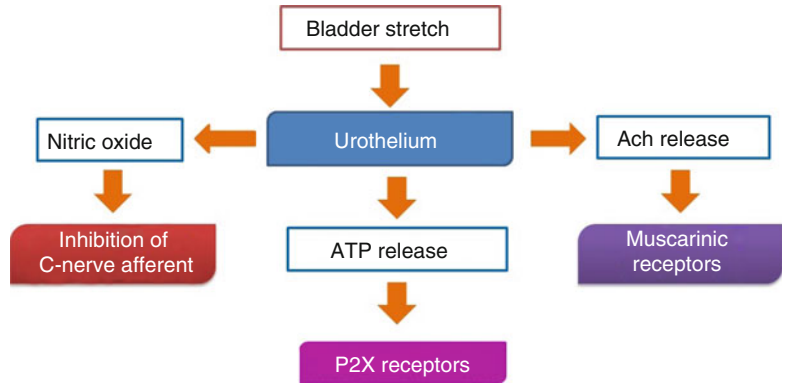

Fig. 3.5 Signaling in urothelial pathways

Purinergic Pathways and ATP

ATP is known to facilitate the non-adrenergic, non-cholinergic component of bladder contraction. It acts on the P2X family of receptors, participating in efferent bladder signaling via the P2X1 receptor on detrusor smooth muscle membranes and in afferent signaling via the P2X2/3 receptors expressed on afferent neurons [38, 39]. Studies of P2X3 in knockout mice reveal detrusor hyporeflexia with a decreased frequency of voiding and an increased bladder capacity [40]. Patients with DO have been found to have substantially elevated levels of P2X3 receptors [41] and ATP [42]. Bladder distension as well as various physiological insults to the bladder stimulates ATP release [43], and the presence of ATP-degrading enzymes in rats is seen to prevent activation of P2X receptors and the manifestation of OAB symptoms [44]. The same study showed that intravesical introduction of ATP conversely induced DO. A study of human subjects undergoing urodynamic studies showed an inverse correlation between voided volume and levels of ATP in voided urodynamic fluid in both individuals with DO and normal controls and an inverse correlation between voided fluid ATP levels and first desire to void in those with DO, but not in the control group [45]. This suggests that abnormal purinergic signaling may be implicated in modulating early filling sensations in pathological states (Fig. 3.5).

Acetylcholine and Muscarinic Receptors

Acetylcholine (ACh) has been classically associated with motor function in the bladder, promoting detrusor contraction. In addition, there is a nonneuronal release of ACh from the urothelium which leads to modulation of sensory bladder function. This concept is reinforced by studies showing that antimuscarinic drugs exhibit activity during the bladder storage phase when effer-

ent nerve pathways are silent [46]. The majority of experimental work proving the nonneuronal release of ACh has been carried out in animal models and has shown a distinct storage and release mechanism from the neuronal cells [47]. Yoshida and colleagues carried out a study using strips of bladder tissue from human subjects, demonstrating that removal of the urothelium substantially decreases release of ACh, as well as an age-related and stretch-induced increase in non-nerve-evoked ACh release [48]. Further studies using antimuscarinic drugs have suggested that blocking of muscarinic receptors reduces afferent stimulation. Studies examining the effect of oxybutynin on single bladder afferent units from rats have shown a desensitizing effect on C-fiber afferents [49, 50]. Similar results have been seen after administration of the M3-selective antimuscarinic darifenacin [51]. A subsequent study by Matsumoto and colleagues [52] suggested that it is M2 receptors that mediate cholinergic stimulation of afferent activity. An animal study using different dosing protocols for antimuscarinic drugs following carbachol-induced DO showed an increase in bladder capacity and inter-contraction interval, with no accompanying decrease in bladder contractility, suggesting a local inhibitory effect for antimuscarinic drugs. Collectively, the evidence suggests a role for muscarinic pathways in the modulation of afferent signaling, although the exact interaction is yet to be determined.

Nitric Oxide Pathways

Nitric oxide (NO) is synthesized within the urothelium and appears to act as an inhibitory transmitter within afferent pathways [53]. Intravesical administration of an NO substrate and an NO synthase inhibitor leads to inhibition and stimulation of both Aδ and C fibers, respectively. In support of this, in vivo studies have shown a decrease in bladder hyperactivity with administration of PDE5 inhibitors, which potentiate the NO/cyclic GMP pathway [54]. Intravesical administration of oxyhemoglobin, (which decreases NO concentration) to healthy rats leads to an increase in bladder pressure and a decrease in bladder capacity, with the converse being observed with administration of an NO substrate [55].

Adrenergic Pathways

The bladder storage phase is predominantly governed by the parasympathetic nervous system, which inhibits parasympathetic nerve-mediated detrusor contractions. Noradrenaline release induces detrusor relaxation via adrenergic β_3 receptors [56] and β_3 receptors are highly expressed in the bladder, particularly in the detrusor muscle and urothelium [57]. Administration of β_3 receptor agonists to detrusor muscle strips leads to smooth muscle relaxation [58]. However, possible afferent effects are also suggested by studies showing that β_3 agonists have an inhibitory effect on afferent nerve firing [59], as well as inducing urothelial NO release [60].

Conclusion

The bladder is unique in that it has a binary mode of action – storage or voiding. Although the regulation of bladder function occurs via a complex network of central and local neural control mechanisms, together with modulation from the urothelium and suburothelium, the actual neural control operates like a simple switching circuit – which in an adult is under the influence of voluntary control from higher brain centers.

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Part II

Lower Urinary Tract Dysfunction

Evaluation of Urinary Incontinence

4

Pranathi Reddy

Urinary incontinence is defined as the complaint of involuntary loss of urine [1], which could be urethral or extra-urethral loss. It is a common problem and affects women of all age groups with a significant effect on their quality of life. The prevalence of urinary incontinence increases with age. Among young adults and middle aged, it is reported in 25 % of women and in those aged 40 and over, the mean prevalence is 34 % [2]. The common types of urinary incontinence in women are stress urinary incontinence (SUI), urgency urinary incontinence (UUI), and mixed urinary incontinence. Urinary incontinence resulting in extra-urethral loss, a feature of fistula, is more of gynecological etiology now rather than obstetric in most parts of the world.

The evaluation of any incontinent patient includes a thorough history, focused physical examination, appropriate investigations including laboratory tests and possibly urodynamic testing.

History Taking

History taking is the cornerstone of urinary incontinence assessment and in combination with physical examination allows categorization of patients into stress urgency, or mixed urinary incontinence [3]. The purpose of history taking is to determine the type of urinary incontinence (UI) that is bothersome to the patient and identify any precipitating factors [1].

Questions should be asked to assess symptoms related to bladder storage and voiding functions. Storage symptoms include frequency, nocturia, urgency with or without incontinence, and leak on cough, sneeze, effort, or physical exertion. Voiding symptoms include hesitancy, slow stream, intermittency, straining to void, spraying of urinary stream, and feeling of incomplete emptying [4]. Complaint of continuous leakage and leak without sensation are suggestive of extra-urethral loss and further assessment should be directed towards it. Symptoms such as recurrent urinary tract infection and hematuria, especially in women over the age of 40, need evaluation of both lower and upper urinary tract to rule out benign and malignant pathology.

The effect of incontinence on quality of life can be assessed by eliciting the frequency of occurrence, severity of leak, need for pads, and limitations of daily activities [4]. A more objective tool would be to use incontinence-specific quality-of-life scales or validated questionnaires to evaluate the severity and relative contribution of UUI and SUI symptoms and response to their therapies. The test–retest reliability of ICIQ, BFLUTS, I-QOL, SUIQQ, UISS, SEAPI-QMM, ISI, and KHQ questionnaires is good. For other scores, the evidence is weak or absent [3].

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A. Tamilselvi, A. Rane (eds.), *Principles and Practice of Urogynaecology*,
DOI 10.1007/978-81-322-1692-6_4, © Springer India 2015

Pelvic floor dysfunction tends to coexist, and patients with UI should be asked about symptoms of pelvic prolapse, such as a sensation of vaginal fullness or pressure or the observation of a bulge in vagina [5]. Bowel problems such as constipation or fecal urgency and/or incontinence can be associated with UI. Bladder pain, urethral pain, and vaginal pain can be associated symptoms in patients with UI.

During clinical assessment, it is important to identify relevant predisposing and precipitating factors and other conditions that might require referral for additional investigations and treatment. A thorough medical and neurologic history should aim to identify conditions, such as diabetes, chronic obstructive airway diseases, connective tissue disorders, and neurologic disorders, such as multiple sclerosis, spinal cord injury, myelodysplasia, stroke, Parkinson's disease, and cardiorespiratory and renal problems which can predispose to UI [4]. The patient should be queried specifically about these conditions as they are known to affect bladder and sphincter function. A list of the patient's current medications and over-the-counter medications should be obtained. Agents that can affect lower urinary tract function include diuretics, caffeine, alcohol, narcotic analgesics, anticholinergic drugs, antihistamines, psychotropic drugs, alpha-adrenergic blockers, alpha-adrenergic agonists, and calcium channel blockers (Table 4.1).

Obstetric history, in particular the parity, mode of delivery, instrumental deliveries, and birth weight, can identify some of the risk factors. Gynecological history such as presence of pelvic mass – fibroids or ovarian cysts – and the menopausal status are relevant. Past surgical history of complex pelvic surgeries, surgery for UI or pelvic organ prolapse, low spinal surgeries, or rectal surgeries can either be inciting or precipitating factors in UI.

The history should be able to categorize the type of incontinence (e.g., stress, urgency, or mixed); however one needs to remember that occasionally urgency incontinence may be triggered by activities such as coughing and can mimic stress incontinence. Mixed incontinence is very common and in these cases further management should be directed towards the predominant symptom [5].

Table 4.1 Example of medications affecting urinary function

Medication	Effect
Alpha agonist	Urinary retention
Alpha blockers	Urinary leak
Alcohol	Frequency, polyuria
Anticholinergics	Urinary retention/functional incontinence
Caffeine	Frequency, urgency, polyuria
Calcium channel blockers	Urinary retention, nocturnal diuresis
Diuretics	Frequency, polyuria
Narcotic analgesics	Urinary retention/functional incontinence

Physical Examination

The primary purpose of physical examination is to exclude confounding or contributing factors to the incontinence or its management. A complete physical examination should be performed, with emphasis on neurologic assessment and on abdominal, pelvic, and rectal examinations. The general examination should include height and weight, which will allow for objective assessment of body mass index, as obesity is an established risk factor of UI [5]. Assessment of the gait and mobility of the patient can help to rule out a functional etiology for the UI.

The abdominal examination will allow evaluation of scars and palpation for possible distended bladder and pelvic mass. The neurologic examination concentrates on the sacral segments predominantly along with lower lumbar segments. This includes, but not limited to, testing of lower limb tone, sensation, reflexes, perineal sensation, and reflexes such as bulbocavernosus or anal reflex.

Local examination concentrates on demonstration of urinary leak, presence of prolapse, atrophic vaginal changes and pelvic mass. Evidence of pelvic organ prolapse (POP) beyond the hymen is consistent with complicated SUI because the prolapse can produce a relative obstruction of urethra that can impair bladder emptying.

Therefore, it is recommended that all pelvic compartments (anterior, posterior, and apical) be assessed individually. When POP is reduced with a nonobstructing pessary or large cotton swabs, SUI may become apparent or worsen [6].

Stress urinary incontinence should be objectively demonstrated before any anti-incontinence surgery is performed. Visualization of fluid loss from the urethra simultaneous with a cough is diagnostic of SUI. Delayed fluid loss is considered a negative cough stress test result and suggests cough-induced detrusor overactivity [5]. The cough stress test can be performed with the patient in the supine position during the physical examination. However, if urine leakage is not observed, the cough stress test needs to be repeated with the patient standing and with a full bladder (or a minimum bladder volume of 300 mL) to maximize test sensitivity [7].

Support to the bladder neck is assessed by evaluating the mobility of the urethrovesical junction. Urethral hypermobility is defined as a 30° or greater displacement of urethra from the horizontal (measured with a cotton tip swab in urethra) with the patient in supine lithotomy position and straining – referred to as the "Q-tip test." This test is not recommended in the routine evaluation of patients with UI. Other methods of evaluating urethral mobility include measurement of point Aa of the POP Quantification system, visualization (inaccurate method), ultrasonography, and lateral cystourethrogram. Women with stress incontinence who have good urethral mobility have a lesser chance for failure of mid-urethral sling procedures. In women with SUI without urethral hypermobility, where leak can be due to intrinsic sphincter deficiency (ISD), bulking agents were considered to be a more appropriate surgical option [8]. This notion is however being increasingly questioned with use of mid-urethral slings, where cure rate of 78 % is quoted with tension-free vaginal tape (TVT) in patients with ISD [9].

Digital assessment of the pelvic floor muscle contraction and grading it using modified Oxford grading scale (Table 4.2) can be helpful in discussing management options such as pelvic floor exercises for SUI.

Table 4.2 Modified Oxford grading scale for pelvic floor muscles

Grade	Definition
0	No contraction
1	Flicker of contraction
2	Weak muscle activity
3	Moderate muscle contraction
4	Good muscle contraction
5	Strong muscle contraction

Investigations

Bladder Diaries

A reliable method of documenting the frequency of incontinent episodes is essential for outcome assessment in both clinical practice and research studies. Bladder diaries, completed prospectively by the patient, have been widely used for this purpose [10]. Bladder diaries are used to document each cycle of filling and voiding over a number of days and can provide information about urinary frequency, urgency, diurnal and nocturnal cycles, functional bladder capacity, and total urine output. They also record leakage episodes, fluid intake, and pad changes and give an indication of the severity of the problem. They may also be used for monitoring the effects of treatment. Encouraging women to complete a minimum of 3 days of diary, covering variations in their usual activities, such as both working and leisure days is useful [3].

In addition, use of disease-specific questionnaires assessing quality of life is invaluable in clinical evaluation.

Urinalysis

Urinalysis determines any evidence of hematuria, pyuria, glycosuria, or proteinuria. Urinary tract infections can be identified using urinalysis and treated before initiating further investigation or therapeutic intervention for UI [5]. If urinalysis tests positive for both leucocytes and nitrites, a midstream urine specimen is sent for culture and analysis of antibiotic sensitivities. If symptomatic,

these women can be prescribed an appropriate course of antibiotic treatment pending culture results. If women do not have symptoms of UTI but their urine tests positive for both leucocytes and nitrites, do not offer antibiotics without the results of midstream urine culture [3].

A urine specimen is sent for cytology if there is hematuria or irritative voiding symptoms to rule out a malignancy. Microscopic hematuria, is clinically significant when three to five red blood cells per high-power field are visible and warrants further investigation by cystoscopy and imaging.

Assessment of Residual Urine

Presence of an elevated post-void residual urine volume can indicate a bladder-emptying abnormality or incontinence associated with chronic urinary retention (previously referred to as overflow incontinence). It should be assessed using a bladder scan in preference to catheterization on the grounds of acceptability and lower incidence of adverse events [3]. A PVR less than 50 mL is not usually significant; one between 50 and 200 mL can be equivocal, and a PVR greater than 200 mL should be referred for specialist evaluation [4]. A single number may not provide the answer to the etiology of a patient's incontinence; the PVR value needs to be taken into consideration as part of the entire workup. An elevated post-void residual urine volume in the absence of POP is uncommon and should trigger an evaluation of the bladder-emptying mechanism, usually with a pressure–flow urodynamic study [5].

Imaging Studies

Imaging techniques that can be used in the assessment of urinary tract include ultrasonography, X-ray, computed tomography (CT), and magnetic resonance imaging (MRI). Ultrasound has particular value in measuring post-void residual volume. Confirmation of alternative pelvic pathology by ultrasound would be an indication for referral to an appropriate specialist. Studies have focused on measurement of bladder wall thickness by transvaginal, transperineal or abdominal ultrasound for the diagnosis of detrusor overactivity (DO) [11]. There are reports of significantly greater bladder wall thickness in women with DO than with any other diagnosis and that bladder wall thickness of more than 5 mm had sensitivity of 37 % and specificity of 79 %, for diagnosing DO [12]. Further studies are required to clarify the role of ultrasound in the assessment of overactive bladder.

There is a lack of evidence regarding use of MRI or CT scanning in assessment of women with UI. Intravenous pyelogram (IVP) has been employed in the evaluation of upper urinary tract such as detection of structural abnormalities and strictures and in assessing unexplained hematuria. With increasing use of CT Urogram, use of IVP even for these indications is becoming obsolete.

Role of Urodynamics and Cystoscopy

In patients presenting with UI, at the end of initial assessment by history, physical examination, and above investigations, it is possible to categorize those with SUI, UUI, OAB, and mixed UI. Patients needing specialist referrals and further evaluation can also be identified at this stage, e.g., those with fistulae, neurogenic etiology, or upper tract pathology. In the former group with SUI, UUI, and mixed UI, conservative management in the form of lifestyle intervention, pelvic floor muscle training, bladder retraining, and pharmacotherapy can be commenced without need for further evaluation. Hence, urodynamics and cystoscopy are not essential in the initial evaluation of patients presenting with uncomplicated UI.

Urodynamic Testing

Practical aspects of urodynamic testing are detailed in the next chapter on urodynamics. The principles underlying the procedure are detailed below. The term "urodynamics" encompasses a number of varied physiological tests of bladder and urethral function, which aim to demonstrate an underlying abnormality of storage or voiding.

Uroflowmetry is a noninvasive measurement of the rate of urine flow over time. It can also be used

to assess bladder emptying but cannot be used alone to diagnose the cause of an abnormality [13]. Cystometry, measurement of intravesical pressure, can be carried out through a single recording channel (simple cystometry) or more commonly, by multichannel cystometry, which involves synchronous measurement of both bladder and intra-abdominal pressures by means of catheters inserted into bladder and rectum respectively.

Videourodynamics involves synchronous radiographic screening of bladder with multichannel cystometry and is so called because originally the information was recorded to a videotape. Ambulatory urodynamics involves multichannel cystometry carried out with physiological bladder filling rates and using portable recording devices, which enables the woman to remain ambulant during test.

There are also numerous tests of urethral function, including urethral pressure profilometry and leak point pressure measurement. These are used to derive values that reflect the ability of urethra to resist urine flow, expressed most commonly as maximum urethral closure pressure (MUCP) or as abdominal, cough, or Valsalva leak point pressures (ALPP, CLPP, VLPP).

Urodynamics should be used selectively in women with UI to answer specific functional questions. After undertaking a detailed clinical history and examination, multichannel filling and voiding cystometry are indicated in women who have [3]:

- Symptoms suggestive of voiding dysfunction
- OAB symptoms refractory to pharmacotherapy
- Symptoms of OAB with uncertain etiology or a clinical suspicion of neurogenic detrusor overactivity
- Prior to surgical intervention in women with SUI
- Urinary symptoms following anti-incontinence procedure

The use of urodynamics has been clarified in one specific population, that of women with pure SUI symptoms. In that population, it is considered safe to forego urodynamics preoperatively [14]. However, diagnosis of pure SUI should be definitive based on history and examination, as most patients with UI present with more than one symptom. Most surgeons including the author however do not undertake surgical intervention without urodynamic evaluation even in this group.

Multichannel cystometry, when it reproduces the woman's symptoms, may reveal the underlying pathophysiological explanation of incontinence. Ambulatory monitoring demonstrates functional abnormalities more often than multichannel cystometry, but the significance of this is unclear [15]. Urodynamics is an invasive testing and has to be used judiciously to provide reliable information and to be cost-effective.

Cystoscopy

Cystoscopy involves direct visualization of bladder and urethral lumen using either a rigid or flexible cystoscope. Examination is used to identify areas of inflammation (such as interstitial cystitis), tumors, stones, foreign body and diverticula, all of which are findings that will require management within a different clinical pathway. Cystoscopy should not be used in the initial assessment of women with UI alone. Cystoscopy may be of value in women with pain or recurrent UTI, following previous pelvic surgery, or where fistula is suspected. Its role in recurrent stress UI without these additional features is less clear [3].

The indications for cystoscopy include:

- Gross or microscopic hematuria, in the absence of an infection (sterile hematuria)
- Bladder pain
- Evaluation of urologic fistula
- Evaluation of bladder or urethral diverticula
- In patients with obstructive voiding symptoms
- In patients with recurrent UTI

Cystoscopy is contraindicated in the presence of an acute cystitis and in patients with severe coagulopathy. A flexible cystoscope is preferable to the rigid for diagnostic purpose, as it can obviate the need for anesthesia.

Conclusion

In most cases, evaluation of a patient with urinary incontinence is straightforward with a thorough history and physical examination directing the appropriate investigations. The initial assessment helps to appraise the effect of UI in day-to-day activities, understand the expectations of patient and in planning appropriate treatment.

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Urodynamics: The Practical Aspects

5

Kalaivani Ramalingam

Urodynamics is an investigative modality in patients with bladder dysfunction. The test evaluates the lower urinary tract function, that of bladder and urethra. Performing a urodynamic evaluation not only requires knowledge of the procedure but also about the pretest requirements and working of the equipment used. Being an invasive investigation, the indications for this test should be clearly defined by the clinician for each patient.

In this chapter, the different parts of the test, namely the uroflowmetry, filling cystometry, urethral function tests, and voiding cystometry are described. Urodynamic traces are provided for interpretation and to avoid pitfalls in diagnosis. Modifications of standard urodynamic evaluation such as video cystometry and ambulatory urodynamics are discussed briefly. Controversies regarding role of urodynamics in the assessment of urinary problems are debated along with the mention of research implications.

The need for a reliable test to analyze urinary symptoms in women has long been perceived essential, as symptoms are often variable and difficult to express and reproduce. The old adage by Bates, "bladder is an unreliable witness," poignantly explains this dilemma. Urodynamics focuses on bladder filling and voiding and defines bladder storage problems of stress and urgency incontinence and the severity of voiding dysfunction. Urodynamics is a good diagnostic tool if used judiciously and interpreted cautiously. It adds valuable information that impacts future management. It helps to study the capacity, sensation, compliance and contractility of the bladder along with urethral function.

Indications

Urodynamic study is considered after a detailed history of patient symptoms, examination and review of the frequency-volume charts if possible. It is commonly considered in the following conditions:

1. Preoperative – before considering surgery for stress incontinence
2. Failed medical treatment for overactive bladder
3. Failure of prior surgical procedure
4. Combination of different urinary symptoms
5. Coexistent neurological disorders
6. Urethral obstruction or voiding dysfunction
7. Medicolegal concerns

Pretest Preparation

As soon as the clinician perceives the need for a urodynamic study, a detailed explanation of the test should be given along with written information

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A. Tamilselvi, A. Rane (eds.), *Principles and Practice of Urogynaecology*,
DOI 10.1007/978-81-322-1692-6_5, © Springer India 2015

Name ---------- Date ----------

	Day 1			Day 2			Day 3		
	In	Out	Wet	In	Out	Wet	In	Out	Wet
7 am							150 ml		
8 am	100 ml	300 ml		150 ml	150 ml			200 ml	
9 am									
10 am					150 ml				
11 am		160 ml	x	200 ml		x	200 ml		
12 am	200 ml							100 ml	x
1 pm					200 ml		200 ml		
2 pm	150 ml	100 ml							
3 pm		100 ml	x	200 ml				100 ml	
4 pm	200 ml				100 ml		200 ml	150 ml	
5 pm									
6 pm	300 ml	200 ml	x		100 ml	x			
7 pm				200 ml			250 ml	200 ml	x
8 pm					100 ml				
9 pm	200 ml	200 ml				x			
10 pm	500 ml			200 ml			300 ml	100 ml	
11 pm		200 ml			200 ml				
Midnight				200 ml			300 ml	160 ml	x
1 am									
2 am									
3 am	500 ml	200 ml			150 ml			100 ml	
4 am									
5 am								100 ml	x
6 am									

Fig. 5.1 A frequency-volume chart, with the amount of fluid intake and volume of urine emptied. "x" denotes the leak episodes

about the test. The patient is requested to fill in a frequency-volume chart for a minimum of 3 days prior to attending the test (Fig. 5.1). A midstream urine analysis is performed to rule out urinary infection. The test is deferred if infection is suspected or confirmed.

Equipment

The test needs to be scheduled in a private room with no interruption or interference. The equipment comprises a specialized commode, the outflow of the commode directed to a beaker that rests on a flowmeter (Fig. 5.2). The most commonly used integrated systems of urodynamic equipment have the monitor, infusion pole, roller pump system, filling tube set, transducer pressure domes with stop cock and printer, all stacked in a single trolley (Fig. 5.3). Two catheters (Fig. 5.4) are needed for the multichannel cystometry test. Bladder catheter is twin channeled or a composite single lumen for filling and to measure intravesical pressure. The other catheter with a balloon is inserted into the rectum to record the intra-abdominal pressure. It may also be inserted into the vagina or a colostomy stoma. The catheters are connected to pressure transducers which are in turn connected to the computer with integrated software and printer. A peristaltic pump system helps to fill the bladder with normal saline, at a predetermined rate.

Procedure

The sequence of testing in urodynamics starts with the noninvasive uroflowmetry, followed by measurement of the post-void residual urine volume and then filling and voiding cystometry.

Fig. 5.2 Commode chair with beaker on flowmeter

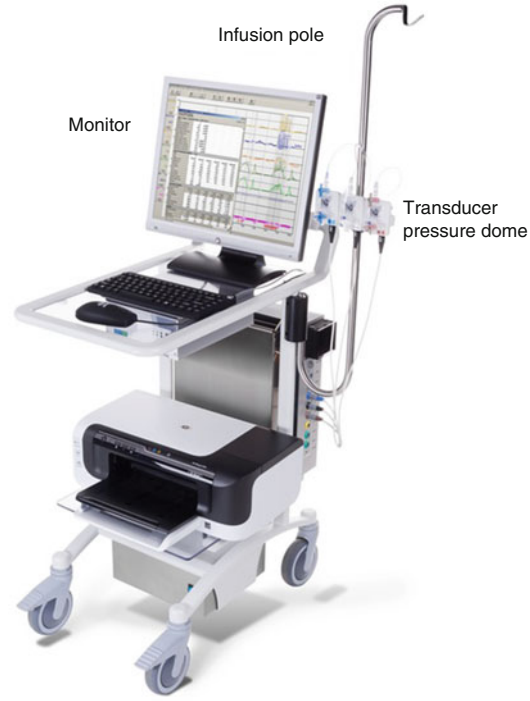

Fig. 5.3 Integrated urodynamic system

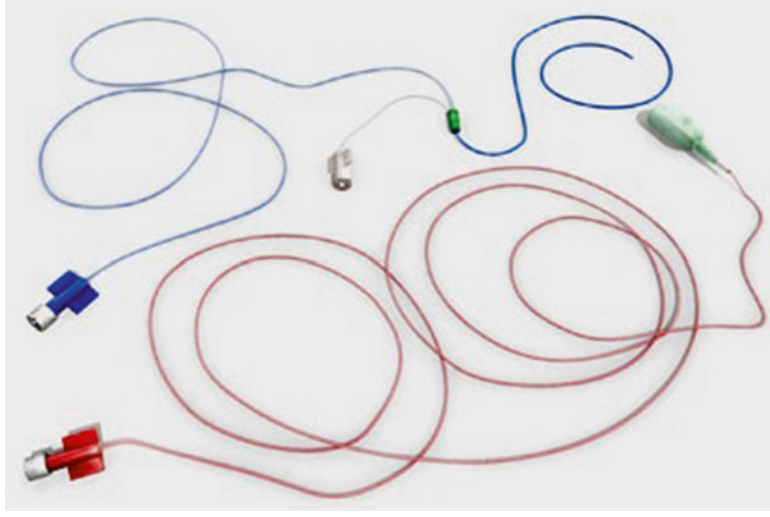

Fig. 5.4 Urodynamic catheters. Double lumen (*blue*) – intravesical catheter. Balloon tip – (*red*) rectal catheter

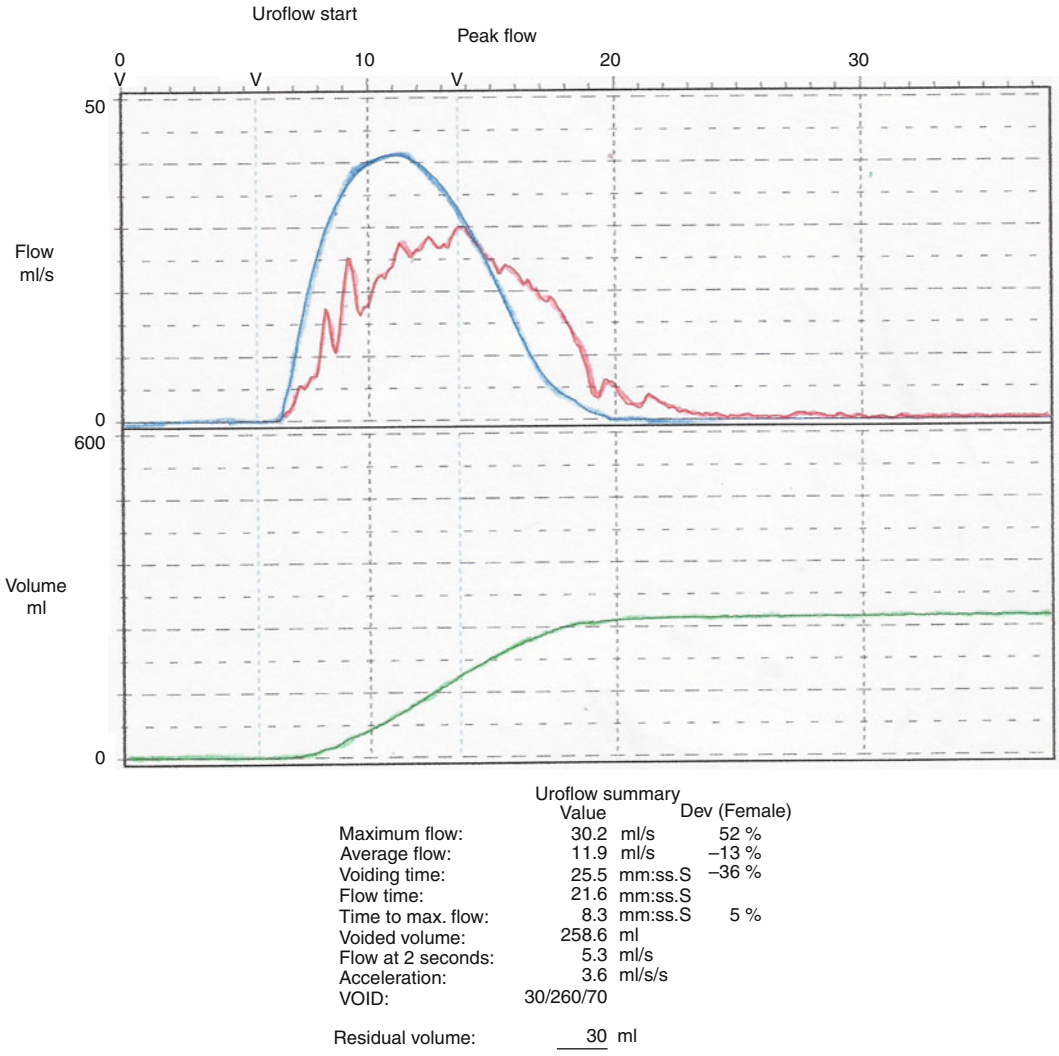

Fig. 5.5 Normal uroflowmetry pattern

Uroflowmetry

The patient is requested to attend with a full bladder and instructed to empty the bladder on the flowmeter commode in privacy. This test is done before insertion of any lines and gives the clinician an idea of the bladder capacity, the flow rate, and the flow pattern. The maximum flow rate should be above 20 ml/s. The voided volume should be at least 200 ml for meaningful interpretation. The normal flow pattern is bell shaped and smooth with the average flow time around 12–30 s (Fig. 5.5). Post-void residual urine volume is checked after completion of voiding by ultrasound scan or catheterization. The value of normal PVR is not clearly defined. In general, a residual of 1/4th to 1/5th of the voided volume is considered normal. Abnormalities in the uroflowmetry can be either intermittent or slow stream pattern (Fig. 5.6). It can signify either an underactive detrusor muscle or an outlet obstruction.

Filling Cystometry

Cystometry can be either single-channel cystometry or multichannel cystometry. In single-channel cystometry, the bladder is emptied by a transure-

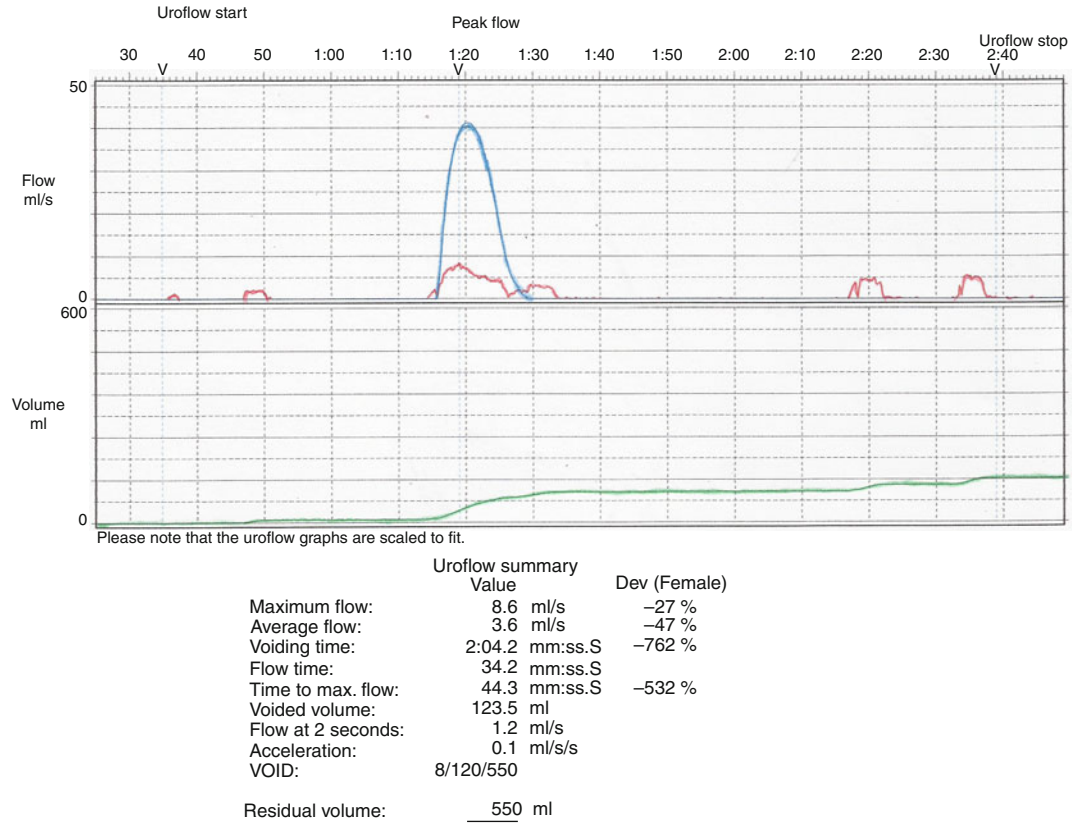

Fig. 5.6 Uroflowmetry intermittent pattern

thral catheter. A 60 ml catheter tip syringe without piston is attached to the catheter and held about 15 cm above the pubic symphysis. The patient is in sitting or standing position. The bladder is filled in 50 ml increments with sterile saline or water. The volume at which patient experiences the first sensation, first desire to void, strong desire and maximum bladder capacity is noted. The meniscus of the fluid in the syringe is observed continuously and any increase signifies detrusor contraction. Multichannel cystometry has largely replaced the single-channel technique currently.

In multichannel cystometry, the patient is requested to lie down on an examination couch and the bladder and rectal lines are inserted. Residual urine if any is noted at this time. The pressure measurement lines are connected to the transducers and flushed with normal saline. All systems are zeroed at atmospheric pressure. The reference point for external transducers is the superior edge of the symphysis pubis. The patient is asked to cough to ensure an adequate rise from baseline of both pressure lines. The pressure measurement from the bladder is termed P_{ves} or vesical pressure and the abdominal pressure or P_{abd} is recorded from the rectal line.

Filling cystometry is a measurement of the pressure/volume relationship during bladder filling. It is performed using measurements of intravesical pressure (P_{ves}) and intra-abdominal pressure (P_{abd}) to calculate the detrusor pressure ($P_{det} = P_{ves} - P_{abd}$). The key features of bladder storage function obtained with filling cystometry include bladder sensation, cystometric bladder capacity, compliance and presence of involuntary detrusor contractions or detrusor overactivity (DO). The aim is to replicate the woman's symptoms by filling the bladder and observing pressure changes or leakage caused by provocation tests.

The filling medium is usually normal saline or sterile water at room temperature. Fluid is infused

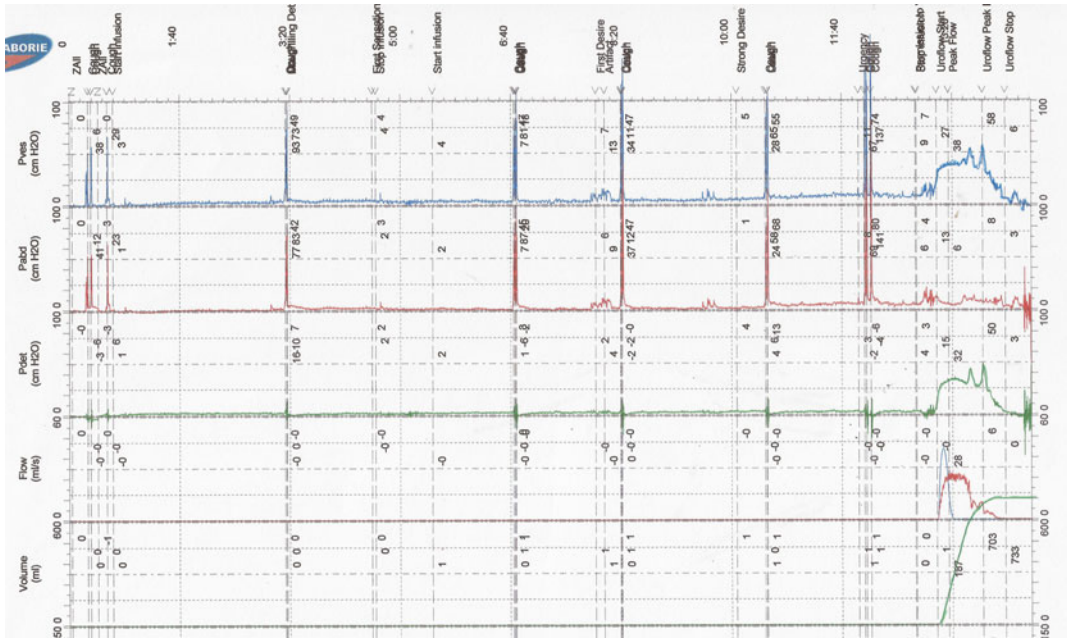

Fig. 5.7 Normal cystometry – with no detrusor overactivity or urodynamic stress incontinence

into the bladder through a peristaltic pump at a predetermined rate, usually around 50 ml/min. Filling rate can be varied depending on the clinical situation. If detrusor contractions are provoked repeatedly at filling rate of 50 ml/min, filling rate can be reduced. In large-capacity bladder, a rapid fill can be considered. A fast filling rate is >100 ml/min and is known to provoke detrusor activity. In patients with suspected neurogenic bladder, a slower fill rate around 20–30 ml/min is recommended.

The patient is asked to cough after every 100 ml of bladder filling, to ensure that the lines are recording appropriately. This will also elicit any stress incontinence if present. Bladder sensations are assessed on the basis of the volume at which patient expresses first sensation of filling, followed by her first desire to void and then a strong desire to void. Pain or urgency at any point in the test is encouraged to be reported. The patient is put at ease with conversation or music during the test. A bladder capacity volume of 400–500 ml as tolerated by the patient is reached. In large-capacity bladder with no sensation, this can be increased up to 600 ml. This is recorded as the maximum bladder capacity on filling cystometry. If urgency/detrusor activity is not demonstrable during the test, the sound of flowing water from tap and hand washing is used as a stimulus.

The bladder compliance during filling describes the relationship between change in bladder volume and change in detrusor pressure. In a compliant bladder, there is very little increase in vesical pressure with increasing volumes of bladder filling. A low-compliant bladder is seen when the elasticity of the detrusor muscle is lost such as in radiation cystitis. The steady increase in vesical pressure from the baseline during bladder filling denotes loss of bladder compliance.

In a woman with normal bladder and urethral function, the bladder compliance is maintained with first sensation around 100–200 ml, normal desire to void around 150–350 ml, and maximum bladder capacity around 600 ml. It is important to remember these are not absolute numerical parameters in urodynamics. The detrusor pressures remain low during filling with no symptoms of pain or urgency reported by patient and there should be no leak on Valsalva (Fig. 5.7). Urinary leak demonstrable with cough or Valsalva during filling cystometry is referred to as urodynamic stress incontinence (USI).

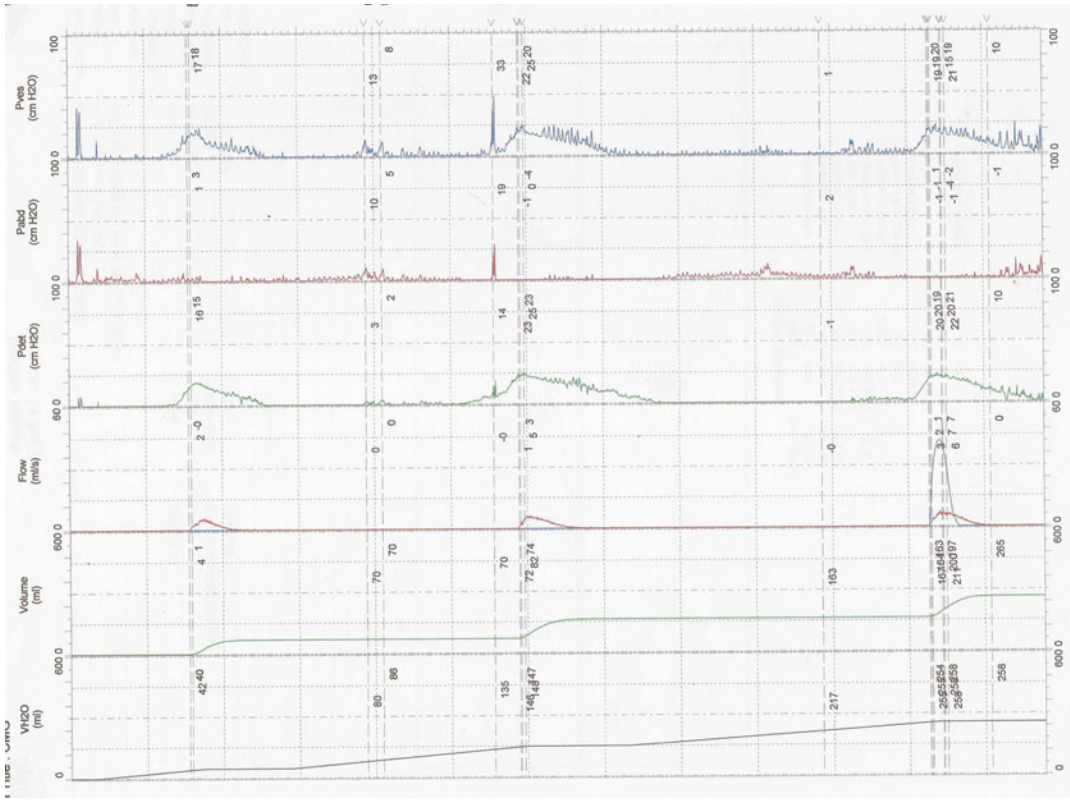

Fig. 5.8 Detrusor overactivity – phasic increase in detrusor pressure associated with leak

After filling, the patient is asked to stand astride an absorbent sheet and encouraged to perform a Valsalva maneuver and/or a series of coughs to provoke stress incontinence, if not present earlier in the sitting posture. If specific measures such as jumping or running provoke the incontinence, they are encouraged to do so, on the spot. Increase in detrusor pressure associated with urgency and/or leak is a feature of detrusor overactivity. The detrusor pressure increase can be phasic in nature (Fig. 5.8) or the pressure might increase steadily with a strong detrusor contraction resulting in elimination of urine.

Voiding Cystometry

The patient is asked to void, once maximum bladder capacity is reached or if patient requests permission to void due to pain or severe urgency. Voiding takes place in the flowmeter with the lines in situ, in privacy, for a more accurate recording of the flow rate and voided volume. The important aspects in voiding cystometry include maximum flow rate (Q_{max}), the detrusor pressure during maximum flow (P_{det}·Q_{max}), the voided volume, post-void residual urine volume, the flow pattern and the presence of abdominal straining. A slow urine flow rate with low detrusor pressure denotes an underactive detrusor. In women, obstruction is defined as a peak flow rate of less than 15 ml/s with a maximum voiding detrusor pressure greater than 20 cm of H_2O.

Electromyography (EMG)

Electromyography is used to assess the activity of the pelvic floor muscle during bladder filling and emptying, using surface electrodes. Used mainly

in research settings, the clinical relevance of EMG is in the diagnosis of dysfunctional voiding such as in detrusor sphincter dyssynergia. During normal voiding there is relaxation of the urethral sphincter and pelvic floor musculature, denoted by absent or minimal activity in EMG tracing. In dysfunctional voiding, there is increased activity in the EMG when voiding commences.

Urethral Function Tests

Urethral pressure profile (UPP) is done in some centers to evaluate voiding dysfunction, using catheter-mounted micro-transducers. Intravesical pressure and the intraluminal urethral pressure are measured simultaneously using pressure transducers that are about 5 cm apart. The pressures are measured during filling and during Valsalva. A mechanical puller withdraws the pressure transducer from the urethra at a set rate of 1–2 mm/s. The difference between the intraurethral and intravesical pressure is defined as the urethral closure pressure (UCP). The maximal urethral closure (MUCP) pressures are calculated along the highest point of the UCP curve. A maximal urethral closure pressure less than 20 cm H_2O denotes intrinsic sphincter deficiency and a high MUCP may denote urethral obstruction or diverticula.

Leak point pressure measurements are another measure of urethral function. This is used to assess intrinsic sphincter function with Valsalva. Abdominal leak point pressure (ALPP) (or Valsalva leak point pressure VLPP) is the intravesical pressure (P_{ves}) created by a Valsalva that leads to urine leak. ALPP is measured with bladder filled to around 200 ml, in the absence of a detrusor contraction. This is used as an indirect measure of the urethral sphincter function. Leakage at an ALPP of less than 60 cm of H_2O is diagnostic of intrinsic sphincter deficiency. Thus, while MUCP measures urethral resistance at filling, the ALPP measures the bladder pressure at leak generated by Valsalva and is dynamic in nature. In contrast, a detrusor leak point pressure (DLPP) is a static measure and is the lowest value of the detrusor pressure at which leakage occurs in the absence of detrusor contraction or increase in abdominal pressure.

Modifications of Urodynamic Testing

Videourodynamics

Videourodynamics is an excellent method for visualization of the bladder neck and its position in relation to the pubic symphysis. A radiopaque fluid is used for filling to allow fluoroscopic visualization of the lower urinary tract. Anatomical abnormalities such as diverticula and reflux may be diagnosed visually. Intrinsic sphincter deficiency can be diagnosed by the presence of open bladder neck and proximal urethra followed by leakage of fluid with straining, without an increase in detrusor pressure. Videourodynamics can be useful in patients with complex symptoms, with failed surgery and where a diagnosis is not possible with conventional urodynamics.

Ambulatory Urodynamics

Ambulatory urodynamics aims to eliminate the problem of non-physiological filling with conventional urodynamic assessment. The basic equipment for ambulatory monitoring comprises the vesical and rectal catheters and a portable storage device to record the data. The recording system is attached to the patient, and an electronic diaper quantifies the amount and time of leakage. It has to be interpreted with the patient's diary of activity during the test time. There is a tendency to overdiagnose detrusor overactivity with this test. However, it is useful in patients who are fearful of having routine urodynamics or when diagnosis is unclear with other investigations.

Pitfalls

Urodynamics is an invasive test that is often embarrassing. A clear explanation and a sensitive approach are required to relax the patient and derive meaningful results. Insertion of urinary catheter carries a small risk of urinary tract infection. The reproducibility of the test is also questionable. The setting in which the testing

takes place may not represent the normal daily activities of the patient and may not be a true representation of bladder function. In spite of its widespread use, there is no consensus on definitive normal values for each parameter.

Conclusion

Urodynamics has been deemed unnecessary in the evaluation of patients being offered conservative management. The main indication for urodynamics is its use in patients with voiding problem and as a preoperative tool before a surgical procedure for urinary stress incontinence. The question whether use of urodynamics improves clinical outcome has not been answered effectively yet. Nager et al. questioned the routine use of urodynamics and demonstrated in a large randomized controlled trial that the patient outcomes were no different with conventional clinical test and the routine use of preoperative urodynamics. When detrusor overactivity is demonstrated on urodynamics in the absence of clinical urgency, it often does not change the management and leads to overdiagnosis of the condition. However, in the absence of a more efficient tool, urodynamics still continues to be the gold standard in the diagnosis of lower urinary tract symptoms in women.

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Overactive Bladder

6

Karen L. Noblett

Overactive bladder (OAB) is an umbrella term that includes the symptoms of urinary urgency, frequency, nocturia, and/or urgency incontinence. Prevalence of this condition is reported to be higher in women and also reported to increase with age, with estimates extending up to 40 % after 70 years [1, 2]. OAB has a significant impact upon quality of life [3], as well as having a significant impact on the healthcare system with annual costs ranging from 66 billion US dollars. These costs are related to the routine costs of pads and diapers to patients, as well as the costs associated with the increased risk for falls and fractures, infections, and physical compromise [1]. Asking patients if they suffer these symptoms and tailoring an appropriate and effective treatment regimen is of great value not only for the patient but also for the healthcare system.

In 2010, the International Urogynecological Association (IUGA)/International Continence Society (ICS) formalized a joint report updating the definitions/nomenclature of symptoms surrounding overactive bladder [4].

1. Increased daytime urinary frequency: Complaint that micturition occurs more frequently during waking hours than previously deemed normal by the woman.
2. Nocturia: Complaint of interruption of sleep one or more times because of the need to micturate. Each void is preceded and followed by sleep.
3. Urgency: Complaint of a sudden, compelling desire to pass urine which is difficult to defer.
4. Overactive bladder (OAB, Urgency) syndrome: Urinary urgency, usually accompanied by frequency and nocturia, with or without urgency urinary incontinence, in the absence of urinary tract infection (UTI) or other obvious pathology.
5. Urgency (urinary) incontinence: Complaint of involuntary loss of urine associated with urgency.

The most common etiology for overactive bladder is idiopathic. Neurologic conditions including multiple sclerosis, Parkinson's, and spinal cord injury can lead to similar symptoms and are termed neurogenic detrusor overactivity. Detailed treatment and diagnostic issues specific to these conditions are beyond the scope of this chapter.

This chapter will serve to review the evaluation of OAB and the algorithm of treatment options for nonneurogenic overactive bladder and the current supporting literature.

Evaluation

The evaluation of patients with overactive bladder includes a thorough medical, surgical, gynecological, medication, and past therapy history.

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A. Tamilselvi, A. Rane (eds.), *Principles and Practice of Urogynaecology*,
DOI 10.1007/978-81-322-1692-6_6, © Springer India 2015

Detailing symptoms of voiding frequency, urgency and urinary leakage and importantly the level of bother to the patient are the key to diagnosis and ultimately the management.

The physical examination should include a pelvic examination with emphasis on pelvic masses, significant prolapse, and/or assessment for urinary retention (post void residuals >200 cc). Urinalysis and culture should also be evaluated to rule out an infection as this can mimic or exacerbate symptoms. Voiding diaries, typically for 72 hrs, are another useful tool to assess excess fluid intake, exacerbating factors, voiding and leakage patterns. Urodynamics and cystoscopy should not be included in the initial evaluation of uncomplicated patients, but can be considered in complicated or refractory cases [5].

Treatment

Based on extensive review of the literature, the American Urological Association (AUA)[1] published guidelines for a treatment algorithm in 2012 consisting of the following therapy recommendations [5]:

1. *First-line*: Behavioral therapy (fluid management, bladder retraining, pelvic floor therapy) with a potential for combination with antimuscarinics.
2. *Second-line*: Antimuscarinic medication (darifenacin, fesoterodine, oxybutynin, solifenacin, tolterodine, trospium, or transdermal preparations). No specific preference between these medications with the exception that extended release medication should be used preferentially if possible.
3. *Third-line*: Sacral neuromodulation, peripheral/percutaneous tibial nerve stimulation, intravesical botulinum toxin.
4. *Additional*: Rare cases for indwelling catheters, diversion, or augmentation cystoplasty.

[1] This AUA review does not address the role of β3-agonists.

Behavioral Modification

Bladder retraining (progressive delay in frequency to an interval of approximately every 2–3 hrs) and fluid management (moderation/avoidance of alcohol, caffeine, and normalization to approximately 1.5–2 l daily) are commonly cited as starting recommendations for patients and important points of discussion. Also, for those who have the primary complaint of nocturia, assessing voided night time volumes and amount of fluid intake after 6 pm may be useful parameters to help guide therapy. For example, if a patient complains of getting up three to four times a night, but each time they void, they are going 300–400 ml, it would indicate someone with nocturnal polyuria that would be addressed in a very different way than if it were drinking a large amount of fluid before bed and/or voiding only small amounts each time.

Pelvic floor physical therapy or muscle training (PFMT) is another commonly employed, low-risk tool. Data on efficacy is somewhat mixed [6, 7]; however, a recent Cochrane review on the subject concluded "the differences in likelihood of cure or improvement after PFMT compared to control are sufficient to be of interest to women" [8]. There is some evidence, however, that the benefit for stress urinary incontinence (a commonly combined measure in these studies) may be greater than that for overactive bladder and urgency incontinence.

Weight loss is a behavioral modification that has received attention as a means to decrease incontinence. A small cohort study demonstrated that a modest 5 % weight reduction decreased incontinence episodes by 50 % [9]. More recently, in the PRIDE (Program to Reduce Incontinence by Diet and Exercise) study, 338 women who were overweight or obese with at least 10 leakage episodes/week were randomized to a 6-month intensive weight-loss program or to four general educational sessions [10]. The intervention group experienced 8 % vs. 1.6 % weight loss, and a 47 % vs. 28 % reduction ($p=0.01$) in incontinence (this included either stress or urgency leakage) when compared to controls. Of note, women

with urgency leakage, specifically, experienced reduction of leakage from 42 % vs. 26 % which trended toward, but did not reach significance ($p = 0.14$) [10].

Pharmacologic Therapy

Antimuscarinics

Historically, the mainstay of pharmacological treatment for urgency incontinence was antimuscarinic medications. Antimuscarinic medications target and block intravesical receptors that promote bladder contractions. Typical efficacy is modest with symptom reduction ranging between 40 and 60 %. Rates of achieving continence range from 5 to 59 % [11]. Additionally, due to common side effects of dry mouth, dry eyes, and constipation, only approximately 25 % of users continue the medication by 1 year [12].

When choosing an antimuscarinic, there is data to support the use of extended over immediate release formulations. The OBJECT trial (Overactive Bladder: Judging Effective Control and Treatment), a multicenter randomized and double-blind study, demonstrated that oxybutynin 10 mg ER was superior to tolterodine 2 mg IR [13] . Solifenacin, in 5 and 10 mg extended release doses, was also shown to be superior to 2 mg immediate release dosing of tolterodine [14].

Additional comparisons include the OPERA and STAR trials. The OPERA (Overactive bladder: Performance of Extended Release Agents) directly compared extended release oxybutynin and extended release tolterodine. Oxybutynin was superior in reduction of urinary frequency, but had higher rates of dry mouth (23 % vs. 17 %) [15] The STAR trial compared solifenacin 5 or 10 mg to extended release tolterodine 4 mg [16]. Findings from this study of 1,177 patients, randomized with an ability for increased dosage in the solifenacin group, demonstrated a significantly greater improvement in the solifenacin group with respect to the number of urgency episodes, urge incontinent episodes, all incontinent episodes, and increases in mean-voided volumes [16].

Table 6.1 Anticholinergic preparations

Drug name	Dosage range
Oxybutynin	2.5–5 mg IR daily thrice daily 5 or 10 or 15 mg ER daily
Tolterodine	4 mg ER daily
Solifenacin	5 or 10 mg daily
Trospium	20 mg nightly, increase to twice daily
Darifenacin	7.5 or 15 mg daily
Fesoterodine	4 or 8 mg daily
Oxybutynin transdermal	3.9 mg patch every 4 days
Oxybutynin gel	84 mg of 3 % gel daily 100 mg of 10 % gel daily

However, as some data provide evidence of modest benefit of one over another, clear superiority is lacking. The recent AUA guidelines do not preferentially distinguish between any of the anticholinergic medications [5]. (An outline of available preparations is listed in Table 6.1.)

When considering side effect profiles, oxybutynin (which has a higher affinity for the parotid gland receptors) has higher rates of dry mouth (up to 61 %), while darifenacin has higher rates of constipation (up to 17 %) [5]. Transdermal preparations of oxybutynin in a patch or gel formulation may decrease these side effects while maintaining efficacy [17, 18]. Of note is the oxybutynin transdermal system (Oxytrol patch (3.9 mg)) recently approved for and is now available over the counter in the United States. In a recent meta-analysis compiling data regarding "trade-offs" between efficacy and side effect profiles, authors concluded that 40 mg/day trospium, 100 mg/g per day Oxybutynin gel, and 4 mg/day fesoterodine were the most favorable formulations [19]. However, acceptance of clear superiority of one antimuscarinic is lacking, and the AUA guidelines do not endorse the favoring of one over another antimuscarinic.

While side effect profiles have been a limitation, overall safety profiles of anticholinergics are good. The main contraindication for antimuscarinics is untreated narrow-angle glaucoma. Caution should also be maintained in patients with poor gastric emptying, frailty, and/or cognitive impairment [5].

β3-Agonists

The first in its class, and the first new class of medications for the treatment of OAB in over 30 years, mirabegron, a β3-agonist, was FDA approved for overactive bladder in June 2012. In contrast to antimuscarinics, this class of drugs targets the β3 receptors in the bladder dome that promote detrusor relaxation. It represents an exciting alternative to the antimuscarinics that have had poor continuity rates of only 25–50 % at 1 year, secondary to side effects [12].

Four phase III trials with mirabegron have demonstrated efficacy and safety [12, 20–22]. These studies included comparisons to placebo in three of the four as well as to tolterodine in two of the four studies. Regarding efficacy among these studies, mirabegron was superior to placebo and similar to tolterodine. Regarding side effects, mirabegron was better tolerated with lower rates of dry mouth compared to tolterodine (2.3–2.8 % vs. 8.6 %) [20]. Specifically, among 1,329 patients randomized to mirabegron 50 mg, 100 mg, or placebo, decreases in incontinence episodes were −1.47 (±0.11), −1.63 (±0.12) and −1.13 (±0.11) respectively. Similarly, the decrease in voids between active and placebo arms was −1.66 (±0.13), −1.75 (±0.12), and −1.05 (±0.13). Both findings were statistically significant [12]. In another randomized, double-blind study, doses of 25 and 50 mg were compared to placebo. Among these 1,306 patients randomized, mean incontinence episodes and number of micturitions were both significantly reduced in the mirabegron groups. The 50 mg dose, but not the 25 mg, also significantly increased the mean-voided volume over placebo [22] (for additional details see Table 6.2).

The safety and tolerability profile of mirabegron has been excellent. Some small, but clinically insignificant increases in pulse rate (0.8–0.9 bpm) and blood pressure (1.5 mmHg SBP and 1.0 mmHg DBP) have been noted [22]. Still, rates of hypertension in another study in both 50 and 100 mg mirabegron dose group were actually lower than placebo [21] . No studies demonstrated significant increases in cardiac events [12, 20–22]. The main consideration for an alternate drug recommendation remains uncontrolled hypertension (blood pressures >180/110). Still, while blood pressure monitoring is indicated, again, the actual clinical impact has not typically been significant.

Intravesical Botox

Onabotulinum Toxin-A (BTX-A), a serotype of the neurotoxin produced by *Clostridium botulinum*, is increasingly utilized as a safe and effective treatment option for refractory overactive bladder. Proof of concept of BTX-A use in the lower urinary tract stems from neurogenic bladder literature and has expanded its use into non-neurogenic cases [23].

BTX-A blocks acetylcholine release at the presynaptic neuromuscular junctions, decreasing detrusor overactivity and incontinence. It is additionally believed to alter urothelial sensory afferent pathways and help alleviate hypersensitivity responses, an explanation as why BTX-A is also effective in decreasing urinary urgency and frequency and increasing bladder capacity [23, 24].

Efficacy and safety of BTX-A have been demonstrated in multiple studies [25–30]. Efficacy typically defined as >50 % reduction in symptoms ranges at 60–80 %, with continence seen in approximately 22 % [24, 28, 29]. Doses of 200 or 300 units are often used in neurogenic cases. However, the literature in nonneurogenic overactive bladder points to an optimal risk/benefit dose of 100 units [27]. Higher doses have been associated with higher rates of retention and need for catheterization, and in one study with 200 units used in 28 women, this rate was as high as 43 % [31].

A recent, larger randomized trial of 242 women directly compared antimuscarinic therapy with intravesical BTX-A in the ABC trial: Anticholinergic versus Botulinum Toxin-A Comparison Trial for the Treatment of Bothersome Urge Urinary Incontinence [26]. Refractory patients with idiopathic overactive bladder were randomized to antimuscarinic therapy plus a saline intravesical injection vs. 100

Table 6.2 Results from phase III clinical trials, mirabegron

Author/year	Comparison groups (*n*)	Study time	Decrease incontinence episodes/24 h	Decrease in voids/24 h	Increase mean voided volume (ml)	Adverse outcomes/additional
Nitti et al. (2013) [12]	Placebo vs. 50 mg vs. 100 mg mirabegron (*n*=1,329)	12 weeks	−1.47/−1.63 in 50 and 100 mg groups vs. −1.13 in placebo ($p<0.05$)	−1.66/−1.75 in 50 and 100 mg groups vs. −1.05 in placebo ($p<0.05$)	18.2/18.0 in 50 and 100 mg groups vs. 7.0 in placebo ($p<0.05$)	Well tolerated. 1–2 bpm elevation in HR in mirabegron group. No difference in cardiovascular events
Chapple et al. (2012) [20]	50 mg or 100 mg mirabegron vs. tolterodine 4 mg ER (*n*=2,444)	12 months	−1.01/−1.24 for 50 and 100 mg mirabegron and −1.26 for tolterodine (no formal statistical comparison)	−1.27/−1.41 for 50 and 100 mg mirabegron and −1.39 for tolterodine (no formal statistical comparison)	17.5/21.5 for 50 and 100 mg mirabegron and 18.1 for tolterodine (no formal statistical comparison)	Tolterodine group with more dry mouth. Rates of hypertension, headache and constipation similar among all. No increased cardiovascular adverse events
Khullar et al. (2013) [21]	Placebo vs. 50 mg or 100 mg mirabegron or tolterodine 4 mg ER (*n*=1,978)	12 weeks	−1.57/−1.46 for 50 and 100 mg mirabegron vs. −1.17 placebo ($p<0.05$) Tolterodine −1.27 vs. placebo (ns) *no direct comparisons between mirabegron and tolterodine	−1.93/−1.77 for 50 and 100 mg mirabegron vs. −1.37 placebo ($p<0.05$) vs. tolterodine −1.57 (ns) *no direct comparisons between mirabegron and tolterodine	Mean increase vs. placebo in 50 mg/100 mg mirabegron/tolterodine =11.9/13.2/12.6 ($p<0.05$)	Changes in systolic and diastolic BP <1.5 mmHg were similar across treatment groups. No significant increase in cardiovascular events
Herschorn et al. (2013) [22]	Placebo vs. 25 or 50 mg mirabegron (*n*=1,306)	12 weeks	−1.36/−1.38 in 25 and 50 mg mirabegron vs. −0.96 placebo ($p<0.05$)	−1.65/−1.60 in 25 and 50 mg mirabegron vs. −1.18 placebo ($p<0.05$)	12.8 in 25 mg (ns); 20.7 in 50 mg dose ($p<0.001$)	No increase in cardiovascular events. Increase in BP: 1.5 mmHg SBP and 1.0 mmHg DBP with 0.8–0.9 bpm. Increase in HR in mirabegron groups

units BTX-A plus a placebo pill. At 6 months, those receiving BTX-A were more likely to be continent: 27 % vs. 13 % ($p=0.003$), with otherwise similar decreases in the number of incontinence episodes daily (initially a baseline of 5 decreased by 3.4 and 3.3/day). Expectantly, urinary tract infection rates (33 %) and intermittent self-catheterization at 2 months (5 %) were both higher in the BTX-A group. However, symptom control at 6 months was also significantly higher in the BTX-A group [26].In a recent cost analysis, the cost comparison was similar between the two treatments over the first 6 months, however, after that time (assuming average efficacy of BTX-A being 9 months), the cost profile favors BTX-A [32].

With BTX-A use, important contraindications/considerations remain: current urinary tract infection, malignancy, obstruction, pregnancy, and neuromuscular junction disorders such as myasthenia gravis (auto-antibodies to acetylcholine receptors) and Lambert–Eaton Syndrome (failure of nerves to release acetylcholine).

Sacral Nerve Stimulation (InterStim Therapy)

InterStim is a form of sacral nerve neuromodulation that is currently FDA approved for: urgency/frequency, urgency incontinence, nonobstructive urinary retention and fecal incontinence. It consists of a lead wire with four electrodes that are positioned along the sacral nerve roots—most commonly S3. This is then attached to an implantable pulse generator (IPG) that is surgically placed in the upper buttocks and provides a nonpainful electrical stimulation. Procedurally, this involves a two-step process (either in office percutaneous nerve evaluation (PNE) or stage I in the operating room) where the patient is able to test the efficacy (reduction in symptoms >50 %) prior to final IPG placement. Proof of concept for InterStim was devised in animal models by Tanagho and Schmidt in the 1970s, and it has been FDA approved in the United States for bladder indications since 1997 [33].

Several advances have been introduced including: a tined lead that has decreased invasiveness of the procedure, and a smaller IPG battery that has improved comfort. Evidence regarding how the tined lead is placed has also resulted in procedural improvements. Use of the curved vs. straight stylet in a randomized crossover trial demonstrated a clear intraoperative superiority with the use of the curved stylet [34]. Furthermore, the safety profile of InterStim, in light of these advances, is excellent. Major complications and morbidity have been uncommon, and estimates of infection (previously up to 10 %) have been closer to 3 % and of chronic pain (previously up to 16 %) have been closer to 8 % in more recent studies [35–41].

Theories on how InterStim works include modulation of the somatic afferents in the pudendal nerves which could both aid inhibitory mechanisms or revive an ability to void by relieving abnormal guarding reflexes—both of which would normalize voiding function [42, 43]. Additionally, recent work has demonstrated that InterStim modulates learning center regions of the CNS [42]. Still, a precise understanding of how InterStim functions remains unclear.

The efficacy of sacral nerve neuromodulation is well supported in multiple clinical trials. The literature demonstrates success for urgency/frequency and urgency incontinence to range between 56 and 68 % (up to 80 %). Efficacy in patients with urinary retention is approximately 70 %, and in fecal incontinence approximately 85 % [35, 36, 44–47]. Success is being defined as 50 % or greater reduction in symptoms.

The recent InSite Trial compared InterStim directly to standard medical therapy (antimuscarinic medications) [48]. In 147 patients with an overactive bladder randomized to these modalities, those receiving InterStim had significantly higher-efficacy rates: 61 % vs. 42 % ($p<0.05$). Quality of life measures were also significantly improved with InterStim compared to medications. 86 % of InterStim subjects compared to 44 % of those undergoing standard medical therapy reported “improved” or “greatly improved” urinary symptoms ($p<0.001$) [48].

Peripheral/Percutaneous Tibial Nerve Stimulation (PTNS)

PTNS is another form of neuromodulation for the treatment of overactive bladder. Procedurally it involves a 34-gauge needle placed 5 cm above the medial malleolus in order to access the posterior tibial nerve and enables stimulation of L4 to S3 nerve roots. This stimulation occurs in an office setting for 30 min on a weekly basis for 12 weeks, with subsequent monthly treatments from then on.

The SUmiT (Study of Urgent PC vs. Sham Effectiveness in Treatment of Overactive Bladder Symptoms) [49] compared active to sham stimulators. Women were randomized ($n=220$) between the two groups and after 13 weeks, the PTNS group demonstrated 54.5 % (vs. 20.9 % in the sham) efficacy defined as "moderately or markedly" improved symptoms. Voiding diaries also demonstrated statistically significant reductions in all overactive bladder parameters [49]. This study additionally supported safety measures of this modality with only 6 of the 110 reporting adverse events which included bruising, tingling, bleeding at the needle site, or discomfort.

Sustainability of this response after the initial 12 weekly treatments with continued monthly sessions has been supported. When 33 PTNS successes were continually treated with monthly sessions for 1 year, efficacy was maintained [50]. When compared to antimuscarinic therapy, PTNS has also demonstrated superiority. Among 100 adults randomized to PTNS or tolterodine 4 mg daily, success was demonstrated in 79.5 % of PTNS vs. 54.8 % in tolterodine ($p=0.01$) [51]. Of note, however, there was no placebo or sham treatment, and no blinding in this study, which may have impacted results.

Conclusion

Overactive bladder represents a chronic, common condition that significantly impacts the quality of life, but multiple treatment options exist. While prudent to initiate conservative options before moving to more invasive therapies (as outlined in the AUA guidelines [5]), a knowledge base and employment of the breadth of these options are valuable. It is important to discuss realistic expectations with patients, as many therapies define success as symptom reduction rather than cure. Regular follow-up to assess and reassess their progress and satisfaction level is essential to ensure compliance. Often it is not through one but through a combination treatment plan that optimal results are achieved.

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Management of Stress Urinary Incontinence

7

Aswini Balachandran and Jonathan Duckett

Introduction

Stress urinary incontinence is a common debilitating condition affecting millions of women worldwide. Conservative treatments are recommended as first-line therapies, but many women need surgical treatment if pelvic floor muscle training is ineffective. In the past the colposuspension operation was the gold standard, but over the last 15 years, this technique has been replaced by mid-urethral sling procedures. These have a high success rate and reduced morbidity. The original retropubic (down-up) sling has been modified and now transobturator slings (inside-out and outside-in) are also available. Recently, short single-incision slings (mini-slings) have been introduced, although current evidence suggests an inferior outcome. We are still learning the benefits and limitations of each type of sling, and many surgeons express a keen preference for one sling over another. Different surgeons obtain very different results with similar slings. As surgeons we should aim to audit our results so that we ensure we give our patients the best possible outcome. This article reviews the current surgical treatment options for stress urinary incontinence.

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Stress urinary incontinence (SUI) is defined by the International Continence Society as the involuntary leakage of urine on exertion, effort, coughing, or sneezing [1]. It adversely affects the quality of life of women of all ages [2]. The reported prevalence of SUI is variable, but several studies suggest that it may be as high as one in four adult women. A postal survey in 2004 conducted in four European countries (UK, France, Germany, and Spain) involving over 29,000 participants found that 35 % of women questioned admitted urinary incontinence [3]. SUI is the most common type of urinary incontinence. Despite this, less than a third of women will seek medical help. Reasons for this reticence are varied and include embarrassment, lack of knowledge of available treatment options, and the belief that incontinence is an inevitable consequence of childbirth or aging [3]. The management of SUI puts a significant burden on healthcare systems with an estimated cost in the USA of over $12 billion [4].

Brief Pathophysiology

In order to maintain continence during bladder filling and urine storage, the bladder outlet and urethra must be closed at rest and remain so during periods of increased abdominal pressure. Normal bladder emptying occurs with a decrease in urethral resistance followed almost immediately by bladder contraction. Relaxation of the

A. Tamilselvi, A. Rane (eds.), *Principles and Practice of Urogynaecology*,
DOI 10.1007/978-81-322-1692-6_7, © Springer India 2015

pelvic floor muscle and urinary sphincters permits urine to flow into the urethra. SUI occurs as a result of a poorly functioning urethra. The mechanism of SUI is from insufficient urethral closure pressure during exertion that raises intra-abdominal pressure. The lack of urethral closure pressure is due to anatomical changes in the bladder and urethra (e.g., cystocele) and weakness in the pelvic floor [2]. Risk factors for SUI include pregnancy, childbirth, menopause, obesity, constipation, and chronic cough. A thorough history taking and physical examination should be performed in all patients presenting with SUI. Voiding diaries and pad tests are important adjunctive assessments. Urodynamics studies are commonly used during the assessment of bladder symptoms [5], although recent evidence suggests that pressure flow cystometry is not mandatory for all women [6].

Treatment Options

There are several well-established treatments for SUI and many have long-term data to support their use [2]. Behavioral modification changes such as weight loss and treatment of underlying constipation or cough are often first-line measures. Other treatments include pelvic floor exercises and medical therapies such as duloxetine. Surgical treatment is currently the mainstay of treatment for SUI [2].

According to the 2009 update of International Consultation on Incontinence Guidelines, the surgical techniques suggested in the treatment of SUI include Burch colposuspension, autologous slings, mid-urethral slings (such as TVT and TOT), insertion of artificial urinary sphincters, and periurethral bulking agents. Many factors should be considered when determining the optimal therapy for a patient with SUI. These include bladder capacity, voiding dysfunction, detrusor overactivity, sexual function, degree of discomfort to the patients, concurrent prolapse, and abdominal or pelvic pathology. The decision to treat symptomatic SUI with surgery should be made when the patient's symptoms are severe enough to warrant an elective operation and nonsurgical therapy is either not desired or has been previously ineffective.

Pelvic Floor Muscle Training

Pelvic floor muscle training (PFMT) is a first-line therapy for women with stress incontinence [7]. It was first described by Arnold Kegel almost 60 years ago. PFMT exercises help to strengthen the muscles of pelvic floor. Numerous studies have evaluated its efficacy and durability with conflicting results. PFMT exercises consist of repeated, high-intensity, pelvic muscle contractions of both slow and fast twitch muscle fibers. Though studies have demonstrated significant improvement in symptoms with PFMT, patient compliance and motivation is essential for continued success [7]. The Cochrane review of PFMT suggests that only 15–20 % of women comply with a regimen [8]. Therefore a successful program of behavior modification and pelvic floor exercises requires a substantial commitment of time and perseverance from both physician and patient. However, in clinical practice, failure rates tend to be high and PFMT has consequently gained a reputation for both futility and poor efficacy [7]. A recent multicenter randomized trial comparing physiotherapy and the mid-urethral sling procedure (MUS) reported a 91 % subjective improvement in the MUS arm compared to 64 % in the physiotherapy arm [9].

Duloxetine

Duloxetine hydrochloride is a potent balanced serotonin noradrenaline reuptake inhibitor (SNRI) and was the first available pharmacological option licensed for SUI [10]. It has a centrally mediated mechanism of action via the neurotransmitter glutamate. A systematic review of 3,063 women showed a significant reduction in incontinence episode frequency, which was decreased by half when duloxetine was commenced. This led to improvements in quality of life and significant increases in voiding intervals [11]. Ghoniem then demonstrated in an RCT that duloxetine was more

effective than pelvic floor muscle training (PFMT) and its effect was synergistic with PFMT [12]. However, duloxetine is poorly tolerated outside of randomized controlled trials with one study reporting only 9 % of patients using duloxetine after 1 year and 82 % opting for a mid-urethral tape [13, 14]. The commonest reason for discontinuation was side effects, specifically nausea in 56 % of women. This was followed by lack of efficacy (33 %) and an unwillingness to take long-term medication (11 %) [11]. The place for duloxetine still remains unclear. The UK's National Institute for Health and Care Excellence (NICE) guidelines from 2013 do not recommend duloxetine as first- or second-line treatment as most women would choose to have a one-off minimally invasive surgical procedure with a higher success rate. Duloxetine may be best considered in women who are unfit for surgery, who do not wish to undergo surgery, or those with severe stress incontinence who are awaiting surgery or are yet to complete their families [10]. Duloxetine is unavailable and remains unlicensed for the treatment of stress urinary incontinence in many countries.

Burch Colposuspension

The aim of surgical treatment for SUI is bladder neck suspension to reduce urethral hypermobility. There have been multiple procedures described, of which, the Burch Colposuspension has been the most extensively studied. It was first introduced in the early 1960s. The basic principle of colposuspension is the fixation of the bladder neck and proximal urethra via suspending sutures placed laterally into the tissue on either side of the paravaginal fascia, at the level of the proximal urethra. These sutures are then placed through the ipsilateral iliopectineal ligament thereby supporting the vesicourethral junction within the retropubic space [15]. It has been described as the most effective surgical procedure for treatment of stress incontinence. Many studies have demonstrated excellent long-term success rates. Burch colposuspension is a time-honored procedure with a 10-year success rate in the range of 55–70 % [16, 17]. However, it is associated with high rates of de novo detrusor overactivity (17 %), voiding dysfunction (10.3 %), and pelvic organ prolapse (13.6 %) [17]. It is also a major surgical procedure and usually involves several days in hospital and a prolonged recovery. In situations where MUS tapes are not available or costly, this may be a valuable option.

Laparoscopic Colposuspension

Laparoscopic Burch colposuspension, one of the first minimal-access techniques for the treatment of SUI was described by Vancaille and Schuessler in 1991 [18]. Laparoscopic colposuspension procedures use similar techniques to open colposuspension procedures with the additional benefits of laparoscopic surgery: less intraoperative blood loss, less postoperative pain, shorter hospital stay, quicker return to normal activities, and shorter duration of catheterization compared to an open procedure. However, it requires a higher level of technical skill and laparoscopic training to perform. The laparoscopic approach is associated with higher complication rates and longer operating times [19]. A study by Dean et al. comparing the outcomes of laparoscopic colposuspension and TVT revealed a statistically significant higher cure rate for TVT [19]. Due to the high success of the MUS procedure, few laparoscopic colposuspensions are now performed.

Pubovaginal Slings

Pubovaginal slings have been described almost 100 years ago [2, 15]. Historically, autologous rectus fascia and fascia lata were among the most commonly used sling materials. Autologous fascial sling procedures were widely used in the late twentieth century [20]. Pubovaginal slings work on the principle of providing support to the proximal or mid-urethra. Long-term subjective and objective cure rates were 82.4 and 85.3 %, respectively. As with the colposuspension, this technique has largely fallen into disregard with the increased use of the MUS. There may still be a place for this technique where MUS procedures have failed.

Mid-Urethral Sling Procedures

Retropubic Tapes: Tension-Free Vaginal Tapes (TVT)

Development of the tension-free vaginal tape (TVT) has occurred in the mid-1990s and works on a similar principle to sling procedures. The original tape was produced by a single manufacturer, but there are now many similar tapes produced from a variety of companies. There is very little direct research comparing tapes produced by different companies.

A synthetic mesh tape is placed suburethrally at the mid-urethral point to create a pubourethral "neoligament" that is anchored suprapubically. A long thin strip of polypropylene mesh tape tightens around the urethra on increased intra-abdominal pressure [2] (Fig. 7.1). A MUS procedure may be performed as a day case procedure under general anesthesia, but it is important to check that the patient is voiding adequately before discharge. Alternatively, MUS can be performed under local anesthesia, although this is usually combined with sedation. Initial follow-up data has shown it to be highly effective treatment for stress leakage with success rates of 95 % at 5 years [21]. Multicenter randomized controlled trials comparing TVT with colposuspension by Ward et al. demonstrated an 81 % objective cure rate for TVT compared to 90 % for colposuspension [22]. These results were supported by a further two meta-analysis. The MUS procedure has a low rate of complications in experienced hands, the most common of which was bladder perforation, which generally causes no long-term effects if identified intraoperatively. Though voiding disorders may occur post-insertion of an MUS, it appears to be less likely than with other incontinence procedures [15]. Other complications include urinary retention rates of approximately 2 % and de novo detrusor overactivity in about 5 % of patients. Compared to open colposuspension, TVT was associated with decreased operative time, analgesia requirement and hospital stay. A further consideration with the MUS is the vast number of different tapes currently available. Each tape varies in pore size and whether they are knitted or weaved, which are important factors when considering tape infection and erosion. To limit the risk of rejection and complications, it is

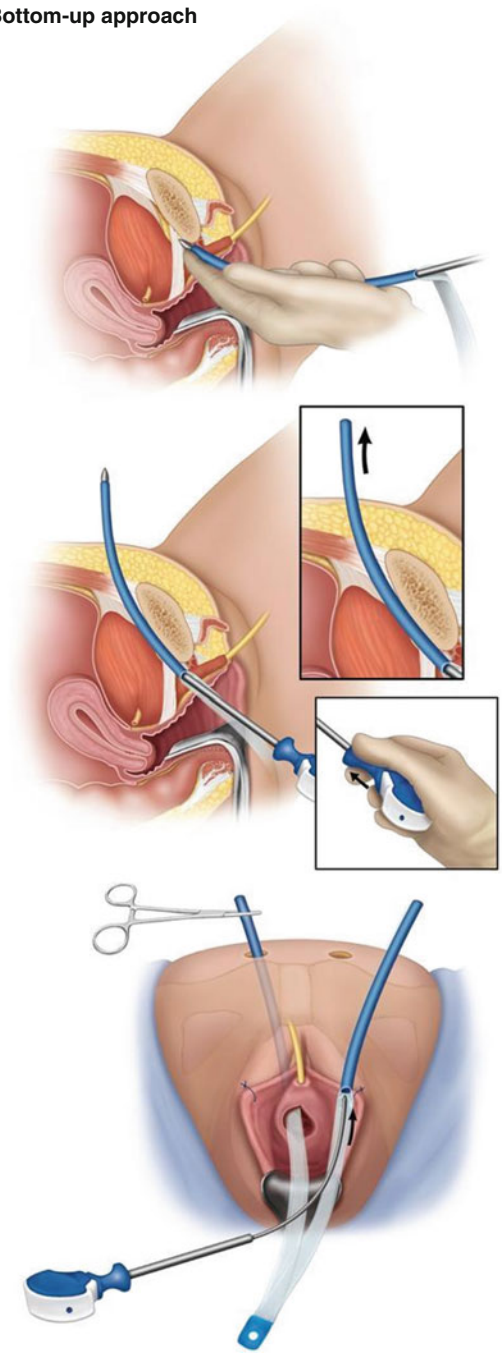

Fig. 7.1 The retropubic approach of mid-urethral sling, with needle passage from bottom-up (From: Noblett et al. [34]; with permission)

important that the tape is macroporous and a monofilament.

Nilsson et al. reported that at 11-year follow-up, objective and subjective cure rates of the TVT were as high as 90 and 77 %, respectively, without any significant late-onset adverse effects [23]. This has been collaborated by a 10-year follow-up data by Svenningsen et al. who demonstrated an 89.9 % objective cure rate and a 76.1 % subjective cure rate with only 2.3 % of patients requiring repeat SUI surgery [24]. Recent publications now describe similar success rates at 17 years [25]. Due to its effective long-term success rates and low complication rates, retropubic MUS procedures are currently considered the gold standard for the treatment of SUI.

Transobturator Tape (TOT)

Transobturator tape (TOT) insertion is a newer development of the MUS and is a modification on the retropubic technique. It dates back to the early 2000s and similarly involves the placement of a manufactured synthetic tape suburethrally. However, in TOT, the tape is anchored through the obturator foramen [2, 15]. Avoiding the retropubic space makes TOT procedures both less invasive and safer [2, 15]. There are two methods of inserting a transobturator tape with the difference being the direction of penetration. With the "inside-out" technique, the needle passes from the midline suburethral position laterally, while for the "outside-in" method, the needle is passed from a lateral position to sit suburethrally. A review by Latthe et al. identified no major difference in efficacy or morbidity between the two techniques [26]. Both techniques avoid the blind passage of the needle through the retropubic space, which is required in the insertion of the TVT [2]. Two meta-analyses demonstrated that TVT and TOT have similar efficacy. However, the risk of bladder perforations and pelvic hematoma are significantly less common in patients treated with TOT [15]. The TOT operation is associated with more groin pain postoperatively. Large studies using observational data suggests that the TOT may have a slightly lower success rate than the retropubic MUS [27].

Mini-slings

Mini-slings were first introduced in 2006. The aim of the mini-sling was to further reduce morbidity related to MUS. The mini-sling is a short tape mesh sling measuring between 8 and 14 cm with paired anchors at each end, inserted under local analgesia via a single vaginal incision. The mini-sling can be deployed either into the retropubic space or in a similar fashion to the TOT. The fixation ends of the tape are often placed in indeterminate soft tissue. Consequently, fixation into good tissue can neither be guaranteed nor tested at time of surgery. The potential benefit of mini-slings is the reduction of adverse events such as pain and visceral injury. This hypothesis has been tested by Smith et al. Their 2-year data showed excellent tolerance of local analgesia, early return to normal, and low morbidity, but very poor success rates [28]. Mini-slings could therefore be potentially used in an outpatient setting. The original mini-slings had a poor success rate [29]. Newer slings with improved design may have a better success rate [30], and this hypothesis is currently being studied in a large trial in the UK.

However, there are a few well-powered studies testing the long-term efficacy of mini-slings resulting in conflicting results. A multicenter randomized trial comparing mini-slings with TOT conducted by Mostafa et al. demonstrated that at 1-year follow-up, there were no differences in terms of subjective satisfaction or quality of life score found between the two groups, with similar success rates [30]. However, a systematic review and other studies suggest a lower cure rate for mini-slings [31]. Longer-term and multicenter outcome data is necessary to evaluate the place of mini-sling in the treatment of SUI.

Artificial Urinary Sphincter (AUS)

These are considered as a "last hope" treatment for recurrent stress incontinence and are only offered after other surgical interventions have failed. They were introduced in the 1970s. The principle of this procedure involves increasing outlet resistance using a patient-controlled inflatable cuff around the proximal urethra. This

allows intermittent deflation and bladder emptying. Good results have been reported when AUS is inserted for SUI secondary to intrinsic sphincter deficiency. Webster et al. (1992) reported over 90 % continence at 2.5 years following AUS insertion in women without previous surgery for SUI [32]. However, longer-term results are less optimistic. In addition, there is a high rate of sphincter removal due to infection or erosion [2]. This should not be unexpected as these devices are usually inserted to tissues which are scarred and damaged by previous failed surgery.

Periurethral Bulking Agents

Periurethral bulking agents have been used for the treatment of SUI in women for decades. They create submucosal cushions ensuring apposition of the urethral wall, which aids continence. It can be carried out under local anesthesia and as a day case procedure. It is therefore associated with a low patient morbidity. A variety of substances have been reported to be safe and effective, but others have been withdrawn from the market after a variety of complications. A Cochrane review published in 2003 by Pickard concluded that bulking agents result in both subjective and objective short-term improvement in women with symptoms of SUI [33]. A study by Corcos in 2001 compared periurethral bulking agents with open colposuspension and TVT. Objective pad weight testing after 12 months revealed an increased curative rate but significantly higher complication rates after the latter two operations [2]. Periurethral bulking agents also have an apparent absence of postoperative de novo detrusor overactivity. However, it is recognized that two or more treatments may be necessary for the majority of patients, and the success rate is probably inferior to other surgical treatments. Despite this, some patients may prefer the low risk of complications and its minimally invasive nature as an initial treatment for SUI prior to considering more invasive surgery. Bulking agents are recommended for use in patients unfit for general anesthesia. They are most commonly used for patients after a failed MUS.

Conclusion

In conclusion, the ideal therapy for SUI has yet to be clearly identified. However, in this area of significant morbidity affecting quality of life significantly, there is good evidence for the efficacy of various treatment modalities.

In treating patients with stress urinary incontinence, the decision on the best course of treatment should be made in light of the available evidence and in conjunction with the patient's own preferences.

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Management of Sling Surgery Complications

8

Jay Iyer and Ajay Rane

The management of urodynamically proven stress urinary incontinence underwent a paradigm shift with the focus changing from bladder neck suspension to support of the mid-urethra in the last two decades. This has resulted in the replacement of Burch colposuspension and pubovaginal slings to synthetic mid-urethral slings as the primary surgical option in women with stress urinary incontinence (SUI). The introduction of synthetic mid-urethral slings has resulted in good objective and subjective cure rates but can be associated with complications that pose a challenge to the treating surgeon. Surgical treatment for SUI has rapidly evolved, and the initially introduced mid-urethral sling, retropubic tension-free vaginal tape (TVT), is accepted worldwide as a standard treatment for women suffering from SUI. However, slings have been associated with a few early and delayed complications which may result in varying degrees of morbidity. Serious complications such as bowel injury, major vascular injury, and even death have been reported with mid-urethral sling procedures.

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Sling complications are related to the sling type, surgical technique and time elapsed postsurgery. Though there are numerous devices available in the market, three different types of mid-urethral slings are commonly described in current practice, namely-retropubic slings (TVT, GYNECARE™; AMS™, RetroArc), transobturator slings (AMS™, Monarc; TVT-O, GYNECARE™) and single-incision mini-slings (AMS™, MiniArc). The rationale for choice of sling has been discussed elsewhere in this book and the surgical technique varies with the type of sling chosen which in turn is intricately related to type of complications seen with slings. This chapter deals with the management of synthetic mid-urethral sling complications in the intraoperative and postoperative period.

Intraoperative Complications

Bleeding, bladder injury and urethral injury are the common intraoperative complications. The overall risk of the intraoperative complications varies between the retropubic and transobturator approach.

Hemorrhage

Bleeding with mid-urethral sling can occur either during the vaginal dissection or with needle passage and perforation of the retropubic or transobturator space. If bleeding is encountered during

A. Tamilselvi, A. Rane (eds.), *Principles and Practice of Urogynaecology*,
DOI 10.1007/978-81-322-1692-6_8, © Springer India 2015

dissection, attempt should be made to control it with pressure and if unsuccessful the best advice is to complete the procedure as quickly as possible [1]. Closure of the periurethral fascia is often all that is necessary to stem the bleeding. If bleeding persists, a pack can be inserted into the vagina and left for a few hours. Bleeding from the venous plexus of Santorini is a risk with the retropubic approach which can be difficult to control. Measures which help in controlling the bleed in this space includes, tamponade with Foley catheter balloon [2], use of hemostatic agents and embolization [3].

The Austrian registry looking at 5,578 TVT procedures reported an overall bleeding rate of 2.7 % [4]. Significant intra-op bleeding requiring surgical intervention occurred in less than 1 % of the procedure. The close proximity of external iliac and femoral vessels with the retropubic approach and the presence of accessory obturator vessels in this area can result in bleeding from direct injury to these vessels. Risk is increased with lateral deviation of the needle, and careful control of the needle during the retropubic passage reduces the risk. In severe uncontrolled bleeding, an open transabdominal retropubic repair may be needed.

Bleeding and its related complications do occur with the transobturator tapes (TOT), and the risk of bleeding with TOT is around 1 %. As with TVT procedures, packing and embolization can help in the control of bleeding [5]. Of concern is an undiagnosed hematoma within the adductor muscles, presenting late as pain in this region.

Bladder and Urethral Injuries

The most frequent intraoperative complication of TVT is bladder perforation and the reported incidence varies from 1 to 15 % with an average rate of 5 % [6, 7]. The risk of bladder perforation is increased in patients with previous retropubic procedures and concomitant vaginal surgeries [8]. In the majority of cases, perforation occurs on the side opposite to the surgeon's dominant hand [9]. Once bladder perforation is diagnosed, the trocar is removed and replaced slightly laterally. Care should be taken to avoid injury to external iliac vessels while going laterally. At the end of the procedure, the bladder is catheterized and drained for 24–48 h. Bladder perforation when diagnosed intraoperatively and treated in this manner has no long-term morbidity. If bladder perforation is unrecognized intraoperatively, it can present with recurrent UTI, bleeding or calculi. Removal of tape later can not only be difficult but may also warrant a cystotomy.

Expert recommendations from the American Urological Association (AUA) state that intraoperative cystoscopy should always be performed to minimize the risk of urinary tract injury [10]. Other measures recommended to reduce bladder injury include emptying the bladder prior to procedure and using finger guidance during needle passage. The risk of bladder injury is considerably lower, less than 1 % with the transobturator approach [5], but it does exist and hence cystoscopy should be considered in TOT procedures as well.

Urethral injury can occur with both retropubic and transobturator approaches and usually results from dissection in the wrong plane. The risk of urethral perforation with needle passage in retropubic approach is around 0.07–0.2 % and with transobturator 0.1–2.5 % [6, 11, 12]. Urethral injury needs to be repaired watertight using 3-0 or 4-0 Vicryl and an indwelling catheter placed. In the presence of a urethral injury, sling placement is contraindicated [13] and the procedure can be undertaken after 6 weeks.

Other Intraoperative Complications

Bowel perforations are rare but a lethal complication reported in 0.03–0.7 % of retropubic procedures [14, 15]. In patients at risk of bowel adhesions in the retropubic space, a transobturator approach is preferable as it avoids entry near the abdominal cavity. In the presence of a bowel perforation, removal of the tape, repair of the damaged bowel, treating and controlling sepsis forms the basis of treatment. Peyrat et al. recommended computed tomography before TVT in patients presenting with risk factors for bowel perforation [16]. Obturator nerve injuries and two cases of loss of clitoral sensitivity with anorgasmia caused by injury to the dorsal nerve of the clitoris have been reported [6]. Although these complications are rare, it is essential that the surgeon is aware of these. It helps in assessment of inexplicable symptoms following sling surgery.

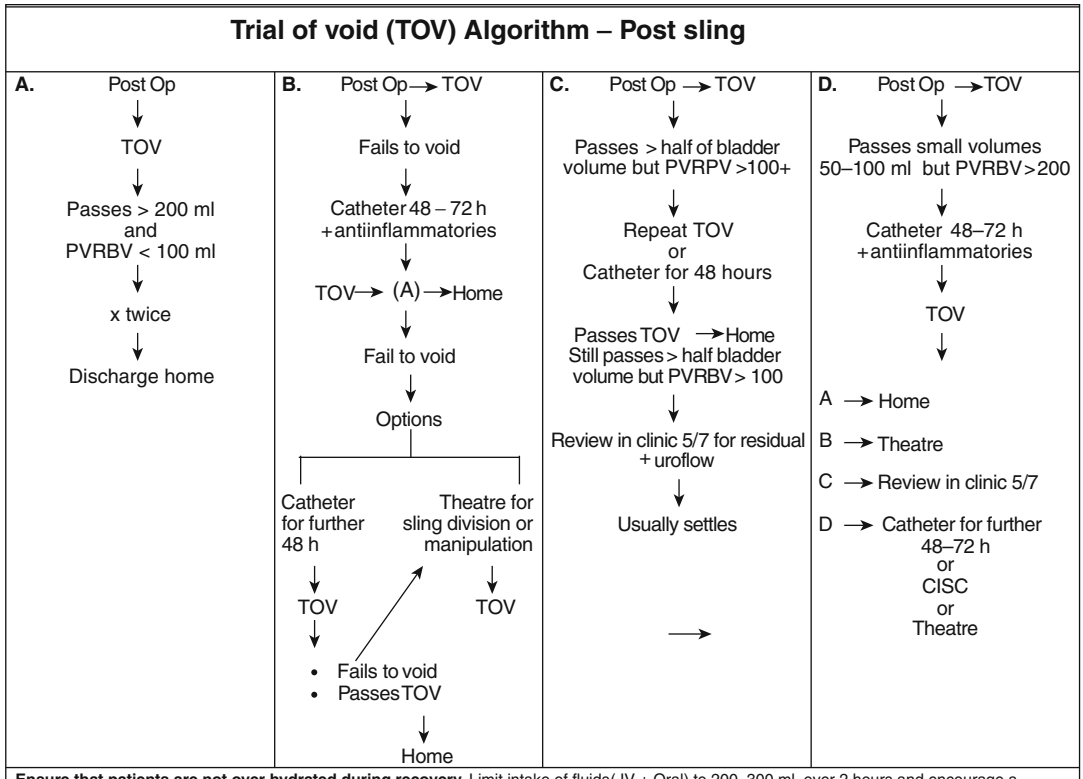
Early Postoperative Complications

Early postoperative complications include urinary retention, lower urinary tract symptoms (LUTS), pain, and infection.

Urinary Retention

Urinary retention is a known complication of anti-incontinence surgery. The incidence of retention with tension-free mid-urethral slings varies from 2 to 25 %, but catheterization beyond 1 week is needed in only 4–8 % of patients [17]. It is thought to result from increased tensioning of tape and overcorrection of the urethral angle. The presenting feature may vary from complete retention to symptoms of frequency and urgency with partial obstruction. The management of postoperative retention following sling surgery varies, depending on the presentation and the type of intervention planned. The timing of any active surgical intervention is still an area where there is no universal consensus yet.

At the first voiding trial postsurgery, if the patient has complete retention, an indwelling catheter is usually placed for 2–3 days. The immediate postoperative voiding problem can be transient and it has been shown that in those who fail the first voiding trial, 36.8 % successfully void on subsequent trials [18]. It is important to rule out an infection in patients with retention. If there is no resolution of retention after a week of catheterization, the options are clean intermittent self-catheterization (CISC) [19], indwelling catheter for 2–4 weeks or early surgical intervention. Some surgeons prefer early surgical intervention and will consider a sling release/urethrolysis at this stage. In those who adopt a more conservative approach, it is important to note that when symptoms persist beyond 28 days, it is unlikely to resolve spontaneously [20] and a more aggressive approach becomes necessary. Long-standing obstruction can lead to irreversible bladder dysfunction. With complete retention following sling, cystometry assessment has no role in evaluation and sling release should be undertaken irrespective of detrusor contractile function (see Trial of void algorithm).



Ensure that patients are not over hydrated during recovery. Limit intake of fluids(IV + Oral) to 200–300 ml, over 2 hours and encourage a Trial of Void 2 h post-operatively. Ensure pain relief as well. (PVRBV= post void residual bladder volume, TOV = trial of void).

The next group with urinary retention can empty their bladder but can have varying degrees of voiding problems (hesitancy, straining to void, weak urinary stream, sensation of incomplete emptying) or storage symptoms (frequency, urgency, and nocturia). Assessment and management of this group is discussed later in this chapter.

Pain

Pain can result from urinary retention, hematoma, urinary tract infection or nerve entrapment. Symptoms and the site of pain provide a clue to the etiology along with urinalysis and ultrasound. Thigh pain and groin pain occur more frequently with the TVT-O approach and a meta-analysis comparing retropubic with transobturator tape found the odds ratio of 8.3 for transobturator [21]. Passage of the needle at the groin results in a pathway through the adductor magnus, adductor brevis and gracilis muscle which can lead to hematoma, myositis, infection or abscess, all of which can result in pain. Pain should be treated expectantly with pain killers for the first 1–2 weeks after surgery and in most patients groin pain resolves within 1 month of surgery [22]. If it persists beyond this period, the possibility of nerve or muscle injury such as obturator nerve entrapment should be considered. This may require sling removal which could be a challenging and daunting task since the transobturator sling occupies the deep tissue space under the thigh muscles.

Lower Urinary Tract Symptoms (LUTS)

Postoperative urinary tract infection (UTI) is a frequent complication with sling surgery with incidence varying from 8.9 to 34 %. The risk is similar with both retropubic and transobturator approaches [23]. A 3-day course of antibiotics has shown to reduce the risk of postoperative UTI following sling surgery [24].

De novo urgency symptoms are another common complication reported in 10–15 % of mid-urethral slings [21]. Trials comparing retropubic with transobturator with regard to de novo urgency have shown conflicting results. While some studies have shown no difference in de novo urgency rates with both approaches [21], some have shown reduced rate of de novo urgency with the transobturator approach [25, 26]. De novo urgency symptoms can result from partial obstruction or by extrusion of sling into the urinary tract. The former is more insidious and difficult to diagnose, but a careful history and urodynamic assessment are helpful. In partial obstruction, the release of sling can lead to symptom resolution in 75 % of patients [27].

The first line of treatment of urgency symptoms would be the antimuscarinics once extrusion and obstruction are ruled out. In refractive cases neuromodulation and botulinum toxin may have to be considered. While de novo urgency postsurgery is considered a complication of sling surgery, the persistence of preoperative urgency in the postoperative period needs to be identified.

Late Postoperative Complications

These include extrusion or exposure of the sling mesh, obstruction/voiding dysfunction and recurrent urinary tract infections. The terminology exposure and extrusion in mesh complications are distinct entities by definition. Exposure is a condition of displaying, revealing, exhibiting or making accessible (e.g., vaginal mesh visualized through separated vaginal epithelium). Extrusion is passage gradually out of a body structure or tissue [28].

Extrusion/Exposure of Slings

Vaginal, bladder or urethral extrusion usually presents after a few months of sling surgery but can occur after many years. The usual presentation of vaginal tape extrusion is either discomfort, pain, dyspareunia, partner dyspareunia (hispareunia) or vaginal discharge, but some

of these patients can be asymptomatic. Meta-analysis has shown that vaginal extrusion of tape appears to be increased with the transobturator approach [21, 23] and is more likely to occur at the lateral vaginal sulci. The horizontal orientation of the needle passage with TOT possibly predisposes to it. Possible reasons for vaginal extrusion include incomplete closure of vaginal incision, wound infection or excessive foreign body reaction.

The management of vaginal extrusion can be conservative with observation only [29] or topical estrogen especially in the early postoperative period. If unsuccessful, surgical approach becomes necessary usually with vaginal excision or trimming of the exposed mesh, with closure of vaginal wall defect. This has been shown to be done successfully in the outpatient setting as well. If the mesh material is infected or the extrusion is recurrent, it becomes necessary to remove the entire sling.

Extrusion into the urinary tract- bladder or urethra is a potential complication with both types of sling approach. In patients who are diagnosed with sling extrusion into the urinary tract, most often it is the result of a missed perforation. Hence the importance of cystourethroscopy following sling surgery needs to be stressed.

Urethral sling extrusion can result from placement of the mesh deep in the periurethral fascia or from excessive tensioning of the sling. Patients can present with recurrent UTI, frequency and urgency, hematuria or decreased urinary stream. The management of urethral erosion involves removal of the sling and repair of the defect over a catheter, with re-approximation of periurethral fascia. Reinforcing the repair with a labial pad of fat can reduce the tension over the repair and promote healing. Indwelling catheter for a minimum of 2 weeks is recommended [30]. There are reports of successful management of urethral slings using endoscopic scissors or laser treatment [31].

Bladder extrusion of the tape can present with irritative bladder symptoms of frequency, urgency, urge incontinence, hematuria or lower abdominal pain. Patients with these symptoms not responding to conservative measures need cystoscopy to identify bladder extrusion. Surgical management of bladder extrusion can be done endoscopically, via cystoscope or laparoscope, or openly through a retropubic or vaginal approach. In extrusions into the bladder involving a large surface area, open excision may be preferred over endoscopic excision; however, the latter is becoming increasingly possible. Laparoscopy-assisted endoscopic excision is another option; indeed, it may be the preferred route. After excision, indwelling catheter for 2 weeks allows healing of the defect.

Voiding Dysfunction

Patients with insidious voiding dysfunction following sling surgery can present with changes in voiding pattern such as hesitancy, poor stream, straining to void, sensation of incomplete emptying, and increased postvoid residual urine volume (PVR) or with development of de novo urgency symptoms. Klutke et al. noted a 2.8 % rate of obstructive symptoms that ultimately required transvaginal sling release in a series of 600 patients who underwent TVT [32]. Voiding dysfunction with the retropubic approach is around 3.4 % and with transobturator approach is 2.0 % in the TOMUS trial [33]. The increased incidence of voiding dysfunction in the TVT group is to be expected, as a sling inserted in this manner has a propensity to cause more obstruction than a sling inserted via the transobturator approach, reflecting the vector of pull. Evaluating the risk factors for voiding dysfunction after sling surgery, the study also showed that concomitant surgery does not increase the risk.

Preoperative urodynamic variables have been evaluated to assess their predictive value in postoperative voiding dysfunction. Some studies have shown that low preoperative peak flow rate, abnormal uroflow pattern or increased PVR can be predictive of postoperative voiding problem [34, 35]. On the other hand, some have stated that there are no preoperative urodynamic variables which can offer successful prediction [32, 36].

Evaluation in patients with post-op voiding dysfunction includes history of preoperative voiding patterns such as the use of Valsalva maneuver for emptying and other incontinence surgeries in the past. Pelvic examination should evaluate for urethral mobility and fecal impaction. Cystoscopy may be needed in ascertaining the symptom etiology.

Early intervention is required in women needing catheterization. The timing of intervention in other cases is determined by the degree of patient distress, time interval between the surgeries, and the onset of obstructive symptoms. In patients with predominant storage symptoms, the initial option would be behavioral modifications such as restriction of fluid intake, timed voiding and double voiding. Antimuscarinics and vaginal estrogen for irritative symptoms and alpha blockers for retention symptoms can be tried. Urethral dilatation has shown some benefit, but recent studies have shown dilatation to be ineffective and may predispose to urethral erosion [37].

Persistent symptoms may justify urodynamics to assess bladder outlet obstruction, but studies have shown it may not carry a good predictive value. In the presence of obstructive features and failure of conservative management, sling release is indicated. Surgical intervention for postoperative voiding dysfunction is needed in 1–2 % of patients and more commonly with retropubic slings [38]. Mobilization of the sling, division of the sling and a formal urethrolysis are the different surgical techniques used in sling release. Mobilization of the sling by loosening it is possible only in early interventions [39]. If this fails, the sling is divided either in the midline or laterally. A transvaginal midline sling division may appear simple, but the risk of urethral injury is high, since the sling is more commonly incorporated into the surrounding tissues in late interventions. With the catheter in place, a 2 cm longitudinal vaginal incision is made below the mid-urethra. The sling is identified by a combination of sharp and blunt dissection and with the help of a sound in the urethra. When the sound is withdrawn gradually with downward pressure, one can feel a "step-off" at the site of the sling. A right angle clamp is passed between the urethra and the sling, and the sling is incised in between the arms of the clamp. It is important to identify the sling, as scar tissue can be confused with the sling [40].

Lateral division of the sling can be helpful in avoiding urethral injury and the technique has been described by Long et al. [41]. The dissection is carried laterally towards the ischiopubic rami from the midline incision, sling is dissected away from the periurethral fascia and divided between two hemostats at 3 or 9 o'clock position. This leaves a "J"-shaped portion of the sling intact.

In a nationwide analysis by Laurikanien et al. [42], after a midline or lateral sling division, 49 % of patients were completely cured of retention. Using the lateral incision technique, Game et al. [43] quoted a 70 % continence rate after sling division. A formal urethrolysis involves dissection and entry into the retropubic space either via the abdominal or vaginal approach with resolution of voiding in 85 % of patients.

Recurrent Urinary Tract Infection

Urinary tract infection appears to be a common problem following sling surgery and recurrent UTI, defined as more than three episodes of UTI symptoms in a year, occurs in 2–4 %. A preoperative history of recurrent UTI, intra-op bladder perforation, occurrence of UTI within first 6 weeks of surgery, and a PVR >100 ml appear to be independent risk factors for recurrent UTI in the postoperative period [44]. In the TOMUS [33] trial, the risk of UTI was increased in those with concomitant surgery compared to those with mid-urethral sling alone. Postoperative recurrent UTI more significantly should alert the possibility of undiagnosed bladder or urethral tape extrusion and/or insidious voiding problem. Recurrent UTI is reported in 53 % of patients with obstructive feature. Identification and correction of the underlying problem are important in treating these patients.

Conclusion

Mid-urethral slings are widely accepted as a standard surgical treatment in women with SUI. However, serious intraoperative as well as remote complications may occur with all types of sling procedures. Complications such as pain, infection, exposure, and extrusion of the tape can occur many years after initial placement, which demonstrates the importance of long-term follow-up of these patients. The surgeon has to carefully review the evidence for efficacy and safety prior to inserting any device for the treatment of SUI in all patients. The understanding that complications can happen even in the hands of an experienced surgeon mandates long-term appropriate surveillance in all.

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Neurogenic Bladder

9

Krishnamurthy Sairam

Introduction

The term "neurogenic bladder" encompasses a variety of clinical and urodynamic entities. In general, it refers to clinical problems related to bladder dysfunction secondary to underlying neurological conditions. It is important to obtain a thorough clinical history followed by examination including a good neurological assessment. While this may give clues to the underlying problem, correct management will not be achieved without the aid of laboratory testing, imaging, and urodynamic evaluation, the last in many patients playing a key role in their management. While certain conditions may warrant specific treatments, management, in general, becomes easy once patients are classified based on their urodynamic pattern. The key determinant in the management of neuro-urological problems is to ascertain whether the bladder is "safe" or "unsafe," i.e., its impact on the renal function. Once this has been determined, management of these entities becomes easy. It is worth remembering that the bladder is a dynamic organ and tends to change physiologically and hence functionally over time. In this chapter, key concepts in identification and management of common clinical entities that cause "neurogenic bladder" will be discussed.

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Neurogenic bladder refers to bladder dysfunction caused by an underlying neurological condition with either involvement of the central or the peripheral nervous system. The bladder innervations are from sympathetic (T10-L1), parasympathetic (S2–S4), and somatic (pudendal S2–S4) nerves. The afferent and efferent fibers relay to the spinal cord and travel via the ascending and descending pathways to the pontine center with neural connections to the cerebral cortex. The neuroanatomy and neurophysiology of the bladder are discussed in depth elsewhere in this book (Chaps. 2 and 3).

Diseases that affect the nervous system, starting from the brain, brainstem and spinal cord up to the peripheral nerves, can cause neurogenic bladder problems. This may result from conditions such as stroke, Parkinson's disease, spinal cord injury, multiple sclerosis, spinal dysraphism, alcoholism, and diabetes mellitus. Childbirth, in addition to other pelvic floor disorders like urinary stress incontinence and fecal incontinence, seems to contribute to voiding problems and parity seems to have an association with it [1].

Clinical Features

The bladder has two primary functions, storage and expulsion of urine, and neurological problems tend to affect one or the other of these two. Depending on the location and level of the neurological lesion, the detrusor muscle or the urethral

A. Tamilselvi, A. Rane (eds.), *Principles and Practice of Urogynaecology*,
DOI 10.1007/978-81-322-1692-6_9, © Springer India 2015

sphincter can be either overactive, normoactive, or underactive [2]. Hence, a patient with neurogenic bladder tends to manifest with either predominantly failure of storage or failure to void symptoms. Clinical acumen determines the dominance of one over the other.

Voiding symptoms include hesitancy, poor urinary stream, dribbling, straining, frequency, nocturia, sensation of incomplete emptying of bladder or fullness of the bladder and total inability to void despite a full bladder (retention). Storage symptoms would include frequency, urgency, urgency urinary incontinence and stress urinary incontinence. Incontinence can also be due to impaired voiding and overflow incontinence, which usually manifests as enuresis, the involuntary loss of urine. This may be sign of a high-pressure "unsafe" bladder, a concept that is discussed further below. Sensation of fullness of the bladder is an important part of the micturition cycle and this is lost or obtunded in patients with neurogenic bladder caused by distal spinal or neural damage.

It is important to note that urgency is a complaint of sudden, compelling desire to pass urine which is difficult to defer [3]. Urgency in an ambulatory fit person may not be a major problem; it will almost certainly result in urgency incontinence in someone with poor mobility like in those with Parkinson's disease, multiple sclerosis or stroke.

Table 9.1 Neurogenic bladder can result from a variety of disorders affecting the central or peripheral nerves

Etiology of neurogenic bladder
Brain and spinal cord related:
Cerebral palsy
Parkinson's disease
Multiple sclerosis, amyotrophic lateral sclerosis
Encephalitis
Stroke
Congenital defects of spinal cord
Brain or spinal cord tumors
Spinal cord injury
Peripheral nerve related:
Diabetic neuropathy
Chronic alcohol use
Vitamin B_{12} deficiency
Herniated disk
Nerve damage from pelvic surgery
Syphilis, poliomyelitis

Clinical Assessment

In women presenting with symptoms suggestive of neurogenic bladder, it is important to obtain a thorough history to identify predisposing factors. The etiological factors implicated in neurogenic bladder are detailed in Table 9.1.

Childhood urological problems such as enuresis, recurrent urinary tract infections, vesicoureteral reflux and congenital spinal problems are relevant. Medical and surgical history focusing on conditions such as diabetes, multiple sclerosis, Parkinsonism, spinal cord injuries and pelvic surgeries is essential, along with the use of alcohol and medications that are likely to affect bladder function. Since the parity, duration of labor, mode of delivery, and menopausal status influence the bladder function, a thorough obstetric and gynecological history is of use. Pelvic floor disorders commonly being interrelated, bowel function needs to be elicited, as neurogenic bowel is likely to coexist with neurogenic bladder, presenting with constipation. In addition, previous treatments and their effect on symptoms can guide subsequent management.

Of significance to the clinician is the identification of an "unsafe" neurogenic bladder. The indications of which could be the presence of hematuria, pain, urinary infections, deranged renal function, or hydronephrosis. Further evaluation should focus on the possibilities of stone or growth.

In spinal cord injury patients with injury level above T6, it is important to get a history or be wary of *autonomic dysreflexia*, a medical emergency that needs to be treated rapidly and adequately. This results from exaggerated sympathetic activity in response to stimuli below the level of injury. Bladder or rectal distension or lower urinary tract instrumentation can result in severe headache, hypertension, flushing and sweating above the level of lesion accompanied usually by bradycardia but occasionally with arrhythmias and tachycardia [4].

The physical examination in these patients is focused on identifying evidence of voiding problem

and a probable causative factor. An abdominal and pelvic examination can identify a palpable or tense bladder and pelvic organ prolapse, a digital rectal examination assesses anal/perianal sensation and sphincter tone and a bulbocavernosus reflex checks integrity of S2–S4 neurons.

The focused neurological evaluation should be supplemented by a general neurological assessment starting with the gait and mobility of the patient, presence of intentional tremors (Parkinsonism) and gross neurological deficits in spinal cord injuries, depending on the level involved.

Investigations

A bladder diary for a minimum of 3 days, with voided volumes, can provide very useful information. It is important to assess renal function and rule out urinary infection as initial part of investigation. In suspected spinal lesions, an X-ray or more commonly magnetic resonance imaging (MRI) is employed in the identification of the site and type of lesion. Other imaging techniques such as ultrasound helps in estimating post-void residual (PVR) urine volume and ruling out hydronephrosis or hydroureter and calculi. CT urogram may be needed in select patients with hematuria, abnormal urinary tract, and spinal dysraphism and in those with reconstructed lower urinary tract to rule out vesicoureteral reflux. Cystoscopy may be needed in those with irritative symptoms, to rule out stone or growth.

Urodynamics, however, is the gold standard evaluation in patients with neurogenic bladder. Uroflow (free flow study) with PVR measurement and cystometrogram (CMG; pressure flow study) will guide the initial management in majority of these patients. Complex testing (videocystometrogram, VCMG; electromyography, EMG; urethral pressure profilometry, UPP) is reserved for those where simple initial evaluation is inconclusive or in those who are at risk of renal compromise and therefore may need surgical intervention. In other cases, such as in patients with complex symptom profile associated with multiple sclerosis, Parkinson's disease, diabetes mellitus, spinal dysraphism, or following radical pelvic surgery, this may be indicated to establish the underlying urodynamic profile. And if symptoms change in the future, further urodynamic evaluation may have to be repeated to guide therapy.

While a detailed elucidation on invasive urodynamic testing is beyond the scope of this chapter, it is important to understand some key determinants such as bladder compliance, detrusor leak point pressure, and abdominal leak point pressure that underpin management of these patients. In patients with suspected neurogenic pathology, slow filling rate is recommended.

Compliance describes the relationship between bladder pressure and volume. It is easy to remember the concept of compliance if one imagines a balloon being blown. Initially, it is difficult to overcome the resistance of the balloon as it is "stiff" or poorly compliant. As one continues to blow the balloon, it becomes easier (one is able to increase the volume of the balloon with little effort), highly compliant, or accommodative. The bladder's viscoelastic property allows it to behave in a similar fashion. Therefore, a compliant bladder allows storage of increasing volumes of urine without proportionate increase in its pressure.

Detrusor leak point pressure (*DLPP*) helps in identification of patients at risk of upper tract damage in neurogenic bladder. DLPP is the lowest detrusor pressure (Pdet) at which urine leakage occurs in the absence of either a detrusor contraction or increased abdominal pressure. The bladder is filled to the point when leakage occurs without increase in abdominal pressure and this detrusor pressure is the DLPP. It measures resistance of the urethra to bladder storage pressure. In the presence of an outlet resistance, high bladder pressure is needed for leakage to occur. When the DLPP is >40 cm H_2O, ureteral peristalsis is hindered and leads to hydroureters, hydronephrosis, and vesicoureteral reflux resulting in renal deterioration [5]. In a poorly compliant bladder with a low DLPP, the patient can be incontinent, but their upper tract is not damaged, as the urethra opens with low pressure. Hence, the "safe" bladder should have a resting pressure of less than 40 cm H_2O.

Valsalva leak point pressure (*VLPP*), or abdominal LPP (*ALPP*) as it is often called, is the intravesical pressure (Pves) at which urine leakage occurs due to increased abdominal pressure in the absence of a detrusor contraction. This is used as an indirect measure of the competence of the sphincter. McGuire showed that 76 % of those with intrinsic sphincter deficiency had an ALPP of <60 cm H_2O [6]. Pelvic organ prolapse can interfere with ALPP assessment and has to be carried out with the anatomy restored (e.g., using a pessary).

It is worth mentioning that urodynamic testing needs to be an interactive session between the patient and investigator and it should ideally reproduce the patient's symptoms. Recognition of associated factors like vesicoureteral reflux and pelvic organ prolapse and their impact on bladder and outlet function is paramount in the management of some of these complex cases.

It is essential to categorize the neurogenic bladder dysfunction following urodynamic testing, into obstructed or non-obstructed and also as due to bladder or outlet problem and the presence or absence of detrusor-external sphincter dyssynergia (DESD).

Management

Medical Goals

- Aim for a low-pressure compliant bladder.
- Protect renal function.
- Maintain continence.
- Address patient's symptom.
- Voiding ideally without the need for catheterization.

Psychosocial Goals

- Patient's desires and suitability for a specific treatment option:
  - Physical ability to deal with treatment option, e.g., can the person perform clean intermittent self-catheterization (CISC)?
  - Cognition, e.g., does the patient understand the complexity of the underlying issues?
  - Mental ability, e.g., the ability to cope with any planned intervention.
  - Social ability, e.g., does the person have any caregivers/support?
- Improve or maintain good quality of life.

It is important that the patient is fully aware of the underlying problem and the progressive ladder of management that may be used in difficult cases. For some, nonmedical management may suffice for life without significant morbidity, while others may require invasive therapies. The treatment plan needs to be individualized for the patient's needs and abilities. Patient involvement is crucial for the success of any management plan.

Treatment of Urgency and Urgency Incontinence

The aim is to improve quality of life by reducing the urgency and urgency incontinence episodes and could be achieved with nonmedical or medical management or a combination of both.

Conservative Management

Provided the upper tract is not at risk, those with minimal symptoms are likely to do well with coping strategies for urgency combined with *pelvic floor exercises*. The voiding diary would have indicated about their fluid intake and the intake of bladder irritants like caffeine. Switching to *decaffeinated beverages* and *reducing intake before key activities* should be part of any successful management plan. A strategy that is commonly taught in practice involves asking the patients to sit down at the first sensation of "urgency." The patient is taught that this is uncoordinated contraction of the bladder muscle, which if ignored is likely to pass. The patient is then asked to count to about 50 very slowly. During this time they are instructed to perform pelvic floor exercise intermittently (Kegel exercise). Once the sensation of urgency passes away completely, they can then resume their activity and pass urine the next time there is a natural call. If they are able to do this, the next step is to start *voiding by*

the clock, i.e., timed voiding. If the patient's urgency incontinence episodes are minimal and there is improvement of their quality of life, then these measures would suffice.

Medical Management

This is usually considered when patients have significant symptom burden, fail with the above conservative measures or require some confidence in the initial stages while undertaking the above measures.

Antimuscarinics are the most commonly used group of agents to treat overactive bladder symptoms [7]. Oxybutynin has been used for the longest period and is perhaps the most studied. In those with significant side effects with oral use, the intravesical instillation of oxybutynin has been found to be equally effective with fewer side effects. Propiverine is another agent that has been studied extensively. The newer antimuscarinics like tolterodine, fesoterodine, solifenacin, darifenacin and trospium are all equally effective and have their own specific advantages.

The other class of drug that has anticholinergic and antispasmodic properties is the *tricyclic antidepressants* (imipramine, amitriptyline), which can facilitate bladder storage.

Mirabegron, a new agent, is a beta-3 adrenoceptor agonist that allows relaxation of the detrusor and increases bladder capacity without affecting voiding [8]. It is reported to have fewer side effects like dry mouth compared to the antimuscarinics.

Neuromodulation

In patients refractory to conservative measures, sacral neuromodulation and posterior tibial nerve stimulation are known to improve the symptoms of overactive bladder. The exact mechanism of neuromodulation on bladder function is not yet clear but improvement of symptoms has been noted in patients with NDO.

Treatment of Urethral (Outlet) Resistance

The aim here is to keep the intravesical pressure low to prevent damage to upper tracts, while improving the patient's quality of life.

Medical Management

Alpha blockers (terazosin, alfuzosin, tamsulosin) have been shown to reduce functional urethral resistance during voiding and can improve bladder emptying [9]. If alpha blockers are effective, it can be used in long-term management of outlet resistance. In patients with poor bladder compliance and neurogenic bladder, combination therapy using two or three different oral agents (antimuscarinics, alpha blockers, and tricyclic antidepressants) has been found to be effective [10].

Botulinum Toxin in Neurogenic Bladder

Schurch pioneered the use of botulinum toxin type A (BoNTA) in 2000 [11]. It is assumed that BoNTA inhibits vesicular acetylcholine release from motor nerve terminals by cleaving the SNARE protein SNAP-25, thus having a strong anticholinergic effect. In addition to the efferent effects, other mechanisms of action have been investigated and confirmed as well, including on the afferent side (sensory inhibition). Thus, it is like temporary denervation of the bladder. This effect tends to last between 6 and 9 months, with higher doses resulting in urinary retention that may require clean intermittent catheterization (CIC).

Botulinum toxin injection into the detrusor muscle is used as a second-line therapy in those with neurogenic detrusor overactivity (NDO) refractory to antimuscarinic or combination therapy. But, there seems to be a paradigm shift happening, especially with NDO where patient demand seems to be changing clinical practice.

There is anecdotal evidence for patients directly seeking intravesical injection of Botox rather than trying the ladder of sequential antimuscarinics. On this background it is not clear where mirabegron will fit in. Further studies will hopefully establish the role of each drug tailored to an individual patient scenario [8].

The optimal dose of BoNTA is yet to be determined but commonly 200–300 IU of onabotulinum toxin (or 300–500 IU of abobotulinum toxin) is used in clinical practice for treating neurogenic detrusor overactivity (NDO). Dosing is based on the principle of "as much as needed, with as little as possible." Antibody formation to the toxin does not seem to be of relevance in current clinical practice [12].

BoNTA is commonly injected cystoscopically into the sub-urothelium/detrusor over 30–50 sites with or without inclusion of the trigonal area. Including the trigone seems to make a difference in patient perception of symptoms including continence with no significant change in urodynamic parameters in the NDO group. It is now standard practice to inject BoNTA via a flexible cystoscope as an outpatient procedure. It is important that patients are taught CIC or at least warned about the risk of requiring CIC after injection of BoNTA.

In neurogenic bladder with outlet resistance, injection of BoNTA to urethral sphincter is effective in reducing the intravesical pressure and PVR for approximately 3–9 months [13].

Catheters in Neurogenic Bladder

There are three modes of catheterization available:

1. Urethral catheterization
2. Suprapubic catheterization
3. Clean intermittent catheterization (CIC or CISC depending on patient ability)

Of the three, CIC or CISC is ideal in long-term catheterization such as in neurogenic bladder. Long-term catheterization, be it urethral or suprapubic, is fraught with a lot of morbidity. In addition to infection, bleeding, need for regular bladder washouts, bladder stone formation, and risk of cancer, long-term catheters result in erosion of the urethra and poor bladder compliance. Suprapubic catheters avoid the problem of urethral erosion and can result in lesser episodes of infection.

The goal of CIC is to achieve emptying of the bladder on a regular basis so that it avoids infection and incontinence. It is particularly important in those with urgency-related incontinence who are treated with antimuscarinics or botulinum toxin.

CIC interval is usually directed by the patient's urine volume, typically aiming for residuals of less than 400–500 ml at each insertion, as this is the volume tolerated physiologically until voiding is triggered in the neurologically intact human.

CIC is the preferred option in treating patients with retention or large post-void residuals following acute disk herniation and major pelvic surgery or in the postpartum period, where spontaneous recovery of detrusor function is anticipated in due course. While PVC and latex catheters are acceptable for short-term use, in those with neurogenic bladder, CIC with hydrophilic catheters is the preferred option [14].

CIC not only affords freedom from catheters and bags but also allows unimpeded sexual intercourse. It empowers these patients with autonomy that allows them to take up or continue with their regular jobs and lifestyle.

Surgical Management in Neurogenic Bladder

Surgery for refractory urgency incontinence and poor bladder compliance involves augmenting bladder capacity – either by detrusor myomectomy or augmentation cystoplasty. This can increase the bladder capacity and decrease the detrusor pressure. In patients with outlet resistance, catheterization either indwelling or intermittent is ideal. A bladder neck or sphincter incision can help in relieving the urethral resistance but carries the risk of bladder neck contracture.

Urinary diversion is usually employed to protect the upper tract if other measures are not helpful or if the patient is unwilling to perform CIC. A continent urinary stoma can be constructed using the Mitrofanoff principle.

Patients with stress urinary incontinence may be treated by sling procedures using autologous tissue. The goal in these patients is to prevent incontinence by obstruction so that they can then be managed subsequently with CIC.

Fowler's Syndrome

Described in 1988, Fowler's syndrome is urinary retention due to a poorly relaxing urethral sphincter and abnormal electromyographic activity of the urethral sphincter, in the absence of a neurological cause [15]. It typically occurs in young women in the second or third decade of life and has known association with polycystic ovaries. It is rare with no particular precipitating factors, although many recall a trigger event (like childbirth, surgery, or dental work). Some of these women often have a prior history of infrequent or prolonged voiding with impaired bladder sensation. They usually present with inability to void for a day or more with no urgency.

Videourodynamic study with sphincter electromyography can be helpful, as it can show the normal function of the bladder neck and proximal urethra during voiding with failure of opening of midurethra. Clare Fowler and colleagues described concentric needle electromyographic findings in the urethral sphincter of these patients, which were abnormal, resulting in a poorly relaxing urethral sphincter.

It is not entirely clear if Fowler's syndrome is a distinct entity but these women do present with urinary retention and respond to sacral neuromodulation. While CISC is a useful therapeutic tool, the majority of these women report a painful or gripping sensation upon removal of the catheter, preferring instead a suprapubic catheter. Botox injection of external urethral sphincter has been attempted but in vain. Sacral neuromodulation appears to be effective in restoring normal voiding in 70 % of these patients [16].

New Developments

Nerve growth factor (NGF), produced by the urothelium and bladder smooth muscle, is involved in the pathogenesis of several neurological problems. NGF levels in urine are elevated in children with neurogenic bladder as well as in those with OAB. NGF levels decrease in those who respond to BoNTA therapy. The precise correlation between this and urodynamic variables like maximum bladder capacity and compliance is yet to be established. Once we understand the relationship better, we may be able to use them as a biomarker for disease burden as well as predicting response to therapies [17]. NGF is also being studied in regeneration of neural tissue following injury.

In the surgical arena, there has been a revival of interest in an earlier attempted concept of lumbar to sacral nerve re-routing in spina bifida patients to improve bowel and bladder function [18].

Conclusion

In managing patients with neurogenic bladder, the clinician should tailor the treatment options with the aim to improve the patients' quality of life and simultaneously protect the upper tract and renal function. The options can vary from conservative to pharmacological to surgical and the clinician's knowledge of all the available options and the new developments will be helpful in planning the management algorithm.

Acknowledgment I am grateful to Prof. Chris Chapple, Consultant Urological Surgeon, Sheffield Teaching Hospitals, and Honorary Professor, Sheffield Hallam University, UK, for his review of the manuscript.

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Voiding Dysfunction

10

Sanjay Sinha

Voiding dysfunction in women is a common but poorly characterized problem [1]. The label of voiding dysfunction is not really a "diagnosis" but represents a subset of patients with lower urinary tract dysfunction with either symptoms or evaluation suggestive of a problem with the voiding phase of micturition.

Terminologies and Evaluation

The ICS-IUGA terminologies document defines voiding dysfunction by symptoms and urodynamic investigations as an "abnormally slow and/or incomplete micturition" [2]. Hence, the condition affects primarily voiding rather than storage although many patients may have associated storage symptoms. Some patients may present exclusively with storage symptoms. Urodynamically, it is a "reduced urine flow rate and/or presence of a raised PVR and an increased detrusor pressure" diagnosed by a simultaneous measure of pressure and flow [2].

Voiding dysfunction broadly speaking can result from poor bladder contractility or outlet obstruction. Abnormal bladder contractility during voiding may be classified as detrusor underactivity or acontractility. ICS-IUGA defines detrusor underactivity as "Detrusor contraction of reduced strength and/or duration, resulting in prolonged bladder emptying and/or a failure to achieve complete bladder emptying within a normal time span" [2]. While acontractility is defined as a detrusor that "cannot be observed to contract during urodynamic studies resulting in prolonged bladder emptying and/or failure to achieve complete bladder emptying within a normal time span" [2].

Furthermore, the term "detrusor underactivity" or "detrusor acontractility" implies that the failure of the bladder to contract stems from abnormalities of the detrusor alone. This fails to recognize that afferent (sensory), efferent (including myogenic factors), and central CNS factors can all be responsible in varying degrees in different patients. Hence, a more generic term "bladder underactivity" has been proposed [3, 4].

Dysfunctional voiding is defined as "an intermittent and/or fluctuating flow rate due to involuntary intermittent contractions of the peri-urethral striated or levator muscles during voiding in neurologically normal women" [2]. Classical detrusor sphincter dyssynergia that occurs in neurogenic bladder is outside the scope of this discussion.

The limitation of the aforementioned definition is readily apparent on further analysis. While the definition mentions abnormally slow and/or incomplete micturition, cutoffs for what constitutes normal flow or emptying are not well defined. The time duration in which the normal

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A. Tamilselvi, A. Rane (eds.), *Principles and Practice of Urogynaecology*,
DOI 10.1007/978-81-322-1692-6_10, © Springer India 2015

bladder should empty itself is also not clearly defined, making it difficult to apply the yardstick of "complete bladder emptying within a normal time span." Moreover, applicability of Western nomograms to Indian patients has pitfalls [5]. There are limitations to the ICS documentation for dysfunctional voiding [6]. Specifically, not all patients with dysfunctional voiding without an underlying neurological problem will show an intermittent flow and the terminologies documents need to make allowance for this finding.

There are no universally accepted criteria for defining bladder outlet obstruction in women [1]. In adult men, prostatic obstruction serves as an excellent model of reversible bladder outlet obstruction and this has helped improve understanding of bladder physiology in men. It is, however, universally accepted that criteria for men cannot be extrapolated to women since many women void with pelvic floor relaxation without a major detrusor contraction. Blaivas and Groutz defined a nomogram for women recommending that women with a maximum flow rate (tube-free) of <12 ml/s and $P_{det.}$ Q_{max} of >20 cm H_2O should be categorized as obstructed [7].

All women with suspected voiding dysfunction must be evaluated in detail by a clinical assessment and judicious investigations (see Table 10.1). Those with high-risk factors such as hydronephrosis, renal dysfunction, or reflux must undergo a full urological evaluation and detailed urodynamics since some of these women might be at risk for long-term renal dysfunction (see Fig. 10.1).

Table 10.1 Basic clinical evaluation for voiding dysfunction

History
Urinary tract: details of storage and voiding symptoms, urinary infections
Gastrointestinal tract: any constipation or encopresis
Neurological evaluation: neurological screening, lower limb and higher function assessment
Gynecological evaluation: assess for symptoms of vaginal or uterine disease, obstetric history
Others: history of relevant surgery
Physical examination
Genitourinary: meatus, labia and vagina, bladder, kidneys
Gastrointestinal: rectal examination, fecal incontinence
Neurological system: specific attention to anal sphincter tone, bulbocavernosus reflex, perianal sensation, gait, spine
Investigations
Bladder diary, urine examination, blood glucose
USG for the bladder with prevoid and post-void bladder volume
Uroflow with EMG, invasive urodynamics or videourodynamics
MRI of the spine, micturating cystourethrogram

Etiology of Voiding Dysfunction

There are two broad categories of problems that can present with voiding dysfunction (see Table 10.2). These are detrusor muscle weakness and bladder outlet obstruction. Iatrogenic voiding dysfunction may occur following pelvic surgery such as stress urinary incontinence surgery. Finally, storage abnormalities such as detrusor overactivity may occasionally present with symptoms suggestive of voiding dysfunction.

Detrusor Underactivity and Acontractility

Detrusor muscle strength can range from normal to acontractility in a continuum. Gross detrusor dysfunction can easily be identified by a constellation of clinical and urodynamic findings including poor flow, large residuals, and obvious detrusor muscle weakness on urodynamics. Unfortunately, many women present with less obvious features.

Detrusor muscle weakness is an important cause of voiding dysfunction which may exist in isolation or in combination with bladder outlet obstruction. Poor detrusor muscle strength varies in a continuum from mild weakness (detrusor underactivity) to a total loss of contractility (acontractility) (see Fig. 10.2). There are several causes for detrusor underactivity (see Table 10.2). Detrusor underactivity is widely prevalent in older women with an estimated prevalence of 12–45 % [4]. It has long been observed that older

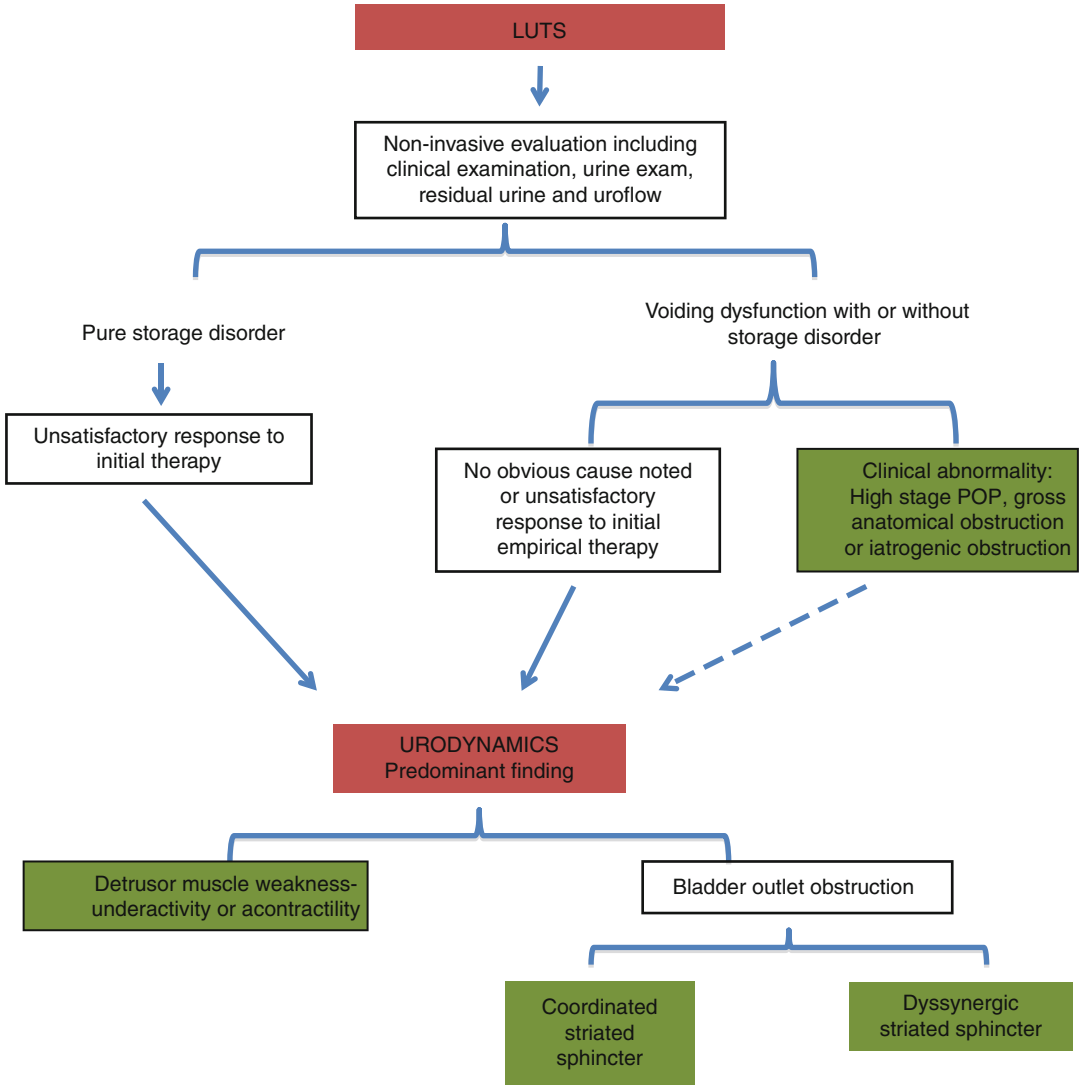

Fig. 10.1 Algorithm for evaluation of voiding dysfunction in women

individuals are more likely to present with underactivity. Research indicates up to two-thirds of elderly nursing home residents show detrusor underactivity. Bladder biopsy reveals changes in the detrusor muscle including axonal degeneration, loss of muscle fibers, and fibrosis [8].

Detrusor underactivity can also occur due to untreated bladder outlet obstruction or retention due to any other cause. Chronic or severe acute stretch injury can lead to detrusor dysfunction. Postpartum urinary retention is a classic example of such injury.

Diabetes is an important medical cause of underactive detrusor. The classical clinical picture of patients with florid diabetic cystopathy is one of acontractility. Such patients usually have long-standing diabetes of more than 10 years' duration and often have other evidence of diabetic peripheral neuropathy. However, studies have shown a higher prevalence of other patterns such as overactive bladder in patients with diabetic cystopathy [9]. Diabetes impacts all levels of neuromuscular control of the lower urinary

Table 10.2 Etiological classification and diagnosis of voiding dysfunction in women

A. Detrusor muscle weakness (underactive or acontractile detrusor)
1. Idiopathic
2. Aging
3. Drug induced
4. Untreated long-standing obstruction
5. Diabetic cystopathy
6. Neurogenic dysfunction, e.g., after a radical hysterectomy
7. Postpartum retention
8. Severe fibrosis of the bladder wall
B. Bladder outflow obstruction
I. With coordinated sphincteric activity (presumed or documented)
1. Pelvic organ prolapse
2. Urethral stricture
3. Functional bladder neck obstruction
4. Iatrogenic following urethral or other pelvic surgeries
5. Large fibroid
6. Urethral diverticula
7. Urethral leiomyoma
II. With dyssynergic sphincteric activity
A. No underlying neurological cause apparent
B. Neurological cause present
C. Classical detrusor sphincter dyssynergia

tract. There is altered afferent sensation possibly linked to changes in the urothelium and detrusor. Afferent and efferent nerves demonstrate degenerative changes in function. Depending on the relative impact on different aspects of lower tract dysfunction, one can expect a wide variety of bladder manifestations [10].

Several drugs can impact lower urinary tract function either by exacerbation of a preexisting voiding dysfunction or by inducing acute-onset symptoms in otherwise normal individuals [11]. Drugs with anticholinergic side effects such as antidepressants and antipsychotic drugs as well as calcium channel blockers are some of the examples. Older women are more likely to be impacted by these side effects due to alterations in neurotransmission, receptor sensitivity, and blood-brain barrier defects. Also, elderly women are more likely to have preexistent lower tract dysfunction such as underactive detrusors that might make them especially susceptible to the adverse effects.

Neurogenic lower urinary tract dysfunction can produce a variety of effects on the lower urinary tract. Classically, lesions above the brainstem do not have an impact on detrusor contractility. Some studies have shown up to 6 % of patients with CVA have acontractile detrusors [12]. It is difficult to judge what proportion of this might be due to preexisting detrusor dysfunction in this elderly age group. Detrusor underactivity may conceivably occur following CVA.

Unrecognized postpartum retention can be a disaster for the bladder with short- or long-term damage. This is now a rare event in the developed world. Pifarotti et al. noted postpartum retention in 105 out of 11,108 (1 %) women undergoing a vaginal delivery. They defined postpartum retention as inability to void within 6 h of delivery. On multivariate analysis, only vacuum-assisted delivery and uterine fundal pressure during the second stage of labor were noted to be independent risk factors [13].

Protracted second stage of labor and vacuum delivery were noted to be risk factors in another large study [14]. In this study, 55 women (0.18 %) developed postpartum retention. Of these, two-thirds recovered by 2 weeks and the remaining by 4 weeks. However, the author has seen anecdotal instances of long-term voiding dysfunction. This is unusual in the Western world where strict labor room protocols are enforced and women are identified before they develop florid bladder damage.

Bladder Outlet Obstruction with Coordinated Striated Sphincter Activity (Synergic Striated Sphincter)

Pelvic organ prolapse (POP) is an important cause for lower urinary tract dysfunction in women. However, one must remember that the only symptom of feeling a vaginal bulge shows consistent correlation with POP. No urine symptoms have been shown to be consistently associated with POP. The situation might be different in women who primarily present with voiding dysfunction and have severe prolapse. Uncorrected

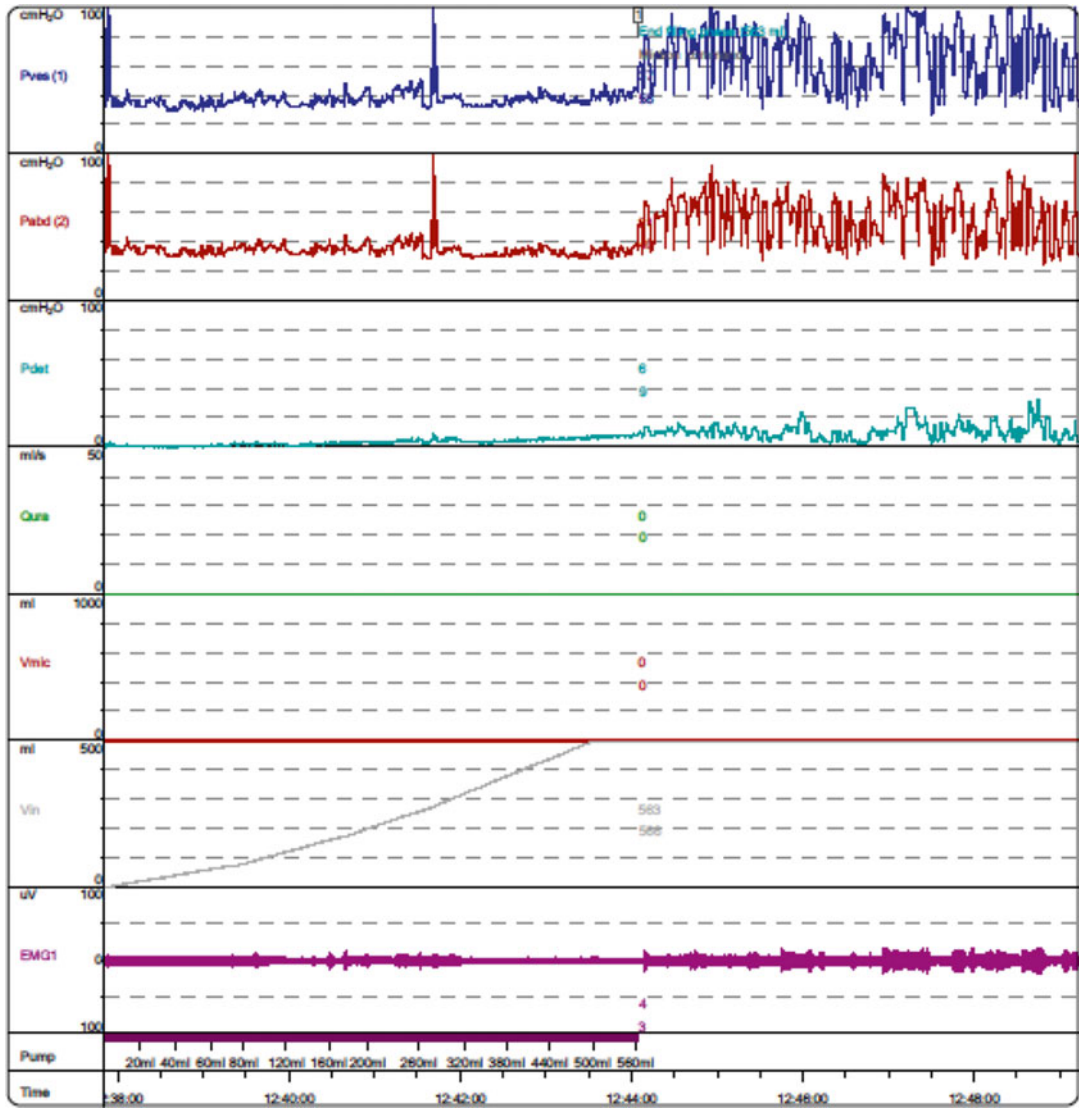

Fig. 10.2 Acontractile detrusor in a 67-year-old diabetic woman with voiding difficulty and storage symptoms for 2 years. One must exercise caution in making this diagnosis. One may find "acontractility" in an uncooperative (but otherwise normal) woman

prolapse may alter the dynamics of the lower urinary tract and elevated residuals are common in such women. In women with significant POP, which usually implies that the prolapsing organ is beyond the introitus, an important clinical question is whether correcting the prolapse will improve voiding. Such women may habitually reduce the prolapse before voiding [15]. One method of resolving this issue is to use a pessary and perform urodynamic evaluation or flow tests with and without the pessary. Women with obvious improvement in lower urinary tract function are likely to improve with surgery (see Fig. 10.3).

Genuine urethral stricture in women is an uncommon entity. Conversely, a large number of women continue to be subjected to urethral dilatation for various forms of lower urinary tract dysfunction.

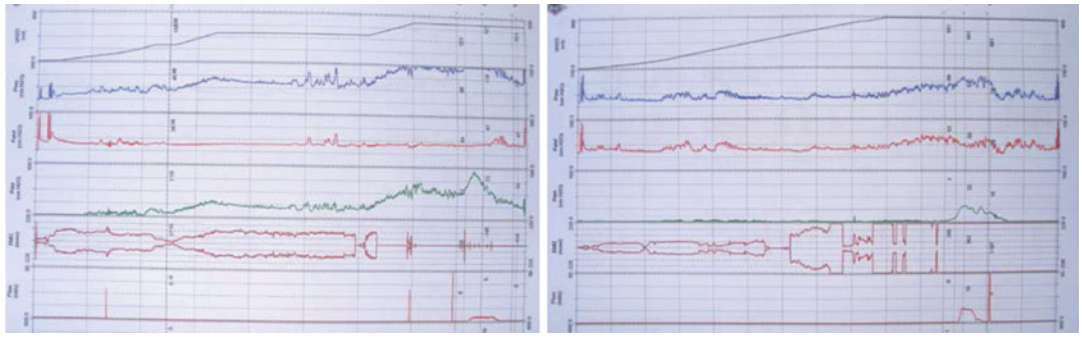

Fig. 10.3 Urodynamics evaluation without (*left*) and with (*right*) a pessary in a 60-year-old woman with grade III pelvic organ prolapse and voiding dysfunction. Substantial improvement in voiding function (reduced voiding detrusor pressure and better flow rates) was noted upon placement of a pessary. She had complete resolution of voiding dysfunction following surgery for prolapse

Functional bladder neck obstruction is less common in women since the bladder neck smooth muscle is not as well organized into a sphincter in women as compared to men (see Fig. 10.4). As such, in most series only a small number of women have urodynamically confirmed functional bladder neck obstruction [16]. However, in a recent prospective multicenter study from China, the commonest form of voiding dysfunction was noted to be functional bladder neck obstruction. It is unclear whether this result represents methodological differences, variations in diagnostic criteria, or a genuine difference in prevalence [17].

Large uterine fibroids can have an impact on urinary tract function. This is more likely in women, when the uterine size is over 12 weeks of gestation size. These women often complain of voiding difficulty and the symptoms usually improve following surgery [18]. Isolated case reports have found leiomyoma of the urethra as a cause of voiding difficulty [19].

Dysfunctional Voiding

The ICS-IUGA terminology defines dysfunctional voiding as "an intermittent and/or fluctuating flow rate due to involuntary intermittent contractions of the peri-urethral striated or levator muscles during voiding in neurologically normal women" [2]. As discussed in a previous review, this author has made a case for modifying this definition to reflect the two key components of dysfunctional voiding, namely, dyssynergia of the striated urethral sphincter-pelvic floor complex and lack of a clear neurological etiology [6]. In a small but significant proportion of these patients, neurological abnormalities may manifest subsequently [20]. Hence, it is important to evaluate these patients carefully for subtle clues to an underlying neurological abnormality and to perform additional assessment judiciously, including MRI of the spine or other forms of nerve-muscle evaluation. In some women, dysfunctional voiding may represent faulty toilet training in childhood. A related condition in which women with polycystic ovarian disease have urinary retention due to an increased sphincteric tone that has hormonal associations has been termed "Fowler's syndrome" [21].

Most women will have a "staccato flow" with fluctuations in excess of the square root of the maximum flow (see Fig. 10.5). Some women may have slow but non-staccato flow or even normal flow rates [6]. Hence, in suspected dysfunctional voiding, urodynamic evaluation is important for diagnosis and management.

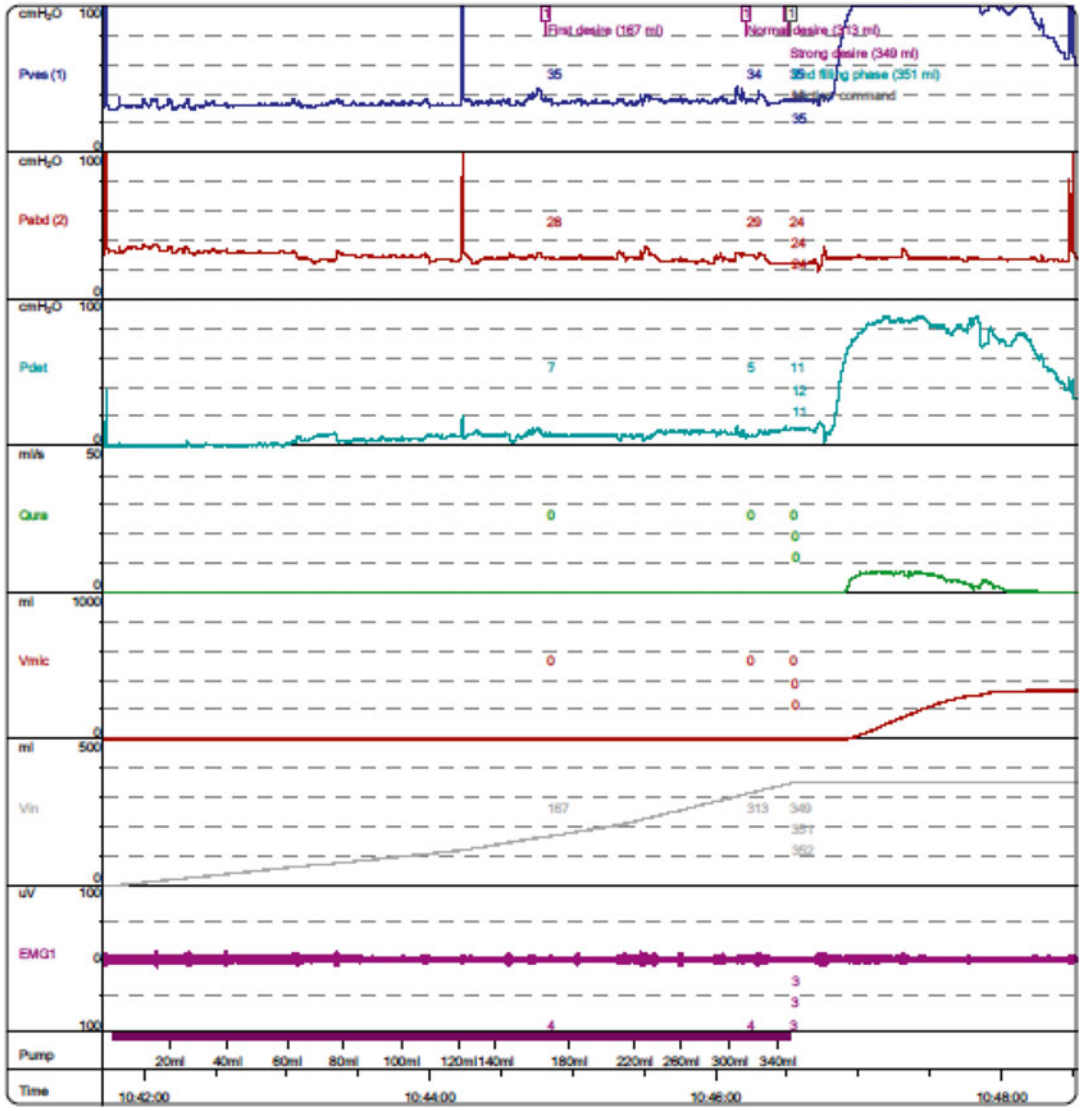

Fig. 10.4 Functional bladder neck obstruction in a 46-year-old nondiabetic woman with voiding difficulty, frequency, and nocturia. It is important to search for consistent findings across multiple cycles of testing preferably including a tube-free uroflow with EMG

Detrusor Sphincter Dyssynergia

Classical detrusor sphincter dyssynergia that occurs in women with neurological disease represents a significant urological problem and a full discussion on this condition is outside the scope of this chapter. A brief summary of the usual neurological features of patients with classical neurogenic bladder follows.

Patients with lesions above the brainstem typically present with detrusor overactivity with coordinated sphincters. However, patients with Parkinsonism may show a delayed ability to relax the striated sphincter presenting with a form of voiding dysfunction. This delayed relaxation has also been termed "bradykinesia" of the striated sphincter [22]. Parkinsonism occurs in 3 % of both men and women above the age of 65

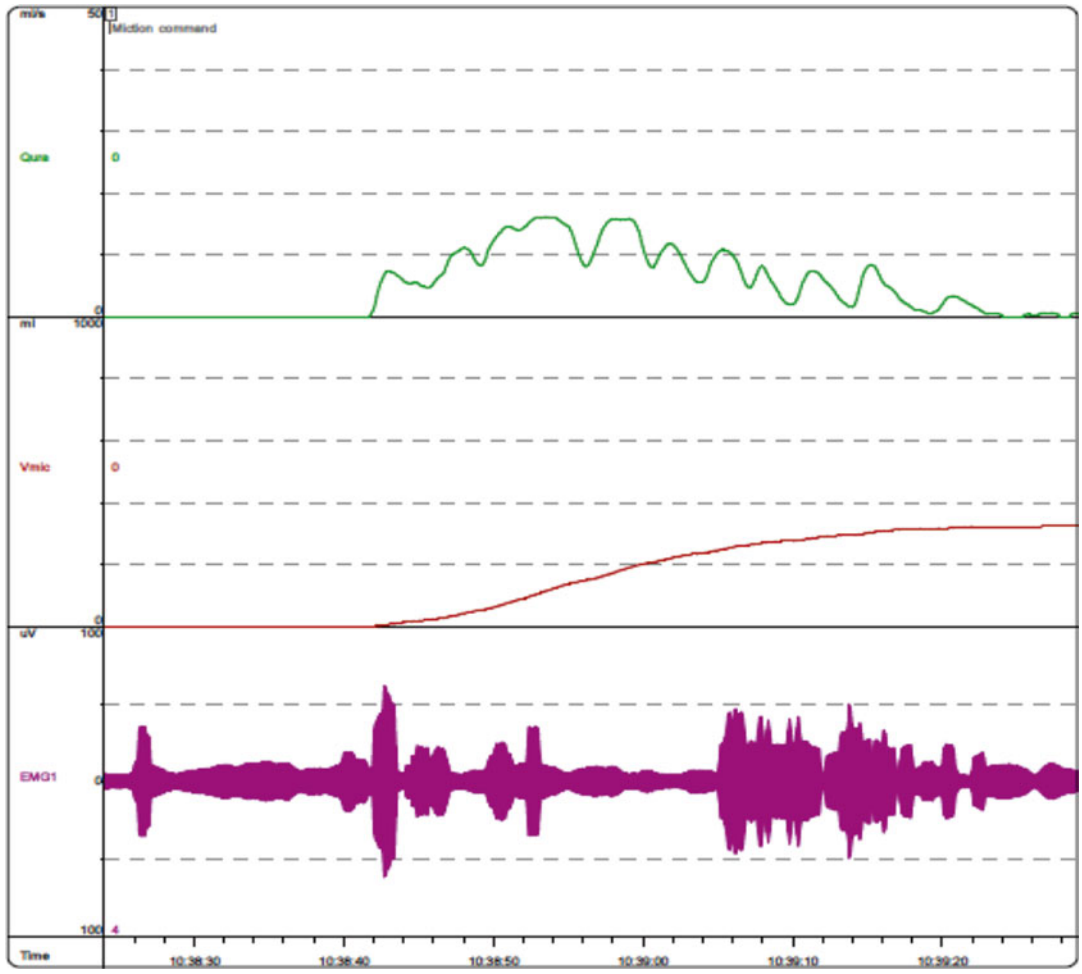

Fig. 10.5 Staccato voiding in a 40-year-old woman with voiding difficulty. Note the EMG contractions during voiding. This finding is only significant if it is clinically consistent and documented on repeated assessment

years. Patients with cerebrovascular accidents have synergic sphincteric activity. In some patients with severe detrusor overactivity, the sphincteric response to phasic contractions may be confused as dyssynergia, and this has been termed "pseudo-dyssynergia." True dyssynergia does not occur in patients with cerebrovascular accidents. Multiple sclerosis patients usually show detrusor overactivity but about half of these patients with storage abnormalities have dyssynergic voiding [23, 24].

Patients with spinal cord injuries most often show both storage and voiding phase abnormalities (see Fig. 10.6). In lesions above T6 level, there is usually detrusor overactivity with dyssynergia of both the smooth and striated sphincter. In lesions between T6 and S2, the striated sphincter alone is most often dyssynergic. In patients with sacral lesions, the sphincter classically demonstrates a fixed and increased tone (non-relaxing sphincter) along with acontractility of the detrusor and an incompetent smooth sphincter. One must however remember the dictum that the level of neurological lesion is not a reliable guide for management. Bladder management in patients with neurogenic bladder is based on urodynamic findings rather than neuroimaging or neurodiagnosis [24].

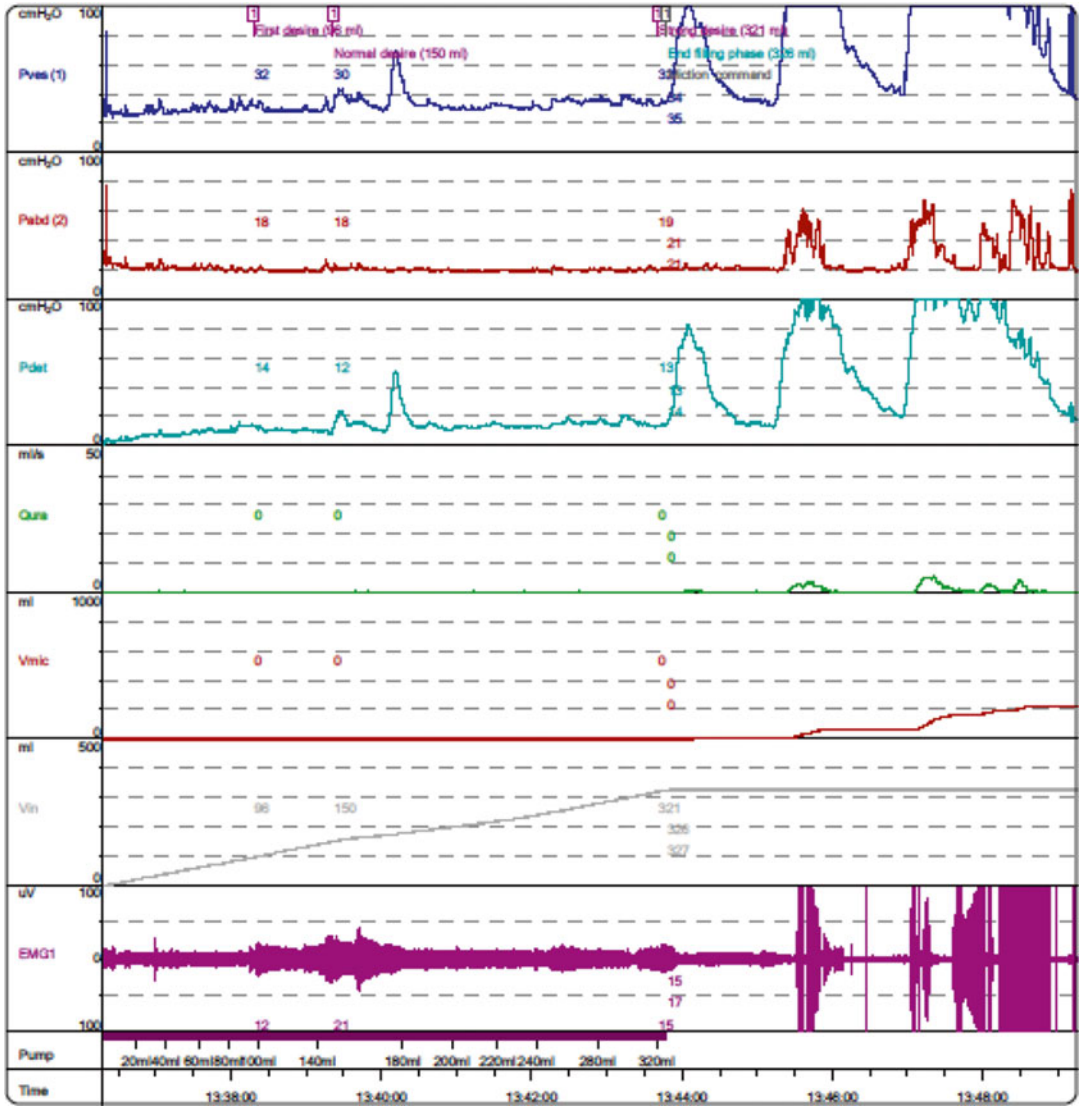

Fig. 10.6 Classical neurogenic bladder in a 16-year-old girl operated for tethered cord at the age of 7 years. Note the detrusor overactivity and reduced compliance during storage and the severe bladder outlet obstruction with classical detrusor sphincter dyssynergia during voiding

Management of Voiding Dysfunction

Treatment of voiding dysfunction is dependent on the clinical setting, the type of abnormality noted, and the risk for consequential damage such as back-pressure effects or infection (see Table 10.3).

Detrusor Underactivity and Acontractility

In patients with an underactive detrusor, lifestyle changes can be quite useful [25]. Avoiding overdistension of the bladder is the key. Fluid restriction and reduction in intake of dietary diuretics

Table 10.3 Treatment of female voiding dysfunction

Timed voiding
Lifestyle changes including fluid restriction
Pharmacotherapy
Surgery for pelvic organ prolapse
Clean intermittent self-catheterization
Urethral dilatation
Optical urethrotomy
Intraurethral botulinum toxin
Neuromodulation
Surgical reconstruction including urinary diversion, augmentation cystoplasty, or other major lower tract reconstructive surgeries

such as tea, coffee, and alcohol can reduce the overall urine volume. This should be combined with timed voiding every 2–3 h during the day. Patients should be instructed to void "by the clock" rather than waiting for a desire to void. Many of these patients have impaired bladder sensation and their strong desire to void may be delayed. Waiting for bladder sensations to trigger a void can lead to progressive overdistension of the bladder and ongoing detrusor damage. The bladder may contract best at a fill of about 300–350 ml and hence limiting the bladder volume to this level can help. Double and triple voiding 20 min after the initial void can also help in minimizing residual urine [25]. There are no medications that can reliably increase the bladder's ability for a coordinated contraction. Although bethanechol is a popular drug, there is no objective evidence to suggest benefit and the drug should not be used for this purpose [26]. Diabetic patients should observe good glycemic control to reduce the progressive neuromuscular damage due to hyperglycemia. In patients with severe underactivity or acontractility, clean intermittent self-catheterization is the solution [25, 27]. Patients are instructed to empty their bladder five times a day using a small catheter that they are taught to self-insert by a clean (as contrasted to a sterile) technique. Catheters can safely be reused several times.

Bladder Outlet Obstruction with Coordinated Sphincters

In women with bladder outlet obstruction with coordinated sphincters, one must address the primary cause of obstruction. Those with obvious anatomical narrowing of the urethra (in extreme cases, a urodynamics study will, in fact, not be possible), the urethra can be dilated with a successful and often durable outcome. However, urethral dilatation is a much misused therapy, and women with all and sundry urine symptoms, are offered urethral dilatation without rationale. Used as such, this therapy is either ineffective or has limited or short-term efficacy at best and can lead to long-term urethral incompetence and stress incontinence at worst. In women with true functional bladder neck obstruction, alpha blockers are often effective and should be tried [28]. Not all women with a functional bladder neck obstruction on urodynamics respond to alpha blockers and this begs the question whether the diagnosis is correct (in other words, has a dyssynergic void been missed) or is the obstruction too severe for oral medication to work (just as alpha blockers do not relieve obstruction in all men with severe functional bladder neck obstruction). A clear answer is currently not forthcoming from literature since most urologists are reluctant to perform a surgical correction for functional bladder neck obstruction for the fear of producing iatrogenic incontinence. There are sporadic reports in literature regarding bladder neck incision in women either by use of a "cold" knife of some kind (under vision or blind) or by the use of electrocautery or laser. A pediatric scope has been recommended by some authors in view of the shorter length of the female urethra and the challenges in obtaining good vision and precise incision using regular instruments designed for adult males [29].

In women with high-grade prolapse, surgical repair of prolapse may resolve urinary symptoms. However, one must counsel such

women carefully. A preoperative urodynamics done with and without vaginal pessary can sometimes predict the resolution of urinary symptoms. One must recollect that the only symptom that reliably correlates with pelvic organ prolapse is a feeling of bulge in the vagina. There is limited evidence that women with large fibroids may benefit from surgery for the fibroid. Again, one must be careful in prognosticating such women since it is by no means certain that removing a fibroid will resolve urinary symptoms. A better approach is to tackle the fibroid on merits and observe for any benefit in urinary symptoms [30].

Iatrogenic urethral obstruction following surgery for stress urinary incontinence is a subject in itself and beyond the scope of this chapter. Briefly, the diagnosis of such an obstruction is challenging since women can present with an array of symptoms, not all of which are voiding symptoms. Many women have storage symptoms alone and are noted to have poor voiding efficiency only on investigations. Certainly, any woman with new-onset voiding difficulty following stress incontinence surgery needs to be carefully assessed for iatrogenic obstruction. No single test can prove obstruction (unless the obstruction is "gross," which is unusual). A constellation of suggestive symptoms and signs along with poor uroflow, increase in residuals, worsening of voiding dynamics (in those who had a preoperative urodynamics study), and cystoscopic evidence of anatomical obstruction can suggest iatrogenic obstruction. Intervention in such women is controversial. In those with gross obstruction, the tape can be divided, usually to one side of the midline. Early intervention at or before 3 weeks might reduce the odds of long-term persistent storage symptoms [25]. Satisfactory voiding is restored in about 75 % and about 40 % have recurrent stress urinary incontinence following tape division [31]. Hence, recurrence of stress incontinence is not a certainty following tape division. In most women, a formal urethrolysis is not necessary and the entire tape need not be removed, unless there is associated sepsis.

Dysfunctional Voiding

Patients with dysfunctional voiding need to be triaged on the basis of their presentation. Those patients with high-risk markers such as reflux, hydronephrosis, marked voiding difficulty, or renal dysfunction should undergo urodynamic evaluation at the outset [6]. Patients with primarily storage symptoms can undergo initial management based on uroflow with surface electromyography recording. However, one must have a low threshold for offering urodynamics in those who fail to respond satisfactorily. All patients with dysfunctional voiding must undergo a basic neurological evaluation and select patients who need more detailed assessment including MRI to search for underlying neurological abnormality [20].

All patients need to be educated about their problem. Some patients may benefit from biofeedback and aggressive constipation care. Biofeedback consists of repeated voiding with simultaneous auditory or visual feedback to the patient regarding striated sphincter activity. In some women, this may help to resolve a "habitual" striated sphincter dyssynergia [32]. Alpha-adrenergic blockers are usually not useful but can be tried in patients with safe voiding pressures [6]. Patients with very high voiding pressures can potentially damage their upper tracts and such patients should not be offered alpha-adrenergic blockers since it is unlikely to work and a more effective treatment option must be chosen.

Botulinum toxin injection into the sphincter has been tried in dysfunctional voiding with mixed results. The injection is usually given periurethrally in women. Fifty to hundred units are diluted in saline and injected into four sites representing four quadrants of a circle [33]. The effect of the injection starts in about 1–2 weeks and can last for several months.

Sacral neuromodulation can also be useful in select patients. In women with the Fowler's syndrome, 78 % of women continued to void spontaneously 10 years following InterStim sacral neuromodulation implantation [34]. However, the procedure carries a high revision rate of about 30–50 % and failure rate of about 30 %.

Clean intermittent self-catheterization is the mainstay of therapy and helps bypass the sphincteric mechanism.

Detrusor Sphincter Dyssynergia

There is no oral medication for classical detrusor sphincter dyssynergia. Treatment revolves around clean intermittent self-catheterization [25]. Additional urodynamic and clinical findings need to be addressed individually. Many patients have concomitant storage pressure abnormalities which need to be managed. Storage abnormalities may consist of various combinations of neurogenic detrusor overactivity, reduced compliance and reduced capacity. Anticholinergics are the first-line treatment with intra-detrusor botulinum toxin and augmentation cystoplasty reserved for nonresponders. Sphincterotomy and stents are not a good option in women. Isolated reports suggest that intrasphincteric botulinum toxin might help in reducing voiding pressures but experience with this is not uniformly good [35]. In women with limited ambulation or other severe comorbidities, an indwelling catheter may represent an acceptable salvage option.

Conclusion

Managing voiding dysfunction in women is complex as identification of the causative factor itself is composite, varying from anatomical abnormalities to neurological (either peripheral or central). The clinical evaluation including urodynamic assessment has a major role in the management algorithm. The management options are varied and not necessarily aimed at addressing the pathology but rather the symptom mostly. In a significant proportion of patients' catheterization, either clean intermittent or continuous drainage forms the mainstay of treatment.

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Bladder Pain Syndrome

11

Arjunan Tamilselvi

Bladder pain syndrome is the occurrence of persistent or recurrent pain perceived in the urinary bladder region, accompanied by at least one other symptom, such as pain worsening with bladder filling daytime and/or night time urinary frequency. The symptoms present with no proven infection or other obvious local pathologies [1]. The condition also known as painful bladder syndrome (PBS) and/or interstitial cystitis (IC) is a chronic debilitating condition of uncertain etiology, and both the terminologies have been used interchangeably (PBS/IC). Guy Hunner is credited with the original description of this condition (1915) [2], but it appears to have been described earlier by Nitze et al. [3]. In keeping with the current definition, the terminology bladder pain syndrome (BPS) will be used in this chapter, instead of PBS/IC.

BPS predominantly affects women with a 9:1 female to male ratio [4]. Women between 40 and 60 years of age are commonly affected though it has been reported in children as well [5]. The quality of life in women with BPS is significantly affected with both daytime and nighttime frequency, pain, and sleep deprivation leading to depression.

The prevalence rate of BPS varies between different countries, and in the general population, it is quoted around 60 per 100,000. This possibly is not a true estimate and the major hurdle in assessing the prevalence is the confusion over the definition and the lack of an accurate clinical diagnostic criterion [6].

This chapter aims to evaluate the diagnostic criteria, pathophysiology of this condition and its management.

Definition/Nomenclature

Defining BPS has been problematic, since the type and severity of symptoms can vary and there has been no clarity on the exact pathological process or etiology of the condition.

The National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK) diagnostic criteria developed in August 1987 defined that clinically the patient must have bladder pain and/or severe urinary urgency with frequency, stable bladder at cystometry, and sterile urine. In addition, cystoscopy shows glomerulations or mucosal splitting (ulcers) [7]. The NIDDK diagnostic criterion was too restrictive for clinical use and alternate clinical diagnostic criteria were proposed by different groups. Hanno et al. stated that by using the NIDDK criteria, more than 60 % of patients with IC could be misdiagnosed [8] (Table 11.1).

The definition proposed by the standardization subcommittee of the International Continence Society [1] (2012) states the

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A. Tamilselvi, A. Rane (eds.), *Principles and Practice of Urogynaecology*,
DOI 10.1007/978-81-322-1692-6_11, © Springer India 2015

Table 11.1 NIDDK definition of interstitial cystitis (1987)

Inclusion criteria
1. Cystoscopy – glomerulations and/or classic Hunner's ulcer
2. Symptoms – bladder pain and/or bladder urgency
Exclusion criteria
1. Bladder capacity greater than 350 cc on awake cystometry
2. Absence of an intense urge to void with the bladder filled to 100 cc during cystometry using a fill rate of 30–100 cc/min
3. Demonstration of phasic involuntary bladder contractions on cystometry using the fill rate described in number 2
4. Duration of symptoms less than 9 months
5. Absence of nocturia
6. Symptoms relieved by antimicrobials, urinary antiseptics, anticholinergics, or antispasmodics
7. Frequency of urination while awake of less than eight times a day
8. Diagnosis of bacterial cystitis or prostatitis within a 3-month period
9. Bladder or ureteral calculi
10. Active genital herpes
11. Uterine, cervical, vaginal, or urethral cancer
12. Urethral diverticulum
13. Cyclophosphamide or any type of chemical cystitis
14. Tuberculous cystitis
15. Radiation cystitis
16. Benign or malignant bladder tumors
17. Vaginitis
18. Age less than 18 years

Reproduced from Ref. [7]

condition as "the complaint of suprapubic pain related to bladder filling, accompanied by increased day time and night time urinary frequency in the absence of proven urinary tract infection or other pathology" and suggested the term IC be restricted to specific cystoscopic and histological features [9] (the features were however not defined). The condition came to be known as PBS/IC or IC/PBS. The diagnostic sensitivity for PBS using these criteria was only 61 % [10]. At the clinical proceedings of the Association of Reproductive Health Professionals in 2007, the definition of PBS/IC was proposed as: "Pelvic pain, pressure or discomfort related to the bladder, typically associated with persistent urge to void or urinary frequency, in the absence of urinary infection or other pathology" [11].

Revisions have been proposed to the terminology by several clinicians and organizations interested in clarifying this issue. This includes the NIDDK Research Symposium: Frontiers in Painful Bladder Syndrome and Interstitial Cystitis in 2006 proposing the terminology bladder pain syndrome (BPS), the NIDDK Multidisciplinary Approach to the Study of Chronic Pelvic Pain Network (2008), and the American Urological Association (AUA) IC/BPS Guideline 2011 attempting to redefine it [12].

The European Association of Urology (EAU) 2012 Guidelines on Chronic Pelvic Pain and the updated International Association for the study of Pain (IASP) Taxonomy 2012 defines BPS as follows: "Bladder pain syndrome is the occurrence of persistent or recurrent pain perceived in the urinary bladder region, accompanied by at least one other symptom, such as pain worsening with bladder filling, daytime and/or night-time urinary frequency. There is no proven infection or other obvious local pathology." Bladder pain syndrome is often associated with negative cognitive, behavioral, sexual, or emotional consequences as well as with symptoms suggestive of lower urinary tract and sexual dysfunction [1, 13]. The European Society for the Study of Interstitial Cystitis (ESSIC) proposed that the definition incorporate features: "Chronic (6 months or more) pelvic pain, pressure or discomfort perceived to be related to the urinary bladder accompanied by at least one other urinary symptom like persistent urge to void or urinary frequency. Confusable diseases as the cause of the symptoms must be excluded" [14].

Risk Factors/Etiology

The etiology of BPS is unknown but there are several hypotheses regarding its pathophysiology. A series of events have been proposed to trigger the BPS such as bladder overdistension, bacterial infections, trauma from pelvic surgery, and bladder trauma, which are believed to damage the bladder epithelium [11] (Fig. 11.1).

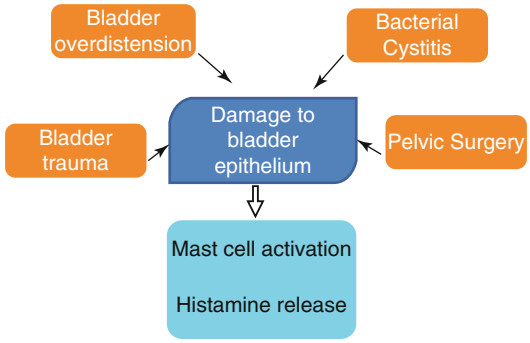

Fig. 11.1 Proposed pathophysiology of BPS

Dysfunction of the bladder epithelium makes the coating of the bladder epithelium, the glycosaminoglycan (GAG) layer, to become defective. This can increase the permeability of bladder epithelium to urine contents such as potassium, which can leak into the interstitium and musculature, leading to mast cell activation and release of histamine. It is also supposed to activate the C-fiber nerve endings and release of substance P. Immunologic and allergic responses are postulated to be triggered as well. Combination of these events is believed to cause progressive bladder damage and chronic neuropathic type of pain [15].

Associated Conditions

Hypersensitivity disorders like irritable bowel syndrome, fibromyalgia, vulvodynia, myofascial dysfunction of the pelvic floor muscles, migraine headaches, and allergies are more common among patients with symptoms of BPS compared to the general population [16].

Clinical Features

Symptom presentation in BPS is variable, usually presenting with pelvic pain or bladder pain, pressure or discomfort relating to bladder, urinary urgency, and urinary frequency. Pain is typically chronic in nature (>6 months), referred in the area of the bladder, vagina, urethra, rectum, or perineum. The characteristic feature of the pain is its relation to bladder filling, increasing with bladder distension which may diminish with voiding [16]. In some patients the pain may persist for a considerable time after voiding, with a persistent urge to void. Urinary frequency is common with BPS and usually occurs both at daytime and night time. Urgency is a feature common to both BPS and overactive bladder (OAB) and is one of the reasons for misdiagnosis of this condition. Urgency in OAB is typically due to concern about an impending incontinence, whereas in BPS it is due to increasing pain with filling and an attempt to relieve it by voiding.

There are no specific features for BPS on physical examination, but most often patients with BPS have bladder base tenderness on pelvic examination and tenderness of levator ani muscle has been noted in 81 % of patients in a study [17]. Pain mapping of the vulva can be done to rule out vulvar/vestibular diseases.

Investigations

The diagnosis of BPS is one of exclusion, and hence assessment of these patients should aim to rule out other conditions which can present with similar features, such as urinary tract infection, endometriosis, bladder carcinoma, radiation cystitis, overactive bladder, and nonbacterial cystitis secondary to drugs such as NSAID, allopurinol, aspirin, and cyclophosphamide. It is important to remember that some of these conditions can be associated with BPS and a diagnosis of BPS is possible only if symptoms persist after treatment of the associated conditions.

Evaluation, along with a detailed history and examination should include a 3-day bladder diary, which can provide information about the frequency and volume of voids. Urine analysis and culture are needed to rule out bacterial cystitis and atypical infections like chlamydia or mycoplasma. In the presence of a hematuria, urine cytology is indicated to rule out bladder carcinoma.

Urodynamics

The role of urodynamics is to rule out detrusor overactivity and there are no specific features

diagnostic of BPS. However in patients with BPS, on uroflowmetry, the volume voided is smaller compared to those with idiopathic detrusor activity. In addition on filling CMG, values for bladder sensation such as first desire to void, normal desire to void, and strong desire to void are significantly lower in patients with BPS due to increased bladder sensation. Pain on increased filling also means bladder may not be increased to its capacity leading to decreased bladder compliance [18].

Cystoscopy

In the past, cystoscopy was primarily employed to identify the Hunner's ulcer, a primary diagnostic criteria in the NIDDK criteria. Currently its role is to exclude intravesical abnormalities in the bladder such as endometriosis, carcinoma, or foreign body. In the presence of mucosal ulcers, glomerulations, or edema on cystoscopy, the symptoms appear to be severe in nature.

Potassium Sensitivity Testing (Parsons Test)

The test involves instillation of 40 ml of sterile water and 40 ml of 40 mEq/100 ml potassium chloride alternatively into the bladder for 5 min. Pain and urgency following each instillation are noted. Increase in pain or urgency with potassium instillation indicates a positive test. The test is painful, has low sensitivity and specificity, and hence is not used anymore.

Urine Markers

Antiproliferative factor, epidermal growth factor, insulin-like growth factor (IGF)-binding protein 3, and interleukin (IL)-6 are noted to be increased in patients with interstitial cystitis, whereas markers such as cyclic GMP and methyl histamine are decreased in these patients [19]. Among these, the antiproliferative factor shows promise as marker for BPS in the future.

Table 11.2 Examples of dietary substances that may trigger symptoms

Coffee	Tea
Carbonated drinks	Alcoholic beverages
Artificial sweeteners	Food preservatives
Citrus fruits and juices	Spicy foods
Tomatoes	Caffeinated beverages

Management

Management of patients with BPS is a therapeutic dilemma and there are basically three components to it: diet and behavior modifications, oral drug treatments and bladder instillation of drugs. The role of surgery is limited and reserved for severe cases.

Diet and Behavior Modification

Certain foods have been shown to trigger the symptoms of BPS and avoiding those can help in symptom control and flare-ups. Some of the possible foods implicated are shown in Table 11.2. The list is not exhaustive and the patients should aim to work out potential dietary factors for themselves, as the symptoms have been shown to occur between 30 min and 6 h after ingestion of the triggering agent [20].

Behavior modifications which have shown to be helpful include gentle exercises like walking and yoga, relaxation techniques like meditation and hypnosis, wearing loose comfortable clothing, and appropriate fluid intake (not fluid restriction).

Oral Drug Treatments

Antihistamines

Antihistamines inhibit mast cell degranulation, reduce histamine release, and thereby provide symptom relief. They also have skeletal muscle relaxant effect and promote sleep which is helpful in patients with BPS. Improvement is seen in 6–8 weeks after continuous use. Cimetidine and Hydroxyzine are the commonly employed antihistamines [21].

Amitriptyline

This tricyclic antidepressant appears to be an effective treatment in BPS and acts by modulating the pain by inhibiting the reuptake of serotonin and norepinephrine in the central nervous system. In addition, it stabilizes the mast cells and is very effective in the treatment of the pain component [22]. It however has anticholinergic and sedative side effects.

Pentosan Polysulfate Sodium (PPS)

Pentosan polysulfate sodium is the only oral agent approved by the U.S. Food and Drug Administration (FDA) for treatment of BPS. The proposed mode of action is by repair of the GAG layer of the bladder epithelium and thereby reducing the permeability. It is taken in doses of 100–200 mg, 3 times a day for a minimum of 3 months. The drug is shown to be effective but it takes time to build the GAG layer. A long-term study has found that 42–62 % of patients had an overall improvement of symptom with pentosan polysulfate sodium [23]. There are no serious adverse effects apart from dyspepsia, diarrhea and reversible alopecia.

Intravesical Drug Therapy

Intravesical instillations are used as second-line treatment or in conjunction with oral drugs. The drugs employed in bladder instillation include dimethyl sulfoxide (DMSO), hyaluronic acid, chondroitin sulfate, heparin, and lidocaine.

DMSO has anti-inflammatory, analgesic, and muscle relaxant effects. It is instilled via a catheter and allowed to remain for 15 min inside the bladder. It is repeated weekly for 6–8 weeks. The efficacy of DMSO has been shown to be around 70 % [24]. The drug can also be instilled as a cocktail with other agents such as heparin, sodium bicarbonate or corticosteroid.

Hyaluronic acid and chondroitin sulfate act by coating and temporarily replacing the defective GAG layer on bladder epithelium. The instillation frequency is weekly for the first 4 weeks and thereafter monthly until symptoms resolve. The response rate for chondroitin sulfate is around 67 % [25] and with hyaluronic acid 71 % [26].

Hydrodistension

Hydrodistension of the bladder under anesthesia during cystoscopy as a diagnostic procedure has been shown to have therapeutic benefit in about 50–70 % of patients over a period of 6–12 months [27, 28]. It is believed to act by disrupting the neuronal bladder pathways and interrupting the pain transmission. Under anesthesia, the bladder is distended, emptied, and refilled to observe for hyperemia, mucosal changes, deposits, or glomerulations. The distension is maintained for 3–4 min and bladder emptied.

Surgical Treatment

In patients refractory to the above measures and where the symptom severity significantly impairs the woman's quality of life, surgical option is considered. This can vary from less invasive endoscopic resection or fulguration of bladder ulcers and local injection of hydrocortisone, saline, and heparin in and around the ulcers to radical surgery such as augmentation cystoplasty, urinary diversion with or without cystectomy, or partial cystectomy [29]. It is important that the patient understands that pain may persist in spite of surgery either due to pelvic floor muscle spasm or central nervous system-mediated pain.

Conclusion

Bladder pain syndrome is an extremely challenging condition both for the patient and the clinician. Despite the patient's persistent symptoms, the clinician has limited confirmatory tests to offer and needs to resort to a process of exclusion to arrive at a diagnosis. Once a diagnosis is established, there is no definitive treatment modality for immediate improvement. Treatment options need to be explored serially and they can take some time before providing symptom relief. It is important for health-care providers to be aware of the diagnostic criteria of BPS and to re-evaluate patients with recurrent UTI and refractory OAB symptoms. Counseling

forms a very important aspect, in care of these patients as various treatment options need to be discussed. Patients must be provided with realistic expectations of the various treatment modalities.

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Part III

Pelvic Floor Dysfunction

Evaluation of Pelvic Organ Prolapse

12

Paul Riss and Marianne Koch

Introduction

Pelvic organ prolapse (POP) affects many women in the course of their lifetime and can be the cause of a severe impairment of quality of life. Many risk factors are known for pelvic organ prolapse such as obstetrics history, physical labor over many years and genetic and familial predisposition. However, usually there is very little a woman can do to reduce the impact of risk factors especially in underserved populations or low-income countries. POP is not a life-threatening condition and fortunately many treatment options – conservative as well as surgical – are available to support women suffering from POP.

In order to help a woman with POP, the first step is a comprehensive and correct evaluation of the patient and her condition. Fortunately the assessment of pelvic organ prolapse does not require expensive equipment, but with a good history and a focused clinical examination, it is possible to get a clear picture of the extent of pelvic organ prolapse and how it affects the life of the individual patient. This assessment forms the basis of developing a treatment plan together with the patient. This plan must be individualized in the context of the available options and take into account the particular health care setting.

The adequate assessment of the POP severity through exact history taking and clinical examination forms the basis of developing an individualized treatment plan in cooperation with the patient. The aim of this chapter, therefore, is to show how with a good history and clinical examination the health care provider is able to get a complete picture of the pelvic organ prolapse without having to use expensive – and often unavailable – additional diagnostic tools.

History

General Considerations

The basis of a good diagnosis of pelvic organ prolapse is, as always, a good, detailed and comprehensive history. The severity of symptoms and the degree of bother experienced by the patient may differ from the assessment of the prolapse. It is very important that the patient is comfortable and put at ease so that a trusting relationship with the physician during the interview can be established. Symptoms of pelvic organ prolapse relate to very personal aspects of a patient and most women are reluctant to share intimate or seemingly embarrassing details of their life. However, it is important to address all aspects of quality of life in order to be able to help the patient with those aspects that concern her most (Table 12.1).

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A. Tamilselvi, A. Rane (eds.), *Principles and Practice of Urogynaecology*,
DOI 10.1007/978-81-322-1692-6_12, © Springer India 2015

Table 12.1 Elements of history taking in patients with pelvic organ prolapse

Symptoms
Feeling of heaviness/descent
Bulge
Pain
Dyspareunia
Urinary incontinence
Anal/fecal incontinence
Need to replace bulge to void/pass stool
Aspects of quality of life
Household work
Professional work
Leisure
Sexual activity
Sports
Hobbies

It is always a good idea to start with an open question such as "What troubles you the most?" It can be surprising to see the range of symptoms and their severity in a patient population with similar degrees of prolapse. From the beginning of the interview, it is important to keep in mind the main complaint of the patient since this will be the symptom which any treatment must address.

It is also extremely important to ask about the family situation and the socioeconomic environment of a patient. It does not make sense to offer surgery to the patient when she – or her family – cannot afford it or when she is unable to come to the hospital at all. Conservative treatment, on the other hand, can only be offered to patients who have the means and the possibility to come to follow-up visits or to physical therapy at regular intervals.

Specific Questions

In order to elucidate the concrete manifestations of pelvic organ prolapse, specific questions should be asked: a sensation of "heaviness" or "descent" and a feeling of a "foreign body" or "bulge" between the labia or in front of the vulva. Further questions are aimed at elucidating how often and at what times of the day these symptoms are noted by the patient. Symptoms of pelvic organ prolapse typically appear during the day and get worse with physical labor and towards the afternoon and evening.

The next question is how these symptoms affect daily activities and quality of life. Finally the patient is asked about urinary incontinence, anal and fecal incontinence and implications on sexual function. It is important to ask whether a bulge prevents complete emptying of the bladder and the rectum and whether the patient has to manually replace the prolapse in order to be able to pass urine or stool.

Although time is often a problem, the main points of pelvic organ prolapse can be elucidated with a few questions as outlined above. Ideally, the history taking has the form of a structured interview. Alternatively, questionnaires can be used. In the mind of the doctor, while taking a history, the outlines of a possible treatment strategy already takes shape. Severe symptoms or the presence of a marked bulge are points in favor of surgery.

Clinical Examination

For the clinical examination, the patient must be placed in a comfortable position, and the examiner must be able to see the vulva and the prolapse. There might be cultural inhibitions to let a doctor see the external genitalia, but if at all possible, an effort should be made to visualize the prolapse. This is important to see the extent of the prolapse and the movement of the prolapse on Valsalva and to see whether the skin and the epithelium are intact.

For the examination, the patient can be in the dorsal or lateral supine position. The patient should also be asked to stand up so that the prolapse can be evaluated in the standing position. The dorsal lithotomy position with the knees apart offers the examiner the best access to the external genitalia and the prolapse and allows the easy use of specula and ultrasound probes. Many women will tolerate this position when a light blanket or bed sheet is placed over their knees and lower abdomen.

The next step is a thorough clinical examination of the patient with the help of speculum and palpation. It is preferable to use two separate specula in order to better visualize the anterior and posterior vaginal wall. Only a gynecological examination with speculum allows for a differentiation between the various forms of a prolapse (anterior, posterior, apical) and the extent of the prolapse (stage). The patient is asked to do a Valsalva maneuver or to cough so that the physician can see the extent of the prolapse (Figs. 12.1, 12.2, 12.3, 12.4, and 12.5). The pelvic organ prolapse quantification (POPQ) system is utilized to objectively assess the severity of POP.

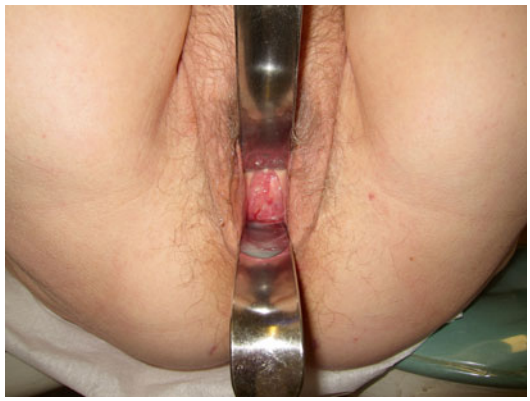

Fig. 12.1 Examination of the patient. Two separate speculae (*blades*) are used for the gynecologic examination to visualize the anterior and posterior vaginal walls separately

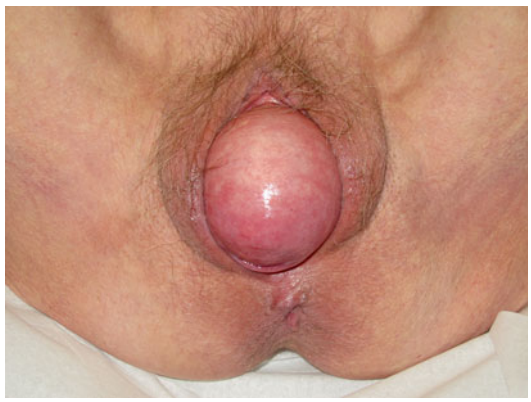

Fig. 12.2 Cystocele. There is a marked bulge on the anterior vaginal wall beyond the hymenal ring. The differential diagnosis of a rectocele is possible only with the help of specula

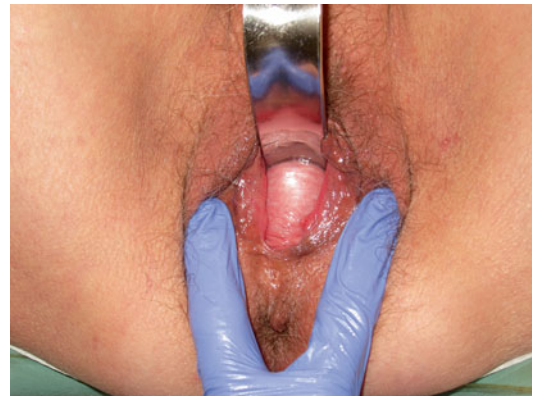

Fig. 12.3 Rectocele. Descent of the posterior vaginal wall. The anterior vaginal wall is held back with a Breisky speculum

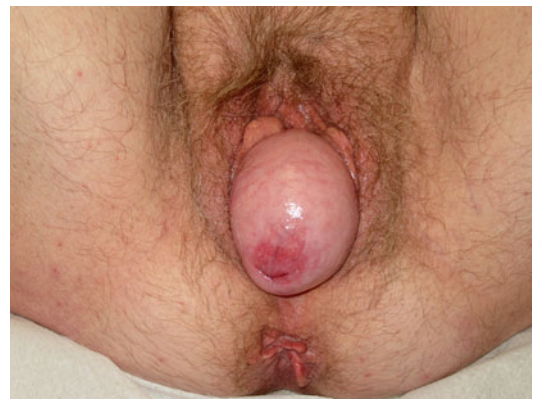

Fig. 12.4 Uterine descent. The cervix protrudes beyond the hymenal ring. With two separate specula, it is possible to make the differential diagnosis of cervical elongation

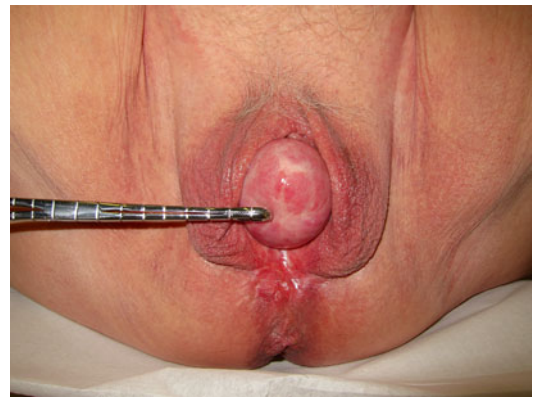

Fig. 12.5 Post-hysterectomy vaginal vault prolapse. The tip of the sound points the post-hysterectomy scar at the top of the vaginal vault (point C in POPQ)

It is important to keep in mind that a pelvic organ prolapse almost always can be manually replaced into the small pelvis. This means that the picture of how a pelvic organ prolapse presents itself is never stable and very often cannot be completely reproduced during a clinical examination and even with good Valsalva maneuvers. The most important anatomical information for further treatment planning is knowledge about compartment and level, which are involved in the POP.

POPQ: Pelvic Organ Prolapse Quantification System

For several reasons, it is very important that the result of a clinical examination of pelvic organ prolapse is clearly described and recorded. Every medical diagnostic or therapeutic intervention must be documented. It becomes part of the patient record, which allows comparison with subsequent examinations or assessments by other health care providers. Over time, it allows the evaluation of changes during the natural course or in conjunction with treatments. Last but not least, it is important to document pelvic organ prolapse for quality control measures and for research.

For these reasons, the need became apparent for a standardized description and documentation of pelvic organ prolapse and in 1996, the result of the combined efforts of several working groups was published as *The Standardization of Female Pelvic Organ Prolapse and Pelvic Floor Dysfunction* [1]. The system quickly became known as the POPQ system – pelvic organ prolapse quantification system – and is now the preferred system to describe and document pelvic organ prolapse. Clinical research on pelvic organ prolapse requires the use of POPQ, but over the years, many clinicians have familiarized themselves with the POPQ and use it in their daily practice [2].

Measuring the POPQ

The patient should be in the supine position. The examiner must have a good view of the vulva but does not touch it. The patient is asked to do a Valsalva, and the points are measured with a ruler.

If the POPQ is taken in the standing position or with a full bladder, this must be clearly stated in the record.

Building the POPQ

The POPQ consists of six points and three measurements. The definition of the points and the measures is given in Table 12.2. The plane of reference is the hymen or the hymenal ring. The

Table 12.2 POPQ definitions of points and measures

Anterior vaginal wall
Point Aa – a point located in the midline of the anterior vaginal wall 3 cm proximal (i.e., inside the vagina) to the external urethral meatus or hymen
Point Ba – a point that represents the most distal (i.e., most dependent) position of any part of the upper anterior vaginal wall between the vaginal cuff/anterior vaginal fornix and point Aa. By definition, in a woman without prolapse, point Ba is identical with point Aa (minus 3)
Apex
Point C – a point that represents either the most distal (i.e., most dependent) edge of the cervix or the leading edge of the vaginal cuff (hysterectomy scar) after total hysterectomy
Point D – a point that represents the location of the posterior fornix (or pouch of Douglas) in a woman who still has a cervix
Posterior vaginal wall
Point Ap – a point located in the midline of the posterior vaginal wall 3 cm proximal to the hymen (i.e., inside the vagina)
Point Bp – a point that represents the most distal (i.e., most dependent) position of any part of the upper posterior vaginal wall between the vaginal cuff/posterior vaginal fornix and point Ap. By definition, in a woman without prolapse, point Bp is identical with point Ap (minus 3)
Measures
Genital hiatus (*gh*) – measured from the middle of the external urethral meatus to the posterior midline of the hymen
Perineal body (*pb*) – measured from the posterior margin of the genital hiatus to the mid-anal opening
Total vaginal length (*tvl*) – the greatest depth of the vagina in centimeters

The hymen is a fixed plane reference. Points can be either at the hymen (zero), above = proximal (minus), or below = distal (plus)

hymenal plane is designated as zero (0), all points above or proximal or inside the vagina are given a minus sign (−), and all points below or distal are given a plus sign (+).

Unfortunately, the definitions and the designation of the points are not intuitive and have been felt to be a barrier to the wider acceptance of the POPQ. It is helpful to remember that there are two kinds of points:

Fixed points: the 2 points A (Aa on the anterior vaginal wall and Ap on the posterior vaginal wall) are fixed points and can be thought of as "birthmarks" on the vaginal epithelium. In a woman without prolapse, they are 3 cm above the hymen (minus 3). In a case of complete prolapse, point A being a fixed point can never have a value greater than plus 3.

Movable points: the 2 points B (Ba on the anterior vaginal wall and point Bp on the posterior vaginal wall) are defined as the most distal (most dependent) point on any part of the upper vagina. "Upper" means that point B is located somewhere between point A and the anterior or posterior vaginal fornix or post hysterectomy between point A and the vaginal cuff or apex. By definition, in a woman without prolapse, point B is identical with point A (minus 3).

Points C and D are easier to understand. Point C is the most distal part of the cervix, and point D represents the position of the posterior fornix (or pouch of Douglas). After total hysterectomy, there is no point D and point C is the position of vaginal cuff or apex (Fig. 12.6).

In a patient without a uterus (Fig. 12.5), the tip of the sound would point to point C. Figures 12.7 and 12.8 give examples of points Aa and Ba in cystocele and of point C in a patient with uterine prolapse.

Point D has also been described as the insertion point of the uterosacral ligaments at the level of the cervix [1]. It follows that a big numerical difference between points C and D would indicate cervical elongation.

The three measures are straightforward: genital hiatus, perineal body, and total vaginal length. The definitions are given in Table 12.2. Except for a very short perineal body, they have less importance for the description of a pelvic organ prolapse.

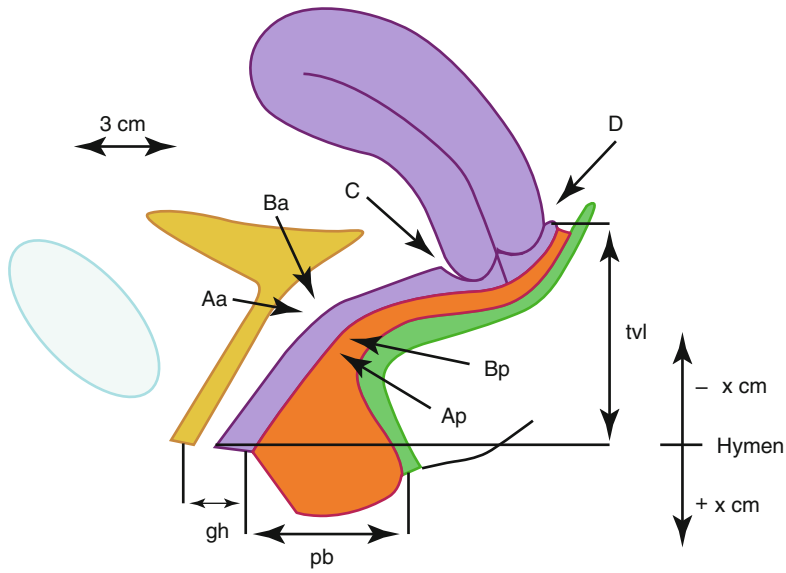

Fig. 12.6 Schematic drawing of POPQ. The plane of reference is the hymen (*zero*). Points above the hymen are given negative numbers (*minus*), points below the hymen are given positive numbers (*plus*)

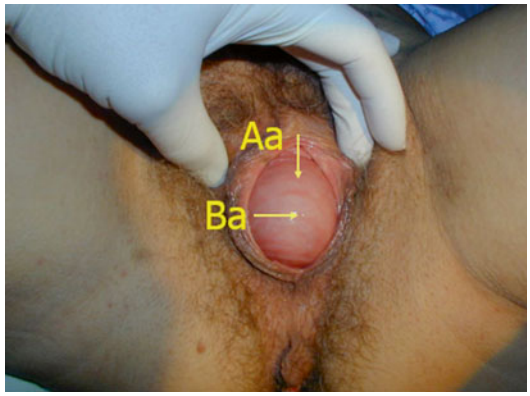

Fig. 12.7 POPQ – example cystocele. Point Aa is 3 cm below the hymen and point Ba 6 cm below the hymen (Aa +3, Ba +6)

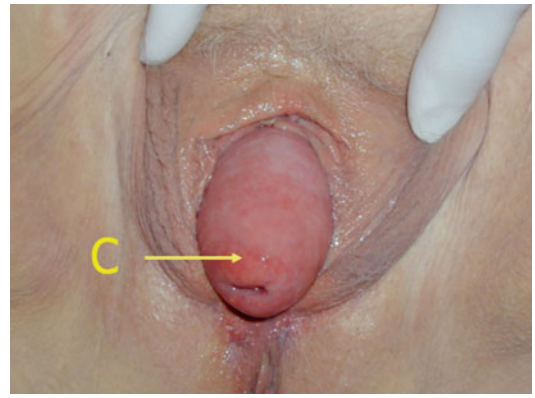

Fig. 12.8 POPQ – example uterine descent. Point C would be recorded as +8 and the prolapse described as uterine prolapse stage III

Fig. 12.9 Grid recording of POPQ measurements. The first line represents the anterior compartment and point C (cervix or vaginal cuff). The third line represents the posterior compartment and point D (Douglas or insertion of uterosacral ligaments)

Aa	Ba	C
gh	pb	tvl
Ap	Bp	D

Recording the POPQ

Figure 12.9 shows how the nine numerical values – the six points and the three measures – are recorded. The classical format is the 3 by 3 grid: the first row contains the 2 points from the anterior vaginal wall and point C (cervix or cuff), and the third line, the 2 points from the posterior vaginal wall and point D (when a uterus is present). In addition the points can be marked on a sagittal line diagram giving a graphic representation of the prolapse (Fig. 12.10).

A simplified representation without the need of using a grid is to write the nine values in a row with slashes according to the lines of the grid (e.g., Aa Ba C//gh pb tvl//Ap Bp D). This format is easy to understand, can be written on paper or typed on a keyboard, and is easy to remember, and there is no loss of information.

POPQ and Staging

The POPQ system still offers staging as a kind of summary of the measurements (Table 12.3). In fact the stages are redundant since looking at the nine numbers of the POPQ gives more information than a staging system. Some clinicians, however, are

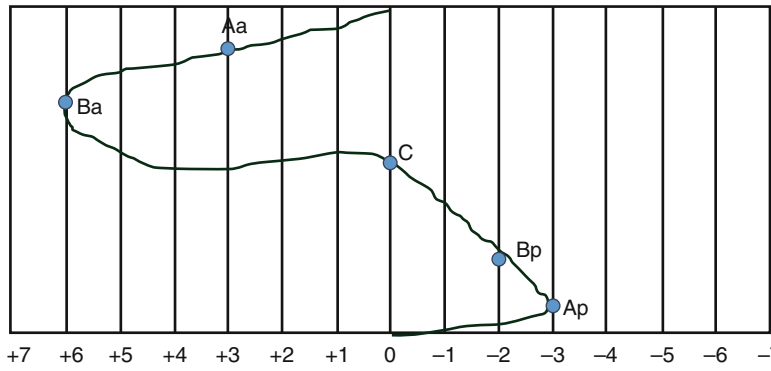

Fig. 12.10 Representation of POP in a sagittal line diagram. This is a stage III cystocele (point Ba 6 cm below the hymen) with a stage II uterine descent (point C at the hymen) and no rectocele (points Ap and Bp above the hymen)

Table 12.3 Stages of the POPQ system

Stage 0	= No prolapse
	Aa, Ba, Ap, Bp are all at –3
	C or D between tvl and ≤ tvl –2 cm
Stage I	= Most distal portion > 1 cm above the level of hymen
Stage II	= ≤1 cm proximal to or distal to (above or below) the plane of hymen
Stage III	= ≥1 cm below the plane of the hymen
Stage IV	= Complete eversion

Reference point for the ordinal stages: most distal portion of the prolapse

still more comfortable with an ordinal system than with having to navigate points.

Two aspects must be kept in mind when assigning stages to a prolapse: the stage is determined according to the most distant part of the prolapse, and second the quantitative description of the prolapse has to be done first. The disadvantage of the staging classification is that it does not say anything about which part of the vagina or internal genitalia is prolapsed. This information has to be given in addition to the stage. The nine numbers of the POPQ include this information.

Limitations of POPQ

A disadvantage of the POPQ system is that all points and measures are taken in the midline. As a consequence, the POPQ does not reflect asymmetries and cannot be used to describe, for example, paravaginal defects. One has also to keep in mind that the POPQ depends on the co-operation of the patient and to the strength of her cough or Valsalva maneuver. It is therefore unreasonable to assume that in an individual patient the POPQ will always be identical. Very unusual forms of prolapse like a perineal hernia cannot be described by the POPQ system.

One also has to realize that the POPQ is only a description. It is not a diagnosis and does not offer specific indications for treatment.

Current Use of POPQ

After almost 20 years, the POPQ system is well established and has stood the test of time [3]. It is widely used in clinical practice and essential in clinical research. It is impossible to think of a paper dealing with pelvic organ prolapse where the POPQ system is not used. The use of the stages is discouraged since they offer no improvement over the presentation of the POPQ in numbers but on the contrary comes with a loss of information. At this time, it is impossible to say whether the POPQ system will and must be modified in the future. Most clinicians who use it are very comfortable after an initial learning phase and appreciate its great advantage [4].

Imaging

The implications of imaging are limited in the diagnosis and treatment of pelvic organ prolapse. Perineal or intravaginal ultrasound can be used as well as defecography or magnetic resonance imaging. Dynamic imaging allows the visualization of the movement of the uterus and the pelvic floor during Valsalva and the evacuation of the rectum. The main use of imaging, however, is in research and therefore with fewer implications on patient management.

From Evaluation to Treatment Decisions

As always in medicine, the same is true in pelvic organ prolapse: first the problem must be identified and a correct diagnosis must be made, and then a management plan is developed. Two basic questions must be answered in the evaluation of pelvic organ prolapse:

1. Which "compartment" is involved? The essential differentiation is between the anterior compartment (anterior vagina, cystocele), the posterior compartment (posterior vagina, rectocele), or apical descent (prolapse of the uterus and prolapse of the vaginal apex post hysterectomy).
2. Which "level" is involved? Level I designates the cervix or the inner vagina, level II the middle vagina (which corresponds to the bladder in the anterior compartment) and level III the outer or most distal part of the vagina (e.g., corresponding to the urethra in the anterior compartment).

Conclusion

Pelvic organ prolapse is not a life-threatening but a very irritating and bothersome condition. The two pillars of the evaluation of pelvic organ prolapse are history taking and clinical examination. Imaging studies are of lesser importance. The result of the clinical examination should be summarized and reported using the POPQ system. On the basis of a complete evaluation of a patient, management decisions can be made. These must be tailored to the individual needs of the patients and the specific circumstances.

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Conservative Management of Pelvic Organ Prolapse

13

George Ralph and Karl Tamussino

Pelvic organ prolapse (POP) refers to the descent of pelvic organs into the vagina. The prevalence of which varies from 40 to 50 % over the age of 40 years. The incidence of prolapse increases with increasing age and surgical intervention might not be an ideal option for all. Conservative management should ideally be offered to women with prolapse while discussing their management options. Conservative measures have played a major role in the management of POP both as a short-term intervention and as a definitive management strategy. This includes lifestyle interventions, pelvic floor exercises, and mechanical support devices (pessaries). In assessing the success of conservative measures, it is usually the improvement of patient's symptoms and the quality of life measures that are analyzed. Prolapse symptoms can include dragging sensation or heaviness in the vagina, backache, and a sensation of lump in the vagina, which may be associated with bladder, bowel, or sexual dysfunction. Objective assessment of POP, with staging using either the Baden-Walker or POP Quantification system, has been undertaken in a few studies evaluating the conservative management.

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Pelvic Floor Exercises/Pelvic Floor Muscle Training (PFMT)

Pelvic floor muscle training is usually suggested along with lifestyle interventions. Measures such as weight loss, avoiding constipation and heavy lifting along with treatment of chronic cough are some of the interventions suggested to improve prolapse symptoms.

The role of pelvic floor muscles in providing structural support to the pelvic organs is well known. PFMT involves initial assessment of the pelvic floor muscle tone, followed by Kegel exercises with or without biofeedback. Assessment of the pelvic floor muscle should take into account both the strength and the tone of the muscle. Vaginal palpation of pelvic floor muscle is the common technique employed in this assessment, prior to PFMT [1]. Other modalities such as ultrasound, MRI, electromyography, manometry and cones can measure different aspects of the pelvic floor muscle but have their own limitations and are not in routine use.

PFMT is likely to increase the muscle function and thereby elevate the levator plate to improve prolapse symptoms. In prolapse stages 3 and 4, PFMT on its own is less likely to help the symptoms and women with symptomatic stages 1 and 2 are more likely to benefit.

A. Tamilselvi, A. Rane (eds.), *Principles and Practice of Urogynaecology*,
DOI 10.1007/978-81-322-1692-6_13, © Springer India 2015

In an assessor blinded randomized control trial, comparing PFMT with no intervention, there was improvement in pelvic floor muscle function in terms of muscle strength and endurance with PFMT [2]. The Cochrane review by Hagen indicates there is improvement of prolapse symptoms and the chance of improvement in prolapse stage is by 17 % in patients with PFMT compared to the controls. There are studies that indicate a significant impact for PFMT, especially with 6 months of supervised training with both anatomical and symptom improvement [3]. A recent multicenter randomized controlled trial on individualized pelvic floor muscle training in women with pelvic organ prolapse (POPPY Trial) shows that one-to-one pelvic floor muscle training for prolapse is effective for improvement of prolapse symptoms [4]. PFMT appears to be an ideal first-line management in patients with POP, as a supervised and intense training program with no adverse effects.

Pessaries

Before the advent of anesthesia, surgical techniques such as vaginal hysterectomy were not used commonly for treatment of POP and pessaries formed a major part of treatment. In the nineteenth century, the pessaries included pomegranates and wax balls, which were apparently of limited efficacy when used vaginally. In the early twentieth century, surgical approaches such as vaginal hysterectomy with anterior and posterior colporrhaphies gradually superseded pessaries. Today pessaries are made out of silicon and with proper handling and care, are associated with little vaginal discharge or bleeding. Pessaries are a good option for the elderly frail patient unfit for surgery and for young women who prefer conservative management over surgery. With proper patient selection, pessaries are well tolerated and frequently a viable alternative to surgical interventions. A potential limitation for both patients and gynecologists with pessaries are the frequent visits, which can be managed by allied healthcare staff, where facilities exist.

Serious complications, such as fistulae, occur only with neglected (forgotten) pessaries; mild problems such as discharge are easily taken care of. Many gynecologists are not familiar with the variety of pessaries available or how to select, insert and manage the different models and training in this aspect is important.

Fig. 13.1 Ring pessary

Selection of a Pessary

In 1830, the American Medical Association registered 123 different pessaries. In practice, four types are useful: the ring pessary, the Gellhorn or shelf pessary, the cube pessary, and cylinders (Figs. 13.1, 13.2, and 13.3). All these models come in different sizes. A survey of the members of the American Urogynecologic Society (AUGS) found that 78 % of providers selected a pessary based on the stage of prolapse, whereas 22 % used a ring pessary as first line in all patients [5]. There is little scientific evidence to guide the choice of a pessary.

The selection of a pessary depends on several factors. Efficacy is paramount, but most try to avoid pessaries that are difficult to insert and remove and those requiring frequent follow-up visits.

Pessary Outcomes

Hanson reported very good results in 1,216 patients with vaginal pessaries [6]. The overall success rate was 71 % in patients with POP and ring pessaries were successful in 89 % and

Fig. 13.2 Space-filling Shelf and Gellhorn pessaries

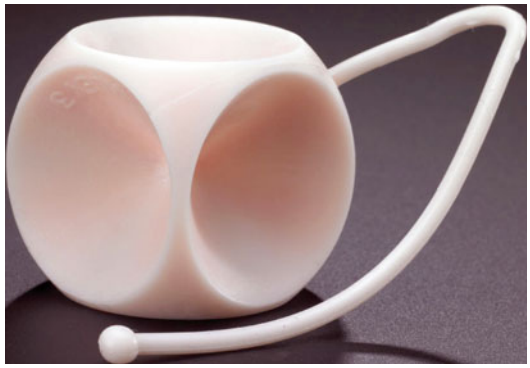

Fig. 13.3 Cube pessary

Gellhorn pessaries in 85 %. In patients with stress urinary incontinence, ring pessaries with or without urethral support, were effective in 78 % and 63 % of patients, respectively. Local hormone replacement treatment seemed to improve the success rate with pessary fitting in this study [6].

In a prospective study, Wu et al. used the ring pessary as a first-line treatment in all patients with all degrees of pelvic organ prolapse, and when a ring pessary failed, other models were used. The success rate was 79 % in this study and they were unable to identify any predictive factors for success with pessaries [7]. Wu et al. found that prior hysterectomy did not influence the success of pessary fitting. In our experience, a long and wide vagina is not a contraindication for management with a pessary.

The compartment of prolapse also does not influence the success of pessary and this should not be taken into account when considering pessaries for POP [8]. Most gynecologists prefer surgical option over pessaries in advanced stages of prolapse in elderly population. In a study by Powers (2006), in those with advanced POP (stages 3 and 4), there was a significant likelihood of success with a success rate of 62 %, among those willing to try the pessary [9].

Urinary tract symptoms however seem to influence the success of pessary fitting. Sulak (1993) showed that only one third of patients with urinary incontinence reported a decrease of their symptoms with pessary and two thirds underwent surgery [10].

In general the success rates with pessary have varied from 41 to 74 %, with variable lengths of follow-up from a week and up to 5 years [7, 11–15]. The majority who decide to go for surgical intervention after a pessary trial, do so within 12 months of pessary fitting. A prospective study on pessary use over a period of 5 years found that if the fitting was successful at the end of 4 weeks, most women continue to use it over 5 years [16]. With the use of supportive pessaries, it has also

Table 13.1 Pessary fitting assessment

Pelvic examination
Width and length of vagina
Atrophic changes
Infection, ulceration
Degree/stage of prolapse

Table 13.2 Fitting of pessaries

Selection of type
Selection of size
Local estrogen
Training of patient

been noted that there is no progression of the prolapse stage and improvement of the stage in 21 % of the women [11].

In a study comparing surgical intervention with pessary use in 554 women, 1 year after treatment, there was similar improvement in bladder, bowel, sexual function and quality of life parameters in both groups [17].

Complications associated with pessary use include discharge, pain, discomfort, ulceration, bleeding, disimpaction and constipation which occur in 12 % of women [16]. Of these, vaginal discharge is the most common.

Contraindications for Pessary

All pessaries are generally contraindicated in the presence of cervical or vaginal ulcerations, undiagnosed vaginal bleeding, active local pelvic infection and patients allergic to silicone and latex. Patients who are unable to attend for regular pessary change and follow-up should not be fitted with one. Space-occupying pessaries ideally should be avoided in sexually active women, unless they are adept at its removal and self-insertion.

Technique of Pessary Therapy (Tables 13.1 and 13.2)

Before inserting a pessary, the patient is examined to rule out atrophy, infection and ulceration of the vagina. Pelvic examination includes quantification of the prolapse and assessment of the width and depth of the vagina. Local treatment with vaginal estrogen for 3–4 weeks prior to pessary insertion is recommended in those with atrophic changes. The provider estimates the size and type of pessary to be used.

The aims of pessary fitting are proper correction of prolapse and proper size of pessary to avoid pain and ulceration of vaginal mucosa and bleeding. Most women can be successfully fitted with a pessary 70–90 % of the time [18].

We usually begin pessary fitting with a ring pessary and move over to other types as indicated.

Step 1 (Figs. 13.4 and 13.5)

The ring pessary is folded between the index finger and thumb. Estrogen cream is placed over the pessary and at the introitus and the labia minora are separated for insertion.

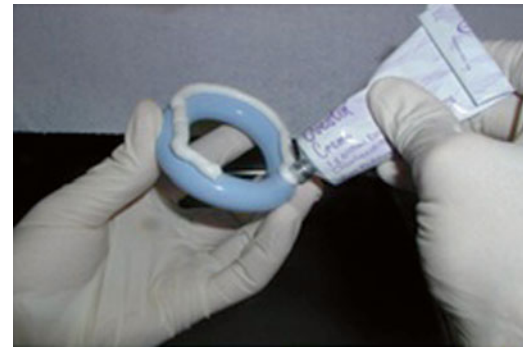

Fig. 13.4 Application of cream

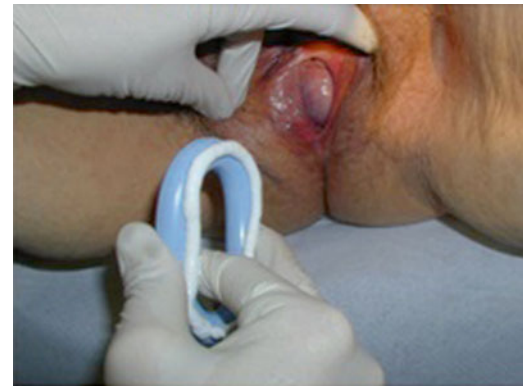

Fig. 13.5 Insertion of pessary step 1

Step 2 (Fig. 13.6)

The ring is inserted in the sagittal plane, folded between the index finger and thumb. Once the ring is inside, it is rotated towards the posterior vaginal wall.

Step 3 (Fig. 13.7)

When the ring is properly inserted, the cervix is supported by the ring.

Step 4

With the labia separated, the patient is asked to perform a Valsalva maneuver. The pessary should stay in position; a slight descent is normal. If the pessary descends to the introitus, a larger size is tried. The position of the pessary is examined in the supine and standing positions and after a brief walk. The fitting is successful if the pessary does not descend and the patient feels neither the prolapse nor the pessary.

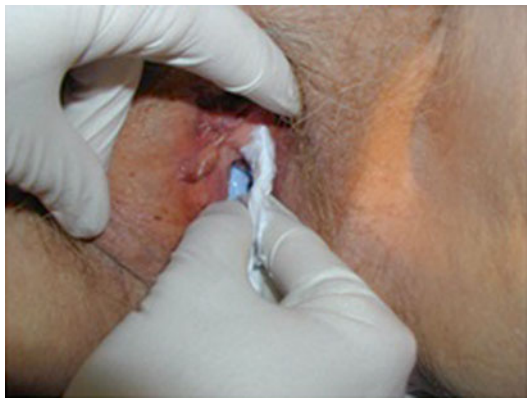

Fig. 13.6 Insertion of pessary step 2

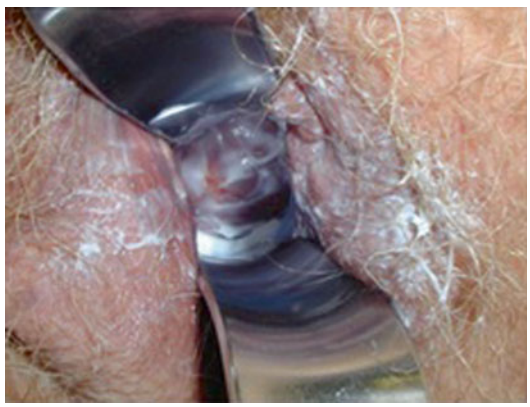

Fig. 13.7 Insertion of pessary step 3

Pessary Removal

Step 1 (Figs. 13.8 and 13.9)

The ring pessary is grasped between the index finger and thumb, rotated into the sagittal plane, and retrieved.

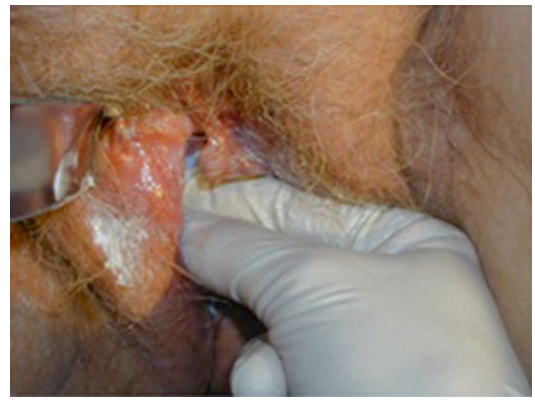

Fig. 13.8 Removal of pessary step 1

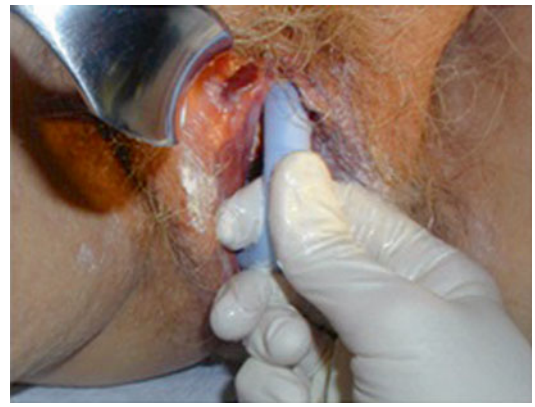

Fig. 13.9 Removal of pessary step 2

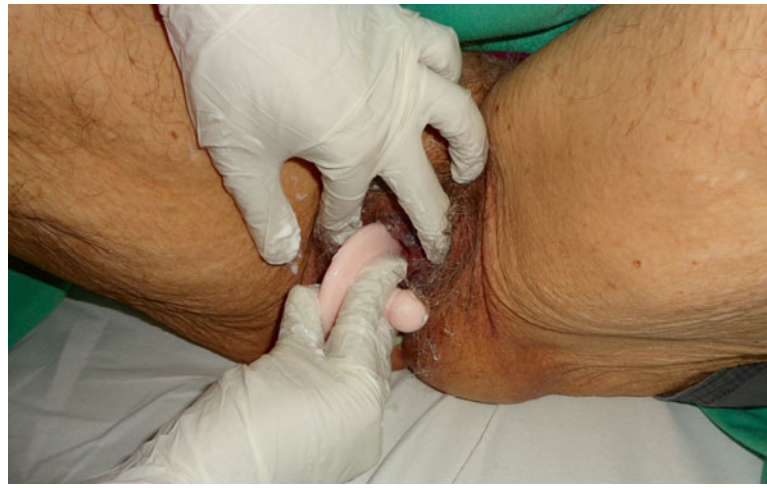

Fig. 13.10 Insertion technique of Gellhorn pessary

Step 2

The vagina is inspected with specula for infection or ulceration. The ring is cleaned with water and soap, applied with estrogen cream, and reinserted.

Gellhorn Pessary

Gellhorn has a concave disk portion attached to a stem. It is similar to the shelf pessary in design but flexible. For insertion, the knob is squeezed toward the disk, and the disk portion is held parallel to the introitus (Fig. 13.10). Once the pessary is inserted behind the pubic symphysis, it will open out, and the concave portion of the disk is made to rest against the leading edge of prolapse, forming a suction. For removal of the Gellhorn (or Shelf), the knob is grasped, generally with the help of a ring forceps, while the concave end of the pessary is rotated to release the suction and the pessary is pulled downward and removed (Fig. 13.11a–c).

Type of Pessary

There is no consensus or guidance on the use of different types of pessaries in the management of POP [19]. In a trial comparing ring and Gellhorn pessary, there was no significant difference in the efficacy between the two types.

Follow-Up

In a multi-professional survey on pessary use, wide variations in the follow-up of patients with prolapse were seen [20]. A variety of recommendations have been published, but from a practical standpoint, follow-up intervals have to be convenient both for the patient and the caregiver.

The first visit should be after about 2 weeks; if the patient is comfortable and able to insert and remove the pessary, we recommend follow-up at 3-month intervals. Women who are able to do so can remove the pessary about once a week. Most silicon pessaries can be used for at least 2 years. When the material starts to become stiff, the pessary is replaced.

Management of Complications (Table 13.3)

At the pessary trial, too small pessaries tend to fall out and too large pessaries can be painful. Alternative sizes should be tried and the patient should be comfortable and not feel the pessary.

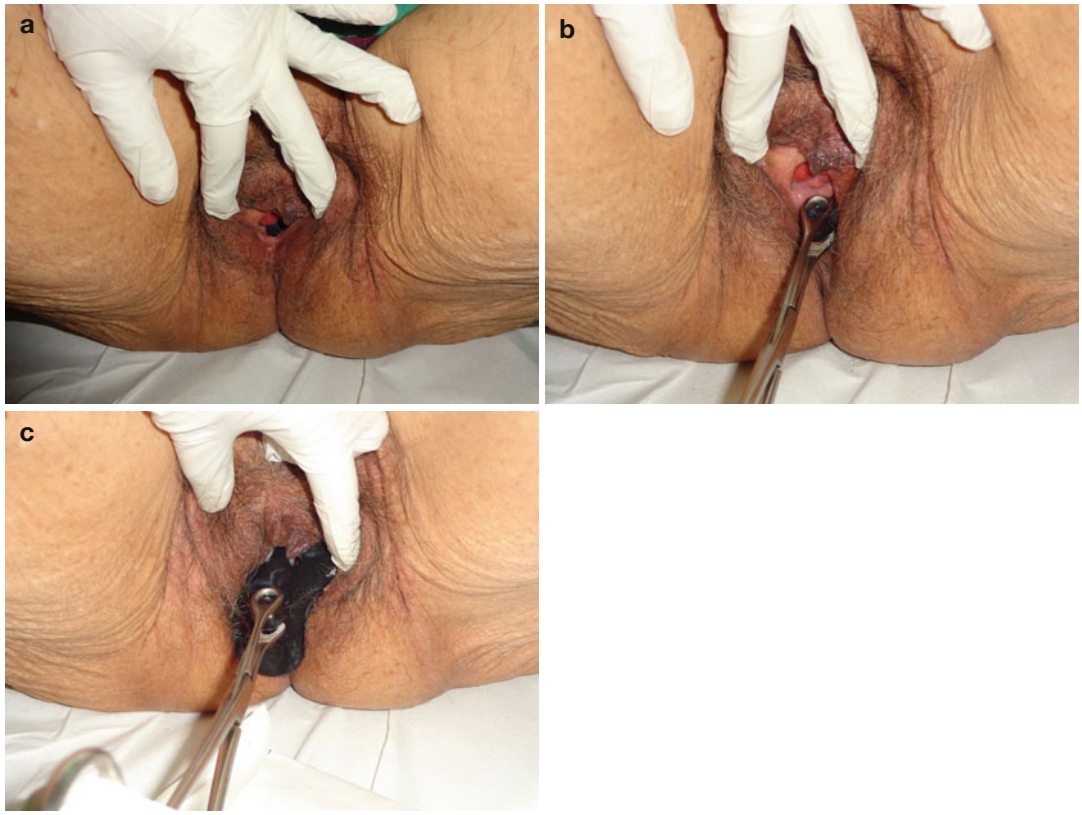

Fig. 13.11 Removal technique of Shelf pessary

Table 13.3 Common problems with pessary use

Vaginal discharge
Spontaneous expulsion
Discomfort/pelvic pain
Bleeding or spotting
Urinary incontinence
Difficulty with defecation

A too large pessary can also cause vaginal erosion and ulceration of the vaginal epithelium. The pessary should be removed and the vagina treated with local estrogen. After healing of the ulceration, a smaller size should be inserted.

POP may mask stress urinary incontinence by kinking the urethra (occult or latent incontinence). Reduction of prolapse with a pessary can unmask SUI. If a patient with a ring pessary complains of stress urinary incontinence, the ring should be replaced with a support and knob ring.

If a patient reports vaginal discharge or odor, the pessary should be removed at least once a month. This problem usually affects people who neglect the pessary or change it infrequently. Fistulae with neglected (forgotten) pessaries have been reported [21].

Difficulty in pessary removal with impaction has been noted. Use of estrogen cream helps with removal but rarely surgical removal may be indicated. In general most women are able to tolerate pessaries well. The factors which can influence the trial of pessary to fail include patient's discomfort after fitting, age <65 years, vaginal length <6 cm, and wide genital hiatus [22].

Conclusion

Surgical management of POP, though effective, might not be the treatment choice in some women. PFMT and pessaries are a good first-line option in all women with

POP, being noninvasive with minimal side effects. Most women with prolapse can be successfully fitted with a pessary and complications with its use are usually minor. In sexually active women, the use of pessaries, which they can self-care for in terms of removal and reinsertion, is likely to increase the use and compliance.

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Anterior and Posterior Pelvic Organ Prolapse

14

Aparna Hegde

Pelvic organ prolapse (POP) is a highly prevalent problem and approximately 200,000 surgical procedures are performed annually for prolapse in the United States [1]. Thirty percent of women who have undergone prolapse or incontinence surgery in the past will require an additional surgery for the same in their lifetime [2]. Women with symptomatic POP experience disruption of their day-to-day life as well as sexual function. Reconstructive surgery may be required in patients where conservative option is not acceptable or feasible. The prevailing view is that a common pathogenesis underlies support defects at different sites of the vaginal wall and various defects can coexist. Apical support is essential and contributes to support of the anterior and posterior wall and hence any surgical repair of the same should include repair of the apical prolapse [3, 4].

Pathogenesis of Anterior Vaginal Wall Defects and the Importance of Apical Support

The current theory regarding anterior vaginal wall defect is that, the herniation of bladder and/or urethra into the vagina, does not result from thinning or stretching of the anterior vaginal wall as previously thought. It results principally from a specific defect in the support structures of the vagina [5, 6]. Hence, performing an anterior colporrhaphy which involves just surgical plication or tightening of the anterior vaginal wall without addressing the specific defect or the apex has been proposed as one of the primary reasons for recurrence, in anterior prolapse repair [7, 8].

Four specific anatomic defect sites for the anterior vaginal support were originally described by Richardson [9] (Fig. 14.1):

Midline Defects

Midline or central defects arise from defects in the pubocervical fascia which extend anteroposteriorly. They are visualized as a bulge of anterior vaginal wall centrally on Valsalva maneuver, but the lateral attachments to the pelvic sidewalls are still intact. These defects could be responsible for stress urinary incontinence as they often interrupt the support of the urethrovesical junction, contributing to urethral hypermobility.

Paravaginal Defects

The detachment of the lateral vaginal wall from the arcus tendineus fascia pelvis (ATFP) unilaterally or bilaterally, leads to paravaginal defects. The lateral vaginal sulcus descends and can lead to the loss of the urethrovesical angle and stress urinary incontinence. These defects are identified

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A. Tamilselvi, A. Rane (eds.), *Principles and Practice of Urogynaecology*,
DOI 10.1007/978-81-322-1692-6_14, © Springer India 2015

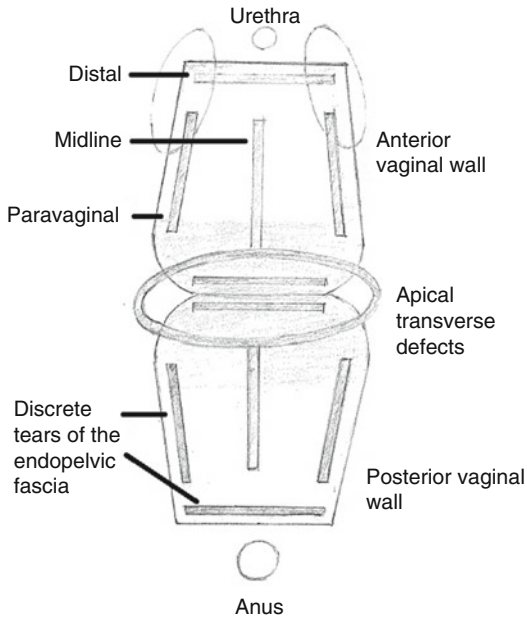

Fig. 14.1 Discrete defects of the anterior and posterior vaginal wall (midline, paravaginal, transverse and distal defects)

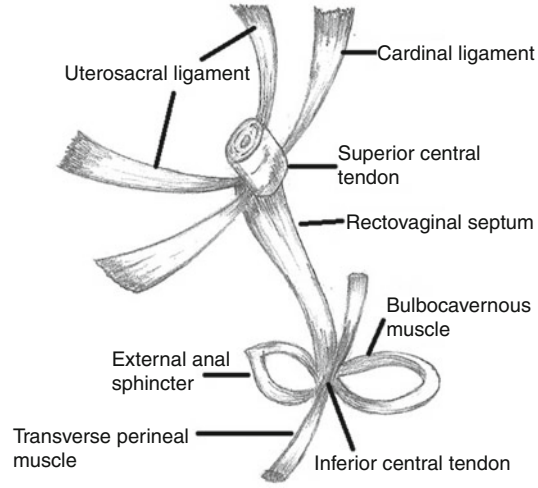

Fig. 14.2 Supports of the posterior vaginal wall

by placing the two blades of a ring forceps in the lateral vaginal sulcus and elevating them. If the prolapse resolves, then it is due to paravaginal defect. Though most clinical practice and research have focused on midline and paravaginal defects, the methods to diagnose them have not been validated.

Transverse Defects

The transverse separation of the pubocervical fascia from its insertion into the ring of connective tissue around the cervix and the uterosacral ligaments can lead to an anterior compartment prolapse that appears to originate high on the anterior vaginal wall or near the cervix [9]. Many recent studies underline the intimate interaction between anterior and apical compartment support systems. Summers et al. performed a quantitative analysis of the relationship between anterior and apical compartment support and found a relative risk – r^2 value of 0.5,3 indicating that half the size of the anterior compartment prolapse is explained by the apical compartment and vice versa [10].

The importance of restoring apical support when repairing anterior prolapse becomes apparent when one considers the trapezoidal anatomy of the endopelvic fascia on which the bladder is supported. The most common site of defect in anterior wall prolapse is at the point of attachment of the wide dorsal part of the fibromuscular fascia to the ischial spines [6]. Therefore, simply plicating the fibromuscular tissue from side to side during anterior colporrhaphy would not reestablish the normal anatomy of the upper one third of the anterior vaginal wall. Chen et al. used a biomechanical model to prove that the magnitude of anterior vaginal wall prolapse is a combined function of both pubovisceral component along with uterosacral and cardinal ligament ("apical support") impairments [11] (Fig. 14.2).

Distal Defects

These are the least common and occur due to a break in the fibromuscular support of the anterior vaginal wall, just before the insertion into the pubic symphysis. These tend to be smaller, but may be associated with urethral hypermobility [6].

Pathogenesis of Posterior Vaginal Wall Prolapse

The upper third of the posterior vaginal wall is suspended by the cardinal-uterosacral ligament complex and the distal third fuses with the perineal body (Fig. 14.2). The middle half is supported by

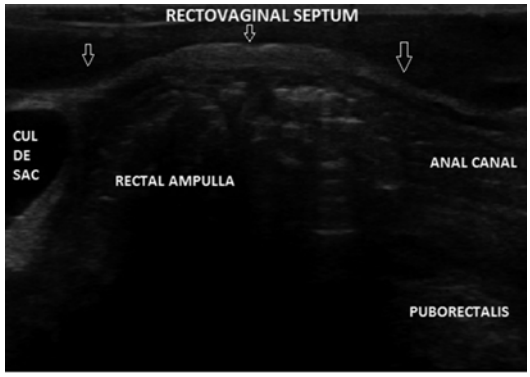

Fig. 14.3 Midsagittal plane ultrasound image of the posterior pelvic compartment showing rectovaginal septum. (using the BK Medical Pro Focus UltraView machine)

fascia attached laterally to the ATFP. There is controversy regarding the presence of a rectovaginal septum; however, histological studies and imaging studies have confirmed its presence [12, 13] (Fig. 14.3).

The levator ani muscle and rectovaginal septum work together, to provide the necessary support to the posterior vaginal wall. The perineal body is suspended by and attached to the puborectalis muscle. The puborectalis muscle forms a sling that creates an angle of 45° at the anorectal junction. Proximally, the vagina is supported by the pubococcygeus and iliococcygeus muscles on which it lies. In a woman with a healthy pelvic floor, the puborectalis muscle is in a resting tone which closes the vaginal canal and causes the anterior and posterior walls of the vagina to lie in apposition. There is little stress placed on the rectovaginal septum and connective tissue support of posterior vagina even during defecation, as the increased pressure placed on the posterior vaginal wall is equilibrated by the opposing anterior vaginal wall. However, with damage sustained during childbirth, the levator hiatus enlarges and the vaginal canal opens [14]. Defecation in such a situation leads to increased strain on the endopelvic fascial attachments and the rectovaginal septum which can lead to discrete tears resulting in rectocele and excessive perineal descent.

Selection of Patients for Anterior and Posterior Prolapse Repair

The two key questions that need to be asked before surgery are whether the symptoms of prolapse are bothersome enough and can they be attributed to the prolapse. Conservative therapy should be discussed prior to surgical intervention. It is important not to expose asymptomatic women or women with non-bothersome symptoms to the risks of surgery. It is also advisable to defer prolapse repair until the woman completes childbearing.

Planning of Surgery

Anterior and posterior vaginal wall defects very rarely occur in isolation. Thorough pelvic floor examination, including POP-Q assessment is essential, to determine whether there is coexistent prolapse of other vaginal compartments. When prolapse is present at more than one anatomic site, a coordinated approach to the repair is required. The outcomes following both anterior and posterior wall repairs have been shown to be significantly improved, when performed concurrently with an apical support repair. In the presence of an apical prolapse, surgical support of apex, either abdominal or vaginal should be performed prior to an anterior colporrhaphy or posterior repair. In combined abdominovaginal procedures, it is always better to move from a clean field (abdominal) to a clean-contaminated field (vaginal) to decrease the risk of infection.

Important issues to address are, if prolapse coexists with urinary tract or bowel problems (urinary incontinence, urinary retention, constipation, or fecal incontinence). An urine analysis is usually done to rule out infection in symptomatic patients. Urodynamic testing should be considered in a patient with urinary incontinence and POP, as the combined condition falls in the category of complicated incontinence for which urodynamics has been recommended by the ICI guidelines [15]. Even if the patient does not complain of voiding dysfunction or incontinence, in the presence of an anterior wall pro-

lapse of grade 3 or greater, it may be preferable to test urethral function with the prolapse reduced [15, 16]. A woman with severe anterior vaginal wall prolapse may be continent because of urethral kinking [17] and it is important to identify occult incontinence by prolapse reduction. Pessaries, large cotton swabs, or ring forceps-type instruments can be used to reduce anterior wall prolapse at the time of urodynamic testing, taking care that the urethra is not compressed. If concomitant stress urinary incontinence is proved to exist along with the prolapse, the choice of performing an appropriate anti-incontinence procedure along with the prolapse surgery can be offered with appropriate counseling.

Sexual dysfunction can be associated with POP and women may also experience dyspareunia or other sexual problems following repair. It is therefore important that whatever repair is chosen, attempt should be made to maintain adequate vaginal length.

The patient also needs to be assessed for pelvic floor dyssynergia (incoordinate contraction of the pelvic floor muscles) on pelvic examination or by ultrasound. In the presence of dyssynergia, the patient should be advised to undergo a course of physiotherapy, to re-learn proper pelvic floor contraction. A patient who pushes instead of squeeze while performing Kegel's maneuver is most likely to be harmed rather than helped by the prescription of Kegel exercises.

In a patient with defecatory dysfunction along with posterior wall prolapse, defecogram or defecating proctogram may be useful to determine the presence of occult rectal prolapse, intussusception, or non-relaxation of the levator ani. If such defects are noted, it is essential to counsel the patient about possible management options. For example, in a patient with non-relaxing pelvic floor during defecation, physiotherapy and biofeedback to relearn appropriate relaxation of the levator ani muscles may be needed before surgery.

Lastly, it is important to determine the patient's expectations from treatment to determine the best possible surgical treatment for her.

Surgical Repair of Anterior Prolapse

George White [7] said at the beginning of the twentieth century that "The only problem in plastic gynecology left unsolved by the gynecologist of the past century is that of permanent cure of cystocele." Irony is that, this is true even now.

The principles in intraoperative care includes, use of prophylactic antibiotics and interventions for thromboprophylaxis. In the modified lithotomy position, the patient is examined under anesthesia, to determine the site-specific defects and the ease of approach to sacrospinous ligaments. The Lone Star vulvovaginal retractor is useful in the absence of trained assistants [16].

Vaginal Repair

Anterior colporrhaphy is the procedure of choice for repair of anterior wall prolapse. The technique described here takes care of any transverse defect, in which the pubocervical fascia is detached from the pericervical ring. The procedure starts with a vertical midline incision. The vaginal epithelium is then dissected off the underlying fibromuscular layer to the lateral vaginal sulci and up to the vaginal apex or cervix. This layer is then plicated in the midline using interrupted 2-0 polyglactin 910 suture (Vicryl) from the level of the bladder neck to vaginal apex. When placing the final most proximal suture, the endopelvic fascia is incorporated with the cardinal ligaments, thus reattaching the anterior vaginal wall to the vaginal apex (either the cervix or vaginal cuff) using permanent suture. When the uterus is still present, the proximal suture is placed through the cervical stoma. Since most cystoceles begin as superior transverse fascial tears from the cervix/apex, incorporation of the apical-most plication stitch to the supportive tissue of the apex is advocated (Fig. 14.4). Addressing this defect, with reattachment of the torn fascia at the time of anterior colporrhaphy, restores the continuity between anterior vaginal wall and apex, which can decrease the recurrences. A study comparing the use of permanent suture for the proximal apical stitch as opposed to

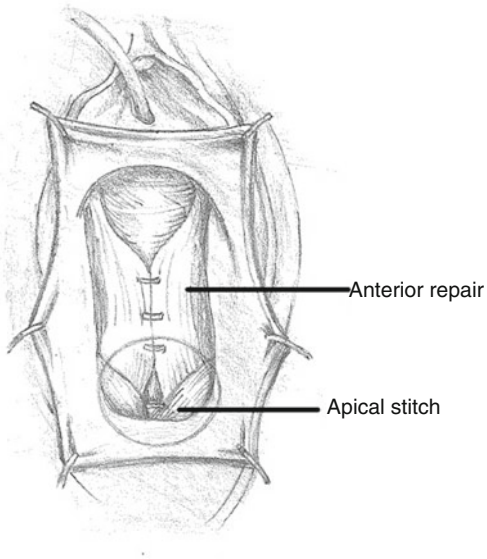

Fig. 14.4 Apical stitch of the anterior repair

absorbable has conclusively shown that the use of permanent suture is associated with improved anatomic correction, however, at the expense of increased suture exposures [18].

Paravaginal repair is the reattachment of the lateral sulcus of the anterior vaginal wall to the ATFP using permanent sutures, by careful dissection through the vesicovaginal space. The two points which mark the boundaries of the linear ATFP to which the vaginal wall will be reattached are, the ischial spine and the inferolateral pubic bone. Grade 0 permanent suture is passed perpendicular to the arcus 1.5 cm anterior to the ischial spine (first suture). Four to five sutures are then passed through the arcus with 1 cm distance between the sutures. Usually four to six sutures are placed on each side in a complete bilateral paravaginal defect. After the sutures are placed through the arcus, anterior fascial plication and other anterior compartment procedures are performed as indicated. The sutures are then sewn to the connective tissue layers of the bladder and the vagina at matching levels. For each of these landmarks, a point halfway between the ramus and the midline is chosen. Sewing the vagina and bladder at points too lateral, results in an inadequate lift, and choosing points too medial yields a very dramatic anterolateral elevation of the vagina that is difficult to close. In contrast, choosing the midpoints result in a tension-free skin closure. The vaginal wall is then closed with 2-0 Vicryl after trimming any excess vaginal epithelium [16].

Abdominal Paravaginal Repair

In the abdominal paravaginal repair, low transverse or vertical midline incision may be used to enter the retropubic space. An intravaginal finger helps in demonstrating the location and extent of the paravaginal defect on either side. The first stitch is near the vaginal apex, the obturator internus muscle and fascia, with the arcus included, all within 1–2 cm of the ischial spine. The permanent sutures are then continued towards the pubis, reattaching the lateral vagina to pelvic side wall about 1–1.5 cm apart. After placing all the sutures, the knots are tied, from the spine up to the pubis. Any bleeding that may occur from the suture site, usually stops when the sutures are tied.

The laparoscopic approach achieves the same repair that would otherwise be done through the abdominal route. Utilizing an infraumbilical open laparoscopy technique and three ancillary ports, the peritoneal cavity is entered, and the superior bladder border is identified. Transperitoneal entry into the retropubic space is then achieved. The repair is then similar to that described for the abdominal approach [16].

Augmented Repair of the Anterior Wall

Augmented repair of the anterior wall with either synthetic mesh kits or biological grafts came into vogue because of the poor long-term success rates reported with traditional anterior repairs. Although the connective tissue is reattached to strong supports in traditional repairs, the tissue itself is weak and the repair is less likely to be durable.

In the last decade, many surgeons moved from the earlier traditional paradigm of addressing single compartments with autologous tissue to

newer multicompartment composite procedures utilizing synthetic mesh or biological grafts. Synthetic mesh-trocar-based kits such as Apogee™, Perigee™ and Prolift™ were developed to simplify the placement of mesh, and studies have shown cure rates in the range of 87–96 % [19, 20]. A second-generation mesh kit using type 1 macroporous polypropylene lightweight mesh (IntePro Lite™) for combined anterior/apical prolapse repair has been studied and found to have anatomic success rates in the range of 92 % with minimal mesh erosion [21]. A recent Cochrane review has confirmed that mesh use in the anterior compartment has a lower failure rate when compared to traditional repair [22, 23]. However, a systematic review of complications by the Review Group of the Society of Gynecologic Surgeons (SGS) found a high incidence of graft erosion, wound granulation and dyspareunia (10.3, 6.8, and 8.9 %, respectively) following the use of synthetic mesh [24]. In light of the recent FDA notifications cautioning the use of synthetic mesh [25], use of mesh-augmented repair has now become controversial and surgeons are going back to traditional native tissue repairs.

Various biological graft materials have also been used for anterior repair with variable success. Anatomical recurrence rates following the use of biological mesh (Pelvicol™) for anterior wall reinforcement vary from 6.9 to 50 % [26–28]. Gomelsky et al. [29] and Mahdy et al. [30] used porcine dermis absorbable graft (InteXen™) and reported failure rates of 12.9 % (mean follow-up 24 months) and 3.8 % (mean follow-up 8 months), respectively. Further studies are required before the efficacy of biological grafts in improving success rates of anterior repair can be validated. Studies assessing outcomes following different surgical techniques in anterior prolapse, has been tabulated in Table 14.1.

Surgical Repair of Posterior Prolapse

Traditional posterior colporrhaphy and site-specific repair are the two main methods of rectocele repair.

Traditional Posterior Colporrhaphy

The perineal incision with which the repair begins may be horizontal, triangular, or diamond shaped, depending on the degree of perineal relaxation present. If the introitus needs to be narrowed with a perineorrhaphy, a triangular- or diamond-shaped incision is made. The posterior vaginal epithelium is then opened in the midline up to the apex of the vagina or to the cephalad border of the rectocele. The rectovaginal septum is then carefully dissected off the vaginal epithelium and plicated in the midline with continuous or interrupted delayed absorbable sutures.

The repair can include a levator myorrhaphy or plication of levator ani muscles. The levator ani muscle along with a portion of the fibromuscular fascia on either side is approximated in the midline with interrupted sutures. This helps in building a muscular posterior shelf, but may constrict the vaginal canal and be a source of postoperative pain and/or dyspareunia. In sexually active women, this can pose a problem.

The excess vaginal epithelium is trimmed at the end of plication and the vaginal epithelium is closed with a running or interrupted absorbable suture. If a perineorrhaphy has to be performed, superficial perineal muscles and the bulbocavernous muscles on either side, approximated in midline using fine absorbable sutures and the perineal skin closed with a subcuticular absorbable suture.

Site-Specific Fascial Defect Repair Technique

The purpose of the site-specific fascial defect repair is to identify the fascial tears contributing to rectocele and re-approximate the edges. The initial dissection is similar to that of posterior colporrhaphy; however, instead of approximating the fascia and/or levator muscles in the midline, the fascial tears are identified and repaired with interrupted sutures. The tears can be identified better by inserting a finger in the rectum and pushing anteriorly [40]. However, this technique is somewhat limited in its dependency on the

Table 14.1 Comparison of anterior compartment surgical outcomes

Author	Study design	No.	Success rate	Success criterion	Follow-up	Others
Anterior colporrhaphy (AC)						
Porges and Smilen [31]	Case series	388	97.5 %	Subjective, no reoperation	31 months	
Columbo et al. [32]	RCT: Burch vs AC for SUI and POP	33 and 35	97 % AC and 66 % Burch	Grade 1 at f/up and, subjective outcomes	13.9 years (mean)	39 % no coitus, severe dyspareunia
Weber et al. [33]	RCT: 3 surgical techniques	33 and 26 and 24	30 % standard AC, 42 % standard + mesh, 46 % ultra lateral AC	Stage 0 or 1 point Aa or Ba	23 months (median)	Polyglactin 910 mesh; 1 mesh erosion
Korshunov et al. [34]	RCT: standard vs polypropylene mesh	24 and 21	88 % standard, 100 % mesh	Stage 1 point Ba	9.5 months (mean)	14.3 % mesh required partial resection
Gandhi et al. [35]	RCT: dehydrated fascia lata vs standard AC	76 78	79 % fascia group 71 % control	Stage 1	13 months (median)	
Abdominal paravaginal repair						
Richardson et al. [36]	Case series	233	95 %	Functional symptomatic outcome	2–8 years	
Shull and Baden [37]	Case series	149	95 %		6–48 months	
Vaginal paravaginal repair						
Shull et al. [38]	Case series	62	93 %	Grade 1 Baden-Walker	1.6 years	
Young et al. [39]	Case series	100	98 %	Grade 1 Baden -Walker	11 months	

Modified from Ref. [20]
No. number of patients, *AC* anterior colporrhaphy, *RCT* randomized controlled trials, *NS* not specified

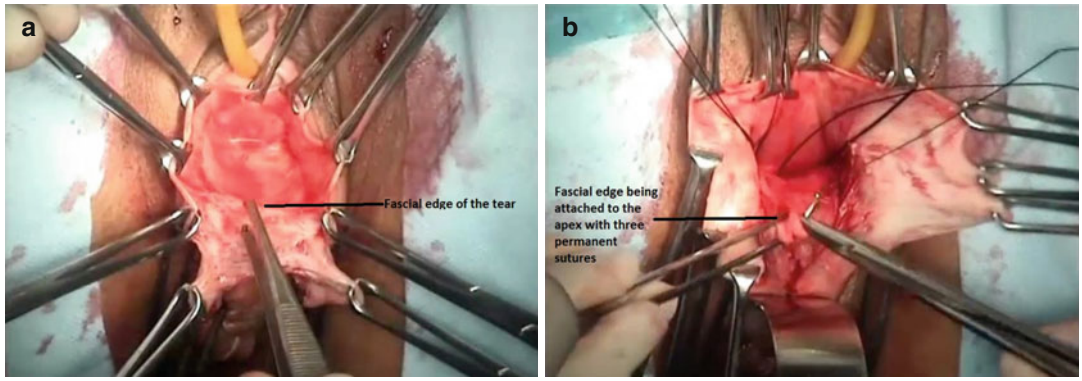

Fig. 14.5 (**a**) Fascial edge of the posterior vaginal wall detached from the apex identified. (**b**) Fascial edge reattached to the apex with three permanent sutures

operator's ability to accurately identify and correct each tear and on the assumption that the rectovaginal septum is strong everywhere else. In a study by Guerette (2004), most fascial defects were found to be apical, with separation of the fascia from the vaginal cuff [41]. Hence, the repair should ideally dissect the fascial edge of the tear and reattach it superiorly to the cervix or cuff usually with three permanent sutures (Fig. 14.5). This is usually followed by a perineoplasty.

Transperineal Repair Technique

In transperineal technique adopted by colorectal surgeons, a transverse incision is made across the bulbocavernous and transverse perineal muscles, thereby entering the plane between the external anal sphincter and vaginal mucosa. Dissection is carried out superiorly until the posterior cul-de-sac is reached taking care not to enter the pouch of Douglas. This technique usually requires the use of mesh over the whole length of the dissection [42]. Plication of the levator muscles and closure of the vaginal mucosa then follows as described above in the traditional posterior colporrhaphy repair.

Transanal Repair Technique

Colorectal surgeons commonly approach the rectocele through the anal canal and not the vagina. Many techniques have been described; however, in high rectoceles, this technique is difficult and excessive dilation for exposure can lead to anal incontinence following the procedure [43, 44]. The procedure involves mobilization of the rectal mucosa which is pulled out until taut. An inverted T-shape incision is made with the horizontal portion above the dentate line and the vertical line extending to the superior edge of the rectocele. A mucosal flap is dissected on both sides and the excess is removed. The rectovaginal fascia is plicated with absorbable sutures and the flaps are re-approximated at the end of the procedure.

Laparoscopic Rectocele Repair Technique

Laparoscopic rectocele repair involves opening the rectovaginal space and dissecting inferiorly to the perineal body. The perineal body is sutured to the rectovaginal septum and rectovaginal fascial defects are identified and closed. There are very few studies validating this approach.

Outcomes Following Posterior Colporrhaphy and Discrete Fascial Defect Repair

Posterior colporrhaphy has been described as the most misunderstood and poorly performed gynecologic surgeries [45]. Posterior colporrhaphy has an anatomic cure rate of around

76–96 % whether levator plication is performed or not. However, conflicting results have been reported regarding bowel and sexual function [46]. In some studies, constipation increased from 22 % preoperatively to 33 % postoperatively, and fecal incontinence increased from 4 to 11 % postoperatively [47–49]. However, Maher et al. reported a reduction in constipation from 76 to 24 % postoperatively in a prospective study [23].

Studies have also reported high sexual dysfunction rates of up to 50 % following posterior colporrhaphy [50]. Many theorize that this may largely be due to spasms of the levator, bulbocavernous and transverse perineal muscles. However, the added support provided by the plication of the muscles may help to reduce the recurrence rate of rectocele, and temporary dyspareunia can be viewed as an acceptable outcome for the long-term success of the repair. Interestingly postoperative de novo dyspareunia (reported rates of 8–26 %) has been shown to occur even in women in whom levator plication has not been performed [51]. Several authors have reported anatomic cure rates of site-specific repair to be equivalent with traditional posterior colporrhaphy, with significant improvement in quality of life measures with less postoperative dyspareunia [52–54]. Sands et al. reported a higher anatomical recurrence rate and similar dyspareunia/bowel symptoms in patients who underwent discrete fascial repair as opposed to traditional posterior colporrhaphy [55].

A randomized trial comparing posterior colporrhaphy to site-specific repair with or without graft augmentation, reported outcomes in favor of the traditional repair [56]. Interestingly, there was no difference in the functional outcomes between the two groups, and the rates of dyspareunia were equal.

Although discussing the outcomes of the transanal repair procedure is beyond the purview of this chapter, it must be mentioned here that infectious complications are more common following transanal repair procedures compared to vaginal repair. Hence, it may not be ideal to perform the transanal repair for enteroceles or high rectoceles.

Graft Augmentation for Posterior Repair

The use of either synthetic or biological grafts for the posterior vaginal wall is not supported by evidence [57]. The use of porcine tissue overlay has been found to have a significantly higher objective failure rate than site-specific or standard posterior colporrhaphy [56]. Poor results have been particularly reported with Pelvicol™, a porcine dermal acellular collagen matrix [58, 59]. In another trial of 161 women, comparable failure rates were found at the 1-year follow-up visit for absorbable synthetic mesh (polyglactin) and native tissue posterior colporrhaphy [60]. Precut mesh kits gained popularity before there was enough safety or outcome data supporting their use, and very few studies have studied their outcomes [20, 61]. Currently, mesh use is not recommended in the posterior compartment, since there has been no conclusive evidence of superior outcomes with mesh compared to traditional repair.

Conclusion

The repair of anterior and posterior wall prolapse should be performed as part of a multicompartmental repair where all vaginal wall support defects are addressed. Traditional repairs and its modifications need to ensure that the continuity of the endopelvic fascia with apical support is restored both anteriorly and posteriorly. Choosing the right technique for each compartment is essential to obtain good anatomical and functional outcomes.

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Surgical Management of Apical Prolapse

15

Arjunan Tamilselvi

Introduction

Pelvic organ prolapse (POP) is a common problem and can affect women of all age groups. The lifetime risk of undergoing an operation for POP is estimated to be around 11 % [1, 2]. With increase in life expectancy, the number of women presenting with POP is likely to increase in the future. The only persistent symptom of POP is the sensation of vaginal bulge, while increasing degrees of prolapse can be associated with bladder, sexual, or bowel dysfunction. POP is not a life-threatening condition but can significantly impair the quality of life of the individual.

The role of pelvic floor muscle training (PFMT) in the treatment of prolapse appears to be limited. PFMT appears to alleviate prolapse symptoms for a short term, but their role in long-term improvement is inadequate [3]. Use of mechanical devices, pessaries in prolapse treatment, appears to be effective and has been described in detail in Chapter 13. The fact that pessary needs to be used lifelong with regular changing may influence some women to choose a surgical option for their POP.

Assessment of POP includes identification of defect in all three vaginal compartments – apical, anterior, and posterior. The three levels of endopelvic fascial support described by DeLancey explains failure of specific aspects of the fascia in each of these compartments [4].

Apical compartment prolapse-uterovaginal and post-hysterectomy vaginal vault, results from failure of the uterosacral and cardinal ligament complex. The anterior compartment prolapse (cystocele) and the posterior compartment prolapse (rectocele) result from defects in the pubocervical and rectovaginal endopelvic fascia, respectively. Though these are described as isolated support mechanisms, the endopelvic fascia is a continuous layer extending from the sacrum proximally to the perineal membrane distally and across the pelvis through their attachments to the arcus tendinous fascia pelvis (ATFP) on either side of the pelvic wall. Apical prolapse is therefore usually accompanied by descent of the anterior and/or posterior compartments. In about 70 % of patients presenting with POP, more than one compartment is involved [2]. Apex appears to be the keystone of pelvic organ support, and attention to the apical compartment repair is vital in decreasing the risk of recurrent POP.

Surgical Management of Apical Prolapse

Uterovaginal (UV) prolapse and vaginal vault prolapse can be surgically addressed via the vaginal or abdominal route. In UV prolapse, the decision of

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A. Tamilselvi, A. Rane (eds.), *Principles and Practice of Urogynaecology*,
DOI 10.1007/978-81-322-1692-6_15, © Springer India 2015

Table 15.1 Surgical options for apical prolapse

Vaginal approach	Abdominal approach
Vaginal hysterectomy + McCall culdoplasty	Abdominal sacrocolpopexy
Sacrospinous ligament suspension	Abdominal sacrohysteropexy
High uterosacral ligament suspension	Abdominal uterosacral suspension
Iliococcygeus fixation	
High levator myorrhaphy	

whether the uterus is to be retained also needs to be decided. The age of the patient, desire to preserve fertility, presence of precancerous lesions or other pelvic pathology can influence the decision regarding uterine conservation. In general, removal of uterus is practiced to provide easier access for a complete pelvic floor reconstruction.

The route of repair, abdominal versus vaginal, is again determined by several other factors such as the age of patient, pre-existing co-morbid problems, site of prolapse, and the surgeon's preference for a particular technique. Of these, the site of defect appears to be the significant factor influencing the route. The commonly performed apical surgical procedures are mentioned in Table 15.1.

Vaginal Approach

In most parts of the world, surgical treatment of uterovaginal prolapse is the traditional vaginal hysterectomy (VH) with or without anterior and posterior repair [5]. Combining this with plication of the uterosacral ligaments (McCall's culdoplasty) or high uterosacral ligament suspension (HUSL) recreates the level I support of vaginal apex.

McCall's Culdoplasty

McCall described culdoplasty in 1957, where purse-string sutures were used to plicate the uterosacral ligaments along with the peritoneum to support the post-hysterectomy vaginal cuff [6]. The technique has been in regular use since then with modifications.

Culdoplasty technique usually involves placement of internal and external sutures on the uterosacral ligament after hysterectomy and plicating them in the midline. About one to three internal sutures are placed from the uterosacral ligament of one side to the opposite side, incorporating the peritoneum in between. This obliterates the cul-de-sac and reduces the risk of postoperative enterocele. The external or distal sutures on the uterosacral ligament anchor the ligament to the vaginal vault. The close proximity of the ureter at the cervical end of the uterosacral ligament should be borne in mind during McCall's culdoplasty.

The shortening and plication of the distal uterosacral ligaments in midline appears to be effective in apical support. The success rate has been quoted as high as 90 % at the end of a year to about 85 % in a 4–9-year follow-up study [7]. In a study comparing the sacrospinous ligament fixation with McCall's culdoplasty, recurrence in the anterior compartment was less frequent with the culdoplasty [8].

High Uterosacral Ligament Suspension (HUSL)

The technique of HUSL suspension was first described by Miller in 1927 [9]. The suspension procedure can be employed for vault support either at the end of the hysterectomy or for vaginal vault prolapse. In HUSL, the uterosacral ligament portion, proximal to the ischial spine, is used to suspend the vaginal apex along with incorporation of the anterior and posterior vaginal walls to create a pericervical ring.

The technique has been described in detail and popularized by Shull et al. in the last decade [10]. The uterosacral ligaments are identified posteromedial to the ischial spines at the 4 o'clock and 8 o'clock positions. The transverse portions of the pubocervical and rectovaginal fascia are identified, and bowels are packed away. Traction is applied on the uterosacral ligaments, and the strong suspensory ligament tissue towards the sacrum is traced. In the original technique, three double-armed, nonabsorbable sutures were placed through the ligament on the sacral side of ischial spine. The first suture is closer to the ischial spine and the other two sutures are then placed posterior and medial to the initial suture. This is repeated on the opposite uterosacral ligament. Once the sutures are placed on either side,

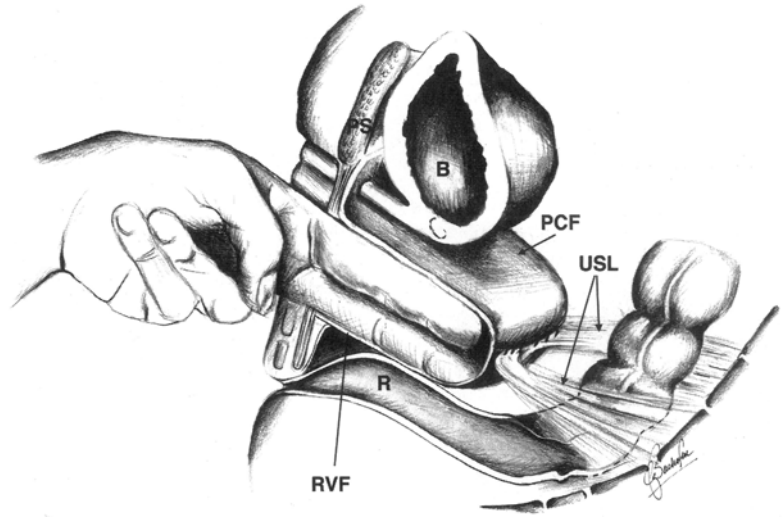

Fig. 15.1 Sagittal section showing suture passing through pubocervical fascia (*PCF*), uterosacral ligament (*USL*), and rectovaginal fascia (*RVF*). *B* bladder, *PS* pubic symphysis, *R* rectum (From Shull et al. [10]; with permission)

pack is removed and the double-armed sutures are used to secure the transverse portions of the pubocervical and rectovaginal fascia. Before the sutures are tied, 5 ml Indigo carmine is given intravenously. The sutures are then tied in sequence bringing the pubocervical and rectovaginal fascia together at the apex. Cystoscopy is performed to check ureteral patency, and the suspensory sutures are trimmed. The risk of ureteral kinking makes it mandatory to perform cystoscopy during this procedure. This technique appears to provide good support using the native tissue in the vaginal approach (Fig. 15.1).

In the case series by Shull et al., the anatomical success rate using Baden-Walker scoring system was 87 % for all sites with follow-up over 3.5 years [10]. A meta-analysis of the HUSL suspension has shown successful outcome for apical compartment to be 98 %, for anterior compartment 81 %, and 87 % for posterior compartment [11]. With a low overall recurrence of 4–18 % and a reoperation rate of less than 7 %, it is an effective procedure addressing the apical prolapse. In addition, the procedure also maintains the normal vaginal axis and appropriate vaginal length.

Complications

The major disadvantage of the procedure is the risk of ureteric injury varying from 1 to 11 % [10, 12]. The average distance between the uterosacral ligaments and the ureter at the cervical end is 0.9–1.4 cm, and moving towards the sacrum, the distance between the two is increased. At the intermediate portion beyond the ischial spines, where the suspension sutures are placed, the ureter is about 2.3–2.7 cm away from the ligament. Ureteral kinking can occur at this site and cystoscopy is important to visualize a free spill of dye on either side after suture placement. If there is no spill of dye-colored urine on any side, the sutures on that side should be cut one by one until spill is visualized. Once patency is established, there is no need to catheterize the ureter (Fig. 15.2).

New-onset neuropathic pain postoperatively has been reported following HUSL suspension. The presenting feature being sharp, stabbing pain radiating from buttocks posteriorly to the legs usually after the first 24 h of the procedure [13]. Entrapment of S1 to S4 nerves appears to be the cause of neuropathic pain. Removal of the uterosacral ligament suture on the affected side has been shown to relieve the pain, with complete resolution of symptoms in 6 weeks [14]. The close relationship of the sacral plexus to the uterosacral ligament makes it vulnerable to injury. The S1 trunk of the sacral plexus passes under the ligament about 3.9 cm superior to the ischial spine, the S2 trunk passes at 2.6 cm under the ligament, the S3 trunk passes at 1.5 cm, and the S4 trunk passes under the ligament at 0.9 cm,

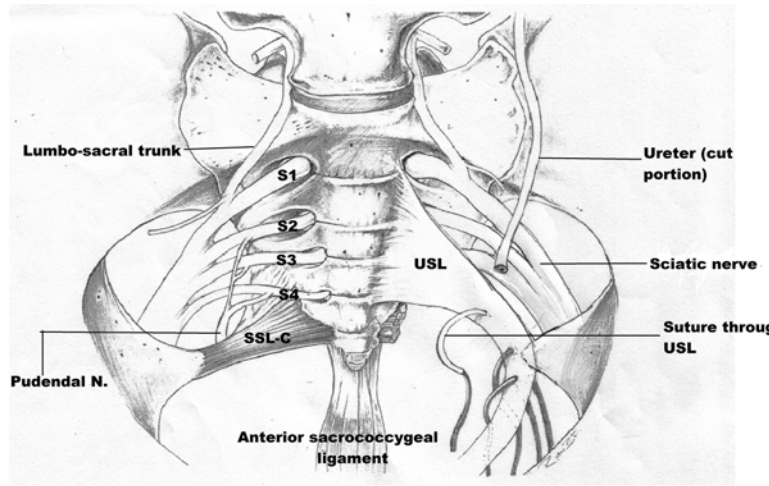

Fig. 15.2 Relation of the uterosacral ligaments to the S1–S4 trunk of the sacral plexus and their close proximity to uterosacral ligament sutures. Ureter cut portion to depict its relationship to uterosacral ligament and sacral plexus. *USL* uterosacral ligament, *SSL-C* sacrospinous ligament coccygeus muscle complex

superior to the ischial spine [15]. The close relationship makes the sacral nerves vulnerable to injury and entrapment.

HUSL in experienced hands is an effective procedure for apical prolapse using the native tissue, but the risk of ureteric injury mandates assessment of ureteral patency when this procedure is undertaken.

Sacrospinous Ligament Suspension (SSLS)

Sacrospinous ligament suspension was first described by Sederl (1958) and was popularized by Nichols and Randall [16]. SSL suspension aims to suspend the vault to the sacrospinous ligament. The ligament can be approached either via the anterior or posterior approach, most surgeons commonly choosing the posterior approach. Sutures are placed on the sacrospinous ligament and are secured to the vaginal vault. Tying these sutures, moves the vault towards the sacrospinous ligament and suspends the apex.

The technique involves a midline posterior vaginal wall incision and entering the rectovaginal space laterally. The ischial spine is palpated and the rectal pillars are dissected by a combination of sharp and blunt dissection. The sacrospinous ligament is palpated and viewed, passing medially and posteriorly from the ischial spine. The upper border of the ligament is palpated and delayed absorbable sutures are placed about one to two fingerbreadths medial to the spine, ensuring the suture lies inferior to the upper border and not around the upper border. The pudendal neurovascular bundle, sacral plexus, and sciatic nerve are in close proximity to the ischial spine and above the superior border of the ligament. Care is also taken to avoid the whole thickness of the ligament during suture placement. Two sutures are usually taken, and in bilateral procedures, the technique is repeated on the opposite side. The sutures are then passed through the vaginal wall on either side of midline and are held. The enterocele, anterior and posterior vaginal walls are repaired if indicated, and the upper portion of the posterior vaginal wall incision is closed. The sacrospinous ligament suspension sutures are then tied, moving the vaginal vault towards the ligament, making sure there is no suture bridge in between. The posterior vaginal incision is then closed entirely.

Success rate of SSLS has been quoted to vary from 67 to 94 % in several case series, with the mean follow-up varying from 22 to 83 months in different studies [17]. The variation partly being related to the fact that the anatomical outcomes have not been evaluated using a common grading system in all studies and also the recurrence in different compartments have been reported together in some (Table 15.2) [18].

Table 15.2 Cure rates for sacrospinous ligament suspension

	Authors	No. of patients	Follow-up (mean duration of follow-up) (years)	Cure rate (%)
1	Benson et al. (1996) [19]	42	1–5	67
2	Sze et al. (1999) [20]	54	0.6–6	77
3	Shull et al. (1992) [21]	81	1–5	82
4	Morley and DeLancey (1988) [22]	92	0.1–11	90
5	Maher et al. (2004) [23]	48	0.6–5	69
6	Meschia et al. (1999) [24]	91	1–6.8	94

In evaluating the recurrence of POP in different compartments following SSLS, the change in vaginal axis appears to be a determinant factor. The vaginal configuration is altered with the suspension, and the study by Rane et al. and Sze et al. using MRI showed there is alteration of the vaginal axis to an exaggerated posterior direction with SSLS [25, 26]. This increases the stress on the anterior compartment in standing and Valsalva disproportionately, which in turn amplifies the risk of anterior compartment prolapse. A study on long-term follow-up after SSLS shows the recurrence in anterior compartment 29 %, posterior compartment 5 %, and the apical 7 % [27]. Several case series have shown similar recurrence rates with anterior wall recurrence around 6–28.5 % and apical recurrence 2.4–19 % with SSLS [28].

Complications

The intraoperative complications reported in an analysis of 195 cases by David-Montefiore includes, vascular injury in 0.5 %, rectal injury in 0.4 %, and the need for blood transfusion in 5.2 % [29]. Buttock pain is a problem with SSLS, reported in about 3 % of patients and usually resolves in 6 weeks time. In pain persisting beyond 6 weeks, there appears to be a 50 % risk of significant long-term pain, and release of suture may have to be considered in these patients [30].

Pudendal and sacral neurovascular injuries are the serious complications of SSLF. The efficacy of the SSLS as a vaginal procedure for apical prolapse is well recognized and studies assessing the route of repair have largely compared SSLS with abdominal sacrocolpopexy.

Iliococcygeus Suspension

The iliococcygeus suspension recommended as an alternative to SSLS was first described by Inmon and involves fixing the vaginal vault to the iliococcygeus fascia just anterior to the ischial spines. The vault is secured bilaterally to the iliococcygeus fascia on either side [31, 32]. It is easier to perform than SSLS, but there are no RCTs favoring the iliococcygeus fixation over SSLS. The objective cure rate of 96 % has been reported in a case series with follow-up over 13 years [33].

Levator Myorrhaphy

A wide midline plication of the levator muscle is performed, and the vaginal cuff is attached to it in levator myorrhaphy [34]. In a prospective randomized study, comparing the high levator myorrhaphy with HUSL suspension, apical suspension is achieved in 96.7 %. However, the mean total vaginal length was significantly shorter after levator myorrhaphy [35], and sexual function is likely to be compromised with this technique.

Abdominal Route

Abdominal Sacrocolpopexy (ASC)

In abdominal sacrocolpopexy, the vault is secured to the anterior surface of sacrum at the level of S1–S2 by a graft material. The procedure was originally described by Lane [36]. Conventionally done as an open technique, ASC can also be performed using laparoscopic and robotic approaches.

In the open technique, the vaginal vault is lifted from below using an end-to-end anastomosis sizer (EEA) or a similar instrument (Fig. 15.3). The peritoneum over the vault is incised, and the vesicovaginal and rectovaginal spaces are entered along the proximal portion of the anterior and

Fig. 15.3 End-to-end anastomosis sizer used for elevating the vaginal vault

Fig. 15.4 Y-shaped manufactured mesh

posterior vaginal wall. This area is used for securing the graft to the vaginal walls. Polypropylene mesh is commonly used as a graft material. Addison et al. promoted the use of two separate graft strips for the anterior and posterior vaginal wall, so that the tension on both is varied and spread out [37]. Two straps of meshes are secured, each to the anterior and posterior vaginal walls using multiple delayed absorbable or permanent sutures. In the recent past, commercially designed Y-shaped meshes have become available for use in ASC (Fig. 15.4). The depth of the graft extent distally is determined by the extent of anterior and posterior wall prolapse. In cases of perineal descent associated with vault prolapse, extending the posterior graft up to the perineal body, termed as colpoperineopexy, has been described [38].

The anterior longitudinal ligament overlying the sacrum is exposed, taking care to identify the sacral vessels in this area and avoiding them. The close proximity of common iliac vein, middle sacral artery, ureter, sigmoid mesocolon, and sacral vessels in this area demands meticulous dissection. Apart from the median sacral artery, other accessory vessels have been shown to traverse the presacral space, and dissection in this area should take into account these anatomical aberrations.

The proximal free ends of both the anterior and posterior graft materials are secured to the anterior longitudinal ligament at the level of S1–S2, using a nonabsorbable suture or bone anchors. It is important to anchor the graft without tension. Anchoring the graft to sacral promontory will place the vagina under tension and alter the vaginal axis. If graft fixation is attempted below the level of S3, risk of hemorrhage in the presacral space is increased. The graft material once secured is placed along the sacral curvature and re-peritonealized. It is recommended that the peritoneum is closed over the graft material to reduce the risk of bowel obstruction.

Several studies have confirmed a good long-term success rate with ASC ranging from 77 to 100 % for the apical compartment [23, 38–42]. It is considered a durable technique for apical prolapse repair (Table 15.3). When success is defined by no recurrence in any compartment, the success rate quoted is 56–100 % [43]. In the study by Sze et al. and Rane et al. evaluating the vaginal axis postoperatively, both showed that with ASC there was better anatomical restoration of the vaginal axis and the near normal vaginal configuration is maintained [25, 26].

The incidence of postoperative stress urinary incontinence following ASC is reported as 4.9 % in a review by Nygaard et al. [44]. In the RCT by Brubaker et al., it was shown that combining abdominal sacrocolpopexy with Burch colposuspension reduced the postoperative symptoms of stress incontinence, without increasing other lower urinary tract symptoms [45].

In spite of its good success rates, not all surgeons offer ASC in patients with apical prolapse. Concerns such as longer operating time, longer duration of hospital stay, and complications of laparotomy limit its use, especially in the elderly,

Table 15.3 Cure rate for abdominal sacrocolpopexy

	Authors	No. of patients	Follow-up (mean duration of follow-up)	Cure rate
1	Cundiff et al. (1997) [38]	19	11 weeks	100 %
2	Timmons et al. (1992) [39]	163	33 months	99 %
3	Reddy and Malik (2002) [40]	11	60 months	100 %
4	Maher et al. (2004) [23]	47	24 months	94 % subjective
5	Addison et al. (1985) [41]	56	39 months	96 %
6	Tate et al. (2010) [42]	100	60 months	77 %

where preexisting comorbid problems favor a vaginal approach rather than an abdominal route. The risk of mesh erosion is also a concern with ASC. In an attempt to reduce the morbidity associated with complications of mesh erosion, several types of graft material have been tried in ASC. Biological graft materials such as fascia lata and rectus sheath have been used in an attempt to reduce the risk of mesh erosion from synthetic grafts. In a case series using biological graft material, the risk of mesh erosion was nil, but the failure rate of ASC was 83 % within a median follow-up of 17 months [46]. In a study by Tate et al. comparing polypropylene mesh with cadaveric fascia lata over a 5 year follow-up, the anatomical success rate with polypropylene was 93 % compared to 62 % with fascia lata [42]. The risk of mesh erosion also varies with the type of synthetic mesh used. With the use of polypropylene mesh, the risk of erosion is around 0.5 %, with Mersilene 3.1 %, with Gore-tex 3.4 %, and with Teflon 5.5 % [44]. It is recommended that some form of graft material is however used between the apex and sacrum, rather than affixing the apex directly to the anterior longitudinal ligament with sutures.

The risk of mesh erosion has been suspected to be increased with concomitant hysterectomy. Procedures such as supracervical hysterectomy or abdominosacral hysteropexy have been proposed to reduce this risk. The evidence on the role of hysterectomy and mesh erosion is however conflicting.

Abdominal sacrocolpopexy can be performed laparoscopically and as robotic-assisted approach as well. The laparoscopic approach to ASC aims to maintain the success rate of open technique with a decrease in morbidity associated with laparotomy. Case series have shown success rates ranging from 60 to 100 % with no increased complication rate with laparoscopic approach [47–49]. The use of laparoscopic approach for ASC is limited by the steep learning curve needed in this technique. In robotic sacral colpopexy, case series has shown that the success rate with the robotic approach is similar to that of open abdominal approach with a failure rate of 6 %. The data obviously is limited with no long-term case series yet [50]. In a study by Paraiso, comparing laparoscopic and robotic approaches in ASC, the operating time was longer with increased cost in robotic approach [51].

Abdominal Versus Vaginal Route in Apical Prolapse

Studies comparing ASC with SSL suspension have been analyzed in the Cochrane review on surgical management of pelvic organ prolapse [52]. The review reported that there was no statistically significant difference between the abdominal and vaginal approach in the number of women reporting prolapse symptoms, although there were more reports of subjective failure in the vaginal group (subjective failure after abdominal surgery 9/84 versus 18/85 after vaginal surgery). This also reported that there was no statistical significant differences in objective failure at any site. ASC was however better in terms of lower rate of recurrent vault prolapse, less postoperative dyspareunia, and less postoperative SUI, but the reoperation rate for SUI was similar in both groups. The operating time was longer, with longer time to recovery and it was more expensive with ASC compared to vaginal SSLS.

Uterus Preserving Surgeries in Apical POP

Hysterectomy as a component of prolapse repair may not be favored by some women though preferring a surgical intervention over conservative measures. The concept of uterine preservation in prolapse surgery is almost a century old, suggested by Bonney in the 1900s. The fact that the uterus is not the cause but rather the effect in uterine prolapse has been clearly defined with anatomical studies. Abdominal sacrohysteropexy, abdominal uterosacral suspension, sacrospinous hysteropexy, and Manchester procedure are some of the commonly employed procedures in uterine preservation.

Manchester procedure (Fothergill's Operation) is employed in the management of uterine prolapse due to cervical elongation. In this vaginal procedure, the cervix is amputated, and the cardinal ligaments are plicated and secured to the front of the cervix. This shortens the ligament and supports the uterus in its normal position. This is usually combined with an anterior and posterior repair.

Sacrospinous hysteropexy provides the transvaginal approach to apical compartment repair with preservation of the uterus. The technique being similar to SSLS, the sacrospinous ligament is approached via the posterior vaginal wall incision, but the incision is extended up to the posterior part of cervix. The sutures passed through the sacrospinous ligament are subsequently attached to the posterior side of the cervix just close to the midline. The sutures are secured to approximate the cervix to the ligament without a suture bridge. In a randomized study comparing the sacrospinous hysteropexy with vaginal hysterectomy for uterine descent, the risk of recurrent prolapse needing surgery was 11 % in the hysteropexy group compared to 7 % in the hysterectomy group [53].

High uterosacral ligament suspension is traditionally a vaginal procedure, but there are case series of laparoscopic uterosacral suspension with uterine preservation, showing good success rates [54]. To be precise, the techniques employed are more of plication of the uterosacral ligaments either involving them in a purse-string manner or plicating them in the midline. The sutures are passed from the left uterosacral ligament through the posterior vaginal wall and cervix and then through the right uterosacral and the serosa of the rectosigmoid in a purse-string manner, ending at the left uterosacral ligament. The case series by Wu using this technique was however small in number with seven patients with a follow-up of less than 2 years [55]. In the case series by Maher et al., the pouch of Douglas was obliterated by culdoplasty, and the uterosacral ligaments were plicated and reattached to the cervix using the laparoscopic technique [56]. The technique had a success rate of 81 % over a follow-up period of 6–32 months with two successful term pregnancies in this group.

In abdominal sacrohysteropexy where the uterus is retained, the proximal ends of the graft strips are secured to the anterior longitudinal ligament. The posterior strip is rectangular, and the distal end of the strip is attached to the posterior wall of the cervix and extended down the vagina depending on the extent of posterior vaginal prolapse. The distal end of the anterior mesh strip is cut into a Y shape, and the two arms are passed through the broad ligament to be secured to the anterior portion of the cervix. In a 5-year follow-up of abdominal sacrohysteropexy, no recurrence of uterine prolapse was observed, with an anterior wall recurrence of 7.7 % and a posterior wall recurrence of 5.7 % [57]. It thus appears to be an effective option in women requiring uterine preservation with apical prolapse.

Most procedures with uterine preservation provide good anatomical outcomes in prolapse surgery. However if the uterus is to be retained, a thorough preoperative evaluation to rule out any associated uterine or cervical pathology and appropriate preoperative counseling are important.

Obliterative Procedures

Rarely, when sexual function is no longer desired and reconstructive procedures are not ideal for the patient, obliterative procedure such as colpocleisis may be more appropriate. Colpocleisis involves denuding the vaginal epithelium off the anterior and posterior vaginal wall and suturing

the walls together. This effectively obliterates the vaginal canal except for the lateral portions, to provide drainage in women with intact uterus. This is referred to as LeFort's partial colpocleisis. A total colpocleisis on the other hand involves removal of all vaginal epithelium and suturing it together. Colpocleisis is an effective option for apical prolapse with low morbidity and usually used in frail elderly. One of the major concerns of colpocleisis is new-onset urinary incontinence, attributed to correction of urethral kinking.

Conclusion

In any patient presenting with POP, there are varied surgical options available. There are patient factors which can determine the route and technique such as age, associated comorbid factors, or other pelvic pathology. In addition, the surgeon's training and experience can also influence this decision-making process. A single approach or procedure based on the surgeon's preference is not always optimal. It is therefore essential for the pelvic surgeon to be skilful in the different surgical techniques employed in the correction of pelvic organ prolapse and tailor the surgery to the patient appropriately.

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Mesh in Prolapse Surgery

16

Jay Iyer and Ajay Rane

Introduction

Prolapse repair with synthetic mesh has become an area of debate in the last few years. The rationale for mesh use in prolapse surgery, the surgical outcomes and its complication profile will help us to understand the concerns and controversies regarding it. The lifetime risk of undergoing surgery for prolapse by age 80 is around 11 % and reoperation rate is quoted around 29 % [1]. The recurrence risk and the need for reoperation in nearly one-third to one-fourth of patients with prolapse surgeries means there is a need for more robust techniques in prolapse repair. Our understanding of pelvic floor anatomy changed dramatically since the description of "levels of pelvic organ support" by John DeLancey [2]. In order to fully understand the dynamics of prolapse surgery, both native tissue and mesh repair, it is important to have a brief overview of the functional anatomy of the pelvic floor, which has been covered adequately in an earlier chapter of this textbook. Many of the treatments for pelvic organ prolapse (POP) offered today have been developed bearing in mind this renewed understanding of pelvic floor anatomy.

Fascial repair also known as native tissue repair has been the mainstay of surgical treatment of pelvic organ prolapse until about two decades ago. The traditional repairs for the anterior and posterior compartments are performed vaginally as these operations are inherently difficult to perform via abdominal or laparoscopic approach. Native tissue repairs traditionally address midline fascial defects, but it is usually difficult to treat paravaginal or lateral defects in the fascial hammock. These defects account for a significant proportion of cystoceles and smaller proportion of rectoceles.

Recurrence risk with prolapse repair appears to be significant in the anterior compartment compared to the apical and posterior. In a study by Weber et al. comparing three different anterior repair procedures with a 23-month follow-up, failure rate of 70 % has been reported after a "standard" anterior repair [3]. The recurrence rate in the posterior compartment after posterior colporraphy is around 12–20 % [4]. The high rate of postoperative recurrence especially in the anterior compartment means there is clearly a potential to devise a mechanism or an operation that would effectively address all "parts" of the fascial hammock. Surgery addressing both level 2 and level 1 support concurrently. Thus, the enthusiasm for mesh surgery in prolapse repair was born out of the need to provide a more strong and reliable technique.

The next step in mesh repair was the selection of an ideal mesh type for prolapse repair.

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A. Tamilselvi, A. Rane (eds.), *Principles and Practice of Urogynaecology*,
DOI 10.1007/978-81-322-1692-6_16, © Springer India 2015

Biological materials and absorbable synthetic materials were not the best as they were not designed to produce permanent support to the weakened tissue and the primary aim with mesh was to reduce the recurrence risk. Among the nonabsorbable synthetic meshes, the type I (Amid classification) mesh was considered better for vaginal prolapse repair [5]. The type I mesh products are macroporous (>75 μm), monofilament fibers in a woven architecture. This type of mesh has been shown to promote better integration into the host tissue through scar formation.

Mesh Repair

Mesh use in prolapse surgery can be either "augmented" mesh repair (mesh overlay) or mesh "replacement" (needle kit). The first-generation needle-driven kits like Perigee™ (American Medical Systems), (Fig. 16.1) used helical needles through the obturator foramen to place a new hammock-type polypropylene mesh. The principle was that it would effectively address multiple defects in the fascial hammock. Anterior PROLIFT™ (Johnson and Johnson, NJ) worked on a similar principle. Similar meshes were developed to address the posterior compartment and apex: Apogee ™ (American Medical system), Posterior and Total PROLIFT™ (Johnson and Johnson, NJ).

In response to safety concerns raised by the US Food and Drug Administration (FDA) advisory statement [6, 7], first-generation mesh kits that involved relatively blind needle passes, resulting in uncommon but serious neurovascular complications, were modified in search of safer alternatives. The second-generation mesh kits use a single vaginal incision for both dissection and introducing the mesh device. These mesh kits use trocar-less delivery systems and lighter meshes and include the Elevate™ system (American Medical System) (Fig. 16.2) and Pinnacle™ (Boston Scientific). These mesh devices obviate the need to use blind needle pass and thereby reduce complication rates related to insertion.

Fig. 16.1 Perigee system

Principles in Mesh Repair

The first principle in mesh repair is the recognition that in most cases, POP can be treated successfully without mesh, thus avoiding the risk of mesh-related complications. Mesh surgery is chosen only after weighing the risks and benefits of surgery with mesh versus all surgical and nonsurgical alternatives [6–8]. The specific technique with each of the mesh kits is beyond the scope of this chapter, but the general guiding principles are discussed below.

Appropriate positioning of the patient is important to have adequate access for needle insertions and movement of trocars. It is incumbent on the surgeon to ensure correct patient positioning. There are no requirements for any special instruments and it is preferable to keep instrumentation simple and to bare minimum. Usually, a Scott retractor or the Lone Star retractor is helpful.

With trocar-based kits, it is good practice to mark the surface anatomy of the obturator foramen, adductor longus tendon, pubic tubercle, ischiopubic

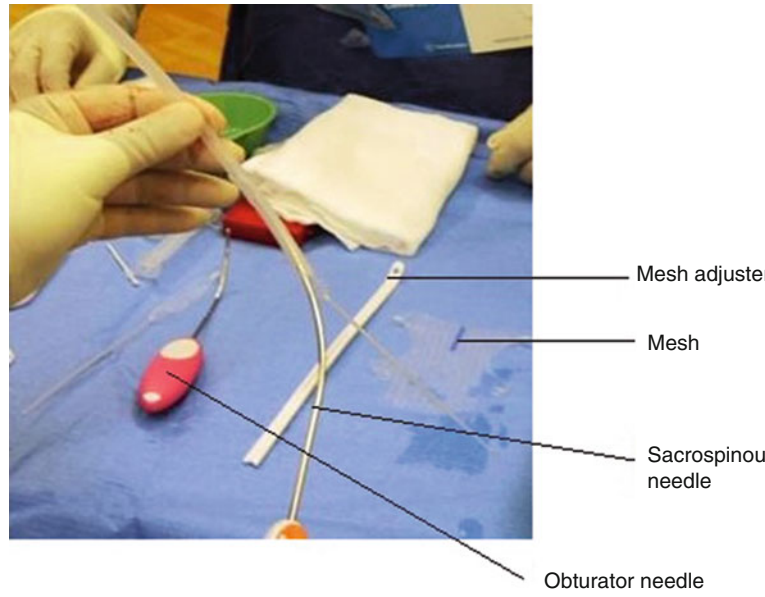

Fig. 16.2 Elevate system

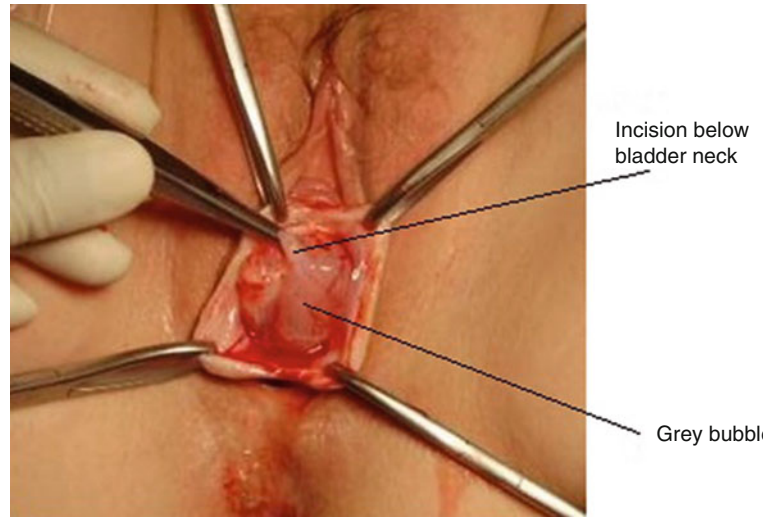

Fig. 16.3 Hydro-dissection and site of incision

ramus and ischial tuberosities before the incision. Additionally, it is useful to mark the bladder neck. Incising below the bladder neck potentially reduces the incidence of postoperative voiding dysfunction. In addition, a gynecologic pelvimetry helps to assess the subpubic angle for adequacy of access and the accessibility of the ischial spines and the sacrospinous ligaments. These are the important landmarks that need to be identified prior to any kind of pelvic floor reconstruction.

Procedure usually starts with hydrodissection with local anesthetic (Marcaine 0.5 %) mixed with diluted epinephrine (1 in 200,000). This helps in developing the natural avascular tissue plane and facilitates full-thickness vaginal dissection. Fluid in the space between the viscus and vaginal wall helps to define the correct plane of dissection (Fig. 16.3). A combination of sharp and blunt dissection carried out in this plane ensures the fascia is left attached to the vaginal

wall rather than to the viscus [9]. This ensures that the mesh lies directly in apposition with the prolapsing organ, reducing the chance of vaginal mesh extrusion.

Anchorage of the mesh to a strong pelvic floor support is pivotal to the success of mesh replacement surgery. The sacrospinous ligament fulfills the role of an "anchor," being relatively avascular, sturdy, with a fixed anatomical location and a well-circumscribed boundary, identifiable even in obese women. The sacrospinous ligament is approached anteriorly in mesh kits and this requires some degree of relearning and is a key skill in mesh repair [10]. To ensure a "four-point anchorage" in the anterior compartment, the needle is inserted through the obturator internus muscles and sacrospinous ligaments on either side, essentially mimicking the original "arcus-to-arcus" support of fascial bladder hammock. Intraoperative cystourethroscopy should be performed as a part of the standard operating protocol to detect inadvertent needle injury or mesh placement.

In posterior mesh placement, needle is inserted via incisions posterior to the anus, passed through ischiorectal fossa and directed towards the ischial spine to be anchored to the sacrospinous ligament complex. Rectal examination is done to rule out rectal injury, following trocar insertion and repeated at the end of the surgery.

It is recommended the mesh is trimmed to the size of the prolapse (Fig. 16.4) and placed in a "tension-free" manner. This reduces the risk of pain-related complications with mesh contracture. There is no need to excise vaginal skin prior to vaginal closure in mesh surgery. Minor trimming for purposes of aligning the edges is acceptable. A two-layered vaginal closure can make vaginal mesh exposure less likely and reduce dead space preventing hematomas. (Rane A., personal communication, 2010).

At the end of the mesh repair, there may be considerable residual laxity of the vaginal skin unlike in native tissue repairs. This in fact denotes appropriate mesh tensioning [9, 11, 12]. Mesh surgery factors in the concept of "vaginal remodeling" that allows surrounding tissues to restructure in much the same way as the vagina involutes following vaginal birth [11].

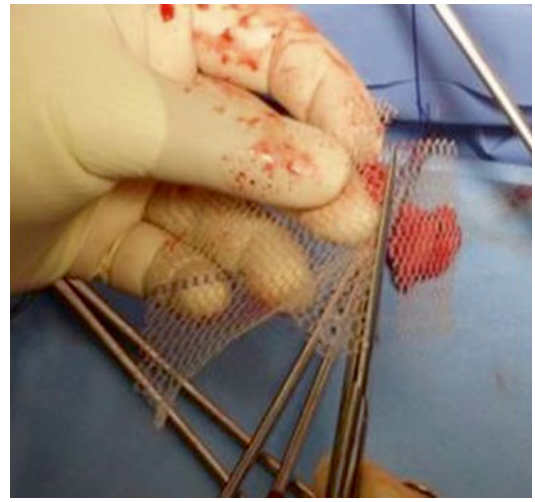

Fig. 16.4 Mesh trimmed to correct "dose"

The major issue with mesh repair is its use without adequate training. Proper training in the use of mesh devices is ideally a three-staged procedure: didactic training and cadaveric workshops, followed by preceptor training at the trainer's operating facility, and finally proctoring at the trainee's own hospital. Reference to the local college/Urogynecological society guidelines will ensure ongoing training, quality control, audit, and peer review. It is also important that mesh surgery be performed as per protocol established by the manufacturer, as any deviations from the accepted technique can cause complications and is medicolegally indefensible.

Surgical Outcomes

Several initial prospective and retrospective cohort studies using mesh kits, showed good anatomical success rates in the range of 80–100 % with follow-up over 3–24 months [13–17]. Studies with medium- and long-term follow-up and randomized controlled trials (RCT) comparing mesh versus fascial repair, showed variable results. In an one year RCT, comparing objective outcomes of mesh versus fascial repair of all compartments, there was an overall recurrence of 63 % in mesh compared to 70 % with no mesh. Most recurrences occurred in the anterior compartment - 46 % in mesh and 60 %

with native tissue [18]. In a RCT comparing anterior colporraphy with mesh, objective assessment at the end of a year showed a success rate of 61 % with mesh and 35 % with colporraphy [19]. Other RCTs comparing the mesh with standard colporraphy have shown failure rates in the range of 9–28 % with mesh [20, 21]. In one long-term outcome analysis of vaginal mesh with native tissue repair in the anterior compartment, the 5-year cumulative risk of any repeat surgery was significantly higher for vaginal mesh, 15.2 % compared to 9.8 %, but risk of surgery for recurrent prolapse was similar [22]. In the posterior compartment, fascial repairs have been shown to give excellent results and there is no evidence to support the use of mesh in posterior repair [23].

The secondary outcomes of cohort studies and RCTs started to highlight the complications rates with mesh. In the RCT by Sokol et al., mesh exposure was reported in 15 % and no statistically significant difference between mesh and native tissue with respect to new-onset dyspareunia [18]. In the study by Altman, the rates of bladder perforation and intraoperative bleed were higher in the mesh group, with mesh exposure rate of 3.2 % [19]. It was the complication profile with mesh repair, lack of evidence for optimal management of these complications and long-term sequelae related to mesh complications that constrained its use. In view of the increasing concerns about mesh-related complications, the US FDA issued statements related to mesh use in prolapse surgery [6, 7].

Complications of Mesh Surgery

Intraoperative

Cystotomy and Urethral Injury

If cystotomy occurs during dissection and is central and accessible, repair of it does not pose a problem. A single- or two-layered closure with 2/0 polyglactin should suffice and a layer of fascia could be interposed over the cystotomy to bolster the repair. It would be usual to continue with the dissection; however, opinion is divided on whether it would be safe to use mesh after a cystotomy. Some surgeons argue that a "clean" midline cystotomy, if adequately repaired, does not contraindicate mesh placement [24]. Most surgeons however would defer mesh in this scenario. More commonly cystotomies occur in the lateral "tunnels" while accessing the sacrospinous ligaments and lateral pelvic wall. Risk is increased in patients with previous surgery and almost always due to improper surgical techniques. These cystotomies are difficult to repair, and the consensus in such cases would be to avoid using mesh and resort to a fascial repair instead. Urethral trauma by needles may occur with the upper needle passes into the obturator foramen with mesh kits. Performing an intraoperative cystourethroscopy is the only reliable method of detecting this complication.

Rectal Injury

If rectal/anal injury was encountered during dissection, standard practice would dictate repair of the laceration and abandonment of mesh repair. A standard fascial repair should be considered in these patients. Rectal injury if unrecognized can lead to rectovaginal fistula.

Fornix Tear

Forniceal puncture is not unusual in women with deep lateral fornices and results during the passage of the anchors into the obturator internus muscle. Creating an adequate subcutaneous tunnel along the length of the fornix, until the ischiopubic ramus reduces this risk. The technique of directing the needle posteriorly along the length of the tunnel before changing direction under the ramus also helps. If fornix tear is identified after the passage of the anchor/mesh, it is reasonable to undermine the vaginal skin at the site of puncture and close vaginal skin over the mesh.

Bleeding

Brisk bleeding can be encountered during dissection, after mesh insertion or while deploying the anchor. Sustained pressure with a pack and gauze usually suffices, as it is usually a venous bleed. In rare occasions, using hemostatic agent like Floseal™ (Baxter, IL) may be needed. Retroperitoneal hemorrhages with large hema-

toma have been reported with transvaginal mesh surgeries [25]. If a major vessel laceration is suspected, help from a vascular surgeon or an interventional radiologist may be indicated. Heavy bleeding from the iliac vessels can be life-threatening, necessitating a laparotomy and surgical vascular control.

Late Complications

Vaginal Mesh Exposure

Vaginal mesh exposure occurs in about 13–15 % of cases [18, 24]. The mean timing of exposure is around 234 days (range of 45–1040 days) [26]. Mesh exposure risk is not limited to vaginal mesh placement and has been reported with mesh use in abdominal sacrocolpopexy (ASC) as well. With anterior mesh, the risk of exposure is 9 %, with posterior mesh 8 % and with ASC risk is around 3 % [26]. The risk of vaginal mesh exposure is higher if the mesh is sutured vaginally during sacrocolpopexy [24]. Vaginal mesh exposure is possibly a healing abnormality when it occurs early, along the suture line and with no signs of infection. It can also be detected in the lateral vaginal wall or fornices. In a proportion of patients where mesh exposure is small (<0.5 cm) and asymptomatic, it can be managed with vaginal estrogen with or without mesh excision as an outpatient procedure. The vast majority, however, need to be reoperated with excision of mesh and fascial repair over the defect. Reoperation rate for mesh exposure is quoted between 8 and 36 % [24].

Visceral Mesh Extrusion

Bladder, urethral and rectal mesh extrusions have been reported after both vaginal mesh surgery and ASC. Bladder extrusion can present with hematuria, recurrent UTI, pain or fistula. Patients who have constant urinary or fecal incontinence immediately after surgery should be evaluated for vesicovaginal or rectovaginal fistula. Treatment involves removal of the entire mesh from the viscus, repair of the visceral defect and closure of vaginal defect. This can be done vaginally, but more often an open abdominal approach is needed. Laparoscopic and cystoscopic transurethral removals have been reported and the important principle is to remove the mesh completely. Urethral erosions are managed with urethrolysis, graft explanation and multilayer closure with Mauritius flap reinforcement.

Pain

The most troublesome and concerning complication of mesh is the pain resulting from contraction and/or hardening of the mesh, leading to dyspareunia and chronic pelvic pain. Feiner et al. defined mesh contraction as an adverse outcome following polypropylene mesh repair where patients experience vaginal pain with movement and dyspareunia [27]. Contraction typically occurs along the fixation arms of the mesh and rarely does the entire implanted mesh contracts. On examination, patient can have localized areas of prominent, tense and tender mesh under the vaginal epithelium. The reported rate of polypropylene mesh-related pain, ranges between 4 and 11 % according to the definition used [24].

Management usually involves meticulous history taking, mapping of the pain with accurate charting of the trigger points and extensive counseling. In-office trigger-point injection of bupivacaine with triamcinolone is useful to accurately identify the location of pain that is causing dyspareunia. After injection, the patient is asked to return home and resume sexual intercourse. If dyspareunia diminishes, surgical removal of the involved mesh segment is likely to ameliorate symptoms. If dyspareunia persists after injection, the problem may not be related to the mesh. This can be helpful in counseling the patient prior to mesh excision.

Mesh contraction should be managed by a surgeon who is experienced in extensive deep pelvic dissection, which is necessary to remove the mesh arms. Complete excision of mesh should be attempted only by experienced surgeons. The most troublesome segments can be excised with full-thickness vaginal dissection. Symptomatic relief is noted in over 90 % of patients, but sadly a few patients may never be cured completely [27]. Therefore, it is essential that women are

adequately counseled before primary surgery and particularly prior to reoperation to treat complications.

Diffuse vaginal pain after mesh implantation is unusual and in these patients, the report of pain has been preceded by an underlying pelvic pain syndrome. Management of such pain is controversial and many patients may not be cured even after the entire graft is removed. An existing pelvic pain syndrome should ideally be elicited in patients where mesh repair is considered.

The US FDA report (2011) stated that vaginal pain and dyspareunia were the most common adverse events reported [7]. Tijdink et al.'s report on surgical management of mesh complications stated that the most common reason for reoperation following transvaginal mesh was vaginal pain and dyspareunia (77 %) [28]. This is in contrast to the common perception that vaginal extrusion is the most common complication.

Infection

The exact rate of infection with vaginal mesh is unknown. With the type 1(Amid classification) mesh, the risk of infection is rare but has been reported. Untreated preoperative bacterial vaginitis is suspected to be an underlying cause. Typically, these patients complain of vaginal discharge and bleeding and can present with vaginal exposure of the mesh. Antimicrobial therapy should cover gram-positive, gram-negative, and anaerobic bacteria and the infected exposed mesh should be removed. Complications such as abscess, cellulitis and spondylodiscitis can occur with mesh repair and are quoted to be <1 % [24].

In order to achieve some uniformity in reporting mesh-related complications and also to simplify the auditing and reporting process for the same, a code-based classification has been proposed jointly by the International Urogynecological Society (IUGA) and the International Continence Society (ICS). This classifies mesh complications based on the category (C), time (T), and site (S) of complication and referred to as the CTS classification [29]. It is suggested that while reporting mesh complications, the CTS terminology and classification is used.

Factors Influencing Mesh Complications

Obesity (BMI >30) and smoking are independent risk factors for mesh exposure [30]. Sexual activity has also been reported to be a risk factor for vaginal mesh exposure. However, this could simply reflect that those who are sexually active are more likely to identify a mesh exposure. The association of concomitant hysterectomy with risk of mesh exposure has been an area of controversy with some studies suggesting increased risk, while others did not find any difference. A meta-analysis demonstrates that the addition of hysterectomy to a transvaginal mesh surgery significantly increases the risk of mesh exposure from 7.3 % without hysterectomy to 19.2 % with hysterectomy [24].

Prevention of Complications

Transvaginal mesh implants should be used with caution in certain group of patients (Table 16.1). In addition, it is important for surgeon to understand the dynamics of the mesh kit being used. Owing to the wide variety of devices available, it is important to appreciate that every "needle" in every "kit" is different: helical needles, open curve needles, self-retrieving needles and needles

Table 16.1 Patients where mesh should be used with caution in prolapse surgery

1. Primary prolapse cases
2. Patients younger than 50
3. Lesser grades of prolapse (POP-Q ordinal grade 2 or less)
4. Posterior compartment prolapse without significant apical descent
5. Patients with chronic pelvic pain
6. Postmenopausal patients who are unable to use vaginal estrogen therapy for any reason
7. Patients with previous irradiation
8. Poorly controlled diabetics
9. Patients on high-dose immunosuppressant and corticosteroids
10. Patients who do not want "foreign material" used in their repairs

with inner and outer sheaths. Knowledge of surgical anatomy, especially an appreciation of the course of the needles in the sagittal, coronal and axial planes of the pelvis, is crucial to the surgical safety. Complications occur when the surgeon fails to appreciate the counterintuitive movement of the needle; for example, with helical obturator needles, the handle needs to be pressed firmly in contact with the patient. If the handle is raised, the tip of the needle which is deep inside the pelvis moves away from the obturator foramen and has the potential to injure vessels and nerves in the lateral pelvic wall. Therefore, appreciating directional reversal of the needle tips with respect to the handles and understanding the spatial relationship of the structures and the needles within the pelvis, in a three dimensional view are critical [9, 31]. Widespread use of these devices without proper training and in the absence of robust trials to address the pros and cons of this new technology has resulted in uncommon yet serious complications [32–35]. The proliferation of different types of synthetic and biologic meshes without comprehending their individual biodynamics can lead to delayed complications [6–8, 36–40]. A "three-step training program" for the nouveau surgeon referred earlier is invaluable.

Current Role of Mesh in Prolapse Surgery

Attempts have been made to analyze the current role of mesh in prolapse surgery after the US FDA safety communication report [6, 7]. Review of the various outcomes with mesh surgery and comparing it with native tissue repairs has led to certain recommendations in each compartment.

In the apical compartment, commonly performed procedures are abdominal sacrocolpopexy (ASC) or uterosacral ligament or sacrospinous ligament suspension with or without vaginal hysterectomy. Comparing the ASC by laparoscopy with vaginal mesh repair, Maher et al. showed a higher objective success rate at 2 years with laparoscopic sacrocolpopexy (77 % vs. 43 %). The reoperation rate was higher with vaginal mesh repair 22 % compared to 5 % with laparoscopic sacrocolpopexy [41]. Comparison of the traditional native tissue vaginal repair in the apical compartment with vaginal mesh repair, the recurrence of POP at operated site was 45 % in native tissue and 10 % in mesh group at the end of 12 months. However, mesh exposure was detected in 17 % [42]. In the apical compartment, ASC has superior outcomes compared to a variety of vaginal procedures including sacrospinous colpopexy, uterosacral colpopexy and transvaginal mesh, with an acceptable risk-benefit ratio [43].

In posterior compartment prolapse, midline fascial plication without levatorplasty is the recommended procedure of choice. No evidence supports site-specific repair or the use of polypropylene mesh or biological graft in posterior compartment repair [23]. Anterior compartment prolapse has the highest potential for recurrent prolapse with traditional native tissue repair [3]. The review at the fifth International Consultation of Incontinence stated that "Consistent level 1 evidence demonstrates superior subjective and objective outcomes following anterior transvaginal polypropylene mesh as compared to anterior colporrhaphy (grade A)." The conclusion based on this was that polypropylene anterior compartment mesh offers improved objective and subjective outcomes compared with native tissue repair. These benefits however, must be considered in the context of increased morbidity associated with anterior polypropylene transvaginal mesh [44]. Newer lightweight single-incision mesh kits show promise in reducing the complications profile and require further evaluation.

Conclusion

The introduction of mesh in pelvic organ prolapse surgery undoubtedly revolutionized the surgical options available for POP. Good anatomical outcomes have been demonstrated with its use especially in the anterior compartment. However, the use of mesh in pelvic reconstructive surgery is associated with a risk of specific complications. Preoperatively, patients must be informed of these risks and informed of conservative and alternative surgical techniques. Mesh has a role in

reconstructive pelvic surgery in complex cases and in those with high risk of failure. Proper patient selection, standardization of the surgical techniques and improved surgical training are of paramount importance. Postoperative evaluation should take into account not only the objective outcomes but also the functional outcomes in POP surgery.

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Part IV

Ano-rectal Dysfunction

Obstetric Anal Sphincter Injuries

17

Aparna Hegde

Introduction

Care during vaginal birth has vastly improved over the last two decades. However, more than 85 % of women sustain various degrees of perineal trauma during vaginal delivery in the UK [1]. The incidence of severe perineal trauma varies between 0.5 and 3 % in Europe [2] and 6–9 % in the USA [3]. Recent years have witnessed a gradual increase in the incidence, with a recent survey in the UK revealing rates as high as 8 % [4]. Though the prevalence of perineal trauma in India is not really known and is impacted by variations in obstetric practice, including episiotomy rates, it is unlikely that the numbers in India are any different. Even more worrying is the fact that sphincter injuries are most often missed clinically at the time of delivery [5]. The aim of this chapter is to provide a comprehensive review of this very important topic including discussion of the risk factors, diagnosis and evidence in the management of obstetric anal sphincter injuries (OASIS).

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Sequelae of Anal Sphincter Injury: Why Is It So Relevant?

Obstetric anal sphincter injury is the commonest cause of anal incontinence [4]. Anal incontinence affects 4–6 % of women up to 12 months after delivery [6–8]. Up to 25 % of primiparous women suffer from altered bowel movement, including fecal incontinence postnatally and almost 1/3 of them have some anal sphincter trauma after their first vaginal delivery [9]. In the majority, both these symptoms and injuries are relatively minor and transient, but persistent incontinence to flatus or urgency of defecation is emotionally and socially debilitating and may delay return to normal after delivery [10]. In 30–50 % of women with clinically recognized anal sphincter injury, symptoms of fecal incontinence, urgency, dyspareunia and perineal pain are more common and may persist for many years [11–13].

It is a matter of great concern that women do not seek medical attention because of embarrassment and the notion that the problem is a normal consequence of childbirth [14]. The true prevalence of anal incontinence and its impact on women following childbirth is hence not really known. It is essential that gynecologists ask specific questions about symptoms of fecal incontinence, especially in the postpartum period.

A. Tamilselvi, A. Rane (eds.), *Principles and Practice of Urogynaecology*,
DOI 10.1007/978-81-322-1692-6_17, © Springer India 2015

Table 17.1 Classification of perineal trauma (RCOG)

Type of tear	Definition
First degree	Injury to the perineal skin
Second degree	Injury to the perineum involving the perineal muscles, but not involving the anal sphincter
Third degree	Injury to the perineum involving the anal sphincter complex:
3A	<50 % of the EAS thickness involved
3B	>50 % of the EAS thickness involved
3C	Both the EAS and the IAS involved
Fourth degree	Injury to the perineum involving the anal sphincter complex (both the EAS and the IAS) and anal epithelium

Classification of Perineal Injury

Classifications of perineal injury used until the end of last century were inconsistent and confusing with significant regional variations. In a systematic review of all relevant obstetric textbooks in the library of the Royal College of Obstetricians and Gynaecologists (RCOG), Sultan and Thakar found that 17 % did not mention any classification and only 29 % classified a third-degree tear as any tear that involves the anal sphincter [15, 16]. Incorrect classification resulting from this lack of standardization may have accounted for the fact that almost 33 % of women were found to sustain occult anal sphincter injury during vaginal delivery in the early 1990s [2].

Since 2001, the classification of anal sphincter tears proposed by Sultan et al. has been universally accepted and has been included in both the RCOG and the International Consultation on Incontinence guidelines [17, 18] (Table 17.1). The classification allows differentiation to be made between injuries to the external anal sphincter (EAS), internal anal sphincter (IAS) and anal epithelium. An isolated rectal tear without involvement of the anal sphincter is a rare occurrence and has not been included as part of this classification.

Occult Anal Sphincter Injury

Occult sphincter injury rates ranging between 6.8 and 28 % have been reported in the literature [19, 20]. Andrews et al. speculated whether these were truly "occult" injuries or simply missed clinically at the time of delivery [21]. The diagnosis of anal sphincter damage is often delayed for many years because the symptoms of fecal incontinence are not commonly reported in the immediate postpartum period and many patients remain asymptomatic for many years.

The importance of early diagnosis of sphincter injury cannot be emphasized enough. Faltin et al. showed a reduction in fecal incontinence symptoms at 12 months, in women who had surgical repair of sphincter injury at the time of delivery when compared with women who had no repair [20].

The role of endoanal ultrasound in the long-term management of patients who have undergone anal sphincter repair and in those presenting with symptoms following delivery is undisputed. However, in the immediate postpartum period, its role is debatable due to poor patient acceptability and poor image quality resulting from edema and should not be used on a routine basis.

Risk Factors for Anal Sphincter Injury

A number of risk factors have been identified by retrospective studies for third-degree tears [22]. These include:

- Primiparity (up to 4 %): the first vaginal delivery carries the greatest risk of new-onset fecal incontinence according to population-based studies.
- Induction of labor (up to 2 %).
- Epidural analgesia (up to 2 %).
- Birth weight over 4 kg (up to 2 %).
- Persistent occipitoposterior position (up to 3 %).
- Second stage longer than 1 h (up to 4 %).
- Forceps delivery (up to 7 %).

Evidence for other suggested risk factors, such as shoulder dystocia and episiotomy is contradictory. Though the rationale for the practice of episiotomy was that it protects the perineum from uncontrolled injury during delivery, data is conflicting. Evidence does report a significantly lower risk of sphincter injury with mediolateral episiotomy when compared with a midline episiotomy (2 % versus 12 %) [23, 24]. However, these

studies may not have accounted for various confounding variables including differences in clinical practice and the experience of the obstetrician. Current evidence does not support the routine use of episiotomy as a method to prevent anal sphincter injury. However, in a recent large retrospective analysis of 17,094 women who underwent operative vaginal delivery between 2000 and 2010, it was found that the use of mediolateral episiotomy when performing operative vaginal delivery is associated with a large reduction of the risk of anal sphincter injury [25].

There have been various attempts at developing an antenatal risk scoring system for sphincter injury [26]. But they have been unsuccessful as many of the risk factors identified are components of normal vaginal delivery and a majority of women with these risk factors deliver without sphincter injury.

A significant fact in recent years is that though the incidence world over has shown an upward trend, it has essentially remained stable at 0.3–0.6 % over the past 15 years in Finland [27]. This has been attributed to the use of traditional methods of perineal support during delivery of the fetal head and shoulders. When Laine et al. introduced this method of delivery, combined with restricted use of episiotomy, the rate of OASIS fell significantly from 4 to 1 % in four Nordic hospitals who adopted this method [28, 29].

Recommended Guidelines

The RCOG national guidelines [30] and the Cochrane systematic review [31] provide recommendations on every aspect of sphincter repair. The salient points are highlighted in this chapter.

All women with perineal trauma after childbirth need to have an assessment of anal sphincter integrity immediately following delivery by visual inspection, digital vaginal and rectal palpation. If sphincter injury is identified, appropriate counseling is an ideal precursor to repair. Patient information booklets which discuss the repair and the outcomes in a reader-friendly manner with clear diagrams are very helpful to prepare the patient.

Technique of Repair

Repair of sphincter injury needs to be performed in an operation theater, in lithotomy position, under aseptic conditions, with adequate light and an assistant. Regional or general anesthesia is preferable as it enables the sphincter muscle to relax, thus making it easier to retrieve the retracted torn ends and bring them together without tension [32–34].

The internal anal sphincter lies between the anal epithelium and the external sphincter, and its muscle fibers are paler than those of the external sphincter and run in a circular fashion. The original description of sphincter injury repair included separate repair of the internal anal sphincter [32]. However identification of the internal sphincter may often be difficult. In fact it was not identified separately in the RCTs conducted on the two methods of repair of EAS [33–36]. However, though the evidence for the same is not yet concrete, it is advisable that if identified, any tears in the IAS should be repaired separately from the external sphincter using an end-to-end technique.

External Anal Sphincter (EAS) Repair

There has been considerable debate on the right method of repairing the EAS: the traditional end-to-end approximation method or the overlap technique (Fig. 17.1). A retrospective study by Sultan et al. in 1999 suggested an improved outcome with the overlap technique [32]. Since then, four RCTs have compared the two techniques [33–36]. The number of recruited women in these studies varied between 41 and 112 with a follow-up duration of 3–12 months. The outcomes assessed included, anal continence scores and quality of life, together with a combination of ultrasound and anal manometry assessment. All studies except one showed no significant difference between the two groups, in terms of fecal incontinence rates. It is important to note that in the study by Fernando et al. [36] in which better outcomes were obtained with overlap technique, only patients with 3B and 3C injuries

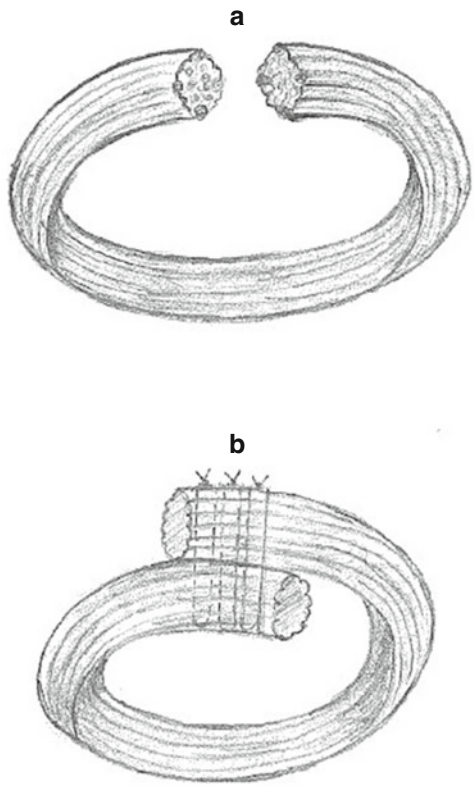


Fig. 17.1 End-to-end anastomosis of external anal sphincter (**a**) and overlap technique (**b**)

were included as opposed to the other studies in which all EAS injuries (3A, 3B, and 3C) were included. Also in the Fernando et al. study, in patients with 3B tears, the remaining EAS fibers were divided to perform an overlap technique unlike in the other studies where the overlap was performed without division of the EAS fibers. Significantly, the repair was performed by only three trained clinicians in the Fernando et al. study, in contrast to other studies in which the repair was performed by a larger pool of trained clinicians. Thus, it needs to be considered that the improved outcome of overlap repair shown by Fernando et al. may not be applicable to other obstetric units throughout the world.

Choice of Suture Material

Only one RCT has assessed suture materials in which no difference was found between 2-0 coated polyglactin (Vicryl) and 3-0 polydioxanone (PDS), when anal incontinence, perineal pain, and suture migration were considered at 12 months follow-up [34]. PDS and Vicryl are both recommended for sphincter repair, as both are absorbable with absorption in 180 and 70 days, respectively.

Monofilament suture materials such as polydioxanone (PDS) or polyglyconate (Maxon) are commonly used for repair of the external anal sphincter muscle because of their longer half-life, increased strength retention and decreased tissue reactivity. They are also less likely to get infected as compared with braided sutures such as polyglactin (Vicryl). However, these long-acting materials are associated with suture migration which can often lead to discomfort when the ends of the suture impinge on the perineal skin [10]. Use of a fine suture such as 3-0 PDS may cause less irritation and discomfort. Burying of the surgical knots beneath the superficial muscle is recommended to prevent knot migration to skin. Internal anal sphincter damage should be repaired with a fine suture such as 2-0 Vicryl. In the presence of a fourth-degree tear, the torn anal epithelium is repaired with interrupted 3-0 Vicryl sutures with the knots tied in the anal lumen. A subcuticular repair of the anal epithelium via the transvaginal approach can also be performed [37].

Who Should Undertake the Repair?

The sequelae associated with third- and fourth-degree perineal tears are of sufficient significance, that it is recommended that sphincter repair needs to be performed by appropriately trained obstetricians under optimal conditions [10]. While repairs performed by inexperienced trainee obstetricians may not be adequate, delay in the repair so that it can be undertaken by colorectal surgeons in a secondary sitting is also not advisable [5]. An audit of obstetric trainees showed that only 13 % of

them were satisfied with their level of experience prior to performing their first unsupervised repair [21]. Hence it is recommended that hands-on workshops are conducted that bridges the gaps in the training of obstetricians. Such workshops have been shown to increase both the awareness of perineal anatomy and recognition of anal sphincter injury [38].

Postoperative Care

Antibiotics

There are no published RCTs regarding the use of perioperative and postoperative antibiotics for third- and fourth-degree tears. The Cochrane review found insufficient data to support administration of prophylactic antibiotics routinely [31]. However third-degree and particularly fourth-degree tears can become contaminated with bacteria from the rectum, significantly increasing the chance of perineal wound infection leading to a higher risk of wound dehiscence, fistula formation and anal incontinence. Given the seriousness of these potential sequelae, it is prudent to prescribe both aerobic and anaerobic antibiotic cover following primary repair. Typically most studies have used intraoperative intravenous antibiotics (cefuroxime 1.5 g and metronidazole 500 mg), followed by a 7-day course of oral broad-spectrum antibiotics (cephalexin and metronidazole).

Pain Relief

Rectal diclofenac is the primary choice of pain relief for obstetric anal sphincter and other perineal injuries [39]. It significantly reduces pain on sitting, walking and defecation, within the first 48 h after delivery.

Use of Laxatives

Traditionally colorectal surgeons, who undertake secondary sphincter repair on patients with fecal incontinence, have recommended the use of constipating agents as it is advisable to avoid the contamination of the wound site by liquid fecal matter. However, primary repair is not associated with preexisting fecal incontinence at the time of repair. Delayed and painful defecation due to hard stools, can lead to considerable discomfort and stress for the patient. It is advisable to use postoperative laxatives and stool softeners following primary repair to ensure significantly earlier and less painful first bowel motion. This helps in early discharge from hospital and reduces the incidence of postoperative wound dehiscence. In the published RCTs, stool softeners (lactulose 10 ml three times a day) along with a bulking agent (ispaghula husk, Fybogel, one sachet twice a day) were used for 10 days following repair [10]. The dose and type of laxatives used should be tailored to the individual and should be customized based on the patient's diet, gut transit time and stool consistency.

Postoperative Physiotherapy

There is very limited data regarding the importance of pelvic floor exercises in the postpartum management of women with OASIS. RCOG guidelines are not very clear and the hospitals world-wide differ in the policy they follow. One study has reported lower anal incontinence rates at 1 year, in women who were taught pelvic floor muscle exercises by a physiotherapist, following third-degree tear. The study however lacked a control group [40].

Outcomes

Endoanal ultrasound and neurophysiological tests, along with patient symptoms of anal incontinence have been used to assess the outcome of primary anal sphincter repair. Poor outcomes have been shown to be related to persistent sphincter defect detected on endoanal ultrasound [41]. The four RCTs described earlier [33–36] comparing end-to-end approximation with overlap repair have shown that 60–80 % of women

will be asymptomatic at 12 months after primary repair of obstetric anal sphincter injury. Based on these results, RCOG guidelines recommend that patients who have had either of the two repairs can be counseled that the outcome of primary repair is likely to be good. However, 19–36 % of women in these studies have been shown to have persistent defects, most of which affect the EAS. The most common symptom experienced is incontinence to flatus.

Long-Term Follow-Up and Future Delivery

Women should be followed up at 6 weeks postpartum by a trained obstetrician. It is valuable to set up a dedicated multidisciplinary perineal clinic to ensure that patients receive dedicated care postoperatively. In addition to discussing the delivery details and the injury, the patient should be specifically questioned about bladder, bowel, and sexual function, symptoms of fecal incontinence, fecal urgency, dyspareunia, and perineal pain. Since these questions are embarrassing to ask, structured questionnaires of bladder, bowel, and sexual function are useful [41, 42]. A careful vaginal and rectal examination should be performed to check for complete healing, scar tenderness and sphincter tone. Ideally, anorectal investigation (i.e., endoanal ultrasound, anorectal manometry, and EMG) should be part of follow-up of every patient who has undergone anal sphincter repair. Hence if these facilities are not available at the primary hospital, at least the symptomatic women and those who have clinically reduced anal tone or contraction should be referred to an appropriate center where these investigations can be conducted.

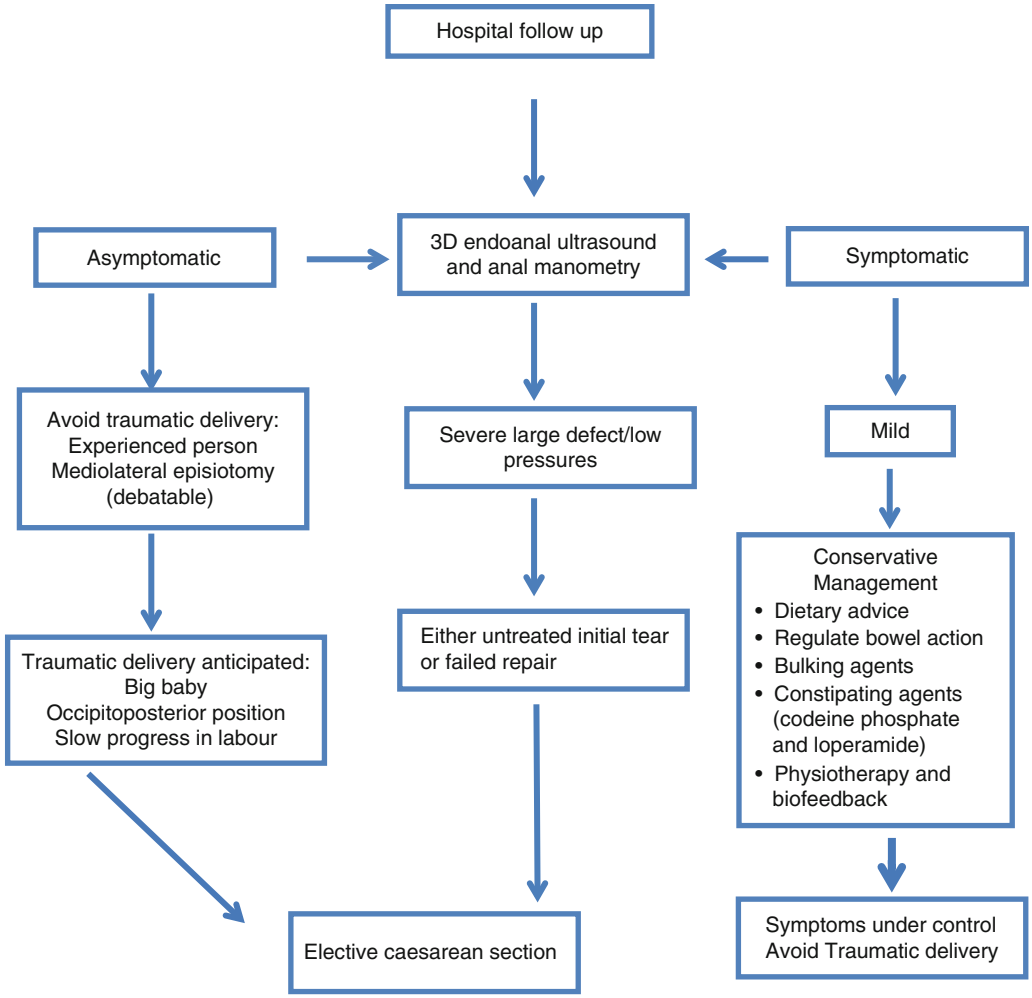
A plan for the management of subsequent pregnancies and the mode of delivery should be part of the follow-up for women who have sustained an anal sphincter injury. Table 17.2 gives an overall protocol for the management of future pregnancies [37]. Harkin et al. found that although anal sphincter injury was increased fivefold at next delivery compared with all multiparae, 95 % of women delivering vaginally after a previous third-degree tear did not sustain further overt sphincter damage [43]. In contrast, two studies concluded that prior anal sphincter laceration is associated with increased risk of laceration in second delivery, particularly in women who deliver infants of high birth weight [44, 45]. Studies assessing vaginal delivery following a third-degree tear have shown worsening fecal incontinence symptoms in 17–24 % of women [46–49] especially in women who had transient incontinence after the index delivery. In a recent cohort study investigating women delivering vaginally after previous third- and fourth-degree tears, the incidence of repeat sphincter injury was 7.2 %. In comparison, in those with no previous sphincter injury, the incidence was only 1.3 % [50]. The risk of a severe perineal tear is increased fivefold in women who had a third- or fourth-degree tear in their first delivery. However future studies including RCTs are needed before conclusive recommendations can be made regarding subsequent mode of delivery following anal sphincter injury.

In general, women who have had a previous third-degree perineal tear and have persistent symptoms of fecal incontinence or significantly abnormal endoanal ultrasound or manometry results are best delivered by prelabor elective cesarean section. Women should also be counseled about the risks of developing anal incontinence or worsening of symptoms with subsequent vaginal delivery. If the women are asymptomatic, there is no clear evidence regarding the best mode of delivery.

Levator Ani Trauma Linked to Childbirth

An important area of research in the last decade linked to obstetric injuries pertains to the trauma to levator ani muscle subdivisions during childbirth. Vaginal delivery increases the odds that a woman will develop pelvic organ prolapse by 4–11 times and stress urinary incontinence by 2.7 times when compared with controls [51, 52]. One partial explanation for the epidemiological link between vaginal birth and pelvic organ

Table 17.2 Flow diagram illustrating management after a third-/fourth-degree tear



prolapse is levator ani trauma. Levator trauma resulting from childbirth may lead to widening of the levator hiatus, particularly in its anterior part. Levator hiatus widening is associated with anterior and central compartment prolapse and may also be a risk factor for recurrence after pelvic reconstructive surgery [53–56]. Several MRI, transperineal and endovaginal ultrasound-based imaging studies have documented damage to the levator ani muscle following vaginal delivery. The injury frequently involving the pubovisceral portion, followed by the iliococcygeus component [54, 57, 58]. Ultrasound imaging of the levator ani subdivision defects and gradation of the damage suffered by the levator ani muscles is currently being researched extensively.

Conclusion

Obstetric anal sphincter injuries are an important adverse outcome of vaginal delivery and the primary cause of fecal incontinence in women, a symptom that can have debilitating effect on a woman's quality of life. It is imperative that they are recognized at the time of delivery and proper repair technique is followed to prevent future adverse outcomes. The obstetrician needs to be adequately trained to handle these injuries primarily at the

time of delivery. Subsequent delivery needs to be conducted by an experienced clinician following a proper follow-up in a perineal clinic where endoanal ultrasound and anorectal manometry is part of the workup.

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Fecal Incontinence

18

Karen L. Noblett and Jessica Hammett

Introduction

Fecal incontinence is characterized by the involuntary loss of liquid and/or solid stool, while anal incontinence also includes the involuntary loss of flatus [1]. The prevalence of fecal incontinence is estimated to impact 2–21 % of people depending on the population studied and the criteria used to define incontinence [2–5]. Fecal incontinence is a debilitating disorder that significantly impacts quality of life and is believed to be a frequent cause of admission to nursing homes, with up to 50 % of nursing home residents suffering from fecal incontinence [6, 7].

Fecal incontinence is often underreported by patients, with surveys suggesting that 70–90 % of patients with fecal incontinence do not discuss it with their health-care providers primarily due to embarrassment and concerns regarding treatment options [8]. Patients who suffer from fecal incontinence routinely score significantly lower than controls in the domains of lifestyle, coping and behavior, depression and self-perception and embarrassment on quality of life questionnaires [9, 10].

Given its significant impact on quality of life, as well as the high prevalence of the condition, it is imperative for health-care providers to query their patients regarding symptoms of fecal incontinence. This is especially important since many treatment modalities exist, ranging from preventative measures and symptom management to surgical treatment options, all of which can have a positive impact on quality of life.

Pathophysiology

The pathophysiology of fecal incontinence is complex. The muscular components of the continence mechanism include the internal and external anal sphincters as well as the puborectalis muscle. The smooth muscle of the internal anal sphincter responsible for 70–80 % of the resting tone is innervated by the autonomic nervous system and is not under voluntary control. In conjunction with the internal anal sphincter, the striated muscle of the external anal sphincter also contributes to the resting tone, however to a much lesser degree. The external anal sphincter, innervated by the somatic nervous system via the pudendal nerve, is under voluntary control and provides additional squeeze pressure when defecation is to be deferred. The puborectalis is a U-shaped muscle that pulls the rectum forward,

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A. Tamilselvi, A. Rane (eds.), *Principles and Practice of Urogynaecology*,
DOI 10.1007/978-81-322-1692-6_18, © Springer India 2015

creating the anorectal angle, which becomes more acute during contraction of the pelvic floor and should relax and widen during defecation.

When the contents of the rectum come into contact with the anoderm, the sensitive squamous epithelial lining of the rectum, the contents are "sampled" to determine its consistency (solid, liquid, gas). Under normal circumstances, the urge to defecate can be delayed by contractions of the puborectalis and external anal sphincter. When socially appropriate, the puborectalis and external anal sphincter are voluntarily relaxed, the anorectal angle straightens, and the diaphragm, abdominal, and rectal muscles contract in order to expel the stool [11–13]. The process of defecation is complex, and continence is dependent on multiple variables including stool consistency, intestinal motility, rectal capacity and compliance, anorectal sensation, sphincteric mechanism pelvic floor musculature and innervation.

Etiology

Risk factors for developing fecal incontinence include multiple childbirths, obstetrical injury, poor general health, pelvic surgical procedures and aging. A more comprehensive list of risk factors is listed in Table 18.1 [2, 3, 12, 14].

The etiology of fecal incontinence is multifactorial and can be broadly divided into traumatic events, neurogenic causes congenital malformations, intestinal causes and others. A more comprehensive list of potential causes of fecal incontinence is listed in Table 18.2 [14–16]. Traumatic damage to the nerves, muscles, and/or blood supply of the rectum and continence mechanism from childbirth, forceps injury, accidental trauma, surgery or radiation may lead to fecal incontinence. Obstetrical injury is the most common cause of fecal incontinence in women and may be the result of injury to nerves and/or muscle that may occur during vaginal delivery, from an episiotomy, a third- or fourth-degree laceration, or an occult injury to the sphincteric complex. This may directly lead to fecal incontinence after delivery or may be a contributing factor to fecal incontinence later in life. Studies have shown that sphincteric defects and/or incompetence may be persistent after a perineal repair despite what was thought to be an adequate repair. Additionally, prolonged compression of the pudendal nerve during delivery may result in permanent nerve injury, which can contribute to both fecal and urinary incontinence [15, 16]. Pelvic radiation, which may compromise rectal compliance through tissue damage and fibrosis, is another form of trauma, leading to fecal incontinence. Similar to bladder physiology, rectal compliance refers to the ability of the rectum to distend while intraluminal pressures remain low. A decrease in rectal compliance can lead to urgency and fecal incontinence [17]. Similarly, a loss of rectal capacity, measured by the amount of material that can be

Table 18.1 Risk factors for fecal incontinence

Advancing age	Diabetes
Obesity	Neurological disorders
Female gender	Urinary incontinence
Caucasian race	Chronic diarrhea
Multiple vaginal births	Constipation
Sphincter tear with delivery	Pelvic organ prolapse
Surgical procedures (colectomy, sphincterotomy)	Gastrointestinal disorders (Crohn's disease, ulcerative colitis, irritable bowel disease)

Table 18.2 Potential causes of fecal incontinence

Traumatic:	*Intestinal:*
Obstetric	Colitis
Surgical	Colorectal tumors
Radiation	Rectal prolapse
Neurogenic:	Decreased compliance or capacity
Multiple sclerosis	Fecal impaction
CVA	Diarrhea
Diabetic neuropathy	*Others:*
Dementia	Aging
Neoplasm	Laxative abuse
Congenital:	Idiopathic
Spinal cord malformation	
Anorectal malformation	

successfully and comfortably stored in the rectum, can lead to fecal incontinence by allowing the rectum to be overwhelmed by severe diarrhea or fecal impaction. The etiology of a patient's fecal incontinence should be determined in order to discern if the incontinence is reversible or treatable.

Evaluation

History and Physical

The evaluation of patients with fecal incontinence should begin with a thorough history and physical examination. Specifically, patients should be queried regarding the duration, frequency and severity of symptoms; type of incontinence (gas, liquid, and/or stool); daily bowel habits, including constipation and diarrhea; obstetrical history; new medications or diet changes and past medical and surgical history. Additionally, the patient should be instructed to fill out a bowel diary so that the patient's dietary habits, number of incontinence episodes per day and quality of the incontinence (measured via the Bristol scale) can be objectively evaluated by the clinician. Incontinence questionnaires such as the Fecal Incontinence Severity Index, Fecal Incontinence Quality of Life Scale, and the Cleveland Clinic Incontinence Score can be employed to assess severity of fecal incontinence and quality of life [10]. These questionnaires can also be useful in determining the degree of improvement in symptoms after treatment.

During the physical exam, a thorough evaluation of the perineum, anus, and vagina should be performed. A digital rectal exam should include evaluation of the rectal tone and the ability of the patient to contract the external anal sphincter as well as the presence of hard stool, internal hemorrhoids, blood and masses in the rectum. External evaluation of the perineum and perianal skin should assess for fecal soiling, scarring, erythema, external hemorrhoids, rectal mucosal prolapse, anal fistulas, and the classic "dovetail" sign suggesting a defect in the external anal sphincter (Fig. 18.1). Additionally, the clitoral-anal and bulbocavernosus reflexes may be tested in order to grossly assess the pudendal nerve. Vaginal examination can aid in determining the presence of an enterocele, rectocele, or recto-vaginal fistula.

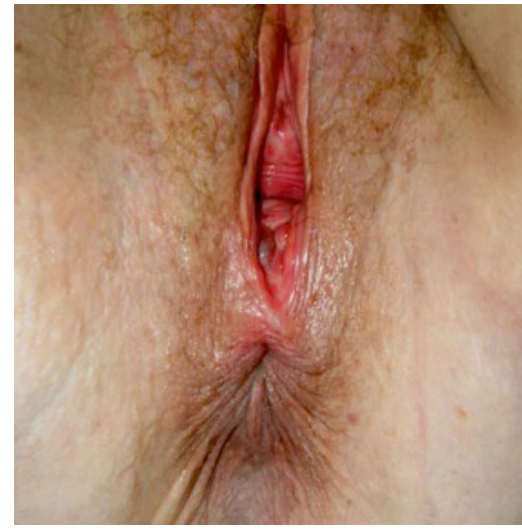

Fig. 18.1 Dovetail sign

Diagnostic Studies

Diagnostic studies may be performed to determine the etiology or possible treatment options for patients with fecal incontinence. Unfortunately, testing is not standardized and may produce normal results despite the presence of fecal incontinence. Therefore, there is some debate over the necessity and use of these tests. However, they may be useful if additional information is required, to determine the most effective treatment options so that the best possible outcomes can be obtained.

Anorectal physiology testing includes anal manometry and neurophysiologic studies such as the pudendal nerve terminal motor latency testing and needle electromyography (EMG) of the external anal sphincter. Anal manometry involves the use of transducer catheters and balloons to measure the resting and squeeze pressures of the external and internal anal

sphincters and the rectum. Additionally, during testing an inflated balloon may be employed to determine rectal capacity, sensation and the presence of the rectoanal inhibitory reflex (RAIR). Pudendal nerve latency testing is performed by stimulating the pudendal nerve and measuring the amount of time required for the stimulus to induce a contraction of the external anal sphincter. A normal delay is approximately 2.0 ms, and a prolongation may suggest a neuropraxic injury. This test is operator dependent, does not correlate well with patient symptoms and is best used when interpreted in conjunction with needle EMG of the external anal sphincter. Needle EMG of the external anal sphincter may be employed to determine the innervation status of the external anal sphincter but can be particularly uncomfortable for the patient. Surface EMG electrodes may be substituted but are not as precise and give more of a global picture of muscle function. A colonoscopy is a generally recommended procedure for colon cancer screening after the age of 50 years, but it may also be employed to investigate fecal incontinence or changes in bowel symptoms as it may reveal the presence of diverticulitis, rectal masses, or a mucosal abnormality.

Imaging studies such as defecography, endoanal ultrasound and MRI may also provide additional diagnostic information. Defecography is a fluoroscopic procedure that involves inserting barium paste into the rectum and obtaining images as the patient contracts the pelvic floor, strains with attempt at maintaining continence, and attempts to defecate. This test may aid in the diagnosis of obstructed defecation, rectoceles, enteroceles and rectal prolapse. Endoanal ultrasound can assess the integrity of the external and internal anal sphincter and may be useful in operative planning if an anatomic defect is discovered. A 360° endoanal transducer is available that can accurately assess the integrity, thickness and length of the internal and external anal sphincters (Fig. 18.2). Additionally, MRI may assess atrophy of the external anal sphincter and levator ani muscles including the puborectalis [18–21].

Treatment

Nonsurgical

Conservative therapy such as dietary modification, pharmacotherapy and pelvic floor therapy are the first line in treatment of fecal incontinence. Patients should be counseled regarding the importance of dietary modification as they can have a significant impact on bowel function, stool consistency and bowel motility. Right amount of fiber in the diet helps with diarrhea and constipation. Fruits, vegetables, whole grains, breads and beans are good source of fibers. Increasing dietary fiber with bulking agents, such as calcium polycarbophil or psyllium, has been noted to have beneficial effects on colonic function as well as cholesterol levels and glucose metabolism. Ironically, increasing dietary fiber can help treat both diarrhea and constipation. In the setting of diarrhea, fiber improves stool consistency, decreases stool frequency and reduces fecal water content, while in the context of constipation, fiber increases stool frequency and fecal weight, without leading to diarrhea.

Another important dietary consideration to discuss with patients is the high rate of lactase deficiency in the general population. Lactose, which is digested by lactase found in the bowel, is a common sugar found in milk and dairy products. Adhering to a dairy-free diet for 2 weeks may have a significant impact on bowel symptoms. Furthermore, the effect of caffeine, a stimulant that may increase urgency in susceptible patients, should be reviewed with the patient. Finally, ingestion of probiotics does appear to have a beneficial effect in patients with irritable bowel disease but there are no reported studies on the effectiveness of probiotics in fecal incontinence.

Due to the variety of underlying causes and contributing factors, there is no one global pharmacological treatment for fecal incontinence. Therefore, treatment with medications should be directed towards controlling symptoms of constipation or diarrhea. Loperamide, diphenoxylate hydrochloride, codeine phosphate, anticholinergics and atropine sulfate are all medications that may be tried to help control symptoms of diarrhea.

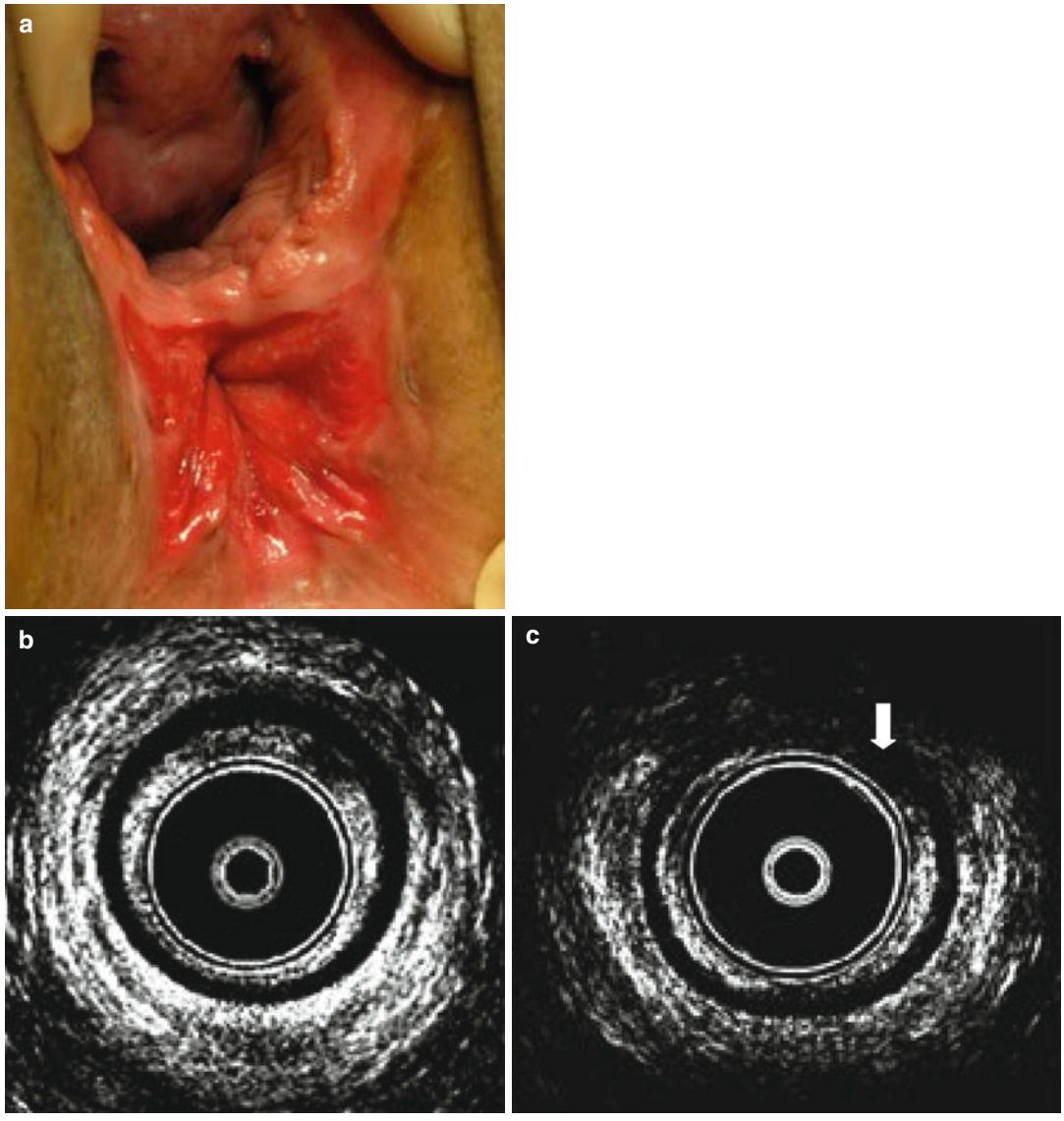

Fig. 18.2 (**a**) Internal and external anal sphincter defects. (**b**) 360° endoanal US with internal and external sphincter intact. (**c**) 360° endoanal US with internal and external sphincter defects

Loperamide is a synthetic agent that binds opioid receptors in the bowel, inhibits small and large bowel peristalsis and has the added benefit of increasing resting anal sphincter tone [22, 23]. Similarly diphenoxylate hydrochloride also binds to opioid receptors in the bowel, but due to this medication's ability to cross the blood-brain barrier, it is a controlled substance in the United States. However, diphenoxylate hydrochloride has minimal potential for physical dependence. For medical treatment of constipation, laxatives and enemas may be employed along with dietary modifications to help the patient from developing impacted stool in the rectum, which may lead to fecal incontinence from loose seepage around the impacted stool. If constipation persists, manual disimpaction is an option to help remove impacted stool from the rectum.

Pelvic floor physical therapy aimed at improving the strength of the levator ani muscle group and the external anal sphincter is another treatment option. Biofeedback, in the form of manual pressure or EMG feedback, rectal distension balloons to train threshold sensation and coordination training are a few tools that pelvic floor physical therapists employ in pelvic floor muscle training. A Cochrane review of recent published trials concluded that currently there is insufficient evidence to definitively define the role of physical therapy and biofeedback in the treatment of fecal incontinence; however, larger studies are needed for future assessment [24].

Surgical

If conservative treatment is not successful, there are several surgical options available, based on the etiology [25].

Sphincteroplasty

When there is a defect in the external or internal anal sphincter, a sphincteroplasty may be performed in order to re-approximate the two ends of the sphincter. The repair can be done end to end, which is the most common postpartum method or overlapping, the most common delayed method. There is limited data available supporting one method compared to the other, but a Cochrane review in 2006 reviewed three randomized controlled trials and found that an overlapping sphincteroplasty performed promptly after an obstetrical trauma may lower the risk of fecal urgency and improve anal incontinence symptoms [26]. However, the rates of anal incontinence and dyspareunia were similar at 12 months post-surgery. Another randomized control trial looked at delayed repair with overlapping versus end-to-end methods, and no difference was seen in the failure rates (17–25 %) or in the complication rates [27]. A list of short- and long-term studies reviewing surgical outcomes after sphincteroplasty may be seen in Tables 18.3 and 18.4.

Table 18.3 Results for sphincteroplasty with mean follow-up of < 5 year duration

Author, year	*N*	F/U mean (range) months	Success (%)	Improved (%)
Fang et al. (1984)	76	35 (2–62)	82	89
Browning and Motson (1984)	83	39.2 (4–116)	78	91
Ctercteko et al. (1988)	44	50	75	–
Laurberg et al. (1988)	19	18 (9–36)	47	79
Yoshioka and Keighley (1989)	27	48 (16–108)	–	74.1
Wexner et al. (1991)	16	10 (3–16)	76	87.5
Fleshman et al. (1991)	55	(12–24)	72	87
Engel et al. (1994)	55	15 (6–36)	60.4	–
Engel et al. (1994)	28	46 (15–116)	75	–
Londono-Schimmer et al. (1994)	94	58.5 (12–98)	50	75
Sitzler and Thompson (1996)	31	(1–36)	74	–
Oliveira et al. (1996)	55	29 (3–61)	70.1	80
Nikiteas et al. (1996)	42	38 (12–66)	60	–
Gilliland et al. (1998)	100	24 (2–96)	55	69
Karoui et al. (2000)	86	40 (3–40)	28	46

Muscle Transposition

Treatment of fecal incontinence with muscle transposition involves replacing or augmenting the external anal sphincter with a skeletal muscle, most often the gracilis muscle and then stimulating the muscle with an electrical pulse generator. The pulse generator transforms the muscle fibers from fast twitch to a slow twitch, making them more fatigue resistant. The pulse generator is not freely available in all countries. Success rates are good at about 60 %, but there are concerns regarding the stimulated gracilo-

Table 18.4 Results for sphincteroplasty with follow-up of >5 years

Author, year	Pt. follow-up/ total (%)	Mean F/U (range) months	Outcomes
Malouf et al. (2000)	46/55 (84)	77 (60–96)	0 % continent 10 % flatus incontinence 79 % soiling 21 % fecal incontinence – solid 8/46 other surgery
Halverson et al. (2002)	49/71 (69)	69 (48–141)	14 % continent 54 % fecal incontinence 7/49 other surgery
Gutierrez et al. (2004)	135/191 (71)	120 (84–192)	6 % continent 16 % flatus incontinence 19 % soiling 57 % fecal incontinence 5/135 other surgery

plasty due to high complication and reoperation rates [28].

Artificial Anal Sphincter

The placement of an artificial anal sphincter was first described in 1987, and initial continence rates of functioning devices were promising (78–100 % solid stool, 56–95 % solid and liquid stool). Unfortunately, due to very high complication rates from infection and ulceration and innervation rates of greater than 80 %, its use is extremely limited [25].

Radiofrequency

Another option for treatment of fecal incontinence involves the administration of temperature-controlled radiofrequency energy to the anal sphincter, also known as the SECCA procedure. An anoscope is placed in the rectum, and needles protrude from the device into the muscle to deliver the radiofrequency energy. Theoretically, this procedure improves sphincter function and restores anorectal sensitivity, although the exact mechanism of action is unknown. Several studies have been performed to evaluate the success rate of radiofrequency administration, but the results have been inconsistent and long-term efficacy rates have not been determined [29, 30].

Sacral Neuromodulation

Sacral neuromodulation is a proven and reliable option for patients who have failed conservative management. The motor nerve supply to the external anal sphincter and pelvic floor as well as the parasympathetic innervations originates from the branches of S2–S4 and modulation of the S3 nerve root has been shown to improve fecal incontinence. Several studies have demonstrated initial improvement rates of 90 % and consistent improvement at 2 years (83 %) and 5 years (69 %) in the intent to treat arm and 89 % in those available for follow-up [31, 32].Additionally, significant improvement was seen in all four domains in the fecal incontinence quality of life index. In a randomized controlled trial evaluating sacral neuromodulation in comparison to conservative treatment (dietary modification, pelvic floor exercises and bulking medications), sacral neuromodulation significantly decreased incontinent episodes from 9.5 to 3.1 a week, achieved continence in 47 % and significantly improved all four domains in the fecal incontinence quality of

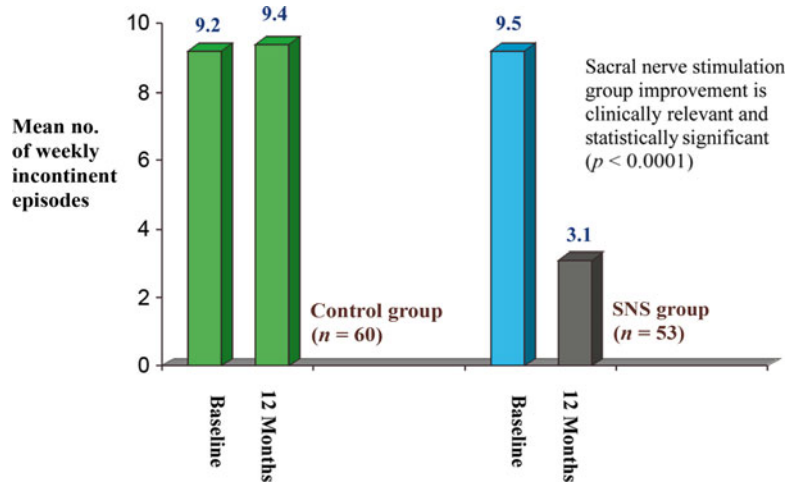

Fig. 18.3 Study by Tjandra et al. [33] investigating sacral nerve stimulation versus medical management for fecal incontinence

life index [33] (Fig. 18.3). Patients with sphincter defects up to 120° were included in this study and the size of the sphincteric defect did not impact the outcome of the procedure.

Sacral neuromodulation consists of a diagnostic phase and a subsequent second stage if the trial is successful. The first stage can be performed in the clinician's office, with the percutaneous placement of a temporary lead, or performed in the operating room, with the percutaneous placement of a quadripolar lead. With the patient in the prone position, the lead is placed into the S3 foramen under fluoroscopic guidance. Stimulation of the sacral nerve is delivered via a portable stimulator, and motor and sensory responses are elicited from the patient. Motor responses include a pulling in of the rectum or "bellowing" and plantar flexion of the ipsilateral toe. Sensory responses are evaluated by questioning the patient on where the stimulation is felt. A bowel diary should be completed prior to and during the trial, and if the patient has a greater than 50 % improvement in symptoms, the internal pulse generator may be implanted (second stage).

Bulking Agents

For patients with mild to moderate fecal incontinence, bulking agents may be injected into the anal canal to create additional bulk and resistance. There are multiple bulking agents available on the market, including silicone implants (Bioplastique), pyrolytic carbon-coated zirconium oxide beads (Durasphere), hyaluronic acid/dextranomer gel (Solesta), and dextranomer in stabilized hyaluronic acid (NASHA Dx). A review of several studies comparing different bulking agents with each other or with isotonic saline injections revealed that there was an improvement in fecal incontinence in at least 50 % of patients. However, many patients required subsequent injections due to deterioration of continence over time and long-term data on continence rates is not yet available [34]. Injection of the bulking materials into the anal canal may be performed in the clinician's office or in an operating room under local, regional or general anesthesia, as dictated by surgeon preference. The patients may be positioned in prone jackknife or in lithotomy. A lubricated bivalve anal retractor may be employed for visualization and the bulking agent is injected submucosally at three to four sites just proximal to the dentate line.

Other

Finally, when other treatment options have failed, intestinal diversion with a colostomy may significantly improve a patient's quality of life by allowing the patient to resume normal activities [35, 36].At the time of this publication,

therapies including autologous muscle cell transfer, perineal puborectalis sling, magnetic anal sphincter, and the use of biologic mesh in sphincteroplasties are currently being investigated and may offer additional future treatment options [25].

Conclusion

Fecal incontinence is a common and debilitating disorder that can lead to social isolation, depression, anxiety and embarrassment. Clinicians need to be aware of the impact fecal incontinence can have on their patients and screen for it regularly. A comprehensive exam and additional testing may be required to determine the etiology of fecal incontinence prior to initiating treatment. Conservative treatment with dietary modification, medical therapy and physical therapy should be explored before progressing to surgical therapies. The management of fecal incontinence is often very challenging and may involve multiple treatment modalities, but the improvement in quality of life can be significant.

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