Anna Padoa
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The Overactive Pelvic Floor

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Preface

This textbook is an expression of the individual professional journeys of its coeditors. As a urogynecological surgeon and a former pelvic floor physiotherapist (currently an individual, couples, and sex therapist), each of us possesses expertise in the biomechanical treatment of pelvic floor disorders. However, our involvement in the treatment of patients with pelvic floor dysfunction inspired each of us in our own direction to expand our professional interests and skills beyond the biomedical approaches common to our professions.

The pelvic floor is complex and multidimensional, and so too, are our patients. Addressing the distress of patients who presented to us with pelvic floor disorders, confronted us with the need to recognize the complexities of the psychosocial, relational and sexual, as well as the physiological components related to pelvic floor complaints. This has included recognizing that treating the pelvic floor means meeting people in their most intimate and vulnerable space. The word "pudendal" is, in fact, Latin for "shame."

The pelvic floor is conceptualized as an anatomical area of the body, responsible mainly for support of the pelvic organs and maintenance of bowel and bladder control. As such, pelvic floor-related texts are classically devoted to reviewing the dysfunctions and treatments of conditions related to compromised integrity of the pelvic floor muscles and fascial support. These conditions typically include pelvic organ prolapse and urinary and/or anal incontinence.

Treatment of conditions related to pelvic floor weakness has been widely researched and protocols have been standardized. Less recognized and not as well understood are conditions related to the overactive pelvic floor. Unlike pelvic floor weakness, which is conceptualized as a condition resulting mainly from mechanical stresses such as pregnancy, delivery, and hormonally induced tissue laxity, the presentation of pelvic floor overactivity, is far more complex.

In conceptualizing the OPF, practitioners must move beyond the view of the pelvic floor as a mere anatomical location. OPF is connected to a complex interaction of psychological, mechanical, functional, and multi-systemic influences. Thus, practitioners who confront patients with symptoms and presentations consistent with an OPF are often faced with practical treatment challenges.

Caregivers who treat the pelvic floor are often trained to address dysfunction in a mechanical manner. Imaging techniques are used to locate the anatomical "fault lines" and urodynamics and manometry measure pressure units.

Physical therapists address muscle weakness with muscle strengthening, and surgeons use their skills much as a handyman would, to lift, tighten, and repair.

In confronting patients with OPF, these tools and skills may be insufficient. While treatment of patients with pelvic floor weakness does require an awareness and appreciation for how symptoms such as prolapse and incontinence affect quality of life and sexual functioning, these variables are addressed indirectly, by providing evidence-based and standardized protocols such as pelvic floor muscle rehabilitation and/or surgical repair. The positive outcome of these mechanical interventions aims to improve quality of life and sexual function.

The classical approach described above may not, however, be satisfactory in addressing the complexities involved in treating patients with pelvic floor overactivity. Treatment requires a comprehensive evaluation to determine the cause and effect dynamic between the pelvic floor, the symptoms related to the OPF, and the psychological contributors as well as outcomes of these multifaceted conditions.

We have, therefore, collaborated with leading researchers and practitioners to present the most up-to-date text that specifically addresses pelvic floor overactivity. Pelvic floor overactivity may be associated with musculoskeletal and neurological impairments, as well as psychological distress and sexual abuse, and is correlated with symptoms that greatly affect quality of life and sexual function. As such, this condition is relevant to several disciplines including urology, gynecology, gastroenterology, sexology, psychology and physical therapy.

This textbook is the first of its kind dedicated to OPF. It provides a comprehensive, state-of-the-art review of the OPF and is intended to serve as a valuable resource for clinicians and researchers with an interest in the pelvic floor. In addition, this text offers clinical tools for medical and mental health practitioners alike for recognition, assessment, treatment, and interdisciplinary referral of patients with OPF and OPF-related conditions.

This book reviews the definition, etiology, and pathophysiology of non-relaxing pelvic floor muscle tone and discusses sexual function and past sexual experience in relation to the pelvic floor. Specific pelvic floor dysfunctions associated with pelvic floor overactivity in both men and women are reviewed in detail. Individual chapters are devoted to female genital pain and vulvodynia, female bladder pain, male chronic pelvic and genital pain, sexual dysfunction related to pelvic pain in both men and women, musculoskeletal aspects of pelvic floor overactivity, lower urinary tract symptoms, voiding dysfunction, and anorectal disorders.

Assessment of the pelvic floor is addressed in distinct chapters describing subjective and objective assessment tools. State-of-the-art testing measures including electromyographic, urodynamic analysis, and imaging techniques are introduced. The final chapters are devoted to medical, psychosocial, and physical therapy treatment interventions with an emphasis on interdisciplinary management.

It is our hope and belief that this textbook will serve as a very useful resource for physicians, nurses, psychotherapists, sex therapists, and physical

therapists and will help guide patient management as well as stimulate investigative efforts. We are deeply indebted to the chapter authors, our collaborators in this meaningful project, for contributing their expertise and wisdom and for their shared commitment to improving the mental and physical health and quality of life of patients with OPF.

Zerifin, Israel — Anna Padoa
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Definitions and Basic Etiology of the Overactive Pelvic Floor

1

Stéphanie Thibault-Gagnon

1.1 Introduction

As the caudal boundary of the abdominopelvic cavity, the pelvic floor acts as the "foundation" of the human body. The anatomical integrity and proper functioning of the pelvic floor muscles and their associated neural, vascular, and connective tissue structures, as well as the interplay between them, are essential for some of the primary functions of life, including: stability for the lumbar spine, pelvis, and hips; support of the pelvic organs (i.e., bladder, uterus, rectum); storage and evacuation of urine and feces; and sexual function. Impairments and/or pain affecting the pelvic floor muscles directly or indirectly via related organs may result in dysregulation of any of these body system functions.

Pelvic floor muscle dysfunction is often thought of in terms of hypotonic, damaged and/or weakened muscles, associated with disorders such as urinary and fecal incontinence, and pelvic organ prolapse. Several other conditions, however, such as elimination disorders of the bladder and bowel, sexual dysfunction and genital/pelvic pain syndromes (e.g., vulvodynia in women, prostatodynia in men) are also commonly associated with pelvic floor muscle dysfunction, whereby, in these cases, the muscles are thought to be hyperactive (overactive), and consequently hypertonic. In this text, we refer to the pelvic floor muscles in a state of hyperactivity and/or hypertonicity as the "overactive pelvic floor" (OAPF).

Symptoms and conditions associated with OAPF are common and may significantly affect the health of women, men, and children. Whether OAPF is present as the primary symptom generator (i.e., trigger) or one component of a complex symptomatic presentation, it is considered to be a significant contributing factor in the circular processes that perpetuate pelvic dysfunction and pain. The contribution of OAPF is often overlooked in the assessment of individuals with pelvic problems, although normalization of pelvic floor muscle activation and tone may be key in the successful management of symptoms.

It is crucial for clinicians to have a broad understanding of the pathophysiological processes that may contribute to OAPF, in order to optimize treatment success for patients. This chapter provides a brief overview of the functional anatomy and neural control of the pelvic floor muscles, and also reviews the definitions and the basic etiological theories for OAPF. The conditions associated with OAPF are only briefly mentioned, as these will be discussed in further detail in later chapters.

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1.2 The Pelvic Floor Muscles

Different skeletal (striated) muscles are found within the successive layers of the pelvic floor; they are collectively known as the pelvic floor muscles. They are attached directly and indirectly (via the endopelvic fascia) to the pubic bones, ischial spines, pelvic sidewall, sacrum, and coccyx. With the exception of the striated sphincters (urethral and anal), the pelvic floor muscles exist as sets of bilaterally symmetrical parts. Clinically, the various muscles of the pelvic floor work as a functional unit. That is, the pelvic floor muscles "normally contract simultaneously as a mass contraction, but contraction quality and contribution of the [different muscles] may differ" depending on the task [1].

The pelvic floor muscles are composed roughly of 70 % type I (slow twitch) and 30 % type II (fast twitch) fibers [2]. The predominance of slow-twitch fibers emphasizes their postural and supportive roles. The fast-twitch fibers, which are found in higher proportion around the urethra and the anus [3, 4], are particularly necessary for dynamic closure of these pelvic openings in response to postural disturbances and increases in intra-abdominal pressure (e.g., with coughing, lifting).

1.2.1 Functional Anatomy of the Pelvic Floor Muscles

1.2.1.1 The Superficial Layer

The most superficial (inferior) layer of the pelvic floor includes the superficial transverse perineal, bulbospongiosus, and ischiocavernosus muscles. The transverse perineal muscles, which have a supportive role [5], arise laterally from the ischiopubic rami and insert into the perineal body centrally, where they join the external (striated) anal sphincter [6]. The external anal sphincter, the proper functioning of which is important for the maintenance of fecal continence and for defecation, may also be considered as part of the superficial layer [7]. In women, the bulbospongiosus muscles arise from the clitoris and run bilaterally along the vestibule of the vagina enveloping the vestibular bulbs (clitoral erectile bodies), before reaching the perineal body [7]. In men, they cover the proximal portion of the corpus spongiosum of the penis and are continuous with the penile fascia on the superior aspect of the penis, and also insert into the perineal body [6, 7]. The ischiocavernosus muscles originate anteriorly from the external surface of the pubic rami on each side, cover the crus of the clitoris in women and the crus of the penis in men, and terminate at the ischiopubic rami [6, 7]. The bulbospongiosus and ischiocavernosus muscles are considered to play important roles in sexual function, specifically, in arousal/erection, orgasm and ejaculation, through both the somatic and autonomic systems [8]. The combined contraction of these two muscles is thought to maintain erection by blocking venous outflow from the penis or clitoris, while their rhythmic contraction is part of the orgasmic response [8]. The ischiocavernosus has been labeled as the "muscle of erection" [9] because its contraction appears to be responsible for elevating the penis into a more upright position [10], while the rhythmic reflex contractions of the bulbospongiosus upon orgasm seem to be responsible for ejecting semen from the penis; it has therefore been called the "muscle of ejaculation" [9].

1.2.1.2 The Intermediate Layer (Perineal Membrane/Urogenital Diaphragm)

The intermediate layer of the pelvic floor is known as the perineal membrane or the urogenital diaphragm. There is controversy regarding the exact anatomical components of this layer [11]. Generally, it is thought to comprise a thin muscular sheet, the deep transverse perineal muscle, that extends across the pubic arch, inferior to the urethra and sandwiched between a superior and inferior fascial layer, which is continuous in men, but transpierced by the vagina in women [6, 7]. The existence of the deep transverse perineal muscle has been debated, especially in women, due to inconsistent empirical findings [7]. It has been suggested that its fibers are actually part of the striated urethral muscles, namely the compressor urethrae and the urethrovaginalis (in women)

[12], which function in conjunction with the external urethral sphincter to maintain urinary continence and allow micturition.

1.2.1.3 The Deep Layer (Pelvic Diaphragm)

The deepest (most cranial) muscular layer of the pelvic floor, known as the pelvic diaphragm, is composed of the coccygeus (ischiococcygeus) muscles and the levator ani muscle group. The coccygeus muscles, which form the posterior portion of the pelvic diaphragm, arise from the ischial spines and sacrospinous ligaments and insert into the lateral sides of the coccyx and lower sacrum [6, 7]. The function of the coccygeus muscle is not fully understood. However, based on its anatomical position and its irregular development, it is considered to be the "homologue of a tail muscle" and may be an evolutionary remnant [6].

In contrast, the levator ani muscle group is considered as the dominant muscular component of the pelvic floor. Although there is still debate regarding its anatomical organization and nomenclature [11], the levator ani is generally thought to be dividable into the iliococcygeus, pubococcygeus, and puborectalis muscles [7]. In the literature, the pubococcygeus and the puborectalis are sometimes combined and referred to as the pubovisceralis muscle because of their connections to the pelvic viscera (vagina, anus) [13]. The iliococcygeus and pubococcygeus portions of the levator ani arise anteriorly from the pubic bone, laterally from the pelvic sidewall via the tendineus arc of the levator ani, and from the ischial spines, and their fibers insert posteriorly into the anococcygeal raphe and the coccyx [6, 7]. The puborectalis muscles arise from the inner surfaces of the pubic bones on either side and fuse posteriorly to form a U-shaped sling that goes around the anorectal junction, connecting with the fibers of the external anal sphincter and the anococcygeal ligament [6, 7]. The puborectalis muscle sling maintains the anorectal angle at approximately 90° when the pelvic floor muscles are at rest. This plays a role, along with anal sphincter functions, in the maintenance of fecal continence. At the time of defecation, relaxation of the puborectalis opens the anorectal angle, allowing stool to pass.

The levator ani muscles play a predominant role in supporting the pelvic organs and in maintaining continence, through their "lifting" and "occluding" actions [1]. Normally, a contraction of the levator ani muscles results in the cranial and anterior displacement of the pelvic organs and the closure, through compression, of the pelvic openings (urethra, vagina, anus) [14]. Moreover, the levator ani muscles are actively involved in the sexual response. Voluntary contraction and/or reflex activation of the levator ani muscles during genital stimulation is thought to enhance the arousal response, and, in turn, contribute to the achievement of orgasm, which itself is associated with rhythmic reflex contractions of the levator ani [15].

1.2.1.4 The Endopelvic Fascia

Superior to the muscles of the pelvic diaphragm is the endopelvic fascia, a continuous layer of dense connective tissue that covers/envelops and provides support to the pelvic floor muscles and the pelvic organs by attaching them to the pelvic sidewall [13]. It encompasses an amalgamation of collagen, elastin, smooth muscle, blood vessels, and nerves, and thus also acts as a neurovascular conduit to the pelvic structures. Areas of varying thickness are found within the endopelvic fascia, with the regions of greater tissue density referred to as ligaments. An important interactive relationship exists between the endopelvic fascia and the pelvic floor muscles, which allows both entities to accomplish their functional roles.

The integrity of the pelvic floor muscles is a critical element to the proper functioning of the endopelvic fascia. The ligaments and fascia act to stabilize the pelvic organs in their positions above the pelvic floor muscles [13]. When the pelvic floor muscles, most notably the levator ani muscles, function properly and support the pelvic organs from below, the ligaments of the endopelvic fascia are not under any undue tension. When the pelvic floor musculature is damaged, and is therefore deficient in providing support to the pelvic organs from below, the connective tissues

must carry a greater load to support the pelvic organs from above [13]. The ligaments and fascia can only sustain this excess tension and maintain the organs in place for so long before they fail and pelvic organ prolapse ensues [13]. Similarly, because the endopelvic fascia provides their anchorage to the bony pelvis, the levator ani muscle function is highly dependent on its integrity [13]. Thus, defects in the endopelvic fascia, for example due to childbirth or pelvic surgery, may impact pelvic floor muscle function.

1.2.2 Neural Control of the Pelvic Floor Muscles

The neural control of the pelvic floor muscles has a complexity surpassing that of other skeletal muscles of the body. The mechanisms involved in the stabilization of the lumbo-pelvic region, pelvic organ support, urinary and fecal continence and elimination, as well as sexual function, depend on the effective and coordinated actions of the pelvic floor muscles and sphincters. These functions also rely on unique interactions between the somatic and autonomic nervous systems [6, 16].

1.2.2.1 Innervations of the Pelvic Floor

The somatic efferent (motor) nerve fibers to the pelvic floor muscles arise predominantly from the second to fourth sacral nerves (S2–S4) [7]. Separate branches from the sacral plexus supply the levator ani (S3–S4) and coccygeus muscles (S3–S4) directly and also form the pudendal nerve (S2–S4) [7]. The pudendal nerve then branches out to supply the remaining pelvic floor muscles, the striated urethral and anal sphincters, and perhaps also part of the puborectalis portion of the levator ani [7, 17]. The transmission of somatic afferent (sensory) information from the pelvic floor region, which includes sensations of touch, pressure, temperature, and pain from the skin, and proprioceptive information from muscles and joints, is mainly via the pudendal nerve (S2–S4) [7]. Additional nerves that have sensory distributions in and near the pelvic floor region include the iliohypogastric and ilioinguinal nerves (L1), the genital branch of the genitofemoral nerve (L1–L2), and the obturator nerve (L2–L4), as well as the anococcygeal nerves [7].

The autonomic nervous system supplies efferent (visceromotor) innervations (sympathetic and parasympathetic) to the smooth muscles of the pelvic organs, sphincters, and blood vessels, and also to the secretory glands of the pelvic region [7]. Sympathetic innervations to the pelvic floor arise from the thoracolumbar region (T10–L2) [7]. The preganglionic sympathetic nerves originate from these segments within the spinal cord and synapse with postganglionic neurons within the sympathetic chain ganglia or the inferior hypogastric plexus [7]. In either case, postganglionic neurons, via the inferior hypogastric plexus, reach the wall of the organ they supply [7]. Parasympathetic innervations to the pelvic floor originate from the sacral spinal segments (S2–S4) [7, 16]. Preganglionic nerve fibers arise from these levels, forming the pelvic splanchnic nerves, and course on through to the inferior hypogastric plexus (without synapsing) toward their end organ, where they synapse with short postganglionic fibers [7]. The autonomic afferent (sensory) nerve fibers run alongside autonomic efferent nerve fibers, following the same routes, to transmit sensory information from the pelvic viscera and other autonomic structures out of the pelvis to the dorsal horns of the spinal cord [7, 16].

1.2.2.2 "Tonic" and "Phasic" Pelvic Floor Muscle Activity

The pelvic floor muscles are the only skeletal muscles in the human body that are known to exhibit myoelectrical activity when they are maintained at rest [18, 19]. Research has shown that the striated urethral and anal sphincters and the levator ani (although not at all sites) demonstrate constant baseline activity [20, 21], commonly referred to as "tonic" activity [22]. This continuous low-level activity is thought to play a role in continence by helping to keep the pelvic openings closed [13] and has also been linked to the support and postural roles of the pelvic floor muscles [22]. At the time of voiding or defecation, an inhibition of the tonic activity of the pelvic floor muscles leads to muscular

relaxation [22], allowing for the evacuation of urine or stool. In this sense, although tonic muscular activity is present without conscious awareness, it may be inhibited voluntarily. This ability to voluntarily relax the pelvic floor muscles is also important in the context of sexual activity, for example, to allow for vaginal penetration during intercourse. The pelvic floor muscles also exhibit "phasic" activity; higher-level activity that occurs for short durations with stronger voluntary or reflex contractions, for example, in response to pain or sudden increases in intra-abdominal pressure [22].

1.3 Defining the "OAPF"

A number of definitions and terms have been proposed to describe conditions involving OAPF muscles, including: hyperactive pelvic floor syndrome (HPFS) [23], hypertonic pelvic floor disorder [24, 25], pelvic floor tension myalgia [26, 27], high-tone pelvic floor [28–30], short pelvic floor [31, 32], levator (ani) or puborectalis syndrome [33], and non-relaxing pelvic floor [34]. There is no firm definition for the OAPF, as there is, to date, no method of objective evaluation that can provide a clear diagnosis. Conditions that are suspected to be associated with OAPF are multifactorial in nature, and include multiple possible etiologies and complex symptomatologies. Individuals with suspected OAPF often present with a mosaic of comorbid urological, gastrointestinal, gynecological, and musculoskeletal manifestations compounded by psychoemotional distress [23, 25, 34]. Table 1.1 presents symptoms and conditions that may be associated with OAPF.

In a 2005 report from the Pelvic Floor Clinical Assessment Group of the International Continence Society (ICS), the term "OAPF muscles" was defined as a condition "in which the pelvic floor muscles do not relax, or may even contract when relaxation is functionally needed, for example during micturition or defecation" [35]. A diagnosis of OAPF is based on both

Table 1.1 Symptoms and conditions that may be associated with overactive pelvic floor muscles

In both women and men	In women	In men
Chronic pelvic pain (CPP)[a]	Urethral syndrome[a]	Chronic prostatitis/prostatodynia[a]
Perineal pain[a]	Urinary retention	Orchialgia (testicular pain)[a]
Perianal pain[a]	Overactive bladder[a]	Penile pain[a]
Sexual dysfunction[a]	Interstitial cystitis (IC)[a]	Ejaculatory pain or obstruction[a]
Voiding dysfunction	Urinary tract infections	Obstructive voiding ("prostatism")[a]
Urinary urgency	Vaginal infections	
Frequent urination	Dyspareunia[a]	
Obstructive defecation	Vulvodynia[a]	
Constipation[a]	Vestibulodynia	
Irritable bowel syndrome (IBS)[a]	Vaginismus	
Proctalgia fugax	Sexual arousal disorder[a]	
Anismus	Orgasmic pain[a]	
Anal fissures[a]	Pelvic congestion[a]	
Hemorrhoids[a]		
Coccygodynia[a]		
Varicocele[a]		
Low back pain[a]		
Hyperventilation[a]		

The list was compiled using information from different sources [23–25, 31, 34].
[a]Indicates the "symptoms known to be associated with pelvic floor dysfunctions" pertaining to the definition for the Hyperactive Pelvic Floor Syndrome (HPFS) proposed by Van Lunsen and Ramakers [23]

symptoms (subjective complaints) such as voiding problems, obstructed defecation, or dyspareunia, and on signs observable upon physical examination, like the absence of voluntary pelvic floor muscle relaxation [35]. Van Lunsen and Ramakers proposed a similar but broader definition for the HPFS [23]. Based on a review of the available scientific data and on clinical observations, the authors proposed three diagnostic criteria for HPFS: (a) comorbidity of three or more symptoms known to be associated with pelvic floor dysfunction (see Table 1.1); (b) evidence of pelvic floor dysfunction based on physical pelvic floor assessment and/or functional tests; and (c) comorbidity of one or more sources of psychological distress [23]. Although these exact diagnostic criteria have not been validated to date, they highlight the need to adopt a biopsychosocial perspective and a multidisciplinary approach in the clinical diagnosis and treatment of OAPF.

Another issue that appears to have perpetuated the lack of a clear definition for OAPF is an apparent general misunderstanding of what constitutes and differentiates skeletal muscle tone from muscle activity. Terms related to an increase in muscle tone (e.g., hypertonicity, hypertonia) have often been used synonymously with, or instead of, terms to designate a state of elevated muscular activity (e.g., overactivity, hyperactivity). This has led to the common misconception that heightened muscle tone is the direct result of elevated muscle activity and that the two physical states are equivalent. A discussion of the distinction between muscle tone and muscle activity is presented below because this knowledge is essential for understanding the pathophysiological processes potentially underlying the development and maintenance of OAPF, and associated symptoms and conditions.

1.3.1 "Muscle Tone" versus "Muscle Activity"

Muscle tone, commonly referred to as muscle tension, is measured as stiffness; the change in resistance or force per unit change in length (Δforce/Δdistance) [18]. Clinically, the tone/stiffness of the pelvic floor muscles is assessed through palpation as the resistance felt when a passive stretch is applied to the muscles [36, 37]. In a normally innervated skeletal muscle, muscle tone comprises both passive (viscoelastic) and active (contractile) components [18]. Muscle activity is an active component of muscle tone and refers to the electrical activity generated by muscle fibers when the motor unit is active and propagation of action potentials is detectable by electromyography (EMG) [18]. The muscular contraction resulting from muscle activity (i.e., electrogenic contraction) contributes to muscle tone. During the measurement of muscle tone, close monitoring of EMG recordings can help identify the presence and relative contribution of electrogenic contraction. Both normal and abnormal electrogenic contraction (i.e., detectable by EMG) can occur [18]. Normal electrogenic contraction refers to contractile activity that occurs in a normal muscle because it is not completely relaxed, but can be controlled voluntarily, or due to reflex activation (e.g., myotactic stretch reflex) [18, 38]. On the other hand, muscle spasm is defined as an abnormal/pathological (involuntary) electrogenic contraction that may or may not be painful [18]. In the context of healthy normally innervated skeletal muscle, a muscle cramp can be considered to be a form of muscle spasm [18]. The pain associated with muscle spasm/cramp may be caused by the shearing forces between the "cramping" and normal (not cramping) parts of the muscle [18, 39]. In addition, pain can also occur if the muscle becomes ischemic and releases pain-producing substances, which can occur if the muscle contracts forcefully for too long, and compresses its own blood vessels [18]. Although the assessment of muscle spasm alone is challenging, its possible contribution to muscle tone must be acknowledged.

Endogenous contracture, defined as a contractile state within a muscle that is not accompanied by electrical activity (i.e., no EMG activity is detected) [18], is also considered to be an active component of muscle tone. In normal muscle, palpable taut bands that are often associated with myofascial trigger points (i.e., hypersensitive/painful spots found within the taut bands of

muscle) [40], which are the hallmarks of myofascial pain syndromes, have been suggested to represent a form of endogenous muscle contracture [18]. Taut bands, within otherwise relaxed muscles, are devoid of action potentials (i.e., EMG-silent) although trigger points have been found to exhibit electrical activity at their loci [18]. To date, there is no conclusive evidence regarding the pathogenesis of trigger points and taut bands although research supports more local factors such as focal ischemia and the associated release of various biochemical substances rather than central processes [18, 38, 41]. Spinal cord mechanisms, however, are thought to mediate pain referral and local twitch responses associated with trigger points [42], and sensitization of central pain pathways is thought to play an important role in the conversion of episodic myofascial pain arising from taut bands and trigger points, to more chronic pain states [43]. It has also been suggested that muscle spasm can induce the development of trigger points, and vice versa [18]. Although trigger points and taut bands can be detected with palpation [40], their exact contribution to muscle tone, as with muscle spasm, is difficult to measure.

1.3.2 "Hypertonic" versus "Overactive" Pelvic Floor Muscles

In a healthy skeletal muscle, an increase in muscle activity/EMG usually results in an increase in muscle tone due to the tension built-up from the contraction of muscle fibers. Hypertonic pelvic floor muscles, however, are not necessarily overactive. Muscle hypertonicity is a general increase in muscle tone that can be associated with either elevated contractile activity and/or viscoelastic stiffening in the muscle [18, 38] and may exist in the absence of muscle activity altogether. Figure 1.1 illustrates possible sources of muscle hypertonicity (measured as increased stiffness) in a normally innervated skeletal muscle.

Several passive (viscoelastic) structures are thought to contribute to the tone of skeletal muscles, including: the cross-bridges between the sarcomeric contractile proteins actin and myosin [44, 45], the extensibility of actomyosin filaments themselves [46, 47], the non-contractile proteins of the sarcomeric cytoskeletons (titin and desmin) and their filamentous connections [48–50], as well as the connective tissues (fascia) linking and covering muscle tissue [51–54]. An increase in muscle stiffness may occur due to changes in any of these passive structures, and in the absence of any detectable EMG. Both an increase (hypertrophy) and a decrease (atrophy) in a muscle's size and mass are associated with increased muscle stiffness due to physiological adaptations of viscoelastic structures [51]. Since the sarcomeric proteins actin, myosin, titin, and desmin all reside within the muscle tissue itself, the increased stiffness that results from muscle hypertrophy [55] has been attributed in part to increases in these subcellular components [51]. Conversely, an increase in muscle stiffness seen with muscle atrophy due to disuse/immobilization is considered to primarily result from an accumulation and increased relative proportion of connective (fibrous) tissue within and surrounding the muscle [51]. Additionally, tissue adhesions (scars) resulting from injury to the muscle may also increase the viscoelastic stiffness of the muscle.

Although by definition OAPF implies a physical state of heightened activity within the pelvic floor muscles, individuals with OAPF are also commonly found to present with pelvic floor muscle hypertonicity from other sources, most notably myofascial trigger points [24, 31, 56], which are not associated with any detectable EMG. Emerging research also suggests that pelvic floor muscle hypertonicity in populations with OAPF symptoms and associated conditions may be in part due to changes in the muscles' viscoelastic properties [57, 58]. Understanding and recognizing the various potential sources of pelvic floor muscle hypertonicity is particularly important for identifying the specific pelvic floor impairments affecting individuals with OAPF, and designing tailored treatment interventions; for example, in the case of physiotherapy, deciding whether to emphasize pelvic floor muscle awareness, control and relaxation exercises, and/or

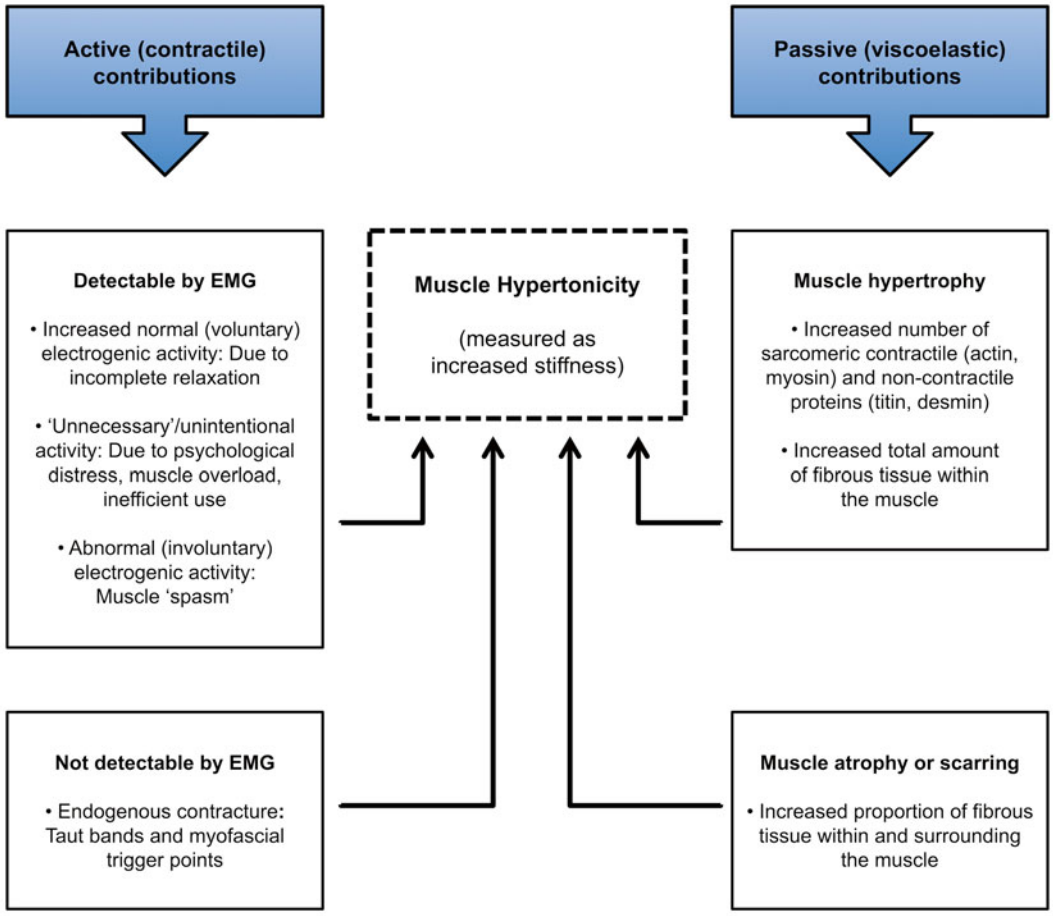

Fig. 1.1 Possible sources of muscle hypertonicity in a normally innervated skeletal muscle. Based on information from Simons and Mense [18] and Gajdosik [51]

manual stretches and trigger point release techniques. In many cases, it is likely that OAPF symptoms are associated with various components of pelvic floor muscle hypertonicity and dysfunction, and a comprehensive treatment program involving a number of different techniques and strategies is usually applied to target all levels of dysfunction [32, 56, 59–61].

The relationships between the different sources of muscle tone (active and passive), and their contribution to muscle hypertonicity, remain under investigation. Although speculative, it is possible that in some cases there is a sequential pattern to the relative contribution of different elements to muscle hypertonicity. For example, a persistent lack of muscle relaxation and/or heightened (but "normal") muscular activity may lead to involuntary muscle contraction (spasm), which may develop further into a myofascial pain syndrome involving taut bands and trigger points. Subsequently, the lack of movement/disuse of the muscles can lead to adaptive changes in their passive structures and result in viscoelastic stiffening. Given that the etiology of OAPF is poorly understood, the existence of relationships between the different components of muscle tone and a possible chronological nature to the development of muscle hypertonicity could be helpful in advancing knowledge on the pathogenesis of OAPF, as well as the duration and severity of impairments. Further research, however, is needed to elucidate these concepts, which for now remain hypothetical.

1.3.3 "Unnecessary" Muscle Tension/Activity

Of high relevance to the topic of OAPF, is the concept of "unnecessary" muscle tension [18]. In addition to the aforementioned active and passive components of muscle tone, Simons and Mense proposed that a type of muscular activity that is unintentional, but that is not a "spasm," exists and is the source of what is often referred to clinically as "muscle tension" [18]. These authors note that this unnecessary/unintentional muscle activity, which is amenable to voluntary control with training (e.g., through biofeedback assistance), may arise from psychological distress or anxiety, overload from sustained contraction or repetitive activity, and/or inefficient use of muscles [18]. These sources of increased muscular activity, as they may pertain to OAPF, are discussed later in this chapter.

1.4 Etiology of the OAPF

OAPF may well be "the organic substrate causing both different kinds of urethral, vaginal, and anal outlet obstruction and different kinds of [genital/pelvic] pain as well as sexual dysfunctions" [23]. According to Van Lunsen and Ramakers this is supported by empirical evidence, including research demonstrating the effectiveness of physiotherapy interventions aimed at improving pelvic floor muscle relaxation on symptoms of conditions associated with OAPF (e.g., vulvodynia, prostatodynia, dysfunctional voiding, constipation) [23]. The mechanisms underlying the onset of OAPF, however, are not fully understood.

The coexistence of different kinds of urinary, anorectal, and gynecological problems, sexual difficulties, genital/pelvic pain, and psychological distress in individuals with OAPF suggests that, as per its clinical presentation, the etiology of OAPF is likely multifactorial. OAPF is thought to occur as a "conditioned response to threat" [23], which may come in different forms. A variety of risk factors or etiological determinants have been proposed to explain the onset and maintenance of OAPF.

Pelvic pain, which refers to pain located anywhere in the genital, pelvic, and lower abdominal region, which may itself result from OAPF, is also considered to be the predominant cause of OAPF [24]. Pelvic pain has several possible origins and perpetuating factors, including physical injury or pathology affecting any biological tissue (musculoskeletal, neural, visceral) within the pelvic area or distant structures with pain referral patterns to this region, as well as psychological, psychosocial, and/or psychosexual distress [33, 62, 63], which in turn are also risk factors for the onset of OAPF. Additional potential triggers for the development of OAPF include abnormal patterns of pelvic floor muscle use, direct trauma and/or pathology, and postural abnormalities resulting from faulty postures, sustained positions, repetitive activities and/or skeletal asymmetries. Identifying the underlying cause(s) and perpetuating factors of OAPF, although often challenging, may be of significant importance when attempting to break the "vicious cycle" of ongoing pelvic dysfunction and pain experienced by affected individuals.

1.4.1 Chronic Pelvic Pain

Pain is defined as "an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage" [64]. Chronic pain is typically defined as "non-malignant" pain that lasts longer than 6 months although duration is not a strict criterion [64]. Important evidence of chronic pain is that the pain is "out of proportion to any initiating pathology or the degree of tissue damage", and is associated with significant psychological, emotional, behavioral, and environmental/social disturbances [65]. The neurophysiological basis for the multidimensional nature of chronic pain can be explained by the body-self neuromatrix described by Melzack [66], a neural network within the brain that integrates various inputs to produce the output of "pain". Melzack explained that somatosensory, thalamocortical, and limbic components of the nervous system interact closely to form the

"sensory-discriminative", "evaluative-cognitive", and "affective-motivational" dimensions of pain [66].

There is a very close relationship between OAPF and chronic pelvic pain (CPP). Although the mechanisms of association are not completely understood, pelvic floor muscle overactivity and hypertonicity have been found to be physical hallmarks of several different conditions involving CPP, including irritable bowel syndrome, (IBS), interstitial cystitis/bladder pain syndrome (IC/BPS), vulvodynia in women, and chronic prostatitis/prostatodynia in men [23–25, 67]. According to Diatchenko and colleagues [68], CPP is an "idiopathic pain disorder" (IPD) which, along with associated pelvic conditions (IBS, IC/BPS, and vulvodynia) and other non-pelvic conditions (such as fibromyalgia and chronic headaches), has two primary pathways of vulnerability that underlie its development. Such pathways include pain amplification and psychological distress and are both mediated by genetic and environmental/social factors [68]. Since CPP and OAPF are so intimately linked, a review of the mechanisms by which enhanced pain perception/pain amplification and psychological distress may occur and contribute to CPP may be useful for illustrating the possible etiological pathways through which OAPF may develop.

1.4.1.1 Neurophysiology of Pain

Pain begins with a noxious stimulus, an actual or potential tissue-damaging event that can be mechanical, chemical, or thermal [69]. Primary afferent fibers (thinly myelinated A-delta fibers and unmyelinated C-fibers from the skin and viscera, and group III and IV nerve fibers from the muscles and joints) transmit nociceptive signals from the periphery to the dorsal horn of the spinal cord where they synapse with interneurons, which mediate spinal reflexes, and with second-order neurons that transmit the nociceptive signals towards higher brain centers [69]. In the case of pelvic pain, the source of noxious input can be from any structure within the "sensory window of the pelvis" [70] (e.g., skin, bones, muscles, nerves, connective tissues, viscera), and as previously mentioned, also from structures outside of the pelvis that refer pain to the region.

Pain is not merely a stimulus–response process. Pain serves as an alarm system to the body, warning it of an actual or perceived threat of harm, and involves physiological processes within the body. The outcome of these processes, however, that is, whether a pain response occurs or not, is ultimately dependant on cognitive awareness and subjective appraisal [66]. As Hilton and Vandyken [71] noted, there is no such thing as "pain fibers" carrying "pain signals"; there are "nociceptive fibers" transmitting nociceptive or "danger signals" from the periphery to the brain. A noxious stimulus is only interpreted as "painful" once nociceptive information is integrated and processed within the brain, and the brain has decided that it is worth paying attention to. Otherwise, there is no pain response.

Tissue injury or pathology is not a prerequisite for a noxious stimulus to occur and cause pain [66]. Thoughts alone have been shown to activate autonomic nervous system mechanisms and produce an inflammatory response in individuals with chronic pain [72].

1.4.1.2 Neuropathophysiology of Chronic Pain

When noxious stimuli occur over a prolonged period of time, a series of processes occur within the peripheral and central nervous systems. The cumulative effect of these processes is the up-regulation of nociceptive system function, which leads to dysregulations in both the peripheral and central mechanisms of sensory and pain processing, and abnormal neuropathic output states [73]. A number of mechanisms are thought to be responsible for the up-regulation of nociceptive nervous system components.

Peripheral sensitization, which refers to the sensitization of peripheral nociceptors, is thought to occur mainly via the influence of biochemical/inflammatory mediators, which are released in response to ongoing noxious input [73]. As mentioned above, such an inflammatory response can occur in the absence of tissue damage [72]. The sensitization of peripheral nociceptors produces a

reduction in their activation thresholds, causing increased firing responses to suprathreshold stimulation as well as spontaneous discharge [73]. Additional peripheral mechanisms that may contribute to the up-regulation of the nociceptive system include: (1) activation of "silent" nociceptive afferents, a special class of C-fibers that remain dormant under normal conditions but are activated by prolonged or highly noxious stimuli, and (2) conversion of myelinated afferents, such that they begin to act like nociceptive fibers [70, 73]. Ultimately, these changes within the peripheral nociceptive system contribute to increasing the noxious influx to the dorsal horn of the spinal cord.

Central sensitization involves a series of neuroplastic changes that occur in response to prolonged noxious stimuli, which result in the up-regulation (sensitization) of the dorsal horn of the spinal cord. The flooding of the dorsal horn by noxious input leads to biochemical and neuroinflammatory events that can produce a reduction in the response thresholds of central neurons that process nociceptive signals and also enhance the signals transmitted by non-nociceptive afferents, such that they start contributing to pain perception [73].

Consequences of nociceptive system up-regulation and sensitization include "hyperalgesia," defined as an increased response to a stimulus that is normally painful, and "allodynia," which is pain due to a stimulus that does not normally provoke pain [64, 73], both of which have been found to be present in individuals with CPP [70, 74–78].

Of equal importance are the neuropathic output states that result from the up-regulation of the dorsal horn of the spinal cord [24, 73]. Since the main outcomes of central sensitization are increased synaptic efficacy and increased excitability of nociceptive central neurons within the dorsal horn, it is likely that these effects also influence the activity of other neurons with which they make synaptic connections [73]. This in turn leads to neuropathic reflexes, which are likely to underlie the sensorimotor and autonomic dysfunctions that occur in individuals experiencing chronic pain [73]. The first neuropathic reflex is known as "neurogenic inflammation" [70]. It involves a dorsal root reflex that causes afferent nerves to fire antidromically (backwards via the sensory peripheral nerve), leading to inflammation and hyperalgesia in the periphery [70]. Neurogenic inflammation and shared neural pathways between the pelvic viscera, known as pelvic organ "cross talk," may be responsible for another neuropathic event known as "viscero-visceral hyperalgesia" [70]. This phenomenon remains the primary explanation for the coexistence of various visceral CPP syndromes (e.g., IC, IBS, vulvodynia) [23, 24, 70]. In addition to the potential effect of dorsal horn up-regulation on alpha motor neuron excitability, a neuropathic reflex known as "viscero-muscular hyperalgesia" [24], which may involve the sensitization of muscle spindle afferents and increased excitability of gamma motor neurons, is thought to contribute to muscular instability and the development of myofascial trigger points [70, 73]. This neuropathic output state may, at least partly, explain how CPP may lead to OAPF [24, 70, 73].

1.4.2 Psychological Distress

Psychological distress and associated cognitive, emotional, and behavioral factors are thought to play a role in triggering and/or perpetuating CPP [79], and consequently OAPF. In acute/subacute states, pain acts to warn the body of actual or potential harm. However, when the threat is no longer present and an individual continues to perceive the pain that he/she is experiencing as threatening, processes are initiated that set the stage for the development of chronic pain. These processes form the fundamental components of the fear-avoidance model (FAM) of chronic pain, a widely accepted conceptual model that explains how negative pain-related cognitions and maladaptive behavioral responses contribute to the development and maintenance of chronic pain [79–82]. The ongoing appraisal of pain as threatening leads to "pain catastrophizing", in

which a person focuses on pain sensations and exaggerates the threat and intensity of pain [79, 81]. This in turn leads to the chief component of the FAM, namely "pain-related fear" [81]. The fear of pain, combined with pain-related anxiety and hypervigilance (i.e., heightened attention) to pain, leads to defensive behaviors, notably muscular reactivity/contraction, in the presence of a painful stimulus or in the anticipation of pain [81]. Ultimately, negative pain cognitions and behavioral responses to pain lead to escape and avoidance behaviors, which in turn lead to disuse, further perpetuating the "vicious cycle" of pain and dysfunction [79–82].

The aforementioned defensive muscular reactions occurring episodically during exposure to threatening situations, with repeated exposure, may become more generalized. For example, in the context of women experiencing dyspareunia, defensive pelvic floor muscle reactions occurring in response to pain or the anticipation/fear of pain upon vaginal penetration [79, 83], over time, are thought to lead to more constant pelvic floor muscle overactivity and hypertonicity [57, 83–87]. Psycho-emotional distress alone, irrespective of the presence of pain, can induce increases in muscular activity. In the case of the pelvic floor muscles, experimental research has shown that these muscles are highly sensitive to emotional distress. Van der Velde and colleagues [88, 89] showed that both women with and without vaginismus exhibited pelvic floor muscular defensive reactions in response to sexual and nonsexual threatening film excerpts. This muscular reactivity can be considered analogous to "unnecessary" or unintentional muscle tension/activity, as described by Simons and Mense [18], which may have a psychogenic origin.

1.4.3 Psychosocial and Psychosexual Disturbances

Psychosocial and psychosexual disturbances are reported to be common in individuals with CPP [62] and those with symptoms of OAPF [23, 34]. Disruptions in intimate relationships and "altered family dynamics" appear to be part of the psychosocial downfall of CPP [62]. Along with traumatic life experiences, including sexual trauma and physical abuse, they may also constitute inciting events that lead to CPP and OAPF in both men and women [90, 91].

Given the aforementioned effects of psychological distress on muscular activity, it is not difficult to understand how a history of traumatic experience may lead to OAPF, especially if the events occur repeatedly or if the person "re-lives" the experiences through, for example, flashbacks or nightmares [90].

1.4.4 Abnormal Behavior/Pattern of Pelvic Floor Muscle Use

Voluntary control of urinary and anorectal functions begins in early childhood. During toilet-training years, children learn to contract or "squeeze" their pelvic floor muscles when it is not yet time to go, and relax these muscles in order to void or pass stool when the time is appropriate. Dysfunctional voiding and/or defecation can result from improper learning of these control mechanisms, or from an abnormal behavior/pattern of pelvic floor muscle use, for example, prolonged voluntary holding [34]. This dysfunctional behavior, which is commonly found in adults and may result from various factors including "habit, lifestyle, occupation, or constant recruitment of [the pelvic floor muscles] to avoid bowel or bladder incontinence" [34], may lead to OAPF in the form of paradoxical pelvic floor muscle and sphincter contractions (inability to relax) at the time of voiding and/or defecation. In this sense, OAPF is considered to be a significant contributor to the development and maintenance of elimination disorders, which are common in childhood and in adulthood [23, 25].

1.4.5 Direct Trauma or Pathology

Vaginal delivery and pelvic surgery, the main culprits of neuromuscular and myofascial injury to the pelvic floor, can also lead to OAPF. Both

obstetric injury and pelvic surgical procedures, especially those that involve fixations to the muscles of the pelvis, have been reported to result in painful and hypertonic pelvic floor muscles [24, 34]. OAPF may be the consequence of inflammation and pain resulting from the trauma, but it may also result from anatomical disruptions. When tissues in one region of the pelvic floor become scarred and tense, or weak and lax, tissue imbalances are created, and both overload/overuse and/or inefficient use of certain muscles can ensue. Pelvic floor muscle compensations may occur, for example, as a counteracting response to imbalances and/or as a stabilizing response to instability/hypermobility in a region (e.g., ligamentous laxity) [18, 67]. This may cause an interesting clinical paradigm where muscle hypotonicity and hypertonicity can coexist [67].

In addition to direct trauma, any pathology or disorder affecting the neuromuscular and connective tissue structures of the pelvic floor region, inherited (e.g., congenital disorders involving decreased collagen content) or acquired, may also lead to OAPF.

Table 1.2 Possible etiological factors for the development of overactive pelvic floor (OAPF)

• Chronic pelvic pain (CPP)[a]
• Psychological distress (e.g., anxiety, fear of pain)
• Psychosocial/psychosexual disturbances (e.g., adverse relationships, sexual trauma, or abuse)
• Abnormal behaviors/patterns of pelvic floor muscle use (e.g., prolonged holding to delay voiding or defecation)
• Direct trauma or pathology/disorder causing tissues changes within the pelvic region
• Postural abnormalities in the region of the spine, pelvis, and/or lower extremities (e.g., faulty sitting and standing postures, prolonged lack of motion and/or repetitive activities, structural/skeletal asymmetries)

[a]Chronic pelvic pain (CPP) itself may have many underlying causes [33, 63], including neuromusculoskeletal and visceral origins, which in turn are etiological factors for the onset of overactive pelvic floor (OAPF). Similarly, all of the etiological factors for OAPF may contribute to the onset and/or maintenance of CPP.

1.4.6 Postural Abnormalities

Any ongoing postural abnormality in the region of the spine, pelvis, and/or lower extremities can contribute to the development of pelvic pain and/or OAPF. Faulty sitting and standing postures (i.e., poor postural habits), prolonged lack of motion (e.g., sitting for long hours) and/or repetitive activities, as well as structural asymmetries (e.g., leg length discrepancy), all have the potential to create asymmetrical loading and excess mechanical stress on the tissues of the pelvic floor region (bone, nerves, muscles, connective tissue) [92, 93]. This in turn can lead to a variety of tissue changes, including atrophy of certain tissues due to lack of physical stress, hypertrophy of other tissues due to an increase in physical stress, the development of myofascial trigger points, as well as injury if there is excessive physical stress [92, 93]. The resultant pain and tissue/muscular imbalances can involve or result in OAPF [18] (Table 1.2).

1.5 Conclusion

A number of terms and definitions have been used in the literature to describe a physical state involving hyperactive and/or hypertonic pelvic floor muscles, which we refer to as the "OAPF." The clinical presentation of individuals with OAPF is varied and complex, usually involving an amalgamation of urinary, anorectal, and/or sexual dysfunction, genital/pelvic pain, and psychological distress. Multiple possible etiologies exist for the onset of OAPF, and it is often difficult to identify the exact cause of OAPF in a given individual. It follows that a consideration of all potential initiating and/or contributing factors is essential in the assessment and treatment of individuals with OAPF in order to successfully break the self-perpetuating cycle of pelvic dysfunction and pain associated with OAPF.

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Overactive Pelvic Floor: Female Sexual Functioning

2

Ellen Laan and Rik H.W. van Lunsen

2.1 Introduction

Pelvic floor dysfunction, most notably pelvic floor overactivity, is often associated with lower urinary tract, bowel, and gynecological symptoms [1–4] that may have negative cognitive, behavioral, sexual, and emotional consequences. In women, pelvic floor overactivity may be involved in bladder pain syndrome/interstitial cystitis, irritable bowel syndrome, chronic pelvic pain, and in sexual conditions such as dyspareunia and Provoked Vulvodynia (PVD) as defined by the International Society for the Study of Vulvovaginal Disease [5–8]. Less is known about the relationship between pelvic floor overactivity and sexual arousal, desire, and orgasm problems.

Pelvic floor dysfunction is generally regarded as a musculoskeletal dysfunction and/or poor sphincter function associated with pregnancy, vaginal deliveries, obesity, a collagen deficiency, or prolonged overexertion of the pelvic floor [9, 10]. The fact that a history of physical or sexual abuse is common among women with pelvic floor overactivity [11–13] suggests that in many cases, pelvic floor overactivity may be a symptom of chronic activation of the defensive stress-system and should thus be regarded a physical manifestation of emotional dysregulation.

This chapter addresses the relationship between pelvic floor overactivity and sexual arousal, desire, and orgasm problems in women and how this relationship may be mediated by trauma such as sexual abuse and attachment problems. The psychosocial aspects of pelvic floor overactivity and sexual pain is the topic of Chap. 20 in this book.

2.2 Pelvic Floor Muscle Anatomy and Function

The pelvic floor muscles attach from the pubic bone anteriorly, to the coccyx (tailbone) posteriorly and form a bowl-like structure, along with ligaments and fascial tissue [14]. The muscles of the pelvic floor consist of superficial muscles including the bulbospongiosus, ischiocavernosus, superficial transverse perineal and external ani sphincter muscles, an intermediate layer consisting of the deep transverse perineal, and the deeper muscles known collectively as the "levator ani" muscles, which consist of the pubococcygeus and iliococcygeus [15]. The levator ani acts to lift up the pelvic organs and is active during defecation. The puborectalis muscles act together with the external anal and urethral sphincters to close the urinary and anal openings, contract the sphincters, and prevent urinary or fecal leakage.

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The joint guideline of the International Continence Society and the International Urogynecological Association denotes function and dysfunction of the pelvic floor muscles as normal, overactive (high tone), underactive (low tone), and non-functioning [16, 17]. "High-tone" pelvic floor functioning refers to a state of muscular tension or contraction when muscle relaxation is desired or functionally required [18]. Pelvic floor muscles that cannot voluntarily contract are characterized as "low tone," whereas the term "non-functioning" signifies the absence of any muscle activity.

The pelvis and the pelvic floor have an important role in protecting and supporting the abdominal organs and maintaining good posture. A flexible pelvic floor that is neither too tense nor too lax is of great significance for effortless micturition and defecation [14]. A "low-tone" pelvic floor may be a consequence of vaginal delivery, damage to the nerves that innervate the pelvic floor muscles or by weakening of the ligaments, but also heavy manual labor, obesity, or frequent coughing may result in long-lasting overtaxing of the pelvic floor. A "high-tone" pelvic floor is often associated with toilet-training executed too early or too intensely [1], but may also be the consequence of a habit to suspend micturition or defecation, of holding in one's abdominal musculature to appear slim, or of performing intense sports that involve extreme flexing of the pelvic floor, such as ballet or gymnastics.

2.3 The Involvement of Pelvic Floor Muscles in Sexual Arousal and Orgasm

To date, little is known about the role of the pelvic floor in sexual arousal and orgasm. In 1948, Kegel was the first to describe a technique for toning and strengthening the striated pelvic floor musculature as a treatment for urinary stress incontinence [19]. During this treatment, several women reported to have enhanced erotic sensations in their genitals and a greater ability to experience orgasm. Kegel's findings were supported by a retrospective correlational study in which pubococcygeal muscle strength was found to be higher in orgasmic than anorgasmic women [20].

In their 1966 book, Masters and Johnson described voluntary and involuntary pelvic floor contractions during sexual arousal in both genders [21]. They obtained their data on the behavior of pelvic floor contractions through direct observation. Involuntary and rhythmic contractions of the pelvic floor muscles were seen during orgasm, with 0.8-s intervals. Masters and Johnson noted that the intensity of the pelvic floor contractions during arousal and orgasm appeared to decrease with age. These involuntary and rhythmic contractions during orgasm may function to restore the vasocongested pelvic tissue to their basal state, or to stimulate the male to ejaculate [21, 22].

Using vaginal and anal pressure probes to study pelvic floor contractions during orgasm, Bohlen and colleagues observed three different patterns of orgasmic contractions during masturbation in eleven women aged 24–33, with unknown pelvic floor function [22]. The first type consisted of a small number of regular contractions at subjectively indicated orgasm onset, and the second type consisted of, on average, twice as many regular contractions followed by additional irregular contractions. The latter type of orgasm lasted almost four times longer than the first type. The irregular contractions may have been the result of continued voluntary stimulation beyond the initial series of regular contractions. A small number of women reported experiencing orgasm without exhibiting any pelvic floor contractions. Anal contractions were found to occur simultaneously with vaginal contractions.

In 1974, Sherfey forwarded her "unromantic view" (p. 102) that there is no such thing as a vaginal, clitoral, or even penile orgasm [23]. According to her, the only type of orgasm is a "myovascular" orgasm (p. 103), which is a strictly mechanic (i.e., muscular) and hydraulic (i.e., venous) affair, with stimulation of an erotic arousal zone—be it the vagina, clitoris, penis, rectum, breasts, or mind—causing the venous networks of the pelvis to expand. According to Sherfey, increased venous congestion and stretching of the pelvic muscles stimulate the muscle nerve endings such that they

begin to contract. These muscular contractions, which are usually perceived as pleasurable, constitute the experience of orgasm.

More recently, using surface electromyography (EMG) and vaginal pressure measures, Shafik observed involuntary pelvic floor activity during stimulation of the clitoris, which he considered to be a clitoromotor reflex [24]. Involvement of the levator ani in vaginal elongation, uterine elevation, and vaginal muscle contractions was mainly described in terms of facilitation of male genital response, resulting from penile thrusting [25].

2.4 Does Pelvic Floor Muscle Training Enhance Sexual Arousal and Orgasm?

Almost four decades after Kegel's first studies, Messe and Geer tested Kegel's assertion about the sexual arousal enhancing properties of pubococcygeus muscle exercises in a psychophysiological study [26]. In their study, they asked women to perform vaginal contractions while engaging in sexual fantasy and compared their genital and subjective sexual responses with engaging in sexual fantasy without performing these contractions and with performing vaginal contractions without engaging in sexual fantasy. Their results showed that performing vaginal contractions without additional sexual stimulation enhanced both genital and subjective sexual arousal relative to baseline. Tensing pelvic floor muscles while engaging in sexual fantasy increased genital and subjective sexual arousal more than tensing alone and fantasizing alone. One additional week of training, the effect of which was tested in an identical second test session, did not further enhance genital and subjective sexual arousal. Messe and Geer speculated about the mechanism through which these contractions may enhance sexual arousal. Increased muscle tone may result in increased stimulation of stretch and pressure receptors during intercourse, leading to enhanced arousal and orgasmic potential. Alternatively, pubococcygeus exercises might focus a woman's attention on her genitals; this shift in attention might result in an increased perception of pleasure, with positive expectations that these exercises would enhance arousal contributing to the effect [26].

In contrast to Messe and Geer's findings, in a small group of women who were coitally orgasmic in less than 30 % of intercourse events, Kegel exercises compared to a waiting list control group and an attention control group did increase pubococcygeus strength [27], but did not show differential improvement on coital orgasmic frequency at posttest compared to the control groups. In a similar study, women with orgasm difficulties were hypothesized be more likely to become orgasmic by practicing exercises to strengthen the pelvic floor muscles over a 12-week period than women practicing relaxation exercises or than women in an attention control group [28]. Results indicated, however, that there was no difference in orgasmic outcome for the three groups during the experimental period. Finally, a recent study in 32 sexually active postmenopausal women who all had the ability to contract their pelvic floor muscles tested the hypothesis that 3 months of physical exercise including pelvic floor muscle training under biweekly guidance of a physiotherapist, and exercises performed at home three times a week, would enhance sexual function [29]. Even though pelvic floor muscle strength was significantly enhanced at posttest, this study found no effect on sexual function.

The majority of studies discussed above investigated the involvement of pelvic floor musculature in sexual arousal and orgasm in individuals of whom pelvic floor status was not reported. Given that these were generally small studies aimed at studying underlying mechanisms of arousal and orgasm, chances are that most participants did not have pelvic floor dysfunction. We conclude that, contrary to the promising findings of the early studies and contrary to common opinion, women who do not have a low-tone pelvic floor and who seek to enhance sexual arousal and more frequent orgasms have not much to gain from pelvic floor muscle training. Actually, a relaxed pelvic floor and mindful attention to sexual stimuli and bodily sensations seem a more effective means of enhancing sexual arousal and orgasm [30].

2.5 Sexual Function in Women with Pelvic Floor Dysfunction

Studies on the prevalence of sexual dysfunction among women with pelvic floor dysfunction, compared with women without such dysfunctions, have been contradictory. In several studies, sexual dysfunction was found in women with urinary incontinence and/or pelvic organ prolapse [31–37]. A large prospective study in women scheduled to undergo hysterectomy for a benign gynecological disorder investigated the relationship between sexual dysfunction and pelvic floor complaints. Of the entire sample, 495 women (38 %) had pelvic floor disorders. Compared to the women without pelvic floor symptoms, sexually active women with pelvic floor disorders were more likely to report reduced or absent sexual desire, sexual arousal problems (vaginal dryness), painful intercourse, decreased rates of orgasm occurrence and intensity, and decreased overall sexual satisfaction [34]. In another study, impaired sexual arousal was significantly associated with lower urinary tract symptoms (LUTS) in women, with 40–46 % of women with LUTS suffering from at least one sexual impairment [37]. However, other studies did not find differences in sexual function among women with or without pelvic floor dysfunction [38–40]. These differential findings may be related to age and partner status, but also to the nature of the pelvic floor dysfunction. In many studies in women with urinary incontinence or prolapse, it is unknown whether these women have a "low-tone" or "high-tone" pelvic floor dysfunction. Although a low-tone pelvic floor may be more prevalent in women with pelvic organ prolapse, many women with urinary incontinence, particularly urge incontinence, have an overactive pelvic floor [16] likely due to sustained contraction as a measure to prevent leakage. Sexual problems may differ depending on the type of pelvic floor dysfunction.

Fortunately, in a recent study [18], 85 mainly premenopausal consecutive patients referred to a physical therapy private practice were divided in a high and low pelvic floor tone group based on presented symptoms [17]. In this study, the majority of women (82.3 %) presented symptoms related to pelvic floor overactivity, and only 6 % reported non-dysfunctional sexual activity. Vulvodynia was the most common complaint (54 %). Results showed that age was significantly related to sexual function, such that women in the middle age group reported better sexual function than younger (<30 years) and older (>50 years) women. In addition, women with low-tone pelvic floor muscles had higher sexual function scores than women with pelvic floor overactivity. Women with a low-tone pelvic floor had lower FSFI sexual pain scores.

There is Level 1, Grade A evidence that pelvic floor muscle training is effective in treating stress urinary incontinence (for a recent review, see [41]). Nevertheless, there is a lack of randomized controlled trials addressing the effect of pelvic floor physiotherapy on sexual dysfunction. This is not surprising, given that most trials treating (mixed) pelvic floor disorders are aimed at enhancing pelvic floor muscle tone, whereas sexual problems are likely often, if not mostly, related to pelvic floor overactivity [18].

In all, the findings in women with pelvic floor dysfunction seem to add to the earlier conclusion that a relaxed or low-tone pelvic floor is associated with better sexual function. Clearly, that involuntary and rhythmic smooth muscle pelvic floor contractions contribute to the peak sensation of pleasure during orgasm does not imply that sexual arousal and orgasmic pleasure are enhanced by high tonus of the voluntary, striated muscles of the pelvic floor.

2.6 Pelvic Floor Overactivity and Sexual Arousal in Women with Sexual Pain

Only a handful of studies directly investigated sexual arousal in women with sexual pain disorders. Using vaginal photoplethysmography, diminished genital and subjective sexual arousal was observed in women with dyspareunia, relative to sexually functional women, during exposure to an erotic film clip depicting intercourse

[42]. In a similar study by Brauer and colleagues, these results were not replicated [43]. Women with and without dyspareunia had equally high levels of genital arousal during an oral sex clip and an intercourse clip.

Apparently, genital response in women with dyspareunia is not impaired. Genital response was found to be impaired, however, by fear of pain. In a second study by Brauer and colleagues, diminished genital arousal was observed in a threatening experimental context, which was created by the suggestion that during erotic film viewing the participant could receive a painful stimulus at her ankle [44]. This detrimental effect of fear of pain—not actual pain as the painful stimulus was never delivered—did not only occur in women with dyspareunia, but was equally great in sexually functioning women. The result of this latter study supports Spano and Lamont's hypothesis that fear of pain results in diminished genital response [45].

Fear of pain may result not only in inhibited sexual arousal but also in increased pelvic floor activity, as part of a defensive reaction. There is accumulating research that supports the idea that the pelvic floor musculature, like other muscle groups, is indirectly innervated by the limbic system and therefore highly reactive to emotional stimuli and states [46, 47]. In line with this, van der Velde and colleagues observed increased pelvic floor EMG in women with and without vaginismus in response to an anxiety provoking film, and suggested that pelvic floor muscles may, involuntarily, contract as part of a defensive response [48].

The few studies that have monitored pelvic floor activity in women with sexual pain disorders using vaginal surface EMG concerned women with dyspareunia diagnosed as provoked vestibulodynia (PVD), as well as women with vaginismus. PVD is the most common form of superficial dyspareunia in premenopausal women and is defined as a sharp/burning pain at the entrance of the vagina in response to vestibular touch or attempted vaginal entry [49, 50]. Vaginismus, described in the DSMIV-TR as recurrent or persistent involuntary spasm of the musculature of the outer third of the vagina interfering with intercourse, may be characterized by high-tone pelvic floor, chronically or in situations of attempted penetration (by any object) [51, 52]. As dyspareunia may be associated with increased pelvic floor muscle tone and most women with vaginismus report pain during attempted penetration, there is considerable overlap between the two diagnoses [53, 54]. Therefore, vaginismus and dyspareunia have been integrated in DSM 5 as genito-pelvic pain/penetration disorder [55]. Studies in women with PVD and/or vaginismus and no-pain controls revealed some evidence for differences in pelvic floor muscle tone or strength; however, these differences are not well established [56]. In women with PVD, an elevated resting EMG and stronger contractile responses to a painful vestibular pressure stimulus have been observed [57, 58]. In contrast, other studies reported lower muscle strength in women with vaginismus or PVD [59, 60].

To avoid difficulties in interpretation between various studies due to differences in specific procedures and equipment, Both and colleagues developed a vaginal probe that enables simultaneous measurement of pelvic floor EMG and vaginal pulse amplitude (VPA) [61]. To investigate the sensitivity of the device for changes in genital blood flow and involuntary changes in pelvic floor activity, VPA and vaginal surface EMG were monitored in 36 women without pelvic floor dysfunction during exposure to sexual and anxiety-evoking film clips. In addition, vaginal surface EMG was monitored during voluntary flick and hold contractions. The device appeared sensitive to changes in vaginal blood flow in response to sexual stimuli and able to pick up small, involuntary changes in pelvic floor activity associated with anxiety. Also, the device was able to record changes in pelvic floor activity during voluntary pelvic floor contractions. Results showed that VPA increased in response to the sexual film, and that EMG values were significantly higher in response to the anxiety-evoking film. Interestingly, higher EMG values in response to the sexual film were associated with lower VPA. This observation provides the first empirical support for the hypothesis that increased pelvic muscle activity may be associated with reduced blood flow to the vagina during

sexual stimulation [4, 62]. According to that hypothesis, in women with PVD the combination of increased pelvic floor muscle activity and lack of lubrication during intercourse results in friction between penis and vulvar skin, resulting in pain and possibly in tissue damage or irritation of the skin. Or, besides making vaginal entry more difficult, increased pelvic muscle activity may result in muscle pain, reduced blood flow to the vulva and vagina, and consequently, as a result of fear of pain, in reduced lubrication.

2.7 Pelvic Floor Overactivity as an Emotional Response

Very relevant when it comes to understanding its role in sexual functioning, is the fact that the pelvic floor is involved in emotional processing. In cases of actual or imminent physical or mental pain, the pelvic floor muscles will involuntarily, and often unconsciously, contract. In a number of psychophysiological studies in which pelvic floor muscle tone was measured using EMG, exposure to threatening film excerpts resulted in a significant increase in pelvic floor muscle activity relative to neutral film exposure, both in women with [47, 63] and without sexual pain problems [63]. Pelvic floor activity in each of these studies was not only significantly enhanced during sexually threatening film excerpts, but also during anxiety-evoking film clips without sexual content. During a film clip with consensual sexual content, pelvic floor activity was not enhanced. In one of these studies, activity in the shoulder muscles (trapezius muscle) was measured concurrently and was also significantly enhanced during exposure to the anxiety-evoking and sexually threatening film excerpts. This suggests that pelvic floor overactivity in threatening situations should be regarded as part of a general defense mechanism [47]. For women who had been sexually abused in the past, the pattern of activity in the pelvic floor was different than for women without such experiences. For them, pelvic floor muscle activity was highest during the sexually threatening film clip and the film clip with consensual sexual content, whereas the women without such experiences had strongest pelvic floor muscle activity during the anxiety-evoking film clip and lowest levels of pelvic floor activity during the consensual sex clip [63]. Reported emotional experience after film exposure concurred with these findings. Women with negative sexual experiences reported significantly greater feelings of threat and lower levels of sexual arousal during the consensual sex clip than women without such experiences. Apparently, for women with sexual abuse experiences even consensual sexual situations can be experienced as threatening and generate a protective pelvic floor response. In support with these findings, Yehuda, Lehrner, and Rosenbaum have recently suggested that sexual difficulties in individuals with posttraumatic stress disorder (PTSD) occur because the hormonal and neural circuit activation that normally leads to positively valenced sexual arousal and activity is already overactive in PTSD, possibly through reduced anterior cingulated activity, but leads to anxiety, fear, and other PTSD symptoms, such that sexual arousal signals impending threat rather than pleasure [64].

It is therefore highly likely that chronically enhanced pelvic floor activity is more prevalent in women with negative sexual experiences such as rape or incest. A systematic literature search of electronic databases from January 1980 to December 2008 found significant associations between sexual abuse and chronic pelvic pain (OR, 2.73) [12]. When the definition of abuse was restricted to rape, the OR for chronic pelvic pain increased (3.27). A prospective study in 89 women with chronic pelvic pain who were individually assessed by all members of a multidisciplinary team showed that irritable bowel syndrome, pelvic floor overactivity, and physical or sexual abuse were the most common diagnosed etiologies [65]. Another study found the effect between documented childhood victimization and pain in adulthood to be moderated by the presence of PTSD in adulthood, such that only individuals who had experienced childhood abuse/neglect and who had PTSD in adulthood were at significantly increased risk for adult pain [66]. PTSD has been found to be a major mediator in the relationship between the experience of

rape and adverse health outcomes [67] and a direct predictor of sexual problems ([68, 69] and see [64], for a review). Hypothetically, therefore, PTSD (as an anxiety disorder) may manifest itself in an overactive pelvic floor, as part of a generalized protective defense mechanism, which in turn might act as a mediator in the relation between rape and sexual problems [47].

In an attempt to find empirical support for the involvement of PTSD in the relationship between pelvic floor overactivity and sexual function in women, in a recent study 89 young Dutch women aged 18–25 years who had been victimized by rape in adolescence were compared with 114 non-victimized controls with respect to sexual and pelvic floor complaints [70]. The rape victims had been successfully treated for PTSD. Three years posttreatment, the rape victims were still 2.7 times more likely to have pelvic floor dysfunction (symptoms of PVD, general stress, lower urinary tract, and irritable bowel syndrome) and 2.4 times more likely to have a sexual dysfunction (sexual arousal difficulties and sexual pain) than non-victimized controls. The relationship between rape and sexual problems was partially mediated by the presence of pelvic floor problems. These findings suggest that rape negatively affects the sexual arousal response, enhancing the likelihood of sexual pain, and that this effect is greater in women who also have pelvic floor overactivity.

2.8 Pelvic Floor Overactivity and Attachment

Characteristically, women with dyspareunia do not cease sexual activity that is painful for them. They ignore the primary function of pain as signaling damage to the body [4]. While intercourse frequency of women with dyspareunia is lower than that of women without sexual pain [71], not engaging in sexual intercourse is, by definition, not a behavioral choice that women with dyspareunia make [72, 73]. The wish to be "normal" seems to be an important underlying mechanism [74]. In heterosexual partnered sex, many women forego their own needs for fear of the negative impact this might have on the male partner's ego [75]. A very recent study found that women with dyspareunia exhibited more mate-guarding and duty/pressure motives for engaging in intercourse and had more maladaptive penetration-related beliefs than women without sexual pain. The factor that best predicted continuation of painful intercourse (attempts) was the partner's negative response to pain [76]. Many women with vaginismus, in contrast, avoid any form of vaginal penetration because of negative cognitions and expectations about vaginal penetration. As a consequence, anxiety-inducing penetration-related thoughts cannot be disconfirmed, and thereby maintain the condition [77–79].

In recent years, attachment processes and attachment styles are increasingly acknowledged as important determinants of sexual problems in intimate relationships [80, 81]. Secure attachment, associated with positive beliefs about oneself of being worthy of love, follows from repeated interactions with an available attachment figure that is reliable and supportive. However, when the attachment figure is unavailable and unresponsive, negative beliefs about the self or the other develop, which is characteristic of insecure attachment [82]. From an attachment perspective, securely attached individuals' beliefs about self and others and their effective emotion regulation strategies allow them to approach sexuality in a relaxed state of mind such that they can enjoy sex for the pleasures involved (for an excellent overview of the interplay between sex and attachment see [82]). In contrast, insecurely attached individuals may use sex to fulfill their attachment needs, leading to sexual experiences tarnished with anxiety, making it difficult to relax and enjoy sex [81]. There is growing evidence that attachment style is related to sexual pain. For instance, Granot and colleagues found that women with dyspareunia were more likely to be insecurely attached [83].

Given that attachment styles develop in interaction with primary caregivers early in life, we hypothesized that individuals who are insecurely attached will show evidence of higher levels of pelvic floor overactivity at a young age. Such elevated levels of pelvic floor overactivity would

reflect a general defensive mechanism in order to cope with the anxiety and stress that comes with insecure attachment. In a pilot study in 49 patients visiting our outpatient sexology clinic with pelvic floor overactivity and dyspareunia, as well as in an age-matched control group of 49 women without sexual and pelvic floor complaints, we assessed the existence of attachment problems using the *Experiences in Close Relationships Revised* [84]. Based on their report of the presence of at least three symptoms indicative of pelvic floor overactivity, the clinical group was divided in a group of women with *primary* pelvic floor overactivity (pelvic floor overactivity present before sexual debut, and painful sexual intercourse as of the first coital experiences; $N=10$) and *secondary* pelvic floor overactivity (pelvic floor overactivity that developed in the years after first sexual intercourse, with dyspareunia developing gradually, most likely as a result of engaging in sexual intercourse without sufficient sexual arousal and genital swelling; $N=39$) [85]. Examples of such early symptoms of pelvic floor overactivity are frequent micturition, abdominal pain, vulvar burning, and defecation problems such as frequent constipation or a pattern of constipation alternating with diarrhea. As predicted, more women with primary pelvic floor overactivity were insecurely attached (70 %) than the women with secondary pelvic floor overactivity (46 %) and non-symptomatic control women (37 %). Even though this was a small study that requires replication, these data are in line with our view of pelvic floor overactivity as an emotionalresponse, resulting from chronic activation of the defensive stress-system.

2.9 Other Sexual Problems Comorbid with Pelvic Floor Overactivity

Patients with complaints associated with pelvic floor overactivity often have other stress-related complaints, particularly in the neck/shoulder area, possibly related to (tension) headache [61]. Women with dyspareunia suffer from tension headache more often than women without sexual pain [86]. Sexual arousal headaches may be conceptualized a specific type of tension headache. The most prevalent type of sexual headache is orgasmic headache, which may be related to a temporary increase of intracranial pressure during sexual activity [87]. Orgasm pain in women can be related to pelvic floor overactivity as well. Van Lunsen and Ramakers depict painful orgasm as being related to the involuntary clonic pelvic floor contractions associated with orgasm, which become painful in women with a chronically overactive pelvic floor [61].

Often, women do not have arousal problems or orgasm problems with masturbation, but they become only moderately sexually aroused as of their first coital attempts. A strong focus on sexual intercourse as the goal of any sexual interaction may be a major disadvantage in a woman's ability to gain sexual rewards (sexual pleasure and orgasm) [88]. Pain during intercourse is frequently associated with a limited noncoital sexual repertoire, adding to the likelihood of sexual arousal being insufficient for pain-free intercourse [76]. Sexual pain problems in the general population are more prevalent in women than in men, and we hypothesize that this may be partly explained by differences in genital anatomy and gender differences in sexual behavior. With sexual intercourse probably being considered the most important type of sexual activity, the goal of heterosexual interactions, in most if not all cultures, it is important to realize that for women, in contrast to men, sexual intercourse is not the most sexually stimulating sexual activity, particularly when intercourse represents the sole source of sexual stimulation. Research clearly and consistently shows that vaginal intercourse without additional glans clitoris stimulation results in orgasm in only about 25–30 % of heterosexual women [89, 90]. This contrasts sharply with research suggesting that over 90 % of heterosexual men always experience orgasm during sexual intercourse [91, 92]. Unfortunately, women's genital anatomy allows for vaginal intercourse without sexual arousal, whereas for men, sexual arousal (producing an erection) is necessary for penetration. Many heterosexual women appear to prioritize their partner's sexual pleasure over their own [74, 75], further reducing the likelihood that

sexual intercourse takes place with sufficient sexual arousal. In many instances, vaginal intercourse without sexual arousal is painful, particularly with enhanced pelvic floor muscle activity, a protective response resulting from earlier painful sexual experiences, or from physical or psychological stressors that were present even before sexual debut.

Anticipation of painful intercourse, painful orgasm, or headache during and after sexual activity may seriously impede sexual arousal, which in itself reduces the likelihood of experiencing orgasm [43]. Even though reduced sexual desire is still conceptualized by many as a biological deficit, as a problem of sexual "drive," incentive-motivation models of sexual desire propose that sexual desire is the consequence rather than the cause of rewarding sexual experiences [93, 94]. It is highly unlikely that the prospect of painful sex, whatever its source or nature, will evoke much sexual desire.

2.10 Pelvic Floor Overactivity and Persistent Genital Arousal Disorder

Persistent genital arousal disorder (PGAD), first described by Leiblum and colleagues as persistent sexual arousal syndrome (PSAS), is a condition characterized by seemingly spontaneous and frequent or persistent sensations of genital "arousal" in the absence of sexual desire or stimulation [95, 96]. These sensations typically do not fully remit with orgasm and are by definition intrusive, unwanted, and distressing. PGAD has been associated with other conditions such as LUTS, overactive bladder, a history of sexual abuse, and restless leg syndrome. The latter association has led Waldinger to renaming the syndrome Restless Genital Syndrome (ReGS) [97]. Because only a minority of the patients with PGAD seem to have (a history of) restless leg syndrome, we prefer the acronym PGAD. In the literature, many possible etiologies of the syndrome are proposed, but in an excellent review, Facelle et al. concluded that causes remain controversial and the syndrome is multifactorial at least [98]. Clinically, women with PGAD report that stress worsens the genital symptoms, whereas distraction and relaxation strategies reduce symptoms [99].

We hypothesize that in many women with PGAD, overactivity of the anterior part of the pelvic floor is responsible for the symptoms of engorgement, throbbing, tingling, and painful contractions that, according to Leiblum and colleagues, are most prevalent in this condition [96]. These symptoms might be the result of vasocongestion and pudendal entrapment resulting from compression of veins and nerves by constricted pelvic floor muscles. In this view, the bothersome vasocongestion may not be caused by increased arterial inflow as in sexual genital arousal, but by obstructed venous outflow. Consequently, we feel that yet another name for this syndrome would be even more appropriate: persistent genital vasocongestion disorder (PGVD).

This hypothesis of PGAD as obstructed venous outflow is substantiated by case histories of women who had PGAD symptoms following entrapment/compression resulting from local tumors [98]. Likewise, Rosenbaum described a case of a pregnant woman whose PGAD symptoms occurred during pregnancy and disappeared as a result of pelvic floor relaxation by physical therapy [100]. In our clinical experience, most women with PGAD benefit from the same multifaceted treatment regimen as offered to other women with pelvic floor overactivity. This treatment includes extensive psycho-education, psychotherapy, and pelvic floor physical therapy aimed at pelvic floor relaxation in daily life.

2.11 Conclusion

We conclude that despite findings showing that involuntary and rhythmic smooth muscle pelvic floor contractions contribute to the peak sensation of pleasure during orgasm, sexual arousal and orgasmic pleasure are not enhanced by high tonus of the voluntary, striated muscles of the pelvic floor. Contrary to the promising findings of early studies and contrary to common opinion, women who do not have a low-tone pelvic floor and who seek to enhance sexual arousal and more

frequent orgasms have not much to gain from pelvic floor muscle training. Findings in women with pelvic floor dysfunction seem to support the conclusion that a relaxed or low-tone pelvic floor is associated with better sexual function. A relaxed pelvic floor and mindful attention to sexual stimuli and bodily sensations seem a more effective means of enhancing sexual arousal and orgasm.

Pelvic floor overactivity, which we conceptualize as an emotional response resulting from chronic activation of the defensive stress-system by trauma such as sexual abuse or insecure attachment, is associated with impaired sexual arousal, desire, and orgasm. Findings from psychophysiological studies suggest that pelvic floor muscles may involuntarily contract as part of a defensive response, and that increased pelvic muscle activity may be associated with reduced blood flow to the vagina. Also, sexual difficulties in individuals with PTSD may occur because sexual stimuli signal impending threat rather than pleasure.

As the above overview shows, evidence is emerging that pelvic floor overactivity may explain comorbidity of sexual problems in women. Given the complex etiologies of pelvic floor overactivity, referral to a pelvic floor physiotherapist for coordination of pelvic floor contraction and relaxation should not be done before underlying emotional/psychological and/or behavioral causes of the pelvic floor overactivity are identified and addressed. After all, if factors that maintain pelvic floor overactivity as a protective response are not addressed and treated, pelvic floor relaxation will prove to be difficult, if not impossible.

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The Pelvic Floor and Male Sexual Function

3

Deborah S. Cohen, Joshua Gonzalez, and Irwin Goldstein

3.1 Introduction

Male sexual dysfunctions, such as erectile dysfunction (ED), premature ejaculation (PE), and orgasmic dysfunction, especially painful ejaculation, are highly prevalent complaints among adult men of various ages [1–3]. Male sexual function is a vital part of overall male health and wellness, and as such, male sexual dysfunction has been found to disrupt quality of life, negatively impacts interpersonal relationships, and is regarded as a herald of other health problems [4].

Pelvic floor dysfunction (PFD) in men includes alterations in activity levels of the male pelvic floor muscles (PFMs) during daily activities and/or at rest, improper muscle response and coordination for activities of urination and/or elimination, shortened endopelvic fascial tissue, and painful myofascial tender points or trigger points in the pelvic floor. PFD in men is highly associated with male sexual dysfunctions such as ED, PE, and ejaculatory pain [5–9].

Chronic pelvic pain syndrome (CPPS) is often associated with a diagnosis of chronic prostatitis (CP), and refers to a persistent situation for more than 3–6 months in which there is pain associated with urination, defecation, or pain that is simply present anywhere in the male pelvis. CP can be intermittent and occur with sitting, standing, certain daily activities, sexual activity, or can remain constant. There is a strong correlation between CP or chronic pelvic pain and male sexual dysfunctions such as ED and PE [5, 10–14]. As well, there is a high rate of pain associated with sexual activity among men with CP/CPPS, in particular pain prior to, during, or following ejaculation [5].

The realms of male sexual dysfunction, male PFD, and male pelvic pain overlap in ways that can be described as shown in Fig. 3.1. In this chapter, the role of the male pelvic floor in men without and with sexual dysfunction is described, and the current state of knowledge regarding best practices of treatment of specific sexual dysfunctions is reviewed with respect to the pelvic floor.

3.2 Anatomy and Physiology of the Male Pelvic Floor

Until recently, the terminology used to describe the anatomy and function of the male pelvic floor has varied across health care disciplines. In an attempt

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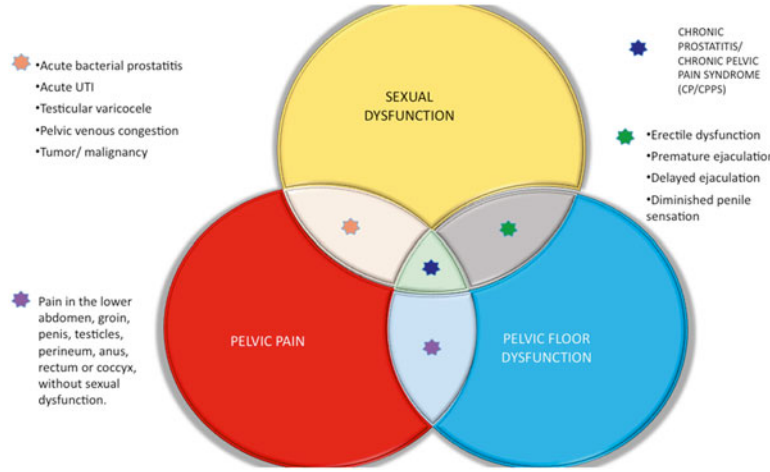

Fig. 3.1 Associations between commonly observed elements of male sexual dysfunction, pelvic floor dysfunction, and pelvic pain

to standardize terminology, The International Continence Society has proposed the following definitions: the pelvic floor is a compound structure that encloses the bony pelvic outlet, while the term PFMs refers to the muscular layer of the pelvic floor [15]. These terms are henceforth used accordingly.

The male pelvic floor consists of several tissue layers, but there has been some nomenclature inconsistency in the literature. Varied names and boundaries have been utilized to describe PFMs and divisions between the muscle layers, a problem that may complicate health care provider communication. Stoker has suggested that the pelvic floor should be thought of as four principal layers: the endopelvic fascia, muscular diaphragm, perineal (or urogenital) diaphragm, and superficial transverse perinei [16]. DeLancey has recognized the peritoneum as the most cranial part of the pelvic floor and the skin of vulva, scrotum, and perineum as the most caudal [17]. Some health care providers believe the puborectalis should be regarded as a component of the external anal sphincter versus the more widely accepted notion that it is a component of the levator ani muscle group [18]. In the end, however, it is well appreciated that male pelvic floor function relies on complex and dynamic relationships between muscles, fascia, ligaments, bone, nerves, and vascular supply and that in men, as it is similarly found in women, these multiple pelvic structures play a crucial role in normal urinary, bowel, and sexual function.

The anatomy and function of the pelvic floor can be best understood when its relationship to the surrounding bony architecture is considered. The bony pelvis is a ring structure composed of the sacrum, and right and left innominate bones. Each innominate bone consists of three parts: the ilium, ischium, and pubis. The pelvic cavity is divided into the false (greater) and true (lesser) pelvis by the pelvic brim [19]. The pelvic brim (or inlet) extends from the promontory of the sacrum, along the arcuate line of the ilium, pectineal line and pubic crest. The coccyx is also part of the bony pelvis, and consists in most people of five coccygeal bones, which articulate with one another and with the sacrum.

The male PFMs attach to the interior of the pelvic ring and thereby serve to stabilize its joints when the muscles are active. The male PFMs extend from the dorsal aspect of the pubic symphysis and pubic rami anteriorly to the coccyx posteriorly. They attach laterally to the ischial tuberosities, interior surfaces of the illia, and to the arcus tendineus levator ani (ATLA)—a tendinous arc that extends from the posterior aspect of the pubic arch anteriorly to the ischial spine posteriorly, in the right and left sides of the pelvis. The male PFMs also function to support the internal organs of the abdomen and pelvis and promote voluntary closure of the urethral and anal sphincters in men [20]. In women PFMs also provide tone to the vaginal vault.

In order to provide optimal mechanical advantage for their functions, the male PFMs are arranged in a dome-shaped sheet that contains a complex network of mostly striated muscle, which provide cover for the entire pelvic cavity [18, 21]. The varied functions of the male pelvic floor are achieved via coordinated activity that involves the full range of contraction, relaxation, and active lengthening, or stretch, of their fibers.

PFMs are often subdivided into superficial and deeper components, each with particular functions [19]. The convention adopted by pelvic rehabilitation practitioners is to describe the PFMs in terms of three layers, progressing from superficial (caudal) to deep (cranial) within the pelvic floor [22]. For the remainder of the description of pelvic floor anatomy, this convention will be adopted.

The structure of the male pelvic floor is shown in Fig. 3.2. The first, most superficial layer of muscle, or the superficial perineal pouch in men, consists of the bulbocavernosus, (the bulbospongiosus in women), the ischiocavernosus, the superficial transverse perineal, and the external anal sphincter. These superficial PFMs function in men to provide urethral and anal closure and maintain continence, as well as to expel the urethral contents. The superficial layer also plays a significant role in penile tumescence and rigidity during erection, as will be discussed later.

The second layer of the PFMs, or the urogenital diaphragm, consists of the deep transverse perineal, the sphincter urethrae, and the compressor urethrae. This second layer of PFMs adds further support to urethral closure during increased intra-abdominal pressure. This second layer has fascial connections into the deep abdominal musculature, and thereby also plays a role in stabilization of the pelvic and lower lumbar joints during movement.

The third most cranial layer of the male PFMs, called the pelvic diaphragm, extends from the dorsal aspect of the pubic symphysis to the coccyx and from the interior surface of one ilium to the other [19]. The pelvic diaphragm consists of the pubococcygeus—which is comprised of the puboureth ralis (pubovaginalis in women), the puborectalis, and the iliococcygeus. The pubococcygeus and iliococcygeus collectively are termed the levator ani, as contraction of this deep layer of PFMs serves to elevate the anal sphincter. This deepest layer of the PFMs is most responsible, along with the peritoneal fascia, for support of the pelvic organs. The tonic activity of

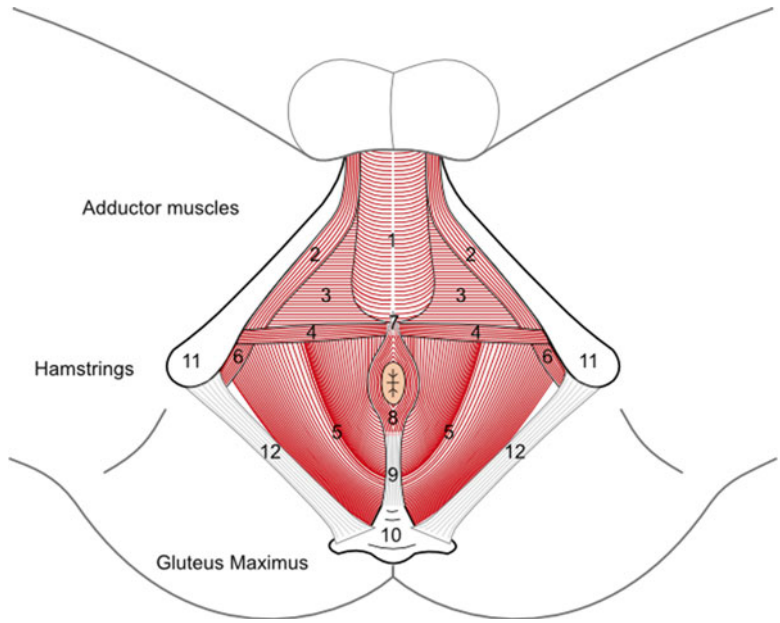

Fig. 3.2 The male pelvic floor, lithotomy view. (1) bulbospongiosus m. (2) ischiocavernosus m. (3) deep transverse perineal m. (4) superficial transverse perineal m. (5) pubococcygeus m. (6) obturator internus m. (7) perineal body (8) external anal sphincter (9) anococcygeal ligament (10) coccyx (11) ischial tuberosities (12) sacrotuberous ligament. *With permission courtesy of Jeff Gibson,*www.malepelvicfloor.com

the pelvic diaphragm prevents the supportive ligaments of the pelvic organs from becoming over-stretched by constant tension [23]. The tonic activity, combined with the minimal elasticity of the endopelvic fascia, gives the pelvic diaphragm its characteristic dome shape. In the presence of pathology, such as low-tone PFD or ligamentous laxity, the pelvic diaphragm appears more basin shaped versus the usual dome shape [16].

The puborectalis is also responsible for controlling the anorectal angle, and thereby maintaining anal continence when it is contracted, and allowing for evacuation of the bowels when relaxed. The deepest layer of the pelvic floor also consists of the ischiococcygeus, or the coccygeus muscle. The coccygeus is not strictly considered to be part of the PFMs, as it does not share it its functions of continence, organ support, or sexual function. Contraction of the coccygeus muscle deviates the coccyx to the ipsilateral side.

As previously mentioned, the relationship between the puborectalis and levator ani muscles remains debatable. Traditionally it has been thought that the puborectalis constitutes a major component to the levator complex. Using MRI, Stoker demonstrated that the puborectalis contributes to both the levator ani and external anal sphincter [16]. However, recent developmental and histological evidence indicate otherwise, suggesting it may be more closely associated with the external anal sphincter [18]. On the other hand, the external anal sphincter is innervated by the inferior rectal branch of the pudendal nerve, whereas the puborectalis and the cranial aspect of the remainder of the levator ani are innervated by the nerve to the levator ani directly [24]. The disparate innervation may imply evolutionary difference between the puborectalis and the levator ani muscles.

The urogenital diaphragm is another component of the male pelvic floor whose structure is debated in the literature. Sometimes referred to as the triangular ligament or the perineal membrane, the urogenital diaphragm occupies the area between the pubic symphysis and ischial tuberosities, lying external and inferior to the pelvic diaphragm [19]. The urogenital diaphragm is composed of a strong muscular membrane that separates the superficial perineal pouch from the upper pelvis and does not constitute a true diaphragm. Historically, the urogenital diaphragm has been described as a tri-layer structure made up of the deep transverse perinei with a superior and inferior fascia [16]. However, recent work suggests that the presence of the deep transverse perinei and superior fascial layer are questionable and that the urogenital diaphragm may represent a single musculofascial layer [25].

Finally, pelvic innervation is fundamental to normal male sexual, urinary, and bowel functions. The muscles of the pelvic floor are innervated by sympathetic, parasympathetic, and somatic nerve fibers. The three types of nerve fibers allow for careful regulation of PFMs including those responsible for erection, emission, ejaculation, urination, and defecation. The hypogastric, pudendal, and levator ani nerves all participate in these sexual, urinary, and bowel functions. Coordinated contraction of the bulbospongiosus muscle is carried out by input from the pudendal nerve (from spinal nerves S2–4), which is necessary for emission and ejaculation. Emission is mediated through the sympathetic nervous system via the hypogastric nerve innervated by preganglionic neurons in the intermediolateral and medial gray nuclei [26, 27]. There is evidence from animal models that contractions of the bulbospongiosus and ischiocavernosus muscles are important for expulsion of seminal fluids and for increased hardness/engorgement of the glans penis [28]. Another study found that contractions of the levator ani in rats act in coordination with the bulbospongiosus to augment penile erectile hardness and that muscle activity is tightly coordinated through somatic innervation during copulation [29]. The role of the PFMs in erection and ejaculation in humans will be discussed further in the remainder of this chapter.

While there still remains discrepancy in terminology and controversy about exact anatomic detail, this review highlights the complex network at play within the male pelvis. A comprehensive understanding of pelvic floor anatomy and physiology is crucial to appreciate the intricacies of normal male sexual, urinary, and bowel function.

3.3 The Pelvic Floor in Male Erectile Function

Normal erectile function includes the ability to obtain an erection sufficiently rigid for vaginal penetration, and ability to sustain this erection long enough to complete sexual intercourse. ED is said to be present when there is a consistent inability to either obtain and/or maintain an erection sufficient for completion of sexual intercourse [30]. Estimates of prevalence of ED range in various countries from 9 to 40 % of men by age 40, and generally increase by 10 % in each decade of life thereafter [3, 31].

The observation that there is a voluntary skeletal muscular component to erectile function and that contractions of the superficial PFMs, in particular, the bulbocavernosus (BC) and ischiocavernosus (IC), are necessary for full penile rigidity, is over a century old [32]. The 1909 edition of Gray's Anatomy referred to the IC as the "erector penis" [33]. Since that time, human and animal models have demonstrated that these two muscles are activated during sexual activity, especially thrusting. It is now understood that there exists a vascular phase and a muscular phase to the formation of a full erection, which correspond to glans tumescence and supraphysiologic erectile rigidity [34].

Contractions of the ischiocavernosus muscles participate in the process of erection by inducing suprasystolic intracavernosal pressures [35, 36] and reducing venous return [7]. The IC also stabilizes the erect penis [37]. Contraction of the BC contributes to engorgement of the glans penis and corpus spongiosum [37], causes increased intraspongiosal pressure [38], and slows venous drainage of blood from the corpora cavernosum, by compressing the deep dorsal vein of the penis [20, 38]. Contraction of the BC and IC can improve erection by increasing maximum inflow pressure, as well as likely compensate for veno-occlusive dysfunction. The degree to which these muscles in the most superficial pelvic floor can participate in penile erection depends on the functional strength and coordination of these muscles. Indeed, voluntary pelvic floor activation has been shown to be more efficient in men who have full erectile function than in those with ED [7–9]. As well, efficiency of maximal pelvic floor contraction was negatively correlated with age in a group of impotent men [8].

A recent study suggested that in order to provide resistance for the IC muscle to increase its strength, a rehabilitation program should include voluntary contractions of this muscle during a state of erection [36]. In this way, the intracavernosal pressure in the corpus cavernosum provides the needed resistance to challenge the IC and induce a training effect. This study also indicated that application of electrical stimulation simultaneously with voluntary contraction may also improve the strength and efficiency of IC contractions. Vibration was applied to stimulate erection, in order to activate the IC muscle reflex contraction via stimulation of mechanoreceptors in the glans.

Rehabilitation of PFM function has long been suggested as an important component of treatment for ED [39–41], and has been found to be effective at improving erectile function [9, 34, 36, 42, 43]. PFM exercise appears to be especially beneficial in men with mild or moderate veno-occlusive dysfunction [34, 44, 45]. Claes noted that in younger fully potent men only a single systolic injection of blood is needed to achieve both tumescence and rigidity. In contrast, he hypothesized that in the group of men who have penile corporal fibrosis and/or reduced relaxation of the corporal smooth muscle and therefore corporal veno-occlusive dysfunction, voluntary contractions of the IC muscle may provide the needed increase in intracavernosal pressure to establish closed hydraulic system needed to maintain penile erection.

Abnormally high pelvic floor tone has been suggested as a cause of ED. Spasm of the PFMs can provide extrinsic compression that impairs pudendal arterial inflow [5, 6]. This is the basis for the current understanding of the high prevalence of ED among individuals with CPPS, as will be discussed later.

3.4 The Pelvic Floor in Ejaculatory Function

The mechanics of ejaculation reflect a muscular event that occurs simultaneously with involuntary contraction of the prostate gland, involuntary closure of the bladder neck, and involuntary relaxation of the urethral sphincter muscles. Just as voluntary contraction of the BC functions to empty the distal urethra at the end of micturition, involuntary contraction of the BC muscle expels contents from the urethra during ejaculation [37, 43]. Shafik also demonstrated that rhythmic contractions of the external urethral sphincter during ejaculation may act as a "suction–ejection pump," sucking the seminal fluid into the posterior urethra while relaxed and ejecting it into the bulbous urethra upon contraction [46].

Strong BC muscle contractions will increase maximal engorgement of the corpus spongiosum, increase urethral pressure and facilitate ejaculation of prostatic and seminal vesicle fluid. Strong BC contraction may also enhance and intensify orgasmic pleasure during ejaculation. PFM training may therefore optimize ejaculatory volume, force, and intensity of sexual climax [37].

The International Society of Sexual Medicine has defined PE as "ejaculation within a minute" [47]. More broadly, PE can be said to include ejaculation beyond the man's control, sooner than he would like, for his own and his partner's sexual satisfaction. PE is the most common male sexual dysfunction [1, 2], and it negatively affects the enjoyment of sexual activity for many men and their partners. This condition also impacts negatively on the self-image and sex lives of many men and adversely affects their relationships with their partners [48]. In a large multinational survey, the prevalence of PE was found to be 23 % overall among participants from the USA, Germany, and Italy [1].

The exact mechanism controlling the ejaculatory reflex, whether by contraction or relaxation of the BC and IC muscles, is not well defined [20], though Pastore suggested that active perineal muscle control could inhibit the ejaculation reflex through intentional relaxation of the bulbo- and ischiocavernosus muscles, which are active during arousal [49].

Several therapies are available to treat PE, with mixed results. Behavioral therapies include precoital masturbation, increasing sexual activity frequency, and manual or physical maneuvers intended to delay ejaculation. The squeeze technique described by Masters and Johnson [50] makes use of the bulbocavernosus reflex, in which sustained pressure is applied to the glans penis causing contraction of the BC muscle and, as a result, diminished ejaculatory urgency. Stopping the movement of intercourse and performing a sustained PFM contraction can also function to defer the urgency of ejaculation, serving as an "internal squeeze" without manual pressure [37].

Pharmacologic strategies include precoital use of topical anesthetic creams to the penile shaft, precoital use of selective serotonin reuptake inhibitors (SSRI), precoital use of opioids such as tramadol, and precoital use of phosphodiesterase type 5 inhibitors [47].

Pelvic floor rehabilitation has been shown to improve control with delaying ejaculation and allowed significant increases in intravaginal ejaculatory latency times [51, 52] in men with PFD [53]. Pelvic floor strategies have also proven to be a viable alternative to use of the SSRI dapoxetine [49]. Most of these studies were conducted with patients who had seen little or no benefit from other interventions.

The application of pelvic floor rehabilitation to the treatment of PE is beginning to be established as a principle, although studies have not yet clarified the features of a specific treatment protocol. Whether emphasis should be on strength, control, or relaxation is not yet well understood but, as is the case with any pelvic dysfunction, treatment should be tailored to individual findings.

3.5 Chronic Prostatitis/Chronic Pelvic Pain Syndrome

3.5.1 Epidemiology

Prostatitis is a common and often debilitating condition that affects millions of men worldwide. The National Institutes of Health Consensus Committee has divided prostatitis into categories

I, II, IIIA, and IIIB. Categories III indicate CP. Category IIIA indicates that leukocytes are present in expressed prostatic fluid, and Category IIIB indicates that these markers are absent. This condition is often called chronic prostatitis/chronic pelvic pain syndrome (CP/CPPS). CP/CPPS is characterized by pain in the pelvis, abdomen, or genitals, and lower urinary tract symptoms of an obstructive or irritative nature, without evidence of recurrent urinary tract infection [54, 55]. Discomfort or pain accompanying ejaculation or afterward are common, as are concurrent sexual dysfunctions, especially ED and PE [5, 10, 11, 14]. CP/CPPS has a prevalence rate of 2–16 % for men in various populations in different regions of the world [11, 56, 57].

CP/CPPS is strongly associated with both sexual dysfunction and PFD and can significantly impact quality of life and relationships of men with this condition [58]. Emphasizing the health status impact of this condition, Wenninger et al. used the Sickness Impact Profile, a generic health status measure, in patients with CP and demonstrated that the mean scores were within the range of scores reported in the literature for patients suffering from other illnesses, such as myocardial infarction, angina, or Crohn's disease [59].

3.5.2 CP/CPPS and Sexual Dysfunction

The most common sexual dysfunctions reported in men with chronic pelvic pain are ED, ejaculatory pain, and PE [5]. Aubin et al. found that men with CP/CPPS have higher rates of sexual dysfunction—lower desire, diminished erectile and orgasmic function, and more frequent pain associated with orgasm and/or ejaculation—than men without pelvic pain [10]. In this study, some of these variables were also related to demographic factors such as age and marital status. However, erectile function as measured by the Brief Sexual Functioning Questionnaire (BSFQ) varied inversely with pain status, independent of these demographic factors. CP/CPPS may also affect female partners who are in relationships with those men who are afflicted. In one case–control study, when compared with controls, men with CP/CPPS had greater rates of sexual dysfunction and depression [11]. When these men and their partners were compared with control couples, the sexual function of men with CP/CPPS was also significantly associated with that of their female partners, who reported higher rates of dyspareunia.

3.5.2.1 CP/CPPSand Erectile Dysfunction

ED is especially common in men with CP/CPPS. Prevalence rates of ED in men with CP/CPPS have been reported in the literature to range from 15 to 40.5 % [5]. The prevalence of ED in a survey of Finnish men with chronic pelvic pain was 43 %, and decreased libido was 24 % [12]. In a survey of 296 Malaysian men with CP/CPPS, 72 % reported sexual dysfunction, which was defined as ED and/or ejaculatory difficulty [13]. The presence of sexual dysfunction in participants of this study was also correlated with greater symptom severity and worse quality of life. A 2002 cross-sectional survey conducted in Singapore also reported that men with CP/CPPS had worse erectile function as measured with the IIEF assessment tool and worse quality of life than men without prostatitis [14].

The higher-than-normal prevalence of ED among individuals with CP/CPPS may be explained by changes in peripheral arterial function in this group. A case–control study involving men with CP/CPPS demonstrated that this group was more likely to have evidence of arterial stiffness associated with nitric oxide-mediated vascular endothelial dysfunction compared to asymptomatic controls [54]. The authors suggest that this may be related to increased autonomic vascular tone associated with pain-induced chronic stress. In addition, they note that vascular endothelial dysfunction may be a contributing factor to the pathophysiology of the chronic muscle spasm and pain experienced by men with chronic pelvic pain. A subgroup of the cases in this study were treated with pelvic floor physical therapy and quercetin supplementation, and 3 out of 4 of these demonstrated improved systemic peripheral arterial function, as well as reduction of their CP/CPPS symptoms [60]. Another possible explanation for the high rate of ED among men with chronic pelvic pain is related to the

presence of abnormally high resting PFM tone, a hallmark of CPPS. As mentioned earlier, elevated pelvic floor tone is thought to be a possible impediment to normal erectile function. One likely mechanism for this association is the potential obstruction of arterial inflow to the penis by extrinsic compression from surrounding musculature [5].

3.5.2.2 CP/CPPS and Premature Ejaculation

PE is another sexual dysfunction commonly found in men with chronic pelvic pain. In one study, the prevalence of sexual dysfunction, which included ED and/or PE, was higher in men with CP/CPPS (49 %) than in the general population, and was negatively correlated with age and duration of CP [61]. A study of Turkish men also showed a significantly higher rate of PE (77.5 %) among a group of men with CP compared with controls (10 %) [62].

One study found a high rate of signs of prostatic inflammation (56.5 %) and chronic prostatic infection (47.8 %) among a group of men with PE [63]. These authors note that the impairment of sensory feedback occurring immediately before orgasm is widely considered the pathogenetic mechanism of PE [64]. Based on their findings, they postulate that the presence of prostatic inflammation may alter sensation and thus the ejaculatory reflex.

Since CP/CPPS is a noninfectious condition, and more recent research has shown that the presence of inflammatory markers in prostatic secretions is not a reliable predictor of or diagnostic tool for CP/CPPS [65], we can adapt this explanation to reflect the musculoskeletal component of PE in the presence of CP/CPPS. That is, PFD can be the pathophysiologic driver of PE, as the presence of muscle spasm impairs the normal sensory feedback mechanism just as prostatic inflammation can.

3.5.2.3 CP/CPPS and Ejaculatory Pain

Ejaculatory pain is a common complaint of men with CP/CPPS. The cause of this association has been the subject of speculation, study, and debate over the past decade in particular. The NIH CP cohort study surveyed 488 men with respect to their prostatitis symptoms [66]. A post hoc analysis of the data from the NIH CP cohort study found that 74 % of the 486 men surveyed may have had ejaculatory pain at least intermittently during the first 3 months of the study [65]. In this follow-up study, the participants were stratified into four categories according to the frequency of their ejaculatory pain. The negative impact of CP/CPPS on individuals increased with the frequency of their ejaculatory pain, and their mental and physical quality of life decreased. Among the four groups, there was no difference in semen bacterial culture or leukocyte count, both markers of inflammation. These authors suggest, therefore, that if ejaculatory pain is due to a specific mechanism, then a likely possibility is that a neuromuscular cause may be at work, such as muscle spasm instigated by the involuntary muscular contractions of emission. In another study of 146 men with CP/CPPS and established PFM spasm, 56 % had painful ejaculation [67]. Others have shown that men with CP/CPPS have significantly more tenderness, muscle spasm, and dysfunction throughout the abdomen and pelvis [68–70].

Pain associated with ejaculation in men with CPPS has been attributed by some to pathology arising from the prostate gland, while others believe it represents a myofascial pain syndrome. Current understanding of chronic painful states such as this suggests that it is neither trivial nor necessary to determine which of these two instigates the pain, as both systems will need to be addressed in a successful treatment model [71]. On the one hand, many chronic pain states depend on feedback cycles from myofascial trigger points and their zones of referred pain, as skeletal muscles easily develop habits of contractedness that can limit movement, impair circulation, and result in chronic pain [72]. As well, visceral and myofascial pain share characteristics of being diffuse and poorly localized. In both cases pain conditions are often chronic, leading to lowering of nociceptive thresholds, and the enlargement of receptive fields of afferent neurons in the dorsal horn of the spinal cord. Pain can be referred from the pelvic viscera such as the prostate gland, to the pelvic and abdominal muscles, or from myofascial structures back to the same viscera. Pain generated as a result of pathology in either system

can mimic that of the other. Distinguishing the two can become impossible for the patient, due to convergence of visceral and somatic afferent activity on the same spinal neuron in the dorsal horn. The pain syndrome can also persist long after any initial precipitating event, making clinical diagnosis more challenging [71]. This understanding of the interaction of somatic and visceral systems should inform our treatment strategies, which should aim to decrease afferent activity from both systems through a multimodal approach.

3.5.3 Pelvic Floor Dysfunction as a Key Functional Component of Pain and Sexual Dysfunction in CP/CPPS

Data have not been able to support theories that persistent prostatic infection with bacteria, viruses, or yeast are at the root of the cause of CP/CPPS [73, 74]. Furthermore, a large multicenter trial found that symptoms of CP were not associated with prostatic inflammation, casting doubt that inflammation can be identified as the direct cause of pain in this condition [75]. This understanding has motivated medical practitioners to seek other explanations for this challenging condition.

Development of the specialty field of pelvic floor rehabilitation within the practice of physical therapy has led to the observation that harmful alterations in patterns of activity in the PFMs can develop as early as childhood. During this time, boys and girls may both learn a habit of holding their urine or bowel movements, which in turn may predispose these individuals to pelvic floor hypertonicity that persists later in life [20].

The notion that abnormal muscle tone and shortening of the levator ani and external rotators of the hips are involved in the pathophysiology of CP/CPPS has been found in the literature for decades [76]. Indeed, there is significantly more PFM spasm and tension in men with CP/CPPS than in healthy men, with up to 50 % of sufferers showing signs of this musculoskeletal dysfunction [70]. As well, tenderness is found in these individuals with palpation of the pelvic floor, psoas, and adductors [68]. In a study by Zermann et al., 88.3 % of the patients with CPPS had pathological tenderness of the PFM and poor to absent pelvic floor function [77]. These authors proposed that because muscle activity reflects neural control, PFD associated with pelvic pain might indicate that there is a primary or secondary central nervous system disturbance in regulation of the PFMs.

The use of real time ultrasound has been shown to be a useful tool in the assessment of pelvic floor function in men with CPPS [78]. In this study, men with CPPS were seen via transperineal ultrasound imaging to have a more acute anorectal angle during pelvic floor contraction as well as at rest. These more acute anorectal angles were correlated with subjects' greater report of pain, sexual dysfunction, as well as with anxiety. Anatomically, the anorectal angle is made increasingly acute by contraction of the puborectalis muscle. A less acute anorectal angle, which occurs with relaxation of the puborectalis, is the position that lends itself to efficient evacuation of the bowels with a minimum of straining to produce the needed intra-abdominal pressure. Men with CPPS have also been shown via transabdominal ultrasound to have reduced mobility of the PFMs [79].

Effective management of CP/CPPS has been demonstrated in programs that emphasize treatment of PFD. Tension myalgia associated with abnormally high PFM tone is a significant component of pain and dysfunction in men with CP/CPPS. Therefore, neuromuscular reeducation must take place, fostering relaxation of the hypertonic levator ani group [37, 76, 80].

Neuromuscular reeducation, with the guidance of electromyography or other methods of biofeedback, has been shown to result in reduced resting baseline tone of the PFMs. This reduction in muscle activity can also produce a reduction in pain ratings, and overall scores on the NIH-CPSI, a valid and responsive instrument which is widely used in assessment of symptoms of CP/CPPS [80–84].

Manual therapy to release painful myofascial trigger points in the pelvic floor, combined with paradoxical relaxation training, in which the patient learns to relax the pelvic floor by first performing a

volitional contraction of this muscle group, has been shown to improve symptoms of pelvic pain, lower urinary tract symptoms, and sexual dysfunction in these men [67].

More extensive PFM training can also be beneficial in this group of men, as it functions to instill awareness of a more relaxed state of these muscles, as once cycles through repetitions of contraction and relaxation [37]. Care must be taken in this group, however, not to aggravate pain as a result of performing voluntary contraction of already tense and tender muscles. Nonetheless, this approach has been used successfully by many physical therapists in the clinical setting, and has demonstrated effectiveness at reducing either pain or overall NIH-CPSI scores [83].

3.6 Conclusion

The fact that the pelvic floor plays a key role in male sexual function is becoming more widely accepted by the health care community. Treatment of PFD has been shown to affect significant improvements in sexual function, in men with sexual dysfunction and PFD.

A full understanding of the anatomical and physiological functions of the pelvic floor in male sexual function is still being developed. At the same time, there is a growing awareness of and participation in a medical treatment model that employs multidisciplinary diagnosis and treatment of sexual dysfunction, including careful consideration of the musculoskeletal system and, in particular, the pelvic floor.

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Female Genital Pain and Penetration Disorders

4

Ahinoam Lev-Sagie

4.1 Introduction

Female genital pain can result from various causes, including inflammation, dermatoses, and infections. In addition, up to 16 % of the female population is diagnosed with vulvar pain syndromes, also known as vulvodynia, defined as genital pain in the absence of an identifiable cause [1].

Often, there is a significant delay in making the actual diagnosis, with a reported mean time of 2 years and evaluation by up to 15 physicians prior to correct diagnosis [2]. This delay adversely affects patients, with subsequent emotional, sexual, social, and interpersonal impact. Causes for this unfortunate situation are multifactorial as it seems that many gynecologists are not familiar with the variety of disorders that cause female genital pain; secondly, in most cases the gynecological exam is not specifically directed at identifying vulvovaginal disorders; and lastly, specialists treating these disorders are scarce. The result is misdiagnoses (usually, "yeast infections") and delay of appropriate treatment.

The objectives of this chapter are to provide practical tools for caregivers who treat patients with genital pain conditions, to provide guidance for the assessment of patients, to discuss the differential diagnosis of female genital pain, and to present current guidelines regarding the management of two vulvar pain disorders, provoked vestibulodynia and generalized vulvodynia.

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4.2 Assessment of Patients with Genital Pain

Vulvovaginitis and vulvar dermatoses may cause itching, irritation, discharge, pain, and dyspareunia. On exam, erythema, edema, discharge, and tenderness may be noted. These symptoms and signs are nonspecific and may represent an inflammatory response to various causes. Most vulvovaginal disorders can be diagnosed by combining medical history, physical examination, and microscopic evaluation of vaginal discharge. In addition, cultures and PCR (polymerase chain reaction) assays are required for the diagnosis of specific pathogens and biopsies may be needed in cases of vulvar skin involvement or suspected neoplastic lesions.

4.2.1 History Taking

History taking may provide many clues required for an accurate diagnosis. A detailed symptom history is important, including the nature of symptoms, their location, severity, duration, whether they are continuous, intermittent or cyclical, whether provoked by touch or spontaneous,

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and what factors worsen or alleviate the complaints. It is important to clarify if the patient experiences discomfort of the external genitalia, the vestibule, or deep in the vagina, as these symptoms may represent different disorders. The patient may use adjectives such as burning, stinging, swelling, or discomfort rather than pain. It is also important to assess the effects on daily life and functioning. Inquire about treatments she might have tried, how these were used, and what was their effect. If a woman reports difficulties with intercourse, it is important to determine whether the pain is superficial, at the point of penetration (likely related to a vulvar or vestibular problem), or deep inside (deep dyspareunia). Inquire about tightening of the pelvic floor (PF) muscles: this may be apparent as painful penetration (vaginismus), urinary frequency, urgency and hesitancy, constipation, hemorrhoids, and anal fissures. It is also important to take a brief gynecological history including menstruation, fertility, parity, and contraception methods. Ask about the presence or history of skin conditions, allergies, and exposure to potential irritants and/or allergens.

While obtaining the history, be familiar with potential diagnoses and open to various possibilities. Don't be distracted as many patients will mention diagnoses they priorly received or have self-diagnosed using the internet. Finally, look for coexisting conditions and avoid the tendency to lump all symptoms into one disorder. It can be helpful to use questionnaires designed for comprehensive evaluation of vulvovaginal complaints (see references below).

4.2.2 Examination

It is important to remember that vulvodynia is a diagnosis of exclusion. Many inflammatory, infectious, and dermatologic disorders present with provoked vestibular pain and dyspareunia, or with unprovoked pain, and they should be ruled out by a comprehensive examination.

The examination should always include observation, evaluation of vestibular tenderness, vaginal examination with assessment of discharge using microscopy, pH measurement and vaginal cultures, pelvic organ and muscle palpation.

The use of a bright light to examine the vulva, vagina, and cervix is recommended. For the evaluation of the vulva, separate the labia with your fingers to look for any lesions, fissures, redness, or swellings. Gently retract the clitoral hood, exposing the clitoris. Examine the vestibule for epithelial thinning associated with estrogen deficiency or skin disorder.

Vestibular tenderness should be evaluated using the Q-tip test in which gentle pressure is applied to several spots in the vestibule using a Q-tip, while asking the patient to quantify the level of discomfort or pain (Fig. 4.1).

Examination of the vagina with a speculum may disclose a normal appearance of the vaginal

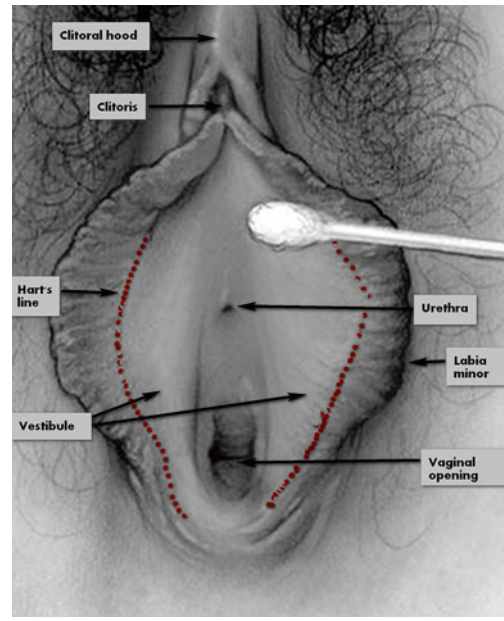

Fig. 4.1 The Q-tip test. The goal of the Q-tip test is to determine if there are areas that exhibit an abnormal pain response in the genital area. Use a cotton-tipped applicator to determine whether pain is provoked by pressure at one or more points. Apply gentle pressure to the following areas: inner thighs, labia major and labia minor, interlabial sulci, clitoris, clitoral hood, and perineum. Sites to be tested within the vestibule can be visualized using a clock face (1–12 o'clock). The anterior vestibular sites [2, 10, 12] are typically assessed first, followed by the posterior sites [5–7]. Apply gentle pressure to each of these sites and ask the patient to rate the pain severity and describe the pain character (burning, raw, etc.)

walls or diffuse redness, erosions, and petechiae, suggesting vaginitis. Notice the presence of discharge, its characteristics (consistency and color), and its origin (vaginal walls or cervix). As the signs on examination are nonspecific, a microscopic examination (wet mount) of vaginal discharge should be performed. Vaginal secretions are placed on microscope slides, and a drop of saline (0.9 % NaCl) and 10 % potassium hydrochloride (KOH) are added to each sample at separate locations. Microscopy can allow for the identification of fungal infection, trichomoniasis, parabasal cells (characterizing vaginal atrophy and estrogen deficiency) and allow for the diagnosis of desquamative inflammatory vaginitis (DIV) (Fig. 4.2).

When appropriate, obtain samples for laboratory studies. These include both cultures for the identification of specific fungal organisms and bacteria, as well as samples for PCR studies, to identify sexually transmitted organisms, including *Neisseria gonorrhoeae*, *Chlamydia trachomatis*, and *Trichomonas vaginalis*.

Next, perform a digital palpation of the levator ani muscles for hypertonicity, tenderness and trigger points, palpation of the urethra, bladder trigone, pudendal nerves, uterus and adnexa. Additional studies, including biopsy, patch tests, or imaging should be performed as needed.

After completion of the history and examination, the provider should be able to diagnose the causative factor such as infectious or noninfectious vaginitis, skin disorders, and estrogen deficiency. After all known causes of genital pain are ruled out, vulvodynia can be diagnosed.

4.3 Causes of Female Genital Pain

Vulvovaginal pain may be caused by a variety of disorders. Pain may be constant or intermittent, provoked by touch or spontaneous, and may be classified in various ways. The most common classification system was developed by the International Society for the Study of Vulvovaginal Disease (ISSVD) [3]. It divides possible causes of vulvar pain into four categories: infectious, inflammatory, neoplastic, and neurologic [3]. In addition, it defines the term vulvodynia as "vulvar discomfort, most often described as burning pain, occurring in the absence of relevant and visible findings, or specific, clinically identifiable, neurologic disorder." This classification distinguishes between generalized and localized pain, and each of these subgroups is further subdivided into provoked, unprovoked, or mixed (continuous pain, exacerbated by touch). The term vulvodynia is reserved for these cases of vulvar pain occurring in the absence of physical findings [4] and therefore can be used only after specific, recognized disorders are ruled out (Table 4.1).

In the presence of inflammatory conditions, pain and dyspareunia may result from friction and damage of irritated tissues, tears, fissures, and irritation of sensitized and inflamed nerve fibers. It is beyond the scope of this chapter to review in detail the various vulvovaginal disorders. Therefore the discussion will be limited only to common conditions that may cause pain and must be ruled out prior to establishing a diagnosis of vulvodynia.

Yeast Infection is a common cause for vulvovaginitis and 80–90 % of cases are caused by *Candida albicans* [5]. Symptoms vary from slight vaginal discharge and discomfort to a severe form of vulvovaginitis including itching, pain, burning, and dyspareunia. In such cases, vulvar swelling, fissures, and erythema are commonly present (Fig. 4.2a). Most vulvovaginal yeast infections do not present with specific findings on vaginal examination, including the classic curdy cheese-like discharge often associated with Candida infections. As symptoms and signs of vulvovaginal candidiasis are often nonspecific, a laboratory diagnosis is often essential. Microscopic examination may aid with the diagnosis (Fig. 4.2b), but the precise species cannot be recognized; therefore, a culture is needed to identify the specific organism and confirm the diagnosis.

A single episode of yeast vulvovaginitis can cause a short duration of pain. However, 5–8 % of adult women suffer from intractable or recurrent candidal vulvovaginitis (RVVC),

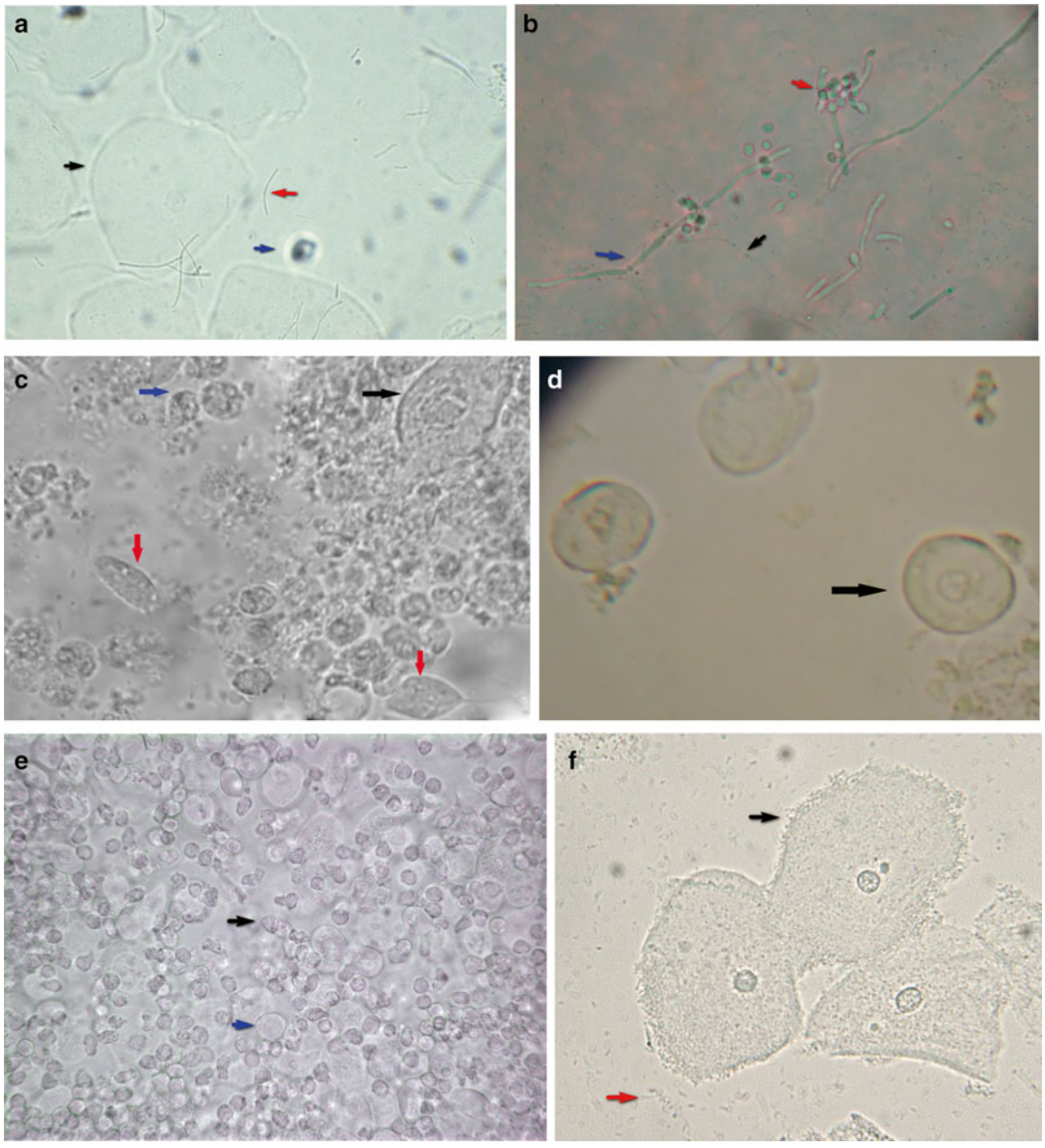

Fig. 4.2 Microscopic findings of vaginal conditions. (**a**) Normal wet mount: Mature squamous epithelial cells (*black arrow*), appear as large, polygonal cells. There is one or no white blood cell (*blue arrow*) per epithelial cell, and gram-positive rods (lactobacillus morphotypes) (*red arrow*) are present. This wet mount represents normal discharge, and is accompanied with normal pH (≤4.5). (**b**) Yeast Infection: mature squamous epithelial cells (*black arrow*), hyphae (*blue arrow*) and budding yeast (*red arrow*). The specific species cannot be recognized from the smear, and a culture is required for identification. (**c**) Trichomoniasis: wet mount reveals an increase of inflammatory cells (*blue arrow*) and parabasal cells (*black arrow*). The parasites *Trichomonas vaginalis* are ovoid shaped and slightly larger than inflammatory cells (*red arrow*), and they are best recognized by their motility. (**d**) Vaginal atrophy: characteristic findings are the presence of parabasal cells (*black arrow*), which are smaller, rounder, and have a large nucleus compared to mature squamous epithelial cells, with scanty vaginal flora as well as an elevated pH (>4.5). (**e**) Desquamative inflammatory vaginitis (DIV): microscopic findings show high number of white blood cells (*black arrow*), an increase of immature, parabasal epithelial cells (*blue arrow*), an absence of lactobacilli and a coccoid flora. The pH is >4.5. (**f**) Bacterial vaginosis (BV): clue cells (*black arrow*) are mature squamous epithelial cells, with adherence of abnormal bacteria to the cell. The flora is comprised of abnormal coccid bacteria (*red arrow*), without elevation of inflammatory cells. BV causes little or no inflammation and therefore it rarely causes pain

Table 4.1 Differential diagnosis of vulvovaginal pain

Vulvar pain related to a specific disorder
Infectious
• Candidiasis
• Trichomoniasis
• Herpes simplex virus
• Group A streptococcus
• (Bacterial vaginosis causes discharge and malodor, but only little or no inflammation and therefore it rarely causes pain)
Inflammatory conditions and dermatoses
• Vulvar contact dermatitis
• Lichen sclerosus
• Lichen planus
• Desquamative inflammatory vaginitis
• Immunobullous disorders
Hormonal
• Vulvovaginal atrophy (estrogen deficiency)
Neoplastic
• Vulvar intraepithelial neoplasia—VIN
• Paget disease
• Squamous cell carcinoma
Neurologic
• Post-herpetic neuralgia
• Multiple sclerosis
• Spinal nerve compression
Systemic disorders
• Behcet's syndrome
• Crohn's disease
Vulvar pain not related to a specific disorder—vulvodynia
1. Generalized
(a) Provoked (sexual, nonsexual, or both)
(b) Unprovoked
(c) Mixed (provoked and unprovoked)
2. Localized (vestibulodynia—previously known as vulvar vestibulitis, clitorodynia, hemivulvodynia, etc.)
(a) Provoked (sexual, nonsexual, or both)
(b) Unprovoked
(c) Mixed (provoked and unprovoked)

which is defined as four or more symptomatic episodes of infection in 12 months. Evaluation of patients for RVVC usually fails to reveal the precipitating cause such as uncontrolled diabetes, immunosuppression, excess of estrogen, or antibiotic usage [6]. The recurrence is believed to result from reinfection or relapse, implying incomplete clearance of candida from the vagina. In such a situation, treatment is directed at control rather than cure, and requires long-term maintenance therapy with a suppressive prophylactic agent [7]. Because of the chronic nature of therapy for RVVC, oral treatments are most useful. The recommended regimen is a weekly oral dose of fluconazole 150 mg. Alternatively, topical prophylactic treatment consists of weekly 500 mg clotrimazole suppositories [8].

Trichomoniasis, a sexually transmitted disease, is caused by the protozoan *Trichomonas vaginalis*. Severity ranges from an asymptomatic carrier state to severe inflammatory vaginitis. Patients may complain of malodorous, yellow-green discharge with vulvar irritation, itch, and dyspareunia. However, many women are asymptomatic [9]. Findings may be absent but are typically characterized by diffuse vaginal erythema and profuse purulent vaginal discharge.

None of the clinical features of trichomonas vaginitis is sufficiently specific to allow for a diagnosis based on signs and symptoms alone; therefore, the definitive diagnosis requires isolation of the organism (Fig. 4.2c). The sensitivity for diagnosing vaginal trichomoniasis by microscopy is low (60–70 %) but for optimal results it is recommended to immediately evaluate a fresh sample of vaginal discharge using a microscope. Other available laboratory methods used for diagnosis include culture, PCR assays, immunochromatographic capillary flow dipstick technology (the OSOM Trichomonas Rapid Test), and more. The recommended treatment is oral metronidazole or tinidazole.

Vulvoaginal atrophy results from inadequate estrogen levels in the vagina. While dyspareunia may affect 10–15 % of fertile women, it affects up to 39 % of postmenopausal women [10]. In this age group, dyspareunia is attributed mainly to vaginal atrophy. Vulvovaginal atrophy occurs most commonly with menopause but can occur due to lactation, medicines, and occasionally, due to usage of extra-low dose contraceptive pills [11]. Estrogen plays a major role in maintaining the normal vaginal environment. Estrogen withdrawal induces significant changes in the vagina causing it to become pale, thin, and less flexible [12]. As a

result, blood flow diminishes, secretions decrease, vaginal flora changes, and pH increases. These changes predispose menopausal women to potential dyspareunia through several mechanisms. Vaginal dryness causes increased friction during intercourse. The thin vaginal walls are friable and prone to mechanical damage and formation of petechiae, ulcerations, and tears occurring with sexual activity. With long-standing estrogen deficiency, the vagina may become shorter, narrower, and less elastic. All of these changes increase the likelihood of trauma, infection, and pain. Furthermore, changes occurring with aging, such as subcutaneous fat loss and decreased skin lipid production, can result in slower healing after injury. Age dependent hypotonia and hypertonia of the pelvic floor muscles can also contribute to dyspareunia [13]. Patients with atrophy may complain of dryness, itching, discharge, pain, dyspareunia, and irritative urinary symptoms [12]. Symptoms are usually progressive and do not resolve spontaneously. Diagnosis is made by identifying characteristics changes on physical examination, noting an elevated vaginal pH and the presence of parabasal cells on microscopy (Fig. 4.2d) [14].

The primary goal of treating symptomatic vaginal atrophy is to alleviate symptoms. First-line therapies include nonhormonal, long-acting vaginal moisturizers and low-dose vaginal estrogen. For women with symptomatic vulvovaginal atrophy who prefer a nonvaginal therapy, transdermal and oral hormone therapy as well as the selective estrogen-receptor modulator, ospemifene, are options [15]. Because the overall dose of estrogen is low and is associated with less absorption, topical estrogen is generally considered safer and is less worrisome to patients. Atrophic changes, including those observed with microscopy, are rapidly and markedly reversed with topical estrogen therapies. Intravaginal estrogen preparations may be insufficient if vestibular symptoms are predominant. This results from preferential distribution of the absorbed hormones to the uterus. The hormonal preparation should be placed in the outer third of the vagina for best clinical results [16].

Desquamative inflammatory vaginitis [14] (DIV) is a rare cause of noninfectious, purulent vaginitis. Its diagnosis is based on symptoms, signs, and laboratory findings that are nonspecific. DIV is a chronic condition, as most patients will have complaints for more than a year before being diagnosed. The most common manifestation of DIV is copious, purulent vaginal discharge. However, 90 % of sexually active patients complain of dyspareunia [17]. As DIV is primarily a vaginal condition, the pain is mainly with thrusting. However, when DIV causes vestibular inflammation or erosions, patients may have pain with intromission as well. Some patients may not know they have dyspareunia because they have ceased having intercourse due to the abnormal discharge. Other symptoms include vulvovaginal burning and irritation [17].

On physical examination, patients may exhibit findings of vulvar erythema and introital ecchymotic spots. A vaginal examination typically reveals diffuse erythema and a purulent yellow or green vaginal discharge (Fig. 4.3b), occasional ecchymotic spots (usually located in the inner third of the vagina), and colpitis macularis. Similar to vaginal atrophy, the pH is elevated and microscopy shows parabasal cells, but there is a marked increase in inflammatory cells (a ratio of inflammatory cells to epithelial cells >1:1), as well as vaginal flora abnormality with the loss of dominant lactobacillus morphotype (Fig. 4.2e) [18].

The differential diagnosis for DIV includes other disorders causing purulent vaginitis. These include infectious vaginitis such as trichomoniasis and Group A Streptococcal vaginitis, dermatologic disorders such as erosive lichen planus and mucosal blistering disorders and usage of chemical irritants. It is therefore essential to exclude other etiologies before confirming diagnosis.

Because of the similarities between vaginal atrophy and DIV, it may be difficult to distinguish them from each another. With atrophy, the epithelial surface remains intact, and response to local estrogen therapy is usually rapid. Thus, failure to reverse the abnormal appearance of the vulvar vestibule or vagina and consequent symptoms with topical estrogen therapy constitutes a diagnostic test.

Little is known regarding etiology, treatment options and long-term follow-up. Both topical vaginal clindamycin and vaginal corticosteroids

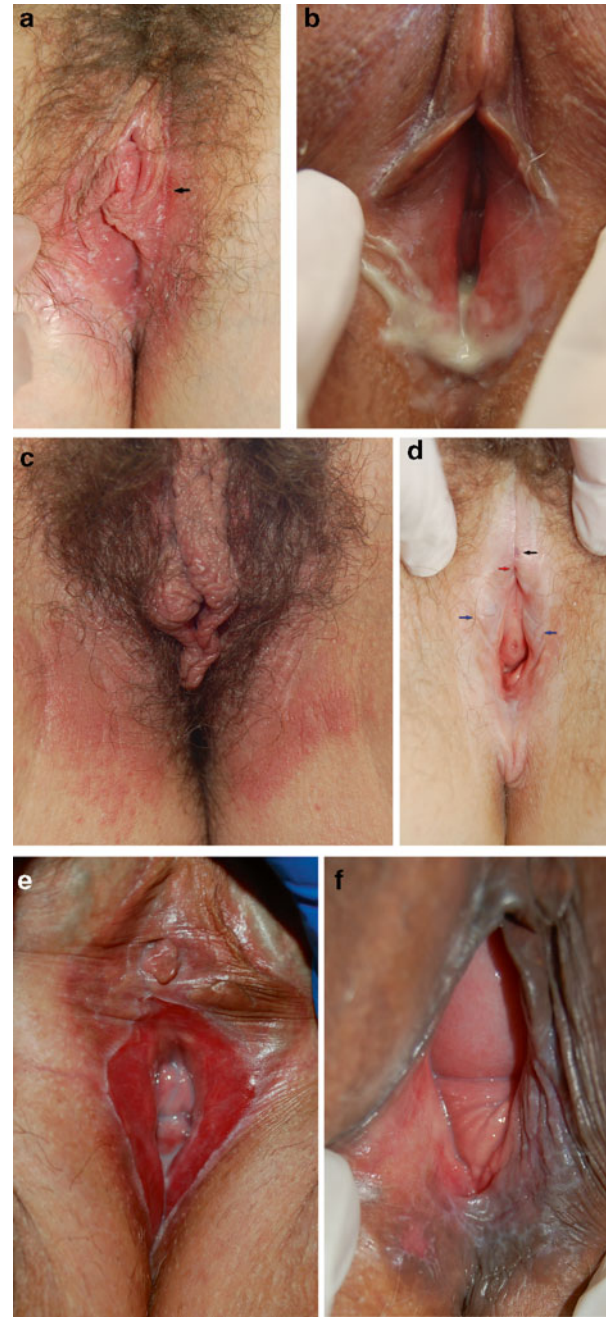

Fig. 4.3 Macroscopic findings in vulvovaginal disorders. (**a**) Vulvovaginal candidiasis—the vulva is inflamed, with erythema and fissures in the interlabial sulci (*arrows*). (**b**) Desquamative inflammatory vaginitis: diffuse erythema and copious purulent discharge. (**c**) Vulvar contact dermatitis—erythema secondary to usage of panty liners. (**d**) Vulvar lichen sclerosus—findings in LS include hypopigmentation, epithelial thinning, hemorrhages (*black arrow*), loss of normal architecture including disappearance of labia minora (*blue arrow*), buried clitoris (*red arrow*) and narrowing of the introital opening. The disease involves the perineal and perianal areas. (**e**) Erosive lichen planus—causes painful vestibular erosion that appears as deep glazed erythema. Note loss of normal architecture with the absence of labia minor, (**f**) Lichen planus—presents with white, reticulate, lacy striae. The lacy pattern is considered pathognomonic

have anti-inflammatory affect and are useful for treating DIV; the choice of treatment should consider the availability and cost of these medications [18]. The treatment, either topical clindamycin or corticosteroids, is administered daily for 2–4 weeks. Initial therapeutic response is extremely encouraging, with 86 % of patients experiencing dramatic improvement; however, relapse and chronic manifestations frequently require long-term topical therapy [18].

Contact dermatitis is an inflammation of the skin due to exposure to an exogenous agent acting as primary irritant or an allergen. As vulvar tissue is more susceptible to irritants than other

tissues, contact dermatitis is common and can complicate all other vulvovaginal conditions. Common vulvar irritants include soaps, panty liners, wet wipes, menstrual hygiene products (pads and tampons), toilet paper, laundry detergents, fabric softeners, cosmetics, spermicides, condoms, lubricants, urine, sweat, and topical medications.

Symptoms are of nonspecific inflammation, and include burning, itching, pain, and fissuring of vulvar tissue. Diagnosis is based on detailed history, exclusion of infectious causes, and high level of suspicion. Physical findings may range from mild erythema (Fig. 4.3c) to severe inflammation (erythema, edema, and fissures), sometimes with a secondary infection. Finalizing the diagnosis may require a biopsy to rule out coexisting conditions. Patch testing may be helpful in cases of allergic contact dermatitis.

Lichen sclerosus (*LS*) is a chronic, inflammatory skin disorder affecting 0.1–1.7 % of women [19] with a distinct predilection for the anogenital region. The mean age at onset of symptoms is 45.5 years, but LS may appear at any age, with 9 % of women experiencing onset of LS in prepuberty, 41 % in the reproductive years, and 50 % postmenopausal [20]. The etiology of LS has not yet been adequately explained, but there is increasing evidence that autoimmune mechanisms play a pathogenic role, with a possible genetic susceptibility.

LS is usually a scarring, chronic progressive or relapsing and remitting, lifelong condition. The characteristic sites involved are the interlabial sulci, labia minora, labia majora, clitoris, clitoral hood, perineum, and perianal area, giving rise to the characteristic "figure-of-eight" appearance. Alternatively, it may involve only small areas of skin. Typically, there is no vaginal involvement.

The typical lesions are porcelain-white papules and plaques with hyperkeratosis. The area evolves into a dry, hypopigmented, sclerotic, and later atrophic lesion. LS causes scarring of vulvar tissue, with loss of normal architecture including disappearance and fusion of labia minora, clitoral adhesions, sealing of the clitoral hood, burying of the clitoris, and narrowing of the introital opening (Fig. 4.3d). Otherwise, it can appear as nonspecific erythema, edema, erosions, fissuring, purpura, and ecchymoses. Tearing during sexual intercourse or physical examination is common.

Clinically, while 10–20 % of patients are asymptomatic, most patients present with itch, chronic scratching, pain, soreness, and dysuria. A high proportion of women report significant sexual difficulties including dyspareunia and apareunia due to continuing inflammatory disease as well as due to anatomic changes and scarring from long-standing active disease.

The diagnosis of LS is usually clinical, and when features are typical, histologic examination is not always essential. However, in the early stages of the disease the diagnosis can be difficult and a biopsy is required.

The gold-standard treatment for LS is topical corticosteroid ointment (such as clobetasol propionate 0.05 %), applied daily or twice a day until active disease has resolved, after which frequency and potency of treatment is gradually tapered to twice weekly. Patients should be counseled that LS is a chronic disease, requiring maintenance treatment and long-term follow-up. Women with LS have higher risk of developing vulvar squamous cell carcinoma.

Lichen planus (*LP*) [21] is a systemic, autoimmune, inflammatory mucocutaneous disorder. It can involve the vagina, vulva and vestibule as well as oral mucosa and skin elsewhere. Vulvovaginal involvement can cause itch, pain, burning, dyspareunia, and dysuria. The most common vulvar variant is erosive LP, causing painful vestibular erosion that appears as deep glazed erythema (Fig. 4.3e). Similar to LS, introital stenosis and destruction of normal vulvar architecture can happen. In the vagina, LP can cause vaginitis or localized erosive lesions. The inflammatory process can subsequently cause adhesions, fibrosis, and even complete vaginal obliteration.

Classic LP presents with white, reticulate, lacy striae (Wickham's striae) (Fig. 4.3f). Diagnosis is based on either typical findings or biopsy. Even though LP is more difficult to treat than LS, the recommended initial treatment is similar, and consists of topical ultrapotent steroids. In unresponsive cases or extensive disease, systemic

immunosuppressive therapy may be necessary. For vaginal involvement, routine usage of vaginal dilators is mandatory. Long-term treatment and follow-up are required.

4.4 Genital Pain Syndromes: Provoked Vestibulodynia and Generalized Unprovoked Vulvodynia

Patients with vulvar pain, in whom there is not a recognized disorder, are diagnosed with vulvodynia. The ISSVD terminology for classification of vulvodynia distinguishes between generalized and localized pain. Each of these two subgroups is further subdivided into provoked, unprovoked or mixed [3]. The majority of clinical presentations is either provoked vestibulodynia (PVD), formerly known as vulvar vestibulitis syndrome, or generalized unprovoked vulvodynia (GVD).

PVD is the term describing a syndrome of provoked, localized allodynia of the vestibule of the vulva, not explained by another condition, and lasting more than 3 months. PVD was first described as a syndrome in 1987 by Dr. Edward Friedrich [22] and was named vulvar vestibulitis syndrome. Friedrich's criteria were [1] severe pain in the vulvar vestibule upon touch or attempted vaginal entry; [2] tenderness to pressure localized within the vulvar vestibule; and [3] vulvar erythema of various degrees.

As inflammation is often not found, in 2003, the term vestibulitis was replaced by the ISSVD to PVD [3], but the diagnostic criteria are similar to those suggested by Friedrich. These include a typical history of pain upon vestibular touch, such as attempted intercourse, gynecological examination, insertion of tampon or other direct contact and a positive Q-tip test, which elicits severe pain or discomfort. Most patients with PVD present with dyspareunia or complete inability to have intercourse.

In *GVD* the patient reports a continuous, unpleasantsensation of pain [3], usually described as burning, stinging, irritating, itching, or a feeling of rawness. Most often the pain is diffuse, without clear borders. Any stimulus which results in pressure on the vulva can exacerbate the pain, including intercourse, tight fitting clothing, sitting, walking, or exercising.

4.5 Causes of PVD

PVD is not a defined disease but rather a symptom. There is a belief that PVD represents a group of distinct disorders that have been classified together simply because they produce pain in the same anatomic location [23]. Causes of these disorders include hormonal imbalance, mainly caused by hormonal contraception [24–27], nerve fiber proliferation in the vestibular mucosa [28–31] and hypertonic pelvic floor dysfunction [32, 33]. PVD may appear with sexual debut or first attempts to insert a tampon (primary PVD) or can be a new onset of pain with activities that did not previously illicit pain (secondary PVD) [34].

Studies found that different factors such as genetic, inflammatory mediators, recurrent vaginitis, allergy, and trauma may be involved in the development of PVD. A high percentage of patients with vulvar pain report an antecedent history of vulvovaginal candidiasis, although it is unknown if this represents a true increase in incidence or a misdiagnosis. It has been suggested that repeated vuvlovaginal infections are a triggering event for some women leading to chronic vulvar pain. This observation has led to hypothesis that in patients with neurogenic vulnerability, an initiating event or series of events may lead to chronic vulvar pain [35–37].

Several studies point to a possible genetic involvement with polymorphisms in genes responsible for regulation of inflammatory response: allele 2 of the IL-1b gene [38], mannose-binding lectin (MBL) [39], melanocortin-1 receptor (MC1R) gene [40], and the gene coding for the inflammasome component NALP3 [41]. The theory suggests that some women with PVD have defective regulation of proinflammatory immune responses due to genetic variations that predispose them to exaggerated inflammatory responses [36, 38, 41, 42]. Chronic inflammation may induce changes in peripheral

nociceptors or may represent an increased exaggerated neurogenic inflammation facilitating central sensitization.

Because the diagnosis of vulvodynia is nonspecific, treatment is not evidence based, and proceeds on a trial-and-error basis. At least 30 different therapeutic interventions have been used for the management of vulvodynia, yet evidence from clinical trials remains largely inconclusive [43]. Recommendations are in favor of a multidisciplinary approach focusing on pain management and re-establishing pelvic floor function [44].

A different approach is suggested by Goldstein [45]. He classifies PVD into groups, based on history and examination findings:

1. Hormonally mediated PVD—the pain began while taking hormonal contraceptive or other medications that affect hormones, after removal of ovaries, breastfeeding or menopause. Typically, patients have a low calculated free testosterone and complain of dryness, decreased libido, and decreased arousal. The entire vestibule is tender and vestibular mucosa is often dry and thin. Treatment includes stopping hormonal contraception and application of topical estradiol (with or without testosterone) to the vestibule [46].
2. Hypertonic pelvic muscle dysfunction—in this subgroup, PF muscles become tight and tender. Patients often have other symptoms suggesting hypertonicity (urinary frequency, urgency and hesitancy, constipation, hemorrhoids, and anal fissures), and predisposing factors, such as musculoskeletal disorders or anxiety, may coexist. Typically, the pain is much worse at 4–8 o'clock position of the vestibule with minimal or no pain in the upper vestibule. Treatment includes PF physiotherapy, with an optional addition of muscle relaxants (valium suppositories), Botulinum toxin injections and cognitive behavioral therapy.
3. Neuroproliferative PVD—in this condition, women have an increased number of nociceptors in the vestibular mucosa. This group is further subdivided into congenital and acquired forms. In the congenital subgroup, vestibular pain has always been present, and there may be sensitivity to palpation of the belly button, due to common embryologic origin (the primitive urogenital sinus) [47]. With acquired neuroproliferative PVD, the pain may begin after a severe allergic reaction or vaginitis. There is tenderness of the entire vestibule. Treatments include topical anesthetics, antidepressants, antiseizure drugs, capsaicin cream, and a surgical procedure, termed "vulvar vestibulectomy." Various surgical approaches were described, in which excision and resection of the painful vestibular tissue is performed [48]. In general, a horseshoe-shaped area of the vestibule and inner labial fold is excised, followed by advancement of the posterior vaginal wall [49].

As with PVD, the term "unprovoked vulvodynia" describes a symptom, and the question is whether we can diagnose a specific cause instead of calling it simply "vulvar pain." It is possible that some cases of GVD may represent pudendal nerve disorders, PF hypertonic disorder, or can be classified as an entity within the spectrum of neuropathic pain syndromes, while in other patients it represents a functional pain syndrome.

4.6 Treatment of PVD and GVD

The state-of-the-art of vulvodynia management is described in "The Vulvodynia Guideline" published in 2005 [50], developed by an expert panel, organized by the ISSVD. The Guideline is largely based on expert opinion and has several disadvantages, including the absence of differentiation between treatments for GVD and PVD, has not been updated with current research and does not clearly state the quality of the supportive evidence utilized. Because the pathogenesis is not defined, treatment of vulvodynia is generally predicated on a trial-and-error basis. The result is that many forms of therapeutic interventions have been used, yet the evidence remains largely inconclusive. Modalities of treatment mentioned in the vulvodynia guidelines include vulvar care measures, topical, oral and injectable medications,

physical therapy, surgery (vestibulectomy) and complementary medicine.

In a review published by Andrews [43], all published studies regarding vulvodynia and PVD were evaluated. The author sought to include only placebo-controlled randomized trials but, not surprisingly, found only a few, so he included studies without a comparator. In most of these studies, the data analyzed was a pretreatment evaluation versus a posttreatment evaluation and the primary outcome was the reported decrease in pain.

Andrews found 447 articles on treatment of PVD and GVD, of which 71 were eligible for review. Fifty-five studies reported 28 different treatments for PVD. The majority of published studies were case series, with success rates varying from no effect to 100 % improvement. There were eleven randomized trials, of which five were placebo controlled. Most of the studies had several methodological weaknesses, including lack of control or placebo group, non-double-blind assessment, no pretreatment pain and functional status assessment, non-validated outcome measures of pain and sexual functioning, and no long-term outcomes.

Of the 71 eligible articles, 16 reported an evaluation of a therapy for vulvodynia, predominantly GVD. Twelve different interventions for GVD were studied. All of the vulvodynia studies had the methodological weaknesses cited above. There were no analytic studies, no randomized controlled trials and all the studies reported a beneficial effect. Andrew's review found insufficient evidence to support that any of the nonsurgical therapies confers a net benefit for patients with PVD, and insufficient evidence for efficacy of any of the treatments studied for GVD.

Furthermore, single placebo-controlled randomized trials have demonstrated evidence for a lack of benefit of topical 5 % lidocaine, oral desipramine, and botulinum-toxin injections. The body of scientific evidence for other interventions was poor, and there was insufficient evidence regarding the efficacy of numerous other interventions, including steroid and anesthetics injections, multilevel nerve blocks, interferon, capsaicin, topical gabapentin, cognitive behavioral therapy, and PF physiotherapy.

Surgical therapy was also evaluated: case series of 1138 patients reported an effect varying between 31 % and 100 %, for patients who reported at least some improvement. Twelve studies reported complete relief as an outcome and the median effect size was 67 %. Therefore, there is fair evidence that vestibulectomy provides a benefit for patients with PVD, but the size of this effect cannot be determined with confidence.

In addition to the above findings, outcomes show a wide range of response to different therapies for vulvar pain syndromes, with 35–79 % of women reporting improvement in pain scores at 6 months [35, 50, 51]. Nevertheless, long-term outcomes are discouraging with up to 66 % of women reporting some improvement, but only 57 % reporting greater than 50 % improvement in pain since diagnosis [52, 53], and more than half still describe severe pain and some level of functional impairment in daily activities [51, 53].

It is suggested that several parameters may be responsible for these discouraging results. First, the expression, clinical presentation, and response to therapy result in a unique pain experience for each patient. There are also other psychological factors that adversely affect the outcome among women with vulvar pain, such as depression, stress, somatization, anxiety, phobic symptoms, and catastrophizing. Failure to identify and address these factors in both the clinical and research settings may affect outcome [23]. Some women have greater evidence of CNS dysregulation, such as enhanced systemic pain responses and more than one pain syndrome. The failure to address this central component may also account for treatment failure [23].

Finally, vulvar pain syndromes may represent a diverse group of disorders with several possible pain mechanisms; therefore, the appropriate treatment varies and is individualized. Unfavorable outcomes to therapy can be explained by grouping patients with different conditions under one diagnosis, and then studying an intervention that might only help one subset of the conditions. This can lead to an apparent lack of effect, due to dilution of the patient subpopulation [43].

Vulvar pain syndromes are complex conditions with a variety of factors contributing to

etiology, symptoms, and response to therapy. The majority of studies involved monotherapy, and demonstrated inadequate results. However, an integrated response addressing a variety of contributing factors offers the best therapeutic result [54]. In fact, several studies found that multidisciplinary approach leads to considerable improvements in vulvar pain and the resumption of intercourse [55–57].

4.7 Summary

Genital pain and dyspareunia are highly prevalent conditions, with a variety of etiologies. The chronic nature of the majority of these conditions as well as the frequent delay in correct diagnosis adversely affect patients emotionally, socially, sexually, and interpersonally.

Accurate diagnosis accompanied by rapid and suitable treatment is of major importance. It may provide immediate cure or control of chronic disorders, and therefore prevent sequelae such as PF dysfunction and emotional stress that may develop if vulvar pain is left untreated. In addition, due to the possibility that intractable vaginitis is a triggering event leading to chronic vulvar pain in vulnerable patients, a quick and accurate diagnosis is important.

Management of vulvodynia can be improved if the etiology of the pain is identified or at least classified better. More research on epidemiology, natural history, and etiology of PVD and GVD is required, as well as standardization of research tools, definitions and outcome measures.

Meanwhile, in the absence of high quality evidence, clinical decisions remain a challenging task. Clinicians should keep in mind that treatment starts with a correct diagnosis. Vulvodynia is a diagnosis of exclusion after other, treatable causes are ruled out through a thorough history, physical examination, microscopy, cultures, and biopsies. Caregivers should believe that a successful outcome is possible and make sure they clarify patients' expectations and goals. A multidisciplinary approach leads to substantial improvements in pain and well-being. Currently, there is no "silver bullet." The patient should realize that adjustments in treatment interventions are typically needed, and that this requires persistence and patience of both the patient and the provider.

4.8 Questionnaires

1. International Pelvic Pain Society
   http://www.pelvicpain.org/pdf/History_and_Physical_Form/IPPS-H&PformR-MSW.pdf
2. ISSVD
   https://netforum.avectra.com/temp/ClientImages/ISSVD/3ef9c6ea-aac7-4d2b-a37f-058ef9f11a67.pdf

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5 Bladder Pain Syndromes/ Interstitial Cystitis and the Overactive Pelvic Floor

Mauro Cervigni, Andrea Morciano, and Giuseppe Campagna

5.1 Bladder Pain Syndrome/ Interstitial Cystitis

Bladder Pain Syndrome/Interstitial Cystitis (BPS/IC) is a condition characterized by symptoms of urgency, frequency, and pain in the bladder and/or pelvic area. These collective terms most probably describe debilitating, chronic bladder disorders of unknown cause, with an exclusion of confusable diseases. Current data show that the condition is much more prevalent than previously thought. BPS/IC is also a cause for pelvic floor dysfunction (PFD), occurring mostly in women (>90 %) [1, 2].

Misdiagnosis and ineffective treatments are common, leaving patients with persistent pain and the potential for neuropathic upregulation and allodynia. Currently, BPS/IC is considered a diagnosis of exclusion because its etiology is not known and clinical characteristics vary among patients. Voiding often relieves the typical symptoms of pain, pressure, or discomfort involving the lower pelvic area, including gastrointestinal organs. Symptoms have to be present for no less than 6 months, obviously in the absence of urinary tract infection (UTI) [1]. Early recognition of BPS/IC is very important because symptoms are quite disabling, affect quality of life and lead patients to seek evaluation by a variety of specialists (usually between five and seven times in a period of 3–5 years). Several comorbidities typically accompany BPS/IC, including allergies, asthma, atopic dermatitis, inflammatory bowel syndrome (IBS), systemic lupus erythematosus (SLE), Sjögren's syndrome, chronic fatigue syndrome, and fibromyalgia [3–6]. Vulvodynia may be present in 20 % of cases [7], as well as endometriosis, identified in 45–65 % of women with pelvic pain of bladder origin [8]. Although less frequently, BPS/IC may occur in men (2.2 % of the population, according to the National Institutes of Health Chronic Prostatitis Symptom Index (NIH-CPSI)) with less frequent urgency and frequency of urination (type 3 prostatitis, non-bacterial prostatitis, or chronic prostatitis) [9].

5.2 Definition

The National Institute of Diabetes and Digestive Kidney Diseases (NIDDK) established a set of consensus criteria, which were developed to ensure the comparability of patients enrolled in clinical studies [10]. These included:

- Hunner's ulcers
- Any two of the following:

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- Pain on bladder filling, relieved by emptying
- Suprapubic, pelvic, urethral, vaginal, or perineal pain for 9 months
- Glomerulations on endoscopy or upon hydrodistention under spinal or general anesthesia

However, over 60 % of patients with possible BPS/IC appear to fail these criteria [11].

The International Continence Society (ICS) in 2002 defined Painful Bladder Syndrome as "The complaint of suprapubic pain related to bladder filling, accompanied by other symptoms such as frequency and nocturia in the absence of proven pathologies" [12].

Most recently, the European Society for the Study of Interstitial Cystitis (ESSIC) named this disease Bladder Pain Syndrome [13] according to the definition by the International Association for the Study of Pain (IASP) [14].

A significant proportion of BPS/IC patients do not complain of pain but report feelings of pressure and discomfort [15]. Such patients would remain undiagnosed for BPS/IC, if only pain syndromes were applicable to a diagnosis of BPS/IC. Therefore, in May 2009 the East Asian Committee of Urology, with representatives from Japan, Korea, and Taiwan, published clinical guidelines for BPS/IC, proposing a new definition of the syndrome as "hypersensitive bladder syndrome" (HBS)—bladder hypersensitivity, usually associated with urinary frequency, with or without bladder pain [16].

5.3 Epidemiology

In the past, the prevalence of BPS/IC was estimated at 18.1/100,000 women [17]. Subsequent studies in 2002 indicated a prevalence of 450 per 100,000 (0.45 %) and more recently such figures have increased up to 680 per 100,000 (0.68 %) for a probable BPS/IC diagnosis and 300 per 100,000 (0.3 %) for a definite one [18, 19].

A recent study of 981 urban women in Vienna showed an overall prevalence of 306 per 100,000 (0.3 %), with the highest number in the 40–59 years age group [20]. BPS/IC has also been reported in children and adolescents [21–23]. In Japan, the prevalence reported from a questionnaire survey of 300 major hospitals was only 2 per 100,000 patients [24]. Patients were older (52.9 years on average) than those in Europe and the USA [25]. This may indicate that patients have had symptoms for a long time before diagnosis. However, a recent epidemiological investigation in Japan found that 1.0 % of the general population experience bladder pain on a daily basis [26]. There is some evidence of genetic predisposition; the prevalence of BPS/IC in first-degree relatives has been shown to be 17 times higher than in the general population [27].

5.4 Pathophysiology

Several etiologic theories have been proposed in recent years, although they remain somewhat speculative and controversial and the precise causes of BPS/IC are still unknown. One aspect has been emphasized: the multifactorial etiology of the disease. Interaction between neural, immune, and endocrine factors creates a vicious cycle, provoking and maintaining the inflammatory response in the bladder.

5.4.1 Infection

To date, no infectious etiology has been identified for BPS/IC. Reverse transcriptase polymerase chain reaction (RT-PCR) for *Chlamydia trachomatis*, adenovirus, cytomegalovirus, herpes simplex virus, papillomavirus, or *Gardnerella vaginalis* [28, 29] have been studied and resulted negative. Lack of efficacy of antibiotic treatment in BPS/IC also excludes the option of an infectious etiology. Symptom flare-ups can occasionally be elicited by infection, which can initiate or exacerbate BPS/IC [30].

5.4.2 Mastocytosis

An increased number of activated bladder mast cells has been repeatedly reported in BPS/IC

[31]. The urothelium of BPS/IC patients contains twice as many mast cells as compared to controls and the detrusor contains ten times more [32]. In addition, more than 70 % of bladder mast cells were activated in BPS/IC as compared to less than 10 % in controls [31]. Mast cells play a pivotal role in the inflammatory process: they release potent inflammatory mediators such as histamine, leukotriene, and serotonin and interact with immunoglobulin E (IgE) antibodies, other inflammatory cells, and the nervous system [32, 33].

5.4.3 Dysfunctional Bladder Epithelium

The protective inner layer of the bladder is made up of glycosaminoglycans (GAGs), chondroitin sulfate (CS), and sodium hyaluronate (SH). The GAG component is hydrophilic and binds a layer of water molecules that is thought to protect the urothelium from potentially harmful agents, including bacteria, proteins, and ions. Proponents of the leaky endothelium theory suggest that the GAG layer may be damaged in BPS/IC [34, 35]; this deficiency allows irritants in the urine to leak through the urothelium and causes inflammation, irritation, and numerous other reactions [36].

Increased urinary levels of CS and SH have been reported in some BPS/IC patients [37, 38], with concomitant decrease of mucosal glycoprotein GP1 [39].

The etiology of the defect in the GAG layer is currently unknown. Antiproliferative factors (APFs), detected in the urine of IC patients, downregulate expression of genes that stimulate proliferation of bladder epithelial cells, and upregulate genes that inhibit proliferation, leading to urothelial undermaturation and dysfunction [40, 41].

5.4.4 Neurogenic Inflammation

BPS/IC is not exclusively an end organ condition; it should be considered a disorder of the peripheral and central nervous systems as they are responsible for the acute and chronic pain. The initiating event can be a noxious stimulus such as trauma, infection, or inflammation. Acute pain is associated with nociception, which results in pain perception modulation by the peripheral and central nervous systems. Conversion of acute pain to chronic begins with activation of visceral silent unmyelinated C-fibers due to prolonged noxious stimulation and inflammation. The neurotransmitter glutamate is released, which activates *N*-methyl-D-aspartate receptors. A chronic pain cycle begins as dorsal horn neurons are activated (wind-up), which causes exaggerated responses to less noxious stimuli (hyperalgesia), or a painful response to normally innocuous stimuli (allodynia). Small volumes of urine in the bladder are perceived as a full bladder. The neuro-transmitter substance P stimulates the release of histamine and nitric oxide, which causes neurogenic inflammation. Once the dorsal horn becomes hypersensitive, pain becomes chronic. Prolonged noxious stimuli can cause dorsal horn cells to transmit efferent signals to peripheral nerve terminals (antidromic transmission). Thus, a self-perpetuating signal is established as a visceral CPP syndrome, causing expression of genes such as c-Fos in the spinal cord and loss of inhibitory neurons, resulting in a decreased threshold for activation.

5.4.5 Reduced Vascularization

A decrease in microvascular density has been observed in the bladder suburothelium of patients with BPS/IC [42]. Bladder vascular perfusion is reduced by bladder filling in BPS/IC, while it is slightly increased in controls [43].

A recent paper showed that hyperbaric therapy seems to relieve the symptoms of BPS/IC [44], indirectly confirming that reduced blood supply may cause a decrease in epithelial function as well as epithelial thinning and denudation [45]. Impaired blood circulation in the bladder and increased apoptotic activity of microvascular endothelial cells have been suggested to occur in BPS/IC [46].

5.4.6 Autoimmunity

Many of the clinical features of BPS/IC suggest an autoimmune element to play a role in the disease process. A concomitant association of BPS/IC and other autoimmune diseases, such as SLE, rheumatoid arthritis, and Sjögren's syndrome, has been reported [47–49].

5.5 Diagnosis

It is important to keep in mind that BPS/IC patients may present with only one of the symptoms, particularly early in the course of the disease. Up to 30 % with BPS/IC present without pelvic pain [50], and approximately 15 % present with pain as the only symptom [51].

The diagnosis of BPS/IC is driven by exclusion but should not necessarily be organ oriented, considering the large number of confusable diseases according to ESSIC Criteria '08 (Table 5.1). A comprehensive medical history should include questions about suprapubic pain, pressure, and discomfort related to bladder filling, as well as increased urinary frequency and urinary urgency, in the absence of UTI or other evident pathology [52].

Table 5.1 Confusable diseases in BPS/IC

Disease type	Confusable diseases
Bladder diseases	Overactive bladder
	Neurogenic bladder
	Radiation cystitis
	Bladder calculus
	Bladder cancer
Prostate and urethral diseases	Benign prostatic hypertrophy
	Prostate cancer
	Urethral stenosis
	Urethral diverticulum
Genitourinary infections	Bacterial cystitis
	Urethritis
	Prostatitis
Gynecologic diseases	Endometriosis
	Uterine myoma
	Vaginitis
	Postmenopausal syndrome
Other conditions	Polyuria
	Overactive pelvic floor

Source: ESSIC criteria 2008

A retrospective analysis from the IC Database (ICDB) pointed out the most common baseline pain site was lower abdominal (80 %), urethral (74 %), and low back (65 %), with the majority of patients describing their pain as intermittent [53].

Questionnaires can be helpful in screening for BPS/IC. The most commonly used screening tools are the Pelvic Pain, Urgency, Frequency symptom scale (PUF) and the O'Leary–Sant Symptom and Problem Index [54, 55]. Both surveys include questions regarding pain, urgency, frequency, and nocturia and how these symptoms affect quality of life.

Physical examination is a critical component of diagnosing BPS/IC. Since the bladder is the pain generator, tenderness with single-digit examination of the trigonal area can help establish a diagnosis of BPS/IC [56]. Pelvic floor tenderness upon palpation of the levator muscles is a common finding [57]. Physical examination should also address high tone of the pelvic floor muscles, and hypersensitivity of the perineal area, using the Kaufman Q-tip touch sensitivity test that might screen for the presence of vulvar vestibulodynia (VVS) [58].

As regards ancillary testing, urine analysis should be carried out to rule out hematuria, urine culture is required to identify bladder infection and cytology can help rule out bladder cancer. Several additional optional diagnostic tests are used but diagnostic evaluation varies among urologists/urogynecologists, in different centers [59–61] and among the USA, Europe, and Asia (Table 5.1) [16, 62].

Urodynamic studies can highlight detrusor overactivity or reduced bladder capacity without detrusor overactivity (bladder hypersensitivity), suggestive of BPS/IC [63–65]. Mild impairment of the voiding phase with detrusor-sphincter discoordination is probably related to the dysfunctional pelvic floor behavior.

Local cystoscopy is not mandatory but is a good preliminary investigation to rule out other conditions (e.g., bladder stone, hematuria, or cancer). Diagnostic cystoscopy can identify Hunner's

ulcer [66], the typical bladder lesion seen in BPS/IC. Typically, the ulcer is recognized as a well-demarcated reddish mucosal lesion lacking normal capillary structure. In addition, some scars or fissures with a rich hypervascularization or a pale mucosal appearance may be present, as an indirect index of hypovascularization.

Cystoscopy with hydrodistention under anesthesia has been proposed by the NIDDK research criteria, but is now considered too restrictive [61]; however, it remains the most common diagnostic procedure performed in patients with BPS/IC, especially in Europe [67]. Hydrodistention is carried out using different methodologies, making comparison between studies difficult [58, 62, 68]. It is useful to exclude other pathologies, to identify the presence of "classic" BPS/IC with "Hunner's lesions" and document urothelial bleeding (glomerulations), even though these have also been noted in the bladders of normal women undergoing tubal ligation [69].

Bladder biopsy should be carried out once the hydrodistention has been completed, to avoid the risk of bladder rupture. Biopsies can show the presence of mast cell infiltration, and orientate specific therapy choices. Biopsy with histopathology may be necessary to exclude neoplasm and eosinophilic or tuberculotic cystitis. The European Society recommends a count of tryptase-positive bladder mast cells with >28 mast cells/mm to be defined as detrusor mastocytosis and considered diagnostic for BPS/IC [61, 62, 70]. An increased number of mast cells has also been recently proposed as a diagnostic criterion for provoked vestibulodynia [71].

There are no recognized blood or urine markers diagnostic of BPS/IC. Recently APF, a substance identified as a frizzled-8 surface sialoglycopeptide [72], has been found to be increased in the urine of BPS/IC patients. APF is detected by its ability to decrease in vitro proliferation of bladder epithelial cells and its activity was enhanced in BPS/IC but not in other urologic disorders [73]. Urine APF levels also seems to discriminate between BPS/IC and CPPS in men [74]. However, APF still needs to be validated and results of its investigation need to be reproduced by further research.

Classic BPS/IC might be differentiated from non-ulcerating disease by its elevated urine nitric oxide (NO) levels [75].

Other urinary markers include heparin-binding epidermal growth factor-like growth factor (HB-EGF), histamine, methylhistamine, and interleukin-6 (IL-6) [76, 77]. Four proteins that differed significantly between BPS/IC and controls have been identified, with uromodulin and two kininogens found to be higher for controls and inter-alpha-trypsin inhibitor heavy chain H4 higher for BPS/IC [78]. Urinary concentration of neutrophil elastase is increased in patients with pain and a small bladder capacity [79]. These markers are not necessarily precise predictors of ulcers and/or symptom severity [80].

Intravesical administration of 40 mL of a solution of 40 mEq of potassium chloride in 100 mL of water (potassium sensitivity test—PST), with pain and urgency scored by the patient as compared to administration of sterile water has been proposed for BPS/IC diagnosis [54]. However, this test's sensitivity and specificity is only about 75 % and the participants at the International IC Consultation in Rome recommended that it should not be used for diagnostic purposes because of its low prognostic value [81].

5.6 BPS/IC and Overactive Pelvic Floor

Many patients with Bladder Painful Syndrome/Interstitial Cystitis (BPS/IC) have concomitant PFD, with muscle tenderness and spasm also known as Overactive Pelvic Floor (OPF).

Several studies found that myofascial pain and OPF are present in as many as 85 % of patients with BPS/IC and/or Chronic Pelvic Pain (CPP) Syndrome [82]. The mechanism is likely very similar to Category IIIB Chronic Prostatitis/Chronic Pelvic Pain Syndrome (CP/CCPS) in the male population. PFD exacerbates BPS/IC symptoms and has been reported to appear in response to events such as bladder inflammation, gait disturbance, and trauma [83, 84].

5.6.1 Pelvic Floor Disorders

PFDs have been classified as hypotonic, or low tone pelvic floor dysfunction (LD), typically associated with urinary and/or fecal incontinence and/or pelvic organ prolapse (POP), or hypertonic dysfunction (HD), which is often associated with Overactive Bladder (OAB), Chronic Pelvic Pain Syndrome (CPPS), BPS/IC, and sexual pain disorders. These dysfunctions are generated by an antidromic reflex that is estimated to be present in 70 % of women [85–87]. In the colorectal literature it is defined also as tension myalgia and levator ani syndrome [88, 89] and can contribute to symptoms of urinary frequency, urgency, dysuria, urinary and fecal retention, dyspareunia, and/or vaginismus [12, 86, 87].

The prevalence of HD is thought to be 50–87 % in patients with BPS/IC [90]. Fibromyalgia, Irritable Bowel Syndrome (IBS), Inflammatory Bowel Disease (IBD), and vulvodynia are all associated with findings of pelvic floor overactivity and myofascial pain [6, 91, 92].

5.7 Pathophysiology

In a normal bladder peripheral neural transmission is mediated by A-fibers which are myelinated, high speed transmission nerves transferring tension, pain, and cold. Unmyelinated C-fibers are low speed transmission neurons in charge of burning, painful and itching stimuli. C-fibers are normally silent and become activated in response to bladder inflammation or irritation. Inflammation, pain, or of pelvic visceral trauma may transfer noxious stimuli to the sacral cord, which can result in pelvic floor muscle dysfunction due to sacral nerve hypersensitivity and initiate a sacral cord wind-up effect [93–97].

The "Guarding Reflex" is a viscero-muscular reflex activated with the aim to increase the tone of the pelvic floor during routine daytime activity [98]. The afferent autonomic bombardment occurring in BPS/IC patients may enhance and maintain such guarding reflex, resulting in pelvic floor overactivity.

5.8 Symptoms

Even though there are symptomatological similarities between BPS/IC and PFD, there are also some differences related to the pelvic floor non-relaxing behavior. Pain symptoms are often vague and poorly localized. Pain is described as achy and throbbing, and is often accompanied by feelings of pressure or heaviness. This feeling is often similar to that described by patients with POP, yet on examination no significant prolapse is observed [92]. Pain often worsens as the day progresses and is typically exacerbated by pelvic floor muscle activities like sexual intercourse or voiding. Factors such as job stress, constipation, or menses may trigger myofascial pain.

Patients with a significant myofascial pain component will often report leg or groin pain occurring with bladder filling and increased urinary frequency during the daytime, in the absence of nocturia. The pressure arising from pelvic floor overactivity may be perceived as a need to void. As the pelvic floor relaxes during sleep, the need to void is generated only by bladder volume and not by pelvic floor pressure. As documented by several authors [99, 100], in patients with BPS/IC pelvic floor physiotherapy combined with bladder-directed therapy is more effective than the latter alone.

5.9 Physical Examination

Clinical evaluation of the pelvic floor begins with observation of pelvic floor muscle activity during the process of actively contracting and relaxation. Mere observation of the perineum and introital area in the dorsal lithotomy position during the performance of an active pelvic floor muscle contraction is often quite revealing. Patients with HPFD are unable to increase contractile strength and therefore cannot produce an effective contraction. Spontaneous muscle fasciculation can often be observed due to muscle fatigue. A lubricated cotton tip applicator is then gently used to evaluate for signs of allodynia and vulvodynia.

At this point the examiner should place a generously lubricated single finger in the vagina to assess pelvic floor awareness and the ability to contract and relax the levator ani. Many scales are available to document strength, tone, and tenderness of any vaginal compartment (Oxford scale -49-50/1). Often patients with HPFD will have a "V" configuration and as a finger is advanced it will drop off the shelf caused by the contracted levator muscles and drop down onto the coccygeus muscle. Active trigger points are often identified as exquisitely tender areas palpable as a small 3–6 mm nodule within a taut band that reproduces the patient's pain, as well as the referral pattern of her pain. Tenderness upon palpation of each ischial spine and underneath the pubic rami should be assessed.

A tool such as Perineometer or an electromyography probe, designed to measure muscle activity, can aid in assessing pelvic flore muscle tonicity [12, 92, 101]. Objective measurements of pelvic floor muscle tone are discussed in Chap. 11 of this text.

5.10 Ancillary Testing

5.10.1 Electromyography

The best objective tool clinically available for the assessment of pelvic floor overactivity is surface EMG. As discussed in Chap. 12 of this text, reliability and validity of this measure continues to present a challenge. The following findings will be suggestive of pelvic floor overactivity:

1. Elevated and unstable resting baseline activity
2. Poor recovery
3. Poor post-contraction and relaxation
4. Spasms with sustained contractions and poor strength [102]

5.10.2 Urodynamic Testing

Typical urodynamic findings include fluctuating or interrupted flow, abnormal voiding phase pressure-flow studies, elevated urethral pressure at rest and urethral instability. Schmidt and Vapnek performed urodynamics in patients with IC or severe urgency and frequency and observed that pain episodes paralleled behavioral changes in the sphincter more than in the bladder [103]. A complete discussion of urodynamic testing is available in Chap. 14 .

5.10.3 Defecography

When symptoms involve obstructed defecation and rectal pain, defecography can be used to identify the presence of a nonrelaxing pelvic floor or paradoxical activity of the pelvic floor during defecation.

5.11 Myofascial Pain Syndrome

Myofascial pain is present in 12–87 % of pelvic pain patients. Myofascial pain disorder can be primary or secondary. The latter is associated with another pain disease such as BPS/IC, Vulvodynia, or IBS. If this is the case, the primary pain generator must be treated as well as the myofascial component.

5.12 Treatment Strategies

5.12.1 Bladder Pain Syndromes/ Interstitial Cystitis

There is no curative therapy for BPS/IC. This is consistent with the fact that the causes of BPS/IC are yet not understood and the pathophysiology remains unclear. Therefore, the therapeutic strategy is to alleviate symptoms, thereby interfering with the potential disease mechanisms and improving quality of life.

Because BPS/IC is a chronic disease, realistic expectation of treatment should be discussed with patients. Remission may be attained but should not be expected, and even when it is attainable, months of medical treatment may be required.

Periods of remission commonly alternate with exacerbations and patients need to be encouraged that therapy is not failing.

5.12.2 Conservative Therapy

Behavioral modification may have modest benefit for IC patients (grade of recommendation B). Barbalias et al. evaluated a bladder training technique as an adjunct to intravesical oxybutynin in patients with IC; there was a modest improvement in O'Leary–Sant questionnaire at 6 months [104]. Chaiken et al. reported similar results with diary-timed voiding and pelvic floor muscle training [105]. There are no randomized controlled trials (RCTs) attesting the efficacy of pelvic floor physical therapy. Biofeedback and soft tissue massage may aid in muscle relaxation of the pelvic floor [105].

Manual physical therapy to pelvic floor myofascial trigger points twice per week for 8–12 weeks also resulted in moderate to marked improvement in 7/10 BPS/IC patients.

Modified Thiele intravaginal massage of high-tone pelvic floor muscle trigger points twice per week for 5 weeks has been shown to improve the O'Leary–Sant Index (grade of recommendation C). A discussion of physical therapy treatment of OPF is available in Chaps. 16, 17, and 18.

Commonsense dietary changes, especially avoidance of potential bladder irritancy as identified by individual patients may be beneficial (grade of recommendation B). A majority of BPS/IC patients seem to have symptom exacerbation related to the intake of specific foods and beverages: coffee, spicy foods, and alcoholic beverages.

However, different patients seem to be affected to different degrees by specific foods and beverages and patients should avoid only those that they find worsen their symptoms.

5.12.3 Medical Therapy

Medical therapies for BPS/IC include oral, subcutaneous, and intravesical agents. These drugs are categorized according to their intended point of action within the disease process.

5.12.3.1 Protection of the Mucosal Surface

One of the theories in the pathogenesis of IC is that deficiency of the GAG layer causes symptoms related to increased permeability of the urothelium. A number of agents have been used to improve the integrity of the mucosal surface.

Pentosan polysulfate (PPS), a branched polysaccharide presumably acting to "replenish" the GAG layer, is the only oral drug approved in the USA for BPS/IC (grade of recommendation B, level of evidence 2). One study of PPS (300 mg/day) used for 3 years showed it was twice as potent as placebo (18 %) in reducing pain but the placebo response was unusually low [1, 83]. A randomized double-blind multicenter study evaluated PPS with a range of doses (300, 600, or 900 mg per day) in 380 BPS/IC patients with >6 months' symptoms and positive cystoscopic examination but no placebo control. With a follow-up of 32 months, they reported a 45–50 % response rate (50 % or greater improvement on the Patients' Overall Rating of Symptoms Index—PORIS), irrespective of dosage [106]. In a recent prospective study, 41 patients with BPS/IC were divided into three groups according to their response to PPS (major, intermediate, minor). They were administered subcutaneous heparin 5000 IU t.i.d. for 2 days, followed by 5000 IU b.i.d. for 12 days plus 300 mg PPS per day, compared to 17 nonmatched patients taking PPS alone. Thirty-two percent of the patients in the minor response group reported a significant improvement in "overall well-being" over that of PPS alone [107].

Hydroxyzine is a histamine-1 receptor antagonist, with additional anxiolytic, sedative, anticholinergic, and mast cell inhibitory properties, which has been shown to reduce neurogenic bladder inflammation [108] (grade of recommendation D, level of evidence 1). Studies on hydroxyzine in treating BPS/IC symptoms report mixed results. One open-label study showed a 55 % reduction in symptoms, particularly in patients who suffered from allergies [109].

Cimetidine is a histamine-2 receptor antagonist (grade of recommendation D, level of evidence 4). It has been reported to decrease median symptom score in 34 BPS/IC patients, but no apparent histological changes in the bladder mucosa were observed [110].

L-Arginine is a natural substrate of nitric oxide synthase (NOS) (grade of recommendation D). It may reactivate NOS activity, which is suppressed in IC, and relieve symptoms. No significant effect was observed in double-blind studies [111].

5.12.3.2 Intravesical Instillation and Bladder Wall Injections

Intravesical and subcutaneous heparin has been used for the treatment of IC since the early 1960s. With intravesical instillation, heparin does not have systemic anticoagulant effects. In one study on 48 patients with IC self-administered intravesical heparin (10,000 IU in 10 mL sterile water three times weekly for 3 months), 56 % of patients attained clinical remission after 3 months [112].

Intravesical dimethylsulfoxide (DMSO) remains the basis of intravesical therapy for IC (grade of recommendation B, level of evidence 2). It has been shown to reduce symptoms for up to 3 months. Its mode of action includes anti-inflammatory and analgesic effects, muscle relaxation, mast cell inhibition, and collagen dissolution. Patients treated with DMSO have reported a 50–70 % reduction of symptoms, although the relapse rate can be up to 35–40 % [113]. Administration in combination with various other agents including hydrocortisone, heparin, and sodium bicarbonate has been reported to improve response [114].

Intravesical hyaluronic acid (grade of recommendation C, level of evidence 4) has been used with long-lasting moderate efficacy and no side effects, but a recent multicenter study found no significant efficacy (Interstitial Cystitis Association—Physician perspectives, unpublished data).

Chondroitin sulfate (grade of recommendation C, level of evidence 3) demonstrated a 33 % response rate [115]. Combined instillation of hyaluronic acid and chondroitin sulfate in refractory interstitial cystitis resulted in significant symptomatic improvement [116]. Botulinum toxin (grade of recommendation C, level of evidence 4) inhibits the release of calcitonin gene-related peptide and substance P from afferent nerves, thus decreasing pain. Small studies of intravesical Botox into the bladder wall reported symptom relief in IC patients, without significant adverse events [117].

5.12.3.3 Pain Modulation

Tricyclic antidepressants (TCAs), especially amitriptyline, are known to have pain-reducing effects (grade of recommendation B, level of evidence 2). One recent RCT of amitriptyline evaluated 50 patients with IC. Improvement in overall symptom scores, as well reduction in pain and urgency, were significantly greater in the treatment group ($P < 0.001$) (Tables 5.2 and 5.3) [118].

5.12.3.4 Immunologic Modulation

Immune stimulants and suppressants have been used in BPS/IC. Intravesical Bacillus Calmette–Guérin (BCG) (grade of recommendation D, level of evidence—no efficacy) was initially reported to have some benefit in BPS/IC; however, a subsequent randomized placebo controlled trial of BPS/IC patients who met the

Table 5.2 Oral medications for treatment of BPS/IC: results

Drug	RCT	Success (%)
Amitriptyline; tricyclic antidepressant	Yes	42
Antibiotics	Yes	48
Cimetidine	Yes	65
Hydrocortisone	No	80
Cyclosporin	No	90
Hydroxyzine	Yes	31
L-Arginine	Yes	Not effective
Nifedipine	No	87
Quercetin	No	92
Sodium pentosan polysulfate	Yes	33
Suplatast tosilate	No	86

Adapted from Hanno P, Baranowski A, Fall M et al. Painful bladder syndrome (including interstitial cystitis). In: Abrams P, Cardozo L, Khoury S, Wein A (eds) Incontinence. Health Publication Ltd., Plymouth, 2005, pp 1457–1520 [52]

Table 5.3 Intravesical medications for treatment of BPS/IC: results

Drug	RCT	Success (%)
DMSO	Yes	70
BCG	Yes	Conflicting RCT data as to efficacy
Resiniferatoxin	Yes	No proven efficacy
Hyaluronic acid	Yes	No proven efficacy
Heparin	No	60
Chondroitin Sulfate	No	33
Lidocaine	No	65
PPS	Yes	Suggestion of possible efficacy

Adapted from Hanno P, Baranowski A, Fall M et al. Painful bladder syndrome (including interstitial cystitis). In: Abrams P, Cardozo L, Khoury S, Wein A (eds) Incontinence. Health Publication Ltd., Plymouth, 2005, pp 1457–1520 [52]

NIDDK research criteria, showed that there was no statistical difference at 34 weeks [119].

Cyclosporin is an immunosuppressant used in organ transplantation (grade of recommendation C, level of evidence 2). It was tested in a small, open-label study of 11 patients. The results were largely positive and bladder pain decreased or stopped completely in 10 patients [120].

Suplataste tosilate (IPD-1151T) is a new immunoregulator that selectively suppresses IgE, IL-4, and IL-5 (grade of recommendation C, level of evidence 4). Treatment in 14 women for 1 year resulted in a significantly increased bladder capacity and decreased urgency, frequency, and pain [121].

5.12.3.5 Multimodal Medical Therapy

To manage the multiple pathophysiological mechanisms of BPS/IC, a multimodal approach combining agents from different classes should be utilized. Attacking the disease at several points seems to improve the therapeutic response. One common multimodal approach is:

1. Restoring epithelial function through heparinoid administration.
2. Treatment of neural activation and pain with tricyclic agents.
3. Allergy control using anthistaminic agents.

In cases with advanced disease, intravesical treatment may be required. We recommend combination intravesical therapy for patients who experience significant flare of symptoms after remission.

5.12.3.6 Neuromodulation

Sacral Nerve Neuromodulation (SNN) is a promising treatment for the management of symptoms in refractory BPS/IC patients. A recent study demonstrated a long-term significant improvement in pain levels, with a reduction of opiate use in a group of SNS patients [122].

Posterior tibial nerve stimulation showed positive effect on pelvic pain management refractory to other previous treatments but the lack of control study suggests further studies to confirm the real efficacy [123].

5.12.4 Procedural Intervention

BPS/IC is a chronic and debilitating disease with an impairment of quality of life due to disabling symptoms. Surgical options should be considered only when all conservative treatment has failed.

Laser resection, augmentation cystoplasty, cytolysis, cystectomy, and urinary diversion may be the ultimate option for refractory BPS/IC patients. Continent diversion may have better cosmetic and lifestyle outcome, but recurrence is a real possibility.

5.13 BPS/IC and HPFD

5.13.1 Behavioral Modification

Improvement in pelvic floor function begins with patient education about the normal function of the pelvic floor at rest and bladder retraining including dietary modification, fluid schedules, bowel programs, timed voiding. Heat applied with heating pads is also very helpful.

5.13.2 Pelvic Floor Rehabilitation

Physical therapy consists of teaching the patients how to contract or relax their pelvic floor. Manual

therapy with direct myofascial release, joint mobilization and strengthening and stretching of levator ani muscles can aid patients improve pelvic floor awareness. Surface electrodes applied to the perianal area or a vaginal probe can monitor levator ani activity and teach the patient how to relax pelvic floor muscles. The Thiele massage was first used for the management of coccydynia. This technique encompasses a deep vaginal massage employing a back-and-forth motion over the pelvic floor musculature as well as a myofascial release maneuver for trigger points. Significant pain improvement can be obtained with biofeedback aimed to learn control over voiding, to relax pelvic floor spasm [124].

5.13.3 Trigger Point Injection

The Trigger Point Injection (TPI) has the objective to inactivate painful trigger points. TPI requires a series of three to five injections utilizing the "wet" (with local anesthetic) or "dry" technique with the advancement of 1–3 mm surrounding the trigger point and its associated taught band. The results show that at least 72 % of patients report an improvement greater than 50 %.

5.13.4 Botulin Toxin Therapy

The mechanism of pain relief with Botox involves muscle relaxation, a direct antinociceptive activity and a secondary decrease in central sensitization. Botulin toxin A injected in the puborectalis and pubococcygeus muscles decreases pain scores and improves quality of life and sexual function scores [125].

5.14 Conclusion

Management of BPS/IC patients requires not only observation of the bladder but general contextual considerations as well. CPP is a very common disorder affecting 16 % of women. There are many causes of CPP and their accurate evaluation is very important.

Patients with bladder tenderness alone respond better to treatment than patients with multiple tender trigger points, possibly because only the bladder is involved in the disease process.

Treatment of this condition deeply impacts the quality of life of patients. While many therapeutic options are available, the earlier the diagnosis, the easier the treatment. Multimodal therapy remains the gold standard in the management of BPS/IC and OPF patients.

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Chronic Pelvic Pain Syndromes in Males

6

Kobi Stav

6.1 Chronic Prostatitis

Chronic Prostatitis (CP) is the most common urologic diagnosis in men younger than age 50 years and the third most common urologic diagnosis in men older than age 50 years after benign prostatic hyperplasia (BPH) and prostate cancer [1]. The estimated prevalence of CP ranges from 2.7 to 7.5 % [2, 3]. Most patients have a poor understanding of their condition, and many are disappointed with the results of treatments. Moreover, many clinicians are frustrated in their attempts to treat patients with prostatitis. The tendency is to tell the patient that he must simply learn to live with his condition or to refer the patient elsewhere. It is now recognized that prostatitis occurs in several separate forms or syndromes.

6.1.1 Anatomy and Physiology of the Prostate

The prostate is a multilobular gland located at the base of the bladder. It has a pyramidal shape, with its base contiguous with the bladder and its apex pointing toward the triangular ligament. The apex of the prostate is continuous with the striated urethral sphincter. Coursing through the gland in a vertical plane is the prostatic or first segment of the urethra. The prostatic urethra presents on its lower posterior surface an eminence, the verumontanum. The ejaculatory ducts enter the prostate at the posterolateral aspects of the base on each side, traverse the gland, and open into the prostatic urethra on the utricle of the verumontanum [4].

The prostate is composed of five groups of lobes: one anterior, two lateral, one median, and one posterior lobe which lies behind the ducts. Circular and longitudinal muscle fibers are scattered profusely throughout the glandular structures. These fibers are connected with those of the bladder. The prostate is composed of approximately 30 % fibromuscular stroma and 70 % glandular tissue. The glandular portions of the prostate are of compound tubular type. These glands form about 20 ducts which drain into the urethra on each side of the verumontanum. At the angle dividing the preprostatic and prostatic urethra, the ducts of the transition zone arise and pass beneath the preprostatic sphincter to travel on its lateral and posterior side. Normally, the transition zone accounts for 10 % of the glandular tissue of the prostate. The transition zone gives rise to BPH, which expands to compress the fibromuscular band into the capsule. The peripheral zone makes up the bulk of the prostatic tissue (70 %) and covers the posterior and lateral aspects of the gland. Most of the prostatic cancers (70 %) arise in this

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zone, and it is the zone most commonly affected by chronic prostatitis [5].

The fibromuscular stroma condenses about the periphery of the glands to form a capsule. The prostate is supported by the endopelvic fascia, the puboprostatic ligaments in front, the triangular ligaments below, and the fascia of Denonvillier posteriorly, separating the prostate from the rectum [6, 7].

The blood supply to the prostate derives from the inferior vesical, internal pudendal, and middle hemorrhoidal arteries. The veins form a plexus on each lateral aspect of the prostate and together with veins from the bladder drain into the internal iliac veins. The nerve supply comes from the pelvic plexus. Parasympathetic nerves end at the acini and promote secretion. Sympathetic fibers cause contraction of the smooth muscle of the capsule and stroma [8].

In women the paraurethral glands are homologous to the male prostate with matching histological appearance. These glands are located caudal to the bladder in a position similar to the ventral prostate of the male. The prostate is the largest accessory sex gland of the male and is dependent on the presence of androgenic hormones. Male ejaculate is approximately 3–5 mL and is composed of spermatozoa and the seminal plasma. The spermatozoa represent less than 1 % of the total ejaculate. The major contribution to the volume of the seminal plasma derives from the seminal vesicles (3 mL), from the prostate (1.5–2 mL) and from the Cowper's gland (0.5 mL). The seminal plasma contains high concentrations of potassium. Zinc, citric acid, fructose, free amino acids, prostaglandins, and enzymes, most notably acid phosphatase, diamine oxidase, lactic dehydrogenase, alpha-amylase and prostatic specific antigen (PSA) [9]. PSA is a glycoprotein that contains 7 % carbohydrate and is almost exclusively in the epithelial cells of the prostate [10]. PSA is a serine protease and an esterase. Its biologic role is to lyse the ejaculate clot [11].

6.1.2 Diagnosis and Classification

Medical history and physical findings may suggest the diagnosis of a prostatitis syndrome, but are not confirmatory. Many of the signs, symptoms, and physical findings of prostatitis syndromes are often indistinguishable. Likewise, Cystoscopy, Urodynamic study, and radiographic studies may assist in differential diagnosis, but they do not confirm the diagnosis of prostatitis.

Microscopic examination of the expressed prostatic secretions is important in the diagnosis and classification of prostatitis. The counts of bacteria and leukocytes in concomitant specimens obtained from the urethra, midstream bladder urine, and prostatic expression should be compared. The localization test (also known as the Meares-Stamey four-glass test), segmentally assesses inflammation and cultures of the male lower urinary tract [12]:

1. **VB1**—first voided 10 mL of urine—represents urethral colonization.
2. **VB2**—next voided 200 mL of urine—represents bladder urine.
3. **EPS**—obtained from the expressed prostatic secretion after prostatic massage.
4. **VB3**—the first 10 mL of urine that is voided after prostatic massage.

Based on clinical experience with this test, a classification system describing four categories of prostatitis was described by Drach and colleagues in 1978: acute bacterial prostatitis, chronic bacterial prostatitis, nonbacterial prostatitis and prostatodynia [13]. This traditional classification was based on an analysis of prostatic fluid, which included microscopy (examination for white blood cells, inflammatory cell clumps, mucus debris, oval fat bodies, and macrophages) and culturing (identifying a traditional uropathogen). The limitations of this classification system included desertion of the meticulous four-glass test [14, 15], the understanding that patients respond to specific treatments regardless of the type of prostatitis, the realization that patients would be diagnosed with chronic bacterial prostatitis quite rarely, and that in many cases patients would not be easily classified into one of the categories of prostatitis [16]. These limitations led to the development of a new classification system—**the National Institutes of Health (NIH) classification system** [17]. The NIH classification of the prostatitis syndromes

has now become recognized as the best system for clinical practice and research. In this system, patients are classified into four main categories:

Category I—acute bacterial prostatitis.
Category II—chronic bacterial prostatitis.
Category III—the presence of chronic genitourinary pain (chronic pelvic pain syndrome—CPPS) in the absence of uropathogenic bacteria detected by standard microbiologic methodology. This is further categorized into:
Category IIIA—inflammatory CP/CPPS—based on the presence of excessive leukocytes in EPSs or post-prostatic massage urine (VB3) or semen.
Category IIIB—noninflammatory CP/CPPS—no significant leukocytes in EPSs or post-prostatic massage urine (VB3) or semen.
Category IV—asymptomatic inflammatory prostatitis—the presence of significant leukocytes (and/or bacteria) in expressed EPS, post-prostatic massage urine (VB3), semen or histological specimens of prostate gland (tissue biopsies) in the absence of chronic pelvic pain.

The four-glass test for now is the gold standard diagnostic evaluation of prostatitis patients. However, studies have confirmed that clinicians have abandoned this complex, time-consuming, and expensive test [14–16]. The modified two-glass test is a simple and cost-effective tool to categorize patients with CP/CPPS [18]. The patient provides a midstream pre-massage urine specimen and a urine specimen (initial 10 mL) after prostatic massage. Microscopy and culturing of these two screening urine specimens allows categorization of the majority of patients presenting with a CP. Table 6.1 describes interpretation of the two and four-glass localization tests for CP/CPPS patients. The Two-glass test has 91 % sensitivity and specificity compared with the gold standard four-glass test [18].

MR imaging plays an important role in the initial detection, localization, and staging of prostate cancer and the assessment of posttreatment changes in prostate cancer. However, detection of prostatitis on MR imaging remains a challenge. Histologically, chronic prostatitis is characterized by extracellular edema surrounding the involved prostatic cells with concomitant aggregation of lymphocytes, plasma cells, macrophages, and neutrophils in the prostatic stroma. This abundance in cells as compared with normal prostatic tissue may lead to an apparent diffusion coefficient (ADC) decrease because of decreased extracellular to intracellular fluid volume ratio.

The size and signal intensity of the prostate gland may be normal [19]. Prostatitis may present as a low T2 signal intensity in the peripheral zone which may be focal or diffuse, usually without contour deformation of the prostate. Yet, there are other possible causes of low T2 signal intensity, including hemorrhage, scarring, atrophy, effects of radiation therapy, cryosurgery, or hormonal therapy. Moreover, current anatomic MR imaging cannot differentiate prostatitis from BPH and sometimes it may mimic low-grade prostate cancer in the peripheral zone [20, 21]. It is possible, that in the near future, with improvement of the MRI techniques, we will be able to use this modality as a diagnostic tool for prostatitis. In patients who have symptoms of chronic prostatitis that are resistant to therapy, MR imaging may be performed to exclude an abscess or other associated structural abnormalities [22].

6.1.3 Symptoms and Clinical Presentation

Category I Prostatitis is an acute bacterial infection of the prostate gland. Generally it is recognized easily because its clinical manifestations are characteristic: an acute onset of perineal and suprapubic pain, external genitalia discomfort combined with storage and voiding symptoms. In addition, usually significant systemic symptoms occur, including fever, chills, malaise, nausea and vomiting, and even frank septicemia with hypotension.

In Category II, patients are usually presented with recurrent lower urinary tract infections. The most common organisms are the Enterobacteriaceae

Table 6.1 Interpretation of the 4 and 2 glass localization tests for CP/CPPS patients

		4 Glass test				2 Glass test	
	Specimen	VB1	VB2	EPS	VB3	Pre-massage	Post-massage
Category II	Leukocytes	–	+/–	+	+	+/–	+
	Culture	–	+/–	+	+	+/–	+
Category IIIA	Leukocytes	–	–	+	+	–	+
	Culture	–	–	–	–	–	–
Category IIIB	Leukocytes	–	–	–	–	–	–
	Culture	–	–	–	–	–	–

family of gram-negative bacteria, which originate in the gastrointestinal flora. Escherichia coli are identified in 65–80 % of infections [23, 24]. Besides Enterobacteriaceae, other microorganisms such as Gram-Positive Enterococci, anaerobic bacteria, *Staphylococcus saprophyticus*, and *Staphylococcus aureus*, have been described [25, 26]. The nidus of infection is the prostate gland [27]. Patients may be relatively asymptomatic between acute episodes, or they may present with a long history of a CPPS [28].

Pain is the main symptom in category III, accompanied by variable voiding and sexual dysfunction symptoms. The clinical presentation of category IIIA is indistinguishable from category IIIB. The pain may be localized to suprapubic area, penis (referred to the tip of the penile urethra), perineum, testes, groin or low back. Some patients complain of painful ejaculation [29, 30]. Many patients have storage and voiding symptoms such as urinary urgency, increased frequency, nocturia, dysuria, hesitancy, diminished urinary stream, and poor interrupted flow.

Records of 1563 category III patients from four databases from Canada, Germany, Italy, and the United States showed that the most prevalent location of pain was the Perineum (63 %) followed by testicular pain (58 %), pain in the pubic area (42 %) and penis (32 %); reports of pain during ejaculation and voiding were 45 % and 43 %, respectively [31].

The neurologic and genitourinary physical examination is usually normal, except for some patients who present tender prostatic and paraprostatic tissues on digital rectal examination. Sexual disturbances such as erectile dysfunction and premature ejaculation are common complaints as well [2, 32–34]. The quality of life of these patients is usually severely impaired [35, 36]. Depression, emotional distress, and anxiety are common characteristics of patients with CPPS [37, 38].

Category IV is asymptomatic prostatitis and is diagnosed when inflammatory cells are identified on prostate biopsy or leukocytes are noted on semen analysis during urologic evaluation for other reasons (elevated PSA, suspicious digital rectal examination of the prostate, infertility evaluation, etc.…). The clinical significance of this type of prostatitis is vague, and treatment is usually based on the primary reason for the urologic evaluation.

6.1.4 Symptom Evaluation

In CP/CPPS there are no objective tools that can evaluate outcomes. Therefore, it is very important to have a validated and a reproducible instrument to measure the symptoms and quality of life of patients for use in research protocols as well as clinical practice. The NIH (National Institute of health) Chronic Prostatitis Collaborative Research Network (CPCRN) developed the NIH-CPSI (chronic prostatitis symptoms index) which is currently the most acceptable validated tool for symptom assessment is CP/CPPS patients [39]. The index consists of nine items that address pain (location, severity, and frequency), urinary function (incomplete emptying, frequency, and urgency) and impact on the quality of life (effect of symptoms on daily activities). The NIH-CPSI has shown validity in clinical trials [40] and has now been accepted by the international prostatitis

research community as an accepted outcome measure [41].

6.1.5 Etiology

The etiology for ***chronic bacterial prostatitis*** (category II) is unknown. The histological findings are nonspecific. Usually, the inflammatory reaction is less marked and more focal than that seen in cases of acute bacterial prostatitis. Infiltration by macrophages and plasma cells is prominent within and around the acini, along with focal invasion of lymphocytes. Because these findings are very common in patients without evidence on prostatitis, they are not diagnostic [42].

Transrectal prostatic ultrasonography often detects prostatic calculi of variable size and number in adult prostates, with an incidence of about 75–100 % [43]. These stones are typically small and tend to occur in clusters. In patients with chronic bacterial prostatitis, the stones are usually multiple and large. These stones can become infected and may serve as a source of bacterial persistence and relapsing infections [44]. The clinical features of men who have chronic bacterial prostatitis with or without stones are indistinguishable. Potential risk factors for prostate bacterial colonization include: unprotected penetrative ano-rectal intercourse, urinary tract infection, phimosis, acute epididymitis, indwelling urethral catheters, transurethral surgery, and intraprostatic ductal reflux [45]. Crystallographic analysis of prostatic stones indicates that certain calculi are composed of constituents commonly found in the urine but foreign to prostatic secretions. This observation suggests that intraprostatic reflux of urine is important in the formation of some prostatic stones [46].

The etiology of ***CPPS*** (category III) is unknown as well. The various hypotheses include genetic, infection (cryptic or otherwise), endocrine, anatomical, physiological, neuropathic, neuromuscular, immune (including autoimmune), and psychological mechanisms, but none of those has been proven to cause all or even most cases of CPPS.

Studies have excluded **infectious agents** as causative agents including fungi, viruses, trichomonads, bacteria, Mycoplasma, and Ureaplasma species [47, 48]. The most controversial supposed agent in CPPS is Chlamydia trachomatis. It is plausible that this organism has an etiologic role in prostatitis since it is the most common causative agent of acute epididymitis and urethritis in young men [49]. However, many studies showed that C. trachomatis appears to play a minor role, if any, in the etiology of CPPS. These studies used cultural and serologic tests of prostatic fluids, immunofluorescent tests of the serum, urethra, and prostate [48, 50–52].

Dysfunctional voiding and functional bladder outlet obstruction is another possible etiology for CPPS. Incomplete funneling of the bladder neck as well as vesicourethral dyssynergic patterns were demonstrated on videourodynamic of many patients with CPPS [53–55]. High pressure voiding may lead to autonomic overstimulation of the perineal-pelvic neural system with subsequent development of a chronic neuropathic pain. However, since painful voiding is quite common in CPPS patients—it is plausible that the functional obstruction is the outcome and not the cause of CPPS.

Intraprostatic reflux due to high pressure voiding has been hypothesized as a central etiologic mechanisms involved in the pathogenesis of CPPS as well. High levels of urate and creatinine were noted in EPS. It was postulated that this may be caused by urine reflux into the prostatic ducts [56]. The inflammatory process in the prostate may be due to chemically induced inflammation, secondary to noxious substances in the urine that have refluxed into the prostatic duct. Another study showed that carbon particles instilled into the bladder can be found in the EPS macrophages and prostatic acini and ductal system after surgery in men with CPPS [57].

Prostatic calcifications are common in patients with CPPS as well [58]. It has been shown that patients with CPPS or chronic bacterial prostatitis have significantly higher prostate tissue pressure than controls, measurable with transperineally inserted pressure transducers [59]. It was postulated that inflammation resulting from chemical,

bacterial, or immunologic stimulation is the main cause for such high pressure and maybe for the formation of intraprostatic stones.

There is some evidence that CPPS may be secondary to **immunologically mediated inflammation**, due to some unknown antigen, or perhaps even related to autoimmunity. Elevated levels of cytokines (IL-1, IL-8, IL-10, and tumor necrosis factor) in the seminal plasma and prostatic secretions have been detected in men with CPPS compared with normal individuals, suggesting an active inflammatory process in the male genital tract [60–62].

This inflammatory reaction may be mediated by an adaptive immune response directed against genital tract antigens [63]. PSA has been suggested as the self-antigen [64]. Fibrinogen and complement C3 have been identified in prostatic biopsy specimens from patients with CPPS [65]. High levels of nonspecific IgA and IgM antibodies were demonstrated in prostatic fluid of patients with CPPS [52].

Psychological factors may play a role in the pathophysiology of CPPS as well. A case control study confirmed that depression and panic disorders are significantly more common in men and women with chronic pelvic pain conditions than in control subjects [66]. It has been shown that hypochondriasis, depression, somatization, anxiety, and hysteria are more common in CPPS patients than control [67–69]. Depressive symptoms, pain intensity, pain catastrophizing, and pain-contingent resting significantly predict a poorer quality of life in patients with CPPS regardless of age and urinary status [70, 71]. This indicates that negative cognitive assessment of the pain experience may be a primary target for psychosocial interventions.

Dysfunctional hypothalamic-pituitary-adrenal axis function was demonstrated in men with chronic pelvic pain syndrome, reflected by insignificant increases in awakening cortisol levels [67]. Comparing to age-matched asymptomatic healthy controls, CPPS patients had significantly higher levels of progesterone, androstenedione and testosterone and significantly lower levels of corticosterone, aldosterone and 11-deoxycortisol. Interestingly, the National Institutes of Health-Chronic Prostatitis Symptom Index total and pain domain scores correlated positively with 17-hydroxyprogesterone and aldosterone ($P<0.001$) and negatively with cortisol ($P<0.001$) concentrations [68]. These findings provide further insights into the biologic basis of CP/CPPS suggesting that the hormonal imbalance is influenced by symptom severity.

6.2 Treatment

At present, antimicrobial agents are the treatment of choice for ***chronic bacterial prostatitis*** (category II). Numerous antibiotics have been tested including erythromycin, minocycline, doxycycline, and cephalexin, in most cases with quite disappointing results. Cure rates of 30–40 % with long-term TMP-SMX (4–16 weeks) have been documented [72, 73]. Direct transperineal injection of antibiotics directly into the caudal prostate showed outstanding success in 24 selected patients with refractory chronic bacterial prostatitis. Remission periods of at least 6 months were obtained in 70 % of these patients after one or two infiltrations. Seven patients had relapse after remission periods ranging from 13 months to 7 years [74]. Fluoroquinolones cover the spectrum of gram-positive and gram-negative bacteria and have been shown to penetrate into prostatic tissue in concentrations approaching or exceeding by several fold those in serum [75, 76]. In a comparative study, 4–6 weeks of norfloxacin was shown to be more effective than TMP-SMX for therapy of chronic bacterial prostatitis caused predominantly by *E. coli* [77].

Prolonged therapy (4–6 weeks) of ciprofloxacin appeared to eradicate *E. coli* prostatitis in 85 % of patients [78]. A prospective open-label study on 116 patients with chronic bacterial prostatitis showed that administering once daily 500 mg of levofloxacin for 28 days a clinical success rate (cured and improved patients) of 77, 66, and 62 % at 1 month, 3 months, and 6 months was achieved [79]. Lower rates of eradication have been associated with therapy given for shorter periods and with prostatitis caused by agents other than *E. coli* [80]. Chronic infections

unresponsive to such treatment are generally managed by continuous, suppressive, low dose of TMP-SMX or nitrofurantoin; neither tends to produce bacterial resistance [81].

Theoretically, removal of the infected material, including potentially infected calculi may be effective in patients with either relapsing or refractory chronic bacterial prostatitis secondary to bacterial persistence within the prostate gland. Patients with prostatic calculi not controlled by medical therapy may be candidate for transurethral prostatectomy. In such circumstances, the procedure may be quite challenging, since the greatest foci of stones are located in the peripheral zone of the prostate [82]. There is no substantial proof in the literature as to the efficacy of prostate surgery in category II chronic prostatitis.

Treating patients with ***CPPS*** (category III) is even more challenging. It is vital to reassure the patient that his condition is not contagious or dangerous and will not lead to serious complications or cancer.

Up to 40 % of the patients with CPPS have symptomatic improvement with antibiotic therapy [25, 48, 83, 84]. Possible explanations for the positive effect of antibiotic on CPPS patients are: independent anti-inflammatory effect of some antibiotics, placebo effect and suppression of noncultured microorganisms. However, two recent randomized placebo-controlled studies which evaluated the efficacy of 6 weeks of levofloxacin [85] and ciprofloxacin [86] on antibiotic-naive patients with CPPS showed no significant difference between drugs and placebo.

Since many patients with CPPS show functional obstruction of the bladder neck and prostate, alpha-adrenergic blocking agents were considered as an important and efficient treatment for this syndrome [87–89]. However, more recent studies concluded that these drugs are no better than placebo [86, 90]. A multicenter, randomized, double-blind, placebo-controlled trial was conducted in order to assess the efficacy of alfuzosin in reducing symptoms in men with CPPS. 272 men were randomized to alfuzosin or placebo. The NIH-CPSI scores did not differ between the groups after 12 weeks of treatment [90].

A short course of anti-inflammatory agents is often helpful for exacerbation of pain and dysuria [91]. Oral corticosteroids showed no benefit over placebo in a randomized trial [92]. A modest improvement with rofecoxib (cyclooxygenase-2 inhibitor) was demonstrated in a placebo-controlled randomized trial [93].

Patients with tension myalgia of the pelvic floor respond best to treatment with muscle relaxants, alone or in combination with an alpha-adrenergic blocking agent [81]. A prospective double-blind crossover study comparing phenoxybenzamine, baclofen, and placebo in 27 patients with CPPS, showed symptomatic improvement in 37 % of the patients treated with baclofen compared with 8 % treated with placebo [94]. A double-blind comparative study between diazepam and minocycline in CPPS patients found no difference in symptom improvement [95].

Hormonal treatment with 5α-reductase inhibitors may reduce the prostatic glandular tissue and therefore improve voiding parameters, especially in patients with a BPH component. A randomized comparison between finasteride and placebo in patients with CCPS showed a significant improvement in the Prostatitis Symptom Severity Index and BPH symptom score. However, there was no difference in pain between the two groups [96]. A randomized placebo-controlled trial evaluated the efficacy of 6 months therapy with finasteride versus placebo [97]. The improvement in the NIH-CPSI and in subjective overall assessment was similar between groups.

A prospectively randomized study compared the efficacy of mepartricin (which lowers estrogen levels in the prostate) versus placebo in 26 patients with CPPS [98]. A significant decrease in the total NIH-CPSI score from 25.0 to 10.0 was demonstrated in the mepartricin group compared to reduction from 25.0 to 20.0 in the placebo group. Moreover, a statistically significant decrease was observed with regard to pain (from 11.0 to 4.0 and from 10.0 to 8.0, respectively).

At the first half of the twentieth century, prostatic massage was the principal treatment for

prostatitis. However, during the years, this practice was abandoned by urologists due to lack of good evidential support. The benefit from prostatic massage is believed to be derived from a combination of several factors, including expression of prostatic secretions, relief of pelvic muscle spasm, physical disruption of any protective biofilm, improved circulation, and consequently improved antibiotic penetration [99, 100]. Repetitive prostatic massages (one to three times per week) combined with antibiotics was evaluated in patients suffering from chronic bacterial prostatitis ($n=52$) and CPPS ($n=19$). Overall, 40 % of this heterogeneous group had complete resolution of symptoms [101]. A more recent study showed no significant difference in response between patients treated with antibiotics alone and those treated with antibiotics and prostatic massage [102]. Despite prostatic massage having been practiced for a long time, there is a paucity of literature on this subject. There is not a single comparative study that has evaluated prostatic massage alone as a therapy for CPPS. In the absence of an ideal sham procedure to mimic prostatic massage, it may never be possible to eliminate the placebo effect, and thus the most robust evidence on this subject may continue to be elusive [100]. Frequent ejaculation may achieve the same function as prostatic massage [103].

It is believed that prolonged chronic tension, distention, or distortion in the muscle bands (e.g., in the perineum) may lead to painful trigger points that are responsible for pain in patients with chronic pelvic pain syndromes. Anderson et al. [104] documented relationships between trigger point sites and pain symptoms in 72 men with CPPS. Pain sensation at each anatomical site was reproduced by palpating at least 2 of 10 designated trigger points. Furthermore, 5 of 7 painful sites could be reproduced at least 50 % of the time ($p<0.05$). The most prevalent pain sites were the penis in 90.3 % of men, the perineum in 77.8 % and the rectum in 70.8 %. Puborectalis/pubococcygeus and rectus abdominis trigger points reproduced penile pain more than 75 % of the time ($p<0.01$). External oblique muscle palpation elicited suprapubic, testicular, and groin pain in at least 80 % of the patients at the respective pain sites ($p<0.01$). This report showed a relationship between myofascial trigger points and reported painful sites in men with CPPS. Manual release therapy of these trigger points may be an effective therapeutic approach in some patients [105]. Combining physical therapy and biofeedback may improve symptoms as well. A study on 31 males with CPPS showed a reduction of the CPSI from 23.6 (range 11–34) to 11.4 (range 1–25, $p<0.001$) after biofeedback physical therapy [106].

Phytotherapy for CPPS patients is another potential treatment for CPPS patients. Some plant extracts have been shown to have alpha-adrenergic blockade activity, effects on detrusor contractility, anti-inflammatory activity, and 5-alpha reductase properties. A prospective, randomized, double-blind, placebo-controlled phase 3 study compared a pollen extract (Cernilton) to placebo in men with CPPS [107]. 139 men were randomly allocated to the pollen extract ($n=70$) or placebo ($n=69$). The individual domains pain ($p=0.0086$) and quality of life (QoL; $p=0.0250$) as well as the total NIH-CPSI score ($p=0.0126$) were significantly improved after 12 weeks of treatment with pollen extract compared to placebo. The bioflavonoid Quercetin was shown to be superior to placebo in a small prospective randomized, double-blind, placebo-controlled trial [108]. Patients taking placebo had a mean improvement in NIH symptom score from 20.2 to 18.8 (not significant), while those taking the bioflavonoid had a mean improvement from 21.0 to 13.1 ($P=0.003$). Twenty percent of patients taking placebo and 67 % of patients taking the bioflavonoid had an improvement of symptoms of at least 25 %. It has been shown that Serona repens (saw palmetto berry) has antiandrogenic actions and anti-inflammatory activity in prostatic epithelial cells [109]. Moreover, Serona repens has been shown to reduce clinical progression rates in men with mild symptoms of bladder outlet obstruction. It also led to improvements in urinary symptoms, QOL scores, and urinary flow rates [110]. However, no published randomized placebo-controlled study has compared the efficacy of Serona repens to placebo in CPPS patients yet.

One of the theories regarding the pathophysiology of symptoms in CPPS is neuropathic pain [111]. Therefore, there is a rational to treat these patients with Neuromodulators. A randomized, double-blind, placebo-controlled trial evaluated the efficacy of pregabalin in patients suffering from CPPS [112]. Compared with the placebo group, men assigned to receive pregabalin had a higher Global Response Assessment rate (31.2 and 18.9 %; $P=0.02$), and showed improvement in total McGill Pain Questionnaire score ($P=0.01$). However, there was no difference between groups in the NIH-CPSI total score, which was the primary end-point. Antidepressants (e.g., amitriptyline), which are well established in the management of other chronic pain syndromes, are sometimes used with some success, but have not been formally evaluated in CPPS.

Electrical Neuromodulation techniques used for chronic pelvic pain conditions include SNS (sacral nerve stimulation), PTNS (percutaneous tibial nerve stimulation) and pudendal nerve stimulation. Only SNS and PTNS are currently approved by the US Food and Drug Administration for the treatment of urinary symptoms and none of these methods are acknowledged as standard therapies for treating chronic pelvic pain syndromes. Although research on PTNS method for male CPPS is sparse, Kabay et al. reported on a trial of 89 men with the diagnosis of medically refractory NIH Category IIIB CPPS who were randomly assigned to receive either PTNS or sham treatment. The PTNS group reported a significant improvement in urinary symptom and pain scores, yet follow-up after the 12-week treatment period was not reported [113, 114]. The most common technique of SNS places a multipolar lead through S3 sacral foramen to stimulate the nerve root. In recent years, several reports have been published regarding the use of SNS to treat IC/BPS symptoms, most commonly using Interstim (Medtronic, Minneapolis, MN). Most of the literature pertaining to SNS presents case series including different types of chronic pelvic pain [113]. Zabihi et al. [115] studied the use of bilateral sacral electrodes and reported >50 % pain improvement in 10 out of 23 patients with debilitating pelvic pain (IC/BPS and male CPPS) who had bilateral sacral electrodes implanted. Currently, there is a lack of studies specifically utilizing SNS and PTNS in men with CP/CPPS. Conclusions of the existing literature must be carefully considered because of paucity of data and the limitations of small studies.

Other potential treatments that may ease symptoms of CPPS patients include: anticholinergic drugs to control bladder storage symptoms (such as urinary urgency and frequency), avoiding irritating foods and beverages (such as caffeine, spicy dishes, alcohol, citrus, sparkling drinks), acupuncture and anxiolytic drugs for patients with significant emotional distress [116–118].

Surgical interventions may be offered to patients who have been refractory to other treatments. Some anecdotal experiences exist for radical prostatectomy and transurethral resection of the prostate; however, these have been largely abandoned due to lack of reliable data to support their use, the potential for significant morbidity, and questionable benefit [119].

Minimally invasive treatments that were evaluated only in small pilot studies include: sacral magnetic stimulation [120], intraprostatic injection of botulinum toxin [121, 122], percutaneous posterior Tibial nerve stimulation [114], and transurethral microwave hyperthermia [123]. Treatments proven to be ineffective include balloon dilatation [124] and transurethral needle ablation [125].

Transurethral microwave thermotherapy (TUMT) was found to be effective in a randomized, double-blind, sham-controlled study. Seventy percent of the patients showed greater than 50 % improvement in symptoms [126]. A more recent study [127] demonstrated similar results, with 63 % of patients maintaining at least a 50 % improvement of pain symptoms 12 months after the procedure.

The use of extracorporeal shockwave therapy (ESWT) has been evaluated for the treatment of CP/CPPS. A placebo-controlled, prospective, randomized, double-blind study of 60 patients noted a significant improvement of pain, quality of life, and voiding symptoms when compared to placebo over a 12-week follow-up period.

This procedure was performed without anesthesia or any noted significant side effects [124]. This study provides level-1 evidence for a potentially promising new therapy for CP/CPPS, but further evaluation of ESWT appears to be warranted.

6.2.1 CP/CPPS and Overactivity of the Bladder Neck and Pelvic Floor

Pelvic floor muscles (PFM) have a close anatomic relation and the same innervation as pelvic visceral organs (e.g., prostate gland). Therefore, the pain of each imitates the pain of the other; this means that increased tension, spasm, and trigger points of PFM may mimic the symptoms of real prostatitis [129]. Some studies have demonstrated PFM tender points in men with CPPS and pain relief after myofascial release of trigger points [31, 130–132]. Another study showed that men with CPPS have significantly lower PFM mobility compared with those without CPPS [133]. These findings may indicate that PFM overactivity is an important factor in CPPS.

The main symptom of CP/CPPS patients is pain. However, a variety of storage and voiding symptoms are associated as well. The causes for these symptoms are unknown. Proposed causes include: obstruction of the urethra, bladder neck dyssynergia or hypertrophy of the bladder neck [82, 134, 135]. Meares postulated that the basis of symptoms in CP/CPPS patients is smooth muscle spasm of the bladder neck and prostatic urethra causing elevated pressures in the prostatic urethra, resulting in intraprostatic and ejaculatory duct urinary reflux, which leads to chemical inflammation [72].

Videourodynamic studies demonstrated that many patients with CP/CPPS show "spasm" of the prostatic urethra and bladder neck. The main findings were reduced urine flow rate, incomplete relaxation of the bladder neck/prostatic urethra, and high maximal urethral closure pressure at rest compared to an age and sex-matched control group. Another prominent feature was incomplete funneling of the bladder neck during voiding with accompanying urethral narrowing at the level of the external urethral sphincter [136]. Typically, the external sphincter is synergic in these patients and detrusor overactivity is unusual. Electromyography of the pudendal nerve is normal and urethral reflexes are intact. Meares called this condition "bladder neck/urethral spasm syndrome" since it is somehow a type of dysfunctional voiding disorder with characteristics of internal sphincter dyssynergia [72].

Some patients with CP/CPPS appear to suffer from tension myalgia of the pelvic floor [137, 138]. In these patients, pelvic pain is associated with sitting, running, or other physical activity. Digital rectal examination may demonstrate a painful prostate gland, discomfort or pain of the anus, paraprostatic tissues and muscles, and sometimes tenderness of the suprapubic area [139, 140].

A study on 103 patients with chronic pelvic pain showed that the majority of men had insufficient conscious control of their somatically innervated striated pelvic floor muscles. None of the patients was able to demonstrate a full range of pelvic floor contraction and relaxation repetitively and easily [141]. This finding can reflect a functional disassociation between the central nervous system and pelvic floor muscles.

About one half of the patients who were previously diagnosed as chronic prostatitis had bladder acontractility with nonrelaxing perineum (striated muscle spasm) during urodynamic studies and 36 % of the patients had detrusor overactivity with appropriate striated sphincter relaxation [142].

It was hypothesized that men are often categorized as suffering and empirically treated for chronic nonbacterial prostatitis when in fact they have chronic voiding dysfunction. This conclusion was based on a variety of videourodynamic findings including 54 % of patients with primary bladder neck obstruction, 24 % with functional obstruction localized to the membranous urethra (pseudodyssynergia), 17 % with impaired bladder contractility, and 5 % with an acontractile bladder [53, 54, 143].

However, a further study [144] showed that very few patients presenting with classic chronic

prostatitis symptoms had urodynamic abnormalities. Videourodynamic records of 201 men (age 18–50 years) who presented to the Urodynamic Unit with any lower tract symptoms (storage and/or voiding with or without pain) were compared with findings in 123 prostatitis patients. Only 37 (18 %) of 201 patients referred to the Urodynamic Unit had pain as a significant symptom and might have been diagnosed as having chronic prostatitis. Of these 37 patients, 4 (11 %) had definite obstruction, 6 (16 %) were equivocal, 6 (16 %) were hypocontractile, 1 (3 %) had pseudodyssynergia, and 7 (19 %) had normal findings. The remainder had abnormalities of bladder filling (hypersensitivity in 30 % and detrusor overactivity in 5 %). Of the 123 patients with prostatitis only 2 (1.6 %, $p=0.03$) had obstruction, 2 (1.6 %) had underactive detrusor, and 2 had urethral strictures. These findings dispute the benefits of urodynamics in CP/CPPS patients.

6.3 Interstitial Cystitis and Painful Bladder Syndrome

Although IC typically occurs less frequently in men than in women, contemporary series have suggested that male IC is much more common than was previously thought [145]. CP/CPPS in men may masquerade as IC/PBS, or vice versa. Both syndromes have rather similar and nonspecific symptoms and are actually diagnosed per exclusion. Probably, many men who are diagnosed as suffering from CP/CPPS actually have IC/PBS [146, 147]. The etiology of these enigmatic syndromes is unknown. However, the pathogenic mechanisms are theorized to be similar in men [148–150]. Undoubtedly, the symptoms of IC/PBS and CP/CPPS overlap to some extent.

Interestingly, men with CP/CPPS diagnoses have findings on cystoscopic [151], urodynamic [142], and potassium sensitivity testing [152] which are very similar to those of patients with IC. Miller et al. [147] observed petechial hemorrhages (glomerulations) in the bladder during hydrodistention under general anesthesia in 12 out of 20 men with CPPS. A study on 30 men who were initially thought to have CPPS and were refractory to treatment revealed the typical appearance of glomerulations after hydrodistention. Cold-cup bladder biopsies revealed increased number of mast cells in the mucosa in all patients [81]. Therefore, the diagnosis of IC/PBS should be considered in CPPS patients that are refractory to treatment.

Interstitial cystitis/painful bladder syndrome (IC/PBS) is discussed in details in a different chapter of this book.

6.4 Chronic Scrotal Pain Syndrome

Acute scrotal pain is usually a result of a well-defined condition such as trauma, torsion of the testis or its appendages, epididymitis, renal colic (referral pain), etc. However, the etiology of chronic testicular pain (orchialgia or orchidynia) is generally ambiguous and the management is often very challenging. It can be disabling and for many patients it is associated with anxiety about cancer. The desired goal of treatment is return to routine activity without significant use of analgesics.

Chronic testicular pain (CSP) was originally defined as intermittent or constant testicular pain for 3 months or longer which significantly interferes with daily activities of the patient so as to prompt him to seek medical attention [153]. The testis and epididymis have a common neuronal supply which can render it difficult to clinically distinguish the main site of pain [154]. Moreover, the majority of epididymectomy and orchiectomy specimens failed to show any pathological abnormalities [153, 155, 156]. Therefore, the definition of CSP was extended by Nickel et al. and included the testis, epididymis, and scrotum [157]. Thus, the terms chronic epididymitis, chronic orchialgia, and CSP are basically the same syndrome of chronic pain.

CSP is a frequently encountered problem in urologic practice, as 3 % of all office visits are attributable to this condition [158]. The estimated incidence for CSP is about 4/1000 [159]. The true incidence of CSP might be even higher since

an unknown proportion of men with CSP are referred to urologists. CSP can occur at any age but the majority of the patients are in their mid to late thirties [154].

The pain can be unilateral or bilateral, constant or intermittent, spontaneous or exacerbated by physical activities and pressure. It can remain localized to the scrotum or radiate to the groin, perineum, back or legs. On clinical examination the testis may be tender but in the majority of patients it is otherwise unremarkable [160].

Testicular pain can be due to defined testicular causes, referred pain or idiopathic. Defined causes of CSP include infection, tumor, intermittent testicular torsion (torsion de-torsion syndrome), varicocele, hydrocele, spermatocele, polyarteritis nodosa, trauma, and previous surgical interventions such as inguinal hernia repair or vasectomy [161]. The pain in postvasectomy patients usually appears during ejaculation. This fact suggests that obstruction or congestion of the vas or the epididymis may be the cause of pain. It has been suggested that the formation of a sperm granuloma at the vasectomy site may be responsible for the chronic pain [160]. Referred pain may be due to pathology or conditions in organs that shares the same nerve pathway with the scrotal contents: ureter (such as ureterolithiasis), hip, intervertebral disc prolapse and entrapment neuropathies of the ilioinguinal or genitofemoral nerve [160]. Idiopathic CSP represents approximately 25 % of the chronic orchialgia cases [162]. Possible etiologies for the idiopathic condition include neurological or rheumatological disorders, post-infectious etiology, post-trauma/injury, psychosomatic disorders, or one of the presenting symptoms of CPPS.

There are no clinical guidelines and no comprehensible recommendations for work-up and assessment. Evidence-based literature concerning the prevalence, diagnostic work-up, and treatment of pain located in the scrotum is sparse.

Strebel et al. [159] assessed the management of CSP by urologists and found that the most commonly used examinations besides clinical evaluation are a urinary dipstick and midstream urinary culture in 96 % and ultrasound examination in 93 %. Interestingly, the diagnostic yield of scrotal ultrasound is low in the presence of a normal physical examination and urinalysis [158]. However, many urologists advise ultrasonography as the best complementary investigation probably because of their concern of missing a testicular tumor [158]. The major benefit of scrotal ultrasound is reassurance to the patient worried about cancer [163]. Testicular microlithiasis and/or epididymal cysts have been reported in patients with orchialgia but the clinical correlation of these conditions remains controversial [160, 164]. Additional assessments included blood sampling (29 %), urethral swab (29 %), duplex ultrasound (19 %), assessment for coexisting chronic prostatitis (15 %), referral to an orthopedist (7 %), rheumatologist (6 %) or psychiatrist (3 %), and PCR on urine specimen (4 %) [159].

The most common noninvasive treatments for CSP among Urologists are antibiotics (quinolones or tetracyclines) and nonsteroidal anti-inflammatory agents, although isolation of bacteria from the genitourinary tract is uncommon [159, 165]. Recurrence rates after conservative treatment with antibiotics and anti-inflammatory analgesics are generally high (about 48 %) [159]. Tricyclic antidepressants sometimes relieve the pain [160].

When symptoms recur, epididymectomy or microsurgical spermatic cord denervation are optional [153, 166, 167]. Microsurgical denervation of the spermatic cord can achieve complete pain relief in 76–96 % of the patients with CSP [166, 168]. This procedure is recommended for pain which is refractory to nonsurgical treatments and for patients who had temporary pain relief after spermatic cord blockade. Patients with CSP without a history of prior vasectomy are not likely to benefit from epididymectomy [167]. Vasovasostomy can achieve complete pain relief in 70 % of patients with postvasectomy orchialgia [169]. A small number of patients fail to respond to both conservative and invasive treatment methods and for them the only available therapeutic option is inguinal orchiectomy [153].

Whenever there is an identifiable intrascrotal pathology it is recommended to treat the specific condition: ligation of the internal spermatic vein

can achieve pain relief in 75 % of the patients with varicocele, 94 % of patients with painful spermatocele experience significant pain relief after spermatocelectomy and almost all patients with painful hydrocele are free of pain after hydrocelectomy [170].

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Musculoskeletal Conditions Related to Pelvic Floor Muscle Overactivity

7

Pamela Morrison

7.1 Introduction

The superficial and deep pelvic floor muscles have many functions important to the musculoskeletal system as well as other systems such as the excretory, digestive, reproductive, and respiratory. They contribute to regulation of intra-abdominal pressure [1] and respiration [2]; contribute to sacroiliac joint [3], pelvic and lumbar spine stability [2]; act as a postural stabilizer to assist with core stabilization [2], contribute to sexual function [4], and act as a support mechanism for maintenance of continence and prevention of pelvic organ prolapse [5, 6].

Overactive pelvic floor muscles are described as muscles that are shortened, tightened, painful, and in spasm. Often, the muscles have tender points that are specific areas within the pelvic floor muscles that elicit pain or point tenderness when palpated. Additionally, the muscles may have myofascial trigger points which are taut bands within the muscles and when provoked or palpated refer pain to another adjacent area. Overactive pelvic floor muscles in the literature have been referred to as pelvic floor muscle hypertonicity or hypertonic pelvic floor muscles [7], high-tone pelvic floor muscle dysfunction [8], shortened pelvic floor muscles [9], non-relaxing pelvic floor muscles [10], levator ani syndrome [11], and pelvic floor tension myalgia [12]. Individuals with overactive pelvic floor muscles have difficulty with volitionally relaxing them and there may be a subconscious clenching or holding pattern occurring. Prolonged shortening of the pelvic floor muscles causes a change in the length–tension relationship resulting in weakness, decreased flexibility, impaired recruitment patterns and motor planning. The length–tension relationship is the relation between a muscle's length at rest and the isometric tension or force it generates when fully activated. Muscle imbalances between the pelvic floor muscles and other surrounding muscles result.

Overactive pelvic floor muscles may be a causal or perpetuating factor in chronic pelvic pain disorders for men and women including vulvodynia, interstitial cystitis or bladder pain syndrome, pudendal neuralgia, prostatitis and prostatodynia, irritable bowel syndrome, constipation, coccydynia, endometriosis, pelvic inflammatory disease, pelvic congestion syndrome, sexual pain, and fibroids. Chronic pelvic pain is defined as pain in the pelvis lasting 3–6 months or longer. Symptoms include intermittent or constant pain, dull aching, cramping, pressure or heaviness in the pelvis, painful intercourse, pain upon bowel movements or urination, and pain upon prolonged sitting.

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Overactive pelvic floor muscles can be a result of or a causative factor in musculoskeletal conditions because of its anatomical relationships and functional relationship to the diaphragm, ribs, abdomen, spine, sacrum, pelvis, coccyx, and hips. Pain and lower quarter musculoskeletal impairments may be caused by holding patterns of the pelvic floor muscles related to lumbopelvic joint mobility losses, abdominal wall adhesions, or hip balance impairment. The abdomen, hips, pelvis, and spine are a connected kinetic chain and any dysfunction along this chain may cause overcompensation and dysfunction of associated muscles [10]. Also, musculoskeletal structures of the low back, abdomen, pelvis, and hips share segmental innervation with the pelvic floor muscles. A prospective evaluation of patients with chronic pelvic pain of various etiologies found abnormal musculoskeletal findings in 37 % versus 5 % of controls [13]. Therefore, overactive pelvic floor muscles can contribute to, cause, or result from musculoskeletal disorders such as pubic symphysis pain, low back pain, sacroiliac joint dysfunction (SIJD), coccyx pain, hip disorders, and abdominal wall pain (Table 7.1).

Most often when discussing overactive pelvic floor muscles the reference is thought to refer to the deep pelvic floor muscles or levator ani which comprises the pubococcygeus, iliococcygeus, and puborectalis. Some anatomy references include the coccygeus as well. However, there is a superficial pelvic floor muscle group and a middle layer that can also be overactive alone, or in concert with the levator ani or and can be a contributing cause or perpetuating factor of musculoskeletal problems. The superficial pelvic floor muscles include the bulbocavernosus (bulbospongiosus), ischiocavernosus, and superficial transverse perineal. The middle layer comprises the deep transverse perineal muscle and sphincter urethra.

Table 7.1 Musculoskeletal disorders related to overactive pelvic floor muscles

Abdominal wall pain
Coccyx dysfunction
Hip disorders
Lumbar spine disorders
Pelvic disorders
Persistent genital arousal syndrome
Piriformis pain/syndrome
Postural dysfunction
Psoas pain
Pubic symphysis disorders
Pudendal neuralgia
Sacroiliac joint dysfunction

7.2 Structure and Function of the Pelvic Girdle

The pelvis has several functions: to protect and support the pelvic viscera, allow for transference of weight bearing forces from the trunk and ground reaction forces from the lower extremities, provide support for the fetus and house the birth canal, and provide attachment sites for ligaments and muscles of the lower extremities and trunk [14]. The body's center of gravity is located within the pelvis at the second sacral segment. The pelvis is the hub or center of stability for control of movement for the entire body.

The innominate bones that are made up of the ilium, ischium, and pubis are fused in adulthood. The ilia and sacrum join on either side to form the sacroiliac joints posteriorly. The sacroiliac joints are intermediate joints between a synarthrosis and diarthrosis called an amphiarthrosis which means only a small amount of movement can occur [15]. Sacroiliac joint mobility is multiplanar and varies based on the position and transfer of load [15, 16]. The main movement of the sacroiliac joints is rotation yet gliding, anteroposterior and vertical movements also occur to a lesser degree. More recent studies demonstrate that sagittal rotation movement of the sacroiliac joint is about 3.6° of movement and translation movement is 2 mm [17];however, prior studies have shown that up to 10 mm of movement can occur in the sacroiliac joint during manual medicine maneuver in normal subjects [18]. The sacroiliac joints are innervated by spinal nerve roots L4–S3.

The apex of the sacrum and the first coccyx segment attaches to create the sacrococcygeal joint. There is a sacrococcygeal fibrocartilage disc also known as the interosseus ligament.

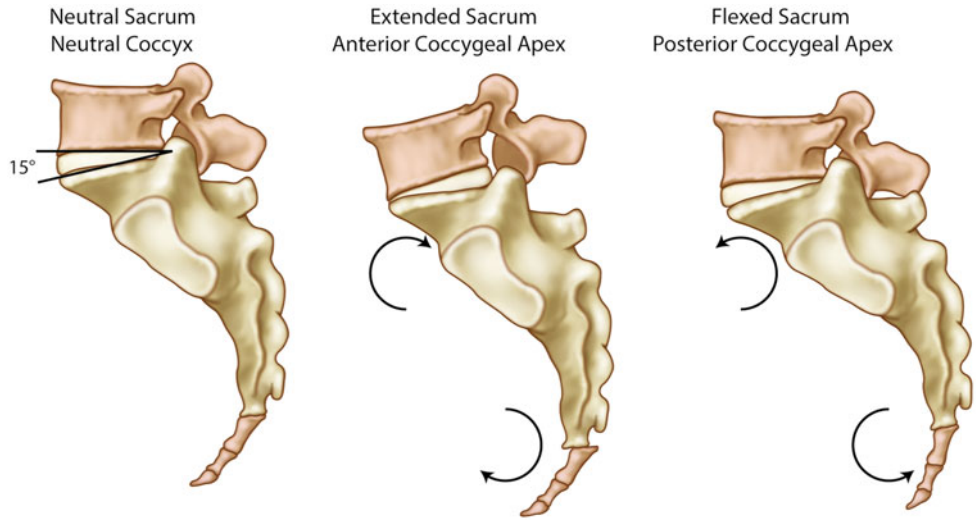

Fig. 7.1 Sacrum and coccyx normal biomechanics. Adapted from: Weiselfish Giammatteo S, Giammatteo T. *Integrative Manual Therapy for Biomechanics, Application of Muscle Energy and Beyond Technique: Treatment of the Spine, Ribs, and Extremities.* North Atlantic Books: Berkely, 2003;225–228 [61]

Normal movement of the sacrococcygeal joint is 15° of flexion and 13° of extension. In normal physiological movement as the sacrum extends, the apex of the coccyx moves anterior or flexes and as the sacrum flexes, the apex of the coccyx moves posterior or extends (Fig. 7.1). The sacrum comprises five fused segments, and the coccyx also comprises five fused rudimentary bones. The interosseous, dorsal sacroiliac ligaments, and anterior sacroiliac ligament stabilize the sacroiliac joints. Other accessory ligaments, such as the iliolumbar, stabilize the superior part of the joint and the sacrospinous, and sacrotuberous stabilize the inferior aspect of the sacrum. At the distal end of the coccyx, the anococcygeal ligament is the attachment site of the rectoanal angle and the levator ani.

Anteriorly at the superior rami, the pelvis joins by way of a fibrocartilaginous joint, called the pubic symphysis that has a cartilage disc in the center between thin layers of hyaline cartilage. The pubic symphysis has 3° of freedom and can also move anterior/posterior, internal/external rotation about its center axis. The sacrum articulates with the lumbar spine by way of the lumbosacral joint between the first sacral segment (S1) and the fifth lumbar vertebral segment (L5). The sacrum moves into nutation and counternutation relative to the ilium. Spinal flexion couples with sacral counternutation (or sacral extension) and spinal extension couples with sacral nutation (or sacral flexion). The femoral head articulates with the acetabulum of the innominate bones to form the hip joints. Hip flexion is coupled with the ilium moving into posterior rotation and hip extension is combined with the ilium moving into anterior rotation. Coupled motions of pelvic and hip flexion and extension are involved in establishing lordosis and kyphosis in the lower spine [15]. Thus, the spine, sacrum, coccyx, and hips all influence the function of the pelvis. Both the sacroiliac joints and pubic symphysis have little motion.

During gait, the pelvis acts as the axis where rotation of the lower extremities is balanced by rotation of the upper extremities. The pubic symphysis moves superior and inferior during gait. In one legged-stance, the pubic symphysis moves vertical [19]. Movement at the sacroiliac joint helps to decrease the shearing forces at the L5–S1 junction during the hip extension phase of gait. Gait mechanic analysis revealed that the SI joints are mobile yet stable for load transfer to

occur to and the load transfers from the lumbar spine and lower extremities [20, 21].

The muscle groups that attach onto the pelvis and influence its function include the superficial and deep pelvic floor muscles, the hip adductors, extensors, flexors, and rotators, abdominals, diaphragm, and the extensors and rotators of the spine. Muscle imbalances such as weakness or impaired flexibility in any of these muscle groups can change the forces on the pelvic girdle and alter biomechanics leading to dysfunction. More specifically, altered motor control of deeper abdominal and pelvic muscles, such as the transversus abdominis, internal oblique, multifidus, respiratory diaphragm, and pelvic floor muscles can result in lumbopelvic pain and impaired pelvic mobility and stability. Lumbar spine and pelvic girdle muscle recruitment is also altered with existent low back or SIJ pain [22, 23]. Along with the large muscles overlying the SI joint, contraction of other muscles such as the transversus abdominis, the pelvic floor muscles, and the respiratory diaphragm can affect the stiffness of the spine and SIJ [24–28].

An important function of the pelvic girdle is to be able to transfer the load created by the weight of the body and gravity during functional activities including walking, transferring, and all activities of daily living. In order for this to occur efficiently, there must be form closure and force closure of the pelvic joints and effective motor control. Optimal lumbopelvic stability is a function of form closure, force closure, and neuromotor control. Form closure is passive and is produced by joints anatomically approximating congruently. Force closure is the force produced by muscular contraction, ligaments, and fascia creating compressive forces and motor control around a joint creating joint stability or locking [21]. Impairments of these mechanisms, especially at the sacroiliac joints, can cause lumbar spine or pelvic pain, instability, impaired lumbopelvic kinematics, and reduced strength and motor control [29].

Motion control of the pelvic joints requires efficient activation which is coordinated and sequenced so that activation of muscle groups and co-activation occurs resulting in the least amount of compression forces. If inefficient motion control at the pelvis occurs, excessive shearing, giving way, and bracing or stiffening of the hips, low back, pelvis, or rib cage may result [20]. Non-optimal strategies can cause an increase in intra-abdominal pressure resulting in suboptimal breathing patterns, impaired diaphragmatic excursion, increased intra-abdominal pressure, and may compromise pelvic floor muscle function and continence.

The lumbo-pelvic-hip complex helps create the core. The core has been presented as a three-dimensional box or canister comprises the respiratory diaphragm, pelvic floor and hip complex, abdominals, spinal, and gluteal muscles [30, 31]. Although there are many global (larger, longer) muscles and local (smaller, shorter) muscles involved in core stability [31], four muscle groups that contribute the most to stabilizing the box or canister are the respiratory diaphragm from above, the transversus abdominis along the sides, the mulitifidi posteriorly, and the pelvic floor from underneath. These muscles help form the core. The core provides a stable base for all extremity movement to occur [30, 32] Impaired recruitment of the pelvic floor muscles, transversus abdominis, respiratory diaphragm, and deep fibers of the lumbar multifidi can result in failure of load transfer through the pelvic girdle. Muscle recruitment and motor patterns for force closure of pelvic joints may be delayed, inhibited, or asymmetric. Repetitive strain of the lumbo-pelvic-hip passive soft tissue structures may result. Studies of patients with chronic low back pain, pelvic pain, and groin pain show delayed activation of transversus abdominis. Delayed contraction of the transversus abdominis impedes the pretensing of the thoracodorsal fascia needed for efficient pelvic stability for load transfer. Pelvic floor muscle function is important in stabilizing the pelvic girdle from underneath and overactivity in the muscles can result in weakness, impaired sequencing, and timing of activation. Force closure of the urethra, bladder stability, and motion control of the sacroiliac joints may be compromised.

7.3 Pubic Symphysis Disorders

Pubic symphysis disorders can include osteitis pubis, pubic symphysis diastasis or separation, or misalignment. Osteitis pubis is a chronic inflammatory pain disorder that involves the pubic bones, symphysis pubis, hip adductors, abdominal muscles, and adjacent fascia [33]. Osteitis pubis is characterized by sclerosis and bony changes at the pubic symphysis [34]. Osteitis pubis is often misdiagnosed or mismanaged and can thus be prolonged and cause disability. Symptoms can include diffuse lower abdominal and pubic pain, and often be mistaken for groin strain, abdominal strain, and bladder pain. Pain can be referred into the hip, groin, scrotum, perineum [35], or labia. Osteitis pubis can be the cause of pain with intercourse. The pain in the pubis may worsen upon running, climbing stairs, or change of positions such as standing from sitting, rolling in bed, or getting out of a car. Sports activities such as soccer and football where kicking is most prevalent can cause or worsen symptoms [33]. Postures in standing whereby the legs are asymmetrical can exacerbate symptoms. Causes of osteitis pubis include trauma [33], repeat exertional forces [33], childbirth [36], infections following urologic or gynecologic procedures [34], and muscle imbalances [33, 35]. Muscle imbalances of the abdominals and hip adductors along with the pelvic floor muscles may be a causative factor and because of their attachments on the pubis can cause uneven torque to the pubic symphysis resulting in repetitive strain. Diagnosis is based on history, physical exam, and radiographic results such as X-ray or bone scan. Palpation pain directly over the pubis, inflammation at the symphysis pubis or lower abdominal region, and a positive symphysis gap test [33] are also common diagnostic criteria. There are four classifications of osteitis pubis for athletes [33]. Stage 1 includes pain in one leg and inguinal pain in the adductors. Stage 2 involves bilateral inguinal pain. In Stage 3, there is bilateral inguinal pain upon changing positions from sitting to standing or changing directions while playing sports. Stage 4 involves pain in the adductor and abdominal muscles and referred pain to the pelvic girdle or lumbar region upon sneezing, bowel movements, and walking on even surfaces. Conservative management may include rest, nonsteroidal anti-inflammatory drugs [33], corticosteroid injections, and physical therapy. Surgery may be needed in 5–10 % of the cases [36, 37].

Pubic symphysis diastasis is a separation of the pubic symphysis that occurs during pregnancy or as a result of vaginal delivery, following osteoarthritis and long-term use of corticosteroids, pelvic trauma, and high velocity injuries [34]. Horseback riding has also been found to be a cause [34]. There may also be injury to the ligaments of the sacroiliac joints that lead to chronic pelvic pain [38]. The short hip adductors and obturator externus function together as a unit. Together they can produce a distraction force at the pubic rami and if chronically tight can become a predisposing factor. The physiologic widening of the pubic symphysis is considered to be a maximum of 10 mm especially during pregnancy, and separation of 1 cm is often symptomatic causing pain [39]. Pubic symphysis diastasis is diagnosed via history, physical exam, and an anterior-posterior pelvic X-ray. Physical exam may reveal weakness in the hip flexors, quadriceps, and hamstrings due to pain. Difficulty with transfers and position changes may be reported and ambulation may be impaired. Symptoms include groin pain, hip pain, swelling over the pubis, sacroiliac joint pain, pubic or groin pain upon leg movement and walking. If unresolved, overactive pelvic floor muscles, abdominal pain, and hip myalgia can result [40]. Conservative management can include rest, analgesia, wearing an abdominal/pelvic binder or pelvic stabilizing belt [41]. Physical therapy is also prescribed. A specific rehabilitation program that focuses on core strengthening and pelvic floor muscle rehabilitation improves the prognosis of decreased pain, improved function and mobility [42]. Overactive pelvic floor muscles are inherently weak because when muscles are shortened the length–tension relationship is altered and are at a disadvantage of optimal force production upon contraction. Ultimately, the goal of rehabilitation is to achieve full pelvic floor muscle length, improved resting

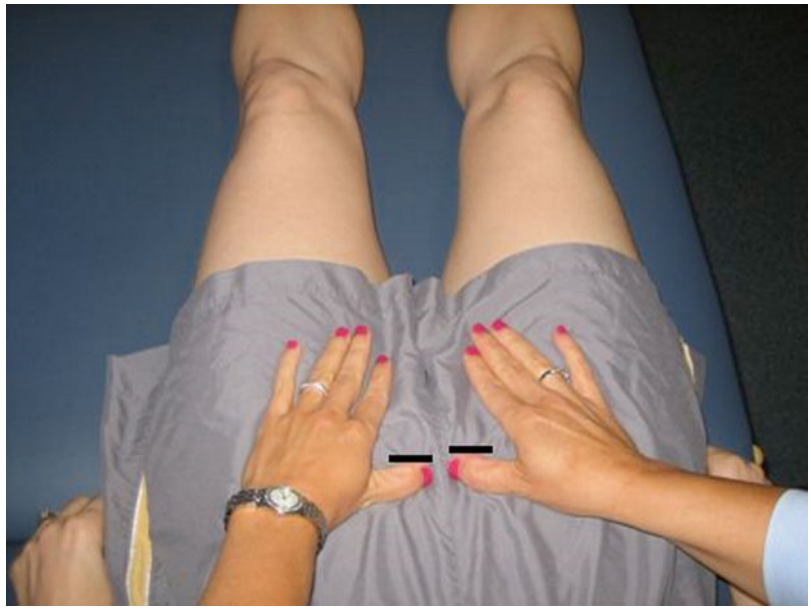

Fig. 7.2 Pubic symphysis misalignment: Noted via palpation of the superior aspect of the pubic symphysis

tone, resolve pain and myofascial trigger points, and then strengthen the pelvic floor muscle through its full excursion or range of motion.

Pubic symphysis misalignment or unleveling or subluxation is another disorder. Unleveling of the pubic symphysis is superior or inferior (cephalad or caudal), also known as a shear [42] (Fig. 7.2). Pubic shears commonly occur with other pelvic innominate rotations or upslip and downslip dysfunctions. An innominate rotation is when one side of the pelvis is rotated anterior or posterior compared to the other side, thus labeled anterior innominate rotation or posterior innominate rotation. A pelvic upslip is when one side of the pelvis is higher than the other side and this is measured by comparing the heights of the iliac crests manually [42]. Innominate rotations and a pelvic upslip or downslip dysfunction can cause pubic symphysis misalignments. Direct causes of superior sheared pubic symphysis include upward forces through the ipsilateral leg, falls onto the ischial tuberosity, weak hip abductors, and tight surgical scars in the suprapubic region such as cesarean incision scar. Inferior shears of the pubic symphysis are caused by lift upward of the body with a fixated foot, tight hip adductors, and pelvic floor muscle tension. The hip adductors have an immediate effect on superior and inferior forces placed on the pubic rami thus an imbalance or chronic tightness can be a contributing factor. The presenting symptom for pubic misalignment is groin pain. Some associated symptoms may include bladder urgency or frequency, urethra pain, clitoral pain, scrotal or penile pain. Superior or inferior pubic symphysis shears alter the anterior and posterior hip rotation motion that occurs with gait. During the normal gait pattern, the pubic symphysis acts as an anterior axis for alternating hip rotation [43, 44]. Perpetual misalignment of the pubic symphysis can eventually result in osteitis pubis, hip pain, and chronic pelvic pain. Physical therapy management includes pubic symphyseal corrective techniques such as muscle energy techniques and direct mobilizations and soft tissue mobilization to the pelvic floor muscles, hip adductors, suprapubic region, and pubic ligaments.

Pubic symphysis compression is caused by trauma, hip hyperadduction or internal rotation forces and pain in the pubis worsens with ascending stairs and walking. Pelvic floor muscle dysfunction such as tightness, spasm, and overactivity is also a cause. Pain presents at the symphysis, perineum, inner thigh, or hip region. Physical

therapy management includes pubic symphyseal mobilization such as decompression or lateral distraction techniques and soft tissue mobilization to the pelvic floor muscles, hip adductors, suprapubic region, and pubic ligaments [43].

Despite pubic symphysis pain disorders referring pain into the pelvic girdle, scrotum, labia, and perineum, the pelvic floor muscles are a frequently overlooked factor in pubic symphysis disorders. Understanding the anatomical connections and relationships are important factors in the physical examination. Two superficial pelvic floor muscles, the bulbocavernosus and ischiocavernosus, and one deep pelvic floor muscle, the pubococcygeus, attach onto the inferior pubis and can be involved in contributing to pubic symphysis pain. The abdominal muscles such as the rectus abdominis and transversus abdominis together with the internal oblique attach to the superior aspect of the pubic symphysis. The abdominal muscles and adductors act antagonistically to the pelvic floor muscles in daily functional activities. When muscle imbalances are present, it can predispose the symphysis pubis to mechanical traction microtrauma such as in osteitis pubis. Overactive pelvic floor muscles will place an abnormal inferior tension or pull on the pubis, especially if only one side of the pelvic floor muscles is tense or shortened. This can contribute to causes and perpetuation of pubic symphysis dysfunction. Likewise, a superior pubic shear will place abnormal tension on the pelvic floor muscles. Over time, if not corrected the pelvic floor muscles may remain in a contracted state and become chronically overactive. Also, the pelvic floor muscles engage as a protective guarding/mechanism as a response to pubic symphysis pain and inflammation. Likewise, the hip adductors can engage as a protective guarding/mechanism.

The hip adductor fascia is continuous with the pelvic floor muscles. The pubococcygeus muscle, one of the levator ani muscles, is also referred to as the pubovisceral can be further subdivided into the puboperinealis, pubovaginalis, and puboanalis. All of these collectively originate on the inner surface of the pubic symphysis and insert into the perineal body, vaginal wall near the level of the urethra, and into the groove between the external and internal anal sphincter. The puborectalis also originates from the pubic bone and forms a supportive sling around the anus.

Pubic symphysis disorders may cause inhibition and weakness of the transversus abdominis, external oblique, and rectus abdominis muscles due to inflammation and pain in the region. Inflammation and pain in a joint can cause inhibition of the surrounding joint muscles. In order for the trunk and pelvis to gain stability, the pelvic floor muscles would be recruited and shortened and remain contracted resulting in overactivity.

7.4 Hip Disorders

Hip impingement, also known as femoroacetabular impingement (FAI), occurs when there is abnormal contact between the proximal femur and acetabulum during motion. Causes of abnormal contact between the proximal femur and acetabulum include muscle imbalances such as a tight psoas muscle and weakened hip extensors or deep rotators pulling the femoral head anteriorly, prior trauma such as femoral neck fractures, or as a result of childhood diseases such as Legg-Calve-Perthes Disease. Even subtle morphologic abnormalities have been seen in active patients that affect either the acetabulum or proximal femur [45]. The symptoms of impingement include medial groin pain, deep hip pain, inner thigh pain, pain along the tensor fascia latae, clicking, locking, and sharp pains [46]. Pain may worsen upon pivoting or turning towards the affected side. Groin pain on hip flexion, adduction, and internal rotation (FADIR) worsen the pain and hip impingement sign test is assessed in this position by orthopedists, physical therapists, athletic trainers, and physiatrists. Resisted hip flexion may also bring on symptoms and a decrease in hip internal rotation range of motion is usually noted. As the hip flexes, the femur abuts on the acetabular rim causing the symptoms of impingement. Recurrence of this can result in acetabular labrum injury such as labral tears and avulsion or shearing of the cartilage. Hip impingement can cause continued

deterioration and lead to early onset of hip osteoarthritis and functional limitations. Tightness of the iliopsoas, quadriceps, tensor fascia latae, deep hip rotators, and pelvic floor muscles are usually concurrent issues. Hip impingement alters hip and pelvic biomechanics during gait [47]. There is a compensatory increased posterior pelvic rotation during active end range hip flexion causing a repeated pull of the pelvic floor muscles [48]. As the pelvic innominate rotates posteriorly, the pelvic floor muscles are tensioned anteriorly. Over time, this constant pulling could cause stress–strain on the pelvic floor muscles leading to overactivity of pelvic floor muscles. Conservative treatment includes analgesics, intra-articular glucocorticosteroid injections, activity modification, and physical therapy [49]. Surgical approaches include arthroscopic or open surgical dissection to debride, repair labrum and chondral surfaces, and address any boney deformities.

Studies show there is a relationship between hip function and pelvic floor muscle function such as seen in a case series of patients with labral tears as a comorbidity of low back and pelvic girdle pain [50]. The deep hip rotators include the piriformis, inferior and superior gemelli, and obturator internus and externus. Patients with chronic pelvic pain received fluoroscopy-guided anesthetic injections into the obturator externus muscle, a deep hip rotator, and 82 % found improvement in their pelvic pain [49].

Acetabular labral tears are a source of hip pain, yet there may be concomitant pelvic girdle and low back pain [51, 52]. The labrum is a cartilaginous ring anchored anteriorly and posteriorly to the transverse ligament and capsule on the acetabular periphery. It aids to the stability of the hip joint by deepening the acetabulum and increasing the surface contact area and distributing ground reactive forces [52]. Labral tears, therefore, may destabilize the hip joint by compromising the seal it provides to the joint, allow for higher stresses to the hip joint, and leading to joint deterioration. Labral tears are usually the result of repetitive sports injuries, torsional movements, and frequent movements into external rotation combined with excessive hip abduction or extension yet can also be due to trauma such as motor vehicle accidents or falls. Structural risk factors include history of hip dysplasia, femoral anteversion, acetabular retroversion, and hip impingement syndrome [52]. Repeated microtrauma over time can also lead to labral tears. Common clinical findings of labral tears include anterior hip pain, groin pain, deep buttock pain, greater trochanter pain, lateral ischial tuberosity pain, clicking, giving way, and locking. There is noted reduced hip range of motion of rotation, flexion, adduction, and abduction. Testing for an anterior labral tear occurs by bringing the hip into end range flexion, internal rotation, and adduction and posterior labral tears are tested in extension, abduction, and internal rotation [52]. A positive test provokes pain or a click. A labral tear is confirmed via magnetic resonance imaging (MRI), magnetic resonance arthrography (MRA), or arthroscopy. Treatment consists of rest, nonsteroidal anti-inflammatory agents, and physical therapy. Surgical intervention involves arthroscopic debridement of the labral tear and repair of associated structures [52].

Hip labral tears have been found to be an etiologic factor in vulvar pain syndromes such as vulvodynia with concurrent overactive pelvic floor muscles. In a preliminary study by Coady et al. [53], 40 women with suspected hip pathology and unprovoked vestibulodynia and/or clitorodynia had anterior labral tears confirmed via MRI. Physical therapy focused primarily on hip rehabilitation proved useful with 39 % reporting improvement in vulvar pain and 7 patients that underwent arthroscopic surgical labral repair reported moderate to marked improvement in both hip and vulvar pain. To better understand how hip and pelvic floor muscles impact one another and how hip dysfunction can cause pelvic floor muscle overactivity one must appreciate the anatomical and functional relationships of the hip and pelvic floor muscles. The fascia covering the pelvic floor muscles is continuous with endopelvic fascia above, perineal fascia below, and obturator fascia laterally [54]. Thus, there is a direct relationship between the hip and the pelvic floor muscles via the fascia. Thickening in the obturator fascia is called the arcus tendinous

fascia pelvis and extends from the pubis anteriorly to the ischial spine and provides attachment to the paravaginal connective tissue. The pubococcygeus originates on the fascia that surrounds the obturator internus. Arising from an adjacent location on the pubis but extending superior to the arcus tendinous fascia pelvis is a thickening of levator ani fascia called arcus tendinous levator ani, which is the origin to the levator ani muscle [54].

The role of the pelvic floor muscles to help create an anchor for the deep stabilizers of the hip is important for optimizing the power of the large hip musculature to both control stability and power for descent and ascent in a squat maneuver and for stair climbing.

7.5 Pelvic Obliquity

Pelvic obliquity is an unleveling or asymmetry of the pelvic girdle bones (Fig. 7.3). One side of pelvis is higher than the other vertically. This can be measured manually by the examiner placing one hand on the posterior superior aspects of the iliac crests and measuring for height differentiation or via X-rays [55, 56]. Causes of pelvic obliquity include leg length discrepancy, hip dysfunction, structural scoliosis, SIJD, visceral dysfunction, muscle imbalances, trauma, falls, jumping on an extended knee or as a combination of two or more of these causes [57, 43]. The superior innominate shear, or the elevated side, is referred to as an innominate upslip dysfunction. The three boney landmarks of the pelvis, such as the iliac crest, the posterior superior iliac spine, and the pubic symphysis are also manually palpated as being elevated compared to the other side. Pelvic obliquity has been found to be a common musculoskeletal evaluation finding in women with chronic pelvic pain including vulvodynia, persistent vulvar pain, and overactive pelvic floor muscles [58, 59].

It is common to find tight hip adductors and pelvic floor muscle tightness or tension on the side of the upslip. Pelvic obliquity can cause a chronic tension of the pelvic floor muscles, piriformis, quadratus lumborum, multifidus, iliolumbar ligament, and iliopsoas on the side that is elevated causing a stress–strain response. Continuous tension placed on the muscle results in strain and elevated resting tone. This leads to overactivity in the pelvic floor muscles and associated muscles in the region.

An upslip also puts stress–strain on the anterior and posterior sacroiliac ligaments, sacrotuberous ligament, and sacrospinous ligament. The pudendal nerve passes between the sacrotuberous and sacrospinous ligament, which is a common area of entrapment leading to pudendal neuralgia. Pudendal nerve irritation can cause overactive pelvic floor muscles. The pudendal and levator ani nerves can become tractioned, compressed, or irritated. Also, the sacral nerve roots that run anterior to the piriformis can become tractioned. Therefore, the irritated nerves can cause upregulation of the end organs such as the levator ani, genitalia, bladder, anus, and perineum.

Correction of pelvic obliquity via various manual physical therapy techniques has been studied and preliminary data suggests it improves low back pain [60] and would therefore likely also improve pelvic pain.

7.6 Sacroiliac Joint Disorders

It has been found that pelvic floor muscle dysfunction can be a concomitant issue in patients with SIJD [25]. SIJD refers to pain in the sacroiliac region caused by pathomechanics from trauma, aberrant postures, degenerative arthritis, pregnancy, altered gait patterns, and inflammatory diseases such as gout, ankylosing spondylitis, and rheumatoid arthritis. SIJD can cause an inflammatory process at the joint and surrounding structures. Surrounding structures include the anterior and posterior sacroiliac ligaments, sacrotuberous, sacrospinous, and iliolumbar ligaments. Sacroiliac pain is felt near the joints and can occur between the medial aspect of the posterior iliac crest and gluteal sulcus. Referral of pain from SIJD can occur across the iliac crest laterally, into the buttock, pelvic floor muscles, and down the lateral and posterior thigh. Sacroiliac dysfunction with hypomobility or compression at

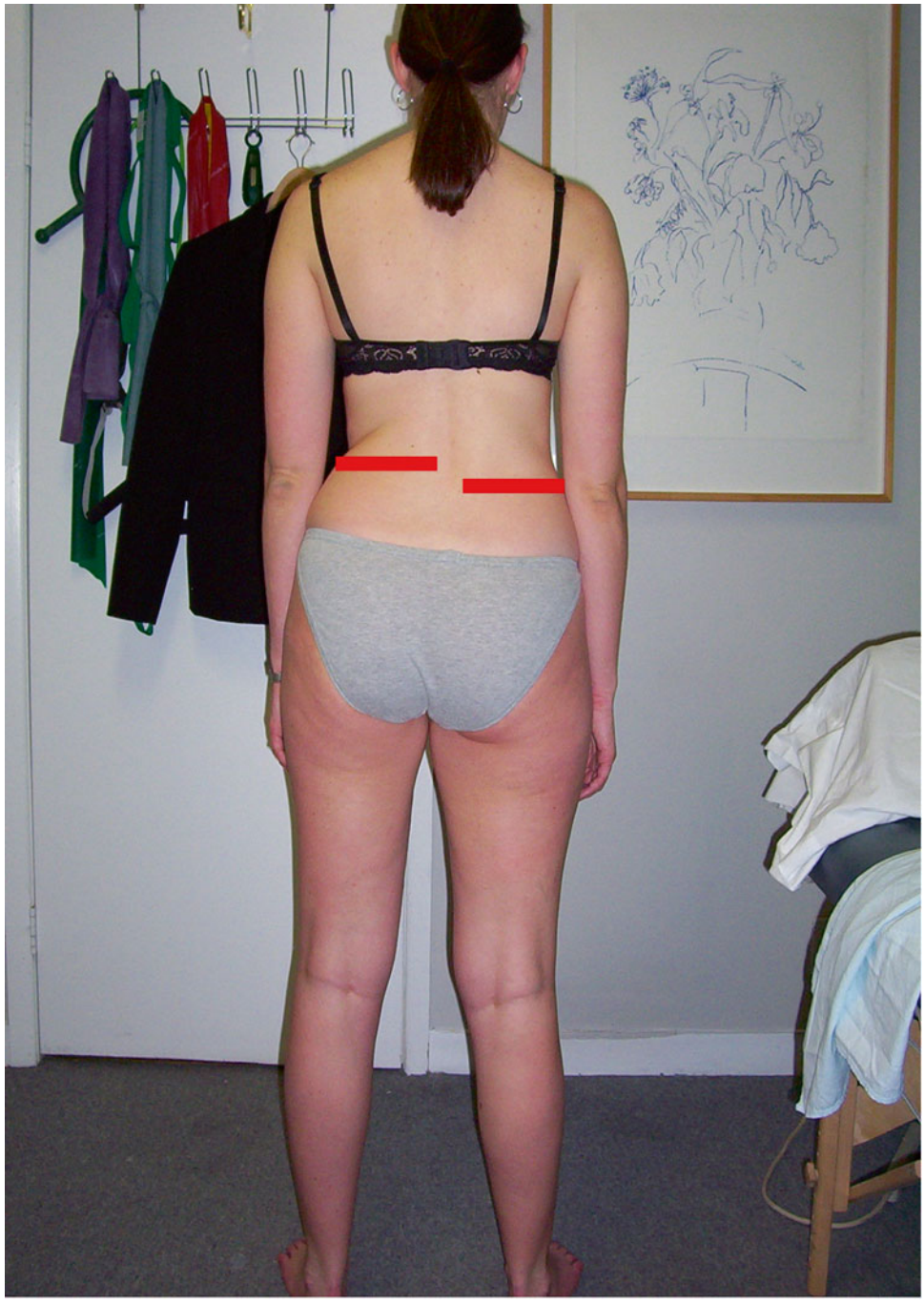

Fig. 7.3 Pelvic obliquity: Noted unleveling or asymmetry of the iliac crests

the joint seems to be more related to overactive pelvic floor muscle dysfunction. Myofascially compressed sacroiliac joint findings include poor motor control, impaired patterns for lumbopelvic stabilization, posteriorly tilted pelvis, hip restrictions in flexion, adduction, and internal rotation motions, trigger points in the obturator internus, piriformis, and coccygeus tension [20]. Whereas,

sacroiliac joint hypermobility or excess motion is related to pelvic floor muscle weakness. In order for the sacroiliac join to effectively transfer load, form closure and force closure ability of the joint has to be intact. The sacrotuberous and long dorsal ligaments have been the focus of research. Loading of the sacrotuberous ligament, which has connections with the gluteus maximus, long head of the biceps femoris, and sacrospinous ligament, restricts sacroiliac joint ventral rotation [15]. The long dorsal sacroiliac ligament is penetrated by S2–4 posterior sacral rami. When sacroiliac dysfunction exists a chronic inflammatory response occurs that can cause "wind up." This physiologic "wind-up" phenomenon begins at the skin, affects the peripheral nerves, and results in a hypersensitivity response from the dorsal horn in the spinal cord and brain and can effect other structures along the same pathways such as the pudendal nerve, direct nerve branches S3–4, and pelvic floor muscles as one of the end organs.

Contraction of the piriformis with a neutral hip can create a sacral torsion or rotation of one side anteriorly which brings the sacral base forward into flexion. This malposition of the sacrum can cause tension in the ipsilateral pelvic floor muscles causing overactivity.

A descended sacrum is a dysfunction whereby forces from above such as a whiplash or something falling on the cranium creates a downward force through the spine causing downward force on the sacrum. The sacral position is inferior to its normal position and is tractioned inferiorly. The coccyx apex is pulled anteriorly or flexed. This creates tension on the pelvic floor muscles also referred to as the pelvic diaphragm by the coccyx apex causing an overactive response (Fig. 7.4) [61].

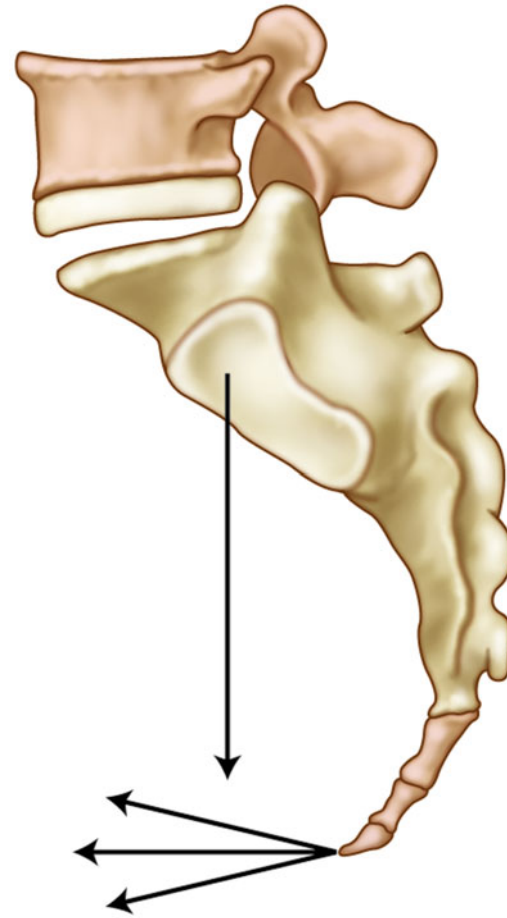

Fig. 7.4 Descended sacrum causes a pulling of the coccyx forward or into excess flexion by the pelvic floor muscles. Adapted from: Weiselfish Giammatteo S, Giammatteo T. *Integrative Manual Therapy for Biomechanics, Application of Muscle Energy and Beyond Technique: Treatment of the Spine, Ribs, and Extremities.* North Atlantic Books: Berkely, California, 2003;225–228 [61]

7.7 Coccydynia

Coccydynia is defined as pain and tenderness in the coccyx or in the region of the coccyx that usually worsens with sitting. Most common cause of coccydynia is abnormal mobility [62]. Other causes of coccyx pain includes trauma, subluxations, fractures, childbirth, chronic poor posture, obesity, pelvic obliquity, leg length discrepancy, repetitive strain, constipation, scar adhesions, lesions, tumors, diseases of the pelvic organs, and muscle imbalances [43, 63–66]. The coccyx may be malpositioned into more flexion, causing a pain response, which is the most common malposition found. However, extension, lateral flexion or rotation, or combinations of these movements may be found because they are accessory motions of the sacrococcygeal joint. With the patient in prone, coccyx alignment can be

best assessed with the clinician covering the coccyx body with the examining thumb (Fig. 7.5). The muscles that directly attach onto the coccyx include the levator ani, coccygeus, and gluteus maximus. The hip rotators attach via fascia of the external sphincter tendon. Chronic flexion position of the coccyx can change the normal resting length of the pelvic floor muscles causing excessive shortening and does not permit the pelvic floor muscles to lengthen through its normal excursion. Overtime, this can lead to chronic overactivity including pain, myofascial trigger points, tender points, and spasm. Likewise, an overactive pelvic floor can pull the coccyx into hyperflexion now being a potential causative factor in coccydynia. Abnormal stress–strain or tension is placed upon the sacrococcygeal joints, sacrococcygeal disc, sacrococcygeal ligaments, coccygeal plexus, and the dura.

A psychological reason proposed for overactive pelvic floor muscles and coccyx pain described by Wise is a fear, anxiety, stress response referred to as "tail-pulled-between-the-legs biological response" [67]. Animals contract their pelvic floor muscles causing their tailbones to pull between their legs to signal predators of defense and preparatory for attack. In a chronic state of stress or fear, continued contraction persists and a flexed coccyx results. This also may be a protective response for the anorectal and genitalia region related to a protective defense mechanism in cases of sexual abuse.

Treatment for coccydynia includes oral medication, ganglion impar blocks, fluoroscopy-guided steroid injections, prolotherapy, manipulation under general anesthesia, Botox injections into the pubococcygeus and puborectalis, and partial or complete coccygectomy. Physical therapy

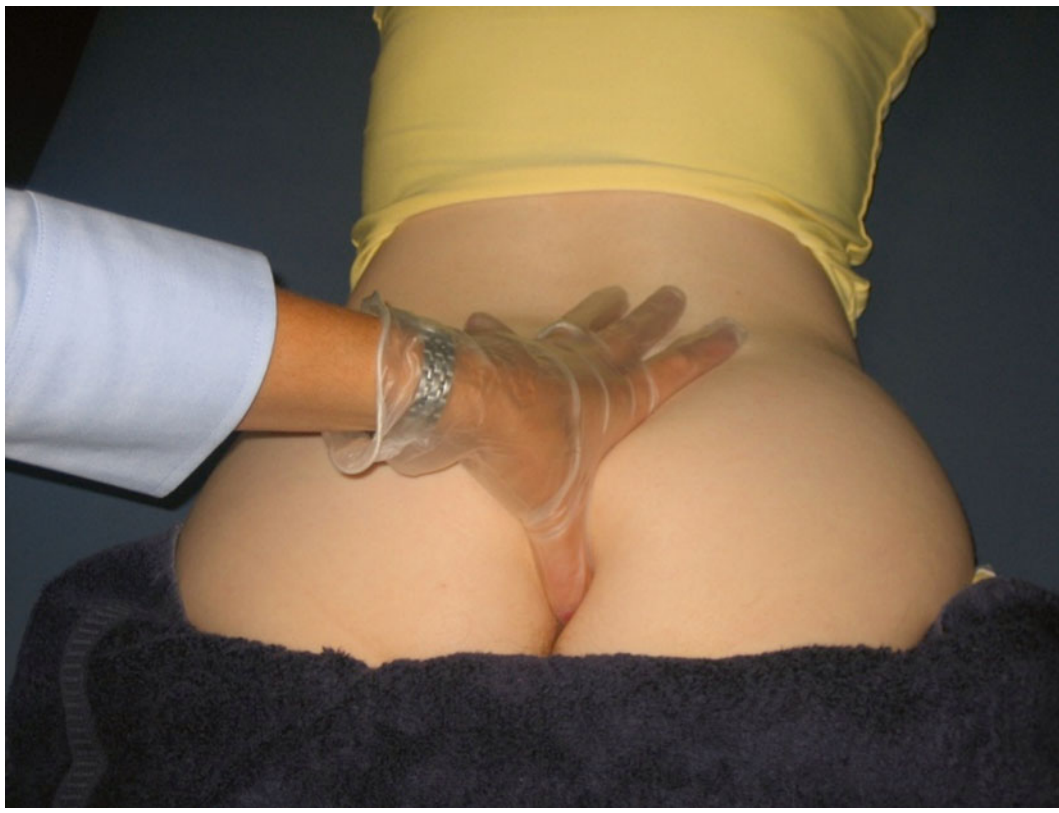

Fig. 7.5 External palpation of the coccyx to determine alignment. Clinician places a thumb along the body of the coccyx to feel for flexion or extension, rotation, or side bending misalignment

that addresses the overactive pelvic floor, coccyx position through external and transrectal mobilization, taping techniques, and pain management modalities is beneficial. Specialized sitting cushions such as donuts and wedges are recommended. Chiropractic care and acupuncture are also adjunct interventions.

7.8 Poor Posture

Poor posture contributes to pelvic floor muscle shortening or tensing. In a stooped or slumped posture in sitting the spine and hips are excessively flexed and hamstrings are shortened causing a posterior rotation of the pelvis. With stooped posture, there is little skeletal support and tension is placed on muscular and ligamentous structures instead. The abdominals, pectoralis, psoas, and suboccipital muscles also shorten. The spine loses height, normal lumbar lordosis decreases, and intradiscal pressure increases [68]. The coccyx is brought into further flexion causing a passive shortening of the pelvic floor muscles. Thus, chronic poor posture will cause a change in the resting length of the pelvic floor muscles and changes the load on the muscles creating a strain and shortened state seen in overactive pelvic floor muscles. Poor posture also impedes normal respiratory diaphragm excursion. Therefore, the diaphragm can become shortened and the rib cage subsequently has impaired mobility in lateral and posterolateral costal expansion. Both abdominal and lower rib cage expansion are necessary for optimal breathing patterns. Because of the fascia links from the diaphragm to the coccygeus, a resultant tightening reaction can occur. Additionally, poor posture such as sitting with one leg crossed over the other can contribute to muscular dysfunction in the pelvis. Habitual unevenness of the pelvis can cause an upslip on one side and tension the pelvic floor muscles, compress the ipsilateral sacroiliac joint and hip, close or overload the lumbar zygapophyseal or facet joints on the upslip side, and create shortening of the quadratus lumborum.

Upon upright posture there is increased intra-abdominal pressure that results in the pelvic viscera and pubic symphysis to rotate anterior. If there is hyperlordotic posture, more pressure is placed on the pelvic floor muscles to have tonic activity to counteract the forces brought further anterior, resulting in overactivity. A common standing posture noted in patients with chronic pelvic pain includes anterior tilted pelvis, increased lumbar spine lordosis, increased thoracic spine kyphosis, and anterior line of gravity to the pelvis and knees. The hip flexors, iliopsoas, tensor fascia latae, thoracolumbar fascia, spinal extensors, piriformis, and coccygeus muscles shorten [69, 14]. Poor sleeping posture such as prone, sleeping on a soft mattress, or sitting up in bed with the main weight bearing on the sacral region can be contributory to pelvic pain disorders resulting in overactive pelvic floor muscles.

Physical therapy treatment consists of proper sitting and standing postural re-education, stretching the muscles that have adaptively shortened, and strengthening muscles that are weak such as the abdominals, and pelvic floor muscle rehabilitation.

7.9 PSOAS/Anterior Pelvic Pain

The psoas muscles are located deep in the lesser pelvic cavity and lateral to the lumbar spine. The psoas major and minor join with the iliacus to become the iliopsoas. The psoas arises from the anterolateral aspect of the lower thoracic and lumbar (T12–L5) vertebral bodies and transverse processes, enters the pelvis, and crosses the hip to insert into the lesser trochanter along with the iliacus. The iliacus originates in the iliac fossa. The psoas is connected to the respiratory diaphragm above and the coccygeus inferiorly via direct fascia links. The actions of the psoas are to flex the hip and spine and rotate and side bend the trunk. Disorders of the psoas such as shortening, spasm, and myofascial trigger points can manifest as abdominal, pelvic, sacroiliac, low back, hip, or groin pain [70]. Gait deviations may also be noted. Clinical examination includes transabdominal palpation for tenderness and myofascial trigger points, length tests, and manual muscle testing for

strength. Organic diseases such as abscess, hematoma, or tumor should be considered. Three nerves emerge on the anterior surface of the psoas: the iliohypogastric, ilioinguinal, and genitofemoral. The iliohypogastric passes inferolaterally through psoas major and emerges from their superolateral border with the ilioinguinal nerve. It then passes over the anterior surface of quadratus lumborum piercing the transversus abdominis above the iliac crest. It branches into the lateral cutaneous and anterior cutaneous nerves. The ilioinguinal nerve also pierces the internal oblique and aponeurosis of the external oblique before entering the inguinal canal. It further branches into the anterior scrotal branches in males and labial branches in females. The genitofemoral nerve emerges from anterior surface of the psoas, where it penetrates the psoas fascia. It further divides into the genital and femoral branches above the inguinal ligament. The femoral branch terminates in the superior thigh and the genital branch enters the inguinal canal through the inguinal ring and follows the spermatic cord in males, giving off branches to the cremaster muscle, testicular autonomic plexus, and the skin of the scrotum adjacent to the thigh. Males with chronic pelvic pain who had pelvic floor muscle overactivity also had pain and tension in their psoas and groin muscles [71]. In the female, the genital branch accompanies the round ligament of the uterus to terminate in the skin of the mons pubis and labium major. The obturator nerve emerges from the medial border of the psoas. Psoas spasm or shortening can cause abnormal neural tension or compression of these nerves running along, through, or adjacent to the anterior aspect which can cause alteration of the mobility of the nerve. Pain along the nerve and its end organs such as the inguinal, hip, groin, pelvic, and genital structures can be triggered.

Psoas myofascial trigger points, according to Travell [72], refers pain to the low back and superior iliac crest posteriorly, abdomen adjacent to the umbilicus, inside the ilium or lateral rectus region near the inguinal ligament, and upper thigh region. Shortening of the psoas is usually associated with shortening of the ipsilateral quadratus lumborum which can cause elevation or an upslip of the ilium and resultant tension of the pelvic floor muscles.

The exam for the psoas includes direct palpation and length tests. The Thomas test performed with the patient on the edge of the table flexes one knee up to the chest and dangles the other over the edge [55]. The clinician passively flexes the knee while extending the hip to determine flexibility or length of the psoas muscle. Direct palpation will help identify tender points or myofascial trigger points. Treatment to correct psoas dysfunction can include physical therapy, steroid injections, and rest.

Physical therapy to correct psoas dysfunction can include myofascial release, deep massage, strain-counterstrain or positional release techniques, stretching techniques, and myofascial trigger point release techniques.

7.10 Piriformis/Buttock Pain

The piriformis muscle is a deep hip external rotator which covers part of the posterior pelvic wall and a portion of the posterior hip capsule deep in the buttock. The piriformis is innervated by spinal nerves S1–2 and sometimes by L5. It attaches onto the anterior surface of the sacrum at the boney portion between the first through fourth sacral foramina and after it exits through the greater sciatic notch it inserts onto the greater trochanter of the hip where its tendon joins with the superior and inferior gemelli, and obturator internus and externus tendons. It also has superior attachment sites onto the sacroiliac joint capsule and sacrotuberous ligament. The piriformis extends, abducts, externally rotates the hip, acts as a portion of the pelvic floor in assisting sacroiliac joint stabilization, and controls anterior innominate movement. Piriformis pain and tenderness is a common finding in patients with chronic pelvic pain and overactive pelvic floor muscles [72]. This can be due to an upregulation of all of the muscles surrounding the pelvic floor muscles which share fascial connections, nerve

innervations, or have anatomical associations. Piriformis syndrome refers to a persistent pain in the buttock or hip caused by spasm, shortening, myofascial trigger points, and resultant compression on the sciatic nerve which lies beneath the piriformis muscle. Although the piriformis muscle has been suspected as the key to the anatomy and presentation of the nerves and blood vessels in the buttock, it is also possible that the obturator and gemelli are another common cause of neural compression. The compression can cause a peripheral neuritis of the sciatic nerve which can result in pain in the buttock that refers down the posterior leg and has also been referred to the deep gluteal syndrome [73]. Referred pain can occur to the posterior thigh and sacroiliac joint. Symptoms of piriformis syndrome can occur as a result of muscular compression of small nerves and vessels. The pudendal nerve and blood vessels can also compress the pudendal nerve as it exits the medial inferior surface of the piriformis [74]. Compression of the pudendal nerve can cause overactivity of the pelvic floor muscles and be the cause of chronic pelvic pain. Spasm of the piriformis muscle and sacral dysfunction such as torsion can tension the sacrotuberous ligament which results in compression of the pudendal nerve or cause mechanical stress on the ilium. This may lead to groin and pelvic pain and be a cause of dyspareunia. This syndrome is often mistaken for lumbar disc pathology, trochanteric bursitis, sciatica, pudendal neuralgia, or pelvic floor muscle dysfunction and can be related or caused by SIJD. Chronic piriformis syndrome can cause pelvic and lumbar spine mobility restrictions because of the muscular attachments. The obturator internus muscle tension along with overactive pelvic floor muscles can be a contributing factor in sciatic neuritis observed in patients with piriformis syndrome [75].

The piriformis sign, Freiberg sign, and Lasègue sign are all helpful in diagnosing piriformis syndrome [76, 77]. When assessing the patient, a positive piriformis sign is noted with the patient supine when the foot rests in external rotation and upon active internal rotation effort to bring the foot to midline causes pain. Freiberg sign is pain felt in the deep buttock region upon passive internal rotation of the hip. Lasègue sign is when localized pain is provoked when pressure is applied over the piriformis as the hip is flexed to 90° with the knee is extended. Patients exhibit tenderness or myofascial trigger points in the deep hip rotators, greater sciatic notch region, or sacroiliac joint upon palpation.

Treatment can include nonsteroidal anti-inflammatories, muscle relaxants, ice, physical therapy, acupuncture, trigger point injections with lidocaine hydrochloride, botulinum toxin type A, or surgical decompression.

7.11 Lumbar Spine Disorders

Lumbar spine disorders can be related to or cause overactive pelvic floor because of the physical attachments of muscles, ligaments, and nerves and the transfer of load of the ground reaction forces to the spine from the pelvic girdle. Lumbar spine disorders affecting the pelvic floor muscles can include disc pathology, end plate fractures, arthritis, facet or zygapophyseal joint irritation or subluxation, lumbar plexus irritation, or stenosis. Additionally, pelvic floor muscle spasm is usually associated with lumbar spine mobility impairments [43]. Joint restriction can be articular or myofascial or a combination of the two [20].

Disc pathology can include bulging, herniated, or degenerative disc disease. Disc pathology, stenosis, and facet joint irritation due to subluxation, misalignment, or hypomobility can cause inflammation or compression around the lumbar plexus nerves (L1–5). These include the iliohypogastric nerve (T12–L1), ilioinguinal nerve (L1), lateral femoral cutaneous nerve (L2–3), femoral nerve (L2–4), genitofemoral nerve (L1–2), and obturator nerve (L2–4). These nerves innervate areas including the mons pubis, pubis, inguinal region, groin, thigh, and genitals and can be a source of chronic pelvic pain which results in overactive pelvic floor muscles. Low

back pain can also result from these lumbar disorders causing a cascade of inhibited lumbopelvic muscle function and stability. Atrophy of the multifidus and poor recruitment of the transversus abdominis results [78]. As a compensation, the pelvic floor muscles could overactivate or stiffen to provide lumbopelvic stability for trunk motion to occur.

Gynecological conditions, such as endometriosis, menstrual cramps, fibroids, and pregnancy, sometimes are the cause of low back pain in women. Women with incontinence, respiratory disorders, and gastrointestinal symptoms have increased risk for the development of back pain.

Myofascial trigger points in the pelvic floor muscles can refer pain into the low back. Evidence of spine dysfunction in women with incontinence and respiratory disorders and the potential for accompanying viscerosomatic pain with gastrointestinal symptoms may provide a physiological explanation for these concomitant clinical challenges.

Medical treatment for low back pain includes medications such as nonsteroidal anti-inflammatories, muscle relaxants, opiate pain relievers, antidepressants, and anticonvulsants. Injections provided may include anesthetics, steriods, and Botox. Surgical options are discussed as well. Along with medications, rest, ice or heat, and exercise is advised. Acupuncture and chiropractic care may be helpful. Physical therapy is usually prescribed to realign the pelvic joints and spine, release neural tension, provide pain relieving modalities, instruct in proper posture and body mechanics, and instruct in therapeutic exercises focused on core strengthening, flexibility training, and improved movement patterns. Pelvic floor physical therapists would address the overactive pelvic floor muscles with myofascial release techniques, surface electromyography pelvic floor muscle biofeedback, and other down-training techniques. Because underlying weakness of the pelvic floor muscles exist when there is overactivity, pelvic floor muscle re-education and strengthening would also be a component of rehab upon resolution of the overactivity.

7.12 Abdominal Wall Pain

Abdominal wall pain can be caused by myofascial trigger points. Chronic abdominal wall pain is commonly misdiagnosed as arising from a visceral source. Lower abdominal muscles consist of the transversus abdominis, rectus abdominis, and internal and external obliques. Myofascial trigger points are taut localized bands within a muscle that upon compression refer pain to another adjacent site and may have autonomic response. Generally, trigger points are classified as active, causing pain at rest, or latent, causing pain when provoked. Abdominal wall myofascial trigger points can also have a twitch response or jump sign when provoked. Somatovisceral responses such as vomiting, nausea, gastrointestinal distress, urinary bladder, and sphincter spasm may result. Myofascial trigger points just above the pubis within the lower rectus abdominis and internal oblique can cause urinary frequency or retention and groin pain and in response the pelvic floor muscles can become hyperfacilitated and overactivity results. Carnett's test is often very valuable to the clinician for diagnostic purposes [79]. As the clinician increases pressure on the site of abdominal pain, the patient curls up their upper body tensing their abdominal muscles. If the pain in the abdomen worsens, it is considered positive for abdominal wall pain versus visceral disease.

Treatment can include physical therapy including massage, dry needling, trigger point release techniques, stretching techniques,cold laser, and therapeutic ultrasound. Other treatments include acupressure, trigger point injections with procaine (Novocaine) or lidocaine (Xylocaine), and oral medications such as antidepressants, neuroleptics, or nonsteroidal anti-inflammatory drugs.

7.13 Pudendal Neuralgia

Pudendal neuralgia (PN) is the existence of pain along the pudendal nerve distribution caused by inflammation, compression, or entrapment. The

pudendal nerve divides into three main branches as it passes near the ischial tuberosity: inferior rectal, perineal, and dorsal clitoral or penile. The pain can be experienced in the genitalia, along the ischial tuberosities, urethra, rectum, anus, lower gluteal, inner thigh, and refers pain to other regions of the pelvis. Symptoms generally worsen with sitting and improve upon sitting or lying down. Patients may complain that they have the sensation they are sitting on a "golf ball" or have a foreign body in their vagina or rectum. Symptoms may also include burning, numbness, aching, difficulty with urination of defecation, painful intercourse, allodynia, hyperesthesia, urination pain, and persistent genital arousal disorder (PGAD). Symptoms worsen as the day progresses. Causes of PN include physical trauma, entrapment, or compression to the nerve from falls, cycling, infections, pelvic surgeries, or inflammation. Other causes can be tumors, endometriosis, and chronic constipation. The pudendal nerve can also be compressed by tension or overactivity in the pelvic floor muscles or surrounding pelvic muscles. Hip capsular restrictions and impingement may cause protective pelvic floor and hip muscle tension and cause neural restriction along the course of or at the branches of the ipsilateral and/or contralateral pudendal nerve [80]. Chronic low-grade hip discomfort may provoke postural compensations and gait changes that places tension or torque on the deep hip rotators such as the obturator internus and thus the pudendal nerve. An inflammatory reaction in the injured hip joint may spread to the pelvic floor muscles and cause secondary inflammation of the ipsilateral pudendal nerve. Symptoms of PN can be unilateral or bilateral. Common sites of nerve compression are between the sacrospinous and sacrotuberous ligaments and at Alcock's canal.

Diagnostic tests include pudendal diagnostic nerve blocks pudendal nerve motor latency test (PNMLT), electromyography (EMG), 3T MRI, and magnetic resonance neurography (MRN). Other diagnostic criteria include the patient's history and symptoms. Manual Tinel sign, lightly percussing the nerve, along Alcock's canal is used by the evaluation as a diagnostic test. Nantes criteria for pudendal neuralgia caused by nerve entrapment is a newly adopted diagnostic guideline which includes: (1) Pain in the anatomical territory of the pudendal nerve, (2) Worsened by sitting, (3) The patient is not woken at night by the pain, (4) No objective sensory loss on clinical examination, (5) Positive anesthetic pudendal nerve block [81].

Treatment includes behavioral modification, physical therapy, analgesics and nerve blocks, surgical decompression, radiofrequency, and spinal cord stimulation. Physical therapy to address the overactive pelvic floor muscles, surrounding pelvic muscle myofascial trigger points, pudendal neural tension, and visceral restrictions may be very beneficial.

7.14 Persistent Genital Arousal Disorder

PGAD is characterized by patient report of spontaneous, intrusive genital arousal which is unwelcomed and disturbing in the absence of sexual interest or desire and persistent genital engorgement and erection can be present [82]. Symptoms include genital fullness, congestion, swelling, tingling, throbbing, and sensitivity for an extended period of time and do not subside completely on its own [82, 83]. This can occur with or without nipple swelling. Dyspareunia can be another complaint. Symptoms commonly occur at the clitoris and vagina while some also include the labia. Although several theories including psychological and physiological etiologies exist, mechanical theories are suggestive of overactive pelvic floor muscles as a cause. Overactive pelvic floor muscles, both superficial and deep, and/or associated overactive obturator internus can cause compression of the pudendal nerve facilitating sensitivity or irritation of the nerve and its end organs such as the perineum, labia, and clitoris. PGAD often presents with concurrent issues such as restless leg syndrome and overactive bladder syndrome proposing involvement of the

pudendal, ilioinguinal, genitofemoral, or iliohypogastric nerve increasing afferent sensations and increased vascular supply of the pudendal vessels [82]. Other causes suggested may include dietary such as consumption of phytoestrogens, medications such as serotonin reuptake inhibitors and serotonin–norepinephrine reuptake inhibitors, vascular issues such as pelvic congestion syndrome and varicosities, epileptic foci and other central nervous system pathology, and Tarlov cysts.

Assessment of patients with PGAD includes a thorough history and physical exam including gynecologic exam focused on the presence of hyperesthesia, engorgement, varices, and a pelvic floor muscle exam. Doppler ultrasound and pelvic MRI might helpful in diagnosing. Treatment options consist of cognitive therapy, acceptance therapy, medications such as anxiolytics and benzodiazepine, antidepressants, antidopaminergics, and antipsychotics. Physical therapy to address the overactive pelvic floor muscles, myofascial trigger points, and adverse neural tension may be very beneficial [82].

7.15 Conclusion

Overactive pelvic floor muscle dysfunction may be causal or a result of musculoskeletal dysfunctions. Local and referred chronic pelvic pain may be related to overactive pelvic floor muscles. Detailed history taking should include investigation of prior physical trauma and possible underlying or concurrent orthopedic conditions. Practitioners should consider the impact of the lumbo-pelvic-hip complex and the orthopedic disorders of each component of the complex on the pelvic floor muscles when evaluating and treating patients with chronic pelvic pain. The practitioner may question why the overactive pelvic floor is present and pay more detailed attention to the musculoskeletal structures. Screening tests appropriate for the gynecologist, urogynecologist, urologist, nurse practitioner, pain management, and family and rehabilitation medicine specialists are presented above. Collaboration among physicians, nurse practitioners, physical therapists, psychologists, and pain management specialists will provide a more comprehensive evaluation and plan of care intended to better serve the patient and to gain a better understanding of overactive pelvic floor muscle clinical presentations.

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Female Voiding Dysfunction

8

Asnat Groutz

8.1 Female Voiding Dysfunction

Lower urinary tract symptoms (LUTS) are very common among women and are usually categorized according to when they occur in the micturition cycle: the storage or emptying phase. Storage symptoms include urinary frequency, urgency, urgency urinary incontinence, and nocturia. Emptying symptoms consist of hesitancy, straining to void, intermittent urinary stream, poor stream, a feeling of incomplete bladder emptying and urinary retention. Most research on lower urinary tract function has previously focused on the storage phase of the micturition cycle, or the study of urinary incontinence. However, the availability and increased use of various treatment modalities, as well as new imaging techniques, have revived the clinical awareness and interest in female voiding phase dysfunction.

Conceptually, voiding phase dysfunction may be due to bladder and/or outlet causes. Bladder causes include detrusor contraction of inadequate magnitude and/or duration to effect bladder emptying (detrusor underactivity), or the absence of detrusor contraction (detrusor areflexia). Outlet causes consist of bladder outlet obstruction due to urethral sphincter over activity (functional obstruction), or anatomical pathologies (mechanical obstruction). The term detrusor/external sphincter dyssynergia (DESD) describes a detrusor contraction concurrent with an involuntary contraction of the urethral sphincter. DESD occurs in suprasacral neurological lesions.

Data concerning the prevalence of voiding phase dysfunction in women are scarce. Previous studies reported 2–25 % prevalence rates among women referred for evaluation of LUTS [1–6]. The most likely reason for this wide variation in reported prevalence rates is the lack of standard definitions for the diagnosis of female voiding dysfunction.

8.2 Diagnosis

No standard definitions exist for the diagnosis of bladder outlet obstruction in women. Relying on a history of obstructive symptoms is too restrictive. Many patients with bladder outlet obstruction present with various LUTS and correlation between obstructive symptoms and objective urodynamic findings is poor [3, 5].

The pressure-flow study is an objective urodynamic examination considered to be the best method to assess the voiding phase of the micturition cycle [6]. A noninvasive ("free-flow") uroflowmetry is a composite measure of the

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interaction between the pressure generated by the detrusor and the resistance offered by the urethra. Thus, a low uroflow may be due either to bladder outlet obstruction, or to impaired detrusor contractility. In order to distinguish between obstruction and impaired detrusor contractility, it is necessary to measure detrusor pressure and uroflow simultaneously. Ideally, the flow pattern in a pressure-flow study should be representative of the equivalent "free-flow" in the same patient. However, factors associated with the pressure-flow technique and setting may affect the voiding process. Specifically, the use of a transurethral catheter may potentially cause urethral irritation and/or relative bladder outlet obstruction during the study. Several investigators suggested strict urodynamic cut-off values of maximum flow (Qmax) and detrusor pressure at maximum flow (pdet.Qmax) for the diagnosis of female bladder outlet obstruction [7]. However, strict urodynamic cut-off values might fail to diagnose patients who are unable to void with urethral catheter in place, or those with "normal" uroflows despite the existence of a relative obstruction. These patients may be further diagnosed by using video urodynamics [4]. Other diagnostic tools are bladder outlet obstruction nomograms. Such nomograms, on the basis of pressure-flow data, are routinely used in the evaluation of obstructive uropathy in men. However, male nomograms are not applicable to women, since normal voiding detrusor pressure in women is significantly lower than in men. We have previously suggested a bladder outlet obstruction nomogram for women on the basis of maximum free uroflow and maximum detrusor pressure during voiding [8]. However, these parameters are still problematic as many women empty their bladder by increasing intra-abdominal pressure, or relaxing their pelvic floor.

8.3 Detrusor Underactivity

Detrusor underactivity and detrusor areflexia are common although poorly understood causes of female voiding dysfunction. Previous studies suggested age-related deterioration in detrusor contractility. Elbadawi et al. [9–11] showed histologic changes consistent with detrusor degeneration, as well as increased collagen content with age. Although these degenerative changes are not necessarily associated with voiding dysfunction, clinical urodynamic studies have demonstrated an age-related impaired bladder emptying. Similarly, detrusor ability to maintain a sustained contractile pressure was found to be reduced in old versus young animals [12, 13]. Pagala et al. [14] reported age-related, region-specific changes in contractile responses of the bladder. Isometric contractions of longitudinal detrusor, circular detrusor, and trigon segments of young and old rats were monitored after electrical, potassium, and bethanechol stimulation. Study results suggest that aging is associated with (1) a decrease in muscarinic receptor-mediated activation of contraction, especially in longitudinal detrusor, (2) an increase in collagen in the circular axis of the bladder that leads to decreased compliance and increased contractile response in the circular detrusor, and (3) decreased membrane depolarization in the trigon. These findings indicate that the effect of aging is specific to different regions and functional components of the bladder, probably due to changes in muscarinic receptors, collagen and depolarization.

Bladder over-distension due to impaired detrusor contractility may occur following pelvic surgery, labor and delivery, epidural anesthesia, anticholinergic medications, or in elderly women, without an obvious cause. Bladder over-distention may further cause ischemic and neuropathic changes within the bladder wall, resulting in irreversible detrusor damage [15, 16]. Treatment modalities include elimination of reversible causes (such as drugs), timed voiding, or intermittent catheterization. Jhang et al. [17] treated 31 women with detrusor underactivity and urine retention in whom medical treatment failed by transurethral incision of the bladder neck. Intermittent catheterization was needed in 27 patients before surgery and in only seven after surgery. Three patients developed transient urinary incontinence, and one developed vesicovaginal fistula after surgery.

8.4 Anatomical Bladder Outlet Obstruction

Previous anti-incontinence surgery and severe urogenital prolapse are the most common anatomic etiologies of bladder outlet obstruction, accounting for half of the cases [5]. Postoperative voiding dysfunction was found to be associated with type of surgery, advance age, previous vaginal bladder neck suspension, increased volume at first sensation on bladder filling, high preoperative post-void residual urine volume, and postoperative lower urinary tract infection [18]. Women who developed voiding dysfunction following anti-incontinence surgery should be managed by draining the bladder with an indwelling catheter or clean intermittent self-catheterization for up to 6 months postoperatively [19]. Alternatively, early mobilization of the tape without division or excision may be undertaken. Price et al. studied 33 patients who underwent early tape mobilization after TVT surgery. Voiding dysfunction resolved in 29 patients with no recurrence of stress urinary incontinence. For women with persistent obstructive symptoms despite conservative management, a more invasive approach may be indicated. Urethrolysis with incision or excision of the tape have been suggested as an effective treatment; however, recurrent stress urinary incontinence was reported in up to 50 % of these cases [20–23].

8.5 Functional Bladder Outlet Obstruction

Normal voiding is achieved by a sustained detrusor contraction synchronized with urethral sphincter relaxation. Inappropriate sphincter activity during voiding, in the absence of known neurological disease, may result in functional bladder outlet obstruction. Voiding dysfunction in otherwise healthy and neurologically intact patients was originally described in children (Hinman syndrome) and young women (Fowler's syndrome). This disorder is believed to be due to a primary failure of relaxation of the striated urethral sphincter. Whether these two syndromes represent two different entities, or share the same pathophysiology, is unclear.

8.5.1 Hinman Syndrome

In 1971, Hinman [24] presented 14 boys with typical characteristics of the nonneurogenic, neurogenic bladder syndrome and suggested that these changes were behavioral as demonstrated by their reversal by suggestion hypnosis, and by the absence of any detectable neurological or obstructive abnormality. Hinman concluded that "since these children usually are toilet trained initially, the incoordination appears to be a learned behavior or habit, perhaps as a response to underappreciated detrusor contractions. Reversal of the syndrome is achieved by suitable medication and by some form of suggestion or retraining." Typically, children with a nonneurogenic, neurogenic bladder present with frequency, urgency, urinary incontinence, recurrent urinary tract infections, or occasionally, encopresis. Further evaluation may reveal signs of obstructive uropathy, such as trabeculated bladder, elevated post-void residual urine volume, hydronephrosis, and vesicoureteral reflux, in the absence of any identifiable neurological or obstructive abnormality. Urodynamically, these children have uncontrolled detrusor contractions that they fail to inhibit, and they do not coordinate these contractions with concomitant sphincteric relaxation. In time, not only do they find it difficult to inhibit detrusor contractions, but they also find it difficult to keep the sphincter relaxed when voiding occurs.

In children, functional voiding dysfunction is usually acquired after toilet training, reaches its peak of destructiveness in late childhood, and tends to resolve after puberty [24–27]. Although it may persist, or even first manifest, later in life, data concerning functional voiding dysfunction in adults are scarce. George and Slade [28] reported a series of 16 men (mean age, 42 years; range, 29±55 years) referred for evaluation of refractory LUTS. Their main symptoms were urinary frequency, hesitancy, intermittent stream, and the inability to void in public places. These symptoms were found to be associated with a high incidence of dyspepsia and anxiety. The investigators suggested the existence of a chronic systemic state and proposed the term "anxious bladder." Jorgensen et al. [29] reported the symptomatology

and clinical manifestations of "idiopathic detrusor sphincter dyssynergia" in neurologically normal patients referred for evaluation of voiding symptoms. Diagnosis was established by the following criteria: (1) two flow curves obtained in privacy showing a characteristic intermittent pattern and (2) simultaneous record of pressure-flow parameters and electromyography (EMG) demonstrating intermittent pelvic floor activity during micturition. Twenty-three patients (0.5 % of the study population) fulfilled these criteria. The mean age of these patients was 27.4 years (range, 5–72). Further differentiation between children and adults was not carried out. Groutz et al. [30] suggested the term "learned voiding dysfunction" and used the following clinical and urodynamic criteria to establish the diagnosis: (1) a suggestive clinical history, i.e., LUTS and difficulty in voiding in public places, or during uroflowmetry/urodynamics, having to concentrate, relax, touch genitalia, listen to running water, etc.; (2) intermittent "free" uroflow pattern; (3) exclusion of neurological disorders, or anatomical causes of bladder outlet obstruction; and (4) demonstration of typical external urethral sphincter contractions during micturition with either needle EMG, or fluoroscopic visualization of the urethra during voiding. The urethra is usually dilated to the level of the external sphincter, while the bladder neck is wide open, distinguishing dysfunctional voiding from primary bladder neck obstruction. Using these strict criteria, 2 % of 1015 consecutive adults referred for video-urodynamic evaluation of LUTS were found to have learned voiding dysfunction. Other patients, with presumed learned voiding dysfunction, who did not undergo video-urodynamics were not included. Thus, the prevalence of learned voiding dysfunction among adults referred for evaluation of LUTS is likely to be even higher. Contrary to children, in whom the main subjective hallmarks of the syndrome are urinary incontinence and recurrent urinary tract infections, adult patients present mainly with obstructive and/or irritative symptoms, while urinary incontinence is less prominent [30, 31]. Functional voiding dysfunction may also be associated with transient postoperative urinary retention. FitzGerald and Brubaker [32] studied 10 women who underwent Burch colposuspension, or sub-urethral sling surgery. Voiding trials were performed 1–2 days after surgery under simultaneous monitoring of the urethral sphincter EMG activity and intravesical pressure. Six patients were unable to void and demonstrated persistent EMG activity. Four of these demonstrated no detrusor contraction, whereas two demonstrated detrusor contractions. The authors concluded that failure of relaxation of the striated urethral sphincter contributes to postoperative urinary retention.

Optimal management of children with Hinman syndrome requires accurate and timely diagnosis. Hypnosis was the first modality successfully applied, in combination with anticholinergics and antibiotics [26]. Over the years, suggestion, retraining, bladder drill, and biofeedback have been combined with pharmacologic therapy to treat detrusor overactivity, obtain striated muscle relaxation, or to inhibit contraction of the α-adrenergic innervated bladder neck. Although potentially successful in more than 80 % of cases, the treatment can span 6 weeks to several years with occasional relapses, requiring cooperation and determination on the part of the child and his/her family [33–36]. Subjects with irreversible renal damage at the time of diagnosis and those who do not respond to, or drop out of behavioral therapy are candidates for reconstructive surgery, dialysis, or renal transplant. Reparative operations will fail if performed before a diagnosis of dysfunctional voiding and optimization of the involved variables [26, 36].

Data concerning the management of functional voiding dysfunction in adults are scarce. The correct diagnosis relies on detailed history corroborated by urodynamic studies with EMG or fluoroscopy, and may be missed due to inadequate awareness or instrumentation. Deindl et al. [37] suggested two different pathogenetic mechanisms of functional urethral obstruction in women with dysfunctional voiding and/or urinary retention. The activity patterns during micturition of both pubococcygeal muscles and the striated external urethral sphincter were assessed using two different EMG techniques. Four women were found to have inappropriate urethral sphincter activation during micturition. Eleven others had inappropriate

contractions of the pubococcygeal muscles with no abnormality of the external sphincter. Biofeedback training led to improvement in women with pubococcygeal activation, but not for those with inappropriate urethral sphincter activation. Minardi et al. [38] randomized 86 women with recurrent urinary tract infections and dysfunctional voiding to receive uroflowmetry biofeedback (group 1), biofeedback training of the pelvic floor muscles (group 2), uroflowmetry biofeedback combined with biofeedback training of the pelvic floor muscles (group 3), or no treatment (group 4). The prevalence of LUTS and urinary tract infections, as well as uroflowmetry parameters, was significantly improved in all three treatments groups, with no improvement in the control group. Chen and Kuo [39] treated 168 women with dysfunctional voiding by antimuscarinic or α-blocker drugs according to their chief complaint of storage or voiding LUTS. The success rates were 41.2 % for antimuscarinic therapy and 51.9 % for α-blocker therapy in patients with storage and voiding LUTS, respectively ($P=0.36$). Botulinum toxin urethral sphincter injection to restore bladder emptying in patients with voiding dysfunction has also been used with some success, but data are limited [40]. Patients who fail conservative therapies are candidates for a neuromodulation trial and, so far, outcome data are encouraging. Peeters et al. [41] recently presented long-term outcome results of sacral neuromodulation for LUTS. Overall, 217 patients (86 % female) received an implantable generator (Interstim™) between 1996 and 2010. The mean duration of follow-up was 47 months. Success (defined as ≥50 % improvement in voiding diary variables) and cure rates for patients with idiopathic urinary retention were 73 % and 58 %, respectively.

8.6 Fowler's Syndrome

In 1988, Fowler et al. [42] presented the concept of voiding dysfunction due to abnormal EMG activity of the urethral sphincter among young women, 64 % of whom also had polycystic ovaries. It was speculated that the EMG disorder is a local, hormonally determined condition, which allows direct muscle to muscle transmission. The mean age of these women at the onset of complete retention was 27.7 years (range 10–50). Mean maximum bladder capacity at the initial episode of complete retention was 1208 mL, and 65 % reported a specific event that had apparently precipitated urinary retention, most commonly a gynecologic surgical procedure using general anesthesia [43]. The nature of the EMG activity was such that it suggested a muscle membrane disorder and, therefore, a primary disorder of the sphincter relaxation rather than inappropriately timed sphincter activity that occurs in neurogenic detrusor-sphincter dyssynergia [42]. Further, the involuntary contraction of the sphincter might cause reflex inhibition of detrusor contractions [44]. The hypothesis of impaired sphincter relaxation was confirmed by the recording of repetitive discharge activity using special hooked wire electrodes [37]. Other investigators reported increased urethral pressure profile and increased sphincter volume on ultrasound in these patients [45]. It was therefore speculated that the abnormal activity in the urethral rhabdosphincter leads to hypertrophy of these myofibers. However, core needle biopsies of the urethral rhabdosphincter failed to reveal any increase in fiber diameter. Further investigation using electron microscopy revealed that these myofibers contain excessive amounts of sarcoplasmic glycogen and mitochondria, suggesting increased metabolic activity of these cells [46]. Urinary retention in patients with Fowler's syndrome is unlikely to resolve without treatment, and sacral neuromodulation is the only intervention that has been demonstrated to restore voiding [43, 47].

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Overactive Pelvic Floor: Gastrointestinal Morbidities

9

Marc Beer-Gabel

9.1 Pelvic Floor Anatomy

The pelvic floor is a complex anatomical unit grouping muscles, connective tissues, and bones. The pelvic muscular diaphragm is composed of the coccygeus muscle posteriorly and the levator ani anterolaterally. The levator ani consists of the iliococcygeus, the pubococcygeus, and the puborectalis muscle group. Through these structures pass the urological, genital, and intestinal tracts. The pelvic floor supports the pelvic organs and the spine. It is a complex functional unit involved in continence and evacuation of urine and stool, sexual function, and childbirth. However, the pelvic floor muscles are also part of a much wider muscular system known as the abdominal core, and act together with the diaphragm, the lower back, and the abdominal wall muscles influencing spinal stability, body posture, and breathing. The pelvic floor muscles connect with some of the muscles of the thigh and as such are activated during walking, running, and other dynamic activities. The complex functions of the pelvic floor, which are both voluntary and reflexive, include visceral and somatic activities; they are explained by their innervation through the co-activation of the somatic and the autonomic nervous system. The pelvic innervation, controlled by the supraspinal centers, explains the connections between the various pelvic organs and between these organs and their muscular links. Therefore, the pelvic floor muscles are part of a complex adaptive system, which is under the control of brain centers. The role of the pelvic muscles is related to the function of the pelvic organs and of the core abdominal muscles and as such, the pelvic muscles represent a link between the inner and the outer world. The consequence of this complexity is that their function may be altered by any of the components of this complex system.

Weakness or relaxation of the pelvic floor results in disorders such as urinary or fecal incontinence and pelvic organ prolapse, which are easily recognized. Overactive pelvic floor is more challenging to diagnose as symptom presentation may be varied and are frequently not attributed to the pelvic floor muscles. Descent of the pelvic floor organs to the perineum isn't typically observed in overactive pelvic floor. The symptoms of an overactive pelvic floor are often nonspecific and are frequently related to abdominal organ dysfunction or to muscle pain, which presents as a dull and diffuse sensation. Defecation disorders, voiding and/or sexual dysfunction and pain are the main expressions of an overactive pelvic floor. These symptoms do not always point clearly to the involvement of the pelvic floor muscles.

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9.2 Pelvic Floor Function and Gastrointestinal Involvement

In this paragraph, we will not describe the entire spectrum of the pelvic floor's functions. Rather, we will restrict the scope to pelvic floor function as it relates to digestion and abdominal and pelvic pain. In recent literature, pelvic floor dysfunction is referred to by various terms. Several terms relate to the pathophysiology of the condition such as short pelvic floor [1], myofascial pelvic pain [2], non-relaxing pelvic floor [3], and hypertonic pelvic floor [4]. Other terms relate to the anatomical aspects of the dysfunction such as levator ani syndrome, puborectalis syndrome, piriformis syndrome, coccygodynia, although the pain was not always located in the coccyx [5]. Some more generalized terms, such as idiopathic perineal pain, are also used. This diverse terminology shows differing pathophysiological understanding for the same condition.

Defecation is a complex integrative function that involves the digestive tract, the anorectum, and the pelvic floor. The foregut is innervated by the vagal system, the hindgut by the sacral parasympathetic nerves, the levator ani by sacral nerves, the internal anal sphincter by nerves originating from L4 level, and the external anal sphincter by the pudendal nerve. Only the latter is volitional. Therefore, synchronization of the autonomic and somatic neural functions of the pelvic floor muscles is essential in order to allow normal defecation and bladder evacuation.

An overactive pelvic floor may inhibit activity in the lower rectum and reduce the urge to defecate or create a sensation of incomplete defecation [6, 7]. If the patient strains and employs Valsalva breathing in order to defecate, the over activation of the abdominal wall muscle will co-stimulate the pelvic floor muscles and impede defecation [8, 9].

9.3 Mechanisms of Overactive Pelvic Floor: A Three-Dimensional Approach

The integration of pelvic floor functions is learned during childhood. To understand the complaints of a patient who suffers from pelvic floor dysfunction, we must investigate the acquisition of the various pelvic floor functions along a three-dimensional system. Those three dimensions are composed of the horizontal axis (pelvic floor structures), the vertical axis (the interaction with the central nervous system), and finally the axis of time. The central nervous system and brain axis are of particular importance as they link the inner and external world. Symptoms of pelvic floor dysfunction or pain generally occur after a long period of imbalance induced by either poor posture, a lack of synchronization between the viscera and the muscles, local persistent injuries, or stress. Each component of this complex circuitry may affect pelvic floor function.

Defecation disorders are a common cause of overactive pelvic floor and may be acquired in early childhood. Toilet training is the beginning of the cognitive control of continence. If the need to defecate is not properly perceived, recognized, or accepted, then continence may be affected. Children may learn to use a withholding mechanism and abstain from defecation, leading to inappropriate activation of the neuronal circuitry linking the rectal nerves to the sacral plexus, the spine and the brain, resulting in future abnormal behavior. Approximately 50 % of constipated children contract rather than relax the external anal sphincter during defecation [10]. This retentive habit may progress to encopresis. Eventually, this mechanical barrage will affect other anorectal functions. In one study, it was demonstrated that 95 % of children with idiopathic constipation have impaired rectal sensation and weakening of rectal contraction during distension. This mechanism contributes to diminished rectal evacuation [11]. One-third of children with idiopathic constipation who were followed up beyond puberty continued to report severe complaints of constipation [12]. Over time, this dysfunction may become painful. About half of children with acute abdominal pain suffer from constipation, which was considered to be the cause of the pain [13]. Prolonged contraction of muscles activate locally the free ends of afferent nerves fibers of the group III (thin myelinated fibers) and group IV (non-myelinated fibers), which transmit pain.

Constipated children frequently complain about urinary dysfunction such as urinary tract

infection (UTI) in 11 % of the cases and urinary incontinence in 63 % of the cases. The association of urinary dysfunction with constipation is supported by the observation that resolution of fecal retention leads to the disappearance of daytime urinary incontinence in 89 % of the cases and of UTI in 100 % of the children [14].

There is a significant association between early sexual abuse and gastroenterological functional symptoms [15]. In a study published in 1995, patients with a history of sexual abuse were more likely to complain of both constipation and diarrhea. Anismus, a condition characterized by anal muscle contraction, was more frequent in sexual abuse survivors, suggesting a perturbation of pelvic floor function [16]. In other studies on sexually abused children, gastrointestinal disorders met the diagnostic criteria for somatization disorder, presenting with hypervigilance, anxiety, and psychiatric disorders. These patients have poor quality of life due to health-related issues, utilize the health care system more often, and report more pain [17].

Visceral insults may express themselves as musculoskeletal pain. Visceral pain is difficult to diagnose when it is not related to obvious inflammation, tumor, or to structural abnormalities. Visceral pain typically is not felt in the organ in which it is generated but in a distant muscular or cutaneous area of reference. This phenomenon is related to the dermatome organization of the nervous system [18]. Organ dysfunction and pain without sign of organic disease are by far more frequent [19].

These syndromes have in common a state of visceral hypersensitivity with a lower threshold of pain. Peripheral nociceptors are more responsive than normal to painful stimuli (allodynia and hyperalgesia) and this leads to central sensitization at the level of the dorsal horn. The phenomenon of viscerovisceral and visceromuscular sensitization appears and a "wind up" mechanism stimulates the brain centers and the autonomic system. The descending pain pathway becomes disinhibited and a state of chronic pain takes place.

In the periphery, the pelvic floor muscles shorten, weaken, and become a source of pain. The pain is myofascial, dull, and diffuse and is characterized by the presence of trigger points. Trigger points are sensitive spots found in tense muscles. Administering pressure to these trigger points produces pain and evokes projected pain in regional muscles. Locally, palpation of these muscles may induce a muscular twitch reaction. Initially, the muscles are thick and overactive. When the cause persists, muscles remain chronically tight, blood flow is decreased and local hypoxemia leading to reduced muscle energy is observed. This process increases muscle pain. Eventually, local muscular shortening will give rise to taut bands and tender points resulting from hypersensitivity of the neural pathway in the muscles. This hypersensitivity is due to sensitization of muscle nociceptor group III and IV afferent fibers, leading to central nervous sensitization in the spinal cord and brain. The patient may also struggle with a state of hypervigilance and stress according to the patient's personality or experiences. If muscle contraction is maintained, a vicious cycle may take place. When this state turns chronic, the muscles will become fibrotic and weak.

The causative factors at the origin of this dysfunction may be related to pelvic viscera or to the perineal muscles. The "cross talk" between pelvic organs and the pelvic muscular layers can enhance further dysfunction and pain. Many patients have more than one underlying cause for their pain.

During chronic irritation, a negative interaction between the organ and muscles may occur. Evacuation disorders may or may not be accompanied by pain. A persistent contraction of the levator ani muscle may complicate organ dysfunction further. Persistent levator ani contraction can be the result of skeletal imbalance, poor learned defecation habits, a chronic visceral injury such as the neuroinflammation seen in irritable bowel syndrome (IBS) or interstitial cystitis, or a guarding reflex. This situation may be clinically confusing since dermatomal referred pain may last years after the primary injury is treated [20].

9.4 Clinical Manifestations

The dominant or initial symptoms of overactive pelvic floor may be triggered either by visceral injury or myofascial pain. The pain may or may

not be associated with GI symptoms. When GI complaints do occur, typical complaints are: The patient can experience constipation with obstructive defecation, a sensation of incomplete rectal evacuation or anal blockage and a change of behavior with excessive straining to defecate and/or rectal digitation. These unpleasant sensations will sometimes induce changes in behavior such as the need to use rectal digitation, recurrent defecation attempts, or vaginal splinting in case of rectocele. Constipation may be associated with pain during or after rectal evacuation, caused by tension of the hypertonic pelvic muscles. Pain may be also related to IBS and to rectal hypersensitivity [21, 22]; however, its origin is often unclear. It is vague, dull, persistent, and enhanced by muscle activation. It is defined as a sensation of deep perineal pressure often described as a "tennis ball stuck in the rectum." The pain may be related to dyssynergic defecation and excessive abdominal strain. In certain cases, the patient describes a cramping pain.

Pain is often more frequent during the second part of the day, absent at night and aggravated by prolonged sitting. It is often erroneously considered as pain of rectal origin although it is mainly of muscular origin. This is maintained by the Rome III classification of functional anorectal and pelvic pain, which is primarily symptom based. It divides these pains into two categories:

- Chronic proctalgia
- Proctalgia fugax

These categories are defined based on duration. Chronic proctalgia lasts more than 20 min. The symptoms must be present for 3 months or more in the last 6 months preceding the diagnosis. All organic causes of rectal pain must be ruled out. Chronic proctalgia is subdivided into two diagnoses:

1. Levator ani syndrome. The diagnosis of levator ani syndrome is made by clinical examination. There is a characteristic discomfort or pain upon digital posterior traction of the muscle. If no tenderness is observed during pressure application, unspecific functional anorectal pain is diagnosed. Pain caused by levator ani tension is classified as a "rectal pain." This classification is misleading as it is of muscular (levator ani) and not visceral (rectum) origin.
2. Unspecified functional anorectal pain. The clinical examination does not help detect location to the pain.

Anorectal functional pain is mainly associated with muscular pain at the level of the puborectalis. Pain originating at the rectum, typically caused by mechanical rectal distension, is felt mainly in the lower left abdomen although it also projects to the S3 dermatome and musculotome as well. Chronic visceral pain is referred and felt in the corresponding dermatome through mechanisms of visceromuscular convergence at the sacral posterior dorsal horn level. The innervation of the rectum and levator ani originates from the sacral plexus S2–S4. This may explain why a tense levator ani and rectal pain may share the same clinical expression. Therefore, pain in these dermatomes can be purely of muscular origin but may be also caused by any of the pelvic organs sharing the same sacral innervation. Pelvic pain may be generated by other pelvic organs such as:

- Painful bladder—characterized by frequent urination, urgency, and chronic bladder-related pain.
- Vulvodynia—characterized by a burning sensation and tenderness of the vulvar introitus.
- Chronic prostatitis.

The link between rectal pain and the pelvic organs is through viscerosomatic convergence, as previously described.

Pain can be also evoked by other pathologies which should be excluded by a thorough examination such as:

- Strained hypertonic pelvic floor muscles related to anal fissure
- Prolapse of internal hemorrhoids
- Rectal mucosal prolapse with a recto-anal intussusception
- Overt rectal prolapse

Pain is a subjective experience triggered by peripheral causes. Symptoms are the result of integration of visceral disease, overactive muscles, and the cross talk between organs and muscles under the control of the central nervous system. It is influenced by the cognitive and emotional status of the patient. It is essential to examine the whole pelvic floor including pelvic organs and to assess for neuromuscular involvement before deciding on the best course of treatment.

9.5 Clinical Examination

There is no well-established standard for clinical assessment of pelvic floor function. Since pain may be generated by any pelvic structure or referred to the pelvic floor, the examination has to address all possible origins. Inspection of the perianal area will exclude any periorificial pathology or itching lesions. Overactive pelvic floor should be assessed by inspection of the perineum while the patient contracts the perineum as if trying to stop micturition and then relaxes. A normal reaction of the perineum would be to be lifted up and then return to the resting position. If the patient cannot relax the pelvic floor muscles upon demand or during a push down effort, a non-relaxing perineum is diagnosed. Some patients will even contract the pelvic floor while bearing down, thus demonstrating a complete inversion of the muscular command, known as dyssynergy.

Digital anal examination is performed in order to assess relaxation or contraction of the perineal muscles. Pain or tenderness of the puborectalis is evaluated by exerting mild pressure with the index finger on both sides of the posterior puborectalis sling. If the patient is reluctant to undergo anal digitation, perineal contraction assessment can be carried out through a single finger insertion in the distal vagina when the patient is lying in the left lateral position. In case of symptoms of obstructive defecation without pain, local examination may be sufficient. In case the pain is associated with other visceral signs, the examination should assess the whole pelvic floor. Other superficial and deep pelvic muscles should be palpated to determine their tonus and map the presence of taut bands and trigger points (from posterior to anterior successively the coccygeus, piriformis, internal obturator, pubococcygeus, iliococcygeus, superficial transverse perineal, perineal body, bulbospongiosus, and ischiocavernosus muscles). The coccyx, the ischial spine, and the pubis should also be palpated to assess for tenderness. When pain is the dominant symptom, a global evaluation of the pelvic floor, the gluteal muscle, lower abdomen, and lower back are also important, since pain may be associated with muscle contractions or be of referred origin. This pain-mapping is of paramount importance for establishing the correct diagnosis and plan treatment accordingly.

9.6 Pelvic Floor Investigations

Examinations are needed to help establish the cause of difficult defecation or to evaluate patients with obstructive defecation. In most cases, an anorectal manometry and balloon evacuation test (BET) will suffice to demonstrate defecating dysfunction.

9.7 Anorectal Manometry and Rectal Balloon Evacuation Test

Manometry is a clinical evaluation tool used to assess anal resting pressure, elevation of the anal basal pressure at strain and rectal sensitivity. Manometry techniques have evolved over the years from water perfusion to solid-state microtransducers, which now allow to perform high-resolution manometry. During normal defecation, the intrarectal pressure increases while the anal pressure is supposed to decrease. In the case of defecation disorders, the rectoanal gradient diminishes [23].

The cause may be low rectal pressure during straining or a paradoxical contraction of the anus. In such cases, strong traction forces on the external anal sphincter and on the puborectalis muscle [24]. As time goes on, this repetitive high pressure may elicit muscular pain from overloaded

muscles. Patients suffering from overactive pelvic floor may have a high anal resting pressure. But there is an imperfect correlation between the elevation of anal pressure at strain during anorectal manometry and the delay to evacuate a rectal balloon at strain [25].

A rectal balloon expulsion test is an excellent adjunct to diagnose obstructive defecation. It is a simple, inexpensive procedure. The technical conditions of this method are not standardized, so the results in the literature are disparate. Most often, patients are asked to expel a rectal balloon filled with 50–60 mL of lukewarm water, in a private commode. The time it takes to expel the balloon or the number of attempts done is rated. As an alternative, variable filling volumes can be used which are superior to the volume perceived by the patient as a need to defecate. The variation in modalities by which this test is carried out explains the wide variation in outcomes reported in the literature. Minguez et al. reported a sensitivity of 88 % and a specificity of 89 % with a negative predicating value of 97 % [26]. On the other hand, Rao et al. found opposite results, showing this test to have low sensitivity and specificity [27]. Combined with a balloon expulsion test, anorectal manometry confirms a diagnosis of obstructive defecation [28].

9.8 Imaging

A detailed description of imaging techniques for pelvic floor assessment is beyond the scope of this chapter and can be found in Chap. 13 of this book.

Imaging studies can be ordered to diagnose a pelvic floor defect, if a rectocele or an enterocele are suspected upon clinical examination. Pelvic floor imaging will define the opening of the posterior anorectal angle at strain, the quality of rectal evacuation and the synchronization of muscle activation. Additionally, imaging may demonstrate organ displacement during rectal evacuation and provide further information regarding posterior pelvic floor function in more complex cases. These investigations can exclude organ pathology. Several imaging techniques are used to diagnose functional or structural pelvic floor disorders which can disturb the defecation process. X-ray defecography is the oldest method. It is however limited to the study of the posterior pelvic compartment. MR defecography can show the anterior and the posterior pelvis without the inconvenience of using pelvic irradiation. It is a good imaging method for the evaluation of defecation disorders in 94 % of patients, selected by clinical complaints [29]. Ultrasound is an excellent way to diagnose the defecation functional and anatomical disorders since it can visualize at the same time the anterior and the posterior pelvic compartments, showing the dynamics of a simulated rectal evacuation and its influence on the whole pelvic floor without exposure to irradiation. We described the technique of dynamic transperineal ultrasound and we recently compared ultrasound defecography to X-ray defecography. The results of our study demonstrated good agreement between DEF and DTP-US for the detection of posterior pelvic floor dysfunctions at strain, in patients suffering from any kind of defecation disorders [30–32].

A very high accuracy of DTP-US was found for the detection of large rectocele, enterocele, intussusception, and rectal prolapse (92 %, 89 %, 83 %, and 94 %, respectively). The level of concordance was good for the diagnosis of mid-size rectoceles (74 %) [33]. In all cases of new onset constipation, in particular those associated with rectal bleeding, cancer of the colon should be ruled out.

9.9 Treatment

Treatment varies according to the clinical presentation. The physician should determine which the dominant complaint is: pelvic dysfunction or pain, and whether they are associated.

9.9.1 Patient Education and Reassurance

In all cases, the first step is to reassure the patient that the pathology is of benign nature [34]. Patients should then be informed about anatomy

and function of the pelvic floor and the anterior abdominal wall muscles. Patient can be trained to palpate their perineal and abdominal muscles and become able to differentiate between relaxed and tense state. A model of integration and synchronization of the pelvic and abdominal muscles can be graphically shown and simply explained. It may be helpful to show the patient a diagram demonstrating the interrelation between anorectal and perineal muscles.

9.9.2 Nutritional Management

Treatment for constipation related to an overactive pelvic floor includes providing suggestions for dietary interventions. The patient's diet should include 20–25 g of fibers with consequent hydration and regular exercise. If this treatment is not sufficient, the patient should take a supplement of osmotic laxatives such as polyethylene glycol or lactulose.

9.9.3 Biofeedback

If obstructive defecation is the main symptom of overactive pelvic floor, biofeedback should be prescribed. Biofeedback treatment of constipation due to OPF has been demonstrated to be more effective than polyethylene glycol [35] and superior to diet and exercise [36]. The course of treatment should be based on clinical examination and anorectal manometry. The patient should be taught to augment the abdominal pressure, while doing a Valsalva maneuver to lower the diaphragm. At the same time, the patient should learn to distinguish the pelvic floor muscles from the abdominal muscles and avoid squeezing the perineal muscles when bearing down to evacuate the rectum and progressively relax them instead. An intrarectal balloon can give a feedback measure of the intra-abdominal pressure. An anal balloon or superficial EMG sensors will show the tension of the perineal muscles on a computer screen. The therapist may help the process by gently pulling out the rectal balloon. In the case of abnormal rectal sensation, the patient should be offered a sensory program to normalize the threshold pressure of the urge to defecate. This method can be used for patients with either high or low rectal sensitivity. Patients will generally improve their threshold sensitivity for the urge to defecate.

When pain accompanies the diagnosis of levator ani syndrome, biofeedback is strongly recommended. Chiarioni et al., in a prospective study comparing various modalities in the treatment of levator ani syndrome, demonstrated adequate relief of pain in 87 % of the biofeedback group and only 45 % of the electrostimulation group. The benefits of biofeedback were maintained for 1 year following treatment cessation [19] A thorough discussion of EMG biofeedback is available in Chap. 12.

9.9.4 Electrical Stimulation

Electrical stimulation has been demonstrated to be superior to digital massage but inferior to biofeedback. In the previously mentioned study by Chiarioni et al., adequate pain relief was reported by 87 % for biofeedback, 45 % for EGS, and 22 % for massage. Pain intensity decreased from 6.8 (0–10 scale) at baseline to 1.8 after biofeedback, 4.7 after EGS, and 6.0 after massage. Improvement was maintained for 12 months [19]. The same protocol of biofeedback as for dyssynergic defecation was used for patients suffering from levator ani pain. This study showed that the improvement in relaxing the pelvic floor muscles when straining and the improved ability to evacuate a balloon was efficient in lowering pelvic pain level. This confirms that levator ani syndrome and dyssynergic defecation appear to represent different manifestations of the same underlying disorder.

9.9.5 Botulinum A Toxin

A small, randomized controlled crossover study compared the effects of 100 units of botulinum A toxin vs. placebo injections into the levator ani showed no significant benefit [37]. However, other uncontrolled studies show that botulinum

toxin injection is an effective therapy for anorectal dysfunction in patients with defecation disorders [38]. A placebo controlled trial evaluating BoTox injection in patients suffering from chronic pelvic floor pain with muscular spasm, demonstrated a reduction in vaginal pressure, but the pain was reduced only partially and did not differ from placebo [39].

9.9.6 Local Analgesic Treatment

Injections of 10 cc of 0.25 % bupivacaine, 10 cc of 2 % lidocaine, and 1 cc (40 mg) of triamcinolone were given in the levator ani in a short-term prospective study. Thirteen of 18 women improved with the first trigger point injection resulting in a comprehensive success rate of 72 %. Six (33 %) of 18 women were completely pain free. However, this study was performed on a small selected group of non-randomized patients [40].

9.9.7 Oral Medications for Chronic Pain

When the pain is severe, it must be treated as a disease that involves the central nervous system and not only its peripheral origins. Tricyclic antidepressants should not be used as the first-line therapy in patients with levator ani syndrome and pain since they may aggravate constipation. Gabapentin and pregabalin can be used, as well as SNRIs, to elevate the pain threshold in the most severe cases, like in other pain management protocols, but there are no studies to evaluate the efficacy of these medications in this context [41].

9.9.8 Sacral Nerve Neuromodulation

It is unclear whether sacral modulation is an appropriate and effective treatment method for treating pelvic pain. There are contradictory results in the literature with only small, non-randomized studies [42, 43]. In a review of studies of seven centers, Baeten reported good short-term outcome in patients treated for severe constipation. Percutaneous nerve evaluation, which is the first step of treatment, is indicative of sacral nerve neuromodulation (SNM) efficacy [44]. In a series of patients with refractory constipation, sacral neuromodulation showed good results in 50 % of the patients. Approximately 90 % of the patients who benefited from SNM maintained improvement over a median follow-up period of 38 months (18–62 months) [45]. In another study, 12 patients suffering from prolonged refractory chronic anal and perineal pain were implanted with sacral neuromodulation. After a mean follow-up of 15 months (range 3–80 months), visual analog pain scores had significantly improved [42].

9.10 Conclusion

GI comorbidities are part of a spectrum of symptoms observed in patients with overactive pelvic floor. GI dysfunction may be the cause, or the consequence, of an overactive pelvic floor, through the process of spinal neural viscerosomatic convergence and should be assessed in each patient presenting with GI complaints. Dysfunction refers to dysregulation of a complex network integrating many components in the central nervous system and in the periphery which together execute a specific function. Defecation is an acquired function requiring sensory and motor coordination, through conscious and unconscious mechanisms. It is under voluntary control through pelvic floor muscle activation, when withholding an urge to defecate. The rectum and most pelvic muscles share the same innervation and act in coordination, at different levels. When mechanical distension is severe or inflammation develops, the dysfunction may become painful. The pain is vague, persistent, and sometimes dominant and may become chronic. All possible underlying causes for pain ought to be diagnosed and treated. Pain can occur in some patients due to prolonged mechanical dysfunction or in association with local injury. The assessment and management of the overactive pelvic floor is an essential component of GI

practice, and GI physicians must increase their awareness of their role in the multidisciplinary approach to the overactive pelvic floor.

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Subjective Assessment of the Overactive Pelvic Floor

10

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10.1 Introduction

An emerging population of female patients is experiencing pelvic hyperactivity that includes voiding symptoms in conjunction with debilitating pelvic pain that can affect gastrointestinal or sexual function. Such patients, deemed as having an "overactive pelvic floor," are currently undergoing comprehensive investigations due to the uncertain etiology of pelvic floor hyperactivity, its alarming prevalence and significant negative impact on quality of life. Pelvic floor overactivity may be associated with neurological or musculoskeletal impairment as well as psychological distress, calling on contributions from medical professionals not only within the field of gynecology. The symptom complex under the "umbrella" of pelvic floor hyperactivity includes overactive bladder syndrome (OABS), chronic pelvic pain (CPP), sexual dysfunction, and associated gastrointestinal disorders. This chapter explores the subjective tools established to assess patients with overactive pelvic floor disorders and aims to characterize the multiple approaches that have been established today.

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10.2 Overactive Bladder Syndrome

OABS is defined by the International Continence Society (ICS) as urinary urgency, usually accompanied by frequency and nocturia, with or without urgency urinary incontinence, in the absence of urinary tract infection or other obvious pathology [1]. This updated definition of OABS is centered around urinary urgency, defined as the complaint of a sudden, compelling desire to pass urine, which is difficult to defer, and must be distinguished from the "normal urge to void" that occurs with normal bladder filling [1–3]. Also, urinary urgency incontinence is no longer essential to the diagnosis of OABS as data from a US study [4] in 2003 indicated that up to 60 % of those with OABS did not actually have urgency incontinence. Reported prevalence rates of OABS in men and women in North America and Europe range between 12 and 17 % [4–6]. Additionally, prevalence of OABS in women in

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North America increases to between 33 and 43 % over the age of 40 years [7]. Not only is the prevalence significant, but the reported health-related quality of life (HRQoL) in these patients is significantly impaired. Symptoms can negatively impact self-esteem, emotional well-being, sexual relationships, and productivity at work [8].

The trio of urinary bladder storage symptoms, which include urinary urgency, urinary frequency, and nocturia, along with urinary urgency incontinence, are subjective findings that can be difficult to measure and rely solely on patients' complaints [2, 3, 9–13]. Thus, OABS is a clinical diagnosis and requires a thorough clinical assessment [2, 3, 10, 12]. Perhaps the most challenging task for physicians is ensuring patients understand the definitions of such urinary symptoms and whether they can accurately recall to what extent they are experiencing them [14]. After working to achieve this goal, physicians can rely on evidence-based instruments that have undergone rigorous validation studies, such as qualitative scales and questionnaires, in efforts to subjectively assess their patients and match the most appropriate and effective treatment method [3, 10, 12–14]. However, physicians and researchers are also continuing to rely on objective measures, such as bladder diaries and urodynamics, to provide insight into pathophysiology, facilitate diagnoses, and evaluate efficacies of treatment measures [3, 12]. Therefore, it is imperative to distinguish between both subjective and objective outcomes as objective assessments may provide results that do not necessarily correlate with or predict such highly individualized subjective outcomes and vice versa.

10.2.1 Evaluating Overactive Bladder Syndrome

Due to its alarming prevalence and impact on quality of life, there continues to be an effort to identify patients with symptoms related to OABS [13]. Studies across Europe and the United States have shown that up to 60 % of patients with bladder symptoms never consulted their physician, an unfortunate statistic that may be due to the stigma associated with bladder problems [13]. Furthermore, a recent study [15] showed that while patients may ultimately consult their physician, they wait a number of years before doing so. This, coupled with its growing prevalence, provides enough evidence to support screening women for symptoms related to OABS. Additionally, screening can help trigger effective communication between patients and health care providers as increased patient education could help eliminate embarrassment and uncertainty about treatment availability associated with bladder problems [13].

One of the first screening tools for lower urinary tract dysfunction, assessing the presence of the four common storage symptoms, was developed and validated in Europe in 2006 [14]. The Bladder Control Self-Assessment Questionnaire's (B-SAQ) first validation study [14] investigated only female subjects from gynecology and urogynecology clinics and was designed to help raise awareness of bladder problems within society and probe patients to seek earlier intervention (see Fig. 10.1). The responses to each symptom and bother item are graded on a 4-point Likert scale: 0 (not at all), 1 (a little), 2 (moderately), and 3 (a great deal). Scores are then added and given an overall score up to 12, which grades severity to either none (0), mild (1–3), moderate (4–6), severe (7–9), or very severe (10–12). Achieving a score greater than 4 on the symptom scale is an indication that seeking medical help may be beneficial to the patient. Assessment of its ease of use, criterion and discriminant validity, and test–retest reliability produced favorable results [14]. Women found the questionnaire to be concise and easy to interpret; there was high internal consistency amongst the questionnaire items, and test–retest analysis showed that the majority of women had the same symptom and bother category assignments [14]. Taken together, these findings support that B-SAQ as a psychometrically robust instrument with good reliability and validity [14]. The sensitivity and specificity were 98 % and 79 %, respectively [14]. Follow-up investigation with male patients demonstrated similar effectiveness, with the exception of a lower specificity (46 %) [16]. Both studies require further assessment in primary care settings.

BLADDER CONTROL SELF-ASSESSMENT QUESTIONNAIRE

ARE YOU: MALE ☐ FEMALE ☐

Please put the NUMBER that applies to you in the boxes shown by the arrows based on the following:

NOT AT ALL = 0 A LITTLE = 1 MODERATELY = 2 A GREAT DEAL = 3

SYMPTOMS **BOTHER**

☐ ← Is it difficult to hold urine when you get the urge to go?

\+ How much does it bother you? → ☐

☐ ← Do you have a problem with going to the toilet too often during the day? +

\+ How much does it bother you? → ☐

☐ ← Do you have to wake from sleep at night to pass urine? +

\+ How much does it bother you? → ☐

☐ ← Do you leak urine? +

= How much does it bother you? → ☐

=

☐ NOW ADD THE TWO COLUMNS DOWNWARDS AND PUT THE SCORES IN THESE BOXES ☐

My symptom score My 'bother' score

THIS SYMPTOM SCORE MEANS:

SYMPTOM SCORE	THIS SYMPTOM SCORE MEANS:	THIS 'BOTHER' SCORE MEANS:	'BOTHER' SCORE
0	You are fortunate and don't have a urinary problem	You aren't bothered by a urinary problem	0
1-3	Your symptoms are mild	You are bothered slightly by your symptoms	1-3
4-6	You have moderate symptoms	You are moderately bothered by your symptoms	4-6
7-9	You have significant symptoms	Your symptoms are of significant bother for you	7-9
10-12	You have very significant problems	Your symptoms are a major problem for you	10-12

if your symptom score (above) is 4 or over you should seek help.

if your bother score (above) is 1 or over you may benefit by seeking help.

IMPORTANT - if you have blood in your urine, have difficulty passing urine, or pain on passing urine. you MUST talk to your doctor about it.

Fig. 10.1 The Bladder Control Self-Assessment Questionnaire (B-SAQ)

Recently, a group of investigators [13] in the United States set their sights on validating a new screening tool aimed at identifying individuals experiencing overactive bladder symptoms in the female population that incorporates current best practices and up-to-date regulatory standards. Originally developed for screening use in patients with multiple sclerosis experiencing urinary problems [17], the Actionable Bladder Symptom Screening Tool (ABSST) was assessed via a prospective, observational study that involved 100 female patients experiencing lower urinary tract symptoms recruited from various gynecology clinics [13]. Each subject completed the eight-item

ABSST that includes questions relating to urgency, micturition frequency, leakage, nighttime voiding, impact on social relations, work interference, and embarrassment over a 7-day recall period [13]. Grading for each item is based on a 4-point Likert scale, similar to the B-SAQ. The questionnaire also includes a question on whether the subject would like to receive help for their bladder problems. Scores greater than or equal to 3 (range 0–8) were indicative of need for further evaluation and/or treatment. Results of the study showed that the ABSST is a reliable, valid, and sensitive tool, which demonstrated an internal consistency coefficient range between 0.88 and 0.91 [13]. The questionnaire was easy to understand and respond to. Analysis of the correlation between ABSST scores and severities of symptoms amongst patients was significantly different, indicating that the ABSST appropriately reflects the severity of symptoms relating to OABS [13]. Sensitivity and specificity were 79 % and 98 %, respectively, which is consistent with the sensitivity and specificity findings in the multiple sclerosis population and supports use of the cut-off score [13]. Additional studies are underway in validating the use of the ABSST in wider population pools [13].

10.2.2 Evaluating Urgency and Its Severity

With the most recently established definition of OABS in 2011, urgency is now regarded as the most pivotal symptom of OABS and, therefore, is essential to evaluate and often the focus of further physician investigations [2, 3, 10, 12]. Possible etiologies for urgency include spontaneous smooth muscle cell contractions, structural changes in the bladder wall, altered release of neurotransmitters acting on smooth muscle or nerves, and altered central nervous system communication with the bladder [18]. Like all symptoms that may be present in OABS, however, urgency poses a challenge for physicians due to its subjective nature and the difficulties associated with ensuring that patients understand what it really means [2, 3, 9–12]. While the exact mechanisms of how urgency is perceived remain unclear, it is critical to differentiate between pathological "urgency" and the physiological "desire to void" that occurs during normal bladder filling [2, 3]. As the bladder fills with volume, an appropriate physiological response (urge) takes place as individuals without symptoms are able to tolerate increases of intensity in their desire to void and defer voiding up to a certain point [3]. At maximal bladder volume, and thus maximal intensity to void, voiding will take place and the regular cycle continues [3]. However, once a sudden, compelling desire to void occurs, which is difficult to defer, patients are experiencing urgency, begin to urinate more frequently (with smaller volumes and at nighttime) and may do so involuntarily [3]. Thus, it is essential to evaluate urgency appropriately so that effective treatment methods can relieve patients of these bothersome symptoms.

The first two subjective tools developed were the Indevis Urgency Severity Scale (IUSS) and the Urgency Perception Scale [10]. Based on the perception that urgency can be perceived differently amongst patients, efforts were made via the IUSS to help distinguish how severe the urgency was on a 4-point qualitative scale. During a clinical trial performed in 2003 [19], patients were asked to rate the severity of their urgency before voiding on a scale from 0 to 4 where 0 represented no urgency, 1 represented mild severity with awareness of urgency but easily tolerated and interruption of daily activities, 2 represented moderate severity with enough urgency/discomfort to interfere with daily activities, and 3 represented the most severe with extreme urgency discomfort that stops the ability to perform all activities [19]. Inclusion criteria for the trial included patients who voided greater than ten times per day and had greater than one urgency incontinence episode per day [19]. The IUSS was later validated in a 12-week randomized controlled clinical trial of trospium chloride in 658 patients with overactive bladder symptoms [20].

The Urgency Perception Scale, which must not be confused with the Urgency Perception Score, represents a subjective assessment of a patient's perception of urgency (with or without incontinence) using a 3-point scale [21]. Patients are asked to describe what they feel when they

experience the desire to pass urine [10]. The three responses can be described either as (1) where the patient reports they are usually not able to hold urine (urgency incontinence), (2) where the patient reports they usually able to hold their urine until they reach the toilet if they go immediately (urgency), and (3) where the patient reports they are usually able to finish what they are doing before going to the toilet (first desire to void) [10]. Although not validated in patients with urinary symptoms, construct validity of the Urgency Perception Scale was established by correlating scores with clinical and patient assessment data from three different clinical trials assessing efficacy of tolterodine in patients with overactive bladder symptoms [21–23].

More recently, other subjective tools have been developed. The Urgency Perception Score is a single-item questionnaire developed to grade urgency based on determining why individual patients choose to void as opposed to use as an index of severity and frequency of urgency episodes [10]. Patents are asked "What is the reason that you usually urinate?" and each response represents a 5-point grading scale that includes 0, which represents voiding out of convenience (no urgency), 1, which represents voiding with delay of an hour (mild urgency), 2, which represents voiding with delay of 10–60 min (moderate urgency), 3, which represents voiding with delay no longer than 10 min (severe urgency), and 4, which represents voiding because of desperate urgency (must stop and go void immediately) [10]. The UPS has been validated in asymptomatic volunteers, patients with lower urinary tract symptoms and patients with OABS through clinical trials evaluating the efficacy of tolterodine extended-release capsules and tolterodine with tamsulosin [24]. With proven test–retest reliability, the UPS represents a clinically useful measure of grading urgency [10].

Perhaps the most used and validated subjective tool for assessing urgency in drug development programs has been the Patient Perception of Intensity of Urgency Scale (PPIUS). The PPIUS asks patients to rate the level of urinary urgency for each void using a 5-point scale [10]. With each void recorded, patients rate the degree of associated urgency ranging from 0, described as no urgency—no feeling of need to empty bladder, but did for other reasons, to 3, described as severe urgency—could not postpone voiding, but had to rush to the toilet in order to avoid wetting oneself, or 4, described as urge incontinence—leakage before arriving to toilet [10]. The content validity and test–retest reliability of PPIUS has been tested in both non-interventional [25] and interventional [26–28] studies including healthy volunteers and patients with urinary symptoms/overactive bladder. Clinical trials assessing the efficacy of solifenacin, mirabegron, and the oxybutynin patch have used the PPIUS and indicated it shows good test–retest reliability and responsiveness [26–28]. Also it was demonstrated to have good value in assessing improvements in major OAB symptoms through correlated changes in PPIUS scores related to patients' perception of bladder condition [26–28]. A recent group from the United Kingdom has worked on incorporating the PPIUS with frequency in efforts to assess two of the major storage symptoms as a single measure [29]. This combination of a subjective urgency assessment with the objective count of urinary voids, termed a Total Urgency and Frequency Score (TUFS), has been tested and validated in patients with OAB [10]. Patients report urgency intensity using the PPIUS with every void and record the number of voids per day in their urinary diary [10]. The PPIUS scores are added to every void and then divided by the total number of days recorded in their diary [10]. While relying on patients to complete their diaries accurately, TUFS has produced favorable results in ongoing clinical trials. Through use in the BLOSSOM [27], SUNRISE [29], SATURN [30], and NEPTUNE [31] trials, TUFS has been shown to have good psychometric properties with high responsiveness and is a useful tool for assessing improvements in major OAB symptoms [10].

Lastly, a unique questionnaire has been developed that couples the assessment of severity of urgency with its impact on quality of life, regardless of the patient's continence status [2]. The Urgency Severity and Life Impact Questionnaire (USIQ) is a 13-question instrument that is divided into a 5-question part examining symptom severity and another 8 question part evaluating the impact of urgency of quality of life (see Fig. 10.2).

URGENCY SEVERITY AND IMPACT QUESTIONNAIRE (USIQ)

I. Urgency is a sudden compelling desire to void, which is difficult to defer due to fear of leakage. Please answer the following questions about your experience of urinary urgency.

During the last month, have you experienced any urinary urgency?

Yes ☐ No ☐

IF YOU ANSWERED NO, PLEASE STOP HERE

The following questions are only about your experience of urinary urgency, NOT about other urinary symptoms.

1. During the last month, what proportion of your urinations had urgency associated with them?

☐ None or almost none of the urinations
☐ About half of the urinations
☐ All or almost all of the urinations
☐ Some of the urinations
☐ Most of the urinations
☐ Don't know

2. When you have urgency, is it typically

☐ Extremely Mild
☐ Moderate
☐ Extremely Severe
☐ Mild
☐ Severe
☐ Don't know

3. Check the best answer for how long can you wait to urinate once you have urgency.

☐ Half an hour or more
☐ Less than 5 minutes but more than 1 minute
☐ I cannot wait at all
☐ Less than half an hour but more than 5 minutes
☐ Less than 30 seconds
☐ Don't know

Fig. 10.2 (**a**–**c**) The Urgency Severity and Impact Questionnaire (USIQ)

4. During the last month, how much has urinary urgency bothered you?

☐ None ☐ Moderate ☐ Extensive

☐ Slight ☐ Considerable ☐ Don't know

5. In an average 24 hour period how much of the time is urinary urgency present?

☐ Never ☐ Sometimes ☐ Always

☐ Rarely ☐ Usually ☐ Don't know

II. The following statements are about the impact of urinary urgency on your life.

		Not at all	Somewhat	Moderately	Quite abit	Always
1	**How much does urinary urgency affect your:** Ability to do household chores, do your work or schoolwork?					
2	**How much does urinary urgency affect your:** Ability to do physical activities such as walking, swimming or other exercise?					
3	**How much does urinary urgency affect your:** Ability to have an intimate relationship or sexual intercourse?					
4	**How much does urinary urgency affect your:** Entertainment activities such as going to a movie or concert?					
5	**How much does urinary urgency affect your:** Ability to travel by car or bus for a distance greater than 30 minutes away from home?					
6	**How much does urinary urgency affect your:** Participating in social activities outside your home?					

Fig. 10.2 (continued)

7	**How much does urinary urgency affect your:** Emotional Health (nervousness, depression, etc)?					
8	**How much does urinary urgency affect your:** Feeling Frustrated?					

Fig. 10.2 (continued)

For both assessments, scores range from 0 to 100 where higher scores correlate with more severe urgency symptoms and a greater impact of such symptoms on quality of life [2]. Validation studies have shown that the USIQ has excellent internal consistency as well as good construct, face, and discriminatory validity [2]. Also, after testing the questionnaire in a controlled trial of symptomatic patients receiving treatment with tolterodine, it was found that the USIQ had excellent test–retest reliability and demonstrated responsiveness following OAB treatment [2].

10.2.3 Evaluating Quality of Life

The effect of OABS on quality of life is significant. Over the past 20+ years, there have been several questionnaires developed and targeted towards assessing the impact of disease on quality of life. Many are used across a wide variety of disease spectrums while others are tailored specifically to urinary tract symptoms. This assessment is essential for physicians to help identify patients in need of immediate therapy, improve their symptoms with various treatment methods, and follow how successful their course of therapy is.

The Urogenital Distress Inventory (UDI), one of the first significant questionnaires developed in the United States in the early 1990s, assesses the amount of distress associated with incontinence and other urinary symptoms [32]. The subjective tool asks about 19 urinary symptoms and patients rate the degree to which these symptoms are troubling to them [32]. The UDI is highly recommended and has been shown to have high validity, reliability, and responsiveness in various populations of women with bladder symptoms before and after treatment [32–34]. A shorter form, UDI-6, has also been developed and has shown equal efficacy in trials [35, 36]. Continued improvements to the UDI are ongoing, and it is now being tested in the male population.

The King's Health Questionnaire (KHQ), available in 26 languages, is another tool that was first developed in London and consists of three major parts [37]. The first part tests the patient's general health and health related to urinary symptoms [37]. The second part includes 19 questions divided into seven domains of quality of life: incontinence impact, role limitations, physical limitations, social limitations, personal relationships, emotions, sleep and energy, severity of coping measures, and symptom severity [37]. The third part includes 11 questions assessing the impact/severity of such symptoms [37]. Similar to the UDI, the KHQ is highly recommended and has been shown to have excellent reliability and validity for women [38–40]. The KHQ also has proven reliability and validity for use in assessing lower urinary tract symptoms in men.

A questionnaire targeted towards the psychosocial impact of urinary symptoms in women was developed and named the Incontinence Impact Questionnaire (IIQ) [32, 41]. The tool consists of 30 questions, 24 related to the degree to which the symptoms affect regular activities and 6 related to feelings caused by them. Scores

Interstitial Cystitis Symptoms Index (ICSI)

During the past month:

How often have you felt the strong need to urinate with little or no warning:

0.__ Not at all
1.__ Less than 1 time in 5
2.__ Less than half the time
3.__ About half the time
4.__ More than half the time
5.__Almost always

Have you had to urinate less than 2 hours after you finished urinating?

0.__ Not at all
1.__ Less than 1 time in 5
2.__ Less than half the time
3.__ About half the time
4.__ More than half the time
5. __Almost always

How often did you most typically get up at night to urinate?

0.__ Not at all
1.__ Once per night
2.__ 2 times per night
3.__ 3 times per night
4.__ 4 times per night
5.__ 5 or more times per night

Have you experienced pain or burning in your bladder?

0.__ Not at all
1.__ A few times
2.__ Fairly often
3.__ Usually
4.__ Almost always

Add the numerical values of the checked entries:

Total score _____

Interstitial Cystitis Problem Index (ICPI)

During the past month:

How much has each of the following been a problem for you.

Frequent urination during the day?

0.__ No problem
1.__ Very small problem
2.__ Small problem
3.__ Medium problem
4.__ Big problem

Getting up at night to urinate?

0.__ No problem
1.__ Very small problem
2.__ Small problem
3.__ Medium problem
4.__ Big problem

Need to urinate with little warning?

0.__ No problem
1.__ Very small problem
2.__ Small problem
3.__ Medium problem
4.__ Big problem

Burning, pain, discomfort, or pressure in your bladder?

0.__ No problem
1.__ Very small problem
2.__Small problem
3.__ Medium problem
4.__ Big problem

Add the numerical values of the checked entries:

Total score _____

Fig. 10.3 The Interstitial Cystitis Symptoms Index (ICSI) and Interstitial Cystitis Problem Index (ICPI)

are added and divided into clusters pertaining to effect on physical activity, travel, social relationships, and emotional health [32]. The IIQ has been tested in several studies including use in incontinent women treated with oxybutynin, tolterodine, or behavioral interventions and has been shown to have good levels of reliability and validity [42, 43].

10.3 Evaluating Pain and Sexual Dysfunction

CPP is defined by the American College of Obstetricians and Gynecologists (ACOG) as localized, noncyclic, pain that persists for 6 months or more and causes a loss of function [44]. CPP affects up to 24 % of women who are

of reproductive age and often requires pharmacologic or surgical intervention, which may not ultimately treat the patient's complaints as pain recurrence is likely [45–47]. The etiology of CPP is unclear as it is thought to result from a complex interplay between gynecologic, urinary, gastrointestinal, neurological, musculoskeletal, and psychological systems [45]. What is certain, however, is the negative effect of CPP on quality of life. Patients with CPP suffer tremendously and have associated stress that affects their marital, social, professional, and sexual lives [45, 46, 48]. Thus, improvement of quality of life is the primary goal in treating patients with CPP [48]. With the aid of subjective instruments such as questionnaires, physicians can appropriately assess the impact of CPP in patients and manage outcomes of their therapeutic interventions. The most recent systematic review of quality of life instruments used in studies of CPP identified a need for the development and evaluation of more specific instruments to assess pelvic pain [48]. Only 19 eligible articles studying use of questionnaires were identified from the 187 articles retrieved after a thorough electronic database search. Of those identified, three of the reports had been studying disease-specific instruments, which were not patient generated and instead developed based on reports from other health professionals [48]. It was determined that, in general, the quality of life instruments reviewed have poor clinical face validity [48]. With regard to the disease-specific questionnaires, compliance with matters of importance to patients varied and only one demonstrated reasonable compliance with quality criteria [48, 49].

Painful bladder syndrome (PBS)/interstitial cystitis (IC) was initially defined by the International Society of Bladder Pain Syndrome in 2005 as "the complaint of suprapubic pain related to bladder filling, accompanied by other symptoms, such as increased daytime and nighttime frequency, in the absence of proven urinary infection or other obvious pathology" [50]. Currently, the European Society for the Study of Bladder Pain Syndrome favors the use of the term "Bladder Pain Syndrome" instead of PBS or IC [51]. This syndrome may be included as a form of CPP in patients and differs from OABS such that pain is the predominant symptom and is occurring in association with bladder symptoms. Parsons and colleagues [52] surveyed several studies in 2007 and reported that BPS has a prevalence of 197 for every 100,000 women over the last 10 years, which was deemed a substantial underestimation of its true prevalence, and affects women more often than men. However, the specific etiology is still uncertain as there is still no absolute definition on how to classify BPS. Genetics, prior pelvic surgery, glycosaminoglycan layer defects, nitrogen oxide metabolism, and autoimmunity have all been linked as possible causes [50].

Various subjective tools have been used and evaluated in the assessment of patients complaining of symptoms of BPS. In efforts to address the initial need for developing broad symptom indexes specifically for BPS, O'Leary and colleagues [53] developed the Interstitial Cystitis Symptoms Index (ICSI) and Interstitial Cystitis Problem Index (ICPI) (see Fig. 10.2). Based off of a 10-year clinical experience with over 400 patients at a New England IC clinic and after statistical validation, these tools were believed to be beneficial in IC management and facilitate clinical research [53, 54]. Both indexes contain questions that address each of the symptoms of frequency, urgency, nocturia, and bladder pain. The two indexes differ, however, in that the ICSI assesses the level of severity of symptoms directly while the ICPI assesses the degree of problem caused by each symptom [53, 54]. Questions such as "have you experienced pain or burning in your bladder" versus how much has "burning, pain, discomfort or pressure in your bladder" been a problem for you, are answered based on a scale of 0–4 where 0 represents either "not at all" or "no problem at all" and 4 represents "almost always" or "big problem," respectively [53, 54]. Both the ICSI and ICPI have been shown to demonstrate excellent ability to differentiate characteristics between patients and controls [54]. The ICSI has also been shown to be responsive to changes in patient condition after use in a clinical trial with pentosan polysulfate sodium [55].

While condition-specific tools such as the ICSI or ICPI exist, few address additional associated symptoms such as dyspareunia or pelvic pain other than bladder pain [56]. In 2009, a single instrument was developed and reported by Clemens and colleagues [56] in Michigan that assessed genitourinary pain symptoms in women using symptom-based criteria. Referred to as the Genitourinary Pain Index (GPI), the 9-question tool initially asks patients to report if they experience pain or discomfort in pelvic areas or in association with bladder activities and sexual intercourse [56]. The GPI also asks patient to quantify how often such symptoms occur (including a scale from never to always) and also challenges patients to assess the average pain on a scale from 1 to 10, 10 being as bad a pain as imaginable [56]. Lastly, the GPI assesses the impact of symptoms on quality of life: how much do patients think about their symptoms, whether it interferes with their daily routine and how patients would feel if they experienced symptoms for the rest of their life [56]. A total score of 0–45 is determined based on each patient's scores from the pain, urinary, and quality of life questions. After a thorough evaluation, the GPI was determined to be a valid and reliable instrument that can be used to assess symptom severity and impact in women, demonstrating excellent internal consistency and responsiveness to change [56]. Similar positive results were found with a gender-specific GPI used when assessing pain and symptoms in males [56].

In addition to CPP and lower urinary tract symptoms, women with hyperactive pelvic floors may experience sexual dysfunction. It has been reported that 43 % of women complain of at least one sexual problem [57]. In accordance with the growing prevalence of sexual dysfunction in women, new instruments for assessing patients' complaints are being devised. Sexual dysfunction in women is complicated, involving both psychological and organic processes, and may draw attention from health care providers outside the realm of female pelvic medicine [57]. In efforts to improve the diagnostic framework for assessing and treating female sexual dysfunction, Rosen and colleagues [57] developed the Female Sexual Function Index (FSFI) questionnaire (see Fig. 10.4). The FSFI is a 19-item self-report questionnaire split between the six domains of desire, arousal, lubrication, orgasm, satisfaction, and pain [57]. Each item has a 5-point response scale (1–5) that correlates with variations in frequency, intensity or degree of satisfaction. For 15 of the 19 items, there exists a zero category that codes for either "no sexual activity" in 12 of them or "did not attempt intercourse" in three. The "satisfaction" domain pertains to global sexual and relationship satisfaction and can be viewed as the "quality of life" domain of the scale [57]. Questions assess patients' satisfaction with amount of closeness with partner, sexual relationship, and overall sex life. The "pain" domain is also a crucial one for investigation in women with coinciding CPP or BPS and assesses patients' pain frequency during and following vaginal penetration as well as pain level during or following vaginal penetration [57]. The FSFI has undergone studies assessing its validity, reliability, and replicability in other languages [58]. Overall, results have shown that the questionnaire has excellent reliability and discriminant validity while also being clear, concise, and easy for patients to answer [57–59]. The FSFI has rapidly found acceptance as a screening tool with use in diverse medical conditions and treatments such as bladder reconstruction, spinal cord injuries, vaginoplasty, vulvodynia, and in correlation with hormonal variations [58].

10.4 Screening for Anxiety, Depression, and Posttraumatic Stress Disorder

There are numerous, validated screening and assessment measures for depression, anxiety, and posttraumatic stress disorder (PTSD), some of which have been specifically developed for use in primary care or general medical (i.e., non-mental health) clinics. The majority of widely used and well-established measures were developed and validated using diagnostic criteria established in the fourth edition of the Diagnostic

Female Sexual Function Index (FSFI) ©

Subject Identifier _____ Date _____

INSTRUCTIONS: These questions ask about your sexual feelings and responses during the past 4 weeks. Please answer the following questions as honestly and clearly as possible. Your responses will be kept completely confidential. In answering these questions the following definitions apply:

Sexual activity can include caressing, foreplay, masturbation and vaginal intercourse.

Sexual intercourse is defined as penile penetration (entry) of the vagina.

Sexual stimulation includes situations like foreplay with a partner, self-stimulation (masturbation), or sexual fantasy.

CHECK ONLY ONE BOX PER QUESTION.

Sexual desire or interest is a feeling that includes wanting to have a sexual experience, feeling receptive to a partner's sexual initiation, and thinking or fantasizing about having sex.

1. Over the past 4 weeks, how **often** did you feel sexual desire or interest?

   ☐ Almost always or always
   ☐ Most times (more than half the time)
   ☐ Sometimes (about half the time)
   ☐ A few times (less than half the time)
   ☐ Almost never or never

2. Over the past 4 weeks, how would you rate your **level** (degree) of sexual desire or interest?

   ☐ Very high
   ☐ High
   ☐ Moderate
   ☐ Low
   ☐ Very low or none at all

Fig. 10.4 (**a**–**e**) The Female Sexual Function Index (FSFI)

Sexual arousal is a feeling that includes both physical and mental aspects of sexual excitement. It may include feelings of warmth or tingling in the genitals, lubrication (wetness), or muscle contractions.

3. Over the past 4 weeks, how **often** did you feel sexually aroused ("turned on") during sexual activity or intercourse?

- ☐ No sexual activity
- ☐ Almost always or always
- ☐ Most times (more than half the time)
- ☐ Sometimes (about half the time)
- ☐ A few times (less than half the time)
- ☐ Almost never or never

4. Over the past 4 weeks, how would you rate your **level** of sexual arousal ("turn on") during sexual activity or intercourse?

- ☐ No sexual activity
- ☐ Very high
- ☐ High
- ☐ Moderate
- ☐ Low
- ☐ Very low or none at all

5. Over the past 4 weeks, how **confident** were you about becoming sexually aroused during sexual activity or intercourse?

- ☐ No sexual activity
- ☐ Very high confidence
- ☐ High confidence
- ☐ Moderate confidence
- ☐ Low confidence
- ☐ Very low or no confidence

6. Over the past 4 weeks, how **often** have you been satisfied with your arousal (excitement) during sexual activity or intercourse?

- ☐ No sexual activity
- ☐ Almost always or always
- ☐ Most times (more than half the time)
- ☐ Sometimes (about half the time)
- ☐ A few times (less than half the time)
- ☐ Almost never or never

Fig. 10.4 (continued)

7. Over the past 4 weeks, how **often** did you become lubricated ("wet") during sexual activity or intercourse?

- ☐ No sexual activity
- ☐ Almost always or always
- ☐ Most times (more than half the time)
- ☐ Sometimes (about half the time)
- ☐ A few times (less than half the time)
- ☐ Almost never or never

8. Over the past 4 weeks, how **difficult** was it to become lubricated ("wet") during sexual activity or intercourse?

- ☐ No sexual activity
- ☐ Extremely difficult or impossible
- ☐ Very difficult
- ☐ Difficult
- ☐ Slightly difficult
- ☐ Not difficult

9. Over the past 4 weeks, how often did you **maintain** your lubrication ("wetness") until completion of sexual activity or intercourse?

- ☐ No sexual activity
- ☐ Almost always or always
- ☐ Most times (more than half the time)
- ☐ Sometimes (about half the time)
- ☐ A few times (less than half the time)
- ☐ Almost never or never

10. Over the past 4 weeks, how **difficult** was it to maintain your lubrication ("wetness") until completion of sexual activity or intercourse?

- ☐ No sexual activity
- ☐ Extremely difficult or impossible
- ☐ Very difficult
- ☐ Difficult
- ☐ Slightly difficult
- ☐ Not difficult

Fig. 10.4 (continued)

11. Over the past 4 weeks, when you had sexual stimulation or intercourse, how **often** did you reach orgasm (climax)?

- ☐ No sexual activity
- ☐ Almost always or always
- ☐ Most times (more than half the time)
- ☐ Sometimes (about half the time)
- ☐ A few times (less than half the time)
- ☐ Almost never or never

12. Over the past 4 weeks, when you had sexual stimulation or intercourse, how **difficult** was it for you to reach orgasm (climax)?

- ☐ No sexual activity
- ☐ Extremely difficult or impossible
- ☐ Very difficult
- ☐ Difficult
- ☐ Slightly difficult
- ☐ Not difficult

13. Over the past 4 weeks, how **satisfied** were you with your ability to reach orgasm (climax) during sexual activity or intercourse?

- ☐ No sexual activity
- ☐ Very satisfied
- ☐ Moderately satisfied
- ☐ About equally satisfied and dissatisfied
- ☐ Moderately dissatisfied
- ☐ Very dissatisfied

14. Over the past 4 weeks, how **satisfied** have you been with the amount of emotional closeness during sexual activity between you and your partner?

- ☐ No sexual activity
- ☐ Very satisfied
- ☐ Moderately satisfied
- ☐ About equally satisfied and dissatisfied
- ☐ Moderately dissatisfied
- ☐ Very dissatisfied

Fig. 10.4 (continued)

15. Over the past 4 weeks, how **satisfied** have you been with your sexual relationship with your partner?

- ☐ Very satisfied
- ☐ Moderately satisfied
- ☐ About equally satisfied and dissatisfied
- ☐ Moderately dissatisfied
- ☐ Very dissatisfied

16. Over the past 4 weeks, how **satisfied** have you been with your overall sexual life?

- ☐ Very satisfied
- ☐ Moderately satisfied
- ☐ About equally satisfied and dissatisfied
- ☐ Moderately dissatisfied
- ☐ Very dissatisfied

17. Over the past 4 weeks, how **often** did you experience discomfort or pain during vaginal penetration?

- ☐ Did not attempt intercourse
- ☐ Almost always or always
- ☐ Most times (more than half the time)
- ☐ Sometimes (about half the time)
- ☐ A few times (less than half the time)
- ☐ Almost never or never

18. Over the past 4 weeks, how **often** did you experience discomfort or pain following vaginal penetration?

- ☐ Did not attempt intercourse
- ☐ Almost always or always
- ☐ Most times (more than half the time)
- ☐ Sometimes (about half the time)
- ☐ A few times (less than half the time)
- ☐ Almost never or never

19. Over the past 4 weeks, how would you rate your **level** (degree) of discomfort or pain during or following vaginal penetration?

- ☐ Did not attempt intercourse
- ☐ Very high
- ☐ High
- ☐ Moderate
- ☐ Low
- ☐ Very low or none at all

Thank you for completing this questionnaire

Fig. 10.4 (continued)

and Statistical Manual of Mental Disorders (DSM-IV), the standard classification of mental disorders developed and published by the American Psychiatric Association and used by mental health providers in the United States [60]. Note that the DSM was revised in 2013 [61], and new or revised measures consistent with new diagnoses and revised diagnostic criteria are likely to be developed.

10.4.1 Depression

The Patient Health Questionnaire-9 (PHQ-9) [62] is the depression module of the PRIME-MD Patient Health Questionnaire, a self-administered questionnaire specifically developed to diagnose DSM-IV mental disorders in a primary care setting [63]. The PHQ-9 asks patients to rate the frequency of each of the 9 DSM criteria for a Major Depressive Episode on a 4-point scale ranging from "0" (not at all) to "3" (nearly every day). These core criteria have not changed from DSM-IV to DSM 5. PHQ-9 scores can be used both for diagnosis and for determination of symptom severity. The PHQ-9 has demonstrated good reliability and validity with patients in primary care and obstetrics/gynecology clinics [62] and in the general population [64], and is strongly associated with functional impairment and quality of life.

10.4.2 Anxiety

A 2-page version of the PRIME-MD PHQ, described above, is also available. The Brief PRIME-MD PHQ can be used in varied ways: the first page includes questions assessing panic disorder in addition to depression; the second page includes questions about psychosocial stressors, one item about physical or sexual violence, and questions about menstruation, pregnancy, and childbirth [65]. This measure was validated in a sample of 3000 patients in 7 outpatient obstetrics/gynecology clinics. The GAD-7 is a brief self-report measure assessing the presence of Generalized Anxiety Disorder (GAD) [66]. This seven item scale has demonstrated good reliability and validity in both primary care and general population samples [66, 67].

10.4.3 Posttraumatic Stress Disorder

PTSD was reclassified in DSM 5 from an anxiety disorder into the new class of trauma and stressor-related disorders, all of which require exposure to a traumatic or highly stressful event. In addition, the diagnostic criteria were reorganized, with some new criteria added. The only currently available screening measure that incorporates these changes is the 20-item PTSD Checklist for DSM 5 (PCL-5), which can be used for provisional diagnosis and for assessment of symptom severity [68]. The PCL asks respondents to indicate "how much they have been bothered" by each of the DSM 5 specified symptoms of PTSD in the prior month, using a scale ranging from "0" (not at all) to "4" (extremely). The PCL-5 can be administered with brief instructions if trauma exposure has already been assessed or disclosed, with a brief assessment of any trauma exposure, or with the Life Events Checklist for DSM 5 (LEC-5), which asks the respondent to indicate any exposure to a list of potentially traumatic events, such as natural disaster, physical and sexual assault, and serious accidents.

10.5 Conclusion

Pelvic floor muscle hyperactivity, as discussed above, can be detrimental to patients and negatively affect many aspects of their quality of life. Thorough evaluations of bladder symptoms, pelvic pain, sexual dysfunction, gastrointestinal dysfunction, and mental health are crucial when evaluating these patients. In order to alleviate such related symptoms, it is important to be well acquainted with the several validated subjective tools with which one can evaluate the different aspects of symptoms that patients experience. Once the diagnosis is made, a multidisciplinary team is often needed to help and treat women suffering from pelvic floor muscle hyperactivity.

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Objective Assessment of the Overactive Pelvic Floor

11

Mélanie Morin

11.1 Introduction

Pelvic floor muscle (PFM) overactivity is recognized as playing a crucial role in several conditions such as bladder and bowel elimination disorders, genital/pelvic pain syndromes, and sexual dysfunctions and, thus, its objective assessment is key to a better understanding of the ongoing pathophysiological processes and critical for properly orienting treatment.

An adequate assessment calls for a thorough comprehension of the muscle physiology underlying muscle tone. As discussed in detail in Chap. 1, general muscle tone, sometimes referred as muscle tension, is a complex phenomenon that can be defined as the resistance provided by the muscle when pressure or a stretch is applied. In normally innervated skeletal muscle, muscle tone is composed of a passive and an active component [1]. The passive component consists of the viscoelastic properties of the muscle tissue related to several structures [2]: (1) the extensibility of actin-myosin cross-bridges (EMG silent); (2) noncontractile cytoskeleton proteins such as the desmin and the titin and (3) conjunctive tissues surrounding the entire muscle (epimysium), muscle fascicle (perimysium), and muscle fiber (endomysium). The active component, in turn, comprises physiological contracture (more commonly defined as trigger point (TP)), electrogenic spasms (which include unintentional muscle contraction with or without pain amenable to voluntary control), and normal electrogenic contraction (involves resting activity in normally relaxed muscle and also myotatic reflex during stretching). Only electrogenic spasms and normal electrogenic contractions involve electrical current propagating along muscle fibers that can be recorded by electromyography (EMG). It has been reported that electrogenic spasms may be related to psychological distress (e.g., anxiety), muscle overload or overuse (due for instance to inadequate posture) and inefficient uses (e.g., failure to fully relax after contraction) [1]. It should be pointed out that the presence of resting activity in normally relaxed muscle is controversial. While studies in skeletal muscle failed to find evidence of resting EMG [3, 4], the PFMs may be an exception as Deindl et al. [5] suggested that some part of the levator ani may present sustained motor unit firing at rest. The term "overactive" describing the pelvic floor may refer to an elevated "electrogenic" resting activity. Throughout this chapter, the objective assessment will be discussed in a wider context of heightened PFM tone as the available literature suggests that the pathophysiology is not limited

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to an electrogenic mechanism. A clear understanding of muscle tone physiology and its active/passive components is mandatory and must be related to the characteristics and limitations of the current PFM assessment tools. For instance, it is of the utmost importance to know which muscle tone component is assessed by each assessment method in order to better understand the role of the PFMs in the patient's symptomatology, presumably associated with an overactive pelvic floor.

In conditions due to overactive pelvic floor, several authors emphasize that elevated PFM tone might not be the only muscle dysfunction and stress the importance of a broader assessment of muscle contractile properties [6–11]. The assessment should therefore go beyond the properties of the muscles at rest to evaluate the contractile properties such as strength, endurance, and speed of contraction.

Several assessment tools and methodologies are available for assessing PFM tone and contractile properties, namely digital palpation, EMG, ultrasound imaging manometry and dynamometry. This chapter reviews these tools in light of muscle physiology (components of tone, for instance) and provides an overview of the psychometric properties (reliability and validity) of the instruments for measuring tone and other PFM contractile properties as well as discussing their limitations. The scientific empirical evidence available related to the implication of the PFMs in men and women with overactive pelvic floor will also be presented.

11.2 Vaginal Palpation

Digital palpation of the PFMs is widely used to evaluate the PFM tone and contractile properties such as strength, endurance, speed of contraction, and coordination [6–9, 12–14]. Although contested as an assessment method for research purposes because of its subjectivity and lack of precision, digital palpation is a fast and practical technique easy to implement in a clinical setting.

Palpation can be used for an overall subjective appraisal of the PFM tone. The examination may start with external palpation to assess the patient's anticipatory and protective reactions, appreciate PFM tone, and detect pain mainly at PFM insertion sites (pubic arch, ischial tuberosity, and coccyx). In cases where intracavity measurement is not possible, the clinician relies entirely on this assessment approach. Intravaginal palpation can be performed in a clockwise manner for detecting pain/tenderness, tension areas, muscle volume, asymmetry, reduced sensation, and scars. Palpation of the obturator internus muscle may also reveal tenderness and tensions. Transrectal palpation can provide a similar overview of the external anal sphincter and levator ani muscles in addition to assessing the coccyx position and mobility, anorectal angle (related to puborectalis tone), and rectal content. Although claimed essential for clinical practice, there is a paucity of evidence regarding the validity and reliability of this global subjective assessment of PFM tone.

11.2.1 Grading Scale for Assessing PFM Tone

Some grading scales have been developed in an attempt to quantify the PFM tone. *Devreese's tone grading scale*, developed in a context of assessing women with incontinence, comprises just three levels (hypotonic/normotonic/hypertonic) for intravaginal assessment [14]. Despite a good interrater reliability (agreement in 96–98 % of patients), the limited number of levels may be less relevant for assessing overactive PFMs. Also developed in women with incontinence and prolapse, *Dietz's tone grading scale* [15], ranging from 0 to 5, shows a moderate test–retest reliability (weighted kappa (K) = 0.55), see Table 11.1 for assessing the PFM tone. Using this scale in asymptomatic women, Loving et al. [9] obtained a higher test–retest reliability (K = 0.7–1.0) and a good interrater reliability (K = 0.7–0.8). The main limiting factor for this quotation is that the grading system incorporates three components, namely hiatus size, resistance to distension, and pain. A woman with pain will thus automatically obtain the highest score of 5.

Table 11.1 Dietz's tone grading scale [15]

Grade	Description
0	Muscle not palpable
1	Muscle palpable but very flaccid, wide hiatus, minimal resistance to distension
2	Hiatus wide but some resistance to distension
3	Hiatus fairly narrow, fair resistance to palpation but easily distended
4	Hiatus narrow, muscle can be distended but high resistance to distension, no pain
5	Hiatus very narrow, no distension possible, "woody" feel, possibly with pain: "vaginismus"

Table 11.2 Lamont's tone grading scale developed in women with vaginismus [16]

Grade	Description
0	Normal muscle tone
1	Perineal and levator spasm (released by reassurance)
2	Perineal spasm maintained throughout the pelvic exam
3	Levator spasm and buttocks elevation
4	Levator and perineal spasm, elevation: adduction of thighs and pelvic withdrawal

Lamont developed a scale (from 0 to 4) for assessing PFM tone in women with vaginismus [16]. Similarly, the scale also includes components other than PFM tone such as hip muscle contractions and withdrawal behaviors (Table 11.2). This scale has not been studied for its psychometric properties and the assessment of combined components may affect the validity of the test for evaluating PFM tone.

Ressing et al. [8] proposed a 7-level scale ranging from +3 very hypertonic to −3 very hypotonic (0 being normal tone) for assessing women with provoked vestibulodynia. Superficial layers of the PFM, including the ischiocavernosus, bulbocarvernosus and transverse superficial, can be assessed by intravaginal palpation at 3, 6, and 9 o'clock. The deeper PFM layers are evaluated at 3, 6, and 9 o'clock for the pubococcygeus sling and 5 and 7 o'clock for the iliococcygeus. The anal examination comprises an evaluation of the external anal sphincter on a scale of 1 (hypotonic), 0 (normal) and +1 (hypertonic) and the puborectalis/pupococcygeus on a scale from +3 to −3 as described in above, the right and left side being evaluated separately. This scale has been studied for its interrater reliability and, overall, a fair-to-moderate reliability was found with correlation coefficients of 0.230–0.514 [8]. Considering the available evidence about reliability and the specificity of the grades assessing only tone component, Reissing's scale is probably the most suitable in a context of assessing heightened PFM tone. With reference to muscle physiology, assessment of PFM tone using these scales evaluates the total contribution of all active and passive components of muscle tone. In other words, it cannot distinguish between specific sources of muscle tone.

11.2.2 Flexibility and Hiatus Diameters

Measurement of flexibility and hiatus diameters has been suggested as a means to assess the maximal PFM length. The distance between the left and right muscle bellies just below the pubic bone is estimated in centimeters by separating two fingers inserted in the vagina (transverse hiatus diameter) [17]. The distance between the back of the pubic symphysis and the midline raphe of the puborectalis is also measured (anteroposterior diameter). Boyles et al. [17] found a good-to-excellent interrater reliability with correlation coefficients of 0.6–0.8. Likewise, Gentilcore et al. [6] assessed the transverse diameter by grading from 0 (less than one finger insertion) to 4 (two-finger insertions with fingers abducted horizontally ≥2 cm). However, this scale was not studied for its validity and reliability. Maximal muscle length measurement is commonly performed in a general skeletal muscle assessment and is determined by the patient's tolerance and increases in EMG in order to ensure adequate muscle relaxation during stretching. Because such monitoring is obviously not part of a palpation assessment, these scales are considered as a global measurement of PFM tone without discriminating between active and passive components.

11.2.3 Relaxation Ability After a Maximal Contraction

The International Continence Society (ICS) stadardization group on PFM function and dysfunction recommended assessing the PFM relaxation ability after a contraction [18]. They suggested describing the relaxation as present/absent and partial/complete [18]. Loving et al. [9] studied the test–retest and interrater reliability of this scale in asymptomatic women and found a kappa of 1.0 and 0.60, respectively. Lower test–retest and interrater reliability was observed by Slieker-ten Hove et al. [13], however, with a weighted kappa of 0.76 and 0.17, respectively. In addition to these qualifiers, Reissing et al. [8] suggested documenting the speed of relaxation (slow/fast). Although not studied for its psychometric properties, a scale developed in women with multiple sclerosis was proposed by De Ridder et al. [19] for grading the ability to relax the pelvic floor: 3—active relaxation after active contraction, 2—hypertonic muscle with temporary relaxation after elongation, 1—spastic muscle, unable to relax even after passive elongation. Reissing et al. [8] proposed a scale to grade the relaxation from 0 to 5 (0 returns to resting state; 5 remains fully contracted). Fair interrater reliability was observed only when the assessment was performed using two fingers ($r = 0.355$–0.400) as opposed to one finger, which yielded unreliable data. Although not studied for reliability, a similar scale was proposed by Gentilcore et al. [6] evaluating the relaxation capacity on a 5-point scale from 0 (which indicated the PFMs were fully able to return to their resting state following a maximal contraction or 100 % relaxation) to 4 (which indicated that the PFMs remained fully contracted after a maximal contraction or 0 % relaxation). One limitation related to these scales is that the scoring refers to the resting state observed before contraction. This is particularly problematic in the case of elevated tone at baseline, whereas a patient would be categorized as having a good relaxation if she returns to the pathological precontraction level.

11.2.4 Myofascial Trigger Point Assessment

The myofascial trigger point (TP) is defined as an identifiable taut band or rope-like indurations palpated in the muscle fiber that can evoke pain both locally (local tenderness) and at distant reference-pain zones specific to each TP [20]. With regard to the muscle physiology [1], TP refers to physiological contracture, one element among the active components of muscle tone. It should be emphasized that TP refers to an endogenous shortening of a specific point in a muscular fiber which is not detectable by global EMG. TP assessment has been deemed important not only because it is associated with pain but it has also been reported that TP can provoke heightened PFM tone (related to electrogenic spasms) [21]. It has been shown that manual therapy for releasing TPs results in PFM relaxation as measured by the reduction in resting EMG activity (i.e., reduction of the electrogenic spasms) [21].

Kavvadias et al. [22] investigated the intensity of pain elicited during palpation of TPs in the anterior levator ani, posterior levator ani, obturator internus and piriformis on both sides using a visual analog scale. They obtained a heterogeneous test–retest and interrater reliability with ICCs of 0.20–0.87 and 0.28–0.87, respectively, in a sample of asymptomatic women. Palpation of the posterior levator ani showed a higher reliability than the anterior portion [22]. Montenegro et al. [23] studied the interrater reliability of the tenderness assessment of the bilateral levator ani, piriformis, and internal obturator muscles in controls and women with chronic pelvic pain. Tenderness was scored according to each subject's reactions for the six positions as follows: 0, no pain; 1, painful discomfort; 2, intense pain; with a maximum total score of 12. Very good interrater reliability was found with a kappa of 0.91. A systematic review about TP in limbs and trunk muscle pointed out that TP assessment should not be limited to tenderness but should notably include taut bands, patient pain recognition, and pain referral [24]. It should be noted

that TPs located in the levator ani have referral zones in the muscle and in both anterior and posterior compartments (rectum, anus, coccyx, urethra, bladder, penis/vulva, buttocks). The obturator internus and piriformis can, in turn, refer to the perineal zone. More information about referred pain patterns in the PFMs and surrounding muscles can be found elsewhere [20, 25, 26]. Hsieh et al. [27] pointed out that the evaluator's experience might influence the psychometrics of TP assessment. In sum, considering that psychometric studies so far have focused mainly on pain intensity with divergent findings, the assessment of TP using digital palpation warrants further investigation.

11.2.5 Contractile Properties

Current evidence in patients with heightened PFM tone converges toward the presence of other concomitant alterations in contractile properties. It is generally recognized that the PFMs are difficult to contract correctly. In fact, even among women without urogynecological problems, over 30 % are unable to adequately contract these muscles following verbal instructions and need further teaching such as vaginal palpation [28]. To facilitate PFM contraction, Messelink et al. [18] suggested instructing women to "squeeze and lift as if to prevent the escape of gas or urine." Crotty et al. [29] demonstrated that verbal cues that included both the anterior and posterior parts of the PFMs yielded stronger contractions: "Squeeze and lift from the front and back together." Palpation was also suggested to be useful in teaching women to perform adequate PFM contraction avoiding muscle compensation such as buttock, adductor, rectus abdominus contraction as well as perineal inversion (i.e., straining) [30, 31].

Several scales have been proposed to grade the PFM contractile properties including Brinks' scale [32], Devreese's scale [14] and Messelink's scale [13, 18] as well as the Laycock PERFECT assessment scheme, which incorporates the modified Oxford scale [12]. In general, these scales show an acceptable intra-observer and test–retest reliability [9, 13, 14, 32–36]. The modified Oxford scale seems to be the most frequently used in women with chronic pain conditions [6–9, 37]. The Laycock PERFECT assessment scheme, presented in Table 11.3, proposes an assessment of strength and endurance, the number of repetitions and fast contractions. It has been shown that this grading system can be used with different patient positions, lying or upright, with good reliability [34], although it was found that women prefer the supine position for internal examination [38]. Furthermore, the reliability of the modified Oxford scale has also been investigated when a "+" or "−" is added to the original quotation but it was found that the original 6-level quotation (without +/−) yielded better reliability. It was recommended by the ICS standardization group on PFM function and dysfunction to limit the number of levels in a strength-assessing scale to preserve an acceptable interrater reliability [18]. On the other hand, this limitation may interfere with the responsiveness to detect differences between individuals and changes following treatment [31].

Table 11.3 The Laycock PERFECT scheme [12]

Scale	Description
P	*P*ower
	0—Nil
	1—Flicker
	2—Weak
	3—Moderate
	4—Good
	5—Strong
E	*E*ndurance is expressed as the length of time, up to 10 s, that a maximal voluntary contraction can be sustained before the strength is reduced by 35 % or more
R	Number of *R*epetitions that a woman is able to achieve (same duration as in E)
F	Number of *F*ast (1-s) maximal contractions performed (up to 10)
E	*E*very
C	*C*ontraction
T	*T*imed (to complete the acronym and reminds the examiner to time and record the above sequence of events)

11.2.6 Overall Considerations

Digital palpation assessment of PFMs can be performed by inserting one or more fingers inside the vagina [6–8]. In chronic pain patients, the presence of pain associated with vaginal distention can bias PFM tone assessment by provoking protective-like muscular reactions and thus result in heightened PFM tone and incomplete relaxation [7, 39]. It is also possible that strength reduction is caused by pain inhibition. Despite the influence of pain on muscle assessment, it is already known from studies both of skeletal muscle and of PFMs that muscle lengthening (i.e., by inserting a finger) resulted in higher muscle strength during voluntary contraction as well as superior passive forces recorded at rest [2]. In our population of interest, it is probable that pain and muscle lengthening affect digital palpation scoring.

In sum, although digital palpation is contested for research purposes because of its subjectivity, it is widely used in clinical settings as it is practical, low-cost, and easy to apply. This tool provides important insight into PFM tone including flexibility, relaxation abilities, and TPs as well as PFM contractile properties. Its use for clinical practice is suggested for evaluating PFM dysfunctions, detecting surrounding muscle compensations, identifying tenderness area, and also orienting treatment.

11.2.7 Evidence in Women and Men with Heightened PFM Tone Using Digital Palpation

The involvement of the PFMs in dyspareunia, especially in provoked vestibulodynia, has been evaluated using digital palpation. Women with provoked vestibulodynia showed higher tone, as assessed with Reissing's scale, in comparison with asymptomatic controls [6–8]. Similar findings were also obtained using Lamont's scale [16]. Lower relaxation capacity and reduced flexibility were also found in women with provoked vestibulodynia [6] as assessed with the aforementioned scales (i.e., relaxation 0 returns to resting state—4 remains fully contracted; flexibility 0 one finger to two fingers abducted more than 2 cm) [6]. Regarding PFM contractile properties, women with provoked vestibulodynia were also found to have lower PFM strength measured with the modified Oxford grading scale [6–8]. Likewise, women with chronic pelvic pain had higher PFM resting tone (Dietz's scale) and decreased maximal PFM strength (modified Oxford grading scale) and relaxation capacity (absent/complete/partial) compared with pain-free controls [9]. Conversely, Fitzgerald et al. [37] did not find any significant difference regarding muscle strength (modified Oxford grading system) in women with and without chronic pelvic pain in a slightly smaller sample. As mentioned earlier, it is not possible to elucidate with palpation alone whether the reduction of pain results from a true weakness or from pain inhibition.

Studies concurred about the importance of muscle tenderness and TPs in women with chronic pelvic pain. The prevalence of TPs in the PFMs and surrounding muscles (e.g., obturator internus, piriformis) ranged from 63 to 89 % in women with chronic pelvic pain (including interstitial cystitis), which was significantly higher than in asymptomatic controls [23, 26, 37, 41–43]. Interestingly, Montenegro et al. [23] reported that TPs were associated with greater depression symptoms and higher rates of dyspareunia and constipation. Similarly in men, TPs were found in 75–88 % of men with chronic prostatitis/chronic pelvic pain syndrome [44]. Andersson et al. [45] also demonstrated that TP palpation in the PFMs and surrounding muscles reproduced the patient's symptomatology of pain in the penis, perineum, rectum, testicle, and groin. In fact, TP assessment is part of the UPOINT phenotyping system along with urinary symptoms, psychosocial dysfunction, organ-specific findings, infection and neurologic/systemic domains to classify urologic chronic pelvic pain [46].

11.3 Electromyography

EMG measurement is basically the recording of the electrical current travelling along the muscle fibers. During voluntary contraction, motor units, consisting of alpha-motoneurons and the

muscle fibers they innervate [47], are recruited resulting in the liberation of acetylcholine (ACh) at the endplate [48]. The binding of ACh at the endplate leads to depolarization of the membrane, which then spreads as an action potential along the muscle fiber allowing liberation of calcium and, thus, the interaction of actin and myosin to produce muscle contraction. Surface EMG assessment will capture this electrical phenomenon and the number of motor units recruited and their frequency of discharge will influence the signal amplitude and, hence, the force output produced. In light of tone physiology, circulating current at rest can only be explained by electrogenic spasms (unintentional muscle contraction) and normal electrogenic contraction (resting activity in normally relaxed muscle, although controversial, and myotatic reflex during stretching). It should be emphasized, however, that neither the passive component (viscoelastic properties) nor the contracture (TP) is captured by EMG.

The literature shows conflicting results when comparing women with and without pelvic /vulvar pain. Some authors have found elevated resting activity [9, 49–51] while others have determined nonsignificant differences between women with pain and controls [7, 52, 53]. This highlights the hypothesis, that, in some women, the involvement of heightened PFM tone is not explained by an electrogenic cause. Various degrees of reliability were also found in the literature when assessing EMG amplitude during maximal contraction as well as resting activity. Such divergences strongly suggest that some confounding factors should be taken into account when interpreting EMG signals. Among other confounding variables, factors related to the detection itself, such as the contact between the electrodes and the mucosa, vaginal lubrication and the thickness of the vaginal tissue, can greatly affect the EMG signal. Moreover, the presence of crosstalk, i.e., contamination from neighboring muscles, should be considered when interpreting the force from the EMG [30]. Chapter 15 is dedicated to EMG and presents thorough discussion of the recommendations and limitations regarding EMG.

11.4 Ultrasound

Ultrasonography imaging is a technology that has aroused great interest in both clinical and research settings for assessing PFM morphology and function in men and women with various urological and gynecological conditions using transperineal and transabdominal approaches [11, 54–57]. It offers significant advantages over the other methodologies as no vaginal insertion is required and is therefore pain-free. Particularly in a context of chronic pelvic pain, this has the benefit of limiting the bias due to pain and also, presumably, the participant's fear of pain or penetration. It should be noted that a detailed discussion of the diagnosis of levator ani trauma post childbirth (i.e., avulsion) [58] and pelvic organ prolapse quantification [59] using transperineal ultrasound is beyond the scope of this chapter.

11.4.1 Transperineal Ultrasound for Assessing PFM Tone and Contractile Properties

Transperineal (also called translabial) ultrasound allows good visualization of the bladder neck, urethra, vagina, anorectal junction and levator ani muscle and measurements of organ movement in relation to a fixed bony landmark, the pubic symphysis, making it more reliable for comparison between subjects. This approach therefore allows quantification of morphological parameters at rest and during contraction. In the mid-sagittal plane, the assessment of organ positioning at rest is attributable in part to PFM tone while organ mobility during contraction is related to PFM contractile properties. In the axial plane using 3- and 4-dimensional (3D/4D) imaging, it can also provide visualization of the levator ani morphometry both at rest and during contraction. Assessment is performed with a curved array transducer (3–6 MHz; 5–8 MHz for 3D/4D), covered with a condom or glove (with conductive gel on the probe and on the condom) and firmly applied on the perineum in a mid-sagittal alignment. The patient is asked to empty her bladder prior to the test and

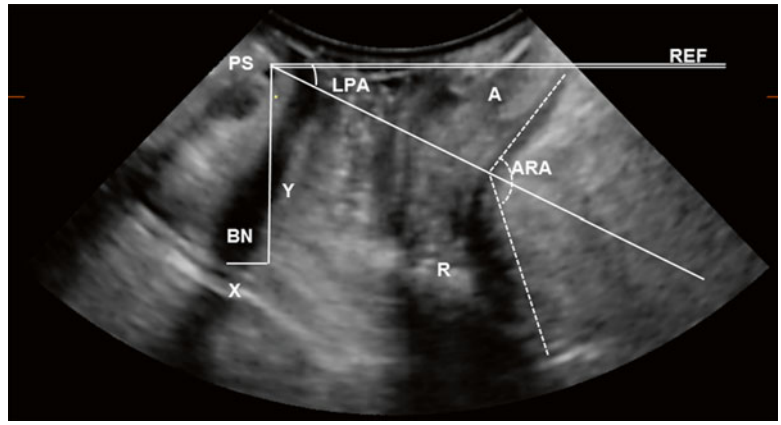

Fig. 11.1 Transperineal ultrasound—mid-sagittal plane. Identifying the anorectal angle (ARA, *dotted line*), the levator plate angle (LPA, *full line*), the bladder neck (BN), the pubis symphysis (PS), the anal canal (A), the rectal ampulla (R), the horizontal reference line (REF, *double line*), and the BN positioning relative to the *X*–*Y* axes

is usually evaluated in a recumbent position, although she can also be evaluated standing [60] or half-sitting [61]. Recent scientific literature abounds with parameters for evaluating PFMs at rest and during contraction.

In the 2D mid-sagittal plane (Fig. 11.1), the position and mobility of the bladder neck can be assessed. Dietz et al. [62] described analyzing this position using *x* and *y* axes relative to a horizontal line drawn from the inferoposterior margin of the pubis symphysis. Other authors propose using the whole pubis symphysis to trace the coordinate system aligning the *x*-axis with the central axis of the pubis symphysis [63, 64]. Both methods yield good test–retest and interrater reliability (coefficients *r* or ICC = 0.60–0.90) [60, 62, 65–67]. The cranioventral displacement of the bladder neck can also be calculated during contraction using the coordinate system [60, 62, 64, 67]. Similarly in men, the displacement of the bladder neck during a contraction demonstrated excellent test–retest reliability (ICC = 0.86–0.95) [61]. Furthermore, functional assessment of the bladder neck movement during coughing has been found useful in women with urinary incontinence in order to assess the reflex activity of the PFMs for stabilizing the bladder neck [68]. The ultrasound unit with a fast acquisition rate has the capacity to capture this rapid contraction. The anorectal angle, defined as the angle between the posterior wall of the rectal ampulla and the anal canal, can be calculated at rest [69]. During PFM contraction, the anorectal angle becomes more acute and moves cranially. This angle is influenced mainly by the puborectalis tone and contractile status [40]; it shows good reliability in both women and men (interrater reliability in men ICC = 0.57–0.70 [55]; test–retest in women 4.6–5.5 % of variation). With regard to the levator plate angle, this is measured between the horizontal reference line at the level of the pubis symphysis and the line from the inferoposterior margin of the symphysis pubis to the anorectal junction. It increases during contraction [60]. As discussed by Raizada et al. [40], the ascent (elevation) and descent of the pelvic floor, as evaluated by the levator plate angle, is hypothesized to be related to the tone and the contractile status of the pubococcygeus, ileococcygeus, and ischiococcygeus muscles. Good reliability was found in both women and men when assessing the angle at rest and its excursion during contraction (interrater reliability in men ICC = 0.90–0.93 [55]; interrater and test retest reliability in women ICC 0.46–0.64 [60, 66]). Instead of calculating an angle for quantifying the elevation of the levator, Stafford et al. [61] assessed the displacement of the anorectal junction using the coordinate system and showed excellent test–retest reliability in men (ICC = 0.83–0.93) for evaluating PFM contractile properties.

The development of 3D/4D ultrasound technology allows visualization of the levator ani muscle in an axial plane (Fig. 11.2) at rest and during contraction in order to assess hiatal biometry, muscle thickness, and muscle damage (i.e., avulsion injury). The measurements are made in the plane of minimal hiatal dimensions determined as the minimal distance between the

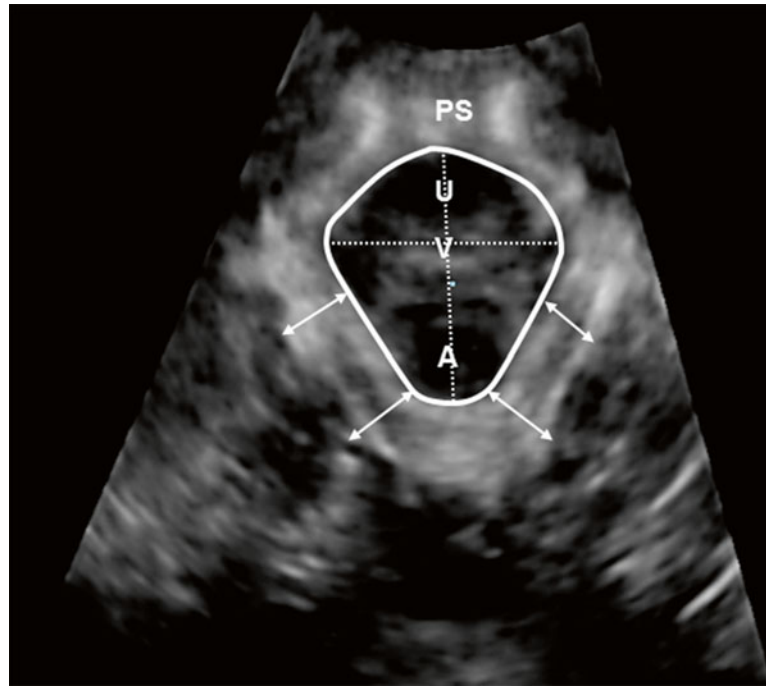

Fig. 11.2 Transperineal ultrasound—axial plane. Measurements taken in the axial plane of minimal hiatal dimensions. Identifying the pubis symphysis (PS), the urethral (U), the vagina (V), and the anal canal (A). Levator hiatus area (LH area) is marked with *lines*. The levator hiatus anteroposterior (AP) and left–right transverse (LR) diameters are drawn as a *dotted line* as well as thickness of pubovisceral muscle lateral to the vagina and rectum (*arrows*)

hyperechogenic posterior aspect of the symphysis pubis and the hyperechogenic back sling of the puborectalis muscle [71]. The levator hiatus area was delimited by the puborectalis muscle, symphysis pubis, and inferior pubic ramus in the axial plane [71]. Inside these borders, the anteroposterior distance corresponded to the levator hiatus anteroposterior diameter and the transverse distance measured at the widest part of the levator hiatus defined the levator hiatus left–right transverse diameter [60, 67, 71]. Measurements of the hiatus area and diameters at rest and their reduction during contraction showed good-to-excellent test–retest and interrater reliability (ICC = 0.61–0.96) [60, 66, 67, 71]. Levator ani thickness measurements can also be performed at rest with ICCs of 0.75–0.82 [60].

Moreover, supporting the validity of the measurements, transperineal ultrasound parameters have shown to be associated with different PFM assessment techniques and diagnostic tools [70, 72, 73]. For instance, dimensions of the hiatus, bladder neck, and levator displacement assessed with ultrasound have been associated with pressure perineometry ($r = 0.43$) [70, 72, 73], MRI measurement (ICC = 0.59–0.78) [74], vaginal palpation (modified Oxford grading system) ($r = 0.47$–0.58) [70, 75, 76], while the anorectal angle has been associated with evacuation difficulties and dyssynergia revealed with defecography findings [77]. In addition to the significant advantage of providing pain-free assessment, it has also been argued that Valsalva maneuver (i.e., straining) gives an insight into the extensibility of the PFMs under the force created by an increase in intra-abdominal pressure (IAP) and downward movement of the pelvic organs [71, 76]. However, when interpreting the findings obtained with ultrasound imaging, it is important to take into consideration that this is not a direct force measurement but rather an image showing the action of the muscle status. Hence, it is not a direct measure of muscle tone as it does not assess the resistance to stretching of the muscle.

11.4.2 Transabdominal Ultrasound for Assessing PFM Contractile Properties

A transabdominal approach has been described to evaluate movement of the posterior bladder wall during PFM contraction. The rationale for

measuring the amount of bladder base movement as an indicator of PFM contractility/strength relies on the fact that the bladder is supported by PFMs and their fascia, and tensioning of the fascia after PFM contraction results in encroachment of the bladder wall. A convex probe with a frequency of around 3–6 MHz, applied on the lower abdomen, can be orientated to visualize the bladder base movement in either the sagittal or the transverse plane (Fig. 11.3). Bladder filling at a standardized volume is required to allow clear imaging of the base of the bladder. Test–retest reliability studies conducted mostly in asymptomatic women showed an excellent reliability for assessing bladder wall movement in the transverse (ICC = 0.81–0.92) and sagittal (ICC = 0.84–0.91) planes [78–80]. Excellent interrater reliability was also observed by Sherbrun et al. with ICCs of 0.86–0.87 and 0.86–0.87, respectively [78]. Similar findings for test–retest and interrater reliability have been reported in men with a history of prostate cancer [54] and chronic pelvic pain [81] when assessing bladder base movement during PFM contraction. Supporting the validity of transabdominal ultrasound, bladder base movement has been correlated to bladder neck displacement obtained with transperineal ultrasound ($r = 0.63$) [79] and vaginal squeeze pressure ($r = 0.72$) [80] in women. Significant correlation was also found with vaginal palpation (modified Oxford grading scheme) in men ($r = 0.57$) [54] and women ($r = 0.62$) [82]. Conversely, Sherburn et al. [78] found no correlation between the modified Oxford and bladder base movement in women (transverse $r = 0.21$ and sagittal $r = -0.13$).

Several limitations should be acknowledged when attempting to quantify muscle contraction using transabdominal ultrasound. As opposed to transperineal ultrasound, the bladder wall remains a surrogate of contraction as the musculature cannot be directly visualized with this approach. Khorasani et al. [81] pointed out that patients with chronic pelvic pain may have more pain with a full bladder and therefore more tension and less PFM mobility, which affects their bladder movement. Also, the measurements are made without reference to a bony landmark and the amount of bladder base displacement is only expressed relative to a moveable starting point. It

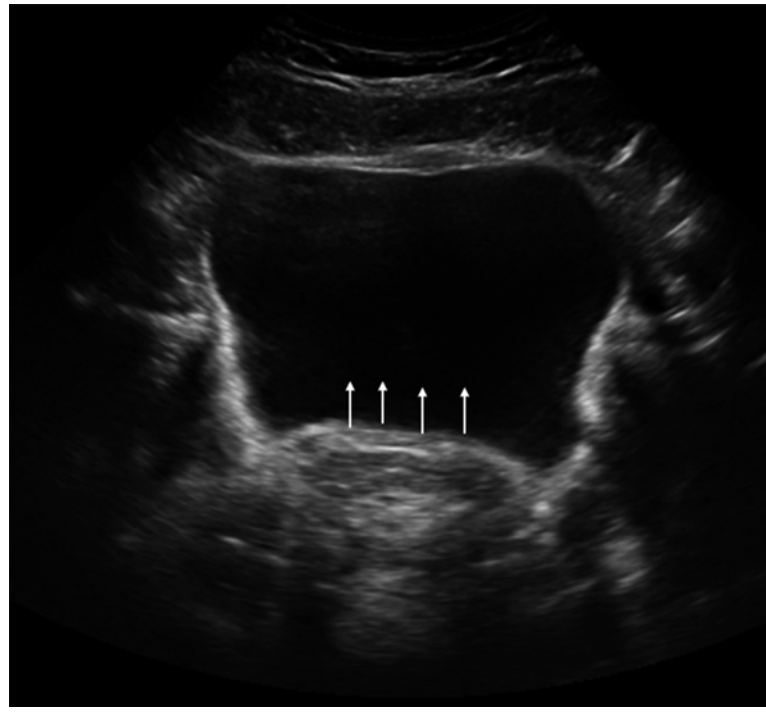

Fig. 11.3 Transabdominal ultrasound—transverse plane. Bladder base caudodorsal movement during PFM contraction

is therefore impossible to evaluate either the PFM tone or its contribution to the bladder movement. In other words, a patient with a hypertonic PFMs may exhibit a limited displacement of the bladder wall because the muscle is in an already contracted state. Moreover, abdominal muscle contraction and the presence of a prolapse can act as confounders. Thompson et al. [83] consequently suggested that transperineal approaches may be more reliable and suitable for inter-subject comparison.

11.4.3 Evidence in Women and Men with Heightened PFM Tone Using Ultrasound Imaging

A study comparing PFM morphology assessed with 3D/4D transperineal ultrasound in women with and without provoked vestibulodynia found that, at rest, women with PVD had a significantly larger levator plate angle, more acute anorectal angle, and smaller levator hiatal dimensions [11]. Taken together, this suggests higher PFM tone in women with provoked vestibulodynia. The fact that ultrasound assessment does not involve vaginal penetration and pain supports the hypothesis that the heightened PFN tone observed is not explained only by protective reactions. Moreover, as an indication of lower strength in the vestibulodynia group, less displacement of the bladder neck, less excursion of the levator plate and anorectal angles, and less levator hiatal narrowing were found compared to controls [11]. When evaluating men with urological chronic pelvic pain syndrome, similar ultrasound findings were reported suggesting heightened PFM tone in this population as well [55]. Using transabdominal ultrasound, Khorasani et al. [81] showed less bladder base movement during contraction, which may indicate lower strength. As discussed previously, this may be biased by an already elevated PFM tone limiting bladder base movement. Finally, transperineal ultrasound was found to be useful for diagnosing rectoanal dyssynergy as compared to defecography in men [84] and women [85] with symptoms of obstructed defecation. They found that rectoanal dyssynergy (paradoxical PFM contraction during straining) can be investigated by measuring anorectal angle during straining. In case of dyssynergia, the anorectal angle stayed small (acute) during straining instead of opening to allow evacuation.

11.5 Manometry

Manometry, also called perineometry or pressure measurement, is commonly used for evaluating PFM tone and contractile properties. It can also be utilized for anorectal investigation (see Chap. 12). It basically consists of a balloon—a vaginal pressure probe—connected to a manometer in order to measure the intravaginal pressure coming from the PFMs in millimeters of mercury (mmHg) or centimeters of water (cmH_2O) or other custom units, depending on the brand of manometers. It was in 1948 that Dr. Kegel [86] developed a perineometer to assess PFM strength in postpartum women with sexual dysfunctions. Since then, several types of pressure probes with different shapes and technical properties have been studied [87–90] and marketed under different brand names, for example, Camtech (Norway), Peritron (Australia), Miofeedback perina (Brazil), and Gymna (Belgium).

Vaginal resting pressure as a measure of PFM tone has been studied mainly in asymptomatic women and in women with incontinence. Using the Peritron device, good-to-excellent test–retest (ICC = 0.74–0.77) and interrater ($r = 0.78$) reliability have been demonstrated [34, 91]. However, resting pressure measurement in the upright position has resulted in poor reliability [38]. It should be pointed out that there are no clear recommendations as to whether the device should be calibrated to zero prior to insertion into the vaginal cavity nor how much the probe should be inflated prior to measurement. The latter would influence the probe size and, consequently, the muscle length and amount of resting pressure recorded. With regard to muscle physiology, intravaginal resting pressure will be influenced by a combination of the active and passive tone components.

Regarding contractile properties, maximal pressure during PFM voluntary contraction has shown excellent test–retest (ICC = 0.88–0.96) and interrater reliability (r = 0.88) [34, 38, 91–94]. These studies were done in a pain-free sample using the Peritron device. It is suggested that the device be recalibrated to zero just before every effort. Maximal strength could be reliably evaluated during a 3-, 5-, or 10-s contraction by considering one trial or the mean of three trials [34, 38, 91–94]. Regarding endurance measurement, Frawley et al. [34] found that endurance assessed during 20 repeated contractions was not reliable. Contrarily, Rahmani et al. [93] demonstrated excellent reliability when assessing endurance during a sustained 60 % maximal contraction (ICC = 0.83).

In support of the validity of manometry, maximal pressure measurement has been found to be correlated with vaginal palpation (modified Oxford scale (r = 0.70–0.81) [33, 95, 96] and Brink's scale (r = 0.68–0.71) [91, 92]), transabdominal ultrasound (bladder base movement (r = 0.72–0.81) [80, 95]) as well as transperineal ultrasound (bladder neck movement (r = 0.43) [70] and muscle thickness (r = 0.49–0.70)). Resting pressure was correlated with the levator hiatus area assessed by transperineal ultrasound (r = −0.46) [97]. However, it is generally recognized that increases in IAP, occurring if a patient co-contracts the abdominal muscles (rectus abdominis), or strain instead of contracting the PFMs can bias pressure measurements. Bo and Sherburn formulated recommendations to ensure the validity of the measurement [98]: (1) performing vaginal palpation before using the perineometer to make sure the patient is able to correctly contract her PFMs; (2) observing the cranial movement of the vaginal probe during measurement of the muscle contraction and (3) not considering the contractions associated with the Valsalva maneuver or retroversion of the hip [99, 100]. Following the last point, Bo and Constantinou [101] wrote a comment explaining that pressure should not be used to assess the reflex contraction of the PFM during coughing. They argue that pressure measurement is a summation of signals including PFM and IAP caused by the cough itself and that it is unlikely that the PFM reflex can be assessed in isolation. Considering these recommendations, the use of perineometry poses a problem when a patient has a really low PFM strength, because no inward movement of the probe is possible in this case. Furthermore, the size of the probe and the brand of the device have also been shown to influence the measurement [102, 103]. Barbosa et al. [103] compared the Peritron with two Brazilian devices and Bo et al. [102] compared the Peritron to the Camtech. Both studies conclude that measurements of vaginal squeeze pressure differ depending on the probe used. Despite these studies focused on maximal squeeze pressure, the results can be transposed to PFM tone assessment according to findings obtained with other tools [104, 105]. Different devices should therefore not be used interchangeably in clinical settings and results using different probes should not be compared or combined in systematic reviews or meta-analyses. The placement of the probe is another factor reported to be important. It was recommended to position the probe at the level of the PFMs that corresponds to the high-pressure zone inside the vagina [106, 107]. In sum, none of these studies on psychometric properties were undertaken in women with an overactive pelvic floor. Further investigation should be conducted in this population.

11.5.1 Evidence in Women with Heightened PFM Tone Using Manometry

Most of the studies so far have used manometry for investigating PFM dysfunctions in women with and without urogynecologic conditions such as incontinence and pelvic organ prolapse. There is thus a paucity of studies documenting its utilization in women with pelvic pain and an overactive pelvic floor. Rogalski et al. [108] demonstrated a significant reduction in resting vaginal pressure in women with a high-tone pelvic floor after intravaginal diazepam suppositories. In conference proceedings, Naess et al. [53] showed that women with provoked vestibulo-

dynia had significantly higher vaginal resting pressure compared to controls. In line with the hypothesis that elevated PFM tone may not always be explained by an electrogenic cause, these authors' results were not corroborated by EMG, as they found a nonsignificant difference in resting activity between the two groups. Furthermore, they observed a reduction in PFM resting pressure after a maximal contraction in women with provoked vestibulodynia indicating that contracting the PFMs can be used as a muscle relaxation technique.

11.6 Dynamometry

Several versions of intravaginal dynamometers have been developed in the last two decades for quantifying PFM tone and contractile properties. Another device, a myotonometer, has also been used for evaluating PFM tone by applying pressure externally on the perineum. Moreover, PFM tenderness and TPs can also be evaluated objectively with dynamometric devices (e.g., palpometer) in order to evaluate the pressure pain threshold.

11.6.1 Intravaginal Dynamometers

In the last two decades, over 11 intravaginal dynamometers have been developed to assess PFM properties. The *tonimetric device* also known as the "pince tonimétrique" developed by M. Caufriez was the first dynamometer to measure the PFM function [109, 110]. This dynamometer consists of two branches that can be opened by pressing on two handles in an angular excursion to increase the vaginal aperture. Although initially designed to assess PFM tone following an anteroposterior vector (i.e., at 12 and 6 o'clock), it can also measure PFM contractile properties. *Row's dynamometer* was described in a brief conference abstract. It consists of a probe with a movable rigid-window section against which the PFMs press during a contraction [111]. However, the latter were not studied for their psychometric properties and not used in other scientific works.

Michigan's dynamometer, used in clinical trials in women with incontinence and prolapse, and described in a patent document [112–115], is composed of two speculum branches equipped with strain gauges and affixed together in order to measure PFM tone and contractile properties at a predetermined static vaginal aperture. Test–retest reliability was only studied for strength measurement and an excellent reliability was found (ICC = 0.83) [116].

The *Montreal dynamometer* [117], designed to evaluate anteroposterior resting and contractile forces, comprises two aluminum branches: the upper one is fixed while the lower one, equipped with gauges, can be moved downward to increase vaginal aperture. The speculum is mounted on a supporting base so that the evaluator cannot bias the device by moving the unit, and the insertion of the speculum can follow the natural angle of the vagina [104, 118]. Several improvements were made to the dynamometer since its initial design in order to assess PFM tone, such as: (1) the mechanism that widens the vaginal opening was modified to create a smoother opening and a numerical linear-position transducer was incorporated to provide real-time measurement of the distance between both branches during a dynamic stretch [104, 118]; and (2) the size of the branches was reduced to that of a pediatric speculum to enable the assessment of women with vulvovaginal pain [10]. The speculum was designed to assess PFM tone more extensively, especially passive properties, by transposing to the pelvic floor a methodology used in the muscles of the limbs. This method consists in passively and slowly stretching the muscle while monitoring EMG activity to detect any electrogenic contributions [119, 120]. The EMG has to remain absent or negligible throughout the test, which can be defined by 1 % of maximal voluntary contraction or increase in EMG higher than two-standard deviation (SD), in order to assess only the passive properties of the muscle [2, 121]. Using four pairs of EMG electrodes affixed to the lower branch to monitor EMG, the following methodology was proposed to evaluate the PFM tone under four conditions [104, 118]: (1) PFM forces were recorded with the speculum closed at its minimal

opening; (2) PMF forces were recorded at the maximal vaginal aperture which corresponds to the maximal muscle length; the maximal stretching amplitude was determined by either the participant's tolerance limit or an increase in EMG activity; (3) To assess PFM passive properties during a dynamic stretch, the PFMs and surrounding tissues were stretched during five lengthening and shortening cycles conducted at constant speed; passive forces and passive elastic stiffness (PES) ($\Delta F/\Delta$aperture − muscle length) were calculated at different vaginal apertures. The muscle that exhibits greater passive resistance (greater force recording) and higher PES is considered "stiffer." The relationship obtained between the muscle length and the passive forces recorded clearly demonstrated that a larger aperture results in higher passive forces in asymptomatic pain-free women. The area between the lengthening and the shortening curve (i.e., the loss of energy associated with lengthening of viscoelastic tissues), called hysteresis, was also computed; (4) The percentage of passive-resistance loss after 1 min of sustained stretching was calculated to evaluate a skeletal muscle behavior known as "stress-relaxation" (i.e., loss of resistance over time with a sustained stretch) can be measured by computing the percentage loss in passive resistance following the application of a steady stretch over a prolonged period [119]. This methodology showed good-to-excellent test reliability in women with SUI with ICCs ranging from 0.66 to 0.88 [118]. Only passive forces at minimal aperture showed fair to good reliability with ICC of 0.51 [118]. Furthermore, contractile properties such as strength, speed of contraction, and muscle coordination as well as muscle endurance can be evaluated with the Montreal dynamometer. Maximal strength, calculated as a maximal force obtained during a 10-s contraction minus the baseline resting force, showed good-to-excellent test–retest reliability in postpartum women with ICC of 0.71, 0.88, and 0.76 for vaginal apertures of 19, 24, and 29 mm, respectively. As explained previously, Dumoulin et al. [122] confirmed that muscle length influences the force output, i.e., higher contractile forces are produced at wider apertures. For the speed measurements, the women were instructed to contract maximally and relax as fast as possible for 15 s. The speed of contraction was quantified by the rate of force development of the first contraction and the number of contractions performed during the 15-s period. Excellent test–retest reliability were found with ICCs 0.79–0.92 [118]. In the endurance test, the women were asked to maintain a maximal contraction for 90 s and the normalized area under the force curve was computed: (area under the curve/maximal strength) × 100 (ICC = 0.81). Various studies have been conducted to support the validity of dynamometric measurements. The maximal strength recorded with the dynamometer was correlated to vaginal palpation (modified Oxford scale, $r = 0.727$) [123]. Moreover, dynamometric measurements have been proven to be minimally influenced by increases in IAP [124]. Furthermore, good sensitivity to detect changes following treatment was also demonstrated [125].

Regarding the dynamometer developed by *Verelst & Leivseth* [105, 126, 127], it is composed of two branches to assess the PFM tone and contractile forces in a laterolateral position (i.e., transverse) rather than an anteroposterior force vector. Both branches can be opened to allow measurement from 30 to 50 mm of transverse opening. They evaluated the test–retest reliability of strength measurements using a coefficient of variability in women without any urogynecological conditions [105]. They obtained coefficients ranging from 11 to 22 %, which indicates good reliability. Although the assessment was done in the transverse direction, the same researchers also observed that the vaginal aperture influences the PFM strength assessment [105]. It should be pointed out that the size of the device may be a problem for the assessment of PFMs in women with pain.

Constantinou et al. [128, 129] developed a probe with four sensors, each mounted on a leaf spring that can be expanded once inserted to contact the vaginal wall and then retracted to a smaller diameter for probe removal. This configuration allows the assessment of spatial distribution of passive and contractile forces for each quadrant (anterior, posterior, left, and right). A

positioning system was added to the probe handle in order to track the orientation/angulation of the probe during PFM assessment [130]. This device has proven useful for discriminating between women with and without urinary incontinence [130, 131]. Likewise, *Saleme* et al. [132] designed a probe that similarly aimed to evaluate the spatial distribution of the PFM forces with the only exception that the sensors are not mounted on extractible leaf springs but are rather positioned on the probe. A study evaluating the positioning of this device using nuclear magnetic resonance imaging confirmed that the device's dimensions and sensor configuration were correctly positioned at the level of muscle mass to measure PFM strength, thereby collaborating validity. The reliability of Constantinou's and Saleme's devices has not been studied and their acceptability in women with gynecological pain also needs to be evaluated.

Three other speculum prototypes (from Nunes, Parezanovic-Ilic, and Romero-Culleres) were developed using the two branches of a conventional gynecological speculum equipped with strain gauges [133–135]. The *Nunes speculum* rests on a support system that can be turned to evaluate the PFM strength in the anteroposterior or transverse direction. The strength measurements were taken at different vaginal apertures which were individually adjusted to reach 4.9 N of passive forces. Evaluation of the test–retest reliability of strength measured in the anteroposterior and transverse directions reveals ICCs of 0.71–0.91 and 0.46–0.76, respectively [135]. In regards to the *Parezanovic-Ilic's speculum*, the information available is somewhat limited, since it was given in an article written in Serbian. The *Romero-Culleres speculum* was presented recently in a conference abstract [133]. Excellent interrater reliability of strength measurement was obtained in incontinent women with an ICC of 0.93 and the measurements were also found to be related to digital palpation (modified Oxford grading system).

The *Elastometer* developed by Kruger et al. [136, 137] was designed to assess the PFM tone in order to investigate its role in predicting delivery-related trauma. The two aluminum branches covered by removable plastic tips are introduced in the vagina and positioned to produce a transverse stretch with the help of a motor incorporated into the speculum. This enables the evaluator to apply a controlled stretch, at a constant speed, to the PFM tone in a transverse direction. The Elastometer reliability study focused on an assessment of PFM tone, as this was the purpose for which it was specifically designed, in asymptomatic women [136, 137]. Data acquisition was automated with the device opening in 20 stepwise increments from 30 to 50 mm over a 60-s period. Test–retest reliability was found to be excellent with ICCs of 0.86–0.92.

Overall, the intravaginal PFM dynamometers differ in terms of size and shape, the force vector recorded anteroposterior, laterolateral, or multidirectional forces and other technical issues. One main advantage is that they provide direct force assessment and most of them have been studied for their reliability. With the exception of the Montreal dynamometer, they evaluate PFM tone as the summative contribution of the active and passive components. Indeed, a methodology combining dynamometry and EMG was proposed to evaluate PFM passive properties while the activity of the muscle remains negligible. The main limitation associated with PFM dynamometers is their lack of accessibility because these devices are mostly used by their designers and not commercially available. Moreover, only the Montreal dynamometer was adapted and used in a sample of patients with pain and heightened pelvic floor conditions.

11.6.2 Myotonometer

A myotonometer (the MyotonPro™) is an instrument that has been used in skeletal muscle of the limbs to assess muscle tone [138–140]. This technology has been used recently for PFM assessment by applying pressures externally on the perineum [141, 142]. The device consists of a hand-held unit that is applied on the muscle at a predetermined level of pressure (preload). The device exerts mechanical impulses followed by release inducing damped oscillation on the muscle

at rest. Several parameters can be extracted from the oscillation curve, such as muscle stiffness which corresponds to the variation of forces divided by displacement of the tissue [138–140]. Interrater reliability was found to be good to excellent (ICC = 0.70–0.86) for assessing perineal muscle stiffness in women with and without vulvodynia [141, 142]. Although it is a new instrument in the field of pelvic pain and it has not yet been published in peer-reviewed journals, its utilization for assessing global PFM tone (i.e., summative contribution of active and passive components) is promising.

11.6.3 Pelvic Floor Tenderness and Pressure Pain Threshold

As discussed above, digital palpation is used to assess TP and muscle tenderness. In an attempt to provide a more objective assessment, Tu et al. [143] developed a device, a palpometer, to investigate pelvic floor tenderness, also called pressure pain threshold or mechanosensitivity. It consists of a force-sensing resistor attached to the index of the evaluator, covered with an examination glove, in order to evaluate the intensity of pressure required to elicit pain in various pelvic floor sites (pubococcygeus, puborectalis, obturator, ischial spine). Good-to-excellent test–retest reliability was found with a coefficient ranging from 0.61 to 0.84 [143]. Although it is not a PFM tone measurement, it provides relevant information about muscle pain and TPs which are closely linked to PFM heightened tone.

11.6.4 Evidence in Women with Heightened PFM Tone Using Dynamometry

The Montreal dynamometer was the only dynamometer used to investigate and compare the PFM passive properties and contractile properties in women with provoked vestibulodynia and controls [10, 144]. Higher PFM passive resistance at minimal aperture, higher stiffness at minimal and 15 mm aperture as well as lower hysteresis was found in women with provoked vestibulodynia [10, 144]. However, it should be underlined that women with provoked vestibulodynia tolerated lower vaginal aperture and some of them had more difficulty maintaining a low level of EMG activity. When excluding women with EMG activation during stretching, the same results were observed, suggesting the relevance of the passive/viscoelastic components of muscle tone to the findings of PFM impairments in women with PVD (unpublished data). Furthermore, women with vestibulodynia also showed lower maximal PFM strength, endurance, and speed of contraction, and achieved a lower number of contractions in 15 s. This suggests that contractile properties are also involved in provoked vestibulodynia pathophysiology [10, 144].

Using the myotonometer, Davidson et al. [141, 142] showed that women with vulvodynia had higher PFM tone (stiffness) compared to asymptomatic controls. With regard to pelvic floor tenderness, enhanced PFM pressure-pain sensitivity measured by palpometer during examination was also found in women with chronic pelvic pain for measurements compared to controls [9, 145]. Fenton et al. [146] even demonstrated that pressure pain threshold assessment can be useful for categorizing women with chronic pelvic pain into two groups (high sensitivity and low sensitivity according to their pain threshold). Such phenotyping should be further studied to determine if it influences the response to treatment. Similarly, in men with urological chronic pelvic pain syndrome, lower thresholds were found on the perineum area compared to controls [147].

11.7 Other Physical Examination Aspects

In addition to PFM tone and contractile properties, other physical aspects should be taken into consideration in conditions related to overactive pelvic floor. Connective tissue abnormalities (also termed “subcutaneous panniculosis”) is reported to be associated with the presence of TP

in underlying muscles and refers to a thickening of the subcutaneous tissues with an increased consistency and resistance to skin rolling [25]. Fitzgerald and Kotarinos [25] explained that connective tissue abnormalities are frequent in women with overactive pelvic floor and they identified commonly affected regions on the abdominal wall, thighs, low back, buttocks, and perineum. Connective tissue assessment as well as mobilization techniques warrant further empirical investigation into psychometric properties and treatment effectiveness. The pudendal nerve may also be involved in conditions related to an overactive pelvic through adverse neural tension (local factors interfering with nerve mobility during body movement), neuralgia, compression/entrapment, and neuropathy [25]. Likewise, a heightened PFM tone may also occur in response to inadequate posture, hip and sacroiliac joint dysfunctions [148]. Although these physical aspects were not in the scope of this chapter, a broader neurological and musculoskeletal assessment is mandatory in women with overactive pelvic floor.

11.8 Conclusion

Several assessment tools are available for assessing heightened PFM tone. Each instrument possesses its own advantages and limitations and evaluates different components of muscle tone. There is no one method capable of identifying the contributions from all the active and passive components of muscle tone. There is consequently neither published nor clinical evidence to suggest the existence of a single tool to comprehensively assess the PFM tone and contractile properties. A combination of tools is probably the most suitable approach to gaining a better understanding of pathophysiology. The available evidence in women and men with conditions related to an overactive pelvic floor suggests an elevated global PFM tone (measured by ultrasound, dynamometry, and manometry) TPs (measured by palpation and palpometer), increased viscoelastic properties (dynamometer and EMG), and, for some patients, elevated tone explained by electrogenic causes (evaluated by EMG). Empirical findings also indicate that the assessment of PFM should not be limited to tone since the contractile properties (strength, speed of contraction, control, and endurance) were also shown to be altered. It is still unclear, however, whether these dysfunctions result from pain inhibition, motor control deficits, or muscle weakness. Future research should be oriented toward further investigating the underlying mechanisms of elevated PFM tone, and studying the specific effects of physiotherapeutic interventions in terms of changes in PFM tone in women and men with overactive pelvic floor.

11.9 Appendix

Throughout this chapter, reliability and validity coefficients were interpreted following accepted standards. Reliability assessed with ICC was characterized as poor (ICC < 0.4), fair to good (ICC 0.40–0.59), good (ICC 0.60–0.74), or excellent (ICC >0.75) [149]. Kappa coefficients were described as poor (<0.21), fair (0.21–0.40), moderate (0.41–0.60), good (0.61–0.80), and very good (>0.80) [150]. Finally, correlation coefficients were interpreted as little or no relation (0.25), fair (0.25–0.50), moderate to good (0.50–0.75) and good to excellent (>0.75) [151].

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Electromyography

12

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Abbreviations

CP/CPPS	Chronic (nonbacterial) prostatitis/ chronic pelvic pain syndrome
DSM	Diagnostic and statistical manual of mental disorders
EAS	External anal sphincter
ED	Erectile dysfunction
EMG	Electromyography
Hz	Hertz
ICC	Intraclass correlation coefficient
MCID	Minimal clinically important difference
ms	Milliseconds
MUP	Motor unit potential
MVC	Maximum voluntary contraction
PFM(s)	Pelvic floor muscle(s)
sEMG	Surface EMG
μV	Microvolts

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12.1 Introduction

Electromyography (EMG) involves recording electrical activity generated during the physiological process of muscle contraction and presenting it back to the patient, clinician, or researcher. The clinical application of EMG is straightforward, where, with minimal training, patients can use such systems at home. However, the proper interpretation of EMG signals requires a sound knowledge of its theories and principles, including the technical specifications required to acquire and interpret EMG signals, and of the strengths and limitations of EMG as an assessment or biofeedback tool.

As discussed in Chap. 12, EMG is the only evaluation approach that can tell us about the electrogenic component of muscle tone. Its use is not, however, limited to the assessment of tone. EMG is used by basic scientists to aid our understanding of neurophysiology of the pelvic floor muscles (PFMs), by clinical neurologists to tell us about myopathic or neuropathic processes affecting the PFMs, by researchers and health professionals to study PFM activation patterns or to validate correct injection sites when botulinum toxin is used, and is beneficial to therapists and patients as a biofeedback tool to reduce PFM tone, enhance strength, or improve motor control.

Despite the ease of EMG application in other skeletal muscles, the PFMs pose several challenges.

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First, the PFMs are positioned in such a way that they cannot be readily accessed using conventional surface electrodes and are tricky to access using intramuscular electrodes. Second, and as discussed extensively in Chap. 1, the PFMs are located close to other larger skeletal muscles, often sharing nerve supplies [1] and fascial connections [2] with them, and have been found to work synergistically with them [3], thus making it difficult to determine what component of the acquired EMG signal represents isolated PFM activity. Third, unlike most striated muscles, the PFMs are rarely electrically silent [4]; they normally exhibit tonic activation when the body is at rest and their tonic activation is influenced by complex physiological reflex loops in addition to voluntary control [5–7].

By adhering to internationally accepted guidelines for acquiring EMG and respecting its inherent limitations, the information gleaned from EMG recordings can provide insight into the pathophysiology and motor control patterns of the overactive pelvic floor, and can be effectively used to provide meaningful, real-time feedback during training programs aimed at improving proprioception and relaxation of the PFMs [8–10].

12.2 Electromyography and the PFMS

12.2.1 The Origin of the EMG Signal

When a motor command is initiated through voluntary or reflex loops, selected nerves are chemically stimulated to allow the movement of ions across their cell membranes, resulting in transient changes in the electrical gradient across the cell membrane, called an action potential [11]. Action potentials propagate along individual alpha motoneurons and, at their end, branch out to a collection of muscle fibers belonging to a functional unit within the target muscle, known as the motor unit. The arrival of electrical current at the neuromuscular junction initiates muscle fiber action potentials that propagate along all muscle fibers belonging to that motor unit, in both directions away from the source of the stimulation. The number of muscle fibers that belong to an individual motor unit can range from tens to thousands and is proportional to the muscle's degree of fine motor control [12]. Muscle fibers belonging to a single motor unit are dispersed in a relatively small region within the muscle [13, 14], and contract almost synchronously [15] to produce a much larger signal than the individual muscle fiber action potential, referred to as a motor unit potential (MUP). The MUP is generally considered to be the smallest detectable functional unit of EMG activity [16].

Clinical EMG investigations examine MUP size and shape characteristics [17] by recording electrical activity from within the muscle or on the skin surface, and these reflect its anatomical and physiologic properties. In the external anal sphincter (EAS), for example, MUPs can be used to assist with the diagnosis of neuropathic disease by providing evidence of neuronal injury and repair [18, 19]. Because clinical EMG evaluations are mainly useful in neuropathic disease processes, which have not been implicated in the overactive pelvic floor, MUP investigations are rarely used in this population.

Normally, during a voluntary contraction, there is an orderly recruitment of motor units [20], beginning with smaller units that generate low force and have high endurance characteristics and ending with the largest motor units that can generate high forces but only for short periods. Because the PFMs must remain active to support the urethra and pelvic organs in upright postures, and produce high forces in response to rapid increases in intra-abdominal pressure (e.g., coughing, sneezing, laughing), the PFMs comprise a predominance (approximately 70 %) of smaller, fatigue-resistant muscle fibers [21–24], but also have fast twitch fibers that are found in higher proportion around the urethra and the anus [23, 25]. The amplitude of a recorded EMG signal generally reflects the number of active motor units at any given time as well as the firing rates of those units.

12.2.2 Overview of the Application of EMG to the Study of the Overactive Pelvic Floor

Over the past 20 years, the use of EMG to assess muscle activation amplitudes and patterns, as well as neuromuscular integrity, injury, and

reinnervation has inspired much research in patient populations where overactivity of the pelvic floor is suspected. EMG is an ideal means of evaluating PFM function and dysfunction, and many different techniques are used to record EMG from the PFMs. Below, we discuss key studies that have shed light on the pathophysiology of the overactive pelvic floor. The discussion is separated by the types of evaluations used, including (1) tonic EMG activation, (2) phasic EMG amplitude, (3) contractile endurance and stability, (4) activation timing and motor control, and (5) evoked activation of the PFMs.

12.2.2.1 Tonic EMG Activation of the PFMs

Pioneers in neuromuscular physiology such as Sherrington [26] and Harrell, Mead, and Mueller [27] demonstrated that striated muscles are normally electrically silent at rest. Tonic muscle activation, which is sometimes referred to as "resting muscle tone,", refers to observations that some striated muscles do not always completely relax, which is generally attributed to functional needs such as postural support [28]. This behavior is normally present in the EAS [29, 30] and the levator ani [4]. Tonic activation in the levator ani may be increased by physiological processes such as bladder fullness [31], postural loading [32], states of high psychological stress [33–35], or subtle increases in intra-abdominal pressure with respiration [4, 30]. Heightened tonic activation of the PFMs has been suspected to be a cause or an effect in several conditions associated with an overactive pelvic floor; research supporting this hypothesis is discussed further below.

Using concentric needle EMG, Shafik and El-Sibai [36] demonstrated significantly higher tonic EMG activation in bulbocavernosus, puborectalis, and levator ani muscles of women with vaginismus ($n=7$) compared to control women ($n=7$). They also found that the women with vaginismus demonstrated EMG activation during dilator penetration while such a response was not observed in the control group. Despite the very small sample size and the lack of psychometric data on their approach, the result is quite compelling as the women with vaginismus had almost double the tonic EMG activation amplitude as the control women.

Findings regarding increased tonic activity of the PFMs have been mixed when evaluating women with dyspareunia. In the mid-1990s, Glazer and colleagues were the first to develop a standardized PFM surface EMG (sEMG) assessment protocol to evaluate the overactive pelvic floor, using an intravaginal probe with longitudinal electrodes and an integrated EMG processing approach [37]. Initial studies suggested that tonic activation was elevated in women with vulvovaginal pain [37, 38]. In 1998, Glazer et al. used their protocol to compare PFM function between women with and without dysesthetic vulvodynia, and found that tonic activation was significantly higher in the women with dysesthetic vulvodynia ($n=25$) compared to healthy controls ($n=25$) [39]. While recording sEMG simultaneously from the superficial and deep PFMs, Gentilcore-Saulnier et al. further demonstrated that women with provoked vestibulodynia ($n=11$) had significantly higher tonic activation in their superficial PFMs but not their deep PFMs when compared to pain-free controls ($n=11$) [40]. Other controlled studies have not reported heightened PFM tonic activation in women with chronic vulvar pain [41–43], in women with overactive bladder [44], in men with chronic prostatitis/chronic pelvic pain syndrome [45], or in postpartum women with chronic pelvic pain and/or low back pain [46]. As mentioned, numerous physiological processes can influence PFM tonic activation, such as bladder fullness, intra-abdominal pressure, and psychological distress. However, as will be discussed in Sect. 12.3 of this chapter, the type of surface electrodes used can also contribute to discrepancies in experimental findings. Surface electrodes mounted on intravaginal probes are highly nonspecific and the resultant EMG signals may represent combined activation from both the superficial and the deep layers of the PFMs as well as other nearby muscles. Further research is required in order to elucidate which among the many PFMs may demonstrate heightened tonic activation in the different conditions associated with an overactive pelvic floor.

12.2.2.2 Phasic PFM EMG Activation

EMG has been recorded from the PFMs during voluntary contractions of varying intensities and tempos [37, 47], and involuntary responses to activities such as coughing [48] or postural challenges [32, 49]. The resultant electromyograms are considered to represent "phasic" responses. Phasic responses include information such as activation amplitude, timing, and rate.

The amplitude of an EMG signal is positively correlated with the number of active motor units within the pick-up area of the electrodes; however, the relationship is not necessarily linear [50]; doubling of EMG signal amplitude does not necessarily represent a doubling of the number of recruited motor units, nor does it reflect a doubling of contractile force. An increase in EMG amplitude during a sustained contraction can indicate increased motor unit recruitment if force output is increasing, or it can indicate a state of muscular fatigue if force output is holding steady or decreasing [51]. Activation amplitude is also affected by contraction type, for example, isokinetic vs. isotonic [50] and concentric vs. eccentric [52, 53]. Because the amplitude of the recorded EMG signal is also dependent on the size, location, and configuration of the recording electrodes relative to the location and number of active muscle fibers [54], as well as the excitability of the motor neuron pool [55], EMG activation amplitude recorded during voluntary activation is inherently highly variable between patients and between sessions [56].

Using phasic EMG activation, Shafik and colleagues [57, 58], as well as others [59], have made valuable contributions to our understanding of the anatomical structure and functional relationships among the different PFMs, demonstrating synergistic relationships between the bulbocavernosus and EAS [60] and the levator ani and the urethral sphincter [3]. Although these studies provide minimal quantitative detail, they have nevertheless enhanced our understanding of PFM function.

Similar to experimental findings where tonic activation amplitudes are compared between cohorts, there are also discrepancies in the literature in terms of phasic muscle activation differences between women with and without vulvodynia. White et al. [38] and Jantos [61] retrospectively reported that women with vulvovaginal pain demonstrated lower than expected phasic contraction amplitudes when they compared results to asymptomatic women. Studies have also demonstrated lower sEMG amplitudes upon sustained contraction in women with dysesthetic vulvodynia [39], and during quick "flick" contractions in women with vaginismus [62]. Engman et al. [41] reported that women with vaginismus generated lower mean sEMG amplitudes during a 60-s contraction compared to asymptomatic women. They failed to detect statistically significant differences in sEMG amplitudes between the groups during any other tasks (for example, during short tonic contractions, rapid contractions, and during relaxation). Other cohort studies have failed to demonstrate that maximal phasic contraction amplitudes in women with pelvic pain conditions associated with overactive PFMs were lower than pain-free control counterparts [40, 42, 43]. Where it is not clear what the sources of these differences are, as they could be related to strength, motor control issues, poor proprioception, or other causes.

In men with sexual dysfunction, EMG amplitudes recorded during phasic PFM contractions have demonstrated that men with erectile dysfunction (ED) generate lower voluntary PFM EMG activation than men without ED [63], and that some men with ED have concomitant PFM overactivity [60]. Among those men with ED and concurrent PFM overactivity, using concentric needle EMG, Shafik et al. (2004) found that the corpus cavernosum had heightened activation, and suggested that such activation may inhibit erection [60].

Investigating the pathophysiology of dyssynergic voiding through studying voluntary PFM contractions has not proven fruitful to date. A prospective case–control study [44] compared sEMG activity in 28 women with overactive bladder to 28 controls and found no difference in PFM EMG amplitudes recorded during voluntary contractions.

In summary, altered phasic activation of the PFMs, specifically lower activation amplitudes,

has been postulated to be associated with conditions where an overactive pelvic floor is suspected, but this phenomenon has been inconsistently observed in the literature. As will be discussed in Sect. 12.3, results may be confounded by the recording systems themselves, where the presence of an intravaginal or intra-anal probe may cause pain and in turn inhibit muscle contraction. Further, low EMG activation may reflect many phenomena, ranging from structural or anatomical differences, to differences in neuromuscular control.

12.2.2.3 PFM Endurance and Contractile Stability

There have been numerous studies reporting on PFM "endurance," where EMG activation has been averaged over sustained contractions held from 10 to 60 s [39, 41–43, 64]. Studies in women with suspected overactive PFMs have reported lower PFM EMG amplitudes during sustained PFM contraction. In these studies, force output was not standardized nor recorded during the tasks, and considering that other factors such as pain, proprioception, and motivation may influence the steadiness of motor output, it is impossible to conclude that differences noted reflect increased PFM fatigability in conditions associated with an overactive pelvic floor. Studies that describe muscle fatigue without a concomitant measure of PFM force or without confirmation of failure of the contractile mechanism through the application of neuromuscular stimulation to the PFMs (called the interpolated twitch technique [65]), cannot confirm the presence of muscular fatigue. The role of EMG in endurance assessment is discussed further below.

The use of EMG to measure muscle fatigue is quite complex as the observed changes in the EMG signal depend on the strength (force output) of the contraction. During submaximal contractions, as muscle fibers (motor units) fatigue, there may be recruitment of other motor units to take over the task, with an associated increase in EMG amplitude [66, 67]. Similarly, during maximal contractions, EMG activation amplitude will increase as the fatigue process progresses, while the motor demand for continued force output results in higher firing frequencies and lower action potential conduction velocities along the muscle fiber membranes in all active motor units [51]. Concomitantly, the frequency characteristics of the EMG signal will shift to a lower frequency band (reflected in the mean or median power frequency) before the force output of the muscle begins to decline as a result of the muscle's inability to sustain the contraction. Although the decline in mean or median power frequency of the EMG signal over the duration of a contraction is easily observed, the exact point of muscle failure is difficult to determine. Measures of endurance are probably better made through measuring the decline in force output over time during sustained contractions [51]. That said, using PFM dynamometry, Dumoulin et al. [68] found that the high variability in PFM force output during sustained contractions in women made it difficult, if not impossible, to quantify differences in endurance between patient populations.

Glazer and colleagues included measures of contractile stability in both men [45] and women [37–39] in their assessment of the overactive pelvic floor where, for example, they have reported that women with vulvodynia have higher standard deviation in their tonic and phasic activity than women without vulvovaginal symptoms [39]. Engman et al. [41] found no significant differences between women with and without vaginismus in the variance of EMG amplitude during tonic and phasic contractions. As discussed in Sect. 12.3and as presented in Fig. 12.3, the variation in baseline activation during sustained PFM contractions is highly dependent on the signal processing parameters used. In addition, the physiological interpretation of a higher standard deviation in tonic activity or during sustained contraction is not clear, as it may result from many causes including pain, poor proprioception, poor motor control, poor endurance, or poor motivation.

12.2.2.4 Activation Timing or Motor Control

The timing of PFM EMG activation relative to other nearby muscles during various tasks such as straining, coughing, and voiding is yet another

indicator of PFM function. Fine wire electrodes are well suited to this application as the muscle activation onsets are abrupt and therefore easy to detect. The detection of EMG onset using surface electrodes is much more complicated than using fine wire electrodes because the resultant signal most often demonstrates a gradual rise in EMG activation; selecting the appropriate point to label as the onset is challenging. Automated onset detection algorithms are reliable, but are highly susceptible to sources of noise interference (e.g., motion artifact and ECG interference as discussed in Sect. 12.3) that will negatively affect their validity. Several approaches to automated onset detection for sEMG data have been cited in the literature over the past 20 years [69–71], but to our knowledge none have been validated against wire or needle EMG activation onset in the PFMs.

Clinically, onset detection from sEMG signals is often done by visual inspection of raw EMG signals. For example, the relative timing of detrusor contraction (through bladder and urethral pressure measurements) and levator ani activity (recorded using sEMG patch electrodes located on the perineum) is used to diagnose voiding dyssynergia, where the PFMs are normally expected to relax when the detrusor muscle is active [72–76]. Patients with postpartum low back and/or pelvic pain [46], vaginismus [77], dyssynergic urination [78], and defecation disorders [72, 73, 79] have all demonstrated the presence of paradoxical sEMG activation recorded from their PFMs during straining activities, where relaxation of the PFMs is normally expected. This observed behavior points to abnormal neuromuscular control, and is particularly well suited to the use of PFM EMG as a real-time biofeedback tool to train or reestablish normal behaviors.

Urodynamic flow analysis using EMG is commonly used to diagnose PFM dyssynergia in children and adults [72–74, 76, 78, 80]. In children, dyssynergic voiding is identified through the presence of a "staccato pattern" to confirm urethral sphincter contraction, rather than relaxation, during voiding [74, 75, 80]. Surface patch electrodes are widely used to compare muscle activation patterns during filling and voiding as part of this routine urodynamic assessment. Kirby et al. [78] demonstrated that surface patch electrodes did not represent the expected activation of the pelvic floor and external urethral sphincter during voiding and relaxation, whereby EMG amplitude was actually higher during voiding compared to filling using both qualitative and quantitative evaluation. As discussed in more detail in Sect. 12.3, this is not surprising considering that EMG recorded using surface patch electrodes on the perineum is a reflection of global muscle activation, and should not be used as a surrogate for urethral muscle activation.

Recently, van Batavia et al. [80] suggested a novel EMG lag-time analysis to assess voiding dyssynergia in children by reporting the time interval between PFM relaxation, and the initiation of voiding as a potential metric. They suggested that normal values range between 2 and 6 s and suggested that detrusor overactivity should be suspected when this delay is shortened. This idea shows promise but has not yet been tested using proper psychometric assessment.

The inability to relax the PFMs during defecation efforts is the main criterion used in the diagnosis of dyssynergic defecation [81] and is part of the Rome III diagnostic criteria [79]. The specific criterion states that dyssynergic defecation is present when there is an appropriate expulsive force concurrently with either an inappropriate PFM contraction or with less than 20 % decrease in resting baseline EMG amplitude. The timing and EMG amplitude criteria are commonly based on visual inspection of the EMG data, and as such, are not well standardized. When looking at timing of PFM quieting during defecation efforts, Ribas [72] examined two cohorts of women with functional constipation using intra-abdominal pressure, defecography and EMG, and concluded that 68 % of women demonstrated pelvic floor paradoxical contraction upon defecation. Bordeianou et al. [73] found that sEMG values recorded during voiding did not predict pelvic floor dysfunction when compared to defecography, considering that among 64 patients who had signs of dyssynergia on sEMG, only half had signs of dyssynergia on defecography, which is considered the gold standard. Based on these

findings, there is a need to reevaluate the Rome III criteria in the diagnosis of dyssynergic defecation.

As will become clear in Sect. 12.3, the difficulties in assessing PFM dyssynergia may result from the highly nonspecific nature of the surface patch electrodes generally used for these investigations. Given the current evidence, further research is urgently needed, where more appropriate EMG electrode types and configurations may more clearly identify the presence or absence of paradoxical PFM activation and alterations in the timing of PFM relaxation prior to voiding to allow us to understand conditions such as urinary retention disorders, chronic constipation, and even pelvic pain, dyspareunia, and ED.

12.2.2.5 Evoked Activation of the PFMs

The study of evoked activation of the PFMs has the potential to inform our understanding of the roles of the central and peripheral nervous systems in the overactive PFM, notably by measuring the excitability of the central nervous system by looking at the amplitude and timing of the EMG response. Evoked activation can be induced using electrical or mechanical excitation of the pudendal nerve bundle or S2–S4 nerve roots via stimulation of the motor neuron pool, and/or stimulation of cortical interneurons using transcranial magnetic stimulation, and by recording the resultant EMG activity from the PFMs. Evoked potential studies involving the PFMs are rare as the nerves innervating the PFMs are difficult to access along their pathways deep within the pelvis. These techniques have high within-subject variability [82–85] partly because the stimulation is imprecise and might lead to activation of nearby muscles, which precludes their usefulness to study the corticomotor pathways to the PFMs and might explain why they have seldom been used. The reliability of the amplitude or latency of other PFM responses has not been discussed in the literature.

Nevertheless, Frasson et al. [86] successfully used concentric needle electrodes to demonstrate hyper-reflexia of the bulbocavernosus muscle in response to clitoral stimulation in women with vulvodynia. Regardless of subtype, women with vulvodynia demonstrated polysynaptic reflex amplitudes that were approximately twice as high as those recorded from a control group, providing compelling evidence of a central nervous system involvement in this condition. This result was consistent with findings from our laboratory of heightened sEMG responses of the bulbocavernosus muscle in women with provoked vestibulodynia when a pressure stimulus was applied to the vulvar vestibule, as compared to pain-free women [40]. It may also reflect the same phenomenon witnessed by Shafik et al. [36] in the evaluation of women with vaginismus, whereby PFM EMG responses were observed during vaginal probe insertion. The presence of PFM spasm was initially included in the definition of vaginismus, but has, however, since been withdrawn due to conflicting evidence [87, 88].

12.2.3 Summary

Although much has been learned about the functional anatomy and neurophysiology of the PFMs through EMG investigations, with results pointing towards the presence of higher electrogenic tone and PFM dyssynergia, high quality research in this area is lacking. Much more can be learned through further investigation, however, several considerations must be taken into account when recording and interpreting EMG signals in both the clinical and research settings such that the resultant EMG information is accurate and reliable. These are discussed in the following section.

12.3 The Acquisition of EMG Signals

EMG signal properties are highly influenced by the instrumentation used to record the signals, including the characteristics of electrodes used, signal acquisition parameters, signal processing techniques, and signal presentation. Each of these is discussed briefly below.

12.3.1 Characteristics of EMG Electrodes

12.3.1.1 Electrode Type

EMG signals can be recorded using needle, fine wire, or surface electrodes, and each electrode type has distinct advantages and disadvantages. First, it is important to consider the purpose of EMG recording. Will the EMG data be used for biofeedback, for research, or for diagnostic purposes? Next, it is important to consider the availability of appropriate equipment, the skill level of the examiner, and what certification is mandated by the jurisdiction when using more invasive techniques. Finally, and perhaps most importantly, specific factors to consider include the patient's acceptance of the electrode (internal vs. external), age, anatomy (size), sexual experience, as well as the possibility of the recording electrode itself inducing muscle activation (e.g., due to pain or fear). A thorough understanding of the strengths and limitations of each type of electrode, summarized in Table 12.1, will guide this decision.

12.3.1.2 Needle Electrodes

Needle electrodes are small and highly specific, picking up only that electrical activity propagating within approximately 0.5 cm^2 of the needle tip [89]. They are mainly used by neurologists and physiatrists in the clinical EMG evaluation of MUP properties to assist with diagnosis of neuropathy or myopathy [90]. Needle electrodes can, however, be used to study activation in muscles that are small and/or situated deep beneath the skin. They can provide a clear indication of the timing of motor unit activation, demonstrating the exact time that the first motor unit near the needle tip becomes active, but are not very useful when determining the state of activation of the muscle as a whole. The presence of a needle within a muscle during contraction can lead to pain inhibition or muscle spasm, thus influencing the validity of the outcomes. In many jurisdictions, needle electrodes can only be inserted by a health professional with specific certification in EMG.

In PFM research, needle electrodeshave been used in key neurophysiologic studies by Podnar et al. [91–96], who investigated MUP morphology and firing rates primarily in the EAS to elucidate the pathophysiology of fecal incontinence and constipation, and by Shafik and colleagues [5, 36, 57, 58, 60, 97–99], who investigated neurophysiologic links between the various PFMs during examinations of sexual function and dysfunction.

12.3.1.3 Fine Wire Electrodes

Fine wire electrodes can be particularly useful when studying small muscles and/or muscles that are located deep beneath the skin surface, and should be considered when electrodes placed on the skin surface are likely to receive electrical contributions from other nearby muscles, a phenomenon known as cross talk. Often they are inserted as pairs, where both wires are threaded into a single needle cannula. The wires are hooked at their ends such that when the needle is inserted into the muscle and then withdrawn, the hooks hold the wires in situ. The fine wires are insulated except for their tips, which register EMG signals within the local muscle region where they are located. As with needle electrodes, fine wire electrodes can pick up single motor unit activity and are very useful when studying the onset of muscle activation because the rise in EMG during activation is more abrupt than it is with surface electrodes, however, as with needle electrodes, they may not record the global level of activation within a muscle [100, 101], particularly when studying larger muscles. The major advantage of fine wire electrodes over needle electrodes in terms of studying muscle activation amplitude and/or timing is that fine wire electrodes are virtually painless once the needle cannula is removed. In some patients with an overactive pelvic floor where pain may prevent the use of intravaginal or intra-anal probes or a pain response may be triggered by the use of probes, fine wire electrodes may be the preferred choice. They might also prove highly useful in the study of dyssynergic voiding, where there is a need to determine accurate timing information.

Table 12.1 Comparative table of EMG PFM recording electrodes

Electrode types	Recording techniques	Advantages	Disadvantages
Surface electrodes	Superficial PFMs: Small (1 × 10 mm bar or 1–3 mm diameter circle), paired electrodes (10 mm separation) on each side of the perineum adhered over the specific muscle of interest (e.g., bulbocavernosus, ischiocavernosus, or EAS)	Noninvasive	Relatively nonspecific
	Deep PFMs: paired electrodes (20–40 mm bar or circular electrodes with diameter up to 10 mm, separated by 5–20 mm), located on a probe or other device that is inserted into the vagina or anus	Does not interfere with normal contractile activity	Difficult to adhere to the skin surface, often need to shave skin under site of electrode application
		Gives a global estimate of muscle activation	Prone to motion between the skin and the underlying muscle of interest therefore prone to motion artifact and to cross talk
Fine wire electrodes	Twisted pair of fine wire inserted into the muscle of interest through a needle cannula. The cannula is removed and hooked wires remain in place	Risks of bleeding, hematoma and infection	More invasive to insert than surface electrodes
	Insertion location determined using insertional activity and activity on contraction or ultrasound guidance	More selective to specific muscle of interest	Possibility of causing a hematoma
	For deep PFMs, can be inserted through the perineum or through the vaginal wall	Less likely to record cross talk than surface electrodes. Will remain situated in muscle despite migration with respect to the skin surface	Prone to motion artifact
Needle electrodes	Can be located through evidence of insertional activity upon insertion of needle, the presence of motor unit activation at rest and on contraction or with ultrasound guidance	Risks of bleeding/ hematoma and infection	Invasive and potential for hematoma
	For deep PFMs, can be inserted through the perineum or through the vaginal wall		Often too selective to record global muscle activity
	The concentric needle remains in the muscle of interest during contractile or reflex efforts	Ensures that the specific muscle of interest is studied with little risk of cross talk	Can be uncomfortable, particularly during muscle contraction therefore may alter natural contractile behaviors
			In certain jurisdictions, can only be inserted by a health professional with specific certification

Comparison of surface, fine wire and needle EMG electrodes based on advantages and disadvantages of their respective use. *PFMs* pelvic floor muscles, *EAS* external anal sphincter, *mm* millimeter

Despite the fact that fine wires are an ideal electrode type for recording EMG from the small PFMs that lie deep within the pelvis where potential for recording cross talk is high [102], fine wire electrodes have not commonly been used when studying the PFMs. Binnie et al. [103] reported high correlations between fine wire EMG recorded from the EAS and sEMG recorded intra-anally using longitudinally oriented electrodes on either side of an anal probe. However, their sample size was small ($n=8$) and the reliability analysis was insufficient to support this conclusion. Stafford et al. [104] recently described a technique for using fine wire electrodes to study the activation of the bulbocavernosus and puborectalis in men, and Auchincloss and McLean [105] used fine wire electrodes to determine that there was no measurable effect of having an electrode probe located in the vagina when women performed voluntary PFM contractions ($n=12$). As with needle electrodes, the insertion of fine wire electrodes requires technical skill, certification, and specialized instrumentation (refer to Sect. 12.3.3), which may explain their limited use. For any fine-wire insertions, and especially for the PFMs, the availability of an ultrasound system to validate the location of insertion is highly advantageous.

12.3.1.4 Surface Electrodes

Surface electrodesare by far the most common method of recording EMG activity as they are noninvasive and provide a global signal that reflects summed action potential activity from a large portion of the active muscle fibers within the muscle. However, surface electrodes are only useful for recording EMG activity from muscles that are located close to the skin surface; otherwise, the resulting EMG signals may contain cross talk which confounds study results by overestimating the EMG activity within a single muscle.

As noted in Sect. 12.2, surface "patch" electrodesare commonly used to study PFM dyssynergia, particularly during clinical urodynamic and defecation studies. These adhesive electrodes are normally placed perianally and are assumed to reflect the activation of the levator ani group of muscles. This approach is, however, highly nonspecific, as the electrodes are located far from the desired signal source, except in the case of the EAS [103]. In general, electrodes mounted on intravaginal or intra-anal probes are preferable; however, there are situations in which intravaginal or intra-anal probes are inappropriate, for example, in pediatric populations, in accordance with certain sociocultural norms, or in women or men with severe pain with penetrative activities.

When recording EMG from the levator ani, surface electrodes are most often mounted on a vaginal or anal probe to record activity in close anatomic proximity of the pubovaginal and puborectal muscles. Intravaginal and intra-anal surface electrodes have been used to evaluate the tonic and phasic activation of the levator ani in women and men with vulvovaginal and pelvic pain, respectively. Some of the discrepancies in the literature described in Sect. 12.2 may be explained by differences in electrode type, size, and configuration.

12.3.1.5 Electrode Configuration and Size

Although the same principles hold true for needle and fine wire electrodes [106], electrode configuration errors are by far most common when surface electrodes are used to record EMG signals, and thus our discussion of electrode configuration will focus on sEMG.

Most modern EMG systems use differential amplifiers, meaning that they subtract (take the difference between) activity seen at two "active" electrodes before the data are recorded. If both electrodes are placed over the same muscle, since MUPs propagate along the muscle, they are "seen" by one electrode before they are "seen" by the other; therefore, signals do not pass by both electrodes at the same time and are not subtracted out of the resultant signal. Signals that are common to both electrodes, for example, electrical noise, are subtracted away. Noise sources, such as interference from other electrical equipment in the area, are further minimized by using a ground electrode placed on the skin over a bony prominence where physiological activity is expected to be minimal, which removes noise that is common

to all channels (termed common mode rejection). In order for this system to work appropriately, the two active electrodes should be oriented along the line of action of the muscle fibers. The levator ani muscle group has distinct origins, insertions, and innervations on each side of the pelvis; therefore, its activation should be recorded using intravaginal probes that make distinct and differential recordings from two active electrodes on each side [107, 108]. In comparison, because the EAS has circumferential fibers, intra-anal probes with one bar on each side of the probe may be appropriate for recording its activation. Results by Binnie et al. [103] support this principle, where they found that electrodes oriented longitudinally on either side of an intra-anal probe recorded higher voluntary and reflex activation amplitudes from the EAS than circumferential electrodes.

In addition, large, widely spaced electrodes will record signals that originate from sources much deeper or farther away than small and closely spaced electrodes [109], and will therefore record higher EMG activation amplitudes, representing activity from a larger motor unit pool, whether those motor units are generated by the muscle of interest or not. The larger amplitude signals, having traveled some distance through the tissues to arrive at the electrodes, undergo a "volume conductor" effect [109], meaning that their peaks will not be as sharp and their timing information will be less precise. Similarly, if an intravaginal or intra-anal probe is tilted, higher EMG amplitudes can be expected on the side of the probe that more closely approximates the PFMs. Hence, researchers and clinicians alike must keep in mind that depending on PFM tone, vaginal/anal laxity, and individual anatomical differences, the distance between the PFMs and the recording electrodes can change within a session, introducing bias in the signals.

Larger, more widely spaced electrodes are most useful when large, superficial muscles are investigated. They are advantageous in terms of the reliability of the resulting EMG signals since they record contributions from a larger proportion of the whole motor unit pool, and they are less susceptible to differences in motor unit recruitment between trials or between sessions [56, 108, 109]. Unfortunately, most commercially available intravaginal and intra-anal probes use electrode surfaces that are larger than the contact area of the PFMs or the EAS, thus increasing the likelihood of cross talk contamination. The impact of electrode size and spacing is evident in recent EMG data published by Auchincloss and McLean [56]. Using identical signal processing techniques, EMG amplitudes reported for maximum voluntary contractions (MVC) of the PFMs ranged from 45 to 50 μV when acquired by the FemiScan™ probe and from 120 to 140 μV when acquired from the same sample on the same day but by larger, further separated Periform® electrodes. According to our research, even though the electrodes are likely too long, the FemiScan™ is the only currently available commercial intravaginal probe that records EMG activation from the deep muscles of the pelvic floor with an appropriate electrode configuration [107].

Other appropriate intravaginal surface electrodes have been reported in the literature; however, none appear to be commercially available at this point. As early as 1985, Lose et al. [110, 111] used paired electrodes (surface area 38.5 mm each) mounted on a sponge that was inserted into the vagina over the lateral vaginal wall to record levator ani activity. Stafford et al. [112] described an intra-urethral electrode to investigate EMG activation of the striated urethral sphincter in which four electrodes were located around a catheter, and suction was used to hold the urethral walls still against the catheter tip, and have used this electrode to study urethral sphincter function in healthy men [104]. Keshwani and McLean [102, 108] developed an intravaginal differential suction electrode with small, closely spaced electrode surfaces that are less prone to cross talk and, because it is held to the vaginal wall using suction, it is less prone to movement with respect to the vaginal wall causing motion artifact (e.g., artifact is noise contamination from nearby muscles that falsely increases EMG amplitudes) in the resulting EMG signals [102]. Voorham-van der Zalm et al. [113] have also reported a vaginal and anal probe design that incorporates small electrodes. However, their

electrode configuration is monopolar, rather than differential, with activity under each small electrode referenced to a surface electrode placed on the skin surface over the pelvis. This probe may prove to be quite useful for the study of different clinical populations [114]; however, it requires further psychometric assessment at this time, since the electrode configuration has potential for recording high levels of crosstalk and noise interference. Readers who desire more in depth information and a critical appraisal of various commercially available probes can refer to a review published in 2015 by Keshwani and McLean [107].

12.3.1.6 The Electrode Tissue Interface

With conventional EMG electrodes and systems [115], it is recommended that users perform a proper skin preparation before adhering surface electrodes to the skin surface over the muscle(s) of interest. This process involves cleaning and gently abrading the skin surface and rubbing conductive paste into the skin in order to reduce the impedance between the skin and the electrode surfaces to minimize the loss of signal amplitude. This and other formerly essential criteria for the electrode skin interface such as recessing electrodes and using conductive paste between the electrode and the skin surface have been relaxed in recent years as modern amplifiers have improved substantially [116]. That said, attention should be paid when placing electrodes on the perineum: The skin should be hairless, clean, and dry, and the electrodes should be securely adhered to the skin surface using adhesive or suction. If one of the electrodes lifts even slightly during the EMG evaluation, the recorded sEMG signals are no longer valid.

Despite following recommended guidelines, the electrode–tissue interface is still a large potential source for noise contamination in sEMG signals when the electrodes move with respect to the tissue surface from which they are recording signals. This is particularly a problem with intravaginal and intra-anal electrodes that do not generally adhere to the vaginal wall. The resultant motion artifact (Fig. 12.2) can be a major problem especially given that the lifting action of the levator ani naturally causes migration of the electrodes relative to the underlying muscles. Motion artifact can also be caused by motion of the leads attaching the electrodes to the amplifiers [117]. Keshwani and McLean [102] investigated motion artifact contamination in EMG data recoded from the PFMs during coughing and found that close to 30 % of files recorded using the FemiScan™ probe were contaminated with motion artifact, whereas only 14 % of files were contaminated when suction was used to hold a differential electrode securely to the vaginal wall.

12.3.2 Signal Acquisition Parameters

The acquisition parameters used to record EMG signals follow basic engineering principles to optimize signal quality and the resolution of signal amplitude, and to capture the signals at a fast enough rate to avoid distortion. However, unlike customizable EMG systems used in research applications, most commercially available EMG systems incorporate technical features that are not adjustable. Commercial EMG systems designed for surface electrodes are rarely compatible with fine wire or needle electrodes because the amplifier filters must be optimized for each application, and the frequency bandwidth of EMG data recorded are dramatically different depending on the electrode type (i.e., 450 Hz for surface electrodes, 1000 Hz for fine-wire electrodes, and much higher for needle electrodes). In parallel with the amplifier characteristics, the signal acquisition rates (sampling rate) of the EMG signal must match the type of electrodes and amplifiers used (i.e., sEMG requires a sampling rate of 900–1000 Hz, whereas fine wire or needle electrodes require sampling rates of 1600–2000 Hz and 8000–10,000 Hz, respectively). These sampling rates are based on a mathematical theory, the Nyquist Theorem [118] whereby the EMG signals should be sampled at a frequency that is at least twice the maximum frequency of the information present in the signal, which can be determined using a mathematical process called Fourier analysis [119]. Sampling at too low a rate dis-

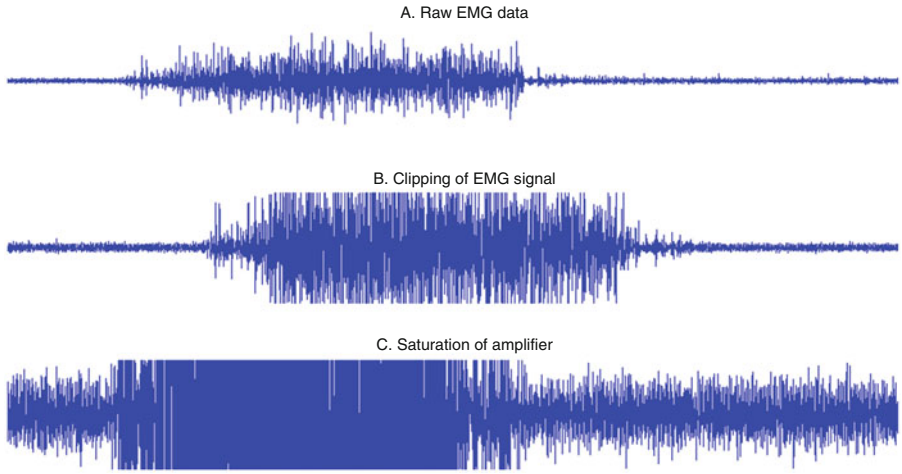

Fig. 12.1 Recording errors when acquiring EMG data. (**a**) Raw EMG without recording errors, note the variation in peak amplitudes. (**b**) Clipping of EMG signals as can be seen by the blunted edges of the recording. (**c**) Saturation of amplifier where the signal amplitude exceeds the resolution of the display

torts the EMG signal. More details on these EMG acquisition principles are widely available through books such as Merletti and Parker [67], and Internet resources [115, 120].

12.3.3 Signal Processing

Particularly for research, it is important to have the capacity to inspect the unprocessed or raw EMG data for errors in collection (Fig. 12.1): Recording errors before processing, as these sources of error can easily be interpreted as physiological data when observers are inexperienced and/or if commercial systems only present processed EMG data, which is the case in most clinical biofeedback systems. The time axis (*x*-axis) plays also a large part in our capacity to see noise such as motion artifact in the EMG signals (see Fig. 12.2). Commercial systems that do not present the raw EMG data for inspection should not be used for research applications.

How EMG signals are presented and interpreted is highly dependent on how the EMG data are treated mathematically or "processed." In order to make use of the physiological information available in EMG recordings, the signals are normally filtered or smoothed. Different approaches to signal processing are presented in Fig. 12.3. Most commonly during data processing and to present a smooth burst of activity, the data are first full-wave rectified (panel b), effectively making all negative values positive, and then averaged, integrated (panel c), or filtered (panels d–i).

As a general rule, when EMG data are averaged over a particular window, the more dynamic the nature of the contraction, the shorter the smoothing window should be. Tonic EMG activation can be smoothed using long windows (200 or even 500 ms), whereas more rapid contractions should not be smoothed over periods longer than the duration of the EMG burst itself; otherwise ,amplitude information will be lost. The effect of smoothing window length on EMG activation amplitude is demonstrated in Fig. 12.3 panels d–f. Another signal processing approach is to use a mathematical filter, such as a Butterworth filter, to smooth EMG data. These filters can be optimized to reduce motion artifact and other specific noise sources [121]. However, a mathematical approach referred to as "forward and back" is normally necessary in order to ensure that fluctuations evident in the smoothed EMG data accurately reflect the time at which the fluctuations occurred in the original EMG signal. This feature is particularly important when muscle activation timing is of interest. The lower

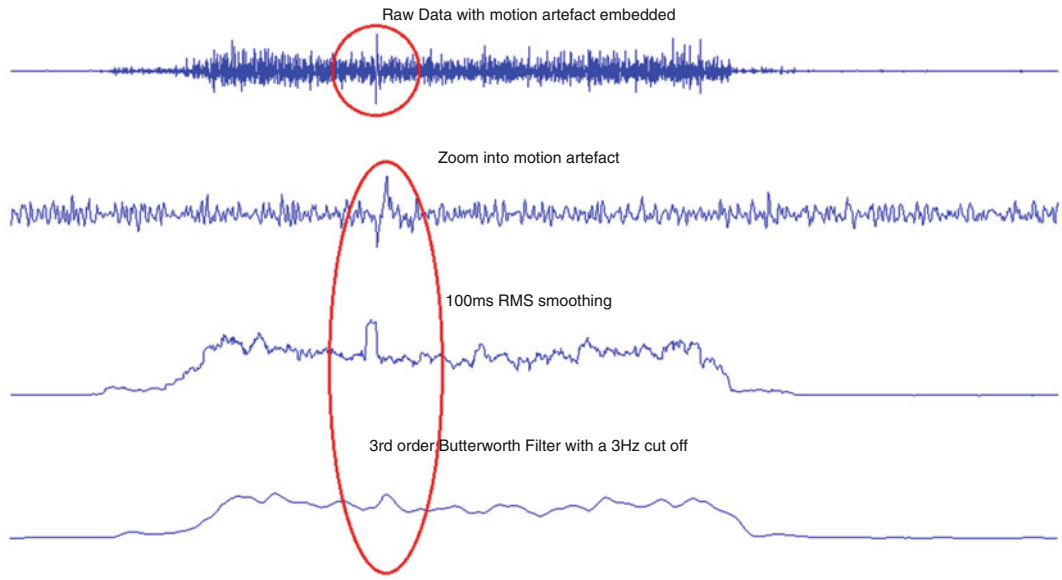

Fig. 12.2 Motion artifactcontamination within the EMG signal. The raw data displayed in the *top row* contains motion artifact as identified by the *small circle*. When the *x*-axis is expanded in the *second row*, the motion artifact becomes more evident. The *third* and *fourth rows* demonstrate how smoothing and filtering processes can mask the motion artifact, reinforcing the importance of careful inspection of the raw EMG signal

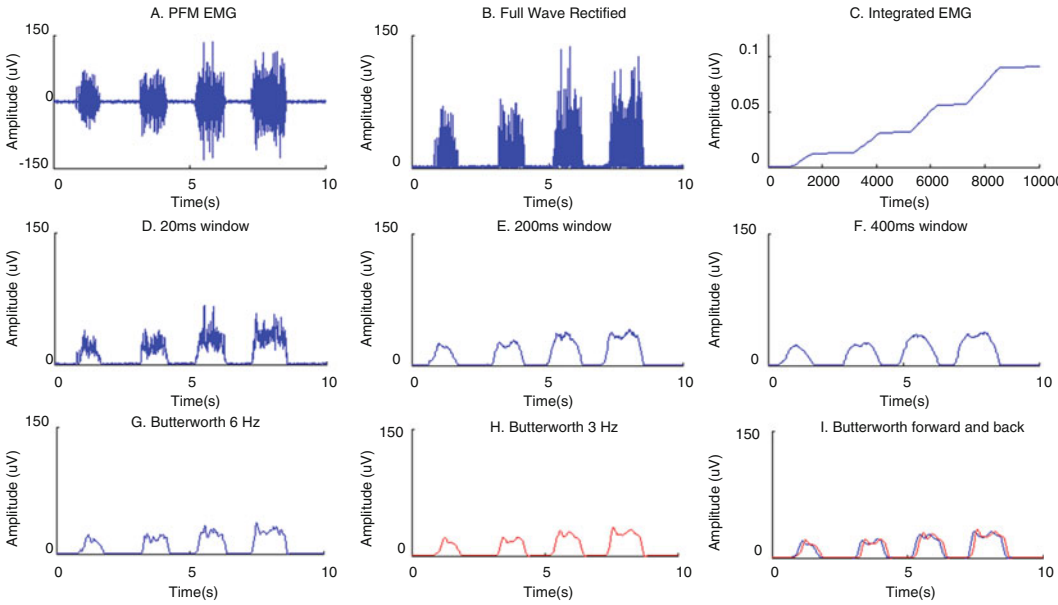

Fig. 12.3 Mathematical processing of EMG data. (**a**) Raw EMG data recorded from the pelvic floor muscles using a differential suction electrode. (**b**) EMG data full-wave rectified (making all the negative values positive). (**c**) Integrated EMG signal. (**d**–**f**) Sliding window technique used to smooth the data where the size of the window increases from 20 ms in (**d**), to 200 ms in (**e**), and 400 ms in (**f**). The signal becomes smoother the longer the window is. (**g**–**i**) Third-order Butterworth filters used to smooth the EMG data. (**g**) Filter cutoff value is 6 Hz. (**h**) Filter cutoff value is 3 Hz. (**i**) Panels (**g**) and (**h**) filter the data in a forward direction, (**i**) filters the data forwards (*red signal*) and then backwards (*blue signal*) to reduce the time shift created with the forward moving filter as can been seen by the separation of the two tracings

panel of Fig. 12.3 demonstrates the impact of different Butterworth filters on the EMG signal amplitude and timing. Using a "forward and back" approach (Fig. 12.3, panel i) restores the correct timing of the signal compared to the distortion caused by using a forward only filter (Fig. 12.3, panel h).

Although specific signal processing approaches are beyond the scope of this chapter, it is important to understand that the method of signal processing used will have a significant impact on amplitude and timing. Researchers are advised to develop a solid understanding of the signal acquisition and processing parameter requirements needed to optimize the quality of their EMG data. Comparing results between studies or between commercial devices that process EMG signals in different ways may lead to misinterpretation.

12.4 Reliability of EMG Signals in the Pelvic Floor

As discussed in Sect. 12.2, in conditions where the PFMs may be overactive, EMG signals have been recorded for a variety of purposes using a variety of approaches. Clinically, EMG has been used to provide biofeedback and in some cases used as an outcome measure to record change over time. As with any tool, the psychometric properties of each EMG outcome must be established and deemed adequate before it can be used as a means of diagnosis or to monitor disease progression or treatment effects.

In recent years, there has been some focus on the investigation of psychometric (i.e., validity and reliability) properties of surface EMG signal amplitudes recorded from the PFMs using a variety of intravaginal devices. The within- and between-day reliability of the EMG amplitude has been of particular interest. A summary of key studies on the psychometric properties of EMG signals recorded from the PFMs is presented in Table 12.2, and demonstrates that EMG data recorded using different intravaginal probes are relatively stable within the same session; however, EMG data are not stable between sessions or days. Further, and as explained in Sect. 12.3, due to the effect of electrode size, shape, material, and configuration [115] on EMG signal properties, EMG data recorded using one type of electrode should not be compared to EMG data recorded using another type of electrode. Similarly, the minimal clinically important difference (MCID) required to detect a true change will vary depending on the specific type of electrode used. As an example, the MCID in EMG amplitude recorded using a Femiscan™ vaginal probe was 10–15 μV, whereas it was 31–41 μV for the Periform® vaginal probe when the same sample of women were studied and the same signal processing was applied [56]. Grape et al. [122] reported similar results (MCID = 14.5 μV) for the Femiscan™ probe using an integrated EMG approach for processing.

To our knowledge, and as shown in Table 12.2, most of the pelvic floor EMG reliability studies in the literature have been done on asymptomatic women, with a majority of them using healthy, nulliparous women [41, 56, 108, 113, 122–128]. A study by Romanzi et al. [126] is one of the only reliability studies that included a mixed population of women ($n = 37$), where approximately half of participants reported urinary incontinence and one third of participants reported fecal incontinence. The authors reported a significant correlation between EMG amplitudes recorded on different days ($r = 0.86$, $p < 0.001$), but did not comment on differences between women who were continent and those who were incontinent and did not perform a comprehensive reliability assessment. Sound reliability investigations have not been reported in the overactive pelvic floor literature.

By definition, reliability is based on the proportion of total variability in a measure that can be attributed to error. Therefore, the greater the total variance observed, the higher the reliability coefficient will be. This is of particular interest when computing the reliability of EMG data recorded from the PFMs. Due to the reasons such as electrode location, tissue temperature, tissue moisture, anatomy and motor control strategies, EMG amplitudes tend to have very high variability between individuals and between days. This in itself can result in overestimation of reliability

Table 12.2 Summary table of PFM EMG reliability studies

Authors	Sample populations	EMG electrode type and configuration	Outcome measures	Psychometric outcomes
Loving et al. 2014 [125]	Healthy women with no history of CPP ($n=10$)	Thought Technology intravaginal probe with large electrodes located on opposite sides of the vaginal wall, configured to record one differential signal	Average EMG activity μV recorded	Between-rater Spearman's $r=1.0$, $p=0.00$ Within-rater Spearman's $r=0.9$, $p=0.00$
Voorham-van der Zalm et al. 2013 [113]	Men and women (19–72 yrs) with no history of urological or urogenital concerns ($n=20$)	MAPLe probe which incorporates a matrix of 24 small (roughly 3–4 mm^2) electrodes arranged anteriorly, posteriorly, left and right at 10 mm intervals from the probe tip. Electrode differential setup: unspecified. Electrodes used for the reliability analysis: unspecified	Mean EMG activity in μV recorded during rest, MVC, endurance tasks, cough, and Valsalva. It is not clear over what time intervals the mean values were calculated	Test–retest ICCs (no confidence intervals provided) at rest, during MVC and during an endurance task ranged from 0.61 to 0.91, 0.53 to 0.77, and 0.54 to 0.79, respectively, for anal recordings, and from 0.73 to 0.85, 0.60 to 0.77, and 0.67 to 0.74 for vaginal recordings
Keshwani and McLean, 2013 [108]	Healthy continent women (26 ± 7 yrs) with no history of CPP ($n=20$)	DSE incorporates a pair of small (1 mm^2) round electrodes separated by 10 mm suctioned to each side of the vaginal wall at the level of the levator ani	Smoothed peak EMG activity during MVC	Between-trial reliability
		The FemiScan™ is located intravaginally and configured to record differential signals from the right and left levator ani separately		DSE: ICC = 0.96–0.97
				FemiScan™: ICC = 0.94–0.97
				Between-day reliability
				DSE:
				ICC = 0.64–0.72
				SEM = 17.5–18.7 μV
				FemiScan™:
				ICC = 0.79–0.92
				SEM = 8.8–14.1 μV
Auchincloss and McLean, 2009 [56]	Healthy nulliparous continent women (30.0 ± 3.9 yrs) with no history of CPP ($n=12$)	FemiScan™ intravaginal electrode which incorporates paired electrodes on each side of the vagina; Periform® probe reconfigured to record monopolar signals from large electrodes located on each side of the vagina	Smoothed peak EMG activity during MVC	Between-trial ICC (3, 1) (CV)
				FemiScan™: 0.72–0.98 (8.5–14.2 %)
				Periform®: 0.87–0.96 (9.6–11.6 %)
				Between-day ICC (3,1) (SEM)
				FemiScan™: 0.41–0.57 (10.1–15.1 μV)
				Periform®: 0.61–0.76 (34.7–41.1 μV)

(continued)

Table 12.2 (continued)

Authors	Sample populations	EMG electrode type and configuration	Outcome measures	Psychometric outcomes
Grape et al. 2009 [122]	Nulliparous continent women (20–35 yrs) with no history of CPP ($n=17$)	FemiScan™ intravaginal electrode which incorporates paired electrode bars to record differential EMG signals separately from each side of the vaginal wall	EMG data from right and left sides averaged. Mean smoothed activity at rest, during MVC	Within-day reliability
				Tonic activity ICC = 0.88, MCID = 2.7 µV
				MVC ICC = 0.90, MCID = 12.5 µV
				Between-day reliability
				Tonic activity ICC = 0.86, MCID = 3.2 µV
				MVC ICC = 0.93, MCID = 14.5 µV
Thompson et al. 2006 [124]	Healthy women (20–55 yrs) ($n=5$)	Periform® probe	Mean smoothed EMG activity of three trials of 3 s contractions and three maximal straining efforts (Valsalva)	Between-day reliability
		Electrode differential set up: unspecified		ICC (SEM) = 0.98 (0.06)
				No units provided but suspected to be µV
				Results from MCV and Vasalva results seemed to have been combined
Engman et al. 2004 [37, 41]	Asymptomatic women (mean age 27.1 yrs, range 20–37 yrs) ($n=27$)	Thought Technology vaginal surface EMG sensor T6050 which incorporates one longitudinal bar on each side of the probe. Electrode differential setup: unspecified	Amplitudes provided as RMS values. Values assessed as per Glazer Protocol (*referenced in author's column*)	Pearson's correlation coefficients computed between days ranged from 0.33 to 0.90 (all but one $p=0.01$)
Aukee et al. 2002 [123]	Healthy women ($n=11$)	FemiScan™ probe recording differential signals separately from each side of the vaginal wall	Smoothed EMG amplitude recorded during MVC	Between trial: Spearman's rho = 0.84–0.97, $p<0.05$
Romanzi et al. 1999 [126]	Non-pregnant women (44.2 ± 4.2 yrs), half with urinary incontinence and one third with rectal incontinence ($n=37$)	Thought Technology intravaginal probe which incorporates one bar on each side of the vaginal wall to record one differential signal	Integrated sEMG root mean square amplitudes during PFM contractions	Between-day reliability
				EMG amplitude:
				Pearson's $r=0.86$, $p<0.001$
				EMG measures (unspecified) were correlated with digital palpation:
				Pearson's $r=0.45$–0.57, $p<0.01$
Thorp et al. 1996 [128]	Healthy non-pregnant nulliparous women (23.7 ± 2.8 yrs) ($n=12$)	Perineometer dumbbell-shaped anal and vaginal probes with one longitudinal electrode on each side. Electrode differential setup: unspecified	Rectal probe: 10 s hold and flick PFMs contractions	Between-day reliability
			Vaginal probe: 10 s hold and flick PFMs contractions	Rectal probe: CV = 0.61–0.71 $p<0.05$
				Vaginal probe: CV = 0.29–0.42 ns

(continued)

Table 12.2 (continued)

Authors	Sample populations	EMG electrode type and configuration	Outcome measures	Psychometric outcomes
Thorp et al. 1991 [179]	Healthy non-pregnant nulliparous women (24.1 ± 4.7 yrs) ($n = 8$ between day, $n = 36$ within day)	Perineometer dumbbell-shaped anal and vaginal probes with one longitudinal electrode on each side. Electrode differential setup: unspecified	Rectal probe: 10 s hold and flick PFMs contractions Vaginal probe: 10 s hold and flick PFMs contractions	Rectal probe and vaginal probe combined within-day reliability
				ICC = 0.89–0.90 $p < 0.05$
				Correlation coefficients (presumably Pearson's)
				Range of r-values = 0.40–0.85 $p < 0.01$
				Between-day reliability
				ICC = 0.76–0.97 no p-values
Gunnarsson & Mattiasson, 1994 [127]	Healthy women ($n = 20$)	Self-designed vaginal probe with longitudinal electrodes. Electrode differential setup: unspecified	Change from baseline during 2 s MVC with visual and auditory feedback	Pearson's correlation coefficients reproducibility $r = 0.92$

Summary of main findings of PFM EMG reliability studies. *CPP* chronic pelvic pain, *DSE* differential suction electrode, *EMG* electromyography, *RMS* root mean square, *MVC* maximum voluntary contraction, *yrs* years-old, *SEM* standard error measurement, *MAPLe* multiple array probe Leiden, *MCID* minimal clinically important difference, *ICC* intraclass correlation coefficient, *CV* coefficient of variation, *PFM(s)* pelvic floor muscle(s), *ns* not significant, *s* second(s), *n* number of participants

of PFM EMG. Further, a number of studies (Table 12.2) use Pearson's correlation coefficients (r) as a measure of reliability. Although correlation coefficients tell us the extent of the association between two sets of scores, they do not tell us about agreement; therefore, systematic error can easily be masked.

Overall, the literature suggests that although sEMG amplitudes recorded during phasic PFM activation may be useful to distinguish between cohorts and thus provide some evidence of the pathophysiology of PFM disorders, it is not a stable enough measure to use for diagnostic purposes.

12.4.1 Amplitude Normalization

One strategy to overcome the often poor test–retest (or between-day) reliability of EMG amplitude is to perform a normalization procedure. This approach has been used extensively in other EMG applications, particularly in biomechanics and motor control research. By having a person perform standardized reference contractions at the start or at the end of an EMG recording session, the data recorded during activities of interest can be reported as a proportion or percentage of that reference activation. In theory, by doing so, the data are more stable, as factors such as electrode location and even electrode type are no longer an issue. For example, if women perform an MVC with an intravaginal or intra-anal probe in place, and then data are acquired while they perform some other activity, then cohorts can be compared in terms of the proportion of maximal activation that was used to perform the task. Of course, this does not correct for cross talk errors if large, widely spaced electrodes are used.

Normalization to an MVChas been criticized in some literature [129] as MVC themselves can have a high degree of variability [56, 122], can be influenced by the type and speed of contraction [130, 131], may be systematically lower in patient groups as compared to control groups [47] due to pain, age differences, or other factors, and as such may induce systematic error into the normalized data. This problem is not unique to the PFMs. In other fields,

researchers have developed non-maximum reference contractions in order to normalize their EMG data [132–134].

As an example of the benefit of using a normalization procedure, Gentilcore-Saulnier et al. [40] found heightened bulbocavernosus EMG responses to a pressure stimulus at the vulvar vestibule in women with provoked vestibulodynia ($n=11$) as compared to pain-free controls ($n=11$). Women in the PVD group demonstrated higher superficial but not deep PFM responses to the stimulus, despite being comparable on their EMG activation during MVC. Although follow-up data after an intervention period were not control matched, they did show a reduction in the superficial PFM EMG response to the same stimulus applied after an intensive 12-week physiotherapy intervention that involved perineal stretching and massage techniques, dilator insertion, and motor control training. In these women, the normalized EMG response to the stimulus after the treatment was complete was not significantly different from that in the asymptomatic control group. Such comparisons would not have been appropriate without the use of the normalization approach.

12.4.2 Summary

Given the wide range of methodologies, the heterogeneity of the populations in studies, and confounding factors such as age, parity, and duration of the chronic pain condition [61, 135], which have all been reported to affect sEMG signals by lowering its amplitude, future studies should account for these variables as possible confounders. Furthermore, the psychometric properties of EMG outcomes suggest that it is not a useful tool for diagnosis [66]. The value of EMG lies more so in helping us to understand pathophysiology, as well as the acute (within-session) effectiveness of different treatment paradigms, whereby the EMG instrumentation remains in place throughout the session such that changes seen in an individual patient within a treatment session likely reflect true changes.

12.5 PFM EMG Biofeedback

One of the most common clinical uses of PFM EMG is in real-time biofeedback applications. In this section, we review the basic principles and goals of clinical EMG biofeedback, its effectiveness in treating the overactive pelvic floor, and we offer recommendations regarding best practice.

EMG biofeedback refers to the measuring, processing, and presenting of EMG signals recorded from relevant muscles back to patients in the form of auditory and/or visual feedback, such that the patient can develop greater awareness of and an increase in voluntary control over their muscles. Typically, PFM EMG biofeedback is provided while the patient is sitting or lying down, with a sensor in place (e.g., surface patch electrodes, intra-anal or intravaginal probe) which sends EMG signals through an amplifier system to be presented on a computer screen or on a small, stand-alone device. The EMG signals reflect the level of muscular activity recorded by the sensor and generally the extent of motor unit activation, as discussed previously.

Due to its conservative nature, relatively low cost and superiority to other forms of biofeedback such as intravaginal and intra-anal pressure [136], sEMG biofeedback has become integral to many multimodal conservative management approaches for PFM dysfunction. EMG biofeedback is the most common form of biofeedback used in the treatment of dyssynergic constipation [137] and vulvodynia [138], and it has been used successfully in different patient populations in whom an overactive pelvic floor is implicated [139] such as in vaginismus [64, 77, 140], chronic constipation [8, 137], overactive bladder syndrome [80, 98, 141], ED [142–144], and CP/CPPS [145–147].

The goal of EMG biofeedback is to enhance the patients' awareness of behaviors that are thought to contribute to their symptoms and to provide positive reinforcement of correct behaviors that may prevent, reduce, or alleviate symptoms [148]. The specific treatment objectives of PFM EMG biofeedback should vary according to the specific findings on clinical examination and

the needs of the patient. In patients with voiding dyssynergia [137] and overactive bladder [98, 149], EMG biofeedback is often used to promote relaxation through quieting tonic activation of the PFMs at rest and during straining activities [150, 151]. In patients with demonstrated weakness of the PFMs, EMG biofeedback during PFM contraction exercises may enhance activation amplitude or the speed of muscle activation [152]. There may be secondary benefits to the use of EMG biofeedback training, such as enhancing blood flow with repeated PFM contraction and/or the alleviation of pain and fear responses to vaginal penetration resulting from the repeated insertion of an intravaginal EMG probe.

12.5.1 Evidence for the Effectiveness of PFM EMG Biofeedback

Three recent Cochrane Systematic Reviews have all suggested that, despite a dearth of high quality randomized controlled trials, there is some evidence for the effectiveness of EMG biofeedback in the treatment of idiopathic constipation in adults [137], fecal incontinence [153], and urinary incontinence in women [152]. Most pertinent to the overactive pelvic floor, the literature reviewed by Woodward, Norton, and Chiarelli [137] reported that 70–80 % of patients with idiopathic constipation and/or dyssynergic voiding reported improvement with EMG biofeedback. The studies that contributed to this review were evaluated as having poor quality with high risk of bias; however, the synthesis suggests that biofeedback training alone [37, 74, 145, 146, 150, 154–158] or in conjunction [10, 40, 61, 140, 141, 159–163] with other therapies, may be more effective than no treatment or than the other therapies alone.

No Cochrane Systematic Review has reported on the evidence of effectiveness of EMG biofeedback in children with dyssynergic voiding; however, a recent review by Desantis et al. [76] included 27 studies on this approach. The review suggested that EMG biofeedback is an effective noninvasive treatment modality, producing a mean perceived improvement in the order of 80 %. Since that review was published, Kajbafzadeh et al. [74] compared EMG biofeedback and diet/behavioral modifications ($n=40$) to diet/behavioral modifications alone ($n=40$) in the treatment of dysfunctional elimination in children. The biofeedback was provided using patch electrodes applied to the perineum during biweekly sessions where children played a computer game that encouraged motor control of the PFMs. Children also performed exercises at home. At a 12-month follow-up visit, 68 % of children who had received the biofeedback treatment in combination with diet and behavioral modifications were symptom free, which was significantly higher than the cure rate of 40 % in children who received the diet/behavioral modification intervention alone. The authors concluded that EMG biofeedback contributed significantly in the resolution of constipation symptoms.

EMG biofeedback has also become a mainstay of treatment for vulvodynia, where 89 % of therapists in the United States reported using EMG biofeedback [138], with a main goal of promoting PFM relaxation. Similarly to the systematic reviews published for other populations, many of the randomized controlled trials performed on the use of EMG biofeedback in women with vulvodynia have major methodological flaws or carry significant risk of bias [10, 37, 40, 140, 154, 155, 163–165]. According to numerous reviews, and despite these flaws, again there is likely some evidence for the effectiveness of EMG biofeedback in this population [10, 35, 139, 158, 166–172].

EMG biofeedback has also been used in the treatment of chronic nonbacterial prostatitis and/or CP/CPPS [146, 150, 156] and erectile dysfunction [144]. Good quality trials describing clear treatment protocols are scarce in these populations as well, although a recent trial of men with chronic nonbacterial prostatitis reported significant improvements in the National Institute of Health Chronic Prostatitis Symptom Index in 97 % of participants (29 % reporting a significant improvement of over 5 points) with 87 % reporting an improvement in the pain subscale [146].

12.5.2 Clinical Application of PFM EMG Biofeedback

The success of an EMG biofeedback intervention likely depends on several factors, including the proper application of the technology, patient motivation, ease of application, and treatment parameters.

The proper application of the EMG technology is essential to generate meaningful signals. Clinicians are encouraged to become familiar with their system specifications and to use strategies to reduce noise, cross talk, and motion artifact during biofeedback sessions as discussed in detail throughout this chapter.

Although it is tempting to use signal amplitudes acquired during EMG biofeedback sessions in order to monitor patient improvement over the course of therapy, this should be avoided. As discussed in Sect. 12.4, EMG signal amplitudes are highly variable between days and therefore an increase in EMG amplitude between sessions does not necessarily reflect higher PFM activity, and similarly, a reduction in tonic EMG activation amplitudes between sessions does not necessarily reflect improved relaxation [56, 150, 151]. Instead, patients and therapists should focus on changes in EMG amplitudes within treatment sessions, where the reliability of recorded amplitudes is relatively high. This might include, during the course of a treatment session, for example, the absence of paradoxical contractions during straining, improved speed during quick phasic contractions or increased recruitment capacity during sustained and maximal phasic contractions.

Progress with treatment is best monitored using patient-oriented outcomes, including, for example, patient perceived improvement [137], reductions in perceived pain [173, 174], reports of improved frequency of defecation in patients with dyssynergic voiding [76], reduced frequency of voiding in patients with overactive bladder [149], and perceived sexual function [32]. Complementary tools to measure outcomes may include the evaluation of changes in muscle tone, strength, or motor control as discussed in Chap. 11. Refer to Table 12.3 offers simple solutions on how to overcome common challenges associated with using EMG biofeedback.

Overall, successful interventions with EMG involve (1) the use of within-session, rather than between-day, changes to monitor improvements in daily performance within the same subject [56, 150, 159], (2) the use of validated and reliable outcome measures (e.g., sensory thresholds or perceived sexual function) rather than EMG data [157], (3) the use of complementary modalities to evaluate passive tone and/or strength, which are not measured directly by EMG (e.g., manual testing, dynamometry), and (4) avoidance of the use of EMG amplitudes for diagnosis.

Patient motivation and compliance with EMG biofeedback can be a major problem [144, 146, 150, 155, 157] and may reflect motivation with exercise in general or an aversion to using the biofeedback technology during exercise. Danielsson et al. [157] specifically reported that a lack of motivation was the main obstacle to compliance with EMG biofeedback training. Portable systems are available such that patients can use them at home, which may enhance compliance with biofeedback-assisted exercises if patients find the sessions interesting, or perhaps more effective, by having proper proprioceptive and visual cues in real time. Recent advances in the development of virtual reality applications may assist with motivation and compliance. For example, children's games have been developed [74] in an effort to add interest and motivation. Similar applications are also now available for women with PFM dysfunction [175].

When EMG is used for biofeedback, the standards for electrode configuration may be relaxed when necessary. For example, for cultural or ethical reasons, it may not always be possible to use intravaginal or intra-anal electrodes, and therefore surface patch electrodes may be chosen instead. Although it may not be necessary to record signals from both the right and left sides of the PFMs [122, 176, 177], electrode configurations should nonetheless follow the general principles of EMG recordings whenever possible, such that both electrodes should be placed on the same side of the vagina, anus, or perineum and in

Table 12.3 Basic recommendations for best EMG data acquisition

Factors affecting EMG data	Effect on EMG data	Recommendations to minimize impact
Electrode characteristics		
Electrode size and geometry [40, 113, 115, 117, 176, 180, 181]	Larger electrode record more EMG activity that might not come from the PFMs (cross talk)	Large electrodes are best for electrical stimulation and smaller, paired, electrodes for data recording
	Electrodes positioned close together (1 cm apart) will pick up less cross talk	Choose probes or place surface electrodes in pairs on same side of muscle and along the line of action of the muscle, approximately 1 cm apart
	Circumferential probe electrodes do not differentiate between right and left PFMs	Assess right and left sides separately
		Assess deep and superficial PFMs separately
Location [182]	Electrodes placed on the perineum are nonspecific	Choose intravaginal or intra-anal recordings when possible, considering ethical and cultural issues as well as comfort. Surface patch electrodes should be used with both electrodes in the pair placed on the same side of the perineum
Configuration [107, 108, 183]	Recordings made from paired electrodes where one electrode is on each side of an intravaginal probe results in data that represents the difference between the sides	Differential recordings are best, especially in research environments
		Monopolar recordings may be adequate for EMG biofeedback if care is taken to minimize cross talk and to ensure proper performance of the task by other means
Data acquisition		
Noise [56, 113, 117, 182]	Noise in the EMG signal has numerous sources (leads, environment, motion artifact) and can create errors in the evaluation of EMG amplitude and timing	Minimize environmental noise (turn off other sources of electricity), use short leads, and twist all leads together. Use adhesive to minimize movement of surface electrode relative to the skin
	If using adhesive electrodes on the perineum: consider that electrodes might touch during a contraction along with the inward lift of the perineum, causing shorting or artifact	Inspect raw EMG for noise, beware of tasks such as cough and Valsalva, which are known to cause artifact
Cross talk [108, 117]	EMG activity form surrounding muscles can mimic PFM activity: When recording using a probe, surrounding hip musculature contracting at more than 25 % of MVC would pollute the signal	Minimize co-contractions of hip musculature and be careful when recording tasks that require walking, jumping, etc.
	Probes with circumferential electrodes record more cross talk than those with longitudinal electrodes	Use small, closely spaced electrodes in a differential configuration

Main solutions for issues with EMG electrode setup and data acquisition common to both clinical and research settings. *MVC* maximum voluntary contraction, *EMG* electromyography, *PFM(s)* pelvic floor muscle(s), *cm* centimeter

line with the muscle fibers of interest whenever possible. When surface patch electrodes are used over the perineum, clinicians should be aware that their signals are at especially high risk of contamination. Clinicians should carefully assess the quality of the contraction and instruct patients to minimize co-contraction of nearby muscles to limit cross talk contamination.

EMG biofeedback treatment regimens are extremely varied and, to date, there are no best practice guidelines. Study protocols by Starr et al. [162] and Gentilcore-Saulnier et al. [40] as

well as recent reviews by Rosenbaum [158], Pedraza [178], and De Andres [169] describe how patient-centered treatment protocols, based on the specific clinical findings of each patient, can be implemented for treating various PFM dysfunctions. These protocols may serve as a good starting point for the development of effective treatment regimens.

There is mounting evidence that certain factors may enhance the success of treatment programs for PFM dysfunction. Program success may be enhanced by: (1) completing more than two sets of PFM exercises daily, (2) using a multimodal treatment approach that includes education, physical examination, a variety or exercises, as well as a psychological approach [149], (3) having a longer overall duration of the treatment (>3 months) [163], (4) attending at least three individualized treatment sessions [162], and (5) having greater compliance with the prescribed exercises [162]. These principles likely hold true with EMG biofeedback-enhanced PFM training.

12.5.3 Summary of the Use of EMG as a Biofeedback Modality

EMG biofeedback has been widely used in the treatment of the overactive pelvic floor, and there is some evidence of success. If applied and interpreted appropriately, EMG can be a powerful and effective tool to track PFM contractile behavior in real time, and to facilitate the achievement of treatment objectives such as promoting relaxation, enhancing strength or motor control. Due to the complexity of this technology, informed clinicians will achieve the most meaningful results for their patients.

12.6 Conclusions

When used appropriately, EMG can provide useful insight into the normal neuromuscular function of the PFMs and neuromotor changes associated with different urogenital and anorectal disorders, can provide insight into inappropriate motor control patterns, and can be used as a training tool to reestablish normal function. Due to its poor reliability and inherent variability between instrumentation systems and data processing approaches, EMG should not be used as a diagnostic tool. When using EMG as a biofeedback tool, it is important to remember that signal amplitude is influenced by many factors, many of which are uncontrollable and highly variable between days. Therefore, the quality of the PFM contraction or relaxation should be, whenever possible, confirmed concurrently through inspection and palpation, and EMG amplitude values should not be used to monitor progress over the course of treatment.

After reading this chapter, it is our hope that clinicians and researchers alike will have gained a thorough understanding of what is known about neuromuscular involvement in conditions associated with an overactive pelvic floor, will be able to identify strengths and limitations in the literature on PFM dysfunction, will understand the appropriate application of EMG, and will recognize the strengths and limitations of EMG in their respective practice in order to properly apply and to accurately interpret the electromyogram.

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13 Female Pelvic Floor Imaging with Emphasis on the Overactive Pelvic Floor

Vered H. Eisenberg

13.1 Introduction

Chronic pelvic pain, frequently associated with pelvic floor dysfunction, is an underdiagnosed and often untreated condition, resulting in years of suffering and decreased quality of life. It seems reasonable to expect that imaging should be part of the evaluation in these patients, but in overactive pelvic floor this is not yet the case.

Pelvic floor imaging is a rapidly evolving topic in urogynecology with the advent of new high-resolution techniques. The increasing availability of ultrasound in the clinical setting, and recent developments of three- and four-dimensional (3D and 4D) ultrasound, has brought new interest in this modality as a main clinical tool in the understanding of pelvic floor anatomy and function [1, 2] Both ultrasound (US) and magnetic resonance imaging (MRI) have been used to evaluate bladder neck (BN) mobility, urethral sphincter volume, pelvic organ descent, morphology of the levator ani, and diameters of the genital hiatus. Imaging has also been used as an adjunct to physical examination in the management of patients with urinary incontinence (UI) or pelvic organ prolapse (POP) and as a research tool to investigate the pathophysiology of both conditions [3]. In clinical practice most examination rooms have an US machine available and it is frequently used as an integral part of the physical examination.

The availability of high-energy magnets (up to 3 T) has increased the accuracy of nuclear MRI. However, MRI suffers from insufficient functional study capabilities because the patient must lie supine in the MRI coil making levator ani functional studies difficult due to conformational changes to the levator ani that occur when upright or partly upright. Dynamic MRI is used in some settings, but suffers from limited availability, much higher costs, and decreased patient acceptability [4].

Transperineal or translabial ultrasound is widely used in urogynecological assessment, with 3D and 4D imaging techniques enhancing its utilization in both research and clinical practice. Several hundreds of papers have been published worldwide allowing the translation of basic clinical research into standard clinical practice. Currently, there is a lack of standardization in some aspects of pelvic floor ultrasound; however, this is a work in progress that will likely be achieved with time and effort. Despite this, knowledge of pelvic ultrasonography is not widespread and the different specialists involved in the care of these patients limit assessment modalities to their area of interest.

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Imaging of the pelvic floor in women with pelvic floor dysfunction can encompass various areas such as the urethral sphincter, urethral and bladder neck (BN) morphology, BN and urethral position and mobility, morphology of the vaginal walls and vault, morphology of the anal canal, and the morphology and function of the levator ani/puborectalis sling muscle complex. Pelvic floor imaging may be influenced by a number of factors such as patient position, degree of bladder fullness, provocative maneuvers such as Valsalva and pelvic floor contraction. Furthermore, the process depends on operator training and experience, and equipment quality, all of which raise the issue of defining and standardization of the available methodologies [3].

This chapter will provide a review of the main uses of imaging in the evaluation of pelvic floor dysfunction, with an emphasis on pelvic floor ultrasound. The available literature regarding overactive pelvic floor-related disorders, including overactive bladder, chronic pelvic pain syndromes, and provoked vestibulodynia (PVD), will be reviewed. A description of ultrasound will be provided as well, in the context of the evaluation of urinary incontinence, POP, and postoperative audit of slings and meshes, all of which may coexist. Additional description of posterior compartment evaluation is provided in Chap. 9.

13.2 Ultrasound Techniques

The currently available ultrasound techniques are summarized in Table 13.1. The main groups who operate in this field have produced a standardization paper in 2011 [5]. Evaluation of the complex pelvic floor anatomy may at times require more than one modality, as all may contribute complementary information.

13.2.1 Transperineal Ultrasound

Standard requirements for two-dimensional (2D) translabial or transperineal ultrasound (TPUS) of the pelvic floor include a B-mode ultrasound machine with cine-loop function, convex transducers of 3–6 MHz, and a field of view of at least 70°. A midsagittal view is obtained by placing a transducer on the perineum [2, 6, 7] (Fig. 13.1). For hygienic reasons the transducer is covered with a glove, condom, or thin plastic wrap. Powdered

Table 13.1 Types of pelvic floor ultrasound

Modality	Probe	Imaging	Dynamic study	Compartment	Levator ani and perineal muscles assessment
Transperineal—2D	Convex 3–6 MHz	Sagittal, coronal	Yes	All	Yes
Transperineal—3D	Convex 4–8 MHz	Axial, multiplanar, tomographic	No	All	Yes
Transperineal—4D	Convex 4–8 MHz	Axial, multiplanar, tomographic	Yes	All	Yes
Transvaginal—2D	Biplanar 5–12 MHz	Sagittal, axial	Yes	A and P	No
Transvaginal—3D	Biplanar 5–12 MHz (180° view)	Multiplanar	No	A and P	No
Transvaginal—3D	360° view 9–16 MHz	Multiplanar	No	A and P	Yes
Endoanal—3D	360° view 9–16 MHz	Multiplanar	No	Anal sphincters	Perineal muscles only

2D two dimensional, *3D* three dimensional, *4D* four dimensional, *A* anterior compartment, *P* posterior compartment

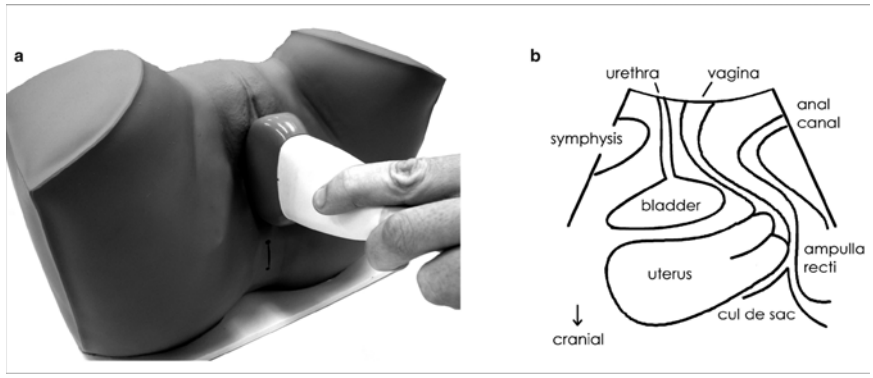

Fig. 13.1 Model demonstrating transperineal transducer placement (**a**) and schematic diagram showing resulting midsagittal view on two-dimensional transperineal ultrasound (TPUS) (**b**). With permission from Dietz HP. Pelvic floor ultrasound: a review. Am J Obstet Gynecol. 2010; 202(4): 321–334 © Elsevier 2010 [2]

gloves can impair imaging quality owing to reverberations and should be avoided. Sterilization (as for vaginal or endorectal transducers) is considered unnecessary; mechanical cleaning and alcoholic wipes can be used for disinfection. The patient lies in the dorsal lithotomy position with the hips flexed and slightly abducted. Pelvic tilt can be improved by bringing the heels closer to the buttocks and by moving the hips towards the heels. The patient is asked to void before the examination in order to allow a full Valsalva maneuver, but bowel emptying is not mandatory. A full rectum can impair diagnostic accuracy, and at times the assessment has to be repeated after bowel emptying. Parting of the labia can improve image quality, especially if the vulva is hypertrophic or hirsute. Vaginal scar tissue and modern mesh implants may impair visibility. Obesity is rarely a problem. Imaging is performed at rest, maximal Valsalva maneuver, and maximal pelvic floor muscle contraction (PFMC). Pelvic floor ultrasound provides both static and dynamic imaging and allows anatomical and functional assessment of the different compartments. It is currently used in specialized urogynecology centers, but can be implemented more widely in any outpatient clinical setting without any discomfort to the patient. An important advantage of this methodology in the context of pelvic pain is that it is noninvasive and more widely acceptable for the patient.

The standard midsagittal view includes from anterior to posterior: the symphysis pubis with the urethra and bladder neck immediately dorsally, vagina and cervix medially and rectum and anal canal posteriorly (Fig. 13.1). Further posteriorly to the anorectal junction, the central portion of the levator plate (or the puborectalis sling) is seen as a hyperechoic structure [2]. Parasagittal or transverse views allow additional information, such as visualization of the urethra, puborectalis muscle attachment, and depiction of mesh implants. Confounders that block the view may be as a result of shadowing from vaginal prolapse, the pubic bone, or a full rectum, particularly in the presence of a rectocele obscuring the posterior compartment, or the vaginal apex.

3D and 4D TPUS is performed with 3D transabdominal probes developed for obstetric imaging (such as RAB 8-4, GE Healthcare Ultrasound, Milwaukee, WI, USA; AVV 531, Hitachi Medical Systems, Tokyo, Japan; V 8-4, Philips Ultrasound, Bothell, WA, USA; 3D 4–7 EK, Medison, Seoul, South Korea). These transducers combine an electronic curved array 4–8 MHz with mechanical sector technology, which allows a fast sweep through the field of view (Fig. 13.2). This technique enables the creation of tomographic multislice imaging in a chosen plane giving a picture similar to CT scanning (without any radiation), called tomographic ultrasound imaging (TUI) (Fig. 13.3). TUI enables assessment of the entire puborectalis muscle and its attachment to the pubic rami, to measure the diameter and area of the levator hiatus, and to determine the degree of hiatal distension on Valsalva maneuver [8].

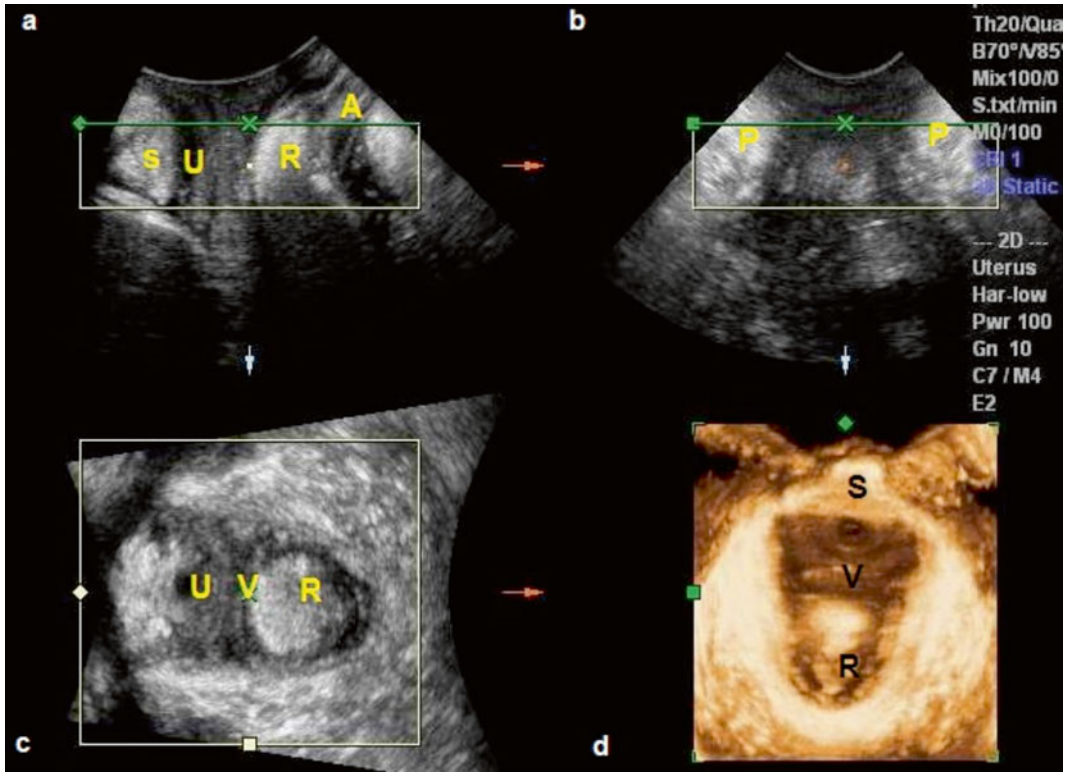

Fig. 13.2 Standard acquisition screen of 3D TPUS: Voluson GE series ultrasound with a RAB 8-4-MHz transducer (GE Healthcare): midsagittal plane (**a**), coronal plane (**b**), axial plane (**c**), and a rendered axial plane (**d**). *A* anal canal, *P* puborectalis muscle, *R* rectal ampulla, *S* symphysis pubis, *U* urethra, *V* vagina

4D imaging allows real-time acquisition of volume ultrasound data, which can be visualized in orthogonal planes or rendered volumes. The 3D data is archived as a cine loop that allows maneuvers such as Valsalva and PFMC. The methodology also allows data storage and offline analysis with the help of dedicated software (4D view, GE Healthcare Ultrasound, Milwaukee, WI, USA, and others) [9].

4D ultrasound imaging of the pelvic floor has significantly enhanced the clinical approach to complex urogynecological conditions, and is rapidly becoming more available in tertiary facilities. Its main advantages are ease of use, availability within the gynecological clinic, lack of radiation or contrast media, and performance by the patient's clinician, all of which make it superior to MRI [10]. It is particularly useful for assessing the dynamic function of the pelvic floor complex, and can provide an enhanced view of soft tissues, particularly the levator ani complex [2]. Furthermore, ultrasound real-time imaging allows the operator to identify and control for common confounders in the clinical examination, such as bladder and rectal filling [11], suboptimal performance of Valsalva maneuver, often seen as levator muscle co-activation [12], and duration of Valsalva maneuver [11]. In order to enhance exam yield it is recommended to ensure bladder (and possibly rectal) emptying, a sufficiently long Valsalva (at least 5 s) a maximal Valsalva effort, and relaxation of the levator ani [11]. Ultrasound allows visual biofeedback, which greatly facilitates achieving an adequate Valsalva maneuver and PFMC.

While in the past MRI was considered the standard investigation for detecting PFM injuries resulting from vaginal birth, high-resolution 3D and 4D ultrasound images can be equally accurate in

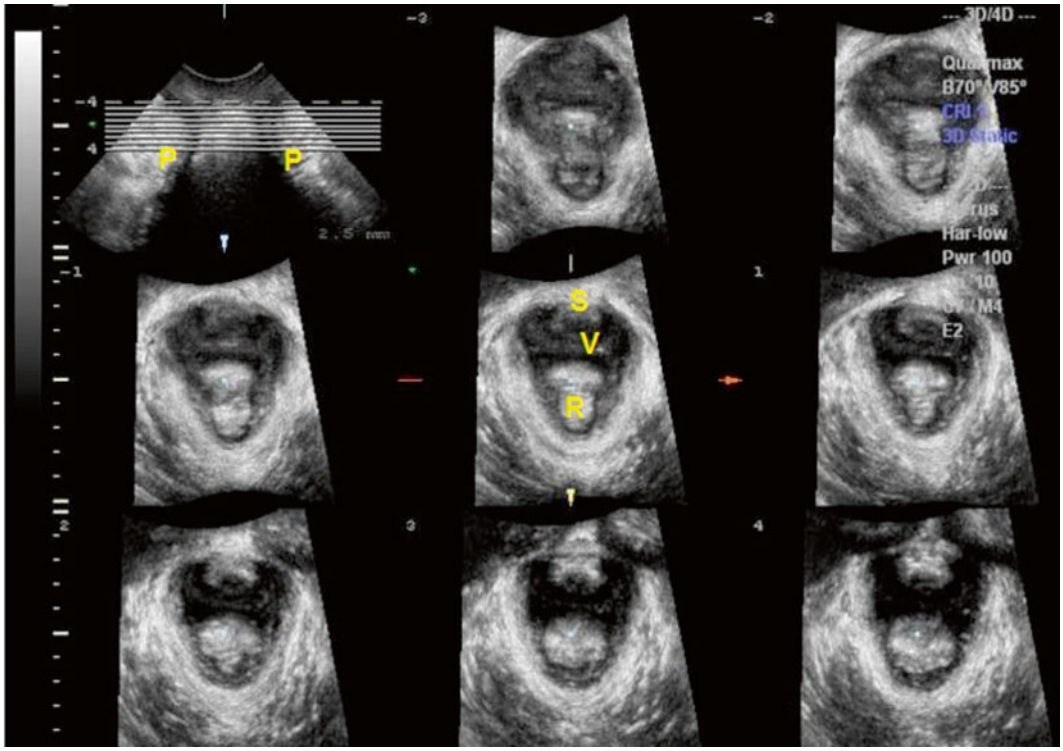

Fig. 13.3 Levator ani assessment on multislice/tomographic ultrasound imaging (TUI) using a Voluson GE Expert machine equipped with a RAB 8-4-MHz transducer (GE Healthcare). *P* puborectalis muscle, *R* rectal ampulla, *S* symphysis pubis, *V* vagina

demonstrating anatomical and functional defects which can result in prolapse and incontinence. Some of these defects may be missed during a clinical examination, which could lead to suboptimal surgical repair and need for repeat surgical procedures [13]. Translabial ultrasound, especially 4D real-time imaging, has the major advantage of providing a global view of the entire pelvic floor, from the symphysis to the anorectum, and includes the lower aspects of the levator ani muscle. As a result, the modality is optimally suited for interdisciplinary assessment and communication [7].

13.2.2 Transvaginal Ultrasound

Transvaginal ultrasound (TVUS) is performed in a position similar to TPUS. There are several available probes: electronic biplanar 5–12 MHz, with high frequency (9–16 MHz), mechanical probes rotating 360°, or radial electronic probes (type 8848, B-K Medical, Herlev, type 2050, B-K Medical, type AR 54 AW, 5–10 MHz, Hitachi Medical Systems Denmark) [1]. Whichever probe is used, it is important to keep it stable avoiding excessive pressure on adjacent structures, which may distort anatomy. The choice of this modality as opposed to TPUS mentioned above will be dependent on operator experience and preference or specific indications as described in Table 13.2.

Biplanar electronic probes allow 2D sagittal and axial sectional imaging of the anterior and posterior compartments. As for TPUS, imaging is performed at rest, maximal Valsalva, and PFMC. This methodology also allows the use of color Doppler to image the vascular pattern of pelvic floor structures [1]. 3D images may be obtained by adding an 8848 transducer to the

Table 13.2 Indications for pelvic floor ultrasound

Indication	
Anterior compartment	Urinary incontinence
	Voiding dysfunction
	Bladder pain syndromes/ interstitial cystitis
	Recurrent urinary tract infection
Posterior compartment	Fecal incontinence
	Obstetric anal sphincter trauma (OASIS)
Pelvic organ prolapse	Cystocele, uterine prolapse, rectocele, enterocele
Obstructed defecation	Chronic constipation, straining at stool, incomplete bowel emptying, vaginal or perineal digitation for bowel emptying, intussusceptions
Pain syndromes	Pelvic, vaginal, anal pain, endometriosis
Suspected pelvic floor dyssynergy	Hypertonic disorders
Follow-up after pelvic floor surgery	Midurethral slings, support meshes, sacrocolpopexy

external 180° rotation mover allowing sagittal, axial, and coronal and any desired oblique sectional images to be obtained [1]. The radial electronic probe and the rotational mechanical probe allow a 360° of the pelvic floor. A 3D volume can be acquired for real-time manipulation, or archiving for offline analysis with dedicated software.

13.2.3 Endoanal Ultrasound

Endoanal ultrasound (EAUS) is performed with a high multifrequency, 360° rotational mechanical probe or a radial electronic probe, as described for TVUS. The patient is placed in a dorsal lithotomy, left lateral, or prone position. In all situations, the transducer is rotated so that the anterior aspect of the anal canal is superior at the 12 o'clock position. 3D acquisition is achieved with the mechanical rotational transducer allowing a thorough investigation, volume manipulation, and measurements in any plane [14]. This methodology is more widely described in Chap. 12.

13.3 Methodology

13.3.1 Anterior Compartment Evaluation

Anterior compartment assessment is easily performed with two-dimensional TPUS or TVS [15, 16] (Fig. 13.1). Measurements are performed in the midsagittal plane with the patient at rest, Valsalva, and PFMC. Bladder wall or detrusor wall thickness usually measures up to 5 mm [17–19]. Post-void residual bladder volume usually does not exceed 50 mL in normal circumstances [19]. Perineal mobility can be measured relative to the lower margin of the symphysis pubis, specifically the bladder–symphysis distance. This measurement allows reproducible assessment of the position and mobility of the bladder neck [20], by calculating the difference between values obtained at rest and on Valsalva. There is no definition of "normal" for bladder neck descent although a cut-off of 30 mm has been proposed, which is still below the 95th percentile of findings in young nulligravid continent women [21]. Measurements of 1.2–40.2 mm (mean, 17.3 mm) have been obtained in a group of 106 stress-continent nulligravid young women of 18–23 years of age. Various confounders such as bladder volume, patient position, and urethral catheterization have been shown to influence measurements. It is essential to abstain from exertion of undue pressure on the perineum so as to allow full development of pelvic organ descent. Increased bladder neck descent may result from congenital or environmental causes or both and is mainly linked with birth trauma and prolonged second stage of labor [22, 23].

The urethral length can be measured from the bladder neck to the external urethral orifice. The retrovesical angle is the angle between the posterior wall of the bladder and the longitudinal axis of the urethra, which usually measures approximately 90–120°. Urethral rotation is the change in angle measured between the proximal urethra and central symphyseal axis on Valsalva as compared with the angle at rest. The extent of rotation can be measured by comparing the angle of inclination between the proximal urethra and any other fixed axis [24]. The urethral rhabdosphincter can

be measured with both 3D-TVS with the biplane electronic transducer and 3D TPUS and all planes can be evaluated including width, length, thickness, and lumen volume [25]. Urethral funneling can be seen in the urethrovesical junction both in women with stress urinary incontinence (SUI) and in asymptomatic patients [26]. Funneling of the internal urethral meatus may be observed on Valsalva and is often associated with leakage.

Descent of the most inferior aspect of a cystocele relative to the symphysis pubis on Valsalva assists in measuring anterior compartment prolapse, which will be discussed later.

13.3.2 Central Compartment Evaluation

As with the anterior compartment, assessment of the central compartment is performed with two-dimensional TPUS (Fig. 13.1). When positioned in the midsagittal section, the cervix and uterus are isoechoic, and may cause an acoustic shadow behind (above) it. The uterine body is seen proximal to the cervix and can be seen as enlarged, retro- or anteverted. Dynamic two-dimensional TPUS allows evaluation of uterine descent or prolapse. TVS is less useful for imaging of the central compartment because the vaginal probe impedes descent of the uterus or vaginal vault.

13.3.3 Posterior Compartment

The anal canal is generally imaged by EAUS [14], one of the cornerstones of a full colorectal diagnostic work-up. The anal canal is divided into three levels of assessment in the axial plane: (1) The upper level corresponds to the hyperechoic sling of the puborectalis muscle and the concentric hypo echoic ring of the internal anal sphincter (IAS); (2) The middle level to the complete ring of the external anal sphincter (EAS), the conjoined longitudinal layer, the complete ring of the IAS and the deep and superficial transverse perineal muscles are visualized; (3) The lower level corresponds to the subcutaneous part of the EAS. At the upper end of the anal canal the puborectalis muscle anchors the anal sphincter complex to the pubic rami (Fig. 13.4).

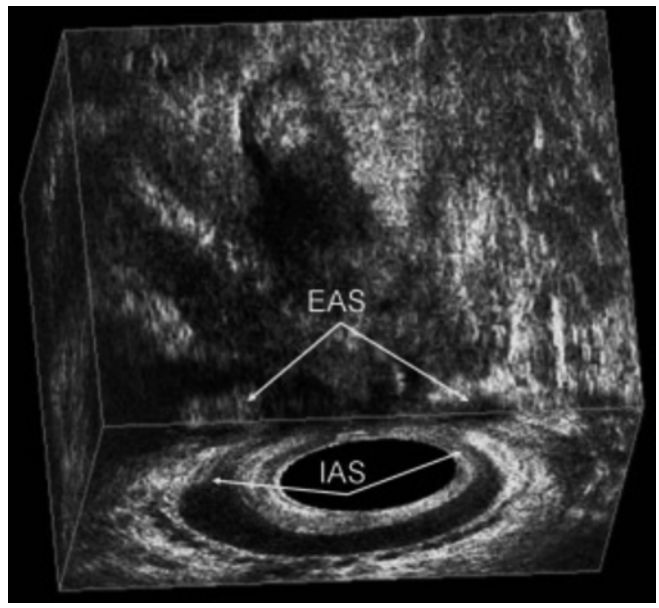

Fig. 13.4 Three-dimensional endoanal ultrasound using a 360° rotational transducer (type 2050, 9–16 MHz, B-K Medical). In the coronal plane a combined anterior defect of the external sphincter (EAS) from 11 to 1 o'clock and of the internal sphincter (IAS) from 9 to 1 o'clock positions can be seen. *With permission from Santoro GA, Wieczorek AP, Dietz HP, Mellgren A, Sultan AH, Shobeiri A,* et al. *State of the art: an integrated approach to pelvic floor ultrasonography. Ultrasound Obstet Gynecol. 2011; 37 (4): 381–396. © John Wiley and Sons 2011* [1]

The most common clinical indication for EAUS is the assessment of sphincter integrity following obstetric trauma. Obstetric anal injury (OASIS) may occur as a result of perineal trauma or extension of an episiotomy during childbirth. Following repair, the healing process is characterized by fibrosis, which appears as a low echogenic band on ultrasound. These perineal tears may involve either or both the EAS and IAS. The incidence of anal sphincter defects following vaginal delivery detected by endoanal ultrasonography is 30 % for primiparae and 9 % in multiparae [27]. Evaluation of the IAS and EAS with EAUS is still considered to be the gold standard in investigating patients with obstetric anal sphincter injuries and anal incontinence, especially with the advent of 3D technology, which allows imaging in the sagittal and coronal planes [28]. Up to 35 % of women after OASIS will have sonographic findings, which may have been missed by clinical examination, with either EAUS or TPUS [29, 30]. Clinical evaluation of the anal sphincter complex is insufficiently reliable, thus necessitating the use of well-established imaging modalities such as EAUS or TPUS, both of which are highly operator dependent.

Two-dimensional TPUS and 2D-TVS with a biplane transducer provide additional information on the posterior compartment [1, 31]. The main advantage of both of these techniques is that they allow access to the midsagittal plane, which enables visualization of the integrity of the perineal body, the integrity of the rectovaginal septum, measurement of the anorectal angle, and dynamic assessment. During Valsalva it is possible to visualize descent of an enterocele, diagnose a rectocele, and to evaluate movement of the puborectalis muscle and anorectal angle to diagnose pelvic floor dyssynergy. As explained before, TPUS is more likely to allow full development of prolapse because the TVS probe may hinder complete descent. Furthermore, TPUS includes a fixed point of reference, the lower symphyseal margin, in the field of view.

Ultrasound is also useful for the assessment of anorectal dysfunction and over the past decade gynecologists, colorectal surgeons, and gastroenterologists have developed slightly different diagnostic approaches and definitions [7]. Translabial ultrasound is a suitable screening tool for conditions involving the posterior compartment, and its results are comparable to defecation proctography [32–34]. While actual defecation is not necessary for the diagnosis of rectocele, enterocele, rectal intussusception, or prolapse, a Valsalva maneuver may be sufficient. An anterior rectocele can be diagnosed on ultrasound using a depth of 10 mm as a diagnostic cutoff, with an incidence of up to 50 % in an urogynecological clinic [7]. Such patients may have symptoms of obstructed defecation, such as straining at stool, incomplete bowel emptying, and vaginal digitation, although they may also be asymptomatic, and the association between bowel symptoms and imaging findings is considered to be weak [35, 36]. This may be due to variable diagnostic criteria or concomitant other pathology such as intussusceptions, perineal hyper mobility, or anismus [7]. Childbirth may be responsible for some rectoceles, or for the enlargement of a preexisting rectocele, through disruption of the rectovaginal septum during vaginal delivery, but rectoceles have been found in approximately 10 % of young nulliparous women and are associated with BMI [37, 38]. In women with obstructed defecation, ultrasound imaging allows for visual biofeedback in order to achieve behavioral modification [7].

13.3.4 Functional Assessment

13.3.4.1 Valsalva

Ultrasound is very useful in the anatomical and functional assessment of the pelvic floor musculature. The Valsalva maneuver, namely forced expiration against a closed glottis and contracted diaphragm and abdominal wall, is used to reveal symptoms and signs of POP and to mimic defecation. A Valsalva maneuver results in dorsocaudal displacement of pelvic organs that can be quantified using a system of coordinates with the inferoposterior symphyseal margin as the reference point. The increased intra-abdominal pressure will unfold compartment prolapse. In the axial plane, the levator hiatus becomes distended, and the posterior aspect of the levator ani is displaced caudally, resulting in varying degrees of perineal descent. It is important to allow the transducer to move with

the tissues, avoiding pressure on the perineum that would prevent full development of any organ prolapse and/or perineal descent [7]. There are several confounders that affect the efficacy of the Valsalva maneuver, namely bladder and rectal filling, levator co-activation, and duration of the maneuver [7, 12, 39]. All women can generate pressures sufficient to reach 80 % of maximal organ descent, provided the maneuver lasts at least 5 s [39]. Real-time imaging allows easily understood visual biofeedback, and this will improve Valsalva performance in most situations. When levator co-activation prevents adequate assessment in the supine position, it may be necessary to repeat imaging in the standing position in order to increase the likelihood of an adequate bearing-down effort.

13.3.4.2 Pelvic Floor Muscle Contraction

A levator contraction is seen as shortening of the levator hiatus in the sagittal plane. This also elevates the anorectum and changes the angle between the levator plate and the symphysis pubis. Other pelvic organs, including the uterus, bladder, and urethra, are displaced cranially during PFMC, and there is compression of the urethra, vagina, and anorectal junction. This explains the importance of the levator ani for urinary and fecal continence as well as for sexual function. As for the Valsalva maneuver, visual biofeedback also aids in teaching PFM exercises, which can be quite effective. A cranioventral shift of pelvic organs imaged in a sagittal midline orientation is taken as evidence of a levator contraction [40]. Measurements of reduction of the levator hiatus in the midsagittal plane [41] or determination of the changing angle of the hiatal plane relative to the symphyseal axis are other methods to quantify levator function. TPUS is considered more reliable than transabdominal ultrasound for the evaluation of PFMC [42], and three-dimensional (3D) ultrasound is regarded as the preferred method. 3D ultrasound allows multiplanar imaging and has been found to measure functional aspects of PFM contraction, such as squeeze and lift reliably [43].

TPUS may be used for the evaluation of pelvic girdle pain (PGP). In a case control study, women with postpartum PGP did not have impaired voluntary PFM function, based on palpation, manometry, and ultrasound [44]. The levator hiatus area, together with BMI, was significantly associated with PGP. Women with PGP had statistically significantly smaller levator hiatus and a tendency for higher vaginal resting pressure compared to controls. A low position of the bladder had a tendency to be associated with PGP and there was a tendency for greater reduction in muscle length during contraction. There was also a tendency for more POP among women with PGP. The finding that women with PGP had a statistically significantly smaller levator hiatus, even at rest, and a tendency for higher vaginal resting pressure may indicate an increased activity of the PFM complex. This corresponds with the findings of Pool-Goudzwaard et al. [45], who showed increased activity, higher resting tone and a shorter endurance time of the PFM as measured with intravaginal palpation and electromyography in women with lumbopelvic pain. It may be that women unconsciously contract their PFM to protect against PGP. The same authors found in a different study [46] that certain patients with PGP may compensate for deficient pelvic stability by increased activity of the PFM.

13.3.5 Clinical Applications of Pelvic Floor Ultrasound

As we have seen, ultrasound allows anatomical and functional assessment of the pelvic floor. The common clinical applications of pelvic floor ultrasound are given in Table 13.2.

13.3.6 Urinary Incontinence

Ultrasound can provide essential information in the management of SUI. Tunn et al. [16] recommended measurement of the posterior retrovesical angle (RVA) with TPUS in patients with SUI. Valsalva maneuver allows for quantitative evaluation of increased urethral and bladder neck mobility. Funneling of the internal urethral meatus may be observed on Valsalva and sometimes even at rest in patients with SUI or urge urinary incontinence (UUI)

In order to maximize pelvic organ mobility, bladder emptying is required [47]. A residual urine measurement can be obtained while assessing the urethra and bladder neck, using the formula (X*Y*5.6) = residual urine in mL, with X and Y the largest bladder diameters measured at right angles to each other, in the midsagittal plane [48]. Provided that residual urine volume is below 50 mL, detrusor wall thickness can be measured, either by vaginal or translabial ultrasound.

TVUS measurement of bladder wall thickness (BWT) is a well-established diagnostic tool in the evaluation of overactive bladder (OAB) symptoms [19]. Khullar et al. found that women with urinary symptoms and detrusor instability had significantly thicker bladder walls than did women with SUI. A BWT greater than 5 mm at TVUS was a sensitive screening method for diagnosing OAB or detrusor over activity in symptomatic women who did not have outflow obstruction [17]. The hypothesis is that BWT is associated with detrusor hypertrophy secondary to isometric contractions [49–52]. Recent systematic reviews have evaluated the various available techniques for measuring BWT and suggested that discrepancies between described techniques cannot allow for safe conclusions about diagnostic accuracy to be drawn [53–55]. BWT has been found to decrease in women with overactive bladder who take anticholinergic therapy, and symptoms improve even though the BWT had stopped decreasing [53]. Other authors have not confirmed these findings [18, 50]. Increased BWT is very likely to be associated with symptoms of the overactive bladder [18, 56], and may be a predictor of postoperative de novo urge incontinence and/or detrusor over activity after anti-incontinence procedures [57].

It has been shown that mid-urethral mobility, rather than general mobility of the bladder neck, is of central importance for continence [58]. Ultrasound can determine segmental urethral mobility, by demonstrating the entire urethra and its mobility relative to the symphysis pubis [59]. Translabial ultrasound can identify an anatomical configuration that is associated with USI. However, sonographic findings are insufficient to predict USI and cannot replace urodynamic testing (Fig. 13.5). For a detailed discussion of urodynamic testing, see Chap. 14 of this book. Urine leakage may also be demonstrated using color Doppler [60]. Other indirect signs of urine leakage on B mode real-time imaging are weak grayscale echoes (“streaming”) and the appearance of two linear (“specular”) echoes defining the lumen of a fluid-filled urethra.

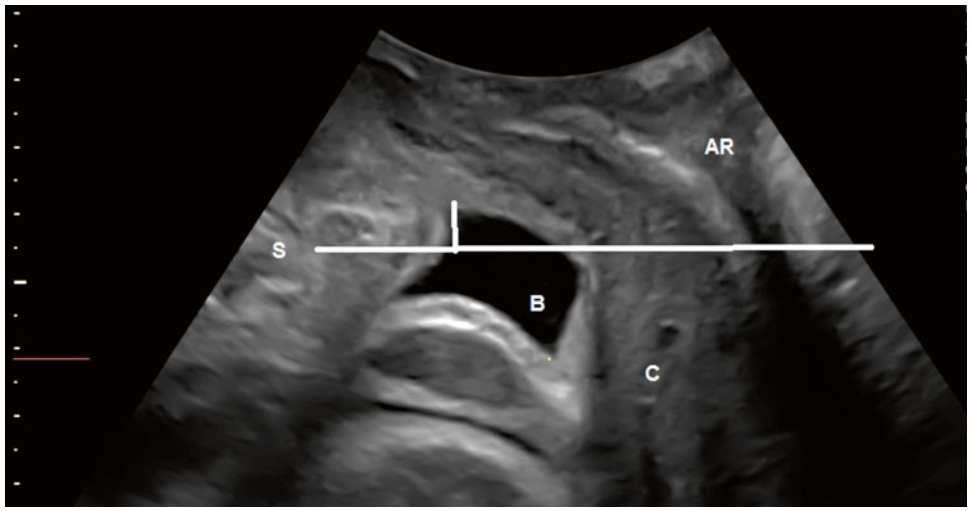

Fig. 13.5 Two-dimensional TPUS (midsagittal view) obtained using a Voluson GE Expert machine equipped with a RAB 8-4-MHz transducer (GE Healthcare). A typical, cystourethrocele is seen as a prolapse of the bladder below the symphysis pubis line (*horizontal line*) during Valsalva maneuver. *AR* anorectal junction, *B* bladder, *S* symphysis pubis, *C* cervix

The urethral musculature can be imaged by transvaginal [61], intraurethral [62], and translabial ultrasound [63]. Issues of ultrasound physics will result in different insonations based on the different techniques. The circular fibers of the rhabdosphincter, depending on their location, are insonated at highly variable angles—some aspects of the sphincter are perpendicular, while others are parallel to the incident beam. This results in variations in echogenicity leading to misconceptions regarding the shape of the rhabdosphincter. On translabial imaging the entire rhabdosphincter is insonated at identical angles, i.e., perpendicular to the incident beam, avoiding artifacts and giving the appearance of a doughnut. An association between sphincter volume and maximum urethral closure pressure, with stress incontinence has been described [64], aswell as the former parameters and surgical outcomes [65].

TPUS and TVS allow comprehensive evaluation of abnormalities of the female urethra, such as urethral diverticula, abscesses, tumors, and other urethral and paraurethral lesions. TPUS is highly useful in the diagnosis of paraurethral abnormalities. Occasionally a "cystocele" will turn out to be due to a urethral diverticulum (Fig. 13.6), a Gartner's duct cyst (Fig. 13.7), or an anterior enterocele, all of which may be missed on clinical examination despite causing disturbing symptomatology [24]. Urethral vascularity may be evaluated by TVS and is believed to contribute to continence.

13.3.7 Fecal Incontinence

EAUS is the historical gold standard for morphological assessment of the anal canal. It can differentiate between incontinent patients with intact anal sphincters and those with sphincter lesions,

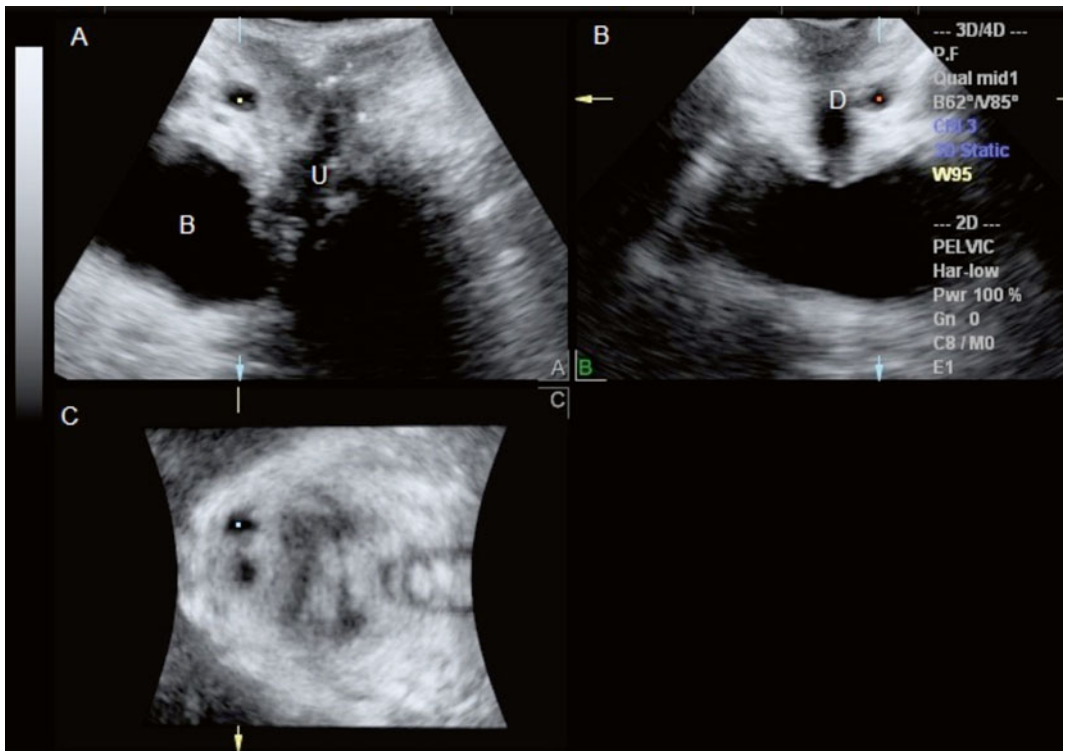

Fig. 13.6 Three-dimensional TPUS using a Voluson GE Expert machine equipped with a RAB 8-4-MHz transducer (GE Healthcare) in a patient with a urethral diverticulum seen connecting to the urethra on panel (**b**). *panel a - midsagittal; panel b - coronal; and panel c - axial view. B* bladder, *U* urethra, *D* diverticulum

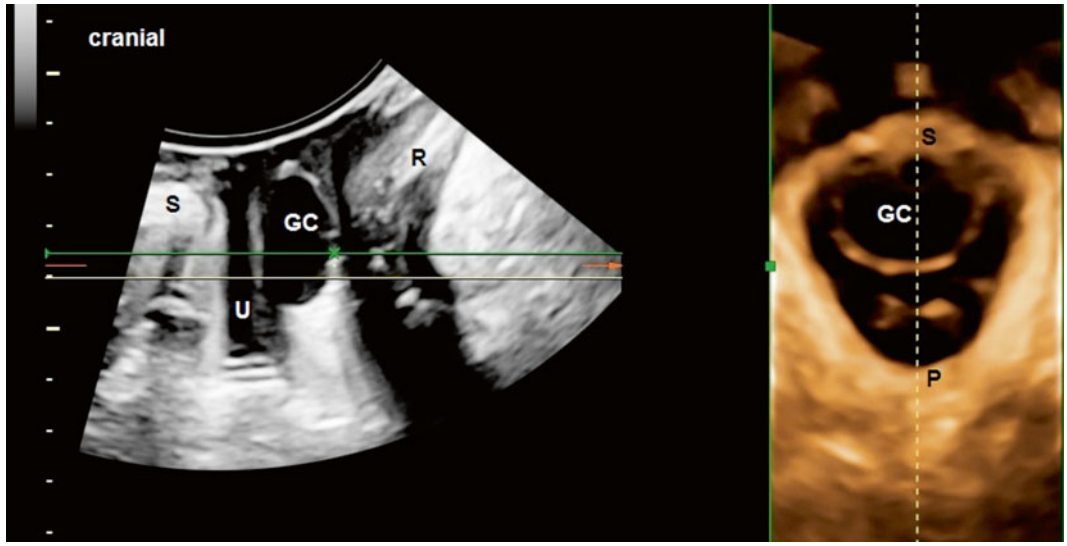

Fig. 13.7 Three-dimensional TPUS and rendered image using a Voluson GE Expert machine equipped with a RAB 8-4-MHz transducer (GE Healthcare) in a patient with a large cyst in the anterior vaginal wall consistent with a Gartner's cyst. *S* symphysis pubis, *U* urethra, *GC* Gartner's cyst, *R* rectum, *P* puborectalis muscle

such as defects, scarring, thinning, thickening, and atrophy [5]. Sphincter tears can be identified by interruption of the circumferential fibrillar echo texture. Scarring is usually seen as a loss of normal architecture, with an area of amorphous texture with low reflectivity. It is possible to identify whether scarring or defects are present in both the IAS and EAS or in either of them. The number of defects and their extent circumferentially (radial angle in degrees or in hours of the clock) and longitudinally (proximal, distal or full length) are also evaluated and reported. Three-dimensional EAUS also allows measurement of length, thickness, area of the sphincter defect in the sagittal and coronal planes, and volume of sphincter damage [66, 67]. EAUS also has an important role in detecting clinically occult anal sphincter injuries following vaginal delivery (Fig. 13.4) [27].

Three-dimensional TPUS has been used to demonstrate the morphological characteristics and normal biometry of the anal sphincter complex [68] and to detect anatomical defects [69–71], and its use is becoming more widespread. One important advantage of the transperineal approach over the endoanal approach is that it avoids distortion of the anal canal by the endoluminal transducer, which can lead to artifacts. Similarly with TPUS, excessive pressure by the transducer on the perineum or an inappropriate angle of incidence of the ultrasound beam to the anal canal may also result in erroneous results. 3D-TPUS usually does not identify clearly the conjoined longitudinal layer, but has the advantage of demonstrating both the IAS and EAS, and also the perineal body, the entire sling of the puborectalis muscle, and the superficial transverse perineal muscles (Fig. 13.8). A further extensive review of posterior compartment assessment is given in Chap. 12.

13.3.8 Levator Ani Assessment

Direct neuromuscular injury to the pelvic floor often causes PFM spasm resulting in dysfunction or pain [72]. This is common in patients who have suffered a traumatic vaginal delivery, particularly if instrumentation was used, with a threefold risk incurred by forceps [73]. Levator injuries may result in POP, postpartum pain syndromes, urinary frequency syndromes, voiding dysfunction, and the levator ani syndrome [74] all of which persist for years after the delivery [75].

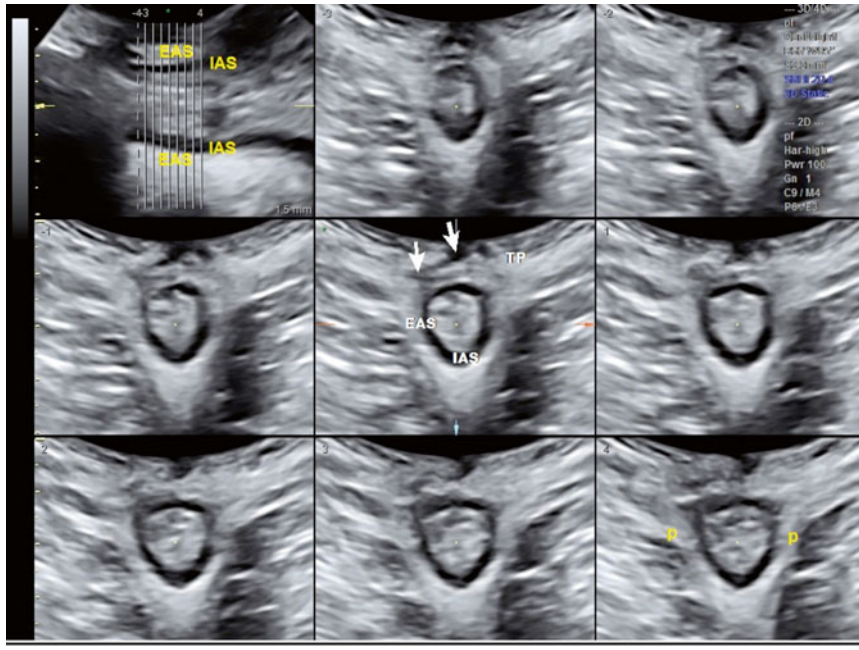

Fig. 13.8 Three-dimensional TPUS and multislice/TUI of the anal sphincter complex in a patient after obstetric anal sphincter injury grade 3A, using a Voluson GE Expert machine equipped with a RAB 8-4-MHz transducer. *EAS* external anal sphincter, *IAS* internal anal sphincter, *TP* transverse perineal muscles, *P* puborectalis muscle

Levator avulsion refers to disconnection of the muscle (usually the puborectalis sling) from its insertion on the inferior pubic ramus and the pelvic sidewall, whereas tears may occur in any part of the muscle. Levator avulsion is usually the result of overstretching of the levator ani during the second stage of labor [76, 77]. The prevalence of levator avulsion defects is approximately 10–36 % of women delivering for the first time [78, 79]. Avulsion is most often occult, but has been demonstrated in the delivery suite in patients with large vaginal tears [80]. Levator avulsion can be palpated clinically [13, 81], but is much easier to detect by imaging, because the lateral attachments of the levator ani to the pubic bone are clearly visualized. Several imaging modalities including 3DTVUS, 3D-TPUS, and MRI can be utilized to document major levator trauma [9, 76, 82]. The clearest visualization of defects is achieved on maximal PFMC, with TUI for quantification of defects (Fig. 13.9) [83].

There are functional and anatomical implications to the presence of levator avulsion defects. An avulsion defect reduces muscle strength by about one-third [82, 84], and there is also a marked alteration in anatomy [85]. The presence of an avulsion may also be a marker for other forms of trauma, such as connective tissue damage to supporting structures (uterosacral ligaments and endopelvic and pubocervical fascia). However, the main effect of levator avulsion is the enlargement of the levator hiatus. An enlarged levator hiatus may result from congenital reasons or due to irreversible over-distension or avulsion injury. This may result in excessive loading of ligamentous and fascial structures, which with time may lead to connective tissue failure, the development of prolapse, and pelvic pain syndromes.

On MRI studies, DeLancey et al. [82] found that women with POP have an odds ratio of 7.3 for having a major levator injury compared with asymptomatic women. A large series using TPUS, confirmed these findings when it found that patients with a levator ani defect compared to those without, are 2.3 times more likely to have a

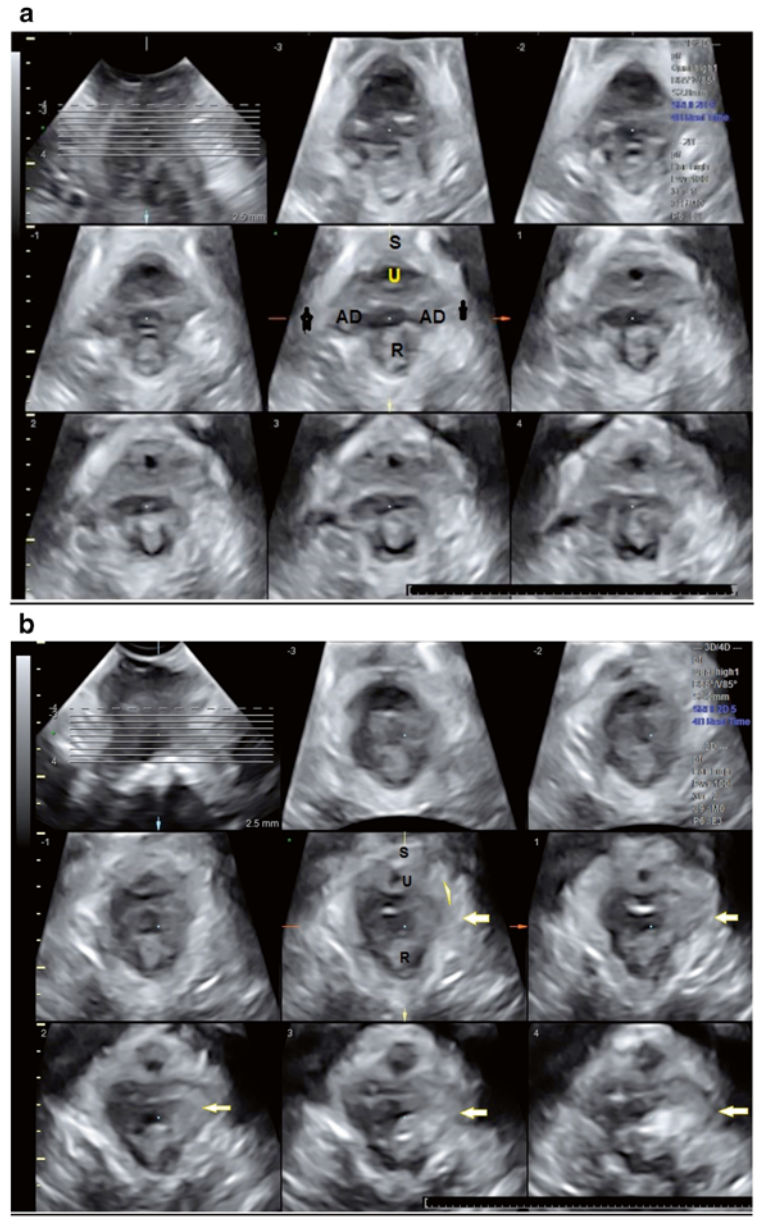

Fig. 13.9 Quantification of levator ani trauma on multislice/TUI using a Voluson GE Expert machine equipped with a RAB 8-4-MHz transducer (GE Healthcare); (**a**) bilateral levator defect (*asterisk*) and AD apparent in all eight slices; (**b**) unilateral left avulsion in a patient referred for trigger point pain on the left side—*arrow*. *S* symphysis pubis, *U* urethra, *R* rectum, *AD* avulsion defect

significant cystocele, and four times as likely to have uterine prolapse [86]. Trauma to the puborectalis component of the levator ani seems to be the most significant in affecting both the size of the hiatus and symptoms and signs of prolapse [87]. There are many women with highly abnormal functional anatomy of the levator ani, even in the absence of a levator avulsion. Athanasiou et al. [88] found that levator hiatus area, measured with 2D-TVS, was significantly larger in women with prolapse than in those without (17.8 vs. 13.5 cm^2). A larger hiatal area was associated with a higher prolapse stage ($P<0.001$), as assessed by maximum descent of the leading organ [8, 89], and this relationship is even stronger on Valsalva. The greater the extent of the defect, the higher the likelihood of symptoms and signs of POP, and the larger the hiatus as measured in the plane of minimal hiatal dimensions [90]. Although 10–30 % of women will suf-

fer avulsion defects, an even greater number will sustain levator micro trauma, which refers to irreversible over distension of the levator hiatus [91]. The predictors of micro trauma may vary from those that predict levator avulsion [91]. The long-term course of such morphological and functional changes is not yet clear, but neither ongoing deterioration nor "healing" is common [92].

The levator hiatus is identified in the midsagittal plane, determining the "plane of minimal hiatal dimensions," which is located in an oblique axial plane. Measurement of hiatal dimensions is useful since the levator hiatus can be interpreted as the largest potential hernial portal in the human body. Hiatal dimensions are strongly and independently associated with prolapse [9, 93, 94]. The hiatal area can be determined in a simple axial plane placed at the location of the minimal anteroposterior diameter of the hiatus [8], and these are comparable to assessment of the hiatus on MRI [95]. Once the plane of minimal dimensions is obtained, it can be used as a reference plane for the assessment of the puborectalis muscle on multislice or tomographic ultrasound (Figs. 13.3 and 13.9) [83]. This is very useful for the identification and evaluation of levator trauma. TUI is easily acquired once the reference plane, the plane of minimal dimensions, is identified. An interslice interval of 2.5 mm enables us to reliably image the entire puborectalis from its most caudal to its most cranial aspects, which can bracket the entire structure, from below its insertion to the inferior aspects of the iliococcygeus muscle [87, 96]. This methodology seems robust enough for clinical practice, and the likelihood of false-positive findings appears very low [97]. Both complete and partial trauma may occur. Partial trauma is of less significance for prolapse and prolapse symptoms [98], and it has to be distinguished from complete trauma which has very different implications and is strongly associated with prolapse [82, 86, 99], and prolapse recurrence [100, 101].

Levator avulsion and ballooning (levator hiatus area in excess of 25 cm^2, [93]) (Fig. 13.10) can be used to select patients for mesh surgery, particularly in the anterior compartment. The effect of anterior compartment mesh on cystocele recurrence may be more marked in women with avulsion [102]. The puborectalis is accessible through a simple distal lateral colpotomy at the level of the hymen and easily dissected off the vagina. 3D-TVUS may also be used to evaluate patients after puborectalis muscle repair with autologous fascia lata [103].

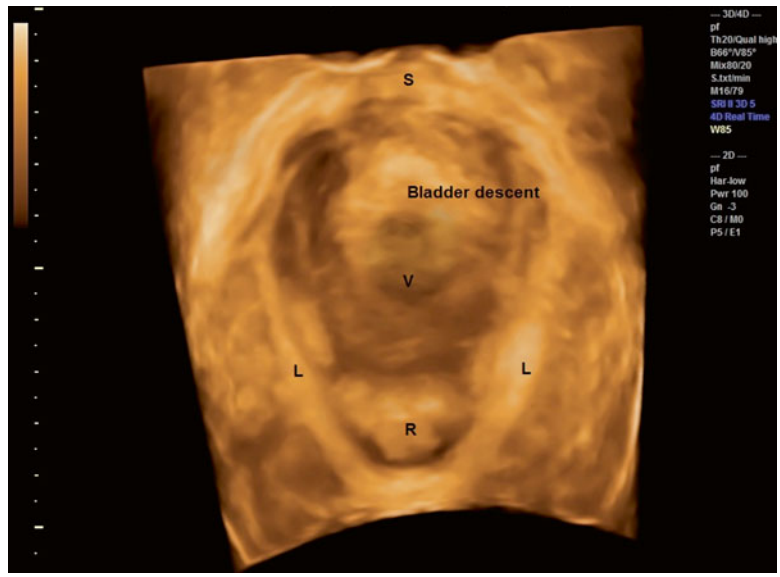

Fig. 13.10 Three-dimensional TPUS and rendered image using a Voluson GE Expert machine equipped with a RAB 8-4-MHz transducer (GE Healthcare) in a patient with moderate ballooning (32 cm^2). *S* symphysis pubis, *V* vagina, *R* rectum, *L* levator

13.3.9 Pelvic Organ Prolapse

Pelvic floor hypertonic disorders may present with symptoms similar to POP; however, on examination no prolapse is found. In the presence of prolapse, typically the leading edge of the prolapse should be at the level of the introitus in order to produce symptoms or even awareness [89].

13.3.10 Anterior Compartment Prolapse: Cystocele

TPUS, being dynamic, can demonstrate downwards displacement of the urethra and the presence of cystocele in the midsagittal plane during maximal Valsalva maneuver (Fig. 13.5) [90]. While clinical examination is limited to grading anterior compartment prolapse, imaging can identify two distinct types of cystocele that can be distinguished clinically [104], and that have very different functional implications [105]. Green first described the different types of cystocele on the basis of radiological imaging [106, 107] using X-ray cystourethrography. A cystourethrocele, in which both bladder base and urethra form one smooth surface and ultrasound shows an open RVA over 140° (Green type II), and isolated cystocele, in which the RVA remains intact and the lowermost point of the bladder is below the level of the bladder neck (Green type III). A cystourethrocele is associated with SUI, while an isolated cystocele is associated with symptoms of prolapse and voiding dysfunction [105]. An associated issue is that of urethral kinking, a common phenomenon in women with Green Type III cystocele with an intact RVA. While in the past, an isolated cystocele had been regarded as evidence of a central defect, and cystourethrocele as evidence of paravaginal defects, it has recently been shown that an isolated cystocele is much more likely to be associated with levator avulsion than is a cystourethrocele (Fig. 13.11) [105].

13.3.11 Central Compartment Prolapse: Uterine Prolapse

Dynamic TPUS can demonstrate the effect of the descending uterus on the bladder neck, the urethra, or the anorectum, which may explain symptoms of voiding dysfunction or obstructed defecation [6, 11, 90]. Mild descent of an anteverted uterus may result in compression of the anorectum and even a mild degree of rectal intussusceptions, explaining symptoms of obstructed defecation. The vaginal vault in a hysterectomized patient may be obscured by a descending rectocele or enterocele and is more difficult to visualize. An anterior cervix in women with an enlarged, retroverted uterus may compress the bladder, explaining symptoms of voiding dysfunction.

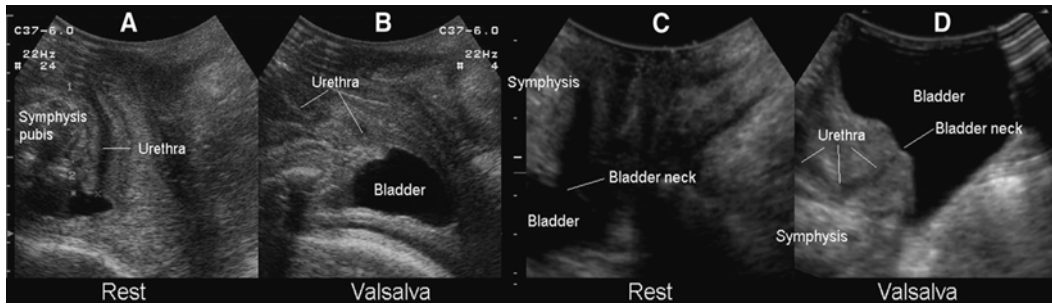

Fig. 13.11 Two main types of cystocele as imaged on maximal Valsalva in midsagittal plane: cystourethrocele (green type II; **b**), associated with urinary stress incontinence and good voiding function, and isolated cystocele (green type III; **d**), associated with prolapse and voiding dysfunction rather than stress incontinence. (**a**, **b**) Retrovesical angle (RVA) on Valsalva is at about 180°, and bladder neck is at lowest point of bladder. (**c**, **d**) RVA on Valsalva, (**d**) is intact at 90–120°, and bladder base is lower than bladder neck. *With permission from Dietz HP. Pelvic floor ultrasound: a review. Am J Obstet Gynecol. 2010; 202(4): 321–334 © Elsevier 2010* [2]

13.3.12 Posterior Compartment Prolapse

13.3.12.1 Rectocele

A prolapse of the posterior vaginal wall or a rectocele may in fact result from different anatomical situations: perineal hypermobility, true rectocele and enterocele and these can be indistinguishable clinically [31]. Several modalities have been used to identify and quantify rectoceles, and defecography has been considered the gold standard for evaluation of this condition [108]. Dynamic TPUS has been shown to demonstrate rectocele, enterocele, and rectal intussusception with images comparable to those of defecography [31, 109]. On ultrasound, a herniation depth over 10 mm has been considered diagnostic [31]. Ultrasound can be used for screening, and when ultrasound imaging reveals a rectocele or a rectal intussusception, there is a high likelihood of this diagnosis being confirmed on proctography [32, 33, 110, 111].

13.3.12.2 Rectal Intussusception

Rectal intussusception is an invagination of the rectal wall into the rectal lumen, and can be classified as intrarectal (remains in the rectum), intraanal (extends into the anal canal), or external (complete rectal prolapse). Both dynamic TPUS and TVS may detect rectal intussusception as an invagination of the rectal wall into the rectal lumen during a maximal Valsalva maneuver [32, 33, 109], providing images comparable to those obtained using MRI and proctography.

13.3.12.3 Enterocele

Dynamic TPUS and TVS may be used as an alternative to evacuation proctography and MR-defecography in the diagnosis of enteroceles [31, 33, 109]. The ultrasound appearance of an enterocele is a herniation of bowel loops into the vagina, and it may coexist with a rectocele. An enterocele can be graded as small (the most distal part descends into the upper third of the vagina); moderate (descends into the middle third of the vagina); and large (descends into the lower third of the vagina). Steensma et al. reported good agreement between 3D-TPUS and defecography for detecting the presence of enterocele [33].

13.3.13 Pelvic Floor Dyssynergy

Pelvic floor dyssynergy, anismus, spastic pelvic floor syndrome, or paradoxical puborectalis syndrome, is a common name for a phenomenon characterized by a lack of normal relaxation of the puborectalis muscle during defecation. This is to be differentiated from an involuntary (reflex) contraction of the levator ani, which is a common reaction, especially in young nulliparous women [12]. Dyssynergy is associated with symptoms of obstructed defecation and incomplete emptying. Dynamic TVS and TPUS may also have a role in documenting pelvic floor dyssynergy [31, 33]. When dyssynergy occurs during a Valsalva maneuver, the anorectal angle becomes narrower, the levator hiatus is shortened in the anteroposterior dimension, and the puborectalis muscle thickens as a result of contraction/levator coactivation. These findings may help to refer the patient for physiotherapy and in evaluating results after treatment.

13.3.14 Bladder Pain Syndrome/ Interstitial Cystitis

Bladder pain syndrome/interstitial cystitis (BPS/IC) is a chronic inflammatory condition of the bladder, which is the cause of pain in more than 30 % of women with chronic pelvic pain, and may be also associated with other pain syndromes, such as inflammatory bowel disease, fibromyalgia, and vulvodynia. Ultrasound can aid the diagnosis by evaluating the anterior compartment as described above. Imaging is indicated as secondary or primary assessment when oral or intravesical therapy fails, or before initiating treatment at the discretion of the clinician [112]. Endometriosis and interstitial cystitis (IC) may coexist in approximately 65 % of women [113–115]. A recent systematic review of the prevalence of BPS/IC and their coexistence with endometriosis in women with chronic pelvic pain found nine studies including 1016 patients. The mean prevalence of BPS was 61 %, of endometriosis was 70 %, and coexistence of both 48 % [116]. This means that even in endometriosis

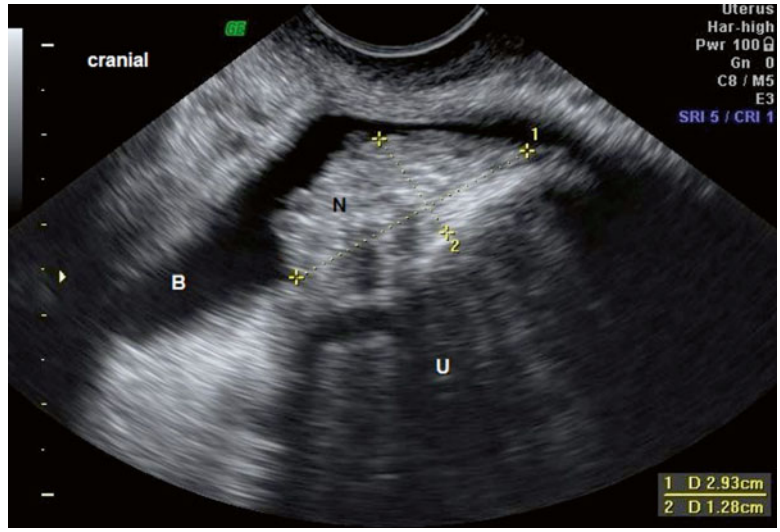

Fig. 13.12 2D TVUS image using a Voluson GE Expert machine equipped with a transvaginal 5–9 MHz transducer (GE Healthcare) of a bladder nodule in a patient with severe bladder pain syndrome who was subsequently diagnosed as suffering from endometriosis. *B* bladder, *N* endometriotic nodule, *U* uterus

patients, one must consider that the bladder may be the source of pain, and particularly in women with refractory pelvic pain associated with endometriosis, evaluation to rule out BPS/IC should be undertaken (Fig. 13.12). The chronic inflammatory state associated with endometriosis may be a trigger for neuropathic changes such as visceromuscular hyperalgesia, resulting in muscular instability and a hypertonic contractile state of the pelvic floor musculature.

13.3.15 Vulvodynia and Vestibulodynia

Women with vestibulodynia often have additional pain conditions, such as fibromyalgia, irritable bowel syndrome, BPS/IC, or chronic fatigue syndrome. It has been suggested that PFMs play an important role in PVD pathophysiology. Morin et al. [117] used 4D TPUS to compare PFM morphometry in women suffering from vestibulodynia and compared them to asymptomatic healthy controls. They found that women with PVD showed a significantly smaller levator hiatus area, a smaller anorectal angle, and a larger levator plate angle at rest compared with asymptomatic women, suggesting an increase in PFM tone. During PFM maximal contraction, smaller changes in levator hiatus area narrowing, displacement of the bladder neck, and changes of the anorectal and of the levator plate angles were found in women with PVD compared with controls, which may indicate poorer PFM strength and control. This provides evidence that women with PVD have increased tone and reduced strength of the PFMs. The advantages of 4D TPUS in this context are the ability to perform a noninvasive pain-free evaluation.

13.3.16 Postoperative Imaging

Surgery to the pelvic floor can be a trigger to the development of hypertonic disorders, and can cause postoperative voiding dysfunction, urinary frequency, urgency, and pain syndromes. This is particularly seen following procedures that involve fixation to muscle sites, such as various mesh and sling operations, levator plications for rectocele correction, or sacrospinous vaginal vault fixation [118].

TPUS has been used in the evaluation of postoperative findings after anti-incontinence surgery for over two decades [119, 120]. Burch and Marshall Marchetti colposuspensions usually produce typical postoperative findings that include bladder neck immobilization, a varying degree of anteriorization, and a "colposuspension

ridge" under the bladder [121, 122]. Fascial slings may also cause a ridge with a pronounced bladder neck immobilization. Over-elevation of the bladder neck when seen on ultrasound may be a marker for postoperative voiding dysfunction and symptoms of the overactive bladder [123]. Laparoscopic colposuspensions also cause distortion of the bladder neck [124], while urethropexies elevate the internal urethral meatus rather than the trigone [125].

Modern suburethral slings are synthetic implants usually of wide-weave polypropylene mesh that are highly echogenic and easily identified in the anterior vaginal wall. TPUS is the most appropriates imaging modality since these implants cannot be seen with plain X-ray, CT, or MRI [126]. Ultrasound can confirm the presence of the sling (Fig. 13.13), evaluate its position [127], or distinguish between transobturator and transretzius slings [128], compare the results of different insertion techniques [129], and evaluate type and material of the sling [130]. Perforation or erosion of the sling into urethra or bladder can also be seen by TPUS [131].

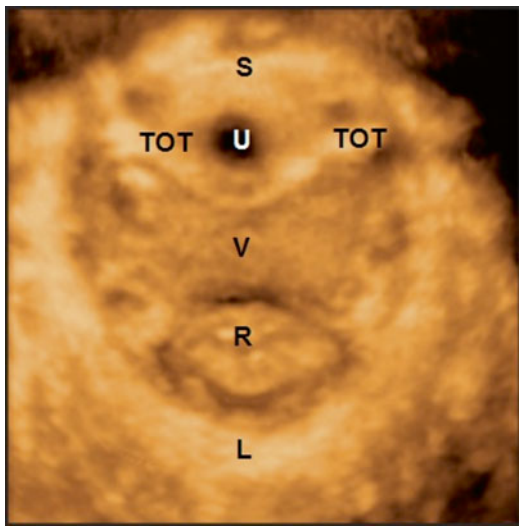

Fig. 13.13 TPUS using rendered view using a Voluson GE Expert machine equipped with a RAB 8-4-MHz transducer (GE Healthcare): transobturator tape is visualized in an axial rendered volume with the patient at rest. *S* symphysis pubis, *U* urethra, *TOT* transobturator tape, *V* vagina, *R* rectum, *L* levator ani

Synthetic or biologic grafts are widely used especially in women with severe or recurrent POP. Levator avulsion is a strong risk factor for prolapse recurrence after conventional surgery [100, 101]. Evaluating for levator avulsion injury by imaging may help in determining which patients are most likely to benefit from mesh use, especially in view of the occurrence of complications such as support failure, mesh erosion, and chronic pelvic pain. Meshes, which are made from Polypropylene, are less visible on X-ray and MRI, but are easily seen on ultrasound because of their high echogenicity. Mesh visibility on ultrasound may be limited by recurrent prolapse or patient tissue composition. Ultrasound imaging allows determination of mesh position, extent and mobility of mesh implants, assessment of surgical techniques, and determination of functional outcome. Mesh may be visualized in the anterior or posterior vaginal wall, behind the trigone and posterior bladder wall or anterior to the rectal ampulla, sometimes extending into the perineal body (Fig. 13.14).

Mesh complications cited in the literature are erosion or mesh exposure and chronic pain syndromes [132], mesh contraction [133], both of which may be the result of physiological wound healing [134, 135]. At times the mesh appears less wide on ultrasound than expected. This has in the past lead to a suspicion of mesh shrinkage, contraction, or retraction [136, 137]. A more plausible explanation is that the mesh did not in fact shrink, but rather became folded on itself, either during the implantation process, immediately after closure, or at any other time during the immediate postoperative period [134, 135, 138].

Mesh inserted transabdominally, such as sacrocolpopexy Y, may also be seen by 3D-TPUS. Eisenberg et al. [138] described the appearance, position, and dimensions of mesh implants following minimally invasive sacrocolpopexy. Folding of the mesh was a common occurrence resulting in decreased mesh dimensions with time.

Ultrasound may uncover complications such as dislodgement of anchoring arms [139] that may occur in different forms causing prolapse

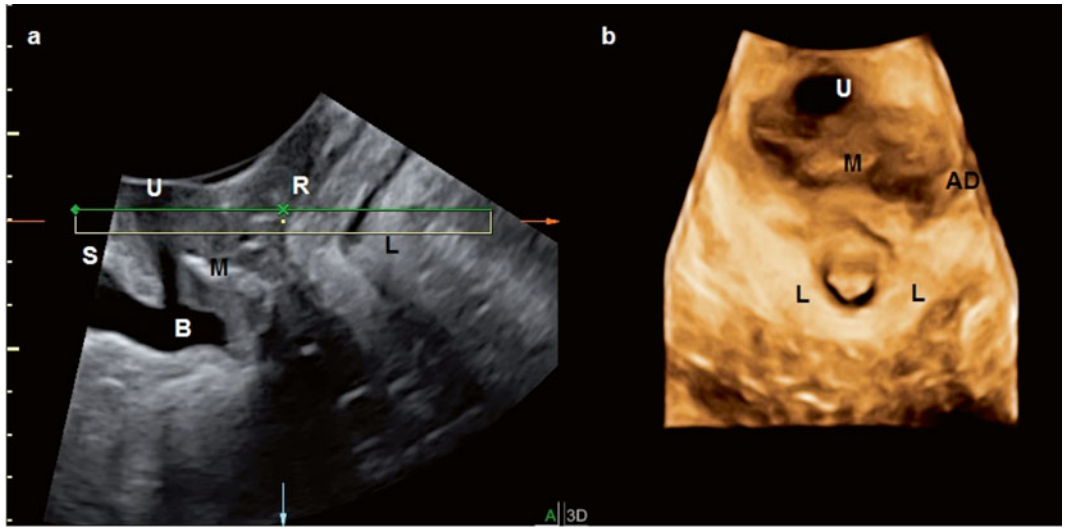

Fig. 13.14 Three-dimensional TPUS using a Voluson GE Expert machine equipped with a RAB 8-4-MHz transducer (GE Healthcare) in a patient with left avulsion injury, after successful anterior mesh implantation. The midsagittal plane (**a**) demonstrates absence of prolapse on Valsalva, despite levator ballooning evident in the axial plane (rendered volume) (**b**). *S* symphysis pubis, *U* urethra B, *B* bladder, *M* mesh, *R* rectal ampulla, *L* levator ani, *AD* avulsion defect

recurrence due to suspension failure. Dislodgment of all four arms occurs rarely (not more than 5 % of all cases). Dislodgment of the superior (cranial) anchoring arms is more common [136]. Patients with ballooning (hiatal distensibility on maximal Valsalva) or levator avulsion are at an increased risk of support failure. Dislodgment of the superior arms occurs in about 20 % of transobturator meshes, resulting in the appearance of a "high cystocele," similar to that observed after Burch colposuspensions [11].

13.4 Other Imaging Modalities

13.4.1 Cystourethroscopy

Endoscopy is helpful in the exclusion of other causes for symptoms of OAB, such as bladder calculi, tumors, or endometriosis, affecting the bladder [140]. Women complaining of hematuria, painful bladder syndrome, or recurrent incontinence should also be referred for cystourethroscopy.

13.4.2 Fluoroscopy

Fluoroscopic techniques such as voiding cystourethrography (VCUG), evacuation proctography, cystoproctography, and cystocolpoproctography are still widely available because of their ability to depict pelvic floor abnormalities with the patient in a physiological position, either standing or sitting as opposed to supine as with MRI or TPUS. The disadvantages of these procedures include their invasive nature, use of ionizing radiation, need for contrast media, and limitation to single compartment investigations. VCUG is used for detecting anterior vaginal wall prolapse in women with a history of urinary incontinence. The patient is imaged in a lateral standing position after the bladder has been filled with iodinated contrast. The imaging is performed at rest, coughing, and voiding in order to evaluate bladder base descent. This also enables evaluation of vesicourethral reflux, bladder and urethral diverticuli and bladder wall trabeculation [141]. Fluoroscopy can be used in combination with filling and voiding cystometry, during video-urodynamic testing. See

Chap. 14 for a discussion of indications and technique.

Evacuation proctography or defecatory proctography is used for assessing rectal evacuation, prolapse, and rectocele in women with constipation and defecatory dysfunction. The study uses evacuation of paste barium enema and oral contrast medium, and is performed with the patient seated on a commode placed on the footrest of the X-ray table, and continuous imaging by video fluoroscopy is performed before, during, and after evacuation. Pelvic floor descent can be measured with reference to fixed bony landmarks, such as PCL (a line drawn from the inferior part of the symphysis pubis to the sacrococcygeal joint, corresponding to the pelvic floor) and the midpubic line (MPL) (an extension of the long axis of the symphysis pubis) [142]. Normally, a complete evacuation involves pelvic floor descent, relaxation of the puborectalis and sphincter muscles and a wider anorectal angle, followed by a return to the pre-evacuation position. Proctography can facilitate in the diagnosis of rectal prolapse, rectocele and rectal intussusceptions. "Anismus," "pelvic dyssynergy" or absent or delayed rectal emptying due to inability of the puborectalis muscle to relax during voluntary evacuation, can also be diagnosed by evacuation proctography. This is seen when more than 66 % of rectal contrast material is not evacuated within 30 s [143].

A complete method of imaging pelvic floor disorders would involve the use of multicompartmental fluoroscopic techniques such as cystoproctography, cystocolpoproctography, and functional MRI with their advantages and disadvantages [144].

13.4.3 Pelvic Floor MRI

High-resolution MRI techniques offer considerable insight into the etiology of pelvic floor structural defects, without the use of ionizing radiation, and are considered superior to fluoroscopy, which has been considered the gold standard for more than 20 years in detecting pelvic floor abnormalities. MRI allows visualization of soft tissue, ligamentous and muscular pelvic floor structures in fine detail. The main disadvantages of its use are considerable cost, need for specialist radiological interpretation, low patient acceptability, and limited access [4]. There is no need for specific patient preparation for static MRI imaging. Images are usually acquired in axial, sagittal, and coronal planes, with the patient in the supine position. If an open magnet is available the images can be obtained with the patient seated.

Dynamic MRI may be useful for diagnosing and staging POP, with similar detection rates when compared with fluoroscopic techniques. MRI is often able to reveal more extensive organ prolapse than physical examination alone [142, 144]. Several lines and levels of reference have been proposed, the most commonly used ones are either a line drawn from the inferior margin of the symphysis pubis to the last sacrococcygeal joint (pubococcygeal line—PCL) or a line extending caudally along the longitudinal axis of the symphysis pubis in the sagittal plane (MPL) [76, 145–148]. The MPL seems to correspond to the level of the hymen [147], but different staging systems exist for the PCL and MPL [149]. The largest measurement from the leading edge of the prolapsed organ (bladder base, cervix/vault, or anorectal junction) perpendicular to the reference line during straining or evacuation is used to stage the presence and degree of POP.

Broekhuis et al. [150] reported low agreement between patients' symptoms, based on validated questionnaires, and findings on dynamic MRI of the pelvic floor, leading them to conclude that that dynamic MRI is unlikely to have clinical benefit. Contrary to this, Hetzer et al. [144] reported that MR-defecography findings led to changes in surgical management in two thirds of patients. Kaufman et al. [4] showed that dynamic MRI led to altered surgical management plans in 41 % of cases. Groenendijk et al. [151] reported a revision of initial management plans in more than a third of cases following diagnostic tests. Overall, defecography was regarded as more valuable (assigned diagnostic value, 49 %) than MRI, which was rated least useful of the tests considered for use (assigned diagnostic value, 20 %). All this information may lead to the conclusion that dynamic MRI should

not be the first modality of choice even in the settings where it is highly available.

The puborectalis muscle can be seen as a separate structure on MRI lateral to the pubovisceralis, but is best imaged in the axial and sagittal planes, while the iliococcygeus muscle is better visualized in the coronal plane. There is considerable variation in the levator ani size and thickness between individuals [146]. MRI has been used extensively to study the impact of vaginal delivery on the various components of the levator ani muscle. Strain forces during vaginal birth can cause the levator ani to stretch as much as 320 %, and up to 20 % of parous women may sustain levator injury, particularly in association with forceps delivery, anal sphincter tears, and episiotomy [76, 145].

The anal sphincter complex can be visualized in the axial and coronal planes: The EAS has low signal intensity, while the IAS has intermediate signal intensity on T2-weighted images. Pelvic floor MRI is considered to be as accurate (91 %) as ultrasound in detecting anal sphincter defects and more accurate (93 %) than ultrasound in demonstrating sphincter atrophy [152]. Endoanal MRI, with the use of an endocoil, is more complex, more time-consuming and less acceptable by the patient than endoanal US, but may provide high quality images of the anal sphincter complex. Endocoil MRI seems to be as equally effective as EAUS in depicting anal sphincter tears; however, EAUS remains the investigation of choice for assessing anal sphincter integrity following obstetric injuries. One of the major indications for endoanal MRI is the assessment of anal sphincter volume and thickness [152]. EAS atrophy is defined by thinning of the muscle and replacement by fat. A normal EAS is 4 mm thick on MRI. A thinning of less than 50 % of the normal muscle is considered as moderate atrophy, whereas >50 % thinning and replacement by fat is considered severe atrophy [153, 154].

Studies using static MRI to study the mechanism of urinary incontinence have found that in continent women the levator plate is nearly parallel to the PCL and the bladder neck is above the PCL and closer to the symphysis pubis in comparison with women with urodynamic stress incontinence [155, 156]. Digesu et al. found that women with Burch colposuspension had a shorter distance between the levator ani muscle and the bladder neck, and that this was associated with continence [157].

13.5 Summary

This chapter provides a current update on the use of pelvic floor imaging for the various indications described in Table 13.2. TPUS, EAUS, and TVS all provide an accurate anatomical assessment of patients with urinary incontinence, fecal incontinence, and presence and grade of POP. When discussing which methodology should be used, one must consider the available instrumentation as well as the expertise of the performer. Advanced ultrasound techniques and the advent of 3D and 4D ultrasound capabilities, new software options, and better training may all allow a better multicompartment assessment of pelvic floor dysfunction. Combining TPUS, TVS, and EAUS may complement the advantages of each modality and to overcome possible limitations in order to improve clinical management.

One of the most significant aspects of sonography for the clinical management of patients with POP is the ability to assess for levator ani morphology and function, because it allows the assessment of the underlying abnormality rather than just the clinical manifestation. Levator ani damage, avulsion defects, abnormal levator ani contractility and ballooning may be diagnosed on TPUS and TVS. TPUS also has the advantage of enabling evaluation of pelvic floor function with various dynamic maneuvers. In patients with urinary incontinence, ultrasound can provide useful information on the anatomy and function of the lower urinary tract, with relation to urethral mobility and vascularity, funneling of the internal urethral meatus, bladder neck descent and BWT, all of which may be evaluated by TPUS or TVS. Ultrasonography allows the evaluation of anti-incontinence procedures and helps in understanding their failure.

Ultrasonography should be performed as an initial examination in patients with defecatory disorders. Positive findings on ultrasound may

avoid more invasive tests, whereas negative findings require confirmation by defecation proctography. Recent years are seeing an increased role of TVS and TPUS in the evaluation of anal sphincter injury owing to its greater availability and patient acceptability.

Ultrasound can be of great utility to the pelvic reconstructive surgeon and to the clinician involved with the care of pelvic floor dysfunction and pelvic pain syndromes. A full clinical assessment in urogynecology should include imaging, especially in complicated and recurrent cases. Both ultrasound and MRI already have a substantial impact on clinical research and audit. Imaging techniques help us to further elucidate the etiology and pathophysiology of pelvic floor dysfunction, help to assess the outcomes of conservative and surgical treatment, and to assist in the development of entirely new therapeutic concepts. It is important to incorporate this vast knowledge and utility in the care of patients with hypertonic pelvic floor disorders and chronic pelvic pain.

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Urodynamic Assessment

14

Sarit Barak and Gil Levy

14.1 Purpose of Urodynamic Testing

The purpose of urodynamic testing is to supplement a patient's clinical history and physical examination with a series of tests that are designed to assess the storage and voiding phases of micturition, using noninvasive and invasive methods [1].

Observations seen during these tests and the clinician's interpretation can help identify potential bladder safety issues, guide treatment, predict outcomes and establish a correlation between symptoms and the patient's quality of life (QoL) [1].

Before performing any urodynamic test, a clinical evaluation should be completed to identify the relevant urodynamic questions. A thorough history is necessary to obtain a clear understanding of the patient's complaints, including type of symptoms (e.g., urgency, frequency, urge incontinence, stress incontinence, pain and other voiding and storage symptoms), severity and duration of symptoms, bother associated with the symptoms, previous therapies, and relevant medical comorbidities. A physical examination can identify specific findings (urethral diverticulum, pelvic organ prolapse, a pelvic mass), which may contribute to, or cause, the symptoms of interest. Patients can also be asked to complete a voiding diary to objectively assess fluid intake, voided volumes, episodes of incontinence, and voiding frequency. Pad-weight testing helps quantify the amount of urine lost during incontinence episodes [2]. Data from validated questionnaires allow to quantify symptoms and their effect on QoL. To be most useful, data obtained from urodynamic testing must be considered as supplemental to clinical evaluation [3, 4].

Both noninvasive and invasive urodynamic techniques can be used to help qualify and quantify lower urinary tract activity during the micturition cycle. Noninvasive tests include uroflowmetry and post-void residual (PVR). Invasive tests include cystometry, sphincter electromyography (EMG) videourodynamics (VUDs), pressure-flow study (PFS), and urethral function tests [1].

14.2 Preparation for Urodynamic Testing

Preparing the patient for invasive urodynamic testing can greatly affect usefulness and efficacy of the test. As with all invasive procedures, informed consent should be obtained and all questions addressed [1]. Patients generally tolerate urodynamic testing well, but feelings of anxiety,

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discomfort, and embarrassment are common. Preparation should include a thorough explanation about the test procedures using lay terminology, as well as the rationale for the testing, which can help reduce feelings of discomfort [5]. Patients should be instructed to maintain their regular diet and to take their scheduled home medications [1]. Patients with overactive pelvic floor syndrome (OPFS) might react with anxiety, fearing the procedure as it involves a sensitive area. In these cases, a detailed explanation of the procedure is required with accommodation of specific requests by the patient. Occasionally a mild tranquilizer is required prior to the procedure. Showing the urodynamic machine and catheters to the patient may elevate anxiety on the one hand, but may increase the patient's sense of control on the other. It is suggested to ask the patient what will make things easier for her and allow extra time for studies. Scheduling the test at the end of the session can be useful for such patients. Before the examination, explain why, how, where, and for how long it will take. The use of local anesthetics in the form of Lidocaine gel may be used prior to the insertion of the catheters. Occasionally, self-insertion of the vaginal catheter and patient's assistance with the insertion of the bladder catheter may be suggested, with the hand of the patient controlling the nurse's sterile hand that holds the catheter. Lubrication of the catheters needs to be visualized by the patient and continuous verbal communication before and during each step is critical to keep the patient's cooperation. In our experience, the presence of relatives in the room, occasionally requested by the patient, might aggravate anxiety during the procedure. We minimize the number of staff present in the room to the nurse and the physician, whom the patient is familiar with in advance. Preparing all the equipment in advance allows for the staff to present a safe and professional environment for the test.

All patients should undergo urinalysis and urine culture before the test, to screen for signs of urinary tract infection before the procedure. Patients with a symptomatic infection should defer the urodynamic test until the infection is resolved (negative culture and symptoms).

Limited data exist regarding the usefulness of pre-procedural antibiotic administration. The American Urological Association (AUA) Best Practice Statement on Urologic Surgery Antimicrobial Prophylaxis states that antibiotic prophylaxis before urodynamic testing is indicated only in patients with risk factors, such as: as advanced age, anatomic anomalies of the urinary tract, poor nutrition status, smoking, chronic corticosteroid use, immunodeficiency, externalized catheters, colonized material, coexistent infection, and recent prolonged hospitalization. Recommended antibiotics include oral fluoroquinolones or trimethoprim-sulfamethoxazole. However, patient allergies, prior urine cultures, and local antibiogram pattern should be considered [6, 7].

14.3 Special Considerations

Patients with spinal cord injuries above T6 are at risk for experiencing Autonomic Dysreflexia (AD) during bladder filling, characterized by an acute increase in blood pressure and bradycardia, accompanied by symptoms such as headache, piloerection, skin pallor, profuse sweating, and flushing. Untreated AD can result in intracranial hemorrhage, retinal detachment, seizures, and death [8]. A prior positive history or risk factors for AD and typical triggers for developing AD in the patient should be noted and appropriate preparations and/or precautions should be followed. Many patients know their typical triggers and these most often involve simulation of the bowel or bladder [9]. Preparations before starting urodynamic testing include monitoring blood pressure and heart rate, which should be continued throughout the study. If symptoms of AD are identified during urodynamic testing, the trigger (usually filling of the bladder or catheter placement) should be removed, by draining the bladder and then removing catheters if needed. Additionally, the patient should be placed in reverse Trendelenburg (head up) to take advantage of any gravitational reduction in blood pressure. Any tight clothing or restrictive devices should be loosened. If blood pressure elevation

does not resolve, 1–2 in. of nitro paste can be applied to the chest and wiped off after blood pressure values normalize [8].

As with all invasive procedures, certain patients may also experience vasovagal syncope during urodynamic testing. For this reason, some centers' policy require that all patients, male and female, perform the voiding phase of the study in the seated position. In contrast to AD, treatment of vasovagal syncope requires the patient be placed in the Trendelenburg position, in order to increase blood flow to the head and/or chest [1].

14.4 Equipment

Single-channel urodynamic studies should be avoided in cases of OPFS patients. Complex filling cystometrogram (CMG) allows for measurement of bladder pressure during filling. Single-channel recording of bladder pressure can offer information about bladder sensation, capacity, compliance, and voluntary detrusor contractions [10, 11]. The urodynamic catheter (6–10 F) is placed into the bladder and room temperature fluid is instilled at 30–50 mL/min. Lidocaine gel can be used in order to achieve better cooperation from the patient with OPFS who present with anxiety prior to the exam. The gel can be applied 10 min prior to insertion of the catheters. Bladder sensation (first sensation, normal desire, strong desire, and maximum capacity) is assessed. Extremes in sensation likely represent a pathologic abnormality [12].

Commonly, disposable air-charged or water-filled urodynamic pressure-measurement catheters are used to perform cystometry. Air-charged catheters are newer in design; therefore, most prior research was based on water-filled systems. There are notable differences in how each catheter responds to transient and sustained pressure changes and they do not give interchangeable results [13]. However, both catheter types are widely accepted for clinical use, and most observed differences are outside the range of what is generally relevant to urodynamic studies.

In urodynamic terminology, Pves is the measure of the bladder pressure and Pabd is the abdominal pressure measured by the catheter in the rectum [1].

Standard double- and triple-lumen catheters are used. Double-lumen catheters have one port for fluid inflow and a second port to measure vesicle pressure (Pves). Triple-lumen catheters provide a third more proximal, sensor port, which can be positioned at the level of the mid-urethra to measure bladder and urethral pressure simultaneously.

Abdominal pressure (Pabd) is measured by rectal or vaginal catheter. This allows the testing clinician to incorporate information about the relative contribution of Pabd changes (i.e., with cough or Valsalva) to bladder pressures and calculate detrusor pressure.

Rectal or vaginal catheters come in multiple styles, including fluid-filled rectal balloon and air-charged catheters. Rectal catheters are placed in the rectal vault, proximal to the anal sphincters. Presence of stool in the rectum can affect Pabd readings. Vaginal catheters are placed in the vaginal vault and considered equal to the rectal catheters. In patients with overactive pelvic floor, the choice for rectal or vaginal is given to the patient. Patients with anxiety from vaginal insertion might prefer rectal balloon. Patients can be offered to insert the vaginal catheters themselves.

Regardless of catheter system, the International Continence Society (ICS) recommends that all urodynamic catheters be zeroed to atmospheric pressure and pressure transmitters are positioned at the height of the upper edge of the pubic symphysis [10].

14.5 The Components of the Urodynamics Test

The components of the test can be divided into noninvasive or invasive tests, and every individual urodynamic test includes residual volume, cystometry, uroflowmetry, pressure flow studies, and electromyography (EMG).

14.5.1 Uroflowmetry: Noninvasive Emptying Assessment

Prior to insertion of the catheters, a free uroflowmetry is performed. This is a noninvasive part of the exam that measures the flow of urine and the duration of micturition. During this test, a well-hydrated patient voids into an uroflowmeter, which generates a "flow curve" [14]. The flow curve is plotted with the urine flow on the *Y*-axis and time on the *X*-axis.

Measurements obtained during uroflow are peak flow rate (Q_{max}), flow rate, voiding time, voided volume, and flow pattern (Fig. 14.1).

Urine flow: Voluntary urethral passage of urine which may be continuous or intermittent.

Flow time (s): The duration of time during which the flow occurs.

Voiding time (s): The total duration of micturition including interruptions. When voiding is completed without interruption, voiding time is equal to flow time [16, 17].

Flow rate: Volume of urine expelled via the urethra per unit of time. It is expressed in mL/s. A reduced flow rate suggests the presence of bladder outlet obstruction, reduced bladder contractility, or both [16, 17].

Maximum urine flow rate (MUFR), or Q_{max}: Maximum measured value of the flow rate. It can be determined by evaluating the flow curve during uroflowmetry and is expressed in mL/s. Q_{max} can be influenced by a number of factors including age, gender, and volume voided. For instance, women generate higher flow rates on average compared to men because of the presence of a shorter urethra, which offers less resistance. Variability among uroflow tests can also occur in the same patient, depending on several factors, including time of day and hydration status. Thus it is important to repeat an abnormal test on a patient who is being considered for surgery or invasive therapy and the interpretation of this value must be in the context of additional clinical information [18, 19].

Average (urine) flow rate (AUFR), or Q_{ave}: Voided volume divided by flow time [16, 17].

Flow pattern: This is one of the most important parts of the exam, specifically in the case of OPFS patients. Flow patterns may be subject to the interpretation of the individual clinician because there is no standard against which to compare them. Flow patterns can be described in various ways, such as "Flat," "Double stream," or "Intermittent" which

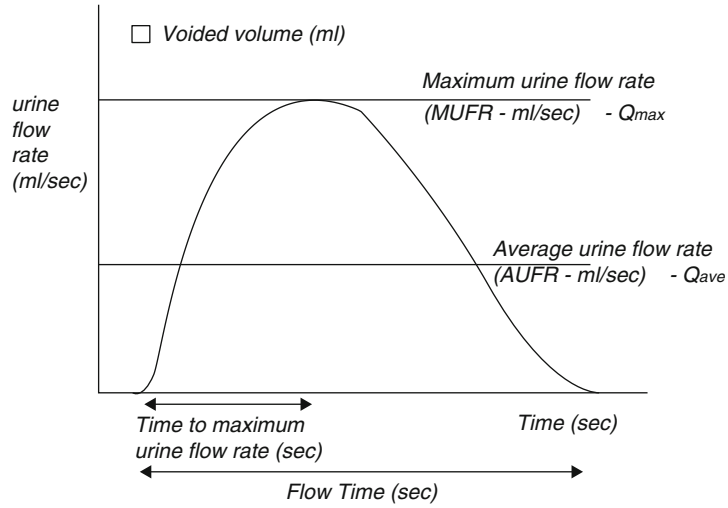

Fig. 14.1 Schematic illustration of a normal uroflowmetry curve, with flow rates measured over time. *With permission from Haylen BT, De Ridder D, Freeman RM, Swift SE, Berghmans B, Monga A, Petri E, Rizk DE, Sand PK, Schaer GN. An International Urogynecological Association (IUGA)/International Continence Society (ICS) joint report on the terminology for female pelvic floor dysfunction. Int Urogynecol J (2010) 21:5–26 © Springer 2010* [15]

may indicate obstruction. Bladder outlet obstruction can be due to many causes that can be suggested by clinical history, but cannot be diagnosed just with PVR testing. Abnormal uroflowmetry results are often further evaluated using more invasive tests, typically a pressure flow study [18].

Voided volume: is the total volume of urine expelled. This volume must be equal to or greater than 150 mL for uroflowmetry results to be valid. A voided volume less than 150 mL may produce an invalid test, because flow patterns and parameters are inaccurate below this volume. Also, the voiding of a very large volume may lead to an abnormal flow test result in a patient with no significant pathology. Such abnormal results are the outcome of overstretching of the detrusor muscle that can cause an inefficient contraction [18].

Time to maximum flow (s): The time from the onset of urine flow to maximum urine flow [16, 17]. There is a strong dependency of urine flow rates on voided volume. The urine flow rates are best referenced to nomograms. The cutoff for abnormally slow rates of MUFR and AUFR has been determined and validated as under the tenth percentile of the respective Liverpool female nomogram [20–24]. As per the Liverpool nomogram the 25th centile (men) and the 10th centile (women) appeared to be most appropriate lower limits of normality for both urine flow rates to identify those men more likely to be obstructed and those women at higher risk of voiding difficulties.

Post-void residual urine volume (PVR):The volume of urine left in the bladder at the completion of micturition [17, 25]. PVR can be measured directly by draining the bladder with a catheter, indirectly with bladder ultrasound, or by fluoroscopy during a videourodynamic exam (if radiopaque contrast has been instilled into the bladder before voiding) [26]. The difference in the upper limits of normalcy may reflect the accuracy of measurement techniques. Transvaginal ultrasound suggests an upper limit of normal of 30 mL [27]. Studies using urethral catheterization with up to 10-min delays quote higher upper limits of normal of 50 mL [25] or 100 mL [27]. An isolated finding of an abnormal PVR requires confirmation before being considered as significant [15].

14.6 Cystometry (The Filling Stage)

14.6.1 Cystometry

This is considered the cornerstone of urodynamic testing and it refers to the measurement of intravesical bladder pressure during bladder filling, for the assessment of bladder storage function [19].

14.6.2 Technical Conditions in Preparation for Cystometry

Pressures: All systems are zeroed at atmospheric pressure prior to insertion with the pressure transducers at the level of the patient's pelvis.

External pressure transducers: Reference point is the superior edge of the pubic symphysis.

Catheter mounted transducers: Reference point is the transducer itself.

Initial bladder volume: Bladder should be empty.

Fluid medium: Usually sterile water or saline (or contrast if radiology is involved).

Temperature of fluid: Should ideally be warmed to body temperature.

Position of patient: The sitting position is more provocative for abnormal detrusor activity than the supine position. At some point in the test, filling might desirably take place with the woman standing.

Filling rate: The filling rate, including any changes during testing, should be noted on the urodynamic report [9, 16, 17].

14.6.3 Cystometrogram

The graphical recording of bladder pressures and volumes over time. Several standard parameters are evaluated during a cystometrogram, including bladder storage pressure, capacity, bladder sensation, bladder stability, and compliance [10, 16].

In preparation for this phase of urodynamics, both urethral and rectal (or vaginal) catheters are

placed. The bladder is then filled with contrast, saline or sterile water through the urethral catheter.

14.6.4 Intravesical Pressure (Pves)

This is the direct measurement of bladder storage pressure during cystometry. **Pves** is measured by the urethral catheter.

14.6.5 Abdominal Pressure (Pabd)

The intra-abdominal pressure (**Pabd**) is measured by a rectal or vaginal catheter. The simultaneous measurement of abdominal pressure is essential for interpretation of the intravesical pressure trace [16, 17].

14.6.6 Detrusor Pressure (Pdet)

This is the component of intravesical pressure created by the bladder wall (passive and active). **Pdet** is calculated by subtracting the abdominal pressure from the intravesical pressure [17]. Whereas the calculated Pdet represents the viscoelastic properties and tone of the bladder wall, when looking at the urodynamic study all three tracings (Pves, Pabd, and Pdet) should be evaluated to monitor for artifacts and other factors contributing to the Pdet tracing [1].

14.6.7 Bladder Sensation

This is assessed by questioning the woman during the filling cystometry about fullness of the bladder. Sensation during cystometry is subjective and can be influenced by the rate of filling, temperature of the fluid medium, position of the patient (supine vs. upright), and the patient's level of concentration [18].

Determining the volumes at which different degrees of fullness occur, the occurrence of pain during filling and evidence of decreased sensation during filling, may all be subtle predictors of disease processes. The greatest value of the cystometrogram with respect to sensation occurs when a symptom arises, and when sensation is correlated to actual Pves changes [28]. Understanding the accuracy of bladder sensation during the exam and its correlation with the patient's symptoms is a key factor in analyzing results, specifically in cases of OPF patients. The way patients describe their symptoms and the simulation of these sensations during cystometry allow the examiner to understand the objective pressure/volume measurements related to pain, pressure, or urgency symptoms. Sensations evoked during the urodynamic exam that are new to the patient or do not mirror her/his usual complaints should be probably ignored in most cases since they do not represent the etiology of the patient's symptoms.

14.6.8 First Sensation of Bladder Filling

The volume at which the patient first becomes aware of bladder filling.

14.6.9 First Desire to Void

The volume at which the patient first wishes to pass urine [16].

14.6.10 Normal Desire to Void

The volume at which the patient wishes to pass urine at the next convenient moment, but voiding can be delayed if necessary.

14.6.11 Strong Desire to Void

The volume at which a persistent desire to pass urine occurs, without the fear of leakage.

14.6.12 Urgency

A sudden, compelling desire to pass urine, which is difficult to defer.

14.6.13 Bladder Oversensitivity

Also known as "increased bladder sensation" or "sensory urgency" [17]. Increased perceived bladder sensation during bladder filling with an early first desire to void or an early strong desire to void, which occurs at low bladder volume or at low maximum cystometric bladder capacity, in the absence of abnormal increases in detrusor pressure.

14.6.14 Reduced Bladder Sensation

Diminished bladder sensation during filling cystometry.

14.6.15 Absent Bladder Sensation

Absent bladder sensation during filling cystometry.

14.6.16 Pain

The complaint of pain during filling cystometry. It is considered abnormal and needs to be investigated.

14.6.17 Bladder Capacity

Measured during urodynamics, it reflects the volume at which a subject with normal bladder sensation can no longer delay voiding [17]. This measurement is different from the functional bladder capacity, which is usually determined by the voiding diary, and the maximum anesthetic capacity, which is obtained under anesthesia [18].

14.7 Detrusor Function During Filling Cystometry

14.7.1 Normal (Stable Bladder) Detrusor Function

This refers to the accommodation of the detrusor to increasing bladder volumes without evidence of an involuntary detrusor contraction. If the detrusor is normal then there is little or no change in detrusor pressure with filling, and there are no involuntary phasic contractions despite provocation with activities (postural changes, coughing, hearing the sound of running water or hand washing) [15].

14.7.2 Detrusor Overactivity (Instability)

The occurrence of involuntary detrusor contractions during filling cystometry. These contractions, which may be spontaneous or provoked, produce a waveform of variable duration and amplitude on the cystometrogram. The contractions may be phasic or terminal. Symptoms like urgency and/or urgency incontinence may or may not occur. If a relevant neurological cause is present, then neurogenic detrusor overactivity is noted, otherwise the term idiopathic detrusor overactivity should be used [15].

14.7.3 Neurogenic Detrusor Overactivity

This defines detrusor overactivity with evidence of a relevant neurological disorder (according to the definition from the ICS) [15].

14.7.4 Bladder Compliance

The change in bladder pressure for a given change in bladder volume [18]. Compliance (C) is calculated by dividing the volume change (ΔV) by the

change in detrusor pressure (ΔPdet), $C = \Delta V / \Delta Pdet$ and is expressed as cmH_2O [16, 17].

A normal bladder displays low pressure during filling because of its viscoelastic properties. Bladder compliance can be affected by various factors such as bladder filling rate (faster filling is more provocative), contractile/relaxant properties of the detrusor, and starting and ending points for compliance calculations.

Loss of elasticity is a result of muscle being replaced by collagen and can be caused by a number of disease processes including neurologic conditions, prolonged catheter drainage, radiation therapy, prior pelvic or urethral surgery, interstitial cystitis, and obstructive uropathy [29]. A poorly compliant bladder displays an abnormal, often linear increase in Pdet during filling. This can result in dangerously high detrusor storage pressures. High storage pressures can distort the normal detrusor anatomy resulting in the development of vesicoureteral reflux and can be transmitted to the upper tracts, with development of hydronephrosis and renal failure. Early studies by McGuire and associates [30] have shown that sustained Pdet greater than 40 cmH_2O is specifically linked to renal or upper tract damage.

14.8 Assessment of Urethral Function During Filling Cystometry

14.8.1 Urethral Pressure Measurement

Urethral pressure and urethral closure pressure represent the ability of the urethra to prevent leakage. Normal urethral pressure value is higher than bladder pressure during non-voiding periods.

Urethral pressure is currently measured by a number of different techniques, which tend to lack consistent results either between methods or for a single method [31]. Urethral pressure might be measured at rest with the bladder at a given volume, during coughing or straining or during the process of voiding [10, 16, 17]. Urethral pressure values summarize all the components that participate in the continence mechanism and include also pressure of the pelvic floor muscles surrounding the urethra. The urethral pressure profile (UPP), maximum urethral pressure (MUP), and maximum urethral closure pressure (MUCP) may be used as part of the urodynamic evaluation for stress urinary incontinence.

14.8.1.1 Urethral Pressure (Intraluminal)

The minimal fluid pressure needed to open a closed urethra

14.8.1.2 Urethral Pressure Profile

The graph of urethral intraluminal pressure produced when urethral pressure is measured by a catheter drawn along the entire length of the urethra [16, 17, 29].

14.8.1.3 Resting UPP

The urethral pressure when the bladder and the subject are at rest.

14.8.1.4 Stress UPP

The urethral pressure measured during applied stress (cough, strain, Valsalva).

14.8.1.5 Maximum Urethral Pressure

The maximum pressure in the UPP.

14.8.1.6 Urethral Closure Pressure Profile (UCPP)

The pressure difference between the urethra and the bladder along the length of the urethra, measured by subtracting the simultaneous intravesical pressure from the measured urethral pressure.

14.8.1.7 Maximum Urethral Closure Pressure

The maximum difference between urethral pressure and intravesical pressure [10, 16, 17]. MUP is recorded on a graph. Maximum difference between urethral pressure and Pves is the MUCP, which is the most commonly used measurement of the urethra in current practice [11].

14.8.1.8 Functional Profile Length

The length of the urethra along which urethral pressure exceeds intravesical pressure in a woman.

14.8.1.9 Functional Profile Length (On Stress)

Functional profile length (on stress) is the length over which urethral pressure exceeds intravesical pressure on stress.

14.8.1.10 Pressure "Transmission" Ratio

The increment in urethral pressure on stress as a percentage of the simultaneously recorded increment in intravesical pressure. For stress profiles obtained during coughing, pressure transmission ratios can be obtained at any point along the urethra. If single values are given, the position of the catheter in the urethra should be stated. If several transmission ratios are defined at different points along the urethra, a pressure transmission "profile" is obtained. During "cough profiles," the amplitude of the cough should be stated when possible [10, 16, 17].

14.8.2 Urethral Closure Mechanism

14.8.2.1 Normal Urethral Closure Mechanism

The positive urethral closure pressure maintained during bladder filling, even in the presence of increased abdominal pressure, although it may be overcome by detrusor overactivity [17].

14.8.2.2 Incompetent Urethral Closure Mechanism

The leakage of urine that occurs during activities that increase intra-abdominal pressure, in the absence of a detrusor contraction.

14.8.2.3 Urethral Relaxation Incompetence (Urethral Instability)

Leakage of urine due to urethral relaxation, in the absence of increased abdominal pressure or a detrusor contraction.

14.8.2.4 Urodynamic Stress Incontinence

The involuntary leakage of urine during filling cystometry associated with increased intra-abdominal pressure, in the absence of a detrusor contraction [17].

14.9 Leak Point Pressures

This measurement is considered to evaluate the summary of the pressure components that cause opening of the urethra. Pressure values should be measured at the moment of leakage. There are two types of leak point pressure measurements.

14.9.1 Detrusor Leak Point Pressure (Detrusor LPP)

This static test measures the lowest value of detrusor pressure at which leakage is observed, in the absence of increased abdominal pressure or a detrusor contraction. High detrusor LPP (>40 cmH_2O) may put patients at risk for upper urinary tract deterioration or secondary damage to the bladder in the cases of known underlying neurological disorders such as paraplegia or MS [32]. There are no data on any correlation between detrusor LPP and upper tract damage in non-neurogenic patients.

14.9.2 Abdominal Leak Point Pressure

This dynamic test is defined by the ICS as the intravesical pressure at which urine leakage occurs due to increased abdominal pressure, in the absence of a detrusor contraction [15, 33].

Abdominal leak point pressure (ALPP) is used for the definition of intrinsic sphincter deficiency (ISD) and low abdominal LPP is suggestive of a poor urethral function, corresponding to more severe stress urinary incontinence [15]. The ALPP can be induced either by cough (cough leak point pressure = CLPP), or by Valsalva maneuver (Valsalva leak point pressure = VLPP). ALPP values can be used to quantify

the severity of stress urinary incontinence and this information can be used to guide treatment selection [16, 32, 33].

LPP values can be affected by many other factors such as the technique used to confirm urine loss, location of catheter, type of pressure sensor, bladder volume, rate of bladder filling, and patient position. The catheter size also has an important influence on the measurement of ALPP values and should be standardized. A trans-urethral catheter can partially occlude the urethral lumen, potentially distorting the value of the ALPP. Although there is no standard catheter size recommended for urodynamic testing, it is generally agreed that smaller catheters have less occlusive potential and are usually preferred.

Urethral pressure measurements are used to assess urethral competence and incontinence severity. Although it is fully accepted that urethral pressure is an important and integral component to urinary continence, it remains challenging to measure and characterize this pressure in a reliable manner [34, 35].

14.10 Pressure Flow Studies

Uroflow studies are good screening tests for identifying patients with low flow rates or abnormal voiding patterns, but they cannot identify whether this is caused by outlet obstruction or poor detrusor contractility.

Pressure flow studies are useful in determining a patient's voiding mechanism and the cause of the abnormal voiding, whether it's caused by low detrusor contractility or by bladder outlet obstruction [18]. A pressure flow study involves the simultaneous measurement of Pves, Pabd, and voiding flow and allows the calculation of Pdet during voiding. Flow is recorded as milliliters per second and Q_{max} is the highest flow rate recorded in the study [1].

The bladder is filled until the patient feels sufficiently full, and then the patient is asked to void. By measuring the Pdet during voiding, specifically at maximum flow, one can determine whether poor flow is caused by obstruction (high Pdet pressures) or whether it is caused by detrusor failure (low or absent Pdet). Similar to noninvasive uroflowmetry, flow rates and the pattern of the flow are also recorded during pressure flow studies.

14.11 Voiding Cystometry (Pressure Flow Studies)

Voiding cystometry is the pressure–volume relationship of the bladder during micturition [25]. It begins when the "permission to void" is given by the urodynamics technician and ends when the woman considers her voiding completed [17]. Measurements to be recorded should be the intravesical, intra-abdominal, detrusor pressures, and urine flow rate (Fig. 14.2).

14.11.1 Measurements During Voiding Cystometry

(a) Pre-micturition pressure: The pressure recorded immediately before the initial isovolumetric contraction.
(b) Opening time: The time elapsed from the initial rise in pressure to the onset of flow. This is the initial isovolumetric contraction period of micturition. It reflects the time taken for the fluid to pass from the point of pressure measurement to the uroflow transducer. Flow measurement delay should be taken into account when measuring opening time.
(c) Opening pressure: The pressure recorded at the onset of measured flow.
(d) Maximum pressure: Maximum measured pressure.
(e) Pressure at maximum flow: Pressure recorded at maximum measured flow rate.
(f) Closing pressure: Pressure recorded at the end of measured flow.
(g) Contraction pressure at maximum flow: The difference between pressure at maximum flow and pre-micturition pressure.
(h) Flow delay: The delay in time between a change in pressure and the corresponding change in measured flow rate [10, 16, 17].

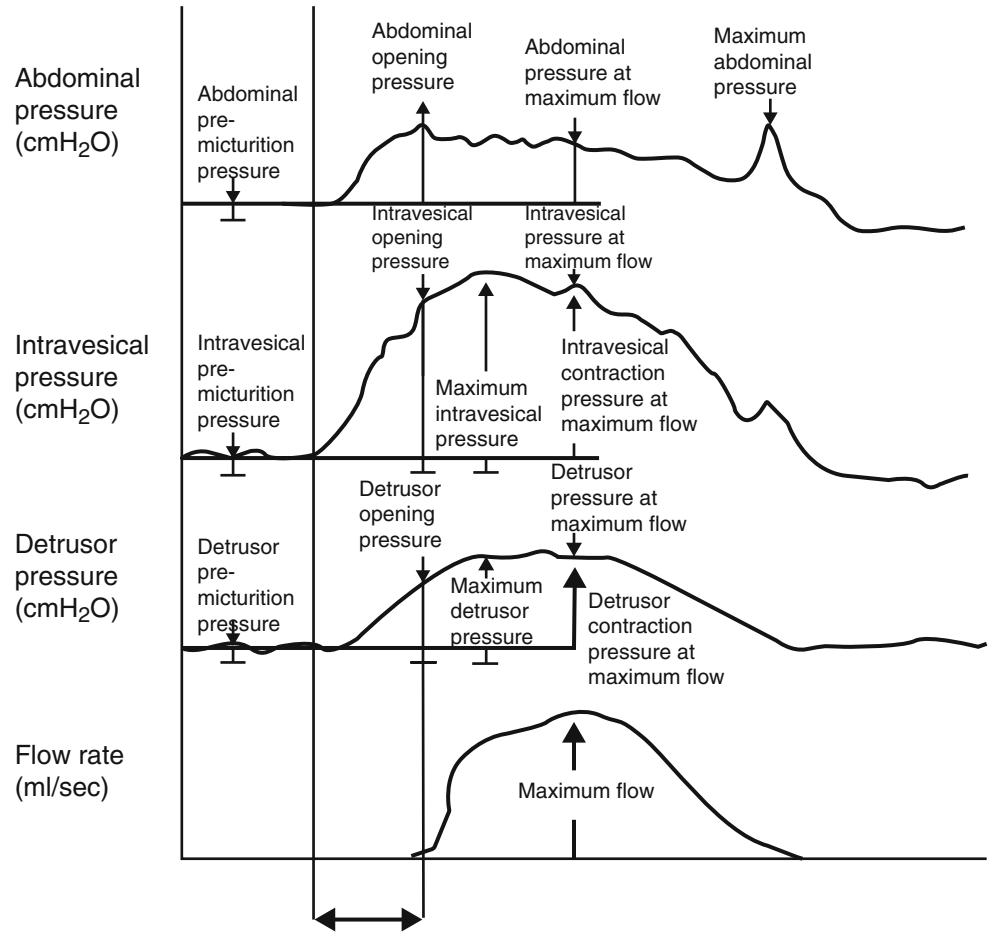

Fig. 14.2 A schematic diagram of voiding cystometry *With permission from Haylen BT, De Ridder D, Freeman RM, Swift SE, Berghmans B, Monga A, Petri E, Rizk DE, Sand PK, Schaer GN. An International Urogynecological Association (IUGA)/International Continence Society (ICS) joint report on the terminology for female pelvic floor dysfunction. Int Urogynecol J (2010) 21:5–26 © Springer 2010* [15]

14.11.2 Detrusor Function During Voiding Cystometry

Normal detrusor function: Normal voiding in women is achieved by an initial (voluntary) reduction in intra-urethral pressure (urethral relaxation) [36]. This relaxation is part of pelvic floor muscle relaxation, which simultaneously eliminates the inhibiting effect on the detrusor. This is generally followed by a continuous detrusor contraction that leads to complete bladder emptying. Many women will void successfully (normal flow rate and low PVR) by urethral relaxation, with a low rise in detrusor pressure [37]. The amplitude of the detrusor contraction will tend to increase to cope with any degree of bladder outflow obstruction (BOO) [38].

Detrusor under activity: 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. Occasionally patients will void by increasing intra-abdominal pressure compensating for detrusor under activity [16, 17].

Acontractile detrusor: The detrusor cannot be observed to contract during urodynamic studies, resulting in prolonged bladder emptying and/or a failure to achieve complete bladder emptying. The term "areflexia" has been used where there is a neurological cause [16, 17].

14.11.3 Urethral Function During Voiding Cystometry (Voiding Urethro-Cystometry)

This technique may assist in determining the nature of urethral obstruction to voiding. Pressure is recorded in the urethra during voiding. This may be at one specific point, e.g., high-pressure zone or it may be measured as a profile. A voiding urethral pressure profile (VUPP) uses a similar technique to that described above for the UPP measured during bladder filling. Simultaneous intravesical pressure measurement is required. Localization of the site of the intraurethral pressure measurement is useful [10, 16, 17].

Normal urethral function: The urethra opens and is continuously relaxed to allow micturition at a normal pressure, urine flow and PVR.

14.11.4 Bladder Outflow Obstruction

Bladder outflow obstruction is the generic term for obstruction during voiding. It presents as reduced urine flow rate and/or presence of a raised PVR and an increased detrusor pressure. It is usually diagnosed by studying the synchronous values of urine flow rate and detrusor pressure and any PVR measurements.

Outlet obstruction in women is most likely caused by pelvic organ prolapse, as a complication after surgery for stress incontinence, or from pelvic floor or external sphincter pathology such as detrusor-sphincter dyssynergia [18].

14.11.5 Dysfunctional Voiding

Dysfunctional voiding is characterized by an intermittent and/or fluctuating flow rate due to involuntary intermittent contractions of the periurethral striated muscles or levator muscles during voiding in neurologically normal women. This type of voiding may also be the result of an acontractile detrusor (abdominal voiding), with an EMG or videourodynamics required to distinguish between the two entities [15]. This is a common finding in patients with OPFS symptoms but can be used to differentiate different etiologies under this category.

14.11.6 Detrusor Sphincter Dyssynergia

Detrusor sphincter dyssynergia (DSD) is defined as lack of coordination between detrusor and urethral sphincter mechanism during voiding due to a neurological abnormality. This is a feature of neurological voiding disorders. Videocystourethrography is usually required to establish a diagnosis of DSD [15].

14.12 Electromyography

EMG is part of a standard urodynamic test that can be performed during cystometry and during a pressure flow study in order to evaluate the striated urethral sphincter and pelvic floor [18]. Sphincter EMG is an indirect measure of pelvic floor and urethral sphincter muscle contractility, as it measures depolarization of the sphincter muscle membrane [39]. Urodynamic questions that can be answered using this information include evaluation of outlet contraction and relaxation in relation to the timing of other components of the urodynamic study [1]. This measurement is important in the evaluation of patients with voiding dysfunction and specifically OPFS patients, who present with increased PVR volume.

EMG monitoring is most commonly done with either perineal surface-patch electrodes or needle electrodes, which are more invasive and uncomfortable. Needle electrodes (the gold standard) are able to isolate electrical activity from specific muscle fibers within a 0.5-mm

radius of the tip [40, 41]. They are, however, invasive, uncomfortable, and can be easily dislodged with movement. Patch electrodes are noninvasive and are the preferred technique in patients with OPFS symptoms. The patches are placed on the perineum, but the signal source may be inferior [40]. The striated urethral sphincter and levator ani are located in close proximity, but they are anatomically and neurologically discontinuous. Thus, perineal surface measurements may not accurately reflect striated sphincter activity, but a compounding of motor unit signals from all muscles of the pelvic floor [42].

A normal EMG study essentially rules out a neurogenic cause in patients with voiding dysfunction; however, an abnormal EMG study using the superficial patch technique may detect muscle spasm secondary to pain or anxiety [18]. In a normal urodynamic study, the sphincter EMG has baseline resting activity that may increase slightly as the bladder fills (guarding reflex). EMG activity will also increase with stress or Valsalva maneuvers. During the first phase of voiding, there should be cessation of activity as the urethral sphincter relaxes.

If EMG activity increases with voiding, this may represent detrusor-sphincter dyssynergia (DSD), dysfunctional voiding, or normal attempts to prevent voiding in the presence of an involuntary detrusor contraction [43].

14.13 Urodynamics in the Evaluation of Pelvic Floor Disorders

14.13.1 Indications

Urodynamic tests may provide better understanding of the underlying pathophysiologic mechanisms of urinary disorders. Although often considered an essential part of the evaluation and management of patients with lower urinary tract symptoms, the clinical use of urodynamics has not yet been clearly demonstrated [44]. Experts agree that the validity of urodynamics is linked to the patient's symptoms, and the ability to reproduce those symptoms during the testing session is controversial. Urodynamic test results should also be interpreted alongside information obtained from the entire clinical evaluation, which includes symptoms, medical history, physical examination, voiding diary, and other selected tests. When evaluating a patient with voiding dysfunction, it is important to have a specific set of questions in mind before performing urodynamics. As with any test, one should know what information he wants to obtain from the test before its execution and the limitations of the test [18].

Urodynamic tests are not necessary in the evaluation of every patient with lower urinary tract symptoms [45]. For instance, urodynamic tests are not recommended in the initial evaluation of uncomplicated female urinary incontinence when conservative treatments are planned [44, 46]. Urodynamic tests should be performed in cases with complex features, when diagnosis is difficult, and before more invasive therapy is considered [44].

Indications for urodynamic testing [18] in patients with urinary incontinence include the following:

- Urinary complaints with complex features
- Incomplete bladder emptying
- Failure of prior anti-incontinence surgery
- Symptomatic pelvic organ prolapse
- Associated neurologic condition
- Before initiating invasive therapy
- Symptoms do not correlate with objective findings
- Failure to improve after initial treatment

14.13.1.1 Stress Urinary Incontinence and Pelvic Organ Prolapse

Provocative cystometry is the key urodynamic test for the evaluation of stress urinary incontinence symptoms. According to the ICS terminology report [17] "urodynamic stress incontinence" (previously "genuine stress incontinence") is the observation of urethral leakage during increased abdominal pressure in the absence of a detrusor contraction. Studies suggest that cystometry has

varying sensitivity and specificity for the symptom of stress incontinence, depending on whether other symptoms (often mixed urinary incontinence) are present [18]. In addition to the observation of urodynamic stress incontinence, urethral parameters measured during cystometry may be used to aid in determining prognosis and for patient counseling. A UPP may be measured at rest with a urethral catheter by slowly withdrawing the catheter while measuring continuous pressures. The maximum urethral closure pressure (MUCP) has been reported to correlate with stress incontinence severity and with surgical outcomes in some studies but not in others and the reproducibility of UPP parameters is poor [44]. The location of the MUCP along the urethra might be related to the external muscular component of the urethral continence mechanism. In patients with OPFS the muscular component of this mechanism might play a role in dysfunctional voiding [18]. If leakage is present, abdominal or Valsalva leak point pressures can be measured [17, 47].

The presence and degree of prolapse may also be assessed. Women with severe prolapse may not have stress incontinence symptoms, but may develop them when the prolapse is reduced. Many recommend that urodynamic testing be performed in women with moderate to severe pelvic organ prolapse to evaluate for "occult" stress incontinence before surgery and to identify which patients should have a concurrent incontinence surgery at time of their prolapse repair. In such patients, cystometry may be performed with the prolapse reduced using a pessary, vaginal pack, or speculum [44].

14.13.1.2 Overactive Bladder

In 2002, the ICS defined overactive bladder as a symptom-based condition characterized by "urgency, with or without urge incontinence, usually with frequency and nocturia in the absence of other etiologies" [17]. This definition focuses on urgency, rather than urge incontinence, because it is increasingly recognized that approximately 2/3 of patients with overactive bladder do not have urge incontinence [48]. Overactive bladder symptoms are however poor predictors of urodynamic detrusor overactivity. Some studies suggest that sensitivity of clinical diagnosis is better than that obtained with urodynamic testing, because greater than 50 % of patients with a subjective history of urge urinary incontinence have normal urodynamic studies and conversely, detrusor over activity may be observed on cystometry in about 10 % of asymptomatic women [44]. Given this, the Third International Consultation on Incontinence recommends that invasive urodynamic testing is not needed before initiation of any conservative therapies in patients with uncomplicated overactive bladder and urge incontinence (no symptoms of voiding difficulties) [44]. Similar to the evaluation of stress urinary incontinence symptoms, urodynamic study is recommended in complex cases.

If symptoms are not improved with typical conservative therapies, urodynamics should be pursued to rule out other causes of incontinence, including poor compliance and occult stress incontinence. Furthermore, evaluation of the pelvic floor by EMG may identify dysfunctional voiding, an entity that may respond well to biofeedback.

14.13.1.3 Neurologic Lesions

Normal storage and evacuation of urine requires a complex interaction between neural centers in the brain, spinal cord, supraspinal and peripheral nerve centers. Interruption or insult at any level can cause voiding dysfunction, and the complex symptoms resulting from neurologic lesions and disorders, generally deserve full characterization using urodynamic tests [44]. Characteristic urodynamic findings may be seen, depending on the type of neurologic lesion involved.

14.14 Video Urodynamics

Videourodynamics or fluorourodynamics involve synchronous radiographic imaging of the bladder with multichannel urodynamic testing [1].

Fluoroscopy is used to offer dynamic images of the anatomy with maneuvers. It combines the use of fluoroscopy with the measurement of bladder and urethral pressures during cystometry or the

pressure flow study, and is desirable when simultaneous evaluation of structure and function is necessary in order to make a precise diagnosis [49].

It was originally called videourodynamic study because the information was recorded to videotape in 1970 [50]. Ultrasonography is an alternative imaging modality but not used widely. Although fluoroscopy is often useful, providers should limit the number of images that do not contribute to the diagnostic value of the study [51, 52]. For many urodynamic studies, the essential static images include one for each of the following stages of the urodynamic study: scout, filling phase low volume, Valsalva maneuver (dynamic) at approximately 200 mL (or lower in patients with decreased capacity), dynamic images during incontinence, cystometric capacity, during voiding and PVR. Additional images without the catheter may be needed during voiding if the patient is unable to void with catheter in place [51].

14.14.1 Indications

1. **Urinary incontinence associated with pelvic organ prolapse.**
   Videourodynamics is very helpful in the work-up of urinary incontinence associated with pelvic organ prolapse. Under fluoroscopy, the urethra and bladder neck are observed during the filling phase, looking specifically for whether or not the bladder neck is closed at rest, and for the position of the bladder in relation to the pubic symphysis. If significant prolapse exists, it may be necessary either to obtain fluoroscopic images with the patient in the oblique position to determine whether urethral hypermobility is present, or to repeat the images with either pessary or vaginal pack reduction of the prolapse. During stress maneuvers (straining and coughing), the bladder neck and urethra are evaluated for opening and leakage [18].
2. **Voiding dysfunction and not accurately vesicoureteral reflux**
   Videourodynamic testing is also useful in the evaluation of voiding dysfunction where it may be used to identify the presence of vesicoureteral reflux caused by poor bladder compliance [18]. In cases where reflux occurs at low volumes it may go unnoticed without the use of fluoroscopy. In cases of non-synchronized pelvic floor muscle relaxation, absence of the normal urethral voiding behavior can be noticed. Although less common in women, physical obstruction can be documented in cases of voiding dysfunction (Fig. 14.3).
3. **Bladder diverticula**

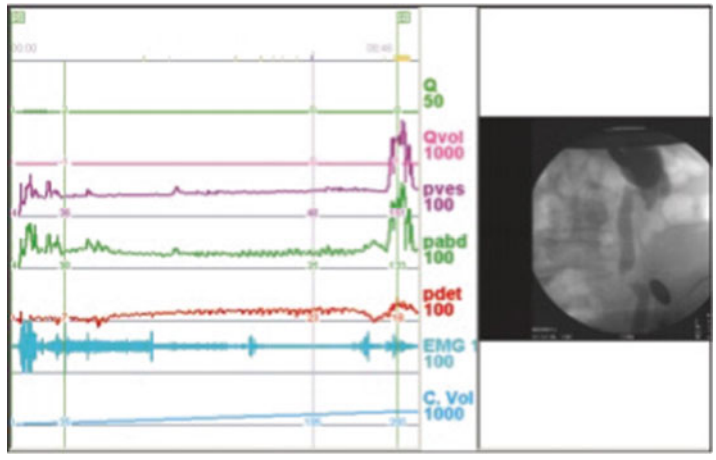

Fig. 14.3 VUDS demonstrated a vesicoureteric reflux. VUDS in a 69 y.o woman after Radical Hysterectomy presented with complanins of urinary incontinence, impered bladder sesation and difficult urination. VUDS revealed the bladder was contracted and the bladder neck as well as urethral sphincter was closed with bilateral vesicoureteral reflux. With permision: Hann-Chorng Kuo, MD. Incont Pelvic Floor Dysfunt 2008;2(3):117–118

Videourodynamics can be very helpful in identified bladder diverticula caused by chronic bladder outlet obstruction that may result in an artificially low Pdet during voiding and may not be recognized during urodynamics without fluoroscopy [18]. When there is a diverticulum and bilateral vesicoureteral reflux, the diverticulum fills more and reflux increases bilaterally with voiding.

14.14.2 Diagnosis of Specific Disorders Involving Voiding Dysfunction

As a concluding section of this chapter, we find it appropriate to describe two conditions in which pathological muscle behavior is involved and for which urodynamic testing plays a major role in diagnosis: Fowler's syndrome and DSD. When evaluating patients with overactive pelvic floor, these conditions are possible differential diagnoses and should therefore be ruled out through a detailed history, physical examination, urodynamic studies and Electromyography (EMG). A detailed description of the EMG technique is given in Chap. 12 of this text.

14.14.2.1 Fowler's Syndrome

This syndrome, which was first described in 1985 by Professor Clare J. Fowler, is considered to be the main cause for urinary retention in young women [54]. A detailed clinical description of this syndrome can be found in Chap. 8 of this book.

In Fowler's syndrome urinary retention is associated with a primary abnormality of the striated urethral sphincter [55], characterized by an abnormal sphincter electromyographic (EMG) signal displaying "complex repetitive discharges" [56] and elevated MUCP [57]. The abnormality that lies in the urethral sphincter prevents its relaxation and disturbs the normal urine flow. Most of the symptoms in Fowler's syndrome are caused by inability to empty the bladder. Some women may experience also back pain, suprapubic pain, or dysuria due to urinary infections [54].

Diagnosis: The gold standard diagnostic test for Fowler's syndrome is an external urethral sphincter EMG. On concentric needle EMG of the external urethral sphincter, these patients show a specific pattern of decelerating bursts and complex repetitive discharges [55, 58]. This abnormal finding is pathognomonic of Fowler's syndrome.

Other tests that might aid to the diagnosis are urine flow rate, residual volume, UPP, and sphincter volume measured by sonography (an overactive sphincter may be enlarge due to continuous muscle activity). Wiseman et al. [57] reported a significantly higher maximal urethral closure pressure (103 ± 26.4 vs. 76.7 ± 18.4 cmH_2O; $p < 0.001$) and a significantly higher external urethral sphincter volume (2.29 ± 0.64 vs. 1.62 ± 0.32 cm^3; $p < 0.001$) in patients with Fowler's syndrome.

14.15 Detrusor Sphincter Dyssynergia

Normal micturition is the result of a synergic action between the bladder smooth muscle (detrusor muscle) and the urethral sphincter striated smooth muscle. This process consists of relaxation of the urethral sphincter followed by a detrusor contraction, which expels the urine stored in the bladder [43]. This synergy between the detrusor muscle and the external urethral sphincter is controlled by the pontine micturition center. Disruption of pathways between this area and the caudal part of the spinal cord often results in detrusor-sphincter dyssynergia (DSD) [59].

The common causes for DSD are spinal cord injury, multiple sclerosis, acute transverse myelitis, and myelomeningocele [60–66]. DSD typically occurs in patients with a supra-sacral lesion. A few weeks after signaling breakdown, detrusor muscle contraction becomes synchronized with external sphincter contractions instead of relaxation and this condition is known as "DSD" [67].

The precise incidence of DSD is unknown [68], but spinal cord injury contributes the most significant portion of DSD. One report estimates that the incidence of DSD in patients with suprasacral spinal cord injury is 75 %, and the

incidence of DSD in MS and spinal dysraphism is estimated at 25–50 % [69–71]. Detrusor-sphincter dyssynergia has been defined as a detrusor contraction concurrent with an involuntary contraction of the urethral and/or periurethral striated muscle (Fig. 14.4).

Although the intra- and peri-urethral striated muscles are usually held responsible, the smooth muscle of the bladder neck or urethra may also be responsible [17]. Dyssynergia between the detrusor muscle and bladder neck refers to a condition characterized by a detrusor contraction, which is coincident with a failure in the opening of the bladder neck demonstrated objectively (Fig. 14.5).

Pelvic floor overactivity or dysfunctional voiding is the term used to describe the external sphincter urodynamic abnormality when it occurs in the absence of neurological disease. Bladder neck dyssynergia (BND) refers to incomplete opening of the bladder neck during voiding. BND has also been referred to as detrusor-internal sphincter dyssynergia, smooth-muscle sphincter dyssynergia and proximal sphincter dyssynergia when it is due to a neurological lesion. Both conditions should be clearly distinguished from the so-called dysfunctional voiding, which consists of striated sphincter hyperactivity in the absence of a detrusor contraction preventing adequate voiding, usually occurring in the absence of a demonstrable neurological condition [43].

Because detrusor-sphincter dyssynergia prevents adequate voiding, it might lead to a low compliance and thick-walled bladder, elevated retrograde pressures in the ureter and renal pelvis, hydronephrosis, renal scarring and terminal kidney failure [60]. Intermittent catheterization combined with anticholinergic medications to reduce detrusor pressures is the most common treatment for DSD [68]. Inability to catheterize and/or development of complications arising from DSD may lead to more aggressive interventions such as external sphincterotomy, intrasphincteric injection of botulinum type A toxin

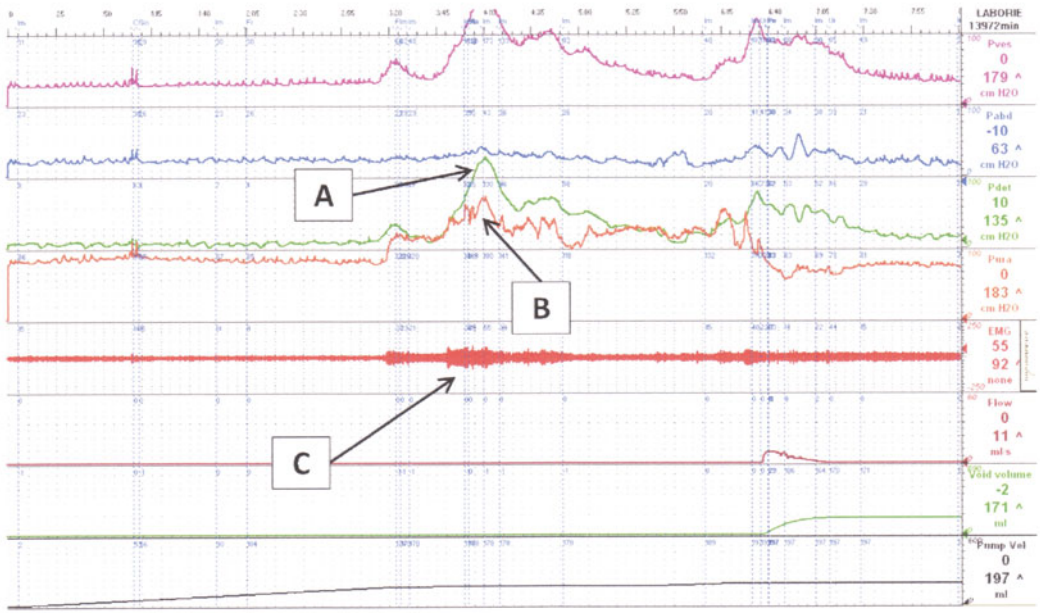

Fig. 14.4 DSD demonstrated by EMG. Strong voluntary detrusor contraction (Pdet) with (*B*) simultaneous increased external urethral sphincter contraction (Pura) and (*C*) increased EMG activity. *With permission from Lenherr SM, Quentin Clemens J. Urodynamics With a Focus on Appropriate Indicate ions. Urol Clin N Am 40 (2013) 545–557 © Elsevier 2013* [1]

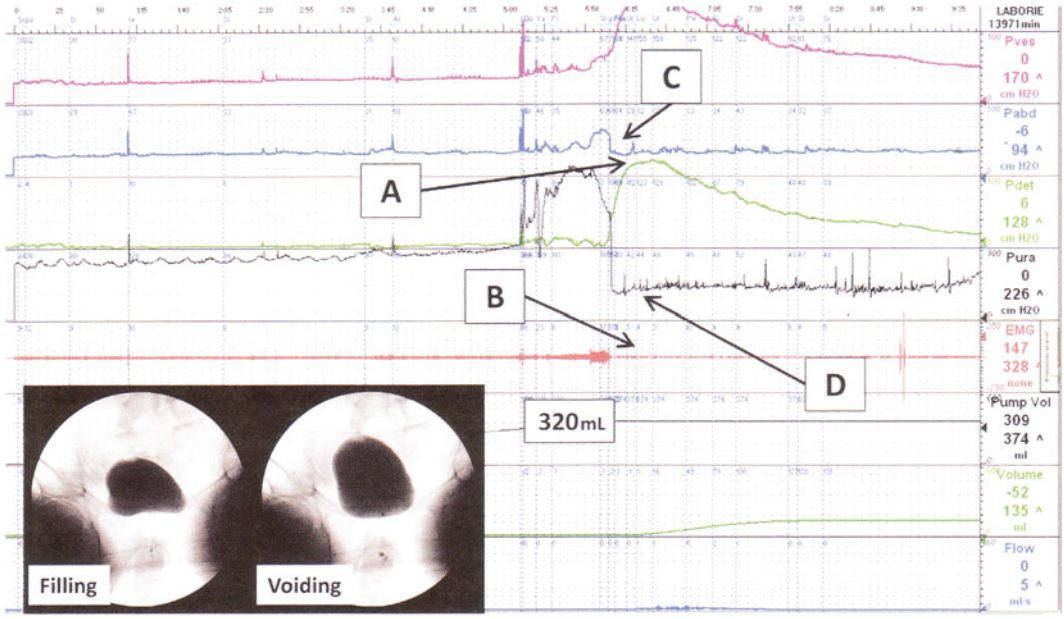

Fig. 14.5 Dyssynergia between detrusor muscle and bladder neck. (*A*) Voluntary detrusor contraction (Pdet) at 320 mL coincided with (*B*) decreased EMG activity and (*C*) no evidence of Valsalva voiding. Fluoroscopy showed the bladder neck did not open with voiding but (*D*) external urethral sphincter pressure (urethral pressure) did decrease. There was urine flow but with high voiding pressure (92 cmH_2O) and Q_{max} of 4 mL/s**.** *With permission from Lenherr SM, Quentin Clemens J. Urodynamics With a Focus on Appropriate Indicate ions. Urol Clin N Am 40 (2013) 545–557 © Elsevier 2013* [1]

and urethral stents [72–75]. As DSD often worsens over time and the chronological courses of many neurological conditions are unpredictable, routine life-long follow-up is required to prevent complications and preserve renal function [76, 77]. DSD can be diagnosed using electromyography (EMG), voiding cystourethrogram (VCUG), and/or UPP [77–80].

14.15.1 Diagnosis of DSD by EMG

This diagnosis requires sphincter over-activity, evidenced by elevated EMG activity during detrusor contraction in the absence of Valsalva and Crede' maneuvers [80, 81].

Diagnosis of DSD with EMG is poorly standardized because of variance in the type of needle, needle vs. patch electrode and electrode placement [80, 81]. Patch electrodes are often preferred for easier placement, patient tolerance and greater mobility [81]. Three types of DSD have been identified by Blaivas et al. [78] depending on EMG findings:

Type 1—Characterized by a simultaneous increase of detrusor pressure and external sphincter EMG activity that reaches its maximum at the peak of detrusor contraction. As the detrusor pressure begins to decline, sudden complete external relaxation occurs allowing urination.

Type 2—Characterized by clonic contractions of the external urethral sphincter interspersed throughout the detrusor contraction. Patients usually void with an interrupted stream.

Type 3—The external sphincter contraction persists throughout the entire detrusor contraction. These patients void with an obstructive stream or cannot void at all.

It has been shown that DSD tends to worsen over time and there is a correlation between the neurological status and the neurological examination. Patients with complete sensory and/or motor deficit have either type 2 or 3 DSD whereas patients with incomplete sensory and motor deficit have type 1 DSD [79]. Types 2 and 3 DSD are considered to have a greater risk of urological complication, as BOO is continuous throughout the detrusor contraction [79]. More recently, Weld et al. [60] has simplified the classification by dividing DSD into two type groups continuous vs. intermittent. Both classification systems are presently used.

14.15.2 Diagnosis of DSD by Voiding Cystourethrography or Videourodynamics

Urodynamic study play a critical role in the detection of DSD and monitoring for associated complications [81]. As EMG is not universally available, diagnosis is often made by voiding cystourethrography. Typical findings on voiding cystourethrography in DSD include a closed bladder neck during filling and subsequent dilation of the bladder neck and proximal urethra to the level of the external urethral sphincter during micturition [80, 81].

In patients with DSD, abnormal urethral images are seen during voiding in videourodynamics and revealed insufficient opening at the external sphincter (intermittent opening or diameter of <3 mm), narrow urethra (an insufficient opening of the entire urethra diameter <3 mm), and detrusor bladder neck dyssynergia (DBND) (an insufficient opening at the bladder neck, diameter <3 mm).

In patients with DSD, videourodynamics show a detrusor contraction with opening of the bladder neck, without concurrent relaxation of the external sphincter. In patients of BND, failure of bladder neck opening is observed [82]. The role of urethral pressure profilometry in the diagnosis of DSD is controversial, and is considered by the ICS to be investigational [31].

Pseudodyssynergia: This term defines the presence of external urethral sphincter contraction occurring during micturition that may be misinterpreted for DSD [74]. Causes of pseudodyssynergia may include abdominal straining, attempted inhibition of detrusor contraction or a response to pain.

Unfortunately, a perfect test for DSD does not exist. Pathology such as BOO, Parkinson's disease, and dysfunctional voiding should be considered before establishing a diagnosis, as they may have similar symptomatology.

In a recent study comparing needle EMG with VCUG, only 60 % agreement between needle EMG and VCUG was found for the diagnosis of DSD. This discordance seems to be due to the fact that BND prevents visualization of the external sphincter [31].

Also in other studies, diagnostic discrepancy between EMG and VCUG ranges from 40 to 46 % [60, 80]. Males were more often diagnosed using EMG whereas females were more often diagnosed by VCUG [60, 80]. It has been suggested that the diagnosis of DSD by VCUG in males may be impaired due to anatomical BOO by the prostate. In female patients diagnosis by EMG may be impaired due to increased electrode artifact [60]. A high index of suspicion, thorough history and clinical examination are necessary to ensure an accurate diagnosis. Urodynamic study using both VCUG and EMG for diagnosis of DSD increases the diagnostic success compared with either method alone [60, 80].

14.16 Conclusion

OPFS involves complicated multiple pathologic interactions involving all pelvic organs. Although these interactions are not completely understood, there is a consensus that bladder symptomatology plays an important part in the pathophysiology equation. The urodynamic test is the only dynamic study that simulates the bladder filling and voiding cycle with the related symptoms. While the inherent shortcomings of the study are recognized, this is still the only test that combines measurable volumes and pressures with assessment of patients' sensations. Performing urodynamic study in an OPFS patient is a unique challenge secondary to patient's anxiety and special sensory

needs. A thorough understanding of the patient's complaints prior to the study is required in order to provide the adequate environment, allowing an optimal study with meaningful results.

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Medical Therapies for the Treatment of Overactive Pelvic Floor

15

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15.1 Introduction

The pelvic floor is a highly compact area comprised of muscles, tendons, ligaments, fascia, and nerves. Overactive or contracted musculature in the pelvic floor is associated with several conditions such as vulvodynia and urinary incontinence, as well as symptoms such as dyspareunia, pelvic pain, and urinary frequency for which patients seek medical intervention. A number of factors are associated with pelvic floor muscle overactivity including a history of recurrent vulvovaginal infections, prolonged sitting, postural dysfunction, history of traumatic labor and delivery, urogenital cancer, history of physical trauma (such as a fall onto the coccyx or pelvic fracture), anxiety, sexual abuse, and constipation. Overactive pelvic floor (OPF) is understood to be a multifactorial condition requiring a multidisciplinary medical, psychological, and physical therapy team. This chapter will focus on medical therapies for OPF and will summarize the evidence-based data regarding medical interventions available for the treatment of pelvic floor overactivity.

15.2 Relationship between OPF and Symptoms

An OPF is related to several possible presentations that physicians are likely to encounter in the clinical setting. Patients describing symptoms of pain, paresthesias, and other sensorimotor pelvic complaints may be suffering from compression of nerves such as the pudendal nerve or any of its branches (dorsal clitoral, perineal, inferior rectal). Other presentations related to OPF include introital dyspareunia, pain with speculum insertion, urinary frequency, the sensation of incomplete emptying with urination, chronic constipation, and rectal fissures. In severe cases, pelvic floor muscle overactivity can cause nonprovoked, chronic vulvovaginal burning and pain (vulvodynia).

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15.3 Medical Assessment

Any evaluation of potential OPF should begin with a thorough history. Patients should be questioned about any of the following symptoms: vulvovaginal burning, throbbing, aching, soreness, introital dyspareunia, vaginal dyspareunia, urinary frequency, sensations of incomplete emptying with urinary, constipation, hemorrhoids, rectal fissures, pain with defecation, injury to the sacrum or coccyx, and injury to the lower back or hips. In addition, patients should be asked if they have a history of "holding urine," aggressive core muscle strengthening exercises, and a history of physical, emotional, or sexual abuse. Affirmative responses to any of these symptoms can be suggestive of OPF.

15.4 Physical Assessment

A brief screening exam can identify the majority of women with OPF and other types of pathologies that are frequently co-morbid with OPF. A description of this exam is as follows: a sensory exam of the vulva is performed using a moistened cotton swab to determine if there are areas that exhibit an abnormal pain response. Women with sexual pain can exhibit allodynia (i.e., the perception of pain upon provocation by a normally nonpainful stimulus such as being touched with a cotton swab) or hyperpathia (i.e., pain provoked by very light touch). This exam should be performed systematically to ensure that all areas of the anogenital region are tested. Initially, the medial thigh, buttocks, and mons pubis are palpated. These areas are typically not painful and this allows the patient to get comfortable with this exam [1]. The labia majora, clitoral prepuce, perineum, and interlabial sulci should then be palpated. Pain in these areas would suggest a process that is affecting the whole anogenital region including vulvar dermatoses, vulvovaginal infections, or neuropathic processes such as pudendal neuralgia. The labia minora are then gently palpated. First, the medial labia minora are gently touched lateral to Hart's line, which is the lateral boundary of the vulvar vestibule. The cotton swab is then used to gently palpate the vestibule at five locations: at the ostia of the Skene glands (lateral to the urethra), at the ostia of the Bartholin glands (4 and 8 o'clock on the vestibule), and at 6 o'clock at the posterior fourchette. Patients with vestibulodynia experience allodynia with the cotton swab test confined to the tissue of the vulvar vestibule but have normal sensation lateral to Hart's line. If the pain is localized to the vestibule, it is important to determine if the pain affects the entire vestibule or just the posterior vestibule as pain throughout the entire vestibule is an indication that there is an intrinsic pathology within the mucosa of the vestibular endoderm whereas, pain confined only to the posterior vestibule suggests that the pain is due to OFP [2].

A manual exam is then performed with one finger (instead of the usual two). The examiner's index finger is inserted through the hymen without touching the vestibule. The finger is firmly pressed downward towards the rectum approximately 2 cm proximal to the hymen. This should elicit the symptom of "pressure" or the "need to defecate" but not pain. Once the patient is normalized to the sensation of pressure, the pelvic muscles are examined. Moderate pressure is applied sequentially to the following muscles: coccygeus, iliococcygeus, pubococcygeus, puborectalis, and obturator internus. When each muscle is palpated, the patient should be asked "is this pressure or discomfort?" In addition, evidence of taught bands, knots, tender points, and trigger points should be noted. Discomfort during this part of the examination is highly suggestive of OFP. Then the urethra and bladder trigone are gently palpated. Intrinsic tenderness of the urethra may be suggestive of a urethral diverticulum or interstitial cystitis, while tenderness of the bladder may be suggestive of either interstitial cystitis or endometriosis.

The ischial spine is then located and the pudendal nerve is palpated as it enters Alcock's canal. Tenderness of the pudendal nerve is suggestive of pudendal neuralgia. Next, a bimanual examination is performed to assess the uterus and adnexa. Abnormalities in the size, shape, or

contour may be indicative of a leiomyoma. A diffusely enlarged, "boggy" and tender uterus may be signs of adenomyosis. Enlargement of the adnexa may represent an ovarian mass, whereas tenderness of the adnexa can often be a sign of a sexually transmitted infection, pelvic inflammatory disease, or endometriosis. A rectovaginal examination is then performed to assess the rectovaginal septum and the posterior cul-de-sac. Thickening or nodularity of the septum, nodularity of the uterosacral ligaments, can be suggestive of endometriosis [1].

If the aforementioned history and physical exam are consistent with a diagnosis of OPF, a referral to a skilled pelvic floor physical therapist is warranted for a more thorough musculoskeletal examination. Medications may be used alone, or as adjuvant treatment in combination with physiotherapy. The authors typically start with diazepam suppositories (Sect. 15.4.2 below) and systemic muscle relaxants such as cyclobenzaprine (Sect. 15.4.3 below) and reserve botulinum toxin type A (BTTA) (Sect. 15.4.1 below) for patients with more recalcitrant OFP. However, there have been no evidence-based algorithms published, therefore, decisions on medications must be made by taking into account each patient's medical history and concurrent medications.

The following medications have been described for the use of OFP.

15.4.1 Botulinum Toxin Type A

Botulinum toxin type A (BTTA) (Botox, Allergan, Irvine, California) has been shown to decrease muscle-related chronic pelvic pain. It does so by interfering with acetylcholine release at the neuromuscular junction, resulting in a decrease in resting tone and maximal contraction ability of the injected muscle. Due to its effectiveness as a paralytic, it is employed for widespread indications such as cosmetic enhancement and hyperhidrosis control as well as for relief from pain of musculoskeletal disorders. In a 2009 review article, Abbott evaluated gynecological indications for BTTA injections for women suffering from chronic pelvic pain. With the few case studies and research prior to 2009, Abbott concluded that vulvar pain may be reduced for a period of 3–6 months in women with provoked vestibulodynia after 20–40 unit injections of BTTA. Furthermore, Abbott compared the benefit of BTTA injection with pelvic floor physical therapy in women with pelvic floor muscle spasm. Those who received BTTA injection appeared to have no significant improvement in pain compared with participants who underwent pelvic floor physical therapy. Therefore, pelvic floor physical therapy is appropriate as an initial, minimally invasive intervention. However, if pain continues to persist despite pelvic floor physical therapy, BTTA injections are indicated as a next-step, more aggressive approach [3].

Another study explored the effects of BTTA on refractory myofascial pelvic pain [4]. Twenty-nine women participated in the study. Patients reported pain during digital palpation of the pelvic floor muscles using the 0–10 pain scale before and after levator ani BTTA injection (100–300 units). Seventy-nine percent of the participants reported improvement in pain on palpation post treatment, and 15 participants elected to undergo a second treatment, an average of 4 months after original injection. Few adverse side effects such as fecal incontinence, urinary retention, constipation and/or rectal pain were reported, all resolved spontaneously [4].

Moldwin and Fariello [5] analyzed the benefit of various myofascial trigger point (MTrP) injection therapy techniques, including BTTA, in treating urological pain syndromes. MTrPs are painful taut muscle bands (also known as "knots") that may create local and/or referred pain. The authors described three types of injection options to treat MTrPs, the first of which was a BTTA injection. The second group of women received intramuscular infiltration with a local anesthetic, lidocaine to deactivate MTrPs and to provide immediate pain relief. Lidocaine has been shown to have effects lasting from several hours to weeks and provide analgesic effect spreads to the tissue surrounding MTrPs, adding

additional therapeutic affect. The last approach, dry needling, involved fine acupuncture needle penetration into the tightest, most painful muscle fibers. All three approaches provided therapeutic benefit in the treatment of urological pain-related disorders with no statistically significant difference [5].

Finally, a study by Nesbitt-Hawes et al. [6] analyzed the benefit of single vs. multiple BTTA injections. Their study included 37 women between the ages of 21 and 52 who presented with at least two out of three criteria: pelvic floor muscle pain upon palpation, vaginal manometry pressure of greater than 40 cm H_2O and chronic pelvic pain. Pain was rated on the visual analogue scale (VAS) regarding dysmenorrhea, dyspareunia, dyschezia, and nonmenstrual pelvic pain. Pelvic floor manometry was performed with an air-filled vaginal probe. All 37 participants were administered 100 IU of BTTA diluted in 4 mL of normal saline. The BTTA was injected into two muscles: the pubococcygeus and puborectalis, bilaterally. Follow-up VAS and manometry assessments were performed at 4, 12, and 26 weeks after the initial injection. Twenty-six participants required only one injection. The remaining 11 participants were offered reinjections when their pain returned following an initial period of remission.

Of the women who received multiple injections, no major adverse side effects were reported. Furthermore, there appeared to be a cumulative effect of both decreased vaginal resting tone and maximal contraction strength. One woman reported vulvar irritation after her initial injection, and 23 women (35 %) reported flu-like symptoms during the 26-week follow-up period. There was no reported incidence of urinary or fecal incontinence. In addition, the researchers did not encounter the same antibody development that was observed in nongynecological BTTA studies, probably due to lower dosages (100 IU vs. ~6000 IU) and longer treatment intervals (>3 months vs. <3 months). In conclusion, multiple injections of BTTA are just as effective as the initial injection, and it is an appropriate treatment should pain return [6].

15.4.2 Diazepam Suppositories

In contrast to BTTA's paralytic properties, diazepam is a benzodiazepine drug that works as a skeletal muscle relaxant, anxiolytic, anticonvulsant and sedative, by enhancing the effects of the inhibitory GABAneurotransmitter. Its widespread effects have made it a drug of choice for many conditions including muscle pain, anxiety, seizures and alcohol withdrawal.

Recently, researchers have begun to explore the effect of diazepam on pelvic floor muscle dysfunction. In a 2010 retrospective study by Rogalski and colleagues indicated that vaginal diazepam suppository usage in high-tone pelvic floor dysfunction, in conjunction with pelvic floor physical therapy and trigger point injections, significantly helped relieve discomfort from pelvic pain. The researchers reviewed charts of 26 women who suffered from bladder pain, dyspareunia and hypertonicity of the levator ani muscles. Sexual dysfunction, including dyspareunia was measured with the Female Sexual Function Index (FSFI) and pain was assessed with a Visual Analog Scale for Pain (VAS-P). Twenty-five of the 26 participants reported subjective improvements, and six out of the seven sexually active patients were able to resume intercourse after suppository usage. On average, VAS-P levels decreased 1.44 on the 10-point scale [7].

In contrast, a 2013 study by Crisp et al. indicated that there is little to no benefit of intravaginal diazepam suppository in decreasing pelvic floor muscle resting tone. However, unlike the Rogalski study discussed above, women in this study were not allowed to use adjuvant treatments such as physical therapy, biofeedback, or trigger point injections. In this triple blinded placebo-controlled randomized trial, 21 women (average age 36.1 years old) with elevated pelvic floor EMG muscle resting tone (greater than or equal to 2.0 μV) were administered either 10 mg diazepam suppository or placebo for 28 consecutive nights. Outcome measures included vaginal surface EMG measurements, FSFI, the Short Form Health Survey 12 (SF-12) VAS, and the

patient Global Impression of Severity (PGI-S) and Improvement (PGI-I). After 4 weeks, no difference was seen regarding the EMG resting tone between the study and control groups, nor were any significant differences detected on the survey results. Consequently, the results of the study indicate that vaginal diazepam suppositories used in isolation, without any other treatment modalities, do not improve pelvic floor muscle resting tone [8].

15.4.3 Cyclobenzaprine

Cyclobenzaprine is a muscle relaxant whose mechanism of action is hypothesized to affect the alpha and gamma motor neurons of the central nervous system to decrease muscle spasm. Shekh and Kunka [9] performed a case study involving using cyclobenzaprine to treat a 26-year-old male who presented with a levator ani syndrome (LAS) symptoms of severe, episodic aching anorectal pain lasting 30–60 min, one to three times a day for 3 weeks. Other causes such as lesions, hemorrhoids, and anal fissure were ruled out. The patient was treated three times daily with 5 mg of cyclobenzaprine for 1 week and reported complete resolution of pain after only 3 days of treatment. The patient continued to remain symptom-free at the 6-month follow-up visit. The only adverse effect reported while on cyclobenzaprine was mild drowsiness, which spontaneously resolved upon discontinued use [9].

15.4.4 Topical Nitroglycerin and Topical Diltiazem

Nitroglycerin has been used for the past 130 years as vasodilator, especially to treat angina pectoris. Nitroglycerin is converted into nitric oxide by the enzyme mitochondrial aldehyde dehydrogenase and is available in multiple forms, including sublingual tablets, patches, sprays, and creams. More recently, nitroglycerin has been demonstrated to reduce musculoskeletal pain through its effects as a vasodilator. A literature review performed in 2011 by Garrick concluded that topical nitroglycerin (TN) significantly helps decrease pain due to acute tendon injuries [10].

Furthermore, Hashmi, Memon, and Khan [11] concluded that TN is an effective medication for chronic anal fissures, frequently caused by spasm of the anal sphincter and puborectalis muscle, and it should be included as a first line of treatment. Their prospective experimental study included 46 women and 46 men, with mean age of 30. Treatment consisted of application of 0.2 % topical TN for 8 weeks. Upon completion of the 8th week treatment period, 76 participants demonstrated anal fissure healing, whereas the remaining 20 participants demonstrated partial improvement in symptoms or nonhealing. The most common side effect reported by 21 participants was headache [11]. In addition, Mari et al. [12] compared the benefit of TN versus lidocaine in treating postoperative pain and anorectal muscle spasm after stapled hemorrhoidopexy procedure. In their single blind, parallel group, randomized controlled trial, the researchers randomly assigned participants to receive twice daily, topical application of either glyceryl trinitrate 0.4 % ointment or lidocaine chlorohydrate 2.5 % gel for 14 days. Pain intensity was measured on the VAS and was reported by participants on days 2, 7, and 14. On all three occasions, average pain scores were lower in the group treated with glycerin trinitrate compared to the group treated with lidocaine (Day 2: 2.5 ± 1.0 vs. 4.0 ± 1.1, $p < 0.0001$; Day 7: 1.4 vs. 2.8, $p < 0.0001$; Day 14: 0.4 vs. 1.4, $p = 0.003$). Furthermore, anal resting pressure was measured in both groups after 14 days of treatment, and the mean pressure was lower in the TN group (75.4 ± 7.4 mmHg vs. 85.6 ± 7.9 mmHg, $p < 0.0001$). The researchers concluded that TN is an appropriate intervention to alleviate poststapled hemorrhoidopexy-related pain and muscle spasm [12].

Another study conducted by Sajid, et al. [13] compares the benefit of topical TN vs. topical diltiazem (DTZ), a calcium channel blocker, to treat chronic anal fissures. In this systematic review,

the authors analyzed 481 patients from seven randomized controlled trials. 283 patients were treated with DTZ and 243 were treated with TN. The results revealed that both TN and DTZ were equally beneficial (RR = 1.10; 95 % CI = 0.90, 1.34; $z = 0.92$; $P = 0.36$); however, participants who were treated with DTZ demonstrated fewer headaches (RR = 0.39; 95 % CI = 0.24, 0.66; $z = 3.54$; $P < 0.004$) and recurrence (RR = 0.68; 95 % CI = 0.52, 0.89; $z = 2.77$; $P < 0.006$) [13].

In addition, Tsunoda et al. [14] performed an open-label, nonblinded, prospective study to assess the benefit of DTZ for the treatment of chronic anal fissures. They used The Short Form 36 Health Survey (SF-36) to assess quality of life changes before and 6 weeks after treatment. Participants also reported pain, bleeding, and irritation using numerical rating scales. After 6 weeks of treatment, 21 of the 30 participants (70 %) demonstrated healed fissures. These individuals also reported improvements in pain, health perception, and quality of life [14].

Despite the encouraging results of these aforementioned trials using diltiazem for anal fissures, there have been no published studies looking at its effectiveness as a treatment for vulvovaginal pain caused by pelvic floor dysfunction. It is possible to surmise, however, that since diltiazem helps anal fissure by relaxing the anal sphincter controlled by the puborectalis muscle, that it potentially could help relax other muscles of the pelvic floor.

15.4.5 Gabapentin

Gabapentin in an endogenous neurotransmitter which acts in part by modulating the action of glutamate decarboxylase (GAD) and branched chain aminotransferase (BCAT) to increase gamma-Aminobutyric acid (GABA) biosynthesis. Though approved to prevent seizures and as an anxiolytic, it is employed for many other uses including neuropathic pain. Gabapentin is recommended as a first line of treatment for diabetic neuropathy-related pain, central neuropathic pain, and postherpetic neuralgia. It is also used to treat anxiety disorders, bipolar disorder, restless leg syndrome, and insomnia. A retrospective study performed by Boardman et al. found topical gabapentin effective in women with vulvodynia. Participants had both generalized (37 %) and localized (63 %) vulvodynia, and were treated with 2–6 % gabapentin for at least 8 weeks. Average pain score decreased 4.77, from 7.26 to 2.49 on a ten-point pain scale, and the mean pain score among the 35 evaluable women was significantly reduced from 7.26 to 2.49. Approximately 80 % of participants experienced at least a 50 % reduction in pain. Furthermore, sexual function improved in the majority of the study group and all patients who had reported decreased participation in intercourse prior to treatment due to pain reported increased intercourse frequency post treatment [15].

Furthermore, gabapentin has been proven to be beneficial in other nongynecological, women's health-related musculoskeletal dysfunction. For instance, pre-operative usage of gabapentin has been shown to reduce post-operative pain in women undergoing total mastectomies due to breast cancer. Bharti et al. [16] performed a randomized controlled trial with 40 adult females, and participants were randomly assigned to study group (gabapentin 600 mg 2 h prior to surgery) or a control group (placebo). Not only did the group who received gabapentin report lower pain levels at 30, 60, and 120 min postoperatively ($p < 0.001$), they also required less intraoperative propofol during surgery compared to controls ($p = 0.009$) [16]. It is important to note however, this improvement is not evident in all types of surgery. Because men who undergo prostatectomy may develop pelvic floor muscle dysfunction or weakness following surgery, Deniz et al. [17] analyzed the effect of gabapentin administration prior to radical retropubic prostatectomy. They discovered that while post-operative pain scales were lower amongst those who received 900 mg of oral gabapentin 2 h prior to surgery, post-operative tramadol consumption at 24 h post surgery were similar [17].

15.4.6 Amitriptyline, Desipramine

Despite the limited use of topical amitriptyline, a tricyclic antidepressant (TCA), for treatment of gynecological disorders is a retrospective review of medical records was conducted by Poterucha et al. [18] to assess the potential benefit of topical amitriptyline (1–2 %) and ketamine (0.5 %) (TAK) for the treatment of rectal, perineal, and genital pain. The researchers amassed data from a single academic medical center in the United States and identified 1068 individuals who were treated with TAK between the January 1, 2004 and November 28, 2011. Of these individuals, 13 received treatment for rectal, perineal, or genital pain. One participant (8 %) reported complete relief after TAK application, six (46 %) reported substantial relief, four (31 %) reported some relief, and only two (15 %) reported no significant change. No significant adverse responses were noted [18].

Additionally, Pagano and Wong [19] performed a prospective study using topical amitriptyline 2 % in sorbolene cream on 150 patients with provoked vestibulodynia and dyspareunia. One hundred and two participants exhibited purely provoked vestibulodynia, and 48 participants suffered both provoked and unprovoked vestibulodynia. On average, participants had experienced symptoms for 4.7 years prior to inclusion in the study. Participants were encouraged to apply a pea-size volume of the cream to the vulvar vestibule twice per day for 3 months. Of the 102 participants in the first group, 84 participants (56 %) responded to treatment. 25 exhibited slight yet appreciable improvement, 44 exhibited moderate improvement, 15 (10 %) exhibited an excellent response defined as completely pain-free intercourse. The response rate was similar in the participants who experienced both provoked and unprovoked vestibulodynia, with 48 % reporting positive response to treatment [19].

Leo and Dewani performed a literature review regarding the effectiveness of oral antidepressant medication in treating vulvodynia. The study included two randomized controlled trials, 1 quasi-experimental trial, 7 nonexperimental studies, and 3 case reports. The majority of the 13 reports involved TCA treatment. The authors concluded that there was a lack of sufficient evidence to support the utilization of antidepressant medication for vulvodynia treatment. Additional research is warranted to identify which patients who present with vulvodynia are the most appropriate candidates for antidepressant pharmacotherapy [20].

In addition, Foster et al. published a randomized controlled trial comparing the effects of topical lidocaine in isolation, oral desipramine in isolation, and a combination of lidocaine–desipramine. They conducted a 12-week randomized, double blind, placebo-controlled trial with 133 participants. The outcome measure used was the level of pain reported during tampon insertion. At the end of the treatment, no significant difference was noted between all three intervention groups (both monotherapies as well as the combined therapy group), as compared with the placebo group [21].

15.4.7 Selective Norepinephrine Reuptake Inhibitors

Serotonin-Norepinephrine Reuptake Inhibitors (SNRIs) increase extracellular serotonin and norepinephrine in the central nervous system thereby helping to treat both depression and neuropathic pain. Milnacipran, an SNRI, has shown to help decrease chronic neuropathic pain and fibromyalgia-related pain. In a study by Ohnami et al. [22], dorsal horn spinal application of milnacipran resulted in prolonged inhibition of C-fiber evoked field potentials after establishment of spinal long-term potentiation. This was accomplished by activation of both the noradrenergic system as well as the spinal 5-hydroxytryptaminergic system. Results of this study deepened prior understanding of the potential usage of SNRI to treat chronic pain, suggesting further possibilities of SNRI treatment for chronic pain secondary to pelvic floor dysfunction [22].

Giannantoni et al. [23] studied duloxetine, another SNRI, in the treatment of chronic prostatitis/chronic pelvic pain syndrome (CP/CPPS) in men. The outcome measures utilized to measure

improvement were the National Institutes of Health Chronic Prostatitis Symptom Index (NIH-CPSI), the International Index of Erectile Function-5 (IIEF-5) questionnaire, uroflowmetry and the Hamilton Anxiety Scale (HAM-A) and Hamilton Depression Scale (HAM-D) to assess psychological status. Measurements were performed immediately after drug administration as well as 16 weeks post treatment. In this randomized controlled trial, the study group received multimodal treatment which included tamsulosin, saw palmetto and duloxetine (60 mg/day), whereas the control group received treatment consisting of equal amounts of tamsulosin and saw palmetto. At the 16-week follow-up assessment, NIH-CPSI pain, quality of life, and total subscores, as well as HAM-A and HAM-D were significantly improved in the study group compared to controls. The researchers concluded that the use of duloxetine, in conjunction with alpha-blocker medication and saw palmetto extract, enables improved CP/CPPS pain reduction, psychological well-being, and quality of life [23].

15.5 Conclusion

Pelvic floor overactivity rarely exists as an isolated condition and instead is related to several neurological, musculoskeletal, urological, oncological, gynecological, and gastrological presentations. As such, physicians in nearly all disciplines will encounter patients challenged with pelvic floor overactivity, which negatively impacts sexual functioning and quality of life. We have presented several options that physicians may consider in offering medical treatment regimens as part of a multidisciplinary approach to the treatment of OPF.

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A Classical Physical Therapy Approach to the Overactive Pelvic Floor

16

Amy Stein and Mary Hughes

16.1 Introduction

Pelvic floor (PF) muscle dysfunction is a global term that incorporates malfunctioning of the musculoskeletal and neuromuscular systems. While the process for determining its underlying cause can be complex, the dysfunction is often a result of a structural malalignment of the skeletal system [1–3], of hyperactivity or hypoactivity of a muscle or muscle group, or of restrictions in the tissues and fascia [4–8]. One may suffer from both overactive and underactive pelvic floor dysfunction, and it is essential to address both for this complex presentation. For instance, a postpartum female may present with stress incontinence related to an underactive or weak pelvic floor condition, as well as dyspareunia, related to overactive pubococcygeus and transverse perineal muscles. In order to address the weakness without causing further muscle shortening, the pelvic floor muscles require lengthening to function at an optimal position, before they are strengthened with pelvic floor exercises. Such traumas as a slip and fall, childbirth, infections, and poor posture can contribute to these structural changes and muscle imbalances.

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Pelvic floor muscles assist with voluntary sphincter control of the bladder and bowel and with sexual performance and function. They provide support for pelvic organs and assist with lumbopelvic stability and mobility. It is estimated that 80 % of pelvic floor muscle fibers are Type I slow-twitch skeletal fibers; 20 % are Type II fast-twitch skeletal fibers. As a result, symptoms of bladder, bowel, and/or sexual dysfunction often accompany PF dysfunction. Pain in the pelvic and abdominal regions, in the back and lower extremities, and in the genital area as well as central sensitization are often correlating findings of PF dysfunction. These symptoms can start as early as childhood and present up until the elder years.

In recent years, there has been an increase in research, specifically regarding treatment techniques [9–19]. It has been described that pelvic floor physical therapy can be an integral and extremely effective treatment for the musculoskeletal causes of functional pelvic pain [20–24]. In this chapter we will review manual therapies, reeducation for both the bladder and bowel, neuromuscular reeducation, posture/body mechanics, therapeutic exercise, and modalities including biofeedback, low-level laser therapy (LLLT), ultrasound, electric stimulation, transcutaneous electrical nerve stimulation (TENS), and heat and cold therapy. This list by no means represents all the treatments available nor does it reflect the research in its entirety.

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It should be noted that to successfully treat pelvic floor dysfunction, it is essential to treat the body as a whole and to address all the biopsychosocial conditions contributing to the patient's pelvic pain. A treatment plan must be constantly monitored by the therapist, and the patient's progress consistently re-evaluated in order to advance the treatment plan [25] and modify it as warranted.

16.2 Manual Therapy Techniques

Myofascial release
Myofascial trigger point release
Connective tissue manipulation
Scar mobilization
Neural mobilization
Visceral manipulation
Joint mobilization

16.2.1 Myofascial Release

It is estimated that a myofascial dysfunction afflicts as many as 23 % of women with chronic pelvic pain (CPP) [26]. Myofascial Release (MFR), a holistic therapeutic approach to manual therapy, was developed by John Barnes as part of a comprehensive approach to the evaluation and treatment of the myofascial system [27]. As the term implies, the main tissue to target for release in the myofascial system is the fascia, described by Barnes as "a tough connective tissue which spreads throughout the body in a three dimensional web from head to foot without interruption." The fascia surrounds all structures in the body including muscles, nerves, vessels, and bones. MFR is reported to be an excellent technique for releasing such restrictions as trigger points, muscle tightness, and dysfunctions in soft tissue that may cause pain and limit motion in all parts of the body [28].

In the MFR process, the therapist evaluates, identifies, and treats fascial restrictions. Such restrictions may be caused by trauma, musculoskeletal conditions, repetitive stress syndrome, or poor posture. The gentle, hands-on techniques the MFR therapist applies to the whole body can bring about positive structural changes, increasing range of motion (ROM), reducing pain, and augmenting fascial mobility [28].

Barnes' approach teaches the therapist to evaluate the fascial system through visual analysis of the human frame's three-dimensional space, by palpating the tissue texture and various fascial layers, and by observing the symmetry, rate, quality, and intensity of strength of craniosacral rhythm.

Craniosacral rhythm is the use of light touch to perform delicate mobilization of the cranial bones and sacrum. This subtle hands-on treatment facilitates the release of connective tissue tightness surrounding the brain and spinal cord, or the Central Nervous System (CNS). This light touch can help to create balance within the CNS, by inhibiting the Sympathetic Nervous System (flight or flight response), and activating the Parasympathetic Nervous System (rest and digest), it reduces stress within the body, decreases pain, and promotes normal function. In addition to assessing the craniosacral rhythm, it is important to have the treating therapist "observe the vasomotor response and their location after a particular fascial restriction has been released. This provides instantaneous and very accurate information enabling the therapist to proceed intelligently and logically from one treatment to the next" [27].

MFR at first releases the elastic component of the fascia; at some later point, the collagenous barrier will be engaged. This barrier cannot be forced. The therapist maintains gentle pressure for some 90 to 120-plus seconds, and as the collagenous barrier releases, the therapist follows the motion of the tissue until all the barriers are released [28].

16.2.2 Myofascial Trigger Point Release

Travell and Simons define a trigger point as a highly irritable spot in a palpable taut band in the muscle or the fascia [8]; according to Travell, a true trigger point is a restriction that has a clear and consistent referral pattern [7, 8]. When pressure is applied, pain or tenderness is often provoked along with the possibility of autonomic nervous system involvement. The autonomic

nervous system plays a role in the regulation of internal organs. Sweating, vasoconstrictions, vasodilation, and cutis anserine are possible responses to trigger points linked to the autonomic nervous system. McPartland and Simons reported the ANS may indirectly exacerbate myofascial trigger points formation via viscerosomatic reflexes [29].

Muscles containing trigger points are characteristically shortened and present with decreased ROM, hyperactivity, incoordination, and substitution patterns [4, 5, 30]. Muscles with trigger points present must expend greater effort than muscles without trigger points to produce the same effects; this may cause changes in surrounding muscles, and it may also disorder the proprioceptive, nociceptive, and autonomic functions of the affected region [31].

It is well documented that trigger points may produce pelvic pain; these trigger points can be found in the levator ani muscle group, obturator internus, coccygeus, abdominals, gluteals, adductors, iliotibial band, tensor fascia latae quadricep, piriformis, quadratus lumborum, paraspinals, and hamstrings [8].

Trigger points in the posterior pelvic region result in symptoms in the rectum, anus, coccyx, and sacrum. Trigger points in the anterior portion may result in more urogenital pain and symptoms. Dyspareunia and bladder and bowel dysfunction can be the result of pelvic floor trigger points and can present with sharp, dull, aching symptoms both deep and superficial [4, 5, 8, 30, 32–34].

Yet trigger points may often go unrecognized. For this reason, it is essential that a skilled practitioner actively palpate the tissue in order to find and treat the restriction. Once identified, the trigger point muscles may be eased and lengthened through stretching and/or with such proprioceptive neuromuscular facilitations, as contract relax, reciprocal inhibition, and active release techniques [5, 13].

Research has found that myofascial trigger point release can achieve up to an 83 % improvement in symptoms; it can lessen the overactivity of the pelvic floor musculature, which in turn can reduce neurogenic bladder inflammation and decrease CNS sensitization [33]. A research study by Fitzgerald et al. concluded that a group of women with interstitial cystitis/painful bladder symptoms responded with significantly higher improvement to myofascial physical therapy, achieving an overall improvement in symptoms, than to global therapeutic massage [18]. Success has also been achieved in the treatment of pelvic pain and bladder, bowel, and sexual dysfunction through the use of many of these manual therapy techniques [5, 7, 13, 18, 31, 32].

16.2.3 Connective Tissue Manipulation

Connective tissue manipulation is the movement of one layer of skin over the other—skin rolling—in order to release tension in the tissue, thereby augmenting the ROM of the joint and restoring neurodynamics [7]. The manipulation creates the sensation of a sharp scratch or of a nail digging at the skin. The more restricted the tissue, the sharper the sensation, and the patient may complain of feeling bruised [24]. Connective tissue manipulation improves circulation to areas with decreased blood flow and pelvic congestion. When tension is released, blood flow to the area increases, flushing toxins from that region. This also improves mobility in the surrounding structures [32]. The therapist will note any changes to the skin texture, color, temperature, and elasticity [24].

16.2.4 Scar Tissue Mobilization

Any restrictions throughout the trunk, pelvic floor, sacral, and lower extremity regions that could be causing restrictions and overactivity contributing to the patient's pain should be addressed through scar mobilization. For abdomino-pelvic floor disorders, it is essential to address both the superficial scars that are visible—i.e., from Caesarian sections, episiotomies, and any abdominal surgeries—and those not visible but formed internally either by a surgical procedure or by such conditions as endometriosis. Some studies suggest releasing the scar with each physical therapy treatment until normal flexibility returns and there is no more adherence to deeper tissues [4].

16.2.5 Neural Mobilization

The theory behind neural mobilization is that in improving axonal transportation, nerve conduction velocity is increased [28]. Manual neural mobilization restores altered neurodynamics by balancing the relative movement of neural tissue and surrounding mechanical interfaces, thus reducing intrinsic pressure and optimizing physiological functions [35]. Treatment is indicated if there are signs of pain due to increased resistance of the tissues and reproduction of symptoms [7]. The therapist will use various techniques to palpate and reduce restrictions in any tissue and musculature that could be restricting the nerve. In addition, neural glides can help free restrictions of the nerve.

16.2.6 Visceral Manipulation

Just as the skeletal system needs to be in proper alignment to maintain homeostasis, so do the viscera. Adhesions, abnormal tone, or displacement may result in a disharmonious movement between internal organs, and this disharmony may in turn lead to chronic irritation and pain. As Carriere and Feldt write, "The viscera of the pelvic floor needs to be very flexible to adapt to the daily filling of the bladder and rectum and to the monthly changes in the endometrium" [36].

Visceral manipulation uses gentle palpation and manual therapy to evaluate and correct the imbalances, returning the organs to their appropriate position and stimulating arterial and venous blood supply so that the organs return to optimal functioning [36]. Please refer to Chap. 21 for more information on visceral manipulation.

16.2.7 Joint Mobilizations

Joint mobilization is a manual therapy intervention, a type of passive movement of a skeletal joint. When applied to the spine, it is known as spinal mobilization. In a patient with pelvic pain, a structural deviation within the skeletal system can often be resolved with these techniques, which decrease muscle guarding, lengthen the tissue surrounding a joint, and affect neuromuscular influences on muscle activity and increased proprioceptive awareness [28].

16.3 Neuromuscular Reeducation

As per O'Sullivan and Schmitz, PNF, which is one of the many techniques of neuromuscular reeducation, focuses primarily on the facilitation of total patterns of movement and posture; this promotes learning—reeducation—in synergistic muscle groups. Pelvic floor muscle weakness, incoordination, adaptive shortening, joint immobility, and alterations in muscle tone may lead to impaired patterns of posture and movement. By applying PNF techniques to the pelvic floor and surrounding structures, those muscles and structures may be strengthened, lengthened, and coordinated [37].

These techniques include *contract-relax* to promote relaxation at a point of limited ROM [37] and *strain-counterstrain*, a form of passive positional release designed to release connective tissue restrictions. In this latter technique, the tissue is initially moved in the direction of ease that shortens the affective structure. According to Jones and Randall, "The purpose of movement toward shortening is to relax aberrant reflexes that produce the muscle spasm forcing immediate reduction of tone to normal levels. This allows the joints influenced by the now relaxed muscle to function optimally increasing its ROM and easing muscle pain. Strain and counterstrain is an effective but extremely gentle technique because its action for treatment moves the patient's body away from the painful, restricted directions of motion" [38].

16.4 Pelvic Floor Muscle Retraining

Pelvic floor muscle retraining requires developing or improving motor control for bladder, bowel, and sexual function and to ameliorate pelvic pain [39, 40]. Patients diagnosed with CPP, dyspareunia, or any urgency-frequency dysfunction often present with overactive pelvic floor

muscles [30]. Pelvic floor muscle overactivity may also be the result of holding patterns, which, over time, can result in shortened muscle fibers, restricted connective tissues, and/or contracted sarcomeres [4].

Behavioral therapy needs to be integrated into the treatment approach in order to break the cycle of dysfunction and pain. Education and training must focus on proper motor control—that is, on relaxing the pelvic floor muscles in voiding and intercourse rather than contracting or tensing the muscles out of fear of pain. Such training entails "learning" the difference between contracting, elongating, and relaxing the pelvic floor through verbal cueing and such other forms of cueing as biofeedback. The techniques for this retraining follow principles of the operant learning model and of cognitive behavioral therapy [41, 42]. In addition, it may be helpful to do any or several of the following behavioral modifications before or after any painful activity, including sexual activity: scheduled voiding, urge control, posture re-training, and pelvic floor muscle relaxation and massage. Such modifications have been shown to be extremely effective in pelvic floor rehabilitation [43, 44].

Various forms of "home remedies" have also been shown to be effective and can be applied by the patient. Stress management, relaxation breathing, pelvic floor muscle relaxation, relaxing time with a walk, hot bath, yoga, and meditation are all examples of behavioral or lifestyle modifications that can help with physiological quieting [43]. Two particularly effective techniques are the use of a dilator, or internal massage device, to help eliminate myofascial trigger points and to further elongate the shortened pelvic floor muscles [41, 44], and passive stretching of the tissues at the vaginal introitus [45].

16.5 Bladder and Bowel Reeducation

Pelvic floor muscles need to be relaxed during urination and defecation, which is why regulating bladder and bowel function is an essential component of the treatment of pelvic pain. For those diagnosed with chronic overactive pelvic floor muscles, getting those muscles to relax and lengthen is often a multi-step process that can be helped by both manual therapies and reeducation techniques. For example, proper positioning is key when voiding. Leaning forward and slightly extending the spine decreases the anorectal angle facilitating bowel movement. Depending on the height of the toilet, it may be necessary to place a stool under the patient's feet to allow for optimal positioning [9]. In addition, taking time on the toilet and allowing oneself time to relax can help avoid the straining that can put a lot of adverse tension on the pelvic floor muscles. Manual therapies, as well as such techniques as imagery, deep breathing, and biofeedback, may all assist in the reeducation of the muscles to address post-void dribble, bladder and bowel frequency, urgency, retention, and hesitancy [43, 46]. Here are some key techniques and tips for addressing these issues.

16.5.1 For Bladder Symptoms

- Eliminate dietary irritants
- Do not restrict water intake; rehydrate to progressively help expand bladder capacity
- Do not push out the urine (it should flow naturally)
- Timed voiding
- Delay the urge to urinate
- Double void
- Don't hover over the toilet
- Breathing with voiding

16.5.2 For Bowel Symptoms

- Increase intake of soluble and insoluble fiber to avoid constipation
- Increase water intake
- Urge delay if patient has bowel frequency
- Use a squatty potty/steps for proper positioning
- Breathing techniques to help relax the pelvic floor and abdominal muscles
- Avoid straining
- Colon massage and abdominal splinting

16.6 Posture/Body Mechanics

One common observation in patients with pelvic pain is that they tend to sit on one ischial tuberosity—often with minimal support on the chair—with legs flexed and crossed with no lumbar lordosis. Pelvic alignment was the biggest difference between women with complaints of pelvic pain and those without [47]. Patients with pelvic pain had a difficult time with single leg stance longer than 10 s [47].

Pelvic and abdominal dysfunction, including increased tone and/or weakness, can result in core instability and faulty postures [1, 2]. According to Howard et al., "Faulty postures are those positions that increase the stress to the joints and use excessive muscle activity. Viewed this way, posture is a mechanical state and when body mechanics are altered in a way that is less functional, the end result is dysfunction and pain." Typical pelvic pain patients present with faulty posture, which is a "contributing cause of weak, deconditioned muscles allowing for imbalances in the pelvis with formation of trigger points and overactivity, and as a result, pelvic pain" [48]. In order to correct these faulty postures and resulting dysfunctions, certain muscles and tissues may need lengthening while others will need strengthening. A healthcare provider with extensive training in the musculoskeletal system can determine what the needs are for each individual patient.

16.7 Therapeutic Exercise

Core strengthening and stretching exercises are also key components when addressing pelvic floor dysfunction.

16.7.1 The Core

Cough, sneeze, or laugh and the pelvic floor and abdominal muscles contract in concert with the action. This normal relationship among the pelvic floor, deep back muscles, diaphragm, and abdominal muscles can, in people with pelvic floor muscle dysfunctions, have adverse effects. The normal co-contraction when one coughs, sneezes, or laughs can be painful when there are active trigger points or irritation in the abdominal/pelvic musculature—including scarring, injury, or inflammation [4].

Certainly, a strong core is essential for mobility and stability of the spine, pelvis, shoulder, and hip girdle and to create a solid base of support for the body as a whole; it is the foundation for facilitating movement from the center of the body out to the extremities. Weakness in the core muscles or an imbalance in the musculoskeletal system can give rise to several different dysfunctions in the back, pelvis, sacroiliac joint, ligaments, discs, and hip joints and may eventually lead to other disorders such as osteoarthritis, degenerative disk disease, incontinence, and prolapsed pelvic organs. And while it is advisable to maintain strong core muscles—diaphragm, trunk, pelvis, and abdomen—through therapeutic exercise, such exercises should *not* be performed in muscles and tissues containing any trigger points, fascial restrictions, hypertonicity, skeletal malalignment, or an acute injury. Only when the dysfunction is resolved should patients return to building core strength through therapeutic exercises, and even then, care should be taken to not increase any pelvic, abdominal, or back pain.

Returning the patient to a neutral postural alignment is extremely important in order to allow for a proper length and tension relationship with the surrounding musculature. Stretching is key to maintaining the proper length and tension. According to Haugstad et al., the most significant restrictions to proper length and tension were found in the iliopsoas, straight abdominal muscles, and femoral adductors in women with CPP [47].

16.7.2 Stretching

The key to healing is letting go of the tension, and that is what stretching can help to achieve. Just as we stretch leg or arm muscles when they feel tight, we can address the tension in the pelvic floor with stretching. Foam rollers and/or a trigger point and MFR ball can help decrease the

tension within the pelvic floor and surrounding musculature. Stretching the muscles surrounding the pelvis—abdominals, hip flexors, extensors, abductors, adductors, and rotators—may also decrease the tension on the pelvic floor. According to a study by Viera and Costa, "The physiological effects of stretches may continue to reduce discomfort and pain" [20].

Vaginal dilators are useful to stretch the internal muscles of the pelvic floor. Vaginal dilators can play an important role in overcoming pelvic floor muscular response that remains and sometimes increases even after pain perception has decreased [41, 49, 50].

16.8 Treatment Modalities

A range of other modalities can complement or augment the primarily manual skills of pelvic floor physical therapy.

EMG Biofeedback provides instantaneous, performance-dependent, visual and/or auditory feedback that measures muscle activity and in so doing helps to increase self-awareness and teach proper muscle coordination. Electrode sensors positioned internally in the vagina or rectum or placed around the anal opening detect muscle activity; the activity is represented on a screen so that the patient can see the difference between what the muscles do during rest versus their appearance during activity. This visual feedback helps the patient learn how to downregulate overactive muscles or re-train pelvic muscle incoordination, and this in turn can help reduce the patient's pain [10, 12, 33, 51].

There are many occasions, however, when the biofeedback shows a low resting tone even though the patient's pain is extremely high. Typically, this occurs in patients with shortened pelvic floor muscles, which may not appear to be overactive on the EMG [4, 5]. For this reason, it is imperative to use manual techniques *with* the biofeedback for these patients. A comprehensive review of EMG biofeedback is provided in Chap. 28.

LLLT is a hand-held modality that assists in healing various pain conditions. LLLT emits photon light without heat, causing bio-stimulation at the cellular level and helping to accelerate the healing process. It uses power densities lower than those needed to heat tissue and low-intensity wave lengths in either scanning or spot form. LLLT may eliminate the trigger points common in pelvic pain by increasing local microcirculation, thus bringing more oxygen to the cells and helping reduce inflammation. It can also be used to temporarily relieve minor muscle and joint aches, reduce pain and stiffness, decrease muscle spasms, break up scar adhesions, and increase lymph flow; some of the research is contradictory, however, with conflicting results due to varying durations of symptoms and differing treatment techniques, parameters, and machine specifications [52–54].

Therapeutic ultrasound is the use of a transducer or applicator that transmits low-intensity and low-frequency sound waves, causing a warming and stimulation of the tissues that leads to vasodilation and thus delivers more blood and oxygen to the affected area. Maximum energy absorption in the soft tissue occurs from 2 to 5 cm beneath the skin's surface, with the intensity decreasing as the waves penetrate deeper. The transmission is absorbed primarily by connective tissue, ligaments, tendons, fascia, and scar tissue. The results are an increase in blood flow in the treated area and a decrease in pain from the reduction of swelling and edema [55].

Ultrasound has been used with success for perineal tears/episiotomies resulting in dyspareunia following vaginal delivery [56], and, in one study in which results were moderate, for interstitial cystitis [57].

Electrical stimulation can be used internally and externally. It works by interfering with the electrical currents of pain signals, inhibiting them from reaching the brain where they would normally induce a pain response. For pelvic pain and overactive pelvic floor muscles, electrical stimulation can help relax muscle spasms, increase local blood circulation, rehabilitate and reeducate muscles, maintain and increase ROM in the tissues, and manage chronic and intractable pain. A small amount of research supports the use of electric stimulation for pelvic and vulvar pain [58–62].

TENS is the application of mild electrical stimulation using skin electrodes placed near to or distant from an area of pain; it acts by interfering with the transmission of painful stimuli—the pain gate theory. Some studies have shown that high-frequency TENS is more effective for pain relief than placebo TENS, while low-frequency TENS is no more effective [63]. There have been many studies on the positive analgesic effects of TENS on chronic, non-pelvic pain conditions [64] and of TENS on dysmenorrhea, pelvic pain and overactive pelvic floor disorders [62, 65–67].

Hot and cold therapies are helpful modalities that have been shown to provide temporary reduction of pain [68, 69]. *Heat* helps to relax tight muscles and thus decrease pain caused by muscle tension or spasms; heat also vasodilates the blood vessels, which increases circulation to the area and helps promote healing. A small, controlled study of patients with anorectal pain showed positive therapeutic effects—compared to people without pain—of anal resting pressure while the patients were immersed in a warm bath [70]. *Cold therapy* causes vasoconstriction of the blood vessels in the area where the cold pack is applied. This helps to decrease the inflammation in the area, thus also decreasing pain. While this suggests that cryotherapy may help with pelvic pain, to date no studies have been done.

16.9 Conclusion

Manual therapy, neuromuscular reeducation, bladder and bowel training, biofeedback, therapeutic exercises, and modalities are all components of physical therapy that can be used as interventions for the effective treatment of pelvic floor muscle overactivity [2, 9–19]. A plan for such treatment properly begins with an evaluation performed by a pelvic floor specialist; this provides the information needed to individualize the plan of care for the patients' dysfunction.

Manual therapy, including MFR, myofascial trigger point release, connective tissue manipulation, scar mobilization, neural mobilization, visceral manipulation, and joint mobilization, as well as patient education, are important components of treatment. By informing the patient about proper posture, body mechanics, and body awareness, education becomes an essential support for successful treatment. Moreover, neuromuscular reeducation is the key to retraining the musculature for optimal positioning and therefore for maximal efficiency—especially in the pelvis where bladder and bowel re-training can reduce the pain and increase the efficiency of voiding and defecation.

Pelvic floor dysfunction can appear at any point throughout an individual's lifespan. As the research increasingly demonstrates, physical therapy can reduce, if not eliminate, the need for surgical interventions and in so doing can save both time and money.

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17 An Alternative Physical Therapy Approach to the Overactive Pelvic Floor

Dee Hartmann

17.1 Visceral Impact on Pelvic Floor Function

17.1.1 Introduction

Viscerally triggered pain is a familiar phenomenon. An impending heart attack may elicit familiar warning signs of chest, left arm, jaw, and back pain along with nausea, vomiting, and anxiety. Diarrhea is often preceded by gastric cramping and pain just as chronic constipation may contribute to worsening low back pain. That feeling of "butterflies in the stomach," though not pain per se, is a viscerally mediated sensation related to fear, anxiety, and nervousness. Premenstrual cramping and lower abdominal pain can be the result of repeated uterine contractions occurring to shed the endometrial lining each month with menses.

The central premise of treating the viscera via visceral manipulation is that the interrelationship of structure and function among the internal organs is at least as strong as that among the constituents of the musculoskeletal system. Much like the musculoskeletal system, manipulation of the viscera can be beneficially used in the treatment of internal organ dysfunction. Visceral manipulation is an ancient, hands-on, gentle technique currently utilized by multiple medical specialists, including but not limited to osteopaths, chiropractors, physical therapists, and body workers. In the 1970s, Drs. Jean-Pierre Barral and Pierre Mercier, French osteopaths, developed a framework of concepts and techniques based on extensive clinical experience as well as studies utilizing radiology, ultrasound, and postmortem reviews. Many of the references mentioned in this chapter are based on the study of their work [1, 2].

When visceral function has gone awry, it is often difficult to determine what physical symptoms may have preceded the presenting dysfunction. If the problem was short-lived, for instance, a urinary tract infection (UTI) that lasted for only a few days, there may or may not have been associated changes in the fascial, muscular, and/or visceral systems around the bladder. However, if the dysfunction was long standing, such as in a recurrent UTI that appeared to continue unresolved for 4 months, the tissues in and around the area of the bladder are more likely to become symptomatic. For instance, the organs, themselves—the urethra, bladder, ureters, and kidneys—may show signs of elevated tension as the functional symptoms of dysuria and urinary urgency and frequency persist. Other structures often join in—the pelvic floor and associated

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muscles, the fascia within the anterior and posterior pelvis, the rectum, and the uterus—by exhibiting increased tension or spasm. However, from another perspective, perhaps there was underlying pelvic floor muscle (PFM) over activity or hypertonicity imposing pressure on the urethra before the complaints of urinary urgency and frequency began. It is possible that preexisting PFM over activity may, in turn, have contributed to reactive urethral and/or bladder spasm. The initial perception of urinary urgency and frequency that is typically included in the differential diagnosis for UTI, may, in fact, be a result of abnormal muscular tension in or near the urethra rather than an active infection. This conceptualizes how the over active PFMs may have created or exacerbated the physical symptoms that mimic an actual infectious condition.

It is far beyond the scope of this chapter to discuss the neuroanatomy and neurodynamic hypotheses proposed for the etiology of visceral pain. Equally, the chapter's purpose is not to instruct practitioners in assessment and manual treatment techniques associated with the viscera. Educational opportunities exist around the world though the Barral Institute (www.barralinstitute.com) as well as osteopathic colleges and continuing education providers. Rather, the chapter's purpose is to suggest how pelvic visceral dysfunction may contribute to pelvic floor muscle dysfunction (PFMD) and overactivity.

17.2 Functional Anatomy of the Pelvis

To understand the functional anatomy of the pelvis, it is essential to appreciate the integration of static support provided by the bony pelvis along with several additional anatomical systems: the musculoskeletal, the fascial, and the visceral systems. To better define pelvic function, Wei and DeLancey suggest that the descriptor "pelvic floor" (PF) should be used to refer to all the structures of support within the pelvic cavity, including the musculature. Pelvic floor support begins with the abdominal peritoneum cranially and continues inferiorly through the viscera (bladder, urethra, uterus, and rectum), the endopelvic fascia, the deep PFMs, and the perineal membrane before ending caudally in the superficial portion of the PFMs (Fig. 17.1) [3, 4]. Together, the levator ani (pubococcygeus, puborectalis, and iliococcygeus muscles) and coccygeus formthe PFMs. The total sum of the muscular attachments of the PFMs, the fascial attachments of the viscera (e.g., pubocervical and uterosacral ligaments), and the static support of bony pelvis combines to form the functional and structural support for the pelvic contents.

Normal functioning PFMs (i.e., the ability to actively and fully contract and relax) contribute to the most inferior support of the pelvic viscera, control of continence via input to the urethral and anal sphincters, maintenance of normal sexual function and orgasmic activity, and core stability at the base of the trunk. The quality of the pelvic support can be altered by abnormalities in any of the supportive systems, be it in the PFMs (e.g., PFM spasm or laxity), the fascia (e.g., uterine or bladder prolapse), or the viscera (e.g., urethral or rectal spasm).

From an anatomical perspective, the sling-like support of the PFMs attaches anteriorly at the inferior pubic rim and travels posteriorly to attach at the coccyx and sacrum. Normal contraction of the PFMs shortens the length of the muscles, creating a lift to the perineal body, pulling it up and in toward the pelvic cavity. Normal relaxation returns the muscles to their original length, allowing the perineal body to drop to its original position. In supine and with normal PFM function, the position of the perineal body should be above the plane of the ischial tuberosities. Hypotonic (i.e., laxity or decreased tone) PFMs allow the perineal body to drop below the plane of the tuberosities whereas hypertonic (i.e., elevated tension or spasm) PFMs cause a pull upward of the perineal body, keeping it more superior to the ischial tuberosities than in normal function. When hypertonicity is present, the resting position of the perineal body is further upward into the pelvis, similar to where it would be at the end of a full, voluntary contraction of normal functioning PFMs. The presence of chronic

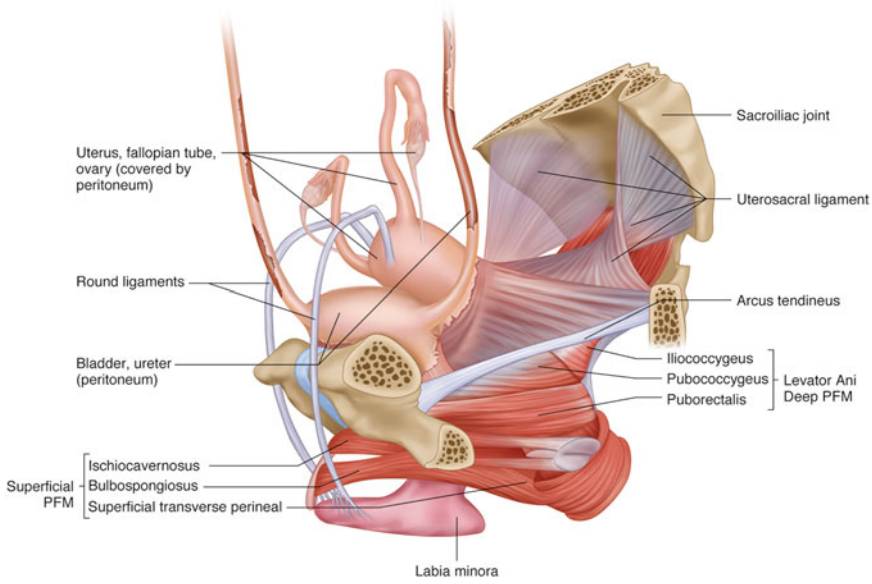

Fig. 17.1 The pelvic floor structures according to DeLancey and Wei begin superiorly at the peritoneum and continue inferiorly throughout the pelvic viscera, endopelvic fascia, levator ani muscles, perineal membrane and superficial genital muscles. *PFM* pelvic floor muscles. Modified from Primal Pictures with labels added (images copyright Primal Pictures Ltd., http://www.Primalpictures.com)

over activity leaves the PFMs unable to release and return the perineal body to a normal position. This chronic over activity can be visualized easily during an external clinical exam of the perineum. Other major components within the anterior compartment are the paired obturator internus muscles. They are fan-shaped and originate from a broad section of the anterolateral wall of the pelvis at the inner surface of the obturator foramen, the ischiopubic ramus, and the inner surface of the femur. Their fibers narrow and become band-like as they traverse inferior, running posterior to the ischial tuberosities where they make a 120° turn upward to insert at the greater trochanters of each femur (Figs. 17.2 and 17.3). Their function is to externally or laterally rotate the hip with extension (i.e., to turn the toes and knee outward in standing) and to abduct the hip when flexed (i.e., drop the knee out to the side when the knees and hips are flexed while in the supine position).

17.3 Pelvic Floor Dysfunction

Chronic pelvic and vulvar pain pelvic floor dysfunction (PFD), and PFMD typically, but not always, coexist. There are rare clinical cases that present with no physical findings; however, those cases are, by far, not the norm. Such complexity is elucidated by the European Association of Urology in their "Guidelines on Chronic Pelvic Pain." They list 15 definitions relating to female pelvic pain, implicating involvement of PFMs, bladder, urethra, uterus, vagina, vulva, clitoris, pudendal nerve, rectum, and perineum [5]. Visceral disorders within the pelvis that are known to contribute to pain include painful bladder syndrome/interstitial cystitis (PBS/IC), irritable bowel syndrome (IBS), dysmenorrhea, and endometriosis. Chronic, abnormal stimuli that are present with these visceral pain(disorders can slowly

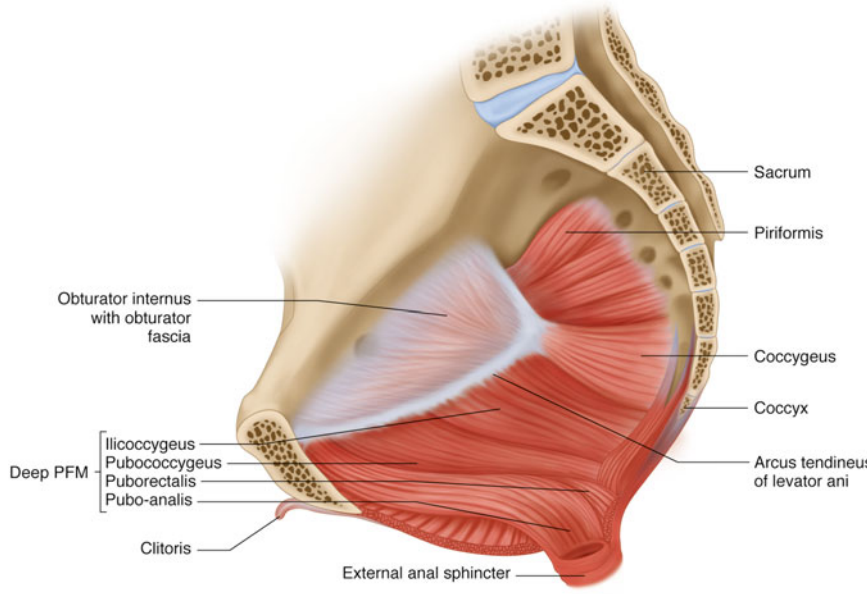

Fig. 17.2 Medial view of the pelvic floor muscles. *PFM* pelvic floor muscles. Modified from Primal Pictures with labels added (images copyright Primal Pictures, http://www.primalpictures.com)

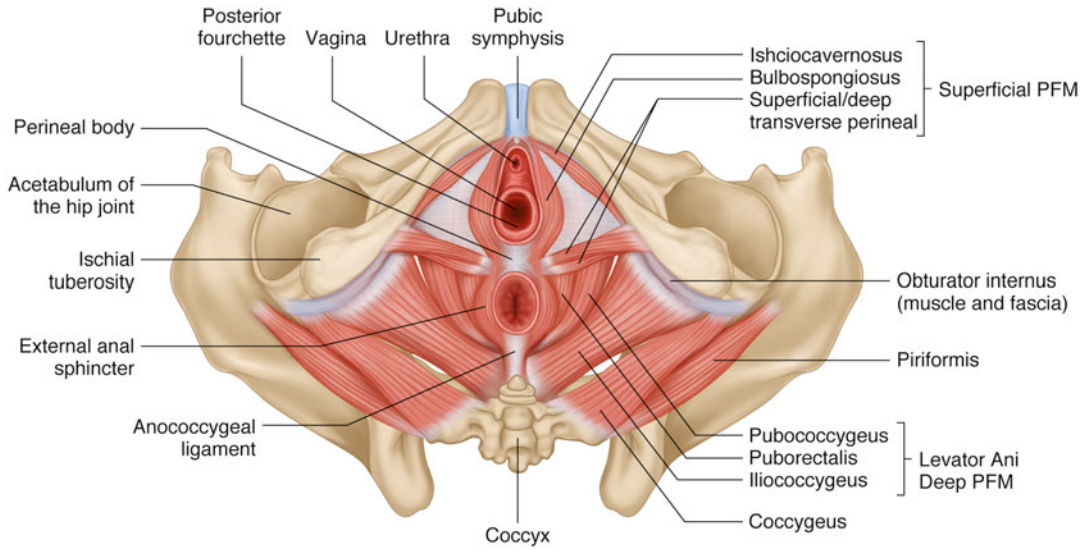

Fig. 17.3 Inferior view for pelvic floor muscles with associated muscles and structures. *PFM* pelvic floor muscles. Modified from Primal Pictures with labels added (images copyright Primal Pictures, http://www.primalpictures.com)

upregulate the spinal cord, disrupting sacral reflexes that regulate sensation and pain [6]. Fascial laxity and resulting organ prolapse may contribute to the pain puzzle, as can bony irregularities (e.g., sacroiliac joint dysfunction and hip pain).

17.4 Pelvic Floor Muscle Dysfunction

Understanding pain related only to PFMD can be equally confusing as it has received multiple labels over time—coccydynia, levator (spasm) syndrome, tension myalgia of the PF, PF spasticity, urethral/anal sphincter dyssynergia, vaginismus, and shortened PF [7]. The progression of PFMD occurs in two stages: the first, neuromuscular, and the second, musculodystrophic. Following some injury or insult (e.g., coccygeal injury with a fall, chronic hip pain, or recurrent yeast or UTIs), free calcium is released, disturbing the sarcoplasmic reticulum and causing over activity within the muscle. In the presence of ATP, calcium ions stimulate the actin/myosin activity, increasing metabolic activity. The release of various neurotransmitters, including serotonin, histamine, kinins, and prostaglandins, stimulate muscle nociceptors and set up a neural circuit between the central nervous system, nociceptors, and motor units [8]. Over time, the hypertonic muscles enter the musculodystrophic phase while attempting to adjust to the overall increase in metabolic activity. When that adjustment fails, localized fibrosis begins and atrophied muscle tissue is replaced by less metabolically active and extensible connective tissue [9]. An example of this phenomenon can be seen when bladder and urethral function are impacted by PFMD. Once PFM over activity becomes chronic and full range of motion is reduced, the tension may obstruct voiding or make it impossible, with severe cases requiring intermittent self-catheterization. Not only will the PFMD cause restriction in the urethra but it can also inhibit the detrusor during bladder filling and emptying, resulting in urinary urgency, frequency, and hesitation [10]. Patients will involuntarily contract their PFMs for extended periods in response to urinary urgency, reflexively inhibiting bladder filling and emptying. With time, the PFMs lose their flexibility and are unable to normally relax. It then becomes impossible to separate the visceral from the muscular dysfunction as each drives the other: more muscular tension creates more urgency and increased urgency creates more PFM tension. The vicious cycle begins (Table 17.1).

17.5 Physical Therapy Assessment

17.5.1 Medical History

The intake interview includes a traditional physical therapist's review of biomechanical systems but adds additional questions related to bowel and bladder function (including intake of fluids), menstrual and vaginal health, and a full history of sexual function, including questions on desire, arousal, and orgasm (Table 17.2). Because of the coexistence of PFM and visceral dysfunction, it is imperative to get a full and comprehensive history that includes a pelvic organ system review. It is often impossible to ascertain which came first: the PFMD that caused the pelvic organ functional complaints (e.g., PFM over activity compromising the ease of bowel movements resulting in a complaint of constipation) or the chronic visceral dysfunction that was driving the PFM tension (e.g., chronic diarrhea that caused PFM over activity). Regardless of the initiating trigger, the bowel dysfunction must be treated along with the PFM dysfunction to reach maximum success with treatment.

17.5.2 Physical Assessment of the Pelvic Viscera

Physical therapy assessment of the musculoskeletal system in those with PFD has been addressed in previous chapters. Because of the interrelationship of visceral to PFM function, assessing the tonicity of the pelvic organs is a necessary addition to the traditional PT assessment.

In women with PFMD, the more common anterior pelvic findings include some combination of the following: elevated PFM resting tone, taut bands and/or active trigger points in the PFM, elevated tone of both obturator internus muscles, increased tension in one or both tendinous arch(es) of the pelvic fascia and/or the levator ani, and increased tone or visceral spasm in the urethra, bladder, uterus, or rectum. Abnormal internal tension or tone is digitally sensed as a stiffness or bulkiness in the tissues being assessed. When palpating the urethra, for

Table 17.1 Possible causes of pelvic floor muscle dysfunction (PFMD)

Faulty biomechanics	1. Dysfunction of the lumbosacral spine or pelvic girdle—hips, pelvis, pubic symphysis, sacroiliac joints, sacrococcygeal joint
	2. Lower kinetic chain irregularities—knees, feet, subtalar joints
	3. Hypermobility throughout the pelvic cavity
Postural and structural dysfunction	1. Scoliosis
	2. Short leg syndrome
	3. Small hemi-pelvis
	4. Perpetuation of typical pelvic pain posture—anterior pelvic tilt, increased thoracic kyphosis, lumbar lordosis
Injury to the pelvic floor muscles	1. Childbirth
	2. Pelvic surgery
	3. Falls with landing on sacrum or coccyx, uncontrolled "slip" or stepping unexpectedly off a curb, creating a shear force at the pubic symphysis
	4. Repetitive movement injuries seen with high velocity sports with altered postures (e.g., gymnastics, dance)
Faulty cumulative behaviors of the pelvic floor muscles	1. Repetitive minor trauma or straining with chronic constipation or urinary obstruction
	2. Abnormal chronic holding patterns
	(a) History of sexual abuse
	(b) Sexual guilt
	(c) Pain with first attempted tampon use, penile intercourse, or vaginal exam
	(d) Urinary urgency or frequency
	(e) Urinary or fecal incontinence
	(f) General stress, anxiety, tension
	(g) Traumatic toilet training, history of bedwetting
	3. Prolonged constriction or extended sitting with unequal weight bearing (e.g., occupational posture) or lack of motion (e.g., long car rides)
Inflammatory pain disorders involving pelvic viscera	1. Irritable bowel syndrome (diarrhea, constipation)
	2. Endometriosis
	3. Chronic cystitis, painful bladder syndrome
	4. Dysmenorrhea

instance, the assessing digit would be at 12:00 in the vaginal canal where the urethra is supported by the periurethral fascia behind the pubic symphysis. With normal tone, the musculature of the urethra is collapsed on itself and feels like a soft, shallow mound of tissue running approximately 4 cm in length along the backside of the symphysis. When the urethra has increased tone, it feels like a straw or a pencil that can be rolled over. Too much pressure on the tense urethra can cause complaints of pain or urinary urgency. Likewise, the rectum, when palpated vaginally at 6:00, has similar properties. Pressure on a tense, round bowel may create pain or the sense of urgency common to passing gas or stool. Elevated rectal tension appears to increase complaints of pressure and pain at the posterior fourchette (6:00). Following release of the tension via physical therapy, the complaints of pain are typically reduced, often to zero.

It is not uncommon as part of the initial patient visit to also complete an assessment of the posterior pelvis via the anus to identify abnormal tension in the anal sphincters, coccygeus muscles, posterior pelvic ligaments (anococcygeal, sacrotuberous, and sacrospinous), and the fascial structures attaching to and around the ischial spines (Figs. 17.2 and 17.3). Bimanual exams of the sacrococcygeal and sacroiliac joints can be completed when dysfunction is suspected. Anterior and posterior PFM function and resting states should not be assumed to be equal as very often they are not.

Table 17.2 Functional history questions

Question	Average function	Possible abnormal function
Bladder function		
How often do you empty your bladder?	6–8×/day, including 1×/night or every 3½–4 h	Every hour or so; up multiple times/night
How much and what do you drink?	1/2 my body weight in ounces	Not a lot, don't want to have to empty
Do you have pain when you empty?	No, not even after intercourse	Yes, very often after intercourse and into the next day
Do you feel empty afterwards?	Yes	No, often have to go again quickly
Do you have to push to start or does the urine just come out?	No, flow just starts after I sit down to go	Yes, often have to think about it and push a little to get it started
Do you ever lose urine?	No, not even with exercise	Yes, often with laughing, lifting, etc.
Do you have a history of chronic urinary tract infections?	No	Yes, often treated with antibiotics, even with no culture or testing
Bowel function		
How often do you have a BM?	3×/day to 3×/week	Often less or more than average
Do you have a history of IBS?	No	Yes (with constipation and/or diarrhea)
Do you need to strain to empty	No	Yes, often even when it's soft
What consistency are your stools?	Formed, soft, easy to expel	1. Large and hard (problem with diet) or
		2. Soft and pencil thin but hard to get out (nonrelaxing anal sphincters)
Do you feel empty after a BM?	Yes	No, still feel like there is more there
Do you ever have pain with BM?	No	Yes (tailbone, abdomen, or low back
Menstrual history		
Do you have a history of painful periods?	No	Yes, with a lot of cramping with long, heavy bleeding; often have used oral contraceptive with help
Do you use tampons?	Yes with no trouble	No, it hurt when I tried it for the first time or it hurts now
Vaginal history		
Do you have a history of chronic yeast infections?	No	Yes, often with no resolution even with, treatment; lots of burning pain
Sexual history		
Are you currently sexually active?	Yes	No, it hurts too much and I don't want it
Do you have pain with sex?	No	Yes
Do you have any sexual desire?	Yes	No, it's been too long; it hurts too much
Did you always have pain with sex?	No	1. Yes, it hurt the first time I tried (primary vestibulodynia/dyspareunia)
		2. No, it was fine before but now it hurts (secondary vestibulodynia/dyspareunia)
Can you have an orgasm?	Yes, with clitoral stimulation and/or intercourse	Yes, with clitoral stimulation only but it's harder to have and it isn't as intense
When you are intimate, do you get wet (arousal)?	Yes	Sometimes but often need to use a lubricant or perhaps not at all
Where does it hurt when you try to have sex?	NA	Mostly at the bottom of the vaginal opening
How long does the pain last?	NA	Ranges from a few minutes to days
Is there anything you can do to make the pain go away?	NA	Sit in a hot tub, ice, go to sleep
Even though you don't have sex, are you still hugging, kissing, and touching?	NA	Yes, I try to do what I can
		No, I don't want to lead him on

Examples given are for average function typically found in women without complaints of pain and possible responses for women with abnormal function and a history of pain

17.6 Visceral Physical Therapy Intervention

Intervention is driven by organ system dysfunction and physical findings. If urinary urgency and frequency are noted in the intake, for example, it is necessary to review dietary as well as bladder habits (Table 17.1). If bladder dysfunction is left unaddressed, it may cause the PFMs to remain in a constant state of excessive holding, thus contributing to additional PFM over activity and possible vulvar pain and sexual dysfunction. The same holds true for bowel dysfunction. Discussions around daily food intake can often lead to suggestions for small changes (e.g., eliminating dairy and/or gluten intake, or decreasing sugar consumption) that can impact overall pelvic function by correcting chronic constipation or diarrhea. Abnormal physical findings throughout the body, including the affected viscera, need to be addressed for full recovery of pelvic health.

Visceral manipulation is often referred to as "organ-specific myofascial release." The manual techniques used to release the abnormal visceral tension are specific, gentle, and slow and can be applied to any organ system in the body.

Treatment of the pelvic organs is very similar to the assessment techniques. Once the organ-specific, abnormal tension is identified, a gentle bimanual approach works well. With regard to urethral and bladder tension, the internal treating digit comes in contact with the bladder trigone, a triangular area of neurologically dense tissue that is formed by the superior ureteral orifices and the urethra inferiorly. The external treating hand is placed just superior to the pubic symphysis over the body of the bladder. Creating and holding a very gentle fascial load with both treating hands for 30–40 s appears to elicit a visceral release of tension in both organs. That visceral release typically results an overall anterior pelvic relaxation which includes the PFM. The same approach works for rectal tension. The internal digit, while in the vagina, places a fascial load at the rectum, superior to the anus, and the external hand does the same just inferior to the umbilicus where the sigmoid colon goes inferior and posterior to form the rectum. As with the urethra and bladder, holding for 30–40 s typically releases tension in the rectum and anus. It's this tension that appears to perpetuate the commonly found posterior fourchette pain in women with vestibulodynia. Once released, the tension relaxes and the pain resolves. These releases have been developed clinically over time by the author and have not been proven.

Any aggression or force applied to the organs results in a negative response, creating yet more tension, pain, and continued dysfunction. Not only do the manual techniques normalize visceral tension, they allow the musculoskeletal and fascial systems to respond as well, leading to restored mobility, reduced pain, and improved overall function. Education regarding these techniques is invaluable, providing practitioners with yet another tool in their toolbox.

17.7 Summary

Treating patients with chronic PFD is challenging for all those in the health-care arena. It is incumbent on all those who deal with this difficult population to be compassionate, understanding, and thorough in their assessment and treatment approach. The unknown factors related to the causes of chronic pelvic pain and PFMD continue to bewilder practitioners. However, therapy that is directed toward finding the comorbid physical abnormalities throughout the body and correcting them appears to be a logical and successful approach. Referral to a WHPT should occur routinely as part of the multidisciplinary approach for all women who present with any type of PFMD. Research indicates that PF physical therapy is safe and effective, and can dramatically improve symptoms related to chronic PFD. Working together, the health-care team can make progress as each member contributes his or her expertise. When provided with the correct tools, women can learn to manage their bodies and recover the function they may have lost due to the pain and dysfunction.

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A Tale of Two Pain States: The Integrative Physical Therapy Approach to the Overactive Pelvic Floor

18

Carolyn Vandyken and Sandra Hilton

18.1 Conceptual Change in Pelvic Health

"It was the best of times, it was the worst of times." This quote is an apt beginning to Charles Dickens' classic novel *A Tale of Two Cities* [1], and is an appropriate metaphor for exploring the challenges of integrating the treatment of central pain mechanisms into clinical practice today. It is the best of times: basic science research has produced over 20 years of evidence supporting the integration of a biopsychosocial approach into the treatment of persistent pain [2–7]. It is the worst of times: established clinical practice patterns are difficult to change; currently, many therapists treat persistent pain with the same tissue-based approaches that are commonly used for acute pain [2, 8]. Despite these challenges, there are good resources available in order to embrace a biopsychosocial approach when integrating the treatment of central pain mechanisms into clinical practice. A biopsychosocial approach, pain biology education, and the development of a toolkit to treat the sensitive nervous system are required elements of this conceptual change [9]. The first step in developing a biopsychosocial framework as a clinician is to begin with an empathetic approach [10].

18.2 The Role of Empathy in Clinical Practice

Charles Dickens used his challenges in life to develop empathy towards the working poor of his time. The connection with his characters is the key to his writing and key to treating patients with chronic pain. When we interact with our clients using an empathetic approach, we validate our patient's journey and acknowledge their experience [11]. Listening with empathy provides clinicians with critical information about potential sources of the real or perceived threats that are contributing to and perpetuating pain. Mantel [12] emphasizes the importance of understanding the individual. "A person in pain is not an empty vessel, filled temporarily with a sensory experience that can be emptied out like water and leave no trace. Pain changes us. It takes more than a pill to reverse or manage that change."

The International Association for the Study of Pain (IASP) defines pain as "an unpleasant sensory and emotional experience associated with actual or potential tissue damage or described in

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terms of such damage" [13]. Importantly, pain can be derived without the presence of nociception or tissue dysfunction [14, 15]. This applies to acute and chronic pain states but is often more relevant in the perpetuation of chronic pain. Being empathetic to the patient's story provides the opportunity to help unravel the meaning of their pain problem **with** them. It is when their pain is associated with "potential tissue damage" as described in the definition above that we may see challenging clinical presentations [15]. The tissues may be healthy, but the sensory input from those tissues, the surrounding area, or the context of the area are being interpreted by the central nervous system as potential threats to the well-being of the person. The central nervous system may determine that there is need for heightened vigilance of the area because of the "potential" for damage. This may result in a protective pain response that is unrelated to the health of the pelvic floor, but may be the driving force in the perpetuation of overactivity [7, 15]. The patient is not imagining this pain; it is not "all in their head." In order to get these concepts across to the patient, the practitioner must use an empathetic delivery.

Expressing empathy can change the outcome of treatment. A 1998 study known as the ACE (Adverse Childhood Experiences) study [16] clearly shows that our life stories matter a great deal to our health. Funded by the Centers for Disease Control and Prevention and Kaiser Permanente in San Diego, the ACE study is one of the largest studies to investigate the effects of childhood emotional and physical trauma on our physical (and psychological) health later in life. With over 17,000 individuals participating, the research demonstrated that childhood abuse and family dysfunction could significantly influence the chances of developing heart disease, cancer, diabetes, obesity, depression and more [16]. By asking the question, "Can you tell me how this adverse childhood experience has affected you later in your life?" and allowing the patient talk about it, the study demonstrated a 35 % reduction in doctor visits and an 11 % reduction in emergency room visits in the following year. Further interaction of a staff psychiatrist conducting hour-long therapy sessions reduced doctor visits by 51 % [16]. Empathy and active listening can make a significant difference in patient outcomes.

18.3 The Scope of the Problem

Chronic pain is a tremendous burden for significant numbers of people around the world. Chronic pain follows no racial, geographic, social, or economic patterns. People suffering with chronic pelvic pain face a uniquely challenging complexity. The pelvic region is essential for survival, procreation, and mobility [17]. Chronic pelvic pain often involves multiple body systems (gynecological, urinary, gastrointestinal, and muscular) likely due to cross talk in the dorsal horn. Social and religious implications of the pelvic area further complicate the experience of pelvic pain. The vast literature on the impact of shame and vulnerability also applies to this patient population [10]. Pelvic pain is underrecognized and under-reported, in part due to the hesitancy of some health care professionals to ask screening questions about bowel, bladder, or sexual function [18].

An overactive pelvic floor and a positive forced FABER's test, a screening test for hip pathology that involves placing the hip in flexion, abduction, and external rotation, have been demonstrated to be strong predictors of chronic pelvic pain in women compared to healthy controls; yet most health professionals have not been properly trained how to assess the pelvic floor and fail to consider its contribution to pelvic pain states [19]. The overactive pelvic floor is a possible "biological" driver of chronic pelvic pain, which needs to be considered in every individual when addressing pelvic pain in a biopsychosocial framework. Health professionals require the proper training to be able to identify an overactive pelvic floor.

18.4 Treating Patients from a Biopsychosocial Perspective

An important component of integrating the treatment of central pain mechanisms into practice is to consistently approach all patients from a biopsychosocial perspective. Key components of a biopsychosocial approach are outlined here.

1. Take the time to listen to your patient and encourage them to share all of the important contributions to their symptoms.
2. Look at the context of the person's experience within all aspects of their life (work, home, social, and recreational).
3. Assure the patient that their pain is 100 % real, and provide reassurance that while the painful area may be in need of better movement or relaxation, the painful area may not be damaged. This requires that the treatment provider accurately understands pain biology and is able to effectively communicate this to the patient in a way that establishes evidence of their safety.
4. Remain committed to looking at the whole person: the biology, their cognitions, and their interaction with the world around them.

18.5 Moving from a Biomedical to a Biopsychosocial Approach

Nijs [8] recommends five steps to be incorporated by clinicians in order to move from a biomedical to a biopsychosocial approach when treating persistent pain. These steps are appropriate for all clinicians, including those working with patients experiencing symptoms of an overactive pelvic floor.

1. *The assessment of the clinician's beliefs and attitudes*: Nijs recommends filling out self-report questionnaires as if you are a patient with persistent pain and evaluate your responses. In order to change interventional strategies it is important to broaden the illness beliefs held by the clinician [20]. Outcomes improved for patients who had a high risk of developing long-term disability and had higher levels of catastrophization or depression, but only if the attitude and beliefs of their treating clinicians changed [2]. Outcomes did not improve with clinical training courses unless they translated into a change in the clinician's belief system [2]. The Pain Catastrophizing Questionnaire (PCS), Tampa Scale of Kinesiophobia (TSK), and the Depression, Anxiety and Stress Scale (DASS) are solid options for outcome measurement tools that can be used with the pelvic pain population.
2. *The assessment of attitudes and beliefs in patients with chronic musculoskeletal pain*: Foster [21] reported that disability at 6 months is predicted by a patient's perceptions that the problem will last long, an expanding array of symptoms, their weak beliefs about self-control, and the patient's low confidence in their own ability to perform activities despite the pain. These attitudes need to be measured and addressed.
3. *Clinical reasoning needs to include the reconceptualization of pain*: Pain biology education is a key component of this goal. Education is necessary to close the gap between the clinician's beliefs and patient expectations. It is not enough to know the science and be able to explain it, the clinician needs to understand WHY the science applies and confidently teach that to the patient.
4. *Understand the patient's social support system and their attitudes and beliefs about the patient's problem.* This should not be minimized.
5. *Match the intervention to the appropriate therapeutic target*: The intervention provided should not be focused on targeting the tissues alone. Interventions should address the sensitized nervous system and cortical structures as well [2]. When planning interventions for the patient it is crucial that the patient understands the clinical reasoning behind each of the therapeutic interventions. Interventions such as

supervised or individualized exercise therapy and self-management techniques enhance exercise adherence and improve self-efficacy [22]. Self-efficacy is one of the main predictors of treatment outcome for patients with chronic musculoskeletal pain [23].

18.6 Transformational Practice

The theme in a *Tale of Two Cities* [1] is one of renewal and rebirth, describing the importance of transformation. Science is leading us towards a transformational period in the treatment of chronic pain. The gate control theory developed by Melzack and Wall is still taught and held as the dominant pain theory today, but Melzack and Wall [14] themselves abandoned this theory many years ago. Melzack developed the neuromatrix theory of pain and Wall called the misnaming of nociception as "pain fibers" and "pain pathways," an oversimplification that complicated the process of understanding pain [24]. Understanding the neuromatrix theory of pain is crucial as clinicians reconceptualize pain for themselves and their patients. Incorporating current pain science into clinical practice through consistent use of accurate pain biology provides credible evidence of safety for the patient. One of the most important things that clinicians can do is to restore or provide hope for their patients that they can and will improve [25]. Health care providers have the opportunity through the careful choice of words and appropriate treatment prescription to ensure that they are not an additional threat that their patient has to protect against. An example of this is the term "chronic" versus "persistent" pain. "Chronic" connotes the notion of permanence. The term "persistent" implies that pain has lasted longer than expected. With the removal of the nociceptive inputs, and a change in thoughts, beliefs, and attitudes, "persistent" pain can resolve. Subtle shifts in the words chosen are integral when treating central pain mechanisms and the beliefs that drive them.

18.7 Evaluation Components

Keith Smart designed and carried out a Delphi study to assess the key factors for identifying central pain mechanisms within an individual based on the subjective and objective evaluation [26]. His research validated four specific areas to assess when completing a subjective and objective evaluation, which provide a predictive likelihood ratio of the relevance of these factors in a patient's presentation in order to identify the presence of central pain mechanisms [26].

1. The presence of disproportionate, nonmechanical pain including hyperalgesia and allodynia represents a 30:1 odds ratio that the patient is presenting with central pain mechanisms.
2. When pain persists beyond the expected time frame for healing of 12–16 weeks there is a 27:1 odds ratio that the patient is presenting with central pain mechanisms.
3. The presence of diffuse or widespread pain represents a 15:1 odds ratio that central pain mechanisms are a significant driving factor.
4. The identification of psychosocial factors such as fear avoidance and catastrophization lead to a 7:1 odds ratio that the patient is presenting with central pain mechanisms.

18.7.1 Subjective Evaluation

It is important to reiterate that central pain mechanisms are present to protect the individual from actual or potential threats, and they represent structural changes within the central nervous system. Through a careful history the clinician is able to identify personal challenges or threats for each patient. Information on what was occurring when the symptoms began, how patients felt at the time of injury, and their current belief system about their symptoms are also critical components of the evaluation. The use of validated screening tools for assessing the presence of negative appraisals and cognitions, which contribute to persistent pain state is key to a biopsychosocial

approach. Tripp et al. [6] provide a biopsychosocial example in their phenotypic approach for assessing women and men with painful bladder symptoms. Fear avoidance and catastrophization are two strong predictors of the presence of central pain mechanisms contributing to the patient's pain presentation [26]. The Pain Catastrophizing Scale (PCS) and Tampa Kinesiophobia Scale (TSK) are simple, validated tools which help to direct assessment and treatment strategies. The presence of high levels of fear avoidance and catastrophization are strong indicators that pain education and other behavioral treatments are warranted in order to address these cortical changes. These questionnaires take less than 5 min to complete and less than 1 min to score. The literature supports the integration of these tools into clinical practice [6, 27, 28]. These screening tools assist clinical decision-making not only in rehabilitation but also in surgical management. A clinical example of using catastrophization measures to assist in decision-making is linked to the surgical decision-making process for elective hysterectomies. The majority of hysterectomies performed in Canada are elective. Pinto et al. [28] demonstrated several risk factors for developing persistent pain in women undergoing elective hysterectomies. One strong risk factor was high levels of presurgical catastrophization. Appropriate pain biology education provided presurgically may help to change this outcome by reducing catastrophization through the understanding of pain [7].

18.7.2 Physical Evaluation

The physical examination also provides important information about biological triggers or perceived threats that are possible drivers of a central pain response. Physical findings may include tight or stiff tissues, weak or deconditioned muscles, scar tissue, peripheral sensitization, signs of central sensitization (i.e., allodynia, hyperalgesia, pain brought on without provocation), vascular issues, and visceral dysfunction. Physical therapists employing a biopsychosocially informed physical examination should:

1. Apply a consistent scientific approach to labeling dysfunctional tissue with respect to normal healing time frames. As stated previously, tissues heal in 12–16 weeks. There are only a few exceptions to this rule: complex fractures and wounds with gaping edges, for example. Telling a patient that they are a "slow healer" is not supported by evidence. In the majority of cases, the patients that you see at 12–16 weeks post-injury have healed. Their tissues are tight, weak, or they may have a demonstrable and predictable dysfunction that responds mechanically. These tissue-based inputs should not be overlooked or ignored as a possible nociceptive input into the nervous system. Mechanical, tissue-based pain is relatively straightforward to treat and should respond in a predictable, time-limited fashion.
2. Describe the possible biological drivers uncovered in the physical evaluation and avoid the use of threatening words and phrases such as "pinched nerve," "degenerative disc disease," "scar tissue," and "you will have to live with it" [15, 29]. These words are not an accurate reflection of available scientific knowledge [2]. Many people have these conditions but do not suffer from pain, which means these conditions may have little clinical relevance [30].
3. Assess the quality of a patient's movement and tissue health. If a patient has global restrictions in multiple areas (diffuse pain), dysfunctional movement patterns such as cogwheeling or poor contraction and relaxation patterns (which is often seen in an overactive pelvic floor) and poor awareness (tactile acuity) of the pelvic floor, it is likely that sensorimotor integration is a dominant driver of the patient's presentation [26]. Asking the patient to contract and relax the pelvic floor repetitively to assess the quality of the movement can test this. Having the patient identify the location of touch during a pelvic floor examination can also test tactile acuity.
4. Test for the presence of hyperalgesia and allodynia with light touch. Clinically there is a lack of objective data to consistently and reliably test pelvic floor pain pressure

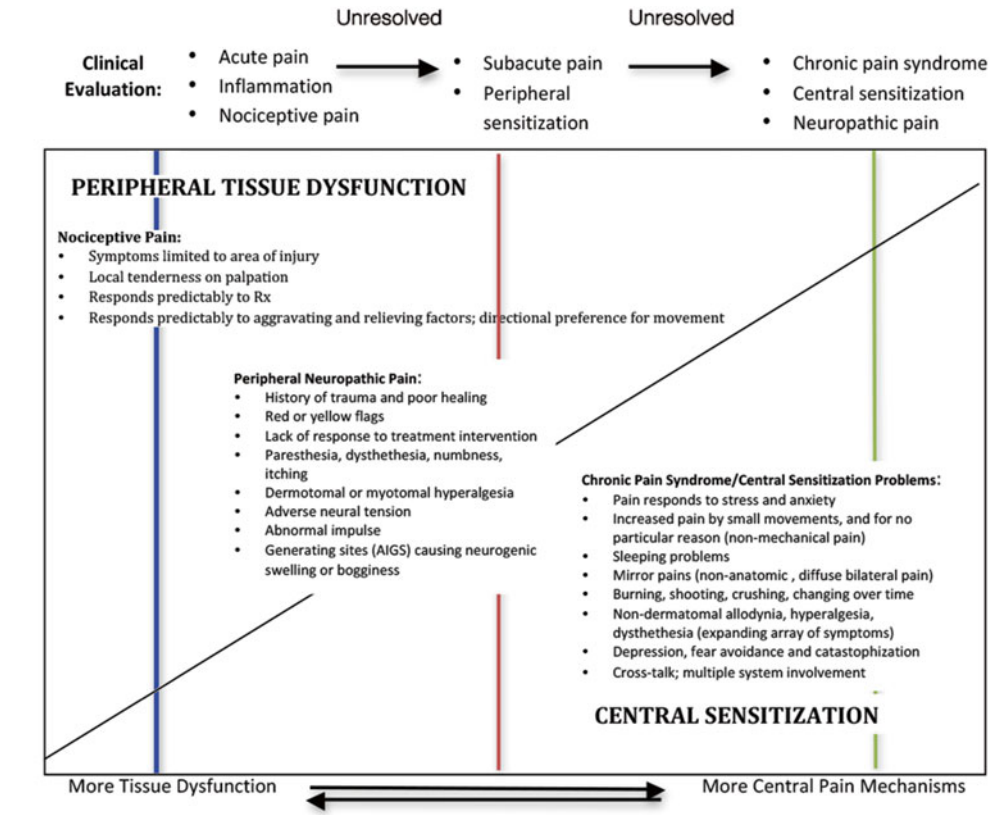

Fig. 18.1 Diagonal diagram. *Adapted from CPA Teleconference, April 16, 2009 by Alejandro Elorriaga Clarac*

thresholds. Pukall has demonstrated the relevance of decreased pain pressure thresholds in patients with vestibulodynia but normative data has not yet been established. There is also a lack of a commercially available means of measuring pressure thresholds in the pelvic floor in an accurate and reproducible way [4]. This is an area for ongoing research and development.

5. Identify the balance between tissue drivers and the influence of the sensitive nervous system. The use of a schematic approach to ascertain the relative contribution of tissue dysfunction versus central pain mechanisms may be helpful (Fig. 18.1). Placing each patient along the *x*-axis of this diagram after a thorough evaluation will help the clinician and patient plan the appropriate balance between targeting tissue dysfunction and the sensitized nervous system. Assessing the individual's response to treatment of the tissues, evaluating their negative appraisals and cognitions, and unraveling the threats that drive the individual's pain state often requires multiple visits.

18.8 Treatment Design for Central Pain Mechanisms

It is important to target cortical structures (the brain) in your treatment plan when there is evidence of central pain mechanisms in the patient's presentation [3]. Inhibitory neurons descending

from the brain to the spinal cord help downregulate the sensitive nervous system and can limit the impact of nociceptive input [31]. Downregulation involves the release of inhibitory chemicals into the synapses to decrease the sum of the neural response that occur when the brain concludes that a threat exists. The activity in descending pathways is not constant and can be modulated [32]. The use of techniques that decrease vigilance and modify the stress response may enhance the activity in the descending pathways and help to decrease the sympathetic nervous system response [33, 34]. As a result, the techniques below may directly or indirectly facilitate decreased activity in the overactive pelvic floor. There are many different techniques that may improve the patient's mind-body connection by targeting the upregulated sympathetic nervous system. Treatment options may include the following:

- *Pain biology education* is required to understand the central nervous system changes in order to give meaning to persistent pain states, and to insure that the patient does not believe that the pain "is in their head." This will be dealt with extensively in a further section. Pain Biology education is integral for patients to understand why they are focusing on therapeutic interventions that target cortical structures.
- *Connective tissue mobilization*: Mobilization of the soft tissue is used to have a direct effect on tissue dysfunction, as muscles, fascia, and neural tissue must move in order to be healthy [35]. Connective tissue mobilization may affect both tissue dysfunction and sensitization through modulation of the nervous system through the somato-visceral reflex [35]. Clinically, treatment of the connective tissue has been shown to be an important component of tissue dysfunction-based treatment in urological pelvic pain [36]. Connective tissue mobilization should not be painful in its delivery, to avoid firing the sensitive nervous system and increasing nociceptive input. Non-painful techniques provide credible evidence of safety and decrease the need for a pain response.
- *Deep breathing*: People with persistent pain tend to have maladaptive breathing patterns, including shallow apical breathing [37]. Retraining deep breathing, with both lateral costal and diaphragmatic techniques, downregulates the sensitive nervous system, particularly the sympathetic nervous system [37].
- *Relaxation and awareness training*: Meditation and mindfulness practitioners teach awareness and relaxation [38]. Different styles of relaxation training include paradoxical relaxation, progressive muscle relaxation, and autogenic training [39]. People who meditate may have more gray matter in regions of the brain that are important for attention, emotional regulation, and mental flexibility [37]. Meditation may also decrease anxiety and improve self-esteem [40]. Mindfulness meditation is the skill of maintaining focus on something by choice while allowing thoughts, emotions, and sensations to come in and out of awareness without judgment [38]. A variety of mindfulness, relaxation, and awareness strategies should be available to find the best fit for your patient. Encouraging a patient to choose her or his preference may help improve consistency of practice and increase the likelihood of success [41].
- *Guided imagery*: Guided imagery allows for individual exploration into unhelpful movement patterns that limit normal movement and function. Imagery engages the power of the mind to reduce anxiety, depression, and stress. Carrico et al. [42] conducted a pilot study, using a guided imagery CD specifically recorded and scripted for women with interstitial cystitis and pelvic pain. 45 % of the treatment group participants responded to guided imagery therapy, noting a moderate or marked improvement on the global response assessment [42]. Pain scores and episodes of urgency significantly decreased in the treatment group compared to the control group [42].
- *Yoga*: The term *yoga* is derived from the Sanskrit verb *yug*, which means to bind or join. This refers to the overarching goal of yoga to unite the mind and body in a way that promotes health [43]. Comprehensive

protocols have been adapted for yoga in the management of chronic pain. Yoga specifically addresses body awareness through body map training, breathing techniques, and increased awareness of mental and physical states, which may help patients better understand their pain response. Several mechanisms could potentially explain the benefits of yoga for persistent pain. Yoga can decrease sympathetic nervous system activity, reduce inflammatory markers, reduce stress markers (cortisol), and increase flexibility, strength, circulation, and cardiorespiratory capacity [43]. Yoga has also been shown to increase the frequency of positive emotions and could potentially undo the physiological effects of negative emotions, broaden cognitive processes, and build physical and psychological resources [43]. Finally, it is possible that yoga can lead to improvements in self-efficacy for pain control [43].

- *Affirmations/positive thinking*: Patients may be able to learn to control and change their thoughts, seeking mastery in the following areas: stress inoculation, assertiveness in dealing with their situation, handling conflict that arises around their pain, and decreasing their resistance to get better [44]. Thoughts are nerve impulses, and negative thinking alone may drive persistent pain states. Moseley et al. [45] demonstrated that the thought of movement alone was sufficient to increase pain and swelling in complex regional pain syndrome. The contribution to persistent pain states from thoughts and beliefs provides a significant opportunity for therapeutic intervention. Clinicians can assist and encourage the use of positive affirmations and can demonstrate good modeling of these techniques.
- *Joy/laughter*: Ongoing stress, particularly in the absence of positive coping skills, lowers resistance, weakens the immune system, and increases susceptibility to health problems [46]. Pain is reduced while undergoing functional magnetic resonance imaging through positive pictures, beautiful music, positive expectations, enticing smells, sweet tastes, social touch, and enjoyable sexual behavior [47].
- *Addressing sleep dysfunction*: A systematic review concluded that there is consistent evidence associating chronic low back pain with greater sleep disturbances and reduced sleep duration [48]. Reid et al. [49] explored the efficacy of engaging in aerobic physical activity with sleep hygiene education to improve sleep, mood, and quality of life in individuals with chronic insomnia. This study concluded that an aerobic physical exercise program (involving two 20-min sessions four times per week or one 30-min session four times per week) with sleep hygiene education could be beneficial to patients with insomnia and depressive mood [49].

18.9 Pain Biology Education

Persistent pain associated with an overactive pelvic floor may have primarily top-down drivers associated with a sensitized nervous system. The evaluation as described previously can provide clues to the necessity for reconceptualizing pain in each individual patient presentation [24]. Pain is a powerful defense. An important question to answer with your evaluation is "what protection does the pain provide for this person?" A follow-up question should be, "Is that protection needed now or is the pain itself driving the symptoms?" If the pain associated with an over active pelvic floor has been persisting for greater than 3 months, it is likely being driven at least in part by central pain mechanisms and careful pain biology education is warranted.

Dickens enjoyed immense success because he published his writing in installments, which made it affordable and anticipatory. It was meant for the common people—not just for the aristocracy or the elite few. Treatment of pain needs to follow the same suit and be available to all. Pain biology education is key to empowering patients—it is affordable, nonaddictive, easily understood and it can be made available individually or in groups [7]. Simply put, pain biology

education is the process of sharing accurate information of the biology of the pain system, using current pain science in clear terms, often with the use of metaphors for explanation in a way that each person can understand. Pain biology education reconceptualizes pain, helping a person in pain to develop an understanding of the nature of his/her pain, and understand the role that he or she plays in treating their pain [24, 50]. Passive pain management with pain medication should only be a short-term solution to help patients get moving [2]. Pearson [51] suggests telling the patient that the purpose of pain medication is to help them move better; essentially, they should be told that they are "movement pills." However, asking a patient in pain to move, even in graded amounts, can be overwhelming and the movement may cause an increase in symptoms in highly protective individuals. Clinically we find that this request to increase movement is best preceded by pain biology education [7].

Pain biology education can be provided in the clinic as part of routine treatment. With an appreciation of the pain system, patients start to reconceptualize that pain is "not in their head." They learn that the nervous system has physically changed as part of a complex protective response [15, 52]. One of the current challenges in medicine is the lack of imaging which can capture these changes in central pain mechanisms for patients and clinicians. Pain needs to be re-conceptualized by the patient so that they have an understanding of the protective nature of pain, often in the absence of tissue damage. Through education, the patient can be empowered to change their beliefs and reduce the threats that perpetuate their pain cycle. This change in the understanding of the nature of pain can lead to a change in their pain response. This is a reasonable and achievable goal that has been studied in back pain and complex regional pain syndrome [24]. Educating patients about pain can change their pain levels more than any other modality that we currently have in persistent pain [7]. Through pain biology education, our goal is to help them to understand, respect, and most importantly, not fear their pain experience. Mantel [11], author and chronic pain sufferer, expressed this importance as "Pain cannot be easily divided from the emotions surrounding it: Emotions sharpen it, apprehension intensifies it, and loneliness protects it, by making hours seem like days. The worst pain is unexplained pain."

18.10 Points to Consider When Teaching Pain Biology

1. Acute pain associated with tissue injury follows a predictable pattern, is straightforward to treat and results are seen within a predicable time.
2. Persistent pain requires treatment that addresses the sensitive nervous system and cortical structures.
3. An empathetic approach is integral to a biopsychosocial treatment framework. Brown [10] distinguishes between empathy and sympathy, highlighting the unhelpfulness of a sympathetic "fix it" approach and teaches key points of an empathetic focus. A skilled practitioner will project empathy in their posture, eye contact and connection with the patient, as well as with their words.
4. Note the descriptive words, thoughts, beliefs, previous diagnoses, and clues the patient uses when completing the subjective evaluation. This allows clinicians to provide personalized patient education with accurate information and pain science based on their individual history and presentation.
5. Use high quality resources to help your patient reconceptualize pain [15, 18, 53]. You must be able to adapt and individualize your pain education for different learning styles, your own teaching style, and the patient's readiness to learn. Pain biology education should be integral in your treatment from your first interaction; however, it is critical to deliver the information in a way that is not threatening to the patient. It takes time to develop the skill of delivering pain education to a variety of patients.
6. Pain biology education is best delivered through metaphors and analogies that fit with the patient's cultural and cognitive framework. Pain biology education provides them with

accurate and alternative beliefs that encourage confidence and safety.

7. Stories and metaphors such as these two examples provide accurate biological information:
   (a) While preparing supper, you cut your finger with a knife. You have immediate pain, and you look at your finger and see blood. You immediately stop what you are doing, clean the cut, and assess the depth of the cut. You need to make a decision about whether a bandage or stitches are necessary. Having pain is purposeful and creates action to stop the immediate threat. Once you apply the bandage, the pain usually subsides within an hour or 2. If you take the bandage off many hours later, you will notice that the cut looks exactly the same as it did when you first cut yourself, except that it is not bleeding any more. If pain is truly produced from the tissues you would still be experiencing pain-it certainly has not healed yet. However, since your brain knows that you took care of the problem—the cut is no longer a threat—pain is no longer produced. The cut heals within days to weeks (depending on the depth) and that is the end of the threat. Pain stops long before the healing has finished; therefore, it cannot be the tissues that are creating the pain. Your brain produces pain 100 % of the time. The same is true for sprains, strains, fractures, and other acute injuries that we may have.
   (b) An understanding that nociception is not necessary nor sufficient to produce pain is paramount when taking a biopsychosocial approach. It will help to ensure that the problem will be looked at outside of the biomedical model. A paper cut, which results in minimal tissue damage, can cause excruciating pain. Conversely, significant tissue damage can cause little to no pain. Bethany Hamilton, a well-known survivor of a shark attack, reported that what she felt was "jiggle, jiggle, bump" when a tiger shark bit off her arm at the age of 13 off the coast of Hawaii. Her story was chronicled in a movie, Soul Surfer, and documents the importance of how the brain assesses the immediate threat of an acute trauma such as this. There was significant blood loss, and if pain immobilized her, she would likely not have survived because of the severity of loss of blood. Her brain made an executive decision and her survival instincts took over-pain would have been counter-productive.

18.11 Practical Application

It is critical for central pain mechanisms to be considered in all pain states as a primary diagnostic indicator in pain that lasts longer than 3 months. The evidence suggests central pain mechanisms play a considerable role in patients with persistent pain, even in those thought to have strong peripheral mechanisms, such as rheumatoid arthritis and osteoarthritis [2]. Lumley cautions [54] "The medical profession has unwittingly created a form of mental imprisonment that I call medicalization, when diagnosis and treatment causes an increase in pain and suffering." We do best for our patients by consistently using a three-pronged approach for both acute and chronic pain, a biopsychosocial framework. The following case series demonstrates this framework in clinical practice.

18.12 Case Series

This case series demonstrates the use of the assessment and treatment framework (Figs. 18.2 and 18.3) first presented in 2011 and 2012 with three distinctive patient presentations [9, 55]. All three patients had significant physiotherapy intervention with an orthopedic physiotherapist prior to their treatment with a pelvic floor physiotherapist as presented in this case series. Orthopedic

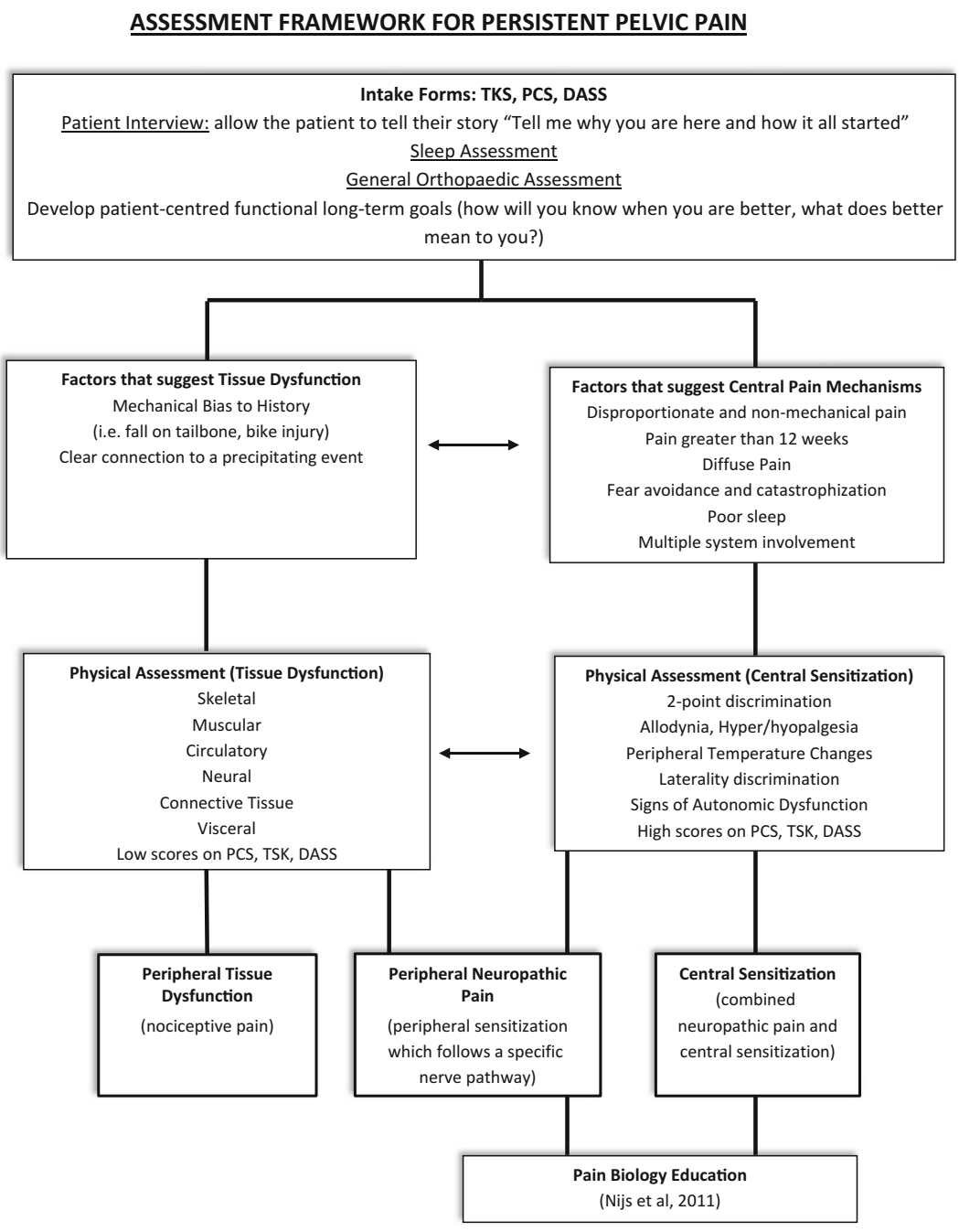

Fig. 18.2 Treatment framework for persistent pelvic pain. *With permission from Hilton S, Vandyken C. The Puzzle of Pelvic Pain—A Rehabilitation Framework for Balancing Tissue Dysfunction and Central Sensitization, I: Pain Physiology and Evaluation for the Physical Therapist Journal of Women's Health Physical Therapy 2011;35(3):103-113 © Wolters Kluwer* [9]

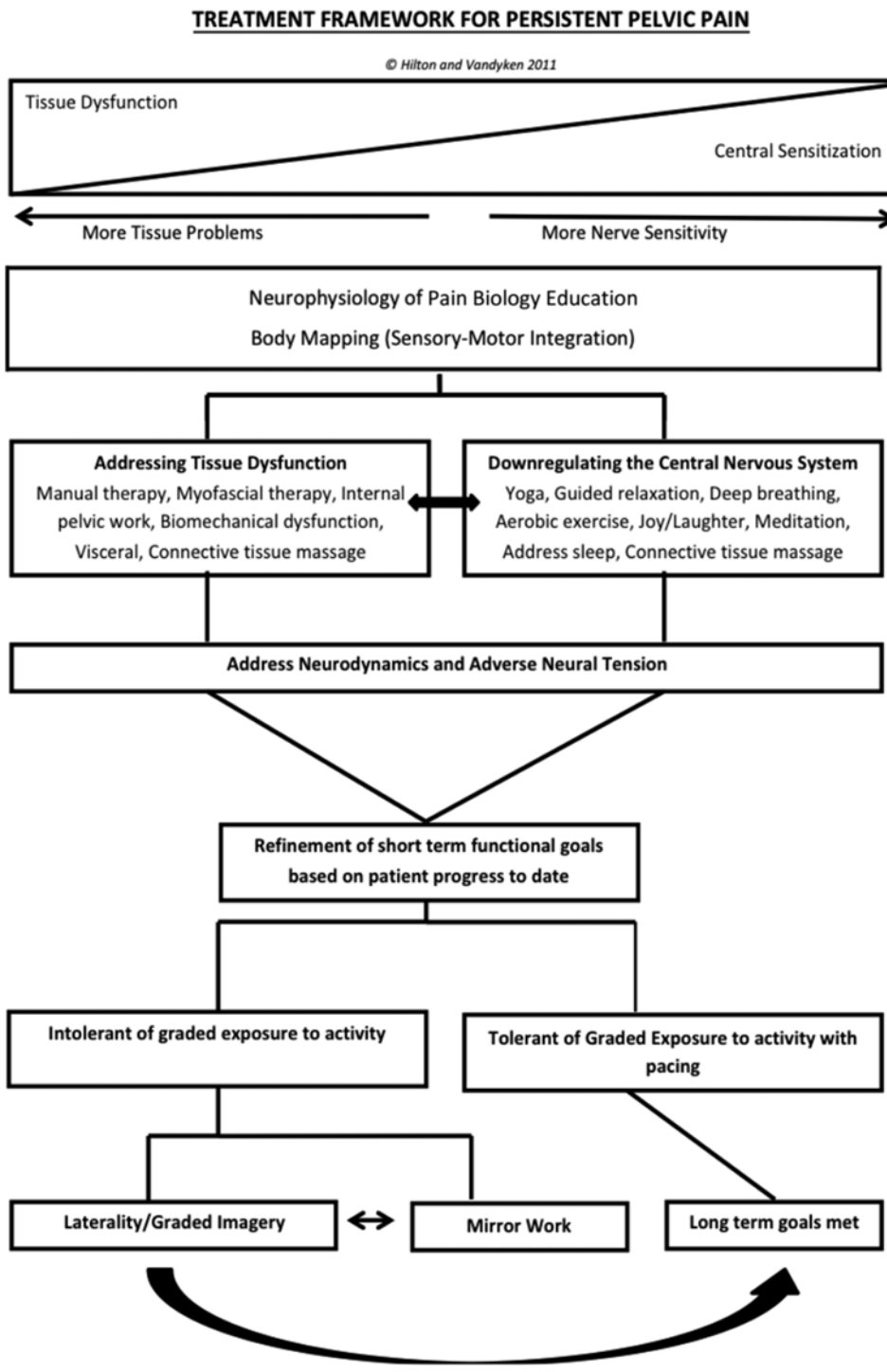

Fig. 18.3 Physical therapy assessment framework. *With permission from Vandyken C, Sandra Hilton S. The Puzzle of Pelvic Pain: A Rehabilitation Framework for Balancing. Tissue Dysfunction and Central Sensitization II: A Review of Treatment Considerations. Journal of Women's Health Physical Therapy 2012;36:44-54. © Wolters Kluwer* [55]

physiotherapists are often missing a deep understanding of the biological tissue drivers of pelvic floor dysfunction; this fact alone can change the outcome of persistent lumbo-pelvic pain states significantly [36, 56]. Furthermore, all physiotherapists need an understanding of a biopsychosocial framework within persistent pain states. This case series provides an example of the possible blend of central pain mechanisms and tissue-based drivers in persistent pain states by using a biopsychosocial framework.

The first case study (Patient #1) demonstrates no specific components of central pain mechanisms. She meets the criteria of persistent pain based on the duration of her symptoms but she responds to treatment in the same way a patient with acute mechanical pain might respond. It is interesting to note that she had been treated with a biomedical approach by another orthopedic therapist; however, symptom resolution was not achieved since the pelvic floor, as a potential tissue driver, was previously overlooked. The second patient (Patient #2) demonstrates a combination of mechanical tissue-based pain and central pain mechanisms. This provides an example of the application of a clinical framework to successfully guide treatment in a time-limited fashion [9, 55]. The third case study (Patient #3) demonstrates dominant components of central pain mechanisms only. Patients with pain, deeply rooted in central pain mechanisms, can respond in a time-limited fashion if the correct tissues are targeted, specifically the cortical structures instead of the musculoskeletal and visceral tissues. In this case series, the information that helped to guide the therapeutic assessment and treatment process has been **bolded**. These **bolded** findings specifically helped to guide the therapists' clinical reasoning.

Treatment sessions were 30 min long with 1:1 care with a physiotherapist but also involved the use of a physiotherapy assistant to teach some of the exercise components. The exercise teaching occurred in addition to the 30-min therapeutic sessions, for an average of 15 min/visit. Audio exercises were used to retrain the sensory-motor cortex with body mapping exercises, Franklin exercises, Feldenkrais exercises, Qi gong, therapeutic yoga, guided imagery and relaxation [57, 58]. The patients were sent these exercises electronically in downloadable format to ensure ease and compliance of their home exercise component for the sensitive nervous system (Tables 18.1 and 18.2).

18.13 Synopsis: Case Series

All three patients continued to work full hours and duties. This correlates accurately with their unremarkable scores on the TSK, a measure of fear avoidance. Only the third patient had high catastrophization scores (PCS), which correlates strongly with her dominant presentation of central pain mechanisms [26]. Pain biology education and strategies to downregulate her central pain mechanisms were sufficient to abolish Patient #3's high levels of catastrophization. Cognitive Behavioral Theory (CBT) or other psychologically based interventions were not required in this case, although they might be reasonable interventions with other patients who present with high catastrophization levels. The number of treatments required for each presentation type fit reasonably within their specific classification system. Mechanical pain, even from multiple mechanical sources as seen in Patient #1 (facet pain, SI joint pain, and pelvic floor muscle overactivity) is relatively straightforward to identify and treat. Although central pain mechanisms can present challenges, treatment directed at the appropriate tissues (nervous system versus musculoskeletal system) will resolve problems in expected time frames of reasonable duration, provided that treatment is proceeded with pain biology education as seen in Patients #2 and #3.

As demonstrated in all three patients, there is a proverbial "no-man's land" between the pubic symphysis and the coccyx, and these structures, although highly muscular and important in all basic physiological functions, are not consistently addressed in Master and Doctorate level physiotherapy programs in the USA and Canada. All of

Table 18.1 Physical evaluation summary for the case series

	Patient # 1	Patient # 2	Patient # 3
Age	45 y/o	34 y/o	57 y/o
Demographics	Married, 1 child, 1 stillborn (full-term), teacher	Married, 2 children (ages 1 and 3), physiotherapist	Committed relationship, 3 grown children, Real Estate Agent
History	Right SI joint (SIJ) pain, stress incontinence, dyspareunia, 1 C-section/1 vaginal delivery	(R) SI joint pain postpartum, **Believed SIJ "unstable"**	**24 months previously, she had surgical removal of coccyx secondary to pain**
	History of breast cancer-put into medical menopause 3 years previously	**Chronic LBP after fall off of bike at age 16 and skiing accident at 18-believed these injuries never fully resolved**	**Sjogren's disease**
		2 C-sections	**+++foot pain (CRPS)**
			Urinary urgency
			Dyspareunia
Pain regions	Pain localized to (R) SI joint and gluteal region but just starting to spread to upper back	Pain localized to (R) gluteal region	Burning sharp pain in (L) sitz bone and perineum, burning, discolored and swollen feet; stiff/rigid posture; neck and upper back pain
List of problems	**Localized pain**	**History of depression**	**Localized pain**
		Chronic constipation	
		Localized pain	
Previous treatment	PT for SIJ externally, Pilates, yoga, core stability exercises, kegels	Pilates (kegels), PT, ART, Anti-depressants (while at University; not at present)	P.T. to external tissues post-surgically for 18 months; acupuncture, Chinese medicine, Pilates (kegels)
Outcome measures-Pre Rx	TSK = 24/68 (low) [26]	TSK = 30/68 (low) [26]	TSK = 28/68 (low) [26]
	PCS = 0 (none) [26]	PCS = 8/52 (low) [26]	**PCS = 46/52 (severe)** [26]
	DASS = no stress, anxiety or depression	**DASS = 10 for stress (moderate)**	
Physical findings	Active Straight Leg Raise (ASLR) = 1/5 (R)	PF trigger points (TP)—(R) piriformis, pubococcygeus, obturator internus and right gluteal muscles	Uses a 6″ wheelchair cushion with a sitting tolerance of <5 min
	Vulvar dryness, vaginal atrophy-medical menopause	**ASLR = 2/5 (R), worse with compression through pelvic girdle**	ASLR = 0
	PFM TP: (R) obturator internus, ischiococcygeus and internal piriformis trigger points released nicely on assessment	**Provocative testing of the SI joint (+ve) in 3/5 tests**	**Provocative testing for SIJ was negative**
	PF muscle strength = 2/3/5 (Using the PERFECT scale)	**Mechanical assessment of low back: directional preference for (R) flex/rot to reduce and centralize symptoms; may be consistent with a lateral disc component and mechanical pain**	**No mechanical LBP**
		Stressors: F/T work, busy mom and commute to work	Minor trigger point (L) obturator internus and coccygeus
			No connective tissue dysfunction externally or internally

(continued)

Table 18.1 (continued)

	Patient # 1	Patient # 2	Patient # 3
Diagnosis	Hypertonic PFM, vaginal dryness, PF muscle weakness, minimal SIJ involvement	Pelvic girdle pain-Excessive force closure category [59]	Beliefs: "Pudendal nerve entrapment"
	No central component	Hypertonic PF	Severe level of catastrophization
	Dominant tissue dysfunction-Blue line on Fig. 18.1	Beliefs: unstable pelvic girdle	**No connective tissue dysfunction externally or internally**
		Chronic constipation	**Dominant central pain mechanisms-Green line on Fig. 18.1**
		Combined tissue/central pain mechanisms-Red line on Fig. 18.1	

the patients in this case series had extensive physiotherapy prior to this intervention. When the previous therapist addressed the pelvic floor muscles in their prior treatment programs, these muscles were presumed to be weak. The patients in this case series were all given kegel exercises in their previous treatment plans, despite underlying pelvic floor overactivity in each of them. Kegel exercises should not be a default exercise program for the treatment of pelvic floor dysfunction. The pelvic floor is critically important in protection, procreation, and basic physiological function [64, 65]. Dr. Woolf's [6] list of conditions driven by central sensitization includes many of the conditions familiar to the pelvic health therapist, such as Interstitial Cystitis, Endometriosis, Vaginismus and Chronic Prostatitis [2]. Therefore, pelvic health therapists should not get mired in simply treating the biological drivers of the tissues either, but need to take a biopsychosocial approach as demonstrated in this case series, in order to maximize each patient's recovery.

18.14 Summary

More research is needed to help select the best strategies to address the sensitive nervous system in all persistent pain states. There is evidence to support the use of CBT, pain biology education, Mindfulness-Based Stress Reduction, yoga and imagery-based exercises within a biopsychosocial framework. Physiotherapists would benefit from further training in these techniques in order to successfully integrate these approaches into their clinical practice when treating patients within this framework.

In writing *A Tale of Two Cities,* Dickens asserts his belief in the possibility of transformation, both on a personal level and on a societal level [1]. We need to transform our practices and provide hope for our patients as well. Persistent pain is pandemic in its cost and toll on our societies. Change and transformation are required. Understanding that pain is complex and highly individual makes treatment challenging but not impossible. Challenge yourself and your colleagues to identify any hesitancy towards using an approach that integrates the biological, social, and psychological components of each patient's presentation. Weave a biopsychosocial approach through each patient interaction from the very first visit by starting with pain biology education. Embrace the complexity and share in the excitement that our patients CAN change their sensitive nervous system, gently, efficiently, and permanently—by practicing what they want to become.

Table 18.2 Treatment summary for the case series

	Patient #1 (Blue line on Fig. 18.1)	Patient #2 (Red line on Fig. 18.1)	Patient #3 (Green line on Fig. 18.1)
Treatment	**First visit:** PFM TP release, education on vulvar care, OI/piriformis stretch given; discontinue Pilates, NO running b/c of grade 2/5 PFM strength	**Pain Education:** Educated on O'Sullivan's Classification system; watched "Jack" video on You Tube with Peter O'Sullivan[59]	**Pain education**: Pain biology education with *Understand Pain, Live Well again* [52]
	Second visit: reported increased (R) SIJ + (R) LBP-produced from ++ painting that week; mechanical LBP needs to be ruled out before SIJ considered [60]	She fits the category of excess tension not "unstable" SIJ	Worked on reconceptualization of information after each visit with patient-therapist handbook
	(R) L-spine facet pain diagnosed-looking back at her mechanical history, she reported performing (R) flexion/rot'n only to stretch the fascia of her left breast every day for the past 3 years: instructed to stop this, and do flexion/rot'n to left 4–6×/day, 10 reps each time	Pain biology education with *Understand Pain, Live Well again* [52]	Patient requested twice-weekly treatments-she was very motivated to improve; she held deeply entrenched beliefs that her pudendal nerve was entrapped
	Third visit: (R) SIJ and LBP resolved with (L) flexion/rot'n; started flexibility stretches for low back in all directions. Released PFM for the third time	**Visits 1–3**: PFM release, rule out mechanical Rx of lateral disc bulge	**Visits 1–6:** examined tissue dysfunction and possible mechanical components including pudendal nerve tension-negative
	ASLR = 0/5 after release of PFM	Starting to see relationship of stress to her symptoms. Goal: to r/o mechanical LBP	Minimal trigger points were present but non-contributing
	Fourth visit: SIJ pain returned but not as intense as it had been-feels "unstable" 2° to completing multi-directional stretches last week; she reports that this happens every time she does generalized stretches	Mechanical LBP r/o after third visit since pain was not responding mechanically	TP's released but sitting tolerance did not change
	ASLR 1/5: completed symmetry routine [61] × 10 min	**Visits 4–6:** Explored SIJ mechanically: Symmetry exercises [63] improved (R) ASLR from 2/5 to 0/5	Started immediately with a short, therapeutic yoga sequence with OI, Gluteal stretch, and Cat/Dog to connect tension of the pelvic girdle and relaxation of nervous system [57]
	ASLR 0/5 after symmetry routine	Pelvic Girdle Pain(+ve)	Used guided relaxation for pain and anxiety [58]
	NO PFM TP on reassessment	**HEP first six visits**: Piriformis, OI stretch, Cat/Dog to stretch the specific pelvic floor muscles that were being released	**Threat Assessment**: Poor health, hypervigilance, 2–3 hrs/day of exercise and medical appointments, rigid posture, would only wear rocker shoes-never walked or stood on bare feet
	Fifth visit: painfree in all areas; PF strength Gr 4/5 on reassessment (PF was initially weak but she couldn't recruit appropriately when overactive on assessment)	Anurex tube: prescribed for hemorrhoids 2×/day for 10 min [61]	**Nervous System exercises:**
	Home program for PFMT alt with dynamic core/PF exercises [62]	Bristol Stool Chart reviewed-Goal: Type 3 or 4 on Bristol Stool chart for stool quality	Remapping exercises for the sensorimotor cortex in sitting [57]
	Taught 20 % PF contraction for recruitment during Pilates-return to Pilates	Fiber intake and toilet positions taught	Started Qi Gong-alternating Lower and Upper Body exercises [57]

	Sixth visit: 6/52 later: painfree; no stress incontinence	**Weeks 7–8:** TP's not resolving; changed home exercise program (HEP) to a guided therapeutic yoga sequence for the pelvic floor and pelvic girdle to release tension more effectively; use sensory awareness while stretching to address tension in the nervous system [57]	Eric Franklin exercises for re-mapping feet (feet are next to genitals on the homunculus and often affected in sensorimotor smudging of the pelvis) [62]
	HEP: dynamic exercises, symmetry, returned to running and Pilates	Patient understands importance of quieting nervous system-start guided audio relaxation before sleep [57]	**3/12 later:** PCS = 22 (significant improvement)
	Total visits: 6	**Weeks 9–14:** Celexa prescription given by family doctor; signed up for Yoga class; No PFM TP any more	Re-evaluated responses to the second PCS: concluded that she needed to work on more positive thoughts and not predicting pain with activity (used Affirmations available on the podcast page of the Kaiser Permanente website) [58]
		Still demonstratinga positive ASLR	**4/12 after assessment:** sitting 8 hours with NO cushion
		Right ASLR = 2/5	Educated patient about normal hypoxia to tissues with prolonged sitting, and the need to move occasionally (wiggle and stand-up); patients often forget that this was normal before their injury/ problem
		Still holding tension in sitting-long commute to work, and sore after her commute	**Total visits: 16**
		Start body mapping for sitting and alternate with lower Qi Gong [57]	
		ASLR (R) = 0/5 by end of 14th visit	
		HEP: Yoga 2×/week, symmetry daily; use deep breathing while driving to work	
		Total visits: 14	
Outcome measure	TSK = 10/68	TSK = 19/68	TSK = 16/68
Discharge	PCS = 0	PCS = 0	PCS = 8
	DASS = 0/0/0	DASS = 6 (stress only)	DASS = not completed
Pain report at discharge and 1-year F/U	Painfree	Painfree; training for 1/2 marathon at 1-year F/U	Painfree sitting
1-year F/U	TSK = 22/68	TSK = not reported	TSK = 28/68
	PCS = 0	PCS = 0	PCS = 1
	DASS = 0/0/0	DASS = 8 (stress/anxiety)	DASS = 8 (stress)

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Complementary and Alternative Therapies for the Overactive Pelvic Floor

19

Rebecca P. Anderson and Sarit O. Aschkenazi

19.1 Introduction

Overactive pelvic floor (OPF) may encompass a multitude of diagnoses resulting in a spectrum of pelvic floor disorders from pelvic pain, voiding dysfunction, defecatory dysfunction, urinary and fecal incontinence. The organs located in the pelvic floor basin include the uterus, vagina, bladder, rectum, and the structures which support them. Addressing these issues presents a quandary to both the provider and patient. The conventional, allopathic therapies mentioned in earlier chapters including medications, lifestyle modifications, biofeedback, and surgery may turn out to be unsatisfactory, leaving women to seek elsewhere for the appropriate treatment. Women will then turn to complementary or alternative medicine (CAM).

These nonconventional or complementary and alternative (CAM) therapies may include acupuncture, guided imagery, herbs, nonprescription supplements, massage therapy, biofeedback, dietary modifications, and other forms of therapy. As defined by the National Center for Complementary and Alternative Medicine (NCCAM), complementary therapies are treatments and healthcare practices not taught widely in medical schools, not generally used in hospitals, and not usually reimbursed by medical insurance companies [1]. According to The National Institute of Health, "CAM is defined as a group of diverse medical and healthcare systems, practices, and products that are not presently considered to be part of conventional medicine" (Accessed August 21, 2014: nccam.nih.gov).

Patients may choose these complementary therapies for a variety of reasons, which have been voiced by patients in our clinics. These include lack of confidence in conventional medicine, seeking a more "natural" approach to therapy, concerns over possible side effects of conventional medicine, failure of conventional medicine to resolve or improve symptoms, cultural influences, and the belief that the body has an innate ability to heal itself. Some of these therapies such as dietary modifications, stress reduction, pelvic floor rehabilitation, and biofeedback are considered both CAM and conventional.

The cost of pelvic floor overactivity/dysfunction can be astronomical. Expenses accumulate

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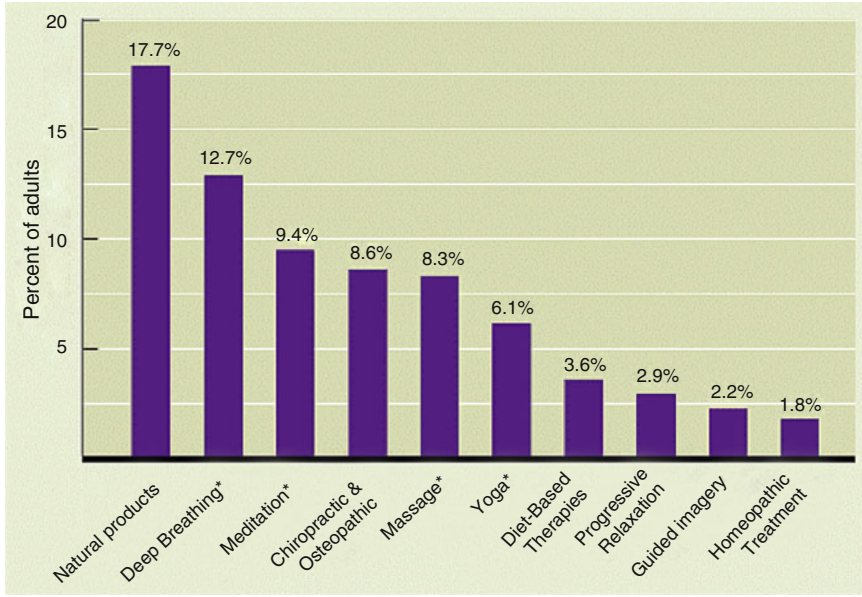


Fig. 19.1 Ten most common complementary health approaches among United States Adults in 2007. *Source: Barnes PM, Bloom B, Nahin R.* CDC National Health Statistics Reports #12. *Complementary and Alternative Medicine Use among Adults and Children: United States, 2007. December 2008*

as the degree of treatment invasiveness increases. As early as 1997, direct costs of pelvic floor dysfunction were $1012 million [2]. Women rarely present with only one pelvic floor disorder and patients could bear spending a significant and burdensome amount on their pelvic floor care. The indirect cost in lost productivity from missed work can be in the hundreds of millions of dollars. These costs do not take into account the reduction in quality of life many of the women experience. It is no wonder women turn to CAM when the conventional methods have failed, have proved insufficient, or when women are not willing to sustain the side effects associated with conventional medication or surgical procedures. Complementary therapies are not cheap and most often will entail out-of-pocket expenses. According to data from the 2007 National Health Interview Survey (NHIS), adults in the United States were spending almost $34 billion out of pocket on complementary therapies and providers [3]. "Given less than optimal success rates for conventional therapy and the growth of widespread CAM use among women, CAM use is likely common and underreported among women with PFD (Fig. 19.1)." This chapter will provide an introduction to the various CAM therapies and how they can be applied to treat the OPF (Fig. 19.2).

19.2 Nutrition

The cornerstone of any therapy or health plan should rest firmly upon nutrition. This section will address common nutrition as it relates to the health of the pelvic floor. Many patients with chronic pelvic dysfunction/pain also have complaints of bowel dysfunction such as bloating, constipation, flatulence, and irritability. Addressing nutrition to improve pelvic floor function will not only benefit the pelvic floor but also the entire body as a whole. By assisting the patient to obtain an optimal weight and stable blood glucose level, improvements in both mood and digestion can be seen. The use of fiber

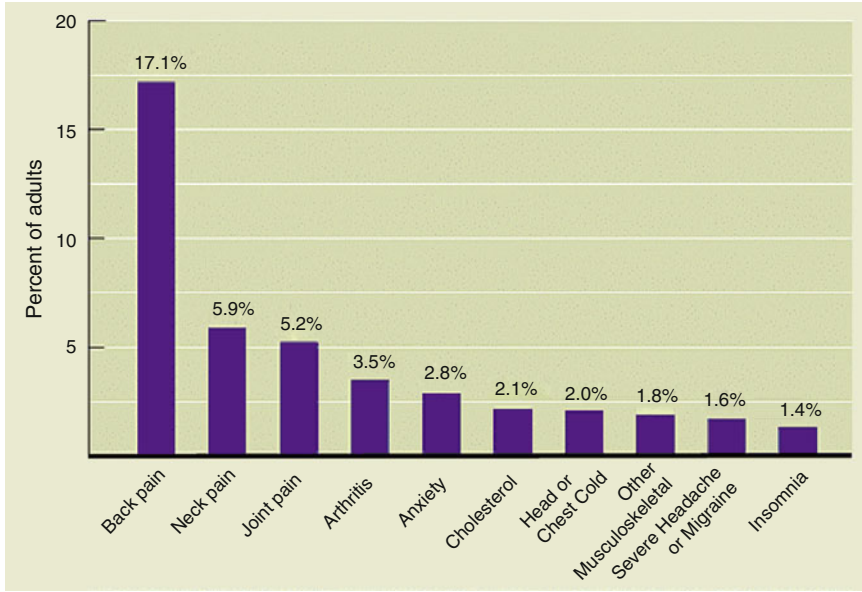


Fig. 19.2 Diseases/Conditions for Which CAM Is Most Frequently Used Among Adults. *Source: Barnes PM, Bloom B, Nahin R.* CDC National Health Statistics Reports #12. *Complementary and Alternative Medicine Use Among Adults and Children: United States, 2007. December 2008*

supplements along with appropriate fluid intake can regulate bowel movements in many patients. The use of probiotics in selected patients can also help to regulate bowel function. In the authors' clinic the recommendation for a Mediterranean diet is given. The use of an elimination diet is also used for selected patients. Referral to a registered dietitian can also be of benefit to those patients who need additional guidance, or coaching in order to improve compliance with implementing the needed dietary changes. Dietary supplements can also be used in patients whose diet is less than ideal. Many patients will find that combining a healthy diet with herbal support can decrease many of their pelvic floor symptoms and lead to an improved generalized well-being.

If an elimination diet is recommended to the patient, guidelines as to which foods should be avoided are provided to the patient. The foods and beverages that are most likely to be problematic include wheat, dairy, soy, corn, sugar, and wine. These foods and beverages should be removed from the diet for 21 days. If the symptoms improve, then it is likely that one of the foods eliminated is the problem source. The patient can then reintroduce one food at a time over a period of weeks to see which food or foods cause symptoms to return.

Patients may get frustrated initially, because they feel there are few foods left to eat, but many of the foods that they give up during the test will turn out to be harmless and can be reintroduced. Foods found to cause problems should be given up indefinitely [see Mediterranean Diet Pyramid, (Oldways Preservation and Exchange Trust, http://www.oldways.com) (Fig. 19.3)].

19.3 Mind–Body Therapies

The mind and the body are intimately connected and communicate via the nervous system, endocrine system, and immune system. Mind–body is defined as *taking into account the physiological, psychic, and spiritual connections between the state of the body and that of the mind* [4].

How our body and mind interprets input can influence our physiological and biochemical response, which in turn affects our health [5]. In

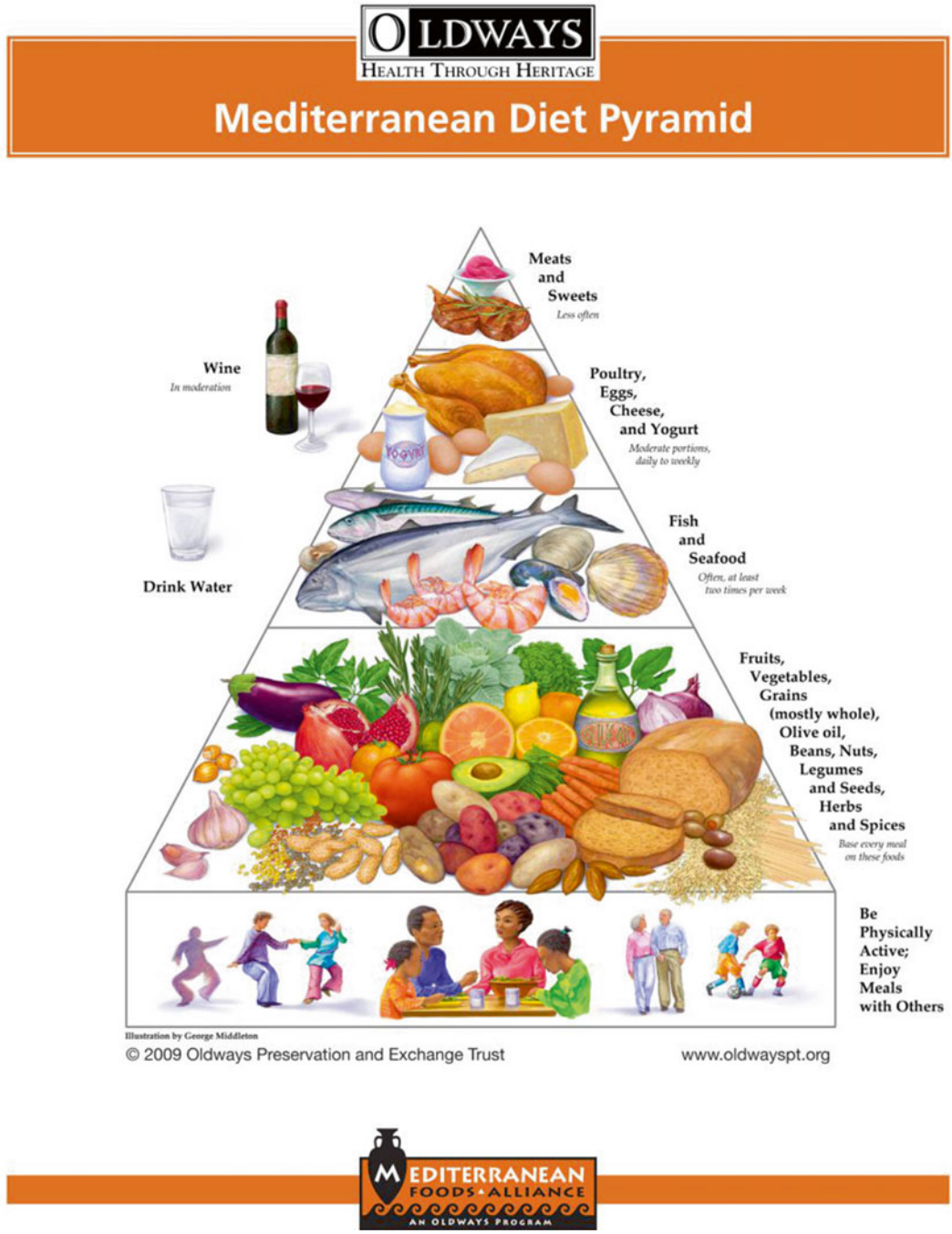


Fig. 19.3 Mediterranean Diet Pyramid. © 2009 Oldways Preservation & Exchange Trust (www.oldwayspt.org), with permission

the 2007 NHIS [6] mind–body therapies ranked among the top ten CAM practices of United States adults. The survey found that deep-breathing techniques were used by 12.7 % of adults, 9.4 % of adults used meditation and 6.1 % of adults practiced yoga. These percentages reflect an increase of use when compared to the previous survey completed in 2002.

What are the therapies that are included in mind–body work? Several of these therapies can be categorized under multiple modalities. Some of these therapies are as follows:

- Relaxation therapy
- Meditation/prayer
- Hypnosis
- Imagery
- Yoga
- Tai Chi
- Pilates
- Feldenkrais
- Music therapy
- Breath work
- Massage
- Art therapy

19.3.1 Relaxation Therapy

Relaxation therapy also known as relaxation technique or relaxation response technique includes a number of modalities such as progressive relaxation, guided imagery, biofeedback, self-hypnosis, and breath work as defined by the U.S. Department of Health and Human Services, NCCAM.

Each of these techniques shares a common goal which is to engage the body's natural relaxation response. These therapies have been used to alleviate medical conditions such as hypertension, nausea and vomiting with chemotherapy, mood state management, epilepsy, and importantly for our discussion, pelvic floor pain and bladder control [7].

The use of relaxation therapy involves more than a change in a state of mind; body functions physically change as well. The "relaxation response" involves the relaxation of breathing to a slower rate, a decrease in both blood pressure and oxygen consumption and in many people a sense of overall well-being. Being able to use this relaxation response may offset the long-term effects of chronic stress. The benefits of relaxation therapy are most useful when practiced regularly and combined with exercise, healthy diet, and social support. Relaxation therapy is a low-risk intervention for most patients, is easy to use, and can be incorporated into the patient's conventional medical care [7].

19.3.2 Relaxation Techniques

There are numerous relaxation techniques which could be used to decrease pelvic floor discomfort and hypertonic muscles. There are multiple modalities of relaxation techniques which patients could employ. Relaxation techniques work on reducing the chronic "fight or flight" response of stress. Using these techniques on a voluntary basis can reduce the negative effects of stress. While neurophysiological responses differ according to gender, stress activates the limbic system which is the area associated with the processing of emotion. The use of relaxation techniques that access these emotional and biobehavioral pathways may have an increased benefit to women [7].

- Autogenic training involves focusing one's attention on the actual sensation of breathing or heartbeat. The body is then pictured as warm, heavy, and relaxed.
- Progressive relaxation is also known as Jacobson's progressive relaxation or progressive muscle relaxation. It is a technique in which one focuses on tightening and then relaxing each muscle group. This therapy is often combined with guided imagery and deep-breathing exercises.
- Guided imagery also uses focus as a means of relaxation. In this technique negative images are replaced with pleasant ones. Guided imagery can be self-guided or delivered by a practitioner either present or on an audio device. This technique is also called visualization. Caution must be used in patients who are suffering from posttraumatic stress disorder or acute mental illness as this can trigger reemergence of negative images. Deep-breathing or breathing exercises are used to consciously slow down breathing and to focus on taking regular, deep breaths. These exercises often

involve holding the breath and then exhaling to a counted measure. A popular breathing technique used by the author is the "4-7-8" breathing exercise taught by Dr. Andrew Weil at the University of Arizona's Integrative Medicine Program. The patient is taught to breath in through the nose for four counts, hold the breath for seven counts, and then breathe out with a rush through the mouth. The tip of the tongue touches the front teeth to complete an energy path according to Ayurvedic practice (author's personal experience).

- Hypnosis uses a phrase or nonverbal cue to induce the relaxation response. It is an altered state of consciousness characterized by an increased responsiveness to suggestion. A hypnotist or hypnotherapist helps the patient to the relaxed state. Once this state is achieved, the hypnotist helps the patient focus the attention on a narrow range of objects or ideas [8]. This shifting of attention can be used to effect positive changes and treatment outcomes.
- Meditation refers to a group of techniques which originated in Eastern religions or spiritual traditions [9]. The use of meditation teaches a person to focus her attention and calm the stream of thoughts that is ongoing in the mind. It can improve psychological balance, increase overall calmness and relaxation, aid in coping with chronic illness, and improve health and sense of well-being.
- Yoga is a system that incorporates simple meditation, controlled breathing, and specific body postures to achieve emotional and body equilibrium. It is practiced for health and relaxation. All forms are based on hatha yoga. Forms or schools are based on the teachings of the preeminent yogi. The use of yoga has been noted to be of benefit in those patients experiencing chronic pain by reducing the intensity of pain.
- Tai Chi. This form of physical meditation is used by all age groups. It is a system of slow, graceful movements that meld mind and body.
- Qi-gong is yet another system of physical meditation which uses slow, gentle, and deliberate movements, breathing, and mediation to improve the life force and emotional health. It can incorporate movements from both kung Fu and Tai Chi.
- Pilates is a movement therapy which increases the core strength. It increases body awareness, reduces stress, and promotes proper body alignment.
- Feldenkrais promotes strategies that release the body from habits, which have become damaging. Body awareness, improved flexibility, and reduction of chronic pain are a few of the goals of this movement therapy.

19.3.3 Herbal/Botanical Therapies

There is a paucity of research to support herbal and botanical therapies. Existing studies are often small, lack proper scientific methodology, and frequently report conflicting results. The use of herbals is based on a synergistic approach to healing. Each herb brings a complex array of constituents to bear on treatment. It is difficult to extract a single agent and define the exact benefit attributed by this component. With any therapy, be it allopathic or integrated, the risks versus benefits must be assessed prior to implementation.

The use of herbal therapies should be based on possible causes of pelvic dysfunction. When the cause is not clear, the treatment approach is one of support and symptom relief. The use of herbal therapies should be considered thoughtfully and holistically and by experienced practitioners.

One of the oldest traditional herbal modalities is known as The Wise Woman Way. According to renowned herbalist, Susun Weed in her book, *Down There: Sexual and Reproductive Health,* healing focuses on nourishment of the whole being: by food, rituals, and trusting the body's wisdom [10]. The Wise Woman Way seeks to empower women in their healing choices. From this point of view, Weed has used the six steps of healing modalities when working with clients. These six are as follows:

- *Step 0*: Do Nothing. An important, vital step in which you listen to the voice inside of you.

Support your health by sleep, meditation, and unplugging.

- *Step one*: Collect information. Research information on your condition. Listen to the wise healers in your community; be they conventional healthcare providers, wise women, or integrative practitioners.
- *Step two*: Engage the energy. Use mind–body medicine such as prayer, guided imagery, ritual, reiki, to name a few.
- *Step three*: Nourish and tonify. Supportive care with food, herbal long infusions, movement, walking, yoga.

The first four steps help build health and should be used daily.

The remaining steps are more invasive and should be approached in a thoughtful and educated manner.

- *Step four*: Stimulate/sedate: CAM such as acupuncture, chiropractic, naturopathy, herbalism, massage. This step is an excellent starting point for those struggling with chronic issues. Herbs used here are more tonics.
- *Step five*: Use drugs: the use of prescribed and over-the-counter drugs. Supplements are also included in this step as well as are essential oils.
- *Step six*: Break and enter: this step includes invasive procedures.

The patient can use any of these steps in no set order. There can be concurrent use of multiple steps by the patient. For example, patients could be using acupuncture for relief of chronic pain while supporting their overall health with organic oatstraw long infusion. Based on these steps, the recommendations of Weed for patients with chronic pelvic floor dysfunction are as follows:

Step 0: Do nothing. The techniques used in this step mirror those of mind–body therapies such as relaxation breathing, meditation, and guided imagery.

Step one: : Collect information and do research. Explore and address possible causes. The link between pelvic floor dysfunction and trauma should be evaluated. Prior pelvic surgeries should also be taken into consideration.

Step two: Engage the energy. The pelvis and abdomen are the center of the body according to Eastern medical and martial arts practice. The Qi or life energy flows from this center. The pelvis is important to the immune system. Chronic pelvic pain or dysfunction is the message from this energy source that the patient does not feel safe in her body.

Step three: Nourish and tonify. The use of whole body massage, Mayan Abdominal Massage (MAM) or electromagnetic therapy relaxes the pelvic muscles and normalizes the nervous system. Weekly sessions are recommended until pain is relieved. MAM is a therapy described as a "noninvasive, external massage which guides internal abdominal organs into their proper positions for optimal health." Additional information on this technique can be found at www.arvigomassage.org (Accessed 5/21/14). The use of nourishing herbal infusions is used to calm nerves, ease muscle spasms, and reduce pain. Oatstraw (*Avena sativa*), comfrey (*Symphytum uplandica* x) and linden (*Tilea europea*) are typically used in pelvic floor dysfunction. Oatstraw is an excellent nervine herb which both nourishes and tones the nervous system [11]. It is safe to use by most individuals. Use of comfrey (*Symphytum uplandica* x) also known as Russian comfrey, has been questioned due to the incidence of liver damage. On further review there are two species of comfrey: the first is wild comfrey (*Symphytum officinale*) and cultivated comfrey (*S. uplandica* x) the x means that the plant is a hybrid. These two plants are very dissimilar in appearance. The wild comfrey is a small plant with yellow flowers. The cultivated comfrey is a large plant with purple or blue flowers. The plants themselves have different constituents. The roots of comfrey contain pyrrolizidine alkaloids or PAs which are harmful to the liver. This underscores the importance of receiving treatment by educated practitioners trained and experienced in herbology. Eliminating foods which are high in omega-6

fatty acids which increase the inflammatory response may be useful.

The use of hot compresses applied to the abdomen can ease pain. Comfrey leaf, castor oil, plantain oil, or calendula oil are those recommended for relief. Patience must be used to see benefit. Compresses can be applied 4–7 times per week, covered with saran wrap and a heating pad can be applied over this compresses. They can be used for 1–3 h to help reduce pain and cramping in the abdomen. The use of compresses is not recommended during menses or when pregnant [12]. To normalize tone of the pelvic muscles, refer to a physical or occupational therapist specialized in pelvic floor dysfunction.

Step four: Stimulate/sedate. The use of acupuncture may reverse nerve disruption, muscle spasms, and muscle pain in patients with an OPF. Biofeedback, electrotherapy, and ultrasound can be helpful for some patients. Magnet therapy used continuously for 1 month has been shown to reduce chronic pelvic pain in a small double-blind study [13]. Using herbal pain relievers to ease the pain often associated with OPF can be safe and nonhabit forming. Skullcap (*Scutellaria lateriflora*) or passionflower (*Passiflora incarnate*) tincture as well as meadowsweet (*Filipendula ulmaria*) can be used for pain relief. St. John's Wort (*Hypericum perforatum*) tincture can be used as a muscle relaxant. Patients who choose to use herbal products should consult a knowledgeable herbalist prior to starting any herb. They also should be encouraged to try one herb at a time to assess for any possible side effects (Fig. 19.4).

Step five: Use of conventional drugs. Nonsteroidal anti-inflammatory medications such as ibuprofen or aspirin can help reduce pelvic pain. Muscle relaxants, anticonvulsants, tricyclic antidepressants, opiates also relieve pain. Trigger point injections with lidocaine can bring relief but is short acting. Many women find taking birth-control pills helpful in decreasing chronic pelvic pain, depending on its cause.

Step six: Break and enter. Orgasm or intravaginal/intrarectal massage can counteract pelvic floor muscle spasms. This can be self or partner administrated or administered as myofascial release by an occupational or physical therapist. Caution should be exercised with this modality as it can cause increased muscle spasms and consequently perpetuate the pain cycle. Surgery to remove inflamed tissue or adhesions caused from prior surgery/inflammatory processes may be helpful but entail the potential of scarring and reformation of adhesions with continuation of the pain.

Dr. Aviva Romm, herbalist and physician, is a world renowned expert in medical botanics skilled in the use of a wide variety of botanical medicines with a scientific approach [14–16]. She is active in organizations currently involved in botanical medicine credentialing [17]. She has published her findings providing additional knowledge on the holistic treatment of chronic pelvic pain. Dr. Romm acknowledges the multifactorial nature of chronic pelvic pain. These factors include, but are not limited to, endometriosis, pelvic inflammatory disease, adhesions, pelvic congestion syndrome, and cyclic uterine pain, uterine myomata, and history of psychosexual trauma [18].

The herbs Dr. Romm recommends are those that are restorative to the nervous system such as adaptogens and nervines, and when appropriate, anxiolytics and antidepressants [18]. The following lists are herbs which address those needs. Several of these listed can also be found in the section on herbalist Weed.

Dr. Romm has published her findings informing much of what we know about holistic treatment of chronic pelvic pain. Dr. Romm approaches the treatment of chronic pelvic pain holistically with acknowledgement of the many factors which are impacted with this diagnosis. She acknowledges that the causes of pelvic floor pain/dysfunction are multifactorial, including but not limited to: endometriosis, pelvic inflammatory disease, adhesions, pelvic congestion syndrome and cyclic uterine pain, uterine myomata, and history of psychosexual trauma [18].

Fig. 19.4 Skullcap (*Scutellaria lateriflora*)

Dr. Romm also touches on the use of Mayan Uterine Massage for uterine displacement. This practice was introduced into the United States by Dr. Rosita Arvigo after studying with a Belizean shaman [19]. Dr. Arvigo trains and certifies people in the use of this massage technique. The treatment is based on the belief that uterine displacement caused by poor posture, childbearing, sedentary lifestyle, or work habits progresses to pelvic congestion, bowel, bladder, and nervous problems.

The herbs recommended are restorative to the nervous system such as adaptogens and nervines, and when appropriate, anxiolytics and antidepressants. She participated in molecular studies of herbs demonstrating multiple mechanisms of vasorelaxation, one of which suggests that the long-term relaxation is due to the opening of potassium channels [20].

The following are herbs which address these needs [21]. Several of these herbs are also listed in the section on Wise Woman Way [10].

- Analgesia (pain relief)
  - Black cohosh (*Actaea racemosa*)
  - Pulsatilla (*Anemone pulsatilla*)
  - Corydalis (*Corydalis ambigua*)
  - California poppy (*Eschscholzia californica*)
  - Kava kava (*Piper methysticum*)
  - Jamaican dogwood (*Piscidea piscipula*)
  - Cramp bark, black haw (*Viburnum* spp.)
- Antispasmodics (pelvic muscle spasm)
  - Yarrow (*Achillea millefolium*)
  - Dong quai (*Angelica sinensis*)
  - Wild yam (*Dioscorea villosa*)
  - Marijuana (*Cannabis indica*)
  - Black cohosh (*Actaea racemosa*)
  - Motherwort (*Leonurus cardiaca*)
  - White peony (*Paeonia lactiflora*)
  - Rehmannia (*Rehmannia glutinosa*)
  - Cramp bark, black haw (*Viburnum* spp.)
  - Ginger (*Zingiber officinale*)
  - Fennel (*Foeniculum vulgare*)
  - Chamomile (*Matricaria recutita*)
- Antidepressants/anxiolytics
  - St. John's Wort (*Hypericum perforatum*)
  - Lavender (*Lavandula officinalis*)
  - Motherwort (*Leonurus cardiaca*)
  - Chamomile (*Matricaria recutita*)
  - Lemon balm (*Melissa officinalis*)
  - Kava kava (*Piper methysticum*)
- Adaptogens (supportive, reduction of stress/anxiety, improvement of sleep)
  - Cordyceps (*Cordyceps sinensis*)
  - Eleuthero (*Eleutherococcus senticosus*)
  - American ginseng (*Panax quinquefolius*)
  - Rhodiola (*Rhodiola rosea*)
  - Ashwagandha (*Withania somnifera*)
- Anti-inflammatories
  - Dong quai (*Angelica sinensis*)
  - Licorice (glycyrrhiza officinale)
  - Evening primrose (*Oenothera biennis* oil)
  - White peony (*Paeonia lactiflora*)
  - Willow (*Salix* spp.)
  - Feverfew (*Tanacetum parthenium*)
  - Ginger (*Zingiber officinale*)
- Antispasmodics (digestive support)
  - Yarrow (*Achillea millefolium*)
  - Wild yam (*Dioscorea villosa*)
  - Chamomile (*Matricaria recutita*)
  - Peppermint (*Mentha piperita*)

- Astringents (Digestive support)
  - Yarrow (*Achillea millefolium*)
  - Goldenseal (*Hydrastis canadensis*)
- Carminatives (Digestive support)
  - Chamomile (*Matricaria recutita*)
  - Peppermint (*Mentha piperita*)
  - Anise (*Pimpinella anisum*)
- Demulcents (Digestive support)
  - Slippery elm (*Ulmus rubra*)
  - Marshmallow (*Althea officinalis*)
- Laxatives (Digestive support)
  - Licorice (*Glycyrrhiza glabra*)
  - Dandelion root (*Taraxacum officinale*)
  - Yellow dock (*Rumex crispus*)
- Nervines (sleep disorders)
  - Kava kava (*Piper methysticum*)
- Anxiolytics (Sleep disorders)
  - Pulsatilla (*Anemone pulsatilla*)
- Sedatives (Sleep disorders)
  - California poppy (*Eschscholzia californica*)
- Uterine tonic (treat for possible pelvic congestion syndrome)
  - Horse chestnut (*Aesculus hippocastanum*)
- Venotonics (treatment for possible pelvic congestion syndrome)
  - Blue cohosh (*Caulophyllum thalictroides*)
  - Lady's mantle (*Alchemilla vulgaris*)
  - Goldenseal (*Hydrastis canadensis*)
  - Partridge berry (*Mitchella repens*)
  - Red raspberry (*Rubus idaeus*)
  - Cramp bark/black haw (*Viburnum* spp.)

Dr. Tori Hudson N.D. naturopathic physician and author is a proponent of the use of nutrition, nutraceutics, herbs, and natural hormones to maintain women's health. She emphasizes that alternative therapies should not be used as primary treatment for life-threatening conditions such as breast cancer, in which they can be associated with increased recurrence and death [22]. She underscores the importance of appropriate use of homeopathy, namely not to replace surgery when it's needed to avoid disease progression. She has years of experience in alternative therapies for the treatment of gynecological and primary care conditions, particularly chronic pain. Many of the herbs she recommends have been mentioned earlier. This brings up an important point in the use of herbals/botanicals: multiple resources citing the same therapy may reinforce the validity of recommendations, when in lack of randomized controlled trials.

Hudson lists the following botanicals as possible treatments for OPF especially when treating pain: ginger, turmeric (*Curcuma longa*), cramp bark, valerian (*Valeriana officinalis*), black cohosh, pine bark (*Pinus pinaster*), chaste tree (*Vitex agnus-castus*), dandelion root, prickly ash (*Zanthoxylum americanum*), motherwort, and green tea (*Camellia sinensis*) [22]. These correspond with those mentioned earlier.

Medical herbalist David L Hoffman has been involved in research on holistic herbalism. He has investigated holistic herbal therapy and its effect on chronic illness and quality of life [23]. Hoffman provides additional information on a broader view of the pelvic floor. His approach is support of the reproductive system as a whole. For functioning, the body should be balanced with attention to the body and spirit [24]. His list of herbs that support the reproductive systems corresponds to those of both Romm and Hudson. These include Lady's Mantle (*Alchemilla vulgaris*), Chasteberry (*Vitex agnus-castus*), Bearberry (*Arctostaphylos uva-ursi*), Corn Silk (*Zea mays*), Marshmallow (*Althaea officinalis*), Yarrow (*Achillea millefolium*), Burdock (*Arctium lappa*), Cleavers (*Galium aparine*), Cramp Bark (*Viburnum opulus*), Skull cap (*Scutellaria lateriflora*), Valerian (*Valeriana officinalis*), Damiana (*Turnera aphrodisiaca*), and Oats (*Avena sativa*) [11].

Of these herbs listed, the authors have recommended to patients with OPF or have personal experience with the following:

- Wild yam
- Motherwort
- Fennel
- Ginger
- Chamomile
- Lavender
- Lemon balm
- Peppermint
- Slippery elm
- Marshmallow

- Dandelion
- Red raspberry
- Turmeric
- Valerian
- Cleavers
- Oats
- Burdock

These herbs have been found to be useful and safe in the majority of patients.

19.4 Aloe Vera

A special emphasis is given to aloe vera. This plant has gained popularity and is available in different preparations and supplement forms. The Aloe vera is a flowering succulent plant which is native to Africa, but also found throughout the world. It has been used traditionally to treat skin conditions such as burns, cold sores, abrasions, psoriasis, sunburn, and frostbite. It has also been shown to prevent oral mucositis during cancer treatment and relieve radiation burns [24].

Possible actions of aloe vera include:

- Antioxidant
- Immunostimulant
- Anti-inflammatory
- Analgesic
- Antiviral
- Purgative
- Antiseptic

Aloe vera is high in vitamins A, B group, C and E, the enzyme carboxypeptidase, minerals sodium, magnesium, potassium, calcium, copper, zinc and iron, polysaccharides, amino acids, and proteins.

The internal use of aloe vera should be taken only under the supervision of a qualified herbalist. Prolonged use of aloe vera can lead to electrolyte imbalances. Using aloe vera along with cardiac or renal medications can increase the effect of these medications.

The use of dried aloe vera juice in pregnancy or lactation is not recommended, due to the possibility of miscarriage [24].

It is beyond the scope of this chapter to discuss the constituents and actions of the herbs listed. The authors strongly recommend finding an experienced herbalist to work with in treating your patients. Being a "natural" product does not mean it is safe to use in all patients and individualized care must be applied by an experienced and properly trained practitioner in this field.

19.5 Supplements

Over one-half of all United States' adults use some form of supplement in 2003–2006 (Fig. 19.5) [25]. The numbers are even greater in Europe and other parts of the world. Women represent the majority of users of supplements. Multivitamins are noted to be the most commonly used supplements followed by calcium and vitamin D.

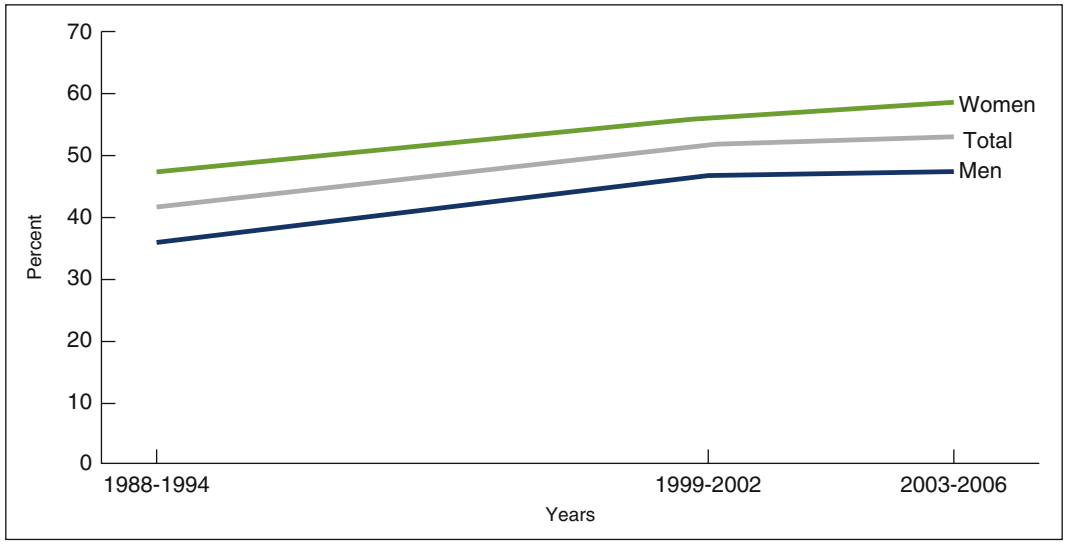
As with the use of herbal supplements, dietary supplements should be assessed during any patient visit. Possible untoward side effects increase when patients chose to take over the counter supplements from questionable sources, and often unknowingly take these in high doses that can potentiate harmful effects. Higher doses of supplements have been documented to cause adverse effects in multiple organ systems.

While supplements play a vital role in supporting patient health, a well-balanced diet which can include many of these supplements is preferable and should be sought after.

19.6 Review of Supplements in the Treatment of Overactive Pelvic Floor

19.6.1 Vitamin D

Vitamin D plays an important role in neuromuscular and immune function as well as a reduction in inflammation. It plays a major role in the absorption of calcium in the intestinal tract and helps maintain both serum calcium and phosphate levels [26]. Vitamin D receptors are present in human muscle tissue. Low vitamin D levels



NOTES: Significant linear trend from 1988-1994 through 2003-2006. Statistically significant ofference for men compared with women for all time periods. 0 < 0.05 for comparison between genders within survey periods. Age adjusted by direct method to the year 2000 projected U.S. population.
SOURCE: COCINCHS. National Health and Nutrition Examination Surveys.

Fig. 19.5 Trends in the percentage of persons using dietary supplements by gender for adults aged 20 years and over. United States 1988–2006. *Source: CDC/NCHS. National Health and Nutrition Examination Surveys*

can impact the function and tonicity of the pelvic floor [23]. According to the results of the National Health and Nutrition Examination Survey completed in 2005–2006 there is a possible correlation between vitamin D levels and reduction in muscle strength and mass which can contribute to the occurrence of pelvic floor disorders. The investigators in the survey were able to find only one study that showed the impact of low vitamin D levels on detrusor muscle functioning. This single study was done on 5816 community dwelling women over the age of 40. It was found that women with a higher level of Vitamin D were at a lower risk of developing overactive bladder. The findings of the survey support the treatment of vitamin D deficiency in both premenopausal and menopausal women to improve pelvic muscle strength and reduce the occurrence of pelvic floor dysfunction.

The use of vitamin D must be based on the individual patient's serum 25-hydroxyvitamin D level. Current guidelines for serum concentration are included in Table 19.1.

The recommended daily allowances for Vitamin D sufficient to maintain bone and calcium health in healthy individuals are listed in Table 19.2. There are some recent studies indicating these values may still be too low, especially in colder countries with restricted sun exposure.

Table 19.1 Serum 25-hydroxyvitamin D concentrations and health

nmol/L[a]	ng/mL[b]	Health status
<30	<12	Associated with vitamin D deficiency, leading to rickets in infants and osteomalacia in adults
30–59	12–20	Generally considered inadequate for bone and overall health in *healthy* (authors italics) individuals
≥50	≥20	Generally considered adequate for bone and overall health in *healthy* (authors italics) individuals
>125	>50	Emerging evidence links potential adverse effects to such high levels, particularly >150 nmol/L (>60 ng/mL)

Source: NIH Office of Dietary Supplements 2011
[a]1 nmol/L = 0.4 ng/mL
[b]Serum concentrations of 25 (OH)D are reported in both nanomoles per liter (nmol/L) and nanograms per milliliter (ng/nML)

Table 19.2 The recommended daily allowances for Vitamin D sufficient to maintain bone and calcium health in healthy individuals

Age	Male	Female	Pregnancy	Lactation
0–12 months	400 IU (10 μg)	400 IU (10 μg)		
1–13 years	600 IU (15 μg)	600 IU (15 μg)		
14–18 years	600 IU (15 μg)	600 IU (15 μg)	600 IU (15 μg)	600 IU (15 μg)
19–50 years	600 IU (15 μg)	600 IU (15 μg)	600 IU (15 μg)	600 IU (15 μg)
51–70 years	600 IU (15 μg)	600 IU (15 μg)		
>70 years	800 IU (20 μg)	800 IU (20 μg)		

Source: NIH Office of Dietary Supplements 2011

It may be useful to check vitamin D levels in patients with OPF symptoms and to offer it as an additional treatment option, supporting the whole body.

19.6.2 Antioxidant Vitamins E and C

The impact of inflammation in OPF has been a topic of discussion among clinicians. Inflammation is a known pain instigator in other chronic pain conditions such as arthritis and fibromyalgia. In a study on patients with endometriosis-related pelvic pain, published in 2013 by Santanam et al. [27], the use of daily antioxidants vitamins E and C for 8 weeks prior to scheduled surgery was found to reduce inflammatory markers in the peritoneal fluid of patients. "Everyday pain" was reduced by 43 % in the supplement use study group but remained the same in 52 %. There was also a decrease in dysmenorrhea in the supplement group.

The National Institutes of Health recommend the following daily allowances for vitamin E:

- For adults older than 14 years-15 mg (or 22.5 IU - International Units)
- For pregnant women of any age: 15 mg (or 22.5 IU)
- For breastfeeding women at any age: 19 mg (28.5 IU)

Dosages above the recommended daily allowance have been shown to cause serious medical issues: Vitamin E can increase bleeding in patients on anticoagulant treatment.

Recommended dosing for Vitamin C are:

- For women over age 18 years: 75 mg
- For pregnant women: 85 mg
- For breastfeeding women: 120 mg

Doses higher than 2000 mg daily may cause abdominal cramping or pain, chest pain, dental erosion, diarrhea, kidney stones, increased risk of lung cancer and Parkinson's disease. High doses can also affect blood clotting.

19.6.3 Glutamine

Glutamine is the most abundant amino acid in the body. During extreme stress such as illness, injuries, or intense exercise, the body is unable to make enough glutamine for its needs. If the body is exposed to prolonged stress, taking a glutamine supplement may be useful.

Glutamine is used for healing after illness, digestive disorders, and infection [28]. It is useful in support of the immune system especially in times of stress. Recommended glutamine dosing for adults: 500 mg taken one to three times daily. Patients with renal impairment, liver disease, or Reye syndrome should not take glutamine.

Information on glutamine is from the University of Maryland's website. The authors recommend this site as a resource for both herbs and supplement information. It is written in an user-friendly format.

19.6.4 Quercetin

Quercetin is a bioflavonoid which has both anti-inflammatory and antioxidant properties [29]. Food sources of quercetin include red wine, apples, onions, black and green teas, leafy green vegetables and herbs such as St. John's Wort and ginkgo. In patients who suffer from OPF, especially chronic pelvic floor muscle spasm, the use an antioxidant such as quercetin may be beneficial. Quercetin reduces inflammation by inhibiting the production of cytokines and tumor necrosis factor, which are inflammatory mediators. The absorption of quercetin is dependent on intestinal flora and function. For this reason, quercetin is often formulated along with bromelain and papain to promote absorption. Quercetin formulas which contain high doses of vitamin C are not recommended, as vitamin C can exacerbate pelvic floor symptoms when ingested in high doses.

Shoskes and Nickel recommend prescribing quercetin at a dosage of 500 mg two to three times per day with food for at least a 6-week trial. If there is improvement within 6 weeks, the patient's symptoms will most likely continue to improve up to 3 months.

The authors of this chapter recommend the use of quercetin in combination with bromelain and papain to patients with interstitial cystitis. There has been positive anecdotal response to this use among women in our clinical setting.

Quercetin is generally considered a safe supplement when used appropriately. It is advised to take periodic breaks from the supplement over time. Extremely high doses may cause renal damage. Major side effects may include headache, joint pain, and nausea. There is also a theoretical danger of interaction if taken along with quinolone antibiotics [30].

19.6.5 Magnesium

Magnesium is responsible for over 300 biochemical reactions in the body. It is essential in the activation of enzymes, contributes to energy production, and helps to regulate calcium levels. It is also important in the regulation of copper, zinc, potassium, and Vitamin D levels in the body. It maintains normal nerve and muscle function. Has been indicated to support a healthy immune system, helps to maintain a regular heartbeat, maintain normal bowel motility, and supports strong bones.

Every organ in the body—especially the heart, muscles, bowel, and kidneys—needs magnesium. It also contributes to the makeup of teeth and bones. Most important, it activates enzymes, contributes to energy production, and helps regulate calcium levels, as well as copper, zinc, potassium, vitamin D, and other important nutrients in the body.

Magnesium deficiency is rare but in the occurrence of certain medical conditions that cause a temporary imbalance in its level. These conditions include vomiting, diarrhea, gastrointestinal disease such as irritable bowel syndrome or ulcerative colitis, diabetes, kidney disease, and the use of diuretics. Too much caffeine, sodium, or alcohol can lower magnesium levels. In patients with chronic, prolonged stress, magnesium deficiency can be found. Magnesium has been shown to improve premenstrual syndrome and regulate uterine hypertonicity. Pertinent symptoms of magnesium deficiency include restless leg symdrom, muscle spasms and weakness, sleep disorders and insomnia.

Recommended dosages of magnesium are as follows:

- For women aged 14–18 years: 360 mg daily
- For women aged 19–30 years-310 mg
- For women 31 and older: 360 mg daily

Magnesium is not recommended in patients with cardiac or renal disease. High doses of magnesium can cause nausea, vomiting, diarrhea, bradycardia, cardiac arrhythmias, respiratory disturbances, and deficiencies of other minerals [31].

The use of supplements can provide a valuable addition to your patients' overall well-being. When choosing supplements your patients should be educated on how to obtain high quality, standardized, pure ingredients. As a provider, it is your responsibility to be aware of quality products.

19.7 Conclusions

The uses of complementary or integrative therapies are a valuable adjunct to allopathic medicine. The choices of therapy must be based on the individual patient with emphasis on risk versus benefit. This chapter offered a brief review of the various therapies which could be used to address the OPF. When recommending a therapy it is wise to research that therapy in further depth. Working with a qualified integrative practitioner will improve safety and benefit both patient and practitioner.

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Psychosocial Management of the Overactive Pelvic Floor

20

Elke D. Reissing and Heather VanZuylen

At first glance, problems with an overactive pelvic floor (POPF) are physiological complaints that require physiological interventions. However, the mind and the body are closely interconnected [1], and there is no better example than pelvic floor (dys)function [2, 3]. As outlined in previous chapters, pain figures prominently in POPF, resulting in reduced sexual activity and lack of enjoyment. Effective treatment interventions aim to address pelvic floor pathology, alleviate pain, as well as increase sexual function and satisfaction; but they are often delivered in a unidisciplinary format: pelvic floor physical therapy [4, 5] *or* psychosocial interventions [6]. However, we would like to suggest that a combination of physical *and* psychosocial interventions is likely the most effective approach [3, 7, 8]. By means of the Fear-Avoidance Model, we will outline how psychology and physiology are linked in POPF. The model has excellent applied value and underscores the call for multimodal interventions [9]. We will discuss practical suggestions to include psychosocial education and interventions in everyday practice—highlighting when a referral to a mental health care professional may be necessary. We conclude with a brief discussion on how lessons learned from the treatment of women suffering from pain with sexual activity may be applied to understudied groups including women in postpartum or postmenopause and men.

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20.1 The Interplay of POPF Problems and Psychosocial Factors

Psychosocial factors are multifaceted and potentially contribute to the etiology, maintenance, and exacerbation of POPF. Furthermore, psychological factors can play a role in treatment adherence and posttreatment adaptation. We will outline a few examples:

Psychological factors can *cause* adverse physiological reactions in women with maladaptive cognitions regarding vaginal penetration. These cognitions can include fear of pain, beliefs of genital incompatibility, disgust, etc. [10, 11]. When intercourse is attempted, a defensive guarding reaction of the pelvic floor muscles occurs. This makes the attempt impossible and/or painful, thus reinforcing negative expectations and false beliefs, potentially resulting in a lifelong inability to experience vaginal penetration [12, 13].

Psychological factors play a role in the *maintenance* and *exacerbation* of pain associated with provoked vestibulodynia, a hypersensitivity of the area of the vestibular region at the entrance of the vagina. With pain, pelvic muscle tension

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increases, resulting in pressure and friction at the entrance of the vagina; this in turn worsens the pain of provoked vestibulodynia—potentially further exacerbating pelvic floor reactivity, hypertonus, and pain [14]. A vicious cycle of pain, distress, and pelvic floor tension results [15].

The success of pelvic floor physical therapy depends to a good degree on *adherence* to home exercises [16]. Patients may not fully commit to exercises for a number of reasons including fear of failure ("If this does not work, I am a lost cause; nothing can help me"). Paradoxically, patients may not follow the therapist's homework instructions to avoid "risking failure" but "keeping hope." Conversely, patients may also be afraid to succeed. A woman during postpartum may worry that—should treatment be successful, she may have to engage in sexual activity more than she desires. In the absence of being able to effectively decline a sexual advance, symptoms such as urinary incontinence and/or pain may have taken the place of "not tonight honey, I have a headache."

Finally, physical therapy intervention may have been successful, but *posttreatment* quality of life and sexual function may not improve. Successful treatment of POPF may leave patients feeling that their sexuality and otherwise private parts of their bodies have become objects of medical intervention and lost their erotic appeal for both the patient and partner. Therapists may not necessarily be informed of posttreatment problems. However, it is not uncommon for women with significant or complete pain reduction following treatment to report continued poor sexual function, not engage in age-normative intercourse frequency, and/or not include vaginal penetration during sexual activity [5, 17].

20.2 The Fear-Avoidance Model of Pain

Whether part of etiology, maintenance, or exacerbation, psychosocial factors figure, sometimes prominently—sometimes tacitly, in the physical therapy treatment of POPF. One way in which a number of researchers and clinicians have come to conceptualize the interplay of physiological and psychological factors is based on the fear-avoidance model of chronic pain [18]. The model has been adjusted to problems of the POPF; in particular, for women who experience pain with sexual activity or the inability to experience intercourse (Fig. 20.1) [13, 15, 19, 20].

According to the model, pain or negative experiences (e.g., urinary leakage during sex, "failed" attempts at intercourse) result in particular ways of thinking about vaginal penetration that may be adaptive ("This is not feeling good, I think I need more sexual stimulation") or maladaptive. Distorted thought patterns include magnification (e.g., "This will get worse"), rumination

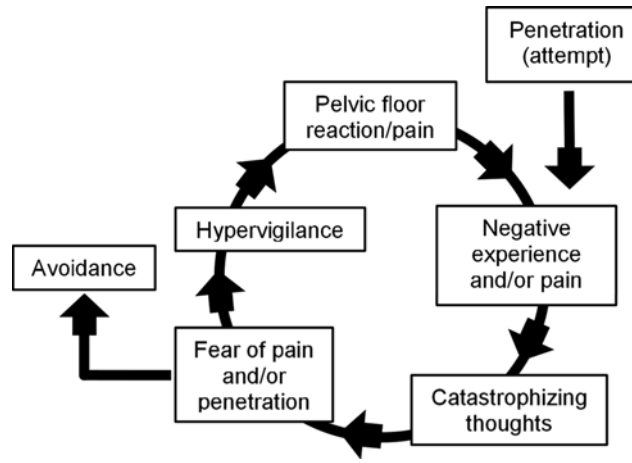

Fig. 20.1 The Fear-Avoidance Model of Pain detailing how maladaptive thinking can perpetuate the cycle of pain and lead to avoidance of or hypervigilance for pain with sexual activity

(e.g., "Something must be seriously wrong with me"), and helplessness (e.g., "Nothing can help me"). Research studies demonstrate the presence of catastrophizing thoughts [21] and their impact on pain maintenance and/or exacerbation [15, 22]. Catastrophizing thoughts result in specific fears of pain and vaginal penetration. In fact, ter Kuile and her colleagues [23] found that reduction in specific "coital fears" was the best predictor for successful treatment outcome in women with lifelong vaginismus. To cope with fear, the patient may avoid all activities related to pain and/or she may be hypervigilant for stimuli that are associated with specific fearful thoughts (e.g., pain, genital incompatibility). The latter can result in an exaggerated attention to physical sensations and increased anxiety that facilitates the experience of pain during attempted vaginal penetration. For example, researchers have noted increased attentional bias and negative affect towards pain-related stimuli in women with provoked vestibulodynia [24]. When sexual activity and intercourse are attempted, the woman experiences preexisting or increasing pelvic muscle hypertonus and/or defensive pelvic muscle reactivity. Increased muscle tone, along with lack of sexual arousal and lubrication, results in further worsening of pain. The inability to "achieve" (pain-free) penetration in turn contributes to negative experiences, confirms negative (maladaptive) expectations, and further exacerbates and perpetuates the cycle of pain and pelvic floor pathology.

20.3 Sexuality-Related Factors

Two psychosocial factors that are not explicitly included in the original fear-avoidance model of pain are sexual function and relationship factors. They are, however, an integral part of our extended model that we call the "vicious cycle of pain" with our patients (see Fig. 20.2).

Researchers have found evidence of low sexual satisfaction, impaired sexual function across the sexual response (desire, arousal, lubrication, orgasm), as well as elevated sexuality-related distress in women who experience POPF [24–26]. It is not surprising that pain affects sex in negative ways; what is disconcerting is that the fear and anticipation of pain can rob a couple of all affection and opportunities to find intimate connection and meaning. In clinical practice, it is not uncommon to hear a woman relate how she is reluctant to kiss or embrace her partner or respond to his affection for fear that this may result in an attempt at intercourse. Fear of a painful experience, "rejecting" the partner yet again, and/or not being able to "finish what she started" will keep a woman at a "safe" but lonely distance. In addition, focusing on intercourse as the predominant, heterosexual script may also detract from other opportunities to experience sexual connection. As a result, women report guilt and shame about not being able to be—what they perceive as adequate sexual partners, above and beyond the distress their partners may actually experience [27, 28]. An attempt at intercourse, motivated by guilt, obligation, fear of losing the partner, or mental calculations of when the couple "should be" intimate results in sexuality that is far from intrinsically motivated and further increases the likelihood of negative sexual experiences and low satisfaction [29–31].

20.4 Relationship Factors

Partners of patients with POPF may be told about symptoms of pain, but they are also involved in triggering pain in a sexual context. Despite this, relationship adjustment has been reported as often quite positive overall [26, 32]. However, partners can influence the symptomatic person's experience of pain and sexual adjustment in numerous ways. Rosen and her colleagues have conducted several investigations highlighting that partner responses to pain that are negative (hostility and frustration) or solicitous (attention and sympathy) are associated with increased pain intensity and decreased sexual satisfaction [33]. On the other hand, facilitative responses (encouragement and adaptive coping) were associated with decreased pain intensity and increased sexual function [34, 35]. The researchers demonstrated that on days when

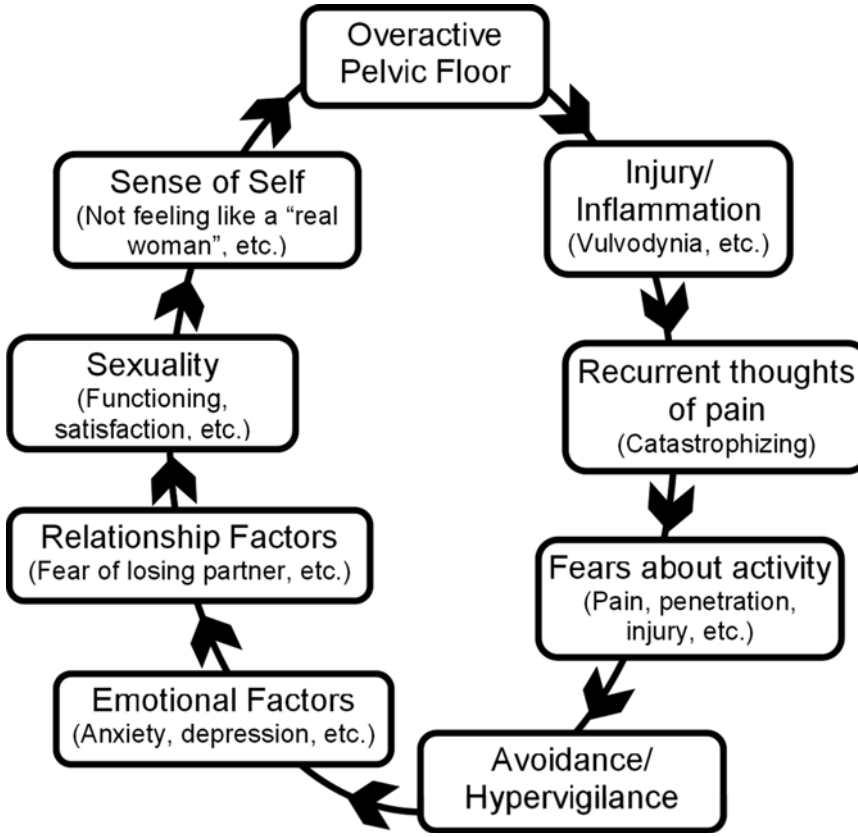

Fig. 20.2 Numerous factors contribute to the cause, maintenance, and exacerbation of the Vicious Cycle of Pain

a woman perceives her partner to respond in a facilitative manner, her *and* the partner's sexual function was improved. In a recent study, the researchers confirmed the negative effects of a woman's experience with pain associated with intercourse on both partners; taken together the available research suggests that that couples may benefit either from treatment that includes addressing relationship factors, or conjoint therapy may be recommended [36].

20.5 Psychosocial Interventions for Non-psychologists

Many health care professionals who are likely to work with persons experiencing POPF are not trained to conduct psychotherapy with their patients. However, the inclusion of psychoeducational aspects to the primary intervention is feasible and likely to increase treatment adherence, success, and maintenance. A good beginning point can be providing your patient with an extended and detailed version of the fear-avoidance model of pain—as described earlier, we refer to it as the "vicious cycle of pain" with patients (see Fig. 20.2). It can be used to highlight the multidimensional and multi-determined dimensions of pain, muscle reactivity, and chronic hypertonus. It can also identify potential roadblocks in treatment (e.g., critical partner, significant anxiety), provide rationale for multimodal treatment (e.g., sex therapy, relaxation), and identify problems that could lead to an iatrogenic effect of physical therapy treatment interventions (e.g., sexual abuse, PTSD).

Draw or hand the model to the patient and ask her to personalize how the "links in the chain" in the vicious cycle apply to her specific situation (in point format as this is more effective for

review). This can be done in the office or at home. Links in the chain can be discussed—if and when appropriate, during your primary treatment. You may also help the client to identify when and whom to consult should sex therapy, pain management, or psychological treatment be necessary. Furthermore, it is very helpful to explain to the patient that treatment can tackle any of the links of the chain but it may take the breaking of more than one link to "break the chain" or "break the vicious cycle" and arrive at satisfactory treatment outcome. This practical approach highlights the integrity of and rationale for a multimodal and/or multidisciplinary treatment approach and motivates the patient to address psychosocial factors in her pain experience.

20.5.1 A Word of Caution

Despite the integral role of psychosocial factors in the "vicious cycle of pain," it cannot be assumed that variables contributing to the development of problems of POPF are the same variables that maintain and exacerbate the problem/symptoms and vice versa. Hence, when highlighting the role of psychological factors, clinicians need to refrain from pathologizing patients and continuously highlight that "the pain is real." Or, as some colleagues explain, "Yes! The pain is in your head!" and then promptly continue to explain the role of central and cortical processes involved in the integration of sensory-discriminative, cognitive-evaluative, and affective-motivational dimensions of pain (see more below) [37–39].

20.6 Pain Management: Breaking the Chains of the Vicious Cycle of Pain

When closely examining the Fear-Avoidance Model of Pain and considering the accumulated literature on the role of mental health in affecting pain and its treatment [40–42], it is of little surprise that pain management techniques have been demonstrated as highly effective for chronic and recurrent pain [43] and pain associated with POPF [8]. Pain management can be delivered in a group format or to individuals in a variety of health care settings. However, in the general practice setting, a rationale for including the "brain" (i.e., the chains in the vicious cycle of pain) in the treatment of what the patient experiences as "physical" pain can be helpful. Once the patient understands that focusing on psychosocial factors does not invalidate her pain experience, she will be motivated to give pain management strategies a chance.

A very basic description of the Gate Control Theory of Pain [38] (see Fig. 20.3) can assist in identifying thoughts, feelings, activities, and circumstances that "open the gate" to (more) pain. Pain signals do not merely travel up the spinal cord (afferent nerve fibers) to be processed in the brain as nociceptive; there are also efferent nerve fiber pathways that descend the spinal cord from centers in the brain that process thoughts and emotions. These messages carry the response to the pain signal back to the receptor organ (e.g., to withdraw one's finger from a flame). Importantly though, they also serve to modulate processing of pain signals in the dorsal horn of the spinal cord, with a potential to augment ("opening the gate"), impede, or even prevent ("closing the gate") processing of pain signals (Fig. 20.3).

The drawing of the "vicious cycle of pain" provides a perfect summary of potential factors that are likely to "open or close the gate" for patients. In addition, a *pain journal* can further identify and elucidate behavioral and situational triggers for pain. In Table 20.1, the example of a pain journal is similar to a "thought record" used in cognitive behavioral therapy (CBT). The advantage of using this expanded and standardized version of the pain journal is that the patient (and therapist) can examine particular behaviors that trigger, maintain, and worsen pain—but also how thoughts and emotions can be adaptive or maladaptive in the management of pain. This clarifies further the different links of the vicious cycle of pain. Discussion of pain responses in the context of the Gate Control Theory of pain and what "opens or closes the gate" for a particular patient will encourage adaptive problem solving and

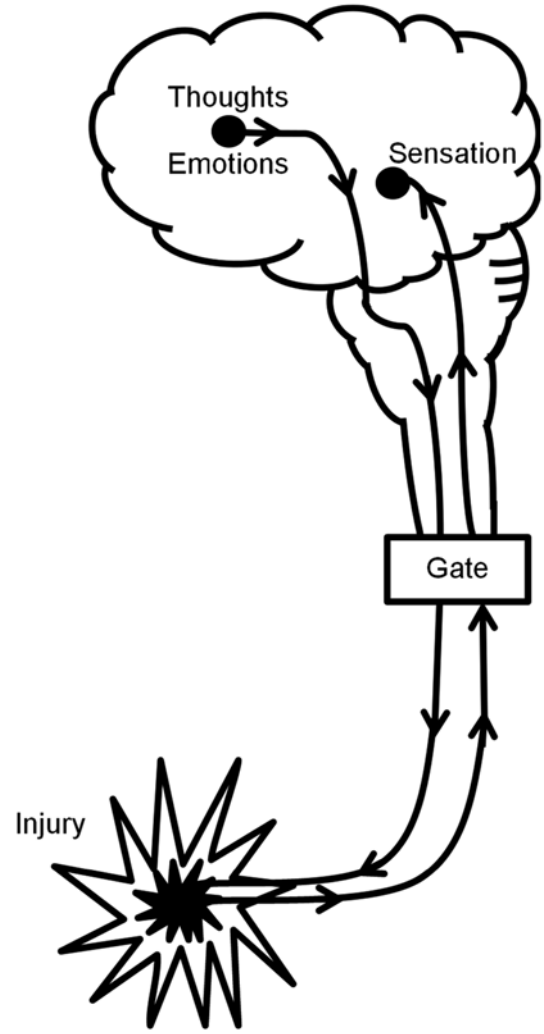

Fig. 20.3 Basic conceptual representation of the Gate Control Theory of Pain, which outlines how thoughts and emotions can alter the experience of pain

help the patient generate alternative re/actions that make sense in her context. Detailed planning and anticipating barriers are crucial to assure that the patient understands the treatment rationale and gives the intervention a try (Table 20.1).

20.7 When Fear and Anxiety Take Center Stage

More often than not, patients present with elevated fear and anxiety. Rosenbaum introduced a number of techniques that can assist therapists in effectively working with the anxious patient. This includes the "Rosenbaum Protocol" (see Table 20.2), a step-by-step exposure to interventions helping patients to recognize and contain growing anxiety and remain present during examination and treatment [44]. Rosenbaum expanded this approach to include mindfulness-based treatment methods to reduce a goal-driven approach and negative self-judgment by the patient (Fig. 20.2) [45].

The mindfulness approach involves teaching a patient how to attend to the pain experience rather than avoid it and then examine thoughts and feelings that accompany the pain. This approach can be complementary to and shares many similarities

Table 20.1 The Thought Record or diary is a systematic record of situational and behavioral triggers, thoughts, and emotions that exacerbate negative experiences, as well as adaptive coping responses

Date	Activity	Pain rating (0–10)	Thoughts	Feelings	Reflections[a]
–	Made love without penetration (finger insertion)	6 with insertion, 3 when his finger was inserted, 1 afterwards (for 10 min)	I can't believe I have pain even with finger insertion; this is really bad; I must be a lost cause; nothing helps me; it is only going to get worse	Defeated, guilty because we can't enjoy sex even without penetration; hopeless, sad, angry (with thought, why can't I have sex like other women?)	This was just before my period and I am often more sensitive and burn more easily, could this be the reason why I had pain with finger insertion? I don't usually have pain unless we include penile penetration? Mmm…feeling a little black & white…a little more hopeful…
–	–	–	–	–	–

[a]The addition of "reflections" allows the patient to use skills and insights already gained, "try out" new thoughts, observe changing emotions with changing thoughts, and consolidate gains

with CBT [8, 46]. Mindfulness differs in that where CBT may aim to alter the content of cognitions, mindfulness focuses on the relationships, and reactions to those thoughts. For example, rather than attempting to change the maladaptive cognitions related to pain (e.g., "This will never get better…"), a mindful approach may advocate attending to the pain while observing one's thoughts in a compassionate and nonjudgmental way.

In addition to learning "from oneself" how psychosocial factors contribute to maintaining the vicious cycle of pain, in a mindful approach to treatment, the client is invited to observe her reactions without judgement. It is normal for the body to react in protective ways when pain is experienced (e.g., eye blink when approached). It may be helpful to explain to a patient that pain is adaptive and people who suffer from congenital abnormalities that reduce pain thresholds are prone to injury and illness [39]. It is also important to note that a mere mental reflection on one's experience with pain may not suffice in changing behavior and/or contribute to treatment adherence, symptom reduction, and better quality of life. Rosenbaum suggests that responses to pain, such as catastrophization, avoidance, and procrastination, may not be cognitively driven and cannot be intellectually resolved [45]. Encouraging the patient to "stay with the feeling" may allow her—sometimes for the first time, to fully experience her feelings and explore her thoughts related to pain. Experiencing intense emotion, realizing that they subside without negative consequences, and that they are limited in time and experience, can be a powerful corrective experience [13]. Occasionally, however, emotions can be overwhelming and cause excessive distress. Some patients appear to be unable to access and/or experience their emotions. This may be a time to consider a referral to a mental health care professional. To paraphrase a patient, "Vulvodynia used to be my enemy…now, vulvodynia is a bit of a friend because it forced me to put a magnifying glass to my anxiety problems and finally tackle them—I am feeling so much better now… even though my pain is still not at zero!"

20.8 Including the Partner in Treatment

Surprisingly, few researchers have examined dyadic factors and partner characteristics in couples with problems associated with POPF. With

Table 20.2 The Rosenbaum Protocol for working with severely anxious patients

Step 1	Lying on the table. The client is asked to lie on the table, fully dressed, covered with a sheet. She is asked to rate her level of anxiety from low to high (0–5), and then asked what she needs in order to reach the number 0. These needs may include lying on her side in a more protected posture, the practitioner moving away from the table, or, she may need to get up off the table and go back to sitting in the chair, where she was able to rate herself at 0. Other "lowering anxiety" tools are introduced including deep breathing techniques. The exercise is repeated until she is able to lie on the table on her back with her knees flexed and together, and rate herself at 0–1
Step 2	Lying on the bed, fully dressed (with pants) and covered with a sheet, the client is asked to bend her knees and separate her legs. She is reminded that if she feels anxious with her knees open, she may do what she needs to relieve her anxiety, which is likely to be a return to the position of knees bent and together. This exercise is repeated until she is able to rate her anxiety level with her legs apart at 0–1
Step 3	As in Step 2 but without the sheet. Covering herself again with the sheet is considered to be one of the lowering anxiety options available to her
Step 4	As in Step 2 but wearing shorts instead of long pants, first with and then without the sheet
Step 5	As in Step 2 but with underwear only, with and without the sheet
Step 6	As in Step 2 without underwear, with and without the sheet

the results of an emergent body of literature [33–35] and borrowing from the chronic pain literature [47], we can assume that partners play a critical role in the pain (and treatment) experience. The couple may use their existing strengths to use a "teamwork approach" that identifies and removes interpersonal roadblocks and accelerates treatment. This can be achieved through partner participation in physical therapy sessions (potentially focusing on educational elements), taking part in homework exercises, and helping the couple improving communication, especially regarding POPF.

20.9 Sexual Intimacy in Couples with POPF

Rosen and her colleagues elegantly demonstrated the importance of adaptive coping in decreasing pain and increasing sexual adjustment and satisfaction for both partners [45]. While treatment may resolve POPF problems for many patients, others will have to adjust to pain or occasional pain and require tools to live a fulfilled life. Communicating about pain and sex and reconsidering sexual scripts and stereotypes are important centerpieces to this.

Basson's circular model of sexual response can be helpful to start the conversation. Figure 20.4 shows the original, basic model [48] but other references are included which outline recent variations and expansions of the model [46, 49, 50]. In essence, it is explained to the couple (or woman) that in long-term, committed relationships, spontaneous sexual desire may occur but more often than not, a woman may be at a relatively neutral place with regard to sex. The first step in starting a cycle of sexual response is arousal (not desire). Stimuli that serve to arouse her range considerably and depend on the individual woman, e.g., having had a particularly good day, romantic dinner, gentle caresses, closeness felt as a result of other daily activities, starting a role play, or a specific song. Some pleasure with arousal will increase the woman's desire to receive or give more stimulation. The dance between arousal and desire results in a physically and emotionally satisfying experience (that may or may not include intercourse). This experience results in an increase in closeness and intimacy in the couple (Fig. 20.4).

In our clinical experience, three important aspects are key to the explanation of the Basson's model and need to be discussed with the patient: First, the model normalizes that several factors can interfere with a physically and emotionally satisfying sexual event (e.g., pain, lack of technique, negative feelings towards the partner). This provides another opportunity for psychosocial intervention—in this case, addressing couple's feelings, sexual technique, and adaptive coping with pain.

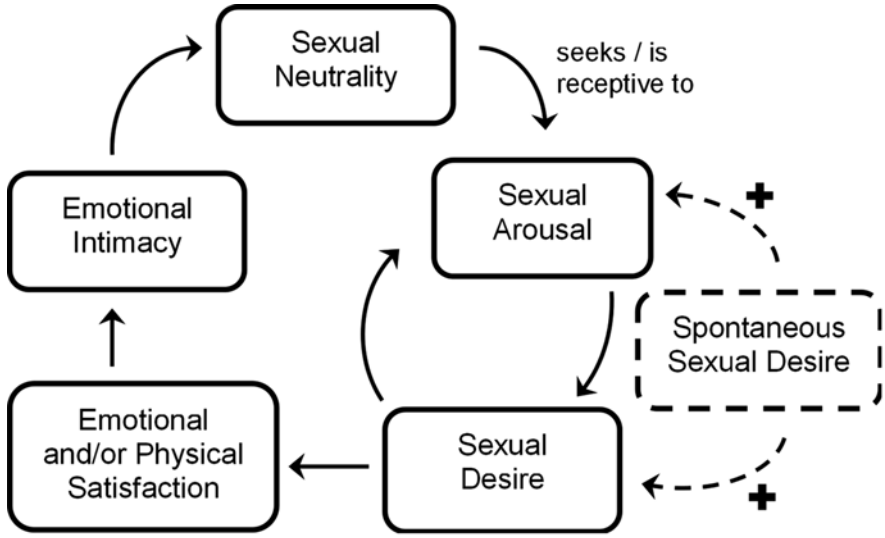

Fig. 20.4 Basson's Circular Model of Sexual Response is an intimacy-based model of arousal and desire

Second, the model normalizes the low levels or even absence of libido in long-term, committed relationships. It is the recollection of and desire for the pleasant feelings of emotional intimacy that become the driving force underlying passionate sexuality in long-term, committed relationships. Finally,and perhaps most important, the woman needs to feel completely assured that she can stop the cycle at any time without guilt, obligation to "go all the way," or negative repercussions. This is crucial for two reasons. If she engages in the cycle, the couple is extremely likely to experience intimacy that will bring them closer and thereby increasing the likelihood of further intimacy—everyone will be happy(er). If, however, she feels that engaging in the cycle means "no return," her likelihood of engaging is potentially more costly and she will be reluctant, evaluating her "desire." This is likely to result in far fewer intimate moments of connection for the couple, ultimately resulting in less emotional intimacy.

20.9.1 Normalizing Alternative Sexual Scripts and Increasing Sexual Connection

Part of discussing Basson's circular model can include an explanation that the heterosexual script of little foreplay resulting in intercourse and ending with his orgasm may not be a particular effective way of pleasure-filled, erotic love-making for couples in general [51] and with POPF problems in particular [45, 52]. Alternatives to intercourse can be discussed, highlighting that these are perfectly adequate and not mere compromise sexual activities in the absence of intercourse. Means of augmenting arousal such as the use of erotica, sex toys, and fantasy can be discussed and thereby normalized.

One intervention that requires little therapeutic know-how is the suggestion to make time and space for intimate connection. Patients with POPF likely present with a history of avoidance of sexuality and situations that could lead to negative experiences (e.g., pain, rejecting partner). Turning attention to investing and planning for sexuality is oftentimes quite a shift. Encouragement to consider any physical closeness that goes beyond what "friends would do" as "intimate connection" can take the pressure off the symptomatic partner. The non-symptomatic partner can be reassured using Basson's cycle of sexual response—once engaged in the cycle, and feeling increased emotional intimacy, increased frequency of sex, and more engaged sexual encounters are the likely result. Creating a conducive space for intimacy helps both partners to validate that their sexuality is important. A warm, subtlety lit, comfortable, and maybe sexy and erotic space (cushions, clothing,

decorations, scents, etc.) can make a substantial difference, provide a refuge from everyday activities and become a special space for the couple. The notion of planning for sexual interactions is often frowned upon by patients who wistfully remember their days of "spontaneous sex" early in the relationship. When these recollections are examined more carefully, it becomes obvious that this spontaneous sex was entirely planned and much anticipated. The proverbial dinner and movie predictably ended with sex. Dates were planned meticulously with regard to personal grooming, clothing, and more. Why not do the same when in a long-term relationship? What specific planning may be involved and how to preserve and protect special time to connect are up to couples and their particular preference. Your office may be a good place to start these conversations.

20.10 When to Refer

Ideally, patients work with a multidisciplinary team using a multimodal treatment approach that is tailored to the specific patient. However, insurance coverage, financial concerns, time constraints, and availability of trained health care professionals can impede access to "ideal" treatment. Patients can, however, achieve excellent treatment outcome with a therapist who conceptualizes POPF from a biopsychosocial perspective and empathically uses psychoeducational interventions, like the ones discussed above. However, for some patients, psychosocial factors may exceed therapist competence and professional practice boundaries including—but not limited to, sexual abuse history, post-traumatic stress disorder (PTSD), affective disorders, and major relationship conflict.

A good yardstick in evaluating whether a psychological problem needs to be addressed prior to, or concurrent with physiological treatment, is the degree to which the problem can interfere with the patient's ability to engage effectively in treatment (e.g., follow instructions to carry out and maintain home exercises). In addition, the level of personal and/or interpersonal distress is a necessary metric. How distressed is the patient and how disruptive is the distress to the patient's everyday function and interpersonal relationships? Patient Reported Outcomes Measurement Information System (PROMIS), funded by the National Institutes of Health (NIH), can assist clinicians in evaluating psychosocial symptoms and distress; PROMIS can be found on the NIH website: http://www.nihpromis.org/.

Finally, a referral for complementary therapies such as pain management, sex therapy, and couple counselling can accelerate physiological treatment and maximize maintenance of gains. Higher rates of anxiety and depression have been found in women who suffer pain with sexual activity, and affective symptoms were associated with increased pain reports [53–55]. Increasingly, researchers highlight the role of partner response and couple communication in the pain experience of the suffering partner and the sexual adjustment of both partners [33–36]. Psychological treatments for sexual dysfunction in general [15] and pain associated with sexual activity in particular [6, 8] have received considerable research attention. Interventions focus on cognitive-behavior therapy (e.g., 16), couple-focused therapy approaches [56], and mindfulness [57], and treatment outcome is uniformly promising. Psychological interventions including sex therapy and pain management as a stand-alone treatment can result in decreased pain and improved sexual function posttreatment [56, 57], and further improvement can be reported with follow-up [16]. A combination of physiological and psychological approaches, as highlighted throughout this chapter may be the ultimate gold standard for treatment of POPF and related sexual symptomatology [58].

20.11 POPF Problems in Men and During Women's Reproductive Lifecycle Events

Much has been learned from the research focusing on women with disorders such as vestibulodynia, on how pain interferes with sexuality and other

domains of life—but also on how treatment from a biopsychosocial perspective offers hope and resolution. However, more research is necessary to better understand Problems with an overactive pelvic floor (POPF) and how to effectively treat them. Researchers are just beginning to pay attention to the role of POPF and pain during lifecycle transitions in women, pregnancy [22, 58], and menopause [59–61]. For example, for women who experience genito-pelvic pain during pregnancy, pain-related anxiety plays a central role in increasing the likelihood of postpartum pain [22]. Men with chronic pelvic pain syndrome and sexual dysfunction may benefit significantly from a multidisciplinary treatment including pelvic floor interventions [58, 62, 63] and psychosocial interventions [64, 65]. For example, in men with chronic pelvic pain syndrome, psychosocial factors are associated with sexual adjustment, symptom experience, and overall quality of life [66–68]. Various components of the fear-avoidance model have also received support, in particular, pain catastrophization [65]. Finally, dyadic factors are associated with increasing the effectiveness of pelvic floor home exercises in men who underwent prostatectomy [69].

20.12 Conclusions

Health care professionals have become progressively more sophisticated in their conceptualization of health problems and acknowledge the role of psychosocial factors in the etiology, maintenance, exacerbation, and posttreatment adjustment of their patients. Working together in multidisciplinary teams can be exceedingly beneficial for both patient *and* provider; while referrals to clinicians offering complementary treatments may be needed by some of our patients. However, a number of psychosocial interventions that are based on education, reflection, and insight into the complexity of POPF can be included in medical and physical therapists' everyday practice. This chapter has provided some concepts and tools that will facilitate and support this initiative and assist in broadening and enriching practitioners' work with their patients.

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