Definitions and Basic Etiology of the Overactive Pelvic Floor

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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

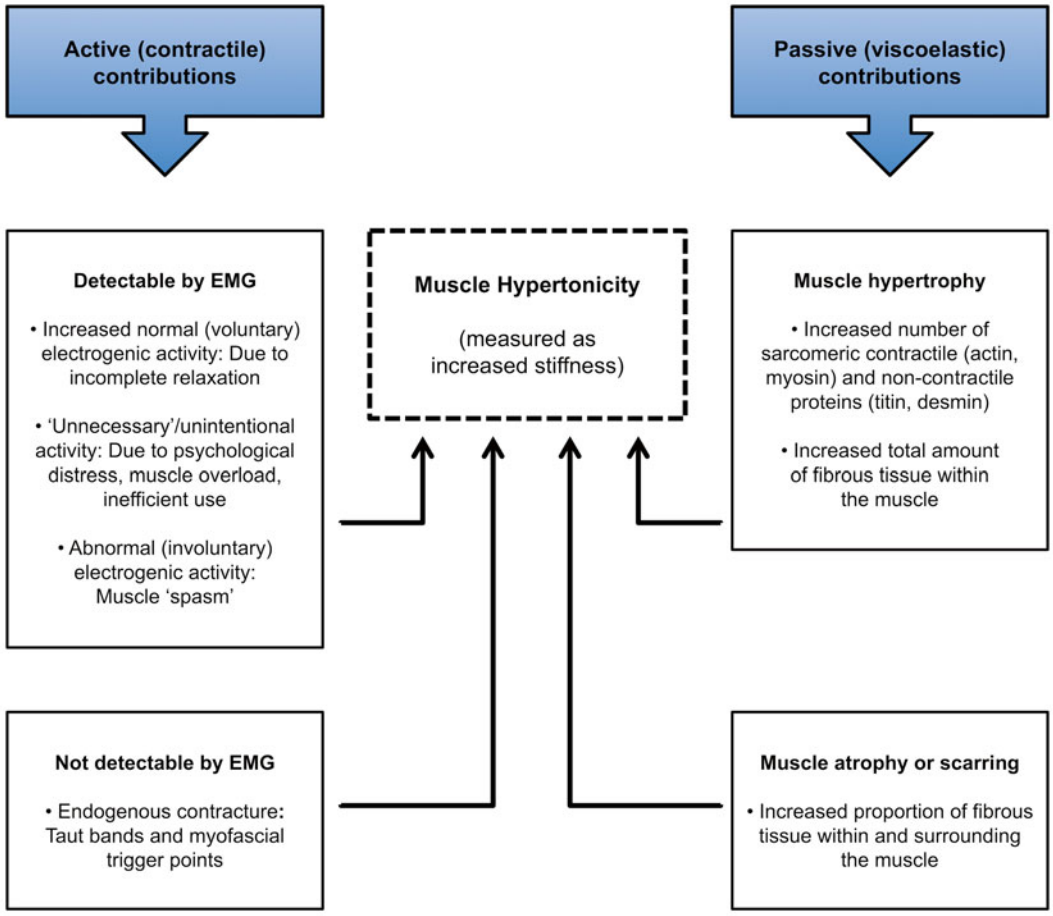

Fig. 1.1 Possible sources of muscle hypertonicity in a normally innervated skeletal muscle. Based on information from Simons and Mense [18] and Gajdosik [51]

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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