Overactive Pelvic Floor: Gastrointestinal Morbidities

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

M. Beer-Gabel, M.D. (🖂)

Neurogastroenetrology and Pelvic Floor Unit, Sheba Medical Center, Tel Hashomer, Israel e-mail: marcobg7@gmail.com 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.

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 termishows differing pathophysiological nology 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:

 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 bladderrelated 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 nonrelaxing 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 highresolution 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.

References

- FitzGerald MP, Kotarinos R. Rehabilitation of the short pelvic floor. I: background and patient evaluation. Int Urogynecol J Pelvic Floor Dysfunct. 2003;14(4):261–8.
- Simons DG, Travell JG. Myofascial origins of low back pain. 3. Pelvic and lower extremity muscles. Postgrad Med. 1983;73(2):99–105. 108.
- Faubion SS, Shuster LT, Bharucha AE. Recognition and management of nonrelaxing pelvic floor dysfunction. Mayo Clin Proc. 2012;87(2):187–93.
- Butrick CW. Pathophysiology of pelvic floor hypertonic disorders. Obstet Gynecol Clin North Am. 2009;36(3):699–705.
- Thiele G. Coccygodynia: cause and treatment. Dis Colon Rectum. 1963;6:422–36.
- de Groat WC, Fraser MO, Yoshiyama M, Smerin S, Tai C, Chancellor MB, Yoshimura N, Roppolo JR. Neural control of the urethra. Scand J Urol Nephrol Suppl. 2001;35:35–43; discussion 106–125.
- Fowler CJ. The perspective of a neurologist on the treatment-related research in fecal and urinary incontinence. Gastroenterology. 2004;126:S172–4.
- Sapsford RR, Hodges PW, Richardson CA, Cooper DH, Markwell SJ, Jull GA. Co-activation of the abdominal and pelvic floor muscles during voluntary exercises. Neurourol Urodyn. 2001;20(1):31–42.
- Klingele CJ, Lightner DJ, Fletcher JG, Gebhart JB, Bharucha AE. Dysfunctional urinary voiding in women with functional defecatory disorders. Neurogastroenterol Motil. 2010;22(10):1094–E284.
- Van Ginkel R. The effect of anorectal manometry on the outcome of the treatment in severe childhood constipation: a randomized, controlled trial. Pediatrics. 2001;108(1), E9.
- Loening-Baucke V. Chronic constipation in children. Gastroenterology. 1993;105(5):1557–64.
- van Ginkel R, Reitsma JB, Büller HA, van Wijk MP, Taminiau JA, Benninga MA. Childhood constipation: longitudinal follow-up beyond puberty. Gastroenterology. 2003;125(2):357–63.
- Loening-Baucke V, Swidsinski A. Constipation as cause of acute abdominal pain in children. J Pediatr. 2007;151:666–9.
- Loening-Baucke V. Urinary incontinence and urinary tract infection and their resolution with treatment of chronic constipation of childhood. Pediatrics. 1997; 100(2 Pt 1):228–32.
- Leroi AM, Bernier C, Watier A, Hémond M, Goupil G, Black R, Denis P, Devroede G. Prevalence of sexual abuse among patients with functional disorders of the lower gastrointestinal tract. Int J Colorectal Dis. 1995;10(4):200–6.

- Drossman DA, Leserman J, Nachman G, Li ZM, Gluck H, Toomey TC, Mitchell CM. Sexual and physical abuse in women with functional or organic gastrointestinal disorders. Ann Intern Med. 1990;113(11): 828–33.
- Leserman J. Relationship of abuse history to functional gastrointestinal disorders and symptoms: some possible mediating mechanisms. Trauma Violence Abuse. 2007;8(3):331–43.
- Cervero F. Visceral versus somatic pain: similarities and differences. Dig Dis. 2009;27 Suppl 1:3–10.
- Chiarioni G, NardoA VI, Romito A, Whitehead WE. Biofeedback is superior to galvanic stimulation and massage for treatment of levator ani syndrome. Gastroenterology. 2010;138:1321–9.
- Giamberardino MA, Costantini R, Affaitati G, Fabrizio A, Lapenna D, Tafuri E, Mezzetti A. Viscerovisceral hyperalgesia: characterization in different clinical models. Pain. 2010;151(2):307–22.
- Walker EA, Katon WJ, Jemelka R, et al. The prevalence of chronic pain and irritable bowel syndrome in two university clinics. J Psychosom Obstet Gynaecol. 1991;12(suppl):65–70.
- Gelbaya TA, El-Halwagy HE. Focus on primary care: chronic pelvic pain in women. Obstet Gynecol Surv. 2001;56:757–64.
- Bharucha AE, Wald A, Enck P, Rao S. Functional anorectal disorders. Gastroenterology. 2006;130:1510–8.
- Rao SS, Welcher KD, Leistikov JS. Obstructive defecation: a failure of rectoanal coordination. Am J Gastroenterol. 1998;93:1042–52.
- Bordeianou L, Savitt L, Dursun A. Measurements of pelvic floor dyssynergia: which tests results matters? Dis Colon Rectum. 2011;54:60–5.
- 26. Minguez M, Herreros B, Sanchiz V, Hernandez V, Almela P, Añon R, Mora F, Benages A. Predictive value of the balloon expulsion test for excluding the diagnosis of pelvic floor dyssynergia in constipation. Gastroenterology. 2004;126(1):57–62.
- Rao SS, Mudipalli RS, Stessman M, Zimmerman B. Investigation of the utility of colorectal function tests and Rome II criteria in dyssynergic defecation (Anismus). Neurogastroenterol Motil. 2004;16(5): 589–96.
- Wald A, Bharucha AE, Cosman BC, Whitehead WE. ACG Clinical Guideline: Management of Benign Anorectal Disorders. Am J Gastroenterol. 2014;109: 1141–57.
- Bharucha AE, Fletcher JG, Seide B, Riederer SJ, Zinsmeister AR. Phenotypic variation in functional disorders of defecation. Gastroenterology. 2005; 128(5):1199–210.
- Unger CA, Pretorius DH, Weinstein MM. Pelvic floor imaging. Obstet Gynecol Clin North Am. 2011;38(1): 23–43.
- Beer-Gabel M, Teshler M, Barzilai N, Lurie Y, Malnick S, Bass D, Zbar A. Dynamic transperineal ultrasound in the diagnosis of pelvic floor disorders: pilot study. Dis Colon Rectum. 2002;45(2):239–45; discussion 245–8.

- 32. Beer-Gabel M, Teshler M, Schechtman E, Zbar AP. Dynamic transperineal ultrasound vs. defecography in patients with evacuatory difficulty: a pilot study. Int J Colorectal Dis. 2004;19(1):60–7.
- Beer-Gabel M, Carter D. Comparison of dynamic transperineal ultrasound and defecography for the evaluation of pelvic floor disorders. Int J Colorect Dis. 2015;30(6):835–41.
- 34. Wald A. Functional anorectal and pelvic pain. Gastroenterol Clin Noth Am. 2001;30:243–51.
- Chiarioni G, Whitehead WE, Pezza V. Biofeedback is superior to laxatives for normal transit constipation due to pelvic floor dyssynergia. Gastroenterology. 2006;130:657–64.
- 36. Rao SS, Seaton K, Miller M, Brown K, Nygaard I, Stumbo P, Zimmerman B, Schulze K. Randomized controlled trial of biofeedback, sham feedback, and standard therapy for dyssynergic defecation. Clin Gastroenterol Hepatol. 2007;5:331–8.
- Bharucha AE, Trabuco E. Functional and chronic anorectal and pelvic pain disorders. Gastroenterol Clin North Am. 2008;37:685–96.
- Maria G, Cadedu F, Brandara F, Marniga G, Brisind G. Experience with type A botulinum toxin for treatment of outlet type constipation. Am J Gastroenterol. 2006;101(11):2570–5. Epub 2006 Oct 4.

- Abbott JA, Jarvis SK, Lyons SD, Thomson A, Vancaille TG. Botulinum toxin type A for chronic pain and pelvic floor spasm in women: a randomized controlled trial. Obstet Gynecol. 2006;108(4):915–23.
- Langford CF, Udvari Nagy S, Ghoniem GM. Levator ani trigger point injections: an underutilized treatment for chronic pelvic pain. Neurourol Urodyn. 2007; 26(1):59–62.
- Faubion SF, Shuster LT, Bharucha AE. Recognition and management of non relaxing pelvic floor dysfunction. Mayo Clin Proc. 2012;8(2):187–93.
- 42. Falletto E, Masin A, Lolli P, Villani R, Ganio E, Ripetti V, Infantino A, Stazi A. Is sacral nerve stimulation an effective treatment for chronic idiopathic anal pain? Dis Colon Rectum. 2009;52(3):456–62.
- 43. Dudding TC, Thomas GP, Hollingshead JR, George AT, Stern J, Vaizey CJ. Sacral nerve stimulation: an effective treatment for chronic functional anal pain? Colorectal Dis. 2013;15(9):1140–4.
- Baeten CG. Status of sacral neuromodulation for refractory constipation. Colorectal Dis. 2011;13 Suppl 2:19–22.
- Sharma A, Liu B, Waudby P, Duthie GS. Sacral neuromodulation for the management of severe constipation: development of a constipation treatment protocol. Int J Colorectal Dis. 2011;26(12):1583–7.