TUTORIAL

Chronic Female Pelvic Pain—Part 2: Differential Diagnosis and Management

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■ **Abstract:** Pelvic pain is a common condition. Treatment interventions have traditionally targeted biomedical conditions with variable success. Utilizing a systematic approach to examination of the pelvic girdle and related organ systems contained within the pelvis will aid the clinician in identifying the painful structure(s) as well as the associated impairments limiting functional recovery. From this, a complete management program can be instituted. The following description of gynecologic, urologic, gastrointestinal, musculoskeletal, and neurologic conditions that can cause or are associated with chronic pelvic pain leads to conservative management proposals based on the available evidence. Finally, nonoperative interventional strategies are described, which target the pain system from a cognitive behavioral perspective, address movement dysfunctions, and address interventional pain technique possibilities.■

Key Words: pelvic pain, rehabilitation, female, intervention, management

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INTERPRETATION, DIFFERENTIAL DIAGNOSIS, AND MANAGEMENT GUIDELINES

Pelvic pain is a common condition with a prevalence of 16% to 25%.[1] Optimizing function is a goal of patients seeking treatment for many conditions. Women with chronic pelvic pain (CPP) face additional barriers in their search to optimize function because of: (1) cultural and psychological barriers to discussing pelvic pain symptoms; (2) commonly utilized biomedical treatment approaches; and (3) the specialization of the many practitioners treating the pelvic region. Applying a systematic examination schema allows the clinician to identify the pain generator(s), as well as contributing factors to the persistence of the pain, and develop intervention strategies to target each (previously described in Part I of this tutorial). The following

is an overview of how conditions within each of the various body systems contribute to CPP, and a description of management options for each.

There are several possible explanations for CPP, including disorders of the gynecological, urological, gastrointestinal, musculoskeletal, and/or the nervous systems (Appendix I). Of the various diagnoses, the most frequently noted are endometriosis (33%) and adhesions (24%), as well as an absence of pathology in 35% of women with pelvic pain who have received diagnostic laparoscopy. Pelvic congestion syndrome (PCS) with dilated pelvic veins resulting in reduced blood flow may be an explanation for some cases of CPP. Janicki proposes that CPP may be a form of complex regional pain syndrome (CRPS) or it may be a form of central sensitization of the nervous system.[2,3]

Pelvic Pain Originating from the Gynecological System

Urogenital function depends on the integrity of the muscular, connective, and neural tissues of the pelvic floor. Dysfunction of any of these tissues may lead to insufficiency of the organ support and loss of sphincter mechanisms for bladder and bowel function, as well as loss of postural support. The need to analyze normal function from dysfunction serves as a foundation to diagnosis and effective treatment.

Key gynecologic conditions that contribute to CPP include pelvic inflammatory disease (PID), endometriosis, adnexa pathologies (ovarian cysts, ovarian remnant syndrome), uterine pathologies (leiomyoma, adenomyosis), and pelvic girdle pain associated with pregnancy. While these painful conditions are typically diagnosed by the gynecologist, obstetrician, or the general practitioner who is practicing obstetrics, it is the resultant pain and sensitization of the autonomic and somatic nerves that innervate these structures, which can lead to the disability experienced by the women suffering from CPP.[4]

Several major and minor sexually transmitted diseases (STDs) can cause pelvic and vulvar pain. Major STDs are reportable to the Centers for Disease Control, because of their public health impact. These include diseases such as syphilis, chlamydia, gonorrhea, and HIV/AIDS. Minor STDs are not required to be reported, including trichomoniasis, vaginitis, and genital herpes. Often, the symptoms associated with these minor STDs are mild to nonexistent, thus delaying treatment. [5,6]

An unfortunate consequence of contracting a STD is the possibility of developing PID, where the highest incidence is observed in women 15 to 25 years of age. This condition that involves the upper genital tract and reproductive organs in women is the leading cause of infertility in women.[7] The most common cause is because of chlamydia and gonorrhea.[5] Use of intrauterine devices (IUDs) and douching exacerbates the infection. Unfortunately, both can be symptom free in women, thus delaying treatment.

Symptoms of STDs include vaginal discharge with a foul odor, frequent urination, and painful intercourse, as well as vaginal bleeding after intercourse, between menstrual periods, or after menopause. Risk factors in this population are high-risk sexual behavior, early onset of sexual activity, history of STDs, and a partner with a STD history. A history of PID is a significant pain problem in the 18- to 25-year-old population, thus contributing to the development of CPP.[8] Treating young women for PID who present with adnexa, or uterine and cervical motion tenderness in the absence of other pathologies, can help prevent progression to chronic pain. Motion tenderness refers to the discomfort experienced when the cervix is palpated and moved in a side-to-side direction with the palpating finger. Normally, this should not be painful or uncomfortable.

For the general practitioner, it is important to identify those patients who are at risk of PID and ensure that they have received proper medical intervention as soon as possible. PID typically responds to antibiotic treatment, but for those without previous treatment or for whom treatment was not effective, a chronic viscerosomatic pain condition can develop whereby abdominal pain varies from intermittent to constant. PID may respond to physical therapy treatment utilizing pain-relieving modalities to the lower thoracic–upper lumbar regions, as well as directly over the lumbar spine and over the lower abdominal area. The expectation for physical therapy is not to fully ameliorate pain, but to provide adequate reduction in pain to allow activities of daily living (ADL).

Endometriosis is defined as the presence of endometrial glands and stroma outside the endometrial cavity.[9] The exact etiology of endometriosis has not been elucidated. Several theories have been proposed, but no 1 theory appears to explain the presence and growth of endometrial tissue outside the uterine cavity. It is generally accepted that endometriosis has a multifactorial, inherited predisposition.[10] Endometriosis

may present as a metaplastic change of normal tissue outside the uterine cavity. Lymphatic or vascular factors may be involved as evidenced by the occasional presence of endometrial tissue in far distant sites, such as the brain, lung, skin, and eye.[10] The theory that has a high level of acceptance is that it is a result of retrograde menstrual flow. However, this concept does not fully explain the development of endometriosis. Instead, there likely is a predisposition to development of endometriosis as a result of immune system or hormonal dysfunction. This is supported by the incidence of endometriosis in women after hysterectomy, tubal ligation, and in men who are undergoing treatment with estrogen.[10,11]

Diagnosis of endometriosis is often made based on the patient's reported symptoms. Typical patients are women in their 30s, nulliparous, involuntarily infertile with secondary dysmenorrheal and pelvic pain.[10] The difficulty in diagnosing endometriosis lies in the lack of visualization of endometrial tissue in 30% to 50% of all women.[10,12,13]

Endometriosis presents as a deep dyspareunia (pain during sexual intercourse), dyschezia (pain or difficulty with defecation), and dysmenorrhea. Endometriosis is relatively common, with prevalence of 1% all of US women.[14] For those who develop endometrial tissue growth outside the uterine cavity, it may not be the cause of pelvic pain, as there are equal numbers of women with painful endometrial tissue as without.[15] In diagnostic laparoscopy for pelvic pain, up to 40% of patients do not demonstrate pathological endometrial tissue findings.[4] The pain from endometriosis is cyclical and can range from intermittent dysmenorrhea that flares just prior to the onset of the menstrual cycle to chronic viscerosomatic pain that interferes with most aspects of life.

Treatment options for endometriosis include medical, surgical, and interventional pain management techniques. Conservative measures are first started as nonsteroidal anti-inflammatory drugs, progestins, androgenic hormones, estrogen–progestin combinations, and gonadotropic-releasing hormone agonists.[16] The goal of hormone treatment is to lead to atrophy of the endometrial implants. Surgical treatment options are laparoscopic excision of endometrial implants, total abdominal hysterectomy with or without salpingo-oopherectomy, presacral neurectomy (PSN), and laparoscopic uterine nerve ablation (LUNA).[17] Patients with endometriosis may also benefit from interventional pain management techniques, such as superior hypogastric plexus blocks and neuromodulation.[18,19] Alternative treatments for endometriosis may include acupuncture. Highfield et al.[20] presented 2 case reports of adolescent girls with CPP secondary to endometriosis who experienced good pain relief with acupuncture treatments.

The clinician must keep in mind the innervation of the gynecological system and the possibility of viscerosomatic convergence when requested to manage the pain of endometriosis. Physical therapy may be helpful by manually or electrically influencing the viscerosomatic convergent pain pathways at the thoracolumbar areas. Application of soft-tissue mobilization, thermal modalities, and Transcutaneous electrical neuromuscular stimulation (TENS) may be warranted. Additionally, as a result of viscerosomatic convergence, the clinician must evaluate posture and stability of the lumbosacral spine and provide appropriate measures to decrease complications for pelvic ring instabilities, sacroiliac joint (SIJ)-related pain, and lumbar neuromuscular dysfunctions.

Pelvic pain during or immediately following pregnancy may develop viscerosomatic sensitization as part of the pathology that contributes to pain. However, this condition may additionally present with musculoskeletal involvement of the pelvic girdle. While pelvic pain during pregnancy affects over 50% of women, 16% of them report persistent pain twelve months postpartum.[21] Such patients present with one or several of a constellation of symptoms. The most common is posterior sacral or buttock pain of variable intensity. Often, there can be complaints of abdominal pain that is deep and difficult to locate. Symptoms may wax and wane, be impacted by the menstrual cycle, and be reported as general fatigue or an overall sense of being unwell.

The mechanisms for persistent pelvic girdle pain are attributed to hormonal and biochemical factors but are not well understood. Mechanical testing of the pelvic girdle can identify those women who have musculoskeletal causes of pelvic pain. O'Sullivan suggested a classification scheme to assist in better understanding the pathophysiologic mechanisms to this condition and in providing causal treatment.[22,23] This testing sequence is included under musculoskeletal assessment of the pelvic ring discussed in Part I of this series.

Treatment for pelvic girdle pain is limited and often self-resolving when the patient's physiologic condition returns back to their normal prepregnancy state. However, a few subsets of patients will continue to have

pain after resolution of pregnancy. A 6-year follow-up study after pregnancy by Ostgaard et al. showed that 7% of women still had continued pain causing severe disability. According to the European Guidelines for the diagnostic and treatment of pelvic girdle pain, treatment should be multifactorial and begin with the use of an individualized exercise program focusing on specific stabilizing exercises, individualized physical therapy, use of a pelvic belt applied for short periods of time, and intra-articular injections for SIJ pain.[24] Medications should only be taken for pain relief if above modalities fail. According to the US Food and Drug Administration pregnancy category system, category A and B drugs have not been shown to cause fetal harm, while category C, D, and X are at risk for fetal harm and not recommended. Safe medications included in category A and B are acetaminophen, opioids, local anesthetics (LA), and epidural steroids given in a limited trial basis.[25,26] However, most experts would agree to avoid any medications if possible during pregnancy. Interventional treatments include local anesthetic steroid injections at the SIJ without fluoroscopy and trigger point injections at hypersensitive tender locations.

First described in 1857, PCS was considered to be a tubo-ovarian varicocele, which is analogous to scrotal varicocele in men. PCS is a disease of the childbearing years, usually seen in the late 20s to early 30s, although other ages have been reported.[27] The pathology is similar to varicose veins with fibrosis seen within the tunica intima and media, as well as muscular hypertrophy and proliferation of capillary endothelium within the uterus.[28,29]

Symptoms produced by PCS can vary from side to side. Deep dyspareunia and postcoital pain lasting from a few hours to several days are hallmarks of this condition. Some women complain of exacerbation of pain with prolonged standing, lifting, walking, or increased intra-abdominal pressure.[27] Uterine enlargement, thickened endometrium, multicystic ovaries, and increased hemorrhoids and varicosities on the vulva are the visible manifestations of congestion. Stones[30] reports dilated uterine and ovarian veins with reduced venous clearance of contrast medium. Thus, trans-fundal venogram may be the diagnostic test of choice. Women may report associated symptoms of irritable bowel syndrome (IBS), interstitial cystitis (IC), frequency urgency syndrome, and chronic headaches.

Treatments with medications that lead to hormonal suppression may relieve symptoms. Farquhar[31] looked at medroxyprogesterone acetate (MPA), which initially looked promising with patients reporting a 50% reduction in pain and improvements in venograms. However, pain returned after stopping MPA, and patients in the placebo group continued to have pain relief.[31]

More recently, a suppression of ovarian function with Goserelin has been shown to be more successful. Soysal et al.[32] showed that goserelin was superior in treating PCS when compared to MPA. At 1 year after 6 months of treatments, the goserelin group had significant pain relief and venogram improvement compared to the MPA group.[32] Ovarian and pelvic vein embolization appears to respond fairly well to these management strategies.[28,29,33] Physical therapy may be of benefit, where the use of manual lymph drainage techniques, exercise, or postural measures may assist in decongesting venous circulation.

Vulvodynia

Vulvodynia is defined as a chronic vulvar discomfort with duration of at least 3 months.[34,35] Several subsets of vulvar pain are described. Generally, this discomfort may be expressed as pain, burning, itching, dyspareunia, stinging, rawness, or ''irritation'' of a constant or intermittent nature.[36] Early results from a survey by the National Vulvodynia Association reported an age range of 11 to 75 years (mean age of 43) in women with vulvodynia. Vulvodynia is classified as primary or secondary. Primary vulvodynia is defined as an onset of symptoms with the first sexual experience or tampon use. Secondary vulvodynia differs from primary in that the onset occurs after first sexual experience or tampon use. Classification of pure vulvodynia implies that it is present with palpation only versus mixed vulvodynia that is present with or without palpation. This condition may be organic or idiopathic in etiology.

Many clinicians suspected a neuropathic etiology for vulvodynia. While most women experience allodynic vulvodynia, the pain can be experienced as hyperalgesia and/or dysesthesia or as all 3 types concurrently. One possible cause for the neuropathic nature of this condition may be related to a stretch injury of the nerve to the levator ani or the pudendal nerve in response to prolonged second-stage labor or pelvic floor descent. Additionally, this injury may be the result of episiotomy or straddle injury in a motor vehicle accident. Other causes include hormonal changes,

tumors and cysts, surgical side effect, or the result of using steroids and antiviral medications. Clinicians should not hesitate to ask the patient regarding their use of pads, deodorant sprays, and/or contraceptives used when assessing etiology. Definitive diagnosis is aimed at identifying painful areas and assessing skin changes. Laboratory tests are useful for ruling out conditions, such as condylomatous vaginitis, lichenoid vaginitis, lichen planus, and other dermatologic conditions.

Management with a multidisciplinary approach appears to be ideal.[37,38] Management should include biofeedback to decrease hypertonicity of the pelvic floor, if this exists. Additionally, the application of manual or electrotherapeutic input to the thoracolumbar and sacral areas to influence somatic and visceral afferents may be beneficial. Lidocaine gel, antidepressant medications, and local Botox injections may be indicated in some women. Psychological support, cognitive behavior therapy, and sexological counseling may be considered.[34]

Vulvodynia, like many of the pelvic disorders, is difficult to treat. Many women may experience this painful syndrome for years, becoming chronic in nature. However, some women spontaneously recover. According to Reed,[39] recent studies have found that one-half of women who report that have had prolonged vulvar pain no longer have symptoms of vulvodynia. Many authors agree that vulvodynia is best managed by a multidisciplinary approach. Treatment strategies include oral medications, such as tricyclic antidepressants (TCAs) as well anti-seizure agents that include gabapentin and pregabalin, topical creams with estrogen or lidocaine, biofeedback, behavioral therapy, surgery (vestibuloplasty, vestibulectomy, and perineoplasty), botulinum therapy, and interventional pain techniques that include local anesthetic and steroid injections, superior/inferior hypogastric plexus cryoneurolysis, and neuromodulation.[18,19,40–53]

Vulvar Vestibulitis Syndrome (VVS)

This condition, which is often idiopathic, is a subset of vulvodynia with pain experienced in the vulvar region, especially characterized by entrance dyspareunia. This condition is classified similar to vulvodynia (ie, primary and secondary). However, the pathogenesis is unclear. Serial use of antibiotics or highly progestational agents appears to trigger the onset of VVS, while the use of panty liners or menstrual pads can exacerbate symptoms. Granot reports low blood pressure, as well as personality factors, predisposes the development of VVS. The inflammation of the Bartholin's gland and/or vestibular glands at the base of the hymen is often observed. [54]

Vulvar vestibulitis syndrome is very difficult to treat, and a lack of treatment efficacy suggests that many factors influence the pathology.[35,55] Because of dyspareunia, psychological and marital difficulties exist. These difficulties merit psychological interventions, where psychotherapy, biofeedback, and counseling can serve as mainstays for treating this condition. Topical estrogen can be helpful in managing the pain of vulvar vestibulitis, as can be a low oxalate diet for selected patients. Vestibulectomy and perineoplasty, or surgical removal of the vulvar vestibule or perineum, appear to be most effective if performed when there is no evidence of viral DNA in vulvar tissue, or in younger patients with a short history of vulvodynia.[34,55]

Dyspareunia

Dyspareunia is another subset of vulvodynia, specifically describing pain with intercourse in the absence of vaginismus.[56] It is not a diagnosis in and of itself, but a set of symptoms with a potential underlying serious organic pathology. It can be experienced at the introitus, midvaginal, or deep vaginal regions. It can be painful only during active penetration or persist after penetration is stopped. Heim has identified physical, psychogenic, and combined causes for this condition, including inadequate lubrication, vaginal mucosal atrophy, infection, and scarring after episiotomy or other surgeries. Deep vaginal dyspareunia may occur in response to PID, endometriosis, or other causes. In addition, it may be an indication of sympathetic pain processes that stem from abnormalities of the cervix or ovaries. Symptoms include burning pain, rawness, and itchiness, which are often accompanied by anxiety and distress. Conservative management may include modalities, massage, mechanical dilators, and relaxation training with the goal of decreasing physical symptoms.[57]

Clitoral Pain

Another subset of vulvodynia is clitoral pain caused by neuralgia of the pudendal nerve. The symptoms associated with this condition are localized to the clitoris with or without accompanying pain syndromes.[58]

Etiology is multifactorial, including metabolic (diabetes), traumatic (tight clothing, violent stimulation), and idiopathic. Pain is always reported with intercourse in response to engorgement of the clitoris and compression of the pudendal nerve, or accompanying tight clothing, exercise, or increased stress levels. It is often exacerbated with sitting. A cotton swab test differentiates between clitoral pain, dysesthetic neuralgia, and vulvar vestibulitis. The test is performed by gently stroking the cotton swab over the clitoris and over the vulva. In the case of clitoral pain, the cotton swab may elicit pain. Conversely, a patient may describe a decreased sensation in the case of dysesthetic neuralgia either over the clitoris or over the vulva. Treatment for clitoral pain may consist of membrane stabilizing medications, such as pregabalin, amitriptyline, nerve blocks, and counseling. Nerve blocks of the pudendal and or dorsal clitoral nerve can be performed.[59,60] In addition, pulsed radiofrequency (PRF) or cryoneurolysis of the aforementioned nerve is possible.[49,61] Lastly, if the nerve blocks or neurolytic techniques fail, sacral neuromodulation may provide relief. Sacral neuromodulation has been used for pudendal neuralgia with success, and although there are no reports of its use for clitoral pain, in theory, it may provide pain relief.[62] The clinician may assist by providing advice regarding loose clothing, exercise modification, use of cold or warm packs, biofeedback, and relaxation training.[63]

Pelvic Pain Originating from the Urologic System

Injury to the muscular, neural, or fascial structures of the pelvic floor, loss of urethral mucosal vascular supply, diseases of the urogynecologic system, as well as age-related changes in the urethral striated muscle complex can lead to functional changes in pelvic floor mechanisms.[64,65] Each of the factors that can impact pelvic floor function should be evaluated and treatment prescribed to improve or correct as many factors as possible.

A common painful condition of the urinary system is IC. This condition affects bladder wall function, presenting with bladder pain along with increased urinary frequency, urgency, and nocturia without infection.[66] The pain from IC is commonly referred to the suprapubic area. However, pain can be referred to the low back, buttock, and perineal areas, where it can wax and wane in such a way that it is often mistaken for urinary tract infection.[67–69] In chronic cases, there can be tension myalgia of the pelvic floor muscles (PFM). Fifty-one percent of patients with IC reported dyspareunia.[70] Frequently, this combination of factors and lack of standard criteria can confound the diagnostic process.[70,71]

Diagnosis of the IC is made based on visual observation of Hunner's ulcers in the bladder mucosa and a positive potassium chloride (KCl) sensitivity test.[70] Use of the Interstitial Cystitis Symptom Index and Interstitial Cystitis Problem Index has been validated in patients diagnosed with the condition.[72] In addition, these can be used as screening tools in patients with undiagnosed CPP.[70] Definitive diagnosis is confirmed through a KCl sensitivity test. Parsons et al. have reported a 100% sensitivity with the test in IC as compared to other pelvic pain diagnoses. Comorbidities for IC include urinary tract infection, endometriosis, fibromyalgia, vulvodynia, CPP, IBS, anxiety disorder, and depression.[73]

Treatment for IC, similar to many of the pelvic pain disorders, should incorporate a multimodal approach. Treatment should include, but not be limited to, dietary restrictions oral agents, behavioral modification, intravesicular therapy, biofeedback, physical therapy, and interventional pain techniques.[74,75] The first line of treatment begins with oral agents, which can begin with nonsteroidal anti-inflammatory drugs (NSAIDs), opioids, penton polysulfate sodium (PPS), amitriptyline, hydroxizine, and gabapentin.[76–78] Unfortunately, NSAIDs are usually not very effective, and practitioners often turn to opioids, which are often equally ineffective. A PPS agent is the only FDA-approved oral agent for IC. It is a semisynthetic sulfonated glycose aminoglycan (GAG) that has properties similar to the naturally occurring GAG layer that protects the urothelium. Such PPS agents can be used alone or in combination with TCAs. A study by Teichman[79] showed that combination therapy of PPS with TCAs produced a better response in patients. Intravesicular therapy with dimethyl sulfoxide (DMSO) alone or combined with heparin has demonstrated an efficacy of 50% to 90%.[80] Recently, a study published by Kuo and Chandler[81] reported that intravesical injections of botulinum toxin A, in combination with hydrodistention, produced significantly better clinical results than hydrodistention alone in patients with IC. When all other therapies fail, neuromodulation techniques are employed. There are numerous reports on sacral nerve root stimulation in patients with refractory IC, which reported that 73% of the subjects experienced at least a 50% improvement in symptoms and a significant

improvement in quality of life.[82] Feler et al.[83] reported significant pain reduction in patients with IC who had failed aggressive treatment and were facing cystectomy for intractable bladder pain. In a more recent study, Zabihi et al.[84] found that sacral neuromodulation for IC patients had significant improvement in pelvic pain, as well as voiding symptoms.

A second urologic condition that can lead to development of CPP is urethral syndrome, which is a noninfectious conditioning presented as midline suprapubic or urethral pain and functional disturbance of dysuria without nocturia. [70] This condition is caused by stenotic or fibrous changes of the urethra from infections, trauma, or atrophy. Newer terminology considers this as a type of painful bladder syndrome that has a similar presentation to IC without the bladder ulcers.[85] Appropriate management of pelvic pain origination in the urological system involves treating each of the previously described conditions. Clinicians are encouraged to implement appropriate pain management strategies (to be further discussed).

Pelvic Pain Originating from the Gastrointestinal System

Pelvic pain is multifactorial and can be caused by pathologies in the gastrointestinal system. While up to 50% of patient visits to the gastroenterologist are for diagnosis and management of IBS, this condition is associated with dysmenorrhea in 60% of cases.[86] Other bowel conditions can contribute to pelvic pain including diverticular disease, Chron's disease, ulcerative colitis, and chronic appendicitis, which are typically well managed by the primary care provider. Functional gastrointestinal disorder is a pain syndrome with a poorly defined pathology of which IBS is one type.[87,88] The development of the Rome criteria to aid diagnosis and treatment of functional gastrointestinal conditions has been ongoing since 1989 with the most recent version, Rome III, published in 2006.[89]

Functional gastrointestinal disorder presents as abdominal pain that is exacerbated by ingesting food and/or engaging in a bowel function. IBS presents with symptoms of abdominal pain, intestinal gas, bloating, constipation, and diarrhea. This encompasses a broad category of possible pathologies and comorbidities that result from alterations in visceral sensitivity, central processing, autonomic and enteric nervous system alterations, and gut motility.[90] IBS is best described by a predominant bowel pattern of constipation, diarrhea, or both (mixed pattern). Using an intake assessment that describes the pain characteristics and relations to food and bowel function can aid in identifying an association and guide the patient and practitioner in developing a conservative treatment plan. Once identified, referral to a gastroenterological specialist is merited.

Pelvic Pain originating from the Musculoskeletal System

Musculoskeletal pathologies that can cause pelvic pain include sacroiliac dysfunction, symphysis pubis and sacrococcygeal joint dysfunctions, coccyx injury or malposition, and neuropathic structures in the lower thoracic, lumbar, and sacral plexi. While the thoracic and lumbar spines, as well as hip dysfunction, can produce comorbid conditions or become involved because of the associated activity and movement changes, these should be considered in the differential diagnosis of pelvic pain.[91,92] The assessment and treatment of these areas has been well described elsewhere and will not be further described in this article.[93–97]

Pelvic ring hypermobility affects between 7% and 14% of all pregnant women.[21] Prolonged pelvic girdle pain, lasting beyond 6 months postpartum, is estimated in 3% to 30% of women.[98] Such dysfunction most often results in pain that localizes to the posterior superior iliac spine and pubic symphysis.[21] There are many theories to the cause of pelvic girdle pain, but the diagnosis remains elusive. As there is no gold standard test for identifying pelvic girdle pain, the following tests must be considered: (1) active straight leg raise (ASLR) according to Mens et al. 1999; (2) posterior pelvic pain provocation test (or thigh thrust described later); (3) positive pain with palpation of the long dorsal sacroiliac ligament; and (4,5) resisted hip abduction and resisted hip adduction tests.[99] A greater number of positive findings indicate greater severity of pelvic girdle pain, and negative findings on these tests suggest that the condition is not present. Mechanical dysfunction of the sacroiliac or symphysis pubis joint and/or pelvic ring has been proposed as a mechanism of this prolonged pain.[22,23,99] However, motor control dysfunction has also been implicated and should be assessed in all patients presenting with prolonged pelvic girdle pain.[100]

As a component of pelvic ring pain, SIJ dysfunction is commonly seen in peripartum pain conditions. The diagnosis of sacroiliac joint dysfunction is best accomplished through the use of clinical provocation tests,

including the (1) dorsolateral pelvic spring tests over the anterior superior iliac spines (ASISs); (2) anteromedial iliac spring over the anterolateral ASISs; (3) thigh thrust (loading the femur through its long axis with the knee fully flexed and the hip positioned at 90 degrees flexion; and (4) ventral sacral thrust provocation tests with the patient in prone.[101] These provocation tests demonstrated increased validity when a cluster of 2 or 3 tests were positive during a given patient examination.[102] In concert with positive provocation, pelvic ring hypermobility can be diagnosed with the ASLR.[99] Mens[103] described position change at the pubic symphysis, as well as pain in the SIJ area with the ASLR, which has been validated for measuring disease severity of the pelvic ring structures in prolonged pelvic pain of pregnancy. While this test does not serve as a pain provocation test, it measures the capacity for load transfer through the pelvic ring. Use of the provocation tests and ASLR is suggested for optimizing differential diagnosis of pelvic girdle pain both with the peripartum patient as well as the patient with pelvic girdle pain unrelated to pregnancy.[93] Management of sacroiliac and pelvic ring disorders has been previously described. Clinicians are encouraged to implement a comprehensive program that may include high velocity thrust manipulation, trunk stabilization, and restoration of motor control (later described), as well as habit changes.

Pain management techniques for SIJ pathology include local and steroid injections, radiofrequency thermocoagulation (RFTC), PRF, cooled radiofrequency, and cryoneurolysis of the nerves innervating the SIJ under fluoroscopic guidance. The local steroid injection may produce results lasting anywhere from 2 weeks up to 12 months. A study carried out by Fischer et al.[104] showed that 87% of patients had a statistically significant decrease in pain with improvement as early as 1.5 weeks and lasting for a mean of 12 months. Conversely, RFTC, PRF, cooled radiofrequency, or cryoneurolysis may last from months up to years.[49,105–107]

Coccygeal joint dysfunction is an uncommon condition that causes pain in and around the coccyx because of a local trauma or overload and is exacerbated by sitting and bending. It is most commonly seen in women as a result of the morphology of the female coccyx and the propensity for hypermobility.[108] The pain is related to dysfunction in the coccygeal joints or disks in the majority of patients.[109] The pathology may be the result of a hypermobility or fracture leading to pain with sitting and transition from sitting to standing. Maigne[110] suggested mobility > 25 degrees of flexion of the long axis of the coccyx in relation to the sacrum as pathological. Maigne et al.[111] has described a method for determining the amount of coccygeal mobility using lateral sitting versus standing roentgenograms. A history of trauma, obesity, and transient exacerbation of pain when standing up from sitting are common features of coccydynia. Coccygeal fracture and dislocation may result in introital dyspareunia and pelvic floor tension myalgia that potentially can become chronic if not properly treated.[112]

Coccygeal pain may be related to neurologic causes. The ganglion impar is located anterior to the sacrococcygeal joint and transmits sympathetic stimuli. Hyperactivity of sympathetic system as a result of visceral pathology may refer pain to this region. Additionally, dural irritation from a lumbar disk has been postulated as a source of coccygeal pain.[108] Computed axial tomography performed by Wray et al.[113] showed the presence of herniated lumbar disk in a portion of patients with coccygeal pain. Whether this was an incidental finding or significant is uncertain but may be a form of double-crush phenomenon. It would be important for the clinician to question the patient regarding history of low back pain prior to onset of coccydynia.

Other conditions that may cause neuropathic coccygeal pain may be schwannomas of sacral nerve roots, neurinomas, arachnoid cysts in cauda equina, and sacrococcygeal meningeal cysts.[108] Space-occupying lesions have been implicated in mixed component pain.[108] The lesion initially affects bony and ligamentous structures causing somatic pain. As the lesion enlarges, neural or visceral structures are affected, leading to neuropathic pain or visceral pain. In these cases, the resulting somatic pain may occur in response to viscerosomatic convergence.

Other than trauma, obesity and lumbosacral disk herniation are risk factors in the development of coccygeal pain.[108] Etiologies relate to muscle spasms of the pelvic floor, arthritis, osteitis, referred pain from lumbar pathology, arachnoiditis of the lower sacral nerve roots, somatization, and idiopathic causes.[113] A high incidence of pathology is seen in debilitated elderly patients.[108] Several reports have been published describing the procurement of a coccygeal fracture during childbirth delivery.[112] Maigne differentiated coccyx pain as arising from spicules (bone spurs), anterior hypermobility (> 25 degree flexion angle of the coccyx comparing sit and stand radiographs), and

subluxation (altered patent of movement, which typically consists of displacement into extension on stand to sit x-rays).[110,114]

Anal pain syndromes have been seen in conjunction with coccyx pain. In this case, diagnosis is made based on the lack of provocation to coccyx manipulation.[108] Attention to the local neural structures is important, as the viscerosomatic involvement of the local visceral nerves from infection or space-occupying lesions in the pelvic organs can lead to sensitization of these structures.

Additionally, coccydynia can be due to muscular hypertonicity of the pelvic floor. In such instances, longitudinal stretching and contract-relax maneuvers applied to the levator ani have been advocated.[109] This is very similar to the press and stretch method used for tender point relaxation.[115] In the presence of an unstable or hypermobile coccyx, manual stretching or manipulation is less effective than local injection.[114]

De Andres[108] proposed an algorithm for coccydynia treatment based on anatomic review. Conservative management includes physical therapy, high velocity thrust manipulation of the sacrococcygeal joint, psychological treatment, and intra-articular injections. Maigne[116] studied the effects of stretching and mobilization of the coccyx and reported that stretching of the levator ani is more successful than mobilization of the coccyx. The use of a donut ring for sitting is helpful only when fibers of the gluteus maximus inserting on the coccyx are not involved. With involvement of these fibers, sitting on a ring may create more traction on the coccyx.[108] When the previous strategies are not effective, coccygectomy is suggested, although Hodges[117] reported a high rate of infection with the surgery. Balain[118] suggested coccygectomy for those patients with degenerative changes of the sacrococcygeal joint. They reported that 83% of patients with degenerative changes recovered well after surgery.

Interventional pain management of coccydynia consists of various methods for blocking the sympathetic or visceral component of pain that travels via the ganglion of Walther. Treatments include injections with local and steroids, RFTC, PRF, cryoneurolysis, and sacral neuromodulation.[61,62,119–122] Those patients who fail these conservative modalities and interventional pain procedures may potentially benefit from coccygectomy.[123] There have evolved various techniques at blocking the ganglion impar, including access via the anococcygeal ligament, sacrococcygeal joint, intracoccygeal joint, and most recently a paracoccygeal approach.[119,124,125] Results with the aforementioned procedures have varied, but overall, there has been good short-term success in treating coccydynia.

Proctalgia Fugax

Proctalgia fugax is described as a sudden cramping rectal pain that is usually present at night. It disappears within several minutes with no objective findings, and its etiology is unclear.[126] Mazza et al. propose that proctalgia fugax may be variant of irritable bowel. Pelvic floor tension myalgia has also been proposed as a source of pain.[127] Takano[128] postulates that it may be a result of pudendal neuralgia. Takano observed pudendal nerve tenderness to palpation in 55 of 68 subjects suffering from this condition. After administrating a nerve block, symptoms completely disappeared in 65% and decreased in 25%. The etiology of this condition may be related to an internal anal sphincter spasm or thickening. Garcia Solanas[129] successfully used sphincterectomy for treating thickened internal anal sphincter demonstrated on ultrasound. Physical therapy may be helpful in applying Thiele's massage or stretching of the sphincter and levator ani.[114]

Several treatment options are available for proctalgia fugax, many with limited success. Typically, treatment begins with hipbaths and a topical nitroglycerin ointment.[130,131] During episodic attacks, oral medications, such as calcium channel blockers (eg, nifedipine) and clonidine, have been incorporated. Inhaled medications, such as salbutamol, have been shown to shorten duration of attacks in isolated cases.[132,133] Intravenous (IV) lidocaine has been used for treatment of proctalgia fugax.[134] Peleg et al. reported that a single dose of and IV lidocaine infusion completely stopped a patient's pain attacks. For refractory cases, digital rectal dilation and internal lateral sphincterectomy have been shown with marginal success when internal anal sphincter hypertrophy is noted during the workup.[135] Interventional pain techniques have shown that 90% of patient's pain syndromes improved with complete resolution of pain in 25% of patients after receiving nerve blocks of the pudendal nerve with LA and steroids.[128] Multiple studies using botulinum A toxin have demonstrated very promising results for proctalgia fugax.[136,137] A preliminary study showed a significant effect in patients afflicted with proctalgia fugax.[137] The investigators found that after 1 injection, 80% of patients remained symptom free, with 1

patient requiring a second injection. All patients in the study remained pain free up to finishing the follow-up of 2 years. Sacral neuromodulation can serve as a treatment option for proctalgia fugax. Recently, Falletto et al. found that sacral nerve stimulation was effective in treating chronic anal pain. Twelve patients diagnosed with chronic anal pain underwent sacral nerve stimulation and were pain free up to a mean of 15 months. The investigators discovered that all but 1 patient had a significant improvement in visual analog and SF-36 scores. The long-term follow-up showed a significant improvement in quality of life, and the investigators concluded that sacral neuromodulation should be considered in patients with chronic anal pain when pharmacologic biofeedback treatments have failed.[62]

Pelvic Pain Originating from the Neurologic System

Nerve irritation or entrapment as a cause of pelvic pain can be related to injury of the upper lumbar segments giving rise to irritation of the sensory nerves to the ventral trunk, or from direct trauma from abdominal incisions or retractors used during abdominal surgical procedures.[138] Location of pain and paresthesias should indicate the nerve structure most likely to be involved. Afflictions of the iliohypogastric, Ilioinguinal, genitofemoral, pudendal, and obturator nerves are of greatest concern in patients with pelvic pain. The goal of nonoperative management for nerve entrapment is to restore an optimal environment to allow the neural tissue time for healing and recovery.[139] Treatment can include local tissue massage to relieve myofascial tightness along the course of the nerve, movements of the nerve to enhance the neurobiological processing, and treatment of the autonomic nervous system via sensory input to the corresponding thoracic segments.[94,139,140]

Injury to the Iliohypogastric nerve can cause pain in the lateral pelvic or suprapubic area and/or abdominal weakness as it supplies motor innervation to some of the abdominal wall muscles.[138] Palpation for paresthesias in the inguinal area, along with clinical findings of lower abdominal pain and pain radiating into the labia, suggests involvement of ilioinguinal and genitofemoral nerve (previously described in Part 1 of this series), especially in the absence of other pathologies. The iliohypogastric, ilioinguinal, and genitofemoral nerves can be tested using the femoral nerve stretch position (Figure 1).

A position of ipsilateral hip neutral flexion with contralateral trunk side bending can be used for mobilization of the previously mentioned nerves (Figure 2).[94,139] The purpose of neural mobilization is to gently move the nerve along its path to stimulate increased cellular transport and minimize local edema. The neural movement should not provoke the patients' familiar pain. Neural mobilization is performed in a rhythmic manner for 1 to several minutes and can be carried out several times each day. Treatment for iliohypogastric nerve irritation can include localized injection or nerve block and surgical nerve excision if nonresponsive to more conservative measures.[138] Injection can be delivered to the area of exit through the abdominal wall or as a foraminal block at L1 to L2 if the local block is unsuccessful and the surgical excision is not possible.

Treatment for the ilioinguinal and genitofemoral nerves is similar to that for the iliohypogastric nerve with the injection targeted to the nerve exits though the abdominal wall. McCrory suggests that the first intervention should be block of the ilioinguinal nerve

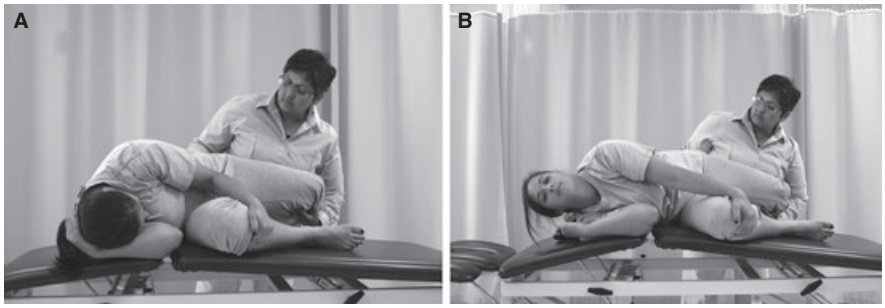

Figure 1. Iliohypogastric, Ilioinguinal and Genitofemoral nerve tension testing for the left side in sidely. The patient is prepositioned with trunk flexed forward and contralaterally sidebent in order to place the neural tissue under tension and to produce pain provocation: The maneuver is performed with (A) tension position; (B) release of tension.

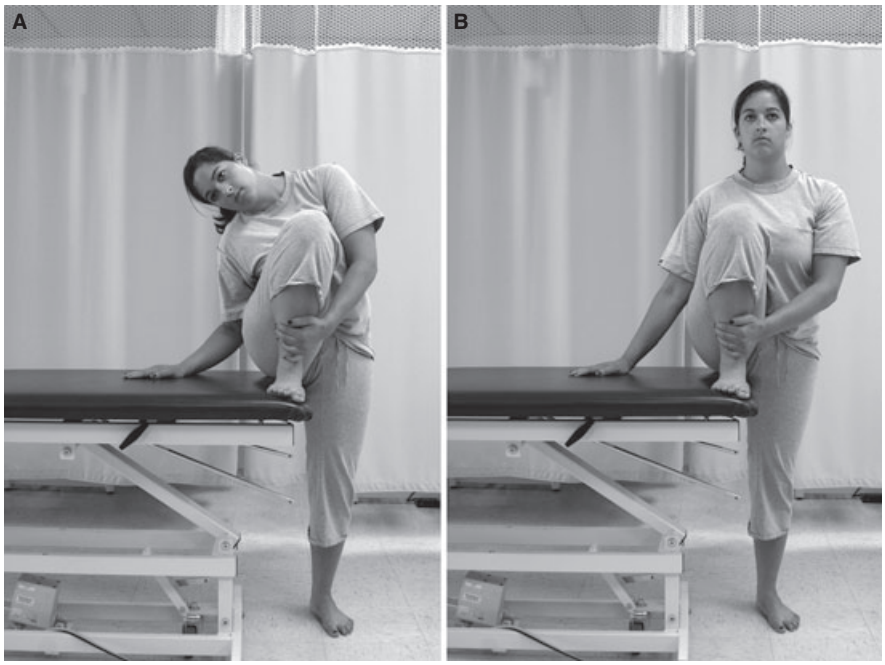

Figure 2. Iliohypogastric, Ilioinguinal and Genitofemoral neural mobilization for the left side in sitting. The patient's hip is prepositioned in neutral and adduction. The maneuver is performed with trunk movement into contralateral sidebending: (A) start position; (B) mobilizing movement into sidebending.

medial to the anterior superior iliac spine. If unsuccessful, then blocking the L1 to L2 roots in the foramen can be attempted. The genitofemoral nerve is difficult to locate in the abdominal area and if thought to be part of the pathology would respond best to a local block at the L1 to L2 foramen.[138] Finally, surgical excision of the genitofemoral and ilioinguinal nerves can be performed if the local blocks are unsuccessful.

The obturator nerve can be involved in chronic pain conditions of the pelvis and lower extremities, potentially accompanied by weakness of the adductors and sensory changes to the medial thigh and knee joint. Because of the course through the pelvis and through the fibrosseous tunnel, the obturator nerve is vulnerable to injury from pregnancy, pelvic masses, visceral surgery, orthopedic injuries, abdominal hernias, and fascial bands narrowing the obturator tunnel, resulting in pain and dysfunction.[138]

Obturator nerve tension testing can be performed (Figure 3). In addition, the knee can be extended for greater obturator nerve tension in those patients with involvement at the obturator canal and medial thigh (Figure 3). The nerve can be therapeutically mobilized (Figure 4). If there is involvement of the thigh, an alternative mobilization can be performed in standing. The patient stands in a stride stance with ipsilateral side bending (Figure 5). The frequency and duration of neural mobilization amount to 5 minutes of movement performed in one of several sessions. The key to success centers on the clinician not exacerbating the symptoms during the neural mobilization. Additional treatment can include local injection to the obturator nerve as it exits the pelvis. In severe cases, the obturator tunnel is surgically released.[138]

The pudendal nerve can contribute to perineal pelvic pain as it supplies the sensory and motor innervation via the nerve to the levator ani, the perineal branch, and the dorsal nerve to the clitoris. Because of the course of the pudendal nerve (previously described in Part I of this series), it can be entrapped or damaged during procedures to correct anal fissures or fistulas, delivery, sustained bicycle riding, and from hypertrophy of the sacrospinous and sacrotuberous ligaments.[141] There are no provocation tests associated with the pudendal nerve; thus, diagnosis must be made from clinical presentation and a process of exclusion.

The typical presentation of pudendal neuralgia is of pain or burning sensation in the perineal area with sitting that improves with standing. The symptoms may be elicited during activities, such as bicycling with an excessively narrow saddle pressing against the pudendal nerves within the pudendal canal. Pudendal nerve

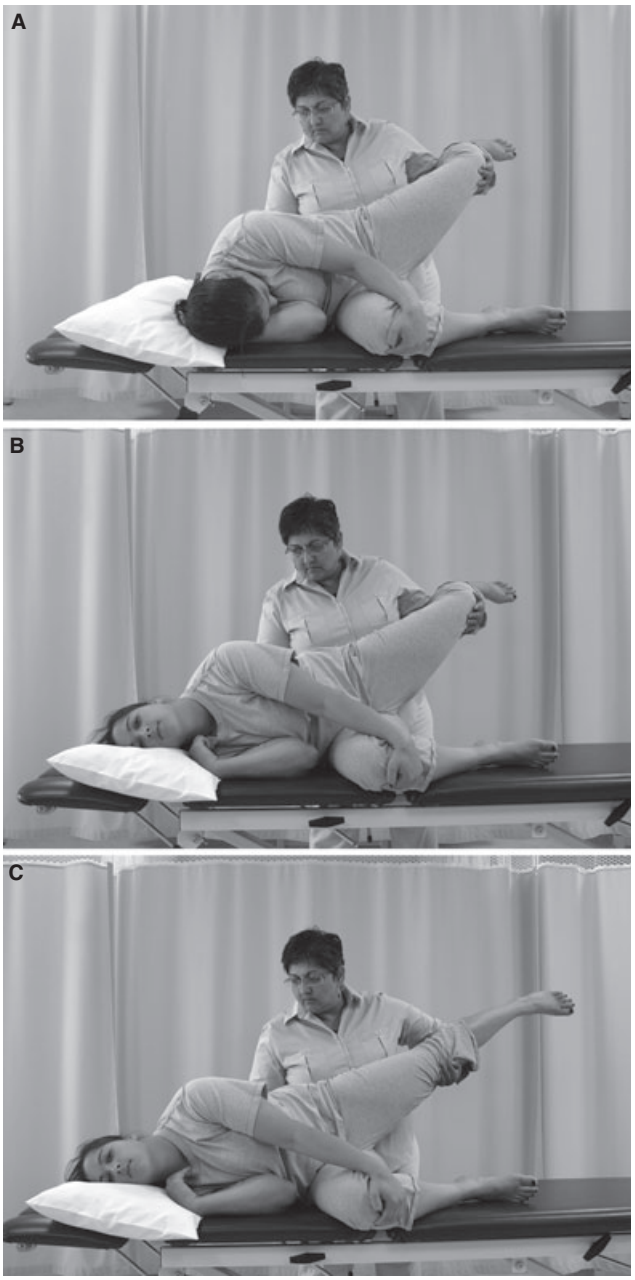

Figure 3. Obturator nerve tension testing for the left side in sidelying. The patient is prepositioned using a contralateral sidebent trunk position with ipsilateral hip extension and abduction for maximal neural tension: The maneuver is performed with: (A) tension position; (B) release of tension; (C) optional position with knee extension to further tension load to the adductors.

entrapment often can be helped by nonoperative measures that include relaxation of the PFM and implementing strategies to minimize compression to this nerve. Use of a sacral sitting pad with a perineal cutout can ease discomfort with sitting. Avoiding deep squatting, as well as sustained sitting in greater hip-flexed postures, can minimize irritation from the sacrospinous ligament and the pudendal nerve along its course.[142] Because of its location, neural mobilization initiated by the patient is not feasible for this nerve. Local injection can aid in pain management.[142]

Interventional pain management strategies for pain involving the iliohypogastric, ilioinguinal and genitofemoral, pudendal, and obturator nerves are very similar and play an important role in their diagnosis and treatment. Treatments of the involved nerves can be performed in injections of local and steroids, RFTC, PRF, cryoneurolysis, and neuromodulation.[143–148] There have been multiple studies using RFTC as well as PRF with very promising results. Malik et al. performed percutaneous radiofrequency lesioning of the sensory branches of the obturator and femoral nerves in 4 patients. All 4 patients had reduction in pain, while 3 of 4 patients had improved functioning. Two of the 4 patients exhibited decreased use of their pain medication. One of the 4 patients reported numbness at the hip, while there were no other side effects.[149] Although thermal coagulation was successful, clinicians must be advised that it carries the potential risk of neuritis. Unlike RFTC, the risk of neuritis is minimized with PFR because no nerve damage occurs. Wu et al. reported 2 cases of patients with groin pain who underwent PRF on the articular branches of the obturator, femoral, superior gluteal, and sciatic nerves. Both patients demonstrated at least 50% reduction in pain and lasted 3–4 months after the intervention along with improved physical function.[145] With respect to neuromodulation, several different approaches have been successfully employed. Spinal cord stimulation (SCS), peripheral nerve stimulation (PNS), and peripheral field stimulation have been used all with good results.[127,150,151] Rauchwerger reported 3 cases of inguinal neuralgia that were successfully treated with PNS.

Goroszeniuk et al. presented 3 case reports of patients with chronic intractable pain in which subcutaneous neuromodulation was successfully introduced. All 3 patients experienced significant pain relief, improved quality of life, and a cessation of narcotic use. Those patients who fail conservative modalities and interventional pain procedures may benefit from surgical management.[152,153] A recent study by Loos et al. demonstrated that peripheral nerve blocks provide long-term pain reduction in selected individuals. For patients that nerve blocks do not work, an

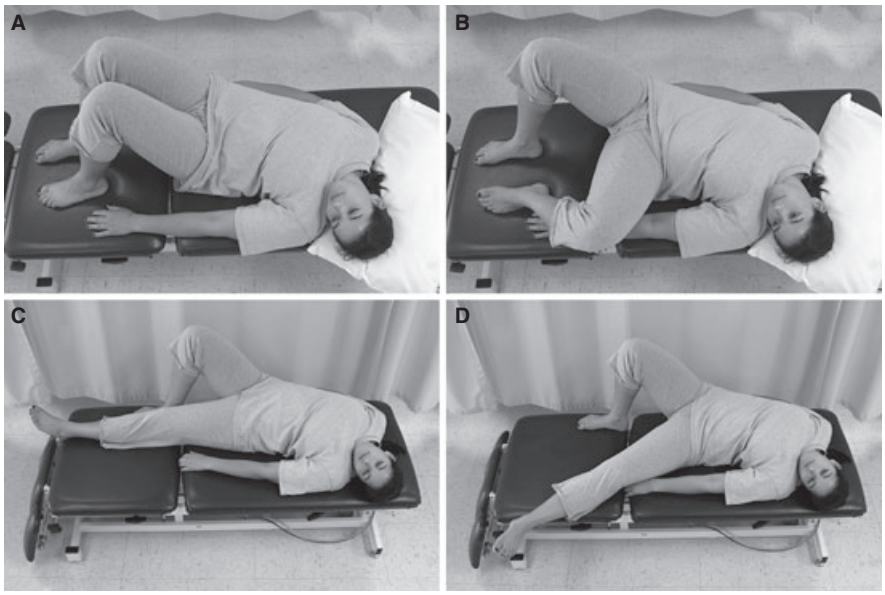

Figure 4. Obturator nerve mobilization for the left side in supine. To mobilize the nerve, the trunk can be positioned into a sidebent position to the side of mobilization in a hook lying position. The thigh is moved into abduction and external rotation to mobilize the nerve within the pelvis: (A) hooklying with ipsilateral sidebend; (B) movement into hip Abduction/External rotation; Alternatively, the lower extremity can be prepositioned with knee and hip in extension (C) alternate start position; (D) alternate mobilization into hip abduction (end position).

iliohypogastric or ilioinguinal nerve neurectomy is a safe and effective procedure.[154]

Pelvic Pain as a Secondary Disease of the Pain System

Chronic pain that presents without identifiable pathology can be considered a secondary disease process because of its identifiable features and its own pathology, symptoms, and signs.[155] Integration of the somatic and afferent input in the central nervous system leads to convergence, activation of silent nociceptors, sensitization of neurons in the peripheral nervous system and dorsal horn, along with alteration of central signal processing that produces an environment where ongoing pain perception is noted. Viscero-visceral activation, as well as viscero-muscular reflex activity, can be seen as features of this sensitization whereby the chronic pain becomes the secondary pathology.[155]

Patients with CPP can present with pain in any part of the pelvis. This pain is characterized by: (1) symptoms that are difficult to isolate; (2) symptoms that are not affected by specific movements or positions; and (3) significant patient distress. Cognitive processes that negatively impact coping, enhance anxiety and depression, and limit opportunities for recovery are known to coexist with chronic pain states. However, no causal link has been identified between those processes and pain.[155,156] In addition, the affective state behaviors employed by the patient can further limit recovery. Only when a biopyschosocial approach to pain management is utilized, will the patient experience full functional recovery.[156,157] This translates into the need for identification of both the primary neuro-musculoskeletal pathology and secondary pain system status, as well as the contributing psychosocial factors, for the management of patients with this condition.[155]

ADDITIONAL NONOPERATIVE MANAGEMENT CONSIDERATIONS

Education: Contractual relationships with the Patient

Any management plan for patients with pelvic pain requires the patient's consent in compliance with treatment. This is especially necessary for the patient with chronic pain in whom interventions often involve behavior modification, cognitive focus change, and mechanical treatments. Preparing the patient for the planned intervention requires education of the patient on the goals of treatment and negotiation on the best strategies to comply with. Such preparation of the patient forms the basis of the therapeutic "contract" or relationship. For patients with greater involvement

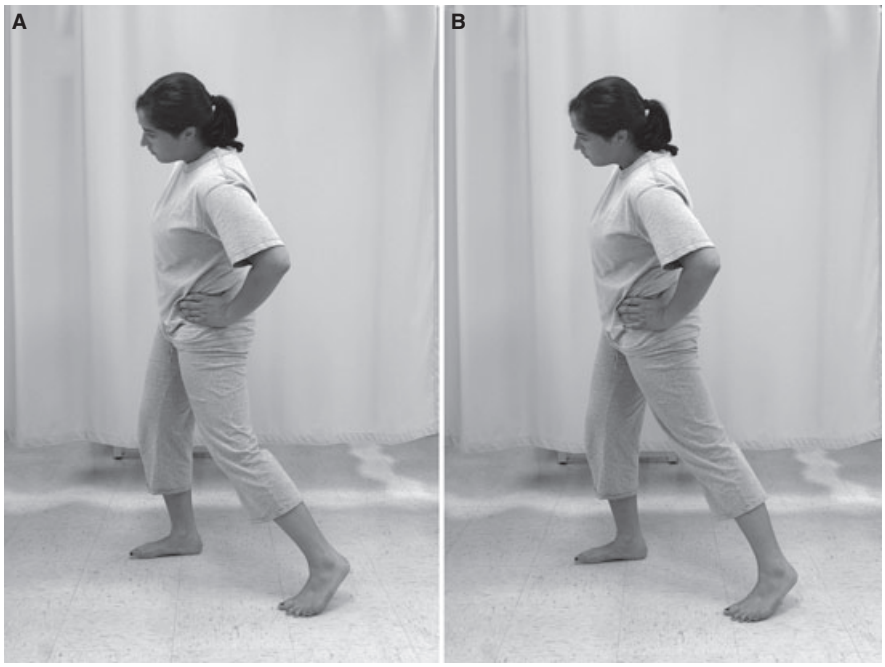

Figure 5. Alternative obturator nerve flossing: the thigh moves the hip into extension, or abduction in a rhythmic fashion (A) preposition in stride with ipsilateral sidebend and knee flexed; (B) movement into hip and knee extension.

of the cognitive and affective systems, this preparation is a critical component to success. [158]

Educational components for CPP management include informing the patient about their pain mechanisms, mechanical pathologies, and the interaction of the visceral system with the musculoskeletal system. The goal of educating the patient on chronic pain as a disease entity is to promote the progression from a search for a ''cure'' of the mechanical or visceral pathology to the process of active coping and development of self-efficacy.[157] The ability of the patient to enhance self-efficacy, to learn positive coping strategies, and to develop thought patterns and movement strategies to enhance recovery of function will become self-sustaining. While the effectiveness of diagnosis-specific pelvic pain management has not been investigated in the peer-reviewed literature, there is sufficient information to support nonoperative management that includes a biopsychosocial approach, which incorporates cognitive behavioral treatment with a multidisciplinary approach.[159]

Reactivation Strategies

Physical strategies to enhance self-efficacy and enhance coping with chronic pain can be selected based on the needs of the patient. These strategies should include pain management techniques, condition-specific local treatment, and movement strategies that incorporate re-establishment of motor control and postural awareness. The clinician may include treatments from one or all categories, depending on the patient's needs and his or her cognitive, affective, and psychosocial status.

Pain management strategies include stress management, sleep hygiene, and relaxation techniques.[159] Specific mechanical strategies to aid pain management include physical agents and manual therapy for joint and soft-tissue structures. Movement control strategies require the skilled training of a clinician to identify abnormal movement patterns that contribute to the patients' disability or dysfunction and to implement training regiment to resolve altered movement control, restore function, and improve muscular strength. A well-designed treatment incorporates all aspects and respects the patients' psychosocial status and readiness for change.

INTERVENTIONAL PAIN MANAGEMENT STRATEGIES

Even though CPP is poorly understood as often undertreated, many treatment strategies have been proposed with some success. Treatment interventions comprising

conservative management including behavioral, physical therapy, pharmacological, surgical, and interventional pain management have been devised. The following section will specifically discuss in detail interventional pain strategies used by pain management specialists.

Diagnostic and Therapeutic Blocks

Given the challenge and complexity of diagnosing CPP, diagnostic blocks are a useful tool in identifying the source of pain. Because many causes of CPP are not readily apparent, diagnostic blocks provide important clinical information when history, physical examination, and imaging techniques cannot elucidate the pain source. Precise diagnostic blocks with LA can clearly delineate the pain source by blocking the site of nociception and the afferent pathway. Thus, the rationale behind diagnostic blocks is to elucidate complicated clinical situations where diagnosis is not clear and determine the source of pain. Once the diagnosis has been established, a therapeutic block may be performed with LA alone in combination with steroids prior to performing more advanced procedures. The choice of LA and steroid varies among practitioners. In our clinical practice, we use 1% to 2% lidocaine or 0.2% ropivicaine for diagnostic blocks with small amounts of LA depending on the site being blocked. For therapeutic blocks, we will use 0.2% ropivicaine and 40 mg of methylprednisolone with the appropriate volume of LA. Diagnostic and therapeutic blocks can be used for a variety of CPP states. Its uses have been documented in vulvodynia, clitoral pain, coccydynia, iliohypogastric, ilioinguinal, genitofemoral, pudendal and obturator nerves, endometriosis, SIJ disorders, and proctalgia fugax.[18,47,48,59,60,104,119,128]

Neuroablative Techniques

Destruction nerve techniques have been employed by surgeons for numerous years with good success. Most techniques were either neurosurgical of chemical neurolysis for the treatment of cancer pain. With more patients developing chronic pain, techniques have emerged with the idea that nerve destruction promotes analgesia. Presently, interventional pain strategies use various techniques, such as RFTC, PRF, cooled radiofrequency, cryoneurolysis, and botulinum toxin for the treatment of CPP.

Radiofrequency Thermocoagulation

Radiofrequency thermocoagulation is application of a continuous electrical current to promote thermocoagulation and eventually nerve destruction. The mechanism of RFTC works by generating heat around the active electrode tip located near the nerve. Heat is generated in the surrounding tissues, not the probe itself, and causes lesioning of the nerve. Nerve destruction begins once the temperature exceeds 45°C. In our clinical practice, we typically perform 2 cycles of 90-second duration to a temperature range from 60°C to a maximum of 80°C. There have been several studies showing good results with RFTC in certain CPP states. Several authors have demonstrated good pain relief with RFTC for SIJ disorder, coccydynia, iliohypogastric, ilioinguinal, genitofemoral, and pudendal neuralgia.[105,120,143] However, we must caution there is a potential risk of postprocedure neuritis associated with RFTC secondary to neuroma formation.

Pulsed Radiofrequency

Pulsed radiofrequency is a neuroablative technique that provides analgesia without destruction of neural tissue. PRF delivers intermittent pulses of current at temperatures typically not exceeding 42°C. The exact mechanism of PRF has not been elucidated. However, while several theories exist, it is known that the intermittent current delivery creates and electromagnetic field that leads to a phenomenon of altering pain signals. This is also known as a neuromodulatory effect on the nerves. The intermittent pulses allow heat to dissipate, thus preserving the nerve from destruction. Normally, the procedure is preformed at a frequency of 2 Hz, pulse width of 20 milliseconds, and duration of 120 seconds at 42°C.[160] PRF is a technique that is felt to be safer than conventional RFTC.[161] A recent study by Tun et al. showed that PRF treatment may lead to separation in myelinated axons. However, all changes in the PRF group compared to the continuous radiofrequency (CRF) group were reversible. This study supports the hypothesis that pulsed RF treatment does not rely on thermal injury of neurologic tissue to achieve its effect,[162] thus making PRF lesioning attractive as a nondestructive method for analgesia while preserving the nerves and preventing neuroma formation and neuritis. Its use has been described for several CPP states. Its use has been applied to meralgia

paresthetica, ilioinguinal neuralgia, pudendal neuralgia, and SIJ disorders.[61,106,146]

Cooled Radiofrequency

Cooled RF is a new neuroablative technique that has recently emerged within the last 5 years in the pain management arena. This technique is similar to RFTC in many ways except for the cooling factor and maximum temperature achieved. Once in correct position, the electrode tips are cooled and are not allowed to exceed a certain set temperature. In a study carried out by Kapural et al., the electrode tips were placed in a suitable location, and the heating protocol was initiated delivering CRF current for 2 minutes and 30 seconds at a maximum temperature of 60°C. This was continued with multiple lesions created 1 cm apart to create strip type of lesion over the nerve. According to Kapural, these continuous lesions provide a greater lesion size as compared to conventional RFTC. Kapural et al. based this theory on work by Lorentzen, Goldberg, and Watanabe where they demonstrated larger lesions compared with noncooled lesions postulated by removal of heat from adjacent tissue, thus allowing greater power delivery to be increased without increasing impedance and tissue charring. [163–165] Kapural went on to perform a small retrospective study with 27 patients involving SIJ pain whereby cooled RF was applied to the sacral lateral branches and L5 dorsal primary rami. According to Kapural,[107] the majority of patients with chronic SI joint pain experienced a clinically relevant degree of pain relief and improved function. This technique shows promising results, and further studies are warranted.

Cryoneurolysis

The use of cryoneurolysis in managing many chronic pain syndromes is gaining acceptance. It is a useful technique when other modalities for pain relief have not worked. It is a nondestructive neuroablative technique that applies extremely cold temperatures to the peripheral nerve to induce a reversible block of condition similar to that produced by local anesthesia. The extent and duration of the effect of cryoneurolysis is a function of the degree of cold obtained and the length of time of exposure to the target nerve. Pain relief from cryoneurolysis occurs because ice crystals create vascular damage to the vasa nervorum, which produces severe endoneural edema to the nerve. This disrupts the nerve structure and creates Wallerian degeneration but leaves the myelin sheath and endoneurium intact. This process is known as second-degree axonotmesis. The Schwann cell basal lamina is spared and ultimately provides the structure for regeneration, because the endometrium remains intact, neuroma formation does not occur, and the nerve is able to regenerate slowly. [49]

The long-term effectiveness of cryoneurolysis varies and is difficult to predict, as few investigations have addressed this issue. Green et al. performed a 60 patient retrospective study for cryoneurolysis of the intercostal nerve. They found that 75% of patients had immediate relief following the procedure and 3 months later, these patients still had at least 50% pain relief.[166] In another study, Zakrzewska[167] found that the mean pain relief for cryoneurolysis was 13 months for the long buccal nerve, 17 months for the mental nerve, and 20 months for the infra-orbital nerves. In our clinical practice, we have seen pain relief anywhere from 2 weeks up to 6 months with > 50% pain relief with patients regaining normal sensation before the return of pain.[168] Cryoneurolysis has been used for vulvodynia, SIJ pain, coccydynia, iliohypogastric, ilioinguinal and genitofemoral, pudendal, and obturator nerves.[49,122,147,148]

Chemical Neurolysis

Operative and nonoperative approaches have been used as a means of blocking or inhibiting sensory pathways involved with visceral pain. Chemical neurolytic treatments are an important nonoperative approach in managing cancer pain. This form of treatments is often initiated when all other treatment options have failed, pain is severe, and life expectancy is short. Neurolytic chemical agents used are alcohol, phenol, and hypotonic saline. Even though chemical neurolysis is not often employed for treatment in chronic pain states, there has been some literature where chemical agents have been applied to nonmalignant forms of chronic pain. Additionally, there is a growing literature showing that PSN either chemical or surgically performed has good results for CPP.[169] De Leon et al. performed presacral chemical neurectomies with 10% phenol for patients with intractable pelvic pain associated with cancer. They reported a 69% success rate at 6 months.[170] Davis et al.[171] had reported performing inferior hypogastric plexus denervation via chemical ablations with alcohol for the treatment of

dysmenorrhea. In a recent study, Weskler et al. used a 4% phenol for chronic nonmalignant pain. Their study found that chemical neurolysis was effective for intercostals, greater occipital, and genitofemoral neuralgia, coccydynia, meralgia paresthetica, and SIJ disorder. Overall, patients had significant pain relief, improved activities of daily living, and decrease use in narcotics.[172] However, as Weskler mentioned, there is a potential risk of flaccid paralysis, and they recommended this technique be reserved for cases far removed from motor nerves and the spinal cord.

Botulinum Toxin

Many causes of CPP can be effectively treated by existing medical and surgical interventions; however, large majorities are either undiagnosed or ineffectively treated. Recognizing that pelvic floor muscle spasms may add to the continuing pain, symptoms may help clarify why some treatments for CPP are ineffective. Generally speaking, pain secondary to muscle spasm has been reported to occur throughout the body, and successful treatments have been developed using botulinum toxin (Botox).[173,174]

With CPP, patients often suffer from a variety of conditions such as pelvic floor muscle spasm to vulvodynia with many successfully treated with Botox. In addition, Botox not only treats these painful disorders, but it also treats associated symptoms, such as dyspareunia, dyschezia, and exacerbation with dysmenorrhea.[175] Abbott et al. performed a double-blinded, randomized, placebo-controlled trial in patients with CPP of more than 2-year duration and evidence of pelvic floor muscle spasm. They found that there was a significant decrease in pain in the botulinum toxin type A group for dyspareunia and nonmenstrual pelvic pain. Additionally, there was a significant reduction in pelvic floor pressure in the botulinum toxin type A group from baseline values.[176] Jarvis et al.[177] performed a prospective cohort study using Botox in the treatment of chronic pelvis pain associated with spasm of the levator ani muscles. His group found women with PFM hypertonicity and pelvic pain may respond to Botox injections into the PFM. Gajraj[178] presented a case of refractory perineal pain that was successfully treated by injecting the obturator internus muscle with a botulinum toxin. Botulinum toxin has also been used for vulvodynia. There are 2 nonplacebo-controlled pilot studies reporting an effect of injections of botox into the vestibule in the treatment of vulvodynia.[45,46] Both studies showed a significant reduction in pain for up to 12 months following treatment in 17 and 20 women.

Botulinum toxin is a strong neurotoxin with 7 distinct types (A-G) that has analgesic effect by several mechanisms. Only 2 types of toxin are commercially used, A and B. Both have similar mechanisms; however, type A (Botox) is more potent, binds to SNAP 25 receptor, and has a longer duration of action than type B (Myobloc), which binds to the vesicle-associated membrane proteins. The mechanism of action for botulinum toxin is to prevent the release of acetylcholine at the neuromuscular junction. However, studies have shown that botulinum toxin analgesic effects often last longer than its muscle relaxant effects.[179,180] Botulinum neurotoxin is a potent inhibitor of acetylcholine release, as well as a number of other neurotransmitters (NTs) and neuropeptides.[181,182] Botulinum toxin type A has demonstrated the ability to decrease neurogenic inflammation, indirectly inhibits central sensitization by decreasing activity of the wide dynamic range neurons, and exhibits an inhibitory effect on substance-P, glutamate, and calcitonin gene-related peptide.[181–183]

With its multiple mechanisms of analgesia effects, the use of botulinum toxin type A is becoming more popular for the treatment of CPP. Clinically, its use has been demonstrated in the treatment of vulvodynia, IC, pelvic floor muscle spasms, and proctalgia fugax.[45,46,81,136,137,176]

Neuromodulation

Neuromodulation is a nondestructive, neuromodulatory technique that delivers electrical current to the spinal cord, or peripheral nerves for the treatment of many chronic pain disorders. The most common indications for neuromodulation are failed-back surgery syndrome (FBSS), CRPS, peripheral vascular disease, and (in the European Union) refractory angina pectoris. Studies have shown that neuromodulation is beneficial in the treatment of many pain syndromes with good results including CPP.[184–186] With such positive results, neuromodulation is rapidly becoming a treatment option for patients with CPP. Its use has been demonstrated for many different CPP states. In this section, we will discuss the different types of neuromodulation used for CPP and its mechanisms of action.

The mechanism of neuromodulation has not been well elucidated, but several theories have been proposed. One of the most popular theories is based on

the gate control theory proposed by Wall and Melzack in 1965.[187] They postulated that spinal modulation occurs at the dorsal horn in the spinal cord. They surmised that increased activity of stimulation of large A-beta fibers was capable of inhibiting painful sensations transmitted along C fibers, thus decreasing the noxious stimulus pathway via C fibers and leading to a balance at the dorsal horn in the spinal cord.

Other postulated mechanisms involve NTs in the spinal cord. Studies have shown that neuromodulation induces a decreased release of the dorsal horn excitatory amino acids, glutamate and aspartate, and a simultaneous increase in inhibitory amino acids, gamma-aminobutyric acid (GABA), and glycine.[188] Stiller et al. found similar results showing an increase in NT levels, specifically GABA with neuromodulation. They predicted that an involvement of spinal GABAnergic mechanisms in SCS is responsible for an improvement in neuropathic symptoms.[189]

Although these theories cannot be absolutely proven, they do merit consideration. The fact that pain relief is still attained after cessation of neuromodulation supports a plausible explanation that NTs are released during stimulation. With all these possible theories, it is difficult to determine how neuromodulation really works, but we can suffice to say neuromodulation does appear to have effects on the dorsal horn of the spinal cord peripherally and possibly at the supraspinal level.[190–192] With its possible multiple mechanistic effects, neuromodulation is a useful tool for CPP.

Spinal Cord Stimulation

Neuromodulation in the dorsal epidural space has been referred to as SCS or dorsal column stimulation. For this article, we will refer to it as SCS. SCS has been used for many chronic pain states including pelvic pain. There are studies demonstrating involvement of the dorsal column pathways with respect to transmission of visceral pelvic pain.[193–195] Kapural et al.[19] performed a retrospective case series of 6 female patients with severe visceral pelvic pain. Their symptoms varied and included deep pelvic, retropubic pain, vulvodynia, dyspareunia, and rectal pains. All patients received repeated hypogastric blocks with a significant pain relief for a period ranging from 1 to 6 weeks. Three received neurolytic hypogastric blocks with the pain relief of 3, 8, and 12 months, respectively. All patients were followed with SCS with significant results. They all experienced a dramatic decrease in pain, improved activity, and decrease in opioid consumption. In our clinical experience, we have placed SCS for severe groin and testicular pain. Our patients had excellent results with significant decreased pain and narcotic use, as well as increased daily sexual activity.

Peripheral Nerve Stimulation

Peripheral nerve stimulation is another route that is safe and effective for the treatment of CPP by directly stimulating the affected nerves. A PNS requires the placement of the neuromodulation electrodes directly on the nerve (surgical) or in close proximity (percutaneous) to elicit a paresthesia over the distribution of that nerve. The first clinical trials using PNS were conducted by Wall and Sweet in 1967. They were able to demonstrate pain relief with direct stimulation of the peripheral nerves for more than 30 minutes after stimulations for 2 minutes.[196] Sweet then performed a follow-up study in 1976 where he used a cuff-life electrode and placed it directly over the injured nerve. Sweet[197] was able to show that a current could deliver paresthesias over the distribution of the nerve with pain relief and not cause a motor response. Today, PNSs are used to treat various pain disorders, including CPP. There are several approaches as mentioned earlier both having their advantages and disadvantages. However, suffice to say either approach has been successful in treating CPP. Recently, Possover et al. reported a case series of 3 patients who underwent surgical laparoscopic implantation of a peripheral nerve stimulator for CPP because of intractable pelvic neuralgia. All 3 patients had successful results with a significant decrease in pain without need for further medical treatment.[198] In another study, Kim et al. evaluated the clinical effect of intermittent percutaneous posterior tibial nerve stimulation (PTNS) in patients with CPP for pain, urgency, and 3-day frequency volume. They found that 90% of patients had significant improvement in visual analog scale scores; however, they noted that there was not statistical difference in the number of voids and bladder volumes. They concluded that PTNS may improve pain symptoms in patients with CPP.[199] As described earlier, the electrode can be placed percutaneously near the nerve to elicit a paresthesia over the distribution of that nerve. Several authors have used this approach successfully.[200–202] Tamimi et al. presented 2 case reports of patients with refractory CPP of unknown etiology with

symptoms referring to the lower abdominal wall. The patients reported excellent relief of their CPP.[200] Stinson et al. presented another series of 3 patients with intractable inguinal neuralgia. All 3 patients had PNS placed and responded favorably with 75% to 100% pain relief at 3, 20, and 12 months. All patients were tapered off their medicines and had a dramatic increase in activities of daily living.[202] In our clinical practice, we have placed peripheral nerve stimulators for various pain states, including CPP, with appreciable results. In conclusion, we believe PNS has a role in the treatment of CPP from a peripheral nerve source.

Sacral Stimulation

Sacral neuromodulation has been well described for pelvic pain syndromes from lower urinary tract disorders to CPP. With multiple pain disorders afflicting the pelvis and urogenital region, sacral nerve stimulation has been shown to have good results regarding pain control. The use of sacral neuromodulation has been described with IC, prostadynia, vulvodynia, coccydynia, proctalgia fugax, and pudendal neuralgia.

Neuromodulation of the sacral nerves has several proposed mechanism for improvement in voiding functions as well as pain control in CPP states. Sacral nerve root stimulation is thought to work by similar mechanisms as SCS. The idea is based on Wall and Melzack's gate control theory (Melzack 1965), whereby sacral stimulation may influence lower urinary tract function and improve pain control through modification of afferent and efferent mechanisms at both spinal and supraspinal levels.[203,204]

Multiple investigators have shown that sacral neuromodulation is effective for the treatment of IC. Peters[205] showed that patients with refractory IC who had sacral nerve root stimulation reported significant pain relief of at least 50% in 92% of patients. Maher et al. investigated women with intractable IC and chronic pain. Their study has similar results concluding that women with intractable IC respond favorable to percutaneous sacral stimulation with significant improvement in pelvic pain, daytime frequency, nocturia, urgency, and average voided volume.[82] Sacral neuromodulation has other applications for CPP. Nair and his group published a case report of a postmenopausal woman who complained of vulvar and vaginal burning and deep pelvic pain for 15 years. After successful trial and implantation of 19 months post-treatment, her pain was reduced by 80% and the patient no longer required medications.[206] The use of sacral stimulation has been shown to be successful in patients where multiple prior therapies have failed. With its multiple applications for CPP, sacral neuromodulation seems to be and effective method for treating pelvic pain and its associated symptoms.

ADDITIONAL MANAGEMENT CONSIDERATIONS

Modalities

Transcutaneous electrical neuromuscular stimulation has been used for the management of pain. The action of TENS is not fully understood, but the effect of high-frequency stimulation appears to modulate hyperalgesia at the spinal cord differently than low-frequency stimulation. The high-frequency stimulation activates the delta-opioid receptors, blocking the release of glutamate as aspartate.[207] Low-frequency stimulation reduces hyperalgesia through serotonin release at the spinal level.[207] However, both high-frequency stimulation and low-frequency stimulation inhibit mechanical allodynia through the rostral ventromedial medulla.[208] As the mechanisms for CPP may include peripheral hyperalgesia or mechanical allodynia, a TENS trial could include the use of both frequencies. Placement of TENS electrodes in the painful area or related sensory distribution reduces hyperalgesia both at the peripheral site as well as through central mechanisms.[208,209] Thus, stimulation location could include the painful area and related somatic or autonomic nerve roots. For the management of abdominal pain, this would include placement of the electrodes from T8 to L3 to target both the autonomic fibers (T8 to L2) and the somatic fibers (L1 to L3), placement of electrodes from L4 to S3 for the management of back and buttock pain, and placement of electrodes from S2 to S5 for the management of perineal pelvic pain.

Treatment of myofascial pain and trigger points can include the use of therapeutic ultrasound to the pelvic floor or abdominal structures. Effective short-term benefit was found with the use of 5-minute ultrasound at 1.0 watt/cm^2 in the upper trapezius myofascial tender point.[210] Pain-pressure thresholds decreased by 44% after 1 session. While there was no lasting effect, this may be an effective modality when used in combination with soft-tissue mobilization or press and stretch to the painful trigger or tender points in the pelvic floor.[211,212]

Manual Therapy

Manual therapy treatment can include massage, myofascial release, and transverse friction massage to painful ligaments, muscles, or trigger points. Additionally, it can include mobilization of a given joint to restore motion if limited, as well as to modulate the pain.[140] Specific studies measuring the benefits of these techniques are lacking in the basic science literature or in condition-specific studies. There are a limited number of studies that include manual therapy techniques as part of the regimen and demonstrate improvement in pelvic pain control and function.[140,213] Clinical experience suggests that this treatment has benefit for pain modulation. Treatments specific to muscular dysfunction can include local techniques to elongate muscles and release trigger points.[115,211,213,214] Treatment of the painful muscles has been demonstrated to be effective in women with IC and is advocated as a mainstay of nonoperative treatment for pelvic pain conditions.[91,213,214] Although the mechanism behind this muscular dysfunction and the relationship of pain to muscular dysfunction is not well understood, manual treatment is effective.[212] For further information on pain modulation, the reader is encouraged to consult the studies of Ruiz-Saez et al., Menck et al., and Pickar.[215–217]

Rehabilitation of pelvic floor dysfunction can include stretching techniques to relax hypertonicity of the PFM as well as techniques to lower trigger point sensitivity or local pain.[115,211,213,214] Incorporation of exercise to enhance gains can be an important part of the recovery of functional pelvic floor control. Identification of related pathology can be important, as trigger points are thought to be caused by local tissue overload.[115,159,218]

Relaxation techniques can be useful for the management of patients with CPP. These techniques can include diaphragmatic breathing, biofeedback, positive thinking, gentle movements such as Qigong, and postural correction. [219] The key with these strategies is to encourage the patient to utilize them when the pain level begins to rise. This can be a time to introduce pacing and the baseline of activity so that the patient is able to perform these without flare-up. Such techniques can decrease the fear of movement and enhance the positive coping strategies that are critical to successful pain management. Specific protocols have not been identified to date, but the clinical use of these strategies has been shown to modulate the affective and cognitive components of chronic pain, which are more challenging to treat with mechanical means.[220]

Management of Abdominal and Pelvic Floor Motor Control Deficit

Motor control deficit of the lumbopelvic girdle can be improved with specific muscular training.[221,222] Specific activation of the transverse abdominis (TrA) muscle can be difficult for some patients to master. Use of the pelvic floor contraction to elicit the transverse abdominal contraction has been shown to augment TrA activation.[223] Thus, the patient should be in a position of comfort that allows for optimal development of pelvic floor muscle awareness. Use of specific biofeedback can be helpful for patients with very poor awareness or with significant difficulty generating an isolated pelvic floor muscle contraction.

The initial training is focused on building awareness of the local muscular structures. This can be performed in any static position. However, it is more difficult to identify in the standing position. Key components to specific muscle activation include the following: (1) developing the ability to position the pelvis in the optimal preferred position; (2) maintaining of developing optimal breathing habits; and (3) muscular activation that emphasizes TrA and PFM without overflow to the more global muscles.

The optimal pelvic position is one where the abdominal contents can be supported by the pubic symphysis, as well as the pelvic floor, and has been described as a neutral lordosis.[23,224] Providing the patient with bony landmarks from which to assess the correct performance of pelvic alignment will enhance self-directed learning and success. Using the heel of the hand on the ASIS and the fingertips on the pubic tubercle bilaterally serves as easily identified landmarks. Aligning these landmarks in the frontal plane requires educating the patient on posterior and anterior pelvic tilt and then guiding them to identify a position between those 2 extremes. From this optimal position, the clinician can then begin training for isolated activation of relaxation of the pelvic floor and the TrA while cueing the identification of global muscle holding, such as the gluteals, abdominal obliques, or thoracolumbar paraspinals.

Sapsford et al. investigated the intensity of pelvic floor muscle contraction during a variety of sitting postures in parous women. The authors conducted an observational study of the level of activity of the pelvic floor and abdominal muscles in slump supported

sitting and very tall, unsupported sitting. Muscular activity in the PFM was significantly greater in the very tall upright sitting and upright sitting and the least in the supported slump sitting. Abdominal activity for internal and external obliques was similarly greater in the more upright positions but did not reach a statistical significance. Six of eight subjects reported a sensation of greatest PFM activity in very tall, unsupported posture followed by upright unsupported and least sensation in the slump supported positions. The authors conclude that investigators interested in treating women with urinary incontinence and weak PFM should assess optimal pelvic floor activation positions for rehabilitation.[225,226] When the findings of the various studies are taken into consideration, it appears that the pelvic floor may be maximally activated in a very tall upright position with expiration. In some cases, this may become the initial position for activation of a pelvic floor, advancing to other positions such as upright sitting and slump sitting as the patient improves. In cases of pelvic ring or sacroiliac hypermobility, application of a pelvic belt may assist by providing external stability as part of the rehabilitative process. In addition, the multifidus muscles may need to be emphasized to promote nutation of the SIJ and to minimize lumbosacral pain as a result of loss of stabilizing function of the PFM.

Once pelvic activation in initiated, the patient can then begin training with diaphragmatic breathing. Diaphragmatic breathing is encouraged in this population. To do so, the patient breathes with diaphragmatic, relaxing the abdominal wall and encouraging lateral rib cage expansion and minimizing rib cage elevation. The correct breathing pattern should be checked with the patient positioned in both recumbence and sitting, both unsupported and supported with the upper extremities. The clinician should incorporate visual feedback using a mirror to encourage the correct breathing pattern.[225] For the patient with persistent pelvic floor hypertonicity, utilization of diaphragmatic breathing with pelvic floor relaxation is an important skill to acquire for restoring functional use and suppleness of the pelvic floor muscular structures.

Besides triggering an indirect activation of the TrA through pelvic floor activity, patients can be instructed to perform additional exercises that emphasize TrA activation. Investigators have reported the use of the abdominal drawing in maneuver (ADIM), where the patient is instructed to draw the umbilicus up and in toward the posterior diaphragm.[224,227–230] Patients can perform the ADIM in the supine, prone, all-4's position on the hands and knees, and standing.[231,232] Other investigators have recommended abdominal bracing (AbB) that produces co-contraction of all of the abdominal muscle groups. To accomplish the strategy, the patient is instructed to tighten the entire abdominal region while maintaining diaphragmatic breathing.[233,234] However, the clinician should be cautioned in using this procedure, because of lack of studies examining the relationship between activation of the pelvic floor muscle function, the risk of inhibition to the pelvic floor, and the increase in intra-abdominal pressure that may be troublesome for patients with selected pelvic pain conditions.[225]

Once the patient is able to find and sustain their optimal position with selective muscle activation, endurance work is initiated.[235] As the PFM are important for force closure of the pelvis, some patients may benefit from strength and endurance training specific to these muscles.[236] To enhance automaticity of stabilization skill development, it is necessary for the patient to be able to self-correct their postural strategy. The in-clinic training can then focus on novel activities that enhance functional recovery but challenge the stabilizing skill ability.

The pelvic floor has a postural support function and assists with trunk, pelvic ring, hip complex, and distal lower extremity support. In addition, it has a significant role in the sphincter function for continence. A specific training program includes postural awareness and sustained holding contraction. The progression of strength and endurance training of the pelvic floor for sphincter function has been well described in the literature.[221,237] The key features of such program include: (1) specific pelvic floor assessment prior to onset of program; (2) 8 to 12 near-maximal pelvic floor muscle contractions twice daily at home, followed by 5 fast contractions; and (3) 12 once-per-week clinic sessions with a clinician who specializes in pelvic floor rehabilitation to enhance neuromuscular training, motivation, and compliance and to incorporate new positions to challenge the stabilizing muscles.

Sapsford goes further, recommending a 5-stage program for restoring PFM activation that integrates diaphragmatic breathing. The first stage begins with isolated diaphragmatic breathing (previously described) that synergistically activates the pelvic floor. Then, patients are asked to initiate pelvic floor muscle tonic activity with 2 strategies: (i) ADIM and (ii) periurethral PFM hold. Investigators have found that

ADIM synergistically activates the PFM groups. For PFM hold, the patient is instructed to tighten and life the PFM up into the pelvic floor region with an initial activation (submaximal) contraction. Here, patients can be encouraged to utilize a muscular contraction similar to that used to stop the flow of a bowel movement. Traditionally, pelvic floor training was cued by asking the patient to stop the flow of urine. However, these instructions could disrupt the normal micturition cycle and is not advised in those patients suffering from disturbances in urinary tract function.

For stage 3, the patient is encouraged to strengthen the PFM groups, where they perform ADIM plus a maximum PFM hold-and-lift for 15 to 40 seconds. This is followed by stage 4, where the ADIM and PFM activation are implemented during functional expiratory patterns, such as nose blowing, sneezing, laughing, and coughing. Finally, for stage 5, the patient performs the same activities during functional impact activities that include walking, running, and sport-specific activities.

Analysis of muscular timing and strength to perform functional tasks is the key to optimize motor control training and is accomplished with the use of palpation or internal biofeedback during the in-clinic practice session. Once the patient has developed the internal awareness, the use of palpation and biofeedback is minimized to enhance motor learning.

Management of Pelvic Ring

Association of pelvic ring disturbances, including instability and load transfer dysfunction, can be managed effectively with the use of pelvic girdle belt to provide external force closure to the pelvic girdle.[238] This is followed by motor control training that has been previously described. While wearing the pelvic belt, the patient is taught strategies to minimize overload to the pelvic girdle. Activities involving heavy load bearing on 1 leg should be avoided, as well as positions of extreme hip flexion/extension or adduction, such as when crossing the legs. The patient is encouraged to use the belt for 4 to 6 months, so to allow external support while the temporal aspects of the pelvic girdle musculature are restored through a specific exercise program.[21] Stiffening of the thoracolumbar fascia through training of its muscular attachments to the latissimus dorsi, gluteus maximus, abdominals, and indirectly the hamstrings is recommended to assist in stabilizing the lumbar spine and pelvic girdle.[239] A patient's management is considered successful when the patient is able to resume typical daily activities without application of the belt.

Management of Viscerosomatic Convergent Pain

Nonoperative treatment for the viscerosomatic convergence consists of alleviating the distress of the secondary pain mechanism. As many women with endometriosis present with IBS, IC, and chronic fatigue syndrome, a central pain processing alteration is likely.[240] The secondary pain can be managed using local treatment provided in a multidisciplinary environment.

Treatment is focused on pain-relieving approaches aimed at calming the central nervous system. Diaphragmatic breathing promotes a healthy breathing pattern, as well as improves self-regulation and relaxation.[219] Specific treatment for somatic complaints is likely to include soft-tissue techniques. These may be carried out in the abdominal wall or in the pelvic floor areas. Several studies have shown improvement or resolution of vulvodynia and IC using myofascial treatment of the sensitized PFM.[211,214,241] Progressing to active movements that do not provoke pain and enhance aerobic function further improves the recovery of movements in those patients with CPP.

Often, such mechanical interventions are conducted within a multidisciplinary team approach to provide the patient with opportunities to enhance coping mechanisms via counseling and to treat the central processing dysregulation component with medications.

Management of Pelvic Floor Somatic Disorders

These disorders are characterized by pelvic floor hypertonicity, which can be addressed with manual techniques to each muscle using the press and stretch method.[115] This technique involves intravaginal palpation of the involved muscle, locating the painful nodule or fibrous band. The clinician manually lengthens each muscle longitudinally pressing into the tissue and taking up the stretch as the muscular tissues give way, repeating this technique for 5 to 15 repetitions depending on the patient's tolerance.[115,211,214] After completing the press and stretch repetitions, the patient is asked to perform a gentle contraction against the resistance to enhance neuromotor programming of the muscle. While the primary pathology of each condition leading to somatic pain syndromes of the pelvic floor

differs, once the condition causes somatic involvement, the treatment is similar to that described earlier. Such treatments are effective for patients with IC, urinary frequency/urgency, and for levator ani syndrome and is likely to help those patients with vulvodynia, vaginismus anismus, and dyspareunia.[211,214] The treatment is focused on alleviating the hypertonicity of the pelvic floor, restoring optimal pelvic floor function, and addressing any coexisting muscular skeletal findings.

Interventional pain techniques for pelvic hypertonicity include trigger point injections, dry needling, and acupuncture. Injections to palpable trigger points complement medical treatment in patients with CPP with a myofascial component.[242] Acupuncture for low back and pelvic girdle pain related to pregnancy has been shown to be better than standard treatment or stabilizing exercises in a single randomized control study.[243] The treatment should include deep needle manipulation and an individualized sequence. [244] Then, the patient can begin training for diaphragmatic breathing.

Acupuncture for dysmenorrhea has been reported to improve quality of life a relive pain better than usual care with a modest increase in overall costs.[245] Likewise, acupressure for dysmenorrhea applied at onset of menstruation improved pain for up to 2 hours postapplication and could be performed as a self-help skill.[246] These techniques might be followed by immediate motor relearning techniques such as manual therapy to the PFM and muscular work to improve the contractility and relaxation response to voluntary movement; however, more research is needed to substantiate these approaches.

CONCLUSION

Chronic pelvic pain is a multifactorial pain condition that is prevalent among women. A systematic examination facilitates differential diagnosis of the pain generators leading to diagnosis-specific management of this condition. While the current understanding of the pain as a secondary disease is developing, utilizing a multidisciplinary approach based on the biophysical model can improve outcomes for patients suffering from this condition and minimize the associated disability.

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APPENDIX I: DIFFERENTIAL DIAGNOSTICS FOR PELVIC PAIN

Adductor muscle lesions	Adenomyosis
Coccydynia	Adhesions
Femoral nerve entrapment	Dysmenorrhea
Hip labrum pathology	Endometriosis
Hip osteoarthritic changes	Leiomyoma
Lumbar disk	Intrauterine device (IUD)
Lumbar facet	Mittleschmerz
Lumbar stenosis	Ovarian pathologies
Muscular imbalance	Pelvic inflammatory conditions
Myofascial pain syndromes	Pelvic congestion syndrome
Nerve Entrapments – non operative	Post surgical hypertonus, scar tissue
Nerve Entrapment – post operative	Prolonged pelvic pain of pregnancy
Pelvic ring hypermobility	Prolapse
Pelvic floor instability, support defects	Vaginismus
Pelvic girdle muscle dysfunction	Vulvodynia
Sacroiliac joint pain	**Affective**
Spinal stenosis conditions	Depression
Symphysis pubis lesion	Anxiety
Thoracic disk	Trauma/Abuse
	Altered coping mechanisms
Gastrointestinal	**Urologic**
Cholelithiasis	Interstitial cystitis
Crohn's disease	Chronic urinary tract infections
Chronic appendicitis	Detrusor instability
Constipation	Cystocele
Functional bowel disorders – Irritable bowel Inflammatory bowel conditions/ulcerative colitis	Urethral syndrome

Adapted from Gunter[92]; Tu[92].