REVIEW ARTICLE

Chronic Pelvic Pain in Females: A Multisystem Perspective for the Osteopathic Physician

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KEYWORDS

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Women's health

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Somatic dysfunction

ABSTRACT

Chronic pelvic pain (CPP) is a debilitating condition that significantly impacts the quality of life of females. It is defined as pelvic pain lasting at least 6 months or longer, and stems from multiple etiologies, including gynecologic, gastrointestinal, urologic, musculoskeletal, neurologic, and psychological issues. Considering its complex presentation, CPP management often demands a multidisciplinary approach. This review describes the osteopathic approach to managing CPP. A discussion of the five osteopathic models (biomechanical, respiratory-circulatory, metabolic-nutritional, neurologic, and biopsychosocial) is evaluated, demonstrating how OMT can improve structural imbalances, fluid dynamics, autonomic tone, and mental well-being in females living with CPP. Integrating OMT with nonosteopathic approaches such as pharmacologic treatment, pelvic floor physical therapy, dietary-lifestyle interventions, and behavioral health support provides a holistic framework to manage CPP. In this review, we provide support for the role of osteopathic physicians in the multidisciplinary care of CPP and emphasize the utility of integrative approaches to optimize therapeutic outcomes.

INTRODUCTION

Chronic pelvic pain (CPP) is a multidimensional symptom that demonstrates the interrelationship between structure and function within the female pelvic system. The structures of the pelvic cavity include bones, muscles, ligaments, and fascia that work together to support the pelvic organs and physiologic functions such as continence, sexual activity, and childbirth. When the pelvic system is imbalanced, it can manifest as pelvic floor dysfunction, pelvic floor hypertonicity, or other somatic dysfunctions leading to long-lasting pain that negatively impacts physical and mental health as well as quality of life. CPP can be attributed to various etiologies, including gastrointestinal, gynecologic, musculoskeletal, neurologic, psychological, urologic, and vascular causes.[1,2]

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Although research on CPP has advanced, it is still underdiagnosed and undertreated in primary care. Treatment is typically centered on pharmacologic agents, surgery, or referrals to specialists. In addition to ordering diagnostic tests and prescribing pharmacologic treatments, osteopathic physicians contribute a complementary skillset by providing OMT, a noninvasive and nonpharmacologic option that may reduce symptoms and enhance quality of life. Recent research on OMT has reported reductions in pelvic pain and overall symptom improvement.[3-6] This evidence highlights the role of OMT in symptom reduction and its value as an adjunct to other modalities in CPP treatment. By integrating OMT with conventional care, osteopathic family physicians can expand treatment options and address somatic dysfunction in patients with CPP.

DEFINITION AND EPIDEMIOLOGY

CPP is a complex and often debilitating symptom that affects millions of individuals worldwide, with a higher incidence among females. It is characterized as cyclic or noncyclic pelvic pain persisting for a minimum duration of

TABLE 1: Diagnostic criteria for chronic pelvic pain.

Criterion	Description
Duration	Persistent or recurrent pain lasting ≥6 months
Location	Localized to pelvis, lower abdomen (at or below the umbilicus, lumbosacral back, and/or buttocks)
Pain pattern	Cyclic or noncyclic
Associated symptoms	Dysmenorrhea, dyspareunia, dysuria, dyschezia
Functional impact	Pain impairs daily function or requires medical care, and is associated with negative cognitive, behavioral, sexual, or emotional consequences

6 months and may be associated with symptoms involving the lower urinary tract, bowel, pelvic floor, myofascial tissues, and/or reproductive systems.[1,2,7,8] CPP is also frequently accompanied by negative cognitive, behavioral, sexual, and psychological effects.[8] Pain is typically localized to the pelvis, the anterior abdominal wall at or below the umbilicus, the buttocks or lumbosacral region, and may cause functional disability, impede daily activities, and potentially require medical attention.[9-12] In conjunction with criteria outlined in Table 1, CPP is a diagnosis of exclusion that should be primarily informed by a comprehensive history and physical examination. Diagnostic workup may be supplemented with appropriate imaging and laboratory testing, as clinically indicated, to rule out other potential underlying etiologies as detailed in Table 2.[10,13]

TABLE 2: Differential diagnoses of chronic pelvic pain.

Cause	Mechanism	Associated Symptoms
GYNECOLOGICAL		
Endometriosis	Ectopic endometrial tissue implantation causes chronic inflammation, fibrosis, and nerve desensitization	Bowel and/or bladder discomfort, cyclical pelvic pain, dysmenorrhea, dyspareunia, infertility
Adenomyosis	Endometrial tissue invades the myometrium, leading to chronic inflammation and increased prostaglandin production	Heavy and painful menstrual periods, pelvic pain, and pressure symptoms
Chronic pelvic inflammatory disease	Persistent infection and inflammation lead to scarring, adhesions, and hypersensitization of pelvic nerves	Abnormal discharge, dyspareunia, low-grade fever, pelvic pain
Ovarian cysts	Expansion and/or rupture of cysts lead to peritoneal nerve irritation and inflammatory responses	Bloating, dyspareunia, pelvic pain
Uterine fibroids	Compression of the structures surrounding the uterus causes local inflammation	Frequent urination, heavy menstrual bleeding, pelvic pressure, and pain symptoms
Pelvic adhesion	Fibrotic bands tether organs, restricting mobility and causing pain and inflammation	Bowel and/or bladder dysfunction, pelvic pain, dyspareunia

TABLE 2: Differential diagnoses of chronic pelvic pain. *cont.*

Cause	Mechanism	Associated Symptoms
GYNECOLOGICAL		
Cervical stenosis	Narrowing of the cervical canal leads to obstruction of menstrual flow and increased intrauterine pressure	Infertility, irregular bleeding, painful periods
Obstetric trauma	Vaginal or perineal tearing, nerve damage, or pelvic floor muscle injury during childbirth can lead to chronic pain	Dyspareunia, pelvic pain, perineal discomfort, urinary and/or fecal incontinence
UROLOGICAL		
Interstitial cystitis	Chronic inflammation and dysfunction of the bladder epithelium lead to hypersensitivity and pain	Chronic bladder pain, urinary urgency, urinary frequency
Recurrent urinary tract infections	Persistent infections cause chronic inflammation and nerve irritation in the urinary tract	Dysuria, pelvic pain, urinary urgency, urinary frequency
Bladder dysfunction	Dysfunctional voiding patterns lead to bladder hypersensitivity and pain perception	Incomplete voiding, urinary urgency, urinary retention
Urethral syndrome	Chronic irritation or inflammation of the urethra leads to pain and discomfort	Burning with urination, pelvic pain, urethral tenderness
GASTROINTESTINAL		
IBS	Visceral hypersensitivity and dysmotility lead to abdominal and pelvic pain	Bloating, cramping, diarrhea and/or constipation, pain relief after defecation
IBD	Chronic mucosal inflammation and cytokine release contribute to pelvic pain	Abdominal pain, bloody stools, chronic diarrhea
Chronic constipation	Increased colonic distension and pressure on pelvic nerves cause pain.	Bloating, hard stools, pelvic pressure, straining
Diverticulitis	Inflammation and microperforations in the colon lead to localized and referred pelvic pain.	Altered bowel habits, fever, nausea, lower abdominal pain
Hernias (inguinal, femoral, obturator)	Entrapment or irritation of nerves near the hernia site contributes to pain.	Groin pain, pelvic pain
Colorectal Cancer	Tumor growth within the colon or rectum can cause obstruction, nerve compression, and inflammation	Pelvic and/or lower abdominal pain, rectal bleeding, weight loss

TABLE 2: Differential diagnoses of chronic pelvic pain. *cont.*

Cause	Mechanism	Associated Symptoms
MUSCULOSKELETAL		
Myofascial pain syndrome	Trigger points in pelvic muscles cause referred and localized pain	Localized pelvic pain, muscle tenderness, trigger points
Pelvic floor muscle dysfunction	Muscle hypertonicity or spasms lead to ischemia, nerve compression, and pain	Dyspareunia, pelvic pain, urinary dysfunction
Fibromyalgia	Central sensitization leads to widespread musculoskeletal pain, including the pelvis	Diffuse pain, fatigue, pelvic pain, sleep disturbances, tender points
Herniated lumbar disc	Nerve root compression can cause referred pelvic pain	Lower back pain, pelvic pain, numbness/tingling in legs, sciatica
Sacroiliac joint dysfunction	Inflammation or mechanical dysfunction of the sacroiliac joint causes referred pelvic pain	Buttock/lower back pain, pelvic pain, pain with walking and/or sitting
NEUROLOGIC		
Pudendal neuralgia	Compression or irritation of the pudendal nerve leads to burning and stabbing pelvic pain	Burning sensation, pelvic pain, perineal pain, pain with sitting
Nerve entrapment (ilioinguinal, genitofemoral, obturator)	Direct irritation or compression of peripheral nerves causes chronic neuropathic pain	Burning, shooting pelvic pain along nerve distribution, hypersensitivity
Central sensitization syndromes	Dysfunctional pain processing in the CNS amplifies pelvic pain perception	Allodynia, cognitive issues, fatigue, widespread pain
PSYCHOLOGICAL		
Anxiety, depression	Altered neurotransmitter levels and heightened pain perception contribute to chronic pain	Fatigue, mood changes, pelvic pain, sleep disturbances
Somatization disorder	Psychological distress manifests as physical pain	Vague, widespread pain without a clear organic cause
History of trauma or abuse	PTSD-related heightened pain sensitivity affects pain processing	Anxiety, avoidance behaviors, hypervigilance, pelvic pain
VASCULAR		
Pelvic congestion syndrome	Venous hypertension and reflux cause chronic ischemia and inflammation, leading to pain	Dull and achy pelvic pain, dyspareunia, lower-extremity edema
Varicose veins in the pelvis	Blood pooling leads to increased vascular pressure and nerve irritation	Pelvic heaviness, pain with prolonged standing
May-Thurner syndrome	Left iliac vein compression causes pelvic venous congestion and pain	Lower-extremity edema, pelvic pain
Deep vein thrombosis	Blood clots in the deep veins of the pelvis or legs lead to venous obstruction and inflammation	Unilateral lower-extremity edema, pelvic/lower abdominal pain
OTHER		
Postsurgical pain	Nerve damage or chronic inflammation after surgeries	Pelvic pain, scar tenderness
Chronic postinfectious pain	Residual nerve damage or persistent low-grade inflammation leads to pain	Pelvic discomfort or pain
Adhesions from surgery or infection	Fibrous bands restrict organ movement and contribute to mechanical pain	Bowel and/or bladder dysfunction, dyspareunia, pelvic pain
Physical trauma (eg, pelvic fracture, nerve injury)	Direct mechanical injury or strain leads to musculoskeletal damage, nerve irritation, or scar tissue formation	Pelvic pain and/or tenderness, bowel and/or bladder dysfunction

Abbreviations: CNS, central nervous system; IBD, inflammatory bowel disease; IBS, irritable bowel syndrome; PTSD, posttraumatic stress disorder

CPP is a significant public health burden affecting females globally. Due to variations in diagnostic criteria, patient underreporting, absence of a standardized definition of CPP, and its association with other medical conditions, CPP is often underdiagnosed, making it challenging to determine accurate prevalence rates.[14,15] CPP prevalence is estimated to be as high as 26% in individuals with female anatomy.[16] CPP is most commonly seen in females between the ages of 18 and 50 years, but it is not exclusive to this range and may extend beyond reproductive years, affecting older women as well.[17] One study demonstrates that its prevalence is comparable to asthma and lower back pain.[15] Endometriosis and bladder pain are the two most prevalent medical conditions associated with CPP, affecting approximately 60%–70% of these individuals. Additionally, other common comorbidities affecting at least 20% of CPP patients include IBS, interstitial cystitis, pelvic floor muscle tenderness, and depression.[18]

PATHOPHYSIOLOGY

The pathophysiology of CPP is multifactorial, involving the interaction between inflammatory, muscular, neurologic, and psychological mechanisms. Peripheral mechanisms of CPP occur when nociceptors are activated through tissue injury and inflammation in chronic conditions such as endometriosis, interstitial cystitis, and IBS.[19] Over time, after the inflammation subsides, a continued nociceptive action occurs, resulting in peripheral sensitization where the threshold for nociceptor activation is lowered, leading to hyperalgesia and allodynia.[20] Another mechanism leading to CPP is central sensitization, where the CNS heightens pain perception. Functional neuroimaging studies demonstrate that structural changes in the brain's pain-processing regions lead to abnormal central transmission of pain signals, which persist even after the initial injury has resolved.[21] Musculoskeletal mechanisms

include pelvic floor muscle dysfunction, hypertonicity, postural deviations, or pelvic asymmetries that lead to alterations in muscle mechanics and dynamics, causing pain and discomfort.[22] Psychosocial factors, including anxiety, depression, and trauma history, including adverse childhood experiences, are widespread among individuals with CPP.[23] These factors can regulate pain sensitivity through dysregulation of the hypothalamic-pituitary-adrenal axis and may increase the likelihood of central sensitization.[24]

CLINICAL EVALUATION

Guided by the osteopathic tenet recognizing the interdependence of structure and function, the physician should conduct a comprehensive history and physical examination to detect pathology as well as somatic dysfunctions contributing to the patient's pain. The patient interview should elicit a detailed medical, surgical, obstetric, and gynecologic history, with attention to pain characteristics including duration, location, quality, intensity, temporal pattern, exacerbating and relieving factors, prior interventions, and any correlation with menstruation, micturition, defecation, or sexual activity.[8,10,25,26] A thorough review of systems should address the gastrointestinal, genitourinary, musculoskeletal, neurologic, and psychological systems to narrow the differential diagnosis.[27] The physician should identify alarming signs such as postcoital or postmenopausal bleeding, hematuria, unexplained weight loss, fever, or pelvic masses that may indicate serious underlying pathology, such as an acute abdomen or potential malignancy, requiring immediate investigation.[9,10,28] The International Pelvic Pain Society offers a structured history and physical examination tool that streamlines systematic evaluation.[29] This free resource includes visual pain mapping, symptom quantification scales, validated screening questionnaires, and an in-depth review of reproductive, urinary, and gastrointestinal systems.[8,10] Additionally, clinicians should understand the patient's perception of the pain's origin and utilize standardized tools such as the Quality of Life Scale to assess the impact on daily function.[8,10]

A physical examination should be performed with a focus on the abdominal and pelvic neuromusculoskeletal systems.[8] This encompasses abdominal palpation and pelvic inspection for focal tenderness, surgical scars, vaginal discharge, pelvic organ prolapse, uterine enlargement, masses, and myofascial trigger points.[10] The pelvic examination should consist of the external inspection to evaluate for signs of infection, dermatologic conditions, vulvar malignancy, and neurologic changes, as well as the internal examination, which should include bimanual palpation to assess for uterine or adnexal tenderness, masses, and cervical motion pain.[10] Palpation of the pelvic floor muscles may reveal hypertonicity, tenderness, or trigger points, which may suggest underlying myofascial dysfunction. A focused osteopathic structural exam should include assessment of the abdomen, lumbar spine, sacrum, and sacroiliac joints to evaluate postural abnormalities, gait disturbances, or other somatic dysfunctions potentially contributing to CPP, while being aware that somatic dysfunction anywhere in the body system can influence CPP. Carnett's test may be used to differentiate between visceral pain and abdominal wall pain.[8,10] Additionally, the cotton swab test, applied to the abdominal skin, can help identify cutaneous allodynia.[8,10]

Laboratory testing has limited diagnostic utility in the setting of CPP. When indicated, laboratory tests typically include a complete blood count, urinalysis, pregnancy test, and screening for sexually transmitted infections.[10,28] Transvaginal ultrasound is the first-line imaging modality for visualizing gynecologic structures, and can detect uterine fibroids, ovarian cysts, and characteristics of endometriosis or pelvic inflammatory disease.[8,28] When pelvic ultrasound findings are inconclusive, pelvic magnetic resonance imaging (MRI) may offer better diagnostic insights. If endometriosis or adhesions are suspected and a noninvasive workup is inconclusive, laparoscopy may be indicated to allow visualization and potential therapeutic intervention. If laparoscopy is unremarkable, CPP may be suggestive of chronic regional pain syndrome.[28] Diagnostic nerve blocks may also be helpful to identify peripheral nerve dysfunction as a contributor to CPP, when neuropathic etiology is suspected.[28]

PELVIS ANATOMIC CONSIDERATIONS

To address somatic dysfunctions of the pelvic region, the osteopathic physician must have an understanding of the pelvic anatomy and biomechanics. The pelvic region itself encompasses the bony pelvis, pelvic cavity, pelvic floor, and perineum.[30,31] The pelvic bowel consists of the sacrum, coccyx, and two innominate bones, each comprised of the ilium, ischium, and pubis, which connect at the sacroiliac joints, pubic symphysis, and sacrococcygeal joint, with the spine at the lumbo-sacral joint, the lower extremities at the acetabulum of the hip, and the pelvic floor musculature.[30] This osteoligamentous ring-like structure is divided into the false and true pelvis, supporting trunk stability and pelvic organ function.[31] The pelvic floor is made of the levator ani muscle group (pubococcygeus, iliococcygeus, puborectalis) and coccygeus, along with surrounding muscles such as the piriformis and obturator internus, which regulate continence, support viscera, and stabilize the pelvis and lumbar spine.[32] The pelvic musculature, with its fascial connections, mediates pelvic balance and transmits biomechanical forces during locomotion.

Somatic dysfunction in these musculoskeletal or fascial aspects of the pelvis can lead to altered pelvic mechanics, visceral dysfunction, and pain.

INFORMED CONSENT

Due to the intimate nature of the pelvic region, informed consent must be obtained before initiating diagnosis or OMT.[33] Physicians should facilitate a discussion with the patient that includes:

- A description of the proposed examination and potential treatment, and how they could help address the patient's symptoms
- An explanation of where physical contact is required and anatomic regions involved
- Potential benefits of treatment responses and post-OMT effects
- Contraindications associated with treatment, especially those pertinent to the patient's history and presentation

When patients are not well informed, they may experience psychological distress or muscle tension during treatment, which can interfere with therapeutic benefits and potentially exacerbate their symptoms.[34] Patients retain the right to decline any medical diagnosis or intervention, including OMT, at any point during their treatment.

THE FIVE OSTEOPATHIC MODELS AND OMT FOR CHRONIC PELVIC PAIN

An osteopathic approach emphasizes the integration of body, mind, and spirit to provide holistic care. Utilizing the five osteopathic models, the physician can systematically evaluate how somatic dysfunction may contribute to CPP. An osteopathic treatment plan can be based on a thorough understanding of the patient's risk factors, comorbid conditions, and individualized goals of care. Treatment should be directed at both the underlying etiology of CPP and its associated somatic dysfunctions. OMT has been demonstrated to be effective in the management of CPP by addressing musculoskeletal imbalances, pelvic floor dysfunction, and central sensitization associated with pelvic conditions, including endometriosis.[5,6] Clinical studies have consistently reported that patients receiving OMT experience significant reductions in pelvic pain symptoms, dyspareunia, and improved quality of life outcomes.[3,4] Importantly, OMT is often used as part of a multimodal treatment strategy. It can be integrated with other evidence-based modalities, including, but not limited to, pharmacologic management, surgical interventions, and physical therapy. This integrative approach aims to optimize therapeutic outcomes and enhance overall patient well-being. The overall goal of integrating osteopathic treatment is to restore normal physiologic motion, optimizing healing and function. Potential OMT that can be applied to address CPP is listed in Table 3.[30,31,35,36]

TABLE 3: Potential OMTs for CPP by osteopathic model.

Technique	Basic Steps	Contraindications
BIOMECHANICAL MODEL		
Soft tissue – prone traction (lumbosacral method)	Patient is placed in a prone position. Using both hands, exert a gentle ventral force and create a separation and distraction effect. A gentle, rhythmic, and kneading fashion, or sustained pressure may be applied	Acute lumbar/sacral fracture, severe hip arthritis, acute sprain, joint hypermobility, deep vein thrombosis
Lumbar soft tissue – prone pressure with counterleverage	Patient is placed in a prone position. Apply a ventral and lateral pressure perpendicular to the paraspinal musculature with the cephalad hand and apply a posterior force to lift ASIS with caudad hand in a gentle, rhythmic, and kneading fashion, or with sustained pressure	Acute lumbar/sacral fracture, severe hip arthritis, acute sprain, joint hypermobility, deep vein thrombosis
Lumbar soft tissue – supine extension	Patient is placed in a supine position. Use both hands to contact lumbar paraspinal musculature and apply a ventral and lateral force. A gentle, rhythmic, and kneading fashion, or sustained pressure may be applied	Acute lumbar/sacral fracture, severe hip arthritis, acute sprain, joint hypermobility, deep vein thrombosis
Innominate MET: • Anteriorly rotated innominate • Posteriorly rotated innominate	Anteriorly rotated innominate: Patient is in a supine position. Flex the hip and knee on the affected side to the restrictive barrier. Patient is instructed to gently push against the physician's resistance by trying to extend the hip Posteriorly rotated innominate: Patient is in a supine position. Extend the hip to the restrictive barrier, then the patient is instructed to gently lift the leg against resistance For all MET, patient is instructed to gently contract a specific muscle group against the physician's resistance. After holding the contraction for 3–5 seconds, the patient relaxes, and the physician repositions the body part to a new restrictive barrier. This process is repeated several times	Acute pelvic fracture, sacroiliac joint inflammation or hypermobility, rheumatoid arthritis, severe hip/knee arthritis

TABLE 3: Potential OMTs for CPP by osteopathic model. *cont.*

Technique	Basic Steps	Contraindications
BIOMECHANICAL MODEL		
Still technique innominate: • Anteriorly rotated innominate • Posteriorly rotated innominate	Anteriorly rotated innominate: Patient is in a supine position. Monitor the PSIS and flex the hip and knee on the dysfunctional side. The leg is abducted and externally rotated until motion is felt, then slight compression is applied while the leg is moved into adduction and extension. The extremity is then returned to the neutral position Posteriorly rotated innominate: Patient is in a supine position. Monitor the PSIS and fully flex the hip and knee on the affected side. The leg is then abducted and externally rotated to the point of ease, with slight compression applied through the femur. While maintaining compression, the leg is returned to the neutral position	Pelvic fracture or instability, sacroiliac or hip inflammation, osteoporosis, hypermobility
Soft tissue – gluteus minimus trigger point	With the patient lying on their side, apply firm pressure between the iliac crest and greater trochanter over the gluteus minimus. Pressure is held until the trigger point softens, and a release is felt	Acute hip fracture or infection
Soft tissue – elbow pressure to piriformis	While the patient lies on their side with hips and knees bent to 90 degrees, apply elbow pressure over the piriformis muscle until a release is felt	Acute hip fracture or infection
Suboccipital release	Patient is in a supine position. Contact the occipital sulcus bilaterally and medially with the index and middle fingers, then apply and maintain a linear traction by rolling fingers out laterally	Acute cervical fracture or instability, rheumatoid arthritis, Down syndrome, Chiari malformation
NEUROLOGIC MODEL		
Anterior lumbar counterstrain AL1: Medial to the anterior superior iliac spine AL2: Medial to the anterior inferior iliac spine AL3: Lateral to the anterior inferior iliac spine AL4: Inferior to the anterior inferior iliac spine AL5: Anterior, superior aspect of the pubic ramus just lateral to the symphysis	Patient is in a supine position. AL1: Flex to L1, side bend toward, knees toward the side of dysfunction to rotate L1 away AL2-4: Flex to spinal level, side bend away, knees away, which rotates the lumbar segment toward AL5: Flex, side bend away, and knees toward which rotates the lumbar segment away All treatments should be positioned correctly and maintained for 90 seconds, or until the physician palpates improvement in at least two to three TART findings	Acute lumbar fracture or strain, severe osteoporosis, hip dislocation, herniated disc
NEUROLOGIC MODEL		
Posterior lumbar counterstrain PL1-5 spinous process: inferolateral aspect/tip of the deviated spinous process of the dysfunctional segment PL1-5 transverse process: posterolateral aspect of the transverse process of the dysfunctional segment	Patient is in a prone position. Extend to the affected spinal level by lifting the extremity or ASIS on the side of the tender point, which rotates the pelvis toward and upper segment away from the side of dysfunction, also adduct the lower extremity. All treatments should be positioned correctly and maintained for 90 seconds, or until the physician palpates improvement in at least two to three TART findings	Acute lumbar fracture or strain, severe osteoporosis, hip dislocation, herniated disc, acute radiculopathy
Anterior pelvic counterstrain Psoas: 2/3 distance from ASIS to midline Iliacus: 1/3 distance from ASIS to midline Low ilium: Superior surface of iliopectineal eminence Inguinal: Lateral aspect of pubic tubercle	Psoas: Significant bilateral hip flexion with lumbar sidebending toward the affected side, may involve some hip external rotation Iliacus: Pronounced bilateral hip flexion and external rotation, knees flexed Low ilium: Notable hip flexion on the affected side Inguinal: Thighs flexed with the opposite thigh crossed over the affected thigh, the lower leg All treatments should be positioned correctly and maintained for 90 seconds, or until the physician palpates improvement in at least two to three TART findings	Pelvic or sacral fractures, sacroiliac joint inflammation/ hypermobility, osteoporosis
Posterior pelvic counterstrain Upper pole L5: Superior medial surface of PSIS High ilium sacroiliac: 2-3 cm lateral to posterior superioriliac spine pressing medially toward PSIS Lower pole L5: on ilium, inferior to PSIS pressing superiorly High ilium flare out: Lateral aspect of ILA and/or coccyx PL3 lateral: 2/3 lateral from PSIS to the tensor fasciae latae PL4 lateral: posterior margin of tensor fasciae latae Piriformis: Midpoint between the lower half of the lateral aspect of the sacrum, ILA, and the greater trochanter	Upper pole L5: Hip extension with fine-tuning through adduction and either internal or external rotation High ilium sacroiliac: Hip extension with adduction and fine-tuning through external rotation Lower pole L5: Hip flexed to 90 degrees, slight internal rotation and adduction High ilium flare out: Hip extension with adduction PL3 and PL4 lateral: Hip extension with abduction and external rotation; adjust positioning as needed for tenderness reduction Piriformis: Marked hip flexion and abduction with fine-tuning using internal or external rotation All treatments should be positioned correctly and maintained for 90 seconds, or until the physician palpates improvement in at least two to three TART findings	Pelvic or sacral fractures, sacroiliac joint inflammation/ hypermobility, osteoporosis

Technique	Basic Steps	Contraindications
NEUROLOGIC MODEL		
Occipitoatlantal decompression	Patient is in a supine position. Support the occiput with both hands, placing the middle fingers on the posterior arch of the atlas. Gentle caudad traction is applied as the patient tucks their chin to their chest and holds deep inspirations to enhance articular release	Acute cervical instability, rheumatoid arthritis, vertebrobasilar insufficiency
CV4 technique	Patient is in a supine position. Cup the occiput, placing the thenar eminences on either side of the squama. Gentle medial compression is applied to resist cranial flexion and encourage extension, held until a still point is reached	Coagulopathy, intracranial mass, vertebrobasilar insufficiency
Ganglia release Celiac ganglion: below xiphoid Inferior mesenteric ganglion: above the umbilicus Superior mesenteric ganglion: halfway between the celiac and inferior mesenteric ganglion	Patient is in a supine position. Apply a gentle, sustained pressure with fingertips over the midline abdomen at the level of the targeted ganglion. Pressure is maintained until a softening or release is palpated in the underlying tissues	Abdominal aneurysm, coagulopathy, active intra-abdominal infection
Paraspinal inhibition	Patient is in a supine position. Contact the paraspinal muscles and apply steady pressure lateral to the spinous processes. This pressure is held for 2–5 minutes to reduce hypertonicity and facilitate muscle relaxation	Acute spinal fracture or tumor, infection, osteoporosis
Sacral inhibition	Patient is in a prone position. Place one or both hands over the sacrum, applying gentle, sustained pressure until a release is felt	Sacral fracture, local infection or abscess, coagulopathy, osteoporosis
Abdominal pump	Patient is in a prone position. Place their hands over the thoracolumbar fascia, applying gentle pressure to engage the tissues. The hands are then twisted in opposite directions while maintaining compression and traction to create a myofascial stretch until a release is felt	Recent abdominal surgery or infection, malignancy, deep vein thrombosis, cardiac/pulmonary compromise, pregnancy
RESPIRATORY-CIRCULATORY MODEL		
Thoracolumbar release	Patient is in a prone position. Place their hands over the thoracolumbar fascia, applying gentle pressure to engage the tissues. The hands are then twisted in opposite directions while maintaining compression and traction to create a myofascial stretch until a release is felt	Acute fracture, open wounds, osteoporosis
RESPIRATORY-CIRCULATORY MODEL		
Thoracolumbar release	Patient is in a prone position. Place their hands over the thoracolumbar fascia, applying gentle pressure to engage the tissues. The hands are then twisted in opposite directions while maintaining compression and traction to create a myofascial stretch until a release is felt	Acute fracture, open wounds, osteoporosis,
Lumbosacral compression/ decompression	Patient is in a prone position. Place one hand on the sacrum and the other on the lumbar spine, then gently compress and decompress the fascia by moving the hands together and apart. The fascia is either followed (indirect) or c hallenged (direct) until tissue release is felt	Acute fracture, open wounds, osteoporosis, hypermobility, acute radiculopathy
Thoracolumbar MFR (diaphragm, thoracic outlet)	Patient is in a supine position. Place one hand on the lower ribs and the other on the thoracolumbar spinous processes and gently compress. The fascia is treated either indirectly or directly until a release is felt	Fracture, soft-tissue infection or abscess, aortic aneurysm, coagulopathy, malignancy
Lumbosacral MFR (pelvic diaphragm)	Patient is in a supine position. Place hands on the lumbosacral junction. The fascia is treated either indirectly or directly until the tissue releases	Fracture, soft-tissue infection or abscess, aortic aneurysm, coagulopathy, malignancy
Pelvic MFR	Patient is in a supine position. Place thumbs on either side of the pubic symphysis and apply gentle forces to indirectly or directly facilitate tissue release	Pelvic fracture, deep vein thrombosis, infection, open wound, osteoporosis
Doming of diaphragm	Patient sits up and leans forward while the physician stands behind and places their fingertips beneath the costal margins. Gentle upward pressure is applied during the patient's inhalation to release tension in the diaphragm	Rib fractures, diaphragmatic hernia, recent thoracic/ abdominal surgery
Pelvic diaphragm release (side-lying)	While the patient lies on their side, place fingers in the ischiorectal fossa to engage the pelvic diaphragm. As the patient breathes, the physician applies gentle upward pressure during exhalation to release fascial tension	Pelvic surgery, infection, hemorrhoids, deep vein thrombosis
Sacral rocking	Patient is in a prone position. Place one hand on the sacral base and the other reinforcing it, applying light pressure. A gentle rocking motion is synchronized with the patient's breathing to encourage extension during inhalation and flexion during exhalation	Sacral or pelvic fracture, open sacral wound, osteoporosis, coagulopathy, sacroiliac joint inflammation

Abbreviations: ASIS, anterior superior iliac spine; MET, muscle energy technique; MFR, myofascial release; PSIS, posterior superior iliac spine; TART, tissue texture changes, asymmetry, restriction of motion; tenderness

1. *Foundations of Osteopathic Medicine: Philosophy, Science, Clinical Applications, and Research*. 4th ed: Wolters Kluwer, 2018.

2. Nicholas AS, Nicholas, E.A. *Atlas of Osteopathic Techniques*. 4th ed: Wolters Kluwer, 2021.

3. DiGiovanna E.L. ACJ, Burns D.K. *An Osteopathic Approach to Diagnosis and Treatment*. 4th ed: Wolters Kluwer, 2020.

4. Chaitow L. DJW. *Clinical Application of Neuromuscular Techniques: Volume 2* – The Lower Body. 2nd ed. Edinburgh: Churchill Livingstone, 2008.

OMT is generally safe when performed mindfully and cautiously; however, physicians must be aware of certain contraindications. Absolute contraindications include compromised or friable tissue susceptible to injury.[30] Relative contraindications include dermatologic conditions (contagious skin diseases, acute burns, painful rashes, abscesses, cellulitis, skin cancers); acute inflammatory or traumatic soft tissue injuries (fasciitis, fascial tears, muscle strains, myositis, ligament sprains); infections and neoplasms affecting muscle, ligament, bone (osteomyelitis, bone tumors, osteoporosis), or joints (septic arthritis, joint instability, joint neoplasms); and visceral pathology (organ infections or neoplasms, organomegaly, gastrointestinal obstruction, acute abdominal or pelvic pain).[30] Vascular concerns such as hematomas, deep venous thrombosis, and coagulopathies also necessitate careful consideration.[30]

Biomechanical Model

Biomechanically, OMT can restore structural integrity, alignment, and functional mobility to correct functional alterations in posture and movement related to CPP. Examples include MET, which targets pelvic musculoskeletal restrictions, correcting somatic dysfunctions at the sacroiliac joints and pubic symphysis, thus reducing pelvic discomfort and biomechanical strain.[37] Soft tissue and MFR techniques can address the pelvic fascia, effectively reducing muscular tension, fascial restrictions, and enhancing local tissue mobility and healing.[35,38]

Respiratory Circulatory Model

The respiratory-circulatory model aims to maximize vascular and lymphatic function. Diaphragmatic release techniques applied to the abdominal diaphragm and pelvic diaphragm help enhance respiratory excursion, leading to better fluid and circulatory movement, contributing to overall pelvic homeostasis.[30,31,34,35] Techniques such as thoracic, abdominal, and pedal pumps, pelvic and mesenteric lift, and sacral rocking open pelvic lymphatic channels and vascular structures, improving venous return and lymphatic drainage. Enhanced venous and lymphatic circulation can reduce local and distal swelling and edema, boost immune responses, improve local tissue nutrition and oxygenation, and facilitate metabolic waste removal, thereby alleviating pelvic congestion and inflammation.[39]

Metabolic Nutritional Model

Metabolic-energy model techniques address inflammatory and metabolic components of pelvic pain. Balanced ligamentous tension (BLT) techniques can be utilized to balance pelvic ligamentous strains. This can promote energy efficiency and reduce strain-induced inflammation.[35] Furthermore, dietary and nutritional counseling complements these approaches by targeting systemic inflammation, optimizing metabolic processes, and facilitating tissue repair and healing. Recent studies highlight dietary interventions such as a low-FODMAP (fermentable oligosaccharides, disaccharides, monosaccharides, and polyols) diet, demonstrating efficacy in reducing endometriosis-related pelvic pain and gastrointestinal symptoms.[40,41] Additionally, supplementation with antioxidants such as vitamins C and E has been shown to significantly reduce pain scores in individuals with endometriosis.[42,43] Personalized healthy diet plans have been increasingly recognized for their potential to reduce inflammation, alleviate gastrointestinal symptoms, and improve quality of life in patients with CPP.[44] These interventions may help mitigate oxidative stress and inflammation contributing to pelvic pain.[43]

Neurologic Model

The neurologic model incorporates viscerosomatic and somatovisceral reflexes, facilitated spinal cord segments, Chapman's reflex points, and autonomic imbalance. Segmental facilitation at spinal levels corresponding to pelvic organs (T12–L2 for sympathetic, craniocervical junction, S2–S4 for parasympathetics) leads to heightened nociceptive input and altered autonomic tone, which can contribute to CPP. On physical exam, findings such as tissue texture changes and tenderness can contribute to pain, pelvic floor hypertonicity, and suggest altered visceral function. Chapman's points may reflect ongoing viscerosomatic activity associated with reproductive, urinary, or gastrointestinal dysfunctions.[39] The following are examples of osteopathic techniques that can help normalize neural input: counterstrain, occipitoatlantal decompression, MFR, paraspinal inhibition, sacral inhibition, sacral rocking, and suboccipital release.[34,45]

Biopsychosocial Model

The biopsychosocial model acknowledges behavioral, emotional, and psychosocial components that influence the perception of pain. In CPP, patients frequently present with comorbid anxiety, depression, trauma histories, and social stressors that exacerbate pain intensity and functional impairment.[46] OMT, integrated with mindfulness-based interventions, can increase patient awareness of tension patterns and stress responses, reducing pain perception and emotional distress. This approach is complemented by patient education, stress management techniques, and collaboration with behavioral health specialists to address psychological comorbidities, improving overall quality of life.[46] Cognitive behavioral therapy, mindfulness-based stress reduction, and trauma-informed care have also been demonstrated to reduce CPP severity and improve quality of life.[10,47] OMT can also modulate autonomic tone and reduce somatic manifestations of stress. Techniques such as compression of the fourth ventricle

(CV4), suboccipital release, rib raising, and thoracic inlet MFR may help rebalance the autonomic nervous system, reduce sympathetic overdrive, and support relaxation responses.[30,37]

ALLOPATHIC AND ADJUNCTIVE THERAPIES FOR CPP

Pharmacologic interventions for CPP commonly include analgesics such as nonsteroidal anti-inflammatory drugs (NSAIDs), acetaminophen, and opioids for acute flareups. Neuromodulatory treatments, including tricyclic antidepressants, serotonin norepinephrine reuptake inhibitors, selective serotonin reuptake inhibitors, and anticonvulsants (gabapentin, pregabalin), are increasingly being prescribed for neuropathic components of CPP.[10,27] Hormonal therapies, including oral contraceptives or gonadotropin-releasing hormone agonists, may be effective, especially when conditions such as endometriosis or dysmenorrhea contribute to CPP.[10,48]

Nonpharmacologic interventions include pelvic floor physical therapy, which focuses on muscular strengthening, relaxation, biofeedback, and manual therapy to alleviate myofascial pain and dysfunction.[10,49] Cognitive behavioral therapy is beneficial for managing associated psychological distress, anxiety, and depression commonly comorbid in CPP.[27,47] Acupuncture and yoga also demonstrate promise in reducing pain severity and improving quality of life.[50-52] Additionally, physical activity and exercise have been associated with improved quality of life and reduced pain symptoms in endometriosis, as well as enhanced mental health outcomes in individuals with CPP disorders.[53,54]

CONCLUSION

CPP remains a complex multifactorial condition that benefits from a personalized, multimodal treatment approach. By utilizing the five osteopathic models, clinicians can identify and treat structural, neurologic, circulatory, metabolic, and biopsychosocial contributors to CPP. While these models and the accompanying table of OMT offer a useful guide, it is not a substitute for thorough osteopathic evaluation and personalized treatment based on each patient's unique findings. OMT integrated with nonosteopathic therapies can maximize the efficacy of treatment for CPP.

REFERENCES

1. Tettambel MA. An osteopathic approach to treating women with chronic pelvic pain. *J Am Osteopath Assoc*. 2005;105(9 suppl 4):S20-S2.
2. Juganavar A, Joshi KS. Chronic pelvic pain: a comprehensive review. *Cureus*. 2022;14(10):e30691. doi: 10.7759/cureus.30691
3. Sillem M, Juhasz-Boss I, Klausmeier I, Mechsner S, Siedentopf F, Solomayer E. Osteopathy for endometriosis and chronic pelvic pain - a pilot atudy. *Geburtshilfe Frauenheilkd*. 2016;76(9):960-963. doi: 10.1055/s-0042-111010
4. Moloney S, Talsma J, Pierce-Talsma S. Osteopathic manipulative medicine considerations in pelvic pain. *J Am Osteopath Assoc*. 2019;119(11):e42-e43. doi: 10.7556/jaoa.2019.130
5. Barnett ME, Henderson KK, Elliott-Burke TL, Heinking KP. Osteopathic manipulative treatment of chronic pelvic pain due to high-tone pelvic floor dysfunction. *Osteopathic Family Physician*. 2023;15(2):12-18.
6. Alboni C, Melegari S, Camacho Mattos L, Farulla A. Effects of osteopathic manipulative therapy on recurrent pelvic pain and dyspareunia in women after surgery for endometriosis: a retrospective study. *Minerva Obstet Gynecol*. 2024;76(3):264-271. doi: 10.23736/S2724-606X.23.05351-4
7. Vincent K, Evans E. An update on the management of chronic pelvic pain in women. *Anaesthesia*. 2021;76(suppl 4):96-107. doi: 10.1111/anae.15421
8. Chronic pelvic pain: ACOG Practice Bulletin No. 218. *Obstet Gynecol*. 2020;135(3):e98-e109. doi: 10.1097/AOG.0000000000003716
9. Royal College of Obstetricians & Gynecologists. *The Initial Management of Chronic Pelvic Pain: Green-top Guideline No. 41*. May 2012. Accessed October 30, 2025. https://www.rcog.org.uk/media/muab2gj2/gtg_41.pdf
10. Speer LM, Mushkbar S, Erbele T. Chronic pelvic pain in women. *Am Fam Physician*. 2016;93(5):380-387.
11. Engeler AP, Baranowski B, Berghmans J, et al; European Association of Urology. *EAU Guidelines on Chronic Pelvic Pain*. 2025. Accessed October 30, 2025. https://d56bochluxqnz.cloudfront.net/documents/full-guideline/EAU-Guidelines-on-Chronic-Pelvic-Pain-2025.pdf
12. Lamvu G, Carrillo J, Ouyang C, Rapkin A. Chronic pelvic pain in women: a review. *JAMA*. 2021;325(23):2381-2391. doi: 10.1001/jama.2021.2631
13. Ayorinde AA, Macfarlane GJ, Saraswat L, Bhattacharya S. Chronic pelvic pain in women: an epidemiological perspective. *Womens Health (Lond)*. 2015;11(6):851-864. doi: 10.2217/whe.15.30
14. Andrews J, Yunker A, Reynolds WS, Likis FE, Sathe NA, Jerome RN. Noncyclic chronic pelvic pain therapies for women: comparative effectiveness. *AHRQ Comparative Effectiveness Reviews*. 2012;11(12):EHC088-EF.
15. Ahangari A. Prevalence of chronic pelvic pain among females: an updated review. *Pain Physician*. 2014;17(4):E141–E147.
16. Meisenheimer ES, Carnevale AM. Chronic pelvic pain in women: evaluation and treatment. *Am Fam Physician*. 2025;111(3):218-229.
17. Ayorinde AA, Bhattacharya S, Druce KL, Jones GT, Macfarlane GJ. Chronic pelvic pain in women of reproductive and post-reproductive age: a population-based study. *Eur J Pain*. 2017;21(3):445-455. doi: 10.1002/ejp.938
18. Arnold MJ, Osgood AT, Aust A. Chronic pelvic pain in women: ACOG updates recommendations. *Am Fam Physician*. 2021 Feb 1;103(3):186-188. Accessed October 30, 2025. https://www.aafp.org/pubs/afp/issues/2021/0201/p186.pdf
19. Stratton P, Berkley KJ. Chronic pelvic pain and endometriosis: translational evidence of the relationship and implications. *Hum Reprod Update*. 2011;17(3):327-346. doi: 10.1093/humupd/dmq050
20. McNamara HC, Frawley HC, Donoghue JF, et al. Peripheral, central, and cross sensitization in endometriosis-associated pain and comorbid pain syndromes. *Front Reprod Health*. 2021;3:729642. doi: 10.3389/frph.2021.729642

21. Sawsan A-S, Kim J, Schmidt-Wilcke T, et al. Functional connectivity is associated with altered brain chemistry in women with endometriosis-associated chronic pelvic pain. *J Pain*. 2016;17(1):1-13. doi: 10.1016/j.jpain.2015.09.008

22. Gyang A, Hartman M, Lamvu G. Musculoskeletal causes of chronic pelvic pain: what a gynecologist should know. *Obstet Gynecol*. 2013;121(3):645-650. doi: 10.1097/AOG.0b013e318283ffea

23. Latthe P, Mignini L, Gray R, Hills R, Khan K. Factors predisposing women to chronic pelvic pain: systematic review. *BMJ*. 2006;332(7544):749-755. doi: 10.1136/bmj.38748.697465.55

24. Wingenfeld K, Heim C, Schmidt I, Wagner D, Meinlschmidt G, Hellhammer DH. HPA axis reactivity and lymphocyte glucocorticoid sensitivity in fibromyalgia syndrome and chronic pelvic pain. *Psychosom Med*. 2008;70(1):65-72. doi: 10.1097/PSY.0b013e31815ff3ce

25. Gunter J. Chronic pelvic pain: an integrated approach to diagnosis and treatment. *Obstet Gynecol Surv*. 2003;58(9):615-623. doi: 10.1097/01.OGX.0000083225.90017.01

26. Steege JF, Siedhoff MT. Chronic pelvic pain. *Obstet Gynecol*. 2014;124(3):616-629. doi: 10.1097/AOG.0000000000000417

27. Fall M, Baranowski AP, Elneil S, et al. EAU guidelines on chronic pelvic pain. *Eur Urol*. 2010;57(1):35-48. doi: 10.1016/j.eururo.2009.08.020

28. Dydyk AS, Singh C, Gupta N. Chronic pelvic pain. StatPearls [Internet]. Accessed: October 30,2025. https://www.ncbi.nlm.nih.gov/books/NBK554585/

29. Pelvic Pain Society. Documents and forms: history and physical. Accessed October 30, 2025. https://www.ucsfhealth.org/-/media/project/ucsf/ucsf-health/pdf/IPPS_english.pdf

30. Seffinger MA. *Foundations of Osteopathic Medicine: Philosophy, Science, Clinical Applications, and Research*. 4th ed. Wolters Kluwer; 2019.

31. DiGiovanna E, Burns D. *An Osteopathic Approach to Diagnosis and Treatment*. 4th ed. Wolters Kluwer; 2020.

32. Bordoni B, Sugumar K, Leslie SW. *Anatomy, Abdomen and Pelvis, Pelvic Floor*. StatPearls [Internet]. 2023.

33. American Academy of Osteopathy. *American Academy of Osteopathy Position Paper: Recommended Guidelines for Pelvic Examination and Treatment*. 2017. Accessed October 30, 2025. https://www.academyofosteopathy.org/assets/docs/PelvicExaminationAndTreatment2.pdf

34. Shermon S, Docherty J, Yao S, Capobianco J. An osteopathic approach to diagnosing and treating perimenstrual disorders. *Osteopathic Family Physician*. 2019;11(4):32-40.

35. Nicholas AS, Nicholas, EA. *Atlas of Osteopathic Techniques*. 4th ed. Wolters Kluwer; 2021.

36. Chaitow L, Delany J. *Clinical Application of Neuromuscular Techniques: Volume 2 – The Lower Body*. 2nd ed. Churchill Livingstone; 2008.

37. Chila AG. Foundations of Osteopathic Medicine. 3rd ed. Lippincott Williams & Wilkins; 2010.

38. Fryer G. Somatic dysfunction: an osteopathic conundrum. *International Journal of Osteopathic Medicine*. 2016;22:52-63.

39. Kuchera WA, Kuchera, ML. *Osteopathic Considerations in Systemic Dysfunction*. 2nd ed. Greyden Press; 2012.

40. van Haaps AP, Wijbers JV, Schreurs AMF, et al. The effect of dietary interventions on pain and quality of life in women diagnosed with endometriosis: a prospective study with control group. *Hum Reprod*. 2023;38(12):2433-2446. doi: 10.1093/humrep/dead214

41. Varney JE, So D, Gibson PR, et al. Clinical Trial: Effect of a 28-Day Low FODMAP Diet on Gastrointestinal Symptoms associated with endometriosis (EndoFOD)-a randomised, controlled crossover feeding study. *Aliment Pharmacol Ther*. J2025;61(12):1889-1903. doi: 10.1111/apt.70161

42. Bayu P, Wibisono JJ. Vitamin C and E antioxidant supplementation may significantly reduce pain symptoms in endometriosis: a systematic review and meta-analysis of randomized controlled trials. *PLoS ONE*. 2024;19(5):e0301867. doi: 10.1371/journal.pone.0301867

43. Zheng SH, Chen XX, Chen Y, Wu ZC, Chen XQ, Li XL. Antioxidant vitamins supplementation reduce endometriosis related pelvic pain in humans: a systematic review and meta-analysis. *Reprod Biol Endocrinol*. 2023;21(1):79. doi: 10.1186/s12958-023-01126-1

44. Shorter B, Gordon B. Diet therapy in the context of chronic pelvic pain. In: Moldwin RM, ed. *Urological and Gynaecological Chronic Pelvic Pain: Current Therapies*. Springer; 2017.

45. Henley CE, Ivins D, Mills M, Wen FK, Benjamin BA. Osteopathic manipulative treatment and its relationship to autonomic nervous system activity as demonstrated by heart rate variability: a repeated measures study. *Osteopath Med Prim Care*. 2008;2:7. doi: 10.1186/1750-4732-2-7

46. Gatchel RJ, Peng YB, Peters ML, Fuchs PN, Turk DC. The biopsychosocial approach to chronic pain: scientific advances and future directions. *Psychol Bull*. 2007;133(4):581-624. doi: 10.1037/0033-2909.133.4.581

47. Urits I, Callan J, Moore WC, et al. Cognitive behavioral therapy for the treatment of chronic pelvic pain. *Best Pract Res Clin Anaesthesiol*. 2020;34(3):409-426. doi: 10.1016/j.bpa.2020.08.001

48. Vercellini P, Buggio L, Frattaruolo MP, Borghi A, Dridi D, Somigliana E. Medical treatment of endometriosis-related pain. *Best Pract Res Clin Obstet Gynaecol*. 2018;51:68-91. doi: 10.1016/j.bpobgyn.2018.01.015

49. FitzGerald MP, Kotarinos R. Rehabilitation of the short pelvic floor. I: Background and patient evaluation. *Int Urogynecol J Pelvic Floor Dysfunct*. 2003;14(4):261-268. doi: 10.1007/s00192-003-1049-0

50. Sung SH, Sung AD, Sung HK, An TE, Kim KH, Park JK. Acupuncture treatment for chronic pelvic pain in women: a systematic review and meta-snalysis of randomized controlled trials. *Evid Based Complement Alternat Med*. 2018;2018:9415897. doi: 10.1155/2018/9415897

51. Qin Z. Guo J, Chen H, Wu J. Acupuncture for chronic prostatitis/chronic pelvic pain syndrome: A GRADE-assessed systematic review and meta-analysis. *European Urology Open Science*. 2022;46:55-67. doi: 10.1016/j.euros.2022.10.005

52. Lin KY-H, Chang Y-C, Lu W-C, Kotha P, Chen Y-H, Tu C-H. Analgesic efficacy of acupuncture on chronic pelvic pain: a systemic review and meta-analysis study. *Healthcare (Basel)*. 2023;11(6)830. doi: 10.3390/healthcare11060830

53. Xie M, Qing X, Huang H, et al. The effectiveness and safety of physical activity and exercise on women with endometriosis: a systematic review and meta-analysis. *PLoS ONE*. 2025;20(2):e0317820. doi: 10.1371/journal.pone.0317820

54. Leventhal EL, Nukavarapu N, Elhadad N, et al. Trajectories of mHealth-tracked mental health and their predictors in female chronic pelvic pain disorders. *J Pain Res*. 2025;18:899-913. doi: 10.2147/JPR.S499102