

## REVIEW ARTICLE

# An Osteopathic Approach to Diagnosing and Treating Perimenstrual Disorders

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**ABSTRACT:** Premenstrual syndrome (PMS), premenstrual dysphoric disorder (PMDD), and dysmenorrhea are Perimenstrual disorders that cause significant physical and emotional distress to reproductive-aged women. The hormonal imbalance associated with perimenstrual disorders impacts multiple organs and somatic structures throughout the body. Many pharmacologic treatments are currently used to treat the various symptoms of perimenstrual disorders, however, these treatments can have a multitude of undesirable side effects. Osteopathic manipulative treatment (OMT) can be incorporated in the treatment of somatic and visceral components of PMS, PMDD, and dysmenorrhea. Osteopathic treatments can target multiple organs and structural components affected by these disorders holistically and with limited potential side effects to the patients. This article describes relevant OMT techniques, which encompass the five models of osteopathic medicine that can be used for specific perimenstrual symptoms.

## INTRODUCTION

Premenstrual Syndrome (PMS), premenstrual dysphoric disorder (PMDD), and dysmenorrhea are a spectrum of menstrual disorders that share pelvic pain as a symptom. PMS and PMDD both present with somatic and emotional symptoms that may last throughout the menstrual cycle. Common somatic symptoms found in each disorder include pelvic pain, abdominal bloating, breast tenderness, and edema. Emotional symptoms such as anxiety, depression, and irritability are also commonly present in both disorders. Unlike PMS and PMDD, dysmenorrhea usually presents only with somatic symptoms such as pelvic pain and only occurs during menstruation.<sup>1,2</sup>

PMS and PMDD have been associated with a reduced quality of life (QOL) in reproductive-age females. Women affected with

these disorders have increased costs, including ambulatory care visit costs, decreased productivity at work, and increased missed workdays. Women also report decreased interest in hobbies, impairment to social activities, and general impairment throughout the day while symptoms are present.<sup>3</sup> Another major concern is the association between major depressive disorder and PMS and PMDD.<sup>4</sup> Dysmenorrhea is suggested to be a leading cause of recurrent missed days at school and/or work in reproductive-age females and can be debilitating.<sup>5,6</sup> Over-the-counter (OTC) medication is of limited effectiveness, not to mention side effects of nonsteroidal anti-inflammatory drugs (NSAIDs) and acetaminophen including gastroesophageal reflux disease (GERD), stomach pain, increased blood pressure, fluid retention and liver injuries: This further supports incorporation of osteopathic treatment for perimenstrual dysfunctions.<sup>3,6,7</sup>

The authors believe osteopathic manipulative treatment (OMT) has a significant role in treating women with PMS, PMDD, and dysmenorrhea, and therefore, this article will focus on osteopathic findings in these disabling conditions, and the manual approach thereof.

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## EPIDEMIOLOGY

The prevalence of PMS is difficult to measure as the ICD-10 definition differs than the American College of Obstetricians and Gynecologists (ACOG), in addition to failure to apply strict criteria by evaluating severity or impairment to function. Studies have found the prevalence of PMS to be 3-98.6% depending on which diagnostic criteria is used, however, research using strict ACOG criteria have found the prevalence to be 3-8%.<sup>3,8,9</sup> Clearly, there are extraordinary differences between these percentages, which may call into question the validity of these statistics and the number of women who are not properly diagnosed. In the experience of these authors, perimenstrual disorders affect a majority of women. PMDD was only added to the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) in 2013 (previously a provisional diagnosis) with prevalence estimated to be 1-5%.<sup>3,10</sup> Studies measure the prevalence of dysmenorrhea to be much higher estimating 45-60%.<sup>11-13</sup>

## DIAGNOSTIC CRITERIA

The definition and diagnostic criteria of PMS have evolved over the years, but several key features have remained the cornerstone of diagnosis. These include:

- 1) The luteal timing of symptom onset, beginning after cycle day 13 and subsiding within 4 days after the onset of menses.
- 2) Symptoms occurring in at least 2 sequential cycles.<sup>7,14</sup>

ACOG sets the standard for diagnostic criteria of female reproductive disorders and diseases in the United States and they stipulate that an identifiable dysfunction in social, academic, or work performance must be present as well. Together with the above criteria, one of the affective or somatic symptoms in *Table 1* must be reported during the appropriate time and without any separate identifiable causes.<sup>1</sup>

**TABLE 1:**

Diagnostic criteria for premenstrual syndrome<sup>1</sup>

AFFECTIVE SYMPTOMS	SOMATIC SYMPTOMS
Angry outbursts	Abdominal bloating
Anxiety	Breast tenderness or swelling
Confusion	Headache
Depression	Joint or muscle pain
Increased irritability	Swelling of extremities
Social withdrawal	Weight gain

The diagnostic criteria for PMDD is set by the American Psychiatric Association (APA) in DSM-5 and has seven criteria that must be met (A-G). (A) Similar to PMS, the timing must be luteal with five symptoms presenting in the week before the onset of menses, improving within a few days after the onset of menses, and being minimal or absent in the week after menses. Symptoms must be

present in more than 50% of menstrual cycles. (B, C) A total of at least five symptoms must be present and contain at least one symptom from criterion B and at least one symptom from criterion C (*Table 2*). (D) The symptoms must cause significant distress and interference with regular activities (social, work, academic, or relationships). (E) Symptoms must not be an exacerbation of another disorder. (F) Symptoms should be confirmed by prospective daily ratings during at least two symptomatic cycles. (G) No other attributable cause can be found (medications, recreational drugs, other medical conditions).<sup>2</sup>

**TABLE 2:**

Diagnostic criteria for premenstrual dysphoric disorder<sup>2</sup>

CRITERION B	CRITERION C
Mood swings, sudden sadness, increased sensitivity	Difficulty concentrating
Anger, irritability	Diminished interest in usual activities
Sense of hopelessness, depressed mood, self-critical thoughts	Easy fatigability, decreased energy
Tension, anxiety, feeling on edge	Marked changes in appetite
	Sense of being overwhelmed
	Changes in sleep
	Physical symptoms, such as those seen in PMS

Dysmenorrhea diagnosis is made clinically in patients with recurrent, crampy, midline, pelvic pain that starts just before or with the onset of menses and then diminishes over 12-72 hours with no other attributable cause.<sup>15</sup>

## PATHOGENESIS

The exact pathophysiology of PMS and PMDD is still unknown and under investigation, however, multiple neurotransmitters and hormones have been implicated in playing a role. The latest theories suggest the serum level changes of estrogen and progesterone are the root causes of PMS and PMDD. One study showed that patients with PMS who received leuprolide, which decreases estrogen and progesterone levels, had decreased symptoms. These symptoms returned when estrogen or progesterone therapy was introduced. Contrastingly, women without PMS who received the same interventions had no response to treatment.<sup>16</sup> This evidence suggests that imbalances in both estrogen and progesterone are involved in the disease process, further suggesting an abnormal response to these hormones with downstream players involved.

Multiple neurotransmitters have been identified as having a possible role in the PMS and PMDD disease process, including gamma-Aminobutyric acid (GABA), opioids, and serotonin. The role of GABA needs additional research; however, benzodiazepines, which agonize the GABA receptor, have shown benefit to patients afflicted with PMS.<sup>17</sup> Opioids also have limited research, but several studies have found reduced peripheral beta-endorphin levels in

patients with PMS and PMDD.<sup>18-20</sup> Serotonin has been found to play a significant role in PMS and PMDD. Cerebrospinal fluid (CSF) serotonin levels were estimated to be negatively correlated with serum estradiol, progesterone, and testosterone. CSF serotonin levels were also estimated to be decreased in patients with PMS.<sup>21</sup> These findings are supported by the effectiveness of selective serotonin reuptake inhibitors (SSRIs) in reducing symptoms of PMS and PMDD.<sup>22,23</sup> Furthermore, tryptophan (serotonin precursor) restriction worsens PMS symptoms and the serotonin antagonist, metergoline, causes a return of symptoms in patients previously effectively treated with SSRIs.<sup>24,25</sup> Altogether, this evidence strongly supports serotonin as a downstream effector of PMS and PMDD.

In contrast to PMS and PMDD, the etiology of dysmenorrhea is less complex. Increased serum prostanoids during menses cause frequent, irregular contractions of the uterus. These contractions increase the uterine pressure, which may overcome arterial pressure to the organ, leading to ischemia and pain.<sup>15</sup> The pathogenesis and symptoms of PMS, PMDD, and dysmenorrhea affect all the osteopathic models, including the biomechanical, respiratory-circulatory, neurological, metabolic-nutritional, and biopsychosocial models. Osteopathic structural exam (OSE) is a key component in revealing areas of somatic dysfunctions that can be addressed by osteopathic manipulative techniques (OMT).

## **HORMONAL AND ORGAN SYSTEM CONSIDERATION IN PREMENSTRUAL SYNDROME AND PREMENSTRUAL DYSMORPHIC DISORDER (PMS/PMDD)**

Multiple organs and structures contribute to the pain and symptoms associated with PMS and PMDD. Organs and structures that play a significant role in these disorders and that require special attention when performing the OSE and OMT are as follows: thyroid, ovaries, uterus, broad ligaments, kidneys, pelvic lymphatic glands, kidneys, adrenals, breasts, sacroiliac area, and the occipital-atlantal (OA) joint.

The thyroid contains sex hormone receptors for estrogen, progesterone, and testosterone.<sup>26</sup> These hormones influence thyroid stimulating hormone (TSH) production.<sup>27</sup> Prior studies have found that both estrogen and progesterone are imbalanced in a patient with PMS, which in response may alter TSH levels.<sup>28</sup> Borderline thyroid function with TSH in the middle to high range and active T3 in the low normal to below normal range have been associated with increased PMS symptoms.<sup>29</sup> Thyroid disease has often been found to lead to menstrual disturbances, reduced fertility, and pelvic pain. Hyperthyroidism is associated mainly with hypomenorrhea and polymenorrhea, whereas hypothyroidism is associated mainly with oligomenorrhea and menorrhagia.<sup>28</sup> For these reasons, a proper history with deviations of normal reproductive gland activity should follow-up with a thyroid workup.

The ovaries are potentially the most vital organs when considering PMS and PMDD due to their role in estrogen and progesterone production.<sup>30</sup> Abnormal production of both hormones has been

found in women with PMS.<sup>31,32</sup> Estrogen has been found to increase baroreflex regulation of sympathetic outflow, increasing norepinephrine activity and can lead to vasoconstriction of blood flow to major pelvic organs thereafter.<sup>33</sup> Vasoconstriction and the resulting ischemia can result in lower thresholds in nociception. In a patient with PMS, it is important to check luteinizing hormone (LH) and follicular stimulating hormone (FSH), estrogen, and progesterone levels to rule out hormonal imbalance.

The effects of imbalanced estrogen also extend to the uterus. Many women with dysmenorrhea and chronic pelvic pain have been found to have uterine fibroids. Estrogen has proliferating effects on fibroids, which may lead to pelvic pain or pressure, heavy menstrual bleeding, and in rare cases, reproductive dysfunction.<sup>34</sup> These fibroids may interfere with uterine contractions and restrict uterine blood vessels, resulting in ischemia, pain, and excessive menstrual bleeding. Heavy menstrual bleeding can still occur in women with PMS who do not have fibroids. Estrogen controls the amount of uterine shedding during the menstrual period. Abnormal estrogen may lead to increased endometrial thickness that leads to increased sloughing and therefore bleeding during menses.<sup>30</sup> Women with PMS are recommended to receive abdominal ultrasounds to rule out uterine fibroids.

The broad ligament is thick mesentery that encapsulates the uterus, ovaries, and fallopian tubes in the pelvis.<sup>35</sup> As mentioned previously, the organs that the broad ligament surrounds contribute to the symptoms of PMS and PMDD. Treating the broad ligament osteopathically to reduce some of its tension may increase blood flow, allowing more oxygen to reach these organs, reducing some of the pain caused by ischemia in PMS. The Chapman's reflex for the broad ligament to enhance lymphatic flow is located on the outer thigh, along the iliotibial band.<sup>36</sup>

The kidneys can contribute to PMS/PMDD pain and bloating via fluid retention through the renin-angiotensin-aldosterone-system (RAAS). During the luteal phase, when estrogen is at its peak, RAAS components also rise.<sup>37</sup> Increased RAAS components lead to vasoconstriction and fluid retention, increasing pelvic pressure and leading to pain.<sup>30</sup> The water retention caused by the elevated RAAS components may also lead to bloating, a common symptom found in women with PMS/PMDD.<sup>14</sup> Renin and angiotensin II levels should be assessed in women suffering from PMS/PMDD who present with bloating or edema. The chemical axis of the RAAS not only involves the kidneys and adrenals, but also the liver and lungs, supporting the osteopathic principal of body unity.

The adrenal glands, which are small glands that produce stress hormones such as cortisol and aldosterone, are also impacted by the imbalanced estrogen levels in PMS.<sup>30</sup> Replacement estrogen therapy leads to a rise in the cortisol levels produced by the adrenal glands, leading to hypertension, suppressed immunity, hyperglycemia, and carbohydrate cravings.<sup>38</sup> Abnormal estrogen and progesterone levels can also result in a rise in aldosterone levels produced by the adrenal glands, leading to salt and water retention, bloating, and possibly edema.<sup>39</sup> Further workup of cortisol and aldosterone may be needed if women who suffer from PMS have alterations in electrolytes such as sodium and potassium or corresponding adrenal symptoms such as hirsutism.

Breast tenderness is a common symptom of PMS/PMDD. Hormonal imbalance may contribute to breast fullness and tenderness.<sup>14</sup> OMT addressing lymphatic drainage and associated chapman points may decrease the severity of this symptom.

### OSTEOPATHIC STRUCTURAL EXAM AND PHYSICAL EXAM FOR THE PATIENT SUFFERING FROM PREMENSTRUAL SYNDROME AND PREMENSTRUAL DYSMORPHIC DISORDER (PMS/PMDD)

Patients with PMS, PMDD, or dysmenorrhea should have a complete and thorough osteopathic structural exam (OSE) and physical exam prior to initiating OMT. On OSE, women diagnosed with any of these conditions may demonstrate abnormalities with their biomechanics, circulation, lymphatics, and autonomies.

Biomechanically, physicians should examine the abdomen, lumbar spine, pelvis, sacrum, and paraspinal musculature for somatic dysfunctions. A gentle palpatory evaluation of the abdomen should be performed to reproduce and localize any pain. Abdominal evaluation can also reveal specific pathology such as uterine or adnexal enlargement or nodularity. The innominates are another important region for osteopathic consideration as it has significant pelvic fascia connections and houses the organs most severely affected by PMS, PMDD, and dysmenorrhea. Additionally, the pelvis is the source of most sex hormones, which, as discussed above, interact with the major glands of stress and exacerbate symptoms. Fascial connections and autonomies also extend to and from the lumbar spine and the sacrum. On OSE, lumbar spinal curves, paravertebral muscle contractions, and sacral base leveling should be assessed for somatic dysfunctions. Abnormal lumbar spine curvature or lumbar paravertebral contraction can reveal whether the dysfunction is acute or chronic, the level of neuromuscular hyperactivity, and which organs are affected based on viscerosomatic reflexes and chapman points. An unlevel sacral base can be indicative of abnormal autonomies.<sup>40</sup>

The parasympathetic nervous system and inflammatory response play a role in PMS. It has been demonstrated that women with PMS had elevated inflammation markers that correlate with the severity of symptoms.<sup>41</sup> The vagus nerve inhibits inflammation via the cholinergic anti-inflammatory pathway (CAP) by interacting with the alpha7 subunit found on the surfaces of macrophages.<sup>42</sup> In addition, the activation of the parasympathetic nervous system leads to vasodilation to pelvic organs, increasing oxygen perfusion, and thereby decreasing ischemia-induced pain. The parasympathetic pathways affecting the abdominopelvic organs, specifically the ovaries, proximal fallopian tubes, kidneys and upper ureters, arise from the vagus nerve. The vagus nerve leaves the cranium through the jugular foramen, accessible to the hands of the operator in the occipital, temporal, occipitoatlantal (OA), and suboccipital regions. Splanchnic nerves from the sacrum innervate the lower uterus and inferior reproductive system.<sup>29</sup> Therefore, diagnosing and treating the jugular foramen and sacral junctional and diaphragmatic areas is vital in women with PMS.

Viscerosomatic reflexes are localized responses found on somatic structures that originated from visceral organ stimulation. The spine and surrounding tissues are common somatic sites to find visceral reflexes. The following spinal regions are important to evaluate during the osteopathic structural exam: T1-4 for reflexes from the thyroid and thoracolumbar region for sympathetic reflexes from abdominopelvic organs. Parasympathetic reflexes for the lower abdominal and pelvic organs are located from S2-S4 and are important to assess during the osteopathic structural examination (OSE) because the pelvic and abdominal organ reflexes are found there.<sup>43</sup> Details pertaining to each organ contributing to PMS can be found in *Table 3*.<sup>36,44,45</sup>

**TABLE 3:**

Osteopathic structural exam autonomic considerations<sup>36,45</sup>

AREA TREATED	VISCEROSOMATIC REFLEXES	CHAPMAN POINTS
Thyroid	T1-T5 bilaterally	Anterior: 2nd ICS Posterior: T2 transverse processes
Ovaries	Sympathetic: T10-T11 bilaterally Parasympathetic: S2-S4	Anterior: Superior and inferior to the pubic symphysis. Posterior: Between T9 and T10, and between T10 and T11.
Uterus	Sympathetic: T9-L2 bilaterally Parasympathetic: S2-S4	Anterior: Between pubic symphysis and obturator foramen. Posterior: Between spinous process of L5 and PSIS.
Broad Ligament	Sympathetic: T9-L2 bilaterally Parasympathetic: S2-S4	Laterally, along the ITB, from the greater trochanter to just superior to the lateral knee along the femoral condyle.
Pelvic (lymphatic) Glands	Sympathetic: T10-L2 bilaterally Parasympathetic: S2-4	Anterior: Along the lower 2/5th of the sartorius muscle (inner thigh) into the proximal, medial tibia. Posterior: At the iliosacral joint, near the PIIS (S3)
Kidneys	Sympathetic: T9-L1 ipsilaterally Parasympathetic: S2-S4	Anterior: 1-inch lateral and one inch superior to umbilicus. Posterior: Between T12-L1
Adrenals	Sympathetic: T8-T10 ipsilaterally Parasympathetic: S2-S4	Anterior: 1-inch lateral to and 2.5 inches superior to the umbilicus. Posterior: Between T11 and T12.
Breast	-----	Posterior: Angles of ribs 5 and 6

Abbreviations- ICS- intercostal space, PSIS- Posterior Superior Ischial Tuberosity, ITB- Iliotibial band, CPP- chronic pelvic pain

Chapman points are small gangliform, myofascial contractions that result in tissue texture changes found just underneath the skin in the fascia, muscle, periosteum, and bone. Posteriorly, these reflexes are found along the short restrictor muscles of the spine and are traditionally considered “stringy” in texture. They are a type of viscerosomatic reflex mediated by the sympathetic nervous system. Based on their location, these points are diagnostic for specific visceral organ problems. The innominate must be treated before directly treating Chapman points to ensure a freeing of the sympathetic trunk termini, named the ganglion impar, which is anterior to the sacrococcygeal joint. Chapman points are treated by applying gentle rotary motion with the endpoint being a reduction of fluid due to lymphatic edema anteriorly and relaxation of tissue texture changes in the muscle in the myofascial of the spinal region. The specific anterior and posterior points found in PMS/PMDD can be found in *Table 3*.<sup>36,44,45</sup>

Women with PMS, PMDD, or dysmenorrhea tend to have stasis in blood and lymph flow, which may lead to ischemic pain and edema, as mentioned in the previous sections.<sup>15,40</sup> Zink’s patterns should be taken into account and the tentorium cerebelli, thoracic outlet, abdominal diaphragm, and pelvic diaphragm should be examined for rigidity and decreased movement. These diaphragms play a vital role in circulating blood and lymph throughout the body, meaning that stasis in any of these will lead to circulatory/lymphatic stasis. The attachment sites of these diaphragms are also important to examine as decreased motility in these sites will lead to diaphragmatic stasis. Therefore, somatic dysfunction of the cranium, spine, ribs, clavicle, and innominates should be evaluated and treated to optimize diaphragmatic motion.<sup>40</sup>

The OSE will not only aid the physician in diagnosing the patient suffering from a perimenstrual disorder, but will also guide them in which treatments the patient would most benefit from based on the corresponding somatic dysfunction.

## THE FIVE OSTEOPATHIC MODELS AND OMT TECHNIQUES FOR TREATING PMS, PMDD, AND DYSMENORRHEA

OMT targets specific organs and structures involved in PMS, PMDD, and dysmenorrhea with limited potential side effects making it a useful treatment compared to pharmacologic therapy. OMT and manual therapy have been effective in treating symptoms of PMS and PMDD.<sup>46-48</sup> Specifically manual therapy has shown to decrease PMS/PMDD symptoms such as fatigue, bloating, and pain.<sup>48</sup> One systemic review concluded OMT to effectively decrease dysmenorrhea pain intensity and duration.<sup>47</sup> In addition to viscerosomatic reflexes from certain organs, chronic pelvic pain can result directly either from either structural or emotional changes, both in which OMT can be useful. Muscle imbalance and posture can affect the structure, and thus, the functioning of pelvic organs. Poor pelvic and lower extremity muscle tone may increase lumbar lordosis and exaggerate anterior pelvic tilt, with resultant crowding of viscera into the pelvic bowl. Repeated alternation of muscle tension and relaxation may lead to nerve entrapment or alteration of blood and lymph circulation to muscles or other

body structures. Techniques that decrease muscle tone, improve lumbar lordosis, and increase lymphatic flow can improve these direct structural changes that lead to pelvic pain.<sup>49</sup>

In osteopathic manipulative treatment, there are five main models that osteopathic physicians adhere to: The Respiratory-Circulatory, Neurologic, Mechanical, Metabolic-Nutritional, and Biopsychosocial. The respiratory-circulatory model addresses respiratory and fluid mechanics in the body such as congestive changes, lymphatic flow, venous return, and edema formation. Treatment goals include restoring the body’s ability to improve respiratory excursion, and thus, lymph and circulatory flow. PMS symptoms that can be addressed with this model include bloating, weight gain, pelvic and thoracic diaphragm pain, salt and water retention, breast tenderness/fullness, inflammation, and lymphatic stasis. The following OMT techniques address this model and can improve the resulting symptoms: pelvic diaphragm release, sacral rocking, ischial tuberosity spread, doming of the thoracic diaphragm, pedal pump, Marian Clark release, assessing and addressing Zink’s patterns to decrease restrictions of the before-mentioned diaphragms. Treatment of the diaphragms can help to maintain appropriate pressures- such as negative intrathoracic- for better fluid movement.<sup>29,50,51</sup>

The Neurologic model addresses facilitated spinal cord segments, viscerosomatic and somatovisceral reflex phenomena, Chapman points, and abnormal parasympathetic effects resulting from cranial or sacral nerve entrapment syndromes. Treatment goals include restoring autonomic balance, alleviation of segmental facilitation, decreasing abnormal afferent signaling, and relief of pain. PMS and PMDD symptoms incorporated in this model include viscerosomatic reflexes to the spine (cervical, thoracic, and low back tenderness), Chapman sympathetic points, OA (vagus nerve) and sacral (splanchnic nerves) somatic dysfunctions that also commonly lead to autonomic para/sympathetic abnormalities. OMT techniques that can be used to resolve these symptoms include lumbosacral decompression, sacral rocking, ischial tuberosity spread, quadratus lumborum release, inhibitory sacral pressure, and suboccipital release. Treating Chapman points in the left 6th intercostal space, on T6-T7, and on the iliotibial band<sup>58</sup> to mobilize stasis of the upper and lower gastrointestinal tract can facilitate better gut motility, which should lead to less bloating.<sup>29,50-52</sup> Owen’s An Endocrine Interpretation of Chapman’s Reflexes discusses treating the ovarian reflex points during the LH surge in the middle of the menstrual cycle, which may alleviate perimenstrual discomfort.<sup>45</sup>

The Mechanical model addresses factors that alter posture, motion, and gait. The goal of treatment is the restoration of free motion within the musculoskeletal system. Cervical, thoracic, and lumbar pain from viscerosomatic reflexes, tender points found in the lumbar and pelvic regions, increased muscle tension, diaphragm restrictions, and lumbar lordosis are common complaints associated with PMS, PMDD, and dysmenorrhea that fall under this model. OMT techniques that can be used to treatment musculoskeletal restrictions include lumbosacral decompression, counterstrain for lumbar, sacral and pelvic tenderpoints, sacral rocking, ischial tuberosity spread, quadratus

lumborum release, inhibitory sacral pressure, myofascial release, and muscle energy.<sup>29,50,51</sup>

The Metabolic-Nutritional model addresses dietary deficiencies and excesses, food allergies, and effect of toxins. Treatment goals include promoting energy conservation/exchange and immune system enhancement. The Metabolic-Nutritional model encompasses many symptoms of PMS and PMDD, such as appetite, food cravings, overeating, fatigability, decreased energy, and increased inflammatory state. Nutritional counseling, exercise encouragement, and OMT techniques directed at somatic dysfunctions including compression of the fourth ventricle, can improve the cranial rhythmic impulse (CRI) and decrease musculoskeletal restriction that increase allostatic load and therefore energy expenditure and can thus help to address PMS and PMDD symptoms. The importance of utilization of muscular activity to modulate blood sugar levels and insulin sensitivity is also a bioenergetics consideration with OMT.<sup>29,50,51</sup>

The Biopsychosocial model addresses the psychological and social components of a patient's health, as stress is a well-known contributor to illness. Treatment goals include optimizing psychological and social components of a patient's health. Mood swings, anger, irritability, tension, anxiety, difficulty concentrating, diminished interest in usual activity, feeling overwhelmed, and sleep disturbances are all PMS and PMDD symptoms that fall under this model. Treatments can include teaching the patient stress reduction strategies, helping the patient improve his or her social interactions, and OMT including CV4 (compression of the fourth ventricle), and suboccipital release. All treatments are described in *Table 4*.<sup>29,50,51</sup>

### Informed Consent

While OMT is a useful treatment for PMS, PMDD, and chronic pelvic pain, informed consent must always be obtained from the patient prior to starting treatment. PMS/PMDD is a condition of that mostly involves organs of sexuality. As with all informed consent, patients must be educated on what techniques they will receive in their treatment sessions, why these techniques are important for addressing their chief complaints, how each technique is performed, and what side effects can be expected after the treatment session is completed. The physician must also inform the patient of any contraindications to the technique (*listed in Table 4*) to assess whether this technique may bring harm to the patient. Lack of informed consent may be detrimental to both patient and physician and the professional relationship between both parties; an uncomfortable patient may tense up amid treatment, which may worsen, rather than alleviate, the original complaint. Finally, each patient should have the right to decline a technique that they are uneasy with and can only do so after being properly informed about it. In the opinion of this author, a choice for a chaperone should be available to the patient. It is the duty of the osteopathic physician to educate patients on treatment goals and OMT: This is best accomplished with a transparent patient-physician relationship.

### Non-Osteopathic Treatments for PMS and PMDD- Pharmacologic and Non-Pharmacologic

The main treatment goals in pharmacologic therapy for PMS/PMDD include suppression of ovulation, suppression of physical symptoms, suppression of psychological symptoms, and potential surgery for intractable PMS/PMDD. For managing PMS/PMDD symptoms anti-depressants are often prescribed. Selective serotonin reuptake inhibitors (SSRIs) are the first line treatment, of which escitalopram seems to be consensus amongst the authors.<sup>53</sup> Serotonin and norepinephrine reuptake inhibitors (SNRIs) are also prescribed because action is quick and often found to be helpful. Sexual dysfunction, fatigue, and weight gain are the most common side effects associated with SSRIs and SNRIs.<sup>54</sup>

Various birth controls can be used including oral contraceptives, progestin only contraceptives, oral micronized progesterone, medroxyprogesterone, transdermal estradiol, and gonadotropin releasing hormone (GnRH) agonists to treat PMS/PMDD symptoms.<sup>1,54</sup> These medications take advantage of the hypothalamic-pituitary-gonadal axis to prevent ovulation from occurring. Despite their effectiveness, oral contraceptives have side effects, including weight gain, nausea, headache, breast tenderness, irritability, depression, vaginal dryness, and the potential for cancer and blood clots.<sup>55</sup>

For suppression of physical symptoms, spironolactone can be used.<sup>56</sup> Spironolactone is an aldosterone-antagonizing diuretic that can be used for water retention and increased aldosterone, both symptoms found in PMS.<sup>54</sup> Metorrhagia, gynecomastia, urticarial, and scalp hair loss are side effects of spironolactone.<sup>56</sup>

For intractable PMS and PMDD, bilateral oophorectomy with estrogen replacement post-surgery is recommended.<sup>54</sup> While all the aforementioned medications are useful in managing PMS, they have unwanted side effects. For this reason, OMT is a very useful treatment because not only can it achieve reductions in symptoms, but it does so with very few, temporary side effects.<sup>29,46,50</sup>

Non-pharmacologic therapy for PMS should address psychosocial and nutritional difficulties that are often present in PMS and PMDD. For psychosocial problems, physicians should educate patients on stress management and coping mechanisms. Physicians should discuss with their patients time management and realistic goals for incorporating exercise and adequate rest and sleep into their lifestyles. Encouraging patients to maintain a diet with adequate amounts of protein, fiber, and carbohydrates, but with low fat, can help promote energy and decrease water retention. Avoiding foods that are high in salt and sugar may also decrease water retention, which will lessen physical discomfort from bloating and edema. Iodine supplementation can be considered for those who experience breast tenderness, as this element prevents the formation of estrone (E1), which is one of the three molecules of estrogen that contribute to mastalgia.<sup>57</sup> Women with perimenstrual disorders should also avoid alcohol and illicit drugs as they often worsen emotional lability.<sup>54</sup>

TABLE 4:

OMT treatments for pelvic pain<sup>29,50,51</sup>

TECHNIQUE	BASIC STEPS	CONTRAINDICATIONS	OSTEOPATHIC MODEL
Pelvic Diaphragm Release	Can be performed in lateral, prone, or supine position. The physician's fingers are placed medial to the patient's ischial tuberosity. Patient then inhales and exhales slowly. With patient exhalation, the physician advances fingers cephalad.	Few contraindications but consider patient positioning.	Respiratory- Circulatory Metabolic-Nutritional
Lumbosacral Decompression	Patient is in lateral position. Place fingers of one hand on sacral base and the other on L5. Traction to separate fingers and gap the lumbosacral junction.	<ul style="list-style-type: none"> <li>Localized and unstable spinal fractures</li> </ul>	Mechanical Neurologic
Anterior Counterstrain Points (Pelvis)	Indirect technique. All treatments need to be placed into proper position and held for 90 seconds or until a change in at least two or three of the TART changes are palpated by the physician <ol style="list-style-type: none"> <li>Psoas-2/3 distance from ASIS to midline</li> <li>Iliacus-1/3 distance from ASIS to midline</li> <li>Low Ilium- Superior surface of iliopectineal eminence</li> <li>Inguinal- Lateral aspect of pubic tubercle.</li> </ol>	Relative Contraindications: <ul style="list-style-type: none"> <li>Cannot voluntarily relax.</li> <li>Cannot discern pain with positional change.</li> <li>Cannot understand instructions.</li> <li>Connective tissue disease (arthritis, Parkinson's)</li> </ul> Absolute Contraindications: <ul style="list-style-type: none"> <li>Severely strained tissue.</li> <li>Instability of treated area.</li> <li>Vascular or neurologic disease.</li> <li>Degenerative spondylosis.</li> </ul>	Mechanical
Sacral Rocking	Patient is prone and physician should cup hands over sacrum. Gentle rocking into nutation and counter-nutation is performed.	<ul style="list-style-type: none"> <li>Pilonidal cyst</li> </ul>	Mechanical Respiratory-circulatory Neurologic Metabolic-Nutritional
Ischial Tuberosity Spread	Patient is prone with the upper body elevated on the elbows and knees flexed. Place thumbs medial to the ischial tuberosities. Have the patient exhale slowly and apply lateral pressure. Repeat three times.	<ul style="list-style-type: none"> <li>Fractures</li> <li>Cannot tolerate treatment position</li> </ul>	Mechanical Respiratory-circulatory Neurologic
Doming of the diaphragm	Fingers are placed under xyphoid process pointing cephalad and posterior. Patient slowly inhales and exhales. Fingers are advanced cephalad with exhalation.	<ul style="list-style-type: none"> <li>Rib fractures</li> </ul>	Mechanical Respiratory-circulatory Metabolic-nutritional
Quadratus lumborum release	Have patient lie on affected side. Lift both feet up to the ceiling. Ask patient to push down to the floor while applying isometric resistance. Re-engage the barrier three times while repeating the procedure.	Few contraindications	Mechanical Neurologic
Pedal Pump	Patient is supine and physician places hands on plantar aspect of feet and moves them rhythmically into dorsiflexion and plantar flexion as if sloshing saline in one total body biologic system. At first physician initiates bodily movement but then the fluid begins to move the physician's hands. The repeated dorsiflexion/plantar flexion should be at about 120 movements per minute. The end point of a true lymphatic pedal pump is that there is no resistance.	Contraindications: <ul style="list-style-type: none"> <li>Venous thrombosis</li> <li>Ankle Sprain</li> <li>Achilles strain</li> <li>Post-surgical patient</li> </ul>	Respiratory-circulatory
Marian Clark Release	Patient is semiprone on all fours with back arched. Physician places fingerpads medial to the patient's ASIS bilaterally. The physician then pulls hands cephalad and tractions abdomen upwards repeatedly.	Pre-existing condition that prevents her from attaining the treatment position.	Respiratory-circulatory
Inhibitory Sacral Pressure	Patient is prone as the physician maintains a steady pressure on the base of the sacrum until pain is relieved. The same may be done at the thoracolumbar region.	Fractures in surrounding area.	Mechanical Neurologic
Suboccipital Release	Patient is supine. Put fingers onto patient's suboccipital area. Apply gentle lateral and superior traction by pushing elbows together and tractioning backwards.	Few contraindications	Neurologic Biopsychosocial
Myofascial C1, C2, L1-5	Direct or Indirect Technique; Doctor places enough force to contact the patient's cervical fascia. The doctor then moves the fascia into either its restriction or ease and holds it in place for 20-60 seconds or until a release is felt.	Relative Contraindications: <ul style="list-style-type: none"> <li>Acute sprain</li> <li>Fracture</li> <li>Neurologic or vascular compromise</li> <li>Osteoporosis or osteopenia.</li> <li>Malignancy</li> <li>Infection</li> </ul> Absolute Contraindications: <ul style="list-style-type: none"> <li>None</li> </ul>	Mechanical
Muscle Energy C1, C2	Direct technique; Patient is supine and physician brings neck to the edge of the restrictive barrier. The patient is then asked to move neck towards his or her direction of freedom while the physician applies an isometric force for 3-5 seconds. The patient then relaxes for 3-5 seconds and the patient is brought further into his or her restrictive barrier. Repeat 3 times and perform a passive stretch into the restrictive barrier at the end of treatment.	Relative Contraindications: <ul style="list-style-type: none"> <li>Severe muscle strain</li> <li>Osteoporosis</li> <li>Post-surgical or ICU patient</li> </ul> Absolute Contraindications: <ul style="list-style-type: none"> <li>Fracture</li> <li>Dislocation</li> <li>Joint Instability</li> <li>Lack of cooperation from patient.</li> </ul>	Mechanical
Chapman Reflexes	Firm and gentle rotary motion on the point until the localized swelling is smoothed out or stringy muscles are relaxed.	Few contraindications	Mechanical Neurologic

## CONCLUSION

The effects of the altered hormonal state in PMS and PMDD is vast and the inflammation pathways of menstruation can be excruciating. These effects extend to not only the soma but also to multiple organs including the thyroid, ovaries, uterus, kidneys, pelvic lymphatic glands, kidneys, adrenals, and breasts. Perimenstrual disorders result in a reduced quality of life for women suffering from them, having impact on their jobs, education, relationships, emotional state, and well-being. OMT can be useful for these patients because of the holistic approach to treating these distinct organs and the limited side effects that can be associated with pharmaceutical medications. OMT can be considered as an adjunctive means of treating perimenstrual disorders and improving the physical and emotional state of the women suffering from them.

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## REFERENCES:

- Guidelines for Women's Health Care: A Resource Manual. (American College of Obstetricians and Gynecologists, 2014).
- American Psychiatric Association. Diagnostic and statistical manual of mental disorders. (American Psychiatric Publishing, 2013).
- Pearlstein, T. & Steiner, M. Premenstrual dysphoric disorder: burden of illness and treatment update. *J. Psychiatry Neurosci.* JPN 33, 291–301 (2008).
- Padhy, S. K. et al. Relationship of premenstrual syndrome and premenstrual dysphoric disorder with major depression: relevance to clinical practice. *Indian J. Psychol. Med.* 37, 159–164 (2015).
- Kannan, P., Chapple, C. M., Miller, D., Claydon, L. S. & Baxter, G. D. Menstrual pain and quality of life in women with primary dysmenorrhea: Rationale, design, and interventions of a randomized controlled trial of effects of a treadmill-based exercise intervention. *Contemp. Clin. Trials* 42, 81–89 (2015).
- Tanaka, E. et al. Burden of menstrual symptoms in Japanese women – an analysis of medical care-seeking behavior from a survey-based study. *Int. J. Womens Health* 6, 11–23 (2013).
- Hofmeister, S. & Bodden, S. Premenstrual Syndrome and Premenstrual Dysphoric Disorder. *Am. Fam. Physician* 94, 236–240 (2016).
- Akiyama, S., Tanaka, E., Cristeau, O., Onishi, Y. & Osuga, Y. Evaluation of the treatment patterns and economic burden of dysmenorrhea in Japanese women, using a claims database. *Clin. Outcomes Res. CEOR* 9, 295–306 (2017).
- Dennerstein, L., Lehert, P. & Heinemann, K. Epidemiology of premenstrual symptoms and disorders. *Menopause Int.* 18, 48–51 (2012).
- Pilver, C. E., Kasl, S., Desai, R. & Levy, B. R. Health advantage for black women: patterns in pre-menstrual dysphoric disorder. *Psychol. Med.* 41, 1741–1750 (2011).
- Burnett, M. A. et al. Prevalence of primary dysmenorrhea in Canada. *J. Obstet. Gynaecol. Can. JOGC J. Obstet. Gynecol. Can. JOGC* 27, 765–770 (2005).
- Polat, A. et al. Prevalence of primary dysmenorrhea in young adult female university students. *Arch. Gynecol. Obstet.* 279, 527–532 (2009).
- Ortiz, M. I. Primary dysmenorrhea among Mexican university students: prevalence, impact and treatment. *Eur. J. Obstet. Gynecol. Reprod. Biol.* 152, 73–77 (2010).
- Yonkers, K. A. & Simoni, M. K. Premenstrual disorders. *Am. J. Obstet. Gynecol.* 218, 68–74 (2018).
- Bernardi, M., Lazzeri, L., Perelli, F., Reis, F. M. & Petraglia, F. Dysmenorrhea and related disorders. *F1000Research* 6, 1645 (2017).
- Schmidt, P. J., Nieman, L. K., Danaceau, M. A., Adams, L. F. & Rubinow, D. R. Differential behavioral effects of gonadal steroids in women with and in those without premenstrual syndrome. *N. Engl. J. Med.* 338, 209–216 (1998).
- Smith, S., Rinehart, J. S., Ruddock, V. E. & Schiff, I. Treatment of premenstrual syndrome with alprazolam: results of a double-blind, placebo-controlled, randomized crossover clinical trial. *Obstet. Gynecol.* 70, 37–43 (1987).
- Chuong, C. J., Coulam, C. B., Kao, P. C., Bergstralh, E. J. & Go, V. L. Neuropeptide levels in premenstrual syndrome. *Fertil. Steril.* 44, 760–765 (1985).
- Chuong, C. J., Hsi, B. P. & Gibbons, W. E. Periovarian beta-endorphin levels in premenstrual syndrome. *Obstet. Gynecol.* 83, 755–760 (1994).
- Giannini, A. J., Martin, D. M. & Turner, C. E. Beta-endorphin decline in late luteal phase dysphoric disorder. *Int. J. Psychiatry Med.* 20, 279–284 (1990).
- Eriksson, E., Alling, C., Andersch, B., Andersson, K. & Berggren, U. Cerebrospinal fluid levels of monoamine metabolites. A preliminary study of their relation to menstrual cycle phase, sex steroids, and pituitary hormones in healthy women and in women with premenstrual syndrome. *Neuropsychopharmacol. Off. Publ. Am. Coll. Neuropsychopharmacol.* 11, 201–213 (1994).
- Shah, N. R. et al. Selective serotonin reuptake inhibitors for premenstrual syndrome and premenstrual dysphoric disorder: a meta-analysis. *Obstet. Gynecol.* 111, 1175–1182 (2008).
- Marjoribanks, J., Brown, J., O'Brien, P. M. S. & Wyatt, K. Selective serotonin reuptake inhibitors for premenstrual syndrome. *Cochrane Database Syst. Rev.* CD001396 (2013). doi:10.1002/14651858.CD001396.pub3
- Roca, C. A. et al. Effects of metergoline on symptoms in women with premenstrual dysphoric disorder. *Am. J. Psychiatry* 159, 1876–1881 (2002).
- Menkes, D. B., Coates, D. C. & Fawcett, J. P. Acute tryptophan depletion aggravates premenstrual syndrome. *J. Affect. Disord.* 32, 37–44 (1994).
- Miki, H., Oshimo, K., Inoue, H., Morimoto, T. & Monden, Y. Sex hormone receptors in human thyroid tissues. *Cancer* 66, 1759–1762 (1990).
- Zagrodzki, P. & Ratajczak, R. Selenium status, sex hormones, and thyroid function in young women. *J. Trace Elem. Med. Biol. Organ Soc. Miner. Trace Elem. GMS* 22, 296–304 (2008).
- Wei, Q. et al. Thyroid hormones alter estrous cyclicity and antioxidative status in the ovaries of rats. *Anim. Sci. J. Nihon Chikusan Gakkaiho* 89, 513–526 (2018).
- An osteopathic approach to diagnosis and treatment. (Lippincott Williams and Wilkins, 2005).
- Hall, John E. Guyton and Hall textbook of medical physiology. (Elsevier, 2016).
- Redei, E. & Freeman, E. W. Daily plasma estradiol and progesterone levels over the menstrual cycle and their relation to premenstrual symptoms. *Psychoneuroendocrinology* 20, 259–267 (1995).
- Munday, M. R., Brush, M. G. & Taylor, R. W. Correlations Between Progesterone, Oestradiol and Aldosterone Levels in the Premenstrual Syndrome. *Clin. Endocrinol. (Oxf.)* 14, 1–9 (1981).



33. Liang, Y. & Yao, S. Potential role of estrogen in maintaining the imbalanced sympathetic and sensory innervation in endometriosis. *Mol. Cell. Endocrinol.* 424, 42–49 (2016).
34. Ciebiera, M. et al. Alpha-Tocopherol Serum Levels Are Increased in Caucasian Women with Uterine Fibroids: A Pilot Study. *BioMed Res. Int.* 2018, 6793726 (2018).
35. Moore, K. L., Dalley, A. F. & Agur, A. M. R. *Clinically oriented anatomy.* (Wolters Kluwer/Lippincott Williams & Wilkins Health, 2014).
36. Arbuckle, BE, personal notations (date unspecified) in *An Endocrine Interpretation of Chapman's Reflexes* by Owens, C, 1937, made available to the American Academy of Osteopathy through the many efforts of individuals led by Dr. Raymond Hruby.
37. Rosenfeld, R. et al. Hormonal and volume dysregulation in women with premenstrual syndrome. *Hypertens. Dallas Tex* 1979 51, 1225–1230 (2008).
38. Gordon, J. L., Eisenlohr-Moul, T. A., Rubinow, D. R., Schrubbe, L. & Girdler, S. S. Naturally Occurring Changes in Estradiol Concentrations in the Menopause Transition Predict Morning Cortisol and Negative Mood in Perimenopausal Depression. *Clin. Psychol. Sci. J. Assoc. Psychol. Sci.* 4, 919–935 (2016).
39. Caroccia, B., Seccia, T. M., Barton, M. & Rossi, G. P. Estrogen Signaling in the Adrenal Cortex: Implications for Blood Pressure Sex Differences. *Hypertens. Dallas Tex* 1979 68, 840–848 (2016).
40. *Foundations for osteopathic medicine.* (Lippincott Williams & Wilkins, 2003).
41. Bertone-Johnson, E. R. et al. Association of inflammation markers with menstrual symptom severity and premenstrual syndrome in young women. *Hum. Reprod. Oxf. Engl.* 29, 1987–1994 (2014).
42. Pavlov, V. A. & Tracey, K. J. The cholinergic anti-inflammatory pathway. *Brain. Behav. Immun.* 19, 493–499 (2005).
43. Nelson, K. E., Glonek, T. & American College of Osteopathic Family Physicians. *Somatic dysfunction in osteopathic family medicine.* (2015).
44. Capobianco, J. *Chapman Reflexes.* in *An Osteopathic Approach to Diagnosis and Treatment* 707 (Lippincott Williams and Wilkins, 2005).
45. Owens, C. *An endocrine interpretation of Chapman's reflexes.* (Academy for Applied Osteopathy, 1963).
46. Ruffini, N., D'Alessandro, G., Cardinali, L., Frondaroli, F. & Cerritelli, F. Osteopathic manipulative treatment in gynecology and obstetrics: A systematic review. *Complement. Ther. Med.* 26, 72–78 (2016).
47. Pinho, L. E. E., Monteiro, A. K. S., Pimenta, A. G. & Santos, A. S. A. dos. TERAPIA MANUAL NO TRATAMENTO DA DISMENORRÉIA PRIMÁRIA: REVISÃO SISTEMÁTICA. *Rev. Pesqui. Em Fisioter.* 7, 224–232 (2017).
48. Hernandez-Reif, M. et al. Premenstrual symptoms are relieved by massage therapy. *J. Psychosom. Obstet. Gynaecol.* 21, 9–15 (2000).
49. Tettambel, M. A. An osteopathic approach to treating women with chronic pelvic pain. *J. Am. Osteopath. Assoc.* 105, S20–22 (2005).
50. Maganito, J. P., D. O., Showalter, A. & Tettambel, M. *A pocket reference guide: osteopathic management of the female patient.* (Melicien Tettambel, Anita Showalter, J.P. Maganito, 2009).
51. Nicholas, A. S. & Nicholas, Evan A. *Atlas of Osteopathic Techniques.* (Wolters Kluwer, 2016).
52. Hruby, R. J. *Exploring osteopathy in the cranial field.* (publisher not identified, 2013).
53. Eriksson, E. et al. Escitalopram administered in the luteal phase exerts a marked and dose-dependent effect in premenstrual dysphoric disorder. *J. Clin. Psychopharmacol.* 28, 195–202 (2008).
54. Nevatte, T. et al. ISPPMD consensus on the management of premenstrual disorders. *Arch. Womens Ment. Health* 16, 279–291 (2013).
55. Oddens, B. J. Women's satisfaction with birth control: a population survey of physical and psychological effects of oral contraceptives, intrauterine devices, condoms, natural family planning, and sterilization among 1466 women. *Contraception* 59, 277–286 (1999).
56. Pearlstein, T. Treatment of Premenstrual Dysphoric Disorder: Therapeutic Challenges. *Expert Rev. Clin. Pharmacol.* 1–4 (2016). doi:10.1586/17512433.2016.1142371
57. Kessler, J. H. The effect of supraphysiologic levels of iodine on patients with cyclic mastalgia. *Breast J.* 10, 328–336 (2004).