Persistent Postoperative Pain: Pathophysiology, Risk Factors, and Prevention

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Objective: Persistent postoperative pain (PPOP) is the second most common reason for a patient to seek care at a chronic pain center. Many of the patients seen with prolapse or incontinence are at risk for developing PPOP as a result of the surgeries done for these problems. The pathophysiology of this disabling pain disorder is well understood, and the risk factors are easy to identify. Once identified, perioperative interventions can be offered to attempt to prevent PPOP.

Methods and Results: Evaluation of articles obtained using a MEDLINE search involving chronic pain and PPOP, including prevalence, pathophysiology, and prevention was reviewed. The concept of central censitization and the key role it plays in chronic pain disorders were also reviewed. A history of chronic pain (anywhere) as well as findings of levator myalgia, allodynia, and hypertonic pelvic floor disorders is critical in identifying patients at risk for PPOP.

Conclusions: Identification of patients at risk allows the clinician to educate the patient about the risk of PPOP and the various perioperative interventions that can be used to prevent its development. Further studies will be required to determine how effective these interventions are in patients undergoing surgery for incontinence and/or pelvic organ prolapse.

Key Words: persistent postoperative pain, chronic pain, central sensitization, pre-emptive therapy, complications of surgery

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Chronic pain affects 34% of all women and 28% of all adults in America.[1] Surgery is recognized as one of the most common causes of chronic pain for patients seen in pain centers. A survey of more 5000 patients seen in chronic pain centers found that 34.2% had pain from degenerative disease and the second most common reason for seeking care was chronic pain after a surgical procedure (22.8%).[2] Persistent postoperative pain (PPOP) is a major cause of reduced quality of life. Although it is a frequently seen clinical problem, it is often unrecognized by those who perform the surgery that triggers this pain disorder.

Persistent postoperative pain is pain that lasts for more than 3 to 6 months after surgery.[3,4] It has been described after many common surgical procedures including breast surgery, where 30% to 50% develop PPOP and, in 5% to 10% of those, it has led to severe disabling pain. Another example is cesarean delivery with 10% of patients having PPOP and 4% with severe disabling pain.[5,6] Persistent postoperative pain is seen after many urogynecologic procedures that we perform on a regular basis including midurethral sling,[7] posterior colporrhaphy,[8] sacrospinous vault suspension,[9] and transvaginal mesh procedures,[10,11]to name a few (Table 1). Persistent dyspareunia and vaginal pain have been reported in both native tissue as well as mesh-augmented repairs as nicely reviewed by Moore and Miklos.[12]

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The proposed pathophysiology of this disabling disorder is understood, and the risk factors for the development of PPOP can easily be identified. The identification of patients at risk allows for patient education and the use of management strategies that have the potential for helping patients avoid a life-altering poor outcome.

This article will review the pathophysiological mechanisms that contribute to the development of PPOP and the risk factors that can easily be identified preoperatively. Various perioperative interventions will then be discussed so that the likelihood of PPOP can be reduced for your patients.

Pathophysiology

The performance of any surgery involves multiple "layers" of surgical wounding. There is physical as well as psychological trauma. The ability for the body to overcome this insult is variable for every patient, for every type of surgery, and also varies with the events that make up the perioperative recovery, such as the presence or absence of complications. At the surgical site, there is significant tissue trauma as well as at the release of multiple inflammatory mediators that affect the function of the nociceptors (sensory nerves) in the area of the trauma. These nerves carry impulses to the dorsal horn of the spinal cord—the first area of sensory processing. The stimulus information is then transmitted to higher centers of the central nervous system where the "awareness" of pain originates. The processing or modulation of noxious stimuli can occur throughout this pain pathway, starting at the periphery and ending at the higher centers of the brain. The key to the transition from acute to chronic pain involves changes in the processing of noxious stimuli. This is the key to understanding the development of PPOP. The complex pathways of nociceptive transmission have been well described elsewhere (Fig. 1).[13,14]

The inflammatory mediators at the site of tissue damage include cytokines, bradykinin, and prostaglandins. These mediators (and many others) induce neural plasticity in the nociceptors in the area of tissue trauma, which results in enhanced pain sensitivity at the operative site (peripheral sensitization).[15] Because tissue healing occurs, this inflammatory stimulus resolves. The peripheral sensitization should resolve, and the tissue sensitivity should return to normal.

At the same time the neural and inflammatory changes are occurring in the periphery, there is a barrage of nociceptive impulses that reach the spinal cord (dorsal horn) and are carried to the higher centers of the central nervous system. The central nervous system—especially in the dorsal horn—also demonstrates plasticity in response to the neuro inflammatory changes that occur at the site of surgical wounding. This is especially true if the pain is prolonged or severe. Pain processing and signaling within the dorsal horn are also enhanced. With ongoing noxious input, the stimulus-response relationship is altered and the central nervous system shows an increase in excitability (central sensitization).[16] This results in the perception of pain arising from a stimulus that normally would not be considered painful (allodynia).

The dorsal horn is where multiple excitatory, inhibitory, and modulatory mechanisms converge. This is the interface between

TABLE 1. Prevalence of PPOP After Common Surgical Procedures

Cesarean delivery	10% with 4% severe debilitating pain
Midurethral sling	0%–30%
Posterior colporraphy	Up to 25% (especially with levatorplasty)
Sacrospinious ligament suspension	10% to 15% (2% requiring intervention for resolution)
Transvaginal mesh	11.7%–19%
Sacrocolpopexy	unknown% (but 10.5% mesh erosion with most presenting with complaints of pain)

the peripheral and the central nervous systems, and the proper functioning of this area is required for the proper processing of sensory information coming from the periphery. Interneurons, glial cells, and descending inhibitory pathways play a key role in sensory processing and pain modulation. It is here that sensory information can be processed abnormally, and the patient's perceptions of stimuli can be altered resulting in hyperalgesia and allodynia. This central neural plasticity can continue long after the original tissue injury has resolved and can result in years of pain and abnormal responses that can induce new pain generators in the periphery. Pain often becomes self-perpetuating as a result of persistent pain at the operative site as well as the triggering of new pain generators in the surrounding areas. This process of spinal windup and the development of multiple visceral and somatic pain generators are well understood and

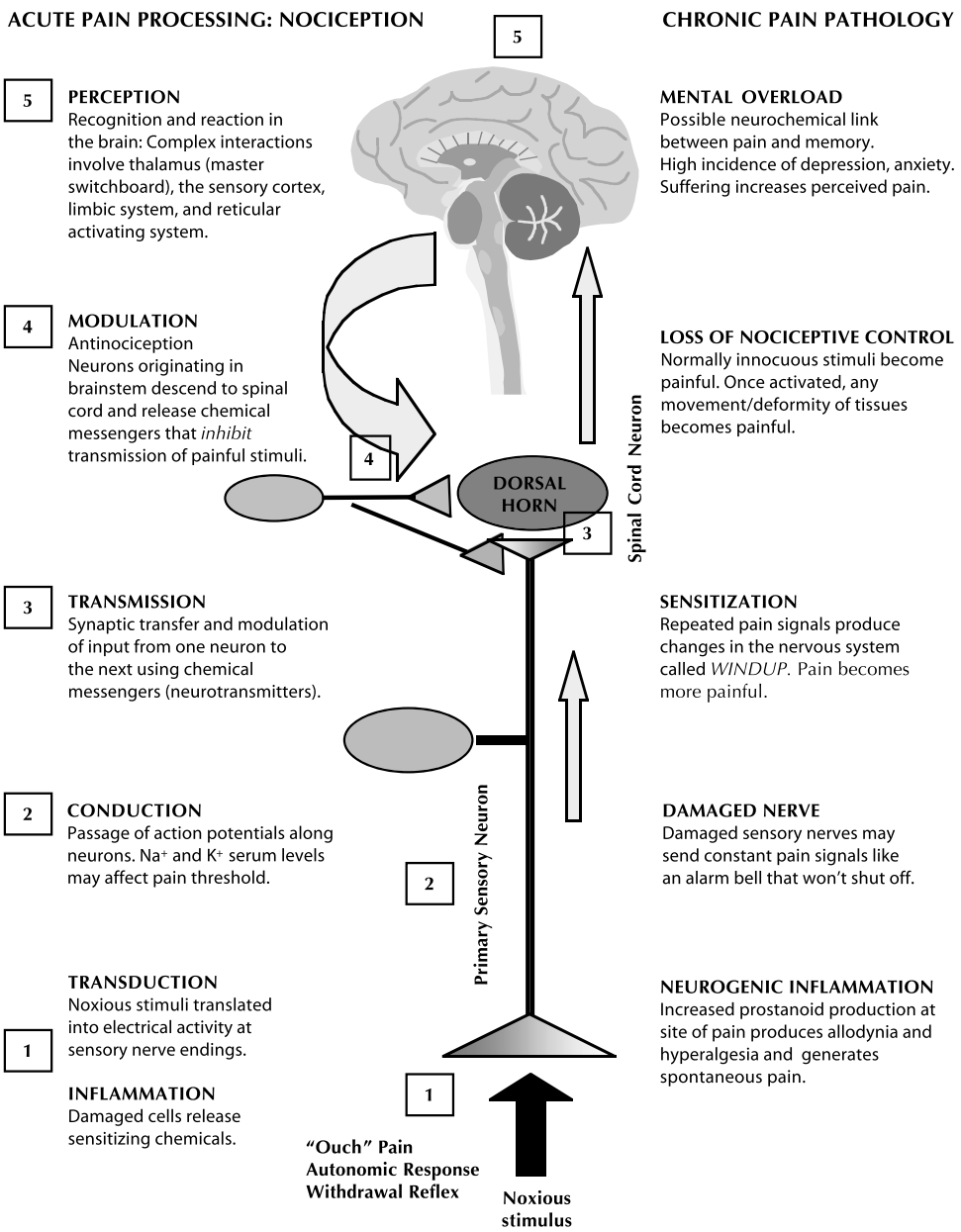

FIGURE 1. Diagram comparing acute and chronic pain processing and pathways. Reprinted with permission from Whitten et al.[13]

documented.[14,17,18] Examples include the development of urgency frequency (bladder pain syndrome), vulvodynia, and various pelvic floor myofascial pain disorders during the initial postoperative recovery in patients who are heading down the path toward PPOP. It is this transition from normal peripheral sensitization during the postoperative period to prolonged central sensitization that is the hallmark of all patients who develop PPOP. Any patient who comes to the operating room with a preexisting dysregulation of sensory processing (central sensitization) is at risk for further upregulation and the development of PPOP. This is obvious in all studies of patients with PPOP.[4,19,20] The biggest risk factor for developing PPOP is pre-existing pain anywhere.

Risk Factor Identification

One of the keys to providing good patient care for those who need surgical procedures is to understand who is at risk for the development of PPOP. The patient's ability to appropriately modulate the barrage of noxious stimuli arriving at the dorsal horn and to maintain proper sensory processing within the central nervous system seems to be the key to preventing PPOP. Once the cascade of events that leads to central sensitization and chronic pain has occurred, it is difficult to reverse the upregulation and abnormal sensory processing. Preoperatively, we must identify patient characteristics that seem to put them at risk. The preoperative state of sensitization, the psychological state of the patient, and metabolic factors all contribute to the modulation of the pain response and need to be considered in every patient. Research looking at risk factors for PPOP after various surgeries demonstrates the validity of this statement, and several examples are given in Table 2.[20]

There are many patient-related factors that have been demonstrated to increase the risk of PPOP. Age (older patients have decreased risk) and sex (females have increased risk) have certainly been identified. Multiple studies demonstrate a previous history of pain (in the area of planned surgical intervention or even pain remote from the site) to be a major risk factor.[3] Many of the chronic pain disorders frequently involve central sensitization.

TABLE 2. Risk Factors for PPOP

Chronic pain disorders	Bladder symptoms
IC/PBS	Urinary hesitancy
Vulvodynia	Urinary frequency
Chronic pelvic pain	Recurrent UTIs
Levator myalgia	Childhood bladder disorders
History of	Urodynamic findings of
Primary dyspareunia	Elevated urethral pressures (>130)
Tampons causing pain	Hesitancy w/pseudo dyssynergia
Prolapse symptoms worse than examination	Urethral allodynia (during or after)
Pessary use caused pain	Miscellaneous
Functional obstructed defection	Repeated surgeries
Dyschesia or anal fissures	Genetic predisposition/family Hx
Life-long constipation	Younger age
Previous surgery with poor PO pain experience	
Anxiety/PTSD/catastrophizing	
Voiding dysfunction after surgery	

IC indicates interstitial cystitis; Hx, history; PBS, painful bladder syndrome; PTSD, post traumatic stress disorder; UTI, urinary tract infection.

TABLE 3. Prevalence of Pelvic Pain Disorders

IC/PBS	2.7%–6.5%
Vulvar pain disorders	16%
Levator myalgia	24%
Chronic pelvic pain (other)	15%

IC indicates interstitial cystitis; PBS, painful bladder syndrome.

Examples of pain disorders known to involve dysregulation of normal sensory processing include fibromyalgia, interstitial cystitis/painful bladder syndrome, vulvodynia, irritable bowel syndrome, and most hypertonic pelvic floor disorders. As demonstrated by Adams,[21] levator myalgia is found in 24% of patients who see an urogynecologist for various pelvic floor disorders such as prolapse and incontinence. Chronic myofascial pain involves central sensitization. Danford[22] has shown a previous history of chronic pelvic pain resulted in an increased risk of persistent pain after surgical removal of transvaginal mesh in patients who presented with pain after the performance of various mesh procedures (odds ratio, 0.28). The more severe the pain is preoperatively, the more likely PPOP will occur.[23] It has also been shown in a small randomized controlled trial (involving amputations) that the better you control that pain before surgery, the less likely PPOP will occur postoperatively.[24] Psychological factors that place patients at risk for PPOP include anxiety, depression, posttraumatic stress disorders, past life traumas,[25] and a history of catastrophizing.[26] Given the multiple historical risk factors associated with the development of PPOP, it is not surprising that even surgeons without many patients with pain disorders will have some who are at risk for the development of PPOP. Let us not forget that 34% of all women have chronic pain and approximately 15% have chronic pelvic pain.

Patient Characteristics Uncovered During Preoperative Evaluation

As described previously, any patient undergoing pelvic surgery with chronic pain—especially pelvic pain disorders—needs to be considered at high risk for the development of PPOP. Many of the symptoms of chronic pelvic pain can be overlooked in those who have come to the pelvic floor surgeon requesting management of problems such as incontinence or pelvic organ prolapse. We must realize that many of our patients will have more than 1 problem. A classic example can be seen in patients who present with pelvic organ prolapse after years of excessive straining due to inability to pass urine or stool due to nonrelaxing pelvic floor dysfunction. Patients with levator myalgia have been demonstrated by Adams[21] to have high bother scores, given their degree of anatomic defects. Treatment of their pelvic floor pain and dysfunction should be considered before the correction of their anatomic problems. The literature demonstrates that many of our patients have pain disorders, and Table 3 attempts to demonstrate the prevalence of common pelvic pain disorders.

During office visits, the surgeon can assess the patient's ability to recover from the emotional and physical stresses of surgery. Patients who demonstrate a significant degree of anxiety, depression, or catastrophizing during their office visits will certainly be at risk. During these office visits, the patient's ability to modulate or control postoperative pain can be predicted by their history and their response to simple office evaluations. As described previously, allodynia is the pathognomonic finding in patients who have already developed a central sensitization, and this allodynia can easily be elicited by a careful history (eg, clothing induces pain, tampons are uncomfortable and voided volumes of <4 oz

in patients without bladder infections) or by performing a careful examination.

At the time of the patient's pelvic examination, the surgeon needs to be mindful of areas of allodynia using pain mapping techniques in all patients but especially in those who have a history of pain and/or allodynia (such as "catheters always bother me severely"). This helps both the surgeon and the patient have a better understanding of the causes for their multiple symptoms. Patients often have the impression that correction of the pelvic organ prolapse will resolve their symptoms of pain. Those with significant pelvic pain complaints and pelvic organ prolapse often have significant myofascial pain because prolapse alone rarely is associated with significant pelvic pain. Patients often demonstrate withdrawal and pain simply with anticipation of a pelvic examination or a light touch at the onset of a pelvic examination. Poor awareness of the pelvic floor muscles and especially poor relaxation are suggestive of an increased risk for postoperative pelvic floor myofascial pain. Bladder base tenderness is associated with bladder pain syndrome[27] (especially if urgency frequency is seen on the bladder diary). During urodynamics, or even with the placement of a small catheter to obtain a sterile urine sample, patients who demonstrate significant allodynia or ongoing burning that lasts for several hours are certainly demonstrating allodynia of the lower urinary tract with instrumentation. Always be concerned with patients developing PPOP when they describe a previous history of surgeries resulting in PPOP or when simple office procedures have caused significant symptoms of pain that continue for hours afterward.

Prevention of POPP

The literature is clear that some patients will develop PPOP after the pelvic floor surgeries. Certainly, those who have preoperative pain disorders are at risk, but any patient undergoing surgery can develop chronic postoperative pain. This phenomenon is well-known to physicians who care for patients with chronic pelvic pain, and it is becoming obvious to pelvic floor surgeons as well. The outcome data from multiple reports concerning surgical removal of vaginal mesh for the management of "mesh-related pain" demonstrate this repeatedly. Although many patients improve, most do not have total resolution and many have worse pain after the various attempts to remove the mesh. Unger et al[28] demonstrated that 30% of patients will have worsening of their pain after surgical attempts at removal of the mesh. This is to be expected once we understand the pathophysiology of chronic pain. These patients have chronic pain (often with a history of pain before placement of the mesh device and many other factors that placed her at risk for development of PPOP after the original placement of mesh as well), and they also may have a problem with the mesh placed (such as contracture, erosion, or even nerve entrapment). Although we can remove part or even all of the mesh, the primary problem is still the chronic pain syndrome that resulted in the perceived need to do mesh removal. Surgery alone will often not be enough in patients who have central sensitization, multiple risk factors for PPOP, and multiple pain generators. Abbott et al[29] demonstrated that 41% of patients with pain and mesh exposure were able to avoid surgery if nonsurgical therapies were used initially. Because we can easily identify patients at risk for PPOP, we must use this to formulate perioperative intervention that will reduce the risk of developing PPOP or even more importantly worsening of their preexisting pain.

The first step is patient education. The physician needs to explain the various steps that can be taken to try to provide a relatively pain-free surgical experience and help the patient understand that her subtle symptoms of chronic pain (pain with tampon use, urinary frequency, vaginal burning, and/or pain with intercourse for years) are all possible manifestations of a pre-existing pain disorder or at least an unrecognized yet symptomatic disorder that might involve central sensitization. I emphasize to the patient that our goal is to help her with her primary complaint such as leakage of urine or pelvic organ prolapse, but I also want to help with her other symptoms. As previously described, anxiety is associated with the development of PPOP. Therefore, offering treatment approaches tailored to her special needs and a discussion of appropriate expectations will tend to help the patient have a better surgical experience, partially by helping her relieve her anxiety and by placing her unique needs and her overall outcome in the forefront.

If patients have preoperative pain disorders, these should be treated before surgery. These problems of pain can be remote to the surgical field or within the surgical field. Even if the patient has minimal symptoms, they can worsen after surgery. A classic example is the patient with the chief complaint of stress incontinence who also demonstrates urinary frequency and urgency ("I have always had a small bladder"). Especially if this patient is found to have high urethral pressures on urodynamics and significant urethral allodynia with the performance of her urodynamics, she should consider simple interventions to down regulate her lower urinary tract before the performance of a midurethral sling. This might simply include a low-acid diet to improve her urinary frequency and physical therapy to correct her hypertonic pelvic floor dysfunction. Advise the patient that if the surgery does cause worsening of her frequency, there are other interventions that can be provided to control those symptoms.

When surgical procedures are being done for pain disorders, it is very important to aggressively treat that pain nonsurgically first. Chronic pelvic pain disorders are best managed by a multimodal approach, and a review of management of the various pain disorders is beyond the scope of this review.[13,17] Often patients with long-standing pelvic floor myofascial, pain will develop new problems of pelvic organ prolapse, and as Adams et al[21] have demonstrated that these patients have higher bother scores on Pelvic Floor Distress Inventory and Pelvic Floor Impact Questionnaire. Obstructed defecation caused by a nonrelaxing pelvic floor often will contribute to the development of pelvic organ prolapse. Pelvic pain or back pain is rarely caused by pelvic organ prolapse, especially when the pain is much greater than one would expect on the basis of the degree of prolapse identified. When patients report that a pessary makes her pain worse, the pain is the result of pelvic floor myalgia and not pelvic organ prolapse. Therapy for hypertonic myofascial pain before to surgery is beneficial for the patient and increases the likelihood that she will have a good surgical experience. Another example is the various vulvar pain disorders such as lichen sclerosis or vulvodynia that sometimes present in patients we see for various pelvic floor disorders. When these patients require an extensive vaginal reconstruction for pelvic organ prolapse, the anticipated postoperative discharge aggravates their pre-existing vulvar pain. It is important to get the vulvar pain under control before the surgery.

If you identify a patient to be at risk for PPOP, you must formulate a surgical approach that minimizes tissue trauma. Minimally invasive procedures such as laparoscopic or vaginal approaches should be preferred more than large abdominal procedures. The likelihood of development of PPOP has been shown to be reduced with these minimally invasive approaches to hernia repair.[30] Important to note, however, is that a vaginal hysterectomy is much less likely to induce PPOP as compared with a laproscopic hysterectomy as demonstrated by Pokkinen et al[31] in a large prospective cohort study (even though the

TABLE 4. Pelvic Surgeries to Avoid if Levator Myalgia Is Present

Levator plications
Sacrospinious fixation procedures
Transvaginal mesh that involves muscle fixation
Transobturator midurethral slings
Any painful surgery with inadequate perioperative pain control plan
Extensive mesh excision
Anal sphincterplasty

amount of acute pain was less with a laproscopic approach).[32] Longer surgical procedures are also associated with PPOP.[26] Muscle sparing procedures should be used especially in patients with pre-existing levator myalgia or other pelvic floor hypertonic disorders (Table 4). Unger and Walters[9] show that 15.3% of patients will have persistent pain at 6 weeks after a sacrospinous ligament fixation and 2.1% will require various techniques of intervention to attempt to control the persistent pain. Most patients with chronic pain after transvaginal mesh procedures have a component of myofascial pain and treatment of this pain disorder before surgical intervention is certainly necessary when mesh is anchored to or traverses these pelvic floor muscles. Many patients with complaints of mesh-related pain have a history of pre-existing pain and/or hypertonic muscle dysfunction that was not identified before the original transvaginal mesh procedure being done. Patient education about the source of the pain is very important as we attempt to prevent the natural tendency for the patient to "squeeze" because of the pain she is experiencing. This is why physical therapy is performed before surgery in these patients. The goal of this therapy is to learn how to relax their muscles and develop the muscle awareness that they often lack. The patient will need to work at keeping her muscles relaxed, using the techniques taught during physical therapy and during the immediate postoperative recovery. The ultimate goal is to try to prevent further upregulation of her pre-existing pain as a result of multiple surgeries to remove mesh rather than understanding that the synthetic mesh is at the most only one of the triggers to her pain disorder and we are treating patients with a pain disorders who happen to have a history of mesh placement. Informed consent must include the awareness of PPOP and even worsening of pain[10,28,33] as a result of surgical attempts of removing the mesh rather than treating the pain disorder.

Perioperative/Pre-emptive Anesthesia and Analgesia Techniques

When operating on patients who are at risk for PPOP, the management strategy is to prevent postoperative pain as much as possible. Our goal is to prevent either the worsening of pre-existing or development of new central sensitization. We must do everything we can to block the barrage of noxious stimuli from reaching the spinal cord and causing this neuropathic dysregulation that is at the heart of PPOP. Regional anesthesia with local anesthetics and opioids, alone or in conjunction with general anesthesia (for laparoscopic approaches), has been shown to decrease analgesia use postoperatively and to improve patients' satisfaction with surgical procedures. Although short-term outcomes consistently seem to be improved, long-term outcomes concerning development of persistent pain disorders have been demonstrated in only 1 study. This study demonstrated that 32% had PPOP and that it was less likely to occur in those who received regional anesthesia.[34] In vitro research has demonstrated a reduction in the neuropathic hyperexcitability that is seen as a result of surgical trauma when pre-emptive anesthesia is used. Randomized controlled trials, however, do not show consistent benefit. The degree of tissue trauma and therefore the amount of noxious stimuli likely alter the potential for showing efficacy when this modality alone is used. Duration of pre-emptive therapies is also key with the goal of blocking the barriage of noxious stimuli during the acute phase of neuroinflammatory input, at least 24 hours after the incision.[35] The addition of long-acting morphine (Dura-morph) and/or with the use of continuous infusion of local anesthesia has been shown to be very effective in postoperative pain management involving obstetrical, gynecologic, and colorectal surgical procedures. Theoretically, these types of interventions should at least decrease the likelihood of development of central sensitization and therefore PPOP.

There are many other pre-emptive therapies to be considered, and in the high-risk patient, a multimodal approach should be considered. Table 5 summarizes the data concerning the use of perioperative drugs that attempt to decrease the likelihood of central sensitization, especially in patients who are at high risk. The use of gabapentin as an adjunctive

TABLE 5. Peri-operative Pharmacologic Interventions

Drug	Protocol	Study Notes	Reference
Clonidine	2 mL/300 μg intrathecal preoperative	Colon resections Decreased hyperalgesia Decreased PPOP	Perkins and Kehlet, 2000[36]
Gabapentin oral	300–1200 mg preoperative	Hysterectomy Decreased postoperative pain	Alayed et al, 2014[37]
Pregabalin oral	300 mg preoperative+ 300 mg/d for 14 d	Orthopedic surgery Decreased postoperative pain	Buvanendran et al, 2010[38]
Amitriptyline	10–25 mg every night at bedtime for 6 wk	Decreased postherpetic neuralgia	Bowsher, 2003 [39]
Ketamine (NMDA antagonist) intravenous	0.5 mg/kg preoperative + intravenous infusion intraoperative and postoperative at 0.25 mg/kg/h	Randomized in posterior exenteration Decreased PPOP 7% vs 71%	De Kock et al, 2001[40]
Cox inhibitors	Variable drugs and dose	Consistently reduces postoperative pain, no long-term studies	Derry, 2013[41]

approach[42] is well supported, but other interventions as outlined might be considered.

As noted previously, the prevention of excessive pain in the immediate postoperative recovery is one of the keys to the prevention of PPOP. When patients have excessive pain in the immediate postoperative recovery, we must first consider significant complications such as hematoma, nerve entrapment, and other potential etiologies, but once these have been ruled out, we must realize that this patient is suffering a complication of inadequate pain management and we must quickly intervene to determine the etiology of her pain and how to control it. Pain management might involve other interventions such as postoperative epidurals or adjustments in pain medications. We must remember that 10% of our white patients lack adequate CYP2D6 function to convert codeine to its active metabolite morphine. Therefore, sometimes, simple adjustments in pain medications are all that is necessary. Myofascial pain likely responds to medications that relieve muscle spasticity (eg, Belladonna/Opium morning suppositories or various muscle relaxers such as benzodiazepines or Tizanadine). Various options to relieve bladder/catheter pain are also available including oral bladder analgesics and bladder rescue cocktails.

SUMMARY

The burden of PPOP is enormous. It affects 10% to 50% of patients undergoing common surgical procedures. It is the second most common reason for patients to seek care from a center that specializes in pain management. Every pelvic floor surgeon has induced a chronic pain disorder in 1 or more patients (hopefully, this happens infrequently), and it is now time for each of us to be aware of and manage this surgical complication. The challenge placed on the surgeon is to identify the patients who are at risk for developing PPOP. We must realize that surgery is controlled trauma that we induce, but we also must do what we can to help our patients recover. We understand the risk factors and the pathophysiology of this postoperative complication and must take steps to do whatever we can to prevent it. Although there is a paucity of evidence-based strategies available to prevent PPOP, perioperative management should be discussed with the patient. Certainly, patient education, behavioral modification, and consideration of perioperatve interventions for those patients at high risk need to be considered. Tailoring surgical approaches as well as simple perioperative pharmacologic and anesthetic approaches will optimize the patient's pain experience postoperatively and potentially reduce the likelihood of developing PPOP. Finally, if the patient manifests a heightened postoperative pain experience, it is paramount to identify this, evaluate its cause, and initiate treatment rapidly to minimize the likelihood of PPOP.

Although we inform our patients about risks of DVTs and infection, the risk of PPOP is much greater. Risk factors are easily identified, and perioperative interventions have the potential to prevent a common postoperative complication. The need for us to inform our patients of this potential life-altering complication and keep striving to provide good outcomes for our patients cannot be overstated. Surgical outcomes such as the correction of stress incontinence or pelvic organ prolapse are important, but we also need to look at long-term quality of life. To only remove mesh in a patient with PPOP and not treat their pain disorder will not provide the level of care that we should be providing these patients. Sometimes, we must protect them from the unintended consequences of our surgical interventions to make their lives better.

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