Chapter 29
Persistent or Chronic Pelvic Pain

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DEFINITIONS

Allodynia—Pain resulting from a nonnoxious stimulus.

Hyperalgesia—Painful sensation of abnormal severity following noxious stimulation.

Neuropathic pain—Pain persisting after healing of disease or trauma-induced tissue damage.

Neuroplasticity—The malleability of central pain perception mechanisms in response to chronic pain states.

Nociceptor—A nerve receptor for pain.

There is not universal agreement on a single definition of chronic pelvic pain (CPP), but most practitioners accept the proposal of the American Congress of Obstetricians and Gynecologists: noncyclic pain of 6 or more months' duration that localizes to the anatomic pelvis, anterior abdominal wall at or below the umbilicus, or the lumbosacral back or the buttocks and is of sufficient severity to cause functional disability or lead to medical care. We are in need of deeper and updated investigation into the epidemiology of pelvic pain, but likely, at least 15% of women are affected by pelvic pain, most commonly during reproductive-age years. When last tabulated in 1996, it was estimated nearly $3 billion was spent annually in physician costs and out-of-pocket expenses. CPP accounts for at least 10% of outpatient visits to a gynecologist and is the indication for 40% of gynecologic laparoscopies.

Endometriosis and adhesions are some of the most common conditions assigned as an etiology of pelvic pain, but the connection between these problems and pain symptoms is actually more tenuous than has been traditionally taught. More and more data confirming the coexistence of multiple chronic pain disorders in patients—conditions such as interstitial cystitis (IC), painful bladder syndrome, irritable bowel syndrome (IBS), temporomandibular joint disorder, migraine headaches, vulvodynia, and fibromyalgia—suggest that perhaps we ought to be treating pelvic pain under an updated paradigm where the disease is pain itself rather than pain a manifestation of a specific etiology. In this chapter, we will apply the concept of central sensitization to CPP and point out common peripheral pain generators—nociceptive stimuli—than can be modulated by gynecologic interventions. We will review important elements in evaluation and describe the criteria for a chronic pain syndrome (CPS), offering a theoretical model to explain the evolution of chronic pain over.

HISTORY

Over the past 60 years, the study of CPP has gone through significant changes in approach. Investigations undertaken before the development of laparoscopy focused on correlations between pelvic pain and psychological distress. In the absence of palpable pathology, the gynecologist of the 1950s and 1960s was understandably reluctant to subject a patient to laparotomy to investigate pain. During this era, the prevailing cartesian theory of pain perception suggested that pain should be somewhat proportional to the degree of tissue damage found. Hence, if the pathology was not big enough to palpate, it was seldom operated on. Although this model was sufficient to address most causes of acute pain, it fails to elucidate the majority of chronic pain disorders, in gynecology as well as other areas of medicine. The gate control theory, promulgated by Melzack

and Wall in 1965, allowed integration of physical and psychological parameters and explained how chronic pain can be quite different from acute pain. The model also suggests that information flows in two directions regarding pain: (a) nociceptive signals from peripheral tissue ascend through the spinal cord to higher centers, and (b) central centers can modulate, via descending signals altering spinal cord neurotransmitter and interneuron activity, the transmission of these nociceptive signals from the periphery. Deterioration of these regulatory processes was thought to potentially account for development of chronic pain states by allowing too many peripheral signals to pass through the spinal cord "gates." Variation in patients' relative degree of gate opening could thus explain why similar amounts of physical tissue damage result in different degrees of pain perception. The concept is similar to the way we view differences in depth of sleep—for some, the brain can easily filter out stimuli; for others, it takes very little exposure (e.g., light, sound) to overcome unconsciousness and bring about wakefulness.

TABLE 29.1 Contributors to Pelvic Pain in Laparoscopy-Negative Patients

Gastrointestinal	
	Constipation
	Irritable bowel syndrome
	Inflammatory bowel disease
	Diverticulitis
Urinary	
	Urethral syndrome
	Interstitial cystitis/painful bladder syndrome
Musculoskeletal or neurologic	
	Pelvic floor tension myalgia
	Piriformis syndrome
	Nerve entrapment
	Ventral hernia
	Rectus tendon strain
	Myofascial pain

	Back or pelvic postural changes
Gynecologic	
	Pelvic vascular congestion
	Cervical stenosis

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While these theory changes were stimulating the field of pain research, gynecologists were busy developing laparoscopy. Previously cherished myths soon fell by the wayside: for example, the incorrect assumption that endometriosis is seldom found in adolescents or women of African descent. With these observations came the hope that laparoscopic and medical treatment of encountered pathology would fix CPP. Reports of CPP from that era focused on “laparoscopy-negative” patients; indeed, some pelvic pain clinics required a negative laparoscopy as an entry criterion, implying that if some pathology were found, it must be a “real” cause for pain. Subsequent experience has shown that even though treatment

of laparoscopically diagnosed pathology is often helpful, the clinical reality is more complex:

1. In many instances, the organic pathology found at laparoscopy may be incidental and not related to the pain.
2. In those with pathology that does contribute to nociception, the pain experienced by the patient may differ from another patient with anatomically similar pathology.
3. Pain from a laparoscopic finding may be the sum of that contribution plus signals from some or all of the disorders listed in **Table 29.1**.

Consider the research of the 1980s that documented a distressingly high prevalence of physical and sexual abuse. Epidemiologic surveys of community samples revealed that as many as 25% to 30% of adult women reported having experienced sexual abuse during childhood. Studies of women attending pelvic pain clinics, especially those based in psychiatric settings, showed that up to 60% of these women had been abused. These observations led to the speculation that the experience of abuse may make a person more vulnerable to the development of CPP or perhaps be a specific cause for pain. Studies using positron emission tomography and functional magnetic resonance imaging suggest that the experience of abuse may indeed leave its neurophysiologic footprints: stressful stimuli produce different central response patterns in abused versus nonabused subjects. In relation to pain, abuse, particularly that which occurs in formative years, may serve to alter response to nociception and central pain processing. That said, not all abused patients go on to have chronic pain nor do all patients with pain have a history of abuse, so it might be the response to trauma that plays a key role in development of chronic pain. Health care providers need to take into account the presence of abuse in a patient’s history when detected, but be careful to avoid necessarily concluding a causal relationship in that patient’s pain.

Melzack neuromatrix theory is an expansion of his original work that includes the notion of neuroplasticity, among other elements. The concept of neuroplasticity suggests that experience can change the neurophysiologic behavior of the central nervous system in a manner that influences the subsequent processing of nociceptive stimuli. It may explain the apparent development of pain responses to stimuli usually thought of as nonpainful (allodynia), as well as exaggerated responses to painful stimuli (hyperalgesia). Every practicing gynecologist has

seen patients whose pain responses seem out of proportion to the pathology found. This may reflect the emotional meaning of the problem for the patient, as well as past or present trauma, but it may also be the result of nociceptive mechanisms not yet fully understood (e.g., the exact mechanism of pain from endometriosis) or the result of sensitization of spinal cord interneurons that have become pain amplifiers as a result of being on the receiving end of peripheral nociceptive stimuli for prolonged periods. When nociception has been emanating from one organ system for a period of time, adjacent organs may join the chorus. This concept may help explain the common finding of coexistent somatosensory disorders in the same patient, an observation that has led some investigators to pursue potential genetic variations in central neurotransmitter networks that might predispose to the development of multiple such disorders. The above is the negative side of neuroplasticity. The positive side of the neuroplasticity concept is that, perhaps, given enough time and the right treatment, even seemingly intractable chronic pain problems may ameliorate to the point of allowing substantially improved function.

The concept of central sensitization adds another layer to theoretical understanding of chronic pain. This idea emphasizes the ramping up of pain signaling with repeated stimuli over time. The centralized pain hypersensitivity helps us understand how multiple organ systems can be recruited into the syndrome, incorporating genetic and social factors in pain amplification.

CONTRIBUTIONS OF PERIPHERAL PAIN GENERATORS

This section deserves an important caveat. Though we believe that types of tissue damage or other nociceptive input can generate pain, they cannot be viewed in isolation of the patient's individual central pain processing. Management strategies will be discussed in more detail, but, in general, the goal of the treating provider involves trying to dampen overall pain signaling sensitivity—"turning down the master volume"— and looking for areas in the periphery that can be "tuned up" toward better functioning. The following peripheral generators represent areas where we can intervene but should not be described to patients with CPP as *the* cause of their pain. These conditions can be completely asymptomatic in many patients, and thus the host where the disease manifests is much more important than the disease itself.

Endometriosis

A review summarized laparoscopic findings from 2,615 patients in 15 studies (nine retrospective, six prospective). Endometriosis was found in 2% to 51% of patients, suggesting that referral biases lead to very skewed samples. Clearly, not every woman with pain has endometriosis, nor does every woman with endometriosis have pain, although women with the disease had pain more often than those without it. A number of previous studies of CPP described only either patients without visible laparoscopic findings or stratified patients according to the presence or absence of such findings. The description of atypical (nonpigmented) endometriosis by Jansen and Russell in 1986 calls these classifications into question. Laparoscopy studies published before that time reported that 11% of women with CPP had endometriosis, whereas three similarly conducted studies published since 1986 reported a 41% prevalence of endometriosis in women undergoing laparoscopy for CPP. The pre-1986 literature on pelvic pain must be reevaluated with this information in mind. Many studies may have included women with endometriosis in the anatomically normal group, thus generating erroneous conclusions about the entirely psychogenic nature of their pain.

There are a variety of proposed mechanisms explaining pain associated with endometriosis, including inflammatory, nociceptive, and neuropathic. There is no pathognomonic symptom associated with endometriosis. Laparoscopic treatment of endometriosis improves pain symptoms more than diagnostic surgery alone, but many of the symptoms often assigned to the disease (e.g., dyspareunia, dysmenorrhea, abnormal bowel or bladder function) are commonly found in functional disorders such as IBS, making it difficult to understand the relevant contribution of endometriosis to CPP. The severity of the pain correlates poorly with the amount of superficial peritoneal disease, and such implants do not localize to the site of patients' symptoms. Deeply infiltrating

endometriosis (DIE)—fibrotic, vascular, desmoplastic tissue destruction—is an exception to this rule. Nodular disease of the colon is associated with dyschezia and hematochezia; urologic tract disease with hematuria, dysuria, and obstruction; and cul-de-sac disease (uterosacral ligaments, rectovaginal septum, ovarian endometriomas) with deep dyspareunia. Fear of worsened pain, impaired fertility, or recurrent disease after treatment may increase pain levels. Of the women for whom we repeat laparoscopy for recurrent pain following complete hysterectomy and adnexectomy for endometriosis, only a small minority (3% to 5%) prove to

have recurrent disease. Most cases of continued postoperative pain are interpreted in the context of the patient's overall sensitivity to pain and other peripheral stimuli functioning under that same sensitivity, such as pelvic floor tension myalgia, painful bladder, or functional gastrointestinal disease.

Those experienced in treating the disease can often detect DIE with clinical exam and imaging. When endometriosis is strongly suspected otherwise, and initial treatment with oral contraceptives (OCs) has failed, diagnostic laparoscopy should be the next step. One widely discussed study by Ling et al. concluded that a careful clinical history and physical examination can predict the presence of endometriosis in approximately 80% of cases. However, this was done in the setting of a strict research protocol; the diagnostic sensitivity of this approach in general clinical practice is likely much lower. Unfortunately, the study is often misinterpreted as implying that pain relief following gonadotropin-releasing hormone (GnRH) agonist treatment is not only a sensitive detector of endometriosis but is also *specific*—that is, it makes the diagnosis of endometriosis. Careful reading of the data reveals that this is not the case: the frequency of relief following GnRH treatment was the same in women with and without endometriosis. In addition, pain sensitivity is known to increase perimenstrually even in women without CPP. This may mean that nociception from pain disorders outside the reproductive tract may also improve when menstrual cyclicity is eliminated. For example, IBS symptoms also decline in women taking GnRH agonists. Hence, although *failure* to relieve pain with a GnRH agonist supports the notion that the reproductive organs are not involved, relief of pain with these medications does not prove that they are to blame.

Pelvic Adhesions

Early reviews supported the role of adhesions as a significant peripheral pain generator in CPP. In one, 6% to 55% of the 2,615 patients who underwent laparoscopy for pelvic pain had pelvic adhesions. In more recent investigation, Latthe et al. demonstrated a relatively weak correlation between adhesions and CPP, much less than factors such as psychosomatic symptoms and substance abuse. Correlation, of course, does not imply causality, and few, if any, well-designed studies demonstrate effective treatment of CPP with adhesiolysis. Unfortunately, in an effort to provide some explanation for complex pain disorders, providers often still posit adhesions as an etiology, even when a patient's surgical history includes only laparoscopy with findings of minimal or no endometriosis, pelvic inflammatory disease, or other conditions associated with meaningful adhesions. This explanation can happen even when the patient's pain escalation is remote from the last surgery. Adhesions may play some role in pain conditions in some women, but the relative contribution is probably small. Also, the putative treatment—repeat surgical intervention—can add new contributions to pain syndromes, such as the impact of surgical trauma, disappointment from lack of pain relief, feeding the psychological need of being "ill" with more surgery, and, in the worst case, generating a complication such as enterotomy.

Pelvic Support

Most women in pain clinics are in their third or fourth decade of life, while pelvic organ prolapse affects significantly older women, suggesting a very minimal role for support problems in CPP.

Pelvic relaxation usually leads to reports of heaviness, pressure, dropping sensations, or aching. In attempting to hold in prolapsing organs, the patient may tense the levator plate, leading to tenderness during daily activities and intercourse. Fear of (or actual) loss of urinary control during coitus can add to the discomfort by impairing

physiologic sexual response.

Uterine retroversion is another potential etiology for CPP, particularly in the form of deep dyspareunia. Clearly for many women, retroversion is an innocent anatomic variant, but for those with pain, uncontrolled clinical series of uterine suspension procedures suggest changing the position of the uterus to an axial or anteverted position can improve dyspareunia by elevating a tender fundus out of the posterior cul-de-sac and allowing for better vaginal expansion as a natural part of the sexual response cycle.

Pelvic Congestion

Overfilling (congestion) of the pelvic venous system has been implicated as a cause of dull chronic aching pain that usually is unilateral and worse at the end of the day after prolonged standing, premenstrually, and postcoitally. Some studies suggest the condition is present in nearly one third of women with CPP, but there is no agreed-upon reference standard for diagnosis, despite individual technical regimens involving venography, MRI, and ultrasonography. Hormonal suppression, percutaneous embolotherapy, and surgery (vein ligation, hysterectomy, and salpingo-oophorectomy) represent available treatments, but study protocols involving these interventions are diverse, and few have been investigated in controlled trials.

Residual Ovary

When the uterus has been removed, with or without removal of one ovary, the remaining ovary or ovaries become symptomatic in 1% to 4% of women. Pain from the ovary can be increased by confinement within postoperative adhesions, rupture or leakage of a cyst prompting additional adhesion formation, or attachment of the ovary to the sigmoid colon or vaginal apex by postoperative adhesions. In the case of attachment to the vaginal apex, deep dyspareunia can result when the area is struck.

Ovarian Remnant

A more difficult situation can develop if a small fragment of ovarian tissue is left behind during attempted oophorectomy. In most instances, this happens when challenging dissection is required, such as cases of extensive pelvic adhesions and/or DIE. Within 1 to 3 years of the attempted oophorectomy, continued folliclestimulating hormone (FSH) stimulation will result in growth of the ovarian fragment, often producing an intermittently symptomatic pelvic mass located along the course of the ovarian vascular supply. A postulated mechanism for pain generation includes the cystic enlargement of the mass confined within fibrotic adhesions. If the remnant developed because endometriosis is made for difficult oophorectomy, that disease is often found in the remnant and probably also serves as a pain generator. Ovarian remnants are uncommon, but not rare, as implied by early case series. Classic symptoms include absence of vasomotor symptoms when bilateral oophorectomy was intended and presence of cyclic unilateral pain. As in the case of the residual ovary, the remnant can produce dyspareunia if it is located close to the vaginal apex. When performing oophorectomy, it is best to open the pararectal space and completely skeletonize the infundibulopelvic (IP) ligament, not only to avoid complications such as ureteral injury but also to prevent ovarian remnant syndrome. In difficult cases, dividing the IP at or above the pelvic brim, as in risk reduction prophylactic oophorectomy, is prudent.

Vaginal Apex Pain

Following hysterectomy, pain may persist or recur because of intrinsic sensitivity of the vaginal apex. Although the cuff may appear to have healed perfectly well, gentle examination with a cotton-tipped applicator may reveal focal sensitivity of

moderate-to-severe degree, many times located in one lateral fornix or the other and often replicating the reported pain of dyspareunia. When this is not done, the unaware examiner may then, noting pain upon traditional bimanual examination, mistakenly conclude that the source of nociception lies cephalad, for example,

in a remaining ovary, pelvic scarring, or bowel adhesions.

The diagnosis may be confirmed by noting elimination of the pain following injection of local anesthetic. The condition is generally considered neuropathic, by virtue of the character of the pain (burning, stinging, sharp), and that neuropathic treatments (overnight application of lidocaine, oral medications such as nortriptyline, amitriptyline, gabapentin, etc.) seem to benefit some patients. Laparoscopic revision of the vaginal cuff may give good initial relief in approximately two thirds of patients, but pain tends to recur to a degree over the subsequent 2 to 3 years.

Musculoskeletal Problems

Musculoskeletal changes can become involved with CPP, either as the primary problem or as a secondary reaction to the pelvic pain. Dysmenorrhea can be referred to the midline of the low back, especially when the uterus is retroverted. Pain can also be referred to the midline of the low back in the presence of cul-desac endometriosis. An ovary fixed to the pelvic sidewall can refer pain to the ipsilateral low back, lower quadrant, and upper thigh.

The muscular problem that most often produces pelvic pain is pelvic floor tension myalgia. Intermittent or constant painful contraction of the levator plate can be present as a primary psychophysiologic problem, but contraction is more often a reaction to some other source of pain. Even when the primary source of pain is successfully treated, the reactive muscle contraction can persist as a learned response, in much the same way that vaginal introital muscle spasm (vaginismus) can persist after transient but repeated painful vaginal events. Pelvic floor tension myalgia is often found in the setting of generalized somatic hypersensitivity, a condition whose worst case includes fibromyalgia.

Lumbar musculature can become tender as a primary problem or in reaction to subtle changes in posture and motion. Trigger points can be present in the low back and gluteal areas in the muscles best inspected by pelvic examination (e.g., levator plate, piriformis, obturator internus).

The piriformis and obturator muscles warrant additional mention because they are seldom appreciated as possible sources of pain. These muscles are external rotators of the leg, and rotation against resistance can allow detection of tender spasm of the muscles during the pelvic examination. The sciatic nerve can traverse the belly of the piriformis as a normal anatomic variant, producing symptoms similar to sciatica when the muscle is in spasm.

Myofascial Pain

Focal lower quadrant abdominal wall pain can be produced by entrapment of the genitofemoral and ilioinguinal nerves, as described by Applegate. Such entrapment appears most often after Pfannenstiel abdominal incisions. Reiter and Gambone reported that 14% of 122 laparoscopy-negative women had myofascial pain probably related to a previous surgical incision. Myofascial trigger points—palpable taut bands of tender skeletal muscle—may be a primary problem or a later reaction to the long duration of pain from some other source.

Medical Comorbidity

Peripheral pain generation in CPP often involves nongynecologic systems (**Table 29.1**). A careful history and close physical examination of gastrointestinal, urologic, musculoskeletal, and neurologic systems are needed to evaluate these additional contributions to CPP. Most of the available literature examines these problems of other systems independently of each other and without reference to their relevance to CPP or to the overall prevalence of these disorders in CPP.

The gastrointestinal system is perhaps the most common nongynecologic contributor to CPP. Constipation and IBS occur most frequently, although inflammatory bowel disease and diverticulitis can at times present with pain

alone. Women are proportionately more affected by IBS, and some have hypothesized increased relaxin levels produced by a dysfunctional corpus luteum as one of many possible contributing factors. As previously mentioned, treatment with a GnRH agonist may reduce symptoms of IBS.

Urologic problems, which are less easily confused with gynecologic disorders, are perhaps second in terms of prevalence. The urethral syndrome (frequency, urgency, and dysuria in the absence of bacteriuria), IC, and bladder spasms are all accompanied by significant anxiety and depression symptoms. The symptoms of these three disorders are very similar to those in a population of gynecologic CPP patients. Structured questionnaires (e.g., the Pelvic Pain and Urgency/Frequency [PUF] scale) help detect symptoms possibly emanating from the bladder. In some patients, however, a "positive" score can be achieved on the basis of other components of pelvic pain alone—without specific bladder symptoms—making the measure perhaps too *sensitive* and hence insufficiently *specific*. A history (whether pain occurs during micturition, daily activities, or coitus) does not always reveal the involved system, but careful pelvic examination with stepwise gentle palpation of the urethra, bladder base, and bladder may help the physician identify the site of the pain the patient is experiencing.

Many patients do not experience the problems described here in pure form, but rather in varying degrees of intensity, with varying contributions to an individual's total discomfort. Indeed, we suspect that shared innervation of pelvic organs may often lead to subsyndromal symptoms in an organ system that neighbors one with different pathology. Such patients have a multifaceted somatosensory disorder, as opposed to being the unfortunate victim of multiple unrelated organ-specific disease processes. Close attention to such nuances of detail is warranted both in clinical management and in published reports.

PSYCHOLOGICAL FACTORS

Personality

The links between chronic pain and individual psychology and personality style have been sought after and discussed in the psychiatric literature for many years. Some early reports implied that women who reported CPP had a high prevalence of feminine identity problems related to conflicts about adult sexuality, psychiatric disturbance characterized by mixed character disorder with predominant schizoid features, high neuroticism, and unsatisfactory relationships. Although these initial studies were an important beginning, the high prevalence of psychopathology in some reported samples did not seem applicable to significant numbers of CPP patients seen in practice. The findings are difficult to interpret, partly because there is a lack of clarity concerning the operational definition of CPP that was used. Biases in patient selection and interviewer information, inadequate control groups, and the absence of diagnostic laparoscopy also contribute to the confusion. Despite these shortcomings, it seems apparent that disorders of personality, especially borderline personality, are overrepresented both in the general population of severe chronic pain patients and in the population of those with CPP.

In primary care, such patients usually are seen less often. In any case, a label of personality disorder should not be applied indiscriminately to every angry patient by her frustrated physician. People who have difficulties maintaining satisfactory relationships and function in life, even when these difficulties are caused in part by subsyndromal personality problems, can be more vulnerable to nociceptive signals from tissue damaged by endometriosis, infection, or surgery. Unmet dependency needs may lead them to seek external solutions such as medications and further surgery, rather than to rely on their own impaired coping skills.

Depression

Focusing specifically on a CPP sample that had been evaluated by diagnostic laparoscopy, Walker and associates found that women with CPP (with and without positive laparoscopic findings) met criteria for lifetime

major depression, current major depression, lifetime substance abuse, adult sexual dysfunction, and somatization more often than did control subjects. Stout and Steege found that 59% of 294 women seeking evaluation at a pelvic pain clinic scored in the depressed range (>16) on the Center for Epidemiologic Studies Depression Scale at the time of their initial visit. Slocumb and colleagues reported that patients with an abdominal pelvic pain syndrome scored higher on scales of anxiety, depression, anger-hostility, and somatization on the Hopkins Symptom Checklist.

Because no study of CPP has assessed its association with depression over time, no statement can be made as to whether depressive symptoms are a predisposing factor leading to, or a reaction to, the pain condition. There seem to be two distinct groups of CPP patients: one in which pain and depression are common final presentations reached by a number of pathways and another in which depression develops in reaction to pain, as is the case with many other acute and chronic medical diseases.

History of Sexual Abuse

Women seeking treatment for CPP have a high prevalence of sexual trauma in their personal histories. In Reiter's study of 106 women with CPP, 48% had a history of major psychosexual trauma (molestation, incest, or rape) compared with 6.5% of 92 pain-free control subjects presenting for annual routine gynecologic examination ($P < 0.001$). The high prevalence of reports of psychosexual trauma elicited from CPP patients supports the hypothesis that pelvic pain is specifically and psychodynamically related to sexual abuse. However, Rapkin and colleagues did not find a higher prevalence of childhood or adult sexual abuse in a group of women with CPP compared with women with chronic pain in other locations. These findings argue against a unique relation between sexual abuse and CPP and suggest that abusive experiences promote the chronicity of many different painful conditions.

When such a history is noted, the clinician and patient together must judge whether the feelings surrounding these events are intense enough to intrude upon the present. If so, psychotherapeutic help may be indicated. If not, although the memories may be painful, further emotional work on this area may not be beneficial. The literature on the sequelae of abuse and subsequent treatment is disappointing, especially when the abuse occurred in the distant past. In any case, it is difficult to judge whether these events are directly relevant to present pain and hence demand attention or whether they contribute to a psychologically vulnerable substrate influenced by subsequent physical and emotional events. In these circumstances, it may be worthwhile to suggest further mental health evaluation as an exploratory measure, being careful not to imply that the patient is being referred because the physician is certain that the abuse is related to the development of the pain.

Sexual Dysfunction

In clinical practice, women presenting with CPP often report a high incidence of marital distress and sexual dysfunction, particularly dyspareunia. Stout and Steege found that 56% of 220 married women scored in the maritally distressed range (<100) on the Locke-Wallace Marital Adjustment Scale at the time of initial visit. A high level of marital distress has also been reported in other chronic pain patients and their spouses. Although some women report satisfactory sexual functioning before the onset of pain symptoms, others appear to have longstanding impairments in sexual response. In our experience, sexual difficulties are often the problem that makes a person seek (or is encouraged by her partner to seek) help for her pain.

DIAGNOSTIC STRATEGIES

Recognizing a Chronic Pain Syndrome

Many women can experience pain for longer than 6 months without becoming debilitated; although their pain is chronic, such women are not described as having a CPS. The following are the common clinical hallmarks of true CPS:

1. Duration of 6 months or longer
2. Incomplete relief by most previous treatments
3. Significantly impaired physical function at home or at work
4. Signs of depression (sleep disturbance, weight loss, loss of appetite)
5. Hypersensitive response to nociceptive stimuli
6. Altered family roles

Of the signs of depression, sleep disturbance is usually the first to appear. Careful questioning is needed to distinguish awakening caused by pain from awakening that just happens. In the true vegetative sign, the person usually cannot get back to sleep even if pain is relieved (by medication or other means).

The alteration of family roles is perhaps the most important of those mentioned. This includes changed responsibilities for household, children, finances, and so forth. Initially intended as helpful, such changes may eventually diminish the patient's self-esteem and progressively reduce her family's interactions with her to little more than checking on her pain. Over time, this covertly reinforces the symptom of pain and imparts to it unintended value as a major means of maintaining communication within the family.

Simultaneous Medical and Psychosocial Evaluation

When the aforementioned markers of CPS are present, one should surrender the need to immediately discover how much of the pain problem is physical and how much is psychological. Rather than guess, it is useful to ask two separate questions: Is there physical disease that requires medical or surgical treatment? Is there emotional or psychological distress that requires treatment?

It is useful to directly state that the precise connection between these two cannot be measured; this can help diminish the patient's fear that she will be told "it's all in her head." In one sense, that statement is true: Pain is, by definition, a product of the brain, spinal cord, and peripheral nervous system, but few patients nefariously endorse nonexistent symptoms for secondary gain. A provider telling a patient he or she believes her pain symptoms are real—they might be modified

by a host of psychological, experiential, or genetic factors that influence the interpretation of peripheral stimuli, but they are real—can establish an important therapeutic bond. The patient may then be more open to sharing her personal and emotional concerns. If this statement is made early in the evaluation, before all physical evaluations have been carried out, the patient is likely to be less defensive. In this framework, a mental health consultant has a better chance of developing rapport with the patient and will be a more helpful collaborator when needed.

History Taking

The site, duration, pattern during activities, relation to position changes, and association with bodily functions are all important elements of pain. For example, pain that is absent in the morning but worsens progressively during the day may be associated with pelvic floor muscle dysfunction, while a tender "spot" of dyspareunia might be related to nodular cul-de-sac endometriosis.

The chronology of a patient's pain is critical. As CPS develops, pain can be present over a progressively larger area despite stable visible pathology. Interpreting this as the breakdown or wearing out of physiologic systems that deal with pain signals has some biologic validity and may make sense to the patient. The clinician may need to counter the idea patients sometimes have that endometriosis "flares" like rheumatoid disease or spreads like a malignancy.

From a cognitive perspective, it is invaluable to discern the patient's and her family's ideas about the causes of and future for her pain. Fears of cancer can be discovered even if this diagnosis was never even remotely considered by the clinician. Less dramatic but equally powerful attributions of cause can emerge, such as pelvic infection that is due to sexual acts remote in time, arguments with a spouse, divine retribution, and so forth.

Physical Examination

It can be helpful to begin the exam with the back, evaluating for tenderness of the spine, paraspinous muscles, and sacroiliac joints. This may identify pain generators and sites for therapeutic intervention, but it also allows for touch to begin in a very nonthreatening way. Gynecologic assessment is uncomfortable to some degree for almost all women, but the patient with CPP is particularly vulnerable. Moving from back to abdomen to pelvis can establish trust and reduce fear. The abdominal wall is examined with and without flexed rectus muscles. A positive Carnett sign (increased tenderness when palpation is done in the presence of abdominal wall flexion) implies at least a contribution to the pain from abdominal wall myofascial sources. Decreased pain during this maneuver implies a higher contribution from visceral sources. On occasion, gentle fingertip palpation of the abdominal wall can detect such trigger points in the musculature. Rarely, a subcutaneous abdominal wall endometrioma is discovered, a diagnosis supported by a history of predictable, cyclic focal tenderness.

Pelvic exam then begins with external review of the vulva and vestibule. Gentle palpation with a cotton swab can detect areas of sensitivity consistent with vestibulitis in the introitus or trigger points higher in the vagina.

Guiding a patient through contraction-relaxation sequences of the abdominal, thigh, and vaginal introital muscles can reduce the discomfort of the examination and can indicate the patient's degree of control over muscle tension. Single-digit palpation of the levator plate, piriformis, and obturator muscles can elicit the tenderness of pelvic floor tension myalgia. This condition can present as a sequel to some other pelvic pain or a problem in itself. Discomfort is usually felt as pelvic pressure and radiation pain to the sacrum, near the insertions of the levator plate muscles.

Single-digit palpation should also be used to discover areas of tenderness in the cervix, uterus, and adnexa as well. Premature addition of the abdominal hand to the exam adds in nociceptive signals from abdominal wall myofascial components that may lead the examiner to overattribute pain to the viscera. Finally, the abdominal hand is added to assess size, shape, and mobility of pelvic structures. Adnexal thickening and mobility, pelvic relaxation, coccygeal tenderness, and foci of pain that reproduce dyspareunia should be noted. A rectovaginal exam is important when DIE is suspected.

During all components of the physical exam, it is important to not only ask the question "Does this hurt?" but also ask the questions "Do you feel pain where I am pressing or somewhere else?" and "Is this the pain you were describing earlier—is this *your* pain?" If a patient answers affirmatively to the final question, it can be helpful to point out what structure you are palpating (e.g., pelvic floor muscle vs. ovary).

Laboratory Tests

Imaging Studies

In the case of CPS, it has already been established that intensity of pain does not correlate well with extent of visible pathology. It follows that if the physical examination is relatively benign and is not severely limited by body habitus, extensive imaging usually adds little to the database needed before laparoscopy is performed. This is especially true in the case of organ-specific studies (intravenous pyelography, barium enema, colonoscopy) in the absence of symptoms or signs pointing to an explicit organ system (e.g., blood in the stools). Ultrasounds, CT scans, and MRIs can, at times, discover unrelated or nonspecific items misinterpreted by the patient and physician. Understandably limited by the setting, many patients have been told by emergency physicians her

pain is due to ovarian cysts. This explanation is supported by an imaging study— either (a) a cyst (physiologic or not) is present or (b) a small amount of fluid is seen, interpreted as cystic rupture. Intervening for CPP on the basis of an imaging study finding alone is unlikely to be fruitful. On the other hand, with a specific question in mind—for example, pelvic sonography to confirm ovarian endometrioma, MRI when adenomyosis is suspected, or lower endoscopic ultrasound to rule out invasive rectal endometriosis—imaging can be quite helpful.

Blood Studies

Relatively few hematologic or chemical measures are of use in diagnosing CPP. An elevated leukocyte count and erythrocyte sedimentation rate may make the clinician suspect chronic pelvic inflammatory disease even when cervical probes are negative for the most common sexually transmitted infections. Serum cancer antigen 125 (CA 125) can confirm suspicions of DIE in those without prior surgical evaluation but is not sufficiently sensitive to detect early-stage disease. In those with advanced endometriosis, anti-müllerian hormone levels can help fertility counseling in a woman considering extirpative surgical treatment for her disease. In patients with post-bilateral oophorectomy with remnant ovarian tissue, FSH and estradiol levels remain in premenopausal ranges. Women using replacement estrogen therapy should stop 3 weeks before these levels are measured.

Anesthetic Blocks

Injection of small volumes of a local anesthetic, 1 to 5 mL of 1% lidocaine or 0.25% to 0.5% bupivacaine, blocks pain from either an entrapped segmental nerve (e.g., ilioinguinal) or an

abdominal wall trigger point. Such blocks can be therapeutic as well as diagnostic. Many anesthesia pain clinics administer epidural or spinal anesthetics to distinguish pain arising from peripheral organs from pain that has become completely central in origin.

In some instances, it is useful to attempt diagnostic/therapeutic transvaginal blocks with the same local anesthetics for vaginal apex pain, as discussed above. A series of three or four blocks administered 1 to 2 weeks apart may provide durable relief in some instances.

In most cases, a history and careful routine physical examination distinguish central from lateral sources of pelvic pain. When this discrimination is difficult, it may be useful to administer a transvaginal uterosacral block (blocking most uterine innervation) and then repeat the pelvic examination. When this relieves the pain, the pain can be assumed to arise from the uterus. If the pain is not relieved, however, one cannot distinguish a failed block from pain of nonuterine origin.

Psychological Tests and Interviews

To distinguish physical from psychological contributions to pain, many studies of CPP have used traditional psychological instruments that were developed to measure general psychopathology or personality factors. In some studies, more psychological abnormalities are detected in women without visible pathology at laparoscopy. In other studies, women with positive laparoscopy who have had pain for a long time appear equally distressed in their questionnaire responses. These psychometric instruments generally have uncertain face value for chronic pain patients, and their use can support the patient's fears that the health care provider thinks she is "crazy" or that the pain is imagined. Once again, the question of whether the emotional distress identified by these instruments is an antecedent to or a consequence of persistent pain remains unanswered. Psychometric tests are most useful when they are interpreted by a psychologist who has interviewed the patient, and they serve best as a means to better understand the patient's strengths and weaknesses, rather than as a means to decide who has "organic" versus "psychological" pathology or who needs surgery.

Laparoscopy

Great strides have been made in operative laparoscopy in the past three decades. Laparoscopy can be useful diagnostically and therapeutically (even in the face of negative findings), but when a CPS is clinically evident, results of laparoscopic treatment alone, despite comparable pathology, are much less impressive. For a patient with the clinical markers of CPS listed earlier, the complete workup as described should be performed before laparoscopy.

In some puzzling cases, we have performed laparoscopy under local anesthesia to "pain map" the pelvis. A 2-mm laparoscope and a small suprapubic probe are placed with the use of short-acting, reversible intravenous analgesia (e.g., remifentanil) and local anesthetic. Having been oriented to the procedure beforehand, as each organ is touched, the patient is asked if the site is painful, to rate it on an ordinal scale from 1 to 10, and if the discomfort represents her pain. It is possible in some cases to block the superior hypogastric plexus during pain mapping to better predict benefit from presacral neurectomy. In this approach, mapping is done before and after injecting 10 mL of 1% lidocaine just underneath the peritoneum over the sacrum, using a 7-inch, 22-gauge spinal needle.

Limiting patient characteristics for using pain mapping include high states of anxiety and obesity, where the torque required to move an instrument against a thick abdominal wall can provide distracting nociceptive information. Once thought to be the "holy grail" of CPP diagnostics, better understanding of the central mechanisms of chronic pain and the relationship among various named pain disorders has led to diminished enthusiasm for laparoscopic pain mapping in routine practice.

MANAGEMENT

Relatively straightforward pain problems are not challenging to manage, such as treating isolated dysmenorrhea with hormonal suppression or a chronic tuboovarian abscess with adnexectomy. More often, however, CPP represents a complex and nuanced syndrome where treatment may vary considerably depending on the patient. When pain itself is the disease, the goal of treatment is not necessarily complete eradication of pain, but rather finding strategies that afford more functional living. Neuromodulatory medications (e.g., tricyclic antidepressants, neurotransmitter reuptake inhibitors, neuroleptics), psychological adjuncts (e.g., cognitive-behavioral therapy, pain psychotherapy, sexual counseling), and complementary strategies (e.g., mindfulness-based medication, yoga, acupuncture) can be useful to dampen central hypersensitivity. For the peripheral elements, physical therapy, diet modification, peripheral nerve blocks, and surgery can be helpful depending on the target pain generator. In all cases, good sleep hygiene, exercise, healthy eating, and social support are important foundational elements that improve the effectiveness of CPP treatment.

General Principles

A complete evaluation of CPS often reveals a number of contributing factors, such as bladder irritability, irregular bowel function, poor posture, and emotional and relationship stresses, in addition to laparoscopically visualized pathology. Treating each component sequentially is common practice but often ends in frustration because each treatment addresses only a part of the problem. Simultaneous treatments often begin with disquieting multimodal therapy but allow better relief. Close follow-up at regularly scheduled visits provides support and a coping mechanism for the patient. When the patient is essentially required to feel worse to be seen again, the pain may be tacitly reinforced.

Medication Use

Analgesics

Analgesics such as nonsteroidal anti-inflammatory drugs and opioid narcotics can be quite effective for acute conditions, but their use in chronic pain is marred by a host of adverse outcomes and limited efficacy associated

with long-term use. Dose-related effects of medications such as acetaminophen (liver toxicity) and cyclooxygenase inhibitors (gastric and renal damage) are well known, but they are not major offenders in the realm of tolerance and withdrawal. Opioid narcotics, on the other hand, are notoriously dangerous in regard to these consequences, in addition to problems such as narcotic bowel syndrome and opioid-induced hyperalgesia. Some patients benefit from structured narcotic therapy, but, if they are to be used for CPP, clinicians should screen carefully for factors associated with high risk for misuse and set nonnegotiable ground rules, such as limiting prescriptions to one provider, not entertaining requests for early refills, mandating scheduled urine drug screens, and refraining from uncontrolled dose escalation. Newer opioid medications, such as oxymorphone and tapentadol, are considered less euphoria generating than the more commonly employed narcotics such as hydrocodone, oxycodone, and hydromorphone.

Antidepressants and Neuroleptics

This class of drugs is commonly employed in the treatment of chronic pain, including CPP, although few controlled trials have been carried out specifically in this population. While no longer first line for mood disorders, tricyclic antidepressants such as amitriptyline, nortriptyline, and desipramine have a long record of being effective in treating chronic pain. Newer-generation neurotransmitter reuptake inhibitors such as duloxetine and desvenlafaxine can also be useful. Neuroleptics such as gabapentin, pregabalin, and lamotrigine are generally employed when symptoms are more specifically neuropathic in nature. It is important to discuss with patients that, although they tend to diminish with continued use, all of these medications have central side effects, some of which are predictable and others quite idiosyncratic. When higher doses or multiple agents are used, it can be helpful to consult with a psychiatrist or psychopharmacologist to avoid complications such as severe mood dysregulation or serotonin syndrome.

Anxiolytics

Anxiolytic drugs are certainly widely prescribed by gynecologists, although it is uncertain how often they are given for pain. In one study, alprazolam, a triazolobenzodiazepine with mixed anxiolytic and antidepressant effects, had a surprising degree of analgesic effect in moderate-to-high doses in patients with chronic pain of malignant origin and concomitant mood changes or anxiety. These patients were already receiving narcotics, which may suggest that alprazolam potentiates the analgesic effect of narcotics. Their role in conjunction with nonnarcotic analgesics is uncertain, and the addiction potential is obvious.

Hormonal Medications

Oral contraceptives are effective in reducing dysmenorrhea and cyclic symptoms associated with endometriosis. It is not uncommon, however, to meet resistance to using these medications in women with CPP—either because they were previously used and did not cure the entirety of the pain syndrome and were thus deemed ineffective or because of sensitivity to side effects such as nausea, an understandable consequence in a viscerally hypersensitive group. Avoiding ultra-lowdose 20-mcg ethinyl estradiol formulations can help reduce unscheduled bleeding, important for women who may closely associate bleeding with pain. Outside of DIE, progestin-only formulations, by enteral or parenteral route, run the risk of exacerbating depressive symptoms in a vulnerable population. A notable exception includes the levonorgestrel intrauterine system (LNG-IUS), which has little systemic absorption and can control dysmenorrhea and pain from endometriosis in a low-risk, reversible, long-acting manner.

The use of GnRH agonists deserves special mention. They have been recommended to distinguish gynecologic from nongynecologic sources of pain; however, these agents also relieve symptoms of other functional conditions. Furthermore, pain thresholds have been shown to be lower premenstrually, even in asymptomatic

women. The impact of the menstrual cycle itself in chronic pain patients has not been well explored, but it seems likely that it may impart some cyclicity even to conditions unrelated to the reproductive tract. Cyclicity of symptoms must therefore be interpreted with caution, and the disappearance of symptoms or of their cyclicity by pharmacologically obliterating the menstrual cycle does not demonstrate a gynecologic cause. To address the most common clinical circumstance, relieving pain with a GnRH agonist does not prove that the pain is due to endometriosis, nor does it prove that pain comes from the reproductive tract, as discussed above. Gonadotropin-releasing hormone agonists can be useful when differential diagnosis includes ovarian remnant syndrome or residual ovary syndrome or in the treatment of DIE, but its utility in treating general CPP is limited by cost and morbidity. Contrary to popular belief, GnRH agonists are not more effective than other more benign hormonal manipulations directed at pelvic pain. For DIE, aromatase inhibitors may work similarly, without as significant hypoestrogenic effects.

Surgery

Two basic surgical approaches have been used to treat CPP: removing pelvic organs and treating visible disease while leaving the pelvic organs in place. The use of both approaches is guided by clinical experience, as scientific data regarding the role of surgery in pelvic pain are sparse. Expansion of literature on the topic, including stratification for characteristics that lead to strong or poor response, would be most welcome.

In the United States, approximately 12% of hysterectomies are performed with pelvic pain as the primary indication. An additional 6.1% are performed for endometriosis or adenomyosis, and 5.1% are performed for pelvic inflammatory disease; no doubt many in these two categories also involve symptoms of pain. In approximately one third of hysterectomies performed for pain, no pathology is found. Despite the frequency of pain as an indication for this procedure, data regarding efficacy are surprisingly sparse. One report notes relief in 78% of women after hysterectomy for pelvic pain of uterine etiology (women with adnexal or other pelvic disease were excluded). Interestingly, the presence or absence of uterine pathology (adenomyosis or leiomyomata) had no bearing on whether pain was relieved.

Others have suggested hysterectomy performed in primary care settings is very effective for the treatment of CPP. In a prospective observational study of private practices in Maine, Carlson and associates reported that at a 1-year follow-up, satisfaction with the outcome of surgical treatment was much higher than satisfaction with the outcome of medical therapy. However, approximately one third of women improved substantially on medical therapy; perhaps, women were more likely to undergo operation when visible pathology was apparent. In the Maryland Women's Health Study, 1,299 women were interviewed at length before hysterectomy for benign disease and at 3, 6, 12, and 24 months after surgery. In more than 90% of cases, the procedure was well tolerated and did not result in postoperative depression or a decline in sexual functioning. In the subset of women with pain as the primary indication for surgery, relief of pain occurred in more than 80%, indicating that the clinicians involved generally used good judgment and technique. In general, women with preoperative depression or sexual dysfunction did not fare as well as their less symptomatic counterparts, although even when hysterectomy is performed in women with both depression and CPP, slightly more than 80% are improved emotionally and in terms of pain at 2-year follow-up.

Rigorous study design is lacking regarding data on adhesiolysis, with reports essentially limited to little more than clinical case series. When treated by laparotomy, 28 of 42 (65%) patients reported cure or improvement of pain. In a sample of mostly primary care patients, 84% of 65 patients had relief of pain after laser laparoscopic adhesiolysis with follow-up intervals of 1 to 5 years. In Sutton's large series, 85% had pain relief at 1 year. Steege and Stout reported that 15 of 20 (75%) patients without a CPS who were undergoing laser laparoscopic adhesiolysis had good relief of pain at a follow-up 6 to 12 months after surgery. However, if a CPS was present, only 4 of 10 (40%) patients with equivalent adhesive disease

obtained relief. The greater the emotional and behavioral disability, the greater the need for combined medical, surgical, and mental health management.

Endometriosis is a more common finding among women undergoing laparoscopy for pelvic pain than those for other indications. Peritoneal disease does not correlate with symptom site or stage of disease with symptom severity. Laparoscopic treatment, even of mild disease, is more effective than diagnostic surgery alone. Some studies suggest superiority of excision to ablation of endometriosis implants, and this approach probably makes good clinical sense, but there is insufficient evidence to conclude one is definitively superior to the other in pain reduction. Specific symptom improvement following DIE (gastrointestinal, urologic, cul-de-sac) resection is best documented in the literature. The benefits of surgical excision may be prolonged by subsequent medical therapy. Numerous studies have been done of postsurgical treatment with hormonal medications. Oral contraceptives, danazol, progestins, and GnRH agonists (with and without add-back estrogen/progestin) have all been shown to be effective. Although the GnRH agonists have become perhaps the most widely used of these, definitive evidence for their superiority is lacking. The more economical and less physiologically intrusive approach would seem to favor sex steroids over GnRH agonists. Most troublesome in reviewing all of these studies is the observation that dyspareunia is the symptom that is most refractory to treatment, testifying to its multifactorial nature.

Presacral neurectomy, as an adjunct to surgical excision of endometriosis, has been evaluated for its effect on pelvic pain. In a retrospective sample of 71 women undergoing conservative resection of endometriosis by way of laparotomy, 35 (50%) who also had presacral neurectomy enjoyed significantly greater improvement in both dysmenorrhea and dyspareunia. Two subsequent retrospective reports noted that similar percentages (approximately 75%) of women obtained pain relief after endometriosis surgery that included presacral neurectomy, compared with approximately 25% who obtained relief without neurectomy. Zullo et al. investigated the question with a double-masked randomized trial and demonstrated a 20% difference in pain improvement when presacral neurectomy was added to endometriosis excision in women with central pain.

An ovarian remnant should be removed if it is persistently symptomatic despite attempts at medical suppression and if menopause cannot be expected in the patient’s near future. The dissection should be detailed and should include all the peritoneum surrounding the mass. The pararectal space, and paravesical space if needed, should be opened systematically, and the ureter and pelvic sidewall vessels exposed and carefully freed from the specimen. Usually, there is vascular supply along the tract of the IP, and it is prudent to divide the pedicle well above the pelvic brim. When a GnRH agonist has been used preoperatively for symptom control or to distinguish the relative contributions made by the remnant and other pelvic pathology, such as adhesions, the remnant tissue may become so small as to make it difficult to identify. Hence, if a palpable (or ultrasonically visible) mass disappears after GnRH agonist treatment, it may be wise to allow time for it to regrow before pursuing surgical excision. When the remnant is small, some surgeons have stimulated the remnant with clomiphene citrate to make it easier to find.

Psychological and Alternative Treatments

Psychological disorders should be treated in CPP, whether independently present or the result of a long-standing pain disorder. Some practitioners may find cognitive-behavioral or biofeedback therapy useful in reducing automatic responses to painful stimuli. Sexual counseling, couples counseling, and psychotherapy can be helpful adjuncts. Alternative strategies, such as mindfulness-based meditation, yoga, and acupuncture, all have their appropriate roles in individual cases, but none is so clearly applicable or effective that its automatic use is supported in cases of CPP.

Management Overview

The most effective clinical approach requires simultaneous treatment of as many factors as possible: anatomic, musculoskeletal, functional bowel and bladder, psychological, and so forth. The patient and physician must contract for the long term and work from a rehabilitation perspective, rather than hope that the latest single addition to the treatment will prove to be *the* answer. The physician, to prevent frustration and feelings of defeat, must often play the role of helping to manage and relieve the pain while helping to maximize function, even when pain persists. To the surgically trained gynecologist who prefers a clear-cut single answer to a clinical problem, this can be the most difficult part of dealing with the problem of CPP. It is important to free oneself from the responsibility of needing to "fix" a patient's CPP. While the compassionate provider can be an invaluable aide, much of the work in improving from pelvic pain is the burden of the patient herself.

THE EVOLUTION OF A CHRONIC PAIN SYNDROME

As is apparent from this discussion, CPP is a heterogeneous problem, not a single diagnosis, and no single etiologic hypothesis is clearly supported. Most of the hypotheses reviewed here have some credible evidence supporting them; none have been sufficiently validated.

Some patients appear to have a pure version of one or the other of the syndromes described, whereas many others present with several or many simultaneously. Psychological and neurologic mechanisms are proposed here to explain how the evolution of chronic pain may occur, regardless of the particular tissue damage or functional disorder that may first have provided nociceptive stimuli. We suggest the following elements (**Fig. 29.1**): biologic events sufficient to initiate nociception, alterations of lifestyles and relations over time, recruitment of neighboring organ systems, anxiety and affective disorders, and a circular interaction (vicious cycle) among these elements.

Biologic Events Sufficient to Initiate Nociception

Sexually transmitted diseases, endometriosis, recurrent bladder and vaginal infections, primary or secondary functional dyspareunia, alterations of bowel habit, muscular dysfunction, pelvic congestion, and gynecologic or other abdominal surgeries (**Table 29.1**) may contribute individually or in combination.

Alteration of Lifestyle and Relations

Physical activities at home and recreational pursuits can suffer. Believing that rest usually helps in the treatment of most causes of acute pain, the patient may assume that the same applies to chronic pain and may thus restrict herself more than actual discomfort dictates. Family members start to regard the patient as sick and leave her out of many activities, thus reducing her

roles within the family structure. With time, concern for and discussion of her pain can become the family's major pattern of communication with the pain victim. If sexual intimacy has been the major means of emotional sharing and smoothing over of differences, and if this intimacy is reduced, then the altered pattern of interactions may take hold more quickly.

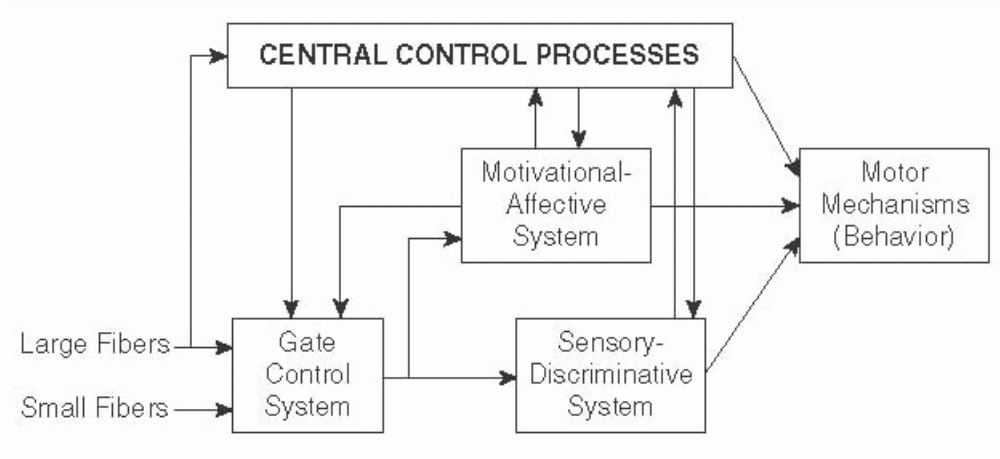

FIGURE 29.1 The gate control theory of pain perception.

Anxiety and Affective Disorders

Depression can occur as a cumulative result of the disability suffered, or the pain can bring on an episode of depression in a patient already biologically vulnerable. The observation most relevant here is that pain patients with a family history of depression can derive the most benefit from antidepressants.

The Vicious Cycle

Diminished activity, altered family roles and social supports, anxiety, and affective disturbances can influence nociception by a variety of central pathways, ultimately altering spinal cord "gating" of nociceptive signals. Cognitions about the pain can play an additional role.

Several important modifying influences can be present in addition to these major pathways (**Fig. 29.2**). Incest and other forms of sexual abuse have attracted the most attention as possible forerunners of CPP. However, CPP is clearly not a unique or specific sequel to sexual abuse, and a large proportion of CPP patients have not been abused in this manner. Victims of sexual abuse have many negative emotional sequelae; pain problems often occur after abuse, but they are not necessarily directly caused by the abuse.

A genetic predisposition to depression also allows the vicious cycle to become easily established and strengthened over time. Antidepressant medications play an important role in the overall therapeutic plan in such cases. A relatively new area of research in pain is dealing with possible genetically determined variations in central neurotransmitter processes that may predispose to the development of pain syndromes.

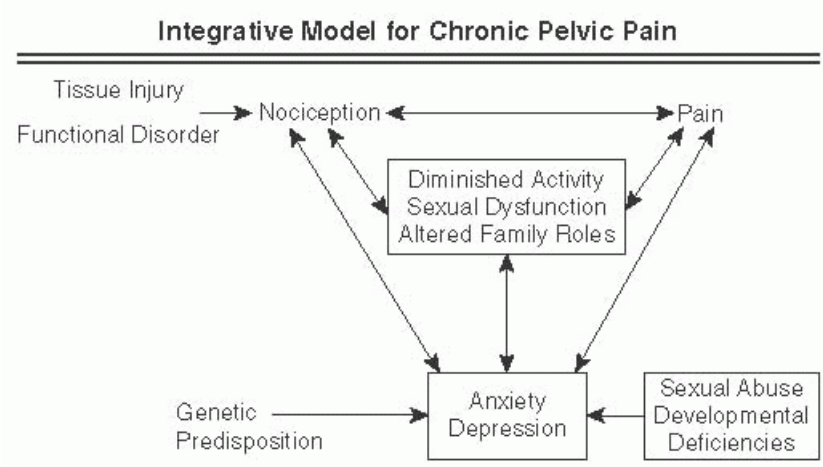

FIGURE 29.2 An integrative model for CPP, including elements of gate control theory, cognitive-behavioral theory, and the operant conditioning model.

Several authors have suggested that the concept of perceived control best explains the development of affective changes accompanying chronic pain, regardless of the location of the pain. The individual who sees herself as having little control over the physical and emotional events affecting her may be most vulnerable to development of a CPS. Another factor thought to influence pelvic pain is a patient's degree of catastrophization, a state of interpreting negative things are far worse than they are. It may be reasonable to consider these variables as a culmination of the effects of affective change, activity, family roles, sexual dysfunction, and previous victimization experiences.

The longer that pain has been a part of the person's life, and the more psychological vulnerabilities she carries forward to the present, the less likely it is that any treatment of the tissue damage itself will be effective in relieving pain and restoring physical and emotional function. However, as treatment studies show, the contribution of peripheral generators to chronic pain can seldom be dismissed entirely. The more difficult task is the selection of an efficient and cost-effective combination of treatment approaches aimed at the most important factors acting in the present.

BEST SURGICAL PRACTICES

- A complete medical and psychosocial history, as well as a pain-oriented physical and pelvic examination, should be completed before diagnostic laparoscopy is performed.
- Neuropathic and musculoskeletal components of CPP often require treatment both before and after appropriate pelvic surgery.
- A minimally invasive (laparoscopic) approach is especially appropriate for chronic pain patients.
- Laparoscopic treatment of endometriosis is more effective than diagnostic surgery alone.
- Presacral neurectomy can be an effective adjunct to endometriosis excision when a central component of pain is present.
- Resection of DIE is effective treatment of organ-specific symptoms.
- Complete skeletonization of the IP vessels, especially in difficult oophorectomy, reduces the risk of adjacent organ injury and ovarian remnant syndrome. Ovarian remnants should be removed with careful opening of avascular spaces and identification of retroperitoneal structures.
- Though not a cure for all components of a woman's CPP, hysterectomy can be an effective treatment, even in women with both depression and pain.

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