Dyspareunia affects 8–22% of women at some point during their lives, making it one of the most common pain problems in gynecologic practice. A mixture of anatomic, endocrine, pathologic, and emotional factors combine to challenge the diagnostic, therapeutic, and empathetic skills of the physician. New understandings of pain in general require new interpretations concerning the origins of pain during intercourse, but also provide new avenues of treatment. The outcomes of medical and surgical treatments for common gynecologic problems should routinely go beyond measures of coital possibility, to include assessment of coital comfort, pleasure, and facilitation of intimacy. This review will discuss aspects of dyspareunia, including anatomy and neurophysiology, sexual physiology, functional changes, pain in response to disease states, and pain after gynecologic surgical procedures.

Pain during intercourse is one of the most common complaints in gynecologic practice. Together with chronic pelvic pain, it is also one of the more difficult clinical problems to assess and successfully treat. This review will discuss the following aspects of dyspareunia: anatomy and neurophysiology, psychological influences on sexual functioning, sexual physiology, functional changes, pain in response to disease states, and pain after gynecologic surgical procedures.

A systematic review of studies of dyspareunia, done by the World Health Organization, reported an incidence of painful intercourse ranging between 8% and 22%. A study of point prevalence in Sweden, involving 3,017 women, showed a peak incidence of 4.3% in the 20–29 year age groups, with lower numbers reported for each subsequent decade. In that study, 39% saw a physician or midwife, 20% recovered after treatment, and 31% recovered spontaneously. In many instances, women did not bring the complaint to the attention of their health care providers. Current practice of medicine in the United States certainly involves limitations of time, opportunity, and skill that would likely mirror these results.

The present focus will be on the evaluations of painful vulvar, vaginal, and pelvic conditions that often manifest as pain during sexual intercourse. When discussing vulvar and vaginal function, the absence of pain during intercourse is a necessarily narrow view. This discussion does not focus upon, but does not forget, the fact that sexual relations are a complex part of an intimate personal relationship.

NEUROPHYSIOLOGY
The external vulva structures demonstrate well-known homologies with male genitalia. Less well known is that the vaginal vestibule originates from the same embryologic anlage as the urethra and the bladder, perhaps explaining the sometimes observed simultaneous appearance of symptoms in both of these areas. Both areas have estrogen receptors, but in titers lower than those found in the vagina.

The nerve supply of the vulva is redundant, but dominated by the branches of the pudendal nerves. Although A delta fibers are prevalent in the vulva, C
fibers, which innervate the viscera, are well represented in the vagina and cervix and to some degree in the vestibule. These fibers are predominantly silent in terms of sensing pain, but with repeated mechanical or chemical stimulation they may come to convey nociceptive signals to the spinal chord. Of particular interest is animal evidence showing that afferents from the reproductive, urinary, and gastrointestinal tracts impinge on the same spinal segments served by nerves from the skin and muscles from the lower limbs, back, abdomen, and peritoneum. These same spinal neurons are known to be influenced by neurons from other segments of the spinal chord and widespread areas on the brain. These observations take us away from the usual rigid interpretations of innervation, and open the door to understanding of some of the peculiar patterns of pain that sometimes present in clinical practice.

At a physiologic level, the more recent concept of neuroplasticity has similarly taken our understanding of chronic pain away from static interpretations and helped us understand that the evolution (especially spread and worsening) of a pain problem may include physiologic alterations in the nervous system quite aside from the biochemistry of affective disorder and the psychology of human personality and relationships. For example, under stress, repeated subthreshold negative stimuli may result in central sensitization, with the result that previously comfortable stimuli may become painful, without requiring changes in peripheral tissues.

Collectively, these observations may help us understand the now common clinical finding that striated muscle groups (eg, pelvic floor and abdominal wall) can become involved in chronic pain syndromes in the pelvis in general and in dyspareunia in particular. Similarly, they also provide the theoretical basis for observations of changes in visceral sensations in structures such as the vaginal vestibule, the cervix, the vaginal apex after hysterectomy, the introitus after obstetric trauma, and the entire vagina after pelvic support surgery.

PSYCHOLOGICAL INFLUENCES ON DYSPAREUNIA

The distress associated with painful intercourse is certainly an important factor regardless of the origins of the problem. Anxiety has been shown to be an independent predictor of the pain of dyspareunia, aside from structural factors. Marital distress was noted to be higher in women without organic pathology as an explanation for their dyspareunia. However, some subsets of dyspareunia that have clear physiologic underpinnings, such as vulvar vestibular syndrome and postmenopausal genital atrophy, have little association with prior or concurrent psychopathology.

Given the current emphasis on posttraumatic stress disorder and its relationship to sexual and physical abuse, is perhaps surprising that a systematic review of 111 articles demonstrated a relatively weak association of sexual abuse with dyspareunia and pelvic pain. Indeed, two controlled studies failed to find any association between abuse or trauma and sexual pain.

For the clinician, these observations would suggest that although it is clinically important to ask women about histories of abuse of a physical or sexual nature, a positive answer requires further inquiry concerning any potential relationship to current pain or sexual complaints. A history of abuse does not preclude successful response to the many treatments for dyspareunia. Considering the above discussion of neurophysiology and its complexities, it seems evident that categorizing sexual pain as either psychologically or physically based becomes limiting on both theoretical and practical levels.

SEXUAL PHYSIOLOGY

The pioneering investigations of Masters and Johnson documented that vaginal lubrication is the product of the vaginal wall epithelium, not of the Bartholin gland or the endocervical glands. Adequate lubrication depends upon vascular supply of this epithelium, as well as estrogen.

The sex response cycle, as originally described at a physiologic level by Masters and Johnson, begins with sexual arousal. Kaplan added the preceding component of sexual desire, but still viewed the process as essentially linear, with a beginning, middle, and an end. More recent formulations think of it more in circular fashion, in which arousal may not always be preceded by desire. It is now felt that fully half of women may not necessarily experience sexual desire before the initiation of sexual contact, but may note the awakening of desire as stimulation and arousal begin. The absence of “desire” on the part of the woman, defined as preexisting interest in sexual contact and a perceptible physical need, is now regarded by mental health professionals as often normal. Many women with this pattern nevertheless find sexual contact pleasurable and desirable once it starts. These observations may be the source of considerable puzzlement even in the well-functioning couple, but may have even greater impact on the couple struggling to deal with problems involving painful intercourse.

Additional observations from the work of Masters and Johnson have clear bearing on our understanding

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of dyspareunia. They documented that the upper end of the vagina, during sex response, may lengthen by 3–4 cm and may widen to an overall width of 6 cm or more (Fig. 1). The anteverted uterus elevates in a cephalad direction. Together with the vaginal lengthening described, this may serve to move sensitive areas (eg, the posterior cul-de-sac with endometriosis) farther away from contact with the penis. When the uterus is retroverted, vaginal expansion and lengthening seem to occur more anteriorly, in the area between the bladder and the uterus. A more anteriorly directed angle of penile entry is likely to be more comfortable in this circumstance. This understanding of sexual physiology is the basis for some of the counseling suggestions described below.

CLINICAL EVALUATION

General History

As demonstrated by the Swedish study reviewed above, many women find it difficult to tell their health care providers about pain during intercourse. Including a question or two about sexual comfort as a routine part of every gynecologic visit legitimizes the subject and makes it easier for the patient to voice concern when there is a problem.

The history begins with a review of the precise location of the pain during intercourse. The differential diagnosis of pain at the vaginal introitus and vulva is, of course, entirely different from that for deep dyspareunia. The relationship to the menstrual cycle is important, especially when endometriosis or uterine disease is suspected, while understanding the timing within the sexual response cycle is paramount for understanding the effect of the pain on the individual’s and couple’s sexuality.

Reviewing the evolution of the pain over time (months, years) will often help clarify clinical symptoms. For example, dyspareunia may start with posterior cul-de-sac endometriosis, and over time, other areas such as pelvic floor and hip muscles may start to contribute pain signals. When the pain has become this complex, aggressive treatment of the only known “disease” (endometriosis) will often fail if the other components are not addressed.

Along with this history, one gathers a more complete picture of the resources the couple has brought to bear on the problem by asking about their interpretations regarding the cause of the pain, their attempts at solution, the nature of the conversation between them about the problem, and finally their expectations of the degree of effect of the problem on their relationship. Is sexual pain followed by caring and problem solving, or distance and anger? If this problem is not solved, what do they think would happen with the relationship?

Physical Examination Techniques

There may be only one physical element in simpler cases, but more often there is a list of factors, including abdominal, pelvic floor, or hip muscle dysfunction or pain, visceral functional disorders, and some inflammatory and/or structural causes.

At times, intrinsic sensitivity of the abdominal wall can be a problem, especially when couples use the male superior position for intercourse. Accordingly, examination of the abdomen should be included. Areas of point tenderness should be examined with and without the patient elevating her head off the table, which provokes contraction of the rectus muscles. If the discomfort is the same or increased with abdominal wall flexion, then the myofascial structures of the abdominal wall may be involved in pain generation.

In addition to customary visual inspection and palpation, if indicated by history, sensory mapping of the vulva and vaginal vestibule should be done with a cotton-tipped applicator. Careful retraction of the labia majora and minora is needed to adequately visualize the posterior vestibule. Erythema and excessive sensitivity of the vestibule tissue to the cotton tip applicator is present in vulvar vestibular syndrome, discussed below.

Inserting one index finger into the vagina just past the introitus, while asking for contraction and relaxation, allows assessment of her control of the bulbocavernosus muscles. Advancing the index finger allows palpation of the pelvic floor (levator) muscles at the 4–5 o’clock and 7–8 o’clock positions, looking for
reproduction of the coital pain. Uncontrolled levator contraction is often accompanied by pain, and may contribute to dyspareunia.

Palpating the urethra and base of the bladder produces some bladder pressure and urinary urgency, whereas in women troubled with painful bladder syndrome, the pain of the chief complaint will often be elicited by this maneuver. The examiner can often discriminate between urethral and bladder components.

A normal-appearing cervix may be sensitive if it has been involved in previous bouts of cervicitis, obstetric trauma, or conization or loop electrosurgical excision procedure. Gentle pressure with a cotton-tipped applicator will elicit abnormal sensitivity (alldynia) of the cervix.

Single digit transvaginal palpation of the adnexal areas on each side will reliably detect tenderness when any substantial adnexal pathology is present. Adding the abdominal hand too soon in this process will confuse the clinician by mixing together nociceptive signals from the myofascial structures of the abdominal wall with whatever signals might be coming from the uterus, adnexa, or other visceral structures.

Having completed the single digit vaginal examination, the abdominal hand may be added to further evaluate the size, shape and mobility of the pelvic viscera. Rectovaginal examination is a standard part of the pelvic examination and merits particular attention when deep dyspareunia is reported, because this will often detect posterior cul-de-sac endometriosis.

**DYSpareunia Syndromes**

**Dyspareunia Without Organic Pathology**

**Vaginismus**

This has been defined as “persistent or recurrent difficulties of the woman to allow vaginal entry of the penis, finger, or any object, despite her expressed wish to do so.” Traditionally, this has been felt to be the result of fear of pain, pelvic floor dysfunction, or behavioral avoidance. Primary vaginismus, (existing from the beginning of any sexual efforts), is often attributed to difficulties with upbringing and attributed to discomfort with sexuality in general. Sexual abuse is not necessarily involved. Secondary vaginismus is that which is reactive to a disease process (eg, vulvar vestibular syndrome) or relationship issues, beginning after a period of successful sexual relations. Of note, before vulvar vestibular syndrome was well described, many women with this disorder may have been mislabeled as having vaginismus.

Levator pain and/or spasm may also occur when the introital muscles have developed the pattern of involuntary contraction. However, the two muscle groups can indeed function quite independently. This means that introital vaginismus can exist without levator spasm and vice versa.

**Diminished Sexual Response**

A host of conditions can contribute to the diminution of sexual response in someone with a previous comfortable response pattern. These may include recurrent bouts of vaginitis, relationship changes or changes of partner, adverse effects of medications such as antidepressants and antihypertensive agents, hypoestrogenism secondary to progesterone-based contraception or medical therapy for endometriosis, and intrinsic bladder or urethral disease, such as interstitial cystitis and chronic urethral pain. Once established, this pattern of diminished response may then continue even if the provoking disease process is controlled.

**Menopause**

Estrogen maintenance or replacement after the age of menopause improves desire, lubrication, and the remainder of the sexual response cycle and diminishes or prevents dyspareunia as well. However, the effect on libido is more variable, because this aspect of sexuality certainly has many ingredients. Topical therapies to the vulva and vagina exert a local effect on vaginal comfort while provoking few systemic symptoms or adverse effects.

**Levator Spasm**

As mentioned, spasm of the levator muscles can accompany vaginismus. Perhaps more commonly, it can develop in the face of more internal visceral discomforts resulting from the presence of pelvic pathology or after surgery to correct this pathology. Once the muscular habit is established, it can certainly persist after its original cause is corrected. This is a common problem in women with daily pelvic pain, as well as those with dyspareunia.

The classic symptoms associated with levator spasm are pain with intercourse, a sense of pressure in the pelvis, or a sense of things about to “fall out.” The patient with levator spasm often cannot comfortably sit straight up in a chair, because this puts uncomfortable pressure on the levator muscles. She will often instead either lean to one side or slide forward on the chair so that the sacrum rests on the chair.

Treatment of pelvic floor muscle pain, in recent years, has rapidly become a major element in the
physical therapists’ contribution to treating dyspareunia and pelvic pain. An entire subspecialty in physical therapy has developed, in which female therapists work directly with pelvic floor muscles, using transvaginal and/or transrectal access to these muscles.

**Dyspareunia Related to Medical Illness**

Systemic illnesses that affect vascularity and/or mucus membranes may also affect the vagina. Examples include Sjögren’s syndrome, diabetes, and systemic inflammatory/autoimmune diseases. In Sjögren’s syndrome, vaginal dryness symptoms may at times precede ocular or oral symptoms, making diagnosis difficult. This is a disorder most commonly discovered in perimenopausal and postmenopausal women.

Diabetes is well known to affect the vagina in the sense of increased vulnerability to yeast infection. Less well known is its effect in terms of diminished lubrication, lower orgasmic frequency, and sometimes intrinsic cervical pain.

Recent literature has included some speculation regarding the role of nonspecific inflammation in causing both pelvic pain and dyspareunia. One study revealed that in women undergoing laparoscopy for pelvic pain, without gross anatomic disease, biopsies of normal-appearing peritoneum revealed minimal endometriosis in 7%, endosalpingiosis at 11%, and “inflammation” in 52%.

Thomson has speculated that inflammation in the pelvis and the vagina could underlie the clinical symptoms. This may be analogous to the current understanding of vulvar vestibular syndrome as a neuroinflammatory disorder in which abnormal sensations become involved with chronic inflammation in a pathophysiologic vicious circle.

**Specific Gynecologic Pain Syndromes**

**External Pain: Vulvar Disease**

**Lichen Planus**

This is a relatively uncommon disorder of the mucous membranes that may affect the gingiva and the vagina. Its three varieties are erosive, papulosquamous, and hypertrophic. The erosive variety is certainly the most disabling and the type most often affecting the vagina with diffuse inflammation, often with apparent shedding of the vaginal epithelium. Histologic confirmation may be obtained by a punch biopsy from the edge of the lesion. Treating this disorder is quite difficult and most often starts with delivery of vaginal steroids. Typically, this is 500 mg of hydrocortisone every day for 3 days, then 200 mg every day for 2–3 weeks, following by response-driven titration down to a maintenance dose of once to twice weekly. If these are not effective, then immune modulators such as tacrolimus (Protopic, Astellas Pharma US, Inc., Deerfield, IL) or pimecrolimus (Elidel, Novartis, East Hanover, NJ) may be employed.

**Lichen Sclerosis**

This disorder is better known to the practicing gynecologist as a whitening of the vulvar epithelium often with loss of vulvar architecture, especially atrophy of the labia minora, and loss of elasticity. Itching is the predominant symptom. Dyspareunia is certainly very common in this disorder, especially in more advanced cases. There is a very low but real risk of vulvar malignancy, and clinical vigilance is appropriate. The most effective treatment is a high-potency steroid, typically clobetasol propionate, 0.05% ointment applied twice per day until the lesions resolve (usually 2–3 months), then tapered to once or twice weekly for maintenance. When the disease produces phimosis around the clitoris, in rare cases surgery may be somewhat helpful in restoring clitoral sensitivity.

**Lichen Simplex Chronicus**

This disorder presents again with the dominant symptom of itching, but instead of the atrophy seen in lichen sclerosus, there is instead hyperkeratosis, and often obvious thickening of the vulvar epithelium. Excoriations from scratching are common, and again, a diagnosis is made by biopsying the edge of a lesion to allow comparison with normal skin. The main therapy is antihistamines, such as hydroxyzine 25 mg, taken 2 hours before sleep, vulvar hygiene, topical doxepin (a powerful antihistamine), and selective serotonin reuptake inhibitors such as citalopram, fluoxetine, or paroxetine or sertraline.

**Vulvar Vestibular Syndrome**

This is by far the most common cause of longstanding introital dyspareunia. It is predominantly found in white reproductive-age women. Prevailing thinking characterizes this as a neurosensory disorder, and attempts have been made to relabel this as “localized provoked vulvodynia.” Its designation as a neurosensory disorder arises in part from the success of treatments aimed at neuropathic pain, together with the recognition that other pain disorders such as temporomandibular disorder are more prevalent in women with vulvar vestibular syndrome.

The clinical criteria for diagnosis are best described as Friedreich’s criteria: 1) severe pain upon touch of the vestibule or attempt at vaginal entry, 2) tenderness or pressure localized within the vulvar vestibule, and 3) vestibular erythema (Fig. 2). Although women affected by vulvar vestibular syn...
drome often develop significant emotional reactions to having the disease, extensive research has failed to demonstrate any preexisting psychiatric or emotional condition as a precursor to developing the disorder. Rather, it seems more likely that there may be a genetically determined intrinsic vulnerability to inflammatory processes. Given this vulnerable substrate, superimposed provocative stimuli such as vaginal infection, long-term use of low-dose oral contraceptives, or intercourse beginning before the age of 16 may then be causative.

A host of treatments have been used in this disorder, most of which are aimed at combating infection, inflammation, and the neurologic component of the pain. Antibacterial and antimonilial agents have been largely unsuccessful except for treating superimposed infections. Topical lidocaine has been documented to be helpful as a 5% lidocaine ointment applied twice daily by fingertip or overnight by a cotton ball. Multiple topical preparations have been used, all without published documentation of efficacy. These include combinations of lidocaine with high-dose estrogen, cromolyn, amitriptyline, antiepileptic medications, and others. Systemic medications with evidence for efficacy include tricyclic antidepressants and gabapentin.

Many women with vestibular pain will also have levator spasm. Although most clinicians would agree that there may be a reciprocal relationship between pain in the levator area and pain in the vestibule, it is unclear whether one or the other is the original offender or whether they both develop simultaneously, with the muscular contraction being a most understandable reaction to intense pain in the vestibule. Nevertheless, self-directed contraction–relaxation exercises and physical therapy approaches to the pelvic floor, including biofeedback, may be additive in treatment.

Complete excision of the inflamed vestibular epithelium (vestibuloplasty) has been performed for approximately two decades. Reported success rates are quite high, some more than 95%, especially when surgery is used as the first approach rather than being reserved for those who do not improve in response to topical or medical therapies. Recently, Bergeron has reported persistence of pain relief at two-and-a-half years after vulvar vestibuloplasty. In view of recent advances in topical therapies, it would seem that surgery should be reserved for those with persistent symptoms after thorough trials of medical treatment.

The surgical technique of vestibuloplasty is relatively straightforward. The full-thickness epithelium of the posterior vestibule is excised, the borders of excision being the junction with external skin, the 3-o’clock and 9-o’clock positions, and the cephalad margin of the posterior hymen. Some authors advocate excising all the way up to the 1-o’clock and 11-o’clock positions, whereas others suggest this is not necessary. The lower vaginal epithelium is then elevated from its attachments, sufficient to close the defect without tension when pulled caudad. Closure is done with vertical mattress sutures, using suture material that will dissolve within a 2-week span. Postoperative care often involves continued topical estrogen and lidocaine, followed by, after 4–6 weeks of healing, local massage and pelvic floor relaxation techniques. Further physical therapy and supportive counseling are sometimes additive at this point.

Vaginal Pain
**Chronic Vaginitis**
This is certainly a common disorder in every gynecologist’s practice. A recently published algorithm provides excellent guidance for evaluation and treat-
ment. For some versions of this disorder, such as chronic moniliasis and chronic recurrent bacterial vaginosis, cure is at least an elusive if not impossible goal. Preventive therapeutic regimens may include premenstrual and or postmenstrual single doses of therapeutic agents such as a vaginal antimonilial or vaginal antibacterial agent, and vaginal acidification with acidic gels or boric acid capsules.

**Oral Contraceptive Adverse Effects**

Perhaps in keeping with the evidence for higher prevalence of vulvar vestibular syndrome in women using very-low-dose estrogen pills for long periods of time, some clinicians have observed that very light menses can be associated with diminished vaginal lubrication, leading to dyspareunia. Simply switching pills to a higher estrogen preparation can resolve this difficulty.

**levator Spasm**

As alluded to above, uncontrolled tightness of the levators can produce pain in some women. However, there is a puzzling lack of clear correlation between the degree of tightness and the degree of pain experienced, suggesting that tone of the muscles is a different parameter from their sensitivity. A woman complaining of this problem, when questioned carefully, often describes the pain as occurring in mid vagina rather than either introital or deep. Careful single-digit pelvic examination will easily localize the pain to the levators.

levator spasm, or “pelvic floor tension myalgia,” may arise spontaneously for uncertain reasons, but more often is a secondary development after discomforts due to pelvic pathology, such as endometriosis or other adnexal disease, uterine retroversion, and virtually any pelvic surgery.

**Obstetric Lacerations**

Multiple studies have documented dyspareunia after perineal/vulvar obstetric injury. Signorello noted that at 3 months after delivery, women with second-degree tears were 80% more likely to have dyspareunia than those without tears, whereas women with third- and fourth-degree tears were more than 270% more likely to have continued pain with intercourse. Approximately 24% of women have de novo dyspareunia at 6 months after delivery, which decreases without focused treatment to about 8% at 1 year after delivery. No relationship to lactation has been demonstrated. Suture selection may play a role in the amount of pain seen at 3 days after delivery but does not seem to affect long-term dyspareunia. Absorbable sutures that include the skin (as opposed to subcuticular) are associated with slightly greater dyspareunia at 3 months postpartum.

When the laceration or episiotomy involves the rectal sphincter, there does seem to be good evidence that an overlapping (as opposed to end-to-end) closure of the sphincter results in less dyspareunia at 6 and 12 months after repair. Between 7% and 10% of women may still be having dyspareunia a year after delivery.

Therapeutic regimens for this have not received much attention. There is some evidence that therapeutic ultrasonography may play a role, but a recent Cochrane review suggests that the evidence is weak. There may be room for the development of physical therapy treatments for this problem.

**Deep Dyspareunia**

The above discussion of neurophysiology suggests that normally silent C fibers may become activated in the development of chronic pelvic pain conditions and certainly in those that have dyspareunia as part of their symptom constellation. Thus, the cervix may not necessarily be abnormally sensitive when a Pap test is performed or even if grasped with a tenaculum. However, as a sequel to repeated traumas or repeated mildly uncomfortable stimulation, greater pain can develop. Examples include obstetric lacerations, chronic cervicitis, and cervical treatments such as loop electrosurgical excision procedure or conization. In such cases, pain is often quite focal, being elicited only by palpation of one quadrant of the cervix with a cotton-tipped applicator.

Appropriate treatments include treating underlying disorders such as cervicitis if it is present and/or treating the neuropathic component. Nightly delivery of 5% lidocaine for weeks to months has been effective in some cases. Serial anesthetic injections, or even neurolytic blocks with 50% alcohol have on occasion been successful. Medications for neuropathic pain may help (Table 1).

**Uterine Pain**

Adenomyosis, or the ingrowth of endometrial glands more than two high-power fields below the basal layer of the endometrium, is common in the multiparous woman in her 30s and 40s. Its role in pain is controversial, but if serial examinations document increased size and tenderness of the uterus in the luteal phase, the process may be legitimately held responsible for dyspareunia.

The retroverted uterus has long been the subject of debate. Uterine retroversion is present in approximately 20% of women as a normal anatomic variant. Most women with uterine retroversion do not have
deep dyspareunia, but it may occur with intrinsic enlargement of the uterus, adenomyosis, small pelvic capacity, and above-average penile dimensions in the partner.

Uterine retroversion has also been associated with pelvic congestion, although this is by no means uniform. Pelvic congestion typically presents with peaking in the luteal phase and pain present in the broad ligament as well as in the uterus itself, in contrast to the more focal pain in adenomyosis. Multiple surgical procedures have been devised to restore uterine anteversion, with variably durable results. Splinting of the entire length of the round ligaments with nonabsorbable suture such as Gore-Tex seem to be associated with more long lasting results, as reported in prospective clinical trials.28,29

Uterine leiomyomas rarely produce dyspareunia if they are the sole pathology, unless there is a low posterior cul-de-sac leiomyoma or cervical leiomyoma that alters vaginal dimensions. Even in that circumstance, the discomfort is more subtle and the complaint has more to do with altered vaginal caliber and depth.

**Adnexal Disease**

**Endometriosis.** Perhaps the most common disorder to present as deep dyspareunia is pelvic endometriosis. The posterior cul-de-sac, followed by the ovaries, is the most common location for this disease to appear. Many reviews on this complex topic have demonstrated that the relationship between the amount of disease and symptoms is obscure at best. Recent demonstration that de novo neural fiber growth occurs around endometrial implants in a rat model suggests they may play a role.30

Common treatment modalities have traditionally focused on surgical cauterity or excision of disease, followed by hormonal therapies. Oral contraceptives have been found to be equivalent to a gonadotropin-releasing hormone agonist in this setting, as has the progesterone intrauterine device. In the patient with uterine retroversion was well as endometriosis, uterine suspension may be a reasonable consideration.

**Pelvic Congestion Syndrome.** This disorder is described as the presence of overfilled pelvic veins that also demonstrate slowed circulation, demonstrated by delayed emptying of contrast dye introduced into the veins. It is classically present in the multiparous woman in her 30s or 40s and presents with greater symptoms in the luteal phase. The pathophysiology of this disorder is obscure, because changes in pelvic blood flow have been demonstrated to occur under stressful conditions, such as the stress interview, even in women without primary gynecologic complaints.31 It is therefore an open question as to whether the observed venous changes are the cause or the effect of pain.

Treatments of this disorder are limited. Continuous oral contraceptives and continuous progestins would seem to be a logical approach, although evidence for the effectiveness is lacking. Continuous progestins have some research data supporting their use, although the best study on this topic demonstrated that supportive counseling was equally important. With the development of venous embolization

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<th>Dosage Range</th>
<th>Taper up Schedule</th>
<th>Adverse Effects</th>
<th>Comments</th>
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<tr>
<td>Antidepressants</td>
<td></td>
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<tr>
<td>Amitriptyline (Elavil)</td>
<td>10–75 mg</td>
<td>Increase by 10 mg q 7 d</td>
<td>Sedation, constipation, weight gain, orthostatic hypotension</td>
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<td>Nortriptyline (Pamelor)</td>
<td>10–75 mg</td>
<td>Increase by 10 mg q 7 d</td>
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<td>Antiepileptics</td>
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<td>Gabapentin (Neurontin)</td>
<td>300–3, 200 mg</td>
<td>Start at qhs. Increase by 300 mg q 3 d. Usually 300 mg bid and 600 mg qhs is enough</td>
<td>Sedation, mental confusion, weight gain</td>
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<td>Lamotrigine (Lamictal)</td>
<td>100–200 mg bid for therapeutic effect</td>
<td>25 mg qhs for 2 wk, 25 mg bid for 2 wk, 50 mg bid for 1 wk, 100 mg bid for 1 wk, 100 mg qam, 200 mg qhs for 1 wk, then 200 mg bid</td>
<td>Mental confusion, sedation (rare), weight gain (rare). Rash—Stevens–Johnson syndrome has been reported</td>
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techniques, some enthusiasm has developed for this approach. However, reports published to date lack adequate standardization of diagnostic and treatment methods.

**Pelvic Support.** Pelvic organ prolapse has been held responsible for some degree of dyspareunia as well. However, the dominant clinical symptom would seem to be loss of urinary control rather than discomfort during intercourse. Given the population in which pelvic support difficulties occur, sexual changes due to support loss are often confounded by aging changes and erectile difficulties in the partners. Dyspareunia present before treatment of pelvic organ prolapse has been dwarfed by concerns regarding new or increased dyspareunia after pelvic support is surgically corrected (see discussion below).

**Surrounding Organ Systems**

**Gastrointestinal Illnesses**

**Crohn’s Disease**

Crohn’s disease is one of the more difficult gastrointestinal diseases to affect reproductive age women. In young women with anal Crohn’s disease, 9% can develop rectovaginal fistulae, which understandably are accompanied by many symptoms, including dyspareunia. Proctocolectomy with ileal pouch anastomosis, done for Crohn’s or ulcerative colitis, is followed by new dyspareunia in about 10% of patients, whereas 3–7% of women note fecal soiling during intercourse. These complaints improve gradually over time in more than one half the patients but remain a significant difficulty in the remainder.

**Irritable Bowel Syndrome**

Women with this disorder may have visceral hyperalgesia in general and may manifest their symptoms as vague discomfort throughout the lower pelvis, including dyspareunia. At the same time, there is an observed association between endometriosis and irritable bowel syndrome. In some settings, successful treatment of the endometriosis may improve bowel function, without other specific intervention.

**Bladder Disease**

**Painful Bladder Syndrome**

This disorder is characterized by intermittent or continuous urinary frequency, urgency, dysuria, nocturia, and discomfort along the urethra during intercourse. Urine cultures are often sterile, because bacterial shedding from the periurethral glands may be intermittent and in low numbers. Even catheterized urine samples are often sterile, because the catheter bypasses the urethral glands entirely. A clean-catch urine culture demonstrating a low titer of a single species is consistent with this diagnosis.

Tenderness of the urethra upon palpation that replicates the dyspareunia confirms the diagnosis. Not uncommonly, some reactive vaginismus and/or levator spasm may be present with this syndrome as well, often as part of a vicious circle of urethral pain and muscle spasm.

Patients with this disorder will often report that a short course of antibiotics improves their symptoms, but only transiently. A more effective treatment is a daily dose of antibiotics such as nitrofurantoin macrocrystals or a sulfa drug for three months, and a single dose of such an antibiotic either right before or after intercourse, for an undetermined length of time thereafter. If vaginismus or levator spasm is detected along with the urethritis, relaxation exercises or physical therapy may help.

**Interstitial Cystitis**

This disorder is commonly blamed for chronic pelvic pain, and is certainly also associated with dyspareunia. Clinical diagnosis is sometimes made using the Pelvic Pain and Urgency/Frequency Patient Symptom Scale, although in women with dyspareunia and daily pelvic pain, this questionnaire lacks sufficient specificity to clearly implicate the bladder as a source of the pain. Cystoscopy with hydrodistention under general anesthesia remains the criterion standard for diagnosis, and may have therapeutic benefit as well. More recently, pelvic examination with and without an anesthetic solution in the bladder has been used to try to separate out the bladder as an element in both dyspareunia and pelvic pain. This method awaits systematic report.

**Urethral Diverticulum**

It is hypothesized that a urethral diverticulum may arise when a periurethral gland becomes infected, forming a chronic abscess. If the abscess cavity then drains, it may seal off, forming a self-contained structure. Dyspareunia has been noted as presenting system in more than one half of women with urethral diverticuli.\(^\text{32}\)

**Postoperative Changes**

Surgery is a trauma to the body, and healing from surgery inevitably involves scar formation. Pain in surgical incision scars is ubiquitous, occurring after a certain percentage of any type of surgery. For example, cosmetic breast surgery is noted to produce long-term incision pain in approximately 10% of women. More familiar to the gynecologist is long-term discomfort from both vertical and horizontal

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abdominal incisions. Less well known are the discomforts associated with vulvar and vaginal surgery and total hysterectomy done by any means.

The common denominator in all these problems is scarring that may cause pain through neuropathic pain mechanisms. Although treatment of scar pain has received relatively little notice in the gynecologic literature, the literature in other specialties points the way to potential new treatment paradigms for the gynecologist.

Total Hysterectomy
An unknown number of women after total hysterectomy will develop focal pain in the vaginal apex. The normal-appearing vaginal apex suture line may be exquisitely sensitive, usually in one fornix, when gently touched with a cotton-tipped applicator, reproducing the pain of the patient’s dyspareunia. If the cotton-tipped applicator examination is omitted, subsequent bimanual examination may elicit pain that may then be erroneously attributed to sources cephalad to the vaginal apex, such as intraabdominal adhesions or intrinsic disease in a residual ovary.

Treatment for this disorder may include serial injections of local anesthetics, nightly applications of 5% lidocaine ointment using a vaginal applicator, and the tricyclic antidepressant and antiepileptic medications (gabapentin, Lamictal) used for neuropathic pain. Repeat laparoscopy to look for recurrent disease such as endometriosis or other intrinsic ovarian disease may be indicated. Of course, levator spasm and pain in the obturator and piriformis muscles that operate the hip may become part of the problem. Although general principles would suggest that physical therapy for these elements should precede surgery, this is not always the best course of action, because muscle difficulties may not resolve until organic visceral components that stimulated their development are successfully treated. In extreme circumstances, surgical revision may be indicated. This may be accomplished either vaginally, or laparoscopically if there is concern about potential visceral adhesions to the vaginal cuff. Unfortunately, in a follow-up study, approximately 70% of patients were noted to have recurrent pain within 2–3 years of surgery, albeit at a somewhat lower intensity.

In cases in which all of these methods fail, there is a theoretical possibility that neuromodulatory techniques such sacral nerve stimulators, spinal cord stimulators, etc may be efficacious, although published trials of these methods in this disorder have not yet appeared.

Pelvic Support Surgery

Cervicouterine Prolapse
A recent Cochrane review summarized three randomized clinical trials involving 287 women which demonstrated that dyspareunia is more common after vaginal sacrospinous ligament suspension than abdominal sacral colpopexy (36 compared with 15.6%). A series of 101 patients operated on by Sarlos et al with laparoscopic sacral colpopexy included one case of de novo dyspareunia.

Anterior Compartment
In pursuit of better long-term outcomes in the surgical repair of anterior compartment prolapse with urinary incontinence, multiple forms of the suburethral sling have been developed, involving a variety of prosthetic materials. Of the many studies reporting on these treatments, most do not adequately evaluate dyspareunia. Those that do inquire report a wide range of results, with de novo dyspareunia occurring in 8–69% and most studies reporting percentages of 15–30%.

Posterior Compartment
Traditional posterior repair with approximation of the levator muscles is well known to produce de novo dyspareunia in 15–30% percent of patients. The more contemporary site-specific repair, when done by experienced surgeons, has a much lower dyspareunia complication rate, less than 5%. Studies with longer-term follow-up of posterior repair report higher percentages of dyspareunia, suggesting that progressive tissue changes over time may be playing a role in this complication.

Multiple studies of mesh placement in pelvic support surgeries demonstrate that new dyspareunia after surgery is substantially more common with these techniques compared with those that do not use mesh material. It would seem, therefore, that complications after mesh placement deserve much more detailed study and that the use of mesh should be a last resort, rather than the first procedure done.

Despite multiple reports of the complication of de novo dyspareunia after pelvic support surgery, few studies suggest treatments for this problem. Preoperative and postoperative estrogen may be helpful. Treatments applied to other areas of scarring may be appropriate to consider for scarring in the pelvis. These might include carefully directed local massage and other techniques by qualified pelvic floor physical therapists, the medications listed above that target the neuropathic component of pain, and neuromodulation techniques. Although it would be appealing to the surgical mindset to consider removing the mesh,
this repeated surgery may entail the risk of further tissue damage and scarring, with possible aggravation of the neuropathic component of pain, rather than its relief.

**Aging Changes**

Although general surveys of the prevalence of dyspareunia suggest that it is indeed more common in the reproductive years, substantial numbers of women who are perimenopausal and postmenopausal and aging women are troubled by this complaint. The fibromuscular tube of the vagina loses elasticity with age and loses lubrication capacity as a product of both hormonal and vascular changes. This is happening at a time when women’s partners are commonly experiencing their own medical illnesses and erectile dysfunction, adding to the list of challenges faced by the couple. Remembering the above discussion that desire may not always precede arousal in the normal woman, it is understandable that a certain number of couples simply agree, either overtly or tacitly, to stop sexual activity at some point.

This constellation of difficulties is an obvious target for well-considered preventive medicine approaches. Although estrogen supplementation is the traditional approach, concerns about potential aggravation of cardiovascular and breast cancer risk have resulted in substantial decline in this practice. Nevertheless, small doses of vaginal estrogen in the form of estradiol tablets or cream maybe clinically very effective, while resulting in only very modest systemic absorption. Once adequate vaginal lubrication is well established, estrogen may indeed no longer be necessary if sufficiently frequent sexual relations, or other forms of vaginal stimulation, are continued.

This effort often requires supplemental lubrication of the vagina to allow comfortable intercourse. The commonly used water-based lubricants have the disadvantage of quick desiccation, meaning that they provide a few minutes of improved lubrication at best. Multiple commercial preparations last longer. However, ordinary vegetable oil has likely been used for thousands of years and is entirely noninjurious to the vaginal mucosa. Its low cost and discrete availability are also pluses.

Other couples find that employing different sexual positions may help. We have found the cross-wise position to be perhaps the best of these options. (Fig. 3) When employed properly, neither partner is supporting their weight, the external genitalia are available for additional stimulation, and both direction and depth of vaginal penetration can be easily adjusted. When offering this advice to patients, it is most helpful to encourage them to discuss this with each other outside of a sexual situation, when the conversation can take place in a calm and mutually supportive manner. The couples who can manage this discussion reasonably comfortably are obviously those who can best realize the benefits of this position change.

**THE ROLE OF PHYSICAL THERAPY**

As in the case of working with pelvic floor pain disorders, many physical therapists have also become proficient at helping individuals and couples work with introital dyspareunia involving substantial muscular factors. Pelvic floor physiotherapy is now widely used and is successful both in primary muscular disorders and in those situations in which the muscle problems are secondary to other gynecologic disease. Pelvic floor electrical stimulation has also been employed with some success, although it may aggravate pain in some cases. Some preliminary studies of botulinum toxin are now appearing, demonstrating that injection into the levator muscles may also be additive. However, most investigators using this approach recommend simultaneous physical therapy as well, so as to make best use of the benefits of both treatments.
COUNSELING

Initial reports on the treatment of vaginismus with intercourse and sex therapy were extremely optimistic. Over the passage of time, it has been recognized that couples presenting with vaginismus may have a collection of difficulties rather than just one. Success rates have been in the 60–100% range and relapse rates have been recognized to be potentially high. Hypnotherapy, biofeedback, abreaction interviews, and other methods have also been employed, with mixed success as well.

The take-home message for the practicing gynecologist is to recognize that asking about such difficulties should be a routine part of clinical practice. The tactful and comfortable discussion of such issues is an important part of helping the patient accomplish the transition to counseling by a mental health professional. Early attention to sexual difficulties may help prevent the development of more severe problems.

THE FUTURE

As research progresses, the future may find us focusing on additional aspects of pain that are presently little understood. In the case of dyspareunia, these may include the role of inflammation in gynecologic disease, neurophysiologic differences in women experiencing pelvic pain and dyspareunia, and an emphasis on treatments for neuropathic components of chronic pain.

Examples of inflammation in gynecologic disease include the inflammatory components of endometriosis; the role of masts cells in endometriosis, interstitial cystitis, and vulvar vulvar vestibular syndrome; and the emerging notion that subclinical inflammation may play a role in pelvic pain in general. Much work in defining appropriate therapies remains to be done.

In terms of host differences, it has now been demonstrated that women with vulvar vestibular syndrome have lower pressure pain thresholds in other areas of the body, such as the leg. Women with endometriosis have wider receptive fields and more intense pain in response to saline injection in the hands, a standard pain-laboratory technique. Thus, either genetically or environmentally introduced differences in neurophysiology may help explain why some women get pain with certain gynecologic diseases whereas others do not.

As discussed above, multiple primarily pain complaints and multiple examples of postoperative pain may be explained on the basis of neuropathic pain, opening up another whole arena of treatment methods for dyspareunia. These would include medications and physical therapy procedures appropriate for neuropathic pain and scar pain. We might do well to learn from the literature of other specialties that deal with pain related to surgical scars. The next decades may see us adding treatments that address more of these characteristics in addition to or instead of our current emphasis on sex steroids, surgery, and counseling.

REFERENCES