

# Dyspareunia: 5 overlooked causes

Disorders ranging from a simple anatomic problem to a complex psychosocial/biologic phenomenon can cause difficult or painful coitus. An expert outlines diagnosis and treatment strategies for 5 common causes and offers guidance on how to conduct the physical exam and elicit information from the patient.

Identifying the cause of a patient's dyspareunia can be just as challenging as getting her to admit to the problem.

Due in part to underreporting of the condition, the incidence and prevalence of dyspareunia—defined as genital pain experienced just before, during, or after sexual intercourse<sup>1</sup>—is uncertain.<sup>2</sup>

Because it is easy to miss subtle physical findings such as small fissures, periclitoral scarring, or a focus of tender vestibulitis under a hymenal remnant, getting to the root of dys-

pareunia can present a significant challenge to clinicians. Adding to the difficulty is the fact that intermittent conditions such as cyclical *Candida albicans* are hard to diagnose.

This review of 5 common but often overlooked causes describes what is known about dyspareunia and how to conduct a complete evaluation, including physical examination, diagnostic tests, and questions to ask the patient.

## ■ CAUSE 1

### Inadequate estrogenization

Vulvovaginal atrophy is the leading cause of sexual dysfunction, affecting up to 50% of women over age 50. It contributes to a lack of vaginal lubrication with sexual arousal and, consequently, dyspareunia and postcoital bleeding.<sup>3</sup> Even when a woman is taking oral hormone replacement therapy, the vagina can lack sufficient estrogen.

Younger women also may experience atrophy and lowered estrogen levels. For example, a 34-year-old woman with premature ovarian failure may experience slight burning, dryness, and pain on penetration.

Tamoxifen can be a source of dyspareu-

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## KEY POINTS

- The leading cause of dyspareunia for women under age 50 is vulvar vestibulitis; for women over age 50, it is vulvovaginal atrophy.
- The skin conditions dermatitis, lichen sclerosus, and lichen planus are a significant cause of dyspareunia complaints.
- *Candida* can be difficult to diagnose; the fissuring experienced by patients with this infection is often attributed to other causes.
- Desquamative inflammatory vaginitis leads to the loss of the lactobacillus, with bacterial overgrowth and clue cells similar to bacterial vaginosis.
- Generalized vulvar dysesthesia involves constant or episodic unprovoked stinging, burning, irritation, rawness, or pain anywhere on the vulva. In contrast, localized vulvar dysesthesia is provoked pain in the vestibule.

nia: It can cause vaginal atrophy in the premenopausal woman or estrogenization with Candidal invasion in postmenopausal patients.

Atrophy also can occur:

- with hypothalamic amenorrhea caused by excessive exercise or marked weight loss
- during the postpartum period and breastfeeding
- with the use of some low-estrogen (20 µg) contraceptives and medroxyprogesterone acetate
- after radiation or chemotherapy

**Resolve the problem with local estrogen.**

Fortunately, atrophy is easily reversed with local estrogen in the form of cream, tablets, or the vaginal ring. Because the latter does not elevate circulating estradiol levels after the first 24 hours of use, many oncologists are willing to allow this therapy for breast cancer patients.<sup>4</sup>

When dyspareunia persists despite local estrogen use, we must seek out other causes.

■ **CAUSE 2**

**A skin disease**

**Dermatitis.** There are 2 types of dermatitis: eczematous, in which the irritant is essentially unknown, and contactant, which arises from known irritants or allergens. In some cases, the exposure to an irritant may be fairly recent. In others, the continuing combination of irritants and tight clothing or abrasive activity eventually leads to symptoms.

Physical findings of dermatitis include erythema (with or without scaling) and fissuring—especially of the perineum. A biopsy is diagnostic.

Recommended treatment includes meticulous vulvar hygiene and the use of 2.5% hydrocortisone cream twice daily for 14 to 30 days, followed by twice-weekly “maintenance” applications. For moderate or severe cases, a medium-potency steroid (betamethasone valerate 0.1%) or an ultrapotent steroid (clobetasol 0.05%) may be used in the same manner. In addition, physicians should edu-

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## Physical causes of dyspareunia

LOCATION AFFECTED	SPECIFIC CONDITION	IMPORTANT HISTORY	REASON FOR PAIN	COMMENTS AND CAVEATS
Vulva and vestibule	Dermatitis (eczema)	Atopic history, other eczema	Erythema, scaling, fissuring	Look for <i>Candida</i>
	Dermatosis: lichen sclerosus	Itching (recent or lifelong), soreness, possibly no symptoms	Fissures, ulceration, scarring	Look for <i>Candida</i>
	Dermatosis: lichen planus	Itching, irritation, burning	Erosions, ulcers, scarring	Many drugs exacerbate
	Ulcerative disease: herpes simplex or zoster, chancroid, granuloma inguinale, aphthae, Behcet's syndrome	Episodic outbreaks	Ulceration varying in size, tenderness	Behcet's very rare, involves oral ulcers, uveitis, other systems
	Labial hypertrophy	Irritation with physical activity	Elongation of labia	Look for vestibulitis
	Female circumcision	Ethnicity and country of origin	Absence of clitoris and prepuce, labial fusion	
	Generalized dysesthesia (vulvodynia)	Episodic or virtually constant burning, stinging, soreness, irritation, rawness	Often no findings or findings of erythema and edema, areas of hyperesthesia or hypoesthesia	Long history of unsuccessful treatments for urinary tract infections, yeast infections, or bacterial vaginosis
	Radiation	Gyn or urinary tumor	Pallor, alopecia, loss of elasticity	
Urethra and bladder	Urinary tract infection	Dysuria, frequency, urgency	Tenderness over bladder	Symptoms with negative cultures suggest vulvodynia
	Urethral diverticulum	Dysuria, dribbling, pain with penetration	Urethral tenderness, mass	
	Interstitial cystitis	Pelvic pain, dysuria, urgency, frequency, nocturia	Tenderness over bladder, along anterior vaginal wall	Look for vestibulitis
Vestibule only	Vestibulitis	Pain mainly with penetration, tampon, or speculum	Tenderness on touch or pressure, erythema	Easily missed without Q-tip testing
Perineum and anus	Episiotomy	Vaginal delivery with episiotomy	Nonhealing, tenderness	Look for <i>Candida</i>
	Dermatitis (eczema)	Itching, irritation	Erythema, edema, fissuring	
	Inflammatory bowel, Crohn's	Diarrhea, bleeding, pain	Edema, tags, fissures, sinuses	May precede bowel symptoms
Rectum	Rectocele	Sensation of vaginal obstruction	Protrusion of stool-filled rectum	
Vagina	Pelvic floor hypertonus	Aching pain, vaginismus	Levator spasm on palpation	
	Congenital anomaly: vaginal agenesis, imperforate hymen	Inability to be penetrated	Absent vagina, abnormal hymen	

\* Lack of foreplay; inadequate stimulation and arousal.

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LOCATION AFFECTED	SPECIFIC CONDITION	IMPORTANT HISTORY	REASON FOR PAIN	COMMENTS AND CAVEATS
Vestibule and vagina	Atrophy: low or absent estrogen	Dryness, irritation, history of breastfeeding, oligomenorrhea or amenorrhea, low estrogen/high androgenic oral contraceptives, medroxyprogesterone acetate, anorexia, exercise, chemotherapy, radiation, bilateral salpingo-oophorectomy, premenopausal tamoxifen, aromatase inhibitors	Reduction in labial size, mucosal color, and textural change; fissures; elevated vaginal pH; atrophic wet prep	Can occur at any age
	Vulvovaginitis: <i>Candida albicans</i>	Antibiotics, steroids, estrogen, immunosuppression	Itching, erythema, edema, discharge, fissures, or few symptoms	Look for superimposed vestibulitis
	Desquamative inflammatory vaginitis	Irritative symptoms, profuse discharge	Erythema, sheets of white blood cells, parabasals, no lactobacilli	Atypical Pap; look for vestibulitis
	Vulvovaginitis: <i>Trichomonas</i>	Itching, discharge, or few symptoms	Mobile trichomonads; positive culture	
	Bartholin cyst or abscess	Swelling and pain	Cystic mass at base of vestibule	
	Seminal plasma allergy	Itching on entry or ejaculation	Edema and erythema postcoitus	Trial of condom helps
	Dermatosis: lichen sclerosus	Itching or no symptoms	Fissures, scarring around introitus	Look for <i>Candida</i> , vulvodynia
	Dermatosis: lichen planus	Itching, burning, discharge	Erosions, ulcers scarring	Look for <i>Candida</i> , vulvodynia; may also see atypical Pap
	Inadequate lubrication, dryness	Poor sexual technique,* sexual dysfunction, Sjogren's syndrome, oral contraceptives, medications, vestibulitis	Dryness, tenderness	No good test for lubrication; hard to judge on exam; history important
Radiation	Gyn or urinary tumor	Pallor, scarring, loss of elasticity		
Pelvis	Retroverted or prolapsed uterus	Pain with thrusting	Uterine retroversion or descent	
	Leiomyomata	Pain with thrusting	Tender uterine masses	Uncommon cause of pain
	Endometriosis, adenomyosis	Cramping, deep dyspareunia, menorrhagia, dysmenorrhea	Tender uterus, fixed uterus, nodules cul de sac, rectovaginal septum, adnexal tenderness or masses	
	Adnexal pathology	Deep dyspareunia, cyclical	Tenderness in adnexa	
	Pelvic inflammatory disease	Chronic pelvic pain, deep dyspareunia	Cervical motion tenderness, uterine, adnexal tenderness	
	Irritable bowel syndrome	Deep dyspareunia	None or pelvic tenderness	

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cate patients with dermatitis about the chronicity of the condition and the importance of eliminating the cause, if possible.

Poorly treated eczema leads to lichen simplex chronicus. One clue to this condition is a history of atopy or eczema elsewhere on the body.

**Lichen sclerosus and lichen planus.** These dermatoses cause changes in the color and texture of the epithelium.

Because lichen planus can produce erosion of the vestibule, it often is mistaken for vestibulitis. With this condition, erosions are intensely erythematous and vary from small areas to involvement of the entire vestibule. You will also note a serpiginous white border or subtle white reticules adjacent to erosions.

Lichen sclerosus, meanwhile, causes whitened epithelium with the thinned and

**Both lichen sclerosus and lichen planus can produce intense itching or progress without clinical symptoms.**

wrinkled appearance of cigarette paper; areas of hyperkeratosis also may be present. Changes may occur from the periclitoral area to the anus in a keyhole configuration.

Both lichen sclerosus and lichen planus:

- can produce intense itching or progress without clinical symptoms
- can scar extensively and cause bridging synechiae at the fourchette, elimination of the labia minora, and fusion of the prepuce over the glans clitoris
- can produce anal fissuring and painful defecation

While lichen sclerosus never involves the vagina, vaginal lichen planus produces inflammatory vaginitis that can scar and reduce the size of the vagina—even obliterate it entirely.

Treatment for both diseases consists of ultrapotent topical steroids to arrest the inflammatory process. Vaginal lichen planus is treated with hydrocortisone suppositories (25 mg at bedtime), with the length of treatment dependent on severity.<sup>5</sup> More potent steroids may be necessary.

■ CAUSE 3

**Candida**

This infection can be extremely difficult to diagnose for a variety of reasons. Patients come in partially treated with over-the-counter antifungals. Many have taken a fluconazole tablet with a long half-life of action. Others have a cyclical candidiasis that is seen only in the luteal phase of the cycle. In these cases, fissuring is often attributed to other causes.

Complicating matters further, a wet mount will be negative in the presence of *Candida* approximately 50% of the time.<sup>6</sup> For these reasons, a culture is essential when there is an index of clinical suspicion and white blood cells are present on the wet mount.

Uncomplicated *Candida* is treated by topical -azole creams for 3 or 7 days or a single fluconazole 150-mg tablet.

Complicated *Candida* (that is, more than 3 infections in a year or infection in a pregnant or immune-compromised host) will require longer courses of therapy.<sup>7</sup>

■ CAUSE 4

**Desquamative inflammatory vaginitis**

Because the intense inflammation produced by the 2 diseases are similar, some people believe desquamative inflammatory vaginitis is a form of lichen planus<sup>8</sup>—in fact, it is sometimes called lichenoid vaginitis. However, desquamative inflammatory vaginitis does not scar the vagina, suggesting a different cause. Its profusely irritative discharge—microscopically characterized by sheets of white blood cells—resembles *Trichomonas* and *Candida*.

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## Discussing dyspareunia: Questions crucial to a thorough exam<sup>1</sup>

Questions such as "Are you sexually active?" and "Do you have any concerns about your sex life?" can begin a discussion of dyspareunia. Other vital questions include the following:

**When did the pain begin?** Primary complete dyspareunia may result from a congenital anomaly or psychosocial issues, but the leading cause is vulvar vestibulitis.<sup>2</sup> Acquired dyspareunia has many causes.

**When and where does the pain or discomfort occur?** Ask the patient to describe its severity, character, duration, location, and time during the menstrual cycle. Superficial dyspareunia usually is due to vestibulitis, inadequate lubrication, or an anatomic abnormality of the introitus.<sup>3</sup> Other causes include vulvar atrophy, infection, urethral disorders, and vulvar dermatitis or dermatosis. Pain associated with deep penetration or thrusting may be related to a retroverted uterus or to impaired mobility of the pelvic organs due to scarring from endometriosis or pelvic inflammatory disease.<sup>4</sup> Cystitis and interstitial cystitis may cause deep mid-line dyspareunia, as well as dysuria and other urinary tract symptoms. Deep dyspareunia can also be due to vaginal dryness or atrophy. Consider adnexal or bowel pathology when the pain occurs laterally.

**Are there other sexual problems?** Pain during intercourse often causes sexual dysfunction, which needs to be addressed before the pain can resolve.

**What have you tried to treat or prevent the pain?** Successful aids can offer diagnostic clues.

**Is there any vaginal discharge, itching, burning, odor, or bleeding?** These may be present with vaginitis or a neoplasm. Increased discharge may be due to vestibulitis.

**Do you have any gynecologic problems, such as endometriosis, fibroids, or chronic pelvic pain?** These conditions have well-known associations with deep dyspareunia. Endometriosis and vulvar vestibulitis occur together.

**Have you had vulvovaginal or pelvic infections, such as candidiasis, herpes, gonorrhea, or chlamydia?** Recurrent herpes or Candidal infection can be painful and difficult to diagnose; pelvic inflammatory disease can cause scarring and decreased mobility of pelvic organs.

**What gynecologic surgery or other procedures have you undergone?** Childbirth, radiation

or chemotherapy, or incontinence procedures may lead to dyspareunia. Female circumcision is practiced in some cultures and should be considered when appropriate. Scarring and fibrosis can distort anatomy, narrow the vagina/introitus, and decrease tissue mobility, thereby causing pain during thrusting. Chemotherapy and radiation may result in premature ovarian failure (hypoestrogenism). Radiation vulvitis contributes to superficial pain.

**What is your natural lubrication like? If it is low, have you tried commercially available lubricants?** Natural lubrication may be reduced from hypoestrogenism, certain drugs, or difficulty with arousal.

**What do you use for contraception?** Latex allergy from condoms or a diaphragm, or an irritant reaction to spermicides may be at the root of the pain. Low-estrogen oral contraceptives or depot medroxyprogesterone acetate contribute to poor lubrication. The intrauterine device is a risk factor for recurrent *Candida*.

**What medical or psychiatric problems are you currently being treated for?** Skin disorders such as eczema and lichen planus may be associated with vulvar dermatitis. Inflammatory bowel disease may be related to pelvic adhesions. Interstitial cystitis can cause both dyspareunia and dysuria.

**What drugs are you taking?** Many medications are associated with dyspareunia due to side effects such as decreased sexual arousal, vaginal lubrication, or serum estrogen levels.

**Have you ever been sexually abused or had a traumatic injury involving your genitals? Did you receive counseling or help for this?** Many women have worked through their trauma, but unresolved issues can contribute to ongoing pain. Sexual abuse is a risk factor for chronic pelvic pain but is not associated with vestibulitis.<sup>5</sup>

**What do you think may be causing this problem?** Often, the patient will provide the answer.

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Sheets of white blood cells and parabasal cells also resemble *Trichomonas*, *Candida*, or severe atrophy.

The inflammation leads to the loss of the lactobacillus, with bacterial overgrowth and clue cells similar to bacterial vaginosis (though bacterial vaginosis never causes such inflammation).

**Vulvodynia consists of unprovoked stinging, burning, irritation, rawness, or pain anywhere on the vulva and can be constant or episodic.**

Though antibiotics may yield transient improvement,<sup>9</sup> management most often consists of 25-mg hydrocortisone suppositories (or compounded as 100 mg for severe cases) at bedtime for 14 days, then every other day for 14 days. After this course of therapy has been completed, clinicians must reevaluate the patient to determine whether she needs extended therapy (in severe cases) or can begin maintenance with a weekly suppository (for cases that are mild but chronic).

If dyspareunia does not resolve after the inflammation abates, superimposed neuroinflammatory pain (vestibulitis) will need treatment.

■ CAUSE 5

**Vulvodynia or vulvar vestibulitis**

**Vulvodynia (generalized vulvar dysesthesia).** This condition—which consists of unprovoked stinging, burning, irritation, rawness, or pain anywhere on the vulva—may be constant or episodic. Dyspareunia in these cases may involve postcoital exacerbation of symptoms.

The cause is unknown, but some suspect a lesion of the pudendal nerve in its long course from the spine to the vulva.

There may be virtually no physical find-

ings, or there may be areas of tenderness, hyperesthesia, or hypoesthesia. A biopsy will be nonspecific. Vulvodynia is therefore diagnosed by ruling out infectious, dermatologic, or other causes of genital pain.

The following treatments are usually successful:

- tricyclic antidepressants such as nortriptyline, starting with a bedtime dose of 10 mg and working up to 50 mg to 150 mg
- the antiepileptic agent gabapentin, starting with a bedtime dose of 100 mg and working up to as much as 1,000 mg (this is usually substituted for the tricyclic antidepressant if that therapy alone is ineffective)

**Vulvar vestibulitis (localized vulvar dysesthesia).** This condition, consisting of provoked pain in the vestibule on contact, is the leading cause of dyspareunia in women under 50.<sup>10</sup>

This condition may be either primary or secondary. With primary vestibulitis, a woman experiences pain with her first use of a tampon, her first exposure to a speculum, and the initiation of sexual relations. For those with secondary vestibulitis, pain on contact results after a period of comfortable sexual relations.

This pain is thought to stem from inflammation or trauma that initially sensitizes nociceptors in the vestibular mucosa, leading to prolonged neuronal firing. This in turn sensitizes the wide-dynamic-range neurons in the dorsal horn to respond abnormally, converting the sensation of touch into pain (allodynia).<sup>11</sup> There often is a history of an unresolved irritative event such as Candidal infection, repeated genital infection,<sup>12</sup> topical treatments,<sup>13</sup> and early and sustained use of oral contraceptives.<sup>14</sup> All are suspected causes.

Diagnosis is made once other pathology has been ruled out and a Q-tip test has demonstrated that contact elicits pain in the vestibule.

Numerous treatment protocols have been described, but current interest focuses on

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## Dyspareunia: Is it organic or psychological?

**R**ecent studies of the vestibule and vagina provide new insights into an organic explanation of dyspareunia in women who are otherwise healthy.

Clinicians have long known that the vulva and vestibule are innervated by the pudendal nerve, composed of both somatic motor efferents and sensory afferents. But autonomic nerve fibers from the inferior hypogastric plexus and caudal sympathetic chain ganglia also provide genital sensation and may contribute to the perpetuation of neuroinflammatory pain.<sup>1</sup> Although the vulvar vestibule is by definition visceral tissue, it is considered to have nonvisceral innervation.<sup>2</sup> Thus, sensations to touch, temperature, and pain are similar to sensations evoked in the skin and can be exquisitely painful.

The traditional view that the vagina has a paucity of nerve endings was contradicted with demonstration of profound innervation, with greater numbers of nerve fibers in the distal areas than in more proximal parts.<sup>3</sup> The vagina itself can hurt.

In addition, the presence of luteinizing hormone and human chorionic gonadotropin receptors on the bladder trigone supports the complaint of cyclic worsening of pelvic dyspareunia.<sup>4</sup> Circumvaginal motor spasm from hypertonicity of the levator plate<sup>5</sup> is an evolving area of study.

**Shifting views.** Nineteenth-century gynecolo-

gists approached dyspareunia primarily from a surgical perspective, using a wide variety of operative interventions. In time, the surgical approach was replaced by an emphasis on psychosocial issues: Women who complained of dyspareunia were frequently classified as "frigid," while physiological correlates were largely ignored.<sup>6</sup> Still later, "deep-thrust" dyspareunia was considered suggestive of an organic source, whereas superficial or entrance dyspareunia was thought to derive from emotional or psychological issues.<sup>7</sup>

Gradually, an integrated and pain-model approach has evolved. It is now theorized that an instigating pain event is perpetuated by other factors.<sup>8</sup>

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addressing neuroinflammatory pain. This can be done at the peripheral afferents by reducing inflammation and hyperexcitability with topical xylocaine 5% in a compounded sterol-lanolin base 5 times daily; centrally, this is accomplished using tricyclics in doses of 50 mg to 150 mg.

Note that primary vestibulitis is extremely difficult to treat; vestibulectomy and perineoplasty improve the condition by 60% to 90% when medical management fails.<sup>15</sup>

■ **Vaginismus**, an involuntary spasm of the perineal and levator muscles, may occur in patients with vestibulitis. While primary vaginismus is psychologic in origin, secondary vaginismus represents a conditioned response to pain,<sup>16</sup> usually vestibulitis. In patients with secondary disease, pelvic-floor motor instability has been well-demonstrated; these women have a reduced ability to contract or relax the pelvic floor and increased muscular instability at rest.<sup>17</sup>

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▪ **Dyspareunia: 5 overlooked causes**

Vaginismus is diagnosed by eliciting muscle spasms in the pelvic floor by depressing the levators. If a woman has primary vaginismus of psychogenic origin, there is no tenderness in the vestibule; note, however, that the exam may be so difficult for her to endure that evaluating the vestibule is not possible.

Pelvic-floor motor instability is treated with physical therapy and biofeedback. In secondary disease, vestibulitis must also be concomitantly addressed. Primary disease, meanwhile, is treated by desensitization techniques that help the patient control the relaxation of her musculature.

**The fine points of examination**

**Asking the right questions.** Before you begin the physical exam, it is crucial to get as much information as possible about the patient's condition. The dyspareunia history includes the following:

- a complete description of the pain problem and any concomitant sexual dysfunction
- exploration of potential gynecologic causes
- exploration of potential medical causes
- psychosocial information<sup>18</sup>

In addition, a series of open-ended questions asked in a nonjudgmental manner will help identify potential anatomic and medical sources of pain (TABLE), as well as psychosocial issues that may be a cause or result of dyspareunia (see "Discussing dyspareunia: Questions crucial to a thorough exam" on page 59).

**The physical exam.** Although guided by the history, the physical examination needs to be as comprehensive as possible. It should include:

- systematic, meticulous inspection of every structure to confirm normal color, texture and architecture, and the presence or absence of lesions.
- gentle palpation of all tissues for the source of the discomfort. This should include a Q-tip test of the vestibule for tender foci.
- a speculum exam with inspection for mucosal integrity without fissure, erosion, or ulceration, as well as a check for the

presence or absence of rugae and discharge. (Testing for pH is done with a reactive cardboard strip while the speculum is in place; then samples for wet mount and cultures are collected.)

- gentle single-digit exam of the vestibule to confirm the Q-tip test, as well as single-digit palpation of the pelvic-floor musculature, anterior vaginal wall, urethra, and bladder to confirm superficial pain and avoid confusion with pelvic sources.
- bimanual examination to evaluate for any nodularity or masses in the vagina, rectovaginal septum, or pelvis, as well as for mobility and tenderness of the pelvic organs.

Before attributing the dyspareunia to a lesion you encounter, it is important to reproduce pain at that lesion site. Whitened skin and some synechiae, for example, are painless; the source of the dyspareunia may be tenderness in the vestibule.

Note that with generalized dysesthesia (vulvodynia), there may be no physical findings.

■ **Take steps to navigate the pain.** Some women cannot tolerate a vulvar or vaginal examination; asking about previous experience will make it easier to tailor the exam appropriately. The following techniques also may be appropriate:

- use of premedication
- presence of a support person
- an agreement to stop the exam if the patient so requests
- use of a pediatric speculum

In rare cases, examination may need to be deferred until desensitization with a sexual therapist is achieved. Fortunately, most women tolerate the examination well and can identify the troubling areas.

It may not be possible to complete all components of an examination at a single visit. For example, a patient may have to return for a vaginal examination and wet mount if menses are present at the initial appointment.

### Essential laboratory studies.

■ **Vaginal pH.** A normal level (3.5 to 4.5) rules out bacterial infection and atrophy. *Candida* grows at any pH. Elevated pH is nonspecific

### Cultures for *Candida* and *Trichomonas* are important when microscopy is negative.

and can represent recent intercourse or a small amount of blood. However, it also can suggest such causes of dyspareunia as atrophy, vaginal lichen planus, desquamative vaginitis, and *Trichomonas*.

■ **Wet mount** reveals 4 important features:

1. **Epithelial cells.** These should be superficial or intermediate. The presence of parabasal cells suggests atrophy regardless of the age group. It also may indicate inflammation from *Candida*, lichen planus, or desquamative inflammatory vaginitis.
2. **Pathogens.** *Candida* or *Trichomonas* may be identified. Microscopy for *Candida* lacks sensitivity; a negative examination in a symptomatic woman mandates a culture for *Candida*.<sup>19</sup>
3. **Background flora.** As mentioned earlier, lactobacillus dominates the normal vagina; when this predominance is seen on microscopy, there is no bacterial infection. A vaginal culture may grow *Escherichia coli*, group B streptococcus, *Gardnerella*, and a variety of normal commensals, but these are not the cause of dyspareunia when pH is normal and lactobacilli dominate the slide.
4. **White blood cells.** Large numbers suggest *Candida*, lichen planus, *Trichomonas*, gonorrhea, chlamydia, or desquamative inflammatory vaginitis.

■ **Cultures** for *Candida* and *Trichomonas* are important when microscopy is negative. Routine vaginal culture is not recommended. Cultures for herpes, gonorrhea, or chlamydia may be necessary.

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■ **Biopsy and blood tests** offer data on hormonal levels or type-specific antibodies for herpes. Biopsy of the vulva or vagina is indicated whenever there is a visible lesion that needs identification.

■ **Urine culture, colposcopy, or imaging** such as ultrasonography and spine films may be indicated.

■ **Specialty referrals** may be helpful for evaluation of the gastrointestinal or genitourinary tract, or for diagnostic laparoscopy for endometriosis. ■

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