Patients presenting with chronic vulvovaginal pain are often perplexing to diagnose and treat, particularly when examination appears to be normal. This first part of a two-part article discusses the aetiology, pathophysiology and clinical presentation of vulvovaginal pain.

Patients presenting with chronic vulvovaginal pain are often perplexing to diagnose and treat, particularly when examination appears to be normal. Although many patients have an observable skin disease of the vulva and/or vagina that explains their symptoms, some do not. These latter patients, we believe, almost always have a physical cause for their pain, but lateral and multidisciplinary thinking is necessary to arrive at the correct diagnosis.

In this first part of a two-part article, the principles of the aetiology, pathophysiology and clinical presentation of vulvovaginal pain are described. The second part will cover the management of vulval pain.

THE DIFFERENCE BETWEEN ‘VULVODYNIA’ AND VULVOVAGINAL PAIN
‘Vulvodynia’ is a term familiar to every doctor with an interest in vulvovaginal disease. The term was developed initially by the International Society for the Study of Vulvar Disease (ISSVD) in 1983. Their current definition is ‘vulvar discomfort, most often described as burning pain, occurring in the absence of relevant visible findings or a specific, clinically identifiable neurologic disorder’.

The ISSVD has also produced a system of classifying vulvovaginal pain, where patients are categorised into those with an identifiable cause and those that fit their case definition for vulvodynia (Box 1). The term ‘vulvodynia’ in...
1. ISSVD TERMINOLOGY AND CLASSIFICATION OF VULVAR PAIN (2003)*

A. Vulvar pain related to a specific disorder
1. Infectious (e.g. candidiasis, herpes, etc.)
2. Inflammatory (e.g. lichen planus, immunobullous disorders, etc.)
3. Neoplastic (e.g. Paget’s disease, squamous cell carcinoma, etc.)
4. Neurological (e.g. herpes neuralgia, spinal nerve compression, etc.)

B. Vulvodynia
1. Generalised
   a. Provoked (sexual, nonsexual or both)*
   b. Unprovoked†
   c. Mixed (provoked and unprovoked)
2. Localised (vestibulodynia, clitorodynia, hemivulvodynia, etc.)
   a. Provoked (sexual, nonsexual or both)
   b. Unprovoked
   c. Mixed (provoked and unprovoked)

ABBREVIATION: ISSVD = International Society for the Study of Vulvar Disease.

*‘Provoked’ means that pain is in response to friction, pressure, intercourse, insertion etc.
†‘Unprovoked’ means that the pain occurs spontaneously.

The mechanisms involved in vulvovaginal pain are still very poorly understood. There are many reasons why the vulva and vagina are a pain-prone part of the body, including the following:

- the complex anatomical structure of the bony pelvis and lower spine, and its vulnerability to damage
- the central position of the vulva and vagina in the pelvic myofascial complex, which facilitates pain referral from other pelvic viscera
- the fact that this area is subject to a great deal of physical stress: urination, menstruation, defaecation, childbirth and friction from clothes and pads
- personal hygiene habits that may inadvertently exacerbate the original problem
- the high levels of anxiety and fear that are often attached to problems involving the genital area
- the crucial importance of the genital area in a woman's sexual wellbeing and self-esteem.

Innervation
The innervation to the posterior vulva and perianal skin is provided by the pudendal nerve, which originates from the S2–S4 nerve roots. The anterior vulval skin is supplied by the ilioinguinal and genitofemoral nerves, arising from the L1 and L1–L2 nerve roots, respectively. The latter nerves are predominantly sensory, but the pudendal nerve contains motor, sensory and sympathetic fibres that supply the complex autonomic reflexes of the pelvic organs. The epithelium of the vagina proper (that is, deep to the hymenal ring) is not normally sensitive to pain.

The pudendal nerve supplies both the anal and urinary sphincters whereas the muscles of the pelvic floor are mostly innervated via direct branches from the sacral plexus (S3–S5), with some input from the pudendal nerve, both voluntarily from higher centres in the brain and reflexly via the spinal cord.

It is known that there is a relationship between muscle function in the pelvis and pain, and studies have demonstrated that patients with pelvic pain have higher levels of resting muscle tone than normal persons.

The convergence in the spinal cord of afferent impulses from viscera, skin and muscle can also lead to the phenomenon of referred pain. Sciatica is an obvious example of this phenomenon, but it is less well known that pain may also be referred to the vulva and distal vagina. This convergence can also lead to alterations of sensation to nearby viscera, particularly the bladder. It is not uncommon for patients with vulval pain to complain of frequency, urgency, lower abdominal pain and burning on urination.

Autonomic dysfunction may lead to loss of control of the vascular system of the vulva. The result is a variable degree of erythema of the vulval skin and the epithelium of the introitus. This is often misinterpreted as a rash, and treated with corticosteroid creams. The vasoconstrictive action of these medications is followed by a reflex vasodilatation that may increase erythema and produce discomfort.

Pain sensation
Pain sensation is mediated by three types of afferent fibres, the large myelinated type A-beta fibres, the smaller myelinated type A nerve fibres and the poorly myelinated or nonmyelinated type C fibres. The type A fibres are responsible for touch and the other types are responsible for pain perception; the pain mediated by type C fibres often has a burning quality. Type A fibres can become involved in pain sensation, and when this occurs patients may develop hyperalgesia. This increased sensitivity to pain explains why patients with vulval pain often find pressure from clothes painful and difficult to cope with.

When patients experience chronic pain, afferent sensory processes appear to become sensitised, with the type C fibres of the vulval vestibule discharging more easily to lower levels of stimulation and at lower thresholds, or even spontaneously. This phenomenon has been termed ‘wind-up’. There may be associated pathological changes in the dorsal horn connection and perhaps also in higher centres. Secondarily, it is common for pelvic floor...
muscle tone to increase and, in turn, to contribute to a pain reflex via the dorsal horn; it is helpful to understand this mechanism of pain perception in a situation when there is no apparent noxious stimulus present.

The exact mechanisms involved in these phenomena are still far from clear and, outside of the specialties of neurology and pain medicine, poorly recognised. The role of higher centres is also not well understood, although it is known that depressed and anxious patients have more problems with chronic pain, and that mental state is integral to pain experience. Descending inhibitory and facilitatory signals are thought to link the limbic system, which is integral to mood and anxiety modulation, to peripheral pain sites via the spinal cord at the dorsal horn.

Despite the gaps in our knowledge it is important to have a concept of how pain can occur in the absence of observable abnormality, particularly when it comes to explaining the diagnosis to the patient and, indeed, accepting it oneself.

**Lesional and nonlesional vulvovaginal pain**

Patients presenting with vulvovaginal pain can be divided into two main groups:

- those with lesional pain – the pain is directly attributable to an observable vulval or vaginal lesion or disease
- those with nonlesional pain – the pain is experienced in the absence of any observable vulvovaginal pathology and the physical examination is normal for the patient’s age and ethnic group.

It must be remembered that the presenting symptoms may sometimes be due to coexisting lesional and nonlesional aetiologies.

**HISTORY TAKING**

Patients with vulvovaginal pain, particularly when the duration of the pain is long, are frequently distressed and frustrated, making the taking of a history challenging. We find it helpful to encourage these women to give the history chronologically. In addition to the usual history taking that applies to any patient with chronic disease, it is important in patients with vulvovaginal pain to make sure that the history of the pain is obtained and a systems review is undertaken.

**History of the pain**

Most patients with vulvovaginal pain will not volunteer the symptoms that help to make a diagnosis. They will, for example, complain about ‘reduced libido’ instead of dyspareunia; similarly, a patient is unlikely to volunteer that her vulvovaginal pain started during an episode of acute generalised psoriasis. A careful and comprehensive history of the pain is therefore essential, and should cover the duration, location and type of pain, triggers and previous treatments, as listed in Box 2.

**Systems review**

As vulvovaginal pain is often referred, a complete systems review should be undertaken (Box 2). Of particular importance is disease, dysfunction or injury to the lumbosacral spine, lower intestinal tract and anus, and lower limbs, because pain referral in the lower pelvis is usually posterior to anterior.

**How history taking helps diagnosis**

Pain due to an observable lesion (such as an ulcer or a fissure) or a dermatosis (i.e. lesional pain) is usually well localised and has the following features:

- frequently provoked by physical stimuli such as friction during intercourse or when inserting or pulling out a tampon, rubbing, scratching or wearing tight clothes
- often accompanied by the symptoms of the causative dermatosis
- usually bilateral
- often worse during the night, when there are fewer external stimuli
- resolves promptly when the underlying condition resolves. Nonlesional pain presents with more complex symptoms than lesional pain. Descriptions of this type of pain used by patients are listed in Box 3, and features include the following:

<table>
<thead>
<tr>
<th><strong>Pain history</strong></th>
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</thead>
<tbody>
<tr>
<td>• Duration of current episode of pain</td>
</tr>
<tr>
<td>• History of similar pain previously</td>
</tr>
<tr>
<td>• Historical triggers:</td>
</tr>
<tr>
<td>- exacerbating/relieving factors</td>
</tr>
<tr>
<td>- associated symptoms, e.g. itch, vaginal discharge, back pain</td>
</tr>
<tr>
<td>- history of skin disease either vulval or elsewhere</td>
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<tr>
<td>• Pain descriptors:</td>
</tr>
<tr>
<td>- sharp/dull/burn/sting/formation/stabbing*</td>
</tr>
<tr>
<td>- associated itch?</td>
</tr>
<tr>
<td>• Continuous or episodic:</td>
</tr>
<tr>
<td>- length of episodes</td>
</tr>
<tr>
<td>- triggers for episodes</td>
</tr>
<tr>
<td>• Pain location:</td>
</tr>
<tr>
<td>- on/within the labia majora</td>
</tr>
<tr>
<td>- central</td>
</tr>
<tr>
<td>- bilateral/unilateral</td>
</tr>
<tr>
<td>- anterior/posterior</td>
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<tr>
<td>- referral patterns</td>
</tr>
<tr>
<td>• Previous treatments:</td>
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<tr>
<td>- effective</td>
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<tr>
<td>- ineffective</td>
</tr>
<tr>
<td>• Impact on quality of life/sex/relationships</td>
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* More pain descriptors are listed in Box 3.
The differences between lesional and nonlesional vulvovaginal pain are summarised in the Table.

### A NOTE ABOUT VAGINAL DYSPAREUNIA

We find it more helpful to consider vaginal dyspareunia as a subset of vulvovaginal pain rather than as a separate entity. Indeed, many patients with this type of pain will say that they experience exactly the same pain when they are not attempting sexual intercourse.

Like any other vulvovaginal pain, vaginal dyspareunia may be lesional or nonlesional. It is essential to differentiate vaginal (superficial/entry) dyspareunia (felt in the vagina) from abdominal (deep) dyspareunia (felt in the abdomen). The first is usually caused by problems in the lower pelvis, and the second by problems in the upper pelvis or abdomen. There is widespread confusion about the term ‘deep’ dyspareunia, which many doctors think means ‘deep in the vagina’. Our experience is that the important distinction is between abdominal as opposed to vaginal dyspareunia, and that it does not matter (in a diagnostic sense) how deep in the vagina it is felt.

Dyspareunia can occur as the only presenting symptom whether the patient has a lesion or not. In other words, the patient has no symptoms except during intercourse and/or tampon insertion. More often dyspareunia is accompanied by pain at other times. It is the history of this background pain that will help make the diagnosis.

### Pubococcygeus muscle spasm

Before discussing vaginal dyspareunia further, it is important to mention pubococcygeal dysfunction, in which the pubococcygeus muscle goes into spasm as soon as any pressure is applied to the introitus. This is found in many patients, either as a result of any condition that causes vulvovaginal pain or, less commonly, in response to emotional stress and anxiety. The patient describes a sharp, tearing sensation on intromission and immediate relief as soon as intercourse ceases. Tampon insertion often causes the same symptoms. It is unusual for this pain to linger after intercourse ceases unless there is also a lesion that has been irritated during intercourse.

Pubococcygeus muscle spasm is detectable on per vaginal examination (Figures 1 to 5). Very severe spasm may sometimes make insertion of the examining finger impossible, and any attempt to do so is described as severe pain by the patient. However, a patient who is relaxed with you as a doctor but apprehensive about intercourse may appear deceptively normal.
Lesional dyspareunia
When dyspareunia is due to an ulcer or fissure, patients usually complain of the sort of pain experienced when a finger is cut – that is, nociceptive pain. This pain, which is mediated by nociceptors in the skin, is usually well localised and often accompanied by a small amount of postcoital bleeding (with bright red blood). If the patient has a skin condition that is prone to fissuring, such as lichen sclerosus, the pain may occur as a result of intercourse and then typically last for several days, until the fissure has healed.

Dyspareunia due to local physical causes usually improves promptly when the underlying dermatological condition is healed. However, it must be recognised that by the time effective treatment for the dermatosis is initiated, secondary pubococcygeus muscle dysfunction may have developed, and the dyspareunia will continue.

Nonlesional dyspareunia
Patients with nonlesional vaginal dyspareunia often have a history of a background of poorly localised or unilateral pain of the types listed in Box 3. The pain mechanism is either a neuropathy or pubococcygeus dysfunction. Some patients with nonlesional vulvovaginal pain deny actual dyspareunia but say that they no longer want to have sex due to fear of possible pain and low libido.

EXAMINATION OF THE PATIENT WITH VULVOVAGINAL PAIN
Patients with vulvovaginal pain are rarely so uncomfortable that they are unable to be examined but they may experience severe tenderness around the introitus and in the vagina. Speculum examination may be difficult, and we recommend a small straight-bladed instrument, even in multiparous women, but only if it is tolerated.

Step one: exclude vulvovaginal dermatoses or lesions
The most common local physical finding that causes acute vulval pain is fissure. Fissuring may occur in almost any vulval dermatosis. The typical location is at the six o’clock position on the introitus or in the sulcus between the labia minora and majora. Even tiny fissures at the introitus may cause severe dyspareunia, so it is important to look closely, especially in vulval sulci, and to gently stretch the skin.

Usually there is an accompanying vulval rash such as dermatitis, candidiasis, psoriasis or lichen sclerosus.

Other abnormalities that may cause pain include erosions and areas of inflammation, including the classic petechial rash seen in desquamative inflammatory vullovaginitis,
a typically painful condition. However, it should be ensured that the abnormal area corresponds to the location of the pain.

Surgical and obstetric scarring often make it difficult to distinguish between normal and abnormal appearances on genital skin. An assessment by an experienced gynaecologist may be necessary.

**Step two: if there is no lesion**

Note the site of the pain, including particularly whether it is unilateral or bilateral and whether it radiates. Ask the patient to locate the pain if possible. Apply light fingertip pressure to determine whether hyperalgesia exists. If the sort of pressure that would normally cause a sensation of light touch produces a sensation of pain, apply the same light pressure to the inner thigh and ask the patient if she can notice a difference.

Note whether there is introital muscular spasm. Tightness, resistance and pain on gentle downward pressure at the six o’clock position in the posterior vaginal introitus indicate this. Patients who may have bony pelvic outlet pain, related to ‘pelvic girdle’ bony dysfunction, often have point tenderness on palpation of the medial aspect of the pubic rami, and this tenderness to palpation will reproduce their pain. This examination should be performed very gently with one finger palpating the bone directly through the vaginal skin (Figures 1 to 5). This site is adjacent to the insertion of the pubococcygeus, and this muscular insertion can be avoided if the examiner is careful.

The literature on dyspareunia commonly refers to the ‘Q-tip test’ where a cotton wool tip is used to explore the introitus. We do not use this technique as we find that a cotton wool tip may irritate and that far more information is gained by the use of the educated examining finger.

The colposcopy and acetic acid technique used on the uterine cervix to detect areas of abnormality is not applicable to the vulva except when vulval intraepithelial neoplasia is suspected. The application of acetic acid to this site will make any inflammatory dermatosis appear white (and is therefore not helpful diagnostically) and also usually provokes intense pain in benign dermatoses.

**SUMMARY**

Vulvovaginal pain has discernible aetiology and pathophysiology, and in this regard is no different from pain elsewhere in the body. The presentation may be challenging, however, because of the distress and frustration caused by the pain and its effects on sexual functioning.