Chronic Abdominal Pain of Gynecologic Causes: Diagnosis and Treatment

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Introduction

Chronic pelvic pain (CPP) is a common disorder in women. One study from the United Kingdom found a prevalence of 3.8 % in women aged 15–73; higher than the prevalence of migraine (2.1 %) and similar to that of asthma (3.7 %) and back pain (4.1 %) [1]. Similarly a study in Seveso, Italy, found that 4 % of all women had moderate to severe, noncyclic pelvic pain [2]. A US study suggested a higher prevalence of 16 %, with a mean average pain score of 5, and 4 % with pain severe enough to cause them to miss work [3]. This study estimated that 9.2 million US women suffer from chronic pelvic pain.

Chronic pelvic pain leads to many medical interventions. It is the indication for 17 % of all hysterectomies in the United States [4] and more than 40 % of gynecologic diagnostic laparoscopies [5]. Overall, it is estimated that direct and indirect costs of chronic pelvic pain in the United States are over $2 billion per year [3]. At an individual level, chronic pelvic pain frequently leads to years of disability and suffering, with loss of employment, marital discord, and divorce.

CPP as defined by the American College of Obstetrics and Gynecology is nonmenstrual pain of 6 or more months' duration that localizes to the anatomic pelvis, anterior abdominal wall below the umbilicus, or the lumbosacral back and causes functional disability or requires medical or surgical treatment [6]. This definition excludes vulvar pain and cyclical pain of dysmenorrhea. However, it is important to recognize that women with chronic pelvic pain often have vulvar pain or dysmenorrhea as part of their symptom complex.

There are a myriad number of gynecologic disorders that are associated with chronic pelvic pain [7]. As stated above, chronic abdomino-pelvic pain patients should always be evaluated for non-gynecologic sources of pain which are commonly found in this population. This chapter will focus on the more commonly diagnosed gynecologic diseases associated with pelvic pain: endometriosis, leiomyomata or uterine fibroids, adenomyosis, pelvic congestion syndrome, ovarian remnant syndrome, ovarian retention syndrome, and pelvic inflammatory disease (PID). Adhesive disease can also occur in the pelvis but postsurgical adhesions will be addressed in chapter XX. Because many women with CPP also have vulvar or vaginal pain, a few of the more important disorders associated with vulvar or vaginal pain—provoked vestibulodynia (previously vulvar vestibulitis) and pudendal neuralgia—will also be briefly reviewed.

History and Physical Examination

More than 80 % of women with chronic pelvic pain have had pain for longer than 1 year when they seek medical care, and about one third have had pain for longer than 5 years [8]. Making accurate diagnoses in women with CPP can be perplexing. There are numerous possible gynecologic and non-gynecologic diagnoses (only gynecologic will be covered in this chapter). Additionally, it is likely that chronic pain itself may need to be considered a diagnosis [9].

The history and physical examination are powerful diagnostic and therapeutic tools in chronic pelvic pain. As diagnostic tools, a thorough history and examination may lead to an accurate diagnosis. This process minimizes the need for

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expensive laboratory testing and imaging or risky operative interventions. It is important that the clinician remember that even a "routine" pelvic examination is emotionally stressful for many patients with chronic pelvic pain as the exam can be painful and often the patient has undergone numerous undesired exams. Establishing rapport and trust with a patient by a compassionately taken history and a sensitively performed examination ensures that the patient feels the physician is caring and competent. Often just by giving the patient the opportunity to express her frustration with the course of her symptoms and validating her suffering allows the patient to leave the physician's office feeling better.

The history of the patient's pain must be thoroughly explored and all pertinent imaging, pathology and operative records obtained, along with the review of systems, with particular attention to the gastrointestinal, reproductive, urologic, and musculoskeletal systems. Because of the complexity of the history in most patients, intake questionnaires are extremely helpful in obtaining details of the history (see, for example, www.pelvicpain.org). However, they should not replace allowing the patient to tell her story.

Establishing the location of the patient's pain may be an important key to accurate diagnosis. Women with somatic nociceptive pain usually describe pain that is well localized to the area of disease. Such clear pinpointing is not always the case, however. For example, sometimes with levator ani pain the symptoms are described as deep, aching, heavy pain along with sharp, shooting pain and the patient cannot accurately localize the pain to the pelvic floor muscles. With visceral nociceptive pain there is almost always poor localization and usually a description of deep, dull, and cramping pain. Furthermore, because the cervix, uterus, and adnexae have the same metameric innervation as the bladder, distal ureter, lower ileum, colon, and rectosigmoid, it is often difficult to determine if visceral abdomino-pelvic pain is of gynecologic, urologic, or intestinal origin. For these reasons it is important to have the patient complete a pain map.

Women with CPP are more likely to have dysmenorrhea and dyspareunia. For example, dysmenorrhea is present in more than 80 % of women with chronic pelvic pain (in contrast to about 50 % in the general population), and dyspareunia is present in at least 40 % (in comparison to 10–15 % in the general population) [8]. The presence of either of these symptoms often is assumed to indicate a gynecological diagnosis, but dyspareunia and increased pain perimensturally are as common with IBS, IC/PBS, and pelvic floor muscle pain as with gynecological disorders.

Exploring the nature of the onset of pain may aid in diagnosis. For example, an immediately antecedent trauma, such as a fall, surgery, or motor vehicle accident, suggests a musculoskeletal cause. Pain that started with a pregnancy or immediately postpartum may suggest peripartum pelvic pain syndrome. Pain that started at or soon after menarche as dysmenorrhea, progressed to premenstrual pain, and then became constant suggests endometriosis or adenomyosis. If pain started soon after a physical or sexual assault, it may have significant musculoskeletal or psychological components.

Elucidation of any temporal pattern to the pain may be helpful. Cyclicity related to menses suggests gynecologic pain, but is not pathognomonic of gynecologic disease. The same pattern may occur with pain of intestinal, urologic, or musculoskeletal origin also. For example, symptoms of IBS or IC/PBS frequently increase premenstrually. A history of pain or increased pain with coitus is frequently present, as just discussed, and may be due to a variety of disorders, including psychological disease, marital problems, endometriosis, vulvodynia, IC/PBS, and IBS. If intercourse is painful, it is important to find out if pain is with entry at the outermost part of the vagina or if it is with deeper penetration high in the vagina or pelvis, or both. Diseases associated with chronic pelvic pain are not generally associated with entry dyspareunia, except as provoked vulvodynia or vaginismus.

The quality or nature of pain should be sought. For example, neuropathic pain is often described as burning or sharp and piercing, with an electric shock-like quality. Muscular pain may be aching in quality, with sharp, lancing pain with changes in position. Similar qualities of aching with occasional intermittent sharp and radiating pains may also be described with visceral pain. Pain with endometriosis is usually described as cramping [10].

Finding out about any prior treatments for chronic pelvic pain and the response to those treatments is a crucial part of the history. It may be important to know about any prior surgery, not just surgical treatment for pain, because postoperative surgical pain may be a risk factor for or proximate cause of chronic pelvic pain.

Ideally a thorough psychosocial history should be obtained on every patient with chronic pelvic pain. An extensive evaluation by a psychologist or similarly educated professional cannot always be done—nor is it always necessary. However, a basic psychosocial history is always important, especially asking about catastrophizing, anxiety, and depression. Depression, in particular, is one of several predictors of pain severity in women with chronic pelvic pain, and it is also a significant indicator of responsiveness to treatment. Asking about abuse, which may be difficult, is nevertheless another important part of the psychosocial history [11, 12]. Although there is a significant association between physical and sexual abuse and the development of chronic pelvic pain, the presence of moderate or severe depression or history of abuse was not found to be associated with a decreased response to treatment [13].

The physical examination should seek to find the exact anatomic locations of any areas of tenderness and, as much as possible, correlate these with areas of pain. This type of "pain mapping" examination requires a systematic and methodical

attempt to duplicate the patient's pain by palpation, positioning, or bodily movement. At any tender areas or painful positions, the patient should be asked whether the pain produced is the same as her chronic pain. The examination should evaluate the musculoskeletal, gastrointestinal, urinary, and neurological systems, not just the reproductive tract. It facilitates the examination to divide it into standing, sitting, supine, and lithotomy components. As the pelvic examination is particularly relevant for gynecologic disorders, it is the only component of the examination that will be reviewed in detail in this chapter.

Vulvar examination starts with careful visualization of the vulva, perineal body, and anus looking for abnormal pigmentation or erythema, masses, and any skin or mucosal lesions. Palpation should be done with a moistened cotton-tipped swab to evaluate the vulva and the vulvar vestibule for tenderness. This is particularly useful in patients with localized provoked vestibulodynia (vulvar vestibulitis), who have exquisite tenderness in localized areas at the minor vestibular glands just external to the hymen, with normal sensation in adjacent vulvar areas.

A single-digit examination, using only one hand, is how the pelvic examination should be initiated. The introital bulbo-carvenosus and transverse perineal muscles, then the levator ani muscles, should be palpated for tone, spasm, and tenderness. In patients with pelvic floor pain this palpation may cause pain consistent with at least part of the patient's clinical pain symptoms. Pelvic floor pain may also result from trigger points of one or more of the muscles of the pelvis. The piriformis, coccygeal, and internal obturator muscles should be thoroughly evaluated using single-digit examination. The piriformis muscles can be difficult to evaluate transvaginally, however. Rectal examination may allow an easier evaluation than vaginal examination. Transvaginally or transrectally the examining finger is pressed posterolaterally just superior to the ischial spine. In the lithotomy position, if the patient is asked to abduct the thigh against resistance as the piriform muscle is palpated, the muscle may be more easily palpated, and there is exquisite tenderness of the muscle if there is spasm or tension myalgia involving the piriform muscle (piriformis syndrome).

The anterior vaginal, urethral, and trigonal areas should be palpated to elicit any areas of tenderness, induration, discharge, or thickening suggestive of chronic urethritis, chronic urethral syndrome, urethral diverticulum, vaginal wall cyst, trigonitis, or interstitial cystitis. With deeper palpation the cervix, paracervical areas, and vaginal fornices should be palpated with the single digit for tenderness or trigger points suggestive of problems such as repeated cervical trauma (usually from intercourse), pelvic infection, endometriosis, ureteral pain, or trigger points.

The uterus usually can be adequately evaluated for tenderness by direct palpation with a single digit. Significant uterine tenderness may be consistent with diseases such as adenomyosis, pelvic congestion syndrome, pelvic infection sequelae, endometriosis, or premenstrual syndrome. A uterus that is immobile and fixed in position, especially a retroflexed one, may suggest endometriosis or adhesions. The coccyx can be palpated with the single digit, and an attempt should be made to move it 30° or less. This may be easier to evaluate during the rectovaginal examination. Normally the coccyx moves 30° without eliciting pain, but in patients with coccygodynia this movement elicits pain. The ureteral and the adnexal areas should be palpated next, still using a single digit without the use of the abdominal hand. All of the preceding evaluations are "monomanual-monodigital"—that is, only one finger of one hand is used. No abdominal palpation with the other hand is involved.

The traditional visual, speculum, and bimanual examinations are still needed for a thorough evaluation, but they should usually follow the single-digit examination. A cotton-tipped swab can be used to evaluate the cervical os and the paracervical and cervical tissues for tenderness. In posthysterectomy patients the full vaginal cuff should be similarly palpated for tenderness with a cotton-tipped swab. Any pain or tenderness elicited with the bimanual examination is less specific, because it involves stimulation of all layers of the abdominal wall, the parietal peritoneum, and the palpated organ or organs. Including the rectovaginal examination is important in most women with chronic pelvic pain, looking particularly for nodularity and tenderness.

Endometriosis

Endometriosis is often blamed as the source for CPP but several studies on CPP patients found that the most frequent disorders were in fact non-gynecologic [8, 13] and one found that only 15 % of patients had confirmed endometriosis [13]. Endometriosis is the presence of ectopic endometrial glands and stroma, that is, endometrium located outside of the endometrial cavity. Endometriosis may be found in many locations in the body, but most often is found in the pelvis. Endometriosis remains an enigmatic disorder in that the etiology, the natural history, and the precise mechanisms by which it may cause pain are not completely understood.

Endometriosis has a well-recognized direct association with dysmenorrhea, dyspareunia, and chronic pelvic pain, but it may also be a risk factor for the development of non-reproductive tract CPP. For example, women with dysmenorrhea or endometriosis have increased episodes and increased severity of pain related to urinary calculi than women without dysmenorrhea or endometriosis [14]. Similar results have been experimentally demonstrated for vaginal pain, as well [15]. Also, women with endometriosis have been shown to have an increased incidence of interstitial cystitis/bladder pain syndrome [10, 16]. Such viscero–visceral

interactions may have a significant role in chronic pelvic pain in women, reflecting central sensitization, explaining in part why some women with a past history of endometriosis have persistent pelvic pain after their endometriosis is gone [17].

Neither the etiology of endometriosis nor the etiology of pain associated with endometriosis is completely understood. The etiology of endometriosis is complex, but it seems certain that both genetic and environmental factors contribute to the disease phenotype. There are several general theories regarding the etiology of endometriosis but none is sufficient to explain the protean manifestations of endometriosis, or the predilection of some women, but not others, to develop symptomatic endometriosis. For example, the most widely spread theory, retrograde menstruation occurs in most women but only 5–10 % develop endometriosis [18].

Endometriosis is a disease of women of reproductive age, so most women with endometriosis-associated pain are 20–45 years of age. However it has been reported in girls as young as 10 years and it may be a more common cause of pain in teenagers than is generally recognized. It may also occur in postmenopausal women, particularly if they are on estrogen replacement.

Classically the woman with endometriosis presents with one or more of the following triad: an adnexal mass (endometrioma), infertility, or pelvic pain [19]. Pelvic pain most often starts as dysmenorrhea and about 90 % of women with endometriosis-associated pelvic pain have dysmenorrhea as a component of their pain symptoms. Perimenstrual exacerbation of pain can also occur in women with IC/PBS and IBS, and menstrual suppression may reduce these pain flares. Also, these three common diagnoses frequently occur together. One study found only 18 % of women with endometriosis had only endometriosis, while 32 % also had IC/PBS and 31 % had IBS [10]. Dyspareunia with deep penetration is also a frequent component of endometriosis-associated pain, occurring in about 40 % of cases. Intestinal involvement occurs in about 15 % of women with endometriosis and may be associated with gastrointestinal symptoms of tenesmus, dyschezia, constipation, diarrhea, low back pain, and, rarely, hematochezia or symptoms of bowel obstruction. Urinary tract involvement occurs in about 10 % of women with endometriosis and may be associated with urinary frequency, pressure, dysuria, or hematuria.

In many women with endometriosis-associated pelvic pain the physical examination is completely normal. In others there is tenderness and other findings only during menses. For this reason it is sometimes helpful to do the examination during the first day or two of menstrual flow in women with suspected endometriosis. Some women with endometriosis have persistent areas of tenderness in the pelvis, whether or not they are menstruating. Classic physical findings include a fixed retroverted uterus with tenderness posteriorly, tender nodularity of the uterosacral ligaments, and cul-de-sac on rectovaginal examination. However, these are not commonly found. Asymmetrically enlarged, tender ovaries that are fixed to the broad ligaments or pelvic sidewalls may also occasionally be found.

The symptoms and signs that lead to a clinical diagnosis of endometriosis are reliable 65–80 % of the time, but an accurate diagnosis can only be made by surgical excision with histologic confirmation [20, 21]. Accurate diagnosis requires microscopic visualization of both endometrial glands and stroma [22, 23]. As endometriosis may have a wide variety of gross appearances, it is essential that the surgeon be familiar with the variety of potential appearances of endometriosis for accurate diagnosis [22, 23]. A negative laparoscopy should lead one to consider other diagnoses such as IC/PBS or IBS, which can mimic endometriosis.

Treatment of endometriosis is also complex and many factors must be considered in planning treatment. It is important to educate the patient about endometriosis and the treatment options and actively involve her in decision-making. The patient's age, reproductive plans, duration of infertility, and attitude toward surgery or toward hormonal medications may be vital components of the patient's needs or concerns.

There are many medical treatments available for endometriosis-associated pelvic pain. Only those used commonly with good evidence of efficacy will be reviewed here.

Medical treatment with gonadotropin releasing hormone (GnRH) agonists, progestins, danazol or combined oral contraceptives effectively relieves endometriosis-associated pelvic pain. The number needed to treat for medical treatments is 2–2.5 [24].

GnRH agonists shut down LH and FSH production and release, leading to a dramatic decline in estradiol levels, induction of amenorrhea, and improvement of pain levels. When patients have a recurrence of pain within 1 year after treatment with GnRH analogs, re-treatment appears to be reasonably effective, with about two-thirds of patients showing a significant reduction of pain levels during re-treatment [25]. Loss of bone density with GnRH analogs is a serious concern. Clinical trials with GnRH agonists show that add-back therapy with conjugated equine estrogen and/or norethindrone acetate significantly decreases bone loss [26].

Danazol, a 17-ethinyl-testosterone derivative, has efficacy similar to that of GnRH agonists, but is not as frequently used due to possible androgenic side effects, including significant weight gain, mood changes, and masculinizing symptoms [27].

Medroxyprogesterone acetate (MPA) has been a recommended treatment for many years. Although a high dose of 100 mg/day was used in the only placebo-controlled trial of MPA, lower doses are used generally in clinical practice [28]. A randomized study of a 104 mg dose of a subcutaneous formulation of MPA compared to depot-leuprolide showed similar efficacies for both treatments [29].

*Oral contracept*ive (OCP) treatment of endometriosis is a long-standing approach, using either cyclical or continuous dosing. Efficacy appears to be similar or somewhat less than the other hormonal treatments [30]. There is some evidence that women not responsive to cyclical administration of OCPs may respond to continuous administration [31].

Some more recent approaches to medical treatment of endometriosis-associated pelvic pain are levonorgestrel-releasing intrauterine device [32], aromatase inhibitors [33, 34], selective progesterone modulators [35], and other progestins [36]. Although data from randomized clinical trials are needed, current evidence suggests that these therapies are effective alternatives in many patients.

Surgical treatment can be done at the time of laparoscopic diagnosis in symptomatic women. Organ-preserving, laparoscopic surgical treatment has been shown to significantly improve pain in women with stage II, III, and IV endometriosis, with a number needed to treat of 2–2.5 [37, 38]. Surgery for advanced-stage endometriosis can be challenging, tedious, frustrating, and prone to complications, so it is likely that surgical outcomes are surgeon dependent. Postoperative medical treatment may improve pain relief for the duration of the medical treatment, but does not improve long-term outcomes [28, 39, 40].

Presacral neurectomy (resection of the superior hypogastric plexus) and uterosacral neurectomy (uterine nerve resection or transection of the uterosacral ligament) have been recommended for relief of CPP associated with endometriosis. Data from clinical trials show that presacral neurectomy somewhat improves pain relief obtained with surgical treatment of endometriosis [41, 42]. However, presacral neurectomy may lead to intractable constipation and urinary urgency in up to 5 % of patients [43]. Data from clinical trials show that uterosacral neurectomy does not improve pain relief when included in surgical treatment of endometriosis and should not be performed [44].

If fertility is not desired, then hysterectomy, with or without bilateral salpingo-oophorectomy, is often recommended for endometriosis-associated pelvic pain. There is no consensus as to the advisability of removal of both ovaries if one or both are not directly involved by endometriosis. In one study evaluating this dilemma, recurrence of pain when one or both ovaries are preserved has been reported to occur in 62 % of cases compared to 10 % when both ovaries are removed, giving a relative risk for pain recurrence of 6.1 (confidence interval 2.5–14.6) [45]. Reoperation for pain was also more likely with ovarian preservation, with 31 % requiring reoperation compared to 4 % if both ovaries were removed at the time of hysterectomy for endometriosis. Although uncommon, endometriosis has been reported to recur after hysterectomy and bilateral salpingo-oophorectomy, with and without estrogen replacement therapy [46–48]. The potential risks of cardiovascular and bone density risks in premenopausal women without estrogen replacement likely outweigh the rare risk of recurrence.

Complications of medical treatment include side effects of weight gain, edema, hot flushes, headaches, nausea, acne, hirsutism, hot flushes, abnormal uterine bleeding, decreased breast size, decreased libido, vaginal dryness, weakness, decreased bone density, and thromboembolic disease. Surgical complications vary with the severity of disease, but injury to pelvic viscera is a potential risk in women with endometriosis. Endometriosis untreated is rarely life threatening, although there are cases of ureteral and bowel obstruction due to endometriosis, as well as invasion of the urinary and gastrointestinal tracts.

It is important to recognize that the finding of endometriosis in women with CPP does not ensure that medical or surgical treatment of endometriosis will result in effective relief of pain. To the contrary, although treatment of endometriosis in women with pelvic pain is clearly indicated based on randomized, placebo-controlled, double-blind clinical trials, pain relief of 6 or more months duration due to treatment can be expected in only about 40–70 % of women with endometriosis-associated CPP (number needed to treat of 2.0–2.5). Also, recurrence rates of CPP are high after medical and surgical therapy. Women with endometriosis need to be evaluated for all sources of chronic abdomino-pelvic pain including non-gynecologic to ensure appropriate treatment for their CPP.

Pelvic Congestion Syndrome

Pelvic Congestion Syndrome (PCS) is defined by the triad of pelvic pain, pelvic varicosities, and abnormal venous function. Abnormal venous function is identified by a pelvic venogram. Pain is usually worse premenstrually rather than having pain levels which peak with menses. Symptoms often develop after childbirth as pregnancy increases the capacity of pelvic veins by 60 % [49]. Both of these are likely related to hormonal changes affecting the pelvic vasculature associated with pregnancy and the menstrual cycle. In addition, pain is usually dull and aching, with exacerbations related to movement and position changes such as sitting to standing, or left to right side while supine, and may improve after lying down. Backaches which worsen with standing can also occur. Deep dyspareunia and post-coital pain are very common. On abdominal exam, palpation at the ovarian point can reproduce the pelvic pain in 80 % of women with PCS. This point lies at the junction of the upper and middle thirds of a line drawn from the anterior superior iliac spine to the pubic symphysis. Adnexal tenderness may also be found on bimanual exam.

Diagnosis is confirmed by a pelvic venogram showing venous stasis, dilation, delayed emptying and a plexus formation of the ovarian or uterine vessels. This can be

performed by transcervical injection of the myometrium at the fundus or by transcutaneous retrograde injection of the ovarian veins by interventional radiology with serial imaging. It cannot be diagnosed on ultrasound or CAT scan or by simply visualizing enlarged pelvic vessels on laparoscopy.

Treatment can be either menstrual suppression with progestin hormonal therapy, with GnRH agonists, with embolization of the dilated ovarian vessels by interventional radiology, or for refractory cases in women done with childbearing, hysterectomy and bilateral salpingo-oophorectomy. Symptom improvement after embolization may take several months, but long-term success in a small 3-year follow up study has been reported at 76 % [50].

Pelvic Inflammatory Disease and Adhesions

Up to 36 % of women with acute PID may develop chronic abdomino-pelvic pain [51]. This association has been reported on extensively in the literature and is widely accepted. Recurrent PID appears to exponentially increase the risk of subsequent CPP in a follow up of patients for 84 months [52]. PID is more commonly found among teenagers and women under age 25; this may be related to increased susceptibility of the endocervical glandular cells to ascending infection in addition to behavioral differences related to age and maturity. PID can result in adnexal adhesions, tubo-ovarian abcesses, hepatic adhesions, and hydrosalpinges.

The mechanism by which 1/3 of women with PID develop CPP is not well understood but may be related to inflammatory mediated changes and possibly adhesion formation. PID can also result from pelvic tuberculosis in women from high-risk countries or those with high-risk status such as HIV patients. PID can be associated with appendicitis due to appendiceal-adnexal adhesions and similarly with colonic diverticulitis or inflammatory bowel disease. The connection between adhesions and CPP is also not well understood as findings of intra-abdominal and pelvic adhesions are often incidental in many patients without pain. Adhesions are easily blamed for pain if present in patients with CPP, but not clearly demonstrated as the etiology. Two randomized trials of adhesiolysis for CPP failed to show significant improvement after both a laparoscopic and laparotomy approach [53, 54]. Additionally, there are no randomized controlled trials which demonstrate effective means by which to prevent adhesions from reforming. Adhesions and chronic abdominal pain will be addressed in more detail in Chapter XX.

Adenomyosis

Adenomyosis is a benign condition in which endometrial glands and stroma are found in the myometrium (invading past the endometrial cavity). The true incidence is unknown, since the final diagnosis is done with a pathological specimen after hysterectomy. It is thought to be most prevalent among perimenopausal and multiparous women between 40 and 50 years of age.

The classic presentation is a triad of abnormal uterine bleeding (50 %), prolonged dysmenorrhea (30 %), and an enlarged "globular," tender uterus [55]. Menorrhagia (heavy menstrual bleeding) can cause dysmenorrhea from stimulation and edema of endometrial tissue within the myometrium [56]. These symptoms start perimenstrually, are cyclic and often last the entire reproductive age if untreated. They can be associated with chronic pelvic pain. The symptoms can overlap with uterine fibroids and endometriosis. However, endometriosis does not classically present with heavy menses or irregular menses, and fibroids are easily distinguished on ultrasound.

As described previously the final diagnosis is made with histological examination after hysterectomy, which would not be an option for someone desiring future fertility. Imaging is another modality that is used. Clinically, dysmenorrhea, a tender, boggy uterus on bimanual exam, the absence of fibroid uterus on ultrasound and an ultrasound report of heterogenous texture of the myometrium with an enlarged uterus without discrete masses will help make the diagnosis. While MRI has a sensitivity of 88–93 % and specificity 66–91 % compared to transvaginal ultrasound, it rarely uses first line as the final diagnosis is made by histology, not by a suspicious MRI, and treatment does not vary by MRI findings.

The definitive treatment for this adenomyosis is hysterectomy. When hysterectomy is contraindicated *or undesired by the patient*, a number of conservative treatments can be considered [55]. Hormonal therapy with oral progestins, levonorgestrel IUD, gonadotropin releasing hormones agonists, aromatase inhibitors, and danazol may control symptoms. Oral contraceptives have not generally been effective, but may be tried as a simple first time treatment. Uterine artery embolization (UAE) has been used for treatment of adenomyosis but is less well studied and accepted than for uterine fibroids. A review article of the six small uncontrolled studies of an UAE for adenomyosis, with a total of 208 patients, found only a 65 % improvement at 40 months of follow up [57]. Larger studies are needed before UAE will be accepted widely as a viable treatment option for adenomyosis. In patients with focal disease or with adenomyomas, surgical excision can be done but long-term recurrence risks are unknown.

Uterine Leiomyomas

Uterine leiomyomas or fibroids are the most common benign gynecologic tumors. They originate from the myometrial cells. These tumors are hormonal dependent, associated mainly with estrogen, growth hormone, and progesterone,

estrogen being the main growth stimulator and progesterone appearing to inhibit growth. There are different types of fibroids: subserosal, intramural and submucosal. Subserosal myomas originate at the external portion of the myometrium and often protrude out into the abdominal cavity and may cause pressure symptoms or be asymptomatic. Intramural myomas are located completely within the muscular wall of the uterus and often cause menorrhagia and dysmenorrhea. Submucous myomas are close to the endometrium and impinge on the endometrial cavity and are associated with irregular heavy bleeding (menometrorrhagia).

The estimated incidence of fibroids ranges from 33 to 77 %, depending on the method of diagnosis (i.e., clinical, ultrasound, pathology) [58]. Fibroids are most often identified during reproductive age, with most women being in their 30–40s. Symptoms vary depending on the number, size, and location. Abnormal uterine bleeding is the most frequent symptom associated with fibroids, usually presenting as cyclical, heavy menses. The mechanism of abnormal uterine bleeding is unknown but may be caused by dysregulation of angiogenic factors [59].

Pelvic pressure and pain are symptoms associated with the size of the uterus and the location of the fibroids. A fibroid pressing on the bladder or ureter may cause symptoms of urinary frequency, incontinence urinary retention, or hydronephrosis secondary to ureteral obstruction. A fibroid pressing on the rectum may cause symptoms of constipation or low back pain.

The diagnosis is made by physical examination and imaging studies. A pelvic exam will show a pelvic mass that can be identified as a large irregular uterus. Transvaginal and transabdominal ultrasounds have a high sensitivity (90–100 %) and specificity (87–98 %), with a positive predictive value of 81–93 % and a negative predictive value of 98–100 % [60]. Ultrasound is the preferred and most cost effective way of diagnosing a fibroid uterus. MRI can be used to assess the location, size, and depth of fibroids for surgical planning of a myomectomy but it is not used for routine screening. Treatment options for leiomyomas are expectant, medical, interventional radiologic, or surgical.

Expectant management or observation is reasonable in patients with fibroids that are asymptomatic.

There are a number of options for medical management. Combined oral contraceptive pills and oral or injectable progestins are useful for menstrual abnormalities. Levonogestrel intrauterine device also is useful for menstrual abnormalities and has the benefit of reduced hormonal side effects in the patients.

Gonadotropin releasing hormone (GnRH) agonists lead to amenorrhea in most patients and provide a 35–65 % reduction in fibroid volume over 3 months. GnRH agonists are most often used as presurgical therapy for 3–6 months to treat anemia and facilitate less complicated surgery. Long-term use has bone density reduction risk which can be prevented with add-back hormone therapy such as an oral norethindrone acetate.

Mifepristone, a progesterone modulator, has been shown to reduce the volume of fibroids and to induce amenorrhea without the concern for bone density loss. Known side effects include risk of endometrial hyperplasia and transient elevation of transaminase levels. Further studies are needed to evaluate the safety and best use of this class of treatment [61].

Uterine artery embolization (UAE) is performed primarily by interventional radiologists. The approach is done via transcutaneous femoral artery approach to identify and embolize major blood supply to fibroids and is performed primarily by interventional radiologists. It should be used with caution when the patient desires to retain her ability to conceive because age-related amenorrhea can occur and abnormal placentation is possible [62]. Several long-term studies show a higher rate of clinical failures and reoperation rates [61]. Higher success rates may be possible in well selected patients who are closer to menopause.

MRI-guided focused ultrasound was FDA approved in 2004 but there are no long-term studies over 24 months. A high intensity, directed ultrasound approach.

Surgical treatment is by myomectomy or hysterectomy. Myomectomy is surgical excision of the fibroids and is an option for patients who wish to retain their fertility or their uterus. However, it is not definitive treatment as studies have shown that the increased risk of recurrence is associated with the number of fibroids present [61]. Women should be appropriately counseled about their recurrence risk and subsequent need for another procedure or hysterectomy in the future. Hysterectomy is the definitive surgical treatment for a symptomatic fibroid uterus.

Provoked Vulvodynia or Vestibulodynia

This disorder (previously called vulvar vestibulitis) is associated with dyspareunia (pain with intercourse). Pain is present on light touch of the vulvar vestibule in the absence of other findings [63]. Vulvovaginal infections such as candida vaginitis and dermatoses such as lichen sclerosis should be ruled out and any visible lesions biopsied to rule out malignancy. The etiology is unknown. Patients with this disorder also are commonly found to have disorders associated with chronic pain such as depression, IBS, and pelvic floor tension myalgia [64]. Dyspareunia related to this disease can severely affect a patient's quality of life.

Due to the unclear etiology, treatments vary widely and include oral tricyclic antidepressants, gabapentin, steroid creams or injections, topical anesthetic ointments, physical therapy, avoidance of surface irritants, biofeedback, and cognitive-behavioral therapy. In patients with hypoestrogenic

vaginal atrophy, correction with topical estrogen may be helpful. Such cases may be postmenopausal women or, more rarely, women on hormones such as depo-medroxyprogesterone acetate. A recent randomized controlled trial of oral desipramine with and without lidocaine ointment failed to show a reduction in pain compared to placebo [65]. This approach is commonly used to treat both central and peripheral neuropathology. The authors theorized that cream massage of the control ointment, weekly telephone surveys, and medication instructions by an RN and the natural history of the disease process may all have contributed to the improvement among the control group.

For treatment of cases resistant to these therapies, surgical treatment with vulvar vestibulectomy is often performed. It involves excision of the vestibule and possibly vaginal advancement. It does result in disfigurement of the introitus; so many patients are hesitant to use this as a first line treatment. A randomized trial by Bergeron et al. compared vestibulectomy, group cognitive-behavioral therapy and surface electromyographic biofeedback. They found that at 6 months, while all groups had decreased pain, vestibulectomy patients had significantly lower pain levels and were significantly more improved than the other two [66]. They cautioned that there were a larger number of patients who refused vestibulectomy and dropped out before treatment. Their subsequent follow up study at 2.5 years (51 of the original 78) found that these results persisted [67]. They found that higher pretreatment pain intensity predicted poorer 2.5 year outcomes for all groups and that erotophobia predicted a poorer outcome for vestibulectomy [67].

Pelvic Floor Tension Myalgia

Musculoskeletal disorders were found in 23 % patients with CPP in a specialty practice [13]. These include disorders such as sacroiliac joint dysfunction, coccygodynia, and low back pain as well, the more common diagnoses of pelvic floor tension myalgia (PFTM) and abdominal wall myofascial pain (which is covered in Chapter XX). A myofascial trigger point is a hyperirritable spot within a tense band of muscle or fascia which is painful on compression and usually causes referred pain and other sensory disturbances [68, 69]. Patients with CPP most often have these points in their abdominal wall (i.e., rectus abdomins and external and internal obliques) and in their pelvic floor muscles (i.e., levator ani and obturator internus). However, they can also have trigger points in the lumbar and gluteus muscles. Trigger points of the abdomen and pelvic floor are found with a single digit palpation applying pressure along the muscle belly and fascial insertions. These myofascial pain syndromes can occur as a sole cause of CPP or can occur in conjunction with other disorders such as IC/PBS, provoked vestibulodynia, or endometriosis [70]. They can develop after an acute traumatic event, repetitive microtrauma or as a result of chronic muscle tension and muscle shortening from pelvic girdle dysfunction, short leg syndrome or as a response to visceral pain and inflammation from the above disorders (IC/PBS, endometriosis, etc.).

Successful long-term treatment can occur with weekly physical therapy to recruit inadequately used muscles of the pelvic girdle and relax the contracted muscles. Other treatment options are trigger point injections with local anesthetics, dry needling, muscle relaxants, moist heating pads, transcutaneous electrical stimulation (TENS) units, yoga or stretching exercises, and massage, and specifically for PFTM, vaginal diazepam 5–10 mg twice daily. The therapist treating PFTM should be specially trained because exercises such as Kegels will worsen their symptoms and the patient will stop therapy. Trigger point injections are less frequently used for PFTM as the pain associated the injections is severe. Adequate treatment by trigger point injections usually requires visits every 2–6 weeks, which may not be feasible for many patients. Successful injections often result in decreased pain for several weeks. There are no prospective randomized trials to promote one treatment over another.

Pudendal Neuralgia

Pudendal neuralgia (PN) currently is best diagnosed using the "Nantes Criteria" [71, 72]: (1) pain in the anatomical distribution of the pudendal nerve; (2) pain more severe when sitting; (3) pain that does not awaken from sleep; (4) no objective sensory loss on clinical examination; and (5) pain that is relieved by diagnostic pudendal nerve blocks. The diagnosis is likely if all five criteria are met. The differential diagnosis includes recurrent vaginitis, non-provoked vulvodynia, provoked vestibulodynia, pelvic congestion syndrome, and pelvic floor tension myalgia.

There sometimes is a history of long bicycle rides, episodes of prolonged sitting, or pelvic trauma immediately preceding the onset of symptoms. Also, reconstructive pelvic surgery can damage the nerve from compression, scar tissue formation, or direct impingement. Frequently no reason for the onset of symptoms can be elicited.

PN can affect any part of the perineum and vagina including the labia, vestibule, mons pubis, urethra, clitoris, anus and rectum, following the distribution of the nerve. Symptoms can include hyperesthesia, allodynia, paresthesias, burning or stabbing pain, sensations of incomplete voiding or inability to void normally, overactive bladder, dyspareunia, along with bowel dysfunction, sensation of a mass in the rectum, and pain with defecation; sensory symptoms should follow the course of the nerve.

The pudendal nerve arises from the sacral plexus (S2–S4) and then splits up into the anorectal, perineal, and clitoral

branches. There are several locations in the course of the nerve where damage or entrapment occurs, but the most common are between the sacrospinous and sacrotuberous ligaments around the ischial spine (80 %), and in Alcock's canal (20 %). Image-guided pudendal nerve blocks at these locations are both diagnostic and therapeutic. If the pain is relieved after injections (local anesthetic with or without steroids) then most likely the source of the pain has been identified. If the pain is not relieved, then the source may not be the pudendal nerve. A series of nerve blocks can be completed which may control the symptoms. Medical management with a tricyclic antidepressant or gabapentin is often helpful. Other treatment options include pelvic floor physical therapy, pregabalin, oral muscle relaxants, and local muscle relaxants (vaginal diazepam and rectal belladonna and opium suppositories) [72]. Surgical decompression of the nerve has been reported to have a number needed to treat of 1.7, compared to medical treatment only, in a non-blinded randomized trial [73].

Ovarian Remnant Syndrome and Ovarian Retention Syndrome (Residual Ovary Syndrome)

Ovarian remnant syndrome is the presence of painful, histologically documented ovarian tissue in a patient who has undergone a previous bilateral oophorectomy. Often the ovarian tissue is found adherent to bowel or to the pelvic sidewall. It may rarely occur in a patient who has undergone a unilateral oophorectomy, with painful, persistent ipsilateral ovarian tissue. It may be a more common cause of chronic pelvic pain than is generally recognized [74]. A normal FSH hormone level in a patient of hormone replacement often can make the diagnosis. In some cases ovarian stimulation with GnRH agonist documented by increase of estradiol levels before and after stimulation will confirm the diagnosis. If the location of the remnant ovarian tissue is unclear despite imaging, stimulation of ovarian tissue to enlarge it with clomiphene citrate may aid in finding the remnant tissue both on imaging and on diagnostic laparoscopy. Although laparoscopy may have a role in diagnosis and treatment in some cases these remnants are often embedded in dense scar tissue and the surgery is difficult laparoscopically unless extensive adhesiolysis is performed [75].

Another uncommon cause of ovarian cysts and chronic pelvic pain is residual ovary syndrome or ovarian retention syndrome. Ovarian retention syndrome is the presence of persistent pelvic pain, dyspareunia, or a pelvic mass after conservation of one or both ovaries at the time of hysterectomy.

Pain due to ovarian retention syndrome is usually relieved by complete ovarian suppression with GnRH-a treatment [76]. Definitive treatment is surgical extirpation and can sometimes be performed laparoscopically [77]. In young women in whom preservation of ovarian function is desired, adhesiolysis and ovarian cystectomy may be tried, but appears to be less likely to relieve pain.

References

1. Zondervan KT, Yudkin PL, Vessey MP, Dawes MG, Barlow DH, Kennedy SH. Prevalence and incidence of chronic pelvic pain in primary care: evidence from a national general practice database. Br J Obstet Gynaecol. 1999;106(11):1149–55.
2. Lippman SA, Warner M, Samuels S, Olive D, Vercellini P, Eskenazi B. Uterine fibroids and gynecologic pain symptoms in a population-based study. Fertil Steril. 2003;80(6):1488–94.
3. Mathias SD, Kuppermann M, Liberman RF, Lipschutz RC, Steege JF. Chronic pelvic pain: prevalence, health-related quality of life, and economic correlates. Obstet Gynecol. 1996;87(3):321–7.
4. Hillis SD, Marchbanks PA, Peterson HB. The effectiveness of hysterectomy for chronic pelvic pain. Obstet Gynecol. 1995;86(6):941–5.
5. Howard FM. The role of laparoscopy in chronic pelvic pain: promise and pitfalls. Obstet Gynecol Surv. 1993;48(6):357–87.
6. ACOG Committee on Practice Bulletins—Gynecology. ACOG Practice Bulletin No. 51. Chronic pelvic pain. Obstet Gynecol. 2004;103(3):589–605.
7. Howard FM. Chronic pelvic pain. Obstet Gynecol. 2003;101(3):594–611.
8. Zondervan KT, Yudkin PL, Vessey MP, et al. Chronic pelvic pain in the community—symptoms, investigations, and diagnoses. Am J Obstet Gynecol. 2001;184(6):1149–55.
9. Tracey I, Bushnell MC. How neuroimaging studies have challenged us to rethink: is chronic pain a disease? J Pain. 2009;10(11):1113–20.
10. Droz J, Howard FM. Use of the short-form McGill Pain Questionnaire as a diagnostic tool in women with chronic pelvic pain. J Minim Invasive Gynecol. 2011;18(2):211–7.
11. Elzevier HW, Voorham-van der Zalm PJ, Pelger RC. How reliable is a self-administered questionnaire in detecting sexual abuse: a retrospective study in patients with pelvic-floor complaints and a review of literature. J Sex Med. 2007;4(4 Pt 1):956–63.
12. Wijma B, Schei B, Swahnberg K, et al. Emotional, physical, and sexual abuse in patients visiting gynaecology clinics: a Nordic cross-sectional study. Lancet. 2003;361(9375):2107–13.
13. Lamvu G, Williams R, Zolnoun D, et al. Long-term outcomes after surgical and nonsurgical management of chronic pelvic pain: one year after evaluation in a pelvic pain specialty clinic. Am J Obstet Gynecol. 2006;195(2):591–8; discussion 598–600.
14. Giamberardino MA, De Laurentis S, Affaitati G, Lerza R, Lapenna D, Vecchiet L. Modulation of pain and hyperalgesia from the urinary tract by algogenic conditions of the reproductive organs in women. Neurosci Lett. 2001;304(1–2):61–4.
15. Berkley KJ, Cason A, Jacobs H, Bradshaw H, Wood E. Vaginal hyperalgesia in a rat model of endometriosis. Neurosci Lett. 2001;306(3):185–8.
16. Chung MK, Chung RR, Gordon D, Jennings C. The evil twins of chronic pelvic pain syndrome: endometriosis and interstitial cystitis. JSLS. 2002;6(4):311–4.
17. Stratton P, Berkley KJ. Chronic pelvic pain and endometriosis: translational evidence of the relationship and implications. Hum Reprod Update. 2011;17(3):327–46.
18. Guo SW. Epigenetics of endometriosis. Mol Hum Reprod. 2009;15(10):587–607.
19. Adamson GD. Diagnosis and clinical presentation of endometriosis. Am J Obstet Gynecol. 1990;162(2):568–9.

20. Sutton C, Hill D. Laser laparoscopy in the treatment of endometriosis. A 5-year study. Br J Obstet Gynaecol. 1990;97(2):181–5.
21. Walter AJ, Hentz JG, Magtibay PM, Cornella JL, Magrina JF. Endometriosis: correlation between histologic and visual findings at laparoscopy. Am J Obstet Gynecol. 2001;184(7):1407–11; discussion 1411–3.
22. Martin DC, Hubert GD, Vander Zwaag R, el-Zeky FA. Laparoscopic appearances of peritoneal endometriosis. Fertil Steril. 1989;51(1): 63–7.
23. Jansen RP, Russell P. Nonpigmented endometriosis: clinical, laparoscopic, and pathologic definition. Am J Obstet Gynecol. 1986; 155(6):1154–9.
24. Ling FW. Randomized controlled trial of depot leuprolide in patients with chronic pelvic pain and clinically suspected endometriosis. Pelvic Pain Study Group. Obstet Gynecol. 1999;93(1): 51–8.
25. Hornstein MD, Yuzpe AA, Burry K, et al. Retreatment with nafarelin for recurrent endometriosis symptoms: efficacy, safety, and bone mineral density. Fertil Steril. 1997;67(6):1013–8.
26. Hornstein MD, Surrey ES, Weisberg GW, Casino LA. Leuprolide acetate depot and hormonal add-back in endometriosis: a 12-month study. Lupron Add-Back Study Group. Obstet Gynecol. 1998; 91(1):16–24.
27. Farquhar C, Sutton C. The evidence for the management of endometriosis. Curr Opin Obstet Gynecol. 1998;10(4):321–32.
28. Telimaa S, Puolakka J, Ronnberg L, Kauppila A. Placebo-controlled comparison of danazol and high-dose medroxyprogesterone acetate in the treatment of endometriosis. Gynecol Endocrinol. 1987;1(1):13–23.
29. Schlaff WD, Carson SA, Luciano A, Ross D, Bergqvist A. Subcutaneous injection of depot medroxyprogesterone acetate compared with leuprolide acetate in the treatment of endometriosis-associated pain. Fertil Steril. 2006;85(2):314–25.
30. Vercellini P, Trespidi L, Colombo A, Vendola N, Marchini M, Crosignani PG. A gonadotropin-releasing hormone agonist versus a low-dose oral contraceptive for pelvic pain associated with endometriosis. Fertil Steril. 1993;60(1):75–9.
31. Vercellini P, Frontino G, De Giorgi O, Pietropaolo G, Pasin R, Crosignani PG. Continuous use of an oral contraceptive for endometriosis-associated recurrent dysmenorrhea that does not respond to a cyclic pill regimen. Fertil Steril. 2003;80(3):560–3.
32. Petta CA, Ferriani RA, Abrao MS, et al. Randomized clinical trial of a levonorgestrel-releasing intrauterine system and a depot GnRH analogue for the treatment of chronic pelvic pain in women with endometriosis. Hum Reprod. 2005;20(7):1993–8.
33. Mousa NA, Bedaiwy MA, Casper RF. Aromatase inhibitors in the treatment of severe endometriosis. Obstet Gynecol. 2007;109(6): 1421–3.
34. Nawathe A, Patwardhan S, Yates D, Harrison GR, Khan KS. Systematic review of the effects of aromatase inhibitors on pain associated with endometriosis. BJOG. 2008;115(7):818–22.
35. Chwalisz K, Perez MC, Demanno D, Winkel C, Schubert G, Elger W. Selective progesterone receptor modulator development and use in the treatment of leiomyomata and endometriosis. Endocr Rev. 2005;26(3):423–38.
36. Vercellini P, Pietropaolo G, De Giorgi O, Pasin R, Chiodini A, Crosignani PG. Treatment of symptomatic rectovaginal endometriosis with an estrogen-progestogen combination versus low-dose norethindrone acetate. Fertil Steril. 2005;84(5):1375–87.
37. Sutton CJ, Ewen SP, Whitelaw N, Haines P. Prospective, randomized, double-blind, controlled trial of laser laparoscopy in the treatment of pelvic pain associated with minimal, mild, and moderate endometriosis. Fertil Steril. 1994;62(4):696–700.
38. Abbott J, Hawe J, Hunter D, Holmes M, Finn P, Garry R. Laparoscopic excision of endometriosis: a randomized, placebo-controlled trial. Fertil Steril. 2004;82(4):878–84.
39. Parazzini F, Fedele L, Busacca M, et al. Postsurgical medical treatment of advanced endometriosis: results of a randomized clinical trial. Am J Obstet Gynecol. 1994;171(5):1205–7.
40. Hornstein MD, Hemmings R, Yuzpe AA, Heinrichs WL. Use of nafarelin versus placebo after reductive laparoscopic surgery for endometriosis. Fertil Steril. 1997;68(5):860–4.
41. Candiani GB, Fedele L, Vercellini P, Bianchi S, Di Nola G. Presacral neurectomy for the treatment of pelvic pain associated with endometriosis: a controlled study. Am J Obstet Gynecol. 1992;167(1): 100–3.
42. Zullo F, Palomba S, Zupi E, et al. Effectiveness of presacral neurectomy in women with severe dysmenorrhea caused by endometriosis who were treated with laparoscopic conservative surgery: a 1-year prospective randomized double-blind controlled trial. Am J Obstet Gynecol. 2003;189(1):5–10.
43. Zullo F, Palomba S, Zupi E, et al. Long-term effectiveness of presacral neurectomy for the treatment of severe dysmenorrhea due to endometriosis. J Am Assoc Gynecol Laparosc. 2004;11(1):23–8.
44. Vercellini P, Aimi G, Busacca M, Apolone G, Uglietti A, Crosignani PG. Laparoscopic uterosacral ligament resection for dysmenorrhea associated with endometriosis: results of a randomized, controlled trial. Fertil Steril. 2003;80(2):310–9.
45. Namnoum AB, Hickman TN, Goodman SB, Gehlbach DL, Rock JA. Incidence of symptom recurrence after hysterectomy for endometriosis. Fertil Steril. 1995;64(5):898–902.
46. Metzger DA, Lessey BA, Soper JT, McCarty Jr KS, Haney AF. Hormone-resistant endometriosis following total abdominal hysterectomy and bilateral salpingo-oophorectomy: correlation with histology and steroid receptor content. Obstet Gynecol. 1991;78(5 Pt 2):946–50.
47. Redwine DB. Endometriosis persisting after castration: clinical characteristics and results of surgical management. Obstet Gynecol. 1994;83(3):405–13.
48. Dmowski WP, Radwanska E, Rana N. Recurrent endometriosis following hysterectomy and oophorectomy: the role of residual ovarian fragments. Int J Gynaecol Obstet. 1988;26(1):93–103.
49. Perry CP. Current concepts of pelvic congestion and chronic pelvic pain. JSLS. 2001;5(2):105–10.
50. Creton D, Hennequin L, Kohler F, Allaert FA. Embolisation of symptomatic pelvic veins in women presenting with non-saphenous varicose veins of pelvic origin—three-year follow-up. Eur J Vasc Endovasc Surg. 2007;34(1):112–7.
51. Haggerty CL, Peipert JF, Weitzen S, et al. Predictors of chronic pelvic pain in an urban population of women with symptoms and signs of pelvic inflammatory disease. Sex Transm Dis. 2005;32(5):293–9.
52. Trent M, Bass D, Ness RB, Haggerty C. Recurrent PID, subsequent STI, and reproductive health outcomes: findings from the PID evaluation and clinical health (PEACH) study. Sex Transm Dis. 2011;38(9):879–81.
53. Swank DJ, Swank-Bordewijk SC, Hop WC, et al. Laparoscopic adhesiolysis in patients with chronic abdominal pain: a blinded randomised controlled multi-centre trial. Lancet. 2003;361(9365): 1247–51.
54. Peters AA, Trimbos-Kemper GC, Admiraal C, Trimbos JB, Hermans J. A randomized clinical trial on the benefit of adhesiolysis in patients with intraperitoneal adhesions and chronic pelvic pain. Br J Obstet Gynaecol. 1992;99(1):59–62.
55. Matalliotakis IM, Katsikis IK, Panidis DK. Adenomyosis: what is the impact on fertility? Curr Opin Obstet Gynecol. 2005;17(3): 261–4.
56. Dietrich JE. An update on adenomyosis in the adolescent. Curr Opin Obstet Gynecol. 2010;22(5):388–92.
57. Popovic M, Puchner S, Berzaczy D, Lammer J, Bucek RA. Uterine artery embolization for the treatment of adenomyosis: a review. J Vasc Interv Radiol. 2011;22(7):901–9; quiz 909.

58. Cramer SF, Patel A. The frequency of uterine leiomyomas. Am J Clin Pathol. 1990;94(4):435–8.
59. Chavez NF, Stewart EA. Medical treatment of uterine fibroids. Clin Obstet Gynecol. 2001;44(2):372–84.
60. Salman T, Davis C. Uterine fibroids, management and effect on fertility. Curr Opin Obstet Gynecol. 2010;22(4):295–303.
61. American College of Obstetricians and Gynecologists. ACOG practice bulletin. Alternatives to hysterectomy in the management of leiomyomas. Obstet Gynecol. 2008;112(2 Pt 1):387–400.
62. Goodwin SC, Spies JB. Uterine fibroid embolization. N Engl J Med. 2009;361(7):690–7.
63. Foster DC. Vulvar disease. Obstet Gynecol. 2002;100(1):145–63.
64. Edwards L. New concepts in vulvodynia. Am J Obstet Gynecol. 2003;189(3 Suppl):S24–30.
65. Foster DC, Kotok MB, Huang LS, et al. Oral desipramine and topical lidocaine for vulvodynia: a randomized controlled trial. Obstet Gynecol. 2010;116(3):583–93.
66. Bergeron S, Binik YM, Khalife S, et al. A randomized comparison of group cognitive—behavioral therapy, surface electromyographic biofeedback, and vestibulectomy in the treatment of dyspareunia resulting from vulvar vestibulitis. Pain. 2001;91(3):297–306.
67. Bergeron S, Khalife S, Glazer HI, Binik YM. Surgical and behavioral treatments for vestibulodynia: two-and-one-half year follow-up and predictors of outcome. Obstet Gynecol. 2008;111(1):159–66.
68. Lavelle ED, Lavelle W, Smith HS. Myofascial trigger points. Anesthesiol Clin. 2007;25(4):841–51. vii–iii.
69. Lavelle ED, Lavelle W, Smith HS. Myofascial trigger points. Med Clin North Am. 2007;91(2):229–39.
70. Hoffman D. Understanding multisymptom presentations in chronic pelvic pain: the inter-relationships between the viscera and myofascial pelvic floor dysfunction. Curr Pain Headache Rep. 2011;15(5):343–6.
71. Labat JJ, Riant T, Robert R, Amarenco G, Lefaucheur JP, Rigaud J. Diagnostic criteria for pudendal neuralgia by pudendal nerve entrapment (Nantes criteria). Neurourol Urodyn. 2008;27(4):306–10.
72. Hibner M, Desai N, Robertson LJ, Nour M. Pudendal neuralgia. J Minim Invasive Gynecol. 2010;17(2):148–53.
73. Robert R, Labat JJ, Bensignor M, et al. Decompression and transposition of the pudendal nerve in pudendal neuralgia: a randomized controlled trial and long-term evaluation. Eur Urol. 2005;47(3):403–8.
74. Steege JF. Ovarian remnant syndrome. Obstet Gynecol. 1987;70(1):64–7.
75. Nezhat C, Kearney S, Malik S, Nezhat F. Laparoscopic management of ovarian remnant. Fertil Steril. 2005;83(4):973–8.
76. Siddall-Allum J, Rae T, Rogers V, Witherow R, Flanagan A, Beard RW. Chronic pelvic pain caused by residual ovaries and ovarian remnants. Br J Obstet Gynaecol. 1994;101(11):979–85.
77. El-Minawi AM, Howard FM. Operative laparoscopic treatment of ovarian retention syndrome. J Am Assoc Gynecol Laparosc. 1999;6(3):297–302.