



ACOG **PRACTICE BULLETIN**

CLINICAL MANAGEMENT GUIDELINES FOR
OBSTETRICIAN–GYNECOLOGISTS

NUMBER 51, MARCH 2004

Chronic Pelvic Pain

Chronic pelvic pain is a common disorder of women that often presents a diagnostic dilemma. It is frequently difficult to cure or manage adequately. Many gynecologic and nongynecologic disorders appear to cause or be associated with chronic pelvic pain. Treatment usually is directed to specific diseases that cause chronic pelvic pain, but sometimes there is no clear etiology for pain, and treatment must be directed to alleviating the symptoms. The purpose of this document is to provide information on the differential diagnosis of chronic pelvic pain and review the available evidence on treatment options for women with chronic pelvic pain.

This Practice Bulletin was developed by the ACOG Committee on Practice Bulletins—Gynecology with the assistance of Fred Howard, MD. The information is designed to aid practitioners in making decisions about appropriate obstetric and gynecologic care. These guidelines should not be construed as dictating an exclusive course of treatment or procedure. Variations in practice may be warranted based on the needs of the individual patient, resources, and limitations unique to the institution or type of practice.

Reaffirmed 2010



Background

Definition and Prevalence

Pain is defined as an unpleasant sensory and emotional experience associated with actual or potential tissue damage or is described in terms of such damage (1). Pain is always subjective. Many patients report pain in the absence of tissue damage or any likely pathophysiologic cause; in such cases, pain may have a psychologic basis. If patients regard their experience as pain and report it in the same ways as pain caused by tissue damage, it should be accepted as pain. The definition of pain avoids tying pain to the stimulus.

There is no generally accepted definition of chronic pelvic pain. In gynecologic publications, most (but not all) authors have used duration of 6 or more months as the major criterion of the definition of chronicity. Specifying only duration allows for significant ambiguity, which has led to marked inconsistency of the patient populations included in published studies of chronic pelvic pain. An acceptable definition of chronic pelvic pain at least needs to specify temporal characteristics and location and possibly severity. Possible temporal characteristics include cyclic (eg, dysmenorrhea), intermittent (eg, dyspareunia), or noncyclic pain. Many have preferred to use only noncyclic pain in the definition of chronic pelvic pain because they think the potential etiologic dis-

orders causing noncyclic pain differ from those associated with dysmenorrhea or dyspareunia. “Pelvic” often is assumed to be an adequate description of location, but visceral pelvic pain often is vaguely sensed at the periumbilical area, whereas somatic pelvic pain usually is well localized, for example, in the sacroiliac joint point at the posterior buttocks area. Additionally, chronic vulvar pain may or may not be included as chronic pelvic pain, depending on the definition of location. In reviewing any research on the causes or treatments of chronic pelvic pain, it is crucial to know which definition was used.

One proposed definition of chronic pelvic pain is noncyclic pain of 6 or more months’ duration that localizes to the anatomic pelvis, anterior abdominal wall at or below the umbilicus, the lumbosacral back, or the buttocks and is of sufficient severity to cause functional disability or lead to medical care. A lack of physical findings does not negate the significance of a patient’s pain, and normal examination results do not preclude the possibility of finding pelvic pathology.

Although the prevalence of chronic pelvic pain in the general population is not accurately established, available data suggest it is far more common than generally recognized. Approximately 15–20% of women aged 18–50 years have chronic pelvic pain of greater than 1 year’s duration (2, 3).

Etiology of Chronic Pelvic Pain

Potential visceral sources of chronic pelvic pain include the reproductive, genitourinary, and gastrointestinal tracts; potential somatic sources include the pelvic bones, ligaments, muscles, and fascia. Chronic pelvic pain may result from psychologic disorders or neurologic diseases, both central and peripheral. It also may be useful to classify etiologies of chronic pelvic pain into gynecologic and nongynecologic causes, but clearly an obstetrician–gynecologist may diagnose and treat many nongynecologic disorders.

Few, if any, of the diseases thought to cause chronic pelvic pain meet traditional epidemiologic criteria of causality. Sufficient evidence strongly suggests that several of the most common disorders in women with chronic pelvic pain are causal, such as endometriosis, interstitial cystitis, and irritable bowel syndrome (see box and Table 1). For many of the diseases often listed as causes of chronic pelvic pain, only limited evidence or expert opinion supports an etiologic relationship. Although the etiologic relationships of many of the proposed disorders are not well established, in clinical practice, most are treated if diagnosed in women with chronic pelvic pain. This ambiguity makes it difficult to interpret cause and effect with regard to treatment in most studies of women with chronic pelvic pain.

Gynecologic Conditions That May Cause or Exacerbate Chronic Pelvic Pain, by Level of Evidence

Level A*

- Endometriosis[†]
- Gynecologic malignancies (especially late stage)
- Ovarian retention syndrome (residual ovary syndrome)
- Ovarian remnant syndrome
- Pelvic congestion syndrome
- Pelvic inflammatory disease[†]
- Tuberculous salpingitis

Level B[‡]

- Adhesions[†]
- Benign cystic mesothelioma
- Leiomyomata[†]
- Postoperative peritoneal cysts

Level C[§]

- Adenomyosis
- Atypical dysmenorrhea or ovulatory pain
- Adnexal cysts (nonendometriotic)
- Cervical stenosis
- Chronic ectopic pregnancy
- Chronic endometritis
- Endometrial or cervical polyps
- Endosalpingiosis
- Intrauterine contraceptive device
- Ovarian ovulatory pain
- Residual accessory ovary
- Symptomatic pelvic relaxation (genital prolapse)

*Level A: good and consistent scientific evidence of causal relationship to chronic pelvic pain

[†]Diagnosis frequently reported in published series of women with chronic pelvic pain

[‡]Level B: limited or inconsistent scientific evidence of causal relationship to chronic pelvic pain

[§]Level C: causal relationship to chronic pelvic pain based on expert opinions

Data from Howard FM. Chronic pelvic pain. *Obstet Gynecol* 2003; 101:594–611.

The proportion of women with chronic pelvic pain and a specific diagnosis (or diagnoses) is unclear and varies greatly in reported series. A large, primary care

Table 1. Nongynecologic Conditions That May Cause or Exacerbate Chronic Pelvic Pain, by Level of Evidence

Level of Evidence	Urologic	Gastrointestinal	Musculoskeletal	Other
Level A*	Bladder malignancy	Carcinoma of the colon	Abdominal wall myofascial pain (trigger points)	Abdominal cutaneous nerve entrapment in surgical scar
	Interstitial cystitis†	Constipation	Chronic coccygeal or back pain†	Depression†
	Radiation cystitis	Inflammatory bowel disease	Faulty or poor posture	Somatization disorder
	Urethral syndrome	Irritable bowel syndrome†	Fibromyalgia Neuralgia of iliohypogastric, ilioinguinal, and/or genitofemoral nerves Pelvic floor myalgia (levator ani or piriformis syndrome) Peripartum pelvic pain syndrome	
Level B‡	Uninhibited bladder contractions (detrusor dyssynergia)	—	Herniated nucleus pulposus	Celiac disease
	Urethral diverticulum		Low back pain† Neoplasia of spinal cord or sacral nerve	Neurologic dysfunction Porphyria Shingles Sleep disturbances
Level C§	Chronic urinary tract infection	Colitis	Compression of lumbar vertebrae	Abdominal epilepsy
	Recurrent, acute cystitis	Chronic intermittent bowel obstruction	Degenerative joint disease	Abdominal migraine
	Recurrent, acute urethritis	Diverticular disease	Hernias: ventral, inguinal, femoral, spigelian	Bipolar personality disorders
	Stone/urolithiasis		Muscular strains and sprains Rectus tendon strain	Familial Mediterranean fever
	Urethral caruncle		Spondylosis	

*Level A: good and consistent scientific evidence of causal relationship to chronic pelvic pain

†Diagnosis frequently reported in published series of women with chronic pelvic pain

‡Level B: limited or inconsistent scientific evidence of causal relationship to chronic pelvic pain

§Level C: causal relationship to chronic pelvic pain based on expert opinions

Data from Howard FM. Chronic pelvic pain. *Obstet Gynecol* 2003;101:594-611.

database from the United Kingdom found diagnoses related to the urinary and gastrointestinal tracts were more common than gynecologic diagnoses (30.8% urinary, 37.7% gastrointestinal, and 20.2% gynecologic) (4). Furthermore, many women with chronic pelvic pain have more than 1 disease that might lead to pain; 25–50% of women who receive medical care in primary care practices have more than 1 diagnosis (4–6). The most common diagnoses are endometriosis, adhesions, irritable bowel syndrome, and interstitial cystitis (2, 7–9).

Women with diagnoses that involve more than 1 organ system have greater pain than women with only 1

system involved. For example, 43% of patients with chronic pelvic pain without gastrointestinal or urologic symptoms had moderate or severe pain (mean visual analog scale score: 3.8), whereas 71% of women with chronic pelvic pain and both gastrointestinal and urologic symptoms had moderate to severe pain (mean visual analog scale score: 5.4) (6). Consistency of pain also is greater in women with multisystem symptoms. Women with chronic pelvic pain are more likely than those in the general population to have dysmenorrhea (81% versus 58%) and dyspareunia (41% versus 14%). The severity of pain with intercourse and with menses is greater in

women with chronic pelvic pain who have gastrointestinal and urologic symptoms than in those with no gastrointestinal or urologic symptoms.

Populations at Increased Risk of Chronic Pelvic Pain

Demographic profiles of large surveys suggest that women with chronic pelvic pain are no different from women without chronic pelvic pain in terms of age, race and ethnicity, education, socioeconomic status, or employment status (2, 6). Women with chronic pelvic pain may be slightly more likely to be separated or divorced (2). Women with chronic pelvic pain tend to be of reproductive age; however, age does not appear to be a specific risk factor (6). Women develop chronic pelvic pain at all ages, although the prevalence of different diagnoses seems to vary at different ages.

Physical and Sexual Abuse

Most published evidence suggests a significant association of physical and sexual abuse with various chronic pain disorders (10, 11). Studies have found that 40–50% of women with chronic pelvic pain have a history of abuse (12–16). Whether abuse (physical or sexual) specifically causes chronic pelvic pain is not clear, nor is a mechanism established by which abuse might lead to the development of chronic pelvic pain (17, 18). Women with a history of sexual abuse and high somatization scores have been found to be more likely to have nonsomatic pelvic pain, suggesting the link between abuse and chronic pelvic pain may be psychologic or neurologic (19, 20).

Evidence suggests that abuse may result in biophysical changes. For example, one study found that, after controlling for history of psychiatric disturbance, adult survivors had lower thresholds for pain (21). It also has been suggested that chronic or traumatic stimulation (especially in the pelvic or abdominal region) heightens sensitivity, resulting in persistent pain, such as abdominal and pelvic pain, or other bowel symptoms (22, 23). In women with chronic pelvic pain, as in all women, if a history of abuse is obtained, it is important to ensure that the women are not currently being abused and in danger.

Pelvic Inflammatory Disease

Approximately 18–35% of all women with acute pelvic inflammatory disease (PID) develop chronic pelvic pain (24, 25). The actual mechanisms by which chronic pelvic pain results from PID are not known, and not all women with reproductive organ damage secondary to acute PID develop chronic pelvic pain. Whether acute PID is treat-

ed with outpatient or inpatient regimens does not appear to significantly alter the odds of developing subsequent chronic pelvic pain (34% with outpatient therapy versus 30% with inpatient therapy) (24).

Endometriosis

Although endometriosis may be a direct cause of chronic pelvic pain, it also may indirectly place women at increased risk for chronic pelvic pain. For example, evidence suggests that women with endometriosis have increased episodes and pain severity of urinary calculoses than women without endometriosis (26). Similar results have been demonstrated for vaginal pain. Such viscerovisceral interactions may have a significant role in chronic pelvic pain in women and may explain why some women with a history of endometriosis have persistent pelvic pain after their endometriosis is gone.

Endometriosis is diagnosed laparoscopically in approximately 33% of women with chronic pelvic pain (other frequent laparoscopic diagnoses are adhesive disease in 24% of patients and no visible pathology in 35% of patients) (27). Although abnormal examination findings correlate in 70–90% of cases with abnormal laparoscopic findings (28, 29), more than one half of those with abnormal laparoscopic findings have normal findings on preoperative pelvic examinations (30).

Interstitial Cystitis

Women with interstitial cystitis are at significant risk of having chronic pelvic pain. Interstitial cystitis is a chronic inflammatory condition of the bladder. It is clinically characterized by irritative voiding symptoms of urgency and frequency in the absence of objective evidence of another disease that could cause the symptoms (31, 32). Pelvic pain is reported by up to 70% of women with interstitial cystitis, and occasionally it is the presenting symptom or chief complaint (32). It has been suggested that as many as 38–85% of women presenting to gynecologists with chronic pelvic pain may have interstitial cystitis (8, 33).

Irritable Bowel Syndrome

Irritable bowel syndrome is a common functional bowel disorder of uncertain etiology. It is characterized by a chronic, relapsing pattern of abdominopelvic pain and bowel dysfunction with constipation or diarrhea. Irritable bowel syndrome appears to be one of the most common disorders associated with chronic pelvic pain. It seems to occur much more commonly in women with chronic pelvic pain than in the general population; symptoms consistent with irritable bowel syndrome are found in 50–80% of women with chronic pelvic pain (9, 34). The

current diagnostic criteria for irritable bowel syndrome are the Rome II criteria (see box).

Obstetric History

Pregnancy and childbirth can cause trauma to the musculoskeletal system, especially the pelvis and back, and may lead to chronic pelvic pain. Although few well-designed trials have assessed the relationship, historical risk factors associated with pregnancy and pain include lumbar lordosis, delivery of a large infant, muscle weakness and poor physical conditioning, a difficult delivery, vacuum or forceps delivery, and use of gynecologic stirrups for delivery (35). Conversely, women who have never been pregnant may have disorders that can cause both infertility and chronic pelvic pain, such as endometriosis, chronic PID, or pelvic adhesive disease.

Past Surgery

A history of abdominopelvic surgery is associated with chronic pelvic pain. In some cases, the relationship is relatively clear, such as unrecognized spillage of gallstones

at the time of cholecystectomy (36, 37) or osteitis pubis or osteomyelitis after the Marshall–Marchetti–Kranz procedure (38). Prior cervical surgery for dysplasia may cause cervical stenosis, which has been associated with endometriosis (39). Additionally, among women without preoperative pelvic pain, 3–9% develop pelvic pain or back pain in the 2 years after hysterectomy (40). A recent case–control study suggests that cesarean delivery also may be a risk factor for chronic pelvic pain (odds ratio of 3.7) (41).

Musculoskeletal Disorders

Musculoskeletal disorders as causes of or risk factors for chronic pelvic pain have not been widely discussed in gynecologic publications. They may be more important, however, than generally recognized.

Pain that started with a pregnancy or immediately postpartum may suggest peripartum pelvic pain syndrome. This syndrome is thought to be caused by strain of the ligaments in the pelvis and lower spine from a combination of factors, including specific hormonal changes, damage to pelvic ligaments, muscle weakness, and the weight of the fetus and gravid uterus (35).

Faulty posture, in particular an exaggerated lumbar lordosis and thoracic kyphosis (called “typical pelvic pain posture”), may account for up to 75% of cases of chronic pelvic pain (42). Faulty posture is a contributing cause of weak, deconditioned muscles, which allow for imbalances in the pelvis with formation of trigger points and hypertonicity and, as a result, pelvic pain.

Other musculoskeletal disorders may cause or contribute to pelvic pain. These include trigger points, fibromyalgia, lumbar vertebral disorders, and pelvic floor myalgia.

Diagnostic Studies

A detailed history and physical examination are the basis for differential diagnosis. In a woman with chronic pelvic pain, the history and physical examination should take into account the risk factors noted previously, as well as the various conditions associated with chronic pelvic pain (see box “Gynecologic Conditions That May Cause or Exacerbate Chronic Pelvic Pain, by Level of Evidence” and Table 1). The history and physical examination also should seek to identify the location, severity, quality, and timing of the woman’s pain. Because of the many nongynecologic conditions associated with chronic pelvic pain, interdisciplinary evaluation and management may be needed.

Up to two thirds of women with chronic pelvic pain do not undergo diagnostic testing, never receive a diagnosis, and are never referred to a specialist for evaluation

Rome II Criteria for Irritable Bowel Syndrome

At least 12 weeks (need not be consecutive) in the preceding 12 months of abdominal discomfort or pain that has 2 of 3 features:

1. Relieved with defecation
2. Onset associated with a change in frequency of stool
3. Onset associated with a change in stool form or appearance

The following symptoms are not essential for the diagnosis, but their presence increases diagnostic confidence and may be used to identify subgroups of irritable bowel syndrome:

- Abnormal stool frequency (more than 3 per day or fewer than 3 per week)
- Abnormal stool form (lumpy, hard or loose, watery) in more than 25% of defecations
- Abnormal stool passage (straining, urgency, or feeling of incomplete evacuation) in more than 25% of defecations
- Passage of mucus in more than 25% of defecations
- Bloating or feeling of abdominal distention in more than 25% of days

Modified from Thompson WG, Longstreth GF, Drossman DA, Heaton KW, Irvine EJ, Muller-Lissner SA. Functional bowel disorders and functional abdominal pain. *Gut* 1999;45(Suppl 2):II43–7.

or treatment (2, 5). Diagnostic studies should be based on the history and physical examination.

Diagnostic Imaging

Transvaginal ultrasonography is particularly useful for evaluation of the pelvis. In patients with a pelvic mass, ultrasonography may help identify the origin of the mass as uterine, adnexal, gastrointestinal, or from the bladder. Magnetic resonance imaging or computed tomography may be useful in rare cases when ultrasound findings are abnormal.

Laparoscopy

Chronic pelvic pain is the indication for at least 40% of all gynecologic laparoscopies (27). Endometriosis and adhesions account for more than 90% of the diagnoses in women with discernible laparoscopic abnormalities, and laparoscopy is indicated in women thought to have either of these conditions. When endometriosis is suspected on the basis of visual findings during laparoscopy, biopsies and histologic confirmation of suspicious areas are important (43) because the visual diagnosis is incorrect in 10–90% of cases (44). Often, adolescents are excluded from laparoscopic evaluation on the basis of their age, but several series show that endometriosis is as common in adolescents with chronic pelvic pain as in the rest of the population (45, 46).

Conscious laparoscopic pain mapping, a diagnostic laparoscopy performed under local anesthesia, can be performed with the goal of identifying sources of pain in women with chronic pelvic pain. It has been suggested that conscious laparoscopic pain mapping can lead to the treatment of subtle or atypical areas of disease that might have been overlooked if the procedure had been done under general anesthesia or may even help patients avoid surgical treatment when no painful lesions are identified (47). However, no substantial data confirm improved diagnostic accuracy or improved clinical outcomes with conscious laparoscopic pain mapping (48–50).

Evaluation of Symptoms of Urinary Tract Infection

An intravesical potassium sensitivity test evaluates pain and urgency after intravesical instillation of 40 mL of potassium chloride (0.4 mEq/mL) compared with symptoms after instillation of 40 mL of water (51). Among patients with interstitial cystitis, 70–90% have positive results on intravesical potassium sensitivity testing. Up to 85% of women evaluated by obstetrician–gynecologists for chronic pelvic pain may have positive intravesical potassium sensitivity test results (8). Whether these findings represent a high prevalence of interstitial cystitis in

women with chronic pelvic pain or viscerovisceral convergence in women with reproductive tract disease has not been determined. Furthermore, the diagnostic validity of the intravesical potassium sensitivity test for interstitial cystitis is still controversial (52, 53).

The interstitial cystitis symptom index is a validated questionnaire that reliably predicts the diagnosis of interstitial cystitis and may be used to help determine whether cystoscopy is indicated (54). For example, 72% of women with a score of 5 or more on the interstitial cystitis symptom index and significant dyspareunia pain levels have interstitial cystitis identified cystoscopically (33). Cystoscopic criteria for interstitial cystitis are the presence of glomerulations (petechiae) or Hunner ulcer with bladder distention to 80–100 cm water pressure under anesthesia and decreased bladder capacity (less than 350 mL) without anesthesia (55). The reliability of bladder glomerulations as a diagnostic criterion for interstitial cystitis has been questioned because similar findings are possible in women without voiding symptoms (56).

Clinical Considerations and Recommendations

- ▶ *Is there evidence to support the following medical approaches to treatment of chronic pelvic pain?*

Antidepressants

Tricyclic antidepressants and selective serotonin reuptake inhibitors (SSRIs) have been approved for treating depression, but new research has shown they can be effective in treating other conditions in patients who do not have depression. Tricyclic antidepressants, such as imipramine, amitriptyline, desipramine, and doxepin, have been shown in placebo-controlled studies to improve pain levels and pain tolerance in some, but not all, chronic pain syndromes (57). It is not clear how effective other antidepressants, such as SSRIs, are in the treatment of chronic pain syndromes (58–61).

Few studies have evaluated the use of antidepressants for chronic pelvic pain (62). One uncontrolled evaluation of the tricyclic antidepressant nortriptyline showed a decrease of pain intensity and duration, but one half of the patients discontinued nortriptyline before completing the study because of drug side effects at doses of 100 mg or less (63). A placebo-controlled, crossover study of the SSRI sertraline, 50 mg twice daily for 6 weeks, showed no improvement in pelvic pain (64).

At this time, evidence is insufficient to substantiate efficacy of antidepressants for the treatment of chronic pelvic pain, although the efficacy of tricyclic antidepressants for other pain syndromes suggests they also might be efficacious for chronic pelvic pain. Nonetheless, the substantial association of depression with chronic pelvic pain supports the use of antidepressants for the specific treatment of depression.

Local Anesthetic Injection of Trigger Points

Chronic pain syndromes associated with myofascial trigger points have been clinically recognized for quite some time (65). Observational data on the use of local anesthetic injection of trigger points of the abdominal wall, vagina, and sacrum for relief of chronic pelvic pain have demonstrated a response rate of 68% (66).

Analgesics

Extensive evidence demonstrates that nonsteroidal anti-inflammatory drugs, including COX-2 inhibitors, relieve various types of pain, including dysmenorrhea (67, 68). No clinical trials have addressed chronic pelvic pain specifically, but moderate analgesic efficacy, as shown for other types of pain, would be anticipated.

Opioids are increasingly used in the treatment of chronic pain (69). Randomized clinical trials suggest significant analgesic effects but not necessarily improvement in functional or psychologic status (70–72). Risk of addiction has been low in patients with chronic pain. There are no published studies of opioid treatment for chronic pelvic pain.

- ▶ *Is there evidence to support the use of hormonal therapy for treatment of chronic pelvic pain?*

Combined Oral Contraceptives

Oral contraceptives provide significant relief from primary dysmenorrhea (73). They suppress ovulation, markedly reduce spontaneous uterine activity, stabilize estrogen and progesterone levels, abrogate menstrual increases in prostaglandin levels, and reduce the amount of pain and symptoms associated with menses. These effects also are thought to make oral contraceptives effective in the treatment of other gynecologic pain disorders.

Oral contraceptives often are recommended for endometriosis-associated chronic pelvic pain (74), but there are limited data from clinical trials to support this recommendation. One clinical trial suggested combined

oral contraceptives are comparable to the gonadotropin-releasing hormone (GnRH) agonist goserelin in relieving chronic pelvic pain and dyspareunia—but less effective in relieving dysmenorrhea—in women with endometriosis (75). A trial evaluating postoperative administration of monophasic combined oral contraceptives after surgical resection of endometriomas suggested oral contraceptives do not significantly affect the long-term recurrence of endometriosis (76). No data address the use of cyclic versus noncyclic combined oral contraceptives. Other hormonal contraceptive methods, such as the levonorgestrel-releasing intrauterine device, may be effective for treatment of dysmenorrhea associated with endometriosis, but evidence is limited (77).

Gonadotropin-Releasing Hormone Agonists

Gonadotropin-releasing hormone agonists are analogues of naturally occurring gonadotropin-releasing hormones that “down-regulate” hypothalamic–pituitary gland production and the release of luteinizing hormone and follicle-stimulating hormone leading to dramatic reductions in estradiol levels. The GnRH agonists available in the United States are nafarelin, goserelin, and leuprolide. Numerous clinical trials show GnRH agonists are more effective than placebo and as effective as danazol in relieving endometriosis-associated pelvic pain (78–86). However, one clinical trial designed to evaluate empiric treatment of chronic pelvic pain with suspected endometriosis suggested GnRH agonists have the same efficacy in women with symptoms consistent with endometriosis, whether or not they actually have endometriosis (87). Although this finding is based on a relatively small number of cases, it strongly suggests the response to GnRH agonists does not depend on surgical confirmation of endometriosis in women with symptoms suggestive of endometriosis-associated chronic pelvic pain. One possible explanation for this finding may be that although obstetrician–gynecologists generally assume GnRH agonist treatment is specific for endometriosis-associated pelvic pain, in fact, symptoms of pelvic congestion syndrome (88), irritable bowel syndrome (89–92), and interstitial cystitis (93) also vary with the menstrual cycle and respond to GnRH agonist treatment.

Good evidence from studies of prolonged treatment of endometriosis-associated pelvic pain indicates that loss of bone density, one of the major adverse effects of GnRH agonists, can be abrogated by add-back treatment with estrogen and progestogen or progestogen only, without significant loss of efficacy (94–96). Postoperative treatment with GnRH agonists also appears to be

efficacious in women with endometriosis (97, 98). In addition, observational data suggest GnRH agonists may be used to treat pelvic pain associated with ovarian retention syndrome (residual ovary syndrome) and ovarian remnant syndrome (98, 99).

Progestins

Clinical trials suggest progestins are effective in the treatment of chronic pelvic pain associated with endometriosis and pelvic congestion syndrome. Medroxyprogesterone acetate, 30–100 mg per day, effectively decreases pain from endometriosis and pelvic congestion syndrome in most studies (100–103). Other progestational agents not available in the United States, such as gestrinone and lynestrenol, also are effective in the treatment of endometriosis-associated pelvic pain (104, 105). Norethindrone is sometimes recommended for treatment of endometriosis-associated pelvic pain but has only been studied in uncontrolled trials (106).

▶ ***What is the evidence for efficacy of proposed nonmedical treatments?***

Many modalities of treatment other than medications and surgery have been recommended for chronic pelvic pain, including exercise, physical therapy, and dietary modifications. Very few of these treatments have been studied in clinical trials.

Exercise

Although most studies suggest dysmenorrhea is decreased by exercise, there are no definitive data to support this suggestion (107, 108). Additionally, no data address the efficacy of exercise for relief of chronic pelvic pain.

Physical Therapy

Observational studies suggest various physical therapy modalities are effective for pain relief. Electrotherapy, fast- and slow-twitch exercises of the striated muscles of the pelvic floor, and manual therapy of myofascial trigger points in the pelvic floor have shown improvement of pain in 65–70% of patients (109, 110). For peripartum pelvic pain syndrome, physical therapy showed no efficacy over that of a pelvic belt with no exercise (111).

▶ ***Are surgical approaches effective for treatment of chronic pelvic pain?***

Various surgical treatments aimed primarily at treating endometriosis, including excision or destruction of endometriotic tissue and hysterectomy, have been pro-

posed to relieve chronic pelvic pain. Other surgical approaches also have been considered.

Excision or Destruction of Endometriotic Tissue

It is suggested that conservative surgical treatment of endometriosis results in significant pain relief for 1 year in 45–85% of women, but only one clinical trial compared conservative surgical treatment with placebo therapy (112, 113). The design of this trial limits its generalizability (only laparoscopic laser ablation of lesions was performed, only women with endometriosis in stages I through III were included, and only 6 patients had stage III endometriosis), but it confirms the efficacy of conservative surgical treatment. Laparoscopic laser treatment of endometriosis showed pain relief at 6 months in 62% of patients versus 20% in the group that underwent diagnostic laparoscopy only.

The recurrence rate of endometriosis after conservative surgical treatment has been reported to range from 15% to 100% (114). The average time to recurrence after initial surgery by laparotomy is 40–50 months. However, the time to recurrence may reflect the thoroughness of the original surgery or effectiveness of subsequent medical treatment.

Hysterectomy

It is estimated that chronic pelvic pain is the principal preoperative indication for 10–12% of hysterectomies (115). In the Maine Women's Health Study (a prospective cohort study), 18% of women had hysterectomies for a primary indication of chronic pelvic pain (116, 117). In the same study, 45% of the women who had hysterectomies for leiomyomata had more than 8 days of pain per month, and 66% of those with bleeding as the preoperative indication had similar pain, which suggests pain is a secondary indication for hysterectomy in many women. In this study, women with chronic pelvic pain treated with hysterectomy had significantly improved outcomes at 1 year compared with women treated medically. Outcomes were better in mean days of pain per month and severity of pain, as well as in indicators of quality of life. Only one half of these women had specific diagnoses, such as endometriosis, leiomyomata, or adhesions.

The Maryland Women's Health Study, also a prospective cohort study but without a control group, showed that almost 90% of women had relief of pain at 1 and 2 years after hysterectomy (40). Data from the United States Collaborative Review of Sterilization, a prospective cohort study, showed that at 1 year after hysterectomy for chronic pelvic pain, 74% of women had complete resolution of pain, and 21% had decreased pain

(118). A retrospective study of hysterectomy for chronic pelvic pain with no extrauterine pathology found that 78% of women were pain-free at follow-up of at least 1 year (119). In 65% of these women, no uterine pathology was detected. Finally, a small study of women with chronic pelvic pain caused by pelvic congestion (demonstrated by venography and ultrasonography) reported marked improvement or relief of pain in 35 of 36 patients after hysterectomy and bilateral salpingo-oophorectomy (120).

Hysterectomy appears to have a role in the treatment of many women with chronic pelvic pain. Although based only on observational studies, it appears that at least 75% of women who have a hysterectomy for chronic pelvic pain thought to be caused by gynecologic disease experience pain relief at 1 year of follow-up.

Adhesiolysis

Adhesions are commonly thought to be a potential cause of chronic pelvic pain, and evidence from conscious laparoscopic pain mapping suggests some women have painful adhesions (50). Observational studies suggest that up to 85% of women improve after adhesiolysis (121), but the only clinical trial of adhesiolysis suggests that only women with dense adhesions involving bowel show any decrease in pain after surgical adhesiolysis (122).

Nerve Stimulation

Sacral nerve stimulation is beneficial in the treatment of chronic voiding dysfunction. Its use in women with voiding dysfunction and chronic pelvic pain has suggested potential efficacy for treatment of chronic pelvic pain. Uncontrolled studies of sacral nerve stimulation in women with chronic pelvic pain and no voiding disorder suggest that 60% of women show significant improvement in their pain levels (123–125).

Presacral Neurectomy

Innervation from the superior hypogastric plexus (presacral nerve) supplies the cervix, uterus, and proximal fallopian tubes with afferent nociception. Surgical resection of this plexus is sometimes useful for central dysmenorrhea unresponsive to other treatments. Approximately three fourths of patients with this symptom have a greater than 50% decrease in pain after presacral neurectomy (126, 127). Presacral neurectomy is significantly more effective for the treatment of primary dysmenorrhea than uterine nerve ablation (128).

Clinical trials show that as a component of conservative surgery for endometriosis-associated pelvic pain, presacral neurectomy provides additional pain relief

mostly of midline pain associated with menses, with little additional effect on dyspareunia and nonmenstrual pain (129, 130). Similar results are obtained in women without endometriosis; that is, only central dysmenorrhea appears to be decreased, with no significant effect on noncentral or nonmenstrual pain (131). Overall, regardless of pathology, pain that is localized in the lateral pelvic area, as opposed to central pelvic pain, has a notably lower response to treatment by presacral neurectomy.

It has been suggested that performing superior hypogastric plexus blocks before deciding to do a presacral neurectomy may improve the outcomes with surgery or avoid the need for surgery altogether (132), but only small case series have been published in support of this concept (133). It also has been suggested that repeated superior hypogastric blocks may reverse central sensitization and sympathetically maintained pelvic pain resulting in prolonged relief of pain (134).

Uterine Nerve Ablation

Uterine nerve ablation involves transecting or resecting the uterosacral ligaments at their insertions into the uterus, which interrupts a significant portion of the cervical sensory nerve fibers. One small clinical trial found uterine nerve ablation significantly decreased the severity of primary dysmenorrhea for at least 3 months ($P < .05$) (135). Uterine nerve ablation is less effective for the treatment of primary dysmenorrhea than presacral neurectomy (128).

Adding uterine nerve ablation to surgical treatment of endometriosis-associated pelvic pain or dysmenorrhea does not improve the outcome of surgical treatment (136, 137). No evidence demonstrates that uterine nerve ablation improves nonmenstrual chronic pelvic pain.

► Is counseling or psychotherapy effective for treatment of chronic pelvic pain?

Psychosomatic factors appear to have a prominent role in chronic pelvic pain (138), which suggests that psychiatric or psychologic evaluation and treatment should be routine in women with chronic pelvic pain. Various modes of psychotherapy, including cognitive therapy, operant conditioning, and behavioral modification, appear to be helpful in women with chronic pelvic pain, but most of the data are observational (139) or include psychotherapy as part of multidisciplinary treatment (140). One randomized clinical trial of psychotherapy for pelvic congestion syndrome suggested that adding psychotherapy to medical treatment improved the response over that obtained with medical treatment only (102).

Up to 50% of women with chronic pelvic pain have a history of physical or sexual abuse (14, 141). Traumatized patients who experienced abuse as children generally benefit from mental health care. For patients who have not sought such care, obstetrician–gynecologists can be powerful allies in patients’ healing by offering support and referral. Efforts should be made to refer patients to mental health professionals with significant experience in abuse-related issues.

When referring a patient to another health care professional, it is especially helpful to indicate to the patient that her past abuse may be contributing to her current health problems and that further evaluation by a therapist would be beneficial. This is likely more effective than telling the patient that her symptoms are all “psychologic” and that she should see a therapist (142). It is important to secure the patient’s express authorization before speaking to the therapist when collaborative practice between the obstetrician–gynecologist and therapist is warranted. If appropriate, to reassure the patient, the physician should emphasize his or her ongoing involvement in the patient’s case.

- ▶ ***Are complementary or alternative medicine therapies effective for treating chronic pelvic pain?***

Herbal and Nutritional Therapies

Treatment of dysmenorrhea has been studied in clinical trials of magnesium, vitamin B₆, vitamin B₁, omega-3 fatty acids, and a Japanese herbal combination (Japanese angelica root, peony root, hoelen, atracylodes lancea root, alisma root, cnidium root). Vitamin B₁ (100 mg daily) and magnesium (doses varied) were significantly more effective than a placebo in numerous studies, but data were insufficient to recommend the other therapies for dysmenorrhea (143). Published clinical trials of herbal or nutritional therapies for nonmenstrual pain are lacking.

Magnetic Field Therapy

The application of magnets to abdominal trigger points appears to improve disability and reduce pain when compared with placebo magnets (144). However, only one clinical trial evaluated the use of magnet therapy, and it had significant methodologic flaws. Whether magnetic field therapy is helpful for other types of chronic pelvic pain is not known, but limited observational data suggest potential usefulness for endometriosis-associated pain, dyspareunia, and dysmenorrhea (145).

Acupuncture

Clinical trials evaluating the efficacy of acupuncture, acupressure, and transcutaneous nerve stimulation therapies have been performed only for primary dysmenorrhea, not for nonmenstrual pelvic pain. All 3 modalities are better than placebo in the treatment of dysmenorrhea (146–149). Only case reports support acupuncture as a modality to treat nonmenstrual chronic pelvic pain (150).

Summary of Recommendations

The following recommendations are based on good and consistent scientific evidence (Level A):

- ▶ Combined oral contraceptives should be considered as a treatment option to decrease pain from primary dysmenorrhea.
- ▶ Gonadotropin-releasing hormone agonists are effective in relieving pelvic pain associated with endometriosis and irritable bowel syndrome, as well as in women with symptoms consistent with endometriosis who do not have endometriosis. Thus, empiric treatment with GnRH agonists without laparoscopy should be considered as an acceptable approach to treatment.
- ▶ Nonsteroidal antiinflammatory drugs, including COX-2 inhibitors, should be considered for moderate pain and are particularly effective for dysmenorrhea.
- ▶ Progestins in daily, high doses should be considered as an effective treatment of chronic pelvic pain associated with endometriosis and pelvic congestion syndrome.
- ▶ Laparoscopic surgical destruction of endometriosis lesions should be considered to decrease pelvic pain associated with stages I–III endometriosis.
- ▶ Presacral neurectomy may be considered for treatment of centrally located dysmenorrhea but has limited efficacy for chronic pelvic pain or pain that is not central in its location. Uterine nerve ablation or transection of the uterosacral ligament also can be considered for centrally located dysmenorrhea, but it appears to be less effective than presacral neurectomy. Combining uterine nerve ablation or presacral neurectomy with surgical treatment of endometriosis does not further improve overall pain relief.

- ▶ Adding psychotherapy to medical treatment of chronic pelvic pain appears to improve response over that of medical treatment alone and should be considered.

The following recommendations are based on limited or inconsistent scientific evidence (Level B):

- ▶ Gonadotropin-releasing hormone agonists should be considered as a treatment option for chronic pelvic pain because they have been shown to relieve endometriosis-associated pelvic pain.
- ▶ Surgical adhesiolysis should be considered to decrease pain in women with dense adhesions involving the bowel, but it is unclear if lysis of other types of adhesions is effective.
- ▶ Hysterectomy is an effective treatment for chronic pelvic pain associated with reproductive tract symptoms that results in pain relief in 75–95% of women and should be considered.
- ▶ Sacral nerve stimulation may decrease pain in up to 60% of women with chronic pelvic pain and should be considered as a treatment option.
- ▶ Various physical therapy modalities appear to be helpful in the treatment of chronic pelvic pain and should be considered as a treatment option.
- ▶ Nutritional supplementation with vitamin B₁ or magnesium may be recommended to decrease pain of dysmenorrhea.
- ▶ Injection of trigger points of the abdominal wall, vagina, and sacrum with local anesthetic may provide temporary or prolonged relief of chronic pelvic pain and should be considered.
- ▶ Treatment of abdominal trigger points by the application of magnets to the trigger points may be recommended to improve disability and reduce pain.
- ▶ Acupuncture, acupressure, and transcutaneous nerve stimulation therapies should be considered to decrease pain of primary dysmenorrhea.

The following recommendations are based primarily on consensus and expert opinion (Level C):

- ▶ A detailed history and physical examination are the basis for differential diagnosis of chronic pelvic pain and should be used to determine appropriate diagnostic studies.
- ▶ Antidepressants may be helpful in the treatment of chronic pelvic pain.

- ▶ Opioid analgesics can be used to provide effective relief of severe pain with a low risk of addiction but do not necessarily improve functional or psychological status and are not well studied in patients with chronic pelvic pain.

References

1. Merskey H, Bogduk N, editors. Classification of chronic pain. IASP Task Force on Taxonomy. 2nd ed. Seattle (WA): IASP Press; 1994. p. 209–14. (Level III)
2. 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;87:321–7. (Level II-2)
3. Jamieson DJ, Steege JF. The prevalence of dysmenorrhea, dyspareunia, pelvic pain, and irritable bowel syndrome in primary care practices. *Obstet Gynecol* 1996; 87:55–8. (Level II-2)
4. Zondervan KT, Yudkin PL, Vessey MP, Dawes MG, Barlow DH, Kennedy SH. Patterns of diagnosis and referral in women consulting for chronic pelvic pain in UK primary care. *Br J Obstet Gynaecol* 1999;106: 1156–61. (Level II-2)
5. Zondervan KT, Yudkin PL, Vessey MP, Dawes MG, Barlow DH, Kennedy SH. Prevalence and incidence in primary care of chronic pelvic pain in women: evidence from a national general practice database. *Br J Obstet Gynaecol* 1999;106:1149–55. (Level II-3)
6. Zondervan KT, Yudkin PL, Vessey MP, Jenkinson CP, Dawes MG, Barlow DH, et al. Chronic pelvic pain in the community—symptoms, investigations, and diagnoses. *Am J Obstet Gynecol* 2001;184:1149–55. (Level II-2)
7. Prior A, Wilson K, Whorwell PJ, Faragher EB. Irritable bowel syndrome in the gynecological clinic. Survey of 798 new referrals. *Dig Dis Sci* 1989;34:1820–4. (Level II-2)
8. Parsons CL, Bullen M, Kahn BS, Stanford EJ, Willems JJ. Gynecologic presentation of interstitial cystitis as detected by intravesical potassium sensitivity. *Obstet Gynecol* 2001;98:127–32. (Level II-2)
9. Longstreth GF, Preskill DB, Youkeles L. Irritable bowel syndrome in women having diagnostic laparoscopy or hysterectomy. Relation to gynecologic features and outcome. *Dig Dis Sci* 1990;35:1285–90. (Level II-2)
10. Green CR, Flowe-Valencia H, Rosenblum L, Tait AR. The role of childhood and adulthood abuse among women presenting for chronic pain management. *Clin J Pain* 2001;17:359–64. (Level II-2)
11. Walling MK, Reiter RC, O'Hara MW, Milburn AK, Lilly G, Vincent SD. Abuse history and chronic pain in women: I. Prevalences of sexual abuse and physical abuse. *Obstet Gynecol* 1994;84:193–9. (Level II-2)
12. Rapkin AJ, Kames LD, Darke LL, Stamper FM, Naliboff BD. History of physical and sexual abuse in

- women with chronic pelvic pain. *Obstet Gynecol* 1990;76:92–6. (Level II-2)
13. Reiter RC, Gambone JC. Demographic and historic variables in women with idiopathic chronic pelvic pain. *Obstet Gynecol* 1990;75:428–32. (Level II-2)
 14. Jamieson DJ, Steege JF. The association of sexual abuse with pelvic pain complaints in a primary care population. *Am J Obstet Gynecol* 1997;177:1408–12. (Level II-2)
 15. Collett BJ, Cordle CJ, Stewart CR, Jagger C. A comparative study of women with chronic pelvic pain, chronic nonpelvic pain, and those with no history of pain attending general practitioners. *Br J Obstet Gynaecol* 1998;105:87–92. (Level II-2)
 16. Walker E, Katon W, Harrop-Griffiths J, Holm L, Russo J, Hickok LR. Relationship of chronic pelvic pain to psychiatric diagnoses and childhood sexual abuse. *Am J Psychiatry* 1988;145:75–80. (Level II-2)
 17. Heim C, Ehlert U, Hanker JP, Hellhammer DH. Abuse-related posttraumatic stress disorder and alterations of the hypothalamic-pituitary-adrenal axis in women with chronic pelvic pain. *Psychosom Med* 1998;60:309–18. (Level II-2)
 18. Harrop-Griffiths J, Katon W, Walker E, Holm L, Russo J, Hickok L. The association between chronic pelvic pain, psychiatric diagnoses, and childhood sexual abuse. *Obstet Gynecol* 1988;71:589–94. (Level II-2)
 19. Ehlert U, Heim C, Hellhammer DH. Chronic pelvic pain as a somatoform disorder. *Psychother Psychosom* 1999;68:87–94. (Level II-2)
 20. Reiter RC, Shakerin LR, Gambone JG, Milburn AK. Correlation between sexual abuse and somatization in women with somatic and nonsomatic chronic pelvic pain. *Am J Obstet Gynecol* 1991;165:104–9. (Level II-2)
 21. Scarinci IC, McDonald-Haile J, Bradley LA, Richter JE. Altered pain perception and psychosocial features among women with gastrointestinal disorders and history of abuse: a preliminary model. *Am J Med* 1994;97:108–18. (Level II-2)
 22. Cervero F, Janig W. Visceral nociceptors: a new world order? *Trends Neurosci* 1992;15:374–8. (Level III)
 23. Drossman DA. Physical and sexual abuse and gastrointestinal illness: what is the link? *Am J Med* 1994;97:105–7. (Level III)
 24. Ness RB, Soper DE, Holley RL, Peipert J, Randall H, Sweet RL, et al. Effectiveness of inpatient and outpatient treatment strategies for women with pelvic inflammatory disease: results from the Pelvic Inflammatory Disease Evaluation and Clinical Health (PEACH) Randomized Trial. *Am J Obstet Gynecol* 2002;186:929–37. (Level I)
 25. Westrom L. Effect of acute pelvic inflammatory disease on fertility. *Am J Obstet Gynecol* 1975;121:707–13. (Level II-2)
 26. 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;304:61–4. (Level II-2)
 27. Howard FM. The role of laparoscopy in chronic pelvic pain: promise and pitfalls. *Obstet Gynecol Surv* 1993;48:357–87. (Level III)
 28. Kresch AJ, Seifer DB, Sachs LB, Barrese I. Laparoscopy in 100 women with chronic pelvic pain. *Obstet Gynecol* 1984;64:672–4. (Level II-2)
 29. Ripps BA, Martin DC. Focal pelvic tenderness, pelvic pain, and dysmenorrhea in endometriosis. *J Reprod Med* 1991;36:470–2. (Level III)
 30. Cunanan RG Jr, Courey NG, Lippes J. Laparoscopic findings in patients with pelvic pain. *Am J Obstet Gynecol* 1983;146:589–91. (Level II-3)
 31. Summitt RL Jr. Urogynecologic causes of chronic pelvic pain. *Obstet Gynecol Clin North Am* 1993;20:685–98. (Level III)
 32. Ramahi AJ, Richardson DA. A practical approach to the painful bladder syndrome. *J Reprod Med* 1990;35:805–9. (Level III)
 33. Clemons JL, Arya LA, Myers DL. Diagnosing interstitial cystitis in women with chronic pelvic pain. *Obstet Gynecol* 2002;100:337–41. (Level II-2)
 34. Walker EA, Katon WJ, Jemelka R, Alfrey H, Bowers M, Stenchever MA. The prevalence of chronic pelvic pain and irritable bowel syndrome in two university clinics. *J Psychosom Obstet Gynecol* 1991;12(suppl):65–75. (Level III)
 35. Mens JM, Vleeming A, Stoekart R, Stam HJ, Snijders CJ. Understanding peripartum pelvic pain. Implications of a patient survey. *Spine* 1996;21:1363–9; discussion 1369–70. (Level III)
 36. Pfeifer ME, Hansen KA, Tho SP, Hines RS, Plouffe L Jr. Ovarian cholelithiasis after laparoscopic cholecystectomy associated with chronic pelvic pain. *Fertil Steril* 1996;66:1031–2. (Level III)
 37. Dulemba JF. Spilled gallstones causing pelvic pain. *J Am Assoc Gynecol Laparosc* 1996;3:309–11. (Level III)
 38. Sexton DJ, Heskestad L, Lambeth WR, McCallum R, Levin LS, Corey GR. Postoperative pubic osteomyelitis misdiagnosed as osteitis pubis—report of four cases and review. *Clin Infect Dis* 1993;17:695–700. (Level III)
 39. Barbieri RL. Stenosis of the external cervical os: an association with endometriosis in women with chronic pelvic pain. *Fertil Steril* 1998;70:571–3. (Level III)
 40. Kjerulff KH, Langenberg PW, Rhodes JC, Harvey LA, Guzinski GM, Stolley PD. Effectiveness of hysterectomy. *Obstet Gynecol* 2000;95:319–26. (Level II-3)
 41. Almeida EC, Nogueira AA, Candido dos Reis FJ, Rosa e Silva JC. Cesarean section as a cause of chronic pelvic pain. *Int J Gynaecol Obstet* 2002;79:101–4. (Level II-2)
 42. King PM, Myers CA, Ling FW, Rosenthal RH. Musculoskeletal factors in chronic pelvic pain. *J Psychosom Obstet Gynaecol* 1991;12(suppl):87–98. (Level II-3)
 43. Walter AJ, Hentz JG, Magtibay PM, Cornella JL, Magrina JF. Endometriosis: correlation between his-

- tologic and visual findings at laparoscopy. *Am J Obstet Gynecol* 2001;184:1407–11; discussion 1411–3. (Level II-2)
44. Howard FM. The role of laparoscopy as a diagnostic tool in chronic pelvic pain. *Baillieres Best Pract Res Clin Obstet Gynaecol* 2000;14:467–94. (Level III)
 45. Laufer MR, Goitein L, Bush M, Cramer DW, Emans SJ. Prevalence of endometriosis in adolescent girls with chronic pelvic pain not responding to conventional therapy. *J Pediatr Adolesc Gynecol* 1997;10:199–202. (Level II-2)
 46. Vercellini P, Fedele L, Arcaini L, Bianchi S, Rognoni MT, Candiani GB. Laparoscopy in the diagnosis of chronic pelvic pain in adolescent women. *J Reprod Med* 1989;34:827–30. (Level III)
 47. Almeida OD Jr, Val-Gallas JM. Conscious pain mapping. *J Am Assoc Gynecol Laparosc* 1997;4:587–90. (Level III)
 48. Demco LA. Effect on negative laparoscopy rate in chronic pelvic pain patients using patient assisted laparoscopy. *JLSLS* 1997;1:319–21. (Level III)
 49. Almeida OD Jr. Microlaparoscopic conscious pain mapping in the evaluation of chronic pelvic pain: a case report [published erratum appears in *JLSLS* 2002;6:192]. *JLSLS* 2002;6:81–3. (Level III)
 50. Howard FM, El-Minawi AM, Sanchez RA. Conscious pain mapping by laparoscopy in women with chronic pelvic pain. *Obstet Gynecol* 2000;96:934–9. (Level III)
 51. Parsons CL, Stein PC, Bidair M, Lebow D. Abnormal sensitivity to intravesical potassium in interstitial cystitis and radiation cystitis. *Neurourol Urodyn* 1994;13:515–20. (Level I)
 52. Gregoire M, Liandier F, Naud A, Lacombe L, Fradet Y. Does the potassium stimulation test predict cystometric, cystoscopic outcome in interstitial cystitis? *J Urol* 2002;168:556–7. (Level II-2)
 53. Chambers GK, Fenster HN, Cripps S, Jens M, Taylor D. An assessment of the use of intravesical potassium in the diagnosis of interstitial cystitis. *J Urol* 1999;162:699–701. (Level II-2)
 54. O'Leary MP, Sant GR, Fowler JF Jr, Whitmore KE, Spolarich-Kroll J. The interstitial cystitis symptom index and problem index. *Urology* 1997;49(5A suppl):58–63. (Level II-2)
 55. Messing EM. The diagnosis of interstitial cystitis. *Urology* 1987;29(suppl):4–7. (Level III)
 56. Waxman JA, Sulak PJ, Kuehl TJ. Cystoscopic findings consistent with interstitial cystitis in normal women undergoing tubal ligation. *J Urol* 1998;160:1663–7. (Level II-2)
 57. Onghena P, Van Houdenhove BV. Antidepressant-induced analgesia in chronic non-malignant pain: a meta-analysis of 39 placebo controlled studies. *Pain* 1992;49:205–19. (Meta-analysis)
 58. Rani PU, Naidu MU, Prasad VB, Rao TR, Shobha JC. An evaluation of antidepressants in rheumatic pain conditions. *Anesth Analg* 1996;83:371–5. (Level I)
 59. Saper JR, Silberstein, SD, Lake AE 3rd, Winters ME. Double-blind trial of fluoxetine: chronic daily headache and migraine. *Headache* 1994;34:497–502. (Level I)
 60. Max MB, Lynch SA, Muir J, Shoaf SE, Smoller B, Dubner R. Effects of desipramine, amitriptyline, and fluoxetine on pain in diabetic neuropathy. *N Engl J Med* 1992;326:1250–6. (Level II-3)
 61. Schreiber S, Vinokur S, Shavelzon V, Pick CG, Zahavi E, Shir Y. A randomized trial of fluoxetine versus amitriptyline in musculo-skeletal pain. *Isr J Psychiatry Relat Sci* 2001;38:88–94. (Level I)
 62. Walker EA, Sullivan MD, Stenchever MA. Use of antidepressants in the management of women with chronic pelvic pain. *Obstet Gynecol Clin North Am* 1993;20:743–51. (Level III)
 63. Walker EA, Roy-Byrne PP, Katon WJ, Jemelka R. An open trial of nortriptyline in women with chronic pelvic pain. *Int J Psychiatry Med* 1991;21:245–52. (Level III)
 64. Engel CC Jr, Walker EA, Engel AL, Bullis J, Armstrong A. A randomized, double-blind crossover trial of sertraline in women with chronic pelvic pain. *J Psychosom Res* 1998;44:203–7. (Level II-3)
 65. Simons DG, Travell JG, Simons LS. Travell & Simons' myofascial pain and dysfunction: the trigger point manual. Vol. I: upper half of body. 2nd ed. Baltimore (MD): Lippincott Williams and Wilkins; 1999. (Level III)
 66. Slocumb JC. Neurological factors in chronic pelvic pain: trigger points and the abdominal pelvic pain syndrome. *Am J Obstet Gynecol* 1984;149:536–43. (Level III)
 67. Owen PR. Prostaglandin synthetase inhibitors in the treatment of primary dysmenorrhea: outcome trials reviewed. *Am J Obstet Gynecol* 1984;148:96–103. (Level III)
 68. Marjoribanks J, Proctor ML, Farquhar C. Nonsteroidal anti-inflammatory drugs for primary dysmenorrhoea (Cochrane Review). In: *The Cochrane Library*, Issue 4, 2003. Chichester, UK: John Wiley & Son, Ltd. (Meta-analysis)
 69. Portenoy RK. Current pharmacotherapy of chronic pain. *J Pain Symptom Manage* 2000;19(suppl):S16–20. (Level III)
 70. Moulin DE, Iezzi A, Amireh R, Sharpe WK, Boyd D, Merskey H. Randomised trial of oral morphine for chronic non-cancer pain. *Lancet* 1996;347:143–7. (Level I)
 71. Jamison RN, Raymond SA, Slawsby EA, Nedeljkovic SS, Katz NP. Opioid therapy for chronic noncancer back pain. A randomized prospective study. *Spine* 1998;23:2591–600. (Level I)
 72. Wilder-Smith CH, Hill L, Spargo K, Kalla A. Treatment of severe pain from osteoarthritis with slow-release tramadol or dihydrocodeine in combination with NSAID's: a randomised study comparing analgesia, antinociception, and gastrointestinal effects. *Pain* 2001;91:23–31. (Level I)
 73. Proctor ML, Roberts H, Farquhar CM. Combined oral contraceptive pill (OCP) as treatment for primary dysmenorrhoea (Cochrane Review). In: *The Cochrane*

- Library, Issue 4, 2003. Chichester, UK: John Wiley & Son, Ltd. (Meta-analysis)
74. Propst AM, Laufer MR. Endometriosis in adolescents. Incidence, diagnosis, and treatment. *J Reprod Med* 1999; 44:751–8. (Level III)
 75. Vercellini P, Trespidi L, Colombo A, Vendola N, Marchini M, Crosignani PG. A gonadotrophin-releasing hormone agonist versus a low-dose oral contraceptive for pelvic pain associated with endometriosis. *Fertil Steril* 1993;60: 75–9. (Level I)
 76. Muzii L, Marana R, Caruana P, Catalano GF, Margutti F, Panici PB. Postoperative administration of monophasic combined oral contraceptives after laparoscopic treatment of ovarian endometriomas: a prospective, randomized trial. *Am J Obstet Gynecol* 2000;183:588–92. (Level I)
 77. Vercellini P, Aimi G, Panazza S, De Giorgi O, Pesole A, Crosignani PG. A levonorgestrel-releasing intrauterine system for the treatment of dysmenorrhea associated with endometriosis: a pilot study. *Fertil Steril* 1999;72:505–8. (Level II-3)
 78. Bergqvist A, Bergh T, Hogstrom L, Mattsson S, Nordenskjold F, Rasmussen C. Effects of triptorelin versus placebo on the symptoms of endometriosis. *Fertil Steril* 1998;69:702–8. (Level I)
 79. Goserelin depot versus danazol in the treatment of endometriosis the Australian/New Zealand experience. *Aust N Z J Obstet Gynaecol* 1996;36:55–60. (Level I)
 80. Henzl MR, Corson SL, Moghissi K, Buttram VC, Berqvist C, Jacobson J. Administration of nasal nafarelin as compared with oral danazol for endometriosis. A multicenter double-blind comparative clinical trial. *N Engl J Med* 1988;318:485–9. (Level I)
 81. Kennedy SH, Williams IA, Brodribb J, Barlow DH, Shaw RW. A comparison of nafarelin acetate and danazol in the treatment of endometriosis. *Fertil Steril* 1990;53: 998–1003. (Level I)
 82. Nafarelin for endometriosis: a large-scale, danazol-controlled trial of efficacy and safety, with 1-year follow-up. Nafarelin European Endometriosis Trial Group. *Fertil Steril* 1992;57:514–22. (Level I)
 83. Prentice A, Deary AJ, Goldbeck-Wood S, Farquhar C, Smith SK. Gonadotrophin-releasing hormone analogues for pain associated with endometriosis (Cochrane Review). In: *The Cochrane Library, Issue 4, 2003*. Chichester, UK: John Wiley & Son, Ltd. (Meta-analysis)
 84. Rock JA, Truglia JA, Caplan RJ. Zoladex (goserelin acetate implant) in the treatment of endometriosis: a randomized comparison with danazol. The Zoladex Endometriosis Study Group. *Obstet Gynecol* 1993;82: 198–205. (Level I)
 85. Wheeler JM, Knittle JD, Miller JD. Depot leuprolide versus danazol in treatment of women with symptomatic endometriosis. I. Efficacy results. *Am J Obstet Gynecol* 1992;167:1367–71. (Level I)
 86. Dlugi AM, Miller JD, Knittle J. Lupron depot (leuprolide acetate for depot suspension) in the treatment of endometriosis: a randomized, placebo-controlled, double-blind study. *Lupron Study Group. Fertil Steril* 1990;54: 419–27. (Level I)
 87. 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;93:51–8. (Level I)
 88. Soysal ME, Soysal S, Vicdan, Ozer S. A randomized controlled trial of goserelin and medroxyprogesterone acetate in the treatment of pelvic congestion. *Hum Reprod* 2001;16:931–9. (Level I)
 89. Mathias JR, Clench MH, Reeves-Darby VG, Fox LM, Hsu PH, Roberts PH, et al. Effect of leuprolide acetate in patients with moderate to severe functional bowel disease. Double-blind, placebo-controlled study. *Dig Dis Sci* 1994;39:1155–62. (Level I)
 90. Mathias JR, Clench MH, Roberts PH, Reeves-Darby VG. Effect of leuprolide acetate in patients with functional bowel disease. Long-term follow-up after double-blind, placebo-controlled study. *Dig Dis Sci* 1994;39:1163–70. (Level I)
 91. Mathias JR, Ferguson KL, Clench MH. Debilitating “functional” bowel disease controlled by leuprolide acetate, gonadotropin-releasing hormone (GnRH) analog. *Dig Dis Sci* 1989;34:761–6. (Level III)
 92. Houghton LA, Lea R, Jackson N, Whorwell PJ. The menstrual cycle affects rectal sensitivity in patients with irritable bowel syndrome but not healthy volunteers. *Gut* 2002;50:471–4. (Level II-2)
 93. Lentz GM, Bavendam T, Stenchever MA, Miller JL, Smallldridge J. Hormonal manipulation in women with chronic, cyclic irritable bladder symptoms, and pelvic pain. *Am J Obstet Gynecol* 2002;186:1268–71; discussion 1271–3. (Level III)
 94. 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;91:16–24. (Level I)
 95. Surrey ES, Hornstein MD. Prolonged GnRH agonist and add-back therapy for symptomatic endometriosis: long-term follow-up. *Obstet Gynecol* 2002;99:709–19. (Level I)
 96. Leather AT, Studd JW, Watson NR, Holland EF. The prevention of bone loss in young women treated with GnRH analogues with “add-back” estrogen therapy. *Obstet Gynecol* 1993;81:104–7. (Level I)
 97. Parazzini F, Fedele L, Busacca M, Falsetti L, Pellegrini S, Venturini PL, et al. Postsurgical medical treatment of advanced endometriosis: results of a randomized clinical trial. *Am J Obstet Gynecol* 1994;171:1205–7. (Level I)
 98. 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;101:979–85. (Level III)
 99. Carey MP, Slack MC. GnRH analogue in assessing chronic pelvic pain in women with residual ovaries. *Br J Obstet Gynaecol* 1996;103:150–3. (Level III)

100. Telimaa S, Ronnberg L, Kauppila A. Placebo-controlled comparison of danazol and high-dose medroxyprogesterone acetate in the treatment of endometriosis after conservative surgery. *Gynecol Endocrinol* 1987;1:363–71. (Level I)
101. 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;1:13–23. (Level I)
102. Farquhar CM, Rogers V, Franks S, Pearce S, Wadsworth J, Beard RW. A randomized controlled trial of medroxyprogesterone acetate and psychotherapy for the treatment of pelvic congestion. *Br J Obstet Gynaecol* 1989;96:1153–62. (Level I)
103. Harrison RF, Barry-Kinsella C. Efficacy of medroxyprogesterone treatment in infertile women with endometriosis: a prospective, randomized, placebo-controlled study. *Fertil Steril* 2000;74:24–30. (Level I)
104. Gestrinone versus a gonadotropin-releasing hormone agonist for the treatment of pelvic pain associated with endometriosis: a multicenter, randomized, double-blind study. Gestrinone Italian Study Group. *Fertil Steril* 1996;66:911–9. (Level I)
105. Regidor PA, Regidor M, Schmidt M, Ruwe B, Lubben G, Fortig P, et al. Prospective randomized study comparing the GnRH-agonist leuprorelin acetate and the gestagen lynestrenol in the treatment of severe endometriosis. *Gynecol Endocrinol* 2001;15:202–9. (Level I)
106. Muneyirci-Delale O, Karacan M. Effect of norethindrone acetate in the treatment of symptomatic endometriosis [published erratum appears in *Int J Fertil Womens Med* 1999;44:215]. *Int J Fertil Womens Med* 1998;43:24–7. (Level III)
107. Sundell G, Milsom I, Andersch B. Factors influencing the prevalence and severity of dysmenorrhoea in young women. *Br J Obstet Gynaecol* 1990;97:588–94. (Level II-2)
108. Golomb LM, Solidum AA, Warren MP. Primary dysmenorrhea and physical activity. *Med Sci Sports Exerc* 1998;30:906–9. (Level III)
109. Weiss JM. Pelvic floor myofascial trigger points: manual therapy for interstitial cystitis and the urgency-frequency syndrome. *J Urol* 2001;166:2226–31. (Level III)
110. Petros PP, Skilling PM. Pelvic floor rehabilitation in the female according to the integral theory of female urinary incontinence. First report. *Eur J Obstet Gynecol Reprod Biol* 2001;94:264–9. (Level III)
111. Mens JM, Snijders CJ, Stam HJ. Diagonal trunk muscle exercises in peripartum pelvic pain: a randomized clinical trial. *Phys Ther* 2000;80:1164–73. (Level I)
112. 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;62:696–700. (Level I)
113. Sutton CJ, Pooley AS, Ewen SP, Haines P. Follow-up report on a randomized controlled trial of laser laparoscopy in the treatment of pelvic pain associated with minimal to moderate endometriosis. *Fertil Steril* 1997;68:1070–4. (Level III)
114. Candiani GB, Fedele L, Vercellini P, Bianchi S, Di Nola G. Repetitive conservative surgery for recurrence of endometriosis. *Obstet Gynecol* 1991;77:421–4. (Level III)
115. Lee NC, Dicker RC, Rubin GL, Ory HW. Confirmation of the preoperative diagnoses for hysterectomy. *Am J Obstet Gynecol* 1984;150:283–7. (Level III)
116. Carlson KJ, Miller BA, Fowler FJ Jr. The Maine Women's Health Study: II. Outcomes of nonsurgical management of leiomyomas, abnormal bleeding, and chronic pelvic pain. *Obstet Gynecol* 1994;83:566–72. (Level II-3)
117. Carlson KJ, Miller BA, Fowler FJ Jr. The Maine Women's Health Study: I. Outcomes of hysterectomy. *Obstet Gynecol* 1994;83:556–65. (Level II-3)
118. Hillis SD, Marchbanks PA, Peterson HB. The effectiveness of hysterectomy for chronic pelvic pain. *Obstet Gynecol* 1995;86:941–5. (Level II-2)
119. Stovall TG, Ling FW, Crawford DA. Hysterectomy for chronic pelvic pain of presumed uterine etiology. *Obstet Gynecol* 1990;75:676–9. (Level II-2)
120. Beard RW, Kennedy RG, Gangar KF, Stones RW, Rogers V, Reginald PW, et al. Bilateral oophorectomy and hysterectomy in the treatment of intractable pelvic pain associated with pelvic congestion. *Br J Obstet Gynaecol* 1991;98:988–92. (Level II-3)
121. Steege JF, Stout AL. Resolution of chronic pelvic pain after laparoscopic lysis of adhesions. *Am J Obstet Gynecol* 1991;165:278–81; discussion 281–3. (Level II-2)
122. 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;99:59–62. (Level I)
123. Abouseif S, Tamaddon K, Chalfin S, Freedman S, Kaptein J. Sacral neuromodulation as an effective treatment for refractory pelvic floor dysfunction. *Urology* 2002;60:52–6. (Level II-3)
124. Siegel S, Paszkiewicz E, Kirkpatrick C, Hinkel B, Oleson K. Sacral nerve stimulation in patients with chronic intractable pelvic pain. *J Urol* 2001;166:1742–5. (Level III)
125. Everaert K, Devulder J, De Muyneck M, Stockman S, Depaeppe H, De Looze D, et al. The pain cycle: implications for the diagnosis and treatment of pelvic pain syndromes. *Int Urogynecol J Pelvic Floor Dysfunct* 2001;12:9–14. (Level II-2)
126. Black WT Jr. Use of presacral sympathectomy in the treatment of dysmenorrhea: a second look after twenty-five years. *Am J Obstet Gynecol* 1964;89:16–22. (Level III)

127. Lee RB, Stone K, Magelssen D, Belts RP, Benson WL. Presacral neurectomy for chronic pelvic pain. *Obstet Gynecol* 1986;68:517–21. (Level II-3)
128. Chen FP, Chang SD, Chu KK, Soong YK. Comparison of laparoscopic presacral neurectomy and laparoscopic uterine nerve ablation for primary dysmenorrhea. *J Reprod Med* 1996;41:463–6. (Level I)
129. 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;167:100–3. (Level I)
130. Tjaden B, Schlaff WD, Kimball A, Rock JA. The efficacy of presacral neurectomy for the relief of midline dysmenorrhea. *Obstet Gynecol* 1990;76:89–91. (Level III)
131. Chen FP, Soong YK. The efficacy and complications of laparoscopic presacral neurectomy in pelvic pain. *Obstet Gynecol* 1997;90:974–7. (Level II-3)
132. Sheld HH, Karamitsos CA, Parker EO 3rd, Shapiro BS. The use of superior hypogastric plexus block in the diagnosis of chronic pelvic pain. *Am J Gynecol Health* 1992;6:96–100. (Level III)
133. Bourke DL, Foster DC, Valley MA, Robinson JC. Superior hypogastric nerve block as predictive of presacral neurectomy success: a preliminary report. *Am J Pain Manage* 1996;6:9–12. (Level III)
134. Wechsler RJ, Maurer PM, Halpern EJ, Frank ED. Superior hypogastric plexus block for chronic pelvic pain in the presence of endometriosis: CT techniques and results. *Radiology* 1995;196:103–6. (Level III)
135. Lichten EM, Bombard J. Surgical treatment of primary dysmenorrhea with laparoscopic uterine nerve ablation. *J Reprod Med* 1987;32:37–41. (Level II-1)
136. Yen YK, Liu WM, Yuan CC, Ng HT. Addition of laparoscopic uterine nerve ablation to laparoscopic bipolar coagulation of uterine vessels for women with uterine myomas and dysmenorrhea. *J Am Assoc Gynecol Laparosc* 2001;8:573–8. (Level I)
137. Sutton C, Pooley AS, Jones KD, Dover RW, Haines P. A prospective, randomized, double-blind controlled trial of laparoscopic uterine nerve ablation in the treatment of pelvic pain associated with endometriosis. *Gynaecol Endosc* 2001;10:217–22. (Level I)
138. Renaer M, Vertommen H, Nijs P, Wagemans L, Van Hemelrijck T. Psychological aspects of chronic pelvic pain in women. *Am J Obstet Gynecol* 1979;134:75–80. (Level II-2)
139. Albert H. Psychosomatic group treatment helps women with chronic pelvic pain. *J Psychosom Obstet Gynaecol* 1999;20:216–25. (Level II-3)
140. Peters AA, van Dorst E, Jellis B, van Zuuren E, Hermans J, Trimbos JB. A randomized clinical trial to compare two different approaches in women with chronic pelvic pain. *Obstet Gynecol* 1991;77:740–4. (Level I)
141. Reiter RC. A profile of women with chronic pelvic pain. *Clin Obstet Gynecol* 1990;33:130–6. (Level II-2)
142. Laws A. Sexual abuse history and women's medical problems. *J Gen Intern Med* 1993;8:441–3. (Level III)
143. Proctor ML, Murphy PA. Herbal and dietary therapies for primary and secondary dysmenorrhoea (Cochrane Review). In: *The Cochrane Library, Issue 4, 2003*. Chichester, UK: John Wiley & Son, Ltd. (Meta-analysis)
144. Brown CS, Ling FW, Wan JY, Pilla AA. Efficacy of static magnetic field therapy in chronic pelvic pain: a double-blind pilot study. *Am J Obstet Gynecol* 2002;187:1581–7. (Level I)
145. Jorgensen WA, Frome BM, Wallach C. Electrochemical therapy of pelvic pain: effects of pulsed electromagnetic fields (PEMF) on tissue trauma. *Eur J Surg Suppl* 1994;(574):83–6. (Level III)
146. Proctor ML, Smith CA, Farquhar CM, Stones RW. Transcutaneous electrical nerve stimulation and acupuncture for primary dysmenorrhoea (Cochrane Review). In: *The Cochrane Library, Issue 4, 2003*. Chichester, UK: John Wiley & Son, Ltd. (Meta-analysis)
147. Taylor D, Miaskowski C, Kohn J. A randomized clinical trial of the effectiveness of an acupressure device (relief brief) for managing symptoms of dysmenorrhea. *J Altern Complement Med* 2002;8:357–70. (Level I)
148. Helms JM. Acupuncture for the management of primary dysmenorrhea. *Obstet Gynecol* 1987;69:51–6. (Level I)
149. Milsom I, Hedner N, Mannheimer C. A comparative study of the effect of high-intensity transcutaneous nerve stimulation and oral naproxen on intrauterine pressure and menstrual pain in patients with primary dysmenorrhea. *Am J Obstet Gynecol* 1994;170:123–9. (Level II-3)
150. Thomas CT, Napolitano PG. Use of acupuncture for managing chronic pelvic pain in pregnancy. A case report. *J Reprod Med* 2000;45:944–6. (Level III)

The MEDLINE database, the Cochrane Library, and ACOG's own internal resources and documents were used to conduct a literature search to locate relevant articles published between January 1985 and November 2003. The search was restricted to articles published in the English language. Priority was given to articles reporting results of original research, although review articles and commentaries also were consulted. Abstracts of research presented at symposia and scientific conferences were not considered adequate for inclusion in this document. Guidelines published by organizations or institutions such as the National Institutes of Health and the American College of Obstetricians and Gynecologists were reviewed, and additional studies were located by reviewing bibliographies of identified articles. When reliable research was not available, expert opinions from obstetrician-gynecologists were used.

Studies were reviewed and evaluated for quality according to the method outlined by the U.S. Preventive Services Task Force:

- I Evidence obtained from at least 1 properly designed randomized controlled trial.
- II-1 Evidence obtained from well-designed controlled trials without randomization.
- II-2 Evidence obtained from well-designed cohort or case-control analytic studies, preferably from more than 1 center or research group.
- II-3 Evidence obtained from multiple time series with or without the intervention. Dramatic results in uncontrolled experiments also could be regarded as this type of evidence.
- III Opinions of respected authorities, based on clinical experience, descriptive studies, or reports of expert committees.

Based on the highest level of evidence found in the data, recommendations are provided and graded according to the following categories:

Level A—Recommendations are based on good and consistent scientific evidence.

Level B—Recommendations are based on limited or inconsistent scientific evidence.

Level C—Recommendations are based primarily on consensus and expert opinion.

Copyright © March 2004 by the American College of Obstetricians and Gynecologists. All rights reserved. No part of this publication may be reproduced, stored in a retrieval system, or transmitted, in any form or by any means, electronic, mechanical, photocopying, recording, or otherwise, without prior written permission from the publisher.

Requests for authorization to make photocopies should be directed to Copyright Clearance Center, 222 Rosewood Drive, Danvers, MA 01923, (978) 750-8400.

ISSN 1099-3630

**The American College of
Obstetricians and Gynecologists
409 12th Street, SW
PO Box 96920
Washington, DC 20090-6920**

Chronic pelvic pain. ACOG Practice Bulletin No. 51. American College of Obstetricians and Gynecologists. *Obstet Gynecol* 2004;103:589-605.