

DYSMENORRHEA: ETIOLOGY, DIAGNOSIS AND THERAPY
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INTRODUCTION

Pain, whether acute, chronic, or recurring, is a major source of morbidity and disability, costing uncounted billions of dollars annually in both direct and indirect costs. In women, pelvic pain is by far the most common type of pain complaint for which treatment is sought. The diagnosis and treatment of pain has taken on increased importance in recent years.

In making a diagnosis and developing a treatment plan for the patient with pelvic pain, it is important to differentiate between the acute and chronic pain syndromes, and the recurrent symptoms of dysmenorrhea. Similarly, when evaluating dysmenorrhea, it is important to carefully separate patients with primary dysmenorrhea from those with secondary dysmenorrhea. With careful attention to the patient's history, and a well thought out approach to physical, laboratory, and other evaluations, most patients may have a diagnosis established and successful treatment provided.

Most patients who experience chronic pelvic pain will note worsening around the time of menstruation. For this reason, their evaluation will be similar to that of women whose symptoms are restricted to the time of menstruation. In most of these patients, an organic cause can be identified and appropriate therapy offered.

Non-cyclic chronic pelvic pain can be one of the most enigmatic of complaints. An exact organic cause is often difficult or impossible to establish. There are frequent associations with symptoms of depression, fatigue, loss of libido, and disturbances in eating and sleeping. Frequently the pain itself takes on the character of the disease and not a symptom. Both careful and thorough diagnosis (including laparoscopy), as well as psychological evaluation and support are required.

The recurrence of pain with each menstrual flow can inflict a tremendous burden upon a patient and her family. Many of these patients lose one to three days a month to debilitating symptoms. This loss from work, school, and home is, for many, intolerable. When specific causes are found therapy may be directed at correcting the underlying problem. With a much-

improved understanding of the physiology of primary dysmenorrhea, effective therapy is often available for these patients who, at one time, were relegated to home remedies and psychologists.

Our understanding of pelvic and abdominal pain has undergone many changes in recent years. New diagnostic techniques and improved therapies have changed the outlook for these patients. Today, with care and thoroughness, not only will a diagnosis usually be established, but effective therapy can be anticipated for the patient with pelvic pain and dysmenorrhea.

DYSMENORRHEA

SECONDARY DYSMENORRHEA AND CHRONIC PELVIC PAIN

INCIDENCE

The incidence of dysmenorrhea in general is difficult to estimate with any accuracy. It is safe to say that between ten and fifteen per cent of women suffer enough disability that they lose time from work, school, or home on a monthly basis. Most authors feel that between fifty and ninety per cent of women will suffer this degree of disability at least once during their reproductive years. The magnitude of this problem can be appreciated when one realizes that there are about 40 million women of reproductive age in the United States. Dysmenorrhea is most common in younger women, but it may extend throughout the reproductive years.

While the incidence of chronic pelvic pain is much lower, it represents a source of significant disability. It is one that often requires a great deal of time and resources, from both physician and patient, in order to make a diagnosis and establish treatment. Because the same processes that can cause secondary dysmenorrhea may also be a source for chronic pelvic pain between menstrual periods, these two subjects will be treated together.

II. MORBIDITY

Menstrual pain may vary from a mild inconvenience to a severe disability. It may be as mild as lower abdominal cramping or pressure. It may be as severe as extreme pain, nausea, vomiting, diarrhea, and collapse. While not directly life threatening, it can be a source of significant morbidity. In 1940, it was estimated that over 140 million hours were lost from the work force due to dysmenorrhea. It takes little imagination to extrapolate the extreme cost to today's society with its much larger work force of women.

III. ETIOLOGY

Secondary dysmenorrhea is caused by, or is secondary to, identifiable pathological or iatrogenic conditions acting on the uterus, tubes, ovaries, or pelvic peritoneum. Pain generally comes about when these processes alter pressure in or around the pelvic structures, change or restrict blood flow, or cause irritation of the pelvic peritoneum. These processes may act in combination with the normal physiology of menstruation to create discomfort, or they may act independently with their symptoms becoming noticeable during menstruation. When symptoms occur between menstrual periods, these processes may be the source of chronic pelvic pain.

IV. DIFFERENTIAL DIAGNOSIS OF ETIOLOGIES

The possible etiologies of secondary dysmenorrhea may be broadly classified as being intrauterine and extrauterine. Almost any process that can affect the pelvic viscera and cause acute pain can be a source for chronic pain or secondary dysmenorrhea. Since the treatment of these pain syndromes is based on treating the underlying cause, the importance of the differential diagnosis should be apparent.

A. Intrauterine causes

1. Adenomyosis

Adenomyosis is a condition characterized by a benign invasion of the endometrium into the uterine musculature, often accompanied by a diffuse overgrowth of the musculature as well. This condition is reported in between 25 and 40 per cent of hysterectomy specimens. Grossly, the uterus will be slightly enlarged and generally symmetrical. A colicky dysmenorrhea and menorrhagia are the most frequent presenting complaints for a patient with adenomyosis. The pain seen in adenomyosis is often referred to the rectum or the sacrum. Endometriosis is thought to be coexistent in about 15 per cent of cases. The final diagnosis of adenomyosis must be made under the microscope.

2. Myomas

Myomas, or uterine fibroids, are the most frequent occurring human tumor and are reported to occur in 20 per cent of women over 30, and 30 per cent of women over 40 years of age. These tumors may vary in size from very small to over 100 pounds in weight. While these tumors can occur in any part of the uterus, cervix, or the broad ligament, those most likely to be a cause of secondary dysmenorrhea are those that cause distortion of the uterus and the uterine cavity. Pain is thought to arise from disruption of the normal uterine muscle activity or from altered intrauterine pressures. The diagnosis of fibroids will generally be made based on the physical

examination findings of an enlarged and distorted uterus.

3. Polyps

Though an infrequent cause for dysmenorrhea, pedunculated masses within the uterine cavity can be a source of menstrual pain. When large enough to be symptomatic, these growths will generally be detectable by virtue of uterine enlargement or herniation through the cervix.

4. Intrauterine Contraceptive Devices

A common iatrogenic cause for secondary dysmenorrhea is the intrauterine contraceptive device (IUCD). The presence of this foreign body causes an increase in uterine activity that may be painful. This is especially common for women who have not had children. History and the presence of the IUCD string on physical examination should provide an adequate clue.

5. Infection

It is the consequences of infection, rather than the infection itself that are generally responsible for secondary dysmenorrhea or chronic pelvic pain. When active infection is present, it will most often present in an acute manner, and will be diagnosed as discussed earlier. Scarring and intraperitoneal adhesions can, and do, lead to restricted motion of the pelvic viscera and pain. This pain may only be apparent during menstruation, intercourse, bowel movements, or physical activity, or it may be constant and chronic in character. A history of pelvic infection, especially of repeated episodes, combined with a painful pelvic examination, thickening of the adnexa, and restricted motion, should all add to the suspicion.

6. Benign diseases of the vagina and cervix

Often blamed in the past as a source of dysmenorrhea, cervical stenosis and cervical lesions are only infrequently the source of menstrual or other pelvic pain. Inspection of the cervix on speculum examination will reveal the presence of a lesion. Cervical stenosis can only be assessed by the use of a probe. Because this is a generally uncomfortable procedure, it is not advised unless all other causes have been evaluated.

A. Extrauterine causes

1. Endometriosis

Endometriosis is the condition in which tissue resembling more or less perfectly normal uterine

mucous membrane occurs aberrantly in various locations outside the uterus. The chief locations in which endometrial implants are found are; the ovaries; uterine ligaments; rectovaginal septum; the pelvic peritoneum of the uterus, tubes, rectum, sigmoid, and bladder; and more distant locations such as the umbilicus and vagina. The implants of endometrial tissue may vary from the size of a pinhead to large pelvic masses of several centimeters. Endometriosis is most common in white women between 30 and 40 years of age. While about eight to ten per cent of patients will present with acute symptoms, most patients present complaining of severe dysmenorrhea with symptoms referred to the back and rectum. The presence of nodules in the uterosacral area, in a patient that otherwise resembles a patient with chronic pelvic inflammatory disease, should make one consider the possibility of endometriosis.

2. Tumors

Tumors that are either benign or malignant, arising in, or spreading to, the uterus, or adnexal structures, may be a cause of dysmenorrhea or pelvic pain. While an unusual cause of pain alone, the presence of a mass on pelvic examination should prompt the physician to consider all possible types of masses, not just "fibroids."

3. Inflammation

Chronic inflammation can be a source for chronic pelvic pain and dysmenorrhea. This may occur because of the active effects of inflammation, or by virtue of the scarring and damage done by past episodes. In the past, chronic inflammatory processes such as tuberculosis were occasionally found. While not absent today, tuberculosis is unlikely.

4. Adhesions

Adhesions arising from old inflammatory processes or surgical intervention can be a source for chronic pelvic pain, and less frequently, dysmenorrhea. Though generally not apparent on physical examination, the patient's history should be helpful in evaluating this possible cause.

5. Psychogenic

"Psychogenic" dysmenorrhea was once thought to be relatively common. As frequently occurs, when we do not have an explanation for the patient's complaint of pain, it is easy to dismiss it as "all in their head." Much has been written about the various personality types believed to be associated with dysmenorrhea and chronic pelvic pain. As we have learned more about both secondary and primary dysmenorrhea, it appears that very few patients truly have "psychogenic"

dysmenorrhea. While there is no doubt that dysmenorrhea affects different patients to different degrees and in different ways, and that any hard to substantiate complaint such as pain can be used as a way to gain attention or other psychological ends, this is not a common reason for the complaint of dysmenorrhea. In cases of chronic pain, there is often a strong overlay of psychological symptoms, as the pain itself becomes the disease. In approximately five to ten per cent of patients with chronic pelvic pain, no definite cause can be identified. This adds to the confusion about the true role of psychological factors in the complaint of pelvic pain. In general, listening to the patient will often give clues as to the likelihood of this etiology. Worsening of symptoms during times of stress, "inconvenient" recurrences that remove the patient from unpleasant situations, and other signs of emotional components should all make the physician suspicious. Only after other physical causes have been eliminated should this diagnosis be made.

6. "Pelvic congestive syndrome"

The term "pelvic congestive syndrome" is generally applied to patients with complaints of either chronic pelvic pain or recurrent dysmenorrhea in whom few or no clinical findings exist. Some authors have reported that some of these patients have "enlarged" or "engorged" pelvic veins when examined by laparoscopy. This has prompted the hypothesis that this engorgement leads to the complaints of pelvic heaviness and pain. This has not been adequately explored as a clinical entity, and would be a tenuous diagnosis to make.

7. Non-gynecological

As with acute pelvic pain, the abdominal wall, bladder, rectum, sigmoid, and skeletal elements of the pelvis can all be a potential source for chronic pelvic pain. Each of these areas should be included in both the history and physical evaluation of the patient with the complaint of pain. The same processes that can cause acute pain can also be responsible for a more chronic process. (See section 1)

V. DIAGNOSIS

A. Signs and Symptoms

The signs and symptoms involved in secondary dysmenorrhea and chronic pelvic pain can be many and varied. In general they will be referable to the underlying etiology. The complaint of gastrointestinal symptoms, urinary difficulties, back problems, or the like should alert the physician to the possibility of non-gynecological causes. The complaints of heavy menstrual flow combined with pain, suggests uterine changes such as adenomyosis, myomas, or polyps.

The complaint of pelvic heaviness, or change in abdominal contour, should raise the possibility of intra-abdominal neoplasia. Fever, chills, and malaise should suggest an inflammatory process. The coexisting complaint of infertility may suggest that endometriosis is a possibility. When the patient notes that her symptoms started only after placement of an IUCD, it is inappropriate not to think of the IUCD as a probable cause.

B. Physical Examination

The physical examination will generally provide clues to the diagnosis, if not the diagnosis itself, in most patients with the complaint of dysmenorrhea or chronic pelvic pain. The presence of asymmetrical, or irregular enlargement of the uterus should suggest myomas, or other tumors. Symmetrical enlargement of the uterus is often present in cases of adenomyosis, and occasionally when intrauterine polyps are present. The presence of painful nodules in the posterior cul-de-sac and restricted motion of the uterus are suggestive of endometriosis. Restricted motion of the uterus is also found in cases of pelvic scarring from adhesions, or inflammation. Inflammatory processes often cause thickening of the adnexal structures. This thickening may be palpable on physical examination. Unfortunately, in many cases of pelvic pain, laparoscopic examination of the pelvic organs may be necessary to complete the diagnostic process.

C. Laboratory, X-Ray, Ultrasonography

The laboratory evaluation of the patient with secondary dysmenorrhea or chronic pelvic pain is very limited. Blood counts may help to evaluate the presence of ongoing or excessive blood loss. Sedimentation rates may help to identify the presence of chronic inflammatory processes, but are, of necessity, nonspecific. Radiological evaluation of the patient is generally restricted to the evaluation of non-gynecological etiologies, such as those of the gastrointestinal or urinary tract. Ultrasound examinations of the pelvis may be of great usefulness. Ultrasound can demonstrate the presence and extent of myomas, adnexal and other tumors, or locate an intrauterine IUCD.

VI. MANAGEMENT

Whenever possible, the treatment of both secondary dysmenorrhea and chronic pelvic pain is directed toward correcting or removing the underlying causative factors. While analgesics, antispasmodics, and birth control pills may have some temporary benefit, only specific therapy aimed at correcting the cause will ultimately be successful. This may range from removing an offending IUCD to anti-estrogen therapy for endometriosis, from removal of a polyp to hysterectomy. In those few patients in whom no specific diagnosis may be established, and intractable, debilitating pain is present, presacral neurectomy may be useful. This is generally

reserved for those patients in whom no other therapy has been useful. While some reports claim up to 85 per cent relief from pain, the procedure is far from innocuous and its use should be limited.

VII. PROGNOSIS

When the proper diagnosis is made, and appropriate therapy is instituted, there is every reason to expect complete return to normal function in almost every case.

DYSMENORRHEA

PRIMARY DYSMENORRHEA

I. INCIDENCE

The incidence of primary dysmenorrhea is similar to that discussed under secondary dysmenorrhea. Primary dysmenorrhea is more common among younger patients, whereas secondary dysmenorrhea becomes more common as the patient ages. It is uncommon for true primary dysmenorrhea to occur during the first three to six menstrual cycles of a young woman. The incidence of primary dysmenorrhea is greatest in women in their late teens to early 20's. The incidence then declines with age, but even women in their 40's may be affected. It appears that childbearing does not affect the incidence.

II. MORBIDITY

The pain of primary dysmenorrhea is often greater than that experienced with secondary dysmenorrhea. In addition to pain, these patients often experience debilitating nausea, vomiting, diarrhea, and symptomatic vasoconstriction. For the women who suffer from primary dysmenorrhea, this can be the source of significant disruption in their lives.

III. ETIOLOGY

For many years the etiology of primary dysmenorrhea was written off as being due to "conflicting feelings about sexuality and childbearing" in "anxious hostile women who reject the feminine role." Through evidence derived from several (or) many (or) some studies and several lines of research, it appears that primary dysmenorrhea occurs because of either an increase in uterine prostaglandin F_{2a}, an increased sensitivity to prostaglandins, or both. Prostaglandin F_{2a} is a potent uterine muscle stimulator. Increased levels of prostaglandin F_{2a} lead to an increase in uterine contractile activity, ischemia, and pain. Prostaglandin F_{2a} is also a potent stimulator of

the smooth muscle of the gastrointestinal tract, leading to the symptoms of nausea, vomiting, and diarrhea often experienced.

IV. PATHOPHYSIOLOGY

Prostaglandins are derivatives of fatty acids commonly found in the cell wall. The production of various prostaglandins is shown in figure 1. Prostaglandin production in the uterus increases under the influence of progesterone, reaching a peak at, or soon after, the start of menstruation. Once menstruation begins, formed prostaglandins are released from the shedding endometrium. In addition, the necrosis of endometrial cells provides increased substrate for the synthesis process. Two main prostaglandins are made in the uterus: Prostaglandin F2a and Prostaglandin E2. Prostaglandin F2a is a potent smooth muscle stimulator and vasoconstrictor. Prostaglandin E2 is a potent vasodilator and platelet disaggregator. Prostaglandin E2 has been implicated as a cause of primary menorrhagia.

The uterine activity found in patients with primary dysmenorrhea can be striking. During normal menstruation, contractions of 50 to 80 mmHg, lasting 15 to 30 seconds, are not uncommon. These generally occur with a frequency of one to four contractions in ten minutes. Resting pressure in the uterus is generally five to 15 mmHg. In women with dysmenorrhea, contractions may have peak pressure in excess of 400 mmHg, last longer than 90 seconds, and have less than 15 seconds of rest at a baseline pressure sometimes as high as 80 to 100 mmHg. Pressures of this magnitude and duration cause significant ischemia.

The exact mechanism that creates the sensation of pain is unknown. Recent studies show a strong correlation between pain and pain relief, and the parameters of uterine work, maximal pressures, frequency and quality of contractions, rate of pressure change, and the quality of "rest" between uterine contractions. With the exception of rate of change, these parameters all have intuitive bearing on perfusion and ischemia. Further research into uterine blood flow and a better understanding of the phenomena of pain itself may help to resolve these questions.

V. DIFFERENTIAL DIAGNOSIS

The most important differential diagnosis to consider is that of secondary dysmenorrhea. While the history and the patient's description of symptoms are often characteristic, the diagnosis of primary dysmenorrhea should not be made without thoroughly evaluating and eliminating other possible causes.

VI. DIAGNOSIS

A. Signs and Symptoms

Patients with primary dysmenorrhea generally present with the complaint of recurrent, month–after–month, spasmodic lower abdominal pain occurring on the first one to three days of menstruation. The pain is diffusely located in the suprapubic area with radiation around and through to the back. The labor–like pain is described as "coming and going," and the patient will often use a fist opening and closing to illustrate their description. This pain is often accompanied by moderate to severe nausea. Vomiting and/or diarrhea are not infrequent. Patients often double up into a fetal position in an effort to gain relief. Many patients will report having tried a heating pad or hot water bottle in an effort to decrease their discomfort.

B. Physical Examination

The physical examination of a patient with primary dysmenorrhea should be normal. There should be no palpable abnormalities of the uterus or adnexa. Speculum and abdominal examinations should similarly be normal. If the patient is examined during the time of actual symptoms, they are often found to be pale and "shocky." The abdomen will be soft and non–tender, and the uterus normal.

C. Laboratory, X–Ray, Ultrasonography

The use of these modalities is limited to evaluating possible causes of secondary dysmenorrhea only.

VII. MANAGEMENT

A. Modification of periods

Because anovulatory cycles are less likely to have symptoms of primary dysmenorrhea, one approach has been to modify the menstrual cycle itself. In 1865 this consisted of surgical removal of the ovaries. Today, though complete suppression of ovarian cyclic function is possible in much less invasive ways, this approach is impractical for most patients. Suppression of ovulation with oral contraceptive agents does provide many patients with complete or partial improvement. The use of oral contraceptives yields a thinner, more atrophic, endometrium, with much less prostaglandin. This, in turn, causes the menstruation to be shorter, lighter, and have less cramping. Relief is not always complete, and many patients do not want, or cannot take,

these medications. Oral contraceptives may be a reasonable choice for those patients who desire contraception and have no contraindications.

B. Modification of Pain

Analgesics have been the mainstay of dysmenorrhea therapy in the past. For mild pain, aspirin, acetaminophen, propoxyphene or their compounds have found wide use. Unfortunately, the pain of primary dysmenorrhea is generally of enough magnitude that these agents are less than fully effective. Potent analgesics are often required. Useful agents and their compounds are: butalbital (Fiorinal®, 1 or 2 every 4 hours), oxycodone (Percodan®, 1 every 6 hours), pentazocine (Talwin®, 1 every 3 to 4 hours), promethazine (Synalgos®, 2 every 4 hours), codeine (30 - 60 mg every 4 hours), or meperidine (Demerol®, 50 - 100 mg every 4 to 6 hours). While these agents, adjusted to the needs of the individual, may give good pain relief, their potential side effects may still render the patient unable to function normally. The use of these potent drugs should be limited and care exercised.

C. Pain Prevention

By suppressing uterine activity it is possible to interrupt the process by which the dysmenorrhea pain is created. Drugs such as calcium antagonists (Nifedipine), or spasmolytic agents (Isoxuprine, Papaverine, Ritodrine) may suppress uterine activity in the laboratory, but their side effects have limited clinical usefulness. The most practical method of suppressing uterine activity has been by reducing the level of prostaglandin through prostaglandin synthetase inhibitors and/or reducing the sensitivity of the myometrial receptors. The agent that can produce these effects are the non-steroidal anti-inflammatory (NSAI) drugs often employed in arthritis treatment.

There are two broad classes of NSAI compounds, each with sub-groups (Figure 2). Drugs of the enolic acid type appear to be primarily Type II inhibitors of prostaglandin synthesis. These agents act through the inhibition of the isomerase/reductase step in the formation of PGE₂ and PGF_{2a}. The most frequently used agent in the enolic acid groups are phenylbutazone and piroxicam. While phenylbutazone (Azolid®, Butazolidin®) is an effective short term analgesic for musculoskeletal pain (through anti-prostaglandin activity), its relative toxicity has limited its use. Piroxicam (Feldene®) has a long half-life (50 hours) which allows once a day dosage. Its action as an anti-inflammatory drug is well established, but its use as an analgesic or for the indication of dysmenorrhea has not been fully evaluated.

It is the carboxylates that most physicians are familiar with and have the most day-to-day use for pain relief and dysmenorrhea. Within this major group there are four families of compounds that have individual characteristics. The Salicylic acids and esters appear to inhibit cyclo-oxygenase by the donation of their acetyl group to the enzyme. The most recognized agent in this group, aspirin, has a very low potency for reducing prostaglandin synthesis and, so, has had little clinical utility in the treatment of moderate or severe dysmenorrhea. Increased potency is seen in the acetic acid groups. While sulindac (Nalfon®) must undergo reduction to a sulfide form before becoming active, most of the drugs in this group are effective as anti-inflammatory and analgesic agents. In several studies, indomethacin has shown usefulness in treating dysmenorrhea, but a moderate incidence of side effects has also limited the use of this and most other drugs in this class for treating dysmenorrhea. The most commonly used drugs for dysmenorrhea come from two classes: arylalkanoic acids (propionic acid derivatives) and anthranilic acids (fenamates).

Of the propionic acid derivatives currently available, only ibuprofen (Motrin, Rufen®) and naproxen (Naprosyn®, Anaprox®) are approved and commonly used for this indication. Other drugs of this class (benoxaprofen, ketoprofen, fenoprofen) have been used for pain relief or arthritis therapy, but are not approved for dysmenorrhea. Ibuprofen was the first drug of this class to be studied in dysmenorrhea and has shown effectiveness in subsequent subjective studies. The most studied (at least most widely reported) drug of this class is naproxen. The subjective studies of naproxen and naproxen sodium have shown good pain relief in dysmenorrhea, even in the presence of intrauterine devices.

The fenamates are potent prostaglandin synthetase inhibitors, but in addition have been shown to antagonize the actions of already formed prostaglandins. Some of the first non-steroidal anti-inflammatory drug studies in dysmenorrhea used these agents, and further studies have proven them to be very effective in reducing the subjective discomfort of dysmenorrhea. In this country, mefenamic acid (Ponstel®) is approved for dysmenorrhea and clinical studies supporting the use of meclofenamate (Meclomen®) are well under way. The dual action of synthesis inhibition and direct antagonism should give these agents an edge in efficacy. New In Vitro studies have shown meclofenamate to inhibit the activity of 5-Lipoxygenase, while members of the propionic acid group have little or no inhibitory ability. The clinical significance of being able to inhibit the production of the extremely potent leukotrienes is yet to be fully explored.

In most clinical settings, the agents most commonly employed will be mefenamic acid (Ponstel®), naproxen sodium (Anaprox®), and ibuprofen (Motrin®, Rufen®). Clinical dosages for these drugs and the other NSAID agents are shown in Table 1. Once an agent is selected it should be tried over the course of two to four cycles before success or failure should be assessed.

If therapy is unsuccessful, some patients may still have favorable response to another NSAID drug. This second choice should be chosen from a different chemical class for the best chance of success. Patients should be reminded to take their medication at the onset of menstruation or symptoms, and maintain consistent medication for as long as symptoms would normally last if medication had not been taken.

The physician should always be aware of the potential for side effects with any medication. While side effects for these three medications are infrequent and generally mild, serious side effects are possible. The short duration of use in dysmenorrhea limits the risk of serious side effect. If pain relief is not complete, patients should be warned not to add additional analgesics, especially other NSAID agents, because of possible potentiation of gastrointestinal and other side effects.

VIII. PROGNOSIS

Through careful evaluation of the individual patient's symptoms and physical findings, it should be possible to make the diagnosis of primary dysmenorrhea and select the most appropriate mode of therapy, be it analgesic, hormonal or NSAID, to allow a full, comfortable, and productive lifestyle. When at least partial relief of the symptoms of dysmenorrhea is not achieved, serious reappraisal of the original diagnosis of primary dysmenorrhea must be made.

Table 1

Families of Nonsteroidal Anti-inflammatory Drugs	
DRUG	TRADE NAME
CARBOXYLATES -	
SALICYLIC ACIDS:	
Acetylsalicylic acid	Aspirin (various)
Diflunisal	Dolobid
Salicylate	Disalcid, Salsalate, Trilisate
INDOLEACETIC ACIDS:	
Diclofenac Potassium	Cataflam
Diclofenac Sodium	Voltaren, Arthrotec (combined with misoprostol)
Etodolac	Lodine
Indomethacin	Indocin
Ketorolac Tromethamine	Acular, Toradol
Sulindac	Clinoril, Sulindac
Tolmetin	Tolectin, Tolmetin
PROPIONIC ACIDS	
Fenoprofen Calcium	Fenoprofen calcium
Flurbiprofen	Flurbiprofen
Ibuprofen [†]	Motrin
Ketoprofen	Orudis, Oruvail, Ketoprofen
Naproxen Sodium [†]	Aleve, Anaprox, Naprelan
Naproxen [†]	Naprosyn
FENIMATES	
Meclofenamate Sodium [†]	Meclofenamate Sodium
Mefenamic Acid [†]	Ponstel
ENOLIC ACIDS -	
PYRAZOLONES	
Oxyphenbutazone	
Phenylbutazone	(Azolid, Butazolidin)
Nabumetone	Relafen
Celecoxib	Celebrex
Refecoxib [†]	Vioxx
OXICAMS	
Piroxicam	Feldene, Piroxicam
Meloxicam	Mobic

[†] FDA approved for primary dysmenorrhea

Modified from: Smith RP: Gynecology in Primary Care. Williams and Wilkins, Baltimore, Maryland, 1996, p. 399.

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He is married, with one son who is a graduate of the University of Southern California in Cinema and a second son who is a Junior at Denison University in Granville, Ohio. Dr. Smith is a collector of antique gumball machines and a semi-professional magician as well.