Chronic abdominal pain is a challenging problem for primary care physicians, gastroenterologists, and surgeons. Many disorders discussed elsewhere in this text can produce chronic abdominal pain. Some of the major ones are listed in Table 5-1. One of these disorders, functional abdominal pain syndrome (FAPS), is the focus of this chapter.

DEFINITION AND CLASSIFICATION OF FUNCTIONAL ABDOMINAL PAIN SYNDROME

FAPS is a distinct medical disorder, part of the group of functional gastrointestinal disorders. These disorders are characterized by chronic, recurrent, or continuous abdominal pain that is poorly related to gut function, often not well localized. They are more properly understood as an abnormal perception of normal (regulatory) gut function rather than as a motility disorder.

Criteria for a diagnosis of FAPS are derived from a consensus of experts in the study of functional gastrointestinal disorders (Table 5-2). These experts have published criteria, the Rome II diagnostic criteria, for diagnosis of these various functional conditions, among them FAPS. Follow-up studies over several years of patients who meet diagnostic criteria for FAPS rarely yield other specific causes of chronic abdominal pain.

FAPS is commonly associated with the reporting of other unpleasant somatic symptoms, and when it persists and/or dominates the patient’s life, it is usually associated with chronic pain behaviors and comorbid psychological disturbances. Patients with FAPS typically define their illness as medical. They are reluctant to receive psychological assessment or treatment. Psychological disturbances, if present, must be considered as comorbid features of FAPS rather than as part of a primarily psychiatric problem.

Physicians may also be concerned as to whether they have the interest, or can provide the time, to care for patients with FAPS. They may feel unable to move the discussion from a focus on symptoms and making a diagnosis to more effective approaches directed toward coping and management of an established functional gastrointestinal disorder. Occasionally, the physician may feel that the patient does not want to be helped, but this is not usually the case. Rather, the chronic pain disorder and associated psychosocial influences may lead to erroneous attitudes and maladaptive (e.g., “catastrophizing”) behaviors that the physician can work to modify and develop an effective physician-patient collaboration.

Epidemiology and Impact on Health Care Systems

In the U.S. Householder Survey of Functional Gastrointestinal Disorders, FAPS was seen in 2% of the sample (primarily women), considerably less than irritable bowel syndrome (IBS) (9%). Patients with FAPS missed more work days from illness than those without bowel symptoms and had more physician visits. A significant proportion of these
Structural Disorders
Chronic pancreatitis
Abdominal neoplasms (carcinomas, lymphomas, others)
Inflammatory bowel diseases
Mesenteric ischemia
Pelvic inflammatory diseases
Endometriosis

Functional Gastrointestinal Disorders
Irritable bowel syndrome
Functional (nonulcer) dyspepsia
Functional abdominal pain syndrome
Levator syndrome
Biliary pain (gallbladder or sphincter of Oddi dysfunction)

people with refractory symptoms are selectively referred to gastroenterology practices and medical centers; they then have a disproportionate number of health care visits and undergo numerous diagnostic procedures and treatments. One study in England evaluated 20 patients with FAPS. All were women; on average, they were seen by 5.7 consultants; underwent 6.4 endoscopic or radiologic procedures; and had 2.7 major operations, primarily hysterectomy and laparotomy, with only temporary benefit. More than 85% were given a psychiatric referral, but most preferred to be seen by medical physicians, and 40% had tried alternative medical treatments.

PATHOPHYSIOLOGY OF CHRONIC PAIN

Chronic pain is a multidimensional (sensory, emotional, cognitive) experience that is best explained by abnormalities in neurophysiologic functioning at the afferent, spinal, and central nervous system (CNS) level. Chronic pain, unlike acute pain arising from peripheral/visceral injury or disease, has no increased afferent visceral stimuli from structural abnormalities and tissue damage. Motility is not abnormal. Pain in FAPS is due to CNS amplification (i.e., failure to down-regulate) of incoming regulatory visceral afferent signals to conscious awareness. The way in which this takes place (discussed later) and how it can be modified are relevant to understanding the clinical features of FAPS and in approaching treatment.

Further, FAPS is neither a medical nor a psychiatric disease but is part of a biopsychosocial disorder related to dysfunction of the brain-gut axis. As shown in Figure 5–1, the clinical expression of FAPS is derived from psychological and gut physiologic input, interacting via the CNS-gut axis. This model enables the physician to integrate the newly emerging clinical, physiologic, and psychosocial features of FAPS into a more comprehensible form. Applying this information when communicating with patients is helpful in establishing an effective plan for care. (A more complete description of the biopsychosocial model on which this approach is based is presented in Chapter 122.)

Ascending Visceral Pain Transmission

(Fig. 5–2)

The afferent transmission of visceral abdominal pain involves (1) first-order neurons that innervate the viscera and carry information to the thoracolumbar sympathetic nervous system. (2) Second-order neurons that synapse in the spinal cord and project to higher brain areas, such as the thalamus and insula. (3) Third-order neurons that project to the limbic system and prefrontal cortex, where pain perception and modulation occur. This pathway is modulated by input from other sensory systems, such as somatosensory and visceral pain pathways. (4) Fourth-order neurons that project to the hypothalamus and amygdala, where pain-related autonomic responses are generated. (5) Fifth-order neurons that project to the spinal cord and limbic system, where pain-related behaviors and memories are formed. This pathway is modulated by input from higher brain areas, such as the prefrontal cortex and amygdala. (6) Sixth-order neurons that project to the spinal cord and limbic system, where pain-related emotional responses are generated. This pathway is modulated by input from higher brain areas, such as the prefrontal cortex and amygdala.
system, subsequently synapsing in the dorsal horn of the spinal cord; and (2) second-order neurons that cross and ascend from the dorsal horn via the spinothalamic and spinoreticular tracts to synapse, respectively, with the thalamus via the spinothalamic system to the somatosensory cortex (sensory-discriminative component), which is involved in the somatotopic or point-specific localization and intensity of afferent signals, and from the reticular formation via the spinoreticular tracts, through the medial thalamus to the limbic system (motivational-affective component), containing the anterior cingulate cortex (ACC) (see also Chapter 4). The limbic system serves as a modulator of the pain experience, based on the individual’s emotional state, prior experiences, or cognitive interpretation of the signal. This multi-component integration of nociceptive (i.e., stimulative) information in the CNS explains the variability in the experience and reporting of pain. Motivational-affective and evaluative levels of the CNS contribute particularly to chronic pain, permitting perception of pain symptoms in the absence of nociceptive input and providing the basis for understanding psychological influences and applying psychopharmacologic treatments.

This conceptual scheme of pain modulation has been demonstrated through positron-emission tomography (PET) imaging, using radiolabeled oxygen. In a group of healthy subjects who immersed their hands in hot water, half were hypnotized to experience the immersion as painful and the other half as not painful, or pleasant. The changes in cortical activation were compared between these two groups, and no difference was found in activity in the somatosensory cortex. However, those experiencing pain had significantly greater activation of the ACC of the limbic system, involved with the affective component of the pain experience, distinguishing it from the somatosensory cortex. Similarly, in a study where cardiac ischemia was induced by dobutamine, patients with “silent” myocardial ischemia (i.e., ischemia without chest pain) had reduced ACC activation compared with patients who experienced angina pectoris. Because the ACC has connections to descending, corticofugal inhibitory pathways and is a site of high endorphin activity (see later), it is postulated that in normal subjects, activation of this region from peripheral/visceral afferent activity may in part serve to down-regulate these signals via a gating mechanism (see later).

**Descending Modulation of Pain**

Descending modulation of painful stimuli is the regulation of afferent pain impulses from the cortex down to the visceral nerves and is explained by what has become known as the *gate control theory*. In this model, the central descending control of this gating system occurs primarily through the descending inhibitory or endorphin-mediated analgesia system. This is an endorphin- or enkephalin-based neural network, originating from the cortex and limbic system and descending to the spinal cord, with major links in the mid-brain (periaqueductal gray) and medulla (raphe magnus) (Fig. 5–3). This system inhibits nociceptive projection either directly on the second-order neuron or indirectly via inhibitory interneurons in the spinal cord. Then the dorsal horn of the spinal cord acts like a gate to increase or decrease transmission of afferent impulses arising from peripheral nociceptive sites to the CNS. Endorphin activity is facilitated by serotonin (5-hydroxytryptamine [5-HT]) and possible norepinephrine release, which are present in high concentration in this system.

**Visceral Sensitization to Pain**

A relatively new concept to advance an understanding of FAPS is that recurrent peripheral stimulation up-regulates afferent signals or inhibits descending pain control mechanisms, sensitizing the bowel and thereby producing a state of visceral hyperalgesia (increased pain response to a noxious signal) and/or chronic pain. A few clinical studies support this concept. In one experiment involving healthy subjects, a repetitive series of balloon inflations in the colon led to a progressive but transitory increase in pain intensity and a 228% increase in the area under the curve. Visceral sensitization may develop at any or several levels of the neuraxis. At the mucosal level, the recruitment of afferent (“silent”) nociceptors has been proposed. The receptors fire only with prolonged or recurrent peripheral stimulation (e.g., from inflammation, enhanced motility, and tissue damage) and appear to change the excitability of somatic and visceral inputs.
ond-order neurons, which outlasts the period of increased peripheral stimulation. This produces enough sensitization ("pain memory") so that after the peripheral stimulation subsides, sensitized second-order neurons continue to fire, and subthreshold regulatory stimuli then are still perceived as painful.

Visceral sensitization may also occur at the spinal level (spinal hyperexcitability). With an enteric infection or bowel trauma, the increased afferent stimuli from the gut increases neurotransmitter release in the dorsal horn, which travels to higher centers and is perceived as painful. This should resolve with resolution of the injury. However, among patients with functional gastrointestinal disorders, the dorsal horn's sensitivity to incoming signals may remain upregulated, so that even baseline regulatory visceral activity is still experienced as painful (pain memory). This is analogous to an amplifier system, where the incoming signal is the same but the "volume" is turned up (at the spinal cord).

Biochemical Mechanisms for Sensitization

The biochemical basis for visceral sensitization is under active study. At the gut level, there is interest in the putative role of 5-HT. 5-HT is found primarily in mucosal enterochromaffin cells, where it appears to serve as a neurotransmitter of the enteric nervous system (ENS) and as a paracrine molecule signaling other (e.g., vagal) neural activity. 5-HT mediates numerous functions (e.g., bowel contraction or relaxation, intestinal secretion, pain and nausea sensations), depending on its subtype and location. 5-HT is actively released from the enterochromaffin cells with mechanical or chemical stimuli, which increases peristalsis, bowel wall tone, and sensory perception. Clinically, patients with IBS postprandially appear to release greater amounts of 5-HT into the blood. This finding suggests that 5-HT may mediate the visceral hypersensitive pain/diarrhea response to a meal (presumably initiated by luminal distention), and this has raised interest in the use of selective 5-HT, antagonists to reduce these symptoms (see Chapter 91).

At the spinal level, repeatedly stimulated afferent fibers increase second-order neuronal responsiveness possibly through the release of stimulatory neuropeptides (e.g., substance P, neurokinin, and calcitonin gene–related peptide, among others) and excitatory amino acids (e.g., glutamate). These substances increase membrane excitability and activate postsynaptic receptors (primarily N-methyl-D-aspartate but also substance P and calcitonin gene–related peptide), which leads to increased release and influx of intracellular Ca2+. This in turn, may activate phospholipase C, protein kinase C, nitric oxide, and other second messengers, which increase neuronal excitability and presynaptic transmitter release, thereby permitting more Ca2+ influx, creating a positive feedback loop. These substances may also increase the expression of proto-oncogenes such as cFos, which act as third messengers in the transcriptional control of genes that encode neuropeptides such as dynorphin. Increased dynorphin gene expression can enhance neuronal excitability for days to weeks. Animal studies show a strong relationship between noxious stimulus–induced Fos protein expression, dorsal horn neuronal excitability, and prolonged behavioral hyperalgesia.

Role of the Central Nervous System in Functional Abdominal Pain Syndrome

Although peripheral sensitization may influence the onset of pain, clearly the CNS is preeminently involved in the predisposition and perpetuation of chronic pain. This is evident by the lack of peripheral motor or sensory abnormalities and the strong association of psychosocial disturbances in these disorders. In addition, comorbid psychiatric diagnosis, major life stress, a history of sexual or physical abuse, poor social support, and maladaptive coping all are associated with more severe and chronic abdominal pain and poorer health outcome. Their presence in patients with FAPS and other functional gastrointestinal pain may impair or diminish descending inhibitory pathways acting on dorsal horn neurons or amplify visceral afferent signals.

One prospective study of patients with postinfectious IBS supports the importance of the brain in the experience of gastrointestinal pain. Of 94 patients hospitalized with gastroenteritis and no prior history of bowel complaints, 72 recovered but 22 continued to have abdominal pain and bowel dysfunction 3 months later. Both groups had similar levels of gut hypermotility and visceral sensitivity 3 months later. What characterized the group with continued symptoms was their greater psychological distress at the time of the infectious episode and a greater number of mucosal inflammatory cells during the 3-month follow-up period. It was proposed that even though both groups had abnormal motility and visceral hypersensitivity, it was the CNS upregulation of these peripheral signals occurring in the psychologically distressed group that raised the afferent signals to a level of awareness and perpetuated the symptoms. This may have been mediated via CNS influence on peripheral inflammatory/cytokine activity.

It is possible that the links between emotional distress and chronic pain may be mediated through impairment in the limbic system's ability to modulate visceral signals. Recent studies now suggest that the motivational-affected component of the central pain system, specifically the ACC (see Figs. 5–2 and 5–3), is dysfunctional in patients with IBS and other chronic painful conditions. PET brain imaging in response to rectal distention of patients with IBS have shown decreased activation of the perigenual area of the ACC and increased activation of the thalamus. Similar results are found in patients with abuse history and post-traumatic stress disorder. Furthermore, the return of ACC reactivity among depressed patients is associated with clinical improvement and predicts response to antidepressant treatment. This suggests that dysregulation of central pain modulation (disinhibition) may occur in various medical and psychological conditions and that treatments (e.g., psychological treatment and antidepressants) may help reverse these findings.

Clinical Applications

The concept of FAPS as a dysregulation of central nervous system–enteric nervous system (CNS–ENS) function at varying levels of the neuraxis rather than a psychiatric or structural gastrointestinal disorder allows for an explanation...
of chronic pain due to enhanced pain perception: (1) by activation of silent nociceptors; (2) by dorsal horn transmission of impulses stimulated by release of cytokines or other substances; or (3) by chronic or frequently recurring psychosocial stresses that influence central pain modulation. This understanding, by linking psychosocial factors to the pathophysiology of chronic abdominal pain, alters the therapeutic approach from one being purely psychiatric in nature to other forms of therapy.

Given that FAPS is not associated with visceral disease or observable dysmotility, the role for visceral sensitivity in explaining the condition is still conjectural but is supported by clinical observation of chronic pain evolving from injury or inflammation of the bowel, and it helps explain why patients report intestinal pain after normal (subthreshold) activities such as eating a meal. It still needs to be scientifically determined if and how chronic pain results from sensitivity due to multiple operations, previous dysmotility (leading to increased gut wall tension), or abusive trauma of the vagina or anus in children (leading to abdominal, pelvic, or rectal pain). Possibly early pharmacologic treatment, either peripherally (via afferent receptor antagonists) or centrally (via psychopharmacologic agents), or psychological treatment may prevent the development of a chronic pain syndrome.

**CLINICAL FEATURES**

Several clinical features characterize patients with FAPS, particularly as seen in referral gastroenterologic practices or medical centers.

**History of Symptoms and Behavior**

Typically, the patient who presents with FAPS is middle aged and usually female. The history is of 10 or more years’ duration, and the patient is often in distress at the time she or he is first seen. The pain is described as severe and often as the worst ever experienced. The pain may be generalized and diffuse, or, on some occasions, may be localized. It soon becomes clear that it is a central focal point in the patient’s life and that she or he will often say that life would be fine if “you would just take the pain away.” The pain may be described in emotional or even bizarre terms, for example as “nauseating” or “like a knife stabbing”; as constant and not influenced by eating or defecating; as one of several other painful symptoms; and as a continuum of painful experiences often beginning in childhood or recurring over time.

FAPS sometimes coexists with other diseases and disorders as pancreatitis, inflammatory bowel disease, or more particularly and more commonly, IBS, functional dyspepsia, and functional biliary tract pain. If such is the case, the clinician must determine the degree to which one or another of these conditions contributes to the FAPS by stimulation of peripheral nociceptors and how much is the result of the effect on the role of the CNS responding to psychological and social stresses. Typically, when pain is related to stimuli derived from structural visceral/organic disease or disorder, the pain is likely to be more recent in onset, more variable or intermittent in intensity, more precise in location and conforming to neuroanatomic pathways, more responsive to antimotility agents and/or peripherally acting analgesics (e.g., NSAIDs), and related to events that affect gut function. Frequently, FAPS will evolve in a patient who has had a well-defined gastrointestinal disorder but who has been operated on one or more times and, following these operations, has developed chronic abdominal pain. Reoperations in such individuals are common and are performed for alleged intestinal obstruction due to adhesions.

**Psychosocial Features of the History**

Although patients with FAPS show no consistent psychological pattern, most have psychiatric diagnoses of anxiety, depression, and somatization. They may deny or minimize a role for psychological factors, possibly learning in childhood that attention is received with reporting of illness but not emotional distress. A history of unresolved losses, including the death of a parent or spouse, surgery such as hysterectomy or ostomy, or abortion or stillbirth is a common feature. Symptom exacerbations frequently develop soon after these events and recur on their anniversary, during the Thanksgiving-Christmas season, or when the physician goes on vacation.

A history of sexual and physical abuse is now frequently noted among patients with gastrointestinal as well as other chronic medical disorders. A history of abuse, no matter what the underlying diagnosis is, predicts poor health, refractoriness to medical care, increased diagnostic and therapeutic procedures, and more health care visits. Possibly, these trauma lead to an increased awareness of bodily sensations and a lowered pain threshold via central mechanisms. Because patients do not usually volunteer an abuse history, physicians should inquire about this possibility, particularly among those with chronic, unexplained, or refractory symptoms.

Finally, patients with FAPS report poor social networks and exhibit ineffective coping strategies. They feel unable to decrease their symptoms and may catastrophize, that is, view their condition in pessimistic and morbid ways without any sense of control over the consequences. These beliefs and cognitions are associated with greater pain scores and poorer clinical outcomes that lead to a cycle of more illness reporting, higher pain scores, more psychological distress, and poorer clinical outcomes. For many, the illness provides social support via increased attention from friends and family and their relationships with physicians.

**Patient Behavior**

Certain behavioral traits are common among patients with FAPS. Often these patients demand that the physician not only diagnose the problem but relieve it rapidly. They likewise deny relationship of their problem to psychologically disturbing issues and often attribute depression to pain rather than recognizing it as a primary force in their situation. An accompanying spouse or parent who takes responsibility for reporting the patient’s history to the physician is frequent, and it suggests the possibility of family dysfunction or “enmeshment” that might require future specialized counseling. A history of use of analgesics and even narcotics is not
Emphatically, these patients have the symptom criteria for the patient's pain is not the result of a specific disease. For occult blood (e.g., cutaneous nerve entrapment, hernia) or a CNS contribution to the pain (i.e., hypervigilance to body contact), whereas a negative test would be consistent with a visceral contribution to the pain.

Physical Examination

During the physical examination, patients with FAPS may not exhibit autonomic arousal. In fact, the presence of tachycardia, diaphoresis, and blood pressure changes suggests a more acute peripheral source for the pain. The presence of multiple surgical scars without clearly understood indications might suggest pain behaviors leading to unneeded surgical procedures. The “closed-eyes sign” may be noted. When the abdomen is palpated, the patient with FAPS may wince with eyes closed, whereas those with organically caused acute abdominal pain keep their eyes open in fearful anticipation of the examination. Often the “stethoscope sign,” that is, gentle, distracting compression on a painful site of the abdomen with the diaphragm, elicits no response or only a minimal one and thereby affords a more accurate appraisal of the complaint. In patients with abdominal pain, which is usually but not always more acute than chronic and is associated with disease of the viscera or peritonium, compression and change in body position often increase the perceived discomfort (see Chapter 4). To try to distinguish visceral or somatic pain from central hypervigilance, Carnett's test can be performed: After the site of maximal abdominal pain is identified, the subject is asked to assume a partial sitting position, thereby flexing the abdominal musculature. A positive test (i.e., increased pain) would suggest a muscle wall etiology (e.g., cutaneous nerve entrapment, hernia) or a CNS contribution to the pain (i.e., hypervigilance to body contact), whereas a negative test would be consistent with a visceral contribution to the pain.

DIAGNOSIS AND DIFFERENTIAL DIAGNOSIS

A complete history, thorough physical examination, and appropriate attention to psychosocial factors in the patient’s life during the first encounter often point the physician clearly to the diagnosis of FAPS. A physical examination that does not betray evidence of other chronic intra-abdominal conditions (such as chronic pancreatitis and serious biliary tract disease), as well as a battery of routine laboratory tests (including complete blood counts, liver function tests, serum electrolytes, renal function, and examination of stools for occult blood) that are normal, rather clearly indicates that the patient’s pain is not the result of a specific disease. Emphatically, these patients have the symptom criteria for FAPS (see Table 5-2), and recognition of these criteria along with the failure to find evidence of other causes for the pain listed in Table 5-1 and discussed elsewhere in this text should lead the physician to make the diagnosis (see Chapter 4). If the features of FAPS are absent or atypical, and if there are abnormalities on physical examination (e.g., abdominal mass, enlarged liver) or in screening laboratory studies (e.g., anemia, high erythrocyte sedimentation rate, low serum albumin level), another diagnosis in Table 5-2 should be strongly considered and pursued accordingly. It is also common for nonspecific abnormalities to be found (e.g., cyst in the liver, palpable lymph node, “nonspecific” bowel gas pattern), so efforts must be made to determine their relevance to the symptoms that the patient presents.

TREATMENT

Establishing a Successful Patient-Physician Relationship

Once other diagnoses have been safely ruled out and the relationship of the patient’s symptoms to the possible concomitant presence of other functional gastrointestinal disorders has been taken into consideration, a successful relationship with the patient with FAPS is the most important contribution to effective management. Several factors must be immediately taken into account to help establish this relationship and to move toward successful treatment. Important among these is an accurate assessment of the patient’s psychosocial situation. The physician must take into account the importance of the length of the history of illness, because long histories of complaints of pain and frequent visits to physicians or other health caregivers predict a poor outlook. One must determine the reason for the current visit and deal with it first. Is it because of concern for a serious illness? Is there an environmental stress? Has there been a dramatic worsening of the pain? Is the patient really seeking narcotics, a disability claim, or some other type of legitimization of illness? Has the psychiatric disturbance been exacerbated? A knowledge of prior traumatic episodes in the patient’s life will be important in future treatment. Some appreciation of the degree of the patient’s understanding of the illness is also important. This is particularly true for the success of a mutual treatment plan.

Answers to other questions must be sought early in the relationship, such as whether there are abnormal illness behaviors. What is the impact of the pain on the quality of life? Is there an associated psychiatric diagnosis? And finally, but extremely important, what is the role of the family or culture in the FAPS suffered by the individual? Usually family experiences with illness lead to emotional support and a focus on recovery. With dysfunctional family interactions, stresses are not well managed, and diverting attention toward illness serves to reduce family distress. Dysfunction is seen when spouses or parents overwhelm the patient, assume undue responsibility in the management, or become the spokesperson for the patient when communicating anger. If such family dysfunction is observed, counseling should be offered as a way to help the family develop better coping strategies.

Cultural belief systems also affect how the patient is presented to the physician and how the patient responds to treatment. For example, open recognition of psychological difficulties may be a stigma for Asians, but physical symptoms may be socially sanctioned. Further, patients may not comply with treatments that are not consistent with their cultural beliefs. In all of this it is important to gain a knowl-
edge of the patient’s psychosocial resources, that is, the availability of social networks such as family church, recreation clubs, and community organizations and effective coping strategies that may help to buffer the adverse effects of stress and improve the outcome.  

The physician should embark immediately on a course that will not only gain this information but that will also begin to establish rapport with the patient.  

Empathy is primary in this matter, because it acknowledges the reality and distress associated with the patient’s pain. It is accomplished by understanding the patient’s experience while maintaining objectivity. It does not mean over-reacting to the patient’s wish for rapid diagnosis and help by overmedicating or performing unneeded diagnostic studies. Diagnostic decisions are based on objective data rather than the patient’s insistence that “something be done.”

Education of the patient to the condition is provided by eliciting his or her knowledge of the syndrome, addressing any unrealistic concerns, explaining the nature of the symptoms in a fashion that is consistent with the patient’s belief system, and ensuring the patient’s understanding in all matters. It helps to explain that chronic abdominal pain is a true disorder relating to abnormal sensations and/or dysregulation of neuroenteric function and that it can be modified effectively by psychopharmacologic or psychological treatments that affect the regulation of pain.

All patients need reassurance, because they may fear serious disease or surgery. After the evaluation is completed, the physician should respond to the patient’s worries and concerns in a clear and objective manner, and then both must negotiate the treatment. The physician should ask about the patient’s personal experience, understanding, and interests in various treatments and then provide choices rather than directives. A recommendation will be more accepted if the patient understands the reasons it is made and believes it will help. Finally, the physician in a busy practice must set reasonable limits in time and effort and recognize that the patient’s care frequently needs to be shared with mental health professionals. Patients’ demand for more time is best handled by scheduling brief, but regular, appointments of fixed duration. The key to success is to maintain an ongoing relationship and proper boundaries.

Instituting a Treatment Plan

Successful treatment rests on a plan that encompasses ongoing interviews to ensure that the patient should not expect a cure. Using an example such as arthritis, the physician can explain that a realistic treatment goal is to improve daily function. Patients must be made to increase their responsibility for the illness, and to this end they are asked to keep a diary of symptoms for a few weeks, particularly identifying the circumstances of the episodes of pain and their emotional and cognitive responses. This technique not only helps the patient to achieve insight into aggravating factors but also characterizes the patient’s style of coping with the problem. Such information helps identify a strategy for behavioral treatment. Further, treatment is based on the severity of symptoms and the degree of disability and is conservative. Symptoms that are intermittent or of moderate severity, or exacerbations that are clearly linked to psychological distress, are frequently amenable to psychological treatment. However, if the pain is continuous and severe, psychoactive medications for central analgesia (e.g., tricyclic antidepressants [TCAs] such as amitriptyline or selective serotonin reuptake inhibitors [SSRIs] such as fluoxetine) may be helpful (see later).

MEDICATIONS. Most analgesics (e.g., aspirin, NSAIDs) offer little benefit, because they act peripherally. Narcotics should not be prescribed because of the potential for addiction and the narcotic bowel syndrome, where chronic use of narcotics leads to impaired motility and increased pain sensitivity. Furthermore, their use subordinates the development of treatment strategies to that of providing medication. Benzodiazepines are of limited value because they can be abused and they may lower the pain threshold. TCAs or SSRIs can be helpful in treating chronic pain, FAPS, and other painful functional gastrointestinal disorders. Because there are few data about which drug category is more effective, the choice depends on the patient’s tolerance of their side effects. In general, the less expensive TCAs have been shown effective but produce more anticholinergic effects, hypotension, sedation, and cardiac arrhythmias and may require some dose adjustment to achieve an optimal effect. They can be given in doses lower than used to treat major depression (e.g., desipramine, 25 to 100 mg/day at bedtime) and to reduce side effects. However, dose ranging up to full therapeutic levels may be needed, particularly if there is psychiatric comorbidity. The SSRIs may cause agitation, sleep disturbance, vivid dreams, and diarrhea but are much safer if taken as an overdose. In most cases a single pill (e.g., 20 mg of fluoxetine, paroxetine, or citalopram or 50 mg of sertraline or fluvoxamine) will suffice. Although the efficacy of SSRIs for pain control is not well established, this class of drug has additional benefits because they are anxiolytic and are helpful for patients having other comorbid conditions, including social phobia (or agoraphobia), post-traumatic stress disorder, panic, and obsessive thoughts related to their condition.

The physician should explain that antidepressants are central analgesics, not simply drugs for psychiatric conditions, and that they effectively treat other painful medical conditions such as migraine, postherpetic neuralgia, and diabetic neuropathy. They increase the release of neurotransmitters that descend from brain centers to block pain transmission from the gut to the brain, and the dosage is usually lower than used for psychiatric disorders. The lag time for effect may take several weeks; most side effects diminish after a few days and can be reduced by temporarily lowering the dosage or implementing other strategies such as offering other medications to reduce symptoms. The medication can be given for a few weeks and then the patient is reevaluated.

MENTAL HEALTH REFERRAL. Patients may be reluctant to see a psychologist or psychiatrist because they lack knowledge of the benefits of referral, feel stigmatized for possibly having a psychiatric problem, or see referral as a rejection by the medical physician. Psychological interventions are best presented as ways to help manage pain and to reduce the psychological distress of the symptoms. The medical visits should continue with the psychological treatment either regularly or occasionally for reassessment, or as needed, but with continuing availability.
SPECFIC PSYCHOLOGICAL TREATMENTS. The mental health consultant may recommend any of several types of psychological treatments for pain management. Cognitive-behavioral treatment, which identifies maladaptive thoughts, perceptions, and behaviors, is popular, and this information is used to develop new ways to increase control of the symptoms. Stress management is usually done in small groups where education and relaxation techniques are provided. Dynamic or interpersonal psychotherapy is a method that proposes psychological distress and physical symptoms are exacerbated by difficulties in interpersonal relationships. As difficulties emerge during the therapy, efforts are made to understand and address them, often improving the pain symptoms. Hypnotherapy has been investigated primarily in IBS, where the focus is on “relaxation of the gut.” Relaxation training attempts to help counteract the physiologic effects of stress or anxiety.

MULTIDISCIPLINARY PAIN TREATMENT CENTERS. Multidisciplinary pain treatment centers provide comprehensive rehabilitation of patients with chronic pain. The approach is theoretically rational, and it may be the most efficient method of treating disability from refractory chronic pain symptoms.

REFERENCES