CHAPTER 39

Pain Management

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- Patients suffering with chronic diffuse pain who lack objective clinical and laboratory findings (e.g., fibromyalgia) frequently are dismissed as not having real pain, which only perpetuates their illness.
- There are four principal categories of pain: nociceptive pain, neuropathic pain, chronic pain of complex etiology, and psychogenic pain.
- Pain assessment should include attention to possible psychological and sociocultural factors that could be contributing to the pain experience.
- Diagnostic waffling, the ordering of frightening tests, excessive use of physical therapy modalities, activity limitation after minor trauma, and overly liberal work release are among the important factors that can convert what should be a self-limited acute pain condition into a chronic pain syndrome.
- If the clinician suspects fibromyalgia, validation of the patient's pain is important.
- Pharmacologic agents that may be useful in the management of individual pain syndromes include nonsteroidal anti-inflammatory drugs, opioids, muscle relaxants, antidepressants, antiepileptic medications, and topical agents.
- Physical therapy, cognitive–behavioral therapy, aerobic exercise, and complementary and alternative medicine approaches may all be useful in the management of pain in selected patients.

All too often in office-based practice, treatment of pain is secondary to diagnosis and treatment of the disease state. This is unfortunate because pain, especially chronic pain, is among the most disabling and costly medical problems in Western countries (1). Patients suffering with chronic diffuse pain who lack objective clinical and laboratory findings (e.g., fibromyalgia) frequently are dismissed as not having "real" pain, which only perpetuates their illness. Presence of pain should be specifically sought and evaluated in all patients and, if present, relief of pain should be a primary focus of the physician's efforts. Indeed, pain should be addressed as a *disease entity,* not as a sensory entity (2).

NATURE OF PAIN

A useful definition adopted by the International Association for the Study of Pain (IASP) defines pain as "an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage" (3). Neurophysiologically, pain is a complex sensation–perception interaction involving the simultaneous processing of nociceptive input from the spinal cord. This input activates a central network that records the pain experience in multiple regions of the brain (Figure 39-1).

In addition to strictly sensory discriminative elements of nociception and afferent input from somatic reflexes, there are major contributions from pathways and regions of the brain concerned with emotional, motivational, and cognitive aspects of pain. These factors influence the subjective unpleasantness and distress associated with pain. The two principal effectors of the stress response, the hypothalamic–pituitary–adrenocortical axis and the sympathetic nervous system, are also activated. The stress response may become maladaptive in chronic pain syndromes such as fibromyalgia. Negative emotions (depression and anxiety), other negative psychological factors (loss of control, unpredictability in one's environment), and certain cognitive aspects (negative beliefs and attributions, catastrophizing) all can function as stressors with actions in these systems.

Pain Categories

There are four principal categories of pain: nociceptive pain, neuropathic pain, chronic pain of complex etiology, and psychogenic pain. *Nociceptive pain* is due to stimulation of peripheral pain receptors on thinly myelinated A delta and/or unmyelinated (C) afferents during inflammation or injury of tissues. The pain experienced generally "matches" the noxious stimulus. However, both peripheral sensitization (reduction in

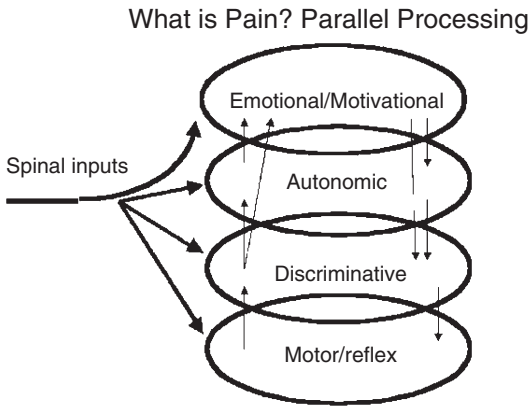

FIGURE 39-1

Pain signals from peripheral sites of tissue injury or inflammation are transmitted simultaneously to multiple areas of the brain through parallel processing. The pain *experience* derives from the combined input of these multiple brain areas. For example, a cognitive brain area gives *meaning* to the pain, which could be trivial (indigestion after eating a pizza) or very frightening (same epigastic pain input from the periphery, but unprovoked in a person recently diagnosed with stomach cancer). The latter pain experience would be much more distressing. Similarly, if a person is depressed, contributions from affective/motivational areas of the brain make the pain more distressing. (Courtesy of Alan R. Light, PhD.)

the threshold of nociceptor endings) and central sensitization [amplification of pain in the central nervous system (CNS)] can occur in "normal" nociceptive pain. These peripheral and central inputs may result in allodynia (an alteration in pain perception such that normally nonpainful stimuli, such as gentle touching, are perceived as painful) and hyperalgesia (increased pain response to a previously painful stimulus). In addition to systemic inflammatory or degenerative rheumatic diseases, nociceptive pain occurs as regional musculoskeletal pain in tenosynovitis, compressive neuropathies, nerve entrapment syndromes, bursitis, and various localized forms of arthritis. Usually self-limited with conventional treatment strategies, regional musculoskeletal pain may become chronic and disabling.

Both peripheral and central nervous system processes also play a role in *neuropathic pain*, which may follow injuries and diseases that directly affect the nervous system. There are three common types: peripheral neuropathic pain (e.g., postherpetic neuralgia, painful diabetic neuropathy, vasculitic neuropathy, radiculopathic pain due to injury to spinal nerve roots); central neuropathic pain (e.g., central poststroke pain, spinal cord injury pain); and cancer-associated neuropathic pain. Complex regional pain syndrome (reflex sympathetic dystrophy; RSD) is another neuropathic pain syndrome. Neuropathic pain may be paroxysmal, perceived as electric shock–like discomfort or burning. Neuropathic pain may be associated with hyperpathia (persistence after the stimulus has ended, spreading or worsening in crescendo fashion with repeated touching). Central sensitization and ectopic firing of peripheral neurons, either spontaneously or through mechanical forces developed during movement, contribute to this peculiar type of pain. Management may require special pharmacologic approaches, as discussed below.

Chronic pain of complex etiology occurs in fibromyalgia and a large number of substantially overlapping regional pain syndromes, such as migraine headache, temporomandibular disorders, irritable bowel syndrome, and atypical chest pain. In practice, the diagnostic label applied to illness in a given patient often depends on which medical specialist evaluates the patient first, for example, a rheumatologist might diagnose fibromyalgia, whereas a gastroenterologist would diagnose irritable bowel syndrome. Previously termed *functional pain syndromes* on the basis of absent structural pathology, these illnesses share very close relationships in terms of etiology and pathophysiology. Recent advances in the understanding of the psychophysiologic/neurophysiologic dysregulation in such illnesses is impelling a unifying reclassification as central sensitivity syndromes (4).

Collectively, central sensitivity syndromes constitute huge personal and societal burdens, but all too frequently such illnesses are not approached effectively by traditional medicine. In fibromyalgia, the prototype of this category, pain radiates diffusely from the axial skeleton over large areas of the body, involving muscles predominately. The patient describes the symptoms as "exhausting," "miserable," or "unbearable." Altered central nociceptive processing results in a decrease in the pain perception threshold and in the threshold for pain tolerance. The hallmarks of fibromyalgia—chronic widespread pain, fatigue, and multiple somatic symptoms—have both psychological and biological bases that derive, at least in part, from chronic stress and distress. Female gender, genes (5), adverse experiences during childhood, psychological vulnerability to stress, and a stressful, often frightening environment and culture are important antecedents. Thus, fibromyalgia and related syndromes should be viewed from a biopsychosocial perspective (6). A useful guideline for the management of fibromyalgia syndrome pain in adults and children has been published recently (7).

More purely *psychogenic pain* is seen in somatoform and somatization disorders and hysteria.

MANAGEMENT OF PAIN

General Approach

The first element in management of pain is accurate assessment and diagnosis of the cause of the pain.

Assessment should include attention to possible psychological and sociocultural factors that could be contributing to the pain experience. In addition, the physician should be aware that fibromyalgia frequently coexists with inflammatory disorders, such as rheumatoid arthritis (RA) and systemic lupus erythematosus (SLE). Diagnostic "waffling," the ordering of frightening tests, excessive use of physical therapy modalities and activity limitation after minor trauma, and overly liberal work release are among the important factors that can convert what should be a self-limited acute pain condition into a chronic pain syndrome. If one suspects fibromyalgia, validation of the patient's pain is important. Comments such as "it's all in your mind" serve only to perpetuate illness. To the patient, the pain is real. On the other hand, it is important to be aware of confounders to recovery, such as pending litigation or compensation claims.

For acute nociceptive pain (< 30 days duration), pharmacological interventions should follow a stepwise approach using non-opioid and opioid analgesics either singly or in combination, as indicated by pain intensity. Depending on the specific musculoskeletal disorder, initially conservative combinations of corticosteroid injections, activity modification, splints, counterforce bracing, local heat or cold, and in some cases, surgical procedures may be indicated for pain relief and/or to preserve function. Education about the nature of the underlying problem, limitations, and prognosis should err on the side of optimism. Whenever possible, rapid return to full activity and work is best.

If there is a significant nociceptive pain element, chronic pain (>6 months duration) may be managed pharmacologically with analgesics using the same stepwise approach outlined for acute pain. Especially important is a multifaceted treatment plan that incorporates various adjuvant medicines, exercise, and psychological and behavioral approaches to reduce distress and promote self-efficacy and self-management. For many regional chronic pain syndromes, referral to an experienced specialist who advocates holistic, nonsurgical approaches is recommended.

Assessment of Pain

Assessment of pain in the physician's office should be based on a biopsychosocial perspective, that is, in addition to identification of biological variables that contribute to pain, the recognition that psychological and sociocultural factors potentially amplify or perpetuate the pain experience (see Ref. 8 for a full discussion). Pain intensity should be measured with either a verbal or numerical rating scale or a visual analog scale. In fibromyalgia, the author finds it useful to determine pain detection threshold (normal = 4 kg/cm^2) at several tender point sites by pressure algometry. Pain behaviors such as guarding, rubbing, grimacing, and sighing vary inversely with patients' self-efficacy for control of chronic pain. A simple self-report form that incorporates validated scales for physical and psychological health status [the modified Health Assessment Questionnaire (HAQ)], visual analog scales for pain, fatigue, patient global self-assessment, a checklist of current symptoms, and scales for helplessness and cognitive performance can be completed in just a few minutes (9). Easily adaptable to a busy practice, such information is invaluable for the psychosocial assessment of pain and in monitoring response to therapy. Marital adjustment, perceived levels of social support, and current stressors in the patient's life are important topics for evaluation. The simple inquiry "how was your childhood?" often reveals adverse childhood experiences, such as abuse, that have increased the patient's vulnerability to chronic pain (10). In multidisciplinary settings, information obtained from the Minnesota Multiphasic Personality Inventory (MMPI), the Social Support Questionnaire (SSQ), the Sickness Impact Profile (SIP), and the Multidimensional Pain Inventory (MPI) is useful for more comprehensive assessments. Subgroups of patients with chronic pain can be identified in this way that can predict response to interdisciplinary therapeutic interventions (11).

Pharmacological Management of Pain

A useful stepwise approach for pharmacological interventions based on nociceptive pain intensity [e.g., in osteoarthritis (OA)] is illustrated in Figure 39-2. Low-dose opioids for patients with OA who fail acetaminophen + nonsteriodal anti-inflammatory drugs (NSAIDs) or a cyclooxygenase-2 (COX-2) inhibitor are effective when used as part of a multimodal approach to pain control, and also may have fewer potentially life-threatening complications (12). Reasonable guidelines for use of opioids in more severe musculoskeletal pain are the exclusion of patients with histories of substance abuse, concomitant attention to psychological and social perpetuators of pain, use of an opioid treatment contract, a one physician/one dispensing pharmacy rule, and close monitoring. Drug-seeking behavior (pseudoaddiction) may indicate that pain is not being controlled adequately.

Opioid Analgesic Drugs

Opioids bind to mu, kappa, or delta opioid receptors (predominately mu for analgesic effects) in regions of the brain involved in integrating pain and to pre- and postsynaptic terminals of peripheral sensory fibers, where they inhibit release of substance P and other

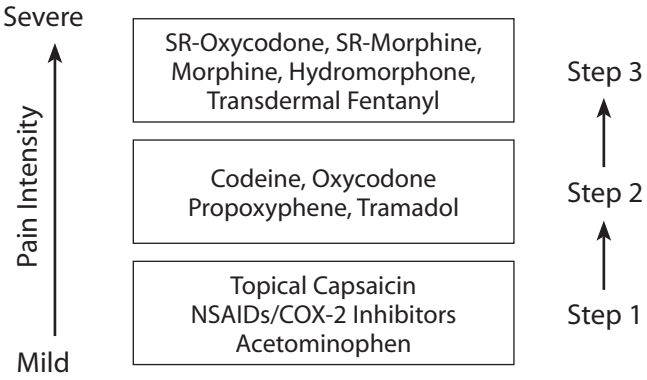

FIGURE 39-2

Stepwise approach to pharmacologic management of pain.

mediators. Tramadol also inhibits reuptake of norepinephrine and serotonin. Table 39-1 lists opioids commonly in use. The side effects of opioids include constipation, nausea and vomiting, sedation, cognitive impairment, miosis, myoclonus, urinary retention, and respiratory depression. Older persons are more sensitive to opioids with respect to both efficacy for pain relief and vulnerability to side effects; starting doses should be reduced 25% to 50%. In the great majority of patients with well-defined chronic rheumatic disease pain, opioids are effective, safe, and well tolerated. Several weeks or months are required to titrate opioid therapy in the outpatient setting. During opioid tapers, which require 2 to 3 weeks, clonidine (0.2–0.4 mg/day) is helpful in controlling withdrawal symptoms. Monitoring of patients taking analgesic medications requires frequent re-evaluation for efficacy and side effects during initiation, titration, dose changes, and maintenance therapy.

Muscle Relaxants

Centrally acting skeletal muscle relaxants, for example, carisprodol (Soma), cyclobenzaprine (Flexeril), metaxolone (Skelaxin), methocarbamol (Robaxin, Robaxisol), tizanidine (Zanaflex), and baclofen (Lioresal, Kemstro) to list a few, have modest benefit as adjunctive therapy for nociceptive pain associated with muscle strains. Used intermittently, or as a single bedtime dose (e.g., 10 mg cyclobenzaprine), they have limited effectiveness in fibromyalgia and other chronic pain syndromes, as well. Sedation and other CNS side effects occur frequently. Abuse may occur, particularly with carisprodol, and abrupt cessation may be associated with withdrawal symptoms.

Antidepressants

Tricyclic antidepressants (TCAs) clearly are effective in neuropathic pain and may be of modest short-term benefit in diffuse and regional chronic pain syndromes, but side effects (dry mouth, drowsiness, and weight gain) limit patient acceptance. Selective serotonin reuptake inhibitors (SSRIs), for example, fluoxetine (Prozac, 10–40 mg daily) or citalopram (Celexa, 20–40 mg daily) have been shown to have limited efficacy in randomized, controlled trials in fibromyalgia; the combination of a TCA with an SSRI in this disorder typically produces greater improvement in pain, sleep, and overall well-being than either drug used alone.

TABLE 39-1. OPIOID ANALGESIC DRUGS.

DRUGS	ORAL EQUIVALENT	STARTING DOSE	COMMENT
Short-acting			
Morphine sulfate (Roxanol)	30 mg	15–30 mg every 4 hours	For all, start low and titrate; begin bowel program early; most of these opioids are available in combination with acetaminophen or aspirin (do not exceed maximum dose). For all, short-acting opioid often is needed for breakthrough pain.
Codeine (Fiornal)	120 mg	30–60 mg every 4–6 hours	
Hydrocodone (Lortab)	30 mg	5–10 mg every 3–4 hours	
Oxycodone (Percodan)	20–30 mg	5–10 mg every 3–4 hours	
Hydromorphone (Dilaudid)	7.5 mg	1.5 mg every 3–4 hours	
Propoxyphene (Darvon)	100 mg	100 mg every 4 hours	
Tramadol (Ultram)	120 mg	50–100 mg every 6 hours	
Methadone (Dolophine)	–	15–60 mg every 8 hours	
Long-acting			
SR-Morphine (MS Contin)	30 mg	5–10 mg every 3–4 hours	
SR-Oxycodone (Oxycontin)	20–30 mg	10–20 mg every 12 hours	
Transdermal fentanyl (Duragesic)	Not available	See package insert	

Dual-action (serotonin/noradrenaline) reuptake inhibitors (SNRIs) such as venlafaxine (Effexor, 150–225 mg daily or duloxetine (Cymbalta, 30–60 mg daily) (13) are superior to SSRIs for pain control and improve many symptoms in fibromyalgia irrespective of comorbid depression. When concomitant depression is present in a chronic pain syndrome, it is essential that depression be treated aggressively. SSRIs and SNRIs must be tapered gradually upon discontinuation. Following the institution of therapy, patients should be observed for worsening of depression and the emergence of suicidal thoughts.

Antiepileptic Drugs

Carbamazepine (Tegretol) and a series of other new antiepileptic drugs have become first-line agents for neuropathic pain (14). Gabapentin (Neurontin, 900–1800 mg daily in three divided doses), a 3-alkylated gamma-amino butyric acid (GABA) analog originally introduced as an anticonvulsant and more recently released pregabalin (Lyrica, 150–425 mg daily in divided doses) (15) are useful in chronic pain states, including fibromyalgia, related syndromes, and various types of neuropathic pain. These agents may ameliorate associated depressed mood and anxiety, as well. The dose of these agents should be escalated slowly over weeks; discontinuance of these agents should likewise be done gradually.

Topical Agents

Obtained from red chili peppers, topical capsaicin binds to vanilloid receptors on peripheral terminals of nociceptive neurons, thereby inhibiting activation of the pain pathway by noxious stimuli. Essentially free of toxicity other than mild burning at the site of application, capsaicin is useful as adjunctive therapy in diffuse and regional musculoskeletal pain syndromes, joint pain in arthritis, and in neuropathic pain disorders. Up to three 5% lidocaine patches (Lidoderm) may be applied to localized painful areas for 12 to 18 hours in a 24-hour period with good efficacy and safety.

Other Pharmacological Agents

Anxiolytics, for example, clonazepam (Klonopin), lorazepam (Ativan), temazepam (Restoril), alprazolam (Xanax), and buspirone (Buspar) have antinociceptive effects in chronic pain and are often used in combination with antidepressants and antiepileptic drugs. Pramipexole (Mirapex, dopamine 3 receptor agonist) has been shown to improve pain scores, fatigue, and function in patients requiring opioids for pain control.

Pharmacologic Management of Fibromyalgia

Low-dose amitriptyline at bedtime, perhaps in combination with cyclobenzaprine (Flexeril), is a well-established therapy for fibromyalgia and related chronic pain syndromes. SNRIs in combination with gabapentin (Neurontin) or pregabalin (Lyrica) are very useful in patients with severe allodynia and hyperalgesia. Corticosteroids and NSAIDs are of no benefit in fibromyalgia pain per se, but are useful treatments for coexisting inflammatory processes ("pain generators"). Opioids should be avoided if at all possible for fibromyalgia pain. However, certain patients with extreme pain unresponsive to other agents will require opioids to improve quality of life and maintain function. If used, opioids should be combined with multidisciplinary approaches, psychotherapeutic interventions, and the cautions mentioned above.

Pharmacological and nonpharmacological treatment of poor sleep is crucial for improving overall sense of well-being. Sleep disturbances should be managed aggressively, beginning with instruction in the elements of good sleep hygiene, such as avoidance of daytime naps and caffeine. Most patients require medication, and many nonbenzodiazepine hypnotics, for example, zolpidem (Ambien), zaleplon (Sonata, 1-hour half-life, useful with mid-sleep awakenings), or eszopiclone (Lunesta) are now available in addition to traditional hypnotics. Oxybate (Xyrem) also is very promising. A formal sleep assessment may be required for patients who do not respond to the above measures.

Fatigue, often a dominant complaint in fibromyalgia, generally improves with effective treatment of pain, depression, and sleep disturbances in combination with a graded aerobic exercise program. Modafinil (Provigil, 100–200 mg q.a.m.) is beneficial in patients for whom overwhelming fatigue is a persistent complaint, and may be useful as a bridge therapy during the early phase of an aerobic exercise program.

Psychological and Behavioral Approaches

The importance of strategies in this area has been emphasized recently (8,16). Depression, anxiety, stress, sleep disturbance, pain beliefs and coping strategies, and self-efficacy all are central to the pain experience in many patients, and frequently determine the outcome of chronic pain. Unless psychosocial and behavioral variables are recognized and approached, strictly pharmacological interventions to reduce nociceptive pain

from inflammation or the diffuse pain in fibromyalgia and related syndromes are of limited benefit. Established behavioral treatments in RA and OA that improve ratings of pain or pain behavior are the Arthritis Self-Management Program (17) and cognitive–behavioral therapy (cognitive–behavioral therapy includes components for education, training in relaxation and coping skills, rehearsals of the skills learned, and relapse prevention). Self-care education and telephone counseling are probably efficacious in OA, but have not been studied in RA. The role of cognitive–behavioral therapy for the diffuse pain of fibromyalgia and related regional pain syndromes remains to be clarified.

Physical Therapy/ Physical Modalities

The objectives here are to diminish pain, improve function, minimize disability, and promote self-efficacy. Although certain strategies and modalities are clearly beneficial, this area needs properly designed trials to establish efficacy.

Exercise

In addition to positive effects on underlying pathological processes in bones, joints, and muscles, exercise is essential to the treatment of fibromyalgia and related chronic pain syndromes. The benefits of exercise, in addition to gains in cardiovascular fitness, muscle tone, and strength, include improvements in both subjective and objective measures of pain and in the overall sense of well-being. Many patients with chronic pain perceive their muscles to be weak and easily fatigued, and bear the negative belief (fear) that activity will exacerbate their condition. Consequently, exercise is avoided and their muscles become deconditioned. Normal activities become challenging. Excessive activity "on a good day" induces a major flare of pain and fatigue, possibly due, in part, to the peripheral and central effects of proinflammatory cytokines (tumor necrosis factor, interleukins 1 and 6) released in response to exercise-damaged myofibers. Ideally, exercise should be low-impact (walking, water aerobics, stationary bicycle, rather than running), beginning very gently and progressing gradually to endurance and strength training. Encouragement and positive reinforcement can reduce the virtually universal problem of poor compliance. Obesity, poor posture, and overloading activities at work and home also contribute to muscle pain and fatigue and should be addressed. Daily stretching exercises after hot showers are very helpful.

Heat and Cold

Heat (hot packs, paraffin, hydrotherapy in its many forms) is of proven benefit in nociceptive pain, especially when combined with exercise (range of motion, stretching, strengthening). Diffuse and regional pain is improved by such strategies as sauna, hot baths and showers, and hot mud. While not superior to superficial heat, cold (cold packs, immersion, or vapocoolant sprays) may provide more immediate analgesic benefit, particularly when applied soon after an injury.

Massage, Trigger Point Injections, Acupuncture, and Transcutaneous Electrical Nerve Stimulation

Gentle massage is well received by patients with diffuse pain syndromes, but as a totally passive modality it fails to promote self-efficacy for control of pain. Injection of "trigger points" is of short-term benefit only and should generally be avoided. Neurophysiologic effects of acupuncture and electroacupuncture include release of opioids and other mediators in the nervous system. Several randomized, controlled trials have shown acupuncture to improve subjective pain and to raise pain thresholds, but its long-term benefit in chronic pain syndromes remains unclear. An advantage of transcutaneous electrical nerve stimulation (TENS) for localized musculoskeletal pain is that the patient can apply this modality at home.

Complementary and Alternative Medicine

The immense popularity of complementary and alternative medicine (CAM) today contrasts with the current paucity of data regarding the biochemical nature and mechanism of action of most alternative remedies and the lack of rigorous studies addressing efficacy, safety, and cost-effectiveness of these strategies. Many physicians lack knowledge in this area and may be overtly hostile toward CAM. Consequently, patients are reluctant to inform their physicians about CAM they are using for self-management. This can be dangerous because of unsuspected drug interactions. Patients with chronic pain and fatigue, for example, fibromyalgia, are the largest users of CAM, often because of frustration with the inefficacy of traditional medicine and lack of empathy and understanding on the part of many physicians. Until neuroscience, behavioral science, and health care systems advance to such a point that effective biopsychosocial treatment strategies are applied in most patients with chronic pain, CAM approaches will continue to proliferate. In the meantime, a practical

approach is to inquire about CAM usage, refrain from expression of negative opinions if a particular CAM treatment is relatively inexpensive and appears to be safe, and to encourage "whatever works" in the context of the power of the placebo effect and the promotion of self-efficacy for control of pain.

Pain in Children

Except for children less than 1 year of age, the approach to the management of pain in children is similar to that in adults. Issues meriting particular attention include the young child's inability to report pain and fear (e.g., of doctors and needles), age-related pharmacological factors, and psychosocial variables that differ from those in adults (e.g., school absenteeism). Although clinically significant pain often is not fully recognized and treated, recurrent complaints of pain all over the body are common in otherwise healthy children. In such cases, the physician must be sensitive and wise, avoid unnecessary testing, and emphasize lifestyle interventions, reduction of school stressors, and aerobic exercise (see Ref. 18 for a useful review).

Pain in Older Persons

Pain, particularly musculoskeletal pain, is very common in older persons and is neither part of normal aging nor better tolerated than in younger persons. Those misconceptions contribute importantly to the unfortunate undertreatment (or lack of treatment!) of chronic pain in the elderly in both community and institutional settings. Indeed, in a study of nursing-home residents, 71% had at least one pain complaint and two thirds had constant or daily pain, but only 15% had received analgesic medication in the previous 24 hours (19). The experience of pain in older persons differs somewhat from that in young and middle-aged individuals: higher pain thresholds, less frequent self-report of pain, atypical presentation of pain (e.g., as confusion, restlessness, or other behavioral change), less prominent anxiety associated with the pain, and frequent coexisting depression. Older persons exhibit lower self-efficacy and tend to use passive coping strategies (e.g., praying and hoping) rather than cognitive coping methods. Their susceptibility to associated impairments is greater.

The American Geriatrics Society has published clinical practice guidelines for the management of chronic pain in older persons (20). Special barriers to accurate pain assessment in this population include reluctance to report pain, use of atypical descriptors of pain, fear of diagnostic tests and medications, and communication difficulties due to sensory and cognitive impairments. With respect to pharmacologic therapy in the elderly, goals, hopes, and tradeoffs should be discussed openly. For mild pain, acetaminophen alone or in combination with celecoxib (Celebrex) is useful. The use of opioids for moderate or severe pain is appropriate, but dosing should follow the "start low, go slow" maxim. The health care provider must be aware of economic barriers that some elderly patients confront in obtaining medications. Nonpharmacologic treatment of pain in older persons should be an integral part of care plans.

Procedure-Based Pain Management

Injection of local anesthetics, epidural techniques, and radiofrequency ablation procedures all have a place in certain cases, but not infrequently are used inappropriately in anesthesia pain clinics. Furthermore, risk–benefit and long-term efficacy of such approaches in chronic diffuse pain have not been fully established.

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