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Management of Chronic Pain in the Acute Care Setting

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The management of chronic nonmalignant pain is challenging in any setting. Chronic pain is commonly associated with a complex interaction of physical, psychological, and social components. Consequently, the evaluation and management of chronic pain requires a long-term relationship and an investment of time beyond what is normally available to acute care physicians. Opioid medications are commonly used in the treatment of chronic pain, but they can further complicate the management of chronic pain, possibly worsening pain through N-methyl-D-aspartate (NMDA)-receptor mediated tolerance and descending pain facilitation. Moreover, opioids are intrinsically self-reinforcing, which can cause them to be taken compulsively even as they contribute to the worsening of the patient's condition.

The purpose of this article is to describe the unique pathophysiology of chronic pain, the function and limitations of opioids, alternative treatments, and strategies through which acute care physicians can participate in the care of chronic nonmalignant pain patients in ways that contribute positively to their long-term management.

Definition of chronic pain

The American Chronic Pain Association defines chronic pain as that which "continues beyond the usual recovery period for an injury or an illness. It may be continuous or come and go" [1]. Other definitions include "persistent or episodic pain of a duration or intensity that adversely affects the function or well being of the patient, attributable to any nonmalignant etiology" [2], and "pain and disability far out of proportion to the

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peripheral stimulus,'' with significant psychological factors [3]. Severe pain from acute injuries, including fractures, rarely persists longer than 2 weeks. When patients present with complaints of severe pain beyond this time period, then one should consider the possibility of a developing chronic pain syndrome [4].

Prevalence

At the emergency department of an urban teaching hospital, approximately 11% of the patients presented for treatment of chronic pain [5]. A Gallup survey found that 89% of Americans age 18 and older have pain at least once a month; 15% of them have severe pain monthly. Among people aged 65 and older, 55% have pain daily [6]. ''Chronic pain,'' by these criteria, is a ubiquitous condition that will affect the majority of the population at some time. The implication of this is that any missteps in the treatment of chronic pain are likely to affect many millions of people, with profound consequences.

Special biological considerations in chronic pain

The function of normal pain is to influence us to protect ourselves from injury. Congenital or acquired insensitivity to pain results in destructive processes such as the Charcot joint [7].

The majority of people have similar pain thresholds; however, it has been shown that the threshold of pain can be increased 50% in most people through acupuncture, relaxation, hypnosis, or imagery [8]. Pain tolerance also varies, and appears to have a genetic component [9]. Some patients are simply less willing or able to cope with moderate levels of pain [10]. A numerical level quantifies how much pain patients tell us they feel, but does not quantify the intensity of noxious physiologic stimulus.

The perception of pain can be divided into three components: sensory, affective (how it affects you emotionally), and evaluative (what you think about the pain and what it means to you) [11]. The pathophysiology of acute pain is covered in the article by Fink elsewhere in this issue.

Chronic pain pathways

There are different kinds of chronic nonmalignant pain with different etiologies. Pain may be felt from stimuli that are not normally painful, referred to as ''allodynia.'' Alternatively, a patient may feel a level of pain that is out of proportion to the level of noxious stimulus, referred to as ''hyperalgesia.'' Both of these are present in many chronic pain syndromes [12].

Pain secondary to nerve injury, sometimes referred to as ''causalgia'' [12], occurs when damaged nerves can fire spontaneously, producing neuropathic pain. When large-diameter sensory fibers are damaged, the patient may

experience numbness, whereas small-diameter C-fiber nociceptors can still transmit pain [13]. This accounts for the paradoxical complaint of feeling both numbness and pain at the same site.

Deafferentation, or the loss of sensory input from afferent nerves, can produce pain. Normal afferent fibers inhibit pain transmission by inhibiting second- and third-order neurons in the spinal cord. Injury to these sensory afferent fibers and the loss of sensory input can result in increased irritability and firing of second- and third-order neurons up the chain [12–14]. After an amputation, deafferentation causes remapping to occur in the subcortical areas of the brain, resulting in "phantom limb pain" [15].

Complex regional pain syndrome (CRPS) is an uncommon type of chronic pain. In Type I CRPS, there is no known nerve injury. Type II CRPS is associated with a known nerve injury. Subsets of each type have been referred to as "sympathetically maintained," because of apparent improvement with a sympathetic block [16], although some studies have shown the efficacy of sympathetic blocks to be no better than placebo [17]. A review of 145 cases [18] found no correlation between pain and autonomic dysfunction, and 41% of the cases had a history of chronic pain before they developed CRPS.

It has been shown that inactivity produces concurrent myofascial pain [12], and that prolonged immobilization is a predisposing factor for Type I CRPS [16]. Some patients, in response to injury or discomfort, are unwilling to move the extremity or tolerate any stimulation of it. One may speculate that they are effectively self-deafferenting, and that they are contributing to the development of a chronic pain syndrome. This may in part explain the effectiveness of physical therapy in some chronic pain syndromes, or the effectiveness of vigorous sensory stimulation of the affected part in complex regional pain syndrome in children [19].

Central sensitization and N-methyl-D-aspartate receptor activation

Central sensitization, or "wind up," has been implicated in the development of chronic pain syndromes. Central sensitization itself is a normal physiologic response to repetitive noxious stimulation. Following the persistent or large-scale activation of alpha-amino-3-hydroxy-5-methylisexazole-4-propionic-acid (AMPA) receptors, a second glutamate receptor, designated as the NMDA receptor, depolarizes the cell, opens the NMDA-associated Na+ and Ca++ channels, and raises the intracellular Ca++ level [20–22]. This process is mediated by protein kinase C [23].

NMDA receptor activation also induces the synthesis of nitric oxide (NO) which diffuses back to nociceptors. There, NO causes the release of the peptide neurotransmitter substance P, which binds neurokinin-1 (NK-1) receptors in the spinal cord, and amplifies the pain signal [21].

Presumably, central sensitization exists to force us to pay attention to whatever is causing the repetitive noxious stimuli, and to alter our behavior.

At this stage it is not pathological, and is not chronic pain; however, persistent activation of NMDA-receptors results in persistent substance P release, which stimulates nerve growth and regeneration within the spinal cord. The remodeling and the formation of new connections with other nerves produces "allodynia." Persistent NMDA-receptor activation may also cause apoptosis (loss) of neurons, resulting in further remodeling [21].

It is important to note that the new neural connections that are formed lack mu-receptors, making them resistant to opioid pain relievers; however, drugs that antagonize NMDA-receptors, such as ketamine, phencyclidine, dextromethorphan, and methadone, can effectively relieve chronic pain [21]. Some anticonvulsant drugs, even though they lack anesthetic properties, are often effective in treating many kinds of chronic pain, particularly neuropathic pain [24]. Because of the different pathophysiology, opioids frequently produce little or no analgesia in chronic pain.

Antinociceptive response

Our endogenous pain-inhibitory antinociceptive response system is a normal part of how our bodies process a pain signal, modulating the transmission of the signal and its effect on the brain. It exists to override the pain stimulus on order to allow vital survival functions such as fighting, escaping, or even hiding ("fight, flight, or hide") in the presence of injury. It is an important and often overlooked aspect of the perception and treatment of pain, particularly in chronic pain, in which psychological and physiological factors may interfere with antinociceptive function. Chronic opioid use also may compromise the antinociceptive response.

The system consists of several parts. Wide dynamic range (WDR) neurons in the dorsal horn can be inhibited by noxious stimuli in noncontiguous areas of the body, contributing to the "gate" response [20]. This may contribute to the mechanism by which rubbing an area after injury or pinching the skin at a distant site reduces the pain from that injury. Vagus and sympathetic nerve stimulation also decreases pain [21].

The endogenous opioids are a very important part of the antinociceptive response, and are particularly relevant as they function at the site of action of opioid medications. Endogenous opioids bind receptors coupled to G proteins on their target cells. Receptor activation decreases the influx of calcium and inhibits adenylate cyclase, resulting in decreased activity of protein kinase A. Receptor activation also alters gene expression and the phosphorylation or other proteins [25].

There are three different types of endogenous opioids: endorphins, enkephalins, and dynorphin.

Endorphins are released in the periaqueductal gray matter of the brain. Beta-endorphin is the most potent endogenous opioid [26]. Endorphins bind mu-opioid receptors on presynaptic terminals of nociceptors and postsynaptic surfaces of dorsal horn neurons, and inhibit the propagation of

pain signals [21]. Mu-receptors are in the highest concentration in the brain. Although mu-receptors are present in the spinal cord, activation of mu-receptors is mostly involved with analgesia above the level of the spinal cord [26]. Mu-receptor activation produces both analgesia and euphoria [27].

Enkephalins are involved in spinal analgesia [26]. They are released in the nucleus raphe magnus of the brain stem. They bind delta-opioid receptors on inhibitory interneurons in the substantia gelatinosa of the dorsal horn, causing the release of gamma-aminobutyric acid (GABA) and other substances, dampening pain signals in the spinal cord. Delta-receptors are stored in the same presynaptic vesicles with the neurotransmitters, so that when the neurotransmitters for pain are released, delta-receptors are simultaneously incorporated into the presynaptic cell membrane. This produces increased sensitivity to the antinociceptive effects of enkephalins at the same time the cells are transmitting a pain signal [21], demonstrating the tight linkage between pain signal propagation and modulation.

Following the release of enkephalins, other pain signal inhibitory molecules, including norepinephrine, oxytocin, and relaxin, are released from spinal cord cells [21].

Dynorphin, released from spinal interneurons, also produces spinal analgesia. It activates Kappa-receptors, leading to the closure of N-type calcium channels in the spinal cord cells that normally relay pain signals to the brain [21]. Kappa-receptor activation reduces pain with less respiratory depression than mu-receptor activation, and less dependence [28], but may also be pronociceptive [29]. Kappa-receptors are the site of action of opioid agonist-antagonists [26].

Within the medulla, there are ''on-cells'' that augment the transmission of pain, and ''off-cells'' that block it. On-cells are activated by the anticipation of pain even before it is painful. Morphine inhibits on-cells and produces continuous firing of off-cells, inhibiting the transmission of the pain stimulus signal. Giving naloxone or withdrawing morphine produces activation of on-cells to a level higher than before morphine was given, causing rebound pain [11].

Physiologic and painful stressors induce an increased tolerance of pain. Congenital differences in this stress-induced analgesia system and the endogenous opioid system appear to account for part of the genetic variation in pain tolerance [9].

While in a state of central sensitization, endorphins and exogenous opioids are less effective [21]. There are also systems within the brain that modulate the antinociceptive response and appear to contribute to the tolerance that develops to opioid medications. This counter-antinociception exists to keep pronociceptive and antinociceptive processes in balance [29]. Presumably, this functions to protect us from further injury.

It is important to note that the endogenous opioid system is only part of the neuromodulary system. Although some pain does not respond well to opioids, it may respond to other measures [28].

Psychological and social considerations

Psychological and social aspects of pain are described in detail in the chapter by Hansen et al elsewhere in this issue. Chronic pain patients commonly have problems with these psychosocial aspects [30]. Pre-existing psychological factors have been shown to be very important in many types of chronic pain [18,31–35]. There is a vicious cycle in which pain causes disability and stress, which in turn worsens pain perception [36]. An unhealthy lifestyle, lack of social support, depressive illness, and substance abuse are predisposing factors [37].

Acute care physicians should understand the role that attention and distraction have in the perception of pain, and encourage patients to distract themselves from the pain rather than focus their attention on it. Physicians can improve their patients' outcomes by raising their expectations of improvement, encouraging them to act as usual and not let themselves become debilitated or deconditioned [38].

Other psychosocial issues, such as patients' beliefs about their pain [39], tendency to "catastrophize" [40], involvement in the "sick role" [41], and "pain behavior" [42], should be appreciated. Physicians should be careful to avoid reinforcing dysfunctional beliefs and behavior.

Overall, some psychiatric morbidity is present in up to 67% of chronic pain patients [43]. Physicians should be aware of the high prevalence of personality and mood disorders in chronic pain patients, and insure that they are referred to a program in which these issues will be evaluated and addressed appropriately.

The role of opioids in chronic pain

Although opioids are now widely accepted for the treatment of acute pain and cancer, the long-term use of opioids for chronic nonmalignant pain remains controversial. Many studies show that some patients' pain and function improve with long-term opioid therapy [44–48]; however, the use of opioids in the treatment of chronic pain is more complicated than is commonly appreciated. Their efficacy for chronic pain is also controversial, as is reviewed below. Prescribing opioids for chronic pain, particularly in the acute care setting, has been discouraged in the past [49,50]. It has been stated that for the most common type of chronic pain patient, opioids do more harm than good [3], and that the use of opioids for chronic pain is associated with poor treatment outcomes [51]. This can become a problem when chronic pain patients demand opioids on a regular basis, particularly when better modalities are available [52].

Function of opioids

To appreciate the actions and limitations of opioids, it is important to understand their mechanism of action. In general, opioid medications work

at the sites of endogenous opioids in the central nervous system's pain control and reward centers. In the spinal cord, they bind opioid receptors, and inhibit the propagation of the pain signal [21]. The analgesic effect occurs mainly at the dorsal horn of the spinal cord, and is not dependent on supraspinal centers or on descending inhibition [53]. The spinal and supraspinal effects of opioids are said to be synergistic [54].

Within the brain, opioids function by both producing analgesia and by altering the emotional components of pain [55]. Opioids have been found to be less effective in relieving experimental pain induced in controlled laboratory conditions. This is because anxiety and context, which are very important parts of the experience of pain, are greatly diminished in a controlled experimental setting [56].

Efficacy

There are genetic differences in the metabolism of opioids, in their effectiveness as analgesics, and in their psychological effects [57]. Codeine is normally metabolized to morphine, which is the form by which it produces what little effect it has. Approximately 10% of Caucasians are deficient in the enzyme for this transformation [9]. Morphine is converted to a more potent form, morphine-6-glucuronide, in the liver and kidney [28]. There are genetic differences in the rate of this conversion as well [57].

Codeine has been shown to be a poor analgesic. Combining 60 mg codeine with acetaminophen adds at most 5% more pain relief to acetaminophen alone, which is lost after the first week [58]. Even 120 mg codeine has been shown to be no better than placebo [52]. Even where codeine was found to have poor efficacy and substantial side effects, many patients requested it as long-term therapy [59]. This demonstrates codeine's self-reinforcing properties despite its poor efficacy.

Propoxyphene is another frequently prescribed opioid analgesic, but is also very weak, with little advantage over non-opioids, and few indications for use [60]. 100 mg of propoxyphene napsylate has been shown to be no more effective than 32 mg codeine, and alone it is no more effective than aspirin or acetaminophen [61]. Not only is propoxyphene no better than aspirin or acetaminophen, it has addictive potential and a risk of renal toxicity [62]. A propoxyphene metabolite produces central nervous system (CNS) excitation and seizures [27].

The opioid agonist tramadol has been found to have no more analgesic properties than codeine or propoxyphene [61].

Some studies of codeine [63], propoxyphene [47], and tramadol [64] have reported relief of chronic pain with these medications. In view of the fact that they have poor analgesic efficacy that is likely to become worse with time, it is probable that the perceived benefits are primarily from the psychological side effects. Even these psychological effects will be lost with

the development of tolerance, after which patients will be taking the medications solely as treatment for withdrawal symptoms.

A review of studies of various opioids yielded highly variable results, with an average pain relief of around 32% [65]. Some studies have found adequate pain relief in some patients, but little or no improvement in employment or social function [46], and worsening among those whose pain did not improve [66].

Opioids have been found to be poorly effective, and were not recommended for chronic pain associated with multiple sclerosis [67], or for "sympathetically maintained pain" [12]. At 2 years follow-up of the long-term intrathecal morphine treatment for low back pain, one half of the patients had less then 25% pain relief [68].

A study was done in which idiopathic pain patients, who believed that opioids were effective for their pain, were blinded and given either an opioid or placebo. When they learned that they did not get significant pain relief from opioids compared with placebo, they became motivated for detoxification and rehabilitation, and decreased their dependence on opioids [69].

Other studies have shown good responses to opioids, but still a substantial percentage of patients received little or no improvement in their pain [47,48,70,71]. Of those patients who appear to improve, it is difficult to distinguish between psychological effects and true analgesic benefit. Again, many patients may have actually been self-treating withdrawal caused by physiologic dependence to medications that initially provided little analgesia.

Neuropathic pain

Neuropathic pain is particularly unresponsive to opioids [54,69,71,72]. This may be in part due to the presence of neurological remodeling, in which the new neural connections generally lack mu-receptors, making them resistant to opioids [21]. In postherpeutic neuralgia, controlled-release oxycodone produced a mean pain relief of only 19 out of 100 points on the visual analog scale; 76% reported side effects [73].

Because of the generally poor response of neuropathic pain to opioids, some authors state that the condition usually should not be treated with these agents [14,71], or that patients who already are on opioids should be detoxified [12].

Side effects

Some degree of sedation and impaired performance commonly occurs when opioid analgesic drugs are initiated, until tolerance to them develops [74]. Respiratory depression may occur if opioids are started at a high dose. Constipation occurs frequently and is potentially serious [47]. The physician should consider prescribing a stool softener along with the opioid.

Tolerance

Tolerance has been defined as a physiologic adaptation to the effect of a drug, with diminished effectiveness at constant dosage, or requiring an increased dosage to maintain the same intensity and duration [75]. This does not necessarily imply addiction, because tolerance is a normal physiologic process [44].

Tolerance to the analgesic effect of opioids occurs commonly [55,61,76,77]. In one study of chronic pain patients [71], 69% of the patients developed some degree of tolerance, and required a steadily increasing dose; 47% of the patients had mild tolerance, but in 22% it was marked and limited treatment. In a formal treatment program, 4% of the patients developed rapid, progressive tolerance with poor improvement in pain, and required discontinuation of their opioid medication [70].

Tolerance occurs through multiple mechanisms. Chronic opioid administration activates pronociceptive descending pathways via upregulation of cholecystokinin (CCK) in the rostral ventromedial medulla. This process, in addition to causing resistance to the analgesic effects of opioids, increases the patient's pain [29]. Opioid tolerance has also been shown to involve a functional uncoupling of opioid receptors to internal mechanisms within neurons [78], along with up-regulation of adenylate cyclase activity [79]. It is detectable after just 1 hour of continuous infusion of alfentanil in rats [80]. Ominously, this process involves the activation of protein kinase C (PKC) and results in increased NMDA-receptor activation—the same process that has been implicated in the development of chronic pain [23].

In rats given morphine injections once daily for 8 days, tolerance has been shown to activate not only these pain-facilitating processes, but also the nuclear repair enzyme Poly (ADP ribose) synthetase (PARS). Activation of PARS leads ultimately to cell death and neuronal remodeling, with irreversible changes similar to those seen in chronic pain syndromes [81].

The clinical consequences of tolerance are significant. Tolerance-induced hyperalgesia in rats is similar to that produced by a thermal burn [22], with a substantially decreased ability to tolerate mechanical painful stimuli [54].

In humans, paradoxical opioid-induced pain has been observed after a single infusion [29]. The ability to tolerate pain has been found to be two times worse in actively-using opioid addicts compared with those who are abstinent. Poor pain tolerance persists even in addicts maintained on methadone, and worsens with increased dose [82]. A low pain threshold and poor pain tolerance persist even in former addicts [83,84]. In this way, the chronic use of opioids can increase the patient's sensitivity to pain, leading to a vicious cycle of increased pain and increased medication requirements [85].

Tolerance also develops if opioid receptors are blocked without coactivation of substance P receptors, and is reversed by stimulating the substance P receptors, suggesting that there must be a balance between opioid receptor activation and painful stimuli [86]. This implies that giving

opioids for mild or psychological pain may induce neuroplastic changes in the patient, resulting in increased pain.

Paradoxically, tolerance to opioids is worse with short-acting opioids than with long-acting opioids. This may be due to the dual effect of opioid receptor occupation plus NMDA-receptor activation by glutamate when the opioid has worn off [87].

In addition to developing tolerance to the analgesic and physiological effects of opioids, tolerance develops to the psychological effects as well, namely sedation and possibly euphoria. This generally occurs more rapidly than the tolerance to analgesic effects [88]. Tolerance to the psychological effects actually complicates the use of opioids further, because the psychological effects are in many cases the only reason many patients prefer them to other types of pain control. Some may interpret the loss of these effects as worsening pain, and this may cause them to seek higher doses.

Ultimately, there is an upper limit to which you can increase the dose of opioids to try to stay ahead of this tolerance [88]. Some authors maintain that if tolerance is mild, then opioids can be continued. If there is a rapid escalation of dose, then opioids should be tapered and discontinued [71].

There is some evidence that cross-tolerance between opioids is incomplete. It is recommended that if the patient is to try a different class of opioid, then it should be started at one half of the equivalent dose [26].

Studies suggest that giving an NMDA-receptor antagonist along with an opioid can prevent tolerance. If tolerance is already present, then giving multiple NMDA-receptor antagonist treatments over several days may reverse the tolerance [22].

Physiologic dependence

Physiologic dependence is closely related to tolerance. It is defined as a physiologic state of adaptation, usually characterized by development of withdrawal symptoms when the drug is discontinued [75]. It develops in the majority of patients who take opioids chronically [89]. In one study [47], it was found to be present in all of 52 patients treated with long-term opioids. Clinical dependence can be detected after just a few days of continuous use [55,90,91]. Subclinical physiologic dependence can be detected after a single dose of morphine in the analgesic range, 10 to 30 mg [92].

Physiologic dependence has a high degree of genetic heritability [93]. In addition to cellular changes seen in opioid tolerance, the mechanism of dependence involves activation of the pain-transmitting on-cells of the medulla. Withdrawal results in a level of activity in these cells higher than before opioids were started, producing rebound pain [11]. A rebound increase in acetylcholine levels in the nucleus accumbens has also been demonstrated, and was shown to persist beyond the period of withdrawal symptoms. This represents long-lasting adverse neuroadaptive effects after even a relatively short period of opioid administration [94].

Withdrawal symptoms include deep bone pain, muscle aches, and back pain [92], and may result in an increase in a chronic pain patient's usual pain [88,95]. It also produces marked increases in depression, hypochondriasis, anxiety, irritability, and feelings of weakness and sickness [96].

Withdrawal symptoms start 8 to 12 hours after the last dose of a short-acting opioid, peak at 48 hours, and subside over 5 to 7 days. Withdrawal to long-acting opioids such as methadone starts after 36 to 48 hours, peaks at 4 to 6 days, and may persist over 14 days. Drug craving may persist even after protracted abstinence [92].

The frequent use of short-acting opioids on a regular basis produces withdrawal symptoms between doses [88], and thereby worsens the patient's condition. This may involve the rebound stimulation of NMDA-receptors by glutamate that is worse with short-acting opioids, similar to the problem of short-acting opioids and tolerance [87].

Dependence may compel patients to seek opioids for withdrawal even if pain has resolved [97]. It has been noted that patients on opioids rarely reduce their doses when the pain improves [50], and that the quantity of opioids taken is dependent on supply rather than on levels of pain [76].

Because of the phenomena of tolerance, dependence, and withdrawal, we should use caution in interpreting the results of studies using long-term opioids to treat chronic pain. For example, a study which claimed excellent relief of chronic pain with codeine [98] also found that patients taking it for longer than 3 months had better relief than those taking it for less time. In view of the known poor efficacy of codeine and the loss of efficacy that occurs with time, it is likely that such patients ultimately find themselves treating withdrawal symptoms rather than their primary pain disorder.

Problems with psychological dependence, abuse, addiction, and pseudoaddiction are reviewed in the chapter by Hansen elsewhere in this issue. They are important issues, but they are controversial, and do not necessarily affect the decision whether or not to use opioids for chronic pain. It is more important to understand the limitations of the true analgesia provided by opioids in chronic pain, and the potential harm done to patients through physiologic dependence, descending pain facilitation, and tolerance-induced, NMDA-receptor mediated worsening of their pain.

The "pain-opioid downhill spiral"

It has been the experience of many pain treatment clinics that the long-term use of opioids makes some patients' conditions worse. This starts with a painful injury, for which opioids prescribed. The pain, however, does not resolve as expected. More opioids are then given, and tolerance develops, with subtle withdrawal and psychosocial changes. Full-blown addiction occurs in predisposed individuals. As psychosocial dysfunction gets worse,

the perceived pain worsens. This, therefore, is iatrogenically worsened pain. The problem becomes opioid maintenance rather than pain management [3].

Many chronic pain patients actually have improvement in their pain when they are weaned off opioids, or when their dose is decreased [3,76,88,95,99–101]. In one series [102], pain decreased 18% to 89%, with an average decrease of 57%. Some patients' pain resolves completely with stopping opioids [95,103]. This is especially likely to occur when patients have been taking short-acting opioids [97,104].

Poor improvements in psychological and functional status have been observed, despite good improvement in pain, presumably due to learned behavior [43]. The global functioning of some patients has also been shown to be worse with the long-term use of opioids, and to improve after withdrawal [3,104].

Long-term opioid therapy for chronic nonmalignant pain appears to be effective for some patients, but clearly some patients are made worse. Consequently, acute care physicians should exercise caution in initiating opioid therapy in chronic pain patients, because many of them may ultimately require detoxification. Detoxification can be very difficult, particularly if the patients have developed psychological or physiological dependence. Moreover, some of the neuroplastic changes may be irreversible.

Are physicians required to provide opioids?

There is a common misconception that the 2001 Joint Commission on the Accreditation of Health care Organizations (JCAHO) pain standards [105] require that all patients' complaints of pain must be completely eliminated, and that physicians are thus compelled to use opioids if the patient demands them. Although the standards state that "patients have the right to appropriate assessment and management of pain" [105], there are no specific references to opioids, nor is any degree of relief specified. Rather, the intent of these standards is that a patient's pain should be evaluated and treated in the best way possible, which does not necessarily include opioids [106]. Treating pain "aggressively and effectively" may include the use of opioids for acute or postsurgical pain, but may exclude opioids for many chronic pain patients.

The term "oligoanalgesia" has been used to describe the undertreatment of pain, which has been reported to occur in emergency departments [107]. Opioids are very effective for treating pain during the relatively brief acute and recovery phases of injury or illness. Chronic pain is far more complicated, however. Rarely should rapid-onset, short-acting opioids be used. Many patients should not be given opioids at all, particularly in the acute care setting. The goal in the treatment of chronic pain is improved function and long-term pain management. Attempts to eliminate pain in the short term can interfere with these goals. Consequently, the term

"oligoanalgesia" should not be applied to the treatment of chronic pain in the acute care clinic.

Conditions for long-term opioid use

Carefully selected chronic pain patients can benefit from long-term opioid therapy when it is managed appropriately; however, multiple studies have shown that non-opioid therapy works for the majority of pain patients. It has been recommended that other therapies be used first before considering long-term opioid therapy [48,70,108]. Ideally, chronic pain patients should not be started on opioid medications without an opioid trial to test for analgesic efficacy, and only after a thorough evaluation, to include a drug history and consideration of behavioral strategies [71,109]. This approach has been tested in a blinded study [69] to determine in advance if opioids would be efficacious for particular chronic pain patients.

Multidisciplinary therapy

When opioids are used for chronic nonmalignant pain, it is usually recommended that they be used in combination with other modalities, including physical and psychological therapies [3,65,66,110–112]. It is important that opioids not be offered as an alternative to multidisciplinary therapy. If the use of opioids is made contingent on the failure of nonopioid therapy, then some patients will not be motivated to pursue it [51].

To maintain control and optimize the patient's drug usage, opioids should be prescribed by only one physician or one chronic pain treatment team whenever possible [90]. Because patients commonly express a preference for medications or treatments that are less effective for long-term pain control, the treating physician or group must have a good understanding of the multiple aspects of chronic pain treatment, and a there must be a good physician–patient relationship, in order to direct the patient toward the most effective pain control strategies.

Complete assessment

It has been stated that opioids should not be started or maintained in chronic pain patients without a complete assessment [90,113]. The JCAHO Pain Standards for 2001 [105] state that the assessment should include physical, cognitive, behavioral, communicative, and emotional and social status, and include substance use and emotional and behavioral disorders. This is similar to the requirements stated by the American Academy of Pain Medicine [109] and the American Society of Anesthesiologists Task Force on Pain Management [2]. This obligates acute care physicians to either complete this assessment themselves, or to ensure that it either has already been done or will be done at follow-up, before opioids are continued.

Patient selection

Appropriate patient selection is vital in the decision to use chronic long-term opioids.

There are studies showing that such a carefully selected subgroup of patients can have reasonably good relief with opioids, with a low frequency of problematic use. Patients who have a well-defined physical etiology are good candidates [60], particularly those near the end of life [74]. Other patients require greater scrutiny. Psychogenic pain should be ruled out or treated appropriately [66]. Patients should have a stable work and family situation, and there should be no history of substance abuse [50]. The risk of addiction approaches 50% in patients who are actively abusing illicit drugs, are not in a recovery program, or have poor social support [114]. All patients should be willing to participate in other treatment modalities and to adhere to the conditions of a contract [110].

Opioids should not be used unless psychosocial issues are also addressed. Even in those chronic pain patients for whom opioids provide pain relief, it is frequently found that psychological and functional status is not improved [46,115]. Cognitive/behavioral and physical therapy should be maintained during opioid maintenance therapy to improve function [46,70,109].

It has been stated that opioids can be used in patients with a history of addiction, but only if the addictive disorder has been treated [88]. In these and other patients who are unable to control or regulate their opioid use, it may be necessary for a pain management clinic to dole their medications out to them on a daily basis.

Set goals

It is also important to establish realistic goals of opioid therapy. For many patients, opioids will not provide the degree of relief that they are seeking [74]. Patients should be informed that the goal of therapy is to improve function, with the intention of tapering the medication to the minimally effective dose. If function is not shown to improve, then opioids are not considered worth their associated risks [114].

Dose and duration

When opioids are used in chronic pain patients, they should be used at the lowest dose that provides reasonable pain relief [99] and improved function [116]. This may also minimize tolerance and opioid-induced NMDA-receptor activation. If moderate or moderately high doses of opioids do not effectively improve pain, then it would be better to gradually withdrawn them, rather than increase to a very high dose [117]. Patients should be made to understand that even though high-dose opioids may give them some psychological relief, it will quickly be lost to opioid tolerance, whereupon they will have developed physiological dependence and possibly worse pain.

The opioid chosen should be long-acting. Short-acting drugs should be used only for acute pain, or for the titration phase of long-term opioid treatment for chronic pain [118]. Many patients' pain improves just by switching to a long-acting medication [108]. Methadone has been used successfully in younger patients, who are at lower risk for drug accumulation [50]. Controlled-release morphine sulfate has been studied in cancer patients [119] and chronic nonmalignant pain patients [120], and has been shown to improve pain and quality of life.

For chronic pain, opioids should be given around the clock rather than as needed for pain [99,121]. It should be noted that even in patients whose pain was decreased and whose mood was stabilized when their opioids were given on a fixed interval, the majority stated a preference for the on-demand schedule [122]. One can anticipate that patients will seek on-demand medications even though they are less effective for pain relief and functional improvement.

The medication should also be slow-onset, to minimize the reinforcing effect of pairing the narcotic effect with the perception or expression of pain. Rapid-onset medication should be avoided in the treatment of chronic pain, particularly when a long-acting medication is given around the clock [28]. Parenteral opioids are the ultimate rapid-onset, short-acting form, even when given intramuscularly. This not only causes a problem with reinforcing pain behavior, but it also provides a short-lived flood of psychological relief or euphoria. When one considers that many patients will get at most 30% relief of their pain even with opioids, then it will be difficult for many of them to resist seeking the psychological effects of an injection. This will only complicate the long-term management of these patients. The use of parenteral opioids for chronic pain should be minimized, if not avoided entirely.

If the dose of medication is increased, it should be done in a gradual manner, by no more than 30% to 50% [27]. If the patient is rapidly developing tolerance with a rapid escalation of dose, then opioids should be tapered and discontinued [71].

''Rescue'' medications provided for breakthrough pain should be limited to six to eight extra doses per month for unexpected exacerbations. If the patient consistently requests additional doses, then there needs to be a reassessment of the dose and schedule, and a consideration of psychosocial issues [60]. To avoid reinforcement issues, rescue medications should not be dispensed on an urgent basis, but prophylactically, by the patient's physician or clinic [97].

To maintain control and optimize the patient's drug usage, opioids should be prescribed by only one physician or one chronic pain treatment team whenever possible [90]. Because patients commonly express a preference for medications or treatments that are less effective for long-term pain control, the treating physician or group must have a good understanding of the multiple aspects of chronic pain treatment, and a there must be a strong

physician–patient relationship, in order to direct the patient toward the most effective pain control strategies.

Pain contracts

A pain contract should also be made when opioids are used frequently for chronic pain, to include documentation of the patient's understanding of the limitations of opioid therapy, treatment objectives, risk of dependence, and need for other treatment modalities [70,110]. Such contracts usually limit the patient to obtaining prescriptions only through the pain clinic, and limit replacements for "lost or stolen" prescriptions [123]. Some contracts specifically prohibit the patient from seeking opioid medication from the emergency room [113].

Preventing chronic pain

Greater attention needs to be paid to the prevention of chronic pain. In surgical patients, there is some evidence that preemptive analgesia with opioids may prevent postoperative pain and the development of chronic pain [124,125]. Preoperative lumbar epidural blockade has been shown to be highly effective in preventing postamputation phantom limb pain [126].

Early recognition of pain and aggressive treatment can help prevent unnecessary functional impairment, secondary physical and psychosocial disability, and chronic pain [37]. Because the self-reinforcing effect of opioids may contribute to the development of chronic pain after acute injury, opioids should be prescribed for a predetermined amount of time [121]. Severe pain from acute injuries, including fractures, rarely persists longer than 2 weeks [4]. If patients seem to need opioids beyond the normal healing period for an acute injury or illness, then they should be considered to be at risk for chronic pain or opioid dependence. Opioids should be discontinued or restricted to a low dose. These patients should be referred to a multidisciplinary pain clinic [127].

Liability

There is potential liability for physicians who prescribe opioids for chronic or poorly defined pain outside of a multidisciplinary pain clinic. Physicians are being sued for causing addiction [77]. A physician was found liable for refilling an opioid despite the fact that the patient had a contract that prohibited it. The patient had a known history of noncompliance, and overdosed on the medication [123].

Follow up

Physicians should always keep in mind that long-term opioid therapy can make some patients' pain worse. Patients should be monitored to ensure

that they are not having accelerated tolerance, and that physical and psychological therapies are maintained.

Nonopioid therapy

There are many alternative treatments to opioid medications that can be very effective in chronic pain patients. It has been established that the majority of people have similar pain thresholds, but this threshold can be raised by 50% using such techniques as acupuncture, relaxation, hypnosis, and imagery. [8] Women in active labor show a sharp decline in pain and negative mood if they have attended a Lamaze childbirth preparation class, relative to controls [128]. Nearly 90% of women can go through labor without medication using relaxation techniques [129]. These methods should be applicable to chronic pain patients, and they should be encouraged to use them and minimize their reliance on medications.

Psychological treatments

Cognitive-behavioral techniques have been applied in the treatment of chronic pain. Re-educating patients about how they should think about their pain and how they should respond to it has been shown to be very helpful in improving patients' pain and function [38]. Operant conditioning [130], relaxation training [131], and the medication technique ''mindfulness'' [132] have also been used successfully; however, it has been noted that cognitive therapy approaches generally do not work well in isolation from other aspects of multidisciplinary treatment [133].

Physical treatments

Physical therapy reinforces adaptive efforts and extinguishes maladaptive pain behaviors [134], produces improvements in activity and well-being, and tends to reduce the use of opioids [135]. It is also thought to retrain the nervous system to re-establish normal neural connections. Pain levels generally do not improve until patients have begun reconditioning, and have increased their activity levels [136].

Many studies have shown that nonspecific or placebo treatments can produce improvement equivalent to physical therapy [137], suggesting that there is therapeutic value in merely getting patients to engage in any form of physical activity, rather than allowing them to be sedentary and avoid movement and stimulation.

Other physical treatments such as manual therapies, acupuncture, and exercise have been also found to be effective. The application of cold raises the threshold of pain for up to 12 hours. Many of these therapies may be effective based on the activation of descending pain inhibitory systems [138]. Acupuncture appears to work through the stimulation of endogenous

opioids [25]. This raises the concern that acupuncture and other physical treatment modalities could be made less effective by tolerance to concurrently administered opioids.

Multidisciplinary pain clinics

Psychological and physical approaches are much more effective in combination. The management of chronic pain frequently requires a combined multidisciplinary approach, including education, psychological counseling, behavior modification, and physical medicine [139,140]. There must also be a "positive physician–patient relationship" [141]. Patients should be referred to a pain clinic within weeks to a few months of the development of chronic or persistent pain, to prevent progressive pain, associated morbidity, and increased costs [127].

Even simple chronic pain requires multiple modalities of treatment, including a psychological evaluation and possible treatment. "Complex" chronic pain, with complex interactions of legal, psychological, medication, and family, requires more intensive psychological and social interventions [142]. Geriatric patients also benefit from multidisciplinary care [74].

A review of multiple studies [130] has shown that combined treatment produces moderately to markedly improved pain, with decreased medication use and increased functioning in the majority of chronic pain patients. Most of the improvements were maintained at follow-up years later. The condition of patients not in multidisciplinary care tends to worsen [143].

Despite the effectiveness of multidisciplinary pain clinics, some patients choose not to participate in them, and prefer instead to seek care at emergency rooms and urgent care facilities; however, it has been noted that those patients who are dissatisfied or who do not complete multidisciplinary pain treatment programs tend to be those who have the greatest drug dependence and psychological pathology [49,144–146]. These patients have the greatest need for multidisciplinary care, and are the worst candidates for chronic opioid therapy outside of the clinic.

Drugs for chronic pain

Acetaminophen and cyclooxygenase inhibitors

Acetaminophen is a safe, effective analgesic for mild to moderate pain. It has a maximal "ceiling" of pain relief, at which increased dosage will not provide more analgesia. Its efficacy is often downplayed because it is commonly available; however, when one considers that nearly all of the pain relief provided by acetaminophen/codeine combinations is derived from the acetominophen, then its efficacy can be better appreciated. Patients should be encouraged to use acetaminophen, but not to exceed the recommended dose, nor to use it in addition to other medications that include it.

Nonsteroidal anti-inflammatory drugs (NSAIDS) have not been shown to be significantly better for musculoskeletal pain than acetaminophen. In osteoarthritis or "tendonitis," there is little or no chronic inflammatory component. NSAIDS are commonly used nonetheless. If there is a risk of gastrointestinal bleeding, a cyclooxygenase-2 (COX-2) inhibitor should be considered [147].

Nonanalgesic drugs

There are many drugs for use in chronic pain that were not originally used as analgesics. These include baclophen (a $GABA_B$-agonist), and clonidine (an alpha-2 adrenergic agonist) [21]. Tapered doses of steroids have been advocated for neurogenic back pain and complex regional pain syndromes [4].

Antidepressants and anticonvulsants may be started in the acute care clinic, preferably in consultation with a pain specialist or personal physician [4]. Carbamazepine has been used successfully in the treatment of neuropathic pain. In a study of carbamazepine used to treat chronic pain patients who were also depressed [148], the patients' pain improved from 8.2 to 4.0 (51%), but the depression improved only 26%. The starting dose of carbamazepine is 100 to 200 mg twice a day [4].

Gabapentin (an N-type calcium channel inhibitor) has also been used successfully for neuropathic pain [4]. In postherpeutic neuralgia, the dose can be slowly increased by up to 300 mg/day, to a target dose of 1800 to 2400 mg/day, up to 3600 mg/day, divided three times daily. Side effects include somnolence and dizziness in approximately 20% and 35%, respectively [149].

Some of the most promising drugs for chronic pain are the NMDA-receptor antagonists. These include ketamine, dextromethorphan, amantadine, and methadone [150]. Morphine sulfate (MS) combined with dextromethorphan (1:1) reduces the amount of morphine required for relief of cancer and other chronic pain by as much as one half [151]. Unfortunately, NMDA receptors are found in high density in the cerebral cortex and hippocampus, and drugs that block these receptors can cause psychological side effects [24].

In summary, there are many effective ways to treat chronic pain that do not involve opioid medications, and do not expose the patient to the risk of tolerance-induced increased pain or physical and psychological dependence. Because long-term opioid therapy often interferes with the management and rehabilitation of chronic pain patients [3], emergency and urgent care physicians should encourage patients to seek nonopioid treatment for their chronic pain. Informing patients about the problems with opioids and their generally poor effectiveness in chronic pain, may help motivate them to seek nonopioid treatments [69].

The role of the acute care physician

Emergency and urgent care physicians have an important role in the care of chronic pain patients. Not only are they immediately available to treat acute exacerbations and are available after hours and on weekends, but they are often the first provider that the patient sees. Moreover, they provide an alternative to the patient's personal physician or pain clinic when the patients are dissatisfied with their care, for better or worse. Acute care physicians should be careful that their practices contribute to the solution and not to the problems associated with chronic pain.

The message

Physicians should emphasize that improved function is vitally important in the treatment of chronic pain [152]. They should inform chronic pain patients that, although there is no quick fix for their condition, they will improve if they take an active role in their therapy [1,101,153]. Chronic pain patients should not be given the false expectation that any single mode of therapy will "cure" their condition [74,106], because this leads them to seek short-term relief at the expense of long-term improvement.

Telling patients to "let pain be your guide" commonly leads chronic pain patients to become deconditioned [121], which in turn can produce concurrent myofascial pain [12]. Instead, physicians should encourage chronic pain patients to increase their levels of activity.

Physicians should be sympathetic and supportive toward their patients, but they should be careful not to overstate the severity or consequences of their condition. It has been shown in a randomized trial that when "idiopathic pain" patients are told that there is no evidence of illness and that they do not require treatment, they are substantially better at follow-up than those who are given a symptomatic diagnosis and a prescription [154]. Patients who have minor whiplash injuries have been shown to have a better outcome if they are told that the actual physical damage is minimal, and that the injury itself does not cause long lasting pain. They should be encouraged to return to normal activity and go back to work as early as possible [38].

Physicians should be careful not to overemphasize medications and testing, because it tends to reinforce patients' perception that their pain is purely physical [121]. This is particularly problematic in patients who have somatization [155].

Consultation and disposition

It is very important that emergency and acute care physicians work closely with the local pain management clinic, or with the patient's personal

physician. Physicians should understand that many treatments for chronic pain require a considerable amount of time and commitment on the part of the patient. Trying to make these treatments work without the coordinated efforts of a pain clinic may result in failure, and a loss of confidence by the patient.

The pain clinic or personal physician should be consulted whenever possible before opioids are given. Ideally, a strategy should be worked out in advance for new patients. This should include a plan for analgesia to help the patient through the period of time until he can be seen in the clinic, but should not establish a pattern of opioid use that may generate the expectation that opioids will be continued indefinitely. Such patterns, once established, are difficult to change, and may result in irreversible adverse changes in the patient's endogenous pain control system.

Close follow up should be arranged after discharge. Hospital admission is rarely indicated for chronic pain control [4].

Documentation

Physicians should document pain levels according to the JCAHO guidelines; however, this should not make physicians feel compelled to use opioids in patients in whom they are not appropriate. In one study [156], the documentation of pain assessment after treatment was associated with a trend for improved patient satisfaction, but it did not achieve statistical significance.

Patients who over-rely on the acute care clinic

The distinction between therapeutic opioid users and recreational drug-seekers is discussed in detail in the chapter by Hansen elsewhere in this issue; however, labeling patients with either designation has not been shown to help them [4]. It is far more important that the acute care physician remember that the frequent use of opioids can be harmful to either class of patients. Unfortunately, emergency and urgent care physicians have been shown to be very poor at restricting opioids in patients in whom they are inappropriate. In a study in which 30 drug-seeking patients were identified [157], 17 were told that they would receive no further narcotics from the facility, but 71% of those patients were given opioids anyway within 2 years.

Habitual patient files

A "habitual patient file" can help identify those patients who are at risk for addiction or tolerance-induce worsening of their chronic pain. Such files can be used in a way that is consistent with state laws, the Health Insurance Portability and Accountability Act of 1996 (HIPPA), and the JCAHO [158].

These are reviewed in greater detail in the chapter by Hansen elsewhere in this issue on drug-seeking patients.

Proactive arrangements

There are many ways in which an acute care facility can improve the care of frequent narcotic users and chronic pain patients who have legitimate needs and well-controlled opioid use. These include patient tracking systems, ''narcotic contracts,'' and pain management letters that inform frequent users that they will be denied narcotics unless they have a detailed letter from their personal physician. One emergency department (ED) reports a system in which frequent narcotic users are referred to the emergency department's care manager. The manager then reviews the patient's history, checks with the patient's doctor, and creates a care plan for the patient. This may include helping the patient with follow-up, arranging referrals to multidisciplinary pain or drug treatment clinics, and educating the patient about alternative treatments, as well as specifying what drugs may be given to the patient when he presents. This system provides much better and more consistent care for both legitimate chronic pain patients and for drug seekers, decreases the number of visits to the ED by both types of patient, and improves the morale of the ED staff. The time invested by the care manager is more than made up by improved care [159].

A system has been proposed to identify pain patients who over-rely on the ED, and direct them toward appropriate outpatient care. Patients presenting to the ED for opioid treatment of a chronic pain problem 10 times in 1 year were reviewed for entry to a chronic pain registry. To be accepted into the registry, they had to have a primary care physician who could be contacted (or have back-up call) 24 hours a day, go to just one ED for treatment of the chronic pain problem, and be willing to undergo evaluation by a physician with expertise in chronic pain and drug dependence issues. Those patients who did not qualify or refuse to enter the registry would be denied opioids at the ED [160].

Whatever system is employed, it is important that the long-term benefit to the patient be kept in mind, and that it be consistent with the practices of the local pain management clinic or with the patient's personal physician (assuming that physician has reasonable expertise in the care of chronic pain).

Specific chronic pain syndromes

Complex regional pain syndrome

Physical therapy, including gentle desensitization and other measures, is one of the few interventions that have been shown to be effective in

controlled studies of CRPS [161]. It produced a nearly 50% decrease in pain with effort at 3 months [162].

Physical therapy has been shown to very effective in the treatment of CRPS in children [19]. The majority of the children in the study were using crutches or wheelchairs, and had chronic changes secondary to maintaining the extremity immobile in a dependent position. The patients were treated solely with intensive physical therapy and vigorous toweling to provide sensory stimulation. The contralateral side was immobilized to force use of the affected side. By the end of therapy, 12 out of 20 had complete resolution of their symptoms. Two others had occasional discomfort and intermittent swelling. All had normal function, and none required a wheel chair or crutches. They were followed for average of 2.4 years, and there was only one recurrence.

Myofascial pain syndrome

Myofascial pain syndrome improves with multidisciplinary therapy [163]. Treatment should include specific musculoskeletal treatments, including physical therapy and trigger point injections. If not treated appropriately, myofascial pain syndrome may progress to a complex chronic pain syndrome, with sleep disturbances, fatigue, and psychosocial difficulties [164]. Trigger-point injections can be done in the acute care setting by physicians who are familiar with the technique.

Fibromyalgia

Aerobic exercise and cardiovascular fitness training produce long-term improvement. Although fitness training may make the patient feel worse initially, it is vital to prevent further deconditioning. Patients tend to be skeptical initially, and proven benefits are lost in the long run if patients are noncompliant [165,166]. Antidepressants [165] and acupuncture can be very effective in fibromyalgia, and may be synergistic [167]. Reducing fibromayalgia patients' ''catastrophic'' thoughts, and convincing them that they have the capacity to be more functional can have a substantial impact on their pain and function [34].

Low back pain

Early, aggressive rehabilitation programs can prevent disability in low back pain [90,168,169]. Nonspecific exercise has been found to be as effective as conventional physiotherapy [170]. Low back pain patients have been found to have poor aerobic capacity. Improving their fitness can improve their pain and disability to the point where they rate it as minimal [171]. Spinal cord stimulation [68] and percutaneous electrical nerve stimulation (PENS) [172] have been shown to produce approximately a 50% decrease in pain, with improved activity. Therapeutic massage was shown to improve

discomfort 32% at 4 weeks; 74% of the subjects rated it ''very helpful'' [173].

A review of the efficacy of multidisciplinary pain clinics in relieving low back pain found that pain decreased 37%, compared with only 4% in control subjects. Prescription medication use, overall pain behaviors, and activity levels improved 65% [174]. Some programs report a success rate of over 80% back-to-work, compared with approximately 40% in controls [175,176].

The most important variable in the successful treatment of chronic low back pain appears to be the reduction of the patient's subjective feelings of disability [177]. It is therefore better if physicians and patients focus on function rather than pain. This encourages patients to be more active, and decreases avoidance behavior and maladaptive attempts to gain sympathetic attention [153].

Osteoarthritis

In a study of the treatment of hip and knee osteoarthritis [178], quadriceps-strengthening exercises produced a 30% improvement in pain. Aerobic exercise and strengthening produced a 24% increase in activity, with substantially decreased pain and use of medication. No flare-ups were observed.

Rheumatoid arthritis

Patients who present for acute exacerbations of rheumatoid arthritis may benefit from a short course of steroids; however, once steroids are started, it is often difficult to discontinue them. Long-term use may produce undesirable side effects without preventing the destructive process of the disease [179]. Consequently, it is important to consult a rheumatologist before initiating this treatment.

Chronic headaches

Chronic daily headaches are commonly caused by overuse of analgesic and other medications [180]. Acute care physicians should be careful not to contribute to this problem. The common practice of administering a short-acting opioid is frequently unsuccessful. In a follow-up study of headaches treated in the ED [181], as few as 13% of the patients treated had sustained headache relief, and almost half of the patients were unable to return to work the next day. Physicians should emphasize abortive or long-term treatment.

In another study [182], placebo plus lying in a quiet darkened room for 60 minutes produced good pain relief, equivalent to or better than meperidine or ketorolac plus an antiemetic. This should serve to remind us that in some diseases, improvement will occur without aggressive analgesia.

Summary

Chronic nonmalignant pain requires evaluation and treatment different from acute pain. The pathophysiology is different, and there is commonly some degree of psychosocial dysfunction. Opioids tend to be much less effective as analgesics for chronic pain, and may increase the sensitivity to pain when given long-term. Because they are self-reinforcing, opioids may be sought and be reported to improve chronic pain, even when they may make the condition worse over time. There are many effective alternatives to opioids for the treatment of chronic pain, but their use is complicated and may require considerable time and effort to determine which ones work. Patients, particularly those who have already been prescribed opioids, may resist these alternatives. An extensive physical and psychosocial evaluation is required in the management of chronic pain, which is difficult if not impossible to achieve in the emergency or urgent care settings. Consequently, emergency and urgent care physicians should work closely with the patient's pain management specialist or personal physician. Systems should be set up in advance to identify those patients whose frequent use of acute care services for obtaining opioids may be compromising their long-term management, putting themselves at risk for psychological and tolerance-induced adverse effects of frequent opioid use. Opioids may be used in carefully selected patients in consultation with their pain management specialist or personal physician, but care must be exercised not to initiate or exacerbate psychological or tolerance-related complications of chronic pain.

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