The hospitalist’s role

In 2006, the Society of Hospital Medicine described pain management as a hospitalist core competency [1]. “Hospitalists should be able to describe the symptoms and signs of pain; assess pain severity using validated measurement tools; formulate a pain management plan; determine appropriate route, dosing and frequency for pharmacologic agents; determine equianalgesic dosing and titrate narcotics to desired effect; anticipate and manage side effects of pain medications; discuss with patients the goals for pain management; employ a multidisciplinary approach to management of patients with pain; use evidence based recommendations; participate in development of pathways that facilitate effective pain management; and participate in efforts to measure quality of inpatient pain control.” Alleviating pain is one of the basic tenets of being a doctor. The goal of alleviating pain is the responsibility of all health care providers, and as leaders of hospital care teams, hospitalists should be at the forefront of pain management.

Pain pathophysiology

The perception of pain begins at the site of perceived or real tissue injury. Injury stimulates nociceptors or free nerve endings to release neurotransmitters. The neurotransmitters activate firing of other nociceptors, which results in information being sent via afferent nerve fibers to the dorsal horn of the spinal cord. In the spinal cord, nociceptive information is sent via the spinothalamic tract to the thalamus. In the thalamus, information is sent to the
cortical areas of the brain, which processes the information. There are different areas of the brain involved in the modulation of pain information. These include the hypothalamus, pons, and somatosensory cortex. Stimulation of these areas causes analgesia.

**Pain assessment**

Any patient-doctor relationship is potentially fraught with personal bias. Participants carry their own personal baggage into each encounter. Encounters to discuss pain may be particularly problematic. For example, patients who view pain as a weakness may be more likely to suffer with pain and present with other somatic complaints. Providers who view pain as a weakness may be less likely to appreciate the extent of patients’ pain. Pain is always subjective, can be very personal and thus, always requires a careful history for a reliable assessment.

Pain assessment tools should be simple, reliable, brief, and sensitive to any changes in pain intensity. Many hospitals commonly use numeric rating scales, where providers ask patients to rate their pain from 0 to 10. The endpoints represent the extremes, ranging from “no pain” to “worst pain possible.” This tool is reasonably easy to administer but does require some abstract thought. For some patients, it may be easier to use a picture-based pain intensity assessment scale, like the Wong-Baker Faces Pain Rating Scale [2]. In this scale, a series of faces ranging from smiling face to a crying face correspond to the numbers on a numeric rating scale. This tool may be easier to use in situations where patients and providers don’t speak the same language. Using the right tool is important because not all hospitalized patients may have the physical stamina or cognitive ability to participate in conversation or elaborate tests [3]. After any therapeutic intervention, a reassessment of the patient’s pain allows providers to make adjustments in a rational manner.

As part of any pain history assessment, it is important to ask about the nature and quality of the patient’s pain. Does the pain come in colicky waves or does it throb and ache? Where is the pain located? How often does the pain occur? When pain occurs, how long does it last? Is there anything that initiates or exacerbates the pain? What has the patient tried previously to alleviate the pain, with success or failure? What does pain mean to the patient? Does it bring back any memories? What is the patient’s attitude toward the use of narcotics or anxiolytics? Is there any history of substance abuse? The patient’s answers are not only important clues to the etiology of the pain, but can also guide the provider’s examination and management decisions. Avoid the use of placebos as part of pain assessment. Deception is unethical and has no role in pain assessment or management.

**Pharmacologic pain therapy**

In 1986, the World Health Organization (WHO) developed the WHO Analgesic Ladder as a tool to address deficiencies in the management of
cancer pain [4]. Despite the fact that no randomized, controlled trials have been conducted to validate its effectiveness, the WHO Analgesic Ladder is widely disseminated and commonly used to treat not only cancer pain but pain arising from any condition [5]. Three steps make up the WHO Analgesic Ladder (Fig. 1).

Step 1 recommends the use of nonopioid analgesics, with or without adjuvants for treatment of mild pain. In Step 2, the WHO advises adding weak opioids for treatment of moderate intensity pain, with or without non-opioid analgesics and with or without adjuvants. In Step 3, they recommend adding more potent opioids, in addition to those drugs recommended in Steps 1 and 2, for treatment of severe pain.

**Nonopioid analgesics**

Commonly used nonopioid analgesics include acetaminophen, salicylates, and nonsteroidal anti-inflammatory drugs (NSAIDs). Acetaminophen is an effective nonopioid analgesic with an average analgesic dose of 325 mg to 650 mg every 4 to 6 hours. At doses greater than 2 grams per day, patients on concurrent warfarin therapy may experience an increase in their prothrombin time. The maximum adult daily dose of acetaminophen is 4 grams, but adults with underlying liver disease should take no more than 2 grams daily. Acetaminophen over-dosage can result in fatal hepatotoxicity.

Discovered in 1897, aspirin (acetylsalicylic acid) remains the most commonly used drug in the salicylate drug class. Aspirin is similar to acetaminophen in its analgesic and antipyretic effects. But unlike acetaminophen, it has anti-inflammatory properties. Aspirin’s usual analgesic adult dose is 325 mg to 650 mg, given once every 4 to 6 hours. The maximum adult analgesic dose is 4 grams daily. The most common side effects involve the gastrointestinal (GI) tract (eg, nausea, abdominal discomfort, dyspepsia, and peptic ulceration) and bleeding (caused by irreversible inhibition of platelet thromboxane production). Aspirin hypersensitivity may occur in association with asthma, urticaria, and angioneurotic edema. Children should

![Fig. 1. WHO Analgesic Ladder.](image-url)
not ingest aspirin because of concern for development of Reye’s syndrome. Others salicylates include diflunisal and choline magnesium trisalicylate. Diflunisal has fewer GI effects than aspirin, and choline magnesium trisalicylate has minimal antiplatelet effects.

Patients with aspirin sensitivity may also be sensitive to some NSAIDs, which can also cause GI side effects. NSAIDs, like aspirin, also inhibit platelet aggregation, but unlike aspirin, the effect is reversible and lasts only for the duration of effective drug concentration. NSAIDs, despite their name, have efficacy for pain with and without associated inflammation [6]. Like acetaminophen and aspirin, NSAIDs offer the advantage of effective analgesia without the risk of physical or psychologic dependence associated with opioid therapy. There are numerous NSAIDs in a number of chemical classes (Box 1).

Unfortunately, there is insufficient data to clearly understand the differences between the NSAIDs. Ibuprofen is purported to have weaker anti-inflammatory effects, but may have the lowest potential for GI side effects [7]. Indomethacin, naproxen, and sulindac present intermediate risks for GI side effects, while tolmetin, ketoprofen, and piroxicam may have the highest risk [8]. Endogenous prostaglandins are responsible for the body’s

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**Box 1. NSAID chemical classes**

*Propionic acids*
- fenoprofen
- flurbiprofen
- ibuprofen
- ketoprofen
- naproxen
- exazoprin

*Acetic acids*
- diclofenac
- etodolac
- indomethacin
- ketorolac
- sulindac
- tolmetin

*Enolic acids*
- meloxicam
- piroxicam

*Anthranilic acids*
- mefenamic acid
- nabumetone
inflammatory response. NSAID inhibition of the cyclo-oxygenase (COX) enzyme prevents the formation of prostaglandins, leading to NSAIDs’ anti-inflammatory effects. NSAID activity in the brain and spinal cord contribute to their analgesic effects. There is a ceiling for analgesia but not for side effects, meaning administration of NSAIDs above recommended doses may increase toxicity without increasing analgesic efficacy [9].

NSAIDs have numerous potential side effects, usually because of inhibition of prostaglandins not involved in the inflammatory process [10,11]. The previously mentioned COX enzyme is expressed in tissue in at least two isoforms: COX-1 and COX-2. COX-1 is present in most tissues and is known to play an important role in the maintenance of the gastric mucosa, preservation of renal blood flow, and platelet function (the latter via production of a platelet enzyme called thromboxane). COX-2 is expressed only in the central nervous system (CNS) and kidneys. NSAIDs can cause renal side effects (via reduction of vasodilatory renal prostaglandins), hematologic side effects (via inhibition of platelet aggregation), CNS effects (via inhibition of central prostaglandin production), and GI side effects (via reduction of prostaglandin production in the gastric mucosa). The concurrent use of proton pump inhibitors or misoprostol (a prostaglandin analog) with NSAIDs may reduce the risk of peptic ulcer disease.

During the past decade, several NSAIDs that selectively block the COX-2 enzyme became commercially available. Selective inhibition of COX-2 with reduced inhibition of COX-1 was thought to offer the benefit of reduced GI, renal, and hematologic side effects. While studies show there may be some reduction in the incidence of GI side effects, the COX-2 inhibitors do not appear to offer any benefit of reduced renal side effects. After-market surveillance revealed that some selective COX-2 inhibitors may be associated with increased incidence of cardiac events. The proposed theory is that selective COX-2 inhibition prevents prostacyclin formation, leading to unopposed platelet thromboxane (a prothrombotic prostaglandin) effects. Celecoxib is the only COX-2 inhibitor which remains on the market in the United States. Its role in pain management is controversial.

**Opioid analgesics**

Opioids should be added to the analgesic regimen when nonopioids alone are insufficient for pain control. However, many hospitalized patients present with acute pain that is moderate to severe in intensity, and it would be inappropriate to initially withhold opioid analgesics pending the response to nonopioid therapies. On initial pain assessment, if the patient has moderate to severe acute pain, consider a pharmacologic regimen that includes both nonopioid and opioid analgesics, along with adjuvant analgesics and non-pharmacologic measures. Opioid agonists, unlike nonopioids, have no ceiling for analgesic effects. Providers should titrate up the opioid dose until there is sufficient analgesic effect or until side effects become intolerable.
Opioids bind to endogenous opioid receptors in several classes: delta, kappa, and mu. Opioids are classified as full agonists, partial agonists, or mixed agonist-antagonists. All available opioids are full agonists that act on the mu opioid receptor. Stimulation of the mu opioid receptor is responsible for the opioids’ analgesic and adverse effects. Adverse effects include urinary retention, pruritis, sedation (typically dose-related), muscle rigidity (more common with higher doses of potent, rapidly acting drugs), respiratory depression (dose-dependent), bradycardia, decreased sympathetic tone, nausea (via stimulation of receptors in the chemoreceptor trigger zone), and constipation. There is significant individual variation in development of side effects, but sedation and constipation appear most frequently. Patients on chronic opioid therapy can develop tolerance to some side effects (e.g., respiratory depression) but typically do not develop tolerance to other effects (e.g., constipation). It is important to differentiate side effects from allergy. True allergy to opioids is rare but if allergy exists, one should switch patients to an opioid in a different chemical class [10].

Morphine is the standard against which all other opioids are compared. This does not mean morphine is the opioid of choice for all patients in all situations. Morphine, available for oral, parenteral, rectal, and intraspinal use, is effective for moderate to severe acute and chronic pain. It is renally excreted, so patients with renal failure may experience prolonged effects. Compared with other opioids, morphine is more likely to cause histamine release, which can increase the risk of vasodilation, flushing, and hypotension.

At equianalgesic doses, all opioid full agonists have similar efficacy. Providers should choose an opioid based on side-effect profiles. Clinicians should always use the equianalgesic conversion chart when transitioning from one opioid to another (Table 1).

Table 1 compares morphine 10-mg intramuscular injection with other opioids and alternative routes. Providers should recognize that the conversion

<table>
<thead>
<tr>
<th>Narcotic</th>
<th>IM / IV (Mg)</th>
<th>PO (Mg)</th>
<th>Duration (Hrs)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Codeine</td>
<td>120</td>
<td>200</td>
<td>4–6</td>
</tr>
<tr>
<td>Fentanyl</td>
<td>0.1</td>
<td>—</td>
<td>1–2</td>
</tr>
<tr>
<td>Hydromorphone</td>
<td>1.5</td>
<td>7.5</td>
<td>4–5</td>
</tr>
<tr>
<td>Meperidine</td>
<td>75</td>
<td>300</td>
<td>2–4</td>
</tr>
<tr>
<td>Methadone</td>
<td>10</td>
<td>20</td>
<td>4–6</td>
</tr>
<tr>
<td>Morphine</td>
<td>10</td>
<td>30–60</td>
<td>3–7</td>
</tr>
<tr>
<td>Oxycodone</td>
<td>—</td>
<td>15–30</td>
<td>4–6</td>
</tr>
<tr>
<td>Oxymorphone</td>
<td>—</td>
<td>—</td>
<td>3–6</td>
</tr>
<tr>
<td>Propoxyphene</td>
<td>—</td>
<td>130</td>
<td>4–6</td>
</tr>
</tbody>
</table>

Abbreviations: IM, intramuscularly; IV, intravenously; PO, by mouth.

a For treatment of ACUTE pain and not for chronic pain.

chart is based on limited data, and there is significant individual response to various opioids. The conversion process from one opioid to another involved five steps:

1) Calculate the total dose of the current opioid over the previous 24 hours.
2) Use the conversion chart to convert to the 24-hour total dose of the new drug or route.
3) Divide the 24-hour total dose into individual doses, based on the schedule.
4) For opioid-tolerant patients where cross-tolerance may not exist, consider reducing the calculated dose of the new drug by 33% to 50% [10].
5) Calculate a breakthrough dose of the new drug. This should be either 10% to 20% of the total daily dose or 25% to 30% of the single standing dose [10].

Meperidine is an opioid with a short duration of analgesic effects because of extensive first pass metabolism. Normeperidine, a meperidine metabolite that accumulates with repetitive dosing, causes numerous CNS side effects, including seizures. Patients who use meperidine for longer than 48 hours, patients who take doses greater than 600 mg per 24 hours, patients with underlying renal failure, or patients treated concurrently with monoamine oxidase inhibitors are all at increased risk for side effects. Naloxone does not reverse the meperidine CNS effects. Given meperidine’s narrow therapeutic window and the availability of safer and more effective alternative agents, it is difficult to justify its use in clinical practice. The American Pain Society states it should not be used for acute or cancer pain [6].

Methadone is most commonly used in management of opioid addiction. Its role in acute pain is controversial. Proponents like its low cost, its effectiveness in neuropathic pain, and its long half-life, which make it an attractive agent to treat persistent pain. But methadone’s pharmacokinetics pose a challenge for those unfamiliar with the drug. Methadone has a short-lasting analgesic effect but a long half-life, which can result in accumulation of the drug with repeated dosing. Because of this fact, it is reasonable to use other short-acting opioids and titrate to therapeutic effects and then transition to methadone using the equianalgesic table. It is important to understand that the equianalgesic table is designed for management of acute pain and not for long-term use. Conversion from other opioids to methadone using the equianalgesic table will result in an inappropriately high dose of methadone. If one is to use the equianalgesic table to convert from another opioid, the dose of methadone should be reduced by 50% or more. Rather than using an alternative opioid and transitioning to methadone, one can use methadone for acute pain by prescribing it initially every 2 to 3 hours for 2 to 5 days, at which point the blood levels will approach a steady state, then reduce the methadone dose by 50%. Without a dose reduction, there is a risk of methadone overdose. For providers unfamiliar with methadone, it may be reasonable to seek consultation with a pain specialist before using this drug.
Tramadol is a centrally acting synthetic analgesic classified as an opioid because it is a weak agonist at the mu opioid receptor. Unlike most opioids, it also appears to block the reuptake of serotonin and norepinephrine, so one would not expect naloxone to fully reverse its effects [6]. However, this activity on serotonin and norepinephrine makes it useful in neuropathic pain. Tramadol, unlike other opioids, is not a controlled substance in the United States. Tolerance and dependence is rare. At the usual prescribed dose of 50 mg by mouth every 4 to 6 hours, its analgesic effect is similar to acetaminophen 300 mg with codeine 30-mg [12]. Side effects, which can be ameliorated if the dose is gradually titrated over several weeks, include nausea, dizziness, dry mouth, sweating, and seizures. At doses greater than 400-mg daily, tramadol is associated with increased risk of seizures. Concurrent use with serotonin selective reuptake inhibitors or tricyclic antidepressants (TCAs) cannot only increase risk of seizures, but also risk the development of serotonin syndrome.

Patient-controlled analgesia

In patient-controlled analgesia (PCA), an infusion pump delivers opioids intravenously (IV). Patients push a button to activate IV opioid boluses. A preset dose of opioid is delivered as long as a predetermined amount of time (the lockout period) has elapsed since the previous dose. PCA is particularly useful in postoperative acute pain because it minimizes any delays in treating pain. Compared with patients who receive opioids for acute pain via other methods, patients on PCAs tend to use less total amounts of opioids because drug delivery more closely matches their pain pattern. When starting a PCA, set the hourly limit at three to five times the projected hourly requirement. For example, if you believe a patient will need 10 mg of a drug per hour, set the hourly limit at 30 mg to 50 mg. Observe the hourly consumption and adjust the hourly limit based on actual need. For opioid-naive patients with acute pain, the usual starting dose for morphine is 1 mg (range 0.5 mg–2.5 mg) with a usual lockout of 8 minutes (range 5–10 min). Setting a lockout time of 8 minutes means a patient would receive no more than one dose of narcotic every 8 minutes, regardless of how often they push the PCA button. The usual starting dose for fentanyl is 10 mcg (range 10 mcg–50 mcg), with usual lockout of 6 minutes (range 5–8 min). The usual starting dose of hydromorphone is 0.2 mg (range 0.05 mg–0.4 mg) with a lockout of 8 minutes (range 5–10 min) [6].

In addition to bolus dosing, clinicians can also administer a continuous (or basal) narcotic infusion. Basal infusions may be particularly useful in opioid-tolerant patients (eg, chronic cancer pain), but their use in opioid-naive patients with acute pain is controversial. Basal infusions increase the incidence and severity of opioid-related side effects and increase the risk of overdose. In opioid-naive patients, there is inconclusive data to support improved pain control [13]. When weaning patients off the PCA, it is important to start oral long-acting, controlled-release opioids (eg, morphine or oxycodone) at least 12 hours before discontinuation of the PCA. Start
with a dose of long-acting opioid, which is equivalent to the amount of drug given by continuous infusion, plus one half of the total dosage given by intermittent boluses [14]. Over the next 24 hours, wean off the PCA by increasing the lockout period and decrease the dose of drug given by boluses. PCA is not appropriate for all patients. Patients who are unable to understand the principle or have altered mental status are not candidates because they may receive inappropriate amounts of drug.

Pain treatment principles

Make every effort to individualize analgesic dose, route, and schedule. There is significant individual variation in doses of opioids required to provide sufficient analgesia in individuals across all age groups, between genders, among ethnic groups, and in opioid-naive and opioid-experienced patients. Frequently re-evaluate patients, especially when one is beginning or changing analgesic regimens.

Route of delivery

Although the oral route of administration is typically most acceptable, IV bolus administration produces the most rapid onset of effect for patients in acute pain. Opioids given rectally or subcutaneously also provide rapid onset of action, but some patients may find the rectal route of administration unacceptable; only limited drug amounts can be delivered in the subcutaneous space. Intraspinal (epidural, intrathecal) administration of opioids is a more invasive route of administration but can produce analgesia with small doses of opioids, as the drug is being delivered directly to the spinal cord. Fentanyl is the only opioid which is also available in oral transmucosal and transdermal formulations. The fentanyl lozenge is indicated only for cancer-related breakthrough pain. Approximately 25% of the dose is absorbed via the buccal mucosa and the remainder of the drug is absorbed from the GI tract. Unlike the lozenge, the transdermal fentanyl patch is indicated for continuous analgesia. The onset of analgesia may occur in 12 to 16 hours but it often takes up to 48 hours to achieve steady-state blood levels [15]. Providers should titrate with short-acting opioids for analgesia before converting to the transdermal fentanyl patch. Unlike the patch, the fentanyl patient-controlled transdermal system (PCTS) is designed for management of acute pain. Similar to a PCA pump, the PCTS delivers fentanyl on demand but does so without an IV line. Unlike the patch, there is no passive absorption of drug. The credit card size PCTS delivers drug only when the patient pushes a button to activate the device. One disadvantage of the device is that it is preprogrammed to deliver a fixed dose of 40 mcg, which may not be appropriate for all patients.

Frequency of dosing

Regardless of the drug choice and route of administration, the frequency of administration is critical to achieving the analgesic effect. Providers
should be familiar with each drug’s recommended schedule. For example, short-acting oxycodone should be administered every 3 to 4 hours. Providers who want to decrease the total daily dose should decrease the drug dosage rather than increase the interval between doses. Less frequent administration would result in break-through pain. Whenever possible, providers should anticipate pain and treat it in a prophylactic manner. Give analgesics before procedures and never rely only on “as needed” dosing of analgesics when you know the patient can expect pain.

Managing side effects

Providers should also anticipate, recognize and treat side effects associated with analgesics. For example, respiratory depression, itching, sedation, constipation, nausea, and vomiting are frequent opioid side effects. When prescribing opioids, providers should discuss these potential side effects with patients. Providers can choose an opioid based on its potential side-effect profile. For example, morphine tends to release histamine more than other opioids, with resultant itching and flushing. Choose an alternative opioid if this side effect occurs. Another method to minimize side effects is to increase the frequency of opioid dosing, which will result in more constant blood levels. It is the peak serum drug levels that are often associated with side effects. Providers can also decrease the incidence of side effects without sacrificing analgesic relief by using combination pharmacotherapy. One can often use less opioids by adding nonnarcotic adjuvants to the regimen. To address the issue of opioid-induced constipation, providers should initiate routinely scheduled stool softeners and cathartics at the same time they initiate opioid therapy. There are also new peripheral opioid receptor antagonists (eg, methylnaltrexone) on the horizon that may help providers address this issue of opioid-induced constipation.

Tolerance, withdrawal, and addiction

Providers must always monitor for opioid tolerance, expect physical dependence, and prevent withdrawal [6]. Within days of starting an opioid, expect some tolerance. Tolerance is the need to increase the amount of a drug to achieve the same effect. Tolerance occurs to some opioid-induced side effects, such as nausea, respiratory depression, and somnolence. Unfortunately tolerance to constipation does not occur. Tolerance to opioid-induced analgesia can also occur in the first 2 weeks of therapy but typically does not occur after that. Patients who experience increased pain after the first 2 weeks of opioid therapy should be evaluated for other causes of pain.

Physical dependence, which also occurs with drugs other than analgesics, is common in patients who take opioid analgesics. Patients with physical dependence develop withdrawal symptoms when a given medication is abruptly withdrawn. Symptoms of opioid withdrawal include anxiety, irritability, tachycardia, abdominal pain, nausea, and vomiting. The drug’s half-life
dictates the time course of the symptoms. To prevent withdrawal symptoms, wean patients, rather than abruptly discontinuing chronic opioid therapy. Do not confuse opioid dependence with opioid addiction. Opioid dependence can occur after just 2 weeks of opioid use. Addiction is an abnormal behavioral condition that may include compulsive use, impaired control over drug use, continued use despite harm, and craving [16]. The risk of iatrogenic opioid addiction is low and should not dissuade clinicians from using opioid analgesics to treat acute pain. “Pseudo-addiction” is a term that describes patient behavior when their pain is under-treated [6]. Patients with insufficiently controlled pain may seem focused on obtaining additional drugs. This may even include illicit drug use and deception as part of his or her efforts. If a patient has pseudo-addiction, rather than addiction, such behaviors will go away if the pain is appropriately treated.

**Adjuvant analgesics**

There are a number of drugs which, when given with narcotics or NSAIDs, can enhance the analgesic effects. These drugs are called adjuvants or coanalgesics. Drugs include the TCAs (eg, amitriptyline, imipramine, desipramine, and nortriptyline), antiepileptic drugs (eg, gabapentin, carbamazepine, topiramate, levetiracetam, oxycarbazapine, phenytoin, lamotrigine, zonisamide, and valproate), glucocorticoids, local anesthetics (eg, lidocaine), benzodiazepines (eg, diazepam, lorazepam, and clonazepam), skeletal muscle relaxants (eg, tizanidine), antihistamines (eg, hydroxyzine), antispasmodil agents (eg, baclofen), and caffeine. Many of these drugs are most effective when used in conjunction with opioid analgesics to treat specific types of pain. Their concurrent use can often result in improved pain control along with reduction in required doses of opioids. Most of these drugs are administered orally, but some are available by injection or topical formulations (eg, lidocaine). Each has potential advantages and possible side effects. For example, in patients with cancer-related pain, glucocorticoid administration cannot only relieve pain but also increase appetite and elevate mood. Glucocorticoids are particularly useful in alleviating pain related to headache caused by brain tumors, pain caused by spinal cord compression, or malignant infiltration of lumbar and brachial plexus. Chronic use, however, can lead to numerous side effects, including osteoporosis, Cushing’s syndrome, and increased risk of GI bleeding. For the terminally ill, such chronic side effects should be of no concern. Care must be taken not to withdraw glucocorticoids rapidly as that can result in pain exacerbation.

**Treating pain in the elderly**

Elderly patients have an increased risk for complications related to treatment of pain. Analgesics may have prolonged effects caused by decreased
elimination from the plasma [17]. For example, elderly patients may be twice as sensitive to parenteral fentanyl as compared with younger patients. When treating elderly patients with pain, consider initiation of opioids at dosage levels 25% to 50% lower than normal adults and titrate carefully. Close monitoring and careful titration can minimize adverse effects. Some drugs should be used with extreme caution or not used at all in the elderly. The Beers Criteria for potentially inappropriate medication use in older adults lists numerous analgesics [18]. Most of these drugs are listed because of narrow therapeutic windows or adverse CNS effects, which may contribute to falls or other adverse events. Drugs listed include the opioids propoxyphene, pentazocine, and meperidine. They discourage the long-term use of all NSAIDs and short-term use of indomethacin and ketorolac. They advise against the use of specific adjuvants, including long-acting benzodiazepines, higher doses of short-acting benzodiazepines, and TCAs.

Neuropathic pain

Neuropathic pain is relatively common. Causes include postherpetic neuralgia, diabetic neuropathy, chemotherapy-induced neuropathy, alcoholic polyneuropathy, spinal cord injury pain, and complex regional pain syndrome (reflex sympathetic dystrophy). The diagnosis of neuropathic pain is based primarily on the pain description. Patients with neuropathic pain are more likely to describe their pain as “burning, tingling, electric shock, cold, pricking and itching” [19]. Physical examination can reveal flushing, sweating, fasiculations, or atrophy. Unfortunately there is no single test that confirms neuropathic pain. Identification of pain as neuropathic is important because not all analgesics are effective in treatment of neuropathic pain. For example, topical lidocaine, antidepressants (TCAs, venlafaxine, duloxetine), antiepileptic drugs, tramadol, gabapentin, and pregabalin appear effective in managing neuropathic pain. NSAIDs can reduce pain in diabetic neuropathy and sciatica [20,21] but, like acetaminophen, appear less efficacious in treatment of other causes of neuropathic pain [22]. The role of capsaicin cream is uncertain and often produces pain when applied [23]. Noninvasive therapies, such as transcutaneous electrical nerve stimulation, can be effective in some patients with diabetic neuropathy and radicular pain [24,25]. Patients with recalcitrant neuropathic pain may benefit from a referral to a pain specialist to discuss more invasive therapy, such as sympathetic regional anesthetic blocks, intrathecal medication (opioids or baclofen), or spinal cord stimulation. The use of oral opioids in treatment of neuropathic pain often requires very high doses. In some patients, the development of side effects at high doses precludes their use. Intrathecal opioids require much smaller doses because the opioids are delivered directly to the dorsal horn of the spinal cord.

It is important to recognize that in some patients, neuropathic pain can coexist with nociceptive pain. For example, patients with radicular and
low back pain may experience pain from both myofacial causes and from nerve root compression [22]. Up to one third of patients with cancer pain have a component of neuropathic pain in addition to their coexisting nociceptive pain [6].

**Nonpharmacologic pain therapy**

Physical measures, such as application of heat or cold, are often underutilized in management of pain in hospitalized patients. Applying heat or cold is easy and can be an effective way to minimize unnecessary escalating doses of pharmacologic therapy and their potential side effects.

Although providers often think of complementary and alternative therapies as outside the mainstream, their use is widespread among patients. These include mind-body therapies (eg, relaxation response, biofeedback, prayer, imagery, and meditation), manipulative therapies (eg, massage, chiropractic and osteopathic manipulation) and new-age therapies (eg, Qi Gong, Reiki). Surveys have demonstrated that physicians are often unaware of their patients’ use of alternative therapies [26]. Not unlike asking about pain, it is important for providers to ask patients about their use of alternative therapies. These questions can be insightful, revealing a patient’s thoughts on why they have pain and belief on whether traditional medicine can address their pain sufficiently.

**Treating pain safely: Joint Commission’s 2007 National Patient Safety Goals**

The Joint Commission’s latest National Patient Safety Goals include goals applicable to pain management [27]. Management of pain in the hospital is a complex system and dangerous medical errors can occur. For example, one Joint Commission on Accreditation of Healthcare Organization goal is to “standardize a list of abbreviations, acronyms, symbols and dose designations that are not to be used throughout the organization.” For example, providers should never abbreviate morphine sulfate because “MSO4” can be confused for magnesium sulfate and vice versa. When writing drug dosages, always write a “zero” before any decimals and never write any “trailing zeros.” Write morphine 0.3 mg and never write “.30”. Another Joint Commission goal is to “identify and, at a minimum, annually review a list of look-alike/sound-alike drugs used by the organization.” For example, some health care providers have mistaken immediate-release oxycodone with controlled-release oxycodone. Overdose or under dosing has occurred with adverse patient outcomes. One strategy to address this problem is to encourage brand name prescribing of controlled-release oxycodone. Another strategy is to stock the products in different locations away from one another.
Summary

Effective management of acute pain should be a primary goal of each health care provider. Acute pain is a complex medical problem with multiple possible etiologies. Management of acute pain requires providers to not only understand how to assess pain, but also how to apply pharmacologic and nonpharmacologic therapies to address pain. There are many barriers to the effective management of pain in the hospital. As leaders of the hospital care team, it is incumbent on hospitalists to overcome these barriers and address the challenge of acute pain in hospitalized patients.

References


