The pathophysiology of pain & prehospital treatment options

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Editor’s note: This article, which focuses on the pathophysiology of pain and various treatment options, kicks off a comprehensive section on pain management in this issue. For a more detailed examination of the prehospital assessment of patients in pain, read “The Face of Pain,” our CE article beginning on p. 74.

Many, if not most, medical conditions or injuries cause pain. In fact, pain is the No. 1 reason people seek health care. Pain is a protective mechanism and occurs whenever any body tissues are being damaged. The pain reflex will eventually cause the patient to react to remove the painful stimulus. This reaction may be rapid, as when you touch a hot pan, or slow, as when you remain seated in the same position for a long period of time. Regardless of the duration, the mechanism is the same.

In the case of the hot pan, tissues in the hand are damaged from the heat, which results in a pain sensation and a reflex to withdraw the hand from the heat. Likewise, remaining seated in the same position for an extended period of time can cause tissue destruction because of lack of blood flow to the skin. When this tissue damage is perceived, the person will shift their weight subconsciously. However, people with spinal cord injuries can’t sense pain from the pressure area, can’t move to eliminate the pressure or experience a combination of both. As a result, they have an increased risk of developing pressure (decubitus) ulcers.¹

One of the oldest roles of medical practitioners is to help alleviate pain and provide comfort for the patient. Pain relief, without a loss of consciousness, is referred to as analgesia. We can provide analgesia through numerous interventions, including drugs, surgical procedures or other modalities that either eliminate the source of the pain or block the pathways that transmit pain impulses to the brain.

In addition to subconscious reflexes, pain elicits a very strong emotional response that is often recorded in our memory. Mark Twain once summed this up well when he stated that “the cat that sits down on a hot stove-lid ... will never sit down on a hot stove-lid again—and that is well; but also she will never sit down on a cold one anymore.”

Analgesic practices vary significantly across the country, and, unfortunately, analgesia is not equally provided to those in need. The reasons behind this are numerous, but, in most cases, unfounded. One study found that Hispanic patients with isolated long-bone fractures treated at a major teaching hospital in Los Angeles were twice as likely to receive no pain...
medication when compared to their non-Hispanic, white counterparts. The same author studied black patients with isolated long-bone fractures treated in a major teaching hospital in Atlanta and found they were less likely to receive adequate analgesia when compared to their white counterparts. A national survey study found that only half of burn patients treated in emergency departments (EDs) received adequate analgesia for their burn pain.

Pain in the prehospital setting
Pain relief is an important and compassionate aspect of prehospital care. Unfortunately, pain in the prehospital setting often goes unidentified, under-treated or both. Example: As a rule, patients with extremity fractures receive inadequate analgesia in the prehospital setting. In fact, in one study, researchers examined the records of 1,073 patients with isolated extremity fractures and found that only 1.5% received analgesia in the prehospital setting.

A recent case conference detailed two instances in which base station physicians refused paramedic requests to administer analgesia to victims of extremity fractures because they feared it would alter the patient’s ability to consent for potential surgery after the patient arrived at the hospital—an unfounded concern. (See “Myths of Prehospital Analgesia,” p. 72.) In another study, only 18.3% of eligible patients received analgesia for lower extremity fractures (including isolated hip fractures) in the prehospital setting. Isolated hip fractures are among the most common orthopedic emergencies encountered by EMS personnel. Despite this, only a modest proportion of these patients receive prehospital analgesia for this painful and debilitating injury.

This article offers an extensive background of the pathophysiology of pain, the various treatment regimens available in the prehospital setting and treatments that may become available in the future (p. 68).

Pathophysiology of pain
The generation of a painful sensation involves an intricate interaction among all parts of the nervous system. Medical knowledge as to how the body senses and interprets pain has increased dramatically in the past 15–20 years. Researchers have mapped out pain-generation pathways and have discovered some of the changes that take place during the development of chronic pain. Pain has been identified as something more than just a feeling or sensation; it’s also linked to the complex psychosocial factors that surround traumatic events.

Before discussing the pathophysiology of pain, suffering is a negative response induced by pain, fear, anxiety, stress, loss of a loved object & other psychosocial events.
ology of pain, we need to first define several terms. First, *nociception* (derived from the word *noxious*, meaning harmful or damaging to the tissues) is a mechanical event that occurs in tissues undergoing cellular injury. A nociceptive stimulus is detected by specialized sensory organs attached to nerve endings in the tissue and relayed to the brain by the nervous system. Pain receptors are principally free nerve endings that are widespread throughout superficial layers of the skin and in the walls of selected internal tissues. Most deep tissues are not well supplied with pain fibers.

Three types of stimuli excite pain receptors: *chemical, mechanical and thermal*. The intensity of pain often corresponds with the rate of tissue destruction. As mentioned previously, pain is an unpleasant sensory and emotional experience associated with actual or potential tissue damage. In short, pain is the brain’s interpretation of a nociceptive stimulus. Several links between the sensory portions of the brain and emotional portions of the brain are hardwired into the nervous system, providing the emotional and long-term psychological response to nociceptive stimuli.

*Acute pain* is defined as pain associated with an acute event. *Chronic pain* is that which persists after the acute event is over. Some clinicians use six months as a time period during which residual pain from an acute event can be termed chronic. As we discuss later in this section, the line between acute and chronic pain is blurred by events that take place in response to nociceptive stimuli. *Suffering* is a negative response induced by pain, fear, anxiety, stress, loss of a loved object and other psychosocial events.

Now that you’re familiar with some of the definitions used in studying pain, let’s look at the generation of a painful response. Locally, sensory nerve endings are located throughout the skin, muscles, bones, connective tissues and organs. The *somatic sensory system* carries sensory input to the brain from the muscles, bones, skin and connective tissues of the body. Pain produced from a nociceptive stimulus received by this system is often called *somatic pain*. Examples: pain produced by a laceration or a fractured tibia.

The *visceral sensory system* carries sensory input to the brain from the hollow and solid organs in the body. Pain produced via this system is often called *visceral pain*. Examples: pain associated with a kidney stone, cholecystitis or a myocardial infarction.

The *somatic sensory system* has two components: a *proprioceptive* component that detects touch, vibration and position and a *nociceptive* component that detects tissue damage. The proprioceptive system is very sensitive, and the sensory organs require significantly less pressure to send a message to the brain. For example, hold your hand out with the palm facing the ceiling, close your eyes and have a friend lightly place a pencil in your palm. You should be able to feel the pencil, weighing only an ounce, as it sits in your hand. Now, put the pencil down and with your eyes closed, touch the tip of your nose with your index finger. If your proprioceptive system is working properly, you’ll be able to touch the tip of your nose and not poke yourself in the eye.

Opiates, the most commonly used centrally acting analgesics, can relieve both physical and emotional pain. Opiates, the most commonly used centrally acting analgesics, can relieve both physical and emotional pain.
divided into two subsections: fast warning (fast pain) and slow warning (slow pain). Fast pain is also described as sharp, electric or acute. Slow pain is usually described as slow and burning, dull, aching, throbbing or chronic.

The actual neurons that make up these components have different characteristics. The fast warning system is linked to the spinal reflexes and fires almost immediately when tissue damage or the threat of tissue damage occurs. In some cases, the spinal reflex occurs so fast that the affected body part is pulled away from danger before the brain interprets the stimulus as painful. The fast warning system can generally well-localize the pain immediately after the stimulus. The motor reaction mediated by the fast warning system is often exaggerated and somewhat poorly controlled because the nerve impulse is processed in the spinal cord and not the brain, which equates to less fine motor control. The slow warning component transmits the stimulus at a slower rate and is believed to be responsible for initiating repair and protection of the affected part.

When a nociceptive stimulus is generated at the sensory nerve ending, the stimulus is sent upstream to the spinal cord. Although the nociceptive message is relayed to the spinal cord, events take place within the tissues that produce a local sensitization to the nociceptive message. Chemicals are released from the nerve ending that initiate the process of inflammation and repair. These inflammatory changes produce the swelling, redness and warmth that occur after injury.

These same chemicals stimulate other local nociceptive nerve endings, causing these nerves to send a nociceptive message to the spinal cord, ensuring the message is received and interpreted correctly. The inflammation and recruitment of additional sensory nerves cause the person to limit activity of the injured body part, protecting it from further harm.

The nociceptive message is received by the spinal cord and will produce changes local to the spinal cord. The message is passed to other neurons that relay the message up to the brain.
Changes occur in these spinal cord neurons that also sensitize the person to pain, again as a protective reflex against additional injury.

The spinal cord has several mechanisms that attempt to both provide natural analgesia and limit the nociceptive response in an attempt to prevent overstimulation.

Natural analgesia occurs from the release of endorphins and enkephalins, specialized chemicals in the spinal cord and brain that are the natural equivalent to opioids. These chemicals bind to the same receptors that morphine and other opioids bind to within the nervous system. Natural endorphins are the reason why an injury sometimes doesn’t cause an initial pain response, but pain then becomes worse 20–30 minutes later as these natural compounds wear off. The brain also attempts to prevent overstimulation by sending messages back down to the spinal cord to decrease the number of nociceptive messages sent to the brain. On a more local level, light touch sensory nerves near the site of injury also limit the amount of nociceptive information produced by the injured body part. This is why rubbing a body part that has just sustained a minor injury can decrease the amount of perceived pain.

The most complex portion of the nociceptive pathway lies within the brain itself. Within the brain, complex interactions take place to interpret the stimulus as painful and to produce the analytical (“Ouch, my leg is damaged”) and emotional (crying, anger and suffering) responses to the pain.

Both long- and short-term memory are involved in storing this painful event and sometimes replaying prior events. The sympathetic nervous system is also stimulated to produce many of the fight-or-flight reactions that normally accompany a painful event, including increased heart rate, respiratory rate and/or blood pressure.

Several biological changes responsible for the development of chronic pain may occur in these pathways. Genes located in the neurons change and can make both the peripheral and spinal cord neurons more sensitive to a stimulus for the life of that neuron. The connecting neurons
in the spinal cord can become more sensitive so that even a small nociceptive message can produce a significant response, including interpretation of significant pain.

In the brain, a similar nociceptive message can trigger an emergence of a past event that increases the pain response to the current nociceptive message. These changes are responsible for the development of chronic pain syndromes in some patients. Some of these physical and biochemical changes begin within 20 minutes of a large nociceptive stimulus. The rest of the changes can occur within hours to days of the event. Several studies have noted that patients who receive early and effective analgesia, even before a surgical procedure, require less overall analgesia than those who initially receive poor analgesia.

Even though chronic pain is arbitrarily defined as pain remaining six months after the inciting event, the changes that set the patient up for chronic pain take far less time to occur. Effective pain management can prevent many of the changes that produce chronic pain. This is why prehospital pain assessment and effective analgesia are important.

Assessment of the pain patient
Pain is one of the most common patient complaints encountered by EMS personnel. In fact, pain is typically what con-
cerns the patient the most; the injury or illness causing the pain is often of secondary concern. The way in which one experiences pain depends upon numerous factors, including cultural conditioning.

Although it would be wrong to stereotype any patient, many cultures encourage patients in pain to express their pain, while others encourage them to hide it. Thus, it might be easy to assume that a patient who expresses their pain verbally and emotionally is suffering more severe pain than a stoic patient. This assumption often proves false. Because of cultural influences, it’s important to inquire about the intensity of the patient’s pain and to use objective systems for measuring or quantifying the patient’s pain. Use a pain scale, such as FACES, CRIES or the 10 scale, if possible. (See “The Face of Pain,” p. 74.)

Other elements that influence pain perception include physical, emotional, social and genetic factors. Age also plays a role. (These factors, methods of assessing pain and assessment tools are discussed in more detail in “The Face of Pain.” See p. 74.)

Prehospital pain management strategies

Pain treatment involves removing or correcting the source of the pain, blocking or attenuating the transmission of pain impulses to the brain or a combination of the two. It can be divided into one of two categories: non-medication and medication therapies.

Non-medication therapies, such as recognition and empathy, distraction, muscle relaxation, position of comfort, temperature regulation and physical therapies, can be quite effective. (These are discussed in more detail in “The Face of Pain,” p. 74.)

The mnemonic RICE offers a
way to remember some important non-medication therapies that may decrease a patient’s pain:

- **Rest:** Have the patient avoid using the affected part to avoid further discomfort or injury;
- **Ice:** Apply ice (bags with crushed ice, cold packs, etc.) to the injured area for the first 24–48 hours to prevent or reduce swelling;
- **Compression:** Wrap an elastic bandage around the injured area to secure the ice in place. Don’t wrap it so tightly that the circulation is cut off. After 10–15 minutes, loosen the bandage, and remove the ice. The patient can reapply ice for 15–20 minutes every one or two hours for the first six hours after the injury. As long as the injury is swelling, the patient should continue to apply ice three to four times a day; and
- **Elevation:** Elevate the injured area above the level of the heart to slow the blood flow to the injury.

Treating the underlying cause of a patient’s pain is a definitive care procedure. **Example:** Placing a patient’s femur fracture under traction reduces their pain significantly. This serves to minimize damage to adjoining tissues and lessens the sensation of pain.

### Medication therapies

Medications that relieve pain are referred to as analgesics. These are generally classified as peripherally or centrally acting. The detection of pain almost always occurs in the peripheral tissues. The pain impulse is transmitted to the brain through the use of chemical messengers and nerves. In addition, localized inflammation, swelling and blood flow are affected by chemicals released secondary to the injury. Thus, some medications, such as non-steroidal anti-inflammatory drugs (NSAIDs), act to block these peripheral effects. Through this, they ease pain, suppress inflammation and provide other beneficial effects for the pain patient.

Centrally acting analgesics affect some portion of the central nervous system. The most commonly used centrally acting analgesics are opiates, which act on specialized receptors within the brain and can relieve both physical and emotional pain. Opiate receptors have differing shapes that influence how well a particular opiate drug interacts with the receptor. The opiate receptors have been designated **mu (µ), delta (δ), sigma (σ), kappa (κ) and epsilon (ε).**

The mu receptors produce euphoria, analgesia and respiratory depression. More specifically, the mu-1 receptors produce analgesia, while mu-2 receptors produce constipation, euphoria, physical dependence and respiratory depression. The delta receptors also produce analgesia. The sigma receptors stimulate the respiratory system and vasomotor activity. They also cause hallucinations and dysphoria. The kappa receptors affect spinal analgesia, sedation and pupillary constriction. The epsilon receptors also
produce analgesia. Opiates, such as morphine, have an affinity for the mu and kappa receptors.10

**Peripherally acting agents**

Although several agents affect the peripheral transmission of pain, the most frequently used medications are NSAIDs. Following cellular and tissue injury, cells release specialized chemicals called prostaglandins, which are responsible for many localized reactions that occur following injury. NSAIDs interfere with prostaglandins, lessening their effect. In addition, NSAIDs are effective in reducing fever.

The first NSAID identified was aspirin. Aspirin is an effective analgesic and anti-inflammatory and still plays a major role in emergency care because it inhibits platelet aggregation. Numerous NSAIDs have been developed and are among the most prescribed drugs in modern medical practice. However, only one NSAID is available in the United States for parenteral injection; thus, the use of NSAIDs for the treatment of prehospital pain is limited.

**Ketorolac (Toradol)**, the only injectable NSAID, has analgesic, anti-inflammatory and antipyretic effects. A peripherally acting agent that does not affect mental status or cause respiratory depression, it’s used for moderate to severe pain—particularly orthopedic and soft tissue injuries. Proven effective in the treatment of ureteral colic (kidney stones), it’s believed to aid in relaxing the ureteral spasm associated with kidney stone passage. It is often used with centrally acting agents, such as morphine, because pain is both a peripherally and centrally mediated event.11

When administered intravenously, ketorolac has an onset of action of less than 30 minutes. Peak effects occur in 45–60 minutes, and the duration of effect is four to six hours. The standard IV dose for an adult is 30 mg.

**Centrally acting agents**

The most frequently used centrally acting analgesics are opiates. In addition, several chemical derivations serve as effective analgesics. Although numerous medications fall within this class, we’ll limit our discussion to those agents most often used in prehospital care.

**Opiates**

Over the past two centuries, opiates have been the mainstay for analgesia in medical practice. Opiates were initially derived from parts of the opium poppy plant (*Papaver somniferum*), although most today are synthetically manufactured. Opiates act on the mu, kappa and delta receptors in the brain; inhibit pain; and cause sedation and respiratory and cardiovascular depression.

Opiates have multiple actions, but exert their primary effects on the central nervous system and organs containing smooth muscle. The principal actions of therapeutic value are analgesia and sedation. A significant feature of
the analgesia is that it occurs without loss of consciousness.

Opiates also suppress the cough reflex and cause respiratory depression, mood changes, mental clouding, euphoria, dysphoria, nausea and vomiting. Opiates depress the cough reflex by direct effect on the cough center in the medulla. They produce respiratory depression by direct effect on brain stem respiratory centers. The mechanism of respiratory depression also involves a reduction in the responsiveness of brain stem respiratory centers to increases in carbon dioxide tension (pCO₂). Opiates also cause pupillary constriction (meiosis). Pinpoint pupils are a common sign of narcotic overdose, but can be caused by other factors.

Opiates decrease gastric, biliary and pancreatic secretions. They cause a reduction in gastric motility, delay digestion of food in the small intestine and, subsequently, decrease propulsive peristaltic waves in the colon, leading to constipation. Some opiates, particularly morphine, can cause a marked increase in biliary tract pressure as a result of spasm of the sphincter of Oddi (the valve that allows bile to enter the small intestine from the biliary tract).

Certain opiates, particularly morphine, produce peripheral vasodilation, which may result in orthostatic hypotension. Release of histamine may occur with some opiates and may contribute to narcotic-induced hypotension. Other manifestations of histamine release include pruritus, flushing and red eyes.

**Morphine** is among the oldest drugs in this class. The name is derived from the word *Morpheus*—the Greek god of dreams. It is a naturally occurring substance in the poppy plant.

Morphine, one of the most frequently used opiates in emergency medicine, is used for moderate to severe pain and provides effective analgesia and sedation. In addition, morphine causes venous system dilation, which decreases cardiac preload. This makes it an attractive treatment for pulmonary edema and congestive heart failure.

Morphine’s onset of action when given intravenously is less than five minutes with peak effects occurring within 20 minutes. The duration of effect is up to seven hours. The IV dose ranges from 2.5–10 mg.

A synthetic opiate that does not chemically resemble morphine, meperidine (Demerol) has a potency about one-tenth that of morphine and is used for the treatment of moderate to severe pain. Like morphine, meperidine can depress respirations. It tends to cause greater histamine release than does morphine or fentanyl and, thus, may cause more side effects.

Meperidine also tends to cause more euphoria than some of the other agents. Drugs that cause more euphoria tend to be more abused than those that don’t. Because of this, and based on the fact that other available opiates are equally effective, many EDs and EMS organizations have removed meperidine from their formulary.

Meperidine’s onset of action when administered intravenously is five minutes. Peak effects are obtained in less than 30 minutes, and the duration of effect is two hours. IV dosages range from 25–100 mg.
A synthetic opiate effective in treating moderate to severe pain, hydromorphone (Dilaudid) is eight to 10 times more potent than morphine and has a more rapid onset of action, but a shorter duration of effect. Hydromorphone reportedly produces less nausea and vomiting than morphine.

Hydromorphone’s onset of action when given intravenously is less than five minutes. Peak effects are reached within 30–90 minutes, and the duration of effect is four to five hours. The IV dose ranges from 1–4 mg.

Fentanyl (Sublimaze) is a synthetic opioid, chemically unrelated to morphine, with short-acting analgesic activity after IV or subcutaneous administration. Its pharmacological effects and degree of analgesia are similar to those of morphine. Fentanyl is used to treat moderate to severe pain. Its duration of action is shorter than both meperidine and morphine. Because of its short duration of action, the side-effect profile is better for fentanyl when compared to morphine.

Initially used as an adjunct to anesthesia, fentanyl does not seem to cause respiratory depression to the same degree as other opiates. In addition, it does not appear to cause a significant release of histamine—and, thus, has fewer side effects.12

Because of its rapid onset and short duration of action, fentanyl is now routinely used in emergency medicine and, to a lesser degree, in prehospital care.13 Because of fentanyl’s lack of hemodynamic effects, it’s used in the treatment of multiple trauma patients.14 It has proven effective in the prehospital treatment of pediatric patients with moderate to severe pain. In fact, in one study, no untoward events occurred during five years of prehospital fentanyl administration to air-transported trauma victims younger than 15.15

Fentanyl’s onset of action is immediate when given intravenously. Peak effects occur within three to five minutes, and the duration of action is 30–60 minutes. The IV dosage ranges from 50–100 micrograms.

**Synthetic opiate agonists/antagonists**

An opiate subclass includes synthetic compounds with both agonistic and antagonistic properties. That is, they activate some opiate receptors but block others. This effect reportedly decreases the likelihood of abuse and helps prevent respiratory depression and other side effects.

Nalbuphine (Nubain) is a mixed narcotic agonist-antagonist with minimal hemodynamic and respiratory effects. The antagonist properties of nalbuphine significantly decrease its potential for abuse and, thus, in most states, it’s not a scheduled substance. Because of this, nalbuphine is widely used in prehospital care as an analgesic agent.

Initial studies suggested that nalbuphine was an effective alternative to morphine.16,17 However, subsequent studies have shown that prehospital use of nalbuphine may be problematic. In an English study of prehospital nalbuphine usage, researchers
found that it offered poor pain control for a high proportion of patients.18

Because of its antagonistic properties, prehospital nalbuphine therapy could be responsible for increased opiate requirements in patients once they arrive at an ED.19,20 It can also interfere with subsequent anesthesia induction and maintenance. Because of these findings, nalbuphine administration in the prehospital setting may not be in the patient’s best interests.

Nalbuphine’s onset of action is two to three minutes when given intravenously, with peak effects occurring within 30 minutes. The duration of effect is three to six hours. The IV dosage range is 5–20 mg.

Many EMS systems use butorphanol (Stadol) as a prehospital analgesic. Like nalbuphine, butorphanol has mixed agonistic and antagonistic properties. Because of these, it generally has a lower incidence of addiction and abuse and remains an uncontrolled drug in most states. Several years ago, an intranasal form of butor-
phanol (Stadol NS) was released for the treatment of headache. Studies found that many patients abused the drug in this form and became addicted.

Butorphanol has a side-effect profile similar to nalbuphine. It can interfere with later ED analgesia and anesthesia. The role of butorphanol in the prehospital setting has not been widely studied.

When given intravenously, butorphanol has an onset of action in less than one minute, peak effects in three to five minutes and a duration of effect of two to four hours. The dosage range is 0.5–2.0 mg IV.

Gasses
Nitrous oxide/oxygen (Nitronox): At high concentrations, nitrous oxide is an anesthetic. At lower concentrations, it’s an effective analgesic. Nitrous oxide has been widely used as an analgesic agent—initially for dentistry and childbirth. It was introduced into the prehospital setting in the late 1970s and has proven effective in treating several types of pain. In the United States, nitrous oxide is supplied in a two-cylinder delivery system (Nitronox) that mixes the nitrous oxide with oxygen at a 50:50 ratio. This is then fed to a demand valve through which the patient self-administers the gas. The unit is designed to shut down if the oxygen cylinder becomes depleted before the nitrous oxide cylinder. If the nitrous oxide cylinder becomes depleted before the oxygen cylinder, the device will allow the continued administration of 100% oxygen.

Nitrous oxide has proven effective at relieving numerous types of pain encountered in the prehospital setting. It can be used for chest or trauma pain and for analgesia during painful procedures, such as transcutaneous pacing. (Note: The National Association of EMS Physicians [NAEMSP] prepared a helpful position paper detailing the prehospital use of nitrous oxide analgesia.)

Summary
How can we better treat pain in the prehospital setting? First, it’s essential that prehospital personnel assess for the presence and severity of pain with every patient. Medical directors must become more aggressive with regard to prehospital pain management. Although the requirements to obtain and store controlled substances can be onerous, the standard of care in emergency medicine is to treat pain aggressively. Shifting prehospital analgesia decisions from online medical control contact to standing orders can help in the earlier provision of prehospital analgesia.

In a New Mexico study, the time interval from scene arrival to administration of the first dose of morphine decreased by 2.1 minutes when pro-
PAIN & COMFORT

When carrying patients to your waiting ambulance, position yourself (as this Clark County, Nev., firefighter did) so you don’t bump a patient’s injury or unnecessarily jostle them. This reduces the chances of causing more pain.

tocols were changed to allow paramedics to administer morphine to patients with isolated extremity trauma without first contacting online medical control. 27

Field personnel, EMS physicians, administrators and representatives from receiving hospitals should organize a comprehensive plan to ensure that adequate analgesia is provided in the prehospital setting. EMS is a compassionate profession, and compassion begins with the relief of pain and suffering. 28

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References


Does your agency document pain management efforts? Take the online poll at www.jems.com.


Several new methods of providing prehospital analgesia are in development or in use in other countries. Some of these are not available in the United States.

**Pharmacological interventions**

**Methoxyflurane (Penthrane) inhalers** have been used with great success for years in Australia. These devices came to the attention of EMS providers in North America following a segment from the television reality series *Survivor*, filmed in the outback of Queensland, Australia. In that segment, a show participant fainted and fell into a fire, sustaining burns to his upper extremities and trunk. Paramedics on the set quickly administered methoxyflurane through a Penthrox inhaler, and viewers could readily see that the patient’s pain improved.

Methoxyflurane is an inhalational anesthetic agent that has potent analgesic properties when used at low doses. A highly volatile liquid that quickly assumes the gaseous state when opened, it has a pleasant, fruity smell that is well-tolerated by patients.

In the United States, methoxyflurane was once used primarily in the veterinary market (trade name: Metofane). However, the manufacturer has stopped production of the drug altogether for the U.S. market. Thus, as of this writing, it’s unavailable in the United States. The decline of methoxyflurane use in the United States was due to reported cases of kidney and, to a lesser degree, liver toxicity. However, these occurred with anesthetic doses considerably greater than the analgesic doses used for prehospital care.

Medical Developments Australia (MDA) still manufactures the drug in Australia under the trade name Penthrane. (See photo above.) The Commonwealth of Australia has concluded that the drug is safe for analgesic therapy and approved it for use by ambulance services throughout Australia and for the Australian Defence Forces.

In Australia, methoxyflurane is used as an adjunct to standard analgesic therapy with morphine or nalbuphine. To administer methoxyflurane for analgesia, 3 mL of the drug is placed onto the wick of the Penthrox inhaler. The device is then gently shaken and any excess drug wiped off. The inhaler is given to the patient to self-administer. If needed, supplemental oxygen can be administered through the inhaler.

Pain relief usually commences after eight to 10 breaths and continues for several minutes after use. The patient should be instructed to use the inhaler intermittently to control pain. The initial dose should last approximately 25–30 minutes. This generally allows enough time to start an IV and administer a parenteral analgesic agent, such as morphine. Methoxyflurane should be used in a well-ventilated area. Following treatment, the inhaler is discarded.

For more information, visit www.asnsw.health.nsw.gov.au, and search for methoxyflurane on the link to A–Z health topics.

**Intranasal fentanyl:** Researchers in Australia have found that the intranasal administration of fentanyl is an effective analgesic for children between three and 12 years of age with acute pain. Administration via the intranasal route allows for early and significant reduction in pain without subjecting the child to needle-sticks and similar preliminary procedures.

In this study, children between three and seven years old received 20 micrograms of fentanyl, while children between eight and 12 years old received 40 micrograms intranasally. Additional 20 microgram doses were provided every five minutes as needed until adequate analgesia was obtained.

Intranasal fentanyl shows great promise for providing quality analgesia to children with acute pain in both prehospital and ED settings.

**Alfentanil (Alfenta)** is a short-acting synthetic opiate similar to fentanyl. Because it has a shorter duration of action, it doesn’t appear to have morphine’s adverse side effects. A Finnish study compared the administration of alfentanil to that of morphine in the prehospital treatment of acute, ischemic-type chest pain. The researchers found that alfentanil provided faster, effective pain relief when compared to morphine. No hemodynamic or respiratory side effects occurred.

**Tramadol**, sold under the trade name Ultram in the United States, shows promise as a prehospital analgesic agent according to a Belgian study. Tramadol is a synthetic analogue of codeine, has weak opioid agonist properties and appears to inhibit pain at the spinal level. It does have some abuse potential, but much less when compared with opiates. In most countries, tramadol is categorized as a non-narcotic drug.

Tramadol is about one-tenth as potent as morphine on a weight basis when given via IV. The onset of action, following administration, is within minutes, with the peak effects noted after 15–30 minutes. The duration of action is 4.5 hours. Tramadol causes very little,
if any, respiratory depression in analgesic doses, and cardiovascular side effects are minor. The most common side effects are dizziness, sedation, dry mouth and nausea.

In a double-blinded, controlled prehospital study, Belgian researchers randomly assigned patients to receive either tramadol (100 mg) or morphine (5 mg for patients weighing less than 70 kg or 10 mg for patients weighing more than 70 kg) via IV. If, after 10 minutes, additional analgesia was needed, the tramadol group would receive an additional 50 mg of tramadol, and the morphine group would receive an additional 5 mg of morphine. The researchers found that analgesia and side effects were similar in both groups and concluded that tramadol is an acceptable alternative to morphine in the prehospital trauma setting. IV tramadol is presently not available in the United States.

**Single-cylinder, nitrous oxide/oxygen mixtures**

Nitrous oxide remains a common analgesic agent in most Commonwealth countries. In the United States, the Food and Drug Administration requires that nitrous oxide analgesia be administered through a two-tank system (Nitronox) in which the separate gasses (nitrous oxide and oxygen) are fed into a blender and mixed at the proper 50:50 mixture. FDA requirements make the device considerably more bulky and expensive than similar devices found outside the United States.

In Canada, Australia and Great Britain, a 50:50 mixture of nitrous oxide/oxygen is provided in a single cylinder. This delivery system, commonly called Entonox or Dolonox, is considerably lighter, more compact and less expensive than the U.S. device. (See photos p. 71.)

A study compared the single-cylinder Entonox system with the twin-cylinder Nitronox system and concluded that, although Nitronox was a safer system in cold weather areas, it
was heavy and bulky, and Entonox was the preferred system of nitrous oxide/oxygen administration. Current FDA regulations forbid mixing nitrous oxide and oxygen in a single-cylinder.

**Non-pharmacological interventions**

A novel Austrian study recently examined the value of using acupressure for prehospital pain management of victims of minor trauma. Acupressure, a traditional Chinese pain treatment, is based on stimulating specific points on the body. In a prospective, randomized, double-blinded trial, patients were assigned to receive acupressure at “true points,” at “sham points” or “no acupressure.” At the end of transport, researchers found that patients who received acupressure at “true points” had less pain, less anxiety, a slower heart rate and greater satisfaction with the care provided. They concluded that acupressure is an effective, easy-to-learn pain treatment for prehospital care.

**References**


Myths of Prehospital Analgesia

By Jeff Myers, DO, NREMT-P

Myths are common in medicine; several relate to the EMS administration of pain medications. Numerous studies have confirmed that the medical profession as a whole does a poor job in providing appropriate analgesia. The prehospital setting is no different. Many myths have been cited as facts to excuse not providing appropriate analgesia in EMS and in the emergency department (ED). Let’s take some time to review and debunk these myths.

MYTH 1: If I give my patient narcotics, they won’t be competent enough to consent to surgery later.

A recent article in Prehospital Emergency Care provided an excellent discussion of this issue.1 The concern over rendering a patient incompetent by administering appropriate narcotic analgesia in the prehospital setting is unfounded. Although the legal specifics vary from state to state, the four general requirements for valid informed consent are: 1) the patient must have the capacity to understand the information and treatment options; 2) the patient must be able to clearly communicate their choice; 3) the patient must not be coerced into making a decision; and 4) the information must be presented in a way that the patient can comprehend the choices and consequences of those choices.

Evaluate patient competence individually on the basis of each patient’s mental status at the time of treatment and the type of decision to be made. Based on the four criteria, even individuals with mental handicaps can be competent to make certain decisions.

Fact: A person who has received narcotic analgesia may be able to more clearly consider treatment decisions than a patient who is experiencing severe pain. In some ways, withholding appropriate analgesia until after consent is obtained can be looked upon as coercion, where the analgesia is a “reward” for consenting to a procedure.

MYTH 2: If I give my patient narcotics for abdominal pain, it will change the physical examination findings, making diagnosis difficult.

The dogma of withholding analgesia for fear that it will alter an abdominal examination stems from a 1921 book by Dr. Zachary Cope, Early Diagnosis of the Acute Abdomen, which stated, “If morphine be given, it is possible for a patient to die happy in the belief that he is on the road to recovery, and in some cases the medical attendant may for a time be induced to share the elusive hope.”2,3

At that time, the belief was that administering analgesia, specifically morphine, to a patient with abdominal pain might cause an inaccurate examination and, thus, allow the patient to die from an undetected condition.

More recently, several researchers have examined this question.4 They randomly assigned patients with abdominal pain to receive either IV morphine or saline. The patients were assessed before and after receiving morphine or saline and then assessed later by a surgeon, if indicated.

The presence of peritoneal signs did not change in the group that had received morphine, and the accuracy of diagnosis did not differ between the two patient groups or between the emergency physicians and the surgeons. In fact, there was a trend toward improved diagnostic accuracy.5 These and other studies help support the contention that early analgesia is appropriate in patients with abdominal pain.

MYTH 3: If I give my patient narcotics, they’ll develop respiratory arrest.

It’s true that respiratory depression is one side effect of narcotics. We’ve all had narcotic overdose patients whose respiratory rate decreased to the point where we needed to intervene. However, treating pain is a different situation than an addict overdosing on an opioid. The respiratory depressant effects of properly used narcotic analgesia is offset by the respiratory rate increase that nociceptive stimuli produces.6 As long as the nociceptive stimulus is present and the narcotic analgesia is used properly, the patient’s respirations won’t be depressed.

This concern is also the focus of a study that examines the reduction of time to analgesia after a protocol change allowing standing order analgesia.7 The authors found no adverse effects in the almost 1,000 patients treated during the study. As with myth 2, the key to preventing respiratory depression was titrating the medication to make the patient more comfortable—not to eliminate pain.

MYTH 4: If I give my patient narcotics, they’ll abuse narcotics.

As medical providers, we occasionally hear this from patients, even those who deny ever abusing drugs, as a reason for not wanting narcotic analgesia. And although other patients may be malingering and/or drug-seeking, that doesn’t warrant withholding analgesia from all patients.

People who become addicted to opioids prescribed for legitimate medical purposes most likely have had addiction problems long before their treatment with narcotics.8 In the prehospital setting, as
EMS departments should work with their medical directors to ensure they carry appropriate analgesics to ensure patient comfort.

well as in the ED, it can be difficult to distinguish drug-seeking individuals from those requiring legitimate analgesia. Patients who have addictions can also become injured or ill and may require analgesia. In these cases, it’s important to remember that if they have previously abused narcotics, they may require larger doses because of the tolerance they develop to opioids. It’s unethical to withhold appropriate analgesia based solely upon addiction concerns.

In short, treat the patient in need of pain relief with an appropriate amount of analgesia. Later, relay any concerns about specific patients privately to the ED staff. Overall, the concern about addiction is largely unfounded. In a five-year review, the medical use of opiates increased, while the incidence of opiate abuse actually decreased.8

References