Chapter 1

Acute Pain Management in the Emergency Department

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Additional information is available at the end of the chapter

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Abstract

The most common presenting complaint to the emergency department (emergency room) is pain. Unfortunately, pain is still undertreated in this setting. Literature has shown that treatment of pain not only improves patient satisfaction but also improves mood, decreases length of hospital stay, and decreases mortality. Various pharmacological options are available for treating acute pain, ranging from oral, intravenous, and intramuscular medications; topical agents; and peripheral nerve blocks. Objectively assessing and documenting a patient's pain is the key to determining treatment. The approach to a patient with acute pain requires an experienced clinician who is aware of the pharmacology of analgesics and anesthetics, contraindications, precautions, side effects, administration methods, and monitoring requirements.

This chapter briefly covers the pathophysiology of acute pain and the different treatment modalities available to the emergency physician.

Keywords: pain, acute, emergency, treatment, management

1. Introduction

1.1. Epidemiology

The most common presenting complaint to the emergency department (ED) room is pain. From 1996 to 2015, ED visits have risen over 46%, from 90.3 million to [1] 136 million [1, 2]. As emergency room visits continue to grow every year, so does the need to treat patients in pain. Roughly, 45% of ED visits involves either moderate or severe pain [3]. The most common pain-related chief complaints in descending order are chest pain, back pain, and headache. Furthermore, the most frequently ordered analgesics, both in the ED and at discharge, are acetaminophen (alone or in combination with hydrocodone), ketorolac, and ibuprofen [3].

1.2. Oligoanalgesia

Despite the trend in increasing ED visits of which the majority involves pain, pain is still undertreated in the ED. Oligoanalgesia is a term used to describe the inadequate treatment of pain and was first studied in a retrospective chart analysis in 1989 by Wilson and Pendleton [4]. In this study, a total of 198 patients were evaluated, and of those who received analgesics, 32% received less than optimal doses [5]. An additional retrospective study, done a few years later, revealed that only 30% of 401 patients treated for bone fractures received analgesics [6]. In a separate study, patients were surveyed after treatment in a fast-track area of the emergency room, and 60% of patients went home with more pain than they were willing to accept [7]. More recently, a prospective, multicenter study enrolled 842 patients (aged 8 years and older) across 20 US and Canadian EDs with presenting pain intensity scores of 4 or greater on an 11-point numerical rating scale and found that only 60% of patients received analgesics, and 74% of patients were discharged in moderate to severe pain. Furthermore, reassessments were uncommon, and analgesics were administered after lengthy delays (median, 90 minutes; range, 0–962 minutes) [8].

1.3. Why is treating pain important?

The Joint Commission on Accreditation of Hospitals Organization (JCAHO) has recommended that assessment and treatment of pain be improved. Moreover, patients expect to have their pain treated fairly quickly and to have it significantly reduced. Studies have shown that patients want their pain treated in less than half an hour, yet the mean time of treatment is at least 78 minutes [9]. Acknowledging and treating pain in the ED improves the rapport between physician and patient. Patients are more likely to characterize physicians who treat their pain as warm and friendly and inspire more confidence to discuss their private health concerns [10]. Inadequately treating pain can contribute to the development of comorbidities such as depression, hypertension, and immune system dysfunction [10].

There are numerous potential reasons for which pain is inadequately treated in the ED, such as concern of masking symptoms, poor communication between clinician and patient (language, cultural), lack of documentation and reassessment of pain, and fear of contributing to and causing addiction. One of the most cited reasons is the concern that analgesics, particularly opioids, may mask symptoms of a surgical abdomen. This concern has been refuted by a number of studies. A randomized double-controlled trial conducted in 2002 evaluated pain control in the diagnosis of appendicitis. Pain was adequately treated, and patients continued to have pain upon palpation on physical examination [11]. An additional study studied the surgeon's confidence in physical signs after administration of morphine to patients with appendicitis. Despite the morphine, these surgeons continued to illicit examination signs such as the obturator sign, Rovsing, and pain upon jumping [12]. Other physicians may argue that opioids may mask the intensity of pain allowing for the progression of the illness to complications such as perforation and formation of an intra-abdominal abscess. A systematic review of six randomized controlled trials evaluating the safety of opioid administration to children with acute abdominal pain showed no significant difference in the rate of perforation or abscess formation [13]

The concern over causing addiction plays an important role in oligoanalgesia in the ED. In 2012, health care providers wrote 259 million prescriptions for painkillers. This is equivalent to every American adult having a bottle of pills. Furthermore, each day, 46 people die from an overdose of prescription painkillers in the United States [14].

In 2007 the cost of prescription opioid abuse was estimate to be $56 billion dollars [15]. Those with a prior history of depression, anxiety, and substance use were most likely to have a propensity for prescription opioid abuse [16]. An additional study confirmed mental disease as risk factor for opioid abuse, as well as males, younger adults, and individuals with greater days of supply of prescription opioid abuse. Thus, the emergency physician must do a full history, including a psychiatric history, prior to considering opioids.

Screening programs not only elucidate a patient's past-filled prescriptions but can also give the physician an idea of the different pharmacies and health clinics the patient has gone to. This information gives the prescriber insight into any drug-seeking behavior and may change their propensity into prescribing a certain analgesic [17].

1.4. Documentation of pain

The effective management of acute pain in the ED requires appropriate assessment of the pain based on the patient's perception of pain using a validated pain scale. Additionally, reassessment of pain is essential to determine the effect of treatment. Pain has been described as a vital sign, and as such it should be documented in the initial assessment of a patient. Verbal pain scores (VPSs) may reveal those who are truly in pain but who may not voice their discomfort, as well as influence the physician to inquire about the patient's pain. One study revealed, that in patients who did not receive analgesics, 42% desired them, but only 31% voiced their concern [8]. A prospective study introducing VPSs in an ED revealed that of those trauma patients who had VPS scores documented, 60% received analgesics versus 33% in those who did not have a VPS score documented. Furthermore, those with higher VPS scores were more likely to receive analgesics [18]. ED crowding has been shown to increase time to analgesic administration and mortality [19]. The use of VPS in this setting may identify those individuals in need of quicker treatment.

2. Pathophysiology

Pain can be divided into acute and chronic, with acute pain being incited by a traumatic injury or pathologic condition. As the causative issue is addressed, acute pain is usually resolved. Acute pain is mediated through nociceptors, of which there are various types ranging from mechanical to thermoreceptors. These receptors are stimulated by chemical, thermal, or mechanical stimuli [20]. As these receptors are stimulated, sensory neurons transmit the stimulus through neuronal pathways made up of various peripheral nerve fibers. "First Pain," which is well localized and sharp, is modulated by Aδ-fibers. The second component of pain,

or the slow phase, is conducted by C fibers and is characterized by dull and poorly localized pain [21].

All pain starts as acute pain; however, not all pain progresses to chronic pain. Acute pain becomes chronic when pain persists despite the resolution of the inciting event. There may be many causative factors that account for prolonged pain. Apart from the psychosocial influences on chronic pain development, physiologic factors that contribute to chronic pain include alterations in the spinal cord that occur when acute pain is inadequately treated. These changes lead to increased excitability, decreased inhibition, and reorganization of certain spinal tracts [22]. The time frame for defining chronic pain varies from 3 to 6 months of ongoing pain. However, some would argue that chronic pain is any pain that persists longer than the reasonably expected healing time for the involved tissues. It is also important to understand that an individual's perception of pain may be influenced by culture, previous painful experiences, beliefs, mood, and ability to cope.

2.1. Somatic and visceral pain

Somatic pain is made up of mostly A-fibers and is located in cutaneous tissues as well as deep tissues such as fascia, tendons, or bone. This pain is described as initially sharp and then as burning or throbbing. On the contrary, visceral pain is primarily composed of C f-fibers, and its primary afferent neuron endings are usually found in internal organs such as intestines, gonads, or heart [21]. For example, at presentation, pain from appendicitis may initially be poorly localized around the periumbilical site and characterized as dull, indicating primarily a visceral pain. However, as inflammation continues, the above fascia tissues become inflamed. At this point, pain is now located at the right lower quadrant and may be sharp. This later presentation of appendicitis is now primarily involving the somatic cutaneous nerves in the corresponding dermatome. Asking the patient to initially describe the pain may hint toward the initial causative pathologic condition. For instance, in a patient with sharp and clearly localized back pain, the causative agent may be musculoskeletal in nature. However, those with dull, achy, and poorly localized pain, back pain radiating to the groin may be due to an internal cause such as pyelonephritis or nephrolithiasis.

2.2. Neuronal pathway

The initiating stimulus of pain is conducted through these peripheral nerve fibers. There are a number of neuronal pathways through neuronal pathways, but the spinothalamic tract is the main pathway. These pathways converge into primary afferent neurons found in the dorsal root ganglion. Afferent neurons have two endings: one signaling the peripheral system and the second signaling second-order neurons in the dorsal horn. The second-order neurons' axons cross the midline of the spinal cord into the contralateral spinothalamic tract, where they ascend into the thalamus. Third-order neurons in the thalamus synapse with the second-order neurons and send signals to the post-central gyrus of the cerebral cortex [21]. As the nerve fibers ascend in the spinal cord, they organize into dorsolateral columns and anteromedial segments [20].

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The dorsal columns and anterior medial segment are divided into different segments called laminae. This is done to organize the type of sensory information sent into each section [23]. Laminae 1 through 6 are located in the dorsal horn, 7 through 10 in the intermediate zone, and 8 though 9 in the anterior/ventral horn. The gray matter surrounding the central cord composes lamina 10. All afferent nerve activity is received in the dorsal horn. Specifically, lamina 1 receives mostly noxious stimuli from cutaneous tissues and deep somatic tissues. Visceral afferent fibers are transmitted to laminas 5 and 1. However, lamina 5 also receives somatic afferent fibers, and it is this convergence that leads to referred pain [21]. Lamina 2, or the substantia gelatinosa of Rolando, mediates the activity of pain and temperature afferent fibers. Next, lamina 3 and 4, known as the nucleus proprius, receive input from lamina 2 and also help regulate pain, temperature, as well as crude touch. Lamina 7 receives afferent input from muscle fibers and joints [24].

Furthermore, the spinothalamic tract is subdivided into a lateral and a medial tract. The lateral tract projects to the ventral posterolateral nucleus of the thalamus and carries fibers sensory input that transmits location, intensity, and duration of pain. The medial tract projects to the medial nucleus of the thalamus and mediates the emotional and autonomic aspects of pain. Collateral fibers from the spinothalamic tract are also projected to the RAS, or reticular activating system, as well as the hypothalamus [21]. These collateral fibers may be responsible for the arousal aspect of pain.

2.3. Modulation of pain

Descending tracts originating from the midbrain and medulla feed into the spinal cord through the dorsolateral funiculus, modulating pain [20]. For example, stimulation of the periaqueductal gray, through projections from the spinothalamic tract, provides widespread analgesia in humans [25, 26]. One investigator noted that stimulation of the periaqueductal gray leads to analgesia with such significance that one could perform an exploratory laparotomy without any chemical anesthesia [26, 27]. Furthermore, these tracts involve transmitters such as norepinephrine, serotonin, and opiates [20]. TCAs and SSRIs through these neurotransmitters have been shown in various studies to significantly reduce chronic pain, regardless of the patient's psychosocial status. A meta-analysis found there is no difference in pain relief from the use of these medications in the absence or presence of depression, and the size of analgesia is not significantly different in the presence or absence of anti-depressant effect [28].

Additional modulation of pain can be seen through the endorphin system. This system consists of neurons that secrete three types of opioids beta-endorphin, net- and leu-enkaphalins, and dynorphins. These chemicals act on the mu, delta, and kappa receptors modulating pain relief [20].

3. Common analgesic agents used in the emergency department

3.1. Opioids

Opioid prescriptions for the management of non-cancer pain have increased over the last 10–20 years. Concerns of opioid dependence and toxicity, such as respiratory depression, have led to the under-dosing of these agents in the ED and the use of other less effective analgesic agents.

3.1.1. Mechanism

The term opioid refers to natural and synthetic substances that act at one of the three main opioid receptor systems (mu, kappa, delta). They can have analgesic and central nervous system (CNS) depressant effects as well as the potential to cause euphoria. The majority of opioids used clinically target μ-opioid (mu) receptors. These receptors mediate analgesia as well as common side effects such as euphoria, constipation, and respiratory depression [29]. One exception is the combination of agonist–antagonist agents such as buprenorphine. Another less commonly targeted receptor is the **κ**-opioid (kappa) receptor, which is important in regulating GI motility and dysphoria. The other endorphin receptors may regulate neuropathic pain, as we all as spinal anesthesia.

3.1.2. Morphine

One of the most commonly used opioids in the ED is morphine. It is considered safe and effective in the monitored setting in the ED [29].

Side effects can range from hypotension, pruritus, nausea, vomiting, and respiratory depression. It is believed that some of these side effects may be due to the destabilization of mast cells that lead to the release of histamine. Respiratory depression is caused by desensitization of the medulla to carbon dioxide, through opioids binding to the mu receptor. The cardiovascular effects of opioids are mediated centrally at the central vagal nucleus and, in the case of morphine, directly into the sinoatrial node. Within the gastrointestinal system, opioids delay gastric emptying and cause constipation [29]. There appears to be no significant differences in side effects between dosages of 0.1 mg/kg and 0.15 mg/kg.

Weight-based dosing for morphine is not necessary in obese patients. A prospective observational study in the ED revealed that patient's weight was not predictive of pain reduction [30]. Thus one should start with the recommended dose of 0.1 mg/kg if side effects are of concern; however, one should be ready to rebolus in 5–15 minutes as studies have revealed this initial dosing is inadequate. A prospective cohort study of 119 patients revealed that 67% of patients who received 0.1 mg/kg of morphine stated less than 50% reduction of pain 30 minutes later [31]. A later study evaluating trauma patients revealed that a dose of 0.15 mg/kg when compared to 0.1 mg/kg significantly reduces pain without any significant difference in adverse events [32].

In the setting of trauma, hypotension may reduce tissue perfusion in patients with significant blood loss. However, in a randomized controlled study in acute trauma patients, hypotension only occurred in 10% of patients who received morphine [32]. A study investigating the use of morphine in the pre-hospital setting in ST segment elevation myocardial infarction patients found no worsening of in-hospital complications or 1-year mortality [33]. The cancer literature has also shown the value and safety of morphine infusions for pain control [34].

3.1.3. Hydromorphone

Hydromorphone is a semisynthetic derivative of morphine that is seven times more potent than morphine. Despite the increased potency, studies have shown that nurses who are concerned about side effects may give a lower dose of morphine versus hydromorphone since the "total milligrams" given in hydromorphone is less when compared to an equal analgesic dose of morphine [35]. Despite the dosing difference, hydromorphone appears to offer better pain control. In a retrospective study involving the use of patient-controlled analgesia (PCA) with either morphine or hydromorphone, more patients receiving morphine required rescue analgesia due to initial inadequate pain control [36].

Pruritus occurs less frequently with hydromorphone. Hydromorphone is conjugated by the liver to hydromorphone-3-glucoronide, an inactive metabolite. However, morphine's metabolite is active, and as a result, hydromorphone is better tolerated [20]. With regard to adverse effects, hydromorphone has not been shown to have an increased risk, and its use does not necessitate increased naloxone administration [37].

3.1.4. Fentanyl

When pain relief is needed quickly for acute severe pain, such as in trauma, fentanyl may be of use. Its time of onset is 1–2 minutes and lasts typically about 30 minutes [20]. The initial IV dose is 1.5 μg/kg, and it has the advantage of a short half-life. This is particularly useful if serial examinations are needed. Fentanyl causes minimal histamine release, making it ideal in patients in whom blood pressure must be maintained. For example, in severe traumatic brain injury patients, in whom MAP must be kept above 80 to maintain cerebral perfusion pressure, and must be examined serially, fentanyl may be a useful analgesic. The safety profile is favorable, particularly in the pre-hospital setting. A retrospective chart review of 2,129 patients transported by Emergency Medical Services revealed that fentanyl affected vital signs in less than 1% of patients [38]. Despite its favorable hemodynamic profile, fentanyl may cause chest wall rigidity when given in doses above 15 μg/ kg leading to inadequate ventilation. This is a rare complication and can be remedied through neuromuscular blockade or naloxone [39, 40].

3.2. Non-opioid medications

Non-opioid analgesics include acetaminophen, non-selective nonsteroidal anti-inflammatory drugs (NSAIDs), and cyclooxygenase 2 (COX-2) inhibitors. NSAIDs and COX 2 inhibitors have anti-inflammatory properties.

3.2.1. Acetaminophen

A common over-the-counter analgesic for mild to moderate pain is acetaminophen (para-acetylaminophenol) or paracetamol (in Europe). Its mechanism of action is through the inhibition of prostaglandin endoperoxide H_2 synthase and cyclooxygenase activity [20, 41]. Its central anti-pyretic effect is of great use when fever needs to be reduced. It has been shown to have good analgesic effects; however, acetaminophen is not anti-inflammatory. Systematic reviews have shown that acetaminophen can significantly reduce pain; however, it may be less effective than NSAIDs in conditions such as back pain and osteoarthritis [42–44]. Acetaminophen can also be combined with opioid medications to reduce the amount of opioid needed. However, concerns about unintentional acetaminophen overdose have led to combination drug products with more than 325 mg acetaminophen per tablet to be withdrawn from the market. Acetaminophen overdose can lead to severe hepatotoxicity and should be used cautiously in patients with chronic alcohol use or liver disease.

Intravenous acetaminophen is being studied for acute pain such as in acute traumatic limb injuries [45] or rib fractures [46] or in postoperative patients [47, 48]. It has also been shown to reduce the need for rescue pain medications such as opioids [49, 50].

3.2.2. Nonsteroidal anti-inflammatory drugs

NSAIDs provide analgesia for mild to moderate pain and also work synergistically when paired with opioids. They work through the inhibition of cyclooxygenase by decreasing the production of prostaglandins and prostacyclins, primarily cyclooxygenase 1 (COX-1) and COX-2. COX-1 mediates platelet aggregation and maintenance of gastrointestinal mucosal integrity. By contrast, COX-2 generates prostaglandins that mediate pain and inflammation [29]. The different NSAIDs can be either selective COX-2 inhibitors or non-selective, thus differing in their side-effect profile. There are many NSAIDs to choose from, but there is little literature showing improved efficacy of one NSAID over another.

Main adverse side effects of NSAIDs include gastrointestinal insult, renal insult, inhibition of platelets, cardiovascular effects, and anaphylaxis. Renal failure is caused by the decreased production of prostaglandins, which aid in afferent glomerular arteriole vasodilation. NSAIDs contribute to arteriolar vasoconstriction, leading to decreased renal perfusion pressure and decreased glomerular filtration rates [51]. This is worsened by dehydration. As selectivity of COX inhibition increases, the renal effects decrease. NSAIDs such as ketorolac and diclofenac have fewer effects on the kidney than naproxen or ibuprofen [51, 52].

The most common side effect of NSAIDs is gastrointestinal injury, such as bleeding or dyspepsia and gastric ulceration. Patients who are at high risk for peptic ulcer disease or its complications, such as the elderly, those with bleeding diathesis, or patients on glucocorticoids, have a relative contraindication to the use of an NSAID. Each NSAID has variability in the risk of gastrointestinal injury it poses. This is due to the selectivity of COX-1 inhibition, so that the relative risk of for ibuprofen is 2.6, while the relative risk for ketorolac is 14.5 [51, 53].

Various studies have shown that COX-2 inhibition is related to increased cardiovascular risk. This is believed to be the result of decreased prostacyclin (prostaglandin I_2) and increased

thromboxane A_2. The effects lead to hypertension, accelerated atherogenesis, and increased thrombotic response to plaque rupture [54]. Myocardial infarction was found to be increased in this class of NSAIDs, resulting in the discontinuation of rofecoxib [55, 56]. Furthermore, it has been shown that specific COX-2 inhibitors may also further inhibit renal perfusion and lead to decreased sodium excretion, which may further worsen congestive heart failure and renal function [57, 58]. Due to COX-2 specific inhibitor side-effect profile and no proven increased efficacy over non-selective NSAIDs, there is minimal to no advantage in using this class in the ED.

There has been no proven efficacy over one type of NSAID, including the route of administration such as intra-muscular versus oral [29, 59, 60]. One should select a particular NSAID based on its side-effect profile and the route of administration that is the most feasible for the patient. Furthermore, prior to using NSAIDs, one must also take into consideration that this class of pharmaceuticals is most useful when used in pain mediated by prostaglandins or inflammation, not in other situations such as neuropathic pain. For instance, NSAID, particularly ketorolac, has been shown to significantly reduce pain in renal colic, and has similar efficacy in pain reduction as morphine [61]. When used in combination, opiates and NSAIDs may reduce the need of additional doses of analgesic rescue therapy in renal colic and have greater pain efficacy than either drug used alone [62]. In acute lower back pain, NSAIDs have been shown to significantly reduce pain and improve daily function [63]. The addition of opioids in this setting of pain was not proven to be more effective than NSAIDs alone [64].

3.2.3. Antispasmotics (muscle relaxants)

Muscle relaxants have been used by the physicians with the intention of alleviating musculoskeletal pain. However, data on this class of medications have produced mixed results since their action may be more the result of sedation rather than muscle relaxation. A systematic review, evaluating the effectiveness of cyclobenzaprine in lower back pain, revealed short-term improvement of pain at 7 days. However, there was no improvement of pain at 14 days, and there was no statistical difference when compared to diazepam [65]. Furthermore, in a second review evaluating the effectiveness of muscle relaxants in neck pain, there was no difference when compared to placebo at 2 weeks [66].

When compared to NSAIDs, muscle relaxants have been shown to have no significant difference in pain relief or improvement in daily function. Moreover, there is little to no added benefit when using muscle relaxants together with NSAIDs [64, 67]. Given the limited data on muscle relaxants, one should consider the side effects. Major side effects range from drowsiness, dizziness, dry mouth as well as other anticholinergic effects. These medications should only be prescribed for short-term use, given the limited data regarding efficacy past 1 week. In addition, one should refrain from prescribing these medications in the elderly, as they are at higher risk of falls and delirium.

3.2.4. Topical analgesics

There are various topical agents ranging from patches, gel, sprays to creams, which may aid in relieving pain. They appear to have several potential advantages over systemic drugs such as delivery at the site of injury, lower levels of systemic absorption, and fewer systemic effects. Although systemic side effects are not as frequent as oral formulations, significant systemic concentration can be achieved by topical application.

Often, these agents have similar efficacy to their oral preparations. For example, a randomized controlled trial comparing a gel preparation of ibuprofen versus oral tablets revealed comparable improvement in patient's pain and functional status at 2 weeks [68]. Topical agents often have fewer side effects than their oral counterparts, and most adverse events are primarily cutaneous in nature such as rashes or pruritus. Decreased adverse events may be due to lower bioavailability in the plasma [69, 70]. Many factors may influence the penetration of the topical agent into the local site. Variability in an individual's skin properties such as the thickness of stratum corneum may be a limiting factor. Furthermore, the agent must be lipophilic and water-soluble [71]. Local site pH, such as acidity in a local cellulitis, may also limit penetration of the agent.

Topical NSAIDs have been shown to have rare incidences of gastrointestinal adverse events such as ulcer formation, as opposed to oral formulations [69]. Topical diclofenac and ibuprofen have been shown to be effective in acute soft tissue injuries, such as ankle sprains as well as arthritic knee pain [68, 72, 73]. Furthermore, topical diclofenac has been shown to be effective in reducing myofascial pain, however, with no effect on the myofascial trigger point pain threshold [74]. Topical NSAIDs have been shown to have equal efficacy as oral NSAIDs, yet various studies have shown topical diclofenac to reduce pain within 2–3 days of treatment [72, 75–77].

Neuropathic pain has been shown to respond to topical agents. For example, topical lidocaine, when used in post-herpetic neuralgia (PHN), has been associated with improved quality of life, improvement of pain and allodynia [78]. Moreover, when compared with oral pregabalin, response rates were higher in patients with either PHN or diabetic neuropathy. The same studies also showed a lower rate of adverse events and improved quality of life [77, 79, 80]. Topical capsaicin cream has also been studied to reduce neuropathic pain; nonetheless, application of this cream has been associated with a burning sensation in up to 81% of patients [81]. Randomized controlled studies of high concentration topical capsaicin revealed significant pain relief in patients with PHN with relief lasting up to 12 weeks [77, 82, 83].

Topical opioids have not been shown to significantly reduce pain. For example, a randomized controlled trial comparing the use of topical morphine sulfate versus traditional Jelonet dressings in burn patients revealed increased need of rescue analgesia and higher pain scores in the topical morphine group [84]. Another study revealed no significant reduction in pain with patients with skin ulcers when topical morphine was compared to placebo [85]. However, in patients with mucositis undergoing chemoradiotherapy for head and neck cancer, oral morphine mouthwash has been shown to significantly reduce pain and reduce length of functional impairment [86]

In summary, topical analgesics may provide additional analgesia in patients who may not be able to tolerate the adverse effects of systemic analgesics. Those with PHN may benefit from topical lidocaine when amitriptyline fails to provide relief. Those with peptic ulcer disease may benefit from topical NSAIDs to treat arthritis, as oral NSAIDs may worsen their condition. By contrast, topical opioids have not been shown to provide significant relief in burns or skin conditions, limiting their role in the ED.

3.3. Peripheral nerve blocks

There are two types of nerve blocks: single injection and continuous nerve blocks.

Single-injection nerve blocks are one-time injections of local anesthetic adjacent to the nerve or plexus for anesthesia and/or analgesia and are most commonly used in the ED. Continuous infusion nerve blocks involve the placement of catheter adjacent to the peripheral nerve or plexus. These are useful in patients who are expected to have prolonged need for analgesia. The effectiveness and duration of the block depend upon the pharmacology of the analgesic/anesthetic agent used, the dose, and the concentration.

Peripheral nerve blocks are important tools for pain management in the ED and have been shown to significantly reduce pain. Analgesia from peripheral nerve blocks can be reached more quickly than intravenous narcotics and often with more efficacy and less rescue analgesics. A randomized controlled trial compared the use of femoral nerve blocks versus intravenous narcotics in femoral fractures and found lower pain scores within 90 minutes in the femoral nerve block group. The incidence of infections was the same in both groups, and there were no reports of paresthesias [87]. In fact, the total amount of morphine required to produce adequate analgesia was up to three times higher in intravenous narcotics group than in patients with a peripheral nerve block [88].

The benefits of peripheral nerve blocks have not only been seen in femoral fractures but also in other traumatic injuries such as hand lacerations, upper extremity fractures, and dislocations. For reduction of forearm fractures, studies have shown that children have less distress and pain when a brachial plexus block was performed versus procedural sedation [89]. Furthermore, length of stay in the ED was also significantly reduced when brachial plexus block was performed with length of stay being reduced almost 3 hours [90]. Similarly, patients with shoulder dislocation that underwent a brachial plexus block also showed reduction in ED length of stay, without any increased adverse events or reduction in patient satisfaction [91].

One of the most concerning complications of peripheral nerve blocks is nerve damage. In a peripheral nerve block, the goal is to position the local anesthetic around the nerve and not "into" the nerve. One should avoid intra-neural injection that may cause direct trauma or toxicity to the nerve. The incidence of nerve damage in the days following the block (including temporary paresthesias) ranges from 0.5 to 15% [92, 93]. However, in significant nerve damage resulting in peripheral neuropathy or symptoms lasting longer than 6 months, incidence was reported to be less than 0.1% in a prospective study [92, 94]. Most complications of nerve damage are transient, with most patients recovering by 3 weeks. Localized infection has been

noted to be rare, with 3% of peripheral nerve catheters in anesthesia studies showing signs of infection or abscess formation [95]. On the contrary, vascular puncture is not uncommon, and incidences of up to 5.7% and 6.6% have been noted when investigators placed femoral or sciatic nerve peripheral catheters [92, 96]. Systemic toxicity, such as cardiac arrest, was found to be rare, with all cases of cardiac arrest noted to be in central spinal anesthesia. Additionally, seizures were noted in 6 out of 50,223 cases [94].

Ultrasound and nerve stimulator techniques have been shown to reduce the complications from peripheral nerve blocks. One study investigating the use of ultrasound or electrical stimulation in the placement of a brachial plexus peripheral nerve catheter resulted not only in decreased time performing the procedure but also no vascular punctures in the ultrasound-guided group [97]. Furthermore, a Cochrane systematic review also confirmed faster procedure times and reduced local anesthetic volume and improved quality of nerve block [98].

Emergency physicians are adept at using ultrasound in central line placement, as well as in other diagnostic procedures, such as in FAST abdominal examinations, in trauma patients. Emergency physicians can be trained in ultrasound-guided peripheral nerve blocks as well. Ultrasound imaging permits direct visualization of needle location relative to target nerves, blood vessels, and related structures, as well as observation of the local anesthetic during and after the injection. A prospective observational study trained emergency physicians in the use of ultrasound guided peripheral nerve blocks in patients with traumatic limb emergencies and found that trained physicians were able to perform the ultrasound-guided nerve blocks in about 9 minutes with no complications and no need of rescue procedural sedation [99].

Prior to the decision to perform a peripheral nerve block, a careful medical history should be obtained including allergies, use of anticoagulants, preexisting nerve damage, active infections at the site, and ability to cooperate with the procedures. During the placement of peripheral nerve bocks, patients should be carefully monitored. It is important to assess for preexisting sensory or motor deficits in the distribution of the block. A patient with neurologic deficits prior to the nerve block may be at higher risk for developing new neurologic deficits following a nerve block than a patient without preexisting deficits. A brief overview of the femoral and brachial plexus peripheral nerve will be explained in the section below.

3.4. Femoral nerve block

The femoral nerve block is used to anesthetize the hip, anterior thigh, and knee. This nerve passes beneath the inguinal ligament and travels lateral to the femoral artery within the femoral triangle (**Figure 1**) [100]. The fascia iliaca separates the femoral nerve from the femoral vascular bundle [101]. The patient is initially positioned in a supine position. The affected extremity is then externally rotated and abducted. With the probe marker to the patient's right, a linear probe is then placed at the inguinal crease parallel to the inguinal ligament, the femoral nerve will then be visualized (it may appear as a hyper echoic, honeycombed structure). Medially, the femoral artery and then the femoral vein will be present. The iliopsoas muscle will be present posteriorly and the fascia lata superiorly (**Figure 2**) [101, 102].

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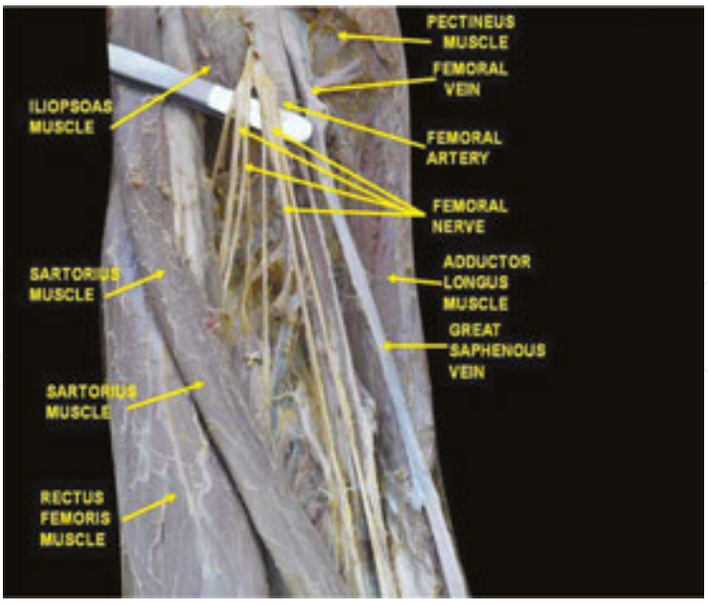

Figure 1. Femoral triangle.

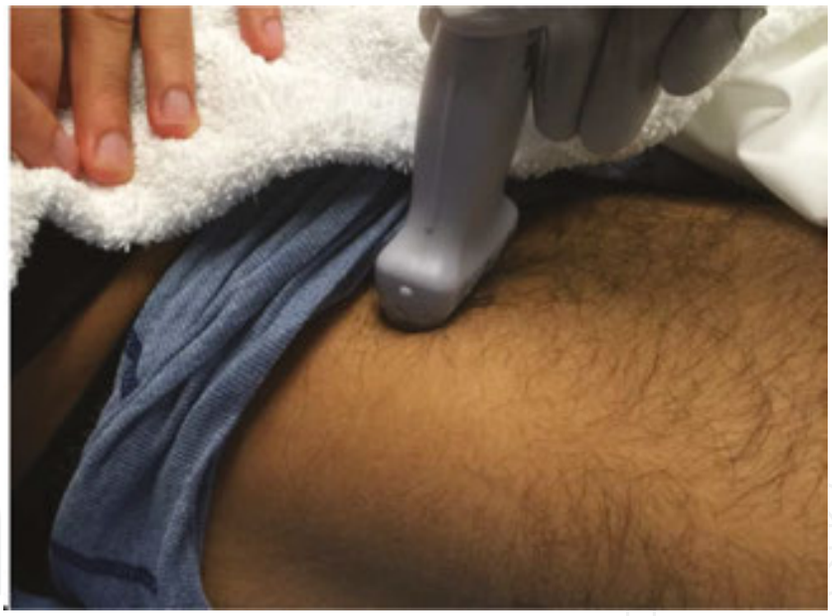

Figure 2. Placement of the ultrasound linear probe for the femoral nerve block. The patient is laid in a supine position with the affected extremity externally rotated and abducted. The linear probe is placed in a transverse fashion inferiorly to the inguinal crease.

Once the structures and anatomical landmarks mentioned above have been identified, aseptic skin preparation is performed and anatomic structures at the block site are again identified using an ultrasound probe in a sterile plastic sheath with sterile conductive gel (**Figure 3**). The structures are once again confirmed on ultrasound and then a skin wheal is made with local anesthetic. When the optimal ultrasound view is achieved, the probe is held immobile; the

block needle is then inserted at the skin on the lateral edge of the probe in-plane, aiming for the space behind the nerve. It is then advanced, with movement only when the needle tip is seen. Often a "pop" will be felt as the fascia iliaca is penetrated with the needle. Next, aspiration of the needle is done to confirm no vascular penetration. About 1–2 ml of local anesthetic is injected to visualize the placement of the needle on the ultrasound screen. The anesthetic should be seen surrounding the nerve. Once correct placement is confirmed, 10–20 ml of the selected anesthetic is injected. It may take up to 10–20 minutes to take effect [101–103].

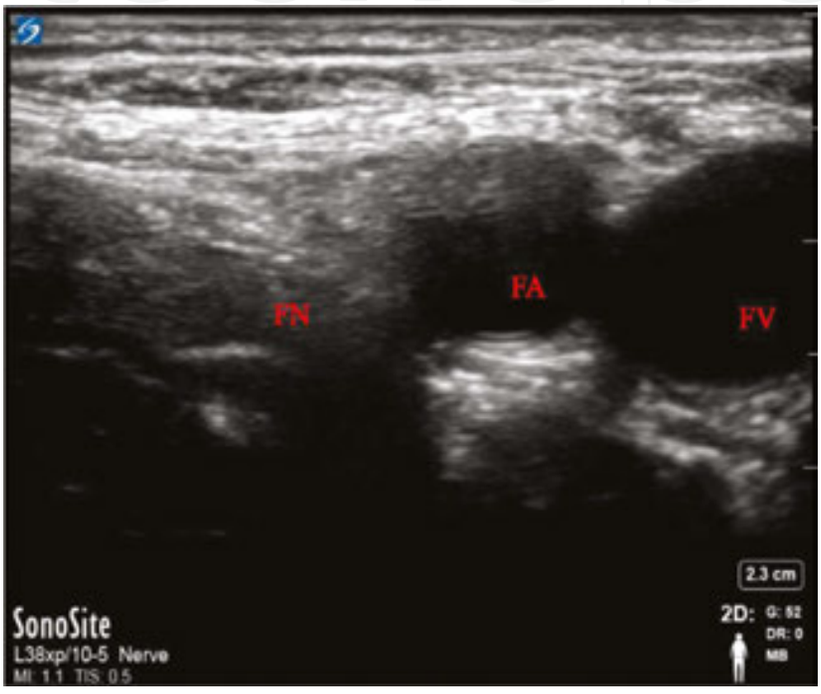

Figure 3. Ultrasound view of the femoral nerve. FA, femoral artery; FV, femoral vein; FN, femoral nerve.

3.5. Brachial plexus block

The brachial plexus block, or interscalene block, can be used to facilitate reduction of upper extremity fractures, lacerations, and even reduce shoulder dislocations. Nerve roots of C5-T1 are the initial part of the brachial plexus, forming a complex configuration before they enter the terminal nerves of the arm (**Figure 4**) [104]. The more proximal one blocks to the plexus, the more proximal the anesthesia is on the arm. Nerve roots of C5-T1 form the superior, middle, and inferior trunks of the plexus at the level of the cricoid cartilage. At this location, the plexus is found superior and posterior to the subclavian artery, with the dome of the lung located anteromedial to the inferior trunk. The interscalene space is the grove between the anterior and middle scalene muscles. This is where one will find the structures mentioned above. However, since the inferior trunk is often not included in this block, one cannot use this procedure for injuries below the elbow [105].

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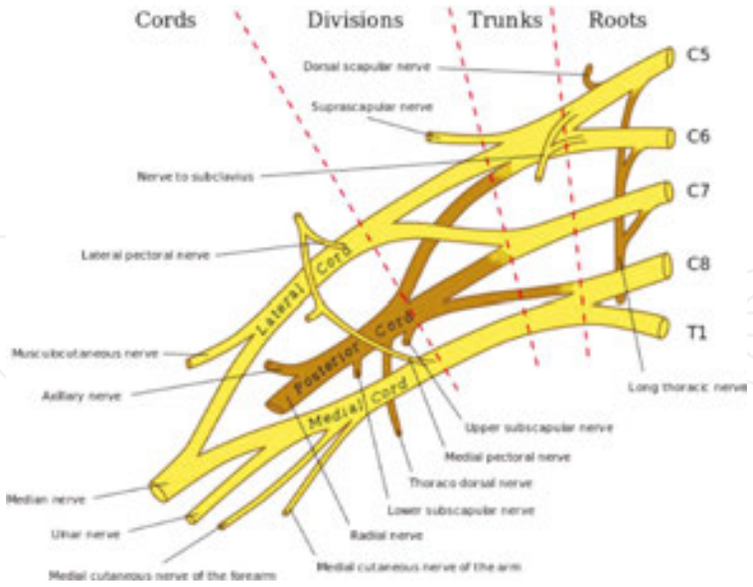

Figure 4. Brachial plexus.

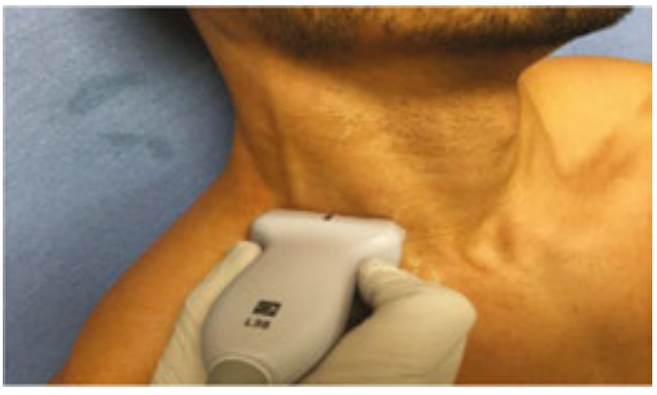

Figure 5. Placement of the ultrasound linear probe for the interscalene brachial plexus block. The patient is laid in a supine position with head turned away. The probe is then placed in a transverse fashion and used to identify the sternocleidomastoid muscle first. Next, one then sweeps posterior laterally to bring into view the interscalene groove.

The patient is initially positioned supine with the head turned 45 degrees to the contralateral side. With a linear probe, one first identifies the sternocleidomastoid muscle (SCM), which is located anteriorly to the carotid artery and internal jugular vein. One then sweeps posterior laterally bringing into view the middle scalene muscle and anterior scalene muscles. This is where the trunks of the brachial plexus may be visualized between the anterior and middle interscalene muscles. As with peripheral nerves, these trunks may appear as hyper echoic honeycombed structures (**Figure 5**) [105, 106]. Once the structures are identified, aseptic skin preparation is performed and anatomic structures at the block site are again identified using an ultrasound probe in a sterile plastic sheath with sterile conductive gel. A skin wheal is made using local anesthetic. Then, in an in-plane approach, the block needle is inserted posterior-

laterally to the probe, at an angle of 45 degrees to the skin. The needle is advanced toward the plexus, aiming toward the space between the top and middle trunks. Next, aspiration is done to check for any vascular puncture, and then placement is confirmed with movement of the trunks on injection of anesthetic. Depending on the agent used, the volume of local anesthetic is about 15–45 ml (**Figure 6**) [105, 106].

A second approach to the brachial plexus block is a supraclavicular block. To perform this block, the patient is once again laid in a supine position with the head turned away from the side being blocked. A linear probe transducer is then placed immediately superior to the clavicle at its midpoint (**Figure 7**). Tilting the probe caudally will bring into view a transverse view of the subclavian artery. Laterally to the artery, one will be able to see a collection of hypo echoic, honeycombed structures, which is the brachial plexus. Underneath these structures, the first rib is visible as a linear hyper echoic structure with lung underneath (**Figure 8**) [107, 108]. After the correct anatomy is identified, the skin is prepped in a sterile manner, and using a sterile probe cover, this area is once again identified. A 27-gauge needle is then used to inject the skin with 1–2 ml of local anesthetic just lateral to the probe. The block needle, 22-gauge, is then advanced in an in-plane approach toward the brachial plexus from a lateral to medial direction. At times one may feel a "pop" once the brachial sheath has been penetrated. One then aspirates to confirm non-vascular penetration and injects 1–2 ml of anesthetic to view the brachial plexus. Next, one then injects about 20–25 ml of anesthetic, until adequate spread is seen surrounding the brachial plexus [107].

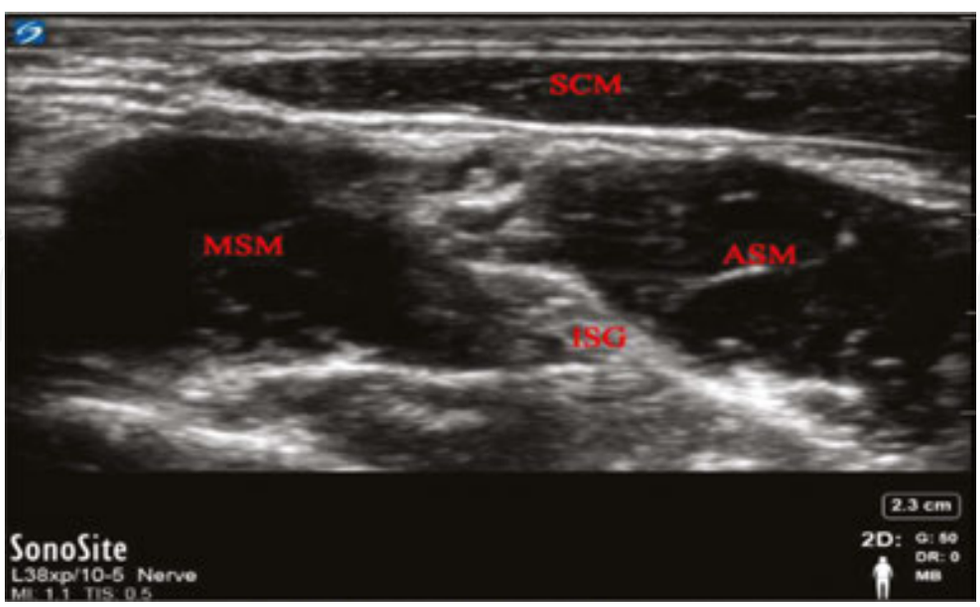

Figure 6. Interscalene view of the brachial plexus. ISG, interscalene groove with the brachial plexus present; SCM, sternocleidomastoid muscle; ASM, anterior scalene muscle; MSM, middle scalene muscle.

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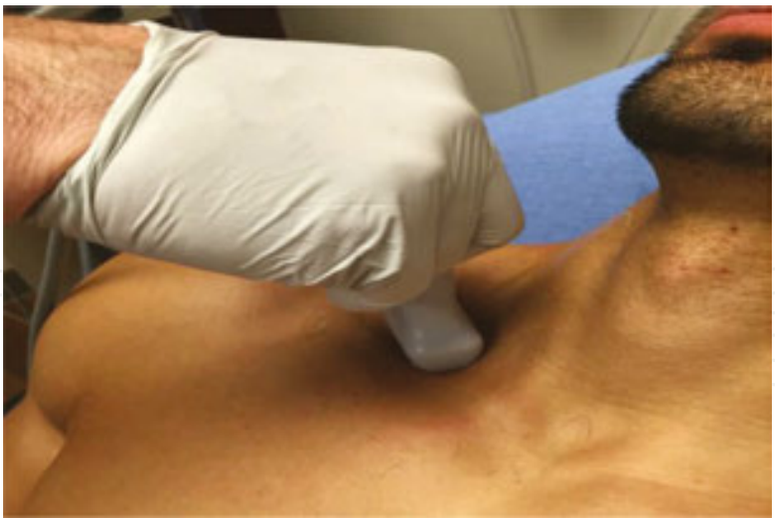

Figure 7. Placement of the ultrasound linear probe for the supraclavicular brachial plexus block. The patient is placed in a supine position or slightly seated position. The linear probe is then placed immediately superior to the clavicle at its midpoint. Tilting the probe caudally will bring into view the subclavian artery with the brachial plexus seen lateral to it, and the first rib and lung underneath.

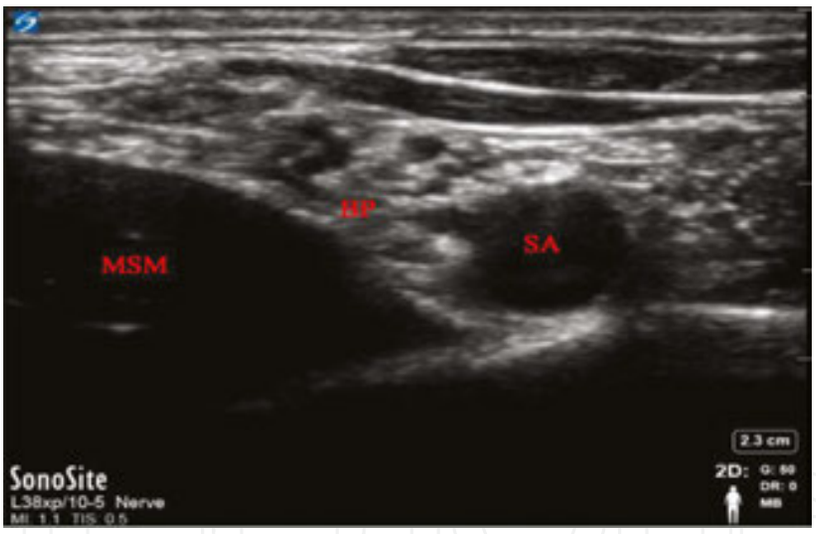

Figure 8. Supraclavicular view of the brachial plexus. SA, subclavian artery; BP, brachial plexus; MSM, middle scalene muscle.

Peripheral nerve blocks are a valuable asset to the emergency physician trained in these procedures. These procedures reduce pain quicker than intravenous narcotics, decrease the amount of sedation needed, and decrease ED length of stays. Peripheral nerve blocks may offer an alternative to avoid respiratory and cardiovascular depression encountered with procedural sedation or intravenous narcotics.

4. Conclusion

Pain is the most common presenting complaint to the emergency room. Appropriate treatment affects not only patient satisfaction and well-being but also patient outcomes. The choice of an appropriate initial therapeutic strategy is dependent upon an accurate evaluation of the cause of the pain and the type of pain syndrome. Effective management of acute pain in the ED requires a systematic approach. First, an accurate assessment of the patient's pain should involve the use of validated pain scales. Second, suitable analgesics given in an acceptable time frame are essential in the diagnosis and treatment. This should include proper monitoring for adverse side effects. Third, pain should be reassessed and documented regularly to determine the effect of treatment.

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