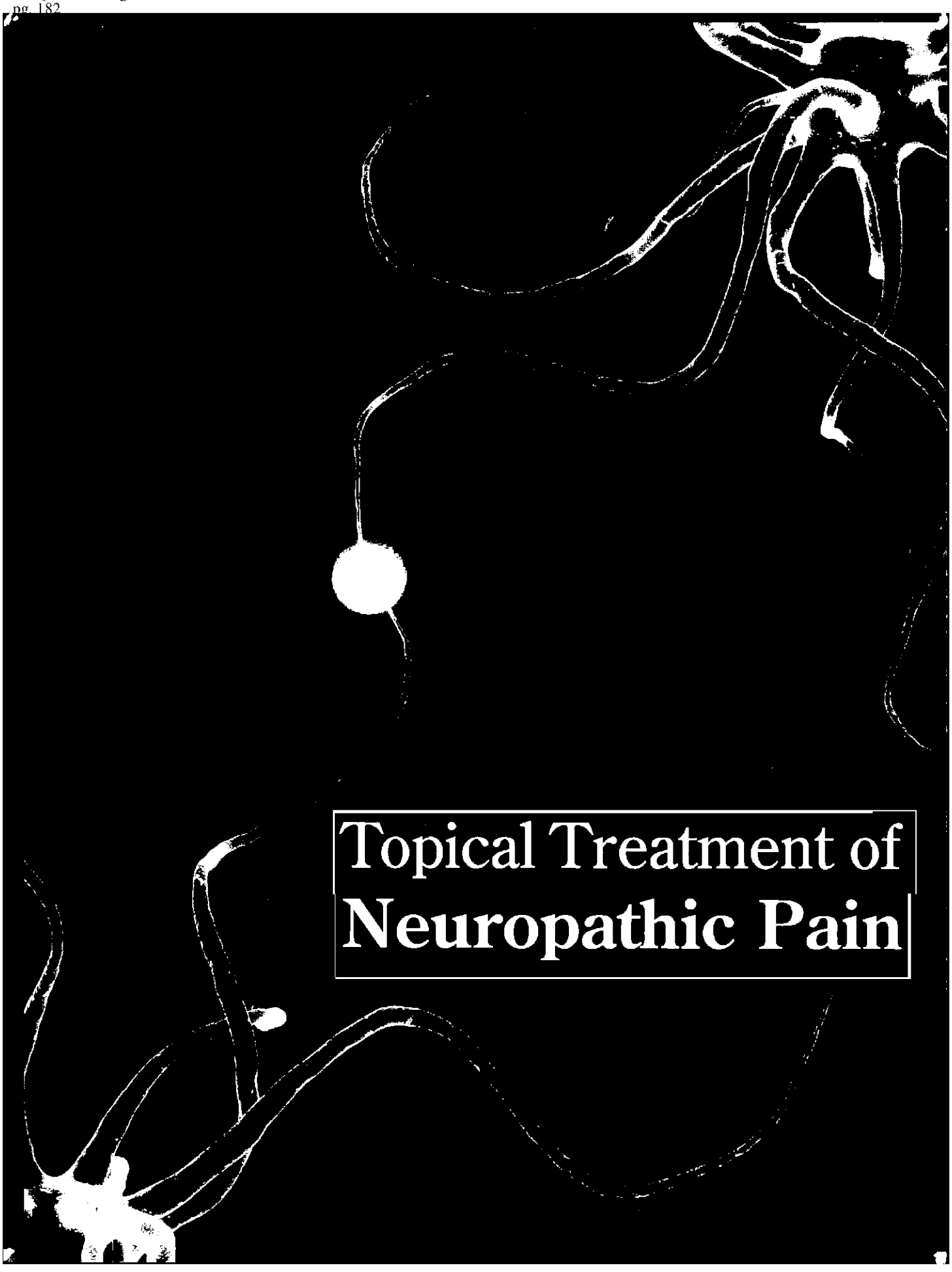

ABSTRACT

Pikes Peak Hospice & Palliative Care, Inc., provides care for more than 200 patients with terminal disease who reside at home, in long-term care facilities, in assisted-living facilities, or in its acute-care Inpatient Unit. Over the past 4 years, its pharmacy has evolved to provide individualized compounded preparations to meet the unique and complex needs of these patients. An integral part of this evolution was development and implementation of topical treatments for pain. These patients often have multiple medical conditions and complicated health issues. Nearly 50% of patients in the care of Pikes Peak Hospice receive a topical compound to treat any of a variety of conditions, including neuropathies, muscle skeletal pain, wound pain, agitation, and anxiety. Topical compounds give practitioners another option in managing patient maladies and, in many instances, compounded topicals have proven as effective, if not more effective, than commercially available products. In some cases, patients have been able to stop oral pain medications or reduce their dose because the topical preparation is so effective. All in all, the topical preparations described in this article have been invaluable to our patients' comfort and an indispensable option for practitioners.

Vince Vadaurri, RPh, BCPS
Palliative Pharmacy of Pikes Peak
Colorado Springs, Colorado

Pain warns of a potential or actual noxious stimulus and prompts an appropriate and coordinate physical and behavioral response. Pain perception and response occur on multiple levels of the nervous system: peripheral nerves, spinal cord, and brain (Figure 1), and neuropathic pain develops at any of these levels from chronic abnormal neuronal processing. Nearly a decade ago, an estimated 4 million Americans had chronic neuropathic pain; with the rising prevalence of diabetes, this number has probably increased.[1] The primary neurological lesion of neuropathic pain can arise from diabetes, trauma, malignancy, chronic inflammation, toxic drugs, impaired immune system, and/or infections; its course is often insidious, the cardinal symptom being pain with no apparent biological purpose.[2]

Given the heterogenous etiology of neuropathic pain, it is associated with an equally diverse and complicated array of molecular changes; predictably, the resulting complex, aberrant sensory process can make the management of neuropathic pain difficult. In general, alterations in nociceptor sensitivity and excitability form the basis for neuropathic pain and for the common symptoms hyperalgesia, allodynia, and hyperpathia. The objective of therapy is to halt, attenuate, or reverse the altered state of nociceptors. To this end, drug therapy targets the specific molecular alterations with the intention of alleviating the neuropathic pain; yet existing treatments prove woefully inadequate for many.[3] Although amitriptyline and pregabalin have been used successfully to treat neuropathic pain, this paper specifically addresses the rationale for the topical use of four agents—tetracaine, methadone, ketoprofen, and gabapentin—for the management of neuropathic pain.

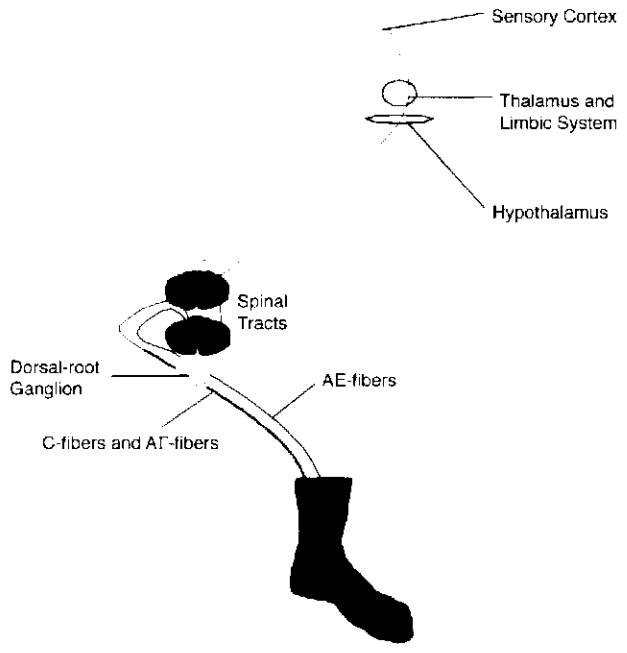

FIGURE 1. Pathways for pain perception and response.

Rx GABAPENTIN 5% IN PLURONIC LECITHIN ORGANOGEL

		For 60 mL
Gabapentin		3 g
Purified water		5 mL
Lecithin and isopropyl palmitate solution		13.2 mL
Pluronic F-127 20% solution	qs	60 mL

Note: For the following formulations, the lecithin and isopropyl palmitate solution can be prepared by mixing 0.2 g sorbic acid, 50 g soy lecithin, and 50 g isopropyl palmitate. The Pluronic F-127 20% gel can be prepared by mixing 0.2 g sorbic acid, 20 g Pluronic F-127, and purified water to make 100 mL.

METHOD OF PREPARATION

1. Weigh the gabapentin.
2. Place the lecithin and isopropyl palmitate solution in a 60-mL luer-to-luer locked syringe.
3. Add the gabapentin and purified water to the syringe.
4. Bring final volume to 60 mL with Pluronic F-127 20% solution.
5. Exchange contents between syringes 15 times.
6. Package and label.

STABILITY

This formula has a beyond-use date of 30 days. Store at room temperature.

Rx KETOPROFEN 10% PLURONIC LECITHIN ORGANOGEL

		For 60 mL
Ketoprofen		6 g
Ethanol 95%		5.5 mL
Lecithin and isopropyl palmitate solution		13.2 mL
Pluronic F-127 20% solution	qs	60 mL

METHOD OF PREPARATION

1. Weigh the gabapentin.
2. Place the lecithin and isopropyl palmitate solution in a 60-mL luer-to-luer locked syringe.
3. Add the ketoprofen and ethanol to the syringe.
4. Bring final volume to 60 mL with Pluronic F-127 20% solution.
5. Exchange contents between syringes 15 times.
6. Package and label.

STABILITY

This cream-colored formula has a beyond-use date of 6 months. Store at room temperature.

TOPICAL ROUTE OF ADMINISTRATION

This section defines the difference between topical and transdermal delivery systems, discusses the characteristics of topical delivery systems and their advantages, and enumerates the reasons for using drug combinations and drugs with multiple mechanisms of action in the treatment of neuropathic pain.

By definition, topical delivery systems act locally, while transdermal delivery systems (e.g., fentanyl patches, clonidine patches, nicotine patches), though applied peripherally, act systemically. A topical dosage form offers the following advantages over transdermal and other systemic dosage forms in treating neuropathic pain:[4]

- Decreased potential for drug interactions
- High concentrations of the drug at the site of injury
- Low systemic drug levels
- Minimal adverse effects
- No need to titrate dose to tolerability

The bioavailability of topical nonsteroidal anti-inflammatory drugs (NSAIDs) has been reported to be generally less than 5% to 15%, while drug concentration at the site of administration can be 30-fold higher than with an oral dose.[4,5] As the molecular processes of neuropathic pain are unraveled, targeting specific pain mechanisms becomes more feasible. Because different sensitizing signal molecules acting on different receptors all contribute to the pain, neither inhibiting a single sensitizing agent nor using one drug is likely to completely ameliorate sensitized neurons. A multi-drug approach or a drug with multiple mechanisms of activity is the most sensible treatment. With these principles establishing the framework for the treatment of neuropathic pain, the remaining sections furnish the substance.

PATHOPHYSIOLOGY OF NEUROPATHIC PAIN

Specialized sensory neuronal fibers known as nociceptors innervate peripheral tissue, responding to noxious stimuli and initiating physiological pain.[6] Nociceptors are categorized as small-diameter unmyelinated C-fibers, small-diameter myelinated Aδ-fibers, and large-diameter myelinated Aβ-fibers.[7] C-fibers are dominant in clinical pain.[8] Innocuous tactile sensation produces low-intensity stimuli activating Aβ-fibers, while high-intensity stimuli activate Aδ-fibers and C-fibers, resulting in pain.[4] Nociceptive sensory nerves consist of a variety of ion channels and receptors that are modified upon stimulation. The three most recognized stimuli are temperature changes, mechanical stimuli, and chemicals. These stimuli interact with specific ion channels to activate the sensory neuron, while other stimuli activate protein kinase A. Protein kinase A phosphorylates sodium channels, causing reduction of the sensory neuron threshold; activated ion channels similarly reduce the sensory neuron threshold.[4] Besides modulating ion channels and sodium channels, noxious stimuli produce an inflammatory response involving infiltration of macrophages, neutrophils, and lymphocytes into the damaged tissue; these cells, with resident cells, produce and release proinflammatory cytokines and endogenous opioids.[7] Moreover, cytokines, damaged tissue cells, and surrounding cells upregulate the expression of cyclooxygenase-2 and nitric oxide synthetases. Cyclooxygenase-2 produces prostanoids and nitric oxide synthetase produces nitric oxide; nitric oxide promotes

vasodilation that facilitates entrance of immune cells and other mediators into the injury site. In other words, inflammation causes nociceptors to become sensitized and easily activated; this may explain the occurrence of mechanical allodynia and thermal hyperalgesia following tissue injury.[9]

Since inflammatory mediators serve an essential role in nociceptor sensitivity, mitigating the inflammatory response should reduce pain. One category of agents used to block the inflammatory process is NSAIDs. NSAIDs inhibit cyclooxygenase enzymes, thus blocking production of prostanoids (e.g., prostaglandin 2). Diclofenac, ketoprofen, indomethacin, and ibuprofen are some the NSAIDs that have been used to treat peripheral pain. In 2007, the U.S. Food and Drug Administration (FDA) approved two topical diclofenac products, Flector patch and Voltaren gel. The patch was approved for the treatment of acute pain in minor strains, sprains, and contusions, and the gel's approved indication was osteoarthritis pain in joints. Other topical NSAIDs are in various stages of seeking FDA approval, and, in the near future, additional topical NSAIDs will be available in the U.S.

As already mentioned, inflammation facilitates access of lymphocytes, monocytes, macrophages, and granulocytes to the damaged areas; these immune cells secrete endogenous opioids: enkephalins, dynorphins, and endorphins; endogenous opioids couple to opioid receptors on nociceptors; inflammation causes dorsal root ganglion to increase the synthesis and expression of opioid receptors (Figure 2).[10-13] Three opioid receptors, mu (μ), kappa (κ), and delta (δ), mediate inhibition of pain throughout the nervous system. Identification of opioid receptors and elucidation of their function in the peripheral sensory neurons have been areas of particular interest recently.[14] When endogenous or exogenous opioids bind to the opioid receptor on nociceptors, G-protein is activated and inhibits adenylyl cyclase. Inhibiting adenylyl cyclase decreases cAMP levels, and this in turn suppresses the activity of voltage-gated sodium channels (VGSCs), high-voltage calcium channels, and inflammatory sensitive nonselective cation currents. The primary effect, however, is modulation of high-voltage calcium channels. Thus, delivery of exogenous opioids to neuropathic sites could result in significant attenuation of nerve excitability

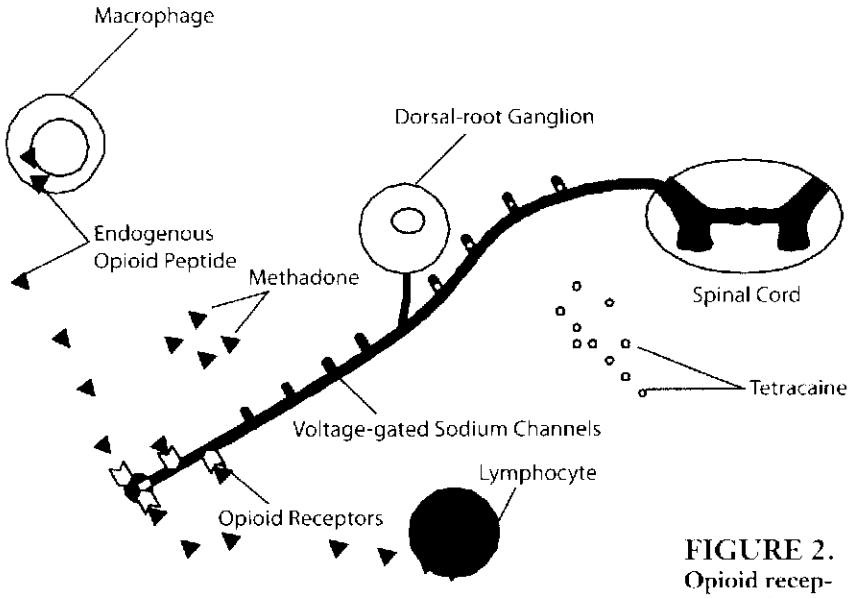

FIGURE 2.
Opioid receptor synthesis and expression.

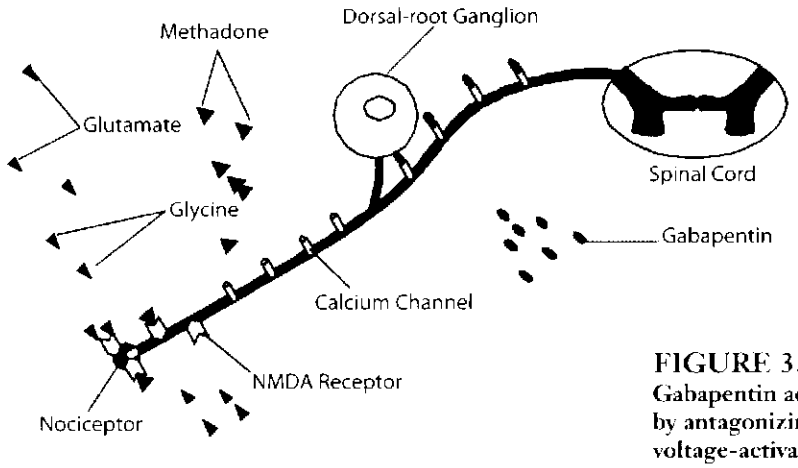

FIGURE 3.
Gabapentin acts by antagonizing voltage-activated calcium channels and modulating activity of GABA.

and consequently a decrease in pain. Moreover, exogenous opioids can work synergistically with endogenous opioids to further suppress pain. Overall, opioids hyperpolarize nociceptors, truncate propagation of action potentials, and prevent release of proinflammatory neuropeptides from sensory nerve terminals. The cumulative results of these actions are analgesia and reduction of inflammation.[6,15]

Oral opioids have been shown to be effective in treating peripheral neuropathic pain but are associated with substantial rates of systemic side effects, such as constipation, dizziness, confusion, mood changes, dry mouth, and somnolence.[15,16] Several studies have shown

Rx KETOPROFEN/TETRACAINE 10%/5% PLURONIC LECITHIN ORGANOGEL

		For 60 mL
Ketoprofen		6 g
Tetracaine		3 g
Ethanol 95%		5.5 mL
Lecithin and isopropyl palmitate solution		13.2 mL
Pluronic F-127 20% solution	qs	60 mL

METHOD OF PREPARATION

1. Weigh ketoprofen and tetracaine.
2. Place the lecithin and isopropyl palmitate solution in a 60-mL luer-to-luer locked syringe.
3. Add the ketoprofen, ethanol, and tetracaine to the syringe.
4. Bring final volume to 60 mL with Pluronic F-127 20% solution.
5. Exchange contents between syringes 15 times.
6. Package and label.

STABILITY

This cream-colored formula has a beyond-use date of 30 days. Store at room temperature.

Rx METHADONE/GABAPENTIN 0.1%/5% PLURONIC LECITHIN ORGANOGEL

		For 60 mL
Methadone hydrochloride		0.06 g
Gabapentin		3 g
Purified water		5 mL
Lecithin and isopropyl palmitate solution		13 mL
Pluronic F-127 20% solution	qs	60 mL

METHOD OF PREPARATION

1. Weigh methadone and gabapentin.
2. Place the lecithin and isopropyl palmitate solution in a 60-mL luer-to-luer locked syringe.
3. Add the gabapentin, methadone, and purified water to the syringe.
4. Bring final volume to 60 mL with Pluronic F-127 20% solution.
5. Exchange contents between syringes 15 times.
6. Package and label.

STABILITY

This cream-colored formula has a beyond-use date of 30 days. Store at room temperature.

that topical opioids offer a beneficial effect in treating cutaneous ulcers, with systemic drug concentrations either undetectable or no more than 20% of the subcutaneous dose.[17-21] In one study, topical methadone was effective in treating wound pain.[22] Besides the benefits of topical administration already mentioned, patients treated with topical opioids do not develop tolerance.[15] The plasticity of neuropathic pain and the findings of published studies offer a rationale for using topical opioids to treat peripheral neuropathies.

Calcium channels are implicated in neuron sensitivity, and, within the chronic nociceptor stimulus model, calcium ion influx contributes to the pathological neuronal plasticity found in peripheral neuropathy.[23] Calcium assists in protein kinase activation, which phosphorylates α-amino-3-hydroxy-5-methy-4-isoxazolepropionic acid (AMPA) and *N*-methyl-D-aspartate (NMDA) receptors. Phosphorylation of AMPA and NMDA receptors increases synaptic efficiency.[2] Antiepileptics represent one class of agents that affect calcium channels. Antiepileptics display several modes of action for the treatment of neuropathic pain. Gabapentin is probably the most widely recognized for its efficacy in this application. Its exact mechanism in relieving such pain has not been fully elucidated, but it does antagonize voltage-activated calcium channels ($\alpha_2\delta$ subunit) and modulates activity of gamma-aminobutyric acid (GABA), an inhibitory neurotransmitter.[24,25] The exact contribution of modulating GABA activity is still being investigated, but antagonizing voltage-activated calcium channels reduces calcium influx into the cell, which prevents neuron sensitivity (Figure 3). Clinically, gabapentin's antinociceptive action attenuates mechanical and thermal hypersensitivity in peripheral neuropathic pain.[26]

Besides inhibition through interference in calcium-mediated activation, NMDA receptors can be blocked directly. NMDA receptors are ligand-gated and voltage-dependent ion channels, requiring both an appropriate ligand and appropriate current for activation. The excitatory neurotransmitter glutamate is the most commonly recognized ligand for NMDA receptor activation.[27] Nociceptor terminals release glutamate in response to noxious stimuli. If the stimulus exceeds the neuronal threshold, the neuron depolarizes. When the membrane depolarizes, glutamate and glycine bind to an NMDA receptor, the receptor is activated, the receptor facilitates sodium and calcium ion influx and potassium efflux, and the neuron further depolarizes (Figure 3).[25] At the site of injury and during the nociceptive transfer of information, not only are NMDA receptors activated, but they also are upregulated, which furthers sensitization. Under physiological conditions, intercellular magnesium blocks NMDA receptor activity, but magnesium binds weakly when the membrane is depolarized and, thus, in pathological conditions, magnesium inhibits the receptor less.

NMDA receptors represent a relatively new drug target for the treatment of neuropathic pain. Current research seeks to develop an NMDA antagonist that will act specifically to block the pain process and not cause undue side effects. Studies show that NMDA antagonists mitigate neuronal sensitization and long-term potentiation and may lead to long-term neuronal resetting or downregulation. In other words, NMDA antagonists may diminish pain and correct underlying pathological processes.

Available exogenous NMDA antagonists include methadone, dextromethorphan, ketamine, and memantine. Methadone provides

a superior pharmocodynamic profile because it acts not only as an NMDA noncompetitive antagonist but also as an opioid agonist.

Under physiological conditions, the sensitized and excited terminal end of nociceptors results in generation of action potentials being transmitted via VGSCs along the sensory nerve axons. VGSCs are obligatory for the initiation and propagation of action potentials.[28] This communication system transfers information from the sensory nerve terminal to the dorsal horn and from the dorsal horn to the spinal cord, then to the thalamus and ultimately to the cortex (see Figure 1).[24] In neuropathic pain, however, VGSCs convey unwarranted signaling, usually spontaneous and unprovoked (Figure 2).[5] Besides generating unwarranted action potentials, VGSCs have an exaggerated response to innocuous stimuli, allodynia and noxious stimuli, and hyperalgesia; accumulation of aberrant VGSCs at the site of injury also may account for neuronal hyperexcitability and instability.[29] Therefore, anesthetics, which block VGSCs, mitigate unsolicited action potentials, prevent activation of accumulated channels, and reduce pain. Topical anesthetics (i.e., tetracaine, lidocaine, carbamazepine, mexiletine) show unequivocal evidence of alleviating pain in damaged tissue.[5] This approach has proven efficacious with lidocaine patches.[30-32]

In neuropathic pain, all these molecular mechanisms are irregular, which results in long-term neural plasticity; neuronal change manifests following repeated activation of nociceptive pathways.[6,23,33,34] The culmination of these changes makes the nociceptive system more sensitive to subsequent stimuli, producing the clinical syndromes of allodynia and hyperalgesia.[2]

PHARMACOLOGICAL TREATMENTS FOR NEUROPATHIC PAIN

Based on the molecular changes in neuropathic pain and specific drugs that can inhibit or alter these changes, the rationale for drug treatment becomes fairly linear. Either a combination of drugs with different mechanisms of action (e.g., methadone, tetracaine) should be used, or a single drug with multiple modes of actions (e.g., methadone).

The vehicle to use will depend on whether the skin is intact or compromised. For intact skin, a Pluronic lecithin organogel (PLO) or other penetrating gel will work well. Penetration enhancers, such as ethanol or D-limonene, may be used to increase absorption. Caution should be exercised in applying these gels to the jawline or face, because application in these areas has been shown to result in significant systemic absorption.

Surgical gels or Manuka honey work well as vehicles on compromised skin such as wounds. Manuka honey has the added benefits of being antimicrobial, a mild debrider, and a good barrier.

Table 1 shows the most common combinations, strengths, and vehicles used. If desired, the strengths may be increased for some of the formulations, but higher concentrations are usually not needed to achieve a therapeutic result, and at higher concentrations working with and maintaining the stability of the vehicles become more difficult. As mentioned in the introduction, other combinations of medications have been and can be used; the combinations listed here have been clinically successful and their molecular targets indicate that they address multiple abnormal sites in the neuropathic pain process. The agents used most frequently to treat pain in patients of Pikes Peak Hospice & Palliative Care, Inc. are the following:

- Gabapentin: long recognized for its efficacy in neuropathic pain
- Ketoprofen: excellent skin penetration; effectiveness in topical pain relief documented in numerous studies
- Methadone: dual activity as opioid agonist and NMDA antagonist, thus addressing neuropathic abnormalities at two distinct sites
- Tetracaine: more favorable pharmacokinetic properties than lidocaine with less skin irritation

TABLE 1. Topical Pain Preparations Most Frequently Used to Treat Patients of Pikes Peak Hospice & Palliative Care, Inc.

Drugs	Strength	Vehicle
Methadone/Tetracaine	0.1-1%/5%	Honey, surgical gel, PLO
Methadone/Ketoprofen	0.1-1%/10%	PLO
Ketoprofen	10%	PLO
Tetracaine	5%	Honey, surgical gel, PLO

PLO = Pluronic lecithin organogel

CASE REPORTS

Case 1

Patient LM, a 79-year-old white woman, was admitted to hospice with a diagnosis of pulmonary hypertension. On admission, one of the patient's primary problems was, "My right lower leg is my trouble spot." The practitioner recorded the pain as "severe, shooting pain across the anterior lower leg that is very bothersome." To control the pain, the patient had been treated with nortriptyline 25 mg at bedtime and hydrocodone/acetaminophen 5/500 mg as needed. The patient continued on oral medications but the pain remained; the practitioner added PLO gabapentin 5%, to be applied 2 mL twice daily to the patient's lower leg. The next day, the patient reported no pain in the right lower leg, and, upon assessment 2 weeks later, the patient reported that, following treatment with the PLO gabapentin, her leg pain had decreased from a level 8 of 10 to 2 to 3 of 10.

Case 2

Patient EH, a 54-year-old white woman, was admitted to hospice with a diagnosis of terminal colon cancer. As a result of chemotherapy, the patient developed bilateral peripheral neuropathies in the hands and feet, and at the time of admission the patient's hands and feet could not

Rx METHADONE/TETRACAINE 0.5/5% IN HONEY

		For 60 mL
Methadone hydrochloride		0.3 g
Tetracaine hydrochloride		3 g
Purified water		5 mL
Honey	qs	60 mL

METHOD OF PREPARATION

1. Weigh methadone and tetracaine hydrochloride.
2. Weigh out honey in beaker with stir bar.
3. Add methadone hydrochloride, tetracaine hydrochloride, and water to the beaker.
4. Heat mixture until methadone and tetracaine dissolve in the honey.
5. Pour into a 60-mL syringe.
6. Mix well.

STABILITY

This formula has a beyond-use date of 30 days. Store at room temperature.

Rx METHADONE/TETRACAINE 0.5%/5% PLURONIC LECITHIN ORGANOGEL

		For 60 mL
Methadone hydrochloride		0.3 g
Tetracaine		3 g
Ethanol 95%		2.5 mL
Lecithin and isopropyl palmitate solution		13 mL
Pluronic F-127 20% solution	qs	60 mL

METHOD OF PREPARATION

1. Weigh methadone and tetracaine.
2. Place the lecithin and isopropyl palmitate solution in a 60-mL luer-to-luer locked syringe.
3. Add the methadone hydrochloride, tetracaine, and ethanol to the syringe.
4. Bring to a final volume of 60 mL with Pluronic F-127 20% solution.
5. Exchange contents between syringes 15 times.
6. Package and label.

STABILITY

This cream-colored formula has a beyond-use date of 30 days. Store at room temperature.

Rx METHADONE/KETOPROFEN 0.1%/10% PLURONIC LECITHIN ORGANOGEL

		For 60 mL
Ketoprofen		6 g
Methadone		0.06 g
Ethanol 95%		5.5 mL
Lecithin and isopropyl palmitate solution		13.2 mL
Pluronic F-127 20% solution	qs	60 mL

METHOD OF PREPARATION

1. Weigh ketoprofen and methadone.
2. Place the lecithin and isopropyl palmitate solution in a 60-mL luer-to-luer locked syringe.
3. Add the ketoprofen, methadone, and ethanol to the syringe.
4. Bring final volume to 60 mL with Pluronic F-127 20% solution.
5. Exchange contents between syringes 15 times.
6. Package and label.

STABILITY

This cream-colored formula has a beyond-use date of 30 days. Store at room temperature.

Rx METHADONE 0.1% PLURONIC LECITHIN ORGANOGEL

		For 60 mL
Methadone hydrochloride		0.06 g
Lecithin and isopropyl palmitate solution		6.6 mL
Pluronic F-127 20% solution	qs	60 mL

METHOD OF PREPARATION

1. Weigh methadone.
2. Place the lecithin and isopropyl palmitate solution in a 60-mL luer-to-luer locked syringe.
3. Add the methadone to the syringe.
4. Bring final volume to 60 mL with Pluronic F-127 20% solution.
5. Exchange contents between syringes 15 times.
6. Package and label.

STABILITY

This formula has a beyond-use date of 30 days. Store at room temperature.

be touched without causing excruciating pain. To control the pain, the patient was taking oxycodone/acetaminophen 7.5/750 mg every 4 hours as needed, tramadol 1 tablet every 4 hours, and transdermal fentanyl 25 mcg patch, applying 1 patch and changing every 72 hours. The neuropathy persisted despite oral and transdermal opioid administration. Oral and transdermal medications were continued, and the patient was started on PLO gabapentin 5% to be applied 1 mL to each hand and foot twice daily. Three days later, the patient reported that the pain was improved, and the staff said they could touch and move the patient's hands and feet without causing pain. Ten days after starting the PLO gabapentin, the patient reported that the pain level in her hands and feet had decreased from an 8 to 9 of 10 to a 2 of 10.

Case 3

Patient RH, a 91-year-old woman, was admitted to hospice with a diagnosis of debility. Cardiovascular disease, pulmonary disease, and type 2 diabetes have contributed to her decline. The patient has bilateral peripheral neuropathies in her legs due to her diabetes. At admission, she was not on any medication to treat the neuropathy. The hospice practitioner prescribed PLO methadone/tetracaine 0.5/5%, applied 2 to 3 mL to each leg twice daily. Several days later, the patient reported her pain level had declined from a 7 of 10 to a 0 of 10, and that she routinely gets about 15 hours of pain relief after application of the PLO.

Case 4

Patient VF, a 61-year-old woman, was admitted to hospice with a diagnosis of malignant breast cancer. The patient experienced tumor eruptions in 80% of her anterior chest and 40% of her right posterior chest. Two of the tumors, one anterior and one posterior, spontaneously erupted and bled, and eventually scabbed. The patient rated her pain level at a background of 7 of 10, and sometimes as severe as 10 of 10. Treatment with Oxycontin 40 mg, 1 tablet twice daily, improved the pain, but she continued to experience episodic pain at a level of 8 of 10 in her anterior and posterior chest. To the patient's regimen the practitioner added methadone/tetracaine 0.5/5% in Manuka honey, to be applied 4 to 5 mL to open lesions every dressing change. After the honey compound was applied, the patient reported her pain level dropped to 0 of 10, and the pain relief lasted 24 hours.

The formulations used in treating these patients' pain are included in this article.

CONCLUSION

Pikes Peak Hospice & Palliative Care, Inc., cares for more than 200 patients with terminal disease who reside at home, in long-term care facilities, in assisted-living facilities, or its acute-care Inpatient Unit. Over the past 4 years, the hospice pharmacy has evolved to provide individualized compounded preparations to meet the unique and complex needs of our patients. An integral part of this evolution is the development and implementation of topical treatments for pain. As illustrated by the case presentations, patients in hospice care often have multiple medical conditions and complicated health issues. Nearly 50% of our patients receive a topical compound to treat any of a number of conditions, including neuropathies, muscle skeletal pain, wound pain, agitation, and anxiety. Topical compounds give practitioners another option in managing patient maladies and, in many instances, compounded topicals have proven as effective, if not more effective, than commercially available products. In some cases, patients have been able to stop oral pain medications or reduce their dose because the topical preparation is so effective. All in all, the topical preparations described in this article have been invaluable to our patients' comfort and an indispensable option for practitioners.

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Rx METHADONE/TETRACAINE 0.1%/5% IN SURGILUBE

		For 60 mL
Methadone hydrochloride		0.06 g
Tetracaine hydrochloride		3 g
Purified water		5 mL
Surgilube	qs	60 mL

METHOD OF PREPARATION

1. Weigh methadone hydrochloride and tetracaine hydrochloride.
2. Fill luer-to-luer lock 60-mL syringes half-full of Surgilube.
3. Add methadone hydrochloride and tetracaine hydrochloride to Surgilube, along with 5 mL of purified water.
4. Bring to volume of 60 mL with Surgilube.
5. Place piston into syringe and eliminate air.
6. Exchange content between syringes 15 times.

STABILITY

This gel formula has a beyond-use date of 30 days. Store in refrigerator.

Rx TETRACAINE 5% IN PLURONIC LECITHIN ORGANOGEL

		For 60 mL
Tetracaine		3 g
Ethanol 95%		2.5 mL
Lecithin and isopropyl palmitate solution		6.6 mL
Pluronic F-127 20% solution	qs	60 mL

METHOD OF PREPARATION

1. Weigh tetracaine.
2. Place the lecithin and isopropyl palmitate solution in a 60-mL luer-to-luer locked syringe.
3. Add the tetracaine and ethanol to the syringe.
4. Bring final volume to 60 mL with Pluronic F-127 20% solution.
5. Exchange contents between syringes 15 times.
6. Package and label.

STABILITY

This formula has a beyond-use date of 30 days. Store at room temperature.

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Address correspondence to Vince Vidaurri, RPh, BCPS, Supervisor, Palliative Pharmacy of Pikes Peak, Colorado Springs, CO 80903. E-mail: pharmacy@pikespeakhospice.org