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PRESCRIPTION COMPOUNDING FOR

PAIN MANAGEMENT

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We customize individual prescriptions for the specific needs of our patients.
SCIATIC PAIN

The following study found that lidocaine reduces both spontaneous and evoked sciatic pain—“Intravenous lidocaine, amantadine, and placebo in the treatment of sciatica: a double-blind, randomized, controlled study” (Reg Anesth Pain Med. 1999 Nov-Dec;24(6):534-40).

BACKGROUND AND OBJECTIVES: Sciatica is a neuropathic pain syndrome caused by compression and/or inflammation of spinal nerve roots by herniated disc material, and its treatment is therefore usually aimed at reducing compression and inflammation. Studies have shown that both systemic local anesthetics and N-methyl-D-aspartate (NMDA) receptor antagonists may produce analgesia in a variety of neuropathic pain syndromes. The present study evaluated the analgesic efficacy of i.v. infusions of the local anesthetic lidocaine, the NMDA receptor antagonist amantadine, and a placebo in sciatica.

METHODS: Thirty patients with sciatica, as confirmed by physical examination and imaging studies, were enrolled in a randomized, double-blind, three-arm crossover trial. Infusions of amantadine (2.5 mg/kg), lidocaine (5 mg/kg), and a placebo were administered over a 2-hour period, 2-7 days apart from each other. Spontaneous pain (visual analog scale) and evoked pain (straight leg raise) were measured every 30 minutes for 3 hours.

RESULTS: Lidocaine reduced spontaneous pain as compared with amantadine and with the placebo for all measurements and at a significant level at the 30 (P < .05), 120, and 180 (P < .01) minute time points. Maximal pain reduction from the baseline was 62 +/- 7% for lidocaine, 43 +/- 7% for amantadine, and 47 +/- 7% for the placebo. Straight leg raise test also significantly improved with lidocaine (from 30 to 37 degrees; P < .05), as compared to amantadine (34-36 degrees) and to the placebo (32-34 degrees). All three treatments were relatively well tolerated.

CONCLUSIONS: Intravenous lidocaine, rather than amantadine, reduces both spontaneous and evoked sciatic pain. PMID: 10588558

An example of how you might prescribe follows:

### COMPOUNDED MEDICATION

<table>
<thead>
<tr>
<th>Gabapentin 6%/Lidocaine 5%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Transdermal Cream</td>
</tr>
<tr>
<td>90gm</td>
</tr>
<tr>
<td>Apply TID PRN</td>
</tr>
</tbody>
</table>

The following case report describes sciatica successfully controlled with gabapentin—“Gabapentin as a potential option for treatment of sciatica” (Pharmacotherapy. 2008 Mar;28(3):397-402).

ABSTRACT: “Gabapentin has been approved in the United States for the treatment of epilepsy and postherpetic neuralgia. Gabapentin has also demonstrated proven efficacy for the treatment of diabetic peripheral neuropathy and trigeminal neuralgia, although these represent off-label uses of the drug. However, to our knowledge, no data have been published regarding the efficacy of gabapentin for treating sciatica. We describe two patients with sciatica who were successfully treated with gabapentin. The first was a 32-year-old man with severe shooting pain in his left leg that was later diagnosed as sciatica secondary to a fifth lumbar-first sacral intervertebral disk herniation. The patient was treated with acetaminophen, nonsteroidal antiinflammatory drugs (NSAIDs), narcotics, and muscle relaxants; he reported only limited pain relief with any of these agents or combination of agents. He was then prescribed gabapentin 300 mg once/day; his pain substantially improved, even after the first dose. The drug was titrated gradually up to 900 mg 3 times/day with good results. The patient subsequently underwent a laminectomy and discectomy on the advice of his neurosurgeon, who assured him that the result would be immediate pain relief. After surgery, the patient continued to experience pain; however, his pain resolved completely after several weeks of receiving gabapentin 600 mg 3 times/day.

The second patient was a 68-year-old Caucasian woman with renal insufficiency who experienced severe burning pain and numbness of abrupt onset in the posterior right leg; this was diagnosed as sciatica. The patient had contraindications for NSAID therapy and was intolerant of hydrocodone. Initial therapy with propoxyphene and acetaminophen, self-started by the patient, was ineffective. Gabapentin 100 mg at bedtime was started and then titrated up to 100 mg twice/day with 200 mg at bedtime. The patient’s pain improved rapidly, and at follow-up approximately 5 weeks later, she was experiencing good pain control with gabapentin. Gabapentin is widely prescribed for management of peripheral neuropathic pain syndromes. To our knowledge, however, these two case reports are the first to describe sciatica successfully controlled with gabapentin. Because gabapentin has the potential to prevent central sensitization, consideration should be given to prescribing this therapy early in the course of sciatica. Further research using randomized, placebo-controlled trials are needed to validate the benefit of gabapentin in the treatment of sciatica.” PMID: 18294119

With our state of the art compounding lab and pharmaceutical knowledge and experience, we can compound lidocaine and gabapentin into one transdermal cream.
The following clinical papers discuss the effectiveness of ketamine and lidocaine in treating allodynia in patients with different types of pain.


**ABSTRACT:** “A double-blind placebo-controlled crossover trial was used to determine the effects of topical ketamine, an N-methyl-d-aspartate (NMDA) receptor antagonist, on the sensory disturbances in 20 patients with complex regional pain syndrome (CRPS). On two occasions separated by at least one week, sensory tests to light touch, pressure, punctate stimulation, light brushing and thermal stimuli were performed in the symptomatic and contralateral limb and on each side of the forehead before and 30min after 10% ketamine cream was applied to the symptomatic or healthy limb. Venous blood for the plasma estimations of ketamine and norketamine was obtained 1h after application of the creams. Ketamine applied to the symptomatic limb inhibited allodynia to light brushing and hyperalgesia to punctate stimulation. Systemic effects of the ketamine are unlikely to account for this as the plasma levels were below detectable limits. Touch thresholds were unchanged, NMDA receptors may contribute to the sensory disturbances in CRPS via actions at cutaneous nociceptors. Allodynia and hyperalgesia were detected in the ipsilateral forehead to a range of stimuli (brushing, pressure, punctate stimulation, cold, heat, and warmth). In several patients, ketamine treatment of the symptomatic limb inhibited allodynia to brushing the ipsilateral forehead, suggesting that the mechanism that mediates allodynia in the symptomatic limb contributed to allodynia at more remote sites. The present study shows promise for the use of topical ketamine as opposed to parenteral and oral forms which often result in undesirable side effects.” PMID: 19703730


**ABSTRACT:** “This study investigated the effect of intravenous lidocaine at two doses (1 mg/kg and 5 mg/kg over 2 hours) and an intravenous saline placebo on the pain and allodynia of postherpetic neuralgia (PHN). Twenty-four patients were studied using a randomized, double-blind, within-patient crossover design. Each patient received normal saline, lidocaine 0.5 mg/kg/h, and lidocaine 2.5 mg/kg/h for a 2-h period. The McGill Pain Questionnaire Short Form, visual analogue scores (VAS), and area of allodynia were measured at intervals during the infusions. Free plasma lidocaine levels were also measured. The results were statistically analyzed using Student’s t-test for paired data. The VAS for ongoing pain showed a significant reduction after all the infusions (P < 0.05). For dynamic pressure-provoked pain, the VAS was unaffected by placebo but showed a reduction at an equal level of significance with both lidocaine infusions (P < 0.05). The area of allodynia of PHN, as mapped by brush stroke, declined in association with intravenous lidocaine (0.5 mg/kg/h = P < 0.05; 2.5 mg/kg/h = P < 0.001). Placebo had no significant effect on the area of allodynia. These findings demonstrate a positive effect on pain and allodynia following a brief intravenous infusion of lidocaine. The higher dose infusion may produce plasma levels in the toxic range, with no significant clinical increase in response.” PMID: 10388248

An example of how you might prescribe follows:

**COMPounded medication**

<table>
<thead>
<tr>
<th>Ketamine 5%/Lidocaine 10% Topical Gel</th>
</tr>
</thead>
<tbody>
<tr>
<td>100ml</td>
</tr>
<tr>
<td>Apply sparingly to affected area(s) 3-4x daily PRN</td>
</tr>
</tbody>
</table>

With our state of the art compounding laboratory and pharmaceutical knowledge and experience, we have the ability to compound ketamine and lidocaine into one topical gel.
PAIN

The following study found that bioadhesive gels of ketoprofen could be used for effective transdermal therapy - “Enhanced transdermal delivery of ketoprofen from bioadhesive gels” (Pak J Pharm Sci, 2009 Apr;22(2):193-8).

**ABSTRACT:** “The aim of this study was to evaluate and compare the in vitro and in vivo transdermal potential of bioadhesive gels of ketoprofen by using gelling polymers like sodium carboxymethylcellulose, xanthan gum, poloxamer 407 and carbopol 934P as bioadhesive polymer with and without penetration enhancer (oleic acid). The effect of oleic acid as a penetration enhancer was examined when it was added to the bioadhesive formulations. Gels were evaluated for bioadhesive force and viscosity. To study the in vitro potential of these formulations, permeation studies were performed with Franz diffusion cell using excised rat abdominal skin. Carrageenan induced rat paw edema model was used to investigate their in vivo performance. The commercial formulation of ketoprofen was used as a reference formulation. The in vitro permeation studies indicate that ketoprofen bioadhesive gel of poloxamer 407 with penetration enhancer was superior to gels of sodium carboxymethylcellulose and xanthan gum with penetration enhancer (oleic acid). The permeation rate of ketoprofen from poloxamer 407 based bioadhesive gel with 15% v/w penetration enhancer was higher (rat abdominal skin flux = 0.421 +/- 0.032 mg/cm(2)/h) than the permeation rate of sodium carboxymethylcellulose and xanthan gum based bioadhesive gel with 15% v/w penetration enhancer. In the paw edema test poloxamer 407 based bioadhesive gel with 15% v/w penetration enhancer showed the best permeation and effectiveness. The in vitro and in vivo studies showed that bioadhesive gels of ketoprofen could be used for effective therapy.” PMID: 19339232

We have the ability to compound ketoprofen in poloxamer as a transdermal gel. This compound has the ability to serve those who are unable to take oral NSAIDs.

An example of how you might prescribe follows:

**COMPOUNDED MEDICATION**

<table>
<thead>
<tr>
<th>Ketoprofen 20% Poloxamer Transdermal Gel</th>
</tr>
</thead>
<tbody>
<tr>
<td>90ml</td>
</tr>
<tr>
<td>Apply sparingly to affected area(s) TID</td>
</tr>
</tbody>
</table>

We have the ability to compound ketoprofen in poloxamer as a transdermal gel. This compound has the ability to serve those who are unable to take oral NSAIDs.
Sciatic Pain

[ ] Gabapentin 6%/Lidocaine 5%  
Transdermal Cream

Quantity 90gm  
Directions: Apply TID PRN

Allodynia

[ ] Ketamine 5%/Lidocaine 10%  
Topical Gel

Quantity 100ml  
Directions: Apply sparingly to affected area(s) 3-4x daily PRN

Pain

[ ] Ketoprofen 20%  
Poloxamer Transdermal Gel

Quantity 90ml  
Directions: Apply sparingly to affected area(s) TID

Directions


Prescriber’s Signature____________________________________  Refills: 1 2 3 4 5 6 7 8 9 10 11 12 NR

All topical compound %s are per 1 ml or 1 gm unless otherwise noted