MEDICAL CONTESTED CASE HEARING NO. 13047

DECISION AND ORDER

This case is decided pursuant to Chapter 410 of the Texas Workers' Compensation Act and Rules of the Division of Workers' Compensation adopted thereunder.

ISSUES

A contested case hearing was held on January 16, 2013, to decide the following disputed issue:

1. Is the preponderance of the evidence contrary to the decision of the Independent Review Organization (IRO) that prescriptions for Avinza, Neurontin, Flexeril, Klonopin and Lidoderm patches are reasonably required health care for the compensable injury of (Date of Injury)?

PARTIES PRESENT

Petitioner/Carrier appeared and was represented by CL, attorney. Claimant appeared by telephone and was assisted by SR, ombudsman. Respondent/Provider appeared by telephone as a witness.

BACKGROUND INFORMATION

Claimant sustained a compensable lumbar spine injury on (Date of Injury), and has had multiple surgeries. He has been diagnosed with failed back syndrome and currently sees TJ, MD for pain management. Dr. J has determined that Claimant is not a surgical candidate and has prescribed multiple medications to manage Claimant's pain and allow him to function. Among the medications prescribed to Claimant are the ones at issue in this hearing. In a determination dated October 10, 2012, an IRO physician reviewer concurred that the medications were reasonably necessary health care for the compensable injury of (Date of Injury). In that decision, the IRO physician reviewer also determined that a compounded topical cream and Protonix were not reasonably required. That portion of the IRO decision has not been appealed.

Texas Labor Code Section 408.021 provides that an employee who sustains a compensable injury is entitled to all health care reasonably required by the nature of the injury as and when needed. The term "health care" includes a prescription drug, medicine, or other remedy (Texas Labor Code §401.011(19)(E)) and is further defined in Texas Labor Code Section 401.011 (22a) as health care that is clinically appropriate and considered effective for the injured employee's injury and provided in accordance with best practices consistent with evidence based medicine or, if evidence based medicine is not available, then generally accepted standards of medical

practice recognized in the medical community. Health care under the Texas Workers' Compensation system must be consistent with evidence based medicine if that evidence is available. Evidence based medicine is further defined in Texas Labor Code Section 401.011 (18a) to be the use of the current best quality scientific and medical evidence formulated from credible scientific studies, including peer-reviewed medical literature and other current scientifically based texts and treatment and practice guidelines in making decisions about the care of individual patients.. The Commissioner of the Division of Workers' compensation is required to adopt treatment guidelines that are evidence-based, scientifically valid, outcome-focused and designed to reduce excessive or inappropriate medical care while safeguarding necessary medical care. Texas Labor Code Section 413.011(e). Medical services consistent with the medical policies and fee guidelines adopted by the commissioner are presumed reasonable in accordance with Texas Labor Code Section 413.017(1).

In accordance with the above statutory guidance, the Division of Workers' Compensation has adopted treatment guidelines by Division Rule 137.100. This rule directs health care providers to provide treatment in accordance with the current edition of the Official Disability Guidelines (ODG), and such treatment is presumed to be health care reasonably required as defined in the Texas Labor Code. Thus, the focus of any health care dispute starts with the health care set out in the ODG. Also, in accordance with Division Rule 133.308 (s), a decision issued by an IRO is not considered an agency decision and neither the Department nor the Division is considered a party to an appeal. The party appealing the IRO decision has the burden of overcoming the decision issued by an IRO by a preponderance of evidence-based medical evidence. The hearing officer is directed to consider the relevant treatment guidelines.

Avinza is an opioid. The low back section of the ODG addresses the prescription and use of opiods as follows:

> Opioids
>
> Not recommended except for short use for severe cases, not to exceed 2 weeks. See the Pain Chapter for more information and studies. When used only for a time-limited course, opioid analgesics are an option in the management of patients with acute low back problems. The decision to use opioids should be guided by consideration of their potential complications relative to other options. Patients should be warned about potential physical dependence and the danger associated with the use of opioids while operating heavy equipment or driving. The studies found that patients taking opioid analgesics did not return to full activity sooner than patients taking NSAIDs or acetaminophen. In addition, studies found no difference in pain relief between NSAIDs and opioids. Finally, side effects of opioid analgesics were found to be substantial, including the risk for physical dependence. These side effects are an important concern in conditions that can

> become chronic, such as low back problems. (Bigos, 1999) Recent studies document a 423% increase in expenditures for opioids for back pain, without demonstrated improvements in patient outcomes or disability rates. (Deyo, 2009) With opioid therapy for nonspecific low back pain compared with no opioids, the odds of chronic work loss were six times greater for claimants with schedule II ("strong") opioids; were 11-14 times greater for claimants with opioid prescriptions of any type during a period of >or=90 days; and 3 years after injury, costs of claimants with schedule II opioids averaged $19,453 higher than costs of claimants in the no opioids group. (Volinn, 2009) This large study found that prescription of opioids was common among patients with back pain, and increasing duration of opioid use was strongly associated with an increasing prevalence of mental health conditions (depression, anxiety, post-traumatic stress disorder, or substance abuse); almost 50% of patients receiving long-term opioids had at least one of these diagnoses. Similarly, negative health habits (obesity, smoking) were associated with duration of opioid use. The wisdom of long-acting opioid use for chronic pain remains controversial. (Deyo, 2011) For more information, and Criteria for Use of Opioids, see the Pain Chapter.

The pain section contains the following regarding the use of opioids:

> This topic is covered under multiple headings. See more specific entries, as follows: **Opioids, criteria for use**; Opioids for chronic pain; Opioids for neuropathic pain; Opioids for osteoarthritis; Opioids, cancer pain vs. nonmalignant pain; Opioids, dealing with misuse & addiction; Opioids, dosing; Opioids, indicators for addiction; Opioids, long-term assessment; Opioids, pain treatment agreement; Opioids, psychological intervention; Opioids, specific drug list; Opioids, screening for risk of addiction (tests); Opioids, state medical boards guidelines; Detoxification; Substance abuse (tolerance, dependence, addiction); Urine Drug Testing (UDT) in patient-centered clinical situations; Weaning of medications; Implantable drug-delivery systems (IDDSs); Methadone; Rapid detox; Testosterone replacement for hypogonadism (related to opioids); Opioid hyperalgesia & Opioids, specific drug list. Opioid drugs are also referred to as opiate analgesics, narcotic analgesics, or schedule C (II -IV) controlled substances. Opioid analgesics are a class of drugs (e.g., morphine, codeine, and methadone) that have a primary indication to relieve symptoms related to pain. Opioid drugs are available in various dosage forms and strengths. They are considered the most powerful class of analgesics that may be used to manage both acute and chronic pain. These medications are generally classified according to potency and duration of dosage duration.

Overall Classification:

Pure-agonists: include natural and synthetic opioids such as morphine sulfate (MS Contin®), hydromorphone (Dilaudid®), oxymorphone (Numorphan®), levorphanol (Levo-Dromoran®), codeine (Tylenol w/Codeine 3®), hydrocodone (Vicodin®), oxycodone (OxyContin®), methadone (Dolophine HCl®), and fentanyl (Duragesic®). This group of opioids does not have a ceiling effect for their analgesic efficacy nor do they antagonize (reverse) the effects of other pure opioids. (Baumann, 2002) Morphine is the most widely used type of opioid analgesic for the treatment of moderate to severe pain due to its availability, the range of doses offered, and its low cost.

Partial agonists-antagonists: agents that stimulate the analgesic portion of opioid receptors while blocking or having little or no effect on toxicity. This group of opiates includes buprenorphine (Suboxone®). Partial agonists-antagonists have lower abuse potential than pure-agonists, however the side effects of this class of analgesics include hallucinations and dysphoria. *Opioid antagonists* such as naloxone are included in this class. They are most often used to reverse the effects of agonists and agonist-antagonist derived opioids. (Baumann, 2002)

Mixed agonists-antagonists: another type of opiate analgesics that may be used to treat pain. They include such drugs as butorphanol (Stadol®), dezocine (Dalgan®), nalbuphine (Nubain®) and pentazocine (Talwin®). (Baumann, 2002) Mixed agonists-antagonists have limited use among chronic pain patients because of their ceiling effect for analgesia that results in the analgesic effect not increasing with dose escalation.

Central acting analgesics: an emerging fourth class of opiate analgesic that may be used to treat chronic pain. This small class of synthetic opioids (e.g., Tramadol) exhibits opioid activity and a mechanism of action that inhibits the reuptake of serotonin and norepinephrine. Central analgesics drugs such as Tramadol (Ultram®) are reported to be effective in managing neuropathic pain. (Kumar, 2003) Side effects are similar to traditional opioids.

Opioid Classifications: Short-acting/Long-acting opioids:

Short-acting opioids: also known as “normal-release” or “immediate-release” opioids are seen as an effective method in controlling both acute and chronic pain. They are often used for intermittent or breakthrough pain. These agents are often combined with other analgesics such as acetaminophen and aspirin. These adjunct agents may limit the upper range of dosing of short-acting agents due to their adverse effects. The duration of action is generally 3-4 hours. Short-acting opioids include Morphine (Roxanol®), Oxycodone (OxyIR®, Oxyfast®), Endocodone®, Oxycodone with acetaminophen, (Roxilox®, Roxicet®, Percocet®, Tylox®, Endocet®), Hydrocodone with acetaminophen, (Vicodin®, Lorcet®, Lortab®,

Zydone®, Hydrocet®, Norco®), Hydromorphone (Dilaudid®, Hydrostat®). (Baumann, 2002)

Long-acting opioids: also known as "controlled-release", "extended-release", "sustained-release" or "long-acting" opioids, are a highly potent form of opiate analgesic. The proposed advantage of long-acting opioids is that they stabilize medication levels, and provide around-the-clock analgesia. Long-acting opioids include: Morphine (MSContin®, Oramorph SR®, Kadian®, Avinza®), Oxycodone (Oxycontin®), Fentanyl (Duragesic Patch®), Hydromorphone (Palladone®). Note: On 01/26/10 Purdue Pharma suspended Palladone® from the US market due to adverse effects with alcohol. (FDA, 2010) The odds of being hypogonadal on long-acting opioids may be 4-5 times higher than the odds on a short-acting equipotent dose. (Rubinstein, 2012)

Claimant receives the Avinza to treat chronic rather than acute pain. The section on the use of opiods for chronic pain says:

Not recommended as a first-line treatment for chronic non-malignant pain, and not recommended in patients at high risk for misuse, diversion, or substance abuse. Recommended as a 2nd or 3rd line treatment option at doses ≤ 120 mg daily oral morphine equivalent dose (MED) as indicated below. Risk-benefit of use should be carefully weighed for substance abuse and overdose risks, including risk of death, and this information should be provided to the patient as part of informed decision-making. Extreme caution is required for any opioid use in patients with the following:

(1) Individuals with a high risk for misuse or diversion;
(2) Individuals with evidence of substance abuse issues;
(3) Individuals with a family history of substance abuse;
(4) Individuals with underlying psychiatric disease.

An accurate diagnosis should be established. At the minimum, screening for opioid risk and psychological distress inventories should occur before starting this class of drugs and a psychological evaluation is strongly recommended. Escalation of doses beyond 120 mg MED should be done with caution, and generally under the care of pain specialists. In certain cases, addiction specialists may need to evaluate patients, with the understanding that many patients who progress to chronic opioid therapy have underlying psychiatric disease and substance abuse issues. While long-term opioid therapy may benefit some patients with severe suffering that has been refractory to other medical and psychological treatments, it is not generally effective in achieving the original goals of complete pain relief and functional restoration. For patients now on high opioid doses who are not benefiting from them, if taken off the medications many would experience severe withdrawal or have to take addiction treatment drugs for years. See

Weaning of medications. To prevent new patients from getting caught in this cycle, ODG recommends consideration of a one-month limit on opioids for new chronic non-malignant pain patients in most cases.

Use for specific disease states

- *Neuropathic pain:* Opioids have been suggested for neuropathic pain that has not responded to first-line recommendations (antidepressants, anticonvulsants). There are no trials of long-term use. There are virtually no studies of opioids for treatment of chronic lumbar root pain with resultant neuropathy. See Opioids for neuropathic pain, where opioids are not recommended as a first-line therapy.
- *Chronic back pain:* Opioids appear to be efficacious but should be limited for short-term pain relief in patients with acute low back pain. Long-term efficacy is unclear (>16 weeks), and there is also limited evidence for the use of opioids for chronic low back pain. (Martell-*Annals,* 2007) (White, 2011) (Franklin, 2009) Failure of activity level to respond to a time-limited course of opioids has led to the suggestion of reassessment and consideration of alternative therapy. There is no evidence to recommend one opioid over another. In patients taking opioids for back pain, the prevalence of lifetime substance use disorders has ranged from 36% to 56% (a statistic limited by poor study design). Limited information indicates that up to one-fourth of patients who receive opioids exhibit aberrant medication-taking behavior. (Martell-*Annals*, 2007) (Chou, 2007) There are three studies comparing tramadol to placebo that have reported pain relief, but this did not necessarily improve function. (Deshpande, 2007) See also the Low Back Chapter for recommendations in acute pain, where opioids are not recommended except for short use for severe cases, not to exceed 2 weeks.
- *Headaches*: Not recommended, in particular, due to the risk of medication overuse headache. (Lake, 2008) (Olesen, 2006) See Medication overuse headache.
- *Osteoarthriti*s: Not recommended as a first-line therapy. Recommended on a trial basis for short-term use after there has been evidence of failure of first-line medication options such as acetaminophen or NSAIDs when there is evidence of moderate to severe pain. Also recommended for a trial if there is evidence of contraindications for use of first-line medications. There is a lack of evidence to allow for a treatment recommendation for long-term use. If used on a long-term basis, the criteria for use of opioids should be followed. See Opioids for osteoarthritis for citations. The American College of Rheumatology guidelines do not recommend opioids for osteoarthritis, except in patients who should have total joint arthroplasty but cannot. (Hochberg, 2012)

- *Nociceptive pain:* Recommended as the standard of care for treatment of moderate or severe nociceptive pain (defined as pain that is presumed to be maintained by continual injury, with the most common example being pain secondary to cancer).
- *Mechanical and* compressive *etiologies*: rarely beneficial.

Evidence for use: A major concern about the use of opioids for chronic pain is that most randomized-controlled trials are limited to a short-term period (1 to 6 months), with high rates of dropout due to adverse effects and/or lack of efficacy (as high as 60%). Studies usually exclude patients with mental health disease or substance abuse, limiting generalizability. Methodological issues result in limitations, with problems of studies including insufficiently comprehensive outcome assessment, and incomplete inclusion of adverse effects. Results suggest modest pain relief compared to placebo (approximately 30%), but there are no long-term studies to determine if pain relief is maintained. Overall, the safety of long-term use has not been adequately studied, and some nonrandomized prospective studies suggest opioid treatment may actually retard functional recovery. This leads to a concern about confounding issues such as tolerance, opioid-induced hyperalgesia, long-range adverse effects such as hypogonadism and/or opioid abuse, and the influence of placebo as a variable for treatment effect. (Eriksen, 2006) (Ballantyne, 2006) (Furlan, 2006) (Ballantyne, 2008) (Franklin, 2008) (Chou, 2009) (Chapman, 2010) (Papaleontiou, 2010) (Furlan, 2010) (Von Korff, 2011) (Manchikanti, 2011)

Upper limits of range of dose: The Washington State Department of Labor and Industries Guidelines suggest that the "upper limit of range" for opioids prior to evaluation with a pain specialist for the need for possible continuation of treatment, escalation of dose, or possible weaning, is 120 mg of oral morphine equivalents a day (MED). These values are based on factors such as evidence of increased risk of alcohol- or drug-related encounters (alcohol or drug intoxication, alcohol or drug withdrawal, or alcohol or drug overdose) at higher doses. Risk increases with a history of previous or ongoing substance abuse and concomitant use of opioids with sedative hypnotics and/or benzodiazepines. Progression to long-term use also increases with prescribing of higher doses of opioids. Other cohort studies have indicated lower rates of return to work, higher rate of healthcare utilization, and higher rates of going on to receive Social Security Disability Income with higher doses of opioids. With the introduction of a definition of high dose of opioids as $\geq$ 120 mg by the Washington State workers' compensation system, there was a 27% decrease in average morphine equivalents a day dispensed, a 35% decrease in the number of patients receiving > 120 mg/day of morphine equivalents (both compared to before 2007), and a 50% decrease in number of unintentional opioid deaths (2009-2010). (Ballantyne,

2006) (AMDG, 2007) (Kidner, 2009) (Kidner, 2010) (Braden, 2009) (Braden, 2010) (Dunn, 2010) (Bohnert, 2011) (Martin, 2011) (Franklin, 2012) See Opioids, dosing (morphine equivalent dose).

Patients most likely to receive high-dose opioids: Cohort studies indicate that small proportions of patients are most likely to receive the majority of opioids (in one study 5% of patients received 70% of opioids dispensed). Patients most likely to receive high-dose opioids in cohort studies are those who have multiple pain complaints, and have mental health and substance use disorders. These are generally patients who are excluded from randomized trials of opioids, which limit the generalizability of current studies. They are also more likely to be receiving concomitant benzodiazepines. Studies show these patients are more likely to have higher rates of medical diagnoses and higher Charlson comorbidity scores. (Sullivan, 2005) (Braden, 2009) (Edlund 2010) (Morasco, 2010) (Kidner, 2010) (Sullivan, 2012)

Risk factors for progressing to long-term opioid use: It is currently suggested that of the patients that proceed to long-term opioid use (90 days or more), two-thirds continue opioids for years later, creating life-long therapy. Current research involves evaluating what subsets of patients are likely to proceed to long-term use, particularly as

(1) the vast majority of patients in randomized-controlled studies abandon opioids after short-term use due to adverse effects and/or lack of efficacy and
(2) a small proportion of patients receive the majority of opioids dispensed.

Subclasses of individuals who continue with long-term use have been identified as patients who use high daily doses (>120 mg morphine equivalent/day) and/or have a history of opioid misuse. The likelihood of receiving long-term opioids increases with number of pain sites, increased baseline pain, decreased baseline function, number of medical diagnoses, nicotine dependence, psychiatric diagnoses, lower self-reported mental health, fear avoidance beliefs, and lower certainty of return to work in the next six months. The most likely mental health diagnoses are anxiety disorder and post-traumatic stress disorder. It is suggested that long-term opioids are often unknowingly being used to treat the sequelae of both physical and psychological trauma. This is based on theories of endogenous opioid system disruption. (Sullivan, 2005) (Webster, 2007) (Dersh, 2007) (Dersh, 2008) (Weisner, 2009) (Braden, 2009) (Franklin, 2009) (Edlund 2010) (Morasco, 2010) (Martin, 2011) (Sullivan, 2012)

Adverse effects: These include serious fractures, sleep apnea, hyperalgesia, immunosuppression, chronic constipation, bowel obstruction, myocardial infarction, and tooth decay due to xerostomia. Neuroendocrine problems include hypogonadism, erectile dysfunction, infertility, decreased libido, osteoporosis, and depression.

Risk of overdose: Since 2003, more overdose deaths have involved prescription opioid analgesics than heroin or cocaine combined. The CDC estimates that in 2008 there were almost 100 drug overdose deaths a day (in numbers nearing that of deaths from motor vehicle accidents). Opioid pain relievers accounted for 73.8% of deaths, with prescription drugs accounting for the largest increase in deaths. (MMWR, 2011) The risk of overdose increases when opioids are used with other drugs (such as benzodiazepines, cocaine, and/or heroin) or alcohol. Other risk factors include a history of substance abuse and/or of mental health disorder. The CDC states that the two main populations at risk for overdose are the approximate 9 million individuals who report long-term use of opioids, and the 5 million individuals who report non-medical use of this class of drugs. The CDC also reports increased risk for individuals on high doses of daily opioids (defined as > 100 mg of oral morphine equivalents a day) who seek care from multiple providers. Individuals with these characteristics were found to represent 40% of overdose deaths. Another concern is that this is a group of individuals who are likely to divert drugs. Statewide data has found that 25% to 66% of those who die of pharmaceutical overdose were taking drugs prescribed to someone else. (Mirakbari, 2003) (CDC, 2012) (CDC, 2011) (Webster, 2011). (Gomes, 2011) (Dunn, 2010) (Bohnert, 2011) (Bohnert 2012)

Concomitant use with other medications: Benzodiazepines and other sedative drugs: Benzodiazepines are commonly implicated in opioid overdose deaths and they lower the lethal opioid dose. Consideration of tapering the use of sedative hypnotics and benzodiazepines before starting opioid use if possible is strongly recommended. (Mirakbari, 2003) (Kahan, 2011) (Gomes, 2011) (Toblin 2010)

Outcomes measures: It is now suggested that rather than simply focus on pain severity, improvements in a wide range of outcomes should be evaluated, including measures of functioning, appropriate medication use, and side effects. Measures of pain assessment that allow for evaluation of the efficacy of opioids and whether their use should be maintained include the following: current pain; the least reported pain over the period since last assessment; average pain; intensity of pain after taking the opioid; how long it takes for pain relief; and how long pain relief lasts. (Nicholas, 2006) (Ballantyne, 2006)

Tolerance and addiction: Opioid tolerance develops with the repeated use of opioids and brings about the need to increase the dose and may lead to sensitization. It is now clear that analgesia may not occur with open-ended escalation of opioids. It has also become apparent that analgesia is not always sustained over time, and that pain may be improved with weaning of opioids. (Ballantyne, 2006) (Ballantyne, 2003) See Substance abuse (tolerance, dependence, addiction).

Behavior reinforcement: A major concern in the use of opioids has been that a focus on this treatment without coordination with other modalities, such as psychosocial or behavioral therapy, may simply reinforce pain-related behavior, ultimately undermining rehabilitation that has been targeted at functional restoration. (Ontario, 2000) It has been shown that pain behavior can be reinforced by the prescribing of opioids, generally on an unintentional basis by the patient. (Fordyce, 1991)

Overall treatment suggestions: Current guidelines suggest the following:

- A trial of opioids for chronic pain as a first step in treatment for appropriate conditions that have not responded to other interventions after careful screening and patient informed consent. The steps involved are outlined in the Criteria for Use of Opioids. The trial includes an initiation phase that involves selection of the opioid and initial dose.
- There is then a titration phase that includes dose adjustment. At this phase it may be determined that opioids are not achieving the desired outcomes, and they should be discontinued.
- The final stage is the maintenance phase. If pain worsens during this phase the differential to evaluate includes disease progression, increased activity, and/or new or increased pre-existing psychosocial factors that influence pain. In addition, the patient may develop hyperalgesia, tolerance, dependence or actual addiction.

(Washington, 2002) (Colorado, 2002) (Ontario, 2000) (VA/DoD, 2003) (Maddox-AAPM/APS, 1997) (Wisconsin, 2004) (Warfield, 2004) (VA/DOD, 2010) See Substance abuse (tolerance, dependence, addiction). See also Implantable pumps for narcotics. See also Opioids in the Low Back Chapter. See Criteria for Use of Opioids.

The ODG notes that Neurontin is a trade name for gabapentin and refers the reader to that section. In it, the ODG says:

Gabapentin (Neurontin®, Gabarone™, generic available) has been shown to be effective for treatment of diabetic painful neuropathy and postherpetic neuralgia and has been considered as a first-line treatment for neuropathic pain. (Backonja, 2002) (ICSI, 2007) (Knotkova, 2007) (Eisenberg, 2007) (Attal, 2006) This RCT concluded that gabapentin monotherapy appears to be efficacious for the treatment of pain and sleep interference associated with diabetic peripheral neuropathy and exhibits positive effects on mood and quality of life. (Backonja, 1998) It has been given FDA approval for treatment of post-herpetic neuralgia. The number needed to treat (NNT) for overall neuropathic pain is 4. It has a more favorable side-effect profile than Carbamazepine, with a number needed to harm of 2.5. (Wiffen2-Cochrane, 2005) (Zaremba, 2006) Gabapentin in combination

with morphine has been studied for treatment of diabetic neuropathy and postherpetic neuralgia. When used in combination the maximum tolerated dosage of both drugs was lower than when each was used as a single agent and better analgesia occurred at lower doses of each. (Gilron-NEJM, 2005) Recommendations involving combination therapy require further study.
Mechanism of action: This medication appears to be effective in reducing abnormal hypersensitivity (allodynia and hyperalgesia), to have anti-anxiety effects, and may be beneficial as a sleep aid. (Arnold, 2007)
Specific pain states:
Acute pain: There is limited evidence to show that this medication is effective for acute pain, and for *postoperative* pain, where there is fairly good evidence that the use of gabapentin and gabapentin-like compounds results in decreased opioid consumption. This beneficial effect, which may be related to an anti-anxiety effect, is accompanied by increased sedation and dizziness. (Peng, 2007) (Buvanendran, 2007) (Menigaux, 2005) (Pandey, 2005)
Spinal cord injury: Recommended as a trial for chronic neuropathic pain that is *associated with this condition. (Levendoglu, 2004)*
CRPS*:* Recommended as a trial. (Serpell, 2002)
Fibromyalgia*:* Recommended as a trial. (Arnold, 2007)
Lumbar spinal stenosis: Recommended as a trial, with statistically significant improvement found in walking distance, pain with movement, and sensory deficit found in a pilot study. (Yaksi, 2007)
Side-Effect Profile: Gabapentin has a favorable side-effect profile, few clinically significant drug-drug interactions and is generally well tolerated; however, common side effects include dizziness, somnolence, confusion, ataxia, peripheral edema, and dry mouth. (Eisenberg, 2007) (Attal, 2006) Weight gain is also an adverse effect.
Dosing Information:
Postherpetic neuralgia – Starting regimen of 300 mg once daily on Day 1, then increase to 300 mg twice daily on Day 2; then increase to 300 mg three times daily on Day 3. Dosage may be increased as needed up to a total daily dosage of 1800 mg in three divided doses. Doses above 1800 mg/day have not demonstrated an additional benefit in clinical studies. (Neurontin package insert)
Diabetic neuropathy (off-label indication) – Gabapentin dosages range from 900 mg to 3600 mg in three divided doses (Backonja, 2002) (Eisenberg, 2007). Gabapentin is 100% renally excreted.
Recommended Trial Period: One recommendation for an adequate trial with gabapentin is three to eight weeks for titration, then one to two weeks at maximum tolerated dosage. (Dworkin, 2003) The patient should be asked at each visit as to whether there has been a change in pain or function. Current consensus

based treatment algorithms for diabetic neuropathy suggest that if inadequate control of pain is found, a switch to another first-line drug is recommended. Combination therapy is only recommended if there is no change with first-line therapy, with the recommended change being at least 30%. (TCA, SNRI or AED). (Jensen, 2006) (Eisenberg, 2007)
Weaning and/or changing to another drug in this class: Gabapentin should not be abruptly discontinued, although this recommendation is made based on seizure therapy. Weaning and/or switching to another drug in this class should be done over the minimum of a week. (Neurontin package insert) *When to switch to pregabalin:* If there is evidence of inadequate response, intolerance, hypersensitivity or contraindications. There have been no head-to-head comparison trails of the two drugs.

Flexeril is the trade name for Cyclobenzaprine. The ODG contains the following entries regarding it.

> Recommended as an option, using a short course of therapy. See Medications for subacute & chronic pain for other preferred options. Cyclobenzaprine (Flexeril®) is more effective than placebo in the management of back pain; the effect is modest and comes at the price of greater adverse effects. The effect is greatest in the first 4 days of treatment, suggesting that shorter courses may be better. (Browning, 2001) Treatment should be brief. There is also a post-op use. The addition of cyclobenzaprine to other agents is not recommended. (Clinical Pharmacology, 2008) Cyclobenzaprine-treated patients with fibromyalgia were 3 times as likely to report overall improvement and to report moderate reductions in individual symptoms, particularly sleep. (Tofferi, 2004) Note: Cyclobenzaprine is closely related to the tricyclic antidepressants, e.g., amitriptyline. See Antidepressants. Cyclobenzaprine is associated with a number needed to treat of 3 at 2 weeks for symptom improvement in LBP and is associated with drowsiness and dizziness. (Kinkade, 2007) Cyclobenzaprine is a skeletal muscle relaxant and a central nervous system (CNS) depressant that is marketed as Flexeril by Ortho McNeil Pharmaceutical. See also Muscle relaxants (for pain), Cyclobenzaprine listing.

With regard to Cyclobenzaprine's use for subacute and chronic pain, the ODG says:

> Recommended as indicated below. Relief of pain with the use of medications is generally temporary, and measures of the lasting benefit from this modality should include evaluating the effect of pain relief in relationship to improvements in function and increased activity. Before prescribing any medication for pain the following should occur:

(1) determine the aim of use of the medication;
(2) determine the potential benefits and adverse effects;
(3) determine the patient's preference.

Only one medication should be given at a time, and interventions that are active and passive should remain unchanged at the time of the medication change. A trial should be given for each individual medication. Analgesic medications should show effects within 1 to 3 days, and the analgesic effect of antidepressants should occur within 1 week. A record of pain and function with the medication should be recorded. (Mens, 2005) The recent AHRQ review of comparative effectiveness and safety of analgesics for osteoarthritis concluded that each of the analgesics was associated with a unique set of benefits and risks, and no currently available analgesic was identified as offering a clear overall advantage compared with the others. (Chou, 2006) There are multiple medication choices listed separately (not all recommended (sic)). See Anticonvulsants for chronic pain; Antidepressants for chronic pain; Antidepressants for neuropathic pain; Antidepressants for non-neuropathic pain; Antiemetics (for opioid nausea); Anxiety medications in chronic pain; Anti-epilepsy drugs (AEDs); Anti-Inflammatories; Benzodiazepines; Boswellia Serrata Resin (Frankincense); Buprenorphine; Cannabinoids; Capsaicin; Cod liver oil; Compound drugs; Curcumin (Turmeric); Cyclobenzaprine (Flexeril®); Duloxetine (Cymbalta®); Gabapentin (Neurontin®); Glucosamine (and Chondroitin Sulfate); Green tea; Herbal medicines; Implantable drug-delivery systems (IDDSs); Injection with anaesthetics (sic) and/or steroids; Insomnia treatment; Intrathecal drug delivery systems, medications; Intravenous regional sympathetic blocks (for RSD, nerve blocks); Ketamine; Medical food; Methadone; Milnacipran (Ixel®); Muscle relaxants; Nonprescription medications; NSAIDs (non-steroidal anti-inflammatory drugs); NSAIDs, GI symptoms & cardiovascular risk; Opioids (with links to multiple topics on opioids); Proton pump inhibitors (PPIs); Pycnogenol (maritime pine bark); Salicylate topicals; Tapentadol; Topical analgesics; Uncaria Tomentosa (Cat's Claw); Venlafaxine (Effexor®); White willow bark; & Ziconotide (Prialt®).

Klonopin is another name for clonazepam, a benzodiazepine. The ODG pain section addresses this drug under the benzodiazepine section, saying:

> Not recommended for long-term use because long-term efficacy is unproven and there is a risk of psychological and physical dependence or frank addiction. Most guidelines limit use to 4 weeks. Benzodiazepines are a major cause of overdose, particularly as they act synergistically with other drugs such as opioids (mixed overdoses are often a cause of fatalities). Their range of action includes sedative/hypnotic, anxiolytic, anticonvulsant, and muscle relaxant. Chronic

benzodiazepines are the treatment of choice in very few conditions. Tolerance to hypnotic effects develops rapidly (3-14 day). Tolerance to anxiolytic effects occurs within months and long-term use may actually increase anxiety. A more appropriate treatment for anxiety disorder is an antidepressant. Tolerance to anticonvulsant and muscle relaxant effects occurs within weeks. Tolerance to lethal effects does not occur and a maintenance dose may approach a lethal dose as the therapeutic index increases. The best prevention for substance use disorders due to benzodiazepines is careful prescribing. (Baillargeon, 2003) (Ashton, 2005) (Dickinson, 2009) (Lader, 2009) Adults who use hypnotics, including benzodiazepines such as temazepam, have a greater than 3-fold increased risk for early death, according to results of a large matched cohort survival analysis. The risks associated with hypnotics outweigh any benefits of hypnotics, according to the authors. In 2010, hypnotics may have been associated with 320,000 to 507,000 excess deaths in the U.S. alone. A dose-response effect was evident, with a hazard ratio of 3.60 for up to 18 pills per year, 4.43 for 18-132 pills per year, and 5.32 for over 132 pills per year. (Kripke, 2012) The AGS updated Beers criteria for inappropriate medication use includes benzodiazepines. (AGS, 2012) See also Anxiety medications in chronic pain; & Insomnia treatment. Benzodiazepines that are commonly prescribed include the following: alprazolam, chlordiazepoxide, clonazepam, clorazepate, diazepam, estazolam, flurazepam, lorazepam, midazolam, oxazepam, quazepam, temazepam, & triazolam. (Clinical Pharmacology, 2010)

The pain section of the ODG contains the following entry on Lidoderm patches, a trademark name for lidocaine patches:

> Not recommended until after a trial of a first-line therapy, according to the criteria below. Lidoderm® is the brand name for a lidocaine patch produced by Endo Pharmaceuticals. Topical lidocaine may be recommended for localized neuropathic pain after there has been evidence of a trial of first-line therapy (tri-cyclic or SNRI anti-depressants or an AED such as gabapentin or Lyrica). This is not a first-line treatment and is only FDA approved for post-herpetic neuralgia. Further research is needed to recommend this treatment for chronic neuropathic pain disorders other than post-herpetic neuralgia. Formulations that do not involve a dermal-patch system are generally indicated as local anesthetics and anti-pruritics. For more information and references, see Topical analgesics. [Lidoderm ranked #2 in amount billed for WC in 2011. (Coventry, 2012)]
>
> **Criteria for use of Lidoderm patches:**
>
> (a) Recommended for a trial if there is evidence of localized pain that is consistent with a neuropathic etiology.

(b) There should be evidence of a trial of first-line neuropathy medications (tri-cyclic or SNRI anti-depressants or an AED such as gabapentin or Lyrica).
(c) This medication is not generally recommended for treatment of osteoarthritis or treatment of myofascial pain/trigger points.
(d) An attempt to determine a neuropathic component of pain should be made if the plan is to apply this medication to areas of pain that are generally secondary to non-neuropathic mechanisms (such as the knee or isolated axial low back pain). One recognized method of testing is the use of the Neuropathic Pain Scale.
(e) The area for treatment should be designated as well as number of planned patches and duration for use (number of hours per day).
(f) A Trial of patch treatment is recommended for a short-term period (no more than four weeks).
(g) It is generally recommended that no other medication changes be made during the trial period.
(h) Outcomes should be reported at the end of the trial including improvements in pain and function, and decrease in the use of other medications. If improvements cannot be determined, the medication should be discontinued.
(i) Continued outcomes should be intermittently measured and if improvement does not continue, lidocaine patches should be discontinued.

The topical analgesic section of the ODG pain chapter contains the following information:

> Recommended as an option as indicated below. Largely experimental in use with few randomized controlled trials to determine efficacy or safety. Primarily recommended for neuropathic pain when trials of antidepressants and anticonvulsants have failed. (Namaka, 2004) These agents are applied locally to painful areas with advantages that include lack of systemic side effects, absence of drug interactions, and no need to titrate. (Colombo, 2006) Many agents are compounded as monotherapy or in combination for pain control (including NSAIDs, opioids, capsaicin, local anesthetics, antidepressants, glutamate receptor antagonists, α-adrenergic receptor agonist, adenosine, cannabinoids, cholinergic receptor agonists, γ agonists, prostanoids, bradykinin, adenosine triphosphate, biogenic amines, and nerve growth factor). (Argoff, 2006) There is little to no research to support the use of many these agents. Any compounded product that contains at least one drug (or drug class) that is not recommended is not recommended. The use of these compounded agents requires knowledge of the specific analgesic effect of each agent and how it will be useful for the specific therapeutic goal required. [Note: Topical analgesics work locally underneath the skin where they are applied. These do not include transdermal analgesics that are

systemic agents entering the body through a transdermal means. For example, see Duragesic® (fentanyl transdermal system).]

Non-steroidal anti-inflammatory agents (NSAIDs): Recommended for the following indications:

Acute pain: Recommended for short-term use (one to two weeks), particularly for soft tissue injuries such as sprain/strains. According to a recent review, topical NSAIDs can provide good levels of pain relief for sprains, strains, and overuse injuries, with the advantage of limited risk of systemic adverse effects as compared to those produced by oral NSAIDs. They are considered particularly useful for individuals unable to tolerate oral administration, or for whom it is contraindicated. There appears to be little difference in analgesic efficacy between topical diclofenac, ibuprofen, ketoprofen and piroxicam, but indomethacin is less effective, and benzydamine is no better than placebo. The number needed to treat for clinical success, defined as 50% pain relief, for all topical NSAIDs combined vs. placebo was 4.5 (95% confidence interval [CI], 3.9 - 5.3) for treatment periods of 6 to 14 days. Current studies indicate 6 or 7 out of 10 patients have effective pain control with topical agents vs. 4 out of 10 with placebo. The reason for the high placebo rate is that most sprain/strain injuries improve on their own. (Massey, 2010) (Mason, 2004)

Osteoarthritis and tendinitis, in particular, that of the knee, elbow, and hand or other joints that are amenable to topical treatment: Recommended for short-term use (4-12 weeks). (See also the Knee Chapter.) (Underwood, 2008) (Mason, 2004) (Biswal, 2006) (Green, 2002) (Niethard, 2005) (Conaghan, 2008) (Altman, 2009) (Wenham, 2010) (Zhang, 2007) (NICE, 2008) (Zhang, 2010) (Altman, 2011) The American Academy of Orthopedic Surgeons recommends topical NSAIDs if there is increased GI risk with use of NSAIDs as one option for treatment. (Richmond, 2010) There are no studies evaluating topical ketoprofen for treatment of hand osteoarthritis. Topical ketoprofen gel has been compared to oral celecoxib, with WOMAC physical function scores significant for the later but not the topical treatment. (Rother, 2007)

Osteoarthritis of the hip and shoulder: There is little evidence to utilize topical NSAIDs for treatment of osteoarthritis of the hip or shoulder.

Osteoarthritis of the low back: There is no evidence to recommend a NSAID dosage form other than an oral formulation for low back pain. (Roelofs, 2008) (Haroutiunian, 2010)

Widespread musculoskeletal pain: Not recommended.

Neuropathic pain: Not recommended as there is no evidence to support use. (Haroutiunian, 2010) (Finnerup, 2005)

General information: The theory behind using a topical NSAID is to achieve a therapeutic concentration in the tissue adjacent to the application, allowing for

safe serum concentration. This would allow for less adverse GI events, eliminate first-pass metabolism and reduce risk of other GI events associated with higher systemic doses provided with oral formulations. Overall, a high concentration of drug is observed in the dermis and muscles (equivalent to that obtained orally), with less gastrointestinal effect. Plasma concentrations are 5% to 15% of those achieved systemically. (Kienzler, 2010) Topically applied NSAIDs appear to reach the synovial fluid of joints, although the mechanism for delivery remains unclear. The efficacy in clinical trials for this treatment modality has been inconsistent and most studies are small and of short duration. Topical NSAIDs have been shown in meta-analysis to be superior to placebo during the first 2 weeks of treatment for osteoarthritis, but either not afterward, or with a diminishing effect over another 2-week period. (Lin, 2004) (Bjordal, 2007) (Mason, 2004) When investigated specifically for osteoarthritis of the knee, topical NSAIDs have been shown to be superior to placebo for 4 to 12 weeks. The effect appeared to diminish over time and it was stated that further research is required to determine if results were similar for all preparations. (Biswal, 2006) These medications may be useful for chronic musculoskeletal pain, but there are no long-term studies of their effectiveness or safety. In terms of acute pain, topical NSAIDs were found to produce a 50% reduction in pain at one week, with the most significant results obtained with use of ketoprofen, while indomethacin was barely distinguished from placebo. (Mason, 2004)

Pharmacokinetics and systemic availability: Absorption and penetration through the skin depends on the active medication, formulation (i.e. gel vs. solution), carrier-medicated transport, and penetration enhancement. Each of these differences produces differences in systemic levels attained. The carrier may also contribute to toxicity. Toxicity by dose has not been established (especially for trials that allowed for more than one joint to be treated). Excessive amounts of topical NSAID may produce higher than desired levels, hindering the advantage of a topical formulation. (Haroutiunian, 2010) (Kienzler, 2010)

Compounded formulations: There is little research available in terms of bioavailability and objective clinical endpoints for these agents. (Haroutiunian, 2010)

FDA-approved agents: At this time, the only available FDA-approved topical NSAID is diclofenac.

Voltaren® Gel 1% (diclofenac): Indicated for relief of osteoarthritis pain in a joint that lends itself to topical treatment (ankle, elbow, foot, hand, knee, and wrist). It has not been evaluated for treatment of the spine, hip or shoulder. Maximum dose should not exceed 32 g per day (8 g per joint per day in the upper extremity and 16 g per joint per day in the lower extremity). The most common adverse reactions were dermatitis and pruritus. (Voltaren® package insert)

Clinical trial data suggest that diclofenac sodium gel (the first topical NSAID approved in the US) provides clinically meaningful analgesia in OA patients with a low incidence of systemic adverse events. (Altman, 2009) The labeling for topical diclofenac has been updated to warn about drug-induced hepatotoxicity. (FDA, 2009) Voltaren Gel was effective in adults regardless of age. Treatment-related application site dermatitis was more common with Voltaren Gel, but gastrointestinal AEs were infrequent. It is recommended for osteoarthritis after failure of an oral NSAID, or contraindications to oral NSAIDs, or for patients who cannot swallow solid oral dosage forms. (Baraf, 2011) (Kienzler, 2010) See also Voltaren® Gel separate listing, where it is not recommended as a first-line treatment.

Pennsaid® (diclofenac topical solution 1.5% containing 45.5% dimithyl sulfoxide): FDA-approved for osteoarthritis of the knee. A recent study on adverse effects of this agent compared to oral diclofenac found that the latter formulation had significantly higher events. Gastrointestinal AEs orally were 39% vs. 25.4% topically ($P< 0.0001$). Cardiovascular events were 3.5% orally vs. 1.5% topically ($P=0.055$). Liver function tests were increased more commonly in those taking oral agents. The most common adverse effect was application-site reaction. Dry skin is thought to result from the DSMO component. Long-term studies were recommended. (Roth, 2011) The dose is 40 drops to the knee four times a day. See also Pennsaid® (diclofenac sodium topical solution) separate listing, where it is not recommended as a first-line treatment.

Flector® Patch (diclofenac epolamine topical patch 1.3%): Indicated for acute strains, sprains, and contusions. Apply one patch twice daily to most painful area. See also Flector® patch (diclofenac epolamine) separate listing, where it is not recommended as a first-line treatment.

Non FDA-approved agents: Ketoprofen: This agent is not currently FDA approved for a topical application. It has an extremely high incidence of photocontact dermatitis and photosensitization reactions. (Diaz, 2006) (Noize, 2010) (Hindsen, 2006) (Devleeschouwer, 2008) (Matthieu, 2004) (Barbaud, 2009) Due to the high incidence of these reactions the French government removed this topical drug from the market in December 2009. This was subsequently overturned, with recommendations made to make the topical formulation available by prescription only, and by strengthening warnings as to adverse effects. (Lechat, 2010) Absorption of the drug depends on the base it is delivered in. (Gurol, 1996). Topical treatment can result in blood concentrations and systemic effect comparable to those from oral forms, and caution should be used for patients at risk, including those with renal failure. (Krummel 2000) *Clinical trials:* Numerous clinical trials are ongoing, including a phase III trial for a ketoprofen patch for treatment of soft tissue injury, acute sprain/strain, and non

articular rheumatism, tendinitis and bursitis, a phase III trial for ketoprofen 10% cream for treatment of acute soft tissue injury, and a topical ketoprofen gel for muscle soreness. Clinical trials show similar results between Diclofenac gel and a ketoprofen patch formulation. (Esparza, 2007) See also Ketoprofen, topical separate listing, where it is under study as a first-line treatment.

Piroxicam: There is no FDA-approved topical piroxicam agent. This drug also is known to produce drug-induced photosensitivity. (Drucker, 2011) (Barbaud, 2009) Numerous adverse effects are noted with systemic delivery of piroxicam including elevated hepatic enzymes in 1-10% in patients who receive the drug.

Adverse effects of topical NSAIDs in general: Topical NSAIDs have a high safety margin with fewer severe gastrointestinal adverse effects. Adverse drug events occur on average in about 12% of individuals, with 75% of these including rash and/or pruritus at the application site. A recent systematic review of use of topical NSAIDs in older adults found the withdrawal rates from topical agents to be similar to that of oral NSAIDs. Gastrointestinal complaints and headaches were reported most frequently in both topical and oral NSAID groups. Anemia, liver function tests, renal abnormalities, and severe gastrointestinal events were higher in oral NSAID users. Examination of drug-related effects, including vehicles used and total dose is needed. (Makris, 2010) The use of oral NSAIDs concomitantly with topical agents is not recommended. (Peterson, 2011) See also NSAIDs, GI symptoms and cardiovascular risk; & NSAIDs, hypertension and renal function.

Cost effectiveness: Current FDA-approved topical agents are approximately six to ten times more expensive than oral over-the-counter preparations. Savings may occur due to lack of serious adverse GI effects, and the lack of necessity of taking an ulcer-protection medication.

Lidocaine: Recommended for a trial if there is evidence of localized pain that is consistent with a neuropathic etiology. See Criteria for use below. Topical lidocaine, in the formulation of a dermal patch (Lidoderm®) has been designated for orphan status by the FDA for neuropathic pain. Lidoderm is also used off-label for diabetic neuropathy. No other commercially approved topical formulations of lidocaine (whether creams, lotions or gels) are indicated for neuropathic pain. Further research is needed to recommend this treatment for chronic neuropathic pain disorders other than post-herpetic neuralgia. Formulations that do not involve a dermal-patch system are generally indicated as local anesthetics and anti-pruritics. In February 2007 the FDA notified consumers and healthcare professionals of the potential hazards of the use of topical lidocaine. Those at particular risk were individuals that applied large amounts of this substance over large areas, left the products on for long periods of time, or used the agent with occlusive dressings. Systemic exposure was highly variable among patients. Only FDA-approved products are currently recommended.

Indications: Recommended for localized pain that is consistent with a neuropathic etiology after there has been evidence of a trial of first-line therapy (tri-cyclic or SNRI anti-depressants or an AED such as gabapentin or Lyrica). Topical lidocaine patches are generally not recommended for non-neuropathic pain (including osteoarthritis or myofascial pain/trigger points). See Criteria for use below. Most studies have utilized the Neuropathic Pain Scale (NPS) as measure of neuropathy when there are questions of whether this is the cause of pain. There is limited information as to long-term efficacy and continued information as to outcomes should be provided to allow for on-going use. (Argoff, 2004) (Galer, 2004) (Argoff, 2006) (Dworkin, 2007) (Khaliq-*Cochrane*, 2007) (Knotkova, 2007) (Lexi-Comp, 2008) (Fishbain, 2006) (Affaitati, 2009) (Burch, 2004) (Gimbel, 2005) (Dworkin, 2003) (Finnerup, 2005) (O'Connor, 2009) Discussion about specific details of these studies are given in detail with references. Trigger points & myofascial pain: Not recommended. (Affaitati, 2009) (Dalpaiz, 2004)
Osteoarthritis of the knee: Not generally recommended unless a component of neuropathy is indicated using measures such as the Neuropathic Pain Scale. All current available studies were sponsored by the manufacturer of lidocaine patches and are non-controlled, and of short-term in duration. (Burch, 2004) (Kivitz, 2008)
Axial back pain (including osteoarthritis): Not recommended unless neuropathy is suggested. Current studies as to use of Lidoderm patches for non-neuropathic low back pain are non-controlled, may or may not evaluate for the presence of neuropathic quality, have included multiple stages of pain (from acute to chronic), have included multiple diagnoses, show limited results in pain reduction, and are generally sponsored by the manufacturer. Acute groups have had better results than chronic pain patients, which may be attributed to natural recovery. (Gimbel, 2005) (Galer, 2004) (Argoff, 2004)

Criteria for use of Lidoderm patches:

(a) Recommended for a trial if there is evidence of localized pain that is consistent with a neuropathic etiology.
(b) There should be evidence of a trial of first-line neuropathy medications (tri-cyclic or SNRI anti-depressants or an AED such as gabapentin or Lyrica).
(c) This medication is not generally recommended for treatment of osteoarthritis or treatment of myofascial pain/trigger points.
(d) An attempt to determine a neuropathic component of pain should be made if the plan is to apply this medication to areas of pain that are generally secondary to non-neuropathic mechanisms (such as the knee or isolated axial low back pain). One recognized method of testing is the use of the Neuropathic Pain Scale.

(e) The area for treatment should be designated as well as number of planned patches and duration for use (number of hours per day).
(f) A Trial of patch treatment is recommended for a short-term period (no more than four weeks).
(g) It is generally recommended that no other medication changes be made during the trial period.
(h) Outcomes should be reported at the end of the trial including improvements in pain and function, and decrease in the use of other medications. If improvements cannot be determined, the medication should be discontinued.
(i) Continued outcomes should be intermittently measured and if improvement does not continue, lidocaine patches should be discontinued.

Capsaicin: Recommended only as an option in patients who have not responded or are intolerant to other treatments. *Formulations:* Capsaicin is generally available as a 0.025% formulation (as a treatment for osteoarthritis) and a 0.075% formulation (primarily studied for post-herpetic neuralgia, diabetic neuropathy and post-mastectomy pain). There have been no studies of a 0.0375% formulation of capsaicin and there is no current indication that this increase over a 0.025% formulation would provide any further efficacy. *Indications:* There are positive randomized studies with capsaicin cream in patients with osteoarthritis, fibromyalgia, and chronic non-specific back pain, but it should be considered experimental in very high doses. Although topical capsaicin has moderate to poor efficacy, it may be particularly useful (alone or in conjunction with other modalities) in patients whose pain has not been controlled successfully with conventional therapy. The number needed to treat in musculoskeletal conditions was 8.1. The number needed to treat for neuropathic conditions was 5.7. (Robbins, 2000) (Keitel, 2001) (Mason-*BMJ*, 2004) Neither salicylates nor capsaicin have shown significant efficacy in the treatment of OA. (Altman, 2009) See also Capsaicin.

Baclofen: Not recommended. There is currently one Phase III study of Baclofen-Amitriptyline-Ketamine gel in cancer patients for treatment of chemotherapy-induced peripheral neuropathy. There is no peer-reviewed literature to support the use of topical baclofen.

Other muscle relaxants: There is no evidence for use of any other muscle relaxant as a topical product.

Gabapentin: Not recommended. There is no peer-reviewed literature to support use.

Other antiepilepsy drugs: There is no evidence for use of any other antiepilepsy drug as a topical product.

Ketamine: Under study: Only recommended for treatment of neuropathic pain in refractory cases in which all primary and secondary treatment has been exhausted.

> Topical ketamine has only been studied for use in non-controlled studies for CRPS I and post-herpetic neuralgia and both have shown encouraging results. The exact mechanism of action remains undetermined. (Gammaitoni, 2000) (Lynch, 2005)
> See also Salicylate topicals; & Glucosamine (and Chondroitin Sulfate).

Claimant has been treating with Dr. J since 2004. He has been on essentially the same medication regimen for the last eight plus years. He has been taking Avinza for only six to eight months, but prior to that he was given prescriptions for Opana and before that, Oxycontin. Dr. J discontinued the Opama and switched Claimant to Avinza because the Opama was not providing the required analgesic effect. Claimant testified that he has taken Klonopin for ten to twelve years, Neurontin for ten to twelve years, Flexeril for eight years, and has been given prescriptions for the Lidoderm patches for eight to nine years. Dr. J testified that he is a pain management doctor and also deals with cases of addiction. He said that the treatment guidelines in Florida deal primarily with invasive procedures and there are no real guidelines for medications, although there are general guidelines for opiates. He testified that studies show that long-term use of opiates without compounded drugs is safe and that the purpose of a treatment regimen such as Claimant's is to maximize function while minimizing pain. In keeping with Florida law, Claimant undergoes a drug screen four times a year. Dr. J testified that he has seen no indication of abuse or diversion in Claimant's case. He testified that the Avinza is used to treat Claimant's pain, the Neurontin and Flexeril are used for neuropathic pain, Klonopin is used to help Claimant sleep, and the Lidoderm patches are for localized pain relief. The ODG indicates that use of Neurontin and Flexeril may help in lowering the effective dosage of Avinza and that the Lidoderm patches are effective for localized treatment of neuropathic pain.

The IRO physician reviewer found that the use of the drugs at issue herein was consistent with the ODG. Carrier asserts that the physician reviewer's opinion should be afforded no weight because he failed to explain why he believed that the use of the drugs was consistent with the ODG. Dr. J, without specific reference to the ODG, has explained the use of each of the medications and that use is consistent with the recommendations of the ODG. While most of the drugs are not ordinarily recommended for long term use, there is support in the ODG for their use in that role when first line treatments have failed. Claimant has undergone surgery, injection therapy, and physical therapy, but still suffers from chronic pain. Claimant testified that his functional ability is enhanced by the use of the drugs prescribed by Dr. J. Carrier's expert noted that there was a surveillance video that captured Claimant's mounting and riding a motorcycle. Claimant testified that without the medications prescribed by Dr. J he would be unable to ride the motorcycle or do many of the daily activities that he is currently capable of performing.

In determining the weight to be given to expert testimony, a trier of fact must first determine if the expert is qualified to offer it. The trier of fact must then determine whether the opinion is relevant to the issues at bar and whether it is based upon a solid foundation. An expert's bald

assurance of validity is not enough. *See* Black vs. Food Lion, Inc., 171 F.3rd 308 (5th Cir. 1999); E.I. Du Pont De Nemours and Company, Inc. v. Robinson, 923 S.W.2d 549 (Tex. 1995). Evidence is considered in terms of (1) general acceptance of the theory and technique by the relevant scientific community; (2) the expert's qualifications; (3) the existence of literature supporting or rejecting the theory; (4) the technique's potential rate of error; (5) the availability of other experts to test and evaluate the technique; and (7) the experience and skill of the person who applied the technique on the occasion in question. Kelly v. State, 792 S.W.2d 579 (Tex.App.-Fort Worth 1990). A medical doctor is not automatically qualified as an expert on every medical question and an unsupported opinion has little, if any, weight. Black v. Food Lion, Inc., 171 F.3rd 308 (5th Cir. 1999).

Dr. J testified that the combination of drugs prescribed to Claimant are monitored and have proven effective to afford pain relief and increased function. The use of the drugs at issue for chronic pain is consistent with the ODG. Under the facts presented here, Carrier has failed to prove that the IRO decision is contrary to the preponderance of the evidence based medical evidence. Even though all the evidence presented was not discussed, it was considered. The Findings of Fact and Conclusions of Law are based on all of the evidence presented.

FINDINGS OF FACT

1. The parties stipulated to the following facts:

   A. Venue is proper in the (City) Field Office of the Texas Department of Insurance, Division of Workers' Compensation.

   B. On (Date of Injury), Claimant was the employee of (Employer), Employer, and sustained a compensable injury.

   C. On (Date of Injury), Employer provided workers' compensable insurance with Travelers Indemnity Company of Connecticut, Carrier.

   D. Carrier denied approval for the medications at issue in this matter: Avinza 120mg, Neurontin 800mg, Flexeril 10mg, Klonopin 1mg, and Lidoderm patches #90.

   E. (Independent Review Organization) was appointed by the Texas Department of Insurance to act as the Independent Review Organization (IRO) in this matter.

   F. (Independent Review Organization) determined that Avinza, Neurontin, Flexeril, Klonopin, and Lidoderm patches were reasonably necessary health care for the compensable injury of (Date of Injury).

2. Carrier delivered to Claimant a single document stating the true corporate name of Carrier, and the name and street address of Carrier's registered agent, which document was admitted into evidence as Hearing Officer's Exhibit Number 2.

3. The prescriptions for Avinza 120mg, Neurontin 800 mg, Flexeril 10mg, Klonopin 1 mg, and Lidoderm patches #90 are reasonably required health care for the compensable injury of (Date of Injury).

CONCLUSIONS OF LAW

1. The Texas Department of Insurance, Division of Workers' Compensation, has jurisdiction to hear this case.

2. Venue is proper in the (City) Field Office.

3. The preponderance of the evidence is not contrary to the decision of IRO that Avinza, Neurontin, Flexeril, Klonopin, and Lidoderm patches are reasonably required health care for the compensable injury of (Date of Injury).

DECISION

Claimant is entitled to Avinza, Neurontin, Flexeril, Klonopin, and Lidoderm patches for the compensable injury of (Date of Injury)

ORDER

Carrier is ordered to pay benefits in accordance with this decision, the Texas Workers' Compensation Act, and the Commissioner's Rules. Accrued but unpaid income benefits, if any, shall be paid in a lump sum together with interest as provided by law.

The true corporate name of the insurance carrier is **TRAVELERS INDEMNITY COMPANY OF CONNECTICUT** and the name and address of its registered agent for service of process is

CORPORATION SERVICE COMPANY
D/B/A CSC-LAWYERS INCORPORATING SERVICE CO.
211 EAST 7TH STREET
STE. 620
AUSTIN, TX 78701-3218

Signed this 24th day of January, 2013.

KENNETH A. HUCHTON
Hearing Officer