The Use of Anesthetics, Steroids, Non-Steroidals, and Central-Acting Analgesics in the Management of Ocular Pain
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INTRODUCTION

It is not uncommon for the optometrist to have patients presenting to the office with complaint of eye pain. Many ocular conditions can present with varying amounts of pain (Table 1). Pain is highly subjective, and is dependent on the type and severity of ocular insult, the chronicity of the pain symptom, and the experiential and psychological
background of the patient. As pain is subjective, the amount of pain for the same ocular insult can vary widely from patient to patient. For example, some patients may not express any pain for a corneal abrasion, while others may relay excruciating pain for a similar injury. Often a scale of 0 to 10 is asked by the optometrist to help them understand the amount of pain their patient is experiencing (Figure 1). Elucidating if the pain is “burning”, “stinging”, “sharp”, “dull”, “itching”, “throbbing”, may be of diagnostic use. Likewise, localizing if the pain is on the surface (i.e. the patient notices it with each blink) versus deeper (i.e. the patient feels a retrobulbar pain on eye movements) is also important diagnostically.

![Universal Pain Assessment Tool](http://www.anes.ucla.edu/pain/FacesScale.jpg)

**Figure 1. Universal Pain Assessment Tool, incorporating 0 to 10 Scale, Verbal Descriptor Scale, Wong-Baker Facial Grimace Scale, and Activity Tolerance Scale.**

The clinician may be hesitant to treat the associated pain, due to misconceptions or fear in using narcotic agents, relying on the primary treatment alone to also reduce the pain, and/or not taking the patient’s symptoms of pain seriously. Pain can affect the physiological state of cardio-respiratory function (often increasing heart rate and breathing, and causing peripheral vasoconstriction – all potentially leading to systemic hypertension), gastrointestinal function (poor food absorption and gastric ulcer formation), and psychological function (increased anxiety, insomnia, depression, and emotional distress). Patients may be less willing to adhere to a treatment plan if pain persists. It is imperative that optometrists judiciously treat for pain along with treatment of the causative eye disease.

This course serves to familiarize the reader to various analgesics used in treating ocular pain, and provide a basic background for the indications and contraindications of each. It also serves to provide fundamentals of pain pathophysiology and management strategies for various ocular conditions that can have associated pain.
Table 1. Various Ocular Conditions that can Present with Accompanying Pain

- Acute Angle Closure Glaucoma
- Abrasion/Laceration/Foreign Body – Cornea, Conjunctiva, Globe, Adnexa
- Adenoviral Keratitis
- Blepharitis
- Chemical Burn – Cornea, Conjunctiva, Globe, Adnexa
- Conjunctivitis
- Corneal Edema/Bullous Keratopathy
- Corneal Ulcer/Infiltrate
- Dry Eye Syndrome
- Herpetic Keratitis
- Hordeolae
- Inflamed Pterygium
- Optic Neuritis
- Orbital Orbital Fractures/Pseudotumor/Tumor
- Pingueculitis
- Post-Herpetic Neuralgia
- Post-Surgical – LASIK, PRK, Cataract, Keratoplasty
- Preseptal/Orbital Cellulitis
- Recurrent Corneal Erosion
- Referred Orbital/Adnexa Pain – Sinusitis, Dental Disease
- Scleritis/Episceritis
- Traumatic Ecchymosis
- Uveitis

Figure 2 shows the physiological pathway of pain. Specific nerve endings in peripheral tissue (usually in the subepithelial layer), called nociceptors, are activated by mechanical, thermal, or chemical stimulation – these stimuli can be external (outside the body) or internal (within the body). Figure 3 shows the arachidonic acid pathway, which produces the inflammatory mediators: prostaglandins, leukotrienes, and thromboxanes. The prostaglandins (primarily PGE₂) and leukotrienes sensitize the nociceptors to other inflammatory mediators – bradykinin (cytokine) and histamine, among others – which interact with substance P (preparation – a tachykinin neuropeptide released from the peripheral neurons) to stimulate the nerve endings. Substance P also stimulates mast cells to release more histamine as well as more prostaglandins and bradykinins, further stimulating the nociceptors. Substance P also contributes to swelling and redness in the area of pain.
Figure 2. Physiological pathway of pain. Injury stimulates peripheral nociceptors, which synapse in the dorsal root ganglion to send signals of pain to the brain. Inflammatory mediators are released which again stimulate the nociceptors, which in turn release Substance P to create mast cell degranulation and peripheral vasodilation (From http://instruct1.cit.cornell.edu/courses/psych396/student2006/the_biology_of_pain_mac_version/receptor2.jpg).

Figure 3. Arachidonic acid pathway responsible for the inflammatory mediators associated with pain (From: Pharmacotherapy 2003, Pharmacotherapy Publications).
Sudden pain stimulus usually produces a fast “reflex arc” response, which may not give a feeling of pain. The sensory neuron synapses with the motor neuron in the dorsal horn of the spinal cord, resulting in a rapid muscular response. An example of this would be removing one’s hand from a hot iron. Soon after this response, a “physiological” response is perceived. This occurs via the pain signal traveling up the spinal-thalamic tract to the thalamus. As the thalamus also controls cardiac and pulmonary function, increased heart rate and breathing may occur. Finally, higher-order synapses in the cerebral cortex create the “pain sensation” perception (Figure 4). People vary widely in terms of their reaction to and severity of each of these responses.

Figure 4. Thalamic and higher cortical pathway of pain perception. Afferent fibers, transmitting the pain signal, synapse in the dorsal root ganglion, transferring the signal up the spinothalamic tract to synapse in the thalamus. A final synapse with primary sensory cortex fibers occurs in the thalamus (From http://www.pharmpedia.com/).

The transmission of ocular pain is primarily via the trigeminal (V) nerve, which has three main branches – maxillary, mandibular, and ophthalmic (Figure 5). Of these branches, the ophthalmic branch is the main transmitter of ocular pain. The corneal nerves are linked to the ophthalmic branch of the trigeminal nerve, and any insult on the corneal nerves is quickly perceived as pain. Likewise, the nerves innervating the iris are also linked to the ophthalmic branch. Other nerves which can transmit pain from the ocular area are the facial nerve (VII) - particularly related to eyelid and adnexal disease - and the optic nerve (II) – related primarily to neuritis or compressive insults.
Clinical evaluation of these three nerves is fairly straightforward. The practitioner can perform a cotton wisp test (or use a corneal anesthesiometer) to elicit the blink reflex, indicating sensory transmission of mechanical stimulus via the ophthalmic branch of nerve V (Figure 6). Nerve VII sensory response can be determined by lightly pressing a dull object (i.e. eraser head) or a sharp object (i.e. needle) randomly on the patient’s skin of the eyelids, adnexa, forehead and cheeks, with the patient’s eyes closed during the testing. The patient then relays to the doctor if they feel a dull or sharp sensation after each press (Figure 7). Nerve II sensory response may be determined through the patient relaying a sense of pain behind the eye or eyes upon extraocular motilities.
Healthcare practitioners have an armamentarium of medications (termed analgesic drugs) to control pain. These medications affect various steps in the pain response, from initial stimulation to final cortical perception. The three main types of analgesic drugs are:

1) Peripheral-acting agents
   a. Blocks peripheral nociceptor stimulation, and the inflammatory pathway that contributes to nociceptor stimulation.
   b. Non-steroidal anti-inflammatory drugs (NSAIDs), acetaminophen, aspirin

2) Anesthetics
   a. Blocks action potential signal from nociceptor to brain or spinal cord
   b. Lidocaine, proparacaine, tetracaine, benoxinate

3) Central-acting agents
   a. Interrupts pain signals and emotional responses to pain at the brainstem to cerebral cortex level
   b. Opioid (narcotic) analgesics
Adjuvant agents, although not directly providing analgesic effects themselves, may enhance the pain-killing effect of analgesic agents. Examples of adjuvant agents are:

- Tricyclic antidepressants (amitriptyline)
- Anticonvulsants (carbamazepine, clonazepam, phenytoin, gabapentin)
- Stimulants (caffeine, hydroxyzine)

**Ocular Pain/Inflammation Treatment**

Ocular conditions such as corneal abrasions, burns or erosions, ultraviolet keratopathy, uveitis, post-surgery symptoms, lid lacerations or other injuries, and zoster infections, can cause variable amounts of pain sensation to the patient. The optometrist may need to prescribe oral analgesics in combination with other treatment. Analgesics can be divided into peripherally acting and centrally acting mechanisms of action. In general, peripherally acting analgesics work to inhibit peripheral pain receptors (through metabolic pathways such as the cyclo-oxygenase route) while centrally acting affect the central nervous system’s opioid receptors involved in pain perception. The goal is to prescribe the lowest amount of medication to adequately control the pain.

**PERIPHERAL ACTING AGENTS**

Peripheral-acting analgesics are relatively safe medications in low to moderate doses, that have what is called a “ceiling effect” – a dosage beyond which no further pain reduction occurs. As a result, they do not result in the tolerance or psychological dependence that central-acting agents cause, and as such are non-addictive. This also allows more long-term duration of pain treatment, if warranted.

**Salicylates**
The salicylate acetylsalicylic acid (ASA or aspirin) is the oldest peripheral-acting analgesic in pain management. It works on inhibiting production of prostaglandin E$_2$ by irreversibly inactivating cyclo-oxygenase, thus reducing the pain sensation. Typical adult dosage is 325-650mg every 4 hours. In higher doses (3-4g per day), it also works to reduce inflammation. However, its blood-thinning properties delegate it more to stroke prevention treatment. It also has a higher risk of causing gastro-intestinal bleeds. Taking aspirin during meals and with plenty of water is recommended to reduce gastrointestinal upset. Aspirin can also cause Reye’s syndrome, a life-threatening brain disease and lipid abnormality, if used on children and teenagers with chicken pox or flu symptoms, so it should be avoided in these cases. Diflusinal (trade name: Dolobid) is a derivative of ASA, and should be regarded similarly.

Because of the irreversible inhibition of cyclo-oxygenase by aspirin, this results in decreased blood platelet aggregation and risk of extended hemorrhaging. Patients who present with subconjunctival hemorrhages should be asked if they are taking aspirin. Consultation with the patient’s primary care provider to determine the risk-benefit of taking aspirin may be needed if prolonged and/or recurrent ocular bleeding occurs.
Acetaminophen (trade name: Tylenol) is a peripheral-acting over-the-counter pain medication that is very popular, and comes in 325 and 500mg tablets, as well as 160 or 325mg/5ml syrup. Generic and pediatric formulations are also available. Typical dosage is 325-1000mg PO q4-6 hours. It is also combined with many central-acting painkillers, some of which will be discussed below.
Acetaminophen has been regarded as the safest pain medication in terms of systemic side effects. In fact, in short-term use at therapeutic dose, it is safe to use during all stages of pregnancy and nursing, and thus has a pregnancy category B rating. Although it is excreted in the breast milk, there are no noted adverse effects on nursing infants. It is the indicated analgesic for mild to moderate pain during pregnancy or lactation. Unlike aspirin, acetaminophen does not inhibit platelet function and as such does not cause the associated bleeding effects. In addition, Reyes syndrome is not associated with its use, and thus acetaminophen can be used safely in children. There is also an antipyretic effect when taking acetaminophen. One important concern, however, is the potential risk of liver damage and/or kidney damage with acetaminophen. This risk is more prevalent with higher dosages and chronic use of acetaminophen. Acetaminophen comes in a myriad of formulations, including tablets, capsules, chewable tablets, suppositories, elixirs, and solutions.

**NSAIDs**

NSAIDs (Non-Steroidal Anti-Inflammatory Drugs) work on blocking the cyclooxygenase pathway of inflammation that leads to pain. There are several classes of NSAIDs, based on molecular structure and chemical characteristics, with the most common class being the propionic acids (Table 2).

<table>
<thead>
<tr>
<th>NSAID Class</th>
<th>Generic Name</th>
<th>Trade Name</th>
<th>Formulation</th>
<th>Typical Adult Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Propionic acids</td>
<td>ibuprofen</td>
<td>Advil, Motrin, Naproxyn</td>
<td>tablet, suspension</td>
<td>200-400mg q4-6hr</td>
</tr>
<tr>
<td></td>
<td>naproxen</td>
<td>Naprosyn</td>
<td>tablet, susp.</td>
<td>250mg q6-8hr</td>
</tr>
<tr>
<td></td>
<td>naproxen sodium</td>
<td></td>
<td>capsule</td>
<td>550mg q12hr</td>
</tr>
<tr>
<td></td>
<td>ketoprofen</td>
<td>Orudis, Oruvail</td>
<td></td>
<td>25-75mg q6-8hr</td>
</tr>
<tr>
<td></td>
<td>fenoprofen</td>
<td>Nalfon, Daypro</td>
<td>capsule, tab. capsules</td>
<td>200mg q4-6hr</td>
</tr>
<tr>
<td></td>
<td>oxaprozin</td>
<td></td>
<td></td>
<td>50mg daily</td>
</tr>
<tr>
<td>Indoleacetic acids</td>
<td>indomethacin</td>
<td>Indocin</td>
<td>cap., susp. tablet</td>
<td>25mg q8-12hr</td>
</tr>
<tr>
<td></td>
<td>ketorolac</td>
<td>Toradol</td>
<td>capsule</td>
<td>10mg q4-6hr</td>
</tr>
<tr>
<td></td>
<td>etodolac</td>
<td>Lodine</td>
<td></td>
<td>200-400mg q6-8hr</td>
</tr>
<tr>
<td>Phenylacetic acids</td>
<td>diclofenac</td>
<td>Cataflam</td>
<td>tablet</td>
<td>50mg TID</td>
</tr>
<tr>
<td></td>
<td>diclofenac Na</td>
<td>Voltaren</td>
<td>tablet</td>
<td>50mg TID</td>
</tr>
<tr>
<td></td>
<td>diflusinal</td>
<td>Dolobid</td>
<td></td>
<td>250-500mg q8-12h</td>
</tr>
<tr>
<td>Acetic acids</td>
<td>nabumetone</td>
<td>Relafen</td>
<td>tablet</td>
<td>500-750mg BID</td>
</tr>
<tr>
<td></td>
<td>sulindac</td>
<td>Clinoril</td>
<td>tablet</td>
<td>150-200mg BID</td>
</tr>
<tr>
<td></td>
<td>tolmetin</td>
<td>Tolectin</td>
<td>tablet</td>
<td>400mg TID-QID</td>
</tr>
<tr>
<td>Anthranilic acids</td>
<td>mefenamic acid meclofenemate</td>
<td>Ponstel Meclomen</td>
<td>capsule capsule</td>
<td>250mg q6hr 50-100mg q4-6hr</td>
</tr>
<tr>
<td>--------------------------</td>
<td>--------------------------------</td>
<td>------------------</td>
<td>----------------</td>
<td>--------------------------</td>
</tr>
<tr>
<td>COX-2 inhibitors</td>
<td>rofecoxib* celecoxib</td>
<td>Vioxx Celebrex</td>
<td>tablet, susp. capsule</td>
<td>50mg daily 100-200mg QD</td>
</tr>
<tr>
<td>Oxicam derivative</td>
<td>piroxicam</td>
<td>Feldene</td>
<td>capsule</td>
<td>10-20mg QOD</td>
</tr>
</tbody>
</table>

*rofecoxib (Vioxx) has been removed from market in 2004

NSAIDs are indicated for mild to moderate pain. While the primary action is on inhibiting the peripheral cyclo-oxygenase pathway to pain, there is some diminished central nervous system response to pain, of which the mechanism is not fully understood. However, no drug tolerance or addiction occurs from the use of NSAIDs. While most of these drugs are metabolized in the liver and excreted in the urine, the acetic acid class does have some biliary excretion.

Figure 12. Popular over-the-counter NSAIDs Advil (ibuprofen) and Aleve (naproxen sodium)

Propionic acid derivatives have been found to have greater analgesic effects than aspirin, with less side effects. Ibuprofen, as an example, has uncommon side effects including kidney and liver toxicity, but is still relatively safe. Ibuprofen (trade names: Advil, Motrin, and Nuprin) is popular, and comes in 100 to 800mg tablets, typically prescribed 300-800mg PO q4-6 hours. It is also available in generic forms.

The indoleacetic acid ketorolac was found to have similar analgesic effect orally to an equivalent dose of intramuscular morphine – however, ketorolac is only indicated for short-term use, due to its gastric inflammation and gastric ulcer risks.

The risk to the gastrointestinal tract is an inherent and variable side effect of many NSAIDs. Painkillers such as ibuprofen affect the cyclo-oxygenase-1 (COX-1) and cyclo-oxygenase-2 (COX-2) inflammatory pathways in the body. COX-1 and COX-2 are two main sub-sets of cyclo-oxygenase. COX-1 catalyzes (helps) the “normal” prostaglandins that benefit renal function, maintenance of the gastric mucosa, and blood coagulation. COX-2 catalyzes the “pathological” prostaglandins that cause inflammation and pain.
COX-2 inhibiting drugs therefore inhibit the “pathological” prostaglandins while leaving the “normal” prostaglandins alone (Figure 13). This was thought to offer a new level of safety above the non-selective peripheral-acting analgesics such as ibuprofen.

Figure 13. Most NSAIDs block both types of cyclo-oxygenase (COX), but COX-2 inhibitors selectively block COX-2. This COX-2 selectivity retains the “good” prostaglandins and thromboxanes involved in normal gastric, renal, and platelet function (From http://www.australianprescriber.com/).

The drugs Vioxx (rofecoxib) and Celebrex (celecoxib) were developed specifically to inhibit only the COX-2, thus reducing the risk of gastric, hematological, and renal side effects (Figure 14).
Celecoxib (trade name: Celebrex) and rofecoxib (trade name: Vioxx) are examples of COX-2 inhibiting painkillers. Celebrex comes in 100, 200 and 400mg tablets, and is typically prescribed 100-200mg PO BID. Vioxx comes in 1.5, 25, and 50mg tablets as well as 12.5 and 25mg/5ml suspension. It is typically prescribed 12.5-25mg PO qd.

Side effects of the COX-2 analgesics include hypertension and heart and blood abnormalities, as well as liver and renal problems. These medications should not be used in conjunction with any other peripheral-acting painkillers over a long period of time. Vioxx was removed from the market in 2004, due to potentially fatal cardiac complications such as heart attack and strokes. Celebrex has carried a warning regarding these potential risks.

**Topical Ophthalmic NSAIDs**

Topical ophthalmic NSAIDS are available for use in various ocular disease conditions, from allergic conjunctivitis to localized inflammatory surface reactions (Table 3). They have analgesic, antipyretic, and anti-inflammatory activity.

<table>
<thead>
<tr>
<th>Trade Name</th>
<th>Generic Name</th>
<th>Concentration</th>
<th>Indication</th>
</tr>
</thead>
<tbody>
<tr>
<td>Voltaren</td>
<td>Diclofenac sodium</td>
<td>0.1%</td>
<td>Post-op cataract extraction inflammation, corneal surgery pain/photophobia</td>
</tr>
<tr>
<td>Acular</td>
<td>Ketorolac tromethamine</td>
<td>0.4%</td>
<td>Post-op cataract extraction inflammation, corneal surgery pain/burning/stinging, seasonal allergic conjunctivitis</td>
</tr>
<tr>
<td>Acular LS</td>
<td></td>
<td>0.45%</td>
<td></td>
</tr>
<tr>
<td>Acuvail PF</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
These topical NSAIDS work by inhibiting cyclo-oxygenase from producing prostaglandin, prostacyclin, and thromboxane A$_2$ inflammatory mediators. This results in reduced inflammation and accompanying pain. Reduction of post-cataract surgery macular edema using topical NSAIDs has been noted clinically; further scientific corroboration of this effect is being investigated. Voltaren is traditionally considered the most potent of the topical NSAIDS, although efficacy of each in treating ocular surface inflammation is similar. Nevanac is an NSAID pro-drug which converts to amfenac, an NSAID, upon penetration of the cornea. Suprofen is contraindicated in herpes dendritic epithelial keratitis. The usual dosage of most of these topical ophthalmic NSAIDS is one drop four times per day in the affected eye; Bromday (bromfenac), is once-daily dosage.

**Figure 15. Topical Ophthalmic NSAIDs (L-R): Voltaren (diclofenac sodium), Acular LS (ketorolac tromethamine), Xibrom (bromfenac), and Nevanac (nepafenac).**

**Corticosteroids**

While not considered a true analgesic class, corticosteroids nonetheless play an important role in the reduction of inflammation and pain associated with various ocular diseases.

Several ocular inflammatory conditions, including uveitis, scleritis, areritic anterior ischemic optic neuropathy, orbital pseudotumor, and optic neuritis, may require the need for an oral corticosteroid, commonly termed steroid. Steroids act higher up on inhibiting the cyclo-oxygenase pathway than the non-steroidal anti-inflammatory medications, and as such tend to have a greater effect in reducing inflammations, as well as reducing pain in many cases.
Figure 16. The inflammatory pathway and its inhibition by steroids and non-steroidal anti-inflammatory drugs.

Prednisone (generic or trade name Prelone) comes in 5mg tablets or 15mg/5ml elixir. Trade names Orapred and Pediapred are 15mg/5ml elixirs. Typical daily dosage is 40-60mg PO qd initially, tapering over a one to two week period. Dosages up to 60mg can be taken at one time, and it is recommended that prednisone be taken with meals to reduce gastric distress. In acute situations where only a short dose is needed, 40-60mg PO qd may be used for two days, then discontinued without taper.

Figure 17. Oral steroids. (L) Prednisolone (R) Methylprednisone Dosepak package

Methylprednisone should be mentioned here as an alternative steroid in the treatment of ocular conditions. It is available in 2, 4, 8, 16, 24, and 32mg tablets generically and as the Medrol “dosepak” blister packages. Dosepaks have six 4mg tablets (total 24mg) that the patient takes on day one, with the number of tablets taken reduced by one tablet each day over six days in a taper schedule. Most practitioners prefer using prednisone to the
lower dosage methylprednisone. The relative anti-inflammatory potencies of various oral steroids are shown in Table 4 below:


<table>
<thead>
<tr>
<th>CORTICOSTEROID</th>
<th>RELATIVE ANTI-INFLAMMATORY EQUIVALENT DOSE (mg)</th>
<th>RELATIVE POTENCY</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cortisone</td>
<td>25</td>
<td>0.8</td>
</tr>
<tr>
<td>Hydrocortisone</td>
<td>20</td>
<td>1</td>
</tr>
<tr>
<td>Prednisone</td>
<td>5</td>
<td>4</td>
</tr>
<tr>
<td>Prednisolone</td>
<td>5</td>
<td>4</td>
</tr>
<tr>
<td>Triamcinolone</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>Methylprednisone</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>Dexamethasone</td>
<td>0.75</td>
<td>25</td>
</tr>
<tr>
<td>Betamethasone</td>
<td>0.75</td>
<td>25</td>
</tr>
</tbody>
</table>

Oral steroids can provide significant analgesic effect in combination with their anti-inflammatory component. However, they should not be used in place of pain medication for conditions that require simply analgesia. Steroids have numerous adverse side effects that can potentially be life threatening, including adrenal insufficiency, “Cushingoid” syndrome, steroid-induced diabetes, menstrual changes, reduced immunity, delayed wound healing, and mood swings. The practitioner should consider prescribing oral steroids only if there is no safer alternative treatment, and consultation with the patient’s primary care provider is recommended before initiating a patient on oral steroids.

Steroids are also available in topical and injectable forms, and are frequently used in these formulations in the management of various ocular diseases such as uveitis and chalazion treatment. Table 5 shows the current topical ophthalmic steroids available.

Table 5. Various commercially-available topical ocular steroids

<table>
<thead>
<tr>
<th>TOPICAL OCULAR STEROID</th>
<th>TRADE NAME</th>
<th>CONCENTRATION/FORMULATION</th>
</tr>
</thead>
</table>

...
<table>
<thead>
<tr>
<th>Steroid</th>
<th>Brand/Manufacturer</th>
<th>Strength/Concentration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prednisolone acetate</td>
<td>Pred Forte (Allergan)</td>
<td>1.0% suspension</td>
</tr>
<tr>
<td></td>
<td>Econopred Plus (Alcon)</td>
<td>1.0% suspension</td>
</tr>
<tr>
<td></td>
<td>AK-Tate (Akorn)</td>
<td>1.0% suspension</td>
</tr>
<tr>
<td></td>
<td>Pred Mild (Allergan)</td>
<td>0.125% suspension</td>
</tr>
<tr>
<td></td>
<td>Econopred (Alcon)</td>
<td>0.125% suspension</td>
</tr>
<tr>
<td>Dufluprednate</td>
<td>Durezol (Alcon)</td>
<td>0.05% emulsion</td>
</tr>
<tr>
<td>Prednisolone sodium phosphate</td>
<td>Inflamase Forte (CIBA)</td>
<td>1.0% solution</td>
</tr>
<tr>
<td></td>
<td>Metreton (Schering)</td>
<td>0.5% solution</td>
</tr>
<tr>
<td></td>
<td>Inflamase Mild (CIBA)</td>
<td>0.125% solution</td>
</tr>
<tr>
<td></td>
<td>AK-Pred (Akorn)</td>
<td>0.125% solution</td>
</tr>
<tr>
<td>Dexamethasone alcohol</td>
<td>Maxidex (Alcon), others</td>
<td>0.1% susp, ointment</td>
</tr>
<tr>
<td></td>
<td>Tobradex (Alcon)</td>
<td>0.1% susp, ointment</td>
</tr>
<tr>
<td>Fluorometholone acetate</td>
<td>Flarex (Alcon)</td>
<td>0.1% suspension</td>
</tr>
<tr>
<td></td>
<td>Eflone</td>
<td>0.1% suspension</td>
</tr>
<tr>
<td>Fluorometholone alcohol</td>
<td>FML (Allergan)</td>
<td>0.1% suspension</td>
</tr>
<tr>
<td></td>
<td>FluorOp</td>
<td>0.1% ointment</td>
</tr>
<tr>
<td></td>
<td>FML-Mild (Allergan)</td>
<td>0.25% suspension</td>
</tr>
<tr>
<td>Rimexolone</td>
<td>Vexol (Alcon)</td>
<td>1% suspension</td>
</tr>
<tr>
<td>Medrysone alcohol</td>
<td>HMS (Allergan)</td>
<td>1.0% suspension</td>
</tr>
<tr>
<td>Lotoprednol etabonate</td>
<td>Lotemax (Bausch &amp; Lomb)</td>
<td>0.5% suspension</td>
</tr>
<tr>
<td></td>
<td>Alrex (Bausch &amp; Lomb)</td>
<td>0.2% suspension</td>
</tr>
<tr>
<td></td>
<td>Zylet (Bausch &amp; Lomb)</td>
<td>0.5% suspension</td>
</tr>
</tbody>
</table>
ANESTHETICS

Local anesthetics play an important role in short-term, in-office management of acute ocular pain. The optometrist uses topical ophthalmic anesthetics each day when performing applanation tonometry, gonioscopy, pachymetry, certain contact lens fits or anterior segment procedures. All anesthetics are synthetic except for cocaine. The mechanism of action of anesthetics is by binding to sodium channels in the nerve cell membrane (Figure 19), blocking the influx of sodium across the cell membrane that creates the action potential. Blocking this action potential stops the transmission of pain signals to the central nervous system. This is shown in below:
Figure 19. Mechanism of anesthetic action. In this image, lidocaine binds to the sodium channel (via key anesthetic residue binding sites F1760 and Y1767), blocking sodium transduction and preventing the membrane depolarization and subsequent action potential (From http://www.nature.com/bjp/journal/v148/n1/fig_tab/0706709f5.html).

Along with blockage of sensory impulses, anesthetics can also block motor impulses, and subsequent paralysis of muscles can occur. Fortunately, this paralysis is usually temporary. Likewise, neuronal function returns to normal after the anesthetic effect wears off. Most topical ophthalmic anesthetics provide optimal anesthesia of between 10 to 20 minutes, and small doses of injectable ophthalmic anesthetics between 30 to 60 minutes.

Local anesthetics are divided into ester and amide classes, with the ester class being the topical ophthalmic anesthetics, and the amide class being the injectable anesthetics (Table 4).

<table>
<thead>
<tr>
<th>ESTHER ANESTHETIC</th>
<th>AMIDE ANESTHETIC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cocaine</td>
<td>Lidocaine (Xylocaine)</td>
</tr>
<tr>
<td>Proparacaine (AK-Taine, Alcaine, Ophthaine)</td>
<td>Mepivacaine (Carbocaine)</td>
</tr>
<tr>
<td>Tetracaine (Pontocaine)</td>
<td>Bupivacaine (Marcaine)</td>
</tr>
<tr>
<td>Benoxinate (Fluress – combo with fluorescein)</td>
<td>Etidocaine (Duranest)</td>
</tr>
<tr>
<td>Chloroprocaine</td>
<td>Procaine (Novocain)</td>
</tr>
</tbody>
</table>
Cocaine is unique in that it possesses both anesthetic and adrenergic agonist properties. Vasoconstriction, pupillary dilation, eyelid retraction and increased respiratory rate are common with cocaine. At 10% concentration, it is also used topically in the diagnosis of Horner’s syndrome. However, its corneal toxicity risk with repeated use, and strong abuse potential, limit its clinical usefulness. Cocaine is listed as Schedule II controlled substance under the U.S. Drug Enforcement Agency.

Proparacaine 0.5% is the preferred topical ophthalmic anesthetic. It has been shown to be superior to tetracaine 0.5% in terms of reduced stinging and burning, less corneal epithelial compromise and less allergic reactions. Proparacaine is also the anesthetic of choice when culturing off the eye, as it does not inhibit bacterial growth as much as tetracaine. Although both anesthetics are in the same class, cross-sensitivity is rare. Figure 20 shows the three most common ophthalmic topical anesthetics.

![Figure 20. Common topical ophthalmic anesthetics (L-R): Proparacaine (Alcaine), benoxinate (in Flurox©), and tetracaine.](image.png)

Some anesthetics are combined to allow better clinical use. Benoxinate is often combined with fluorescein sodium dye to allow easy applanation tonometry measurement (Fluress©, Flurox©). Lidocaine is often combined with the vasoconstrictor epinephrine (1 part epinephrine to 50,000-200,000 lidocaine) to decrease bleeding and maintain the anesthetic in the local area of injection (Figure 21). This is useful in providing nerve block for procedures such as chalazion or cyst removal. Reaction to epinephrine can occur in the form of heart palpitations, anxiety, difficulty breathing, and pallor, so noting these reactions in the patient’s record is important.
Toxicity with anesthetics is not uncommon, but is generally mild. Corneal epithelial desquamation and punctate keratitis is common after instillation of topical anesthetics, but generally does not decrease vision significantly, unless the patient has a pre-existing corneal compromise such as Sjogren’s syndrome or epithelial basement membrane disease (EBMD). In rare cases, corneal stromal edema and Descemet’s folds can occur, significantly reducing visual acuity.

Self-administration of topical anesthetics is not advised, as epithelial compromise and poor healing, sub-epithelial infiltrates, stromal opacification (in the form of a ring opacity) and melt, corneal neovascularization, and progression to uveitis with hypopyon can occur. Loss of the eye has been documented with prolonged use of topical anesthetics. As such, topical anesthetics should only be used for temporary in-office management of eye pain, and not for out-of-office use.

Systemic reactions to topical ophthalmic anesthetics are rare. Cardiovascular signs such as tachycardia and arrhythmia in the early stages, followed by hypotension and reduced pulse strength in the later stages, can occur if the blood levels of anesthetic are high. Respiratory failure can occur in cases of very high anesthetic in the bloodstream. Hypersensitivity is rare with local anesthetic use, and may be limited to blepharitis or conjunctivitis, keratitis, hives and angioedema. Anaphylactic reactions from local anesthetics are extremely rare.

Contraindications to using local anesthetics include:
- any hypersensitivity to anesthetics
- liver disease (increased anesthetic due to anesthetic being unmetabolized)
- taking anticholinesterases (anticholinesterases increase anesthetic concentration)
- dry eye testing (due to tear function and epithelial changes from the anesthetic)
- perforating eye injury (due to endothelial damage from the anesthetic)
CENTRAL ACTING AGENTS

When the pain is severe enough that a stronger analgesic is necessary, the optometrist may need to prescribe a central-acting narcotic agent. These agents are also called opioid (derived from opium) analgesics, and as such have morphine-like properties. Opioids bind to opioid receptors in the brain and spinal cord, mimicking the endorphins that normally bind at these receptors. The specific mechanisms by which they affect these receptors are not certain, but the result is a reduction in both pain and emotional distress associated with that pain (Figure 22).

Figure 22. Effect of opioid analgesics on the central nervous system. (Opioids such as morphine) bind to opioid receptors (primarily mu receptor subtypes) in the brain, causing a central reduction in pain sensation. This binding also causes an addictive psychological “reward” effect similar to endorphins (image from http://images.businessweek.com/ss/05/03/sciwinners/27.htm).

Unlike peripheral-acting analgesics, there is no “ceiling effect” with opioids. As a result, tolerance to the medication over time can occur, resulting in reduced effect and the potential for physiological/psychological addiction. The standard opioid by which other narcotics are compared to is morphine. Morphine can be highly addictive, and is typically only used in hospital settings, when no other opioid is indicated. The most
commonly-prescribed medication in the U.S. as of the writing of this article is hydrocodone, usually used in combination with acetaminophen (Vicodin). Codeine is also a common opioid, usually combined with acetaminophen in various amounts (Tylenol #2, 3, 4). Oxycodone and hydrocodone are several times more potent than codeine, and are also available in combination with acetaminophen or aspirin. Although hydrocodone is several times more potent than codeine, it tends to cause less sedation and gastro-intestinal problems than codeine. Oxycodone and hydrocodone may create a greater euphoria sensation compared to codeine. Common opioid analgesics are listed in Table 5 below:

### Table 5. Common Opioids (Narcotics), their Controlled Substance Schedule, and Typical Adult Dosages

<table>
<thead>
<tr>
<th>Opioid Name</th>
<th>Trade Name</th>
<th>Formulation</th>
<th>Controlled Substance Schedule</th>
<th>Typical Adult Dosage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Codeine</td>
<td>Tylenol #2, 3, 4</td>
<td>Codeine 15, 30, 60mg + acetaminophen 300mg Codeine 12mg + acetaminophen 120mg</td>
<td>C-III</td>
<td>1-2 tablets q4hr</td>
</tr>
<tr>
<td></td>
<td>Elixir (generic)</td>
<td>Codeine 12mg + acetaminophen 120mg</td>
<td>C-V</td>
<td>15ml q4hr</td>
</tr>
<tr>
<td>Hydrocodone</td>
<td>Vicodin, Lortab</td>
<td>Hydrocodone 5mg + Acetaminophen 500mg Hydrocodone 2.5mg + acetaminophen 120mg</td>
<td>C-III</td>
<td>1-2 tablet q4-6hr</td>
</tr>
<tr>
<td></td>
<td>Lortab liquid</td>
<td>Hydrocodone 5mg + acetaminophen 120mg</td>
<td>C-III</td>
<td>5ml q4hr</td>
</tr>
<tr>
<td>Oxycodone</td>
<td>Percocet</td>
<td>Oxycodone 5mg + acetaminophen 500mg Oxycodone HCl 4.5mg + oxycodone terephthalate 0.38 + aspirin 325</td>
<td>C-II</td>
<td>1 tablet q6hr</td>
</tr>
<tr>
<td></td>
<td>Percodan</td>
<td>Oxycodone 5mg + acetaminophen 500mg Oxycodone HCl 4.5mg + oxycodone terephthalate 0.38 + aspirin 325</td>
<td>C-II</td>
<td>1 tablet q6hr</td>
</tr>
<tr>
<td>Propoxyphene</td>
<td>Darvocet-N 100</td>
<td>Propoxyphene mapsylate 100mg + acetaminophen Propoxyphene HCL</td>
<td>C-IV</td>
<td>1 tablet q4hr</td>
</tr>
<tr>
<td></td>
<td>Darvon</td>
<td>Propoxyphene HCL</td>
<td>C-IV</td>
<td>1 capsule q4hr</td>
</tr>
<tr>
<td>Tramadol</td>
<td>Ultram</td>
<td>Tramadol 50mg</td>
<td>------</td>
<td>1-2 tab q4-6hr</td>
</tr>
</tbody>
</table>
Figure 23. Narcotic analgesics: Tylenol #3 (acetaminophen 300mg + codeine 30mg), Vicodin (acetaminophen 500mg + hydrocodone 5mg)

Tylenol comes in several 300mg acetaminophen:codeine combinations, designated by number (Tylenol #2 includes 15mg codeine, #3 includes 30mg, and #4 includes 40mg). Typical dosage is 300mg:30mg PO q4 hours, maximum to 12 tablets in 24 hours. Hydrocodone (with acetaminophen in Vicodin) is available in 500mg:5mg tablets, typically prescribed as 1-2 tablets PO q4-6 hours. It also comes in stronger 660:10mg (Vicodin HP) and 750:7.5mg form (Vicodin ES) forms as well as in combination with ibuprofen (Vicoprofen).

Figure 24. Dual-action NSAID: Ultram (tramadol)

Tramadol (trade name: Ultram) is a good choice when pain is not responding to peripheral-acting analgesics, but there is concern over using a stronger central-acting narcotic painkiller. Ultram has a dual central-acting mechanism, in that it weakly binds opioid receptors while also weakly inhibiting serotonin and norepinephrine reuptake. It comes in 50mg tablets. Typical dosage is 50-100mg PO q4-6 hours. Due to its weaker
effect, there may be less narcotic side effects than other opioid analgesics, although the practitioner should still be wary of adverse reactions.

Narcotic analgesics should be used for short duration only, as addiction can occur with these central-acting agents. Respiratory depression is another potential serious side effect of these medications, as well as sedation and gastrointestinal problems. The optometrist should determine if there is a patient history of narcotics dependency, and prescribe with discrimination. Careful prescription documentation, such as indicating “no refills” and spelling out the quantities rather than writing a number (i.e. ‘Twenty tablets’ rather than ‘20 tablets’) as well as communicating verbally with the patient’s pharmacist will help to prevent any potential forgery and abuse. In the United States, the inclusion of a DEA number is federally required when prescribing narcotic analgesics, as they are considered controlled substances.

Some of the behavioral and physical warning signs of substance abuse may include:

- Increased tolerance to analgesic effect
- Patient complains of “hurt and pain” in spite of medication
- Patient ‘pleads’ and ‘demands’ or states:
  - “I want (specific drug) for this pain”
  - “I will need more than three days worth”
  - “That one never works for me. I really need…”
- Irritability
- Sweating
- Rhinorrhea (runny nose)
- Tremor
- Restlessness
- Mydriasis (usually subtle)
- Anorexia
- Patient ‘figits’ in chair

The patient should be informed of the potential side effects of narcotic analgesics. Some key side effects include:

- Drowsiness, dizziness – avoid driving or operating machinery
- Blurred vision, diplopia, pupillary miosis
- Shortness of breath, heart palpitations
- Nausea & vomiting, gastro-intestinal upset
- Avoid alcohol, muscle relaxants, depressants

Contraindications to using opioid analgesics include the following:

- Narcotic hypersensitivity (cross-sensitivity common)
- Bronchial asthma, COPD
- Kidney or liver dysfunction
- Pregnancy or nursing
- Cardiac or vascular disease
Severe overdose of narcotic analgesics can on rare occasions be treated with naloxone or naltrexone (these are opioid antagonist). These injectable drugs rapidly reverse the effects of opioids – however, a life-threatening counter-effect can occur, leading to tachycardia, rapid breathing, hypertension, seizures, and even sudden death. Naloxone or naltrexone should only be administered in life-threatening opioid reactions, such as when severe respiratory depression occurs. Keeping the patient alert in a well-ventilated area and monitoring vitals may be a preferred approach to using naloxone or naltrexone.

As many analgesics fall under federal Drug Enforcement Agency (DEA) Controlled Substances classifications, it is useful to be familiar with these five schedule classifications, described in Table 6 below:

<table>
<thead>
<tr>
<th>Schedule</th>
<th>Description</th>
<th>Example Drugs</th>
<th>Prescribing Guideline</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>High abuse potential No significant medical benefit</td>
<td>Heroin, marijuana, LSD</td>
<td>LSD, heroin not available therapeutically</td>
</tr>
<tr>
<td>II</td>
<td>High abuse potential Severe dependence risk</td>
<td>Codeine, cocaine, Demerol, morphine, amphetamines, barbiturates, methadone</td>
<td>Written, signed prescription required</td>
</tr>
<tr>
<td>III</td>
<td>Moderate-low physical dependence or high psychological dependence</td>
<td>Tylenol #3, Vicodin, Fiorinal Phenergan + codeine Tussionix, Empirin #3</td>
<td>No more than 5 refills or dispensed more than 6 months after Rx date</td>
</tr>
<tr>
<td>IV</td>
<td>Less abuse potential Limited dependence</td>
<td>Valium, Xanax, Librium, Darvon, Phenobarbitol</td>
<td>As Schedule III</td>
</tr>
<tr>
<td>V</td>
<td>Limited abuse potential</td>
<td>Tylenol + codeine elixir, Phenergan + codeine syrup, Robitussin DAC syrup</td>
<td>Some over-the-counter allowed if 18 years of age or older; otherwise Rx needed</td>
</tr>
</tbody>
</table>

The optometrist may have had patients request marijuana (cannabinoids) for glaucoma treatment. Although this treatment modality is highly questionable and controversial, some patients have noted benefit of cannabinoid intake for chronic systemic pain (related to conditions such as terminal cancer). The centrally acting mechanism of action involves creating a ‘euphoric’ state that reduces the emotional suffering connected with pain. However, the obvious addictive effects of the drug limit its practicality for most treatments. The regional medical regulatory body determines if the use of marijuana for treatment of chronic pain is warranted. In other geographic regions, the use of cannabinoids for pain management is prohibited.

**ADJUVANT AGENTS**
Adjuvant agents may often serve an ‘assisting’ role to analgesics in reducing pain sensation. Tricyclic antidepressants such as amitriptyline (Elavil) 10-25mg per day are often used in cases of herpes zoster neuralgia pain, along with NSAID and/or narcotic pain relief. Likewise, stimulants such as caffeine and hydroxyzine serve to reduce the pain sensation when used concurrently with analgesics. Anticonvulsants such as carbamazepine, clonazepam, phenytoin, and gabapentin often work to reduce the gastrointestinal side effects that often accompany NSAID and opioid treatment. Topical creams such as oleoresin capsicum (the active component of jalapeno peppers) cream (Zostrix) serves to stimulate the P receptors on nociceptors, resulting in a later reduction of substance P from surface sensory nerves and ultimate reduction of pain sensation (Figure 25). Usually the effects are noted within 2 to 4 weeks of treatment with the cream used in a three- to four-times daily regimen. It is important to note that Zostrix is for dermatological use only, and that it can cause severe pain and stinging if applied in the eye directly.

Figure 25. Zostrix (oleoresin capsaicin) 0.025% and Zostrix HP 0.075% topical analgesic dermatologic creams

Unfortunately, there are some rare situations when pain persists even after maximum safe treatment has been prescribed. In these cases, referral to a pain management clinic for evaluation may be indicated, in coordination with the patient’s primary care provider.

CASE EXAMPLES

Corneal Abrasion/Foreign Body

A 26-year-old male was working in the yard when a branch scraped his left cornea. He is in extreme pain, and unable to open his eye to take visual acuities. The eyelids appear edematous and red in the affected eye.
In this situation, one drop topical proparacaine 0.5% was instilled to allow both visual acuities and non-contact tonometry to be taken, as well as evaluation of the cornea and conjunctiva. After informed consent was made regarding corneal foreign body removal and epithelial debridement, a second drop of proparacaine 0.5% was instilled and the foreign body and loose epithelium was removed without complication. A bandage therapeutic soft contact lens was placed on the cornea with topical antibiotics qid QID, which provided immediate comfort. The patient was also prescribed acetaminophen (Tylenol) 500mg every 4 hours for the pain. Follow-up the next day showed notable improvement of signs and symptoms, and the bandage contact lens was removed. The acetaminophen and antibiotic regimen was continued, with complete resolution 3 days later.

*Key points: The use of proparacaine was both diagnostic (to allow acuities, pressure, and evaluation) and short-term therapeutic (to relieve pain and allow foreign body removal and debridement). The contact lens served as a protective shield and the acetaminophen served to reduce the overall pain symptoms.*

**Uveitis**

This 45-year-old female presented with notable photophobia, blurred vision and pain in her right eye.
Diagnosis of uveitis was made, and the patient was placed on Pred Forte (prednisolone acetate 1% ophthalmic suspension) one drop q2hour, and scopolamine 0.25% one drop TID in the affected eye. As posterior cells and vitreal haze was also seen along with anterior chamber cells and flare, the patient was placed on 60mg oral prednisone (divided into 15mg at breakfast, 15mg at lunch, 15mg at dinner, and 15mg at bedtime) after consultation with the patient’s primary care provider. Referral to a uveitis specialist was made, and the patient was seen each following day, with slightly improved signs each day. Diagnosis of Vogt-Koyanagi-Harada syndrome was made. The treatment regimen was continued for 5 days, at which point signs and pain/photophobia symptoms were significantly improving. Tapering of the oral prednisone over the next 2 weeks was made, while the topical Pred Forte/cycloplegia dosage continued unchanged. By the end of the second week, discontinuance of the scopolamine and tapering of the topical Pred Forte was prescribed, with ultimate resolution of the patient’s signs and symptoms.

Key points: The cycloplegia provides comfort by mechanically fixing the inflamed iris, while the topical prednisolone reduces the ocular inflammation and associated pain. Oral prednisone serves to manage more posterior inflammation (vitreitis), only prescribed after systemic contraindications were ruled-out through the primary care provider/specialist. Tapering of both topical and oral steroids is necessary to prevent re-flare-ups.

**LASIK Flap Dehiscence**

A 49-year-old male inadvertently rubbed his left eye under his eye shield within 24 hours of having LASIK surgery, resulting in extreme eye pain and blurred vision. Examination showed corneal flap dehiscence and wrinkling, with flap movement with each blink.
After re-positioning the eye shield, the patient was informed to fill his post-operative prescription of Vicodin (acetaminophen 500mg + hydrocodone 5mg) and take one tablet every 6 hours for the pain, and promptly have his spouse drive him to see his refractive surgeon that day for re-positioning of the flap. The patient was informed not to squeeze or rub his eyes in the interim.

Key points: Topical anesthetic was not used in this case, to prevent possible corneal endothelial compromise. The opioid/acetaminophen analgesic combination allows excellent pain reduction with sedation to relax the patient – however, driving by the patient is not recommended.

Herpes Zoster Ophthalmicus

A 62-year-old female presented with “searing pain” on the left side of her face for the last 3 days, emanating from the eye on the same side. Her visual acuity was also reduced in that eye. Examination showed pustular lesions along the forehead and temple dermatome, with positive fluorescein staining dendrite-like corneal opacities in the eye on the same side.
Figure 29. Herpes zoster ophthalmicus dermatological lesions.

Diagnosis of herpes zoster ophthalmicus was made, and the patient was prescribed 800mg acyclovir five times daily for 1 week, as well as acetaminophen (Tylenol) 500mg every 6 hours and ibuprofen (Advil) 200mg every 6 hours. Topical trifluridine (Viroptic) one drop 9 times daily was also prescribed in the affected eye. Daily evaluation showed some resolution of the corneal opacities, with crusting of the skin lesions occurring. Although the visual acuity improved, the pain persisted, causing the patient great distress. The trifluridine was tapered to QID, capsaicin cream (Zostril) was prescribed QID, and the patient’s primary care provider was consulted in order to consider amitriptyline (Elavil). 10mg Elavil daily was prescribed in conjunction with the other medications, and the patient noted improvement of her pain symptoms over the next 2 weeks. The patient discontinued Elavil, Zostril, and Advil at that point, but continued the Tylenol TID. The patient tapered off the trifluridine by the third week, and a maintenance dose of Acyclovir 400mg BID was prescribed.

Key points: Post-herpetic neuralgia may require several approaches to pain management. In this case, combination of acetaminophen, an NSAID, a tricyclic antidepressant, and capsaicin cream were required to reduce the pain.

SUMMARY

The optometrist has several pharmaceutical choices to help patients with managing pain secondary to ocular disease. It is important to understand the potential side effects and contraindications to treating with analgesics, as well as knowing the current state, provincial, and federal laws for prescribing topical, injectable, and oral analgesics. It is also important to take a detailed case history, obtain a confident assessment of the patient’s pain symptoms, and apply proper treatment of the underlying disease causing
the pain, in conjunction with analgesic treatment. Factor in the cost, lifestyle modification, and minimum dosage required by the patient to effect optimal pain reduction, and adjust the treatment based on the patient’s response.

Judicious, safe prescribing of analgesics, when indicated, will allow better comfort for the patient, along with better compliance by the patient in their treatment regimen. And the reward obtained from the trust patients have in you, after resolving their pain, is immeasurable.

**DISCLOSURE**

The author has no proprietary interest or financial investment in any of the products mentioned in this article.

**REFERENCES**


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