Did You Know?
A patient’s pain may not come from the site where it is perceived.

Compounded preparations often allow lower dosages than commercially available medications.

CASE REPORT
Ketoprofen and Gabapentin in Anhydrous Gel Base for Migraines
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A 42-year-old white woman was referred to us by a local neurologist in September 2002. She had had severe, debilitating migraines for the past 13 years. During that time, she had taken a variety of medications, both prescription and nonprescription, without relief. At the time of the consultation, she was having eight to ten migraines per month. She had been prescribed the antimigraine agent zolmitriptan (Zomig) 5 mg tablets and would take 1 tablet as soon as she felt a headache coming on, but got little relief. She rated the headache pain as 9 or 10 on a 10-point scale with 10 as most severe. The pain was severe enough that she would have to stay home from work, and she was missing about 8 days of work each month.

The patient was in good general health and had no other medical problems. She took no other medications. Tired of living with the pain, she consulted a neurologist. He

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INTERVIEW
Compounding for Orofacial Pain Management
An Interview with Gary M. Heir, DMD, AAOP

Gary M. Heir, DMD, an expert in the management of orofacial pain, is an associate clinical professor in the Department of Oral Medicine at the Newark campus of the University of Medicine and Dentistry of New Jersey. A past president of the American Academy of Orofacial Pain (AAOP), he maintains a solo private dental practice in Bayonne, New Jersey. Here, Dr. Heir explains the use of compounded medications to treat chronic pain disorders that affect the head, neck, and face.

Explain the pathogenesis of orofacial pain.
Contrary to popular belief, temporomandibular disorders account for only a portion of orofacial pain, which is defined as any chronic complex pain disorder of neurologic, muscular, vascular, or psychogenic origin that affects the head, neck, and/or face. Orofacial pain is not caused by the average toothache or a locked temporomandibular joint, and it is not an acute-onset condition for which an immediate cause and effect can be established and an obvious treatment identified. It is usually caused by one or more chronic conditions that can elude diagnosis, and it may persist for months or years if an effective treatment is not identified.

Describe some of the most common types of orofacial pain and how compounds are used to treat them.
The more peripheral the pain, the more likely I am to prescribe a compounded medication. Compounds are very effective in treating orofacial pain because they can be formulated for the specific medical needs of the patient.

Trigeminal deafferentation toothache
A very small number of dental patients experience trigeminal deafferentation toothache (atypical odontalgia)
A very small number of dental patients experience trigeminal deafferentation toothache, which can develop weeks, months, or (occasionally) years after trauma to a tooth or even after an innocuous dental treatment.

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such as an endodontic procedure or the use of deep curare in dental cleaning. Recognizing the source of that pain is very important. The pain is caused by damage to the sensory innervation of the treated oral region and is characterized by chronic pressure, pain, or a burning sensation that can spread into the gum and cheek and may be referred to the opposite side of the oral cavity. Treatment with common analgesics such as aspirin, acetaminophen, or codeine is ineffective. Attempting to relieve that pain by repeating an endodontic procedure in a patient with no obvious dental pathology and performing an apicoectomy may be a mistake, as is removing the painful tooth. In fact, additional invasive procedures may worsen the symptoms. Because deafferentation toothache is a type of neuropathic pain that affects the central nervous system, it cannot be eliminated with a local injection of an anesthetic such as procaine (Novocain). A compounded formulation of medications including lidocaine, a nonsteroidal, and antinflammatory drug, and (occasionally) capsaicin often provides rapid relief.

Postherpetic neuralgia

Facial postherpetic neuralgia can develop when herpes simplex virus that was dormant in the trigeminal ganglion for years somehow becomes reactivated. As the virus leaves the ganglion of the spinal cord, it makes its way along the nerve route to the peripheral nerve, tiny tissue receptors, and the patient notices a tingling sensation in the distribution of the affected nerve. The erupted ulcers eventually coalesce, form a crust, dry, and resolve. Most patients experience no residual symptoms. In some cases, however, any of several factors (the virulence of the virus, the immunoresistance of the patient, and the extent of damage to the nerve route or the sites on the skin at which the eruptions occurred) may produce an enduring, painful sensitization of the peripheral receptors. Damage to the primary afferent nociceptor nerves can also cause postherpetic pain, as can central neuroplastic changes in the dorsal root ganglion of the spinal cord. Selectively compounded pharmacologic agents can target those pain-trigger sites and provide relief. A compounded topical analgesic that targets exposed nerve endings is often the only medication necessary to produce long-term relief, or it may be used in combination with systemic medications that can then be given at a reduced dose. Eliminating or reducing the use of systemic medications limits or prevents the adverse effects caused by many of the medications used to treat postherpetic neuralgia. This is important because many patients afflicted with that disorder are medically compromised or elderly. In one of my patients, facial postherpetic neuralgia is located in an area in one ear. The pain from that condition prevented her from lying comfortably with her head on a pillow, holding a telephone receiver to her ear, and going outside, because the pressure of wind on the affected area was painful. I knew that a tricyclic antidepressant (the first choice of therapy) would produce a constellation of adverse effects such as increased appetite, thirst, urinary retention, and somnolence. A standard dosage of gabapentin (Neurontin) might have relieved the pain, but the patient may then have been too sleepy to work. This patient’s facial postherpetic neuralgia has been successfully relieved by a compounded antiepileptic medication formulated for topical application. When occasional breakthrough pain occurs, she applies a standard lidocaine patch (Lidoderm patch 5%) that has cut to the size of a 50-cent piece and wears it for the remainder of that day.

Trigeminal neuralgia

Compounded medications are also effective in the treatment of traumatic trigeminal neuralgia, which is characterized by brief, sharp bolts of paroxysmal pain that may be spontaneous or triggered by innocuous light touch. As a test for that disorder, I apply a Lidoderm 5% patch to the area of suspected neuropathic pain. If the patch provides relief, I know that topical analgesics may work. Neurologically active medications (lidocaine, capsaicin, ketoprofen, ketamine, carbamazepine, gabapentin, and sometimes lidocaine) that stabilize the membranes of sensitized nerves can then be compounded for topical application either as monotherapy or in various combinations. Patients can also relieve their trigeminal neuralgia or trigeminal deafferentation toothache by using Orabase Protective Paste (pectin/gelatin/trigeminal deafferentation toothache by using Oral Phosphate Protective Paste (pectin/gelatin/sodium carbonate/ethylcellulose in a plasticized hydrocarbon gel) to affix a neurosensory stent (a custom-made acrylic appliance designed to fit over the affected area) filled with the compounded medication. The medication is conveyed directly to the site of the patient’s pain, and the appliance can be refilled and reapplied to sustain relief. For many patients, that treatment has proven remarkably effective. Some of my patients who initially recommended a topical preparation that has been shown to alleviate pain in a variety of strengths.1 He prescribed a combination of ketoprofen 4% and gabapentin 5% in an anhydrous gel base for good skin penetration. The patient dispensed the gel on September 27, 2002, and the patient was instructed to apply a small dab (smaller than a pea) to a trigger point on her forehead every day, regardless of whether she had a migraine. The woman followed the neurologist’s advice and began using the gel preparation as prescribed. Using the gel to support the gel on the evening before going to bed. Within weeks she experienced substantial relief of her migraines. After 14 months, she continues to use the gel daily. At a recent follow-up consultation with pharmacy staff, she reported that she is having only one or two migraines per month and that the severity of the pain is greatly reduced. She continues to take one 5-mg zolmitrin tablet at the first sign of migraine. She rated the severity of her migraine pain as 3 on the 10-point scale. She has experienced no adverse effects or other problems using the topical preparation. She is very pleased with the treatment, and so is her employer.

According to the National Headache Foundation (NHF), more than 45 million Americans suffer from headache. The pain and associated symptoms are very disabling, and impact can be so severe that the person’s ability to perform everyday work, school, family, and social activities is adversely affected. The NHF estimates industry losses of approximately $50 billion per year owing to absenteeism, lost productivity, and medical expenses due to headache.4

References