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Headache: Is it a migraine? Think again

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For many years, headaches or "migraines" have been felt to be intracranial in nature. However, recently a large number of "migraines" have been found to be due to extracranial causes which are amenable to interventional pain techniques. Recognition of the causes of extracranial headaches will result in effective and rewarding headache treatment.

"*If you would test the skill of a young physician, give him a patient with a headache to treat.*"
William Sunderham, MD
University of Michigan 1935

Interventional pain physicians have traditionally focused on spinal pain as their area of interest. Despite the fact that many back pain patients develop chronic headaches as part of their disease process, most interventionalists have had neither the training nor the interest in treating headache patients. Since headaches are felt to be primarily a "female disease," there has been little interest in the diagnosis and treatment, much in the same way as premenstrual syndrome has been largely ignored. Headaches appear to be considered the "property" of neurologists, and because headaches (especially "migraines") are felt to be primarily intracranial, they have been of little interest to an interventionalist. However, as the pathophysiology of extracranial headaches becomes better known, it becomes clear that there is a role as well as a need for interventional treatment of this potentially debilitating condition. This treatise is an attempt to introduce the pain physician to a practical approach to the diagnosis and treatment of headaches and "migraines." I have deliberately used the term "migraine" in quotes, because much of the confusion regarding headache management has come from the use of this term. The International Headache Society (HIS) has defined "migraine" as an intracranial headache, unilateral and throbbing, associated with photophobia, phonophobia, and nausea. Unfortunately, extracranial headaches can present symptoms in exactly the same way, and patients usually use the term "migraine" to mean a "sick headache." As a result, the term is meaningless and confusing, and I will use the term "headache" instead.

In the evaluation of headache patients, the same principles of history, physical examination, and diagnostic studies apply. However, the diagnostic studies are primarily to identify secondary causes of headaches, such as tumors. The true diagnosis is best made at the time of the patient's headache by the use of a diagnostic injection. It is amazing and gratifying (as well as diagnostic) to be able to provide nearly instant relief to a patient with an acute headache, by performing a diagnostic (as well as therapeutic) injection. "Almost before the needle is out of the head," the headache, nausea, and photophobia start to rapidly resolve. Within a few minutes, the patient will start to feel "normal."

As with all diagnostic injections, an injection of a structure with a small volume of local anesthetic that gives at least temporary relief of the pain is considered diagnostic of a pain generator. If the patient does not get relief, that structure is not considered to be the source of the pain. However, that assumes that the patient gets numb from that local anesthetic. As was reported in *Pain Physician*,[1] skin testing with lidocaine, bupivicaine, and mepivicaine revealed that 7.5% of the studied patients only got numb from mepivicaine, and another 4% only get numb from lidocaine. In these patients, bupivicaine (the most commonly used local anesthetic because of its long-lasting effect) would be either very slow in onset or totally ineffective, potentially even resulting in increased pain. In my practice, therefore, all patients are skin tested to evaluate the most effective local anesthetic before any injections.

As with every disease, once a diagnosis is made, the long-term treatment plan can be formulated. Practitioners must realize that the term "headache" is not a diagnosis, but rather a symptom; just as is the way that low back pain must

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be viewed. Treating a patient with low back pain by using epidural steroids for sacroiliac pathology will give no greater long-term relief than treating an occipital neuralgia patient with triptans.

The pertinent history of a headache patient centers on the site of the initial pain sensation, the referral pattern, and the contributing factors. For example, a patient complaining of unilateral or bilateral temple pain (triggered by talking, chewing, and menses), which awakens the patient at 3 or 4 o'clock AM, should immediately bring to mind auriculotemporal neuralgia (ATN), as I will describe later. A confirmatory physical examination of the ATN region showing tenderness of the region would be supportive of this diagnosis. However, similarly presenting pathologies, such as temporomandibular joint (TMJ), sternocleitomastoid muscle (SCM), supraorbital, and occipital, may also be supported or refuted by the physical examination. The diagnosis is then confirmed by a small-volume injection of the presumed pathology, such as the ATN. If local anesthetic (as well as deposteroid if indicated) gave excellent but only temporary relief, more long-term therapies such as cryoneuroablation, radiofrequency lesioning, botulism toxin, regenerative injection therapy, or subcutaneous stimulation, in addition to rational drug therapy (anticonvulsants for nerve pathology, muscle relaxants for muscle pathology, etc.) could then be offered. The dictum "You can not treat what you can not diagnose" has never been truer than in the treatment of headaches.

Supraorbital neuralgia

Clinical presentation

The headache of supraorbital neuralgia is often confused with frontal sinusitis, cluster headaches, and menstrual migraines. The headache may have been preceded by trauma recently, or many years later, as a cicatrix forms around the nerve over time. The nerve can become entrapped by fluid retention such as during the perimenstrual time frame or by salt intake. The nerve can also be injured by poorly fitting glasses or entrapped by frowning or squinting. The supratrochlear nerve, which is present medially, may also have pathology, and may need to be treated concurrently or sequentially. The patient will complain of a frontal headache, either dull or throbbing, unilaterally or bilaterally. There may be severe nausea and emesis. Triptans will help but only temporarily. There can be lacrimation, scleral injection, and sudden stabbing pain that can mimic cluster headaches. The patient will often self-treat with "sinus medications" without relief.

Anatomy

The supraorbital nerve is the termination of the first division of the trigeminal nerve. Irritation of the nerve occurs primarily at the supraorbital notch. A small ligament completes the inferior border of a foramen through which the nerve passes before passage through the orbicularis oculi.

Diagnostic injection

On physical examination, there is marked tenderness over the supraorbital notch, and/or over the supratrochlear nerve at the lateral border of the nasal bone. This area is very sensitive, and a 30-gauge needle is appropriate. Less than 1 cc of total volume should be used, and care must be taken to avoid directing the needle into the foramen itself, since injection of a large volume in the foramen could cause increased entrapment of the nerve.

Further therapies

Supraorbital neuralgia usually responds well to cryoneuroablation, with a dramatic and sustained relief of the headache. It is interesting that Botulin toxin, first used by plastic surgeons to treat frown lines, was noted to relieve certain types of headaches. Also a retrograde effect on the DRG has been proposed as a potential mechanism of the headache relief from Botulin toxin. I think it is much more likely and logical that this represents a decrease in pressure on the supraorbital nerve with frowning, which is then released with the relaxation of the muscle. Pulsed radiofrequency has not been described, but theoretically might be appropriate. Early experience with subcutaneous electrical stimulation suggests that there may be a role for this therapy in intractable cases.

Auriculotemporal neuralgia

Clinical presentation

Auriculotemporal neuralgia (ATN) is probably the most common of the trigeminal headaches. The pain is primarily in the temple, with radiation to the retro-orbital region, and may be unilateral or bilateral. There may be associated ear and lower jaw pain. The headache is often throbbing in nature, probably due to its proximity to the temporal artery, and can be associated with severe nausea and vomiting. This is the headache that wakes the patient up at 3 or 4 AM, presumably due to clenching or bruxing during this lightest stage of sleep. It is misdiagnosed as TMJ pathology (though the auriculotemporal nerve gives innervation to the joint).

Anatomy

The auriculotemporal nerve derives from the third division of the trigeminal nerve. It runs inferiorly under the pterygoid muscle to the neck of the mandible and then turns cephalad to travel with the temporal artery between the external ear and the condyle of the jaw, giving branches to the TMJ and the anterior ear as it passes cephalad to the temporalis muscle.

Diagnostic injection

There are two sites of pathology and therefore two potential injection sites. The most common is the distal en-

trapment that occurs at the temporalis muscle. This spot is usually identified as an exquisitely tender area at the apex of an isosceles triangle with the base composed of lines connecting the tragus and the corner of the eye. This is usually in close proximity to the artery. One cc of local anesthetic and depot steroid are then injected in a cephalad direction, parallel to the path of the nerve. The artery is usually slightly more posterior, so, if the pulsations of the artery are not obvious, I usually try to place the injection slightly anteriorly to avoid a hematoma. Careful evaluation will confirm that the artery itself is not tender, therefore decreasing the likelihood of a missed diagnosis of temporal arteritis. The second site is more proximal and less common. It is located as the nerve passes anterior to the TMJ, just inferior to the zygoma. This is close to the facial nerve, and the patient must be warned of the possibility of temporary facial weakness. For that reason, small volumes (less than 0.5 cc) and meticulous needle placement are important. There is occasionally a "double crush" syndrome with the nerve trapped at both sites, and both would then need to be treated.

Further therapies

The distal ATN entrapment is very amenable to cryoneuroablation. However, because of the proximity of the facial nerve to the proximal ATN, special care and meticulous motor stimulation are advisable for the proximal technique. The use of clonazepam or tizanidine at night will keep the patient in a deeper plain of sleep, decreasing the bruxing and therefore decreasing the entrapment. Standard bite blocks are usually ineffective, but an anterior occlusion splint, which places the front teeth in opposition and prevents clenching, may be useful. (You may wish to try this yourself. First, place your finger on your temple and clench, feeling the temporalis contraction under your finger. Then, place your front teeth together and try again. Neither the masseter nor temporalis can contract.)

Facial neuralgia

Clinical presentation

Although the facial nerve is primarily a motor nerve, it does carry sensory fibers to the face and can be a cause of headaches. The most common entrapment is the zygomatic branch at the coronoid notch. This headache usually occurs in the morning after the dentures have been out all night, with the loss of the dentition height leading to a narrowing of the coronoid notch, which results in entrapment. Other less common entrapment sites include the temporal branch, lateral to the orbicularis oculi.

Anatomy

The facial nerve exits the cranium at the stylo-mastoid foramen. The nerve runs within the body of the parotid gland and then splits into five branches to supply five areas of the face: temporal, zygomatic, buccal, mandibular, and cervical. The zygomatic branch of the facial nerve lies just inferior to the zygoma. A small volume of local and deposteroid (total volume less than 2 mL) can give nearly instant relief.

Further therapies

Most of the small branches of the facial nerve are amenable to cryoneuroablation. However, it is important to remind the patient of the risk of motor weakness. Luckily, the motor function is the first to return, usually long before sensory function, and many desperate patients would gladly exchange a small amount of facial weakness in exchange for relief of intractable headaches.

Posterior auricular neuralgia

Clinical presentation

Pathology of the posterior auricular nerve (also called the greater auricular nerve) will cause a parietal headache associated with ear pain. This nerve is injured by trauma to the mastoid such as seen with blows to the head. For that reason, these headaches are more common on the left, since most male abusers are right handed. Flexion/extension injuries, especially with the head turned, will also traumatize the nerve. The entrapment may be seen many years after the trauma as a cicatrix of scar tissue forms around the nerve. Palpation of the posterior auricular area will reveal a palpable groove just posterior to the SCM attachment, which will be tender and will replicate the pain.

Anatomy

The posterior auricular nerve is a branch of the superficial cervical plexus, and runs along the posterior border of the SCM muscle, superficially and immediately posterior to the mastoid.

Diagnostic injection

The tissues of the posterior auricular region are very thin, and the nerve is usually easy to identify. The apparent entrapment is just posterior to the SCM attachment, and the diagnostic injection should enter just at the base of the skull, aiming cephalad and slightly anteriorly. The injection should be of a small volume (less than 1 cc) of a depot steroid and local anesthetic, taking care that the needle remains on bone.

Further therapies

Cryoneuroablation of the posterior auricular nerve can be very successful. Care, however, must be used to avoid "frostbite" of the superficial tissues since the skin can be quite thin at this region.

Occipital neuralgia

Clinical presentation

Occipital neuralgia is probably one of the most recognized of the extracranial pathologies. These headaches usually start at the base of the skull, and radiate anteriorly, often to behind the eyes. However, as noted earlier, the presentation may be first in a trigeminal distribution, often described as an "ice pick" in the retro-orbital region with pain then radiating to the occipital region. Alternatively, the pain may start as a "tension headache" in the upper cervical region and then center at the base of the skull. The headache can be sharp, dull, or throbbing, and may be unilateral or bilateral, with nausea and photophobia. The nerve can be entrapped by the trapezius muscle, and is commonly seen in flexion/extension injuries.

Anatomy

There are actually three occipital nerves. The largest, the greater occipital nerve, is the dorsal ramus of C2. It originates lateral to the lateral atlantoaxial joint, deep to the inferior oblique muscle, where it may receive a communicating branch from C3. It then ascends up the neck over the rectus capitus and then pierces the semispinalis capitus muscle, deep to the trapezius. It then exits the neck through an aponeurotic sling composed of the insertions of the trapezius and sternocleidomastoid muscles, close to the occipital artery. The lesser occipital nerve is also a branch of C2, but travels more laterally, and exits the neck just lateral to the occipital prominence. The third occipital nerve also comes from C2 and C3, running medially to the occiput, innervating the suboccipital region.

Diagnostic injection

The classic description of the occipital nerve was for surgical anesthesia of the posterior scalp. Ten cc of local anesthetic was injected subcutaneously from lateral to medial across the nuchal ridge. This would usually effectively anesthetize all three nerves, but not at the site of the entrapment. In addition, because the tissue is so adherent to the scalp at this level, large volumes could potentially create an entrapment of the nerves. Two alternative techniques are being described here.

For my technique, the injection site (in this case describing the right side) is identified by placing the thumb of the right hand at the foramen magnum (which identifies midline and avoids the cisternal injection); the index finger is then placed at the conjoined tendon attachment, and the second finger will then identify the injection site at the base of the skull. The needle is directed cephalad and slightly medially until contact is made with bone. Small volumes (less than 2 mL) of local and steroid are thereby injected underneath the tendon where the nerve pierces the tendon attachment.

Dr. Gabor Racz (personal communication) instead recommends a cephalad to caudad approach to reach the potential space anterior to the splenius muscles, using large volumes to "decompress" the nerve proximal to the fascia piercing. A specialized curved blunt-tipped "stealth" needle has been developed to facilitate this approach.

Further therapies

Cryoneuroablation of the occipital nerve is probably for most practitioners the most familiar of the cryoablation techniques. If the small volume technique described above gives good but only temporary relief, the cryo probe can be advanced from a caudad to cephalad direction to the occipital bone, and the nerve frozen at that site. Pulsed RF is being used, but I would strongly caution against heat RF or surgical resection of the nerve because of the high incidence of neuroma formation. However, if the pathology is more proximal, the C2 or C3 nerve root may need to be addressed. There has also been a good deal of interest in the use of occipital subcutaneous stimulation for intractable occipital headaches.

Myofascial pathology (masseter, SCM, trapezius)

Clinical presentation

Myofascial pain or trigger points can present as a variety of headache conditions. Chronic stress leading to teeth clenching, bruxism, dental malocclusion, and TMJ pathology can all cause spasm of the masseter muscle, which will refer pain to the temples, jaw, and over the eye. The SCM muscle will refer pain to the ear, temple, and face, especially over the eye. Patients often complain of fullness in the ear with decreased hearing, leading to unnecessary ENT evaluations. There can also be tinnitus and vertigo, mimicking vestibulitis. Since flexion/extension injuries will traumatize the SCM, what have been considered coup-contra coup brain injuries are now being recognized as myofascial pain. "Tension headaches" is a term that seems to trivialize the intractable occipital and retro orbital headaches that are caused by trapezius spasm. The pain can be caused by stress, chronic postural problems (for instance with prolonged neck flexion for reading), or flexion/extension injuries.

Anatomy

The anatomy of these muscles is well described in Travell.[2]

Diagnostic injection

Trigger point injections such as described in Travell can give immediate, dramatic, and sustained relief.

Further therapies

Injections with botulin toxin has been shown to give long-term relief, up to 3 to 4 months. This provides a window of opportunity for neuromuscular reeducation.

Cervical ligament pathology

Clinical presentation

In 1954, Feinstein[3] followed up on work done by Kellgren,[4] which showed that irritation of the cervical ligaments can refer pain to the head and face as well as the extremities. These cervical ligaments are traumatized in flexion/extension injuries but also can occur with chronic low-grade trauma. The subsequent ligament laxity no longer allows support of the 30-pound head, and the cervical muscles will go into spasm to hold the head up. This ligament pathology results in a straightening of the cervical lordosis. Thus, the common x-ray diagnosis of "loss of cervical lordosis secondary to spasm" is actually the reverse—contraction of a muscle above and below the lordosis must cause *more* lordosis if the muscles are the pathology. On physical examination, palpable defects in the ligament can be observed, and patients often describe the sensation that their head is "too heavy to hold up."

Anatomy

The dorsal median branch of the dorsal median nerve innervates the interspinous ligament, as well as the facets and paravertebral muscles. Then, it enters the spinal column at the level of the cervical nerve roots. Whereas facet pathology will give a unilateral referred pain, interspinous ligament pathology will give bilateral pain. Pain can be referred to the face or occipital region, as well as down the shoulder or arm, just as is seen with facet pathology. In fact, there is an argument that facet pathology (as well as disc pathology) cannot occur without ligamentous disruption.

Diagnostic injection

The interspinous ligament injection is performed by injecting 1 cc of local and depo-steroid into the superficial ligament, dorsal to the level of the lamina. To decrease the risk of entering the epidural or intrathecal space, the needle should be directed inferiorly, ideally contacting the spinous process to confirm depth.

Further therapies

If the diagnostic injection gives good but only temporary relief, Regenerative Injection Therapy (RIT), formally known as prolotherapy, may give long-term relief. An old technique that fell out of favor but has gradually returned into mainstream therapy, RIT relies on the injection of deliberately irritating material at the fibro-osseous junction to stimulate the proliferation of fibroblasts, causing the production of new tissue at the area. Typical solutions are based on high concentrations of dextrose with local anesthetic. Not only is there symptomatic relief, but also the reinforcement of the ligament can be palpated as a decrease in the ligament defect. However, patients on chronic anti-inflammatory agents or anticoagulants, as well as smokers, do not usually respond well, probably due to suppression of the fibroblast activity. In addition, this technique is currently not approved by Medicare. An alternative therapy may be to use heat radiofrequency lesioning at the same fibro-osseous junction. This results in a denervation of the painful periosteum, but may also create a thermal stimulus for fibroblastic activity.

Conclusion

Aggressive treatment of headaches and "migraines" can be gratifying and technically satisfying for the interventional pain physician with the knowledge and skill to provide injection therapy to patients with these pain problems.

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