

NEUROGENIC HEADACHE AND NECK PAIN IN ATHLETES

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INTRODUCTION

Head/neck pain is common in elite athletes but the diagnosis and treatments are often not clear. Nerve injury/compression represents a head/neck pain pathology that has historically been underrecognized and requires a specific treatment plan¹. Athletes commonly suffer head and neck trauma and these injuries can result in swollen, painful nerves. Identifying nerve compression is critical as effective treatments are available. Unfortunately, too often, nerve compression is missed leading to persistent suffering and inability to return to play.

The recognition of the morbidity caused by painful compressed nerves in the head and neck has blossomed over the last twenty years. Dr. Guyuron serendipitously noticed the role of compressed nerves in pain in his plastic surgery practice. He then proceeded to study in detail the pathology and demonstrate the efficacy of surgical decompression². Since his 2000 paper, his work has provided a guide to the nerves at risk of compression and others have built upon this foundational knowledge improving our care of these patients³. Now physicians are better equipped to diagnose and treat these painful nerves.

Athletes represent a population with unique risks for nerve injury and compression. In many sports, play puts the head and neck at risk for blunt forces or whiplash. Whiplash is a sudden forceful flexion and extension of the neck and occurs in many high-impact sports⁴. Concussions and traumatic brain injury also occur frequently in sports and there are international efforts to minimize their impacts⁵. After whiplash or concussion, many athletes experience post-traumatic headaches with the prevalence of acute post-traumatic headache as high as 90%. At 6 months post-injury, 30–50% of patients still experience headaches. It is now clear that a portion of post-traumatic head pain is caused by peripheral nerves that become swollen and subsequently compressed⁶. This article will provide information regarding relevant nerve anatomy and clinical presentations to aid in recognition, treatment, and return to play.

DIAGNOSIS

The diagnosis is made by gathering many pieces of information to localize the pain generator. First, a full history is required. If symptoms occur after a trauma this points

to a post-traumatic process. If a patient states that the head pain is orthostatic, improved when lying flat, or associated with ringing in their ears, this can suggest a cerebral spinal fluid (CSF) leak leading the provider down a different treatment pathway. CSF leaks should be considered in patients with head pain after skull fractures and have been described in many different athletes⁷.

Immediately following a detailed history comes the physical exam. Palpation along the anatomical course of the affected nerve paying particular attention to common compression points is crucial. Tenderness at known points of compression is an important finding. Additionally, in the case of the brachial plexus and dorsal scapular nerves, provocative actions, like head rotation and cervical flexion may reproduce or intensify symptoms. Sensory examination can show dysesthesia or decreased sensation in the distribution of the compressed nerve. Thus, a strong understanding of the nerve sensory distributions is critical (Figure 1).

If a diagnosis of nerve compression is suspected, the next diagnostic step is a focal injection with local anesthetic with or without steroids. The diagnostic block

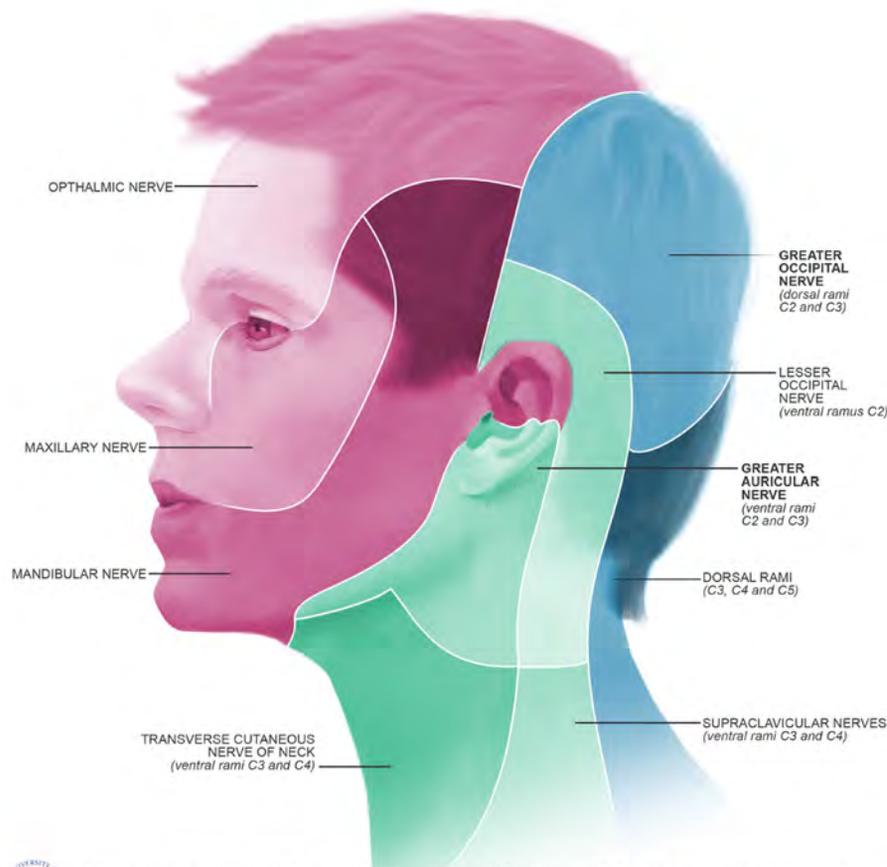


Figure 1: “Dundee - Drawing Dermatome map of head - English labels” at AnatomyTOOL.org by Annie Campbell, ©University of Dundee School of Medicine, license: Creative Commons Attribution-NonCommercial-NoDerivs. Reviewed by: Dr. Penny Lockwood, Dr. David Stewart, Dr. Patrick Spielmann.

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is considered positive if the patient has at least 70% reduction of pain and symptoms following injection. It is important for patients to understand that the relief from the block only lasts for a short time. The patient should record the results of their block and alert their provider the next day. Otherwise, as time passes the patient can forget the impact of the block on their pain.

Imaging, especially ultrasound, can be a valuable tool in diagnosis. Recent advances in ultrasound technology along with the increased use of point-of-care ultrasound have allowed for bedside examination of nerve and musculoskeletal anatomy. These exams can show increased diameter of the nerve suggesting swelling and compression. Ultrasound can also be used to guide diagnostic and therapeutic nerve injections improving test accuracy and symptom relief.

TREATMENT

Botox

Botox has been used for decades in the treatment of dystonia, seizures, muscle hypertrophy, and rhytids in cosmetic patients. With its controllable dose-

dependent paralytic effect, it is postulated that the therapeutic effect of this medication lies in its ability to provide muscle relaxation reducing the pressure on the nerve. Recent literature suggests that botulinum toxin also has an analgesic mechanism of action independent of its effect on muscle relaxation^{8, 9}. The PREEMPT protocol is the standard protocol utilizing Botox in the treatment of head pain¹⁰.

Therapy

Physical therapy by means of stretching, massage, and traction therapies serves as another nonsurgical option to attempt to reduce pain. For patients with nerve compression, the success level of correcting the compression is variable but can be invaluable to address posture compensations that negatively impact return to play.

Surgery

If the physical exam supports nerve compression and the patient has had a positive response to a diagnostic injection, surgical decompression of the nerve serves as an effective treatment. Detailed

descriptions of each surgical release technique are beyond the scope of this article, however, there is one central guiding principle for these procedures – surgeons should take care to identify and release all possible sites of compression paying special attention to the anatomical course of the nerves.

Specific Nerves at Risk

Nerves are commonly at risk for compression along transition points: crossing a joint, passing between tissue planes, exiting a bony foramen, et cetera. This next section will review the anatomy of common nerves and known compression locations in the head and neck. This is not an exhaustive list and providers should be aware that any nerve could be at risk of compression. If pain is focal, a review of nerve anatomy can provide insights into possible pain generators.

ANATOMY

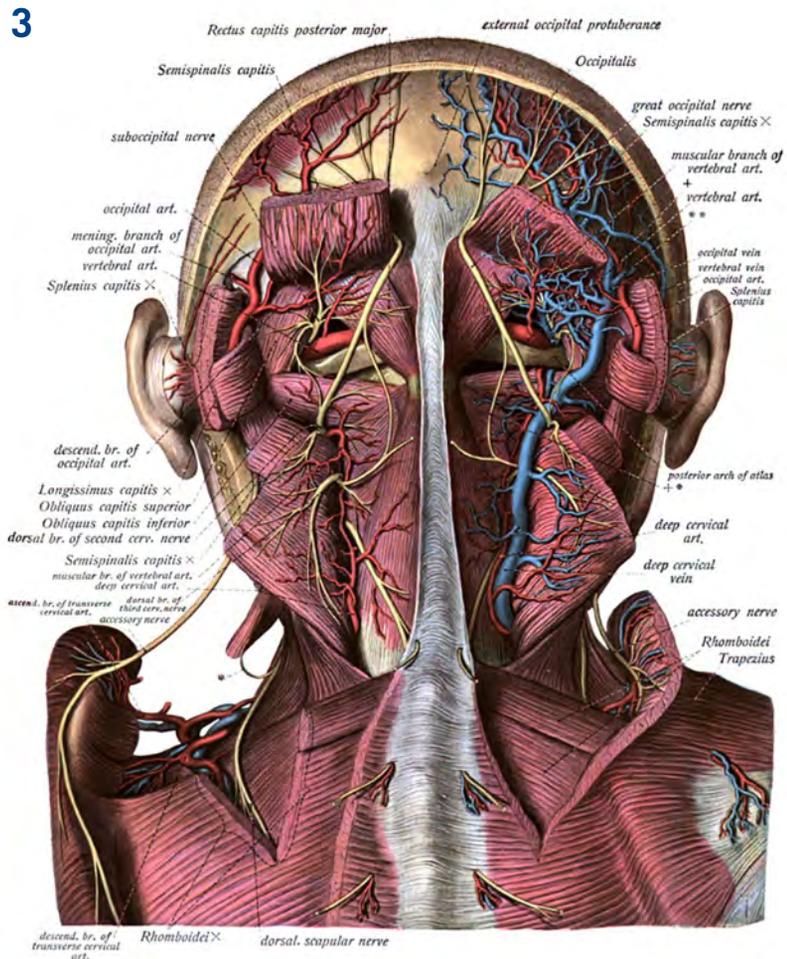
*Greater Occipital Nerve*¹¹⁻¹⁴

The greater occipital nerve arises from the lateral dorsal ramus of C2 to innervate the semispinalis capitis muscle and provide



Figure 2: The “x” marks the point where the greater occipital nerve transitions superficially through the semispinalis. “OP” represents the occipital protuberance.

Figure 3: Anatomy of the posterior neck “Sobotta 1909 fig.696 - Nerves and vessels of the nuchal region, deep layer - English labels” at AnatomyTOOL.org by Johannes Sobotta is in the Public Domain.



cutaneous sensation to the posterior scalp. The nerve runs lateral to midline ascending between the obliquus capitis inferior and the semispinalis before entering the semispinalis muscle medially around 5cm inferior to the occipital protuberance and then travels superolaterally within the muscle before exiting the muscle. This exit point, which is often the point of compression, is typically 1.5 cm lateral of midline, 3.5 cm inferior to the occipital protuberance, and 2 cm deep to the skin (Figure 2 and 3).

There are several well-recognized points of nerve compression. The surgeon should identify the nerve as it travels through the semispinalis and then explore the nerve proximally and distally assessing for other points of compression such as a crossing artery and as the nerve traverses the trapezius muscle insertion at the nuchal line.

Lesser Occipital Nerve^{21,12,15}

The lesser occipital nerve is the ventral ramus of C2 and sometimes C3 supplying sensory innervation to the superior, postauricular

skin, and lateral neck. The nerve emerges from beneath the sternocleidomastoid muscle (SCM) about 6-7cm lateral to midline, at the middle portion of the muscle, superior to the emergence of the great auricular nerve. The nerve then runs parallel to the SCM before penetrating the subcutaneous plane at a variable location (Figure 4).

Supraorbital Nerve¹⁶⁻²⁰

The supraorbital nerve is a branch of the frontal nerve, which originates from the ophthalmic branch of the trigeminal nerve supplying sensation to the frontoparietal scalp. The nerve exits the bony skull via the supraorbital notch/foramen along the superior orbital rim around 3 cm lateral to midline, corresponding to the medial limbus of the eye topographically. Beyond the supraorbital foramen, the nerve most commonly forms superficial (medial) and deep (lateral) divisions. The superficial division divides into smaller branches that pass cephalad over the frontalis muscle to innervate the scalp in a fanlike pattern. The deep division typically travels between the periosteum and the galea running

superolateral toward the temporal fusion line then running parallel and 0.5cm medial to this fusion line prior to dividing into smaller branches to innervate the scalp.

Supratrochlear Nerve^{21,22}

The supratrochlear nerve is a terminal branch of the frontal nerve, which is a branch of the trigeminal nerve ophthalmic division (V1) that passes through the superior orbital fissure on its way to supply sensation to the skin and soft tissues of the glabella, lower forehead, and upper eyelid. The nerve runs along the medial roof of the orbit exiting the orbit via the frontal notch around 1.5cm lateral to midline, and always medial to the supraorbital neurovascular bundle. After this, the nerve splits into two branches before entering the corrugator muscle. It then travels intramuscularly before exiting the muscle about 1.5cm superior to the orbital rim and then continuing superficially to supply sensation to the skin.

Dorsal Scapular Nerve^{23,24}

The dorsal scapular nerve arises from the ventral ramus of C5 within the posterior

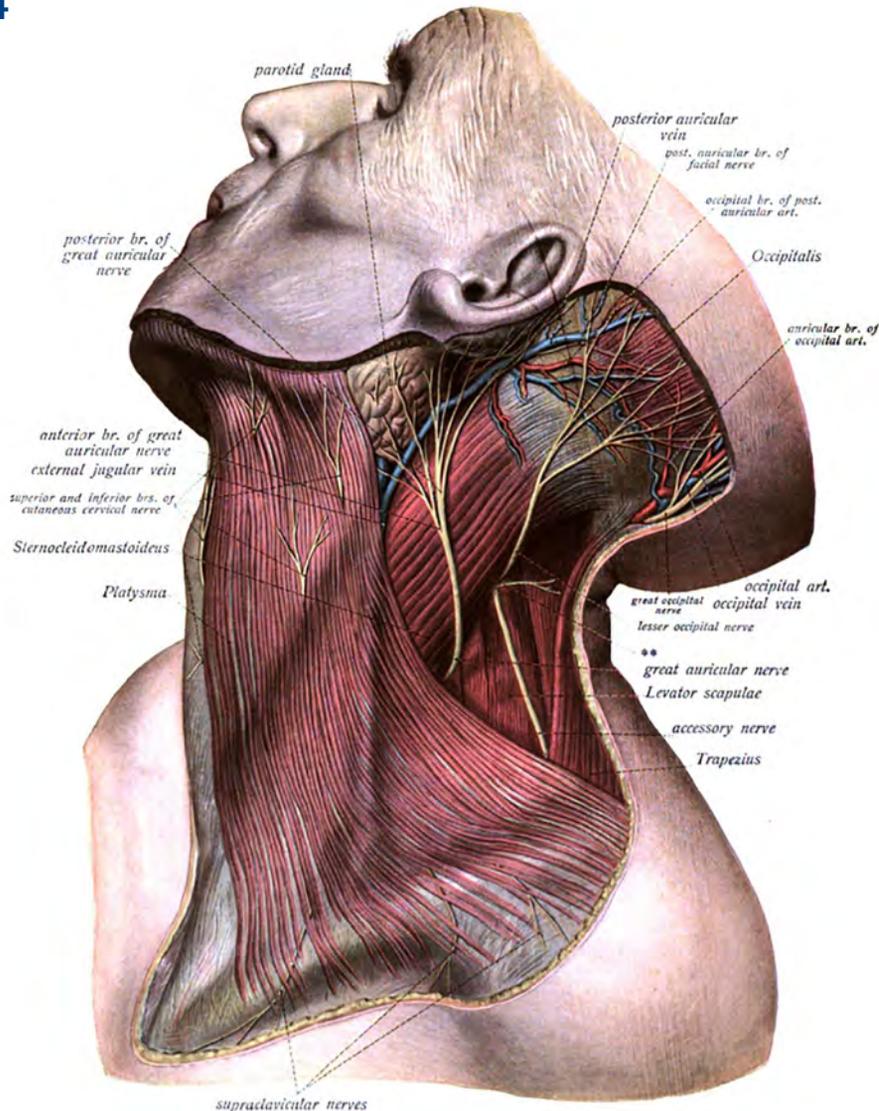


Figure 4: Lateral neck nerve anatomy “Sobotta 1909 fig.697 - Nerves and vessels of the neck, first superficial layer - English labels” at AnatomyTOOL.org by Johannes Sobotta is in the Public Domain.

cervical triangle. It then proceeds deep to the prevertebral fascia and pierces the middle scalene muscle traveling between the posterior scalene muscle and the serratus posterior superior and levator scapulae muscles to innervate the rhomboid muscles. It passes under the levator scapulae muscle and then becomes superficial between the rhomboid major and minor muscles. It then travels caudally along the medial border of the scapula.

Suprascapular Nerve

The suprascapular nerve originates from the upper trunk of the brachial plexus with contributions from C5 and C6. It travels

with the brachial plexus before proceeding posteriorly to the superior border of the scapula and then passes through the suprascapular notch. The nerve courses caudally until it reaches the scapular spine, where it passes through the spinoglenoid notch and under the spinoglenoid ligament. The suprascapular nerve innervates the supra- and infraspinatus (Figure 5).

CLINICAL PRESENTATION

Greater and lesser occipital nerves^{5,25}

Greater occipital nerves can be injured from direct blows to the back of the head or be stretched from a whiplash injury. These patients typically present with pain

localized to the sub-occipital and occipital areas. Patients typically have tenderness where the nerve transitions superficially (Figure 2). Sometimes, this pain can radiate frontally to trigeminal-innervated areas of the head due to connections between the extracranial occipital nerves and the intracranial trigeminal nerves. When frontal radiation is present, photophobia and nausea can often accompany the localized posterior pain.

This injury can derail an athlete’s career as was seen in the case of Briana Scurry. She was an elite football goalie and Olympic gold medalist who had a concussion during play and continued to have severe pain in her suboccipital region after her initial trauma. Ultimately, she retired from her sport before she was diagnosed with greater occipital nerve injury and underwent successful nerve decompression surgery (<https://www.brainandlife.org/articles/olympic-soccer-goalie-briana-scurry-brain-injury>).

Lesser occipital nerves present with pain just posterior to the sternocleidomastoid and the pain can radiate behind the ear. Palpation is very helpful for this diagnosis with the nerve localized beneath the area. There can be more than one branch of the lesser occipital nerve and surgeons should look systematically in the area to ensure full release.

Supraorbital/supratrochlear nerves²⁶⁻²⁸

Patients with supraorbital and supratrochlear neuralgia typically present with localized headache or pain in or above the eyebrow with possible extension up into the scalp above the nerve. Pain related to compression of these nerves is often reproducible by compression over the supraorbital notch or the area directly overlying the nerve with associated tenderness. This injury can be associated with compressive glasses and goggles. It should be considered when treating a patient with “swimmer’s migraine”²⁹.

Dorsal scapular nerve³⁰

Patients with compression of the dorsal scapular nerve often report pain just medial to the scapula. Patients can feel tightness and weakness along the levator scapulae, rhomboid major, rhomboid minor, particularly with scapular retraction. Patients may present with winging of the scapula. Interestingly, patients can

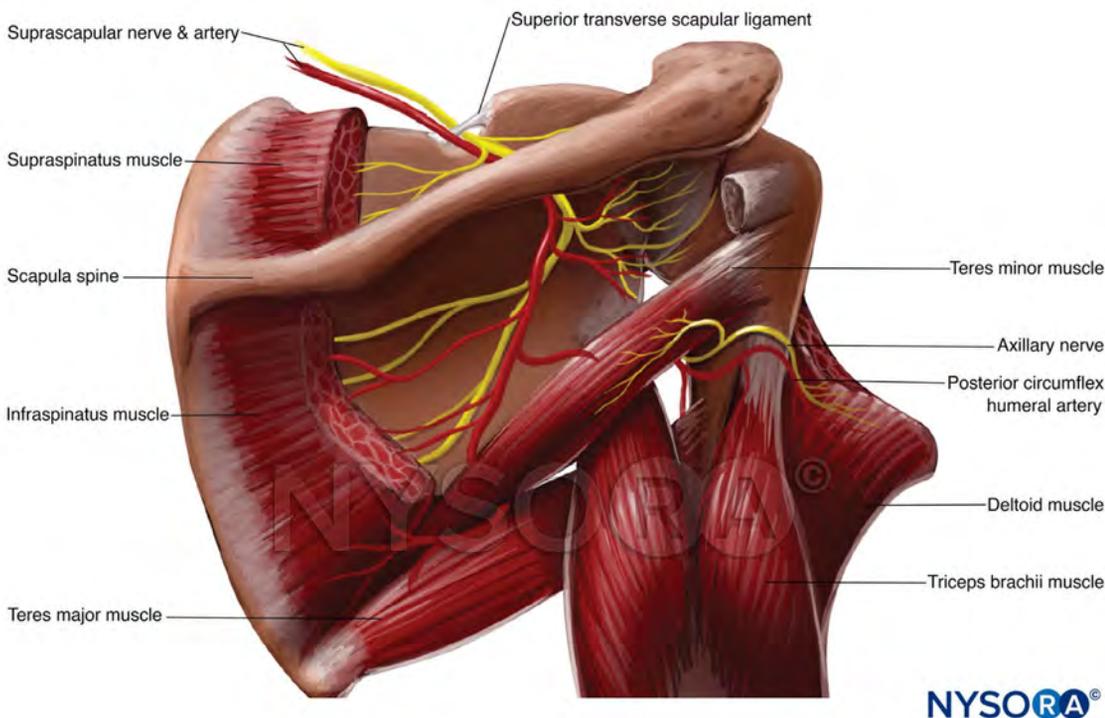


Figure 5: “NYSORA - Drawing Posterior view of shoulder - English labels” at AnatomyTOOL.org by New York School of Regional Anesthesia and VisionExpo.Design, license: Creative Commons Attribution-NonCommercial-NoDerivs.

also have pruritus of the midscapular region along with radiation of pain along the posterolateral shoulder, arm, and forearm. This nerve can be injured in athletes with repetitive overhead movements such as volleyball, tennis, or baseball but has also been seen in other sports such as boxing and following whiplash injuries.

Suprascapular nerve

Patients with suprascapular nerve compression will complain of shoulder pain and often a feeling of weakness. On physical examination, there can be weakness in shoulder abduction and external rotation. There can also be isolated atrophy of the infraspinatus with entrapment at the spinoglenoid notch. The suprascapular is a common nerve compression in athletes. It is frequently seen in overhead athletes such as volleyball, tennis, and baseball pitchers. This entrapment can be amenable to arthroscopic release making for an easy return to play³¹.

Surgery Outcomes

Surgical decompression was once thought to be a placebo treatment. As evidenced by recent studies, this is far from the case. Evans et al³² published a meta-analysis reviewing the outcomes of the surgical treatment of migraines. The 18 studies reviewed demonstrated a reduction in

migraine headache severity and intensity. Similarly, ElHawary et al³³ published a meta-analysis reviewing nerve decompression for the same indication and found a reduction in severity, intensity, and duration. Furthermore, they were also able to analyze the safety profile of the procedure with the most common complication being transient paresthesia and numbness. Other complications reported included itching, hyposensitivity, hair loss, and seroma. For the patient with the correct diagnosis, surgery to address the compressed nerve is safe, effective, and has low morbidity.

CONCLUSIONS

Neuropathic pain is becoming an increasingly recognized etiology for head and neck pain, especially following sports injuries. Furthermore, surgical neurolysis can provide sustained resolution of symptoms when recognized. This article provides a detailed review of relevant anatomy and clinical presentation highlighting treatment options for patients presenting with common nerve compressions as an etiology for head and neck pain. The first step to recovery is recognition.

References

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