Nerve Entrapment Headaches at the Temple: Zygomaticotemporal and/or Auriculotemporal Nerve?

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Background: Temple headaches are common, yet the anatomic etiology of headaches in this region is often confusing. One possible cause of temple headaches is dysfunction of the auriculotemporal nerve (ATN), a branch of the third division of the trigeminal nerve. However, the site of pain is often anterior to the described path of the ATN, and corresponds more closely to a portion of the path of a small branch of the second division of the trigeminal nerve called the zygomaticotemporal nerve (ZTN).

Objectives: We present the anatomic and clinical differences between these 2 nerves and describe treatment approaches. Diagnosis is made by physical examination of the temporal fossa and the temporomandibular joint, and injection of local anesthetic over the tenderest nerve.

Results: In general, treatments of headaches that generated from the peripheral nerve attempt to neutralize the pain origin using surgical or interventional pain techniques to reduce nerve irritation and subsequently deactivate stimulated migraine centers.

Conclusions: Treatment of temporal nerve entrapment includes medications, nerve injections, dental appliances, cryoneuroablation, chemical neurolysis, neuromodulation, and surgical decompression.

Key Words: Headache, migraine, trigeminal nerve, Frey’s syndrome, zygomaticotemporal nerve, auriculotemporal nerve, temple pain, jaw pain, ear pain, tooth pain

Headaches are one of the most prevalent neurologic disorders. They affect about half of all adults worldwide at least once a year, occur more than 2 weeks per month in 1.7–4%, and are the third leading cause of years lost to disability (1). Headache patterns have provided practitioners and researchers with basic guidelines to understand and treat these debilitating conditions (2). Many now propose that headaches, including some migraines, are not always isolated intracranial phenomena, but rather the result of interactions between the intracranial components of the brain and the extracranial nerves. At the beginning of this century, plastic surgeons noted relief of migraines with corrugator muscle resection (3) or injection of botulinum toxin, suggesting a peripheral headache trigger. Headache specialists see
Surgical approaches to treatment of headaches in the temple region not manageable with non-operative approaches have been described (14,19,20) and are increasingly accepted (19,21). Of these, most reports focus on ZTN entrapment; indeed, this nerve has been classified as one of several major migraine trigger sites (Fig.1 site II) (21). Although the ATN had been considered a relatively minor migraine trigger site (Fig. 1 site V) (21), extensive anatomic review of its peripheral course has increased the understanding of its potential contributions to migraine pain in this area (16), including documented persistent ATN pathology after treatment of ZTN entrapment (22). Other authors have commented on the interdependence between the 2 nerves (23).

Distinguishing between dysfunction of the ZTN or the ATN as the source of any particular problem can be difficult because of the complex and variable anatomy of the area between where the nerves emerge from the skull, including confusion regarding the anatomic layers of the temporoparietal region (Fig.2), and their termination in the skin of the temple (24). The purpose of this paper is to provide a direct comparison of what is known of the anatomy of each nerve (including their common anatomical variations), and review some methods to diagnose and treat painful conditions that result when irritation of one or both nerves contributes to headaches in this area.

Fig. 1. Guyuron’s major and minor migraine headache trigger sites. Site I = supraorbital/supratrochlear nerves; site II = temporal zone; site III = rhinogenic trigger zone; site IV = lessor occipital nerve; site V = auriculotemporal nerve; site VI = greater and/or third occipital nerve. (21) (Image courtesy of Andrea Trescot, MD)
Nerve Entrapment Headaches at the Temple

ANATOMY

The temporal fossa is a shallow depression on the outside of the skull between the superior temporal line and the zygomatic arch. Its medial wall is formed by 4 bones: the frontal and parietal bones superiorly and the greater wing of the sphenoid and the squamous portion of the temporal inferiorly (Fig. 3). The H-shaped site where the sutures between these bones meet is called the pterion. The infratemporal fossa is the region medial and inferior to the zygoma. It is almost entirely filled with at least parts of the 4 muscles of mastication (the lateral pterygoid, medial pterygoid, masseter and temporalis muscles). The pterygopalatine fossa is medial to and continuous with the infratemporal fossa.

The trigeminal nerve (the 5th cranial nerve, often designated by its Roman numeral, V) is the sensory nerve of the head (25). As part of the trigeminovascular system, it is increasingly implicated in the genesis of migraine headaches (7-10). The trigeminal nerve consists mostly of somatic afferent (sensory) fibers with some special visceral efferent (motor) fibers in its mandibular (V3) division. Its spinal nucleus (the trigeminal ganglion) is the source of pain, temperature, and touch in all 3 divisions. The trigeminal nerve emerges from the pons at the level of the 4th ventricle and soon splits into its 3 somatic divisions, each of which leaves the middle cranial fossa via a different passage. The ophthalmic division (V1) enters the orbit through the superior orbital fissure, the maxillary division (V2) enters the pterygopalatine fossa via the foramen rotundum, and the mandibular division (V3) goes into the infratemporal fossa (lateral to and continuous with the pterygopalatine fossa) by way of the foramen ovale. Two peripheral branches of the trigeminal nerve, one from each of its lower 2 divisions (the ZTN from V2 and the ATN from V3), terminate in the soft tissues over the temporoparietal region and are responsible for similar headache symptoms. The anatomy of the soft tissue layers in this region is complex, and historically

Fig. 2. The anatomic layers of the temporoparietal region. The temporoparietal fascia is often called the superficial temporal fascia; the superficial temporal artery (STA) lies within this plane. The temporal fascia covers the temporalis muscle and fuses superiorly with the periorbita of the frontal and parietal bones at the superior temporal line, and inferiorly with the periostium of the zygomatic arch. At the level of the superior orbital margin, the superficial and deep layers of the temporal fascia split to surround the fat pad of the temporal fascia; the zygomaticotemporal nerve crosses this fat pad. From Davidge KM, van Furth WR, Agur A, Cusimano M. Naming the soft tissue layers of the temporoparietal region: unifying anatomic terminology across surgical disciplines. Neurosurgery. 2010;67:S120-129. With permission
inconsistent nomenclature has been used to identify them. Where possible, the naming system proposed by Davidge et al (24) will be used in this report (Fig. 2).

**Anatomy of the Zygomaticotemporal Nerve**

The ZTN is a major terminal branch of the maxillary (second, V2) division of the trigeminal nerve; it crosses the orbit, a canal in the zygomatic bone and the temporalis muscle to end in the skin of the anterior temple near the lateral eyebrow (Table 1).

The zygomatic nerve (ZN) separates from the maxillary nerve in the pterygopalatine fossa, enters the floor of the lateral orbit through the infraorbital fissure, and divides into the ZTN and zygomaticofacial (ZFN) nerves (Fig. 4). The ZTN is joined by a communicating branch from the lacrimal nerve; it then may cross a small opening in the frontal process of the zygomatic bone called the zygomaticotemporal foramen (ZTF) or pass directly behind the lateral rim of the orbit. The ZTN enters the temporal fossa approximately 1 cm below the frontozygomatic suture (FZ suture) (26). Unlike the ZFN and many other nerves, the ZTN is not associated with a satellite artery (27). Hwang and colleagues (26)

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**Table 1. Anatomy and entrapment.**

<table>
<thead>
<tr>
<th>Anatomy</th>
<th>ZTN</th>
<th>ATN</th>
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<tbody>
<tr>
<td>Origin</td>
<td>Maxillary division of the trigeminal nerve (V2) exits the middle cranial fossa through the foramen rotundum.</td>
<td>Mandibular division of the trigeminal nerve (V3) (51)</td>
</tr>
<tr>
<td>General route</td>
<td>The zygomatic nerve (ZN) separates from the maxillary nerve in the pterygopalatine fossa (continuous laterally with the infratemporal fossa), enters the lateral orbit through the infraorbital fissure where it divides into the zygomaticofacial (ZFN) and zygomaticotemporal (ZTN) nerves. The ZTN enters the temporal fossa as a single trunk via a canal in the frontal process of the zygomatic bone, and runs between the bone and the temporalis muscle. Approximately 2 cm above the zygomatic arch, the ZTN pierces the temporal fascia to end in the skin over the anterior temple. Branches may communicate with the lacrimal nerve, the auriculotemporal nerve (ATN) and/or the facial nerve.</td>
<td>In the infratemporal fossa (continuous medially with the pterygopalatine fossa), the ATN surrounds the middle meningeal artery. Its 6 named branches separate within the parotid gland at the posterior border of the mandible just below the external auditory meatus. The main branch (also called the superficial temporal ramus) runs very close to the medial capsule of the temporomandibular joint (TMJ) (46) then loops below the posterior part of the zygomatic arch and becomes progressively superficial. It travels within the temporoparietal fascia near (and directly related to in 34% of 50 cadaver hemiheads) the superficial temporal artery (STA) to terminate in the subcutaneous tissue of the posterior temple. (16) Smaller branches communicate with the facial nerve, and innervate the external auditory meatus and auricle (51).</td>
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<tr>
<td>Sensory distribution</td>
<td>Skin over the anterior temple</td>
<td>TMJ (also supplied by the deep temporal nerve and the masseteric nerve), parotid gland, external auditory canal, tympanic membrane, superior auricle and the skin over the temple posterior to that of the ZTN</td>
</tr>
<tr>
<td>Motor innervation</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>Autonomic innervation</td>
<td>Post-ganglionic parasympathetic fibers: from the superior salivatory nucleus, leave the facial nerve as part of the greater petrosal nerve, synapse in the pterygopalatine ganglion, travel as part of the zygomatic nerve and terminate in the lacrimal gland to increase tear production (12,27).</td>
<td>Post-ganglionic parasympathetic fibers: from the inferior salivatory nucleus, leave the glossopharyngeal nerve as part of the tympanic nerve, synapse in the otic ganglion, travel as part of the auriculotemporal nerve and terminate in the parotid gland to increase saliva production (16). Sympathetic innervation of the scalp and face</td>
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define the “vulnerable point” (Vp), where the ZTN curves around the edge of the zygoma, as a place at which it is vulnerable to damage during surgical procedures (Fig. 5). Once it reaches the soft tissues, the ZTN runs between the bone and the temporalis muscle, then through the muscle until it pierces the temporal fascia (another potentially vulnerable point). It then divides into its terminal branches (27,28) and ends in the skin, forming a plexus over the anterior temple below the superior temporal line (Fig. 6) (12,26,27). A superficial branch may communicate with the ATN (12).

Highlighted anatomic variations in the pathway of the ZTN (from central to peripheral) include differences in its relationship to the inferior orbital fissure (26), the location of the ZT foramen (13,29-32), the variability of branching and connections to the ATN, lacrimal and facial nerves (12,13,27,28), patterns of its course within the temporalis muscle (13) and the site at which it pierces the temporal fascia (12,13,27,28).

**Orbital Fissure:**

Hwang and colleagues (26) dissected 20 hemifaces from Korean adults, looking at the path of the ZTN. They found that in 30% of cases, the ZTN did not cross the inferior orbital fissure but rather passed posterior to the greater wing of the sphenoid. The authors did not consider this of particular clinical significance, but emphasized the importance of the FZ suture (they call it the zygomaticofrontal suture) as a landmark. Other authors (33) stressed the importance of precise orbital location of the ZN and its branches in maxillofacial surgery, especially during treatment of zygomaticomaxillary fractures.

**Zygomaticotemporal Foramen**

In a study of 200 dry skulls (400 sides from adult cadavers dissected at St. George’s University in Grenada, West Indies, and at Harvard Medical School in Massachusetts), Loukas et al...
reported that 30% of the samples had one ZT foramen, 20% had 2 or 3, and in 50% it was absent. They noted a large, possibly race-based, difference between their findings and those of previous investigators.

Other authors, in the report of a study of fresh tissue cadavers, describe the ZT foramen as posterolateral to the edge of the lateral orbital rim rather than on its surface. Discussion of the foramina in this area is complicated by the convention of labeling these foramina as zygomatico-orbital (ZO) when viewed from the inside of a skull, but as ZT or ZF when viewed from the outside; and by the fact that this convention is not consistently followed. In dry skull studies of ZF foramina that do not specifically refer to the ZT foramen, the authors may be using “ZF” to refer to both ZF and ZT foramina. In some cases, there are a different number of foramina on the inside and on the outside of the skull, indicating that nerve division has occurred within the bone during development. In general, the reports are consistent in the conclusion that zygomatic foramina are not reliable surgical landmarks due to their highly variable frequencies and locations.
Branching of the ZTN and its connections with other nerves are also variable. One study (27) reported that the ZTN was always (24/24) a single trunk with 1 to 3 terminal branches, while (12,13,28) in others a single trunk was found in less than half of the specimens. This difference is likely due to differences in technique (cadaver or endoscopic studies) or definitions as to the point at which branching begins. More importantly, the presence of multiple branches to the point that the nerve was described as “arborized” in the subcutaneous tissues relatively close to the ZT foramen in a substantial number of specimens in one report (12/50) makes it difficult to make precise statements as to its course and connections (13). Horizontal links to the ATN (3/24 or 13%) and/or lacrimal nerves (2/24 or 8.7%) were detailed in one cadaver study, (27) and described in an investigation of patients undergoing endoscopic forehead surgery (12). Facial nerve connections were not seen in one study, (27) but are mentioned in others (36-38).

Temporalis Muscle

The relationship of the ZTN to the temporalis muscle is likewise variable, though specifically investigated in only 1 study. The authors report that in 22% of the specimens (11/50), the ZTN had a short intramuscular course and in 28% of the specimens (14/50), it had a tortuous intramuscular path. In the other half of the specimens, after its exit from the ZT foramen, the ZTN passed between the bone and periosteum, and then pierced the periosteum directly into the fat pad of the temporal fascia without entering the muscle (13).

Temporalis Fascia

The place at which the ZTN pierces the deep temporal fascia is sited by one group as on average 2.2 cm (1.5 - 2.7 cm) above the superior border of the zygomatic arch and 1cm (0.7 - 1.3 cm) posterior to the FZ suture (28), and by another as 1.7 cm (1.2 – 3.1 cm) lateral to and 0.6 cm (0.4 – 1.1 cm) cephalad to the lateral palpebral commissure (often called the lateral canthus) (12). In both of these reports, “deep temporal fascia” probably refers to the superficial layer of the temporal fascia (24). The authors describe a palpable “hollow area” at this point (12). A third group measured the intersection between the nerve and the superficial temporal fascia as 2.3 cm (1.9 - 2.6 cm) above the superior border of the zygomatic arch, at the level of the pterion (27). Given that in neurosurgery the term “superficial fascia” usually refers to the superficial layer of the temporal fascia (24), all 3 groups are likely talking about the same point.

Entrapment of the Zygomaticotemporal Nerve

Few papers specifically report isolated ZTN entrapment (39). It has been described as vulnerable to injury in the orbit (29,33), at the point where it crosses or curves around the edge of the zygoma, (26) and more peripherally where it could be entrapped in a scar after repair of a zygomatic fracture (26,29), plastic surgery in the area (26) or craniotomy (40-43). Most commonly, ZTN entrapment is discussed in the context of migraine headaches centered on the temple (12,13), and has been designated Guyuron’s major migraine trigger site II (Fig. 1) (21,23). Its course through and near the temporalis muscle and its fascia makes the ZTN vulnerable to temporalis hypertrophy due to bruxism or the tissue edema resulting from the hormonal changes of pregnancy or menstruation (6,44,45).

Anatomy of the Auriculotemporal Nerve

The ATN is a major terminal branch of the mandibular (V3) division of the trigeminal nerve. It has close association with 2 arteries (the middle meningeal and the superficial temporal arteries), the major muscles of mastication, and the temporomandibular joint (TMJ) (46-48). It ends in the skin over the high posterior temple (Fig. 5) (15,16).

The mandibular division of the trigeminal nerve (V3) crosses from the skull via the foramen ovale to the soft tissues of the infratemporal fossa, deep (medial) to the lateral pterygoid muscle (LPM) (49,50). It immediately splits into a posterior division (ATN, inferior alveolar and lingual nerves) and an anterior division (buccal, deep temporal, and masseteric nerves). The 2 roots of the ATN leave the posterior aspect of the mandibular nerve, just distal to where it divides (Fig. 7). These roots surround the middle meningeal artery, and then converge within the upper part of the parotid gland to form a short (~6 mm) trunk from which 6 named branches (the superficial temporal ramus, 2 nerves to the external acoustic meatus, the anterior auricular nerve, and 2 communicating branches to the facial nerve) separate almost immediately (51). This separation occurs at the posterior medial border of the mandibular condyle just below the external auditory meatus. The main branch (the superficial temporal ramus) runs laterally, very close to the medial capsule of the temporomandibular
The auriculotemporal nerve (ATN) arises from the temporal bone and supplies sensory fibers to the joint (TMJ) and gives off some sensory shoots to the joint (46,48,52-54). Multiple small branches communicate with the facial nerve; together they are known as the central communicating branches to the facial nerve (CATNs). Taken together, the cross-sectional area of the CATNs is equal to or greater than that of the superficial
temporal ramus (51,55). Following the most common custom, we will call this superficial temporal ramus the ATN. The ATN then loops below the posterior part of the zygomatic arch and becomes progressively more superficial. It travels within the temporoparietal fascia near (and is often directly related to) the superficial temporal artery (STA) 0.8 – 2 cm anterior to the upper origin (root) of the ear helix (56) to terminate in the subcutaneous tissue of the temple (Fig. 5), roughly at the site of tenderness for many patients with pain in the temple (Fig. 1, site V) (16). Superficial branches of the ATN sometimes communicate with the ZTN, (12) the facial nerve, the lesser occipital nerve, and the greater occipital nerve (56).

The ATN carries sensory information from the TMJ (which is also innervated by the deep temporal nerve and the masseteric nerve), the parotid gland, the external acoustic canal, the tympanic membrane, the superior auricle, and the skin over the posterior superior temple. It has no motor function, but it does carry the post-ganglionic parasympathetic fibers that terminate in the parotid gland to increase saliva production (57). These parasympathetic fibers originate in the inferior salivatory nucleus, leave the glossopharyngeal nerve as part of the tympanic nerve, synapse in the otic ganglion, and join the ATN. In addition, the ATN provides sympathetic innervation to the scalp and face: post-ganglionic fibers from the superior cervical ganglion travel to the parotid and sweat glands and cutaneous blood vessels via the ATN and its branches (16).

Reported anatomic variations of note in the course of the ATN (from central to peripheral) are the number of roots (48,51), the relationships to the middle meningeal artery (51,54), the communication with the inferior alveolar nerve (IAN) (51,58,59), the association with other structures in the infratemporal fossa (57,60), the relationship to the TMJ (46,52), the communications with the facial nerve (38,51,54-56,61-63), as well as the incidence and type of interactions with the STA (15,16). Additional confusion results from differences in naming conventions. For instance, some groups might classify a particular structure as a root (48,59,64), while others call it a branch (65,66).

**Roots**

The classic description is that the ATN is formed by 2 roots from the posterior aspect of the mandibular nerve (51). However, variable patterns from 1 to 5 roots have been reported, with 2 roots seen in 31-72% of the dissections (48,54,59,67,68). Anatomic variations in the relationship of the ATN to the middle meningeal artery are closely related to the number and pattern of its roots (51,54). The ATN has been reported to have connections to the IAN in 10-30% (48,58,59). The intricate root system of the ATN can provide explanations for unexpected symptom patterns (58,59,69) or responses to IAN block (70).

**Muscles of Mastication**

The infratemporal fossa is almost entirely filled with at least parts of the 4 muscles of mastication (the LPM, medial pterygoid muscle, masseter muscle and temporalis muscle), so hypertrophy of these muscles can compress the ATN (48). Of these, the LPM is of particular interest because of its close proximity to the mandibular nerve and the ATN. The LPM (sometimes called the external pterygoid muscle) has superior and inferior parts and assists in opening the mouth (46,57,66,71,72). It runs between the greater wing of the sphenoid bone and the articular disc of the TMJ (superior part) and between the pterygoid process of the sphenoid and the mandibular condyle (inferior part). The posterior division of the mandibular nerve, (57,69,72) the lingual nerve (69) as well as ATN “roots” (48) and “branches” (66) are very close to and sometimes (on average about 5% of the time) pierce the LPM.

The pterygoalar ligament is another occupant of the crowded infratemporal fossa. It is a band of connective tissue between the lateral pterygoid lamina and the greater wing of the sphenoid bone, lateral to or directly below the foramen ovale. With age, this ligament can ossify into a partial or complete bar (in 2 - 32%, depending on the population examined) and potentially impinge on the mandibular nerve and/or the ATN (60).

**Temporomandibular Joint**

The TMJ is very mobile, with movement in many directions, and is a common source of pain. The ATN runs just adjacent to the medial surface of the TMJ capsule (46,48,52,53). Variable reports exist about the distance between the ATN and the joint, an important measurement with respect to the possibility of ATN injury due to TMJ dysfunction or inflammation. The nerve has been measured as 0 – 1 cm horizontal to the neck of the mandibular condyle and 0 – 1.3 cm below the superior border of its articular surface (46,54). Of particular interest is the work of Johansson et al (52) who measured the relative positions of these structures with the jaw in different positions. They found that the
ATN was at or below the attachment of the disc capsule when the jaw was in a neutral position, but moved more superiorly when the mouth was opened. Its position was also changed by displacement of the articular disc. Some additional variability in measurements of the distances between the ATN and the TMJ may be the result of small sample sizes (16 to 40 sides) and of differences in cadaver preparation and dissection approach. Some researchers used formalin-embalmed cadavers (46,53,54), while others used frozen ones (52). The infratemporal fossa is difficult to access; some investigators approached it from above by removing the base of the skull (53,54) and some approached from the side after detaching the zygomatic arch (46). One group removed specimens en bloc, refroze them in different jaw positions, then sliced, stained and reconstructed the nerve paths from the slices (52).

**Facial Nerve**

Communications with the facial nerve are reliably present, though with a variable pattern (38,51,54-56,61-63) and are the most consistent of the connections between the facial nerve and branches of the trigeminal nerve (51,55,63). The facial nerve fibers to the muscles of facial expression leave the skull via the stylomastoid foramen (substantially posterior and lateral to the foramen ovale), travel anteriorly through the parotid gland, and then divide into the temporal, zygomatic, buccal, marginal mandibular, and cervical branches to innervate the superficial muscles of the face and upper neck. Deep in the parotid gland, 1 to 3 or more CATNs can be found (51,61,62). These branches of the ATN pass forward from behind the neck of the mandible and join the temporal branches of the facial nerve at the posterior border of the masseter muscle. CATNs may supply proprioceptive input to the muscles of facial expression (55,62,63), and could provide a pathway for re-establishment of motor function after facial nerve injury (55,62). ATN dysfunction may lead to pain in the upper muscles of facial expression (57,63). These connections may also provide a route for perineural tumor spread (73,74).

The ATN (sometimes called the superficial temporal ramus of the ATN (51), see above) and the other terminal branches are closely associated with branches of the maxillary artery (51). The ATN usually splits into anterior and posterior divisions in front of the ear; these further ramify to supply the skin of the temple.

Superficial temporal artery: The STA is a branch of the external carotid artery closely associated with the temporoparietal fascia (the most superficial fascial layer) (Fig. 2) (24). Its pulse is felt just anterior to the external auditory meatus. The STA has been cited as a potential source of ATN entrapment (15) as well as a way to find the temporal branch of the facial nerve just anterior to the ear (24). The ATN has a complex relationship with the STA: the STA was found to have a direct interaction with the ATN in 34% (16) to 80% (15) of individuals. Some of these interactions are single crossovers of the artery over the nerve (10/16 = 62%), some are helical spirals (3/16 = 19%), and in some there was no interaction (4/20 = 20%) (15). The most proximal crossover was found 10.8 ± 1.8 cm lateral to the midline and 3.8 ± 1.5 cm above the continuation of the horizontal line between the nasion and the lateral orbital rim (16).

**Entrapment of the Auriculotemporal Nerve**

The ATN can be entrapped at 2 distinct anatomic locations: in the infratemporal fossa and, perhaps more commonly, in the soft tissues of the temporal region (6). The infratemporal fossa is a complex area with relatively tight boundaries, so hypertrophy or spasm of the muscles of mastication (which in some cases may be caused by TMJ dysfunction) (69) can compress the ATN (48,57). The ATN is at particular risk for compression by the LPM: the posterior trunk of the mandibular nerve (including the ATN, IAN and lingual nerves) (57,69) or the ATN itself (48,66) passed through the body of the LPM in about 5% of cases. This sets up the possibility of a “double crush” phenomenon in which proximal compression of a nerve, such as from LPM spasm, intensifies symptoms of injury to that nerve at a more distal site (16,75). Entrapment in the infratemporal fossa could explain persistent pain after TMJ repair, pain in the muscles of facial expression (57) and, depending on the portion of the posterior division of the mandibular nerve involved, sensory changes in the ear, tongue, chin, and/or lower lip (46,48,57,58) and/or impaired salivation (48). The medial capsule of the TMJ is also located in the infratemporal fossa. Several authors (46,48,76) have argued that the ATN could be injured at or near the TMJ by joint dysfunction, inflammation or disc protrusion, although other authors disagree (53,54). Johansson (52) emphasized the possibility of entrapment medial as well as posterior to the mandibular condyle.

More distally in the soft tissues of the high temple, ATN dysfunction is considered to be a minor migraine trigger site (Fig. 1, site V) (21). It can be entrapped by...
the superficial temporal artery (15,16,77) (Fig. 5), and/or by 1 or 2 preauricular fascial bands (15). Chim et al (15) dissected 20 temples and identified 2 consistent fascial bands in the preauricular area: the inferior band (13.1 ± 5.9 mm anterior and 5.0 ± 7.0 mm superior to the external auditory meatus) was present in all of the dissections, while the superior one (11.9 ± 6.0 mm anterior and 17.2 ± 10.4 mm superior to the external auditory meatus) was present in 85% (17 of 20) of the specimens.

**Signs, Symptoms and History of Zygomaticotemporal Nerve Dysfunction**

Entrapment and other injuries of the ZTN have been reported to cause diffused pain over the zygomatic arch and anterior temple near the lateral canthus (18,39), as well as post-craniotomy (27) or migraine (12-14,18,22) headaches. ZTN dysfunction-associated symptoms are usually the result of accidental or surgical maxillofacial trauma and of orbital disorders (26,33,39,43).

**Signs, Symptoms and History of Auriculotemporal Nerve Dysfunction**

The anatomic relationships between the ATN and the muscles of mastication, the TMJ, and the surrounding vascular structures set up the potential for entrapment, so that ATN dysfunction can play a role in headaches as well as face, ear and TMJ pain. In addition to “high temple” headaches, entrapment or injury of the ATN can cause several distinct symptom constellations: auriculotemporal neuralgia (ATN) (47,73,78-84), as well as the less common auriculotemporal syndrome (ATS, also called gustatory sweating, or Frey’s syndrome), which is often due to aberrant regeneration of nerves injured during parotid surgery (16,85-88). It may also cause TMJ neuralgia (Costen’s syndrome) (46,48,52,76,89,90).

ATN is a syndrome of severe, often episodic, unilateral facial pain in the temple, ear, TMJ, preauricular and parotid areas. Occurrences can be triggered by pressure over the ATN and resolved with a local anesthetic block (47,78,79,81-84). This disorder may be the result of undiagnosed compression of the nerve (15) and can be treated with the injection, neurolytic, and/or surgical techniques described below.

Patients with ATS complain of sweating, flushing, and/or sensory disturbances, including pain in the ATN distribution with eating sour or spicy foods that produce strong salivary stimuli (86,88). It is commonly accepted that these symptoms are due to the presence of both sympathetic (sweating) and parasympathetic (salivation) fibers in the ATN. When the nerve regenerates after being injured by surgery or infection, the parasympathetic fibers can mistakenly re-innervate sweat glands in the skin (16,85-88). ATS can be diagnosed with the Minor’s starch-iodine test (86,88). In one study of 372 patients who had undergone unilateral parotidectomy, almost 25% of them developed ATS (91). Less than half of them were symptomatic, but those who were symptomatic considered it an unpleasant complication.

There are many causes of TMJ pain, but TMJ neuralgia (Costen’s syndrome) should be considered as part of the differential diagnosis. The symptom constellation includes TMJ dysfunction associated with headaches in the temple region, ear pain, a “stuffy deaf” sensation, tinnitus, dizziness, headache, and a burning sensation of the throat, tongue, and side of the nose (76,89,90). These symptoms were originally believed to be the result of either TMJ pathology or a lack of posterior teeth leading to a “mandibular over-closure” and ATN injury (see above) (76,89). More recent authors (46,48,52,92) have demonstrated that the ATN is often close enough to the TMJ that it could be damaged by THJ joint or disc dysfunction.

**Physical Examination**

The physical exam should be performed systematically to evaluate each possible area of pathology. Following each nerve from proximal to distal is one way to be sure that nothing is missed. Calandre et al (11) listed 2 separate areas of the temporalis muscle to explore during physical examination (“proximal/anterior” and “medium”) fibers. Although the authors do not provide the data underlying their “typical” versus “atypical” division, they describe the anterior fibers as one of the “typical” trigger point sites, leaving the “medium” fibers to the “atypical” group. The “proximal/anterior fibers” are most likely the site where the ZTN crosses the temporalis muscle (Fig. 1 site II) and the “medium fibers” are probably near the ATN (Fig.1 site V).

Examination of the ZTN is relatively straightforward. Find the “hollow point” (HP) (also called the “hollow area”) in the temple, about 2 cm lateral to and 1 cm above the lateral palpebral commissure, where the ZTN passes through the temporal fascia (12). Tenderness at this site indicates the presence of ZTN irritation.

Since the LPM may be involved in ATN entrapment, it should be examined by palpation (46) [though there is some question as to this technique (93)] and by a provocative test (57), pressing on the chin and ask the
patient to protrude the mandible. This maneuver activates the lower portion of the LPM and would increase symptoms if the posterior trunk of the mandibular nerve and/or the ATN were entrapped by the LPM. Further examination of the ATN should include examining the TMJ (placing the examining fingers over the joint as the patient opens and closes their mouth).

The most common site of ATN entrapment is at the temple. Trescot (6) described finding the ATN by placing the index finger at the apex of the equilateral triangle created by resting (for the right-sided exam) the thumb on the tragus and the middle finger on the corner of the eye (Fig. 8). The examiner should also palpate the remainder of the temporalis muscle (looking for trigger point tenderness) and the STA (to evaluate for temporal arteritis).

**Differential Diagnosis**

Although patients with ZTN and/or ATN dysfunction may report the presence of a generalized headache in the temple region, when they are asked specifically to point with one finger to the location of maximum discomfort they are often able to do so. Those with ZTN irritation will point more anteriorly and may have a history of craniotomy, facial plastic surgery or orbital fracture (Table 2). Patients with ATN-associated symptoms will point to a spot closer to the hairline or ear. Their history may include dental or TMJ problems as well as parotid or jaw surgery.

Other potential causes must also be considered (Table 3). Tests that may help distinguish ZTN and/or ATN entrapment are listed in Table 4.

**Identification and Treatment of Contributing Factors**

Bruxism is a common problem and can cause temporalis muscle spasms of varying severity (94). An anterior occlusion splint (which puts the front teeth together, precluding activation of the temporalis) may prevent this spasm that can contribute to ATN entrapment. A low dose, long-acting benzodiazepine such as clonazepam (0.25 to 0.5 mg at night) or muscle relaxant such as tizanidine (2 to 4 mg at night) has been effective in preventing the ATN irritation (6). If TMJ movements aggravate the patient's symptoms, a dental evaluation would be appropriate.

**Injection Techniques**

Injections of these nerves serve several purposes; they provide a diagnosis of the extracranial source of

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**Table 2. History and Physical Examination.**

<table>
<thead>
<tr>
<th></th>
<th>ZTN</th>
<th>ATN</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clinical history</td>
<td>Temple migraine headaches  (21)</td>
<td>Preauricular migraine headaches  (15)</td>
</tr>
<tr>
<td></td>
<td>Craniotomy  (27,41,115)</td>
<td>Toothache (15,81)</td>
</tr>
<tr>
<td></td>
<td>Facial plastic surgery  (26)</td>
<td>TMJ pathology or trauma  (46,82,83)</td>
</tr>
<tr>
<td></td>
<td>Zygomatic fracture  (26,116) or osteotomy  (29)</td>
<td>Parotid surgery or severe inflammation (mumps)  (87)</td>
</tr>
<tr>
<td></td>
<td>Orbital fracture  (33)</td>
<td>Infant with a history of forceps-assisted delivery  (117,118)</td>
</tr>
<tr>
<td></td>
<td>Congenital orbital hypoplasia  (33)</td>
<td>Neurofibromatosis (119)</td>
</tr>
<tr>
<td></td>
<td>Orbital tumor  (29,33)</td>
<td>Jaw abscess (87)</td>
</tr>
<tr>
<td></td>
<td>Orbital radiation  (33)</td>
<td>Trigeminal neuralgia (tic douloureux) (16,87)</td>
</tr>
<tr>
<td>Signs &amp; Symptoms</td>
<td>Pain is more in/near the eye and anterior temple</td>
<td>Pain is more in/near the teeth and the temple at or behind the hairline</td>
</tr>
<tr>
<td></td>
<td>May be throbbing due to association with the STA.</td>
<td>May have TMJ pain with jaw movement (52,67)</td>
</tr>
<tr>
<td></td>
<td>May have pain in muscles of facial expression (57,59)</td>
<td>May have anterior ear pain and/or “stuffy deaf” sensation (76,89)</td>
</tr>
<tr>
<td>Physical Exam</td>
<td>Tenderness in the anterior temporalis and/or masseteric muscle (23)</td>
<td>Tenderness in the temporalis muscle behind the hairline near the ear (114)</td>
</tr>
<tr>
<td></td>
<td>Excessive tooth wear indicating bruxism (23)</td>
<td>Unilateral impaired salivation (67)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Paresthesias of the external acoustic meatus (57,67)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>LPM provocative test (57)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Numbness of lateral tongue, chin and/or lower lip (57)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Doppler to look for an arterial signal at the site of the pain (114)</td>
</tr>
</tbody>
</table>
Nerve Entrapment Headaches at the Temple

Table 3. Differential diagnosis of temple and face pain.

<table>
<thead>
<tr>
<th></th>
<th>Potential distinguishing features</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cervicogenic Headache</td>
<td>Neck pain, cervical facet tenderness, occipital nerve tenderness</td>
</tr>
<tr>
<td>Hemicrania Contiuana</td>
<td>Usually improves with carbamazepine and indomethacin</td>
</tr>
<tr>
<td>Myofascial Pain</td>
<td>Palpable trigger points such as one in semispinalis cervicis or splenius cervicis (120)</td>
</tr>
<tr>
<td>TMJ dysfunction</td>
<td>Pain over the TMJ</td>
</tr>
<tr>
<td>Otis</td>
<td>Otologic exam is abnormal</td>
</tr>
<tr>
<td>Tooth pain</td>
<td>Dental exam is abnormal</td>
</tr>
<tr>
<td>Atypical facial pain</td>
<td>Diagnosis of exclusion (everything else is negative)</td>
</tr>
<tr>
<td>Trigeminal Neuralgia</td>
<td>Trigger zones</td>
</tr>
<tr>
<td>Temporal arteritis</td>
<td>Systemically ill, elevated ESR</td>
</tr>
</tbody>
</table>

The headache, they deliver therapeutic medication to the targeted area, and they allow prediction as to whether further surgical or neurolytic treatment might offer benefit. However, the diagnosis is predicated on the patient actually getting numb from the local anesthetic used, and, if questioned, a significant number of patients will report difficulty getting numb with sutures, dental procedures, or pain procedures.

In 2003, Trescot (95) described a skin test to determine the most effective local anesthetic to use on an individual patient. Of nearly 1,200 patients interviewed, 250 had a history of difficulty getting numb. Of those, 90 only lost sensation after testing with mepivicaine, a local anesthetic not often used in clinical practice; an additional 43 only responded to lidocaine. Although this was only 11% of the total group, it was 53% of the patients with a history of “local anesthetic resistance”. Consideration might therefore be given to skin testing patients prior to injections.

The injections can be done purely for diagnosis with just local anesthetic, or local anesthetic and a deposteroid can be mixed together and injected. Total volumes injected here should be small (less than 1mL); because methylprednisolone is available in a high concentration (80 mg/mL), it is therefore the medication of choice for one author (AT). Because the skin is thin in this area, there is a significant risk of steroid-induced atrophy, and so steroid dose should be less than 40 mg of methylprednisolone equivalents. Small gauge needles (27 to 30 gauge) are used both for patient comfort and to avoid inadvertent injury to the nerves; a 3mL syringe is used for ease of injection.

Injection Technique for ZTN

**Landmark-Guided Technique**

ZTN injections are performed at the site at which the nerve pierces the deep temporal fascia (12,13). Palpate the hollow exit area approximately 1.5 cm lateral to and 0.5 cm above the lateral palpebral commissure and insert a 1-inch 27- to 30-gauge needle approximately 1 cm posterolateral to and in the direction of...
the hollow point (Fig 9). Note the increase in resistance as the needle crosses the deep temporal fascia (12); be careful not to inject more than about 0.5 to 0.75 mL in this area, because of the small space.

**Injection Technique for ATN**

**Landmark-Guided Technique**

The distal ATN injection is more commonly used. Identify the tender area at the apex of a triangle with a base composed of the line connecting the tragus and the corner of the eye (Fig. 8) (6), typically 1 – 2 cm anterior to the root of the ear helix (56,96). This site is usually very close to the artery; confirm that the artery itself is not tender to decrease the likelihood of a missed diagnosis of temporal arteritis. Inject one mL of local anesthetic and depot-steroid in a slightly cephalad direction, parallel to the path of the nerve (Fig. 10-A). The artery is usually marginally more posterior; if arterial pulsations are not obvious, place the injection slightly anteriorly to avoid a hematoma. Take care to avoid injecting directly into the artery, especially with particulate steroids, to avoid a potential embolization (which has never been reported).

Proximal (less common) landmark-guided ATN injections are performed where the nerve passes the posterior aspect of the mandibular condyle, slightly below the zygoma. Insert a 3.5-inch, 27-gauge needle anterior to the junction of the tragus and the lobule (Fig. 10-B). Withdraw the needle 3-5 mm so as to avoid an intracapsular TMJ injection (47,97,98). This site is close to the facial nerve, and the patient must be warned of the possibility of temporary facial nerve weakness. For that reason (and because the region is very vascular) small volumes (0.5 mL) and meticulous needle placement are recommended. If temporary facial nerve palsy occurs, tape the eyelid closed, thenpatch the eye until the local anesthetic has worn off.

Speciali and Gonçalves (47) reported a series of 6 patients with ATN. They injected...
the proximal ATN using 0.5mL of 2% lidocaine with dexamethasone 2.5 mg (up to 1.5 mL). Only one patient had recurrence of pain after a single injection, but the authors report a short follow-up of less than 1 year.

There is occasionally a “double crush” syndrome of the ATN, with the nerve trapped at both the proximal and distal sites. These patients describe persistent temple pain after injection of the distal ATN, with tenderness anterior to the tragus. The proximal site would then need to be treated.

Fluoroscopy

There are no bony landmarks for fluoroscopy to be useful.

Ultrasound-guided Technique

Several authors (99,100) have described an ultrasound (US)-guided technique for distal ATN injection. Place the probe transversely just above the TMJ, and identify the STA with color Doppler. The ATN is a small hypoechoic structure next to the superficial temporal artery (Fig. 11-A). Rotate the probe for a longitudinal scan to track the nerve cephalad (Fig.11-B). The authors used a 25-gauge needle from an out-of-plane approach to deliver 2mL of 1% lidocaine and 10 mg of Depo-Medrol® around the nerve. This selective targeting provided symptom relief for a patient with an injury-related unilateral headache who had failed to respond to less specific injections (99).

Neurolytic and Surgical Techniques

Chemical Neurolysis

Chemical neurolytic techniques involve the use of botulinum toxin, alcohol or phenol. Botulinum toxin is commonly injected into the temporalis muscle to diagnose and/or treat parieto-temporal migraines, which presumably results in decreased entrapment of the ZTN (12,13).

Because of the potential spread of medication, alcohol or phenol is not recommended, except in special cases such as the treatment of cancer pain.

Surgery

Over the last 15 years, surgical treatment of patients with severe chronic migraine headaches has been well established on the basis of retrospective reviews (3,14), a pilot study (101), a prospective randomized clinical trial (19), a long-term outcome study (21), and demonstration of its cost-effectiveness (102). Initially, surgery was reserved for patients whose symptoms responded to diagnostic local anesthetic and/or botu-
linum toxin injections (14,20,101), although with more experience some have omitted this step (103).

Surgical treatment of migraine headaches is not without controversy, and further careful investigation of this matter is warranted (104-106).

**Neurolytic/Surgical Techniques for ZTN Dysfunction and their Potential Complications**

**Chemical Neurolysis**

Dilute 12.5 to 25 units of botulinum toxin in 0.5mL of saline in a 3mL syringe, and attach a 1-inch 30-gauge needle. Palpate the hollow point area (HP), approximately 1.5 cm lateral to and 0.5 cm above the lateral palpebral commissure, and insert the needle approximately 2 cm posterolateral to and in the direction of the HP. Note the increase in resistance as the needle crosses the deep temporal fascia. Inject some of the botulinum toxin into the temporalis muscle at this point then direct further injections cephalad, posteriorly and caudally to paralyze the muscle fibers immediately next to the ZTN (12). These (107,108) authors assert that the toxin will diffuse in a circle with a radius of approximately 1.5 cm.

Complications reported after ZTN landmark-guided botulinum toxin injections include diplopia (13) and decreased strength of mandibular closure (12). Diplopia is a debilitating, though rare, complication that is thought to be due to diffusion of botulinum toxin injections (14,20,101).

![Image of ultrasound evaluation of the auriculotemporal nerve. A = vertical probe placement; B = horizontal probe placement. (Image courtesy of Andrea Trescot, MD)](image-url)
toxin through the ZT foramen, weakening the nearby lateral rectus muscle. The authors recommend a relatively superficial injection into the temporalis muscle, well lateral to the orbital rim (13).

**Surgery**

The hollow point (HP), recognized as the exit point of the ZTN through the deep temporal fascia (on average 1.7 cm lateral and 0.6 cm cephalad of the lateral palpebral commissure), allows identification of the surgical site in individual patients (12). In the traditional surgical avulsion method, about 3 cm of the intramuscular portion of the ZTN is removed and the proximal end retracts into the orbit; this is a common occurrence in procedures done in this area for other reasons (12,109). Recently, Guyuron and colleagues reported the results of a prospective randomized clinical trial to evaluate 2 endoscopic surgical techniques for treatment of temporal migraine headaches (103). Due to the fact that neuromas have occurred after avulsion of other nerves and that surgeons and patients consider avulsion more invasive than decompression, they randomized 19 patients with severe bilateral temporal migraine headaches to have traditional avulsion of the ZTN on one side and decompression by fasciotomy and cauterization of the accompanying blood vessels on the other. At 1 year, they found a dramatic reduction in frequency, migraine days, severity and duration in 89% of operative sites (34/38), with complete elimination in 55%. On average, patients' headaches decreased from 14 to 2 per month. No patient had a complication and there was no difference in outcome measures between the 2 groups. The authors recommend decompression as the preferred first option, and offer avulsion neurectomy to the few patients whose decompression sites achieve less than 50% relief.

In addition to endoscopic approaches to the ZTN (12,103), a case series (20) describing an open procedure with an incision just posterior to the temporal hairline has been reported. The authors investigated the open technique because substantial experience is required to become proficient at endoscopy, long instruments are used to manipulate these very fine nerves, and endoscopic techniques require extensive dissection, especially when the procedure is performed bilaterally. The authors assessed the ZTN for viability and decompressed it as described above (although they label the blood vessel accompanying the ZTN through the fascia as “a sentinel vein” rather than an artery). If the nerve was not thought to be viable, they transected it and buried the end deep in the temporalis muscle. Some patients also had ATN decompression through the same incision. After one year, they found that 84% (16/19) of patients had at least 50% reduction in symptoms and 37% (7/19) had no further headaches in the temple region. There was no difference in outcome between the 2 groups. The authors hope that this simpler technique will “lower the bar” to adoption of surgical treatment of temple migraine headaches.

**Neurolytic/Surgical Techniques for ATN Dysfunction and their Potential Complications**

**Chemical Neurolysis**

Intracutaneous botulinum toxin has been used to treat ATS (Frey's Syndrome) (110). Since this site is very close to the path of the facial nerve in the parotid gland, there is at least a theoretic risk of facial nerve paresis from the toxin.

**Cryoneuroablation**

Cryoneuroablation has been an effective therapeutic technique for ATN. Trescot (5,6) described the use of cryoneuroablation for the treatment of extracranial headache syndromes including ATN entrapment. The cryo probe is placed parallel to the nerve and, using sensory stimulation, the probe is swept side to side to find the nerve.

**Peripheral Nerve Stimulation**

Until recently, peripheral stimulation of the ATN has been limited by technical considerations, but recent advances have increased their use (96). In 2010, Simopoulos and colleagues (111) described bilateral ATN stimulation; the next year Deshpande and Wininger (112) described a combined stimulation of the ATN and the greater occipital and lesser occipital nerves (Fig. 12).

**Surgery**

A 5-year randomized study of 69 migraine patients comparing surgery to botulinum toxin of “migraine trigger points” (including the ATN area) showed an 88% improvement in headaches after surgical release (21). Some authors (18) emphasize the importance of intraoperative Doppler monitoring to identify the precise location of the STA so as to minimize dissection trauma. Endoscopic ATN surgery was pioneered by Guyuron (101) and expanded by Sanniec (18). This procedure is easily combined with ZTN decompression due
to the close proximity of the 2 nerves at the temple. In order to avoid injury to the temporal branch of the facial nerve, the authors recommend releasing the main branch of the ATN just above the TMJ.

DISCUSSION

Headaches, including migraine headaches, are common, disabling and expensive. Recent work on the role of injuries to and sensitization of peripheral nerves as a trigger for migraine headaches has provided new hope for patients and spurred research into anatomic understanding of the anatomy of the nerves involved. Based on the dramatic and often complete relief of “migraines” with injections of these nerves, these extracranial nerve entrapments may represent the actual etiology and not just the trigger of headaches that have been misdiagnosed as “intracranial”. Investigation of failures of medical and surgical treatment served as a particular incentive to understand anatomic details (13,16).

Some discrepancies in the descriptions of ZTN anatomy can be explained by differences in purpose and methods among the reports. For example, some authors (26) interested in bony procedures such as zygomatic fracture reduction involving the lateral orbital wall began their cadaveric dissection in the orbit after the eye and accessory visual structures had been removed. Others with a particular interest in craniotomies traced the course of the ZTN in cadavers from the periphery, past the pterion [the region where the frontal, parietal, temporal and sphenoid bones join - a common neurosurgical approach to the middle cranial fossa (113)] to the ZT foramen (27). A third group focused on surgical treatment of migraine headaches and investigated patients undergoing endoscopic forehead surgery (12) or fresh cadavers (13) and also began their anatomic explorations at the periphery.

Because symptoms of ATN dysfunction have been of long-standing interest, its anatomy is better understood, although the course of the nerve around the TMJ is still a matter for discussion (76,85,89).

As is evident from reports in both the neurology (11) and neurosurgical (114) literature, it is very likely that either or both nerves can be the cause of a headache in the temple region and imply that the ZTN is involved more frequently than the ATN. Investigation into the cause of the headache in an individual patient should include systematic evaluation of both nerves.

SUMMARY

Both the ZTN and the ATN have been associated with headaches in the temple region. Dysfunction of the ZTN is likely after surgery or trauma to the orbital wall, and is associated with temple-region headaches as well as neuropathic pain and paresthesias in the area that it innervates (39). Disturbances of ATN function also cause temple-region headaches, although one-finger demonstration of the area of maximum pain at the start of the headache usually reveals a more posterior location. These patients have histories and other symptoms more related to the jaw and ear. As always, the presence of anatomic variability underlines the importance of careful physical examination and diagnostic injections before substantial further intervention in any individual patient (6).
Acknowledgments

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