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Temporomandibular Disorders

Greater Auricular Nerve Block Reduces Dental Hypersensitivity to Intraoral Cold Water Swish Challenge: A Retrospective Study

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Abstract

Objective

To reduce cold tooth hypersensitivity by injecting a local anesthetic into the neck to block the Greater Auricular Nerve (GAN), a sensory branch of superficial cervical plexus which carries unmyelinated sympathetic fibers along its path to the lateral face. The null hypothesis was that no change in the cold sensitivity would occur post-injection.

Methods

Ninety-one TMD and/or orofacial pain patients that experienced a disproportionate pain response in their maxillary and mandibular dentition to a 5 second cold water swish challenge were re-tested 5 – 10 minutes after the Greater Auricular Nerve was injected (n = 91). Pre-injection cold-water swish values were compared to post-injection ice swish values using the Wilcoxon Signed-Rank test. In the verbal analog scale (VAS) a zero signified absolutely no cold sensitivity whatsoever while 10 signified intolerably painful cold sensitivity.

Results

The mean VAS level of the group was significantly reduced from 6.49 pre-injection to 3.97 post-injection ($p < 0.00001$). A total of 61 of the 91 subjects (68 %) experienced a reduction in cold sensitivity. Only 18 of those subjects (30 %) experienced total elimination of cold sensitivity. After the GAN anesthetic block wore off, the sensitivity to cold returned to baseline in all patients.

Conclusion

The injection of the Greater Auricular Nerve using local anesthetic immediately posterior and superficial to the muscle fibers of the sternocleidomastoid muscle produced a significant temporary reduction in the tooth sensitivity to cold-water swish within 61 of the 91 TMD and/or orofacial pain subjects studied.

INTRODUCTION

The greater auricular nerve (GAN) arises bilaterally from the ventral aspect of C-2 and C-3 and is the largest sensory branch of cervical plexus.[1] This cutaneous sensory nerve tracks along the inferior border/angle of the mandible on its way up towards the auricle to specifically innervate the inferior preauricular region, the ventral pinna, and the mastoid region. It carries unmyelinated sympathetic fibers along its course.[2] Blocking the GAN with local anesthetic along its course typically leads to transient numbness of these aforementioned anatomic structures.

Abundant reports in the literature relate that innervation of dental pulpal tissues are comprised of not only trigeminal afferent fibers, but cervical sensory and vasoconstrictive sympathetic fibers as well.[3-6] Sympathetic fibers increase tension to incoming blood vessels toward the pulp. The presence of vasodilatory parasympathetic fibers within the dental pulp is a contested topic, since most involved researchers proclaim that parasympathetic fibers have never been definitively shown to exist within histological studies of human pulp tissues.[4,7] Hence, vessels that bathe pulp tissues are stimulated to readily constrict via sympathetic innervation, but unlike most tissues within the body, the vasculature present within the hard, encased dentin shell that surrounds the dental pulp lacks the parasympathetic innervation to counter this vasoconstrictive effect. Compartment syndrome may develop if chronic hypoxia leads to localized edema, which could explain why some do not respond to the GAN block.

Traditional theories relating to dental hypersensitivity involve primarily open dentinal tubules and trigeminal innervation, giving little to no clinical consideration to the contribution of cervical sensory with accompanying sym-

Table 1. Comparison of entire group means of this GAN block investigation between pre-op and post-op.

n = 91	Whole Mouth Cold Sensitivity Pre-Op (0 - 10)	Whole Mouth Cold Sensitivity Post-Op (0 - 10)
Mean	6.49	3.97
Standard Deviation	2.43	2.68
Wilcoxon Signed-Rank test	p < 0.00001	

pathetic inputs to the clinical expression of cold hypersensitivity in teeth. This clinical pilot study seeks to demonstrate that dental cold hypersensitivity can be related to cervical sensory and/or sympathetic neurosensory inputs by demonstrating that anesthetic blockade of the greater auricular nerve temporarily sequesters and decreases the intensity of a painful response to an intraoral whole mouth cold water swish of 5 seconds' duration. We carried out the present study with the assumption that blocking the GAN with local anesthetic during diagnostic workups for patients who presented with TMD complaints (which include cold dental hypersensitivity to a cold-water swish challenge) can alter their perception to cold. The purpose of the GAN block is diagnostic only.

Clinical experience in dentistry indicates that inflamed teeth may be sensitive to cold for various reasons, including vascular irregularities due to neurological reasons outside of the trigeminal nerve. We believe based on Ajcharanukul et al[8] that dental cold hypersensitivities can be resultant of an increased intra-pulpal inflammation due to cold exposure, and that this inflammatory problem can be countered resultant of the suppression of autonomic sympathetic intra-pulpal influences and proffer possible reasons for this hypothesis in this work.

A Verbal Analog Scale (VAS) was developed by Yiannios in a 2017 publication.[9] He notes that the basis of the VAS recordings come from an earlier publication in 2015.[10] It consisted in the placement of water into a cup of ice for a minimum of 5-6 minutes to attain an average tested temperature of 37 degrees F. Patients were then asked to place this cooled water into the mouth for 5 seconds and record a level of tooth sensitivity from 0 to 10, zero being no sensitive response and ten as the most severe. This cold-water swish VAS result was then recorded pre-op as well as post-op to whatever treatment occurred. In this case, a GAN block was used to determine if any change occurred to cold hypersensitivity according to the VAS criteria. We cite Table 1 for posted findings of the GAN block.

In dentistry, hypersensitive teeth are routinely encountered in daily practice when open or exposed dentinal tubules are typically implicated as the causative factor for thermal, air, and/or tactile hypersensitivity. There are other terms that are used to describe this hypersensitivity phenomenon, including, but not limited to, dentin(e) hypersensitivity (DH), hypersensitive teeth, and cervical dentinal hypersensitivity (CDH). DH (the most used term) refers specifically to dentin. Just before the turn of the century, authors began differentiating hypersensitive teeth causation into two basic subtypes, DH and CDH, ascribing sensitivity derived from the occlusal (DH or pulp) as well as cervical regions of the dentition (CDH) ascribed by Pashley in 1993.[11] The updated 21st century definition of DH refers to a dull and lingering pain originating from the pulp, whilst a definition of CDH refers to a sharp, fast, and intense pain of short duration arising from the cervical aspect of the dentition.[12] Importantly however, if one were to create a more objective and concise label for hypersensitive teeth which would also encompass alternative sources (other than the dentinal tubules) for hypersensitivity of the dentition, the authors suggest that the term dental allodynia (DA) would be a far more accurate and all-encompassing alternative descriptor. In "pain medicine," an allodynia refers to a disproportionate sensation of pain after exposure to a non-noxious stimulus[13] or put another way; a pain that is disproportionate to the inciting event. For example, recent research has shown statistically that occlusal mechanisms can and do result in dental cold allodynia, irrespective of the lack of missing enamel with exposed dentin and/or recession[9-11,13] as an example of a hypersensitivity response unrelated to the condition of the dentin. The diagnosis of a dental allodynia is arrived at via a systematic process of exclusion[14] whereby the clinician rules out the various other pathologies that may be responsible for a dental hypersensitivity response to cold, such as modified from Yiannios.[12]

MATERIALS & METHODS

Written patient consent was previously obtained from patients that were seeking treatment for various temporomandibular disorders. Ninety-one patient records were serially selected from a patient database for this clinical study according to inclusion and exclusion criteria. Each patient had responded with a moderate to severe tooth pain during a 5 second cold water swish.

The Verbal Analog Scale (VAS) records of these 91 patients who were found to have experienced a disproportionate pain response to a 5 second cold water swish challenge were selected for study (n = 91). The subjects were previously routinely tested as follows:

- Water was steeped for 5-6 minutes minimally in a cup of ice to obtain a consistently cold temperature at an elevation of 1,300 feet above sea level.
- The subjects were instructed to swish the ice water for 5 seconds before swallowing or expectorating and queried as to the pain level that they perceived on a scale of 0 to 10 (10 being intense pain; 0 being no pain whatsoever)
- Approximately 0.25 - 0.5 cc 0.5% Bupivacaine w/ 1:200 epinephrine was injected into each side of the neck, targeting the GAN nerve as it courses superficially above the sternocleidomastoid muscle (both right & left sides) using a 12.7 mm needle with less than 5 mm of penetration. The efficacy of the nerve block was confirmed by the loss of sensation in the distribution of the GAN.

- About 10-15 minutes after the GAN injection, a post-injection ice water re-swish that included a repeat VAS rating was subsequently recorded by the clinician.

The current analysis includes a retrospective records search originating within a single private general dental practice, which often focuses on diagnosing confounding chronic orofacial pain conditions. A single clinician injected the GAN of all 91 patients, without the use of ultrasound, but rather with a combination of an appreciation of the anatomy of the region coupled with infrared Accuvein® technology to help avoid intravascular injection of nearby vascular structures. Additionally, aspiration always preceded injection of the anesthetic. All patients were TMD patients who had consented to this diagnostic procedure after informed consent was gleaned, to ascertain if a percentage of their confounding orofacial symptomatology (headaches, muscle tension, cold hypersensitivity, muscle dystonia, etc.) would abate for the duration of the neck block, thereby implicating a complete or partial non-trigeminal sourcing for their orofacial pain patterning.

INCLUSION CRITERIA

1. Patient report of a VAS score indicating tooth sensitivity to cold,
2. Older than 18 years of age,
3. All 91 subjects had previously sought treatment for temporomandibular disorders (TMD) and/or orofacial pain
4. Acceptance of the injection procedure

CRITERIA FOR EXCLUSION OF TREATMENT

1. Allergy to bupivacaine (Marcaine)
2. Patients with active periodontal and/or pulpal pathology
3. Patients with dental pathology requiring operative interventions
4. Patients with severe enamel wear with multiple concurrent regions of exposed dentin
5. Patients with profound and generalized areas of gingival recession
6. Patients with anterior open bites
7. History of past temporomandibular joint surgery of any kind
8. Patients detected with open restorative margins, fractured tooth syndrome with pulpal reactions, or retentive dentin pins often in heavily restored vital teeth which can produce symptoms to cold.
9. Palato-gingival groove or other enamel invaginations

The GAN emerges at the posterior border of the sternocleidomastoid muscle (SCM) in an easily accessible superficial location as described below. The specific anatomical site targeted for the GAN block made access to the nerve fibers safe without the aid of ultrasound imaging. No adverse effects occurred during any of the injections.

NOTE: *limiting the injection to not more than 0.5cc of local anesthetic to a depth not penetrating the inferior aspect of the SCM is clinically safe. Injecting anesthetic well below the SCM when injecting local anesthetic could theoretically block the phrenic nerve. The patient may experience a temporary shortness of breath, but the contralateral nerve should keep the diaphragm active. The injecting clinician had previously employed this GAN block on well over 1,000 patients over the course of many years and has not once run into a phrenic complication. The usage of an extra-short ½ inch (12.7 mm) 30-gauge needle coupled with the judicious limiting of anesthetic volume ensured that the anesthetic would not be placed deep enough to affect the phrenic nerve.*

The null hypothesis was "No change in cold sensitivity would occur after local anesthesia block of the Greater Auricular nerve."

CLOSED COMPARTMENT SYNDROME

The dental pulp is a classic case for compartment syndrome; a low compliance environment with no room for volumetric expansion in which a small increase in tissue volume due to inflammation can have serious consequences.[15] Edema within this compartment can readily cause inflammation and hypoxia. Injecting the GAN with its associated sympathetic fibers could theoretically block sympathetic vasoconstriction, leading to a decrease in hypoxia. This likely is the primary reason why these GAN blocks temporarily and quite profoundly decrease the dental allodynia to the cold-water swish. It is likely that the sympathetic blockade leads to vasodilatation, improved oxygenation and a gradual resolution of inflammation and edema. The response when positive was close to instantaneous suggesting that the GAN block decreases sympathetic pulpal input based upon this pilot study.

GAN LANDMARK INJECTION TECHNIQUE

After placing the patient in a supine position, the patient is asked to turn their head away from the side intended to be blocked. Figure 1A is an example of a GAN block for the patient's left side. A sterile 30 gauge extra short ½ inch (1.3 cm) needle is then attached to a sterile dental syringe. Figure 1B is a photo of the extra short needle with local anesthetic loaded into a syringe. It is a 1.8 ml carpule of 2% bupivacaine with 1:200 epinephrine. The area to be injected is then disinfected with alcohol, betadine (povidone iodine), or ozonated water. The ½ inch 30- gauge needle is then inserted at the posterior border of the SCM muscle at the level of the cricoid notch. After advancing the needle 2-4 mm at a 90- degree angle to approximate Erb's point, approximately 0.25 – 0.5 ml of anesthetic was deposited after aspiration confirms no vascular penetration had occurred. See Figure 2. The patient should be warned preoperatively of a transient paresthesia along the course of the nerve distal to the injection site that will likely last 6-9 hours when this local anesthetic is used. The patient should also be warned of the highly unlikely but remotely possible phrenic nerve complication mentioned above within the informed consent.

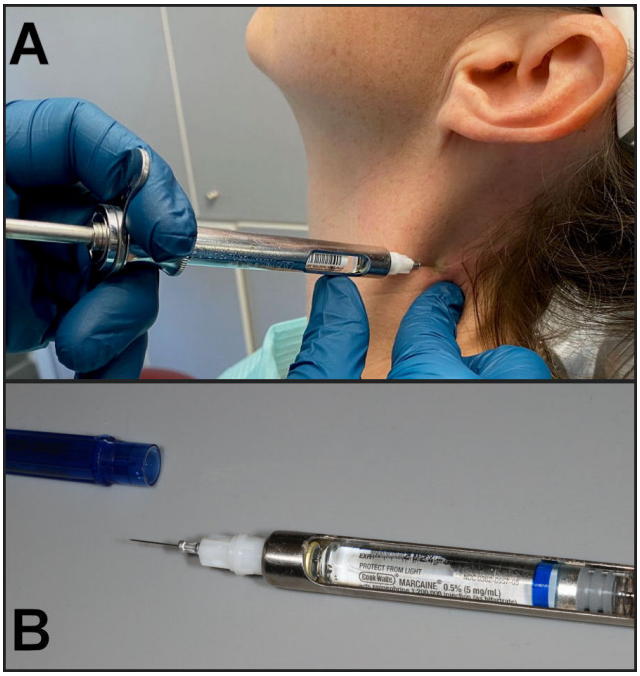

Figure 1AB. A) Injection approach and site for anesthetizing the GAN. B) A photo of the extra short 30- gauge needle as well as the Marcaine solution injected for the GAN block.

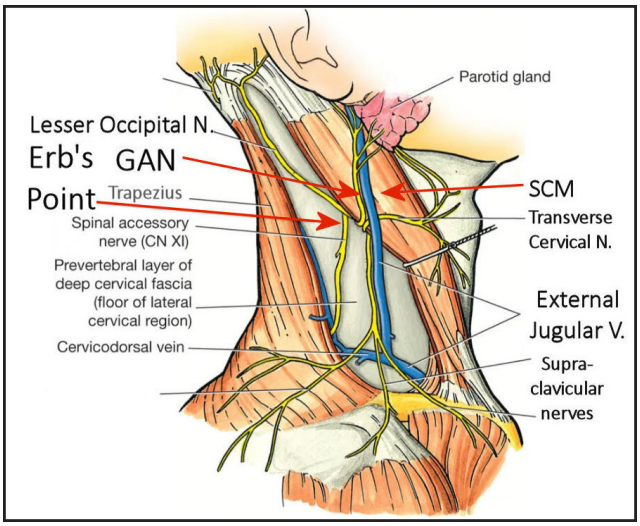

Figure 2. Internal neck anatomy on right side.

GAN ANATOMY

The GAN pierces the cervical fascia and passes superiorly and forward at a point just lateral and inferior to the lesser occipital nerve before curving around the sternocleidomastoid (SCM) muscle at Erb's point. Figure 2 shows this anatomy for the patient's right side. The GAN then pierces the superficial cervical fascia to move more superiorly and superficially to provide cutaneous sensory innervation to the angle of the mandible, the external auditory canal, and the skin overlying a portion of the parotid gland.

Table 2. Comparison of the responder's means of this GAN block investigation between pre-op and post-op.

n = 61	Whole Mouth Cold Sensitivity Pre-Op (0 - 10)	Whole Mouth Cold Sensitivity Post-Op (0 - 10)
Mean	6.60	2.89
SD	2.52	2.13
Wilcoxon Signed-Rank test	p < 0.00001	

STATISTICAL ANALYSIS

The Verbal Analog Scale (VAS of 0 – 10) data series that rated the sensitivity to cold, both pre-injection and post-injection, were determined to be normally distributed using the Jarque-Bera normality test. However, the subjective nature of the VAS values suggested that a non-parametric statistic would be most appropriate. Thus, the Wilcoxon Signed-Rank test was chosen to evaluate for any significant change in the group's mean VAS response to the 5 second cold water swish post-injection.

RESULTS

Table 1 reveals the mean and standard deviation values of the 91 group members prior to local anesthetic injection compared to post-injection. The mean VAS score of cold sensitivity from the group was significantly reduced from 6.49 pre-injection to 3.97 post-injection ($p < 0.00001$).

Among the 91 subjects 61 (68 %) reduced their VAS scores (the responders) and 30 (32 %) indicated no change. Table 2 reveals the greater change in the mean VAS score of the responders. Within the responders, 18 (30 %) indicated a VAS score of 1 or less after the GAN injection. It was very revealing that no subject experienced any increase in cold sensitivity, opposing any claim that vasodilatation might increase the pain or sensitivity in a closed space like the dental pulp.

DISCUSSION

DENTAL PULP SENSORY AND AUTONOMIC OVERVIEW

In previous studies local anesthesia to the GAN has been demonstrated to aid in obtaining a pain free environment for inadequate inferior alveolar nerve blocks during lower mandibular third molar extractions.[16,17] A soft mesenchymal tissue, the dental pulp is densely innervated by sensory afferent fibers, sympathetic fibers, and theoretically (unsubstantiated) parasympathetic fibers.[18,19] Sympathetic fibers arise from both the trigeminal ganglion and the cervical sympathetic ganglion[20] and sensory nerves with sympathetic axons accompany blood vessels and even encircle them.[4] Hence, sympathetic fibers essentially "hitch-hike" into the pulpal environment, following blood vessels, all the way down to the arterioles.[21]

In 1989, Kim et al. reported that electrical stimulation of the cervical sympathetic nerves causes pulpal arteriolar constriction and a reduction of arteriolar and venular flow rate (in mm^3/s) in rat incisor pulps and a decrease in pulpal blood flow (in ml/min/100 g) in canine pulps of cats and dogs.[22] Importantly, sympathetic nerves vasoconstrict, leading potentially to hypoxia, ischemia and claudication within the myriad of different tissues that they innervate.[18] Conversely, parasympathetic nerves vasodilate, increasing blood flow to a region. For example, a combination of sympathetic and parasympathetic innervation to cutaneous vessels are closely related to the body's adaptation to heat and cold stresses.[23] This oppositional and synergistic combination is typical regarding systemic autonomic regulation throughout the body. However, as previously stated, pulpal tissues are generally believed to lack this oppositional parasympathetic neurological input.

The null hypothesis was rejected based on the results revealed in Table 1, finding that a GAN block did indeed significantly reduce dental cold sensitivity for two thirds of the subjects. Since 32 % of the subjects did not respond and only 30 % of the responders' sensitivities were temporarily fully resolved, these results support the theory that tooth sensitivity to cold may be considered as multifactorial. However, the non-responders may have had more inflamed and edematous pulps, which might not respond immediately to a block, but repeated block over time could theoretically allow for a more gradual reduction in inflammation and edema reducing sensitivity too. Also those with a partial response may have had some lessor level of edema too and could have benefited after repeated injections.

The activation of sensory nerves within the pulp induces a long-lasting pulpal blood flow decrease within the pulp, with a concomitant increased vascular permeability.[24] This increased permeability can lead to edema, and subsequent inflammation. Additionally, neurogenic inflammation is thought to be mediated by neuropeptides released from sensory nerves, such as calcitonin-gene-related-peptides (CGRP) and substance P (SP). These inflammatory stimuli could also become dominant, which may further contribute to the progression of pulpal inflammation.[18,25] It is commonly well known in dentistry that a common symptom of pulpitis is thermal sensitivity to cold. Inflammatory changes that are present from the increased vascular permeability and resultant edema can lead to hypoxia and cold hypersensitivity.

LIMITATIONS

It is well accepted that calibration and blinding of examinators can be an important issue in clinical research to minimize bias. However, in this analysis the clinician made no estimates, judgements or decisions regarding the patient reports, only documented them. Thus, there was no opportunity for clinician bias to interfere with the collection of the data as reported by the patients. The vast previous experience of the administering clinician obviated any need for additional calibration.

A placebo injection might have been concurrently utilized if this were a.) a prospective study or b.) if sufficient volunteers could have been found post facto. This was neither a prospective study nor an experimental procedure. Since the comparison was made only intra-patient, from pre to post injection, it reflects what a practicing TMD provider can expect to see. Since 1/3 of the subjects did not respond to the injection, a placebo test could not have been very useful as a test for a Somatic Symptom Disorder (SSD). However, SSD is a psychiatric diagnosis and falls outside of dentistry.

Blinding of the clinician was not possible without a second participant (present) and the use of a placebo injection (not present). There was no need to blind the patient without any placebo.

The GAN injection is not a common general dental procedure, but a useful diagnostic procedure for those practitioners that treat TMD and oral surgeons that do third molar extractions. The GAN injection is far less invasive than many dental procedures, most of which are not at all reversible. This practitioner has undertaken this procedure for many years and found it to be very useful for not only TMD diagnostics, but as an adjunct for profound mandibular anesthesia, and as a treatment to reverse intraoperative trismus. Additionally, the implementation of the GAN by this practitioner has on several occasions staved off the need for endodontic therapy in cases in which that pathway was initially thought to be required. For these diverse applications, the practitioner should always ask oneself if it is possible that a ramped up sympathetic nervous system might be etiological for symptomology, and if so, determine and address the cause of this chronic affliction.

CONCLUSION

The injection of the Greater Auricular Nerve immediately posterior to the sternocleidomastoid muscle produced a significant reduction in the tooth sensitivity to cold within this group of 91 TMD and orofacial pain subjects. Central dysregulation of the brainstem trigeminal-autonomic circuits is a possible reason as to why teeth respond to decreased cold sensitivity following a GAN block. As explained previously, temporarily blocking the sympathetic vasoconstrictive innervation to the dental pulp reverses the edema that is built up within the pulp compartment, leading to elimination of hypoxia for the duration of the block. It is possible that the resolution of this hypoxia and inflammation by decreasing sympathetic tone relates to the loss of thermal sensitivity following a GAN block. The GAN block is safe to perform by any dental practitioner who is well-versed in the relevant anatomy and is an effective diagnostic tool for elucidating potential sources of orofacial pains, including confounding cold hypersensitivity of the dentition. Although this report shows the beneficial effect of a GAN block reducing cold hypersensitivity, more information is needed for the sympathetic system as it responds to a closed compartment system. Additionally, investigations are indicated to verify and define the implications

that the novel concept of Sympathetic Dental Hypersensitivity (or SDH) has upon the practice of dental medicine.

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DISCLOSURE STATEMENT

Yiannios N, DDS - Private Practice in Rogers, AR USA. Director & Founder of the Center for Neural Occlusion

Coleman T, DDS - Retired from practicing dentistry, Middlebury, VT USA

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