Diagnostic Blocks for the Orofacial Pain

BAOS
BRITISH ASSOCIATION OF ORAL SURGEONS
Promoting excellence in Oral Surgery through education, training and research for better patient care

Faculty of Dentistry, Oral & Craniofacial Sciences

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KING'S College LONDON

Learning objectives for Diagnostic blocks for the orofacial patient

- This presentation will aim to provide the delegate with an overview of;
  - the various types of available diagnostic block interventions for orofacial pain
  - their limitations and the related evidence base
  - guidelines for the case selection, application and assessment of the block intervention will also be highlighted.

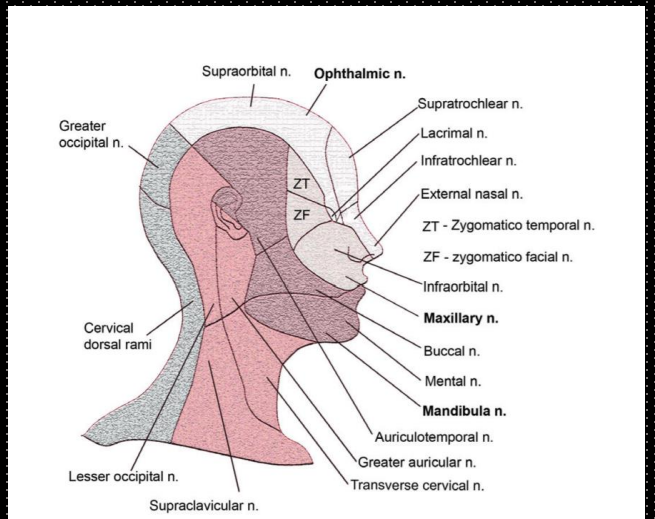

Fig. 1. Cutaneous nerve distribution of the maxillofacial region emphasizing trigeminal nerve divisions.

Disclosures

- NHS work only
- 3x3M Local anaesthesia lectures 2017
- Never prescribe Opiates or opioids

Legality

- There is a open case against a dentist in NWS giving cervical trigger point injections
- The dentists indemnity doe not cover him
- Always check with your indemnity body that you are covered
- Its probably easier if you are registered oral surgeon

Disclosures

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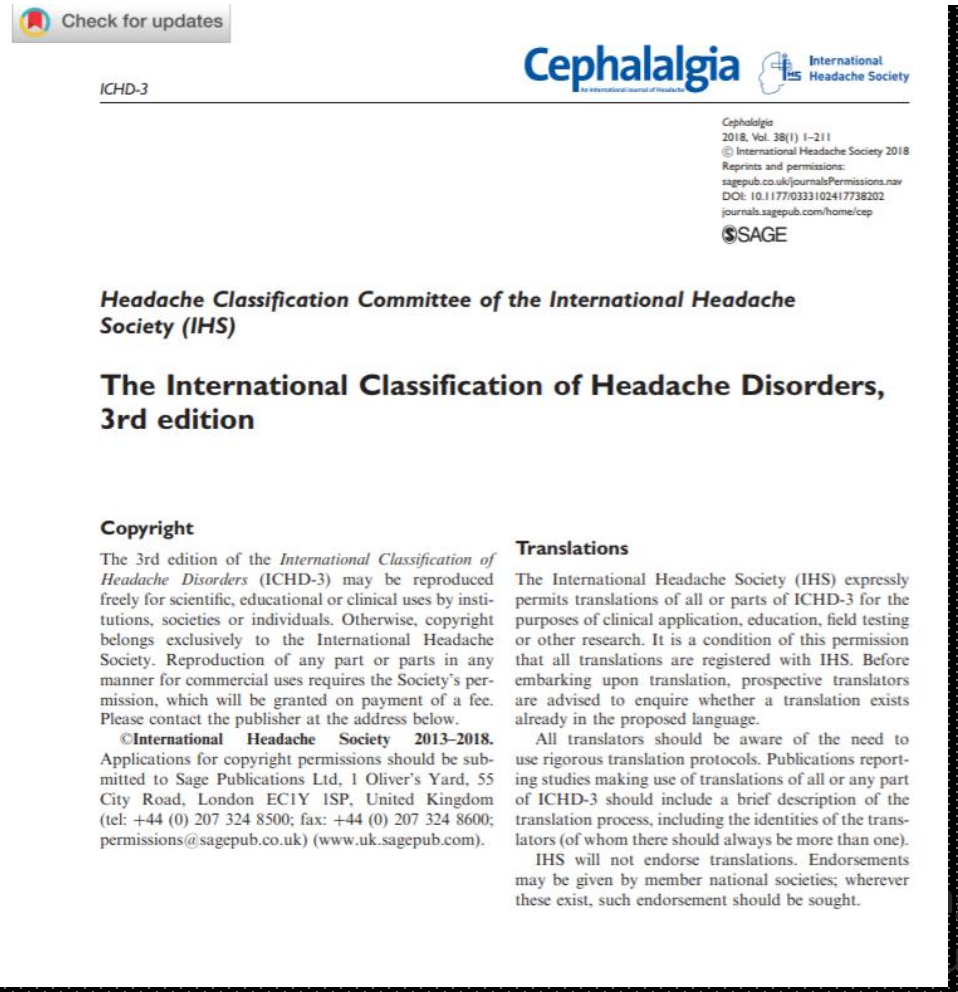
- Silent weapon…….

Facial pain – A diagnostic challenge

Geoffrey Quail



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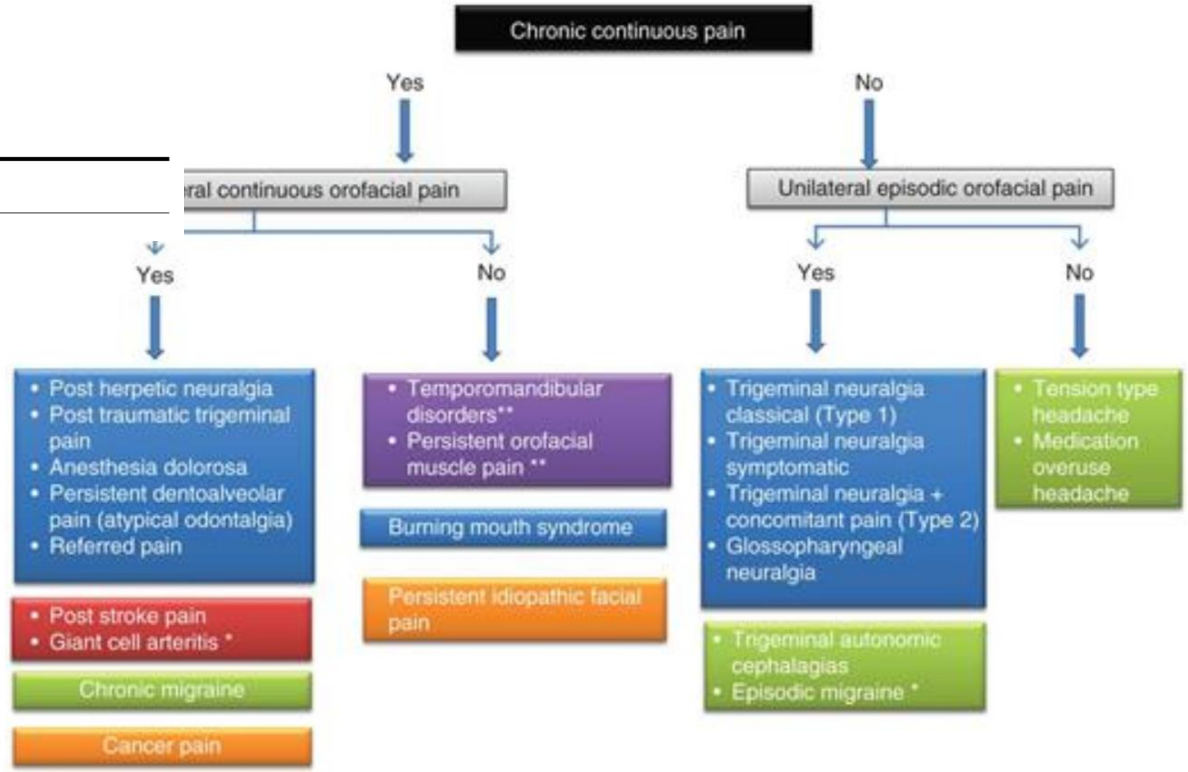

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Causes of chronic orofacial pain. *Can be bilateral, **can be unilateral. Types of pain: blue box, neuropathic; red, vascular; purple, musculosketetal; green, primary headaches; orange, mixed, or unknown.

gnosis of facial pain and guidelines for management *BJA British Journal* e 1, 1 July 2013, Pages 95–104, https://doi.org/10.1093/bja/aet125

Diagnostic procedures consist of nerve blocks aimed to isolate the peripheral nerve implicated, whereas therapeutic interventions either modify or destroy nerve function.

The role of neural blockade as a **diagnostic tool** in painful conditions may be compromised due to several characteristic of chronic pain including;

- social, emotional, financial, and legal factors effecting the patient
- the pathophysiology of clinical pain
- the site of nociception
- the pathway of afferent neural signals.

Information gained from blocks may then be applied to the choice of medicines, **therapeutic blocks**, or surgical therapy or neuroablative therapies.

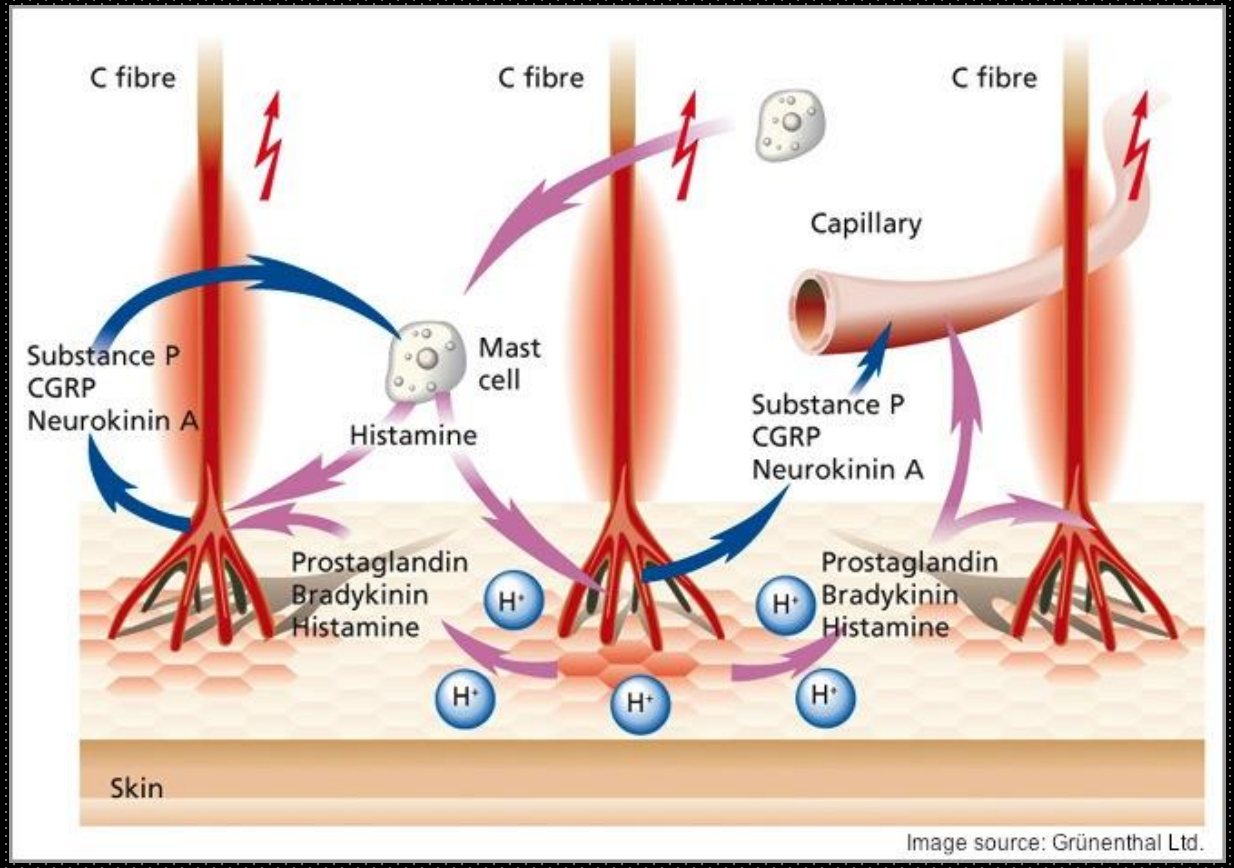
Biopsychosocial model of pain
Championed by Butler and Moseley and others. 2000

Diagnostic blocks issues

- However, there is limited critical examination of the theoretic basis on which diagnostic blockade rests, nor an evaluation of the published support for the diagnostic use of neural blockade.
- The diagnostic use of neural blockade rests on three premises.
  - First, pathology causing pain is located in an exact peripheral location, and impulses from this site travel via a unique and consistent neural route.
  - Second, injection of local anaesthetic totally abolishes sensory function of intended nerves and does not affect other nerves.
  - Third, relief of pain after local anaesthetic block is attributable solely to block of the target afferent neural pathway. The validity of these assumptions is limited by complexities of anatomy, physiology, and psychology of pain perception and the effect of local anaesthetics on impulse conduction.

Nociceptor activity

- Nociceptor Activity
  - Although pain perceived in somatic structures is generally associated with activation of nociceptors, peripheral nerve activity associated with pain perception **also may arise from injured nerves independent of nociceptor activity**.
  - Dorsal root ganglia of injured nerves participate in abnormal impulse generation. **Blockade of such nerves proximal to the injured segment but distal to the dorsal root ganglion may not relieve pain if spontaneous activity continues at the level of the dorsal root ganglion.** This may lead to the false assumption that the injured nerve is not responsible for the patient's pain.



Afferent and efferent interactions

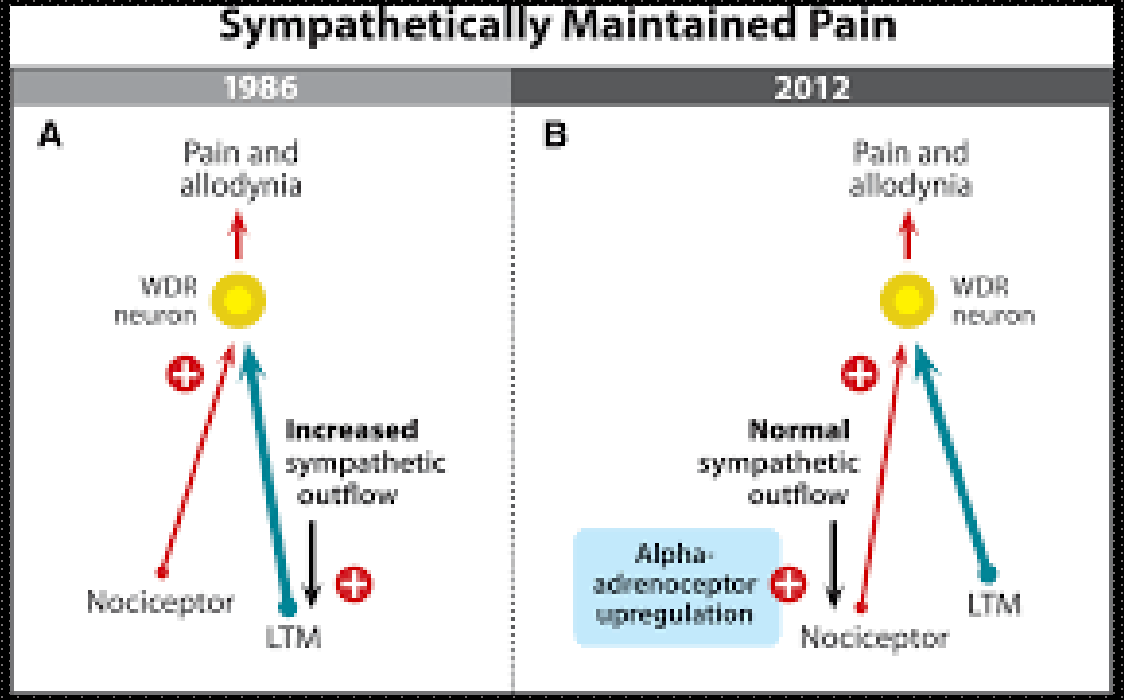
- NBs are effective on afferent neural activity, but important efferent traffic must be considered.
- **Impulse generation arising from an injured nerve fibre is likely to be propagated both orthodromically toward the spinal cord and antidromically toward the innervated tissues.**
- Therefore, nerve block distal to the primary site of nerve pathology may alter pain perception by interrupting antidromic impulses, contrary to the common assumption that axonal function must be interrupted proximal to the area of injury to provide relief.
- For example peripheral blockade of the sciatic nerve has been shown to provide profound relief of pain for patients with documented lumbosacral radiculopathy, perhaps by blocking antidromic impulses that arise from the nerve root or dorsal root ganglion and are propagated to the periphery, producing changes in nociceptor sensitivity.

Sympathetic contributions

When sympathetic motor activity is blocked during diagnostic procedures, such as with most peripheral and central nerve blocks, sympathetic influences on sensory mechanisms should be considered.

Injured nerves

- Receptors at the terminals of C fibres from an injured nerve become excited during sympathetic stimulation or norepinephrine application and show enhanced responsiveness to irritating stimuli.
- At the site of the nerve injury, sympathetic efferent impulses may depolarize nociceptive afferent fibres (ephaptic transmission), potentially producing both orthodromic and antidromic activity.
- Increased sympathetic activity or high levels of norepinephrine increase discharge rates of spontaneous impulses arising from neuromas, and injection of epinephrine in the vicinity of neuromas in patients with pain aggravates pain.

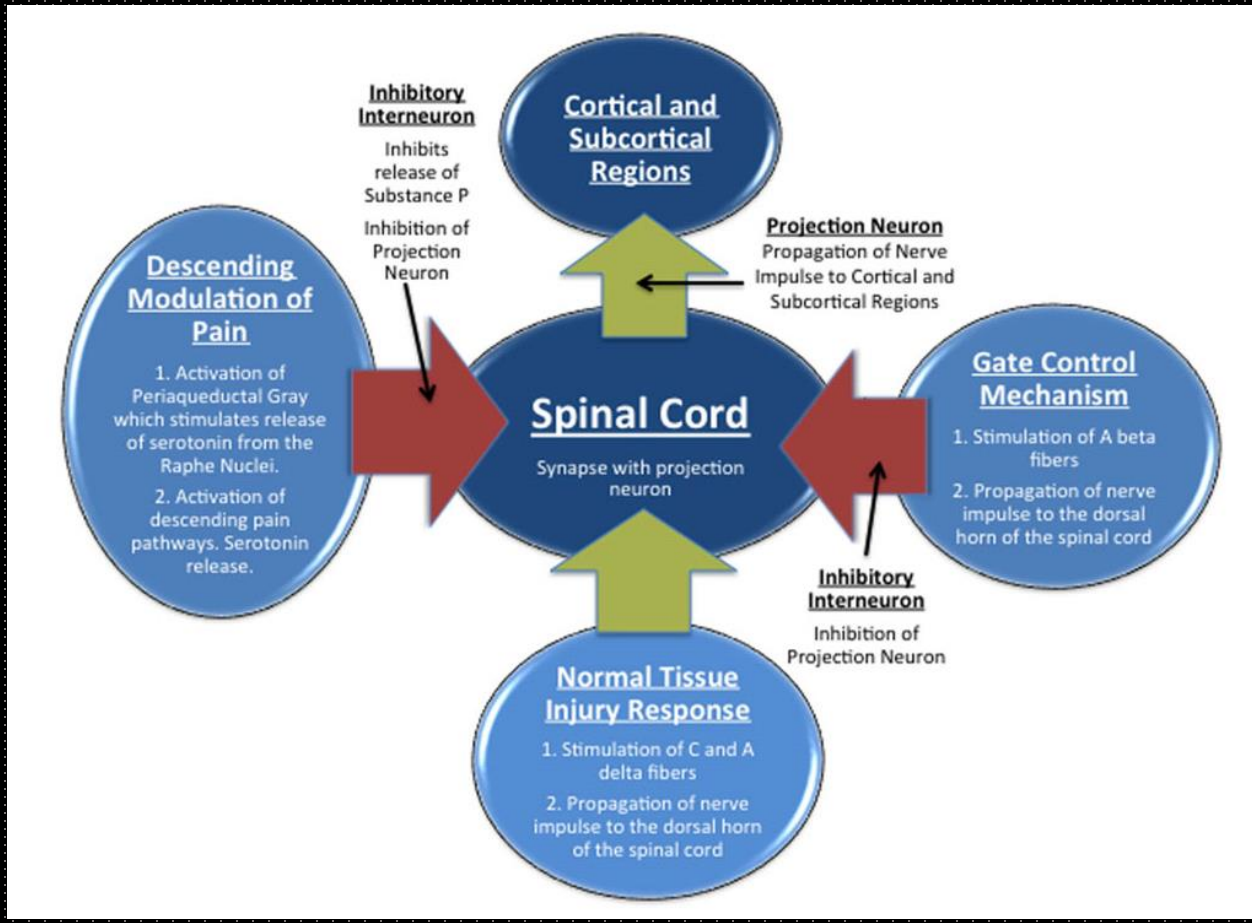


It is well accepted that sympathetic supply can modulate sensory responses in **uninjured nerves**, but the role of this mechanism in producing pain is less certain.

- Mechanoreceptor sensitivity is heightened by increases in sympathetic discharge rates, and aberrant central processing of these signals by sensitized wide dynamic range (WDR) neurons in the dorsal horn may result in the allodynia
- Pain relief after peripheral block may be due to interruption of any of these efferent mechanisms rather than somatic sensory fibres.

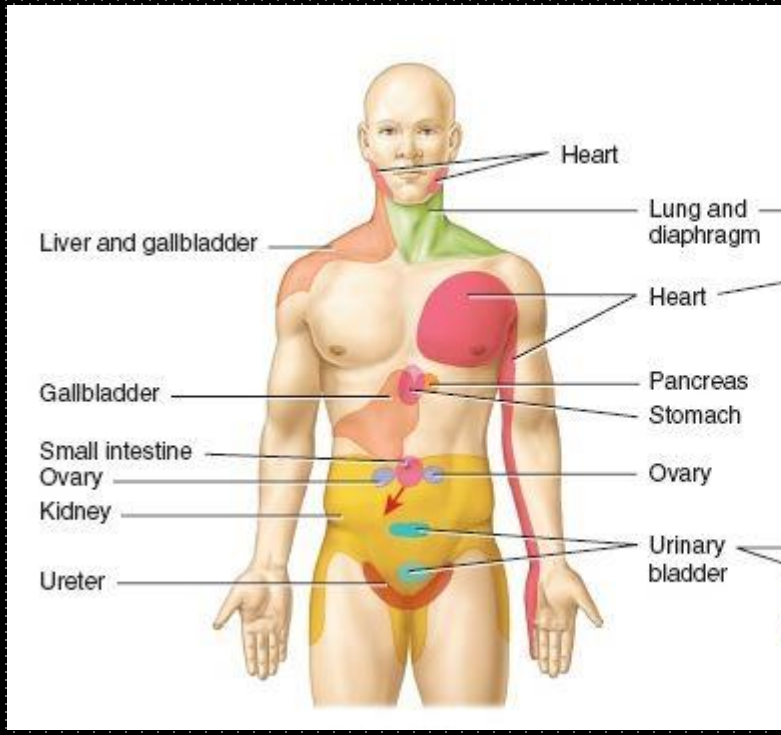
Spinal Processing

- **Variable processing in the spinal cord** involving a balance between large and small fibre inputs is an important determinant of the response of dorsal horn neurons to noxious stimulation.
- Conceivably, **loss of large fibre activity after peripheral or neuraxial blockade could increase dorsal horn cell activity**, particularly if there is preservation of C-fibre input, producing a paradoxical increase in pain.
- Conversely, it is likely that **mechanical allodynia** in neuropathic pain states is **conveyed by large fibre (A beta) input.**
- A **diagnostic block that interrupted small, but not large**, fibres **could fail to relieve touch-evoked pain** even if the remainder of the extremity is insensitive to nociceptive or thermal stimuli, **whereas selective large fibre block would create the opposite effects.**



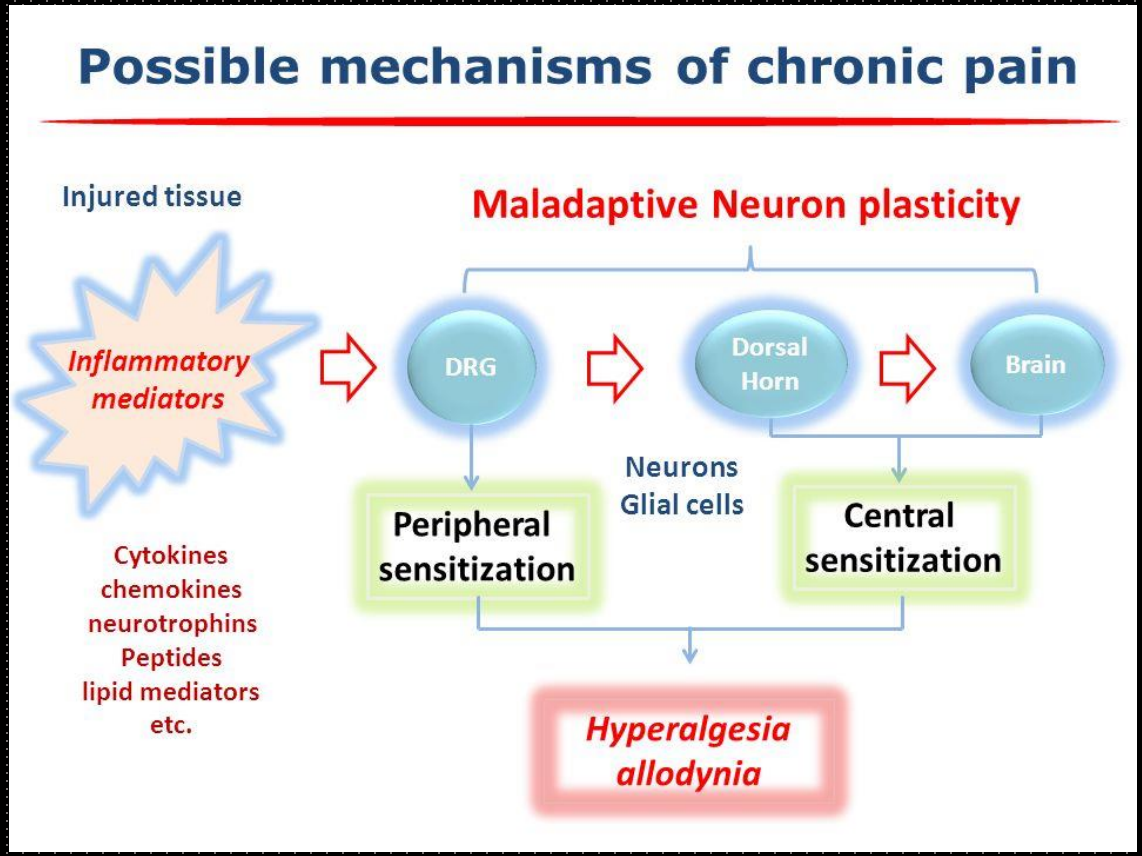
Convergence and Referred Pain

- Many second-order neurons in the spinal cord respond to a variety of input from primary afferents with either visceral and somatic receptive fields, an example of convergent input.
- In other instances, convergence is the result of primary afferent C-fibres that have both visceral and cutaneous collaterals.
- When afferent input arises from both somatic and visceral structures or from separate somatic foci, the perception of pain may depend on a level of combined neuronal activity from both components.
- **Interruption of one limb of the convergent inputs may be sufficient to provide complete pain relief, leading to false assumptions about the source of the pain.** For instance, a patient with pain of pancreatic cancer may have nociceptive inputs from splanchnic nerves plus from myofascial pain in the paravertebral muscles.
- **Infiltration of a painful trigger point in the affected muscle may reduce the combined input to a level below the pain threshold, and the mistaken interpretation would be that the pain is entirely somatic, without any visceral source.**



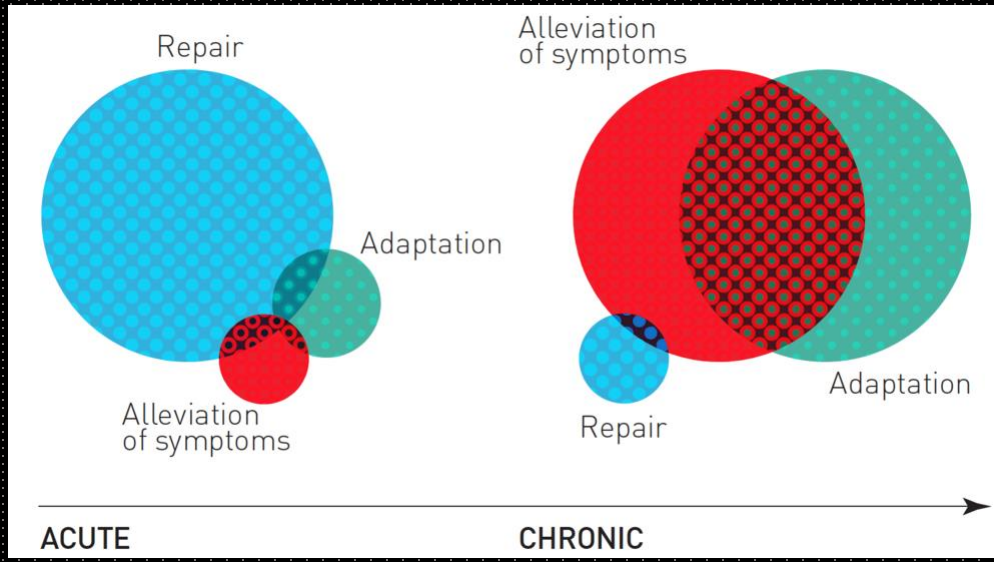
Plasticity

- Sensory processing is not stable but depends on preceding events, a phenomenon called neuronal plasticity.
- Small fibre (nociceptive) activity initiates a series of events in the dorsal horn that leads to heightened responsiveness of second-order neurons that are activated by noxious stimuli.
- Sensitization in response to noxious stimulation is known to affect WDR neurons, which ordinarily respond at very low firing rates to non-noxious inputs and at high firing rates to nociceptor activity. After sensitization, these cells may respond to non-noxious stimuli at sufficiently high firing rates to cause pain perception (allodynia).
- **It is impossible to predict responses to local anaesthetic blockade of afferent impulses under conditions of dorsal horn sensitization**.
- Afferent blockade of conditioning stimuli could lead to **normalization of dorsal horn responsiveness and profound, prolonged relief**. In other circumstances, however, **spinal sensitization might persist independent of afferent activity, with little or no change in pain.**



Plasticity after injury

- Decreased afferent input also can lead to functional changes in the dorsal horn. After periods of deafferentation, cells that respond to noxious stimulation become hypersensitive to remaining afferent inputs, and their receptive field may expand.
- Denervation of peripheral afferent fibres has been shown to cause dramatic functional changes in responses of WDR neurons in the dorsal horn.
- Denervation may additionally produce sufficient sensitization of WDR neurons that non noxious stimulation, including stimuli from outside the original receptive field, can produce pain. **Blockade of such stimulation could falsely indicate the site of pathology.**
- **Alternatively, blockade of an injured nerve may not provide relief of pain and allodynia if the receptive field of sensitized dorsal horn neurons has spread beyond the distribution of the injured nerve, again leading to the mistaken conclusion that the injured nerve is not involved.**



Criteria for Interpretation of the Outcome of PNB

Ratios Describing Efficacy of Tests

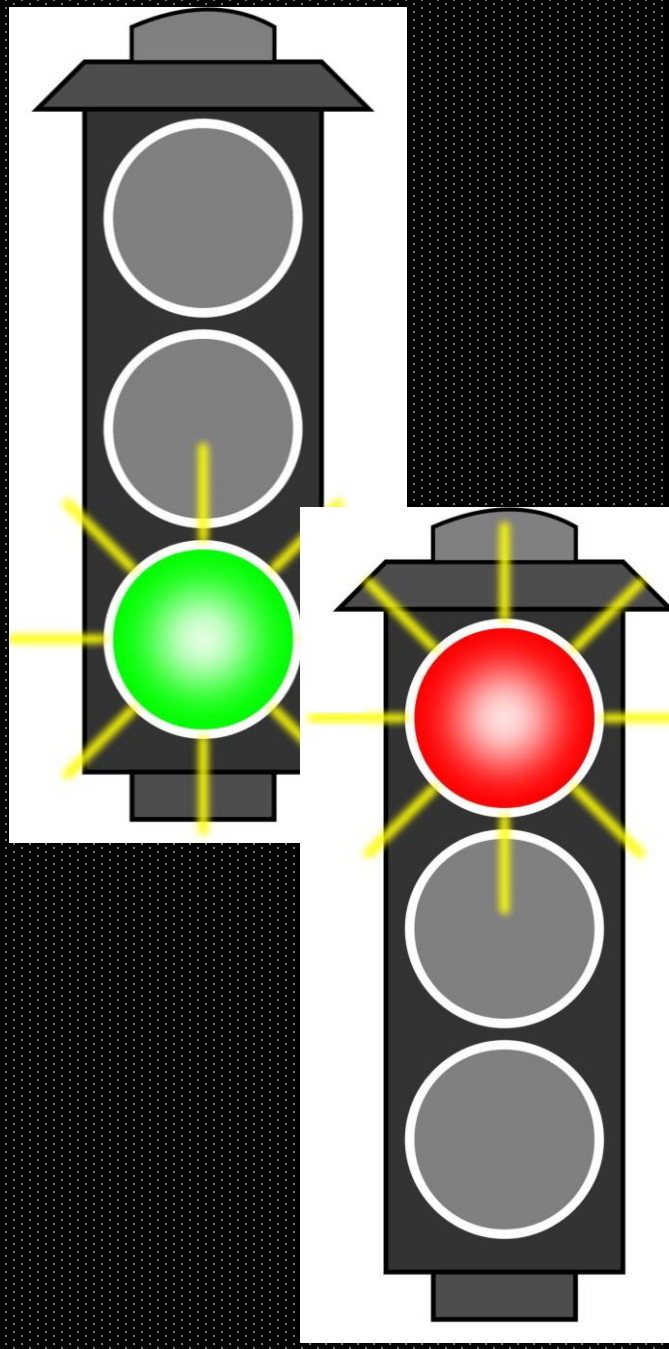
	Disease Present	Disease Absent
Test positive	a	c
Test negative	b	d
Sensitivity (true-positive rate)	=	a/(a + b)
False-positive rate	=	c/(c + d)
Specificity (true-negative rate)	=	d/(c + d)
False-negative rate	=	b/(a + b)
Positive predictive value	=	a/(a + c)
Negative predictive value	=	d/(b + d)

The proper interpretation of a positive test must take into consideration the prevalence of the condition. **For example, a test with a 95% specificity rate will have a positive result in 5% of healthy subjects**. If the condition being sought is rare (e.g., occurs in only 2% of the test group), false-positive responses will outnumber true-positive tests, and the majority of positive results will occur in subjects who actually are healthy.

Hogan QH, **Abram** SE. Neural blockade for diagnosis and prognosis. A review. Anesthesiology. 1997 Jan;86(1):216-41. Review.

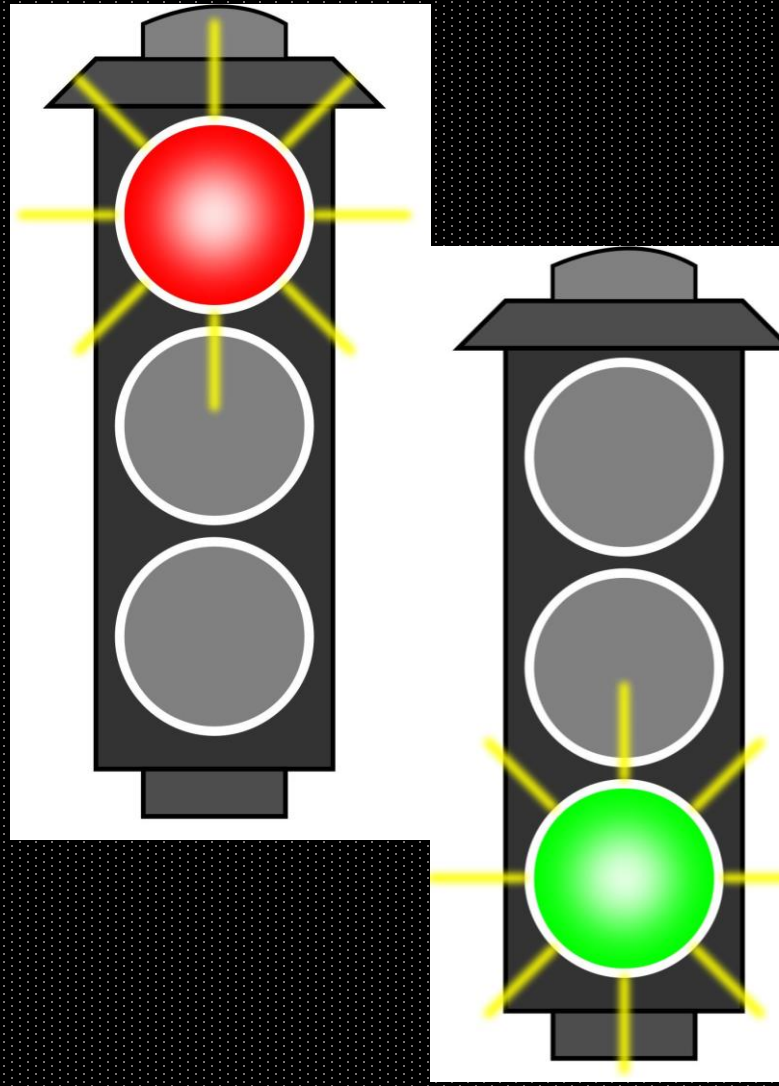
Therefore.....

- **False positive response to PNB**
  - A nerve block distal to the primary site of nerve pathology may alter pain perception by interrupting antidromic impulses, contrary to the common assumption that axonal function must be interrupted proximal to the area of injury to provide relief.
  - If pain relief follows sympathetic blockade, lack of anaesthesia to touch does not assure that pain relief is by sympathetic interruption, because a subtle somatic block could produce analgesia without anaesthesia, resulting in pain relief independent of a sympathetic mechanism.
  - A selective large fibre block would create eliminate touch evoked pain but not response to nociceptive or thermal stimuli
  - Interruption of one limb of the convergent inputs may be sufficient to provide complete pain relief, leading to false assumptions about the source of the pain.
  - Infiltration of a painful trigger point in the affected muscle may reduce the combined input to a level below the pain threshold, and the mistaken interpretation would be that the pain is entirely somatic, without any visceral source.
  - Afferent blockade of conditioning stimuli could lead to normalization of dorsal horn responsiveness and profound, prolonged relief.
  - Denervation may additionally produce sufficient sensitization of WDR neurons that non noxious stimulation, including stimuli from outside the original receptive field, can produce pain. Blockade of such stimulation could falsely indicate the site of pathology.



Therefore.....

- **False negative response to PNB**
  - Dorsal root ganglia of injured nerves participate in abnormal impulse generation resulting in poor response to PNBs this may increase after peripheral blockade
  - Conceivably, loss of large fibre activity after peripheral or neuraxial blockade could increase dorsal horn cell activity, particularly if there is preservation of C-fibre input, producing a paradoxical increase in pain
  - A diagnostic block that interrupted small, but not large, fibres could fail to relieve touch-evoked pain even if the remainder of the extremity is insensitive to nociceptive or thermal stimuli.
  - Afferent blockade of conditioning stimuli with spinal sensitization pain might persist independent of afferent activity.
  - blockade of an injured nerve may not provide relief of pain and allodynia if the receptive field of sensitized dorsal horn neurons has spread beyond the distribution of the injured nerve, again leading to the mistaken conclusion that the injured nerve is not involved.



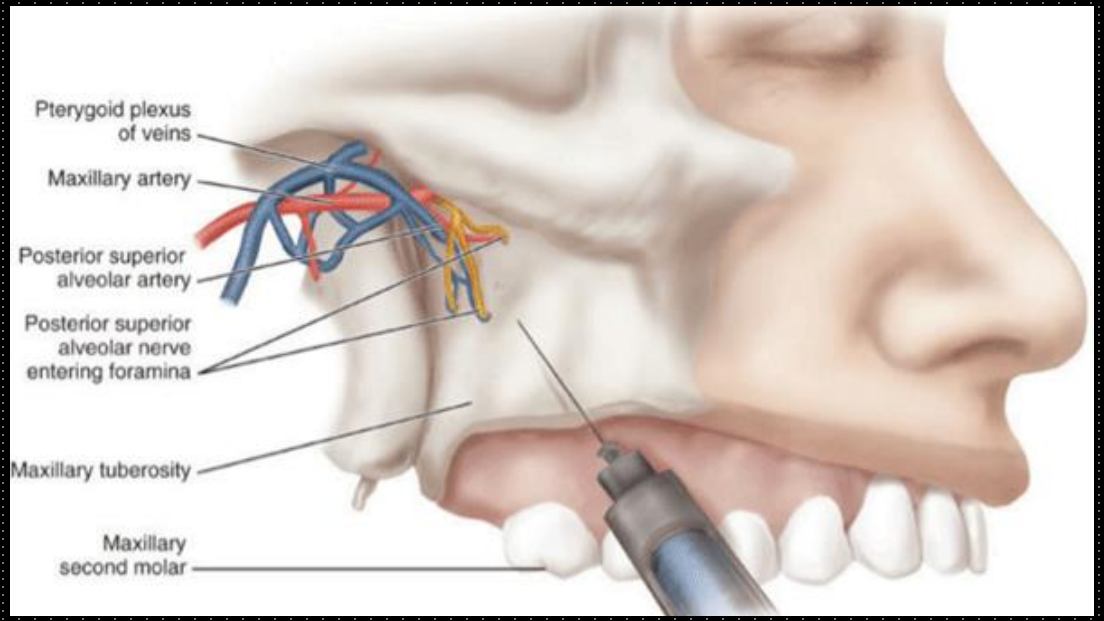
And yet more confounding factors……

Practical issues with Periperhal LA block

Consideration of the subtle, complex, and variable action of local anesthetics should inspire caution in the interpretation of blocks.

Variability may be due to;

- Block Infiltration
- Type LA agent, adjunctive, volume
- Intensity of Blockade
- Differential block
  - Fibre type
  - Degree of blockade
- Systemic effects
- Psychosocial Issues
- Site -Anatomical issues
  - Evidence indicates that anatomic uncertainties with regard to neural connections and structural variability degrades the accuracy of diagnostic information obtained by neural blockade.

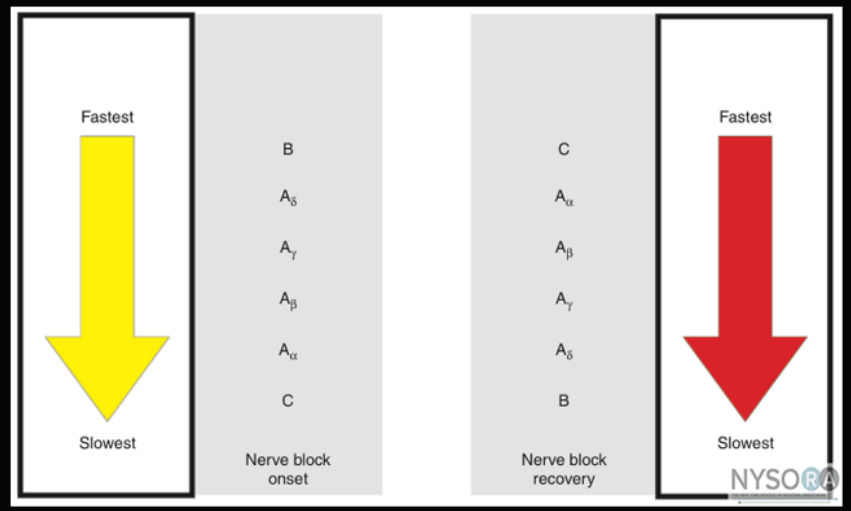


Hogan QH, **Abram** SE. Neural blockade for diagnosis and prognosis. A review. Anesthesiology. 1997 Jan;86(1):216-41. Review.

Intensity of Blockade

Diagnostic and prognostic blocks are accomplished by the action of local anaesthetics on nerves.

- **It has long been recognized that neural blockade is not an all-or-none response**.
- For instance, **analgesia is usually evident earlier, and to a greater extent, than loss of perception of mechanical stimuli after peripheral neural blockade**.
- In the opposite sense, **apparent intense blockade with complete insensitivity to touch and pain is nonetheless not a complete afferent blockade, because studies of different types of blocks with various agents uniformly demonstrate incomplete elimination of somatosensory potentials evoked by stimulation of the anesthetized region.**
- If pain continues after a diagnostic block, one cannot be certain that the injected pathway is not involved, because neural blockade is often not absolute.
- Skin conduction responses, a manifestation of sympathetic action at sweat glands, is often present in areas of apparently complete somatic blockade, weakening the predictive value of sympathetic blocks, unless monitoring confirms the loss of sympathetic activity in the affected area concurrent with the onset of relief.



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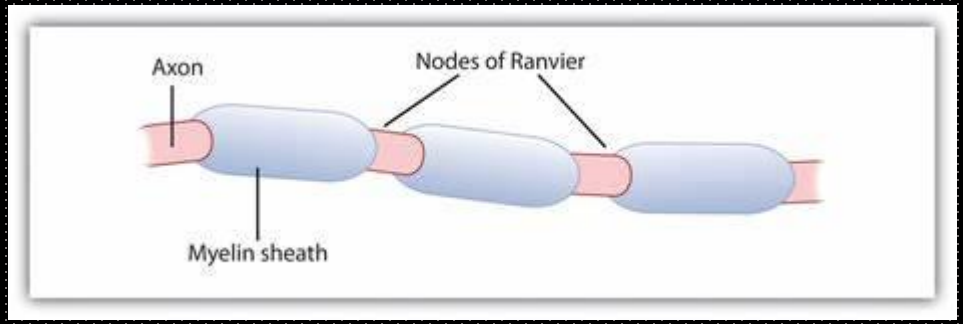
Differential Block

Type of Fiber	Myelination	Fiber size (in microns)	Speed of Conduction (m/sec)	Sensations associated with Nerve Fiber	Sensitivity
B	Yes	3	3-14	Pre-ganglionic & autonomic	4
A-delta	Yes	1-5	6-30	Sharp pain & temperature	3
C	No	0.5-2	0.5-2	Dull pain, temperature, & crude touch	3
A-gamma	Yes	1-10	6-60	Deep pressure & touch	2
A-beta	yes	5-15	30-90	Vibration, deep pressure, touch	1
A- alpha	Yes	10-20	60-120	Muscle length & force	1

- The variable effects of local anesthetics on fibres conveying different functions is termed differential block.
  - **fibre size**, which predicts that small, nonmyelinated C fibres are the most sensitive to local anesthetics, followed by small myelinated B fibres, whereas large myelinated A fibres are the most resistant. Despite the appealing simplicity of this model, it has not withstood the test of time.
  - **intrinsic sensitivity** of nerve fibre types to local anesthetics is probably A greater or equal to B > C.
  - **range conduction speed** and, therefore, fibre size within a fibre type, and the lack of correlation of size and necessary anesthetic concentration for blockade (CM) within the group.
  - The overlap between different groups “appears to negate any possibility of obtaining steady state differential interruption” by local anesthetics.
  - Difference in **diffusion barriers of the various fibre types** probably explains a large part of clinically evident differential effects.

Degree of blockade



- To prevent conduction, at least three nodes of Ranvier in succession must be blocked completely. If local anesthetic is limited in longitude, large fibres with long internodal distances may lack exposure to three nodes, whereas smaller fibres have the necessary three nodes exposed and are blocked.
- At concentrations that produce incomplete sodium channel blockade, the influence of exposure length extends several centimeters, and CMis inversely related to exposed nerve length.
- These phenomena dictate that anesthetic potency and the degree of differential effects varies with the length of nerve exposed, an added variable that is hard to control.

Further subtle influences on local anesthetic action may cloud the interpretation of diagnostic blocks.

- Sodium channel closure by local anesthetics depends on nerve use.
  - The block that develops when the axon is firing at very low rates (tonic block) is less intense than the block that develops while the nerve is active (phasic block).
  - Local anesthetic will affect more completely those fibres that are most active. The spectrum of anesthetic effects will, therefore, depend on the pattern of activity of the subject's various neuron types when the diagnostic block is undertaken.
  - Because the earliest perturbation of nerve function at very low anesthetic concentrations is prolongation of the latent interval for refiring, information encoded with bursts will be transformed into a more uniform signal.

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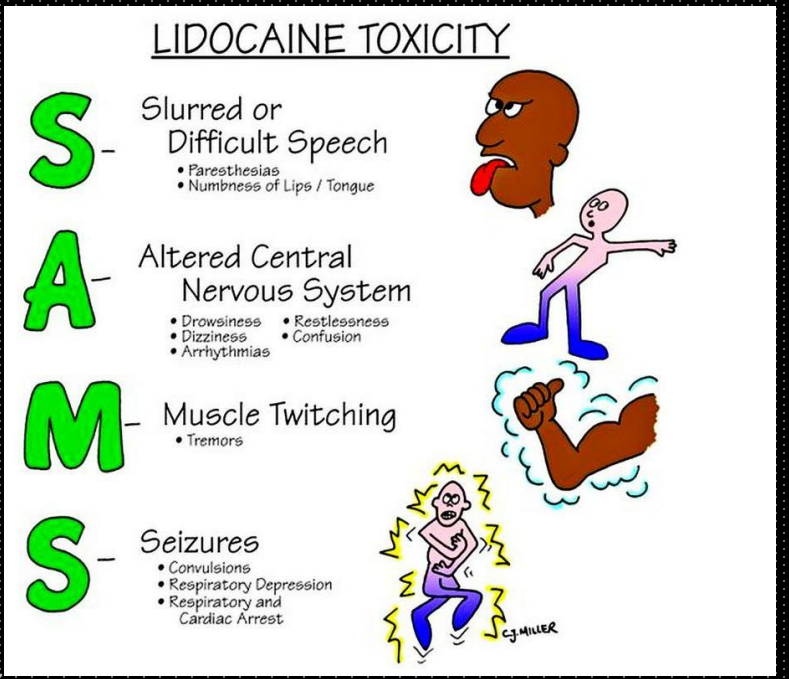
Systemic Effects

- Local anesthetic is absorbed from the site of injection during diagnostic blockade, raising the question of a systemic analgesic contribution.
- At local anesthetic blood concentrations that are insufficient to produce side effects in humans (e.g., 1–5 micro gram/ml lidocaine), there is little or no appreciable effect on impulse conduction in normal peripheral nerves or on cutaneous C-fibre terminal function.
- However, there is considerable evidence that systemically administered local anesthetics affect spontaneous and mechanically stimulated impulse generation arising from injured nerves.
- Nontoxic doses of systemic local anesthetics also depress spinal transmission of nociceptive inputs, but the principal effect of systemic local anesthetics on neuropathic pain is peripheral.
- There have been several clinical reports of the efficacy of intravenous lidocaine in patients with neuropathic pain. Whereas some cite very transient effects, others indicate analgesic effects that last several days or longer. Doses of local anesthetic required to relieve neuropathic pain are generally 1–3 mg/kg. It would be unlikely, therefore, that a selective nerve root block with 3 ml 1% lidocaine (30 mg) would produce pain relief by a systemic effect. In contrast, a lumbar sympathetic block using 15 ml 1% lidocaine might relieve neuropathic pain at a location distant from the site of injection.



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Psychosocial Issues

The potency and frequency of the placebo effect is underestimated by the majority of physicians and nurses.

To diminish ambiguities created by these psychosocial factors, a physician might choose to inject a placebo, an inert substance with no known pharmacodynamic effect. Interpretation of a favourable response to a placebo is problematic.

Patients obtain relief from placebos administered during acute pain approximately one third of the time, but obtain relief from chronic pain in approximately two thirds of cases after administration of a placebo.

For instance, **in patients with causalgia, 3 ml of subcutaneous normal saline relieved spontaneous pain in 68% of patients, and also relieved mechanically induced allodynia in 56% and Tinel's sign (a tingling sensation in the distal end of a limb during percussion of the injured nerve) in 67%.** Probability of analgesia from a placebo is proportionate to the intensity of pain.

No personality features predict a placebo response, individuals are not consistent in being responders or non-responders, and most individuals will eventually respond to a placebo if administered repeatedly.

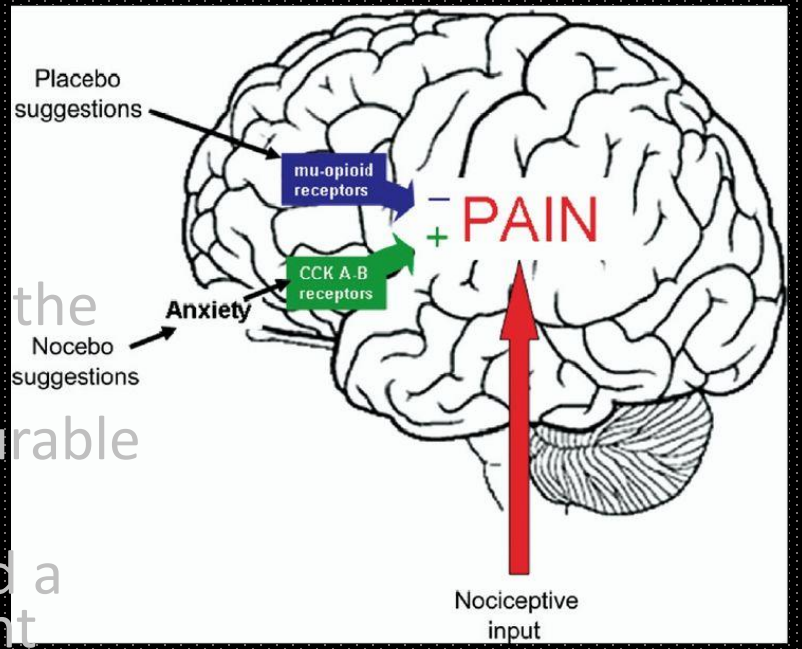
Placebo action may be as intense as the active agent, usually mimics the active agent in dose-response and time-effect relations, and may develop over as prolonged an interval as 60 min.

Injections, like surgery, are especially potent placebos compared with pills.

The same problems that accompany intentional placebo use make it difficult to determine whether analgesia after a diagnostic block with an active agent is, nonetheless, a placebo response.

Hogan QH, **Abram** SE. Neural blockade for diagnosis and prognosis. A review. Anesthesiology. 1997 Jan;86(1):216-41. Review. [60,61]

The Placebo effect



- Psychologic theory that explains placebo response focuses on the subject's expectations and on conditioning.
- In the context of diagnostic blocks, the expectation of a favourable response may make analgesia more likely.
- Most subjects can be trained to have a placebo response, and a placebo response is more likely if the test with the active agent precedes the placebo administration.
- It is evident that the physician's convictions play a large role in generating placebo responses, and that, even in carefully blinded protocols, unintended communication from the examiner to the subject takes place.
- On a neurophysiologic level, the placebo response is a demonstration of descending modulation of nociception.

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Placebo

Compelling evidence with regard to placebo responses leads to the conclusion that the ambiguity created by these responses is a major impediment to the valid use of neural blockade for diagnosis.

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Anatomic issues

- The use of blocks for diagnosis and prognosis depends on an assumption of anatomic consistency. **The sensory innervation of a particular site cannot be assigned, with certainty, to any segmental level,** and sensory changes after local anaesthetic injections near the vertebral column are variable.
- **Most anatomic parameters show variability about a norm**. Surface and palpation landmarks are unreliable indicators of deep structures, which is borne out by a 50% accuracy in guessing vertebral level of needle placement without x-ray imaging.
- **In a variety of injection procedures, accurate needle placement requires imaging. Idealized textbook descriptions of anatomic structures hold in only approximately 50–70% of actual subjects.**
- Separation of somatic input into a discernible segmental pattern is a fundamental concept that underlies many diagnostic blocks. There is, however, variability in the formation of segmental spinal nerves and their peripheral distribution.
  - Multiple interconnections of adjacent rootlets and roots are found within the dural sac in all subjects, with between 3 and 9 such intersegmental anastomoses at the upper cervical region and a similar number at the lumbosacral level. [
  - The pattern of spinal nerve contributions to the limb is highly inconsistent.
  - The distribution of spinal nerve root fibres to the skin has been mapped using zoster eruptions, residual sensation after sectioning the roots on either side of an intact segment, absent sensation after root section or anaesthesia, vasodilatation during stimulation of roots, or pain with nerve root compression and visceral disease.
  - The dermatome diagrams these methods produce show considerable disagreement, especially in the extremities. Also, extensive overlap between consecutive peripheral dermatomes is evident because the division of an individual root rarely produces an appreciable loss of sensibility.

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Type of pain pathophysiology

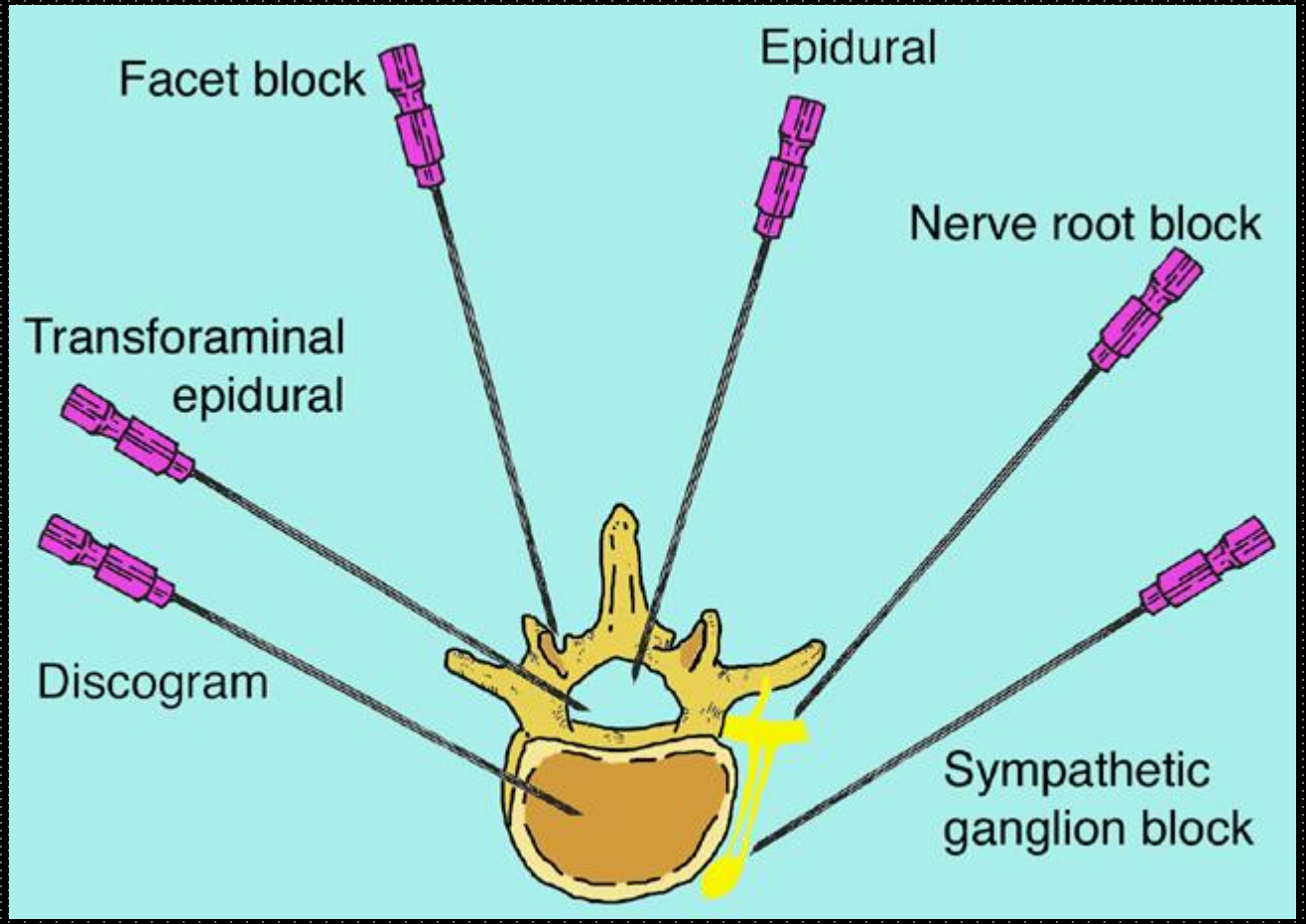
- Deep somatic pain from bones, joints, muscles, and fascia shares many features of visceral pain, including poor somatotopic localization, referred pain, and a generalized increase in central nervous system excitability with motor and autonomic reflexes.
- In addition, the fibres from many deep somatic elements (costovertebral joint, posterior and anterior longitudinal ligaments, annular ligament of the intervertebral disc, and dura) traverse the sympathetic rami and chain.
- It is likely that some pains relieved by sympathetic blocks are unrelated to sympathetic efferent activity (sympathetically maintained pain) and, instead, are deep somatic pains transmitted by sympathetic pathways.

PAIN CATEGORY	DEFINITION AND EXAMPLES	CHARACTERISTICS
Somatic	Pain resulting from injury to or inflammation of tissues . Examples: burns, lacerations, fractures, infections, inflammatory conditions	In skin and superficial structures: sharp; pulsatile; well-localized In deep somatic structures: dull; aching; pulsatile; not well-localized
Visceral	Pain resulting from injury to or inflammation of viscera Examples: angina, hepatitic distention, bowel distention or hypermobility, pancreatitis	Aching and cramping; nonpulsatile; poorly localized (e.g., appendiceal pain perceived around umbilicus) or referred to distant locations (e.g., angina perceived in shoulder)
Neuropathic	Pain resulting from injury to, inflammation of, or dysfunction of the peripheral or central nervous systems. Examples: complex regional pain syndrome (CRPS),	Spontaneous; burning; lancinating or shooting ; pain may be perceived distal or proximal to site of injury, usually corresponding to innervation pathways (e.g., sciatica)

Hogan QH, **Abram** SE. Neural blockade for diagnosis and prognosis. A review. Anesthesiology. 1997 Jan;86(1):216-41. Review.

Thus there are many issues with nerve diagnostic blocks for assessment for therapeutic interventions

- Refractory patients
- Lack of evidence
- Psychological overlay
- Pain mechanisms



Somatic Nerve Block Rationale.

A common reason to perform diagnostic peripheral nerve blocks is to predict the success after surgical decompression or neurolysis of a peripheral nerve.

- Diagnostic blocks also may be performed before a planned peripheral nerve section, neurolytic block, or cryoanalgesia lesion.
- Somatic nerve block also may be used to predict outcome after decompression of entrapment neuropathies such as of the digital nerve (Morton's neuroma) and the median nerve in the carpal tunnel.

Consent and training

What basic principles should be followed to ensure a safe and successful peripheral nerve block?

- Patients should be informed about the potential risks and benefits of PNB and allowed to decide on the anaesthetic they prefer.
- Not all patients are good candidates for regional anaesthetics. For example, performing PNB after trauma or on highly anxious patients.
- The clinician must have knowledge of the anatomy, technique, and equipment necessary to perform the most appropriate block for a given situation.
- The use of aseptic technique, correct equipment (B-bevel needles, nerve stimulators, ultrasound), and basic physiologic monitoring is mandatory.
- The area in which the PNB is performed should have immediate access to resuscitative equipment and medications.

Minimise risks

- It is essential to ensure correct needle placement
  - Avoid intravascular placement
  - Avoid epineural and or intraneural placement
  - Always aspirate
- Knowledge of the anatomy of the target region and the surrounding structures is necessary.
- Knowledge of the equipment and the pharmacology of local anesthetics is also required.
- Do not perform a PNB with which you are unfamiliar or not trained to do.

Risks

- There may be inadvertent damage to anatomic structures by the advancing needle. Examples include
  - direct trauma to the nerve or spinal cord by intraneural injection of local anesthetic,
  - nerve laceration
  - vascular injury with resulting hematoma formation
- The drugs that are injected may have undesirable local and systemic effects.
  - Allergic reactions to local anesthetics are rare. Ester local anesthetics are derivatives of paraaminobenzoic acid, a known allergen, and therefore more likely to cause allergic reactions than the amide local anesthetics.
  - Any local anesthetic injected intravascularly has the potential for systemic reactions, including seizures and cardiovascular collapse.

Table 5 Potential adverse reactions to anaesthetic blocks and recommended actions.

Potential adverse effects	Actions
Local pain	Perform infiltration slowly, with fine-gauge needle. Avoid lateral motions. Limit steroid use. Local cold application.
Lesion to peripheral nerve	If patient experiences sharp radiating pain, remove needle and insert again.
Haematoma	Be aware of any anticoagulant or antiplatelet drugs. Palpate to avoid the temporal and occipital arteries. Apply local compression for several minutes.
Local infection	Avoid infiltration if infection is present. Aseptic measures (sterile technique, local antiseptic)
Vasovagal syncope	Where possible, no blockades on fasting patients Consider performing nerve block on the patient in a decubitus position; delay return to a standing position if the situation recommends it. Limit the number of nerves to be blocked in a single session. For elderly patients or those with a history of syncope, avoid lidocaine at high doses (5%). In a vasovagal episode, place patient in the Trendelenburg position; if no response, start atropine and fluid replacement.
Allergy to local anaesthesia	Anaesthetic block is contraindicated in patients with a history of drug allergies. Limit treatment to corticosteroids in these cases. For anaphylactic shock, 0.3-0.5 mg adrenaline, life support, and transfer
Intradural infiltration	Nerve block is contraindicated in cases of craniectomy or open cranial defect.
Teratogenicity	In pregnancy: – Lidocaine preferred to mepivacaine – Avoid betamethasone and dexamethasone (they accelerate development of foetal lungs). – Exercise caution with any corticosteroid
Local anaesthetic systemic toxicity	Use small doses and volumes. Avoid intravascular infiltration.
Alopecia Corticoid-induced dermal atrophy Hypochromia	Avoid infiltrating the trigeminal branches with corticost Alert patients to potential aesthetic changes. No methylprednisolone doses higher than 80 mg in the o

Procedure Patient information

NHS
Tameside and Glossop Integrated Care
NHS Foundation Trust

Trigeminal Nerve Block For Non Acute Pain

Patient information Leaflet

February 2017

Please read this leaflet carefully.

If you do not follow the instructions given your procedure may be cancelled

What is a Trigeminal Nerve Block?

The **trigeminal nerve** is a nerve responsible for sensation in the face and certain motor functions such as biting, chewing, and swallowing. Irritation of this nerve causes an increase in the number of messages sent to the brain leading to pain. A Trigeminal Nerve block involves the injection of local anaesthetic medication and sometimes steroids into the area surrounding the Trigeminal nerve. This blocks the pain messages thereby reducing the amount of pain felt. The effect of the block is usually temporary, but the benefit can sometimes be prolonged.

Your procedure will be performed under X-Ray guidance. If you are female please ensure there is no risk of you being pregnant on the day of your procedure. Please contact the Pain Nurse if you have any concerns.

What Are The Benefits?

- Temporary relief of pain, however the longer the symptoms have been present, the less successful the outcome.
- Aid in diagnosis

What are the possible side effects/complications of the procedure?

All procedures in medicine carry a risk of complications. Precautions are always taken to minimize the risk as far as possible. Generally injections are safe but occasionally the following risks may occur:

- Failure of procedure to help
- Worsening of pain (Temporarily or permanently)
- Bleeding/ bruising to the injected area e.g. around the eyes
- Infection
- Hypotension/ low blood pressure
- Temporary numbness or paralysis of the tissue around the mouth and face.
- Allergic Reactions
- Dizziness/light headedness/fainting
- Roaring in the ears
- Nerve Damage
- Local anaesthetic toxicity (rare)
- fitting

Potential side effects with using steroids

If steroids are used there are few side effects associated with either single or occasional use of steroids. These include hot flushes, feeling sick, mild abdominal pain, fluid retention, raised blood sugar in diabetics and occasionally menstrual irregularities. These symptoms should settle in a few days.

- If you take water tablets (Diuretics) on a regular basis then please take an extra water tablet the day after your procedure.
- If you are diabetic you should closely observe your diabetic control for the next fortnight.

Specific Procedures Assessment of effectivity

Clinical studies of the blocks are variable quality.

- Important considerations include **entrance criteria**, study size, and the use of control subjects.
- **Diagnostic criteria for condition**
- The **prevalence of placebo responses** in patients with pain greatly weakens the relevance of studies in which no control subjects or blinding was used.
- Where possible, neural blockade tests are evaluated numerically**, using standard definitions**.
- The importance of false-positive rate (how often patients without a condition will nonetheless have a positive test) and false-negative rate (how often a patient with disease will have a negative test for it) is evident because they vary inversely with specificity and sensitivity, respectively

- Condition (ICHD)
- Patient
  - Demographics
  - Psychology
  - Medical co morbidities
- Current treatment
- Previous treatment
- Region
- Diagnostic response
  - Onset
  - Duration
  - Intensity
  - Other signs

What is the evidence for diagnostic PNBs in pain diagnosis?

Non OFP Prediction of diagnostic blocks in treatment outcomes?

Anesth Analg. 2007 Dec;105(6):1756-75, table of contents.

The ability of diagnostic spinal injections to predict surgical outcomes.

Cohen SP[1], Hurley RW.

CONCLUSIONS: The ability to evaluate the effect of diagnostic blocks on surgical outcomes is limited by a lack of randomized studies, methodological flaws, and wide-ranging discrepancies with regard to injection variables, surgical technique, and outcome measures. More research is needed to optimize injection techniques and determine which, if any, diagnostic screening blocks can improv ical outcomes.

Nat Rev Rheumatol. 2013 Feb;9(2):101-16. doi: 10.1038/nrrheum.2012.198. Epub 2012 Nov 20.

Facet joint pain--advances in patient selection and treatment.

Cohen SP[1], Huang JH, Brummett C.

Author information

s, and sacroiliac (SI) joint injections. We garnered materials via MEDLIN gines, aphic references, and conference proceedings.

Cephalalgia. 2015 Apr;35(4):359-62. doi: 10.1177/0333102414541685. Epub 2014 Jul 2.

Occipital nerve block prior to occipital nerve stimulation for refractory chronic migraine and chronic cluster headache: myth or prediction?

Kinfe TM[1], Schuss P[2], Vatter H[2].

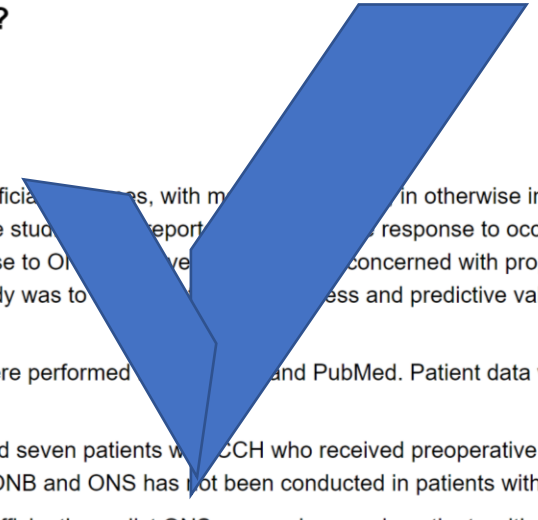
Author information

Abstract

BACKGROUND: Occipital nerve stimulation (ONS) results in beneficia s, with m in otherwise intractable chronic migraine (CM) and chronic cluster headache (CCH). Some stud report response to occipital nerve block (ONB) administered prior to ONS predicts a positive response to O ve concerned with proper patient selection claimed no predictive value for ONB. The aim of this study was to ess and predictive value of ONB prior to ONS.

METHODS: Literature searches on the predictive value of ONB were performed nd PubMed. Patient data were extracted and a pooled analysis was performed.

RESULTS: The literature review revealed 133 patients with CM and seven patients w CCH who received preoperative ONB. To date, a randomized controlled study examining the relationship between ONB and ONS has not been conducted in patients with CM.



Predictive value of a diagnostic block in focal nerve injury with neuropathic pain when surgery is considered

Martijn J. A. Malessy, Ralph de Boer, Ildefonso Muñoz Romero, Job L. A. Eekhof, Erik. W. van Zwet, Michel Kliot, Albert Dahan, Willem Pondaag

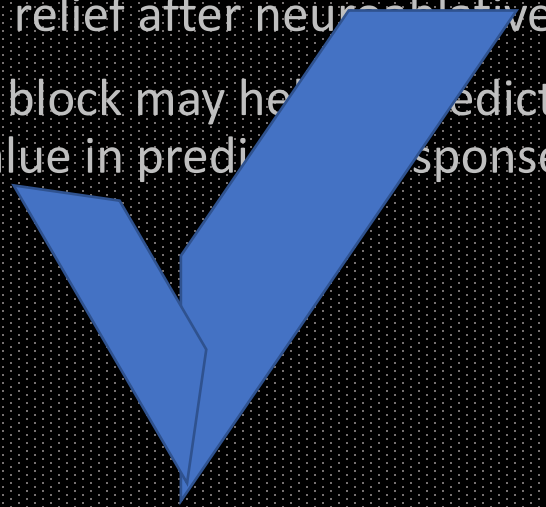
Published: September 12, 2018 • https://doi.org/1

conclude that screening blocks improve

Fig 4. Case illustration of a 63 year patient (number 8, Table 1) in whom blocking a nerve had no pain relieving effect, but nerve surgery had.
Following the current selection algorithm, this patient would not have been operated. The patient had complaints befitting ankle arthrosis. An isolated subtalar arthrodesis was performed by placing compression screws. Immediate postoperative, the patient had severe neuropathic pain with allodynia in the sural nerve area limiting the walking distance to around 150 meters. Conservative treatment failed. A: At inspection 38 months after the onset of the pain, a scar of the screw placement was seen around 4

Specific Procedures- PNB evidence Carpal tunnel

- The diagnostic use of injection of lidocaine and steroid has been examined in patients suspected to have carpal tunnel syndrome.
- The test identified most patients with the disease, demonstrated subsequently at surgery (sensitivity rate 85%), but it indicated lack of carpal tunnel syndrome in only 38% of those surgically negative (specificity).
- Even when peripheral local anaesthetic nerve block produces profound relief, there is poor prediction of long-term relief after neuroablative procedures.
- Relief of the peripheral block may help predict response to neural decompression, but has unproved prognostic value in predicting response to neuroablation.



Specific Procedures- PNB evidence Headaches

- Level II-IV for ONB in Migraine, Cluster headache
- Level II Cervicogenic HA
  - The best available studies indicate that the C2–3 zygapophysial joints are the most common source of cervicogenic headache,16,19,39,40 accounting for about 70% of cases.

Neurología. 2017;32(5):316–330

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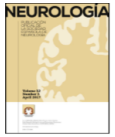

REVIEW

Consensus recommendations for anaesthetic peripheral nerve block☆

S. Santos Lasaosa[a,*], M.L. Cuadrado Pérez[b], A.L. Guerrero Peral[c], M. Huerta Villanueva[d], J. Porta-Etessam[b], P. Pozo-Rosich[e], J.A. Pareja[f]

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KEYWORDS
Anaesthesic block;
Cervicogenic headache;
Cluster headache;
Migraine;
Greater occipital nerve;
Pericranial neuralgia

Abstract
Introduction: Anaesthetic block, alone or in combination with other treatments, represents a therapeutic resource for treating different types of headaches. However, there is significant heterogeneity in patterns of use among different professionals.
Development: This consensus document has been drafted after a thorough review and analysis of the existing literature and our own clinical experience. The aim of this document is to serve as guidelines for professionals applying anaesthetic blocks. Recommendations are based on the levels of evidence of published studies on migraine, trigeminal autonomic cephalalgias, cervicogenic headache, and pericranial neuralgias. We describe the main technical and formal considerations of the different procedures, the potential adverse reactions, and the recommended approach.

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Cervicogenic headache: an assessment of the evidence on clinical diagnosis, invasive tests, and treatment

Nikolai Bogduk, Jayantilal Govind*

Cervicogenic headache is characterised by pain referred to the head from the cervical spine. Although the International Headache Society recognises this type of headache as a distinct disorder, some clinicians remain sceptical. Laboratory and clinical studies have shown that pain from upper cervical joints and muscles can be referred to the head. Clinical diagnostic criteria have not proved valid, but a cervical source of pain can be established by use of fluoroscopically guided, controlled, diagnostic nerve blocks. In this Review, we outline the basic science and clinical evidence for cervicogenic headache and indicate how opposing approaches to its definition and diagnosis affect the evidence for its clinical management. We provide recommendations that enable a pragmatic approach to the diagnosis and management of probable cervicogenic headache, as well as a rigorous approach to the diagnosis and management of definite cervical headache.

Lancet Neurol 2009; 8: 959–68
See Reflection and Reaction page 875

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*Dr Govind died on June 16, 2009
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Introduction

Cervicogenic headache is pain referred to the head from a source in the cervical spine. Unlike other types of headache, cervicogenic headache has attracted interest from disciplines other than neurology, in particular manual therapists and interventional pain specialists, who believe that they can find the source of pain among the joints of the cervical spine. Neurologists differ in their acceptance of this disorder. The International Headache Society recognises cervicogenic headache as a distinct disorder[1] and one chapter in a leading headache textbook acknowledges that injuries to upper cervical joints can cause headache after whiplash,[2] although another chapter indicates that this concept is

The mechanism underlying the pain involves convergence between cervical and trigeminal afferents in the trigeminocervical nucleus (figure 1).[4,5] In this nucleus, nociceptive afferents from the C1, C2, and C3 spinal nerves converge onto second-order neurons that also receive afferents from adjacent cervical nerves and from the first division of the trigeminal nerve (V), via the trigeminal nerve spinal tract. This convergence has been shown anatomically and physiologically in laboratory animals.[5–9] Convergence between cervical afferents allows for upper cervical pain to be referred to regions of the head innervated by cervical nerves (occipital and auricular regions). Convergence with trigeminal afferents allows for referral into the parietal,

Best Practice & Research Clinical Anaesthesiology
Vol. 16, No. 4, pp. 565–578, 2002
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BEST PRACTICE & RESEARCH

7

Diagnostic nerve blocks in chronic pain

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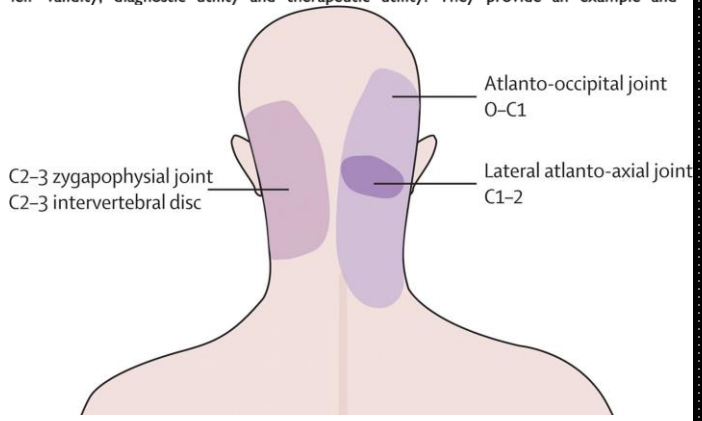
Diagnostic blocks are used to obtain information about the source of a patient's pain. As such they differ in principle and in practice from regional anaesthetic blocks. In order to be valid, diagnostic blocks must be precise and target-specific. They must be controlled in order to exclude false-positive responses. Sympathetic blocks have traditionally been performed without pharmacological controls, but studies have shown that the features of complex regional pain syndromes can be relieved equally well when normal saline is administered as when local anaesthetic is used. This warns that sympathetic blocks must be controlled in each and every case lest false conclusions be drawn about the response. Medial branch blocks of the lumbar and of the cervical dorsal rami have been extensively investigated in order to establish their validity, diagnostic utility and therapeutic utility. They provide an example and



Headache
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Review Article

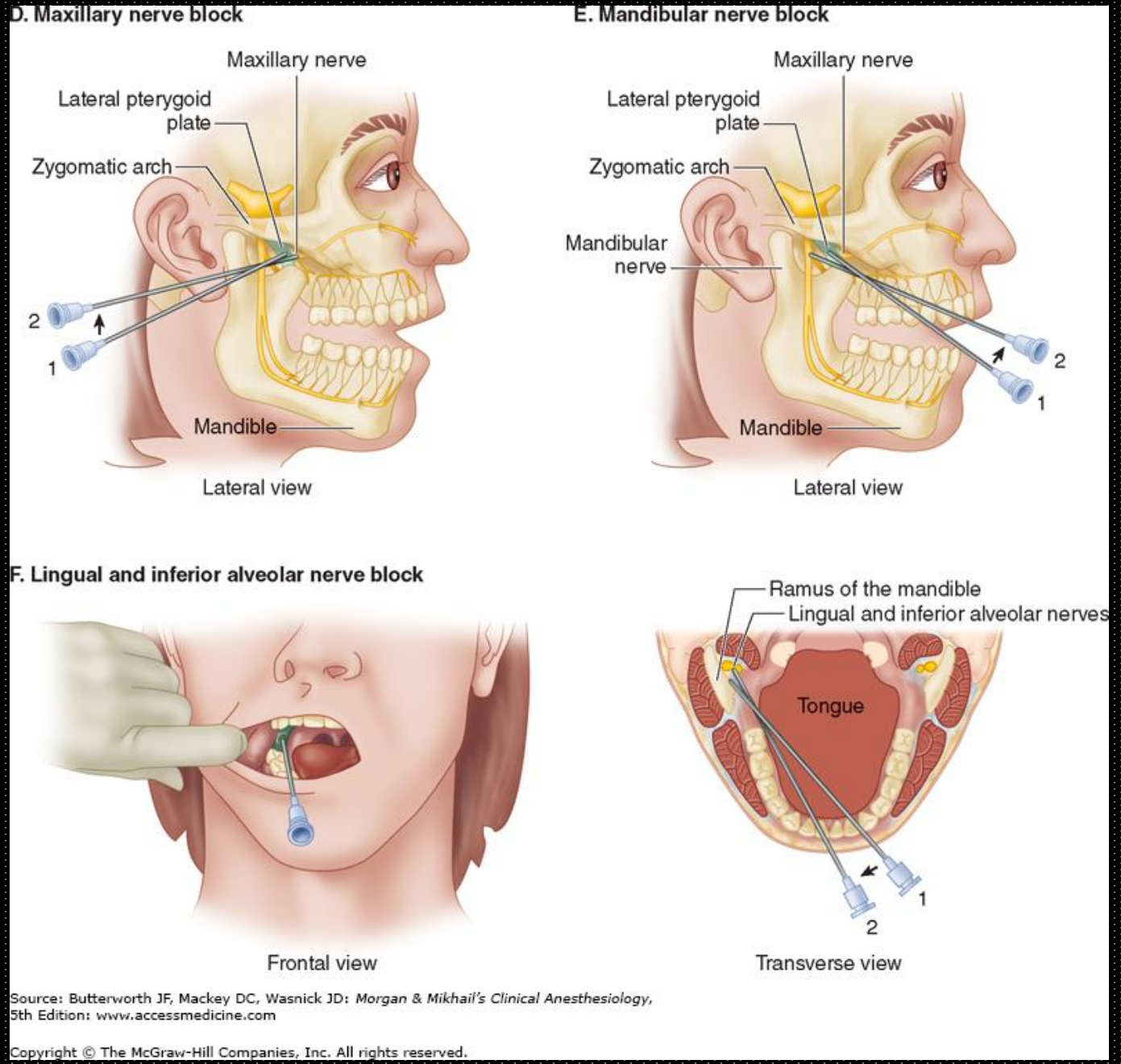
Expert Consensus Recommendations for the Performance of Peripheral Nerve Blocks for Headaches – A Narrative Review

Andrew Blumenfeld, MD; Avi Ashkenazi, MD; Uri Napchan, MD; Steven D. Bender, DDS; Brad C. Klein, MD; Randall Berliner, MD; Jessica Ailani, MD; Jack Schim, MD; Deborah I. Friedman, MD, MPH; Larry Charleston IV, MD; William B. Young, MD; Carrie E. Robertson, MD; David W. Dodick, MD; Stephen D. Silberstein, MD; Matthew S. Robbins, MD

From The Headache Center of Southern California – Neurology, Encinitas, CA, USA (A. Blumenfeld and J. Schim); Doylestown Hospital, Doylestown, PA, USA (A. Ashkenazi); Headache Clinic at Middletown Medical, Middletown, NY, USA (U. Napchan); North Texas Center for Head, Face & TMJ Pain, Texas A&M University, Baylor College of Dentistry, Plano, TX, USA (S.D. Bender); Abington Headache Center – Neurology, Warminster, PA, USA (B.C. Klein); Albert Einstein College of Medicine/Montefiore Medical Center, Bronx, NY, USA (R. Berliner and M.S. Robbins); Department of Neurology, Georgetown University Hospital, Washington, DC, USA (J. Ailani); Departments of Neurology & Neurotherapeutics and Ophthalmology, University of Texas

Somatic Nerve Block OFP Studies

- No evidence



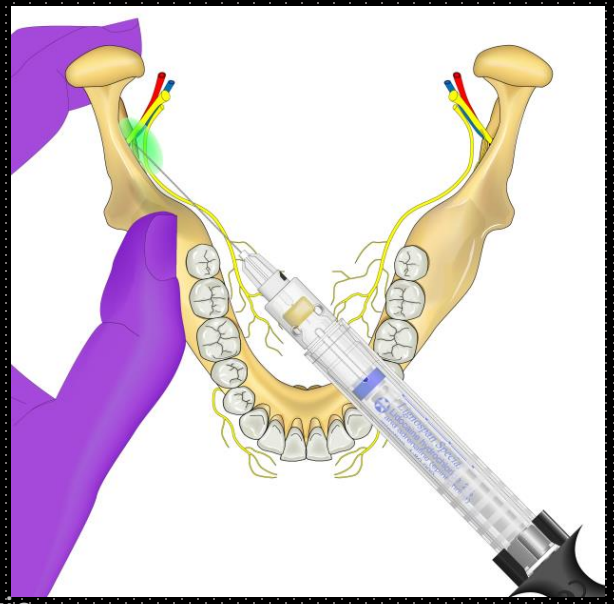
Diagnostic Nerve bocks for OFP
Type of block

- Region
  - **Trigeminal**
    - **Inferior alveolar**
    - **Lingual**
    - **Intra oral infiltration**
    - **Auriculotemporal**
    - **Infraorbital**
  - Temporomandibular
    - Muscular
    - Intracapsular
    - Extracapsular
  - Trigger point
    - Dry needling
    - Injection
    - Acupuncture
  - Cervical nerves
    - Occipital nerve block
  - Sympathetic Stellate

Variables

- Type of injection
  - Infiltration
    - Epithelial
    - Subepithelial
  - Nerve Block
    - Peripheral
    - Central
- Number of injections
- Agent
  - LA
  - Steroid
  - Botox
  - Anti inflammatory
  - Dry needling
  - NGF,
  - Combined

Inferior alveolar nerve block



Only one study assessed efficacy of IANB for diagnosis of Osteo myelitis M related pain

The aims of this study were:

- (1) to demonstrate how reproducible variations in incomplete anaesthesia of the inferior alveolar nerve can be used as a guide to locate the etiologic sites of referred trigeminal pain emanating from the mandible;
- (2) to describe the salient histopathologic features of a low grade, non-suppurative osteomyelitis seen in this patient population.
- **Forty-six patients** with idiopathic facial pain were subjected to a specific protocol of local anesthetic injections to sequentially block branches of the mandibular nerve to determine the effects on his/her pain.
- If this significantly reduced or altered the pain on three separate appointments, then exploratory surgery was conducted near identified zones of unanesthetized gingiva.
- Blocking (92%), bridging (4%), and divergence (4%) were observed patterns of anaesthetic resistance of the mucogingival tissues used to categorize the incomplete anaesthesia.
- A 100% correlation was found between the identified zones of unanesthetized gingiva and the discovery of intramedullary pathology.
- Medullary fibrosis with ischemic and degenerative changes in the cancellous bone were common findings, along with chronic inflammatory cell infiltrates and clusters of lymphocytes. It is concluded that Ratner's method of diagnostic anaesthesia be implemented when searching for occult painproducing pathology of the jaws.

McMahon RE, Griep J, Marfurt C Saxen MA Local anesthetic effects in the presence of chronic osteomyelitis (necrosis) of the mandible: implications for localizing the etiologic sites of referred trigeminal pain. Cranio. 1995 Oct;13(4):212-26.

Lingual nerve block
Experimental LA in Burning mouth syndrome

Tested the effect of a topical anaesthetic (dyclonine HCl) on patients' intensity ratings for oral burning, taste dysgeusia and the taste of two chemical stimuli (1.0 M NaCl and 1.0 M sucrose).

A total of 33 patients (9 male and 24 female, average age: 60 yr) are included in this analysis. The anaesthetic reduced the perceptual intensity of both chemicals in these patients on four out of five post anaesthesia trials ($p < 0.01$).

The BMS cohort included **12 patients whose burning increased ($p < 0.001$),** 14 patients whose burning did not change, and **7 patients whose burning decreased ($p < 0.001$)**

Baseline dysgeusias (n = 13) decreased in intensity ($p < 0.001$) after anaesthesia, suggesting BMS dysgeusia is related to the activation of peripheral taste mechanisms. The results also suggest that BMS oral burning may be a disorder of peripheral pain pathways in some patients

ORIGINAL ARTICLE

Effect of a local anesthetic lozenge in relief of symptoms in burning mouth syndrome

C Treldal[1], CB Jacobsen[1], S Mogensen[1], M Rasmussen[1], J Jacobsen[2], J Petersen[1], AM Lynge Pedersen[3], O Andersen[1]

[1]*Clinical Research Centre, Copenhagen University Hospital, Hvidovre;* [2]*Section for Pharmaceutical Design and Drug Delivery, Department of Pharmacy, Faculty of Health and Medical Sciences, University of Copenhagen, Copenhagen;* [3]*Section for Oral Medicine, Clinical Oral Physiology, Oral Pathology and Anatomy, Department of Odontology, Faculty of Health and Medical Sciences, University of Copenhagen, Copenhagen, Denmark*

OBJECTIVE: Patients with burning mouth syndrome (BMS) often represent a clinical challenge as available agents for symptomatic treatment are few and often ineffective. The aim was to evaluate the effect of a bupivacaine lozenge on oral mucosal pain, xerostomia, and taste alterations in patients with BMS.

METHODS: Eighteen patients (4 men and 14 women) aged 39–71 years with BMS were included in this randomized, double-blinded, placebo-controlled, crossover trial. Lozenges (containing bupivacaine or placebo) were administrated three times a day for 2 weeks for two separate treatment periods. Assessment of oral mucosal pain, xerostomia, and taste alterations was performed in a patient diary on a visual analog scale (ranging from 0 to 100 mm) before and after the lozenge was dissolved.

RESULTS: The bupivacaine lozenge significantly reduced the burning oral pain ($P < 0.001$), increased the sense of taste disturbances ($P < 0.001$), and had no impact on xerostomia, when adjusted for the treatment period.

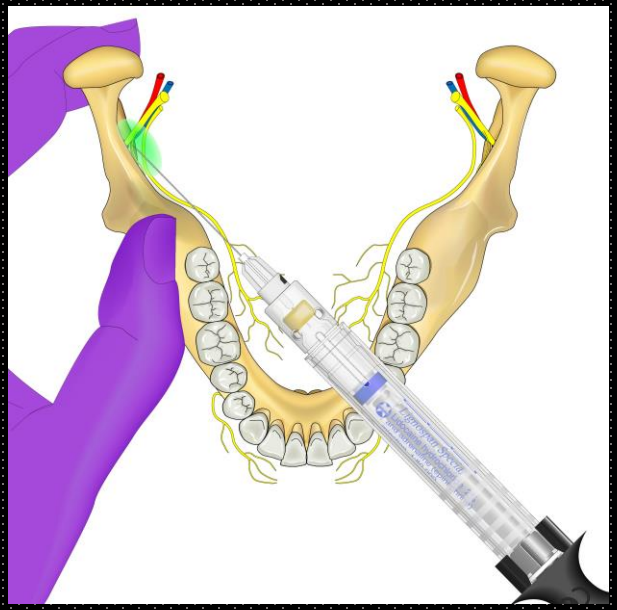
CONCLUSIONS: Our results indicate that the bupivacaine lozenge offers a novel therapeutic modality to patients with BMS, although without alleviating effect on the associated symptoms, taste alterations, and xerostomia.

Oral Diseases (2016) **22**, 123–131

Introduction

Burning mouth syndrome (BMS) is an oral pain condition characterized by a persistent painful burning, itching, or stinging sensation in a clinically normal-appearing oral mucosa, where no dental or systemic causes have been identified (Zakrzewska *et al*, 2005; Jaaskelainen, 2012). Psychological factors may impact the pathogenesis but it is still unsettled whether BMS is caused by a psychosomatic or psychogenic disorder or whether the persistent oral pain influences the psychosocial behavior (Pedersen *et al*, 2004). It is a severe, disabling condition that often impairs the patient's quality of life, as no optimal treatment exists (Lopez-Jornet *et al*, 2008; Ni Riordain *et al*, 2010). The burning sensation is mainly located on the tongue, but frequently affects other areas of the oral mucosa such as the anterior part of the hard palate and the lips (Grushka, 1987; Bergdahl and Bergdahl, 1999; Pedersen *et al*, 2004). In addition to the burning sensation patients often present with altered perception of taste (dysgeusia) and sensations of dry mouth (xerostomia) despite an often normal salivary secretion (Bergdahl and Bergdahl, 1999; Pedersen *et al*, 2004; Zakrzewska *et al*, 2005). The diagnosis of BMS is based on a clinical examination and ruling out any systemic or local explanations for the symptoms (Jaaskelainen, 2012; Sun *et al*, 2013). The prevalence in the adult population is not well estimated and ranges from 0.7% to 15% due to lack of adherence to

Diagnostic lingual nerve block for BMS



- A randomised, double-blind crossover design was used to investigate the effects of lingual nerve block on spontaneous burning pain and a possible correlation with the effects of topical clonazepam, the patient's response to a psychological questionnaire, and the taste and heat thresholds. **17 patients**
- The spontaneous burning was measured with a visual analogue scale (VAS) just before and 15 min after injection.
- The decreases in VAS score after lidocaine or saline injection were not significantly different (2.7+/-3.9 and 2.0+/-2.6, respectively; n=20).
- Two groups of patients could be identified: in a **"peripheral group"** (n=10) the VAS decrease due to lingual nerve injection was 4.3+/-3.1cm after lidocaine and 0.9+/-0.3 cm after saline (p=0.02).
- In a **"central group"** (n=7), there were an increase in pain intensity score (-0.8+/-2.6 cm) after lidocaine and a decrease (1.5+/-3.0 cm) after saline (p=0.15).
- An **increase** in the hospital anxiety and depression **(HAD) score** and a **decreased taste sensitivity and heat pain threshold** of painful oral area were seen in patients compared with age-and-sex-matched controls ($p<0.05$).
- Topical clonazepam treatment tended to be more effective ($p=0.07$) and **HAD score lower ($p<0.03$) in the peripheral than in the central group**. These results suggest that the neuropathic disorder associated with stomatodynia may be predominantly peripheral, central or mixed depending on the individual.
- Topical application of clonazepam and HAD may serve as indicators of which mechanism is dominating.

Grémeau-Richard C, Dubray C, Aublet-Cuvelier B, Ughetto S, Woda A. Effect of lingual nerve block on burning mouth syndrome (stomatodynia): a randomized crossover trial. Pain. 2010 Apr;149(1):27-32. doi: 10.1016/j.pain.2009.11.016. Epub 2010 Jan 18.

Our research on BMS effect LA infiltration

Dr Kiran Beneng Dr Matthew Howard and Tara Renton

Methods

- Patients diagnosed with BMS in accordance with the International Association for the Study of Pain (IASP) criteria (Zakrewska et al., 2001) were invited to join the study (n=9) along with age and gender matched controls (n=9). All BMS patients had symptoms affecting the anterior tip of tongue, with a daily visual analogue pain intensity of at least 3/10.
- All patients were placed in the MRI scanner and a total of 6 cASL scans, each of six minutes in duration were carried out; three prior to, and three following administration of local anaesthestic (LA) to the left tip of tongue, as means of blocking peripheral sensory and nociceptive input.

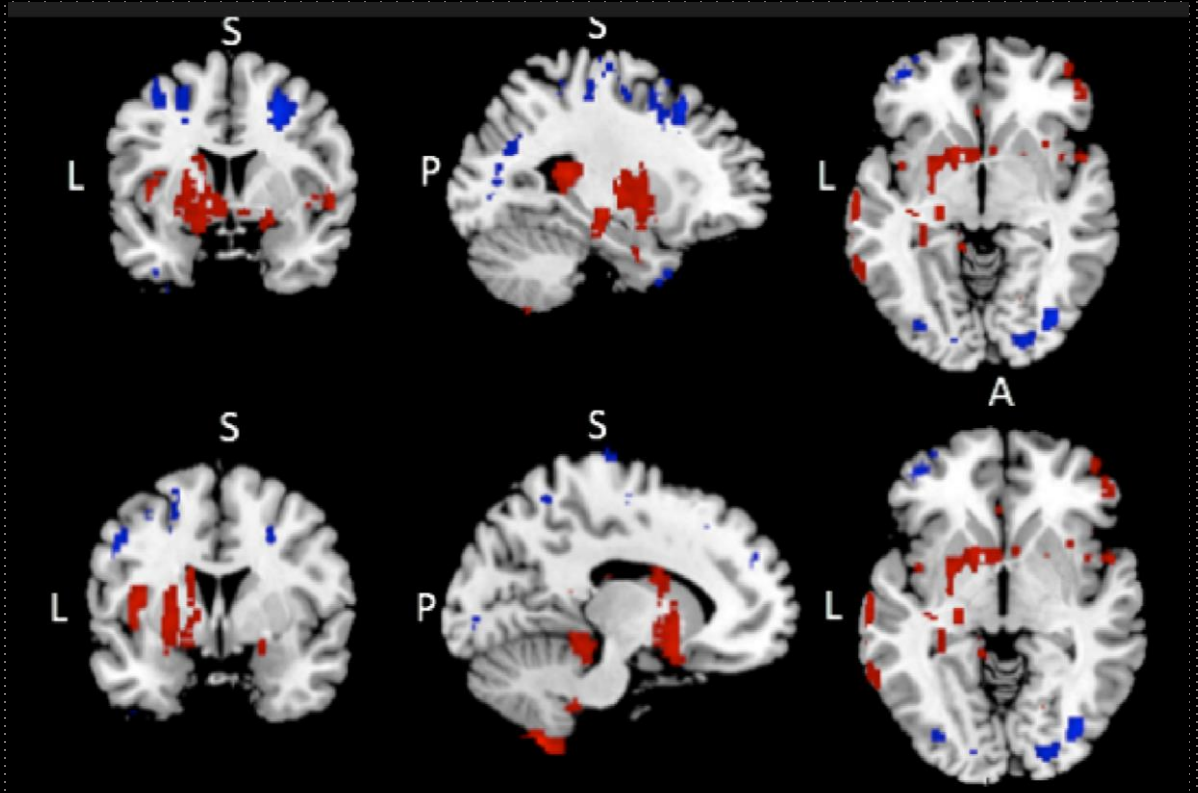
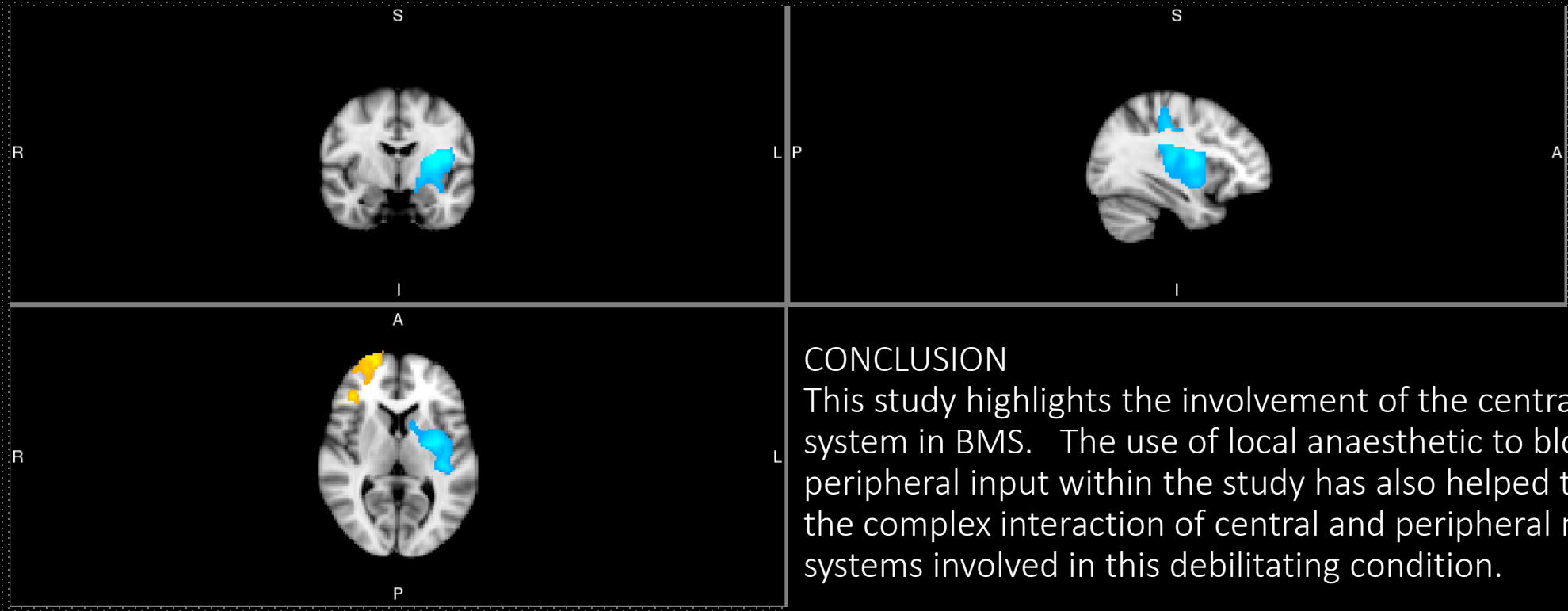


Figure 1. Pre-treatment cortical and subcortical increases in rCBF across patients (red). Post-treatment cortical increases across patients(blue).

Results

- Three groups were identified;
  - Peripheral group with decrease in pain after LA (n=5)
  - Central group with increase in pain after LA (n=2)
  - Mixed group no change in pain after LA (n=2)
- A significant increase in CBF was noted in BMS patients compared to controls with a cluster seen unilaterally within the insula extending anteriorly and inferiorly to the fronto-orbital complex.
- Bilaterally, CBF increases were identified in the most anterior aspect of the frontal lobe, including the frontal pole, extending posteriorly to include the paracingulate and cingulate gyrus.
- Following treatment with LA, rCBF decreases were seen in the BMS group on the ipsilateral side to the LA. CBF changes were noted within S2, insula cortex, fronto-orbital cortex, the primary auditory complex and the putamen.
- Conversely, there was a significant increase in CBF in controls, compared to BMS patients following LA administration with changes predominantly noted around the midline in the cingulate gyrus, hippocampus, parahippocampal gyrus, cerebellum and brainstem.

Analysis of the Group BMS Treatment interaction , indicated significant rCBF differences on the side ipsilateral to LA affecting S1, S2, insula cortex and the putamen. (Figure 1)



CONCLUSION

This study highlights the involvement of the central nervous system in BMS. The use of local anaesthetic to block peripheral input within the study has also helped to illustrate the complex interaction of central and peripheral nervous systems involved in this debilitating condition.

The effect of using topical or infiltration LA for BMS concur with pathophysiology of BMS but to date there is no relation to preferred management for each group

ORAL DISEASES

Oral Diseases (2015) 21, 937–948 doi:10.1111/odi.12345

www.wiley.com

REVIEW ARTICLE

Pathophysiology of primary burning mouth syndrome with special focus on taste dysfunction: a review

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Primary burning mouth syndrome (BMS) is a chronic oral condition characterized by burning pain often accompanied with taste dysfunction and xerostomia. The most compelling evidence concerning BMS pathophysiology comes from studies on the somatosensory system using neurophysiologic or psychophysical methods such as blink reflex, thermal quantitative sensory testing, as well as functional brain imaging. They have provided convincing evidence for neuropathic involvement at several levels of the somatosensory system in BMS pain pathophysiology. The number of taste function studies trying to substantiate the subjective taste disturbances or studies on salivary factors in BMS is much more limited, and most of them suffer from definitional and methodological problems. This review aims to critically evaluate the existing literature on the pathophysiology of BMS, paying special attention to the correctness of case selection and the methodology used in published studies, and to summarize the current state of knowledge. Based on the recognition of several gaps in the current understanding of the pathophysiology of BMS especially as regards taste and pain system interactions, the review ends with future scenarios for research in this area.

Oral Diseases (2015) **21**, 937–948

Keywords: primary burning mouth syndrome; pathophysiology; taste dysfunction; saliva; taste

Introduction

Burning mouth syndrome (BMS) is a chronic oral condition characterized by burning pain, often accompanied with taste dysfunction (dysgeusia, taste phantoms) or dry mouth sensation (xerostomia). Noticeable is that hyposalivation by itself can also induce burning sensation in the mouth without being true BMS.

Burning mouth syndrome remained an enigma for a long time, but during the last years, knowledge of the pathophysiology of BMS has considerably increased. Research has particularly focused on the trigeminal somatosensory system to unravel the background of BMS pain, and various types of abnormalities have been found at several levels of the somatosensory system.

Much less attention has been paid to the other aspects of the syndrome, taste disorders and xerostomia. The numbers of taste function studies trying to substantiate the subjective taste disturbances or studies on salivary factors are much more limited. The aim of this article was to review the existing literature on the pathophysiology of BMS, with special focus on studies on taste dysfunction and xerostomia in BMS.

BMS – clinical features

Burning mouth syndrome is characterized by burning mucosal pain that is not due to any other local or systemic causes, and arises from a clinically normal, healthy mucosa (Bergdahl and Bergdahl, 1999; Woda and Pionchon, 1999; Zakrzewska and Hamlyn, 1999; Zakrzewska *et al*, 2005; Markman and Eliav, 2013). The International Classification of Headache Disorders defines BMS accordingly as 'an intraoral burning or dysesthetic sensation, recurring daily for more than 2 h per day over more than 3 months, without clinically evident causative lesions' (The International Classification of Headache Disorders, 2013). In addition to pain, BMS patients often complain of a feeling of oral dryness or taste disturbances justifying the use of the term 'syndrome' (Scala *et al*, 2003; Granot and Nagler, 2005; Zakrzewska *et al*, 2005).

Burning mouth syndrome diagnosis is in practice based on the exclusion of local and/or systemic factors that could cause the oral burning or other sensory symptoms. Many studies, especially earlier ones, have not distinguished between BMS and oral burning symptoms, that is

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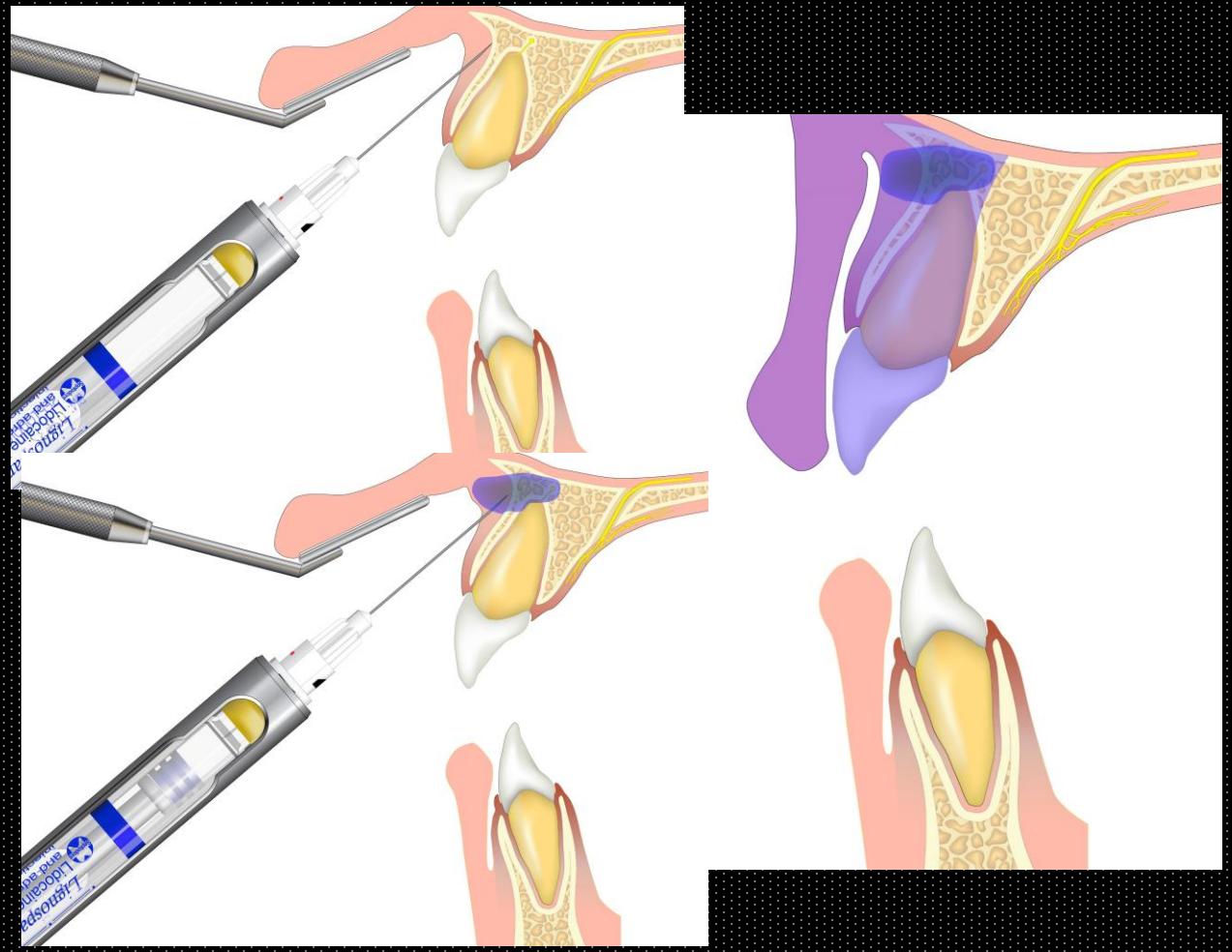
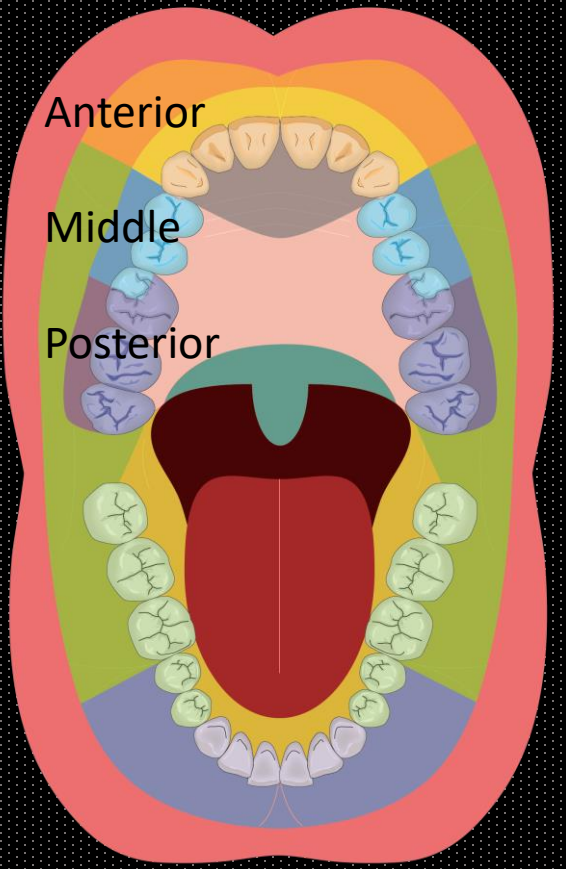
Received 4 March 2015; revised 13 April 2015; accepted 19 April 2015

Trigeminal nerve infiltrations

Anterior Middle Posterior Superior Blocks

Mandibular ridge infiltrations

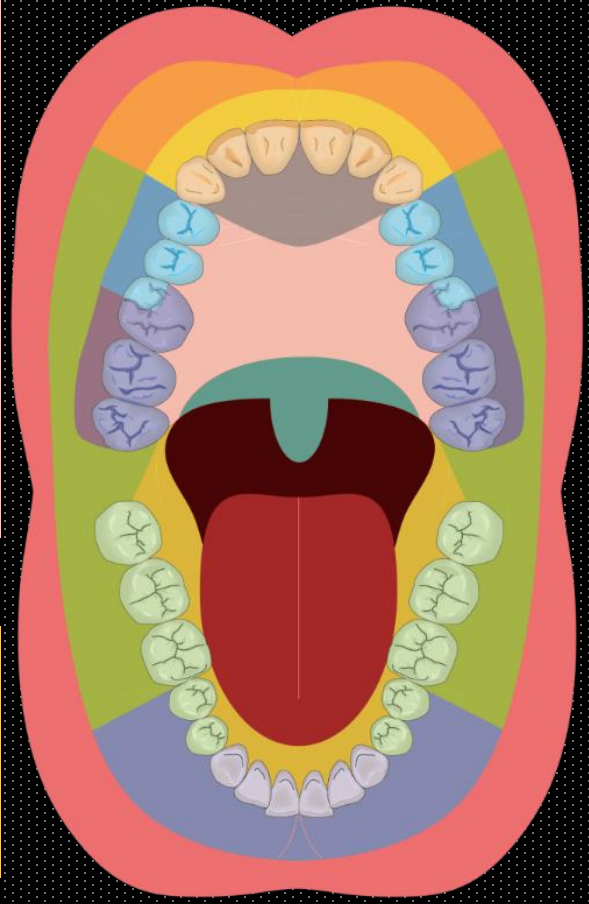




Our experience in therapeutic PNBs

Maxillary dentistry can be performed entirely using Lidocaine 2% with adrenaline for all procedures
Buccal infiltration with intra-septal injections
No additional benefit using 4% Articaine
No palatal or incisal blocks are indicated

Posterior mandibular molar Endodontic procedures may require IDBs or higher techniques (Gow Gates or Akinosi)



Mandibular 7s and 8s for **perio, restorations or implants**
Articaine 4% buccal infiltration and Lidocaine 2% lingual infiltrations OR for **extractions** intraligamental
If fails may need lidocaine IDB

Mandibular 1st molars for **perio, restorations or implants**
Articaine 4% buccal +/- Lidocaine 2% crestal or lingual infiltration s OR for **extractions** add lidocaine lingual **of** intra-ligamental

Mandibular premolars, canines incisors for **perio, restorations or implants**
Articaine buccal infiltration (incisal nerve block using 30% cartridge) adjacent not in the mental foramen and massage over region. If fails repeat or add crestal or lingual infiltration OR for **extractions,** intra-ligamental

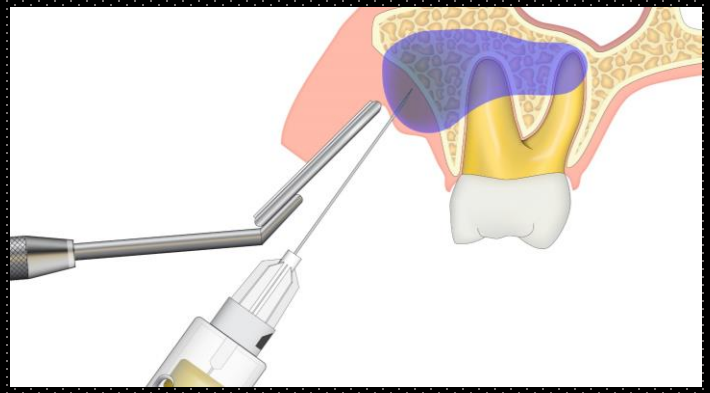
Illustration modified from figure courtesy of Andrew Mason University Dundee

Pre Botox LA injections for focal neuropathic pain

Lidocaine 2% (1:80K epinephrine) 1-2mls infiltrations
positive response prerequisite for BTX treatment

PDAP 1 or primary localised intra oral Ne Pain

- 7 patients
- Mean age 55yrs
- 60% Female
- Site
  - 40% mandibular posterior molar region
  - 40% posterior maxillary molar region
  - 20% anterior maxilla
- Response rate
  - Complete 3
  - Partial 2
  - None 2



PTPN localised intra oral Ne Pain

- 18 patients
- Mean age 42 yrs
- 75% female
- Site
  - 15% mandibular posterior molar region
  - 5% posterior maxillary molar region
  - 80% anterior maxilla
- Response rate
  - Complete 14
  - Partial 2
  - None 2

No predictive effect on outcome of Botoxin injections for PTPN!

TN single diagnostic = ‘therapeutic’ block

The study evaluated the therapeutic effect of combination of pharmacotherapy and lidocaine block.

Thirteen patients with CTN managed with pharmacotherapy were recruited and assigned either to no additional treatment (Group I) or to additional analgesic block (Group II).

The primary endpoint was the reduction in the frequency of pain episodes in a month assessed at 30 and 90 days.

- Comparisons of measurements of pain, general health and depression scales were secondary endpoints.
- The results from the follow-up visits at 30 and 90 days showed the Group II to have larger reduction in the frequency of pain and exhibited a bigger improvement in the scores of the pain, general health and depression scales.
- The results from this preliminary study suggest a clinical benefit of the combination of pharmacotherapy and lidocaine block.

Di Stani F Ojango C, Dugoni D, Di Lorenzo L, Masala S, Delfini R, Bruti G, Simonetti G, Piovesan EJ, Ruggeri AG. Combination of pharmacotherapy and lidocaine analgesic block of the peripheral trigeminal branches for trigeminal neuralgia: a pilot study. Arq Neuropsiquiatr. 2015 Aug;73(8):660-4. doi: 10.1590/0004-282X20150077.

LA infiltrations or NB for TN

DOI: 10.1590/0004-282X20150077

ARTICLE

Combination of pharmacotherapy and lidocaine analgesic block of the peripheral trigeminal branches for trigeminal neuralgia: a pilot study

Combinação de farmacoterapia e bloqueio analgésico com lidocaína sobre os ramos periféricos trigeminais no tratamento da neuralgia do trigêmeo: um estudo piloto

Fabrizio Di Stani[1], Christine Ojango[2], Demo Dugoni[1], Luigi Di Lorenzo[3], Salvatore Masala[2], Roberto Delfini[1], Gianluca Bruti[1], Giovanni Simonetti[2], Elcio Juliato Piovesan[4], Andrea Gennaro Ruggeri[1]

ABSTRACT

Classical trigeminal neuralgia (CT
study was carried out to evaluate t
managed with pharmacotherapy
(Group II). The primary endpoint w
of measurements of pain, gener
30 and 90 days showed the Grou
of the pain, general health and de
pharmacotherapy and lidocaine b

Keywords: analgesic block, class

RESUMO

A neuralgia clássica do trigêmio (
Avaliamos o efeito terapêutico
portadores de NTC tratados com
medicamentos e Grupo II paciente
30 e 90 dias após o bloqueio. Secu
uma redução significativa na freq

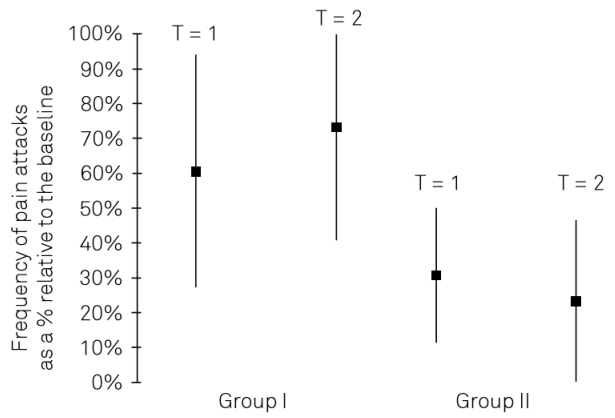


Figure 2. The average frequency of pain attacks in a month measured at T = 1 and T = 2 and expressed as a percentage relative to the baseline (0%- free from attacks; 100%- same frequency of attacks as at baseline) in both groups.

Demographic characteristics of study patients.					
		Group I		Group II	
Age (years)	Mean ± SD	63.0	11.8	68.2	10.8
Gender	Women n %	4	57.1	4	66.7
Duration of symtoms (years)	Mean ± SD	5.0	2.90	16.8	9.20
Symtomatic facial side	Right n %	2	28.6	3	50
	Left n %	5	71.4	3	50
Pain location (trigeminal branches)	V2 or V3 n %	3	42.9	3	50
	V1 + V2 or V2 + V3 n %	4	57.1	2	33.3
	V1 + V2 + V3 n %	0		1	16.7
Frequency of pain attacks	dd/month	30		28.3	

SD: standard deviation; n: number of patients; dd/month: days per month.

	Group I		Group II	
	Mean ± SD		Mean ± SD	
SF-36 physical functioning	50.7	35.76	66.7	26.3
SF-36 physical role functioning	25.7	36.56	25	38.7
SF-36 bodily pain	25.9	14.38	39.8	21.1
SF-36 general health perceptions	38.6	19.32	46	11.8
SF-36 vitality	43.1	18.37	50	14.8
SF-36 social role functioning	39.2	25.45	52	14.7
SF-36 mental health	34.3	25.81	60.7	19.6
SF-36 emotional role functioning	19.6	26.59	38.9	49.0
BDI	26.7	16.18	14	11.2
BPI severity index	5.1	1.999	5.3	2.55
BPI interferference index	4.3	1.599	3.4	3.35

Baseline assessment of The Medical Outcomes Trust 36-Item; SF-36®: Short Form, Health Survey; BDI: Beck Depression Inventory; BPI: Brief Pain Inventory scales.

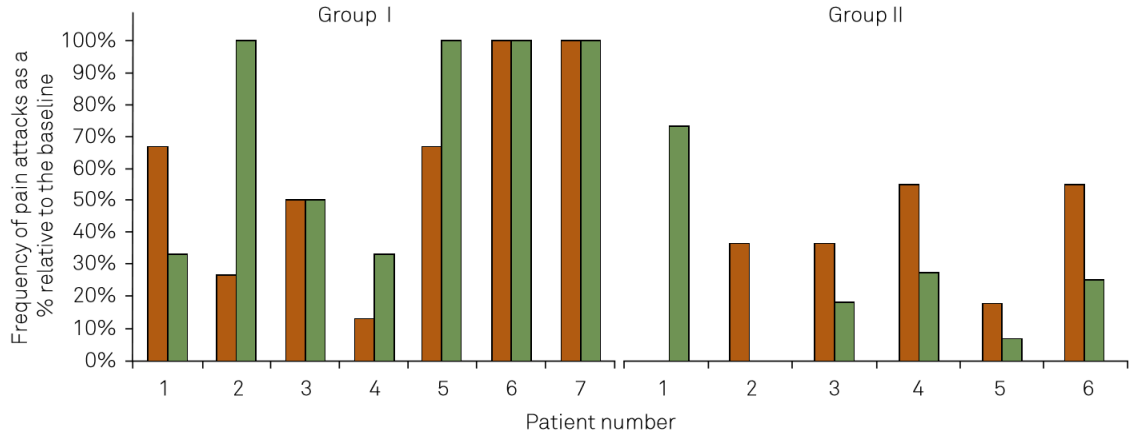


Figure 1. Frequency of pain attacks in a month measured at T = 1 and T = 2 expressed as a percentage relative to the baseline (0%- free from attacks; 100% same frequency of attacks as at baseline) in all patients. Brown: follow-up visit T = 1; Green: follow-up visit T = 2.

TN preoperative nerve block

- Prospective study to establish the preoperative and perioperative analgesic effects of preoperative single peripheral nerve block.
- Sixty patients with classic trigeminal neuralgia who were scheduled to undergo radiofrequency thermocoagulation of the gasserian ganglion were randomly divided into a control group (n = 30) and a nerve block group (n = 30).
- Patients in the nerve block group were treated with single peripheral nerve block using **1% lidocaine and betamethasone** on the day of admission.
- Average pain, worst pain, quality of sleep, and analgesia satisfaction were evaluated before surgery. The incidence and intensity of perioperative episodic pain were determined before the needle reached the gasserian ganglion.

- Compared with the control group, a single peripheral nerve block significantly attenuated
  - average pain ($P < 0.01$)
  - worst pain ($P < 0.01$),
  - ameliorated the quality of sleep ($P < 0.01$),
  - increased analgesia satisfaction ($P < 0.01$).
- Moreover, patients in the nerve block group experienced a decrease in incidence ($P < 0.01$) and intensity ($P < 0.01$) of episodic pain during surgery as compared with the participants in the control group.
- These results demonstrate that a single peripheral nerve block may be an effective way to relieve preoperative and perioperative intolerable pain of trigeminal neuralgia.

Weng Z, Halawa MA, Liu X, Zhou X, Yao S. Analgesic effects of preoperative peripheral nerve block in patients with trigeminal neuralgiaundergoing radiofrequency thermocoagulation of gasserian ganglion. J Craniofac Surg 2013 Mar;24(2):479-82. doi: 10.1097/SCS.0b013e31827c7d6f

Diagnostic Nerve bocks for OFP
Type of block

- Region
  - Trigeminal
    - Inferior alveolar
    - Lingual
    - Intra oral infiltration
    - Auriculotemporal
    - Infraorbital
  - **Temporomandibular**
    - **Muscular**
    - **Intracapsular**
    - **Extracapsular**
  - Trigger point
    - Dry needling
    - Injection
    - Acupuncture
  - Cervical nerves
    - Occipital nerve block
  - Sympathetic Stellate

Variables

- Type of injection
  - Infiltration
    - Epithelial
    - Subepithelial
  - Nerve Block
    - Peripheral
    - Central
- Number of injections
- Agent
  - LA
  - Steroid
  - Botox
  - Anti inflammatory
  - Dry needling
  - NGF,
  - Combined

TMD

Types of blocks

Diagnostic

- Intracapsular?
- Intramuscular injections valuable in determining the source of pain and therapeutic value

Fine PG, Milano R, Hare BD (1988) The effects of myofascial trigger points injections are naloxone reversible. Pain 32: 15-20.

- Auriculotemporal block helps to identify whether the painful structure is a site or source of pain

Schmidt B, Pogrel MA, Necoechea M, Kearns G (1998) The distribution of the auriculotemporal nerve around the temporomandibular joint. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 86: 165-168.

Therapeutic

- Intra articular injections

Henny FA (1954) Intra-articular injection of hydrocortisone into the temporomandibular joint. J Oral Surg 12: 314-319.

- Dry needling
  - Gonzalez-Perez LM, Infante-Cossio P, Granados-Nuñez M, Urresti-Lopez FZ (2012) Treatment of temporomandibularmyofascial pain with deep dry needling. Med Oral Patol Oral Cir Bucal 17: e781–e785.
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- Trigger point injections
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  - Travell JG, Simons DG (1999)Travell and Simons' myofascial pain and dysfunction; the trigger point manual. (2ndedn),Lippincott Williams & Wilkins, Baltimore.
  - Fernandez-Carnero J, La Touche R, Ortega-Santiago R, Galan-del-Rio F, Pesquera J, et al. (2010) Short -term effects of dry needling of active myofascial trigger points in the masseter muscle in patients with temporomandibular disorders. J Orofac Pain 24: 106-112.

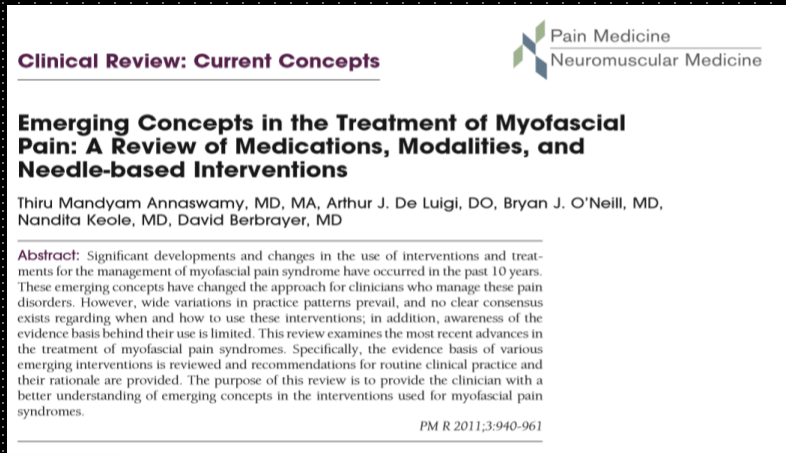
TMD Criteria for LA interventions

Please use updated classification of TMDs (INFORM)

- Degenerative (Arthritis)
- Myalgia
- Arthralgia
- Myofascial
- Disc displacement with or without reduction (ysfunction) +/- pain, D +/- locking
- Headache attributable to TMD

None reported or trialled

Re reported prognostic indicators for treatment outcome



Clinical Review: Current Concepts

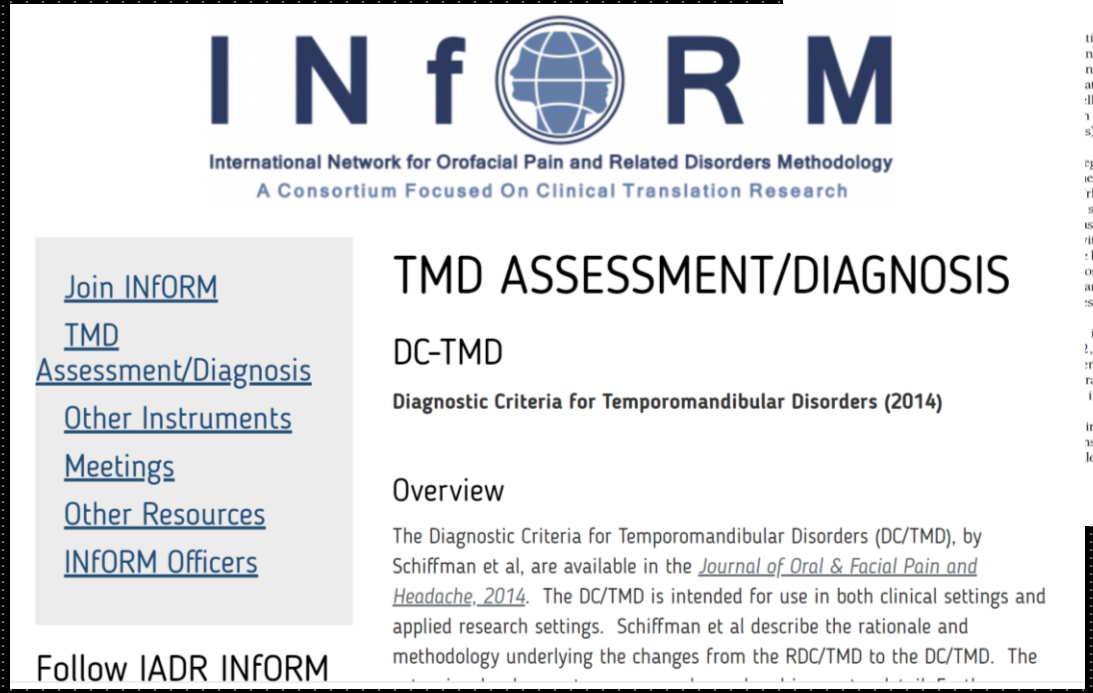
Pain Medicine
Neuromuscular Medicine

Emerging Concepts in the Treatment of Myofascial Pain: A Review of Medications, Modalities, and Needle-based Interventions

Thiru Mandyam Annaswamy, MD, MA, Arthur J. De Luigi, DO, Bryan J. O'Neill, MD, Nandita Keole, MD, David Berbrayer, MD

Abstract: Significant developments and changes in the use of interventions and treatments for the management of myofascial pain syndrome have occurred in the past 10 years. These emerging concepts have changed the approach for clinicians who manage these pain disorders. However, wide variations in practice patterns prevail, and no clear consensus exists regarding when and how to use these interventions; in addition, awareness of the evidence basis behind their use is limited. This review examines the most recent advances in the treatment of myofascial pain syndromes. Specifically, the evidence basis of various emerging interventions is reviewed and recommendations for routine clinical practice and their rationale are provided. The purpose of this review is to provide the clinician with a better understanding of emerging concepts in the interventions used for myofascial pain syndromes.

PM R 2011;3:940-961



INfORM

International Network for Orofacial Pain and Related Disorders Methodology

A Consortium Focused On Clinical Translation Research

Join INfORM
TMD Assessment/Diagnosis
Other Instruments
Meetings
Other Resources
INfORM Officers

Follow IADR INfORM

TMD ASSESSMENT/DIAGNOSIS

DC-TMD

Diagnostic Criteria for Temporomandibular Disorders (2014)

Overview

The Diagnostic Criteria for Temporomandibular Disorders (DC/TMD), by Schiffman et al, are available in the *Journal of Oral & Facial Pain and Headache, 2014*. The DC/TMD is intended for use in both clinical settings and applied research settings. Schiffman et al describe the rationale and methodology underlying the changes from the RDC/TMD to the DC/TMD. The

tient's visit to the outpatient clinic, and
n nearly a third of patients who have
niversally accepted biochemical, electro-
ation criteria exist for a diagnosis of MPS.
ll and Simon [2] is that MPS is a disorder
that affects a small number of muscles
s) that usually are located in tight bands

egrated hypothesis" theory that incorpo-
ervous system factors that could account
rPs. This integrated hypothesis includes
synaptic, and postsynaptic mechanisms
se of acetylcholine; acetylcholinesterase
ity; the energy crisis theory; abnormality
hypothesis. Postural stresses, inefficient
ost frequently described etiologies [1].
and a poor understanding of underlying
se reasons, various treatments have been

included medications such as muscle
2,4]. Several recent developments and
nts for the management of MPS have
ractice patterns continue, and no clear
in addition, awareness of their evidence

in the treatment of MPS. Specifically, the
ns are reviewed, recommendations for
les for the use of these interventions are

T.M.A. Electrodiagnostic and Spine Sections, PM&R Service, Dallas VA Medical Center, 4500 S Lancaster Rd, Dallas, TX 75216; Department of Physical Medicine and Rehabilitation, The University of Texas Southwestern Medical Center at Dallas, Dallas, TX. Address correspondence to T.M.A.; e-mail: thiru.annaswamy@va.gov
Disclosure: nothing to disclose

A.J.D.L. PM&R Residency, PM&R Service, Department of Orthopaedics & Rehabilitation, Walter Reed Army Medical Center, Washington, DC; Department of Neurology, Uniformed Services University of Health Sciences, Bethesda, MD.
Disclosure: nothing to disclose

B.J.O. The Neurologic Group of Bucks/Montgomery Counties, North Wales, PA; Department of Rehabilitation Medicine, Thomas Jefferson University, Philadelphia PA
Disclosure: nothing to disclose

N.K. Oncologic Rehabilitation and Pain Management, Integris Cancer Institute of Oklahoma, Oklahoma City, OK
Disclosure: nothing to disclose

D.B. Rehabilitation Medicine, Sunnybrook Health Sciences Centre, University of Toronto, Toronto, Ontario, Canada
Disclosure: nothing to disclose

Disclosure Key can be found on the Table of Contents and at www.pmrjournal.org

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Vol. 3, 940-961, October 2011
DOI: 10.1016/j.pmrj.2011.06.013

http://www.iadr.org/INfORM/DC-TMD

International TMD RDC recommended outcomes for TMD -efficacy of care?

- Numeric pain rating scale
- Maximal mouth opening
- TMS indices questionnaire

No mention of diagnostic or therapeutic blocks

- Axis II
  - Chronic Graded Pain scale (GPS)
  - Patient Health Questionnaire PHQ4
  - Characteristic pain intensity (CPI)
  - Oral Behaviours Checklist (OBC)
  - JFLS Jaw function limitation test

Fricton J (2007) Myogenoustemporomandibular disorders: diagnostic and management considerations. Dent Clin North Am 51:61-83.
Schiffman E, Ohrbach R, Truelove E, Look J, Anderson G et al. (2014) Diagnostic Criteria for Temporomandibular Disorders (DC/TMD) for Clinical and Research Applications: recommendations of the International RDC/TMD Consortium Network and Orofacial Pain Special Interest Group. J Oral Facial Pain Headache28:6-27.

TMD innervation

Only ligaments to meniscus are innervated
Not the capsule or meniscus

The general dentist's ability to perform an anaesthesia block of the temporomandibular joint (TMJ) can be very beneficial, especially when trying to diagnose or treat patients with temporomandibular dysfunction who have joint and/or muscle pain.

There are three common types of internal joint disorders--orthopedic, inflammatory, and degenerative--producing pain in the ligaments, TMJ capsule, or retrodiscal tissues. Secondary muscle splinting also may be involved.

Subjects with these disorders can have pain, limited opening, or difficulty with extended opening. Dentists providing care for these individuals may need to schedule longer appointments and deal with mid-treatment facial or TMJ pain as well as more postoperative discomfort.

An anaesthesia block for the TMJ can reduce pain and protective muscle splinting, increase the mandibular range of motion, and assist in providing a more manageable treatment.

DuPont JS Jr Simplified anaesthesia blocking of
the temporomandibular joint. Gen Dent 2004 Jul-Aug;52(4):318-20.

TMD intramuscular

- To compare the efficacy of a regional masseteric nerve block (MNB) in the management of myofascial pain of masseteric origin, relative to trigger point injection (TrP-Inj) and intra-oral stabilization appliance (IOA).
- A retrospective chart **review of 200 patients treated for myofascial pain of masseteric origin** was performed. **Sixty patients** met the eligibility criteria and were grouped based on their treatment regimen; IOA, TrP-Inj or MNB. Pain scores recorded at pre-treatment (baseline), 30 minutes post-treatment, and 2 weeks post-treatment were analyzed.
- Treatment with MNB resulted in significant reduction in pain at 30 minutes and two weeks post-treatment compared to TrP-Inj and IOA.
- MNB provided an immediate and sustained therapeutic effect for the management of myofascial pain for at least up to two weeks. MNB is a simple and valuable tool in the management of myogenous pain, especially for the non-orofacial pain practitioner.

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Efficacy of regional nerve block in management of myofascial pain of masseteric origin

Samuel Y. P. Quek, Gayathri Subramanian, Jasma Patel, Sowmya Ananthan, Julyana G. Zagury & Junad Khan

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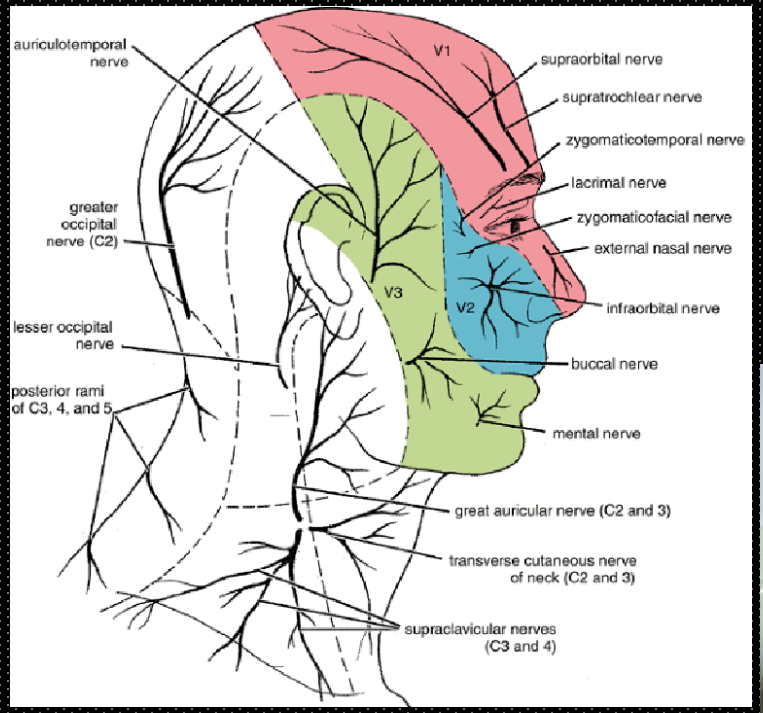
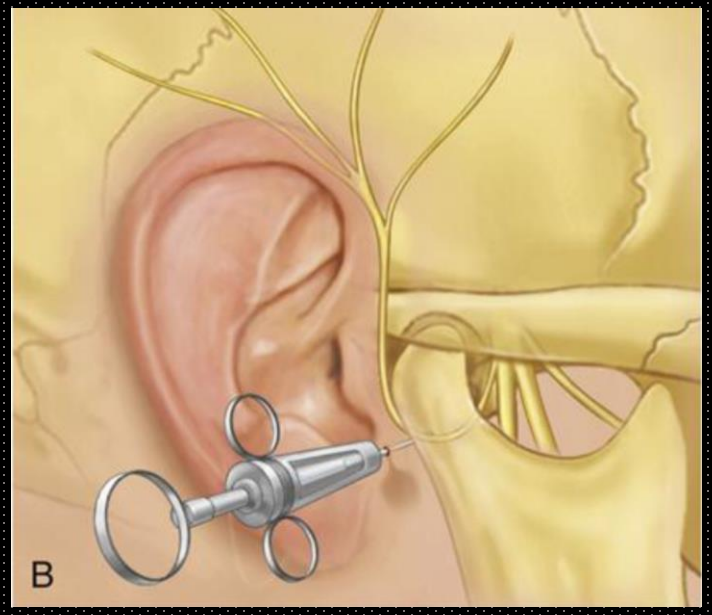
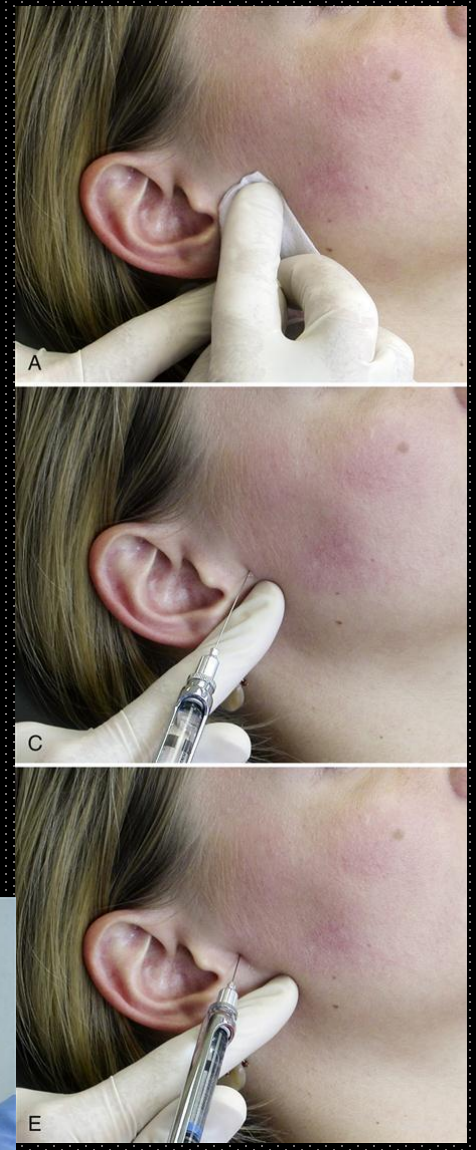
To link to this article: https://doi.org/10.1179/2151090314Y.0000000026

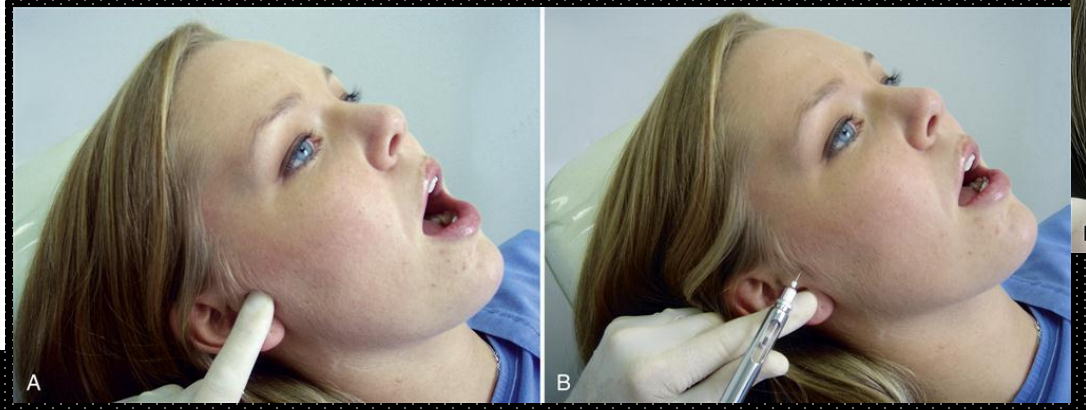
TMD Auriculotemporal NB

- Land marks and technique









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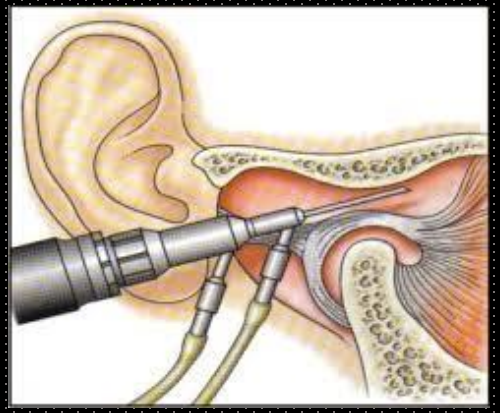
TMD -Auriculotemporal > intra articular NB in healthy subjects

- There is a need for systematic studies regarding the pathophysiology and pain mechanisms of somatosensory function in the temporomandibular joint (TMJ). So far, the effects on somatosensory functions of local anesthetics (LA) applied to the auriculotemporal (AT) nerve or intraarticularly (IA) into the TMJ have not been studied systemically.
- This study aimed to examine in a double-blinded, placebo-controlled manner the effects of LA on mechanical and thermal sensitivity in the TMJ area.
- **Twenty-eight healthy subjects** (27.4 +/- 6.2 years) without temporomandibular disorders (TMD) participated.
- The subjects received an **AT nerve block (n = 14)** or an **IA injection (n = 14)** with LA (**Bupivacaine, 2.5 mg/ml**) on one side, and **a placebo injection (isotonic saline) on the contralateral side**.
- Mechanical (tactile and pin-prick) and thermal sensitivity (40 and 5 degrees C) were assessed at 11 standardized points in the TMJ area before injections (baseline) as well as 30 min, 1 and 2 h after injections. All stimuli were rated by the subjects on a 0-100 numerical rating scale (NRS). TMJ pressure pain threshold (PPT) and pressure pain tolerance (PPTOL) were assessed laterally over both TMJs using an algometer.
- IA injections with LA were not associated with any changes in sensitivity of the TMJ or surrounding area.
- In contrast, AT nerve blocks with LA caused a decrease over time in the pin-prick sensitivity ($P = 0.016$), which however, did not differ significantly from saline, and an increase of the PPTs 30 min ($P = 0.010$) and PPTOLs 30 min, 1 h and 2 h ($P < 0.05$) after LA injections in comparison to saline.
- No other measures showed a significant change after the injections. **Our results showed that IA bupivacaine injection in healthy subjects has no effect on the sensitivity of the TMJ or surrounding area, while AT nerve block has a more pronounced effect on deep mechanical, but not on superficial mechanical or thermal sensitivity**

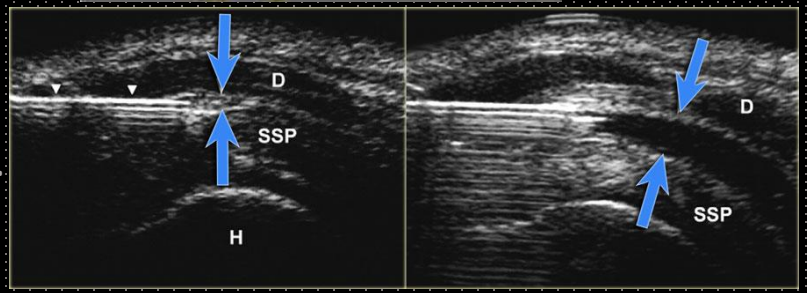
Ayesh EE, Ernberg M, Svensson P. Effects of local anesthetics on somatosensory function in the temporomandibular joint area. Exp Brain Res. 2007 Jul;180(4):715-25. Epub 2007 Feb 15.

TMJ
intra articular/ intra capsular NB

On occasion it is indicated to inject directly into the temporomandibular joint (TMJ). This type of injection would **be indicated for therapeutic and not diagnostic reasons**.



- Intracapsular injection of corticosteroids significantly reduces TMJ pain. It is indicated for acute and painful arthritic TMJ that has not responded to other modalities of treatment and when the joint is still acutely inflamed, such as in the case of polyarthritic disorders and in acute disc displacements without reduction.
- The use of triamcinolone or dexamethasone, in addition to 2% lidocaine without epinephrine, is generally used for TMJ injections
- Ultrasound may be required prior to injecting into the joint space. It has been suggested in animal studies that steroid injections may increase osteoclastic activity.
- There is no evidence that a single steroid injection causes damage; however, multiple injections may do, therefore the quantity of steroid injections should be carefully considered due to the possibility of bone resorption in the site of injection.
- Temporomandibular joint injection. Injections of sodium hyaluronate in osteoarthritis of the knee has shown improvement of symptoms; however, results for the management of TMD have been inconclusive and more studies are warranted.



Wenneberg B, Kopp S, Gröndahl HG. Long-term effect of intra-articular injections of a glucocorticosteroid into the TMJ: a clinical and radiographic 8-year follow-up. J Craniomandib Disord. 1991;5(1):11–18. [PubMed] Kopp S, Akerman S, Nilner M. Short-term effects of intra-articular sodium hyaluronate, glucocorticoid, and saline injections on rheumatoid arthritis of the temporomandibular joint. J Craniomandib Disord. 1991;5(4):231–238. [PubMed] Samiee A, Sabzerou D, Edalatpajouh F, Clark GT, Ram S. Temporomandibular joint injection with corticosteroid and local anesthetic for limited mouth opening. J Oral Sci. 2011;53(3):321–325. [PubMed] Stoll ML, Good J, Sharpe T, et al. Intra-articular corticosteroid injections to the temporomandibular joints are safe and appear to be effective therapy in children with juvenile idiopathic arthritis. J Oral Maxillofac Surg. 2012;70(8):1802–1807. [PubMed] 38. El-Hakim IE, Abdel-Hamid IS, Bader A. Tempromandibular joint (TMJ) response to intra-articular dexamethasone injection following mechanical arthropathy: a histological study in rats. Nt J Oral Maxillofac Surg. 2005;34(3):305–310. [PubMed] Toller PA. Use and misuse of intra-articular corticosteroids in treatment of temporomandibular joint pain. Proc R Soc Med. 1977;70(7):461–463. [PMC free article][PubMed Manfredini D, Piccotti F, Guarda-Nardini L. Hyaluronic acid in the treatment of TMJ disorders: a systematic review of the literature. Cranio. 2010;28(3):166–176.

Diagnostic Nerve bocks for OFP
Type of block

- Region
  - Trigeminal
    - Inferior alveolar
    - Lingual
    - Intra oral infiltration
    - Auriculotemporal
    - Infraorbital
  - Temporomandibular
    - Muscular
    - Intracapsular
    - Extracapsular
  - **Trigger point**
    - **Dry needling**
    - **Injection**
    - **Acupuncture**
  - Cervical nerves
    - Occipital nerve block
  - Sympathetic Stellate

Variables

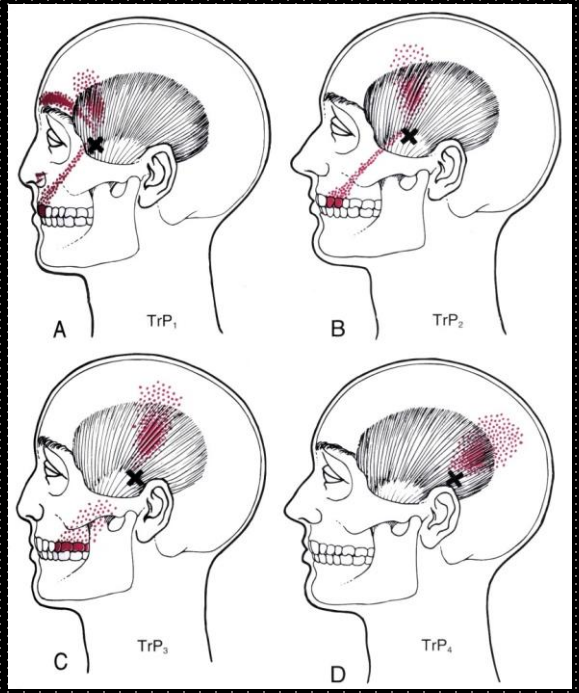
- Type of injection
  - Infiltration
    - Epithelial
    - Subepithelial
  - Nerve Block
    - Peripheral
    - Central
- Number of injections
- Agent
  - LA
  - Steroid
  - Botox
  - Anti inflammatory
  - Dry needling
  - NGF,
  - Combined

TMJ injections

Trigger point injections

Myalgia

- Local anaesthetics are primarily used when a myofascial trigger point is present.
- Myofascial trigger points are usually detected in the mastication muscles, but can also be found in numerous other muscles, such as the splenius capitis and upper trapezius. Due to its low toxicity to muscles, 1% procaine (1 cc) is recommended, but 1% or 2% lidocaine is also commonly used.
- The trigger point injection technique involves locating the trigger point, which is usually found in a taut band of muscle, and needling the area. The patient should be instructed that the muscles may be sore for the first 48 hours after the injection, but should begin to improve thereafter.
- The efficacy of trigger point injections is highly variable and dependent, for the most part, on the patient's compliance with a strict physical therapy regimen in conjunction with the injections. In addition, local anesthetics can be used to block the likely source of pain to confirm a diagnosis.



Journal of Pain Research
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REVIEW

Orofacial pain management: current perspectives

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James M Uyanik

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Abstract: Some of the most prevalent and debilitating pain conditions arise from the structures innervated by the trigeminal system (head, face, masticatory musculature, temporomandibular joint and associated structures). Orofacial pain (OFP) can arise from different regions and etiologies. Temporomandibular disorders (TMD) are the most prevalent orofacial pain conditions for which patients seek treatment. Temporomandibular disorders include a number of clinical problems that involve the masticatory musculature, the temporomandibular joint (TMJ) or both. Trigeminal neuropathic pain conditions can arise from injury secondary to dental procedures, infection, neoplasias, or disease or dysfunction of the peripheral and/or central nervous system. Neurovascular disorders, such as primary headaches, can present as chronic orofacial pain, such as in the case of facial migraine, where the pain is localized in the second and third division of the trigeminal nerve. Together, these disorders of the trigeminal system impact the quality of life of the sufferer dramatically. A multidisciplinary pain management approach should be considered for the optimal treatment of orofacial pain disorders including both non-pharmacological and pharmacological modalities.

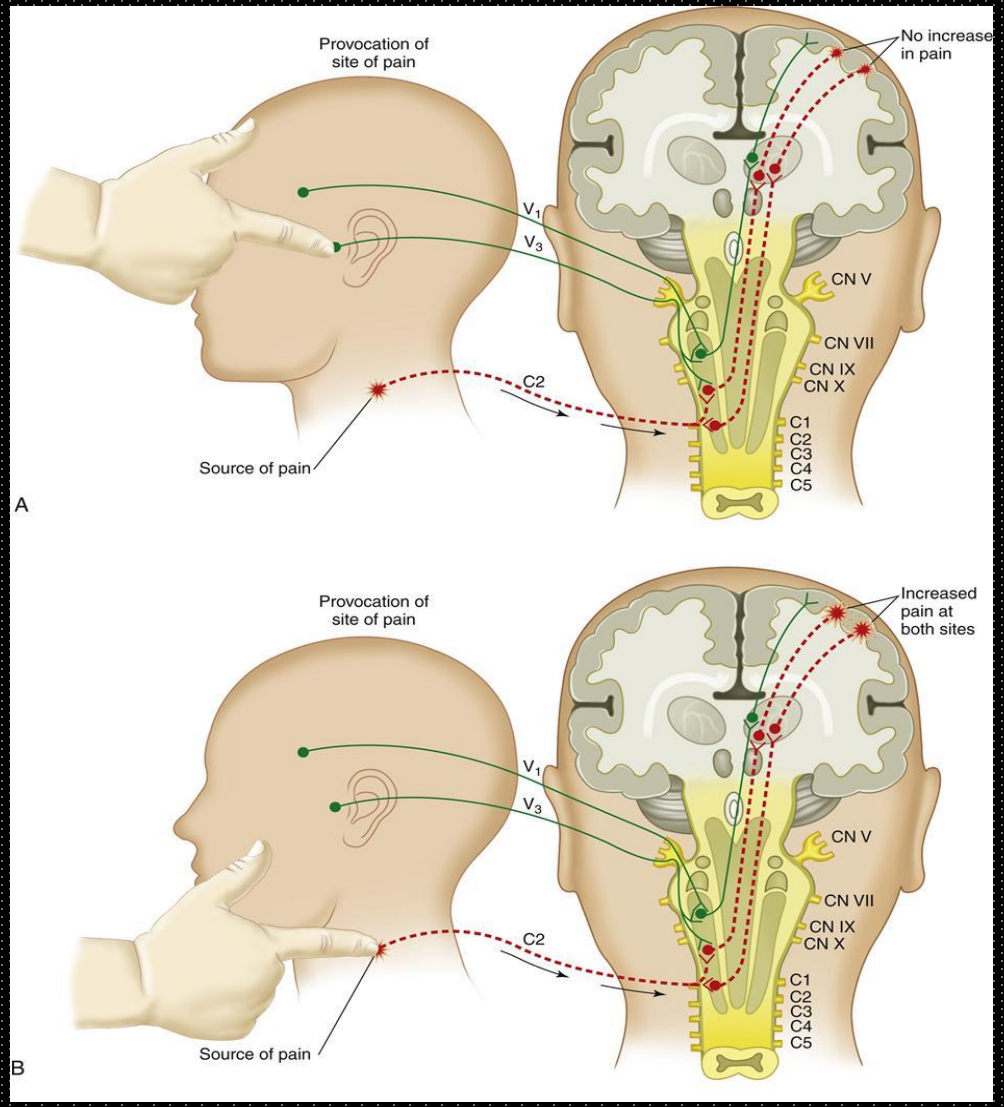
Keywords: pain, orofacial, neuropathic, TMD, trigeminal, headache

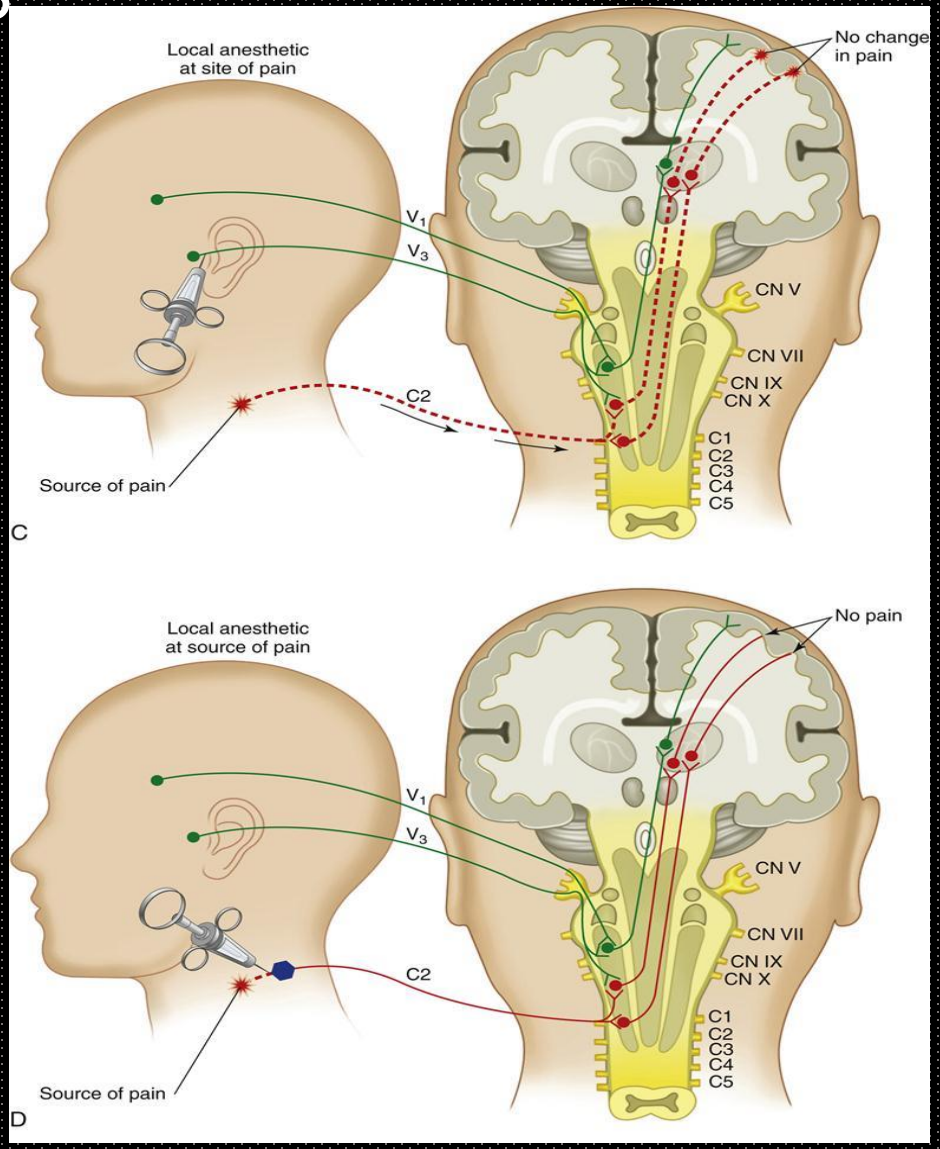
Orofacial pain disorders

Orofacial pain disorders are highly prevalent and debilitating conditions involving the head, face, and neck. These conditions represent a challenge to the clinician since the orofacial region is complex and therefore, pain can arise from many sources. The clinician needs to have solid knowledge of the pain conditions that arise from these structures for proper diagnosis and a multidisciplinary approach of management is

TMD
Trigger point/Dry needling

Uemoto L, Garcia MA, Gouvêa CV, Vilella OV, Alfaya TA Laser therapy and needling in myofascial trigger point deactivation. J Oral Sci. 2013;55(2):175-81.





Trigger Point Injection
Rationale

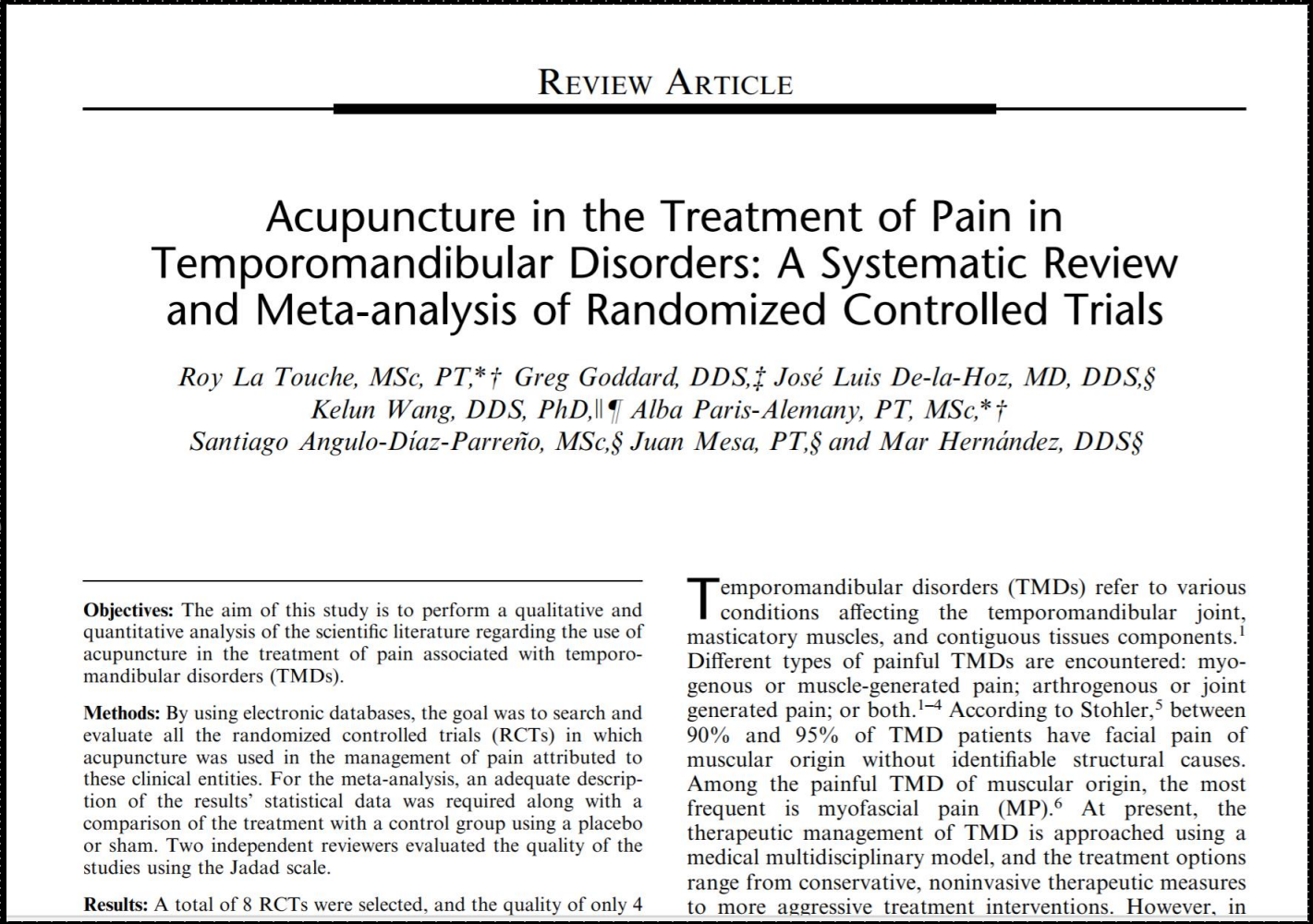
- Myofascial pain syndrome is characterized by pain associated with movement of affected muscles and reproduction of pain with palpation of well localized trigger points in the affected muscle.
- Myofascial syndrome is commonly found in association with other painful disorders, such as TMDs, cervicogenic pain, headaches, facet arthropathy or radiculopathy, and it is often helpful to determine whether a patient's pain is predominantly myofascial, because appropriate treatment may be very different if such is the case.
  - Reproduction of pain during injection into the area and relief of pain after injection for the expected duration of local anesthetic are used to indicate that myofascial pain is at least partially responsible for the patient's pain.
  - Other means of documenting myofascial pain, such as electromyography, have not been proved reliable.
  - Muscle tenderness is also seen in fibromyalgia, which differs from myofascial pain syndrome in that tender points in the muscle are much more diffuse and numerous and usually symmetrical, and palpation generally produces local, but not referred, pain.
  - Trigger point injections, particularly if repeated several times, may have therapeutic benefit for myofascial pain syndrome but are generally regarded as ineffective for fibromyalgia.
  - The predictable and selective destruction of mature myocytes by local anesthetic infiltration might be the therapeutic mechanism of long-term response to trigger point injection because it encourages the growth of a new generation of myocytes.

Trigger point injections
Limitations

- Undesired spread to adjacent nerves should be considered in interpreting trigger point injections.
- Doubt regarding the specificity of diagnosis by trigger point injection is raised by reports showing comparable efficacy from less specific techniques, such as needle insertion without injection, and from jet injection of local anaesthetic into the skin that overlies trigger points.
- Controlled studies have not confirmed the ability of intramuscular local anaesthetic injection to identify muscle or fascia as the pain source, although the simplicity of the procedure for superficial muscles is persuasive. Evidence base D

TMD
Dry needling

- Dry needling uses a thin filiform needle to penetrate the skin and stimulate underlying myofascial trigger points, muscular, and connective tissues for the management of neuro-musculoskeletal pain and impairment in movement. The advantages of dry needling are increasingly documented and include an immediate reduction in local, referred, and widespread pain, restoration of range of motion and muscle activation patterns, and a normalization of the immediate chemical environment of active myofascial trigger points.
- The aim of the treatment was to deactivate the myofascial trigger points in the orofacial muscles.



REVIEW ARTICLE

Acupuncture in the Treatment of Pain in Temporomandibular Disorders: A Systematic Review and Meta-analysis of Randomized Controlled Trials

Roy La Touche, MSc, PT,† Greg Goddard, DDS,‡ José Luis De-la-Hoz, MD, DDS,§ Kelun Wang, DDS, PhD,‖¶ Alba Paris-Alemany, PT, MSc,*† Santiago Angulo-Díaz-Parreño, MSc,§ Juan Mesa, PT,§ and Mar Hernández, DDS§*

Objectives: The aim of this study is to perform a qualitative and quantitative analysis of the scientific literature regarding the use of acupuncture in the treatment of pain associated with temporomandibular disorders (TMDs).

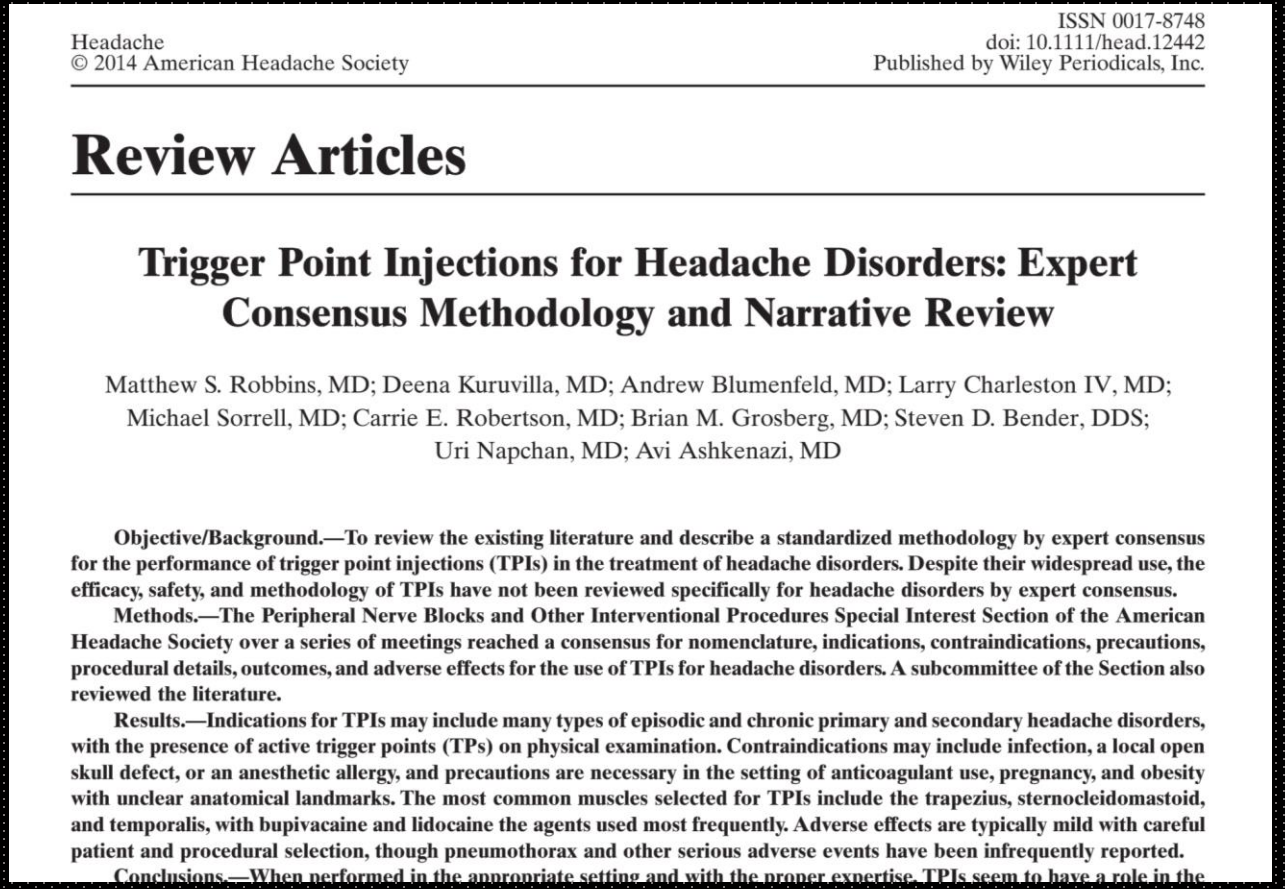
Methods: By using electronic databases, the goal was to search and evaluate all the randomized controlled trials (RCTs) in which acupuncture was used in the management of pain attributed to these clinical entities. For the meta-analysis, an adequate description of the results' statistical data was required along with a comparison of the treatment with a control group using a placebo or sham. Two independent reviewers evaluated the quality of the studies using the Jadad scale.

Results: A total of 8 RCTs were selected, and the quality of only 4

Temporomandibular disorders (TMDs) refer to various conditions affecting the temporomandibular joint, masticatory muscles, and contiguous tissues components.[1] Different types of painful TMDs are encountered: myogenous or muscle-generated pain; arthrogenous or joint generated pain; or both.[1–4] According to Stohler,[5] between 90% and 95% of TMD patients have facial pain of muscular origin without identifiable structural causes. Among the painful TMD of muscular origin, the most frequent is myofascial pain (MP).[6] At present, the therapeutic management of TMD is approached using a medical multidisciplinary model, and the treatment options range from conservative, noninvasive therapeutic measures to more aggressive treatment interventions. However, in

Headache
Trigger point injections

- When performed in the appropriate setting and with the proper expertise, TPIs seem to have a role in the
- adjunctive treatment of the most common headache disorders. We hope our effort to characterize the methodology of TPIs by
- expert opinion in the context of published data motivates the performance of evidence-based and standardized treatment protocols.



Headache
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Review Articles

Trigger Point Injections for Headache Disorders: Expert Consensus Methodology and Narrative Review

Matthew S. Robbins, MD; Deena Kuruvilla, MD; Andrew Blumenfeld, MD; Larry Charleston IV, MD; Michael Sorrell, MD; Carrie E. Robertson, MD; Brian M. Grosberg, MD; Steven D. Bender, DDS; Uri Napchan, MD; Avi Ashkenazi, MD

Objective/Background.—To review the existing literature and describe a standardized methodology by expert consensus for the performance of trigger point injections (TPIs) in the treatment of headache disorders. Despite their widespread use, the efficacy, safety, and methodology of TPIs have not been reviewed specifically for headache disorders by expert consensus.

Methods.—The Peripheral Nerve Blocks and Other Interventional Procedures Special Interest Section of the American Headache Society over a series of meetings reached a consensus for nomenclature, indications, contraindications, precautions, procedural details, outcomes, and adverse effects for the use of TPIs for headache disorders. A subcommittee of the Section also reviewed the literature.

Results.—Indications for TPIs may include many types of episodic and chronic primary and secondary headache disorders, with the presence of active trigger points (TPs) on physical examination. Contraindications may include infection, a local open skull defect, or an anesthetic allergy, and precautions are necessary in the setting of anticoagulant use, pregnancy, and obesity with unclear anatomical landmarks. The most common muscles selected for TPIs include the trapezius, sternocleidomastoid, and temporalis, with bupivacaine and lidocaine the agents used most frequently. Adverse effects are typically mild with careful patient and procedural selection, though pneumothorax and other serious adverse events have been infrequently reported.

Conclusions.—When performed in the appropriate setting and with the proper expertise, TPIs seem to have a role in the

Headache
Trigger Point Injections

Review Article

Peripheral Nerve Blocks and Trigger Point Injections in Headache Management – A Systematic Review and Suggestions for Future Research

Lipton

syndromes. Moreover, there is no widely accepted agreement among headache specialists as to the optimal technique of injection, type, and doses of the local anesthetics used, and injection regimens. The role of corticosteroids in this setting is also debated.

- We performed a PubMed search of the literature to find studies on PNBs and TPIs for headache treatment. We classified the abstracted studies based on the procedure performed and the treated condition. We found few controlled studies on the efficacy of PNBs for headaches, and virtually none on the use of TPIs for this indication.
- **The most widely examined procedure in this setting was greater occipital nerve block,** with the majority of studies being small and non-controlled. The techniques, as well as the type and doses of local anesthetics used for nerve blockade, varied greatly among studies. The specific conditions treated also varied, and included both primary (eg, migraine, cluster headache) and secondary (eg, cervicogenic, posttraumatic) headache disorders.
- Trigeminal (eg, supraorbital) nerve blocks were used in few studies. Results were generally positive, but should be taken with reservation given the methodological limitations of the available studies. The procedures were generally well tolerated. Evidently, there is a need to perform more rigorous clinical trials to clarify the role of PNBs and TPIs in the management of various headache disorders, and to aim at standardizing the techniques used for the various procedures in this setting

Diagnostic Nerve bocks for OFP
Type of block

- Region
  - Trigeminal
    - Inferior alveolar
    - Lingual
    - Intra oral infiltration
    - Auriculotemporal
    - Infraorbital
  - Temporomandibular
    - Muscular
    - Intracapsular
    - Extracapsular
  - Trigger point
    - Dry needling
    - Injection
    - Acupuncture
  - **Cervical nerves**
    - **Occipital nerve block**
  - Sympathetic Stellate
  - Sphenopalatine block

Variables

- Type of injection
  - Infiltration
    - Epithelial
    - Subepithelial
  - Nerve Block
    - Peripheral
    - Central
- Number of injections
- Agent
  - LA
  - Steroid
  - Botox
  - Anti inflammatory
  - Dry needling
  - NGF,
  - Combined

Greater Occipital Nerve Block
Rationale

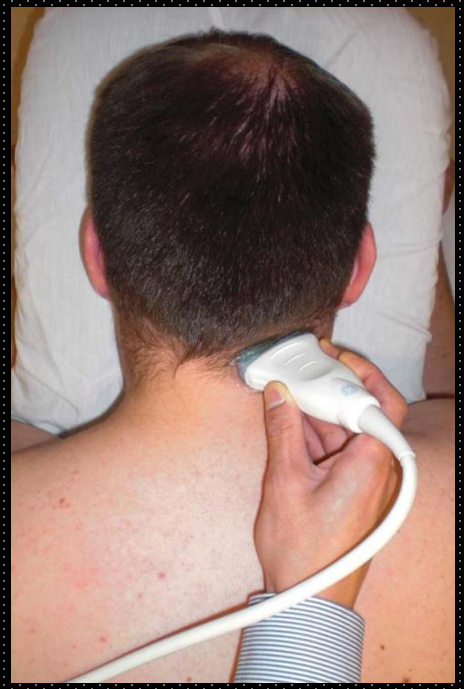
- The greater occipital nerve is the continuation of the medial branch of the posterior primary ramus of the C2 spinal nerve, which distributes cutaneous sensory fibres to the scalp as far rostral as the vertex.
- Several theories have proposed involvement of the C2 spinal and greater occipital nerves in production of headache. Initial analysis suggested that the origin of the spinal nerve or the posterior root ganglion may be pinched between the atlas and axis by extension and rotation.
- Further research proved that this is mechanically unlikely.
- A more popular theory invokes irritation of the greater occipital nerve as it penetrates muscle layers. The passage through the muscular portion of the semispinalis capitus is rarely restricted, but the aperture through the trapezius is by a non distensible channel that typically deforms the nerve.
- Entrapment here may be the origin of nerve irritation that initiates neuralgic pain.
- Greater occipital neuralgia and cervical facet arthropathy are putative sources of cervicogenic headache, which is clinically distinguished from migraine and tension-type headaches by unilateral pain, symptoms and signs of neck involvement (ipsilateral neck, shoulder or arm pain; tenderness or postural pain in the neck; decreased range of neck motion), nonclustering moderate pain that throbs and spreads forward from the neck, and a history of head or neck trauma.
- Transient elimination of pain by greater occipital nerve block is used as a key criterion in the evaluation of cervicogenic headache.

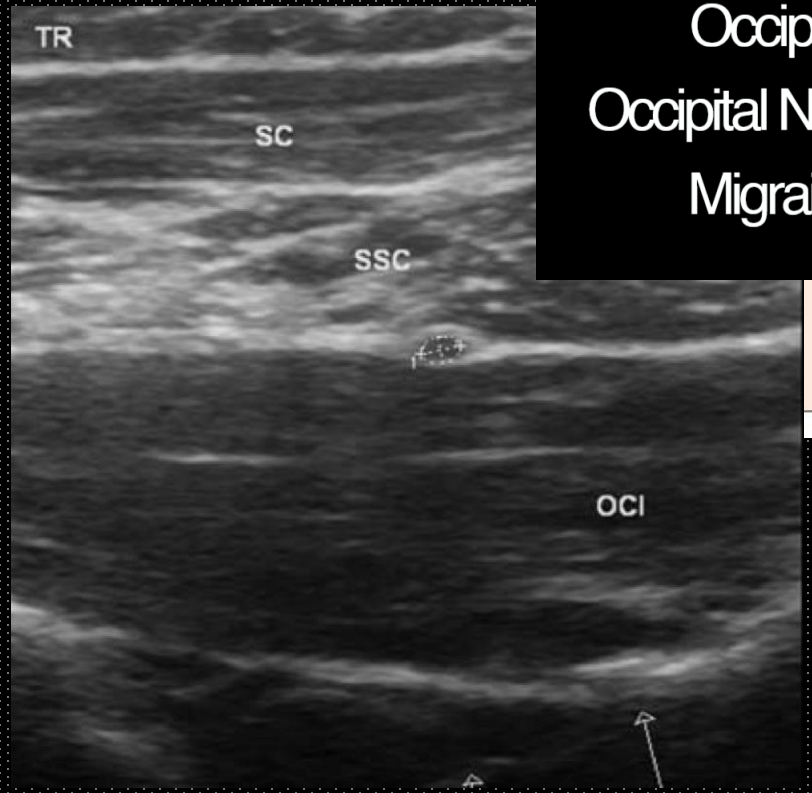
No evidence for efficacy of ONB in diagnosis for OFP

Nerve blocks for OFP

Occipital nerve block

TR Trapezius muscle
SC Splenius capitis
SSC semi spinalis capitis
OCI Obliqus capitis





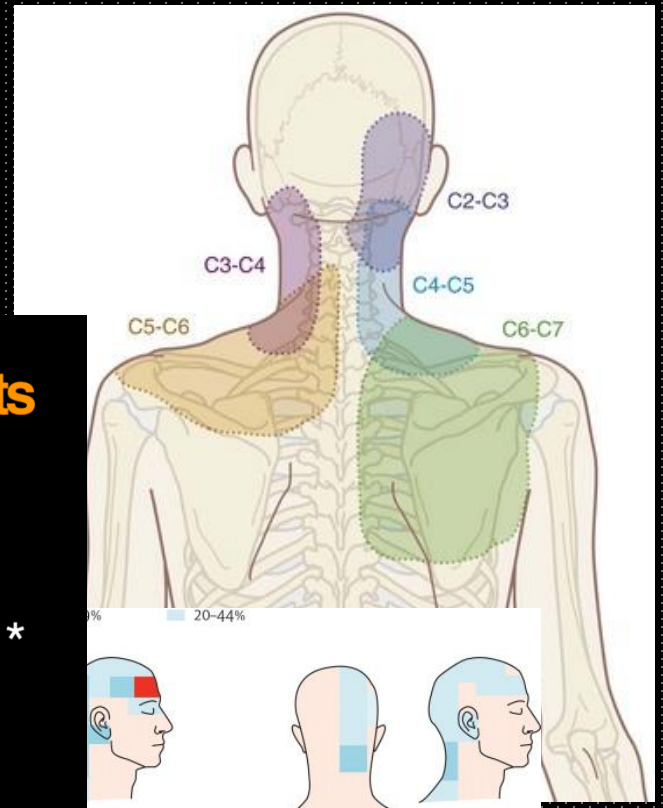
US GUIDED ON BLOCK - 7pts

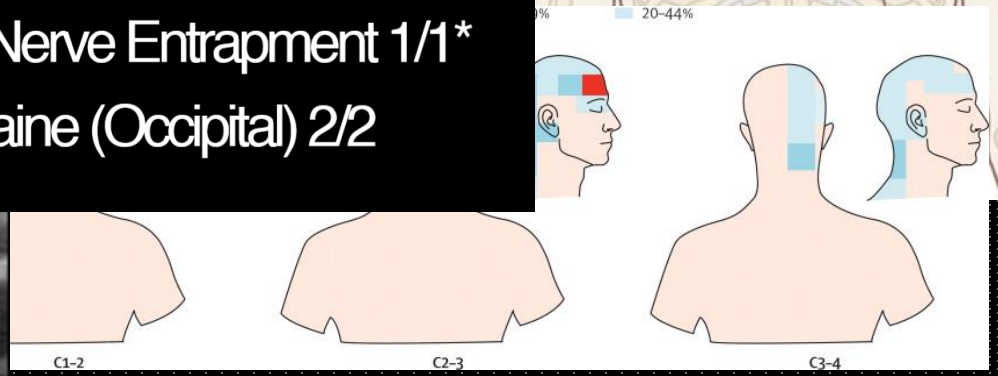
SUNCT/SUNA 0/2

Occipital Neuralgia 2/2

Occipital Nerve Entrapment 1/1*

Migraine (Occipital) 2/2





No evidence published for orofacial pain conditions……
Our experience 65 patients
Positive outcome treating migraine related TMD

Controversies in Neurology

Greater Occipital Nerve Block: A Diagnostic Test?

Often a neurosurgeon or orthopedic surgeon requests a diagnostic nerve block to determine, prior to attempting any surgical procedure, whether a specific cervical nerve root is the generator of the patient's symptoms.[1]

This statement from a contemporary textbook reflects the commonly held view that in pain management nerve blocks are diagnostic.

There is wide variability of headache syndromes treated by greater occipital nerve (GON) blockade.[1] The putative mechanisms by which they might relate to the GON are unclear. It seems a priori improbable that such diverse conditions as migraine (with its complex cere-

Are nerve blocks diagnostic?

Neural blocks may be useful as an empirical way of treating diverse head and neck pains, but such a response is also often used as the criterion for diagnosis.[7,8] But such diagnoses, though clinically useful, are inexact and the procedure may be valid (if proven by properly designed trials) only as an empirical mode of controlling pain.

Blondi rightly notes that *"Occipital nerve blockade, … often results in a nonspecific regional blockade rather than a specific nerve blockade and might result in a misidentification of the occipital nerve as the source of pain."* And he says: *"occipital neuralgia is believed to arise from trau-*

JMS Pearce MD, FRCP is an Emeritus Consultant Neurologist at the Hull Royal Infirmary. His interests are in Clinical Neurology

In respect of diagnosis, the current evidence appraised suggests that the use of nerve blocks as the defining or pathogenetic criterion is both unsound and unreliable

Neurotherapeutics: The Journal of the American Society for Experimental NeuroTherapeutics

Nerve Blocks in the Treatment of Headache

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Summary: Nerve blocks and neurostimulation are reasonable therapeutic options in patients with head and neck neuralgias. In addition, these peripheral nerve procedures can also be effective in primary headache disorders, such as migraine and cluster headaches. Nerve blocks for headaches are generally accomplished by using small subcutaneous injections of amide-type local anesthetics, such as lidocaine and bupivicaine. Targets include the greater occipital nerve, lesser occipital nerve, auriculotemporal nerve, supratrochlear and supraorbital nerves, sphenopalatine ganglion, cervical spinal roots, and facet joints of the upper cervical spine. Although definitive studies examining the usefulness of nerve blocks are lacking, reports suggest that this area deserves further attention in the hope of acquiring evidence of effectiveness. **Key Words:** Nerve blocks, occipital nerve, greater occipital nerve, occipital neuralgia.

Cervicogenic headache: Techniques of diagnostic nerve blocks

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words: vicogenic headache, diagnostic e block, greater occipital nerve k, intervertebral disc block, minor occipital nerve block, segmental nerve block, zygapophyseal joint block.

ABSTRACT

The term cervicogenic headache (CEH) was introduced by Sjaastad and coworkers in 1983. In 1990 Sjaastad et al. published diagnostic criteria for CEH. In 1998 refinements of these criteria were published, emphasising the use of diagnostic nerve blocks in patients with CEH as important confirmatory evidence. However, the standardisation of diagnostic nerve blocks in the diagnosis of CEH remains to be defined. Herein we present an overview of diagnostic nerve blocks in the cervical area. Suggestions as to their role in the diagnosis of CEH are given.

Introduction

Cervicogenic headache (CEH) is a clinically defined headache syndrome which is hypothesised to originate from nociceptive structures in the cervical spine. Early publications of this concept were

afferents from the field of the trigeminal nerve and the receptive fields of the first three cervical nerve roots (7-11). This may imply that CEH mainly emanates from structures innervated by the first three cervical nerve roots, whereby the C2 cord segment provides an important relay of afferent fibres (12,13). However, other observations suggest that headache may also arise from structures in the lower cervical spine (14-16). Various structures in the cervical spine are capable of causing neck pain and headache such as the zygapophyseal joints, segmental nerves, dorsal root ganglia, intervertebral discs, muscles and ligaments (11, 15, 17-19). Other authors have reported the existence of venous vascular and non-vascular compression of the upper cervical roots in patients with CEH (20, 21).

Although diagnostic nerve blocks are an obligatory point in establishing the di-

High-quality studies on PNBs are scarce and no official guidelines pertaining to their use have been formulated. The American Headache Society Special Interest Section for PNBs and other Interventional Procedures published expert consensus recommendations in 2013 regarding the use of PNBs in the treatment of headache disorders.[5] These recommendations were partly based on responses from a physician survey on PNB drug dosages, injection volumes and schedules, and other aspects of PNBs.

Diagnostic Nerve bocks for OFP
Type of block

- Region
  - Trigeminal
    - Inferior alveolar
    - Lingual
    - Intra oral infiltration
    - Auriculotemporal
    - Infraorbital
  - Temporomandibular
    - Muscular
    - Intracapsular
    - Extracapsular
  - Trigger point
    - Dry needling
    - Injection
    - Acupuncture
  - Cervical nerves
    - Occipital nerve block
  - **Sympathetic Stellate**
  - Sphenopalatine block

Variables

- Type of injection
  - Infiltration
    - Epithelial
    - Subepithelial
  - Nerve Block
    - Peripheral
    - Central
- Number of injections
- Agent
  - LA
  - Steroid
  - Botox
  - Anti inflammatory
  - Dry needling
  - NGF,
  - Combined

Selective Sympathetic Blockade
Rationale

- Sympathetic efferent activity is a suspected pathogenic component in a number of conditions.
  - hyperhidrosis, the participation of sympathetic fibres is well documented.
  - sudden sensory-neural hearing loss
  - peripheral vascular disease
  - dysrhythmia from long-QT syndrome
  - central pain, pain after plexus injury,
  - trigeminal or postherpetic neuralgia,
  - large category of poorly defined pain states that are grouped under the terms reflex sympathetic dystrophy or causalgia, a sympathetic contribution is suspected because blood flow and trophic changes are evident, but the pathophysiology is largely obscure.

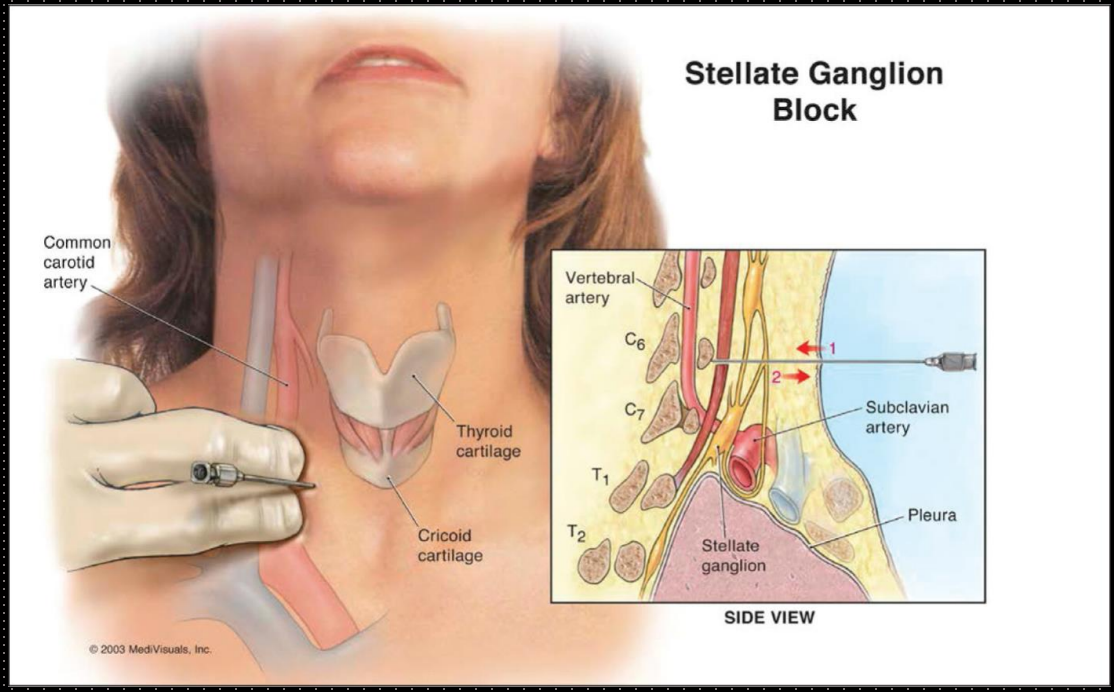
Sympathetic system and chronic pain

Changes in the sympathetic nervous system associated with chronic pain have been described for well over a century.

A complex relationship exists between the somatosensory and sympathetic nervous systems, with sympathetic involvement most likely to be a phenomenon rather than a cause.

The mechanism of sympathetic involvement is still poorly understood and debated. The diagnostic criteria of complex regional pain syndrome (CRPS) include sympathetically-mediated changes.

Sympathetic blocks can be used in the treatment of pain conditions, in conjunction with a multidisciplinary approach including the physical and psychological therapies. The evidence base remains weak for many treatments.



Limited evidence for efficacy of sympathetic blocks in diagnosis for OFP

Diagnostic stellate ganglion blocks for reflex sympathetic dystrophy of the face

The Laryngoscope
Lippincott–Raven Publishers, Philadelphia

Reflex Sympathetic Dystrophy of the Face: Current Treatment Recommendations

Richard L. Arden, MD; Samer J. Bahu, MD; Marcos A. Zuazu, MD; Ramon Berguer, MD, PhD

Reflex sympathetic dystrophy (RSD) of the face is an infrequently reported clinical pain syndrome characterized by dysesthesia, hyperalgia, hyperpathia, and allodynia. Treatment strategies, extrapolated from RSD and causalgia of the extremities, remain variable and poorly defined. Sympathetic blockade is generally the diagnostic and therapeutic treatment of choice; however, the frequency, timing, and duration of injections; need for neurolytic blocks; and role of sympathectomy are not well understood. The objectives of this report are to highlight the clinical behavior of facial RSD and contrast its essential differences from extremity RSD in response to standard treatment regimes. The case studies of two patients with this syndrome, following vascular surgery in the neck, are retrospectively reviewed with existent reported cases. Age, gender, etiology, symptoms, onset, triggers, and examination findings; timing, duration, and method of treatment; and outcome are summarized, forming the database for this study. Findings demonstrate an infrequent association of vasomotor and sudomotor changes with facial RSD, and lack of progression to a dystrophic or an atrophic stage, in contrast to extremity RSD. Furthermore, treatment response to sympathetic blockade is durable and less critically dependent on timing. The authors conclude that facial RSD has a favorable prognosis and should be managed conservatively with nonneurolytic stellate ganglion blocks, even when initiated as a delayed and repetitive injection series.

***Laryngoscope*, 108:437–442 1998**

INTRODUCTION

Sympathetically mediated pain syndromes include causalgia and reflex sympathetic dystrophy (RSD), which fundamentally differ in their precipitating event, histopathologic correlate, and potential for clinical progression. Initially described by Mitchell et al.[1] in 1864, causalgia is a syndrome of sustained, burning pain following partial, incomplete nerve damage, typically following a high-velocity missile injury to a major peripheral nerve. The level of injury is characteristically confined to nerve lesions above the elbow and knee (most commonly median and sciatic nerves) and accounts for 2% to 5% of peripheral nerve injury cases.[2] The more severe and proximate the neural lesion to the spinal ganglia, the greater the degree of causalgic response. The burning pain (dysesthesia) commonly appears immediately or soon after the injury and is spontaneous, continuous, and felt superficially in the hand or foot. For most patients (75%) the pain gradually subsides within 1 year, but during recovery they often experience lowered pain thresholds (hyperesthesia), elevated thresholds to touch, and overreactions/aftersensations to stimuli (hyperpathia). Exacerbating factors have been associated with dependent posturing, mechanical or thermal stimuli (allodynia), muscular activity, and disturbances in the sensory neuronal pool (i.e., auditory, visual, somatosensory, emotional stress). Treatment responses to neuronal blockade, truncectomy, or rhizotomy have been poor or unsuccessful in most cases, in contrast to sympathetic block (or sympathectomy), which has provided significant temporary (occasionally permanent) pain relief.

Reflex sympathetic dystrophy is a term first used by Evans in 1947 to describe a pain syndrome following various types of mild injuries in the absence of demonstrable nerve damage.[3] Unlike the rapid and violent neural deformation associated with causalgia, precipitating events in RSD have included fractures (>50%), lacerations, infections, operations, angina/myocardial infarction, peripheral vascular disease, degenerative joint disease, and injuries to muscles, ligaments, or soft tissue. In 10% to 26% of cases, no precipitating factor can be found.[4] Similar to causalgia, RSD pain possesses the components of dysesthesia, hyperesthesia, and hyperpathia, which seem to follow the topography of the sympathetically innervated vascular system rather than a true radicular or dermatomal pattern. In contrast to

From the Departments of Otolaryngology—Head and Neck Surgery

TABLE II.
Treatment and Outcome Summary.

Case #	Report (year)	Treatment Initiated	Method	Outcome
1	Bingham[5] (1947)	13 mo after injury	Single (R) SG block, procaine/alcohol	Recurrence facial/pain tenderness at 3 w
		14 mo after injury	Cervical sympathectomy	Pain-free at 3 mo follow-up
2	Bingham[5] (1947)	11 mo after injury	Single (L) SG block, procaine/alcohol	Recurrence mild pain/hyperesthesia at 2 mo, severe at 9 months
		20 mo after injury	Cervical sympathectomy	Symptom resolution; no follow-up
3	Hanowell and Kennedy[6] (1979)	7 mo after surgery	Diagnostic (L) SG block, bupivicaine Alternate day, 5 block series	60% improvement at 2 d Pain-free at 3 mo follow-up
4	Khoury et al.[7](1980)	7 y after surgery	Diagnostic (L) SG block, bupivicaine	Pain relief for 6 h
			20 block series	75% improvement after last injection
5	Jaeger et al.[8](1986)	1 y after extraction	Diagnostic (L) SG block (local anesthetic unspecified)	Relief beyond anesthetic duration
			15 block series	Pain-free at 15-mo follow-up
6	Jaeger et al.[8] (1986)	3 y after surgery	Diagnostic bilateral SG blocks (local anesthetic unspecified)	Near-complete facial pain relief
			Bilateral morphine sulfate SG blocks (number unspecified)	66% improvement facial pain; persistent dysesthetic scar pain
7	Veldman and Jacobs[9] (1994)	1 y after surgery	*N*-acetylcysteine, 600 mg tid	Partial decrease facial pain; decreased size red, swollen, warm areas
8	Saxen et al. (1995)	10 y after extraction	Diagnostic (L) SG block, bupivicaine therapeutic (L) SG block	Pain relief for 24 h
			Clonidine, 0.1 mg bid	Not specified
			Responded well; follow-up not specified	
9	Arden et al. (1998)	6 w after surgery	Diagnostic (R) SG block, bupivicaine	Relief beyond anesthetic duration
			Weekly, 6 (R) SG block series × 1.5 (R) SG block, phenol	40–50% improvement facial pain
			3 weekly, (L) SG blocks	No change from baseline
			5 monthly, (L) SG blocks	60% improvement in pain
				70% improvement at 6 mo, 80%–85% improvement at 8 mo
10	Arden et al. (1998)	1 mo after surgery	Diagnostic (R) SG block, bupivicaine	Relief beyond anesthetic duration
			Weekly, 5 (R) SG blocks × 1.5 mo	50%–70% reduction in facial pain

Stellate Ganglion Block for OFP Evidence

- Kojitani et al. reported that SGB added to amitriptyline medication successfully alleviated neuropathic pain after simple tooth extraction [5].
- Matsuura et al. performed SGB 2 times a week in 35 patients with postoperative ocular pain that was resistant to anti-inflammatory drugs [22].
- It was found that SGB, performed an average of 5.9 times, was effective for 96.6% of patients with nociceptive pain [22].
- In a report by Lynch et al., SGB was performed in 14 patients with orofacial neuropathic pain. They observed that five patients noted 50-100% improvement in pain severity 12 months after SGB [19].
- Recently, it was suggested that a trial of SGB in the early stages of various orofacial pain disorders could result in greater reduction in pain severity [21,30,31]. It was also suggested that SGB could prevent facial nerve damage caused by herpes zoster and postherpetic neuralgia that did not respond to medication including acyclovir, steroids, and antidepressants [31].

Bilateral stellate ganglion blockade for recalcitrant oral **pain** from Burning Mouth Syndrome: a case report.
Walega DR, Smith C, Epstein JB.
J Oral Facial **Pain** Headache. 2014 Spring;28(2):171-5.
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Sphenopalatine block for facial pain

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DOI 10.1186/s10194-017-0826-y

The Journal of Headache and Pain

REVIEW ARTICLE Open Access

Sphenopalatine ganglion: block, radiofrequency ablation and neurostimulation - a systematic review

Kwo Wei David Ho[1*], Rene Przkora[2] and Sanjeev Kumar[2]

Abstract

Background: Sphenopalatine ganglion is the largest collection of neurons in the calvarium outside of the brain. Over the past century, it has been a target for interventional treatment of head and facial pain due to its ease of access. Block, radiofrequency ablation, and neurostimulation have all been applied to treat a myriad of painful syndromes. Despite the routine use of these interventions, the literature supporting their use has not been systematically summarized. This systematic review aims to collect and summarize the level of evidence supporting the use of sphenopalatine ganglion block, radiofrequency ablation and neurostimulation.

Methods: Medline, Google Scholar, and the Cochrane Central Register of Controlled Trials (CENTRAL) databases were reviewed for studies on sphenopalatine ganglion block, radiofrequency ablation and neurostimulation. Studies included in this review were compiled and analyzed for their treated medical conditions, study design, outcomes and procedural

Table 1 Summary of evidence level and grade of recommendation for SPG block, radiofrequency ablation and neurostimulation

Medical condition	Application/ Medication used in controlled studies	Number of controlled studies	Highest level of evidence	Grade of recommendation
SPG block				
Cluster headache	Cotton swab/cocaine or lidocaine	1	2b	B
Second-division trigeminal neuralgia	Lidocaine spray	1	2b	B
Reducing the needs of analgesics after endoscopic sinus surgery	Needle injection, transnasal and palatal approach/lidocaine, bupivacaine, l evobupivacaine, tetracaine	6	1b	B
Reducing the pain associated with nasal packing removal after nasal operation	Needle injection, infrazygomatic approach/lidocaine	1	3b	B
Migraine	Tx360 device/ bupivicane	1	2b	B
Postdural puncture headache, sphenopalatine maxillary neuralgia, facial neuralgia, sympathetic neuralgia, post-traumatic atypical facial pain, atypical odontalgia, pain from midline granuloma, herpetic keratitis, hemifacial headache,paroxysmal hemicrania, nasal pain, hemicrania continua, trigeminal neuropathy, cancer pain, seizures associated nasal pathology, arthritic pain and muscle spasm, intercostal neuritis, persistent hiccups, ureteral colic, dysmenorrhea, peripheral painful vascular spasm, complex regional pain syndrome and hypertension	Various protocols	0	4	C
Myofascial pain	Cotton-tipped applicator, nasal spray/lidocaine	2	2b	Not recommended
SPG radiofrequency ablation				

Summary
Diagnostic PNBs in OFP

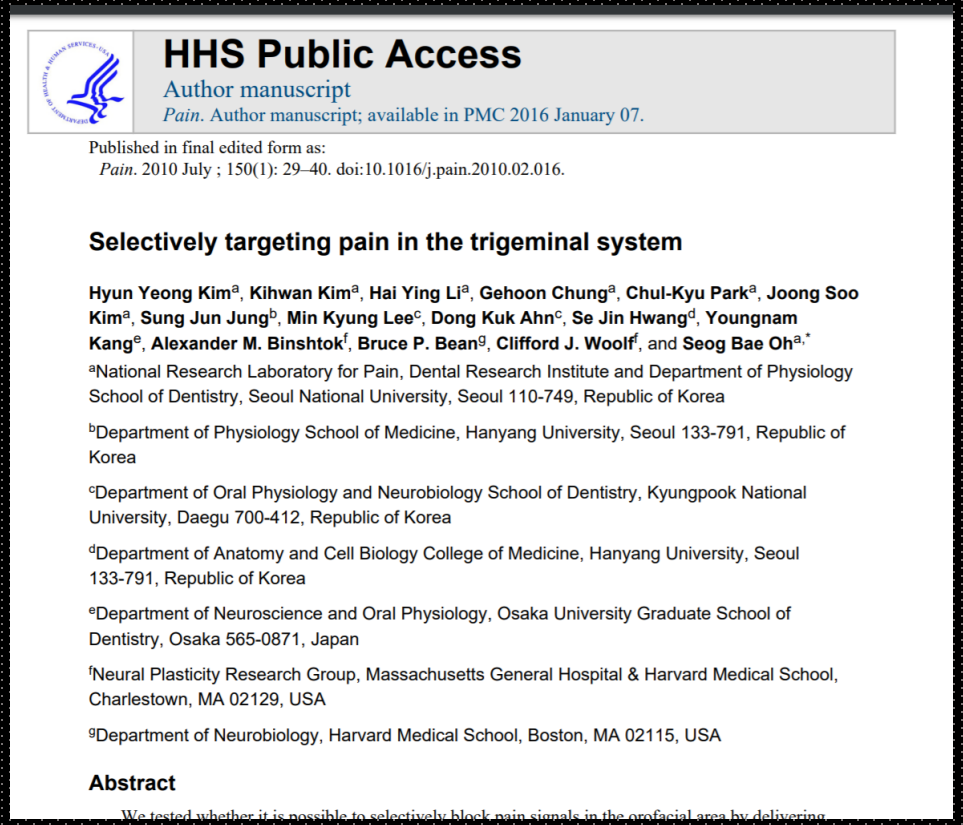
The role of neural blockade as a **diagnostic tool** in painful conditions may be compromised due to several characteristic of chronic pain including;

- social, emotional, financial, and legal factors effecting the patient
- the pathophysiology of clinical pain
- the site of nociception
- the pathway of afferent neural signals.

- Clinical studies of the blocks are variable quality.
- Important considerations include **entrance criteria**, study size, and the use of control subjects particularly **Diagnostic criteria for condition**
- The **prevalence of placebo responses** in patients with pain greatly weakens the relevance of studies in which no control subjects or blinding was used.
- The evidence baselevel rarely exceeds Grade D for all PNBs for diagnosis of OFP

The Future

- Diagnostic Blocks may assist in phenotyping OFP conditions
- Diagnostic Blocks may be able to predict treatment outcome for therapeutic blocks and other treatments but not yet!
- Selectively blocking pain signals in the orofacial area by delivering the permanently charged lidocaine derivative QX-314 into nociceptors via TPRV1 channels.



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Selectively targeting pain in the trigeminal system

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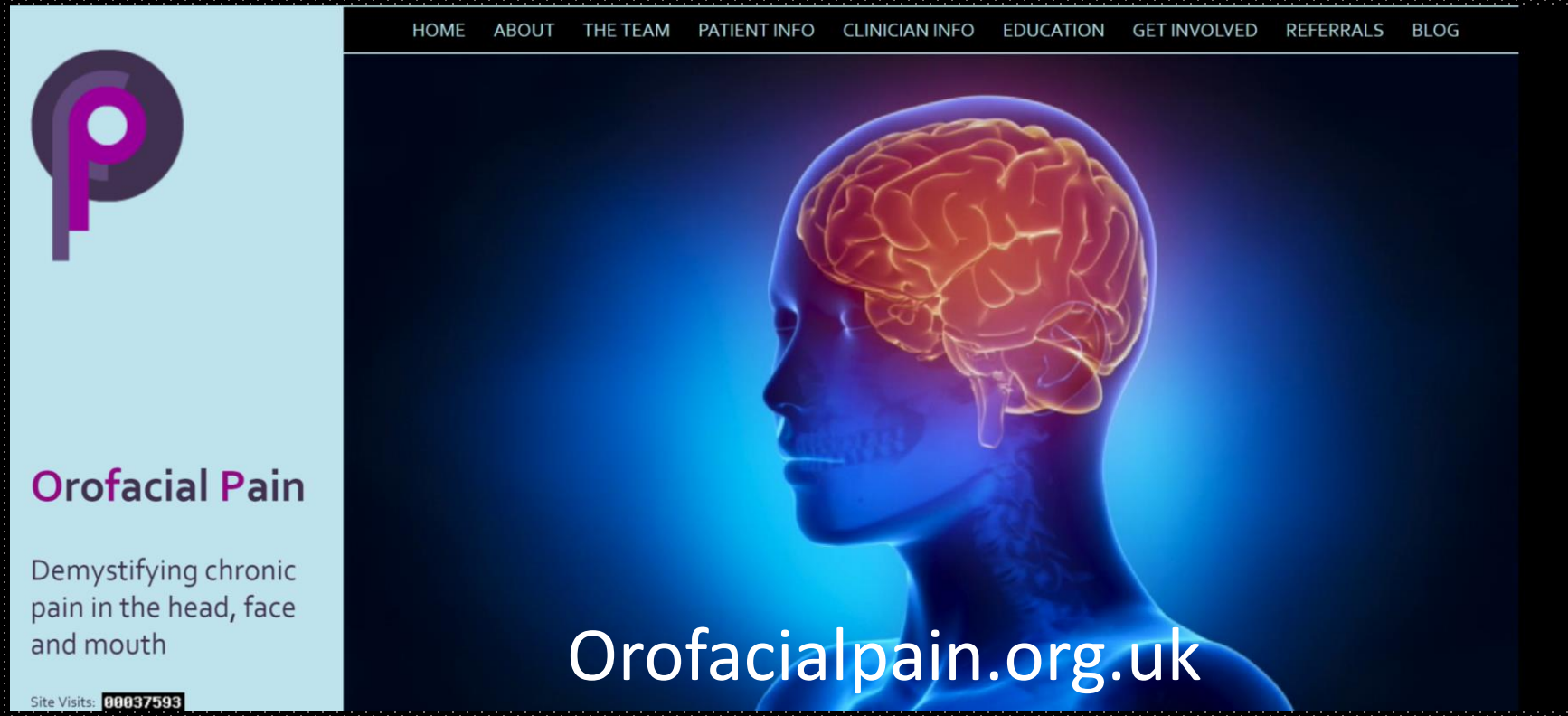
Abstract

We tested whether it is possible to selectively block pain signals in the orofacial area by delivering

Kim HY, Kim K, Li HY Chung G, Park_CK, Kim JS, Jung SJ, Lee MK, Ahn DK, Hwang SJ, Kang Y, Binshtok AM, Bean BP, Woolf CJ, Oh SB Selectively targeting pain in the trigeminal system. Pain. 2010 Jul;150(1):29-40. doi: 10.1016/j.pain.2010.02.016. Epub 2010 Mar 16.

Thank you



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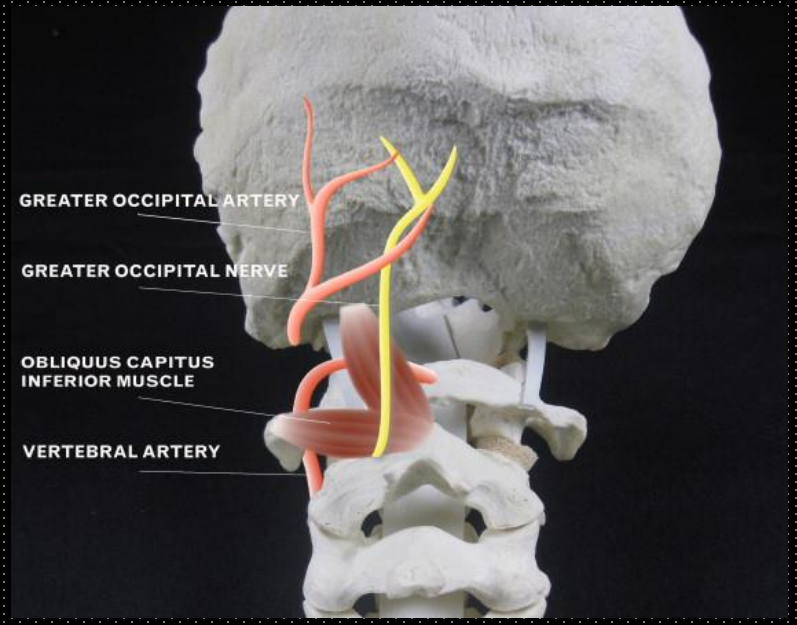
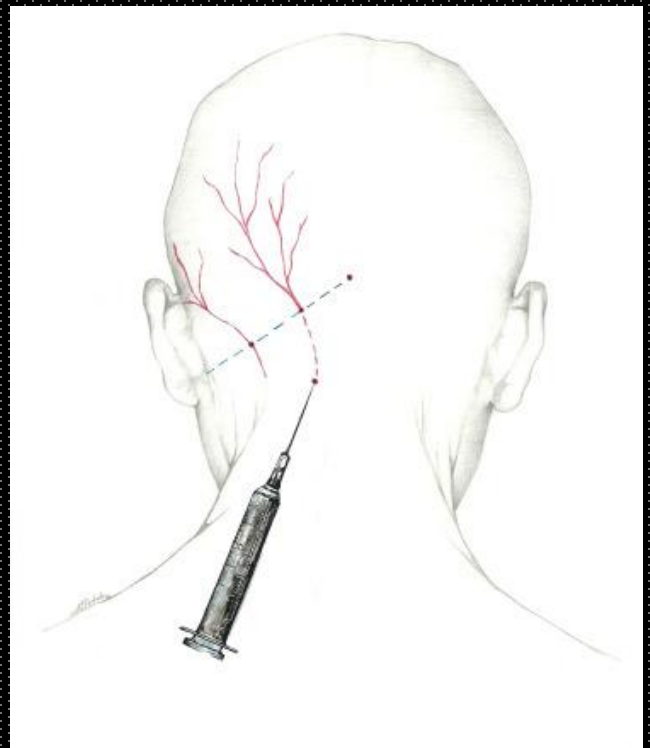
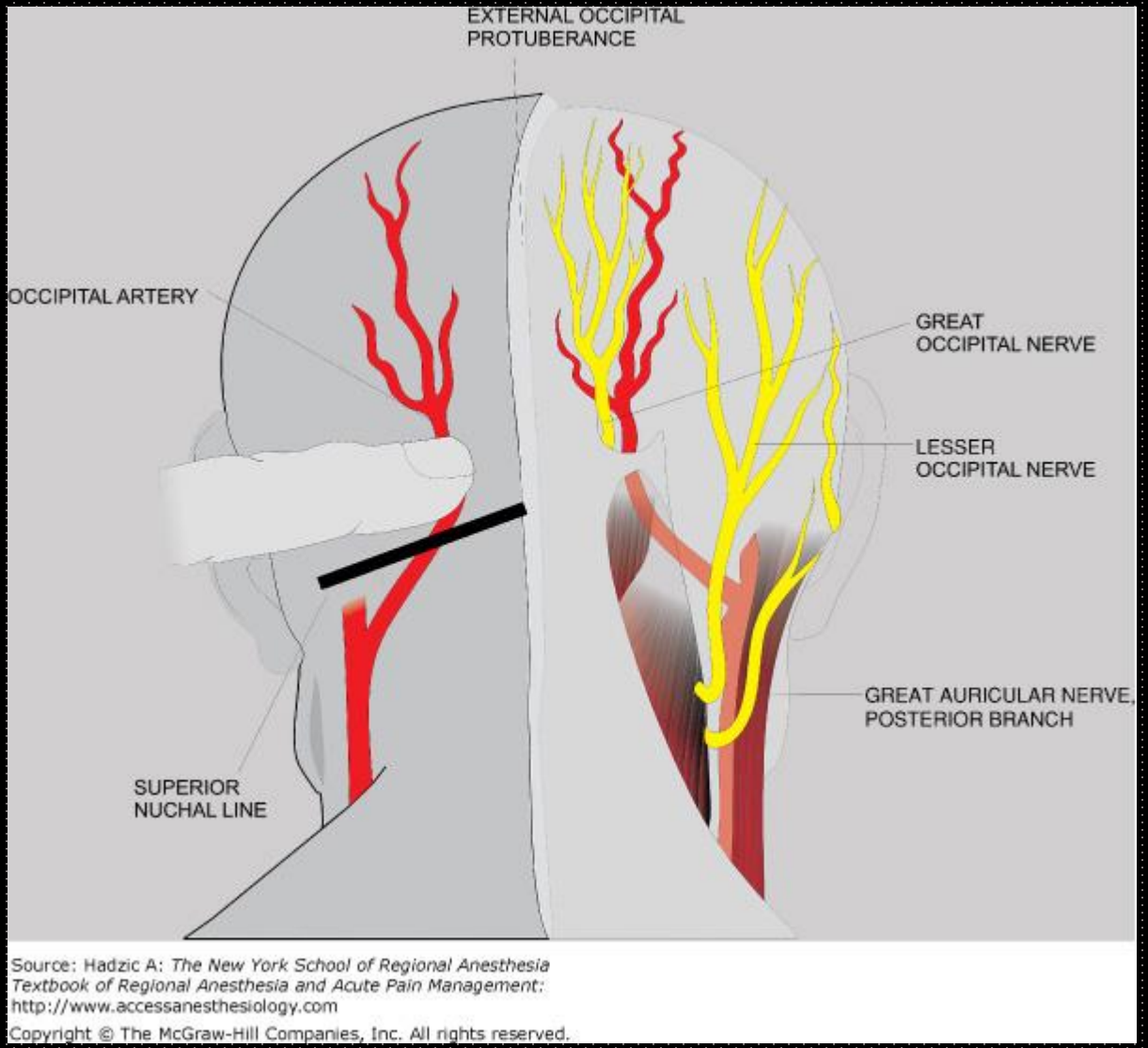
Video https://www.youtube.com/watch?v=T8







Greater Occipital Nerve Block
Studies

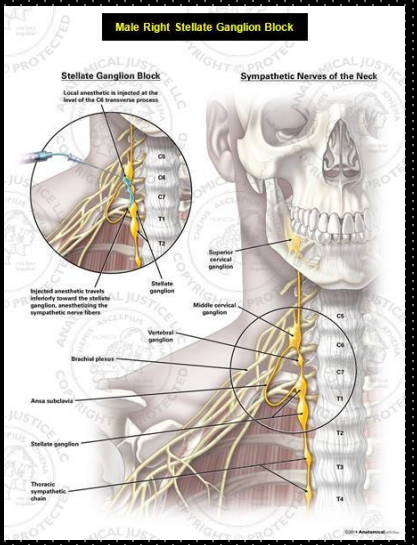
- No information is available regarding rates of successful greater occipital nerve blockade. The ability of the block to identify patients with disease is hampered by inexact definition of cervicogenic headache and no means of confirmation.
- Most studies, as well as the definition of the condition, come from a single group of authors. In one report, [200] patients clinically categorized as having migraine, cervicogenic, or tension-type headaches were tested with greater occipital and supraorbital nerve blocks, the latter as control subjects. Cervicogenic headache patients were most relieved by occipital injection.
- However, supraorbital block also produced relief (about half as much, and not selective for cervicogenic patients), and the two blocks relieved pain at the other poles of the head. Although this calls into question the basis of relief, a mechanism is offered in which sensory tracts converge on common upper cord and brain stem centers. [201] In another report, [192] the ability of greater occipital nerve block (confirmed successful by sensory examination) to provide relief was compared with selective blocks of cervical spinal nerves and the C2/3 facet in patients with symptoms of cervicogenic headache. The patterns of responses were thought to discriminate between various origins of pain, but analgesia, to some degree, followed most blocks.
- Conclusion. **The anatomy of the greater occipital nerve is well defined and the block easily confirmed, but the diagnostic meaning of a favorable response is clouded by the lack of pathophysiologic understanding of cervicogenic headache**.

Greater Occipital Nerve Block Limitations

- Selective blockade of the nerve at the proposed pathogenic site requires injection where it penetrates through the trapezius, but the site shows marked interindividual variability.
- Effective block is confirmed when anaesthesia develops in the distribution of the nerve.
- Cervicogenic headache is a poorly documented entity with no consistent histopathologic or radiologic findings.
- The typical lack of sensory deficit in the area of distribution of the greater occipital nerve does not support a neuropathic mechanism.
- Alternatively, it is possible that pain radiating in the distribution of the greater occipital nerve represents converging deep somatic input from the lateral atlanto-axial joint, which is innervated by the C2 anterior ramus, or from irritation of suboccipital muscles and periosteum, which has been shown to produce ascending headache.
- Because all the proposed pathophysiologies of cervicogenic headache are unproved, the meaning of blockade responses do not rest on a solid mechanistic base. Therefore, no defined process has been proved when relief follows greater occipital nerve block. Also, the therapeutic plan is not well defined after a favorable response to test injections. There are no data on the use of this block for treatment, and the surgical therapy for presumed greater occipital neuralgia has not been promising.
- Favorable responses to radiofrequency lesions of the greater occipital nerve have been claimed.
- Patients who had pain relief after bilateral greater occipital nerve block with 10–15 ml of local anesthetic on each side received heat lesions to the nerves during general anaesthesia. Although good-to-excellent relief was reported in 85% of cases, neural interruption was not documented, and there were no control subjects.

Selective Sympathetic Blockade
Diagnostic

- In these settings, **selective interruption of sympathetic neural traffic to the involved area may provide diagnostic insight and guide future therapy**. If blockade relieves pain, indicated therapies might include further local anesthetic blocks, systemic treatment with sympathetically active drugs (e.g., clonidine and prazosin), or destructive therapy with neurolytic injection or surgery. Failure of relief after sympathetic blockade would argue against the use of these treatments.
  - Horner's syndrome is easily observed but documents only blockade of sympathetic fibres to the head.
  - blockade of sympathetic activity to the extremities produces vasodilatation, vasoconstriction follows segmental block of sympathetic fibres to the trunk, possibly by blockade of sympathetic vasodilator fibres.
  - Skin temperature in pathologic conditions is controlled by a balance between sympathetic vasoconstriction from norepinephrine release and vasodilatation from release of vasoactive peptides from C nociceptors during antidromic activity.
  - Temperature change in the field of a blocked peripheral nerve will depend on the relative contribution of these opposing systems. From the available information, it is apparent that completeness of sympathetic block may depend on the monitored parameter chosen.
- The cervical trunk may be blocked independent of the stellate ganglion or fibres to the brachial plexus, so occurrence of ptosis, meiosis, facial anhydrosis, conjunctival hyperemia, or nasal stuffiness does not assure sympathetic block of fibres to the arm.
- Stellate, thoracic, or lumbar sympathetic injections that produce no measurable evidence of sympathetic blockade cannot reveal disease pathophysiology, regardless of the response of pain.



Stellate ganglion block Limitations

- Stellate ganglion injection may fail to produce sympathetic denervation for several reasons. Alternative routes allow sympathetic fibres to reach peripheral sites without transit through the stellate ganglion.
- These include passage in the nerves of Kuntz from the second and third intercostal nerves to the brachial plexus, distribution via the carotid, subclavian, and vertebral arteries, and by directly entering the peripheral nerves after synapses outside the sympathetic chain in intermediate ganglia located in spinal nerves.
- Sympathetic fibres can probably also bypass the sympathetic chain in the sinuvertebral nerve of Luschka.
- The principal reason for failure of injection to produce stellate ganglion blockade is lack of delivery of anaesthetic to the ganglion. Whereas the ganglion resides at the lower edge of the head of the first rib, solution injected at cervical levels passes anterior into the mediastinum. [A fundamental limitation of diagnostic sympathetic blockade is a lack of understanding about the role of the sympathetic nervous system in pain production.
- Evidence now indicates that excessive sympathetic activity is almost certainly not the explanation of pain.
- The enigmatic pathophysiology and ambiguous definitions of reflex sympathetic dystrophy and other painful conditions in which the sympathetic nervous system plays a putative role frustrate the interpretation and application of findings from blocks.

Limited evidence for efficacy of SGB in diagnosis for OFP

Sympathetic Stellate block

- The stellate ganglion is located on the transverse process of the C7 vertebra, just below the subclavian artery. It is composed of inferior cervical sympathetic ganglion and the first thoracic sympathetic ganglion. Therefore, the sympathetic nerves A receptors. that innervate the head, neck, and upper extremity pass through the stellate ganglion [10].
- Indicated
  - post herpetic neuralgia
  - postoperative pain
  - atypical facial pain
  - orofacial neuralgia [19,21-24].

Review Article
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Therapeutic potential of stellate ganglion block in orofacial pain: a mini review

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Orofacial pain is a common complaint of patients that causes distress and compromises the quality of life. It has many etiologies including trauma, interventional procedures, nerve injury, varicella-zoster (shingles), tumor, and vascular and idiopathic factors. It has been demonstrated that the sympathetic nervous system is usually involved in various orofacial pain disorders such as postherpetic neuralgia, complex regional pain syndromes, and atypical facial pain. The stellate sympathetic ganglion innervates the head, neck, and upper extremity. In this review article, the effect of stellate ganglion block and its mechanism of action in orofacial pain disorders are discussed.

Keywords: Head; Orofacial; Pain; Stellate ganglion block; Sympathetic Nervous System.

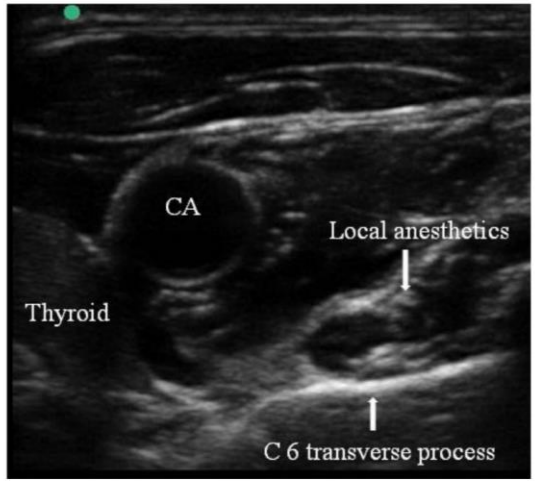


Fig. 1. Stellate ganglion block using ultrasound-guided technique. Local anesthetic was injected at the C6 transverse process. CA: carotid artery.

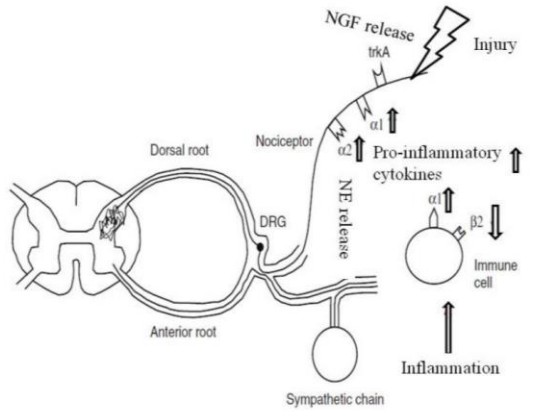
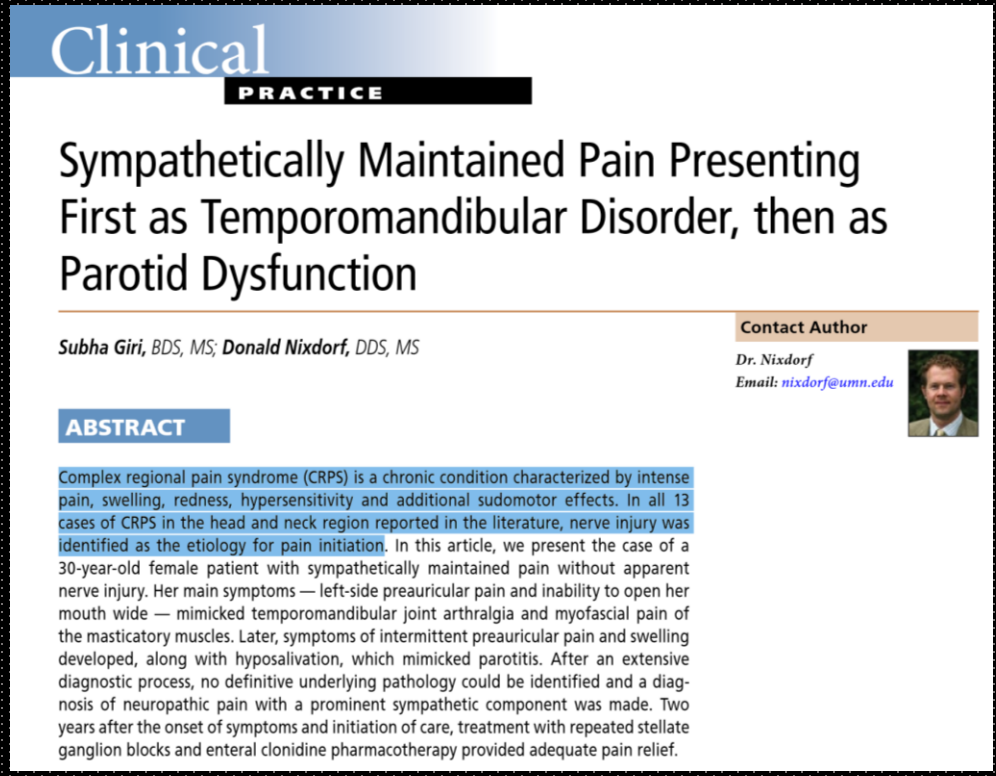


Fig. 2. The sympathetic nervous system (SNS) and pain. Inflammation activates immune dendritic cells. β-2 receptors are downregulated and α-1 receptors are up-regulated on these immune cells. Following nerve injury, functional adrenoreceptors are expressed on peripheral nociceptors. Activation of the SNS increases the level of norepinephrine (NE), which activates α-adrenoreceptors on the afferent fibers, and releases nerve growth factor (NGF). NGF sensitizes peripheral nociceptors through trk A receptors.

sympathetic ganglion. Therefore, the sympathetic nerves

Sympathetic maintained OFP pain

- Complex regional pain syndrome (CRPS) is a chronic condition characterized by intense pain, swelling, redness, hypersensitivity and additional sudomotor effects. In all 13 cases of CRPS in the head and neck region reported in the literature, nerve injury was identified as the aetiology for pain initiation



Clinical PRACTICE

Sympathetically Maintained Pain Presenting First as Temporomandibular Disorder, then as Parotid Dysfunction

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ABSTRACT

Complex regional pain syndrome (CRPS) is a chronic condition characterized by intense pain, swelling, redness, hypersensitivity and additional sudomotor effects. In all 13 cases of CRPS in the head and neck region reported in the literature, nerve injury was identified as the etiology for pain initiation. In this article, we present the case of a 30-year-old female patient with sympathetically maintained pain without apparent nerve injury. Her main symptoms — left-side preauricular pain and inability to open her mouth wide — mimicked temporomandibular joint arthralgia and myofascial pain of the masticatory muscles. Later, symptoms of intermittent preauricular pain and swelling developed, along with hyposalivation, which mimicked parotitis. After an extensive diagnostic process, no definitive underlying pathology could be identified and a diagnosis of neuropathic pain with a prominent sympathetic component was made. Two years after the onset of symptoms and initiation of care, treatment with repeated stellate ganglion blocks and enteral clonidine pharmacotherapy provided adequate pain relief.