

Temporomandibular Disorders (TMDs): A New Hypothesis

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Received: July 01, 2019; **Published:** August 08, 2019

Abstract

Till date there is no known cause of temporomandibular disorders, but it is thought to be related to muscles of mastication and the temporomandibular joint.

We describe in this case series for the first time a new injection gate for the auriculotemporal nerve, using a sodium channel blocker (lignocaine) without steroids that resulted in pain relief of the temporomandibular joint pain lasting more than five months in two patients.

This injection has been named Gate M by the authors (Figure 1).

This is different from auriculotemporal nerve block described by Pinosky, *et al.* in 1996 being done 1.5 Cm anterior to the ear at the level of the tragus for cranial surgeries [1].

The author hypothesises that temporomandibular disorders (TMDs) are not related to the temporomandibular joint or the muscles of mastication per se but rather neuralgia of the auriculotemporal nerve [2].

Also, the hypothesis is diffusion will occur through the uninterrupted myelin sheath covering the main nerve and its branches and by injecting one division of the main nerve the lipid based chemical will diffuse to the main nerve and its other branches as myelin sheath is characterized by a high proportion of lipid (70 to 85%) [3].

Keywords: Temporomandibular Disorders (TMDs); Lignocaine

Introduction

Temporomandibular disorders (TMDs) are thought to originate in both the joint and the muscles of mastication.

There is still though a lack of understanding about the aetiology, diagnosis, and mechanism of TMJ clicking and locking.

Psychological stress was recognized as a contributing etiologic factor [4].

Dentists routinely provide patients with initial medical management for their temporomandibular disorders (TMD). When patients are refractory to medical management, triaging them for further care is challenging due to the lack of evidence-based guidelines.

Four treatment strategies are exercised in the management of these disorders and they include medical management, non-surgical rehabilitation, arthroscopic surgery, and arthroplasty using outcome measures recommended by the International Association of Oral and Maxillofacial Surgeons (IAOMS).

Clinical assessments include intensity and frequency of TMJ pain, mandibular range of motion, TMJ sounds, and impairment of chewing [5].

The auriculotemporal nerve trunk has a close anatomic relationship with the condyle and the temporomandibular joint capsular region, and there is evidence of a possible mechanism for sensory disturbances in the temporomandibular joint region.

There is a hypothesis that the anatomic and clinical relationship of the auriculotemporal nerve to the condyle, articular fossa, and lateral pterygoid muscle may be causally related to compression or irritation of the nerve, producing numbness or pain, or both, in the temporomandibular joint region [6].

The auriculotemporal nerve arises as two roots from the posterior division of the mandibular nerve. The mandibular nerve is a branch of the trigeminal nerve (the fifth cranial nerve).

The auriculotemporal nerve comprises exclusively somatosensory fibers, which ascend to the superficial temporal region. There, it supplies the auricle, external acoustic meatus, outer side of the tympanic membrane and the skin in the temporal region (superficial temporal branches). It also carries a few articular branches that go on to supply the temporomandibular joint.

We describe in this case series for the first time a new injection gate for the auriculotemporal nerve, using a sodium channel blocker only without steroids, lignocaine that resulted in pain relief of the temporomandibular joint pain.

This injection has been named Gate M by the authors (Figure 1).

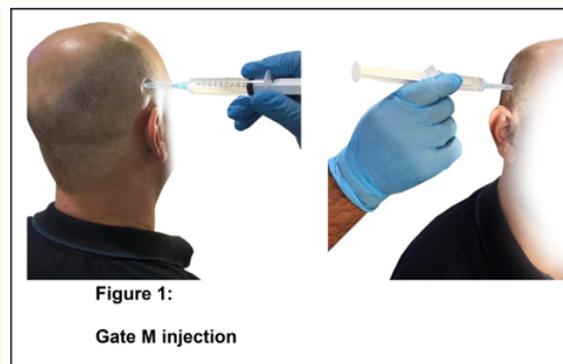


Figure 1: Gate M injection.

10 ml syringe, 26G needle, 10 ml Volume comprising 3 ml of 2% lignocaine plus 7 ml distilled water.

Patient is sitting up.

Temporal area of the skull, 2.5 cm above upper part of ear, the needle is introduced horizontally till the temporal bone is touched and then the volume is injected.

The injection is very smooth without any resistance as the volume is pushed in and there will be elevation of the soft tissues from the skull bone and the formation of a swelling which will subside within ten minutes post the injection.

Case 1 Presentation

A 25-year-old man with no past history of significance presented with pain in his right temporomandibular joint for the last two years. Different oral medications were used by him but failed to cure his pain.

His pain was constant and increased by eating and he had normal mandibular range of motion with no sounds but tender right temporomandibular joint.

He was injected in Gate M as described above with lignocaine only which made the pain vanish completely and he continues to be pain free six months post the injection.

Patient reported parasthesia in his right forehead post the injection which ended in one hour time after the injection.

Case 2 Presentation

A 44-year-old man, a known case of gout disease with no other co morbidities, presented with pain and oedema at the first metatarsophalangeal joints bilaterally with unilateral ankle joint pain and oedema for more than two years with right temporomandibular joint pain for six months.

Blood tests were done on him and were all normal except blood uric acid which was high measuring 8 mg/dl.

He was given Febuxostat and NSAIDS for a month on which he improved in his feet but the right temporomandibular joint continued to be painful with a click.

Mouth opening was painful with some crepitus on full mouth opening.

Patient was injected in Gate M on the right side and within ten minutes his pain and clicking disappeared and he continues to be symptoms free for the last five months.

Discussion

The three major branches of the trigeminal nerve-the ophthalmic nerve (V_1), the maxillary nerve (V_2) and the mandibular nerve (V_3)-converge on the trigeminal ganglion (also called the semilunar ganglion or gasserian ganglion), located within Meckel's cave and containing the cell bodies of incoming sensory-nerve fibers. The trigeminal ganglion is analogous to the dorsal root ganglia of the spinal cord, which contain the cell bodies of incoming sensory fibers from the rest of the body.

It seems that the role of the trigeminal ganglion mimics to certain degree that of the dorsal root ganglia of the spinal cord and the latter has been the focus of many treatment techniques like ablation or modulation of the DRG via continuous thermal radiofrequency, pulsed radiofrequency, electrical DRG neurostimulator technologies, modification of DRG cellular function using viral vectors and gene silencing, dorsal root ganglionectomy and most recently the advent of the sodium channel blockers gates discovered by the author [7].

Also, the role of sodium channels in nerve function and pain needs to be emphasized.

After nerve injury hyper excitability and spontaneous firing develop at the site of injury and also in the dorsal root ganglion cell bodies. This hyper excitability results at least partly from accumulation of sodium channels at the site of injury [8].

Pain is related to excited nerves and disturbance in Sodium Channels.

Simply by giving lignocaine which is a sodium channel blocker you are resetting the electricity of nerves like resetting a jammed laptop.

Gate M injection is a safe and an easy injection that can be done to treat temporomandibular disorders (TMDs).

The local anaesthetic molecule consists of three components: (a) lipophilic aromatic ring, (b) intermediate ester or amide chain, and (c) terminal amine. The aromatic ring improves lipid solubility. The nerve membrane consists of a double lipid layer and a protein layer and therefore the property of enhancing lipid solubility contributes to increased potency of the anaesthetic agent as more of the available drug can diffuse through the membrane [9].

Accordingly, Lignocaine will diffuse through the myelin sheath covering the nerve and in case of blocking the auriculotemporal nerve the diffusion will carry on to the mandibular nerve and then all trigeminal nerve divisions blocking the sodium channels which are accumulated at the site of injury.

The hypothesis is diffusion will occur through the uninterrupted myelin sheath covering the main nerve and its branches and by injecting one division of the main nerve the lipid based chemical will diffuse to the main nerve and its other branches as myelin sheath is characterized by a high proportion of lipid (70 to 85%) [3].

Trigeminal nerve gives off the mandibular nerve which gives off the auriculotemporal nerve.

There will be no motor and sensory deficits in all divisions of the trigeminal nerve (the ophthalmic, the maxillary and the mandibular nerves), however, the cascade of sodium channel blocking effect will be in all nerves sharing the same continuous myelin sheath.

So, injecting any division of a nerve will work to produce analgesia in the parts of the body supplied by that main nerve, and the duration of analgesia will vary from one nerve to another and future studies need to be conducted to scrutinize what factors are affecting the length of analgesia like the size of the peripheral nerve being injected and the injected chemical and any other factors involved in that.

Conclusion

This case series proves that Temporomandibular disorders (TMDs) are not related to the muscles of mastication or the temporomandibular joint itself but is just a neuropathy of the auriculotemporal and masseteric branches of mandibular branch of the trigeminal nerve.

The authors concur that more large studies need to be done to reach a solid evidence of this new hypothesis and treatment.

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Volume 10 Issue 9 September 2019

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