To MVD or not to MVD?
Trigeminal Neuralgia
– A cervicogenic model.

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Authors Note

The classification of head and facial pain is constantly evolving and by its nature incomplete, with various bodies from institutions, to the IASP to the IHS playing their part.

Whilst a classification system is useful in facilitating, communication between doctor/doctor/patient, diagnosis, treatment and treatment outcomes, it can also be detrimental. Many patients’ symptoms don't fit a particular box yet this is where they are placed, boxed in, and consequently on a treatment protocol that fits that box.

For example, in a rigid box system of classification, the migraine patient who goes on to develop Trigeminal like pain finds themselves in two boxes with two different treatment protocols and as such the possibility that the two have a single cause is often ignored to the detriment of the patient and future research into the patient’s condition. I use migraine as a possible example but the same could be said of various head and face pain conditions; TMJ, occipital neuralgia, atypical odontalgia, Type2 TN, GN, GPN to name but a few. Head and facial pain disorders as such are not immune to junk diagnoses due to presenting and often overlapping symptoms.

Put simply Trigeminal Neuralgia means pain in the distribution of the nerve with no more meaning than the term sciatica, it is not a diagnosis but a description of symptoms which is why the pathogenesis (be it microvascular compression or other) of TN remains a hypothesis, and is open to debate. A diagnosis of Trigeminal Neuralgia may as well mean Tough Nut to crack.

For the purpose of this article therefore TN can be read as “TN like” pain.
Introduction

Cervicogenic Trigeminal Neuralgia (CTN) is poorly understood, often ignored and therefore often misdiagnosed leading to the inevitable, incorrect treatment. If a doctor does not consider CTN in the differential diagnosis of Trigeminal Neuralgia, then the patient cannot make an informed decision as to best treatment. Despite the literature and the available evidence, CTN is greatly understudied. Ignoring CTN does nothing to encourage appropriate clinical studies and denies the patient evidence based therapeutic intervention. The role of the health care provider is comprehensive and should consist of the least invasive treatment modality that rewards, both practitioner and patient with the best outcome.

Bonica’s Management of Pain(2012), p955, by Scott M. Fishman states:

"The various pathological findings reported and complex theories advanced in the TN literature attempt to explain the combination of unique clinical feature of TN such as:

- Stereotyped paroxysms of lancinating pain which occur in a limited part of the trigeminal territory.
- Separation of the trigger area from the painful region.
- Non-noxious triggers.
- Absence of sensory or motor deficit.
- Characteristic response of TN to antiepileptic medications.

New diagnostic techniques are now challenging theories that were previously advanced to resolve the many questions which remain regarding the etiology and pathophysiology of TN" 

A cervicogenic model for TN offers an explanation of the above points, in addition to a few others:

- Periods of remission.
- Increasing incidence with age.
- Bilateral TN.
- The absence of neurological findings on examination.
- Many folk have microvascular compressions but no TN
- Why common surgical procedures fail.
- Anaesthesia Dolorosa.
- Why the ophthalmic branch V1 is least effected.
- Other cranial neuralgias occur in the same TN patient.
- Why pain and temperature are the main sensory disturbances of TN and other sensory/ motor deficit is largely absent.
Trigeminal Neuralgia

Trigeminal neuralgia (TN) is a rare, but extremely painful, disorder of the fifth cranial (Trigeminal) nerve, and is characterised by pain in any of its three nerve branches that supply the face. Typically the pain is unilateral and affects one branch, but it may affect one or more branches and also both sides of the face (Bilateral TN).

"The pain resulting from TN imposes a substantial burden on patients. During severe attacks, affected patients may be unable to speak or eat. Even between attacks, some patients are gripped by an overwhelming fear that the pain could suddenly return at any time [Cheshire, 2003]. This poses serious impairment in daily function and reduces quality of life. Pain severity correlated with reduced measures of daily functioning, quality of life, well-being, sleep, mood and overall health status [Tolle et al. 2006]. TN impacted employment in 34% of patients. Two-thirds of patients reported moderate to severe pain within the previous 24 h. Depressive symptoms are frequent in patients suffering from TN [Zakrzewska, 2006; Zakrzewska et al. 1999; Marbach and Lund, 1981]."[23]

There are several classifications of face pain arising from the Trigeminal nerve; type 1 TN, type 2 TN, secondary symptomatic TN, post herpatic neuralgia, trigeminal neuropathic pain and trigeminal deafferentation.

The focus of this literature/anatomical review, is type 1 and type 2 TN.

Burchiel et al, make the following distinction between the two types:

Type 1 TN, idiopathic sharp, shooting, electrical shock-like, episodic pain.

Type 2 TN, idiopathic aching, throbbing, burning, >50% constant pain[i].

"The aetiology of TN remains unclear and several theories have been proposed. Many medical and surgical methods have been applied with only partial effectiveness and several side effects. New hypotheses and therapeutic methods are urgently needed."[22]
Pathophysiology of Trigeminal Neuralgia

Over the years there have been several theories on the pathophysiology of idiopathic trigeminal neuralgia, the favored cause being peripheral with a pathogenesis within the central nervous system. The hypotheses put forward are as follows:

- Microvascular compression in the prepontine cistern of the nerve root by a vein or artery. First hypothesised by Dandy in 1934 then Gardner 1963 and then Jannetta who made microvascular the mainstay of surgical treatment.

- Ignition hypothesis. Nerve injury at the trigeminal root or ganglion leads to nerve hyper excitability followed by sudden painful attacks\(^{(44)}\).

- Trigeminal convergence-projection theory. Based on the fact that nociceptive input from virtually the entire head and neck (cranial nerves V, VII, IX and X and cervical nerves 1-4) converge on the trigeminal spinal nucleus\(^{(42)}\), hence pain signals from multiple regions can be projected onto a single neuron for processing in the brain leading to patterns of referred pain. Convergence theory is based on the work of W.A. Sturge and J. Ross from 1888 and later TC Ruch in 1961, and others.

- Bioresonance hypothesis, states that when the vibration frequency of a structure surrounding the trigeminal nerve becomes close to its natural frequency, the resonance of the trigeminal nerve occurs. The bioresonance can damage trigeminal nerve fibers and lead to the abnormal transmission of the impulse, which may finally result in facial pain\(^{(43)}\).

- Allergy hypothesis. Allergens triggering an inflammatory and consequently an immune response, causing temporary nerve damage and consequently neurological changes\(^{(45)}\).

- Petrous ridge and fibrous band compression as a cause of TN. Malis, demonstrating success in a series of 43 patients using surgical decompression of these bands\(^{(46)}\).

- Compression of the individual trigeminal branches as they pass to the periphery through their respective foramen.
The anatomy of the trigeminal nerve, Cranial Nerve V. and its connections

A basic understanding of the anatomy is important for patients, in that it leads to a better understanding of their complaint and consequently, they can use this knowledge towards making informed decisions as to the best treatment options.

The trigeminal nerve (the fifth cranial nerve, or simply CN V) is the nerve responsible for sensation in the face and motor functions such as biting and chewing. The largest of the cranial nerves, its name ("trigeminal" = tri-, or three and -geminus, or twin; thrice-twinned) derives from the fact that each trigeminal nerve (one on each side of the pons) has three major branches: the ophthalmic nerve (V1), the maxillary nerve (V2), and the mandibular nerve (V3). The ophthalmic and maxillary nerves are purely sensory, and the mandibular nerve has sensory (or "cutaneous") and motor functions. The motor function is primarily responsible for movement of the jaw. Each side of the head/face has its own trigeminal nerve.

Sensory impulses from these divisions include pain, temperature, touch modalities, conscious and subconscious proprioception (the ability to sense stimuli arising within the body regarding position, motion, and equilibrium).

Individual branches and their sensory areas are as follows:

- **V1 function**: Sensory impulses from skin of nose, cornea, forehead, and scalp.
- **V2 function**: Sensory impulses from upper teeth and gums, nasal mucosa, palate, upper lip, and skin of cheek.
- **V3 function**: Sensory impulses from temporal region, tongue, lower teeth and gums, anterior two thirds of the tongue, skin of chin, lower lip, upper jawline and external ear (along with cranial nerves VII, IX and X). Motor impulses to muscles of mastication and muscle that tenses the eardrum. Proprioception from muscles of mastication, teeth, and the temporomandibular joint (TMJ).

Somatosensory pathways are involved in processing these impulses. Each pathway consists of a chain of neurones, starting from the receptor organ and ending at the cerebral cortex. Different sensations have their own somatosensory pathway utilising different nuclei within the central nervous system:

- Proprioception: Sensory nerve impulses to the cortex travel via the Mesencephalic nucleus.
- Discriminative touch, fine touch and vibration: Sensory nerve impulses to the cortex travel via the chief (principal pontine) trigeminal nucleus
- Pain and temperature and crude touch: Sensory nerve impulses to the cortex travel via the Spinal trigeminal nucleus.
Motor nerve impulses are from the motor nucleus. The location of these nuclei and sensory pathways are shown below:

![Trigeminal complex and its nuclei](#)

Figure 1 Trigeminal complex and its nuclei

The Spinal Trigeminal Nucleus extends as far as the second cervical vertebrae, some report as far as the fourth cervical vertebrae \(^{[14]}\).

The location of these nuclei is of clinical importance when considering the aetiology and pathophysiology of Trigeminal Neuralgia.
Trigeminal Neuralgia, The Cervicogenic model

With an understanding of the anatomy of the Trigeminal Complex, the hypothesis of Cervicogenic (caused by the neck) Trigeminal Neuralgia, gains momentum. A review of the literature supports this model.

"Neck stiffness due to irritation of the trigeminal nerve endings innervating the cranial meninges is a well-known clinical sign of meningismus. This sign suggests that there exists a connection between the trigeminal nerve and the posterior neck muscles. The trigeminal system is involved in a number of reflexes such as the blink, jaw, head retraction, defense and orienting reactions, which require connections between the face and the neck motoneurones."

Ivan Milanov et al (1).

Several studies have demonstrated the cervical-trigeminal reflexes in humans proving the connection between the trigeminal nerve and the neck. Serrao M et al in their study of cervical-trigeminal reflexes in humans demonstrated an interaction between nociceptive trigeminal afferents and both upper and lower cervical spinal cord motoneurones (2). C. Ertekin et al studied the head retraction reflex and specifically demonstrated activation of the posterior neck muscles after stimulation of the superior and inferior orbital nerves (V1 branch of the trigeminal nerve) (3). Serrao M et al, showed painful stimulation of any one of the three trigeminal branches produced a reflex contraction of the contralateral trapezius muscle as well as head rotation and neck bending away from the stimulated side (4).

The mechanism by which nerve impulses from the neck give rise to pain in the head is convergence (the exciting of a single sensory neuron by incoming impulses from multiple other neurons). This theory states that there is a convergence of trigeminal and cervical afferents in the spinal trigeminal nucleus/tract which allows referral of pain from neck origins to the head. Only structures innervated by C1, C2 and C3 cervical nerve roots have been shown to be capable of causing headache. These are the muscles, joints and ligaments of the upper three cervical segments, but also include the dura mater of the spinal cord and posterior cranial fossa and the vertebral artery (5). This mechanism was described as early as 1961 when Kerr found that C1-2 stimulation could refer pain to the eye, forehead, vertex and rarely the back of the head (9).

Initially this mechanism was demonstrated in animal models but more recently in humans.

Browne PA et al in their review of the empiric and basic science evidence concluded the animal experimental data support the findings of human empiric and experimental studies, which suggest that strong connectivity exists between trigeminal and cervical motor and sensory responses (8). Piovesan EJ et al demonstrated evidence of convergence in humans by injecting sterile water over the greater occipital nerve (a branch of the second cervical nerve, C2) on one side of the neck causing trigeminally
distributed pain \(^6\). Busch V et al also demonstrated anatomical and functional convergence of trigeminal and cervical afferent pathways using greater occipital nerve blocks \(^7\).

The cervical spine, its influence on the trigeminal complex and convergence is recognised by the medical profession as to the pathophysiology of numerous headache complaints from migraines to cervicogenic headache but is largely ignored in the case of Trigeminal Neuralgia.

**Review of the literature supporting a cervicogenic model.**

Individuals suffering with neck pain and soft tissue injury to the cervical spine have been shown to have impaired Trigeminal sensitivity.

Knibestöl M et al, found impairment of vibration and temperature sensibility of the face in patients with previous soft tissue injury of the cervical spine \(^1\). In another study patients with chronic mechanical neck pain had bilateral pressure-pain hypersensitivity in the trigeminal region \(^2\).

Pathologies of the cervical spine have been shown to cause trigeminal neuralgia, and trigeminal hypersensitivity. In one case a vertebral artery loop was found to be compressing the spinal cord at the C1 C2 level, corrective surgery resolved the trigeminal pain \(^3\). Barakos JA et al, report a case Trigeminal sensory neuropathy caused by cervical disk herniation \(^4\). A neurinoma at the C1 C2 level was found to be the cause of a case of trigeminal neuralgia. The authors, as long ago as 1979 wrote:

"*It is emphasized that since trigeminal fibres descend as far as the upper part of the C2 segment, trigeminal neuralgia should not be considered as an exclusively supraspinal symptom.*" \(^5\).

In the above, abnormal afferent impulses converge at the spinal trigeminal nucleus causing trigeminal sensory impairment and neuralgia. If these abnormal afferents due to pathology/mechanical impairment of the upper cervical structures cause trigeminal pain, it would seem reasonable to surmise that abnormal afferent nerve impulses can arise from the other musculoskeletal elements of the upper neck, from the muscles, ligaments, facet joints etcetera. Sada Teresa Ovalle et al, describe four cases of trigeminal neuralgia due to pathology of the cervical spine, including cervical arthritis and cervical stenosis, that were successfully treated with upper cervical spine nerve blocks, concluding that cervical pathology should be considered as a differential diagnosis in trigeminal neuralgia \(^6\). Interestingly, Sada Teresa Ovalle et al state that pain in the cervical spine was not present initially in the TN patients but evolved as a symptom, this would suggest that lack of neck pain on initial presentation does not rule out TN symptoms from cervical origin.
This being the case, modalities of physical therapy, should relieve the symptoms of trigeminal neuralgia in these cases as shown in the literature. Rodine RJ et al, discuss a case in which chiropractic treatment using soft tissue, manipulation and mobilisation therapies relieved the pain in a patient who had suffered with trigeminal neuralgia for over 7 years (16). Lay EM, describes the osteopathic management of trigeminal neuralgia, using osteopathic techniques directed at the cervical spine (18).

An additional paper discusses twelve cases of cervical spondylosis and trigeminal neuralgia, with relief of symptoms with cervical manipulation (19). Manipulation of the upper cervical spine has shown to result in an increased pressure pain threshold in the trigeminal sensory field, in women with mechanical neck pain (20). Grgić V, presents the case of a 43 year old female who found a complete cessation of symptoms of trigeminal neuralgia with manual therapy of the cervical spine (21).
Microvascular Decompression (MVD)

The lack of certainty regarding the aetiology and pathogenesis of Trigeminal Neuralgia and the fact most clinicians rarely come across the condition (incidence <15/100,000) means treatment is a challenge. The etiological basis of trigeminal neuralgia (TN) is unknown but vascular (arterial and venous) compression of the trigeminal nerve roots has been put forward as the likely cause in most cases (25).

Dandy, in 1934 wrote:
“Whatever the cause of trigeminal neuralgia might be, it must be located in the sensory root” (50).

This however was before the spinaltrigeminal nucleus was termed and since has been implicated in cervicogenic TN.

First line of treatment is pharmaceuticals, and then for patients for whom these don't work invasive surgical procedures are considered. The ‘gold’ standard procedure is microvascular decompression (Microvascular decompression may be considered over other surgical techniques to provide the longest duration of pain freedom (24), where an artery or more rarely a vein is seen to be compressing the trigeminal nerve root and then is surgically removed away from the nerve, protective padding sometimes being placed between the two. This procedure is considered to have a high success rate, defined as no pain and off medication, although the procedure is not without risks, may be fatal and failure not uncommon.

The average mortality rate ranges from 0.2% to 0.5%, and up to 4% of patients suffer from:

1. Major problems such as:
   a. Cerebrospinal fluid (CSF) leakage,
   b. Infarcts or haematomas.
2. Long term complications are [Cruccu et al. 2008; Gronseth et al. 2008] (23):
   a. Aseptic meningitis (11%),
   b. Sensory loss (7%),
   c. Hearing loss (10%).

Microvascular decompression achieves the most sustained pain relief with 90% of patients reporting initial pain relief and over 80% still pain free after 1 year, with 75% after 3 years and 73% after 5 years remaining pain free (23).

Over time patients with an initially successful MVD manifest a relentless rate of Trigeminal neuralgia recurrence (26).
Comment and discussion

Magnetic resonance imaging (MRI)

Generally used to exclude pathology, MRI imaging sometimes shows MVC but the absence of it still does not exclude the possibility of MVC being present, hence the MVD procedure is often used for diagnostic purposes before intervention.

Sgarbi Nicolás et al, in their paper 'Anatomy of the Trigeminal Nerve. Key Anatomical Facts for MRI Examination of Trigeminal Neuralgia', a total of six medical professionals, recognise the neck as a source of facial pain stating:

“Finally, the inferior nucleus occupies the tegmentum of the medulla, extending caudally to the first segments of the cervical spine, and is in charge of thermal and pain information. Its location explains the possible appearance of symptoms in the facial territory in patients with a degenerative/inflammatory disorder of the upper cervical spine.”

They don't go on to suggest analysis of the MRI in relation to the upper cervical spine but place emphasis on finding vascular compressive elements. This is most unfortunate, because of the territory of the trigeminal complex, the upper cervical spine should have already been imaged on the MRI scan, so for a little extra consideration, degenerative/inflammatory disorders of the upper neck might be identified.

Additional articles on Trigeminal Neuralgia and evaluation with MRI, make no reference to the consideration of MRI of the cervical spine or any differential diagnosis associated with it. It is simply not in the Radiologist's teaching.
Microvascular controversy

Vascular compression, arterial or veinous is widely accepted as the likely cause of classical trigeminal neuralgia. This however is debatable. Trigeminal neuralgia occurs in people who have no sign of vascular compression \[^{26}\].

Adamczyk M et al. conclude:

"Contact between a trigeminal nerve root and an artery in the prepontine cistern is a frequently seen anatomical variant. Therefore, detection of such a variant is not equivalent to finding the cause of a patient's complaints." \[^{28}\]

Thomas KL et al 2014, state:

"The reviewed data suggest that the theoretical support for a vascular compressive etiology of TN is weak, albeit the surgical outcome data are relatively convincing." \[^{25}\]

Lee a et al 2014, state:

"The hypothesis that TN is caused by neurovascular conflict must be challenged."

They also note that 99.94% of individuals with trigeminal neurovascular compression (NVC) do not have TN \[^{26}\]. The fact that NVC, is seen in a large number of individuals without TN, could or should NVC be considered normal anatomy?

There are patients suffering TN, who have no vascular compression either on MRI or found during the microvascular decompression procedure. Ko AL et al (2015), conclude:

"Neurovascular compression (NVC) is neither sufficient nor necessary for the development of TN. Patients with TN without NVC may represent a distinct population of younger, predominantly female patients. Further research into the pathophysiology underlying this debilitating disease is needed." \[^{60}\]

Is the trigeminal nerve root not under constant microvascular compression/ irritation by its own arterial supply, the trigeminal arteries?

The hypothesis of microvascular compression also cannot explain the effectiveness of other therapeutic interventions/ surgeries that alleviate the symptoms of TN.

Although rare, patients may present with trigeminal neuralgia on both sides of their face, this is termed bilateral trigeminal neuralgia, this would suggest a cause within the central nervous system as opposed to NVC. This would support the cervicogenic model.
Why is TN in the case of Microvascular compression confined to the Sensory Root and only affects pain touch and temperature sensory modalities?

As previously written the trigeminal nerve has a sensory and a motor root. The sensory root transmits all sensation; gross touch, discriminative touch, proprioception and pain and temperature. The main sensory impairments in patients with trigeminal neuralgia whether as a symptom or as a trigger, are pain and temperature (according to the IHIS classification, pain is the only sensory modality disturbance). These sensory modalities are 'processed' in the lower most part of the spinal trigeminal nucleus (Fig1 p8), extending down and into the cervical spine to the levels of the second/ third cervical segments. Sensory modalities of proprioception and discriminative touch are not a symptom of trigeminal neuralgia, and these sensory modalities are processed in different nuclei as depicted, away from the cervical spine. This suggests a central cause i.e. within the central nervous system for trigeminal neuralgia, instead of or in addition to the peripheral cause MVC. If MVC of the sensory root at the periphery was solely responsible we would see proprioceptive disruption, patients smashing teeth together or routinely biting their tongue.

If MVC is an aetiological factor in TN solely or in addition to another cause, its action would seem to be specific to the sensory root, we see no evidence of MVC acting on the motor root which lies closely to the sensory root and running parallel to it. TN patients generally don't express symptoms of motor origin, difficulty in opening and closing, or deviation of the jaw when opening and closing. The motor processing nucleus also sits outside of the cervical territories of the spinal trigeminal nucleus.

A search of PubMed reveals no research paper with regard to MVD of the motor root, even though the motor root is said to have a small number of sensory fibres.

With MVC and TN the most common vessel responsible is a tortuous/elongated superior cerebellar artery (SCA) that is said to compress and distort the trigeminal nerve. Indra Yousry et al, found that the SCA contacted a significant number of sensory roots in addition to reporting a significant contact with the motor root (48%+ superior motor root, 20%+ inferior motor root). Given that the average width of the motor root is much smaller than the sensory root, would it not be more vulnerable to SCA contact than the sensory root? With a vascular compressive force would we not expect to see an equal and opposite reaction and hence changes to the vascular vessel as well as the nerve, and a resulting vascular dysfunction as a result? Is it a groove/distortion or growth of the nerve due to collagen over production in the extra cellular matrix, not uncommon in nerves?
MVD doesn’t account for the variation in trigeminal branches affected

In addition, the sensory root carries afferent impulses from the three trigeminal branches along fibres which are arranged within and along the sensory root in distinct areas. Sensory fibres of the ophthalmic region are uppermost and medially situated in the nerve root, those from the mandibular branch lowermost and laterally, whilst the maxillary fibres are situated in the middle of the root.\(^{(33,52)}\)

K Gudmundsson et al\(^{(48)}\) note at the pontine level, the first division fibers are usually dorsomedial and the third division fibers caudolateral within the main sensory root. However, the third division fibers may vary from being almost directly lateral to directly caudal to the first division fibers. This would suggest that TN due to MVC should least likely affect the mandibular division. Given the anatomical relationship of the SCA to the trigeminal sensory root, the ophthalmic division would be expected to be the most affected due to MVC, yet the opposite is true. The same would apply to MVC by the superior petrosal vein.

Mila Ćetković et al (2011), stated most common microvascular contact of the trigeminal root as Petrosal vein 24%, SCA 20% followed by the AICA at 12%\(^{(37)}\). Others have stated ‘Considerable rotation occurs as the root travels towards the root entry zone where the ophthalmic fibres lie caudally (lowermost) and the mandibular fibres rostrally (uppermost)\(^{(47,53, p28)}\), though seemingly contradicted in the same writing\(^{(53, p33)}\). Given that Lovely and Jannetta emphasise that neuro vascular compression occurs throughout the length of the root, rotation does not account for the low incidence of ophthalmic symptoms. Isolated first division, ophthalmic trigeminal neuralgia occurs in less than 5% of cases.
In the case of MVC why don't we see signs of peripheral nerve compression?

The site of action of MVC is on the peripheral portion of the nerve, outside of the central nervous system, yet we don't see clinical signs of peripheral nerve compression. The diagnosis of idiopathic TN pain, type 1 and type 2 states that there is no abnormal neurological findings on examination, including the absence of sensory loss to light touch or pin prick by normal sensory testing \(^{(49)}\), yet other peripheral nerve compressive syndrome shows neurological deficit on clinical examination.

In peripheral trigeminal sensory root irritation/compression, especially in long suffering individuals, one would expect to see symptoms of sensory deficit in the total area of its dermatome(s) (an area of the skin supplied by nerves from a single nerve root), Figure 2 shows this is not the case.

Why is minor compression (the vascular component maybe less than 1mm in width) of the peripheral trigeminal sensory root so painful compared with other larger compressions of other peripheral nerves? Peripheral nerve compression usually occurs at sites where there is a constriction of movement away from any compression, e.g. in carpal tunnel syndrome, the median nerve is constricted in its movement by the carpus, with accompanying clinical signs of peripheral nerve compression.
In contrast watching videos of microvascular compression of the trigeminal nerve there seems to be enough space to drive a bus through, certainly enough space to surgically place a pad to separate the vascular entity and the sensory root. This in its self raises questions. If the space is large enough to place a pad in, when the sensory root is flexible, as is the vascular component, surely it has enough room to move away from vascular compression, before any adhesions are formed between the two. In a confined space, necessary for compression, adding extra substrate in the form of padding would increase the mechanical pressure on the trigeminal nerve, if not on the side of the padding then on the opposite side, and hence if it was the cause of symptoms would potentially make the symptoms worse.

It has been stated:

"It appears that classical trigeminal neuralgia may be an exception to the rule that nerve injuries typically produce symptoms like constant pain and allodynia"[38].

Rather than it being an exception to the rule, a simpler explanation would be that in TN the symptoms of nerve compression are not present because microvascular compression is not the issue. Why are we not seeing microvascular compression of the extracranial peripheral nerves?

The cervicogenic explanation for lack of peripheral neuropathy symptoms

With the cervicogenic model for TN we do not expect to see signs of peripheral nerve compression because the mechanism is of convergence/fasciculation between dorsal root fibres of the upper three cervical segments and second order neurones of the trigeminal branches at the spinal trigeminal nucleus (STN). Clinically we see what we would expect, no complete dermatomal sensory loss, no sensory or motor deficit, and symptoms related to pain, touch and temperature which as previously stated are processed in the STN specifically in the nucleus caudalis, the portion receiving dorsal root fibres from the upper three cervical segments. The topography of the three trigeminal branches in the nucleus caudalis is as follows:

a) Arrangement of the three trigeminal branches in the nucleus are sequential, with the ophthalmic branch ending at the front and the mandibular branch at the back of the nucleus [54], thus we would expect dorsal root fibres to converge with mandibular neurones most. This in addition to the fact that mandibular neurones are the most numerous within the nucleus accounts for the rarity of neuralgia of the ophthalmic division.

b) The central or oral area of the face projects to the upper end of the nucleus and the outer areas of the face project to the lower end (See Fig2 p17). During percutaneous trigeminal tractotomies at high cervical levels, responses from all trigeminal nerve branches and from the seventh, ninth and tenth cranial nerves are easily elicited [53]. Cervical convergence has therefore the potential to affect any part of the trigeminal territory.
The Valsalva manoeuvre questioning arterial compression as a cause of TN

Another symptom or clinical sign that would be expected in the case of TN due to arterial compression, would be an increase in pain during the valsalva manoeuvre (holding your breath as you bear down/blowing out with a closed mouth/coughing or sneezing), as in some other vascular cranial disorders. Valsalva, has been known to increase arterial pressure resulting in rupture of cranial arteries, yet there is nothing in the literature in relation to valsalva and TN which would support the theory of arterial microvascular compression.

MVC is actually a hypothesis and MVD a diagnostic procedure

Although MVD surgical intervention has shown positive outcomes in the cessation of trigeminal symptoms, there are no randomized double blind placebo control studies. DR Ken Casey a respected advocate of MVD, humorously likens this to the fact that there is also no such studies in the case of the parachute. This is all very well, the MVD procedure is shown to alleviate the symptoms of TN, but is it the procedure or the MVD that is the cause of relief? In the case of a cervicogenic model for TN the MVD would also be expected to provide relief. If abnormal afferent neuronal signals from the cervical spine from say muscles or ligaments, were causing the neuralgia, would an induced coma via general anaesthetic for 2-5 hours not reset this abhorrent mechanism by relaxation of these tissues? General anaesthesia is used to relax the muscles of the body, in addition inhibiting normal body reflexes to make surgery safe and easier to perform. There is no reason against a trial in patients with TN who opt for surgical MVD, to undergo the procedure but without the MVD, after all surgeons are in many cases using the MVD procedure, as a diagnostic procedure. Baechli H et al, describe five cases of trigeminal neuralgia in which there was no sign of microvascular compression, yet all five were cured using the microvascular decompression procedure.
Added uncertainty as the value of MVD in the treatment of TN

At around the time Janetta was conducting MVD, Malis in 1967 was reporting favorable outcomes in a group of 44 patients, by surgical decompression of the trigeminal nerve at the petrous ridge due to fibrous bands \(^{46}\). This quickly fell out of favor with the rising popularity of the MVD procedure. This raises questions as given the rarity of TN in the general population and the reasonable patient sample number in the Malis surgical group, coupled with the quoted 90% success rate for MVD it would seem at least some of Malis's group would have been candidates for MVD, yet both procedures offered similar results. The odds of Malis falling on the 10% of patients that MVD doesn't give satisfactory results to, would have been huge. This would suggest neither MVC or fibrous bands are responsible for TN, but a common factor in both procedures as relieving the symptom, such as surgical trauma to the nerve, general anaesthesia or another factor.

Adams CB discussing the hypothesis of MVC and its pathophysiology concludes it to be insufficient and unconvincing and puts forward the theory that results are due to surgical trauma of the trigeminal root producing dampening of the brainstem activity \(^{51}\). This theory, however doesn't address the cause of the original excitation of the trigeminal complex.

Why are TN patients more likely to experience other cranial nerve neuralgias

Patients with trigeminal neuralgia often have or go on to experience other head and facial neuralgias; occipital (pain in the back of the head and neck), geniculate (pain and temperature of the ear- cranial nerve VII), and glossopharyngeal (pain and temperature of the ear, upper pharynx, posterior one third of the tongue and auditory tube- cranial nerve IX). Glossopharyngeal neuralgia is often treated surgically by MVD \(^{29}\). These neuralgias are also uncommon, and yet occur more frequently in TN patients than the general population. Is this further evidence that the spinal trigeminal nucleus is responsible in the aetiology of trigeminal neuralgia? The cervicogenic model of trigeminal neuralgia explains the case of multiple neuralgias, in that the cranial nerves responsible share neuronal connections with the spinal trigeminal tract and the spinal trigeminal nucleus (Fig1 p8). Abnormal cervical afferent nerve impulses converge in the spinal nucleus with these cranial nerves giving rise to these additional neuralgias.
TN patients are often diagnosed with comormid TMJ dysfunction, is this an over diagnosis?

TN patients often perceive pain in the temporal-mandibular (jaw) joint and are often diagnosed with having coexisting temporal-mandibular joint dysfunction. Sensory pain afferents from the jaw joint are also processed in the spinal trigeminal nucleus, and are therefore susceptible to convergence.

**Pharmaceutical interaction of TN and the cervicogenic model**

Interestingly, Carbamazepine the drug of first choice for the treatment of symptoms of trigeminal neuralgia, has been shown in animal studies to have its mechanism of action in the spinal trigeminal nucleus (30), and more specifically in the spinal trigeminal subnucleus caudalis (31), the portion that extends to the upper cervical segments, where convergence occurs. Therefore symptoms of cervicogenic TN would also be relieved with carbamazepine, being a central aberration.

**TN and Dental Procedures**

A large number of patients develop TN as a result of dental procedures. Whilst dental procedures can directly damage branches of the trigeminal nerve a likely scenario is cervical trauma due to sustained/excessive contraction of the cervical muscles, backward bending of the neck and strain of its associated structures. The functional anatomical relationship of the jaw (TMJ), bite and the cervical spine is widely documented and referenced. Dental procedures do cause cervical dysfunction including cervical disc infection (34), including radiculomyelopathy due to minor trauma in the presence of cervical spondylosis and stenosis (35).

"The following risk factors are associated with the potential of bony or ligamentous compromise of the upper cervical spine.....recent head/neck/dental surgery" (36).

Dentistry can cause cervical dysfunction/ pathology. Cervical dysfunction can cause Trigeminal Neuralgia.
The cervicogenic case for Anaesthesia Dolorosa

**Anaesthesia Dolorosa**, the IHS classification states:

"Persistent and painful anaesthesia or hypaesthesia in the distribution of the trigeminal nerve or one of its divisions or of the occipital nerves.

**Diagnostic criteria:**

Persistent pain and dysaesthesia within the area of distribution of one or more divisions of trigeminal nerve or one of its divisions or of the occipital nerves.

Diminished sensation to pin-prick and sometimes other sensory loss over the affected area

There is a lesion of the relevant nerve or its central connections

**Comment:**

Anaesthesia dolorosa is often related to surgical trauma of the occipital nerves or trigeminal ganglion, evoked most frequently after rhizotomy or thermocoagulation has been performed for treatment of 13.1.1 Classical trigeminal neuralgia.\(^{(40)}\)

As far as the literature is concerned Anaesthesia Dolorosa is a condition of the trigeminal and occipital nerves more often than not due to surgical procedures. It is not reported to occur following procedures for pathology of other peripheral nerves, phantom limb pain yes, but an entirely different complaint. The literature available suggests there is no cure. Many neurologists and pain clinics are using the same anticonvulsant drugs that treat TN including carbamazepine with some relief in symptoms\(^{(41)}\).

Should Anaesthesia Dolorosa, be such a surprise as a side effect of destructive procedures, most commonly rhizotomy? No not at all, and it probably provides some of the best indicators for the cervicogenic model for TN. During surgical procedures at the wrong end of the trigeminal complex you aren’t addressing the abnormal nociceptive afferent impulses which converge on the cervical-trigeminal nucleus and tract which are consequently processed in the spinal trigeminal nucleus leading to pain and temperature sensations in the face. Destructive procedures of the trigeminal complex at the periphery hence cause numbness but still pain/burning due to continuous convergence at the spinal trigeminal nucleus.

Hence treatment of Anaesthesia Dolorosa could possibly be directed at the cervical spine, though this won’t unfortunately address the facial numbness due to the destructive procedure. A better option would be to include cervicogenic trigeminal neuralgia in the initial differential diagnosis and hopefully examine the likely possibility that the neck is a common source of TN thus avoiding MVD and destructive procedures, and consequently the possibility of Anaesthesia Dolorosa, in addition to successful resolution of symptoms.
Conclusion

There is enough evidence for a cervicogenic model in the aetiology and pathogenesis of trigeminal neuralgia. This model would work via:

a) Cervical somatic afferents at the spinal trigeminal nucleus converging with trigeminal pain sensory pathways resulting in referred pain from the neck to the head.

b) Convergence of cervical somatic afferents at the spinal trigeminal nucleus causing hypersensitivity within the trigeminal complex, making it sensitive to normally non-noxious stimuli, including that of microvascular compression at the sensory root.

c) Convergence may excite, inhibit, desensitise or centralise the trigeminal complex.

A cervicogenic model explains; bilateral symptoms, remission of symptoms, coexisting head/facial neuralgias, sudden onset of symptoms, symptoms occurring due to whiplash and dental procedures, neck pain and trigeminal neuralgia, postural aggravating and relieving factors, trigeminal neuralgia in the absence of microvascular compression, the progressive nature of symptoms, and the action of pharmaceutical intervention of choice, carbamazapine. In contrast MVC falls short or fails in its explanations.

How many patients with TN have a cervicogenic cause? Without the necessary research we can only speculate. Neurosurgery states approximately a quarter of patients with trigeminal neuralgia had experienced trauma in the preceding six months and that a third of patients have a relieving/aggravating factor in rotating their neck one way or the other (56), and

“In the early years, the pain is frequently precipitated by changes in position and maybe eased by returning to the former position” (57).

This starts to suggest that there may be numerous patients with Trigeminal Neuralgia of a cervical cause.

The cervicogenic model and hypothesis accounts for the major positive and negative signs and symptoms of trigeminal neuralgia, for its pathogenesis, and for the efficacy of pharmaceutical treatment modalities. The medical profession needs to concentrate more resources into research, assuming money to be no object.

Any patient consulting a medical professional who denies Cervicogenic Trigeminal Neuralgia, should follow in Mr Bolt’s slipstream.
References

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