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Vasomotor Rhinitis (VMR) and Cerebral Spinal Fluid Flow: A Chiropractic issue

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Purpose: To provide a qualitative report using peer-reviewed published articles, reports, and contemporary textbooks Allopathic, Osteopathic, and Chiropractic to demonstrate the neurological aetiology of VMR. Documenting its systemic repercussions especially as it concerns cerebral spinal fluid flow and the benefit that Chiropractic care offers.

Methods: A Narrative Analysis was undertaken of the literature expanding outwards from accepted definitions of vasomotor rhinitis to identify peer reviewed published studies. The studies concern linking VMR to its neurological aetiology and structural consequences. We will also link that aetiology to other health conditions demonstrating comorbidities and further documenting the autonomic nervous system (ANS) affect in various bodily functions. There will be discussion of the fascial and neurological patterns, the disruptions of those patterns and their ramifications in postural, spinal distortions, and cerebral spinal fluid (CSF) flow. Considering the neurochemicals and neurotransmitters, we will delve into the functions and dysfunctions many times where we find the factors that connect certain pathologies. This enables us to change our perspective from merely looking at a myriad of separate diseases, to seeing that so many have the common underlying causes and possible common resolutions.

Conclusions: VMR is more than nasal congestion. The mechanisms that provoke and maintain this "stuffed nose" collaterally produce problems that are far more detrimental to one's health. The appearance of VMR should be considered a prodromal symptom and should alert us to examine further to observe what other health problems are simultaneously occurring. Chiropractic appears to offer an effective avenue for evaluation and treatment via ANS dysfunction or dysautonomia. Chiropractic presents a treatment with no secondary effects for VMR and the concomitant related cranial dysfunction. Further study is recommended.

]**Indexing terms:** Chiropractic; Autonomic Nervous System; vasomotor Rhinitis; Sacral Occipital Technique; SOT; glymphatic system; neurogenic inflammation; migraine; dysautonomia.

Introduction

Vasomotor rhinitis (VMR) is a nonallergic, non-infection condition which even the allopathic literature states is triggered by autonomic nervous system dysfunction. This occurs when nerves in the nasal mucosa overreact to environmental stimuli like temperature changes, smells, ambient airborne hydrocarbons or esters, stress, or other environmental elements.

... Chiropractic offers a targeted specific resolution to many organic system malfunctions and with the minimal collateral symptomatology, side effects, or complications...'

It is characterised by an ANS imbalance typically hyperactive parasympathetic system, leading to increased blood flow (vasodilation) and excessive, clear mucus secretion, chronic nasal congestion, runny nose (rhinorrhea), and postnasal drip. This has been managed with nasal sprays and trigger avoidance. (1, 2, 3) The most common prescription is Ipratropium Bromide (Atrovent®) a prescription anticholinergic medication. However, introducing an anticholinergic, or any medication, into the nasal mucosa is a double-edged sword.



The nasal mucosa is a formidable vehicle for access to the blood stream, i.e. Narcan®. So, an anticholinergic can cause systemic reactions as well such as; swelling of the eyes, face, lips, tongue, throat, hands, feet, ankles, or lower legs, hoarseness, difficulty breathing or swallowing. Other warnings concerning this drug's literature include prostate and bladder neck blockage. Label warnings state that patients with enlarged prostate, Narrow angle Glaucoma should 'Use with caution'. The product literature also states that the drug may make these conditions worse: Kidney disease, Liver disease, stating 'Use with caution'. However, might there be a safer avenue for therapeutic action, one with minimal adverse effects?

Background

VMR is the most common form of nonallergic rhinitis, comprising approximately 71% of all nonallergic rhinitis conditions. It is estimated that 14 million Americans suffer from vasomotor rhinitis, with a worldwide prevalence approaching 320 million. The most recent report being the report of the 2008 AAAAI Joint Task Force on Practice Parameters, The diagnosis and management of rhinitis: an updated practice parameter, which defines VMR (idiopathic rhinitis) (4) and states '*that patients with vasomotor rhinitis (VMR) often suffer from concomitant gastric distress, particularly irritable bowel syndrome (IBS) or gastroesophageal reflux disease (GERD)*'. Studies show that over 70% of VMR patients may have IBS, and 67% of IBS patients experience VMR, suggesting a strong etiological link between these problems and autonomic nervous system imbalances. (5, 6) This connection is suspected to be through the same ANS imbalance which affects the nasal mucosa.

Otorhinolaryngologic (ENT)ramifications of VMR have been found to be sinus headache, facial pressure, postnasal drip, coughing, and throat clearing are common symptoms shared among both allergic and nonallergic rhinitis. However, Vasomotor rhinitis (nonallergic rhinitis) can cause non-ENT symptoms like 'brain fog' or cognitive difficulties, including confusion, trouble concentrating, and fatigue. (7) This in addition to the GI symptoms already mentioned.

Patients with vasomotor rhinitis tend to be categorised into two groups depending on predominant symptomatology: 'blockers' with congestion and 'runners' with rhinorrhea. Patients with rhinorrhea tend to have an enhanced cholinergic (parasympathetic) response. Those with nasal obstruction tend to have nociceptive neurons with a heightened response to normally harmless stimuli. (8) Nociceptive neurons with a heightened response to harmless stimuli (a phenomenon known as allodynia) are primarily associated with the sympathetic nervous system. (9)

Neuroceptors

The receptors primarily associated with the sympathetic nervous system in these cases are Transient Receptor Potential Vanilloid 1(TRPV1) It is essential to look at the biochemical changes that occur as that is what will illuminate the etiological commonality in health conditions. These TRPV1 receptors are often acting as an upstream controller of sympathetic activity. They act as nociceptors (pain sensors) on sensory neurons, and their activation can trigger sympathetic responses, including increased heart rate, blood pressure, and thermogenesis (heat production). (10, 11, 12)

TRPV1 receptors are non-selective cation channels that function as molecular sensors for noxious stimuli, including temperatures above 43°C (109°F), acidic conditions, and chemical irritants like capsaicin in chilli peppers, that is what makes the nose water as you eat them. They are primarily found on peripheral nociceptive neurons and play a crucial role in pain sensation and modulation. Those patients with TRPV1 over-expression are those patients who often have an increased density of TRPV1 (capsaicin receptors) on their nasal sensory nerves. Patients with Nerve Growth Factors (NGF) issues demonstrate Inflammatory mediators that are known to up-regulate TRPV1 synthesis in sensory neurons and increase the transport of these receptors to nerve endings. People with altered Nerve Growth Factor (NGF) levels often experience conditions related to neurodegeneration, chronic pain, or mental health disorders, as NGF is critical for the survival, growth, and maintenance of neurons. It acts as a signalling protein, and its dysregulation has been connected to conditions like Alzheimer's disease, schizophrenia, and neuropathic pain. Environmental exposure complications are observed where there is frequent exposure to pollutants, cigarette smoke, strong odours, or ozone. This in turn can lead to increased TRPV1 expression, contributing to chronic airway hyper-responsiveness. (13)

There is also a Reduced Activation Threshold (RAT) in these patients. When normally innocuous stimuli, such as a drop in temperature or a strong perfume, which under normal circumstances do not activate normal neurons, will activate these hypersensitive ‘pain-sensing’ neurons in the presence of RAT. What is seen also is something called Antidromic Neuropeptide Release. A process where action potentials travel backward along sensory nerves from the axon branches toward the peripheral terminal, rather than toward the spinal cord. This triggers the local release of neuropeptides. This mechanism drives neurogenic inflammation, causing vasodilation, plasma leakage, and immune cell activation. (14, 15)

They also release neuropeptides like Substance P (SP) and Calcitonin Gene-Related Peptide (CGRP) directly into the nasal tissue. Calcitonin Gene-Related Peptide, is a 37-amino acid neuropeptide that acts as a potent vasodilator and neurotransmitter, playing a key role in pain signalling and neurogenic inflammation. It is widely distributed in both the central and peripheral nervous systems, with elevated levels heavily linked to the activation of the trigeminovascular system in migraine headaches. (16) This demonstrates a clinically established link between VMR and migraine that needs to be explored in more detail. Vasomotor reactivity, the capacity of cerebral blood vessels to adapt to changes in pressure or CO2 levels, is frequently altered during migraine attacks. Migraines cause complex hemodynamic dysregulation and share a well-documented connection with both vestibular symptoms and certain types of vasomotor rhinitis. (17, 18) Another problem well documented is Neurogenic Oedema. When these neuropeptides

cause rapid vasodilation (blood vessel widening) and increased vascular permeability it has found to lead to the 'boggy'" swollen nasal lining typical of VMR. (19, 20, 21, 22, 23, 24)

Pathophysiology

The pathophysiology of nonallergic rhinitis (VMR) is complex, with still much to be discovered. As we search for aetiology it is observed that it appears to be firmly based on the imbalance between parasympathetic and sympathetic stimuli on the nasal mucosa. (25, 26) It is continually observed that mucous secretion is regulated primarily by the parasympathetic nervous system, whereas the sympathetic nervous system controls vascular tone. (27, 28)

There have been studies investigating transient receptor potential (TRP) cation channels that emerged as important players in type 2 upper airway inflammatory disorders; those being chronic inflammatory conditions driven by a specific immune response (involving interleukins 4, 5, and 13) that often coexist and affect the nose, sinuses, and throat. Key disorders include chronic rhino-sinusitis with nasal polyps (CRSwNP), allergic rhinitis, and eosinophilic laryngitis. These frequently present with congestion, facial pain, loss of smell, and runny nose. (29, 30) Clinically, Maspero's study found that 100% of patients with rhinitis demonstrate this ANS dysfunction pattern, as well as 55% to 70% of severe asthmatics. Multiple clinical studies demonstrate these same observations in ANS dysfunction. (31, 32, 33)

ANS

In the Parasympathetic nervous system (PNS) Acetylcholine (ACh) is a primary stimulant of mucus production and secretion, particularly in the airways, acting through parasympathetic nerves and non-neuronal sources. It binds to muscarinic receptors mainly M3 on submucosal glands and surface epithelial cells, inducing rapid secretion and increasing mucus gland activity. (34) Acetylcholine acts through muscarinic receptor signalling, an increase in intracellular calcium Ca2, and causes the fusion of secretory granules with the cell membrane. This leads to rapid mucus exocytosis. The excessive mucus secretion in the airway can at times produce symptoms as severe as chronic obstructive pulmonary disease (COPD) and asthma when left unchecked. (35)

In addition to muscarinic receptors, Alpha 7 nicotinic acetylcholine receptors (nAChRs) are critical in mediating airway mucus production, potentially playing a role in mucus metaplasia (an increase in mucus-producing cells). In chronicity one finds that beyond nerve release, inflammatory cells and airway epithelial cells can produce acetylcholine on their own, providing an autocrine/paracrine mechanism for mucus production during inflammation. This local reaction feeds into the chronic loop of nasal congestion. (36, 37)

The sympathetic nervous system (SNS) in general promotes decrease in overall nasal mucus secretion primarily by causing vasoconstriction of nasal blood vessels, which reduces tissue swelling and congestion. While it can stimulate mucin output from submucosal glands via adrenergic receptors in some instances, its dominant effect is that of reducing congestion and drying the mucosa. (38, 39, 40) Sympathetic effects on mucus production can be seen as

vasoconstriction (Decongestion). Sympathetic nerves release norepinephrine, which constricts blood vessels (especially in the nose), reducing interstitial oedema and decreasing the volume of the nasal mucosa. In contrast to the parasympathetic nervous system, which causes heavy, runny mucus production, sympathetic stimulation results in less but often thicker mucus, allowing for better airflow.

The Alpha-adrenoceptor agonists (which mimic sympathetic nervous system activation) can decrease the frequency of the mucociliary wave. (41, 42, 43, 44) The mucociliary wave, or wave-like motion of cilia, is the coordinated beating movement of microscopic hair-like structures (cilia) throughout the respiratory tract. These cilia act as a 'metachronal wave', a sweeping motion that acts to move mucus, pathogens, and inhaled particles upward to the throat to be cleared out. In essence, the mucociliary wave is the primary innate defence mechanism on the lung. (45) The repercussions of disrupting the mucociliary wave mechanism is that it is not only the respiratory system that utilises this wave motion. The ciliary wave mechanism is also utilised in the spinal canal for the movement of CSF. (46, 47) The sympathetic nervous system acts as a natural decongestant both mechanical and ANS.

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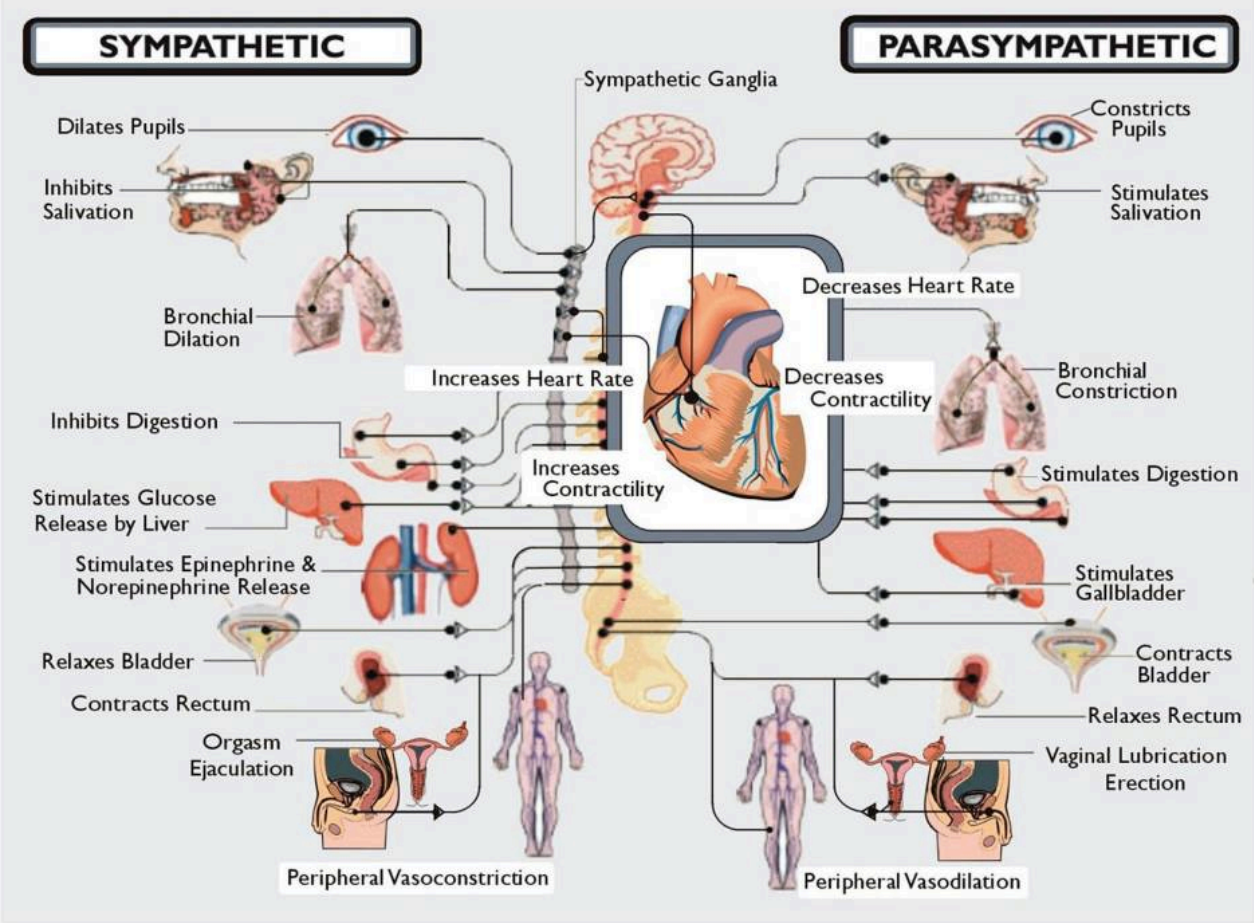
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Figure 1: ANS. Source and copyright 2019 Dysautonomia International (DI)



The latest studies using a cardiovascular testing laboratory convincingly demonstrated that asthmatics with gastro-oesophageal reflux demonstrate evidence of autonomic dysfunction and that these patients tended to have VMR. This testing methodology has also been used to study various disorders such as small fibre neuropathy, orthostatic imbalance or syncope, and degenerative dysautonomia such as Shy-Drager Syndrome. (63) We can now understand that ANS dysfunction exists as a spectrum of conditions. This spectrum might have simple pain and VMR on the lower end with such diseases as Shy-Drager syndrome (SDS) at the extreme high end. In this extreme case we observed an ANS imbalance causing severe organ and system collapse. This brings new appreciation for ANS–function connection. (64, 65, 66)

Structure

Considering components involved with neurological dysfunction it is interesting what else has been documented in Allopathic literature. It has been found that Vasomotor rhinitis (VMR) (nonallergic rhinitis) can occur following a cervical spine injury causing dysfunction of the autonomic nervous system that controls the nasal mucosa. (67, 68) Harlor stated that his study found 'a traumatic injury to the neck can damage the sympathetic nerves, leading to an imbalance where the parasympathetic system becomes overactive, with depression of the sympathetic system resulting in symptoms like chronic, watery, or mucoid nasal discharge'. (69 ,70) Petersen's study found that 'the great majority of patients who suffer from chronic sinus problems have lost the normal curve in their neck and have spondylotic changes in the cervical spine'. Petersen documented that 83.9% of chronic rhinitis patients in the study reported neck pain prior to onset of sinus congestion and pain and presented loss of cervical lordosis.

> 'Neck pain and cervical musculoskeletal dysfunction are common among persons with sinus congestion and may be a comorbid feature or contributing factor to headaches attributed to rhino-sinusitis'. (71)

While plain sinus X-rays or computer tomography of the sinus show little to no pathology in cases of vasomotor rhinitis, cervical spine X-rays in fact do. 'Vasomotor rhinitis and cervical curve degeneration (such as the loss of cervical lordosis) are linked to mechanical stress or instability in the upper neck that irritate the cervical sympathetic chain and/or vagus nerve, disrupting autonomic control and triggering nasal congestion or rhinorrhea'. (72)The cervical spine injuries can interrupt the sympathetic nerve pathways, leading to parasympathetic overactivity in the nasal cavity. (73)

Dicpinigaitis et al reported in their study concerning cervical spinal injury that bronchial hyperresponsiveness observed in the test population most likely reflects the loss of sympathetic airway innervation and resultant unopposed cholinergic bronchoconstrictor tone. 'Blockade of methacholine hyperresponsiveness with ipratropium bromide® suggests a muscarinic receptor-mediated phenomenon'. (74) In a clinical report there was documentation of a patient developing persistent left-sided rhinorrhea for up to 1.5 years after a motor vehicle accident that fractured the left carotid canal, injuring the sympathetic plexus. (75)

Misalignments (subluxations) in the upper cervical spine can cause irritation to the sympathetic nervous system, which in turn causes the sinus membranes to produce excessive mucus. This condition manifests as chronic, watery nose running (rhinorrhea) and nasal congestion that often gets worse with temperature changes, stress, or even bending over. (76, 77) It appears that structurally when the neck curvature is lost it creates a posture where tensile stress on the spinal cord influences the brainstem where the 7th Cranial nerve nuclei is located. In response to the loss in curve of the neck, also the suboccipital muscles located at the back of the head become spastic interfering with sympathetic flow. (78)

Neurology

Discussing the underlying neurology, in the case of the sinus membranes, these tissues are parasympathetically controlled by the 7th Cranial nerve, better known as the facial nerve. The sympathetic innervation originates from the upper thoracic spinal cord (T1-T4), ascends the cervical sympathetic chain, and synapses in the superior cervical ganglion. Post-ganglionic fibres then form the deep petrosal nerve, which passes through the Vidian nerve (nerve of the pterygoid canal) to the pterygopalatine ganglion to regulate blood flow and reduce nasal congestion. (79)

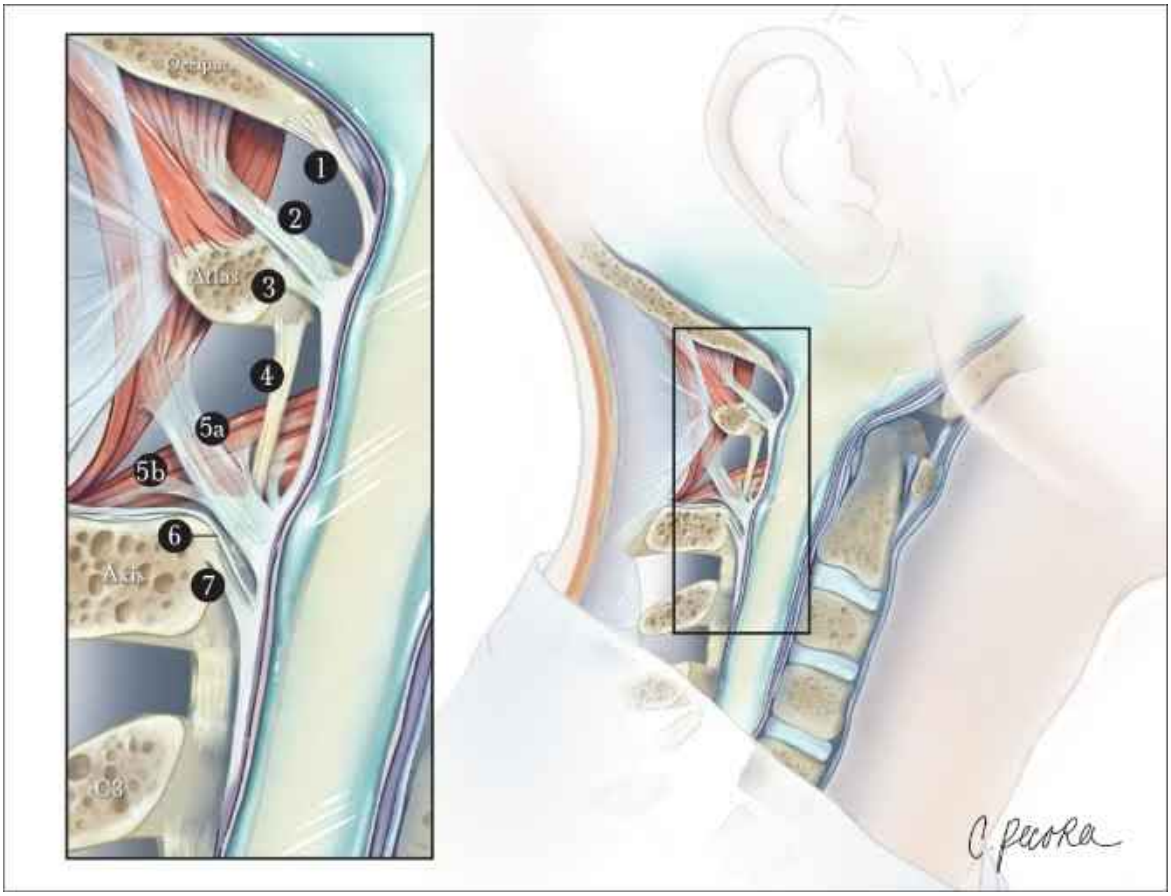
Observing the ascending fibres, they travel up the cervical sympathetic trunk to the superior cervical ganglion (located at C2-C3 level). They then synapse in the superior cervical ganglion which acts as the source for sympathetic nerves to the head. There the post-ganglionic fibres form a plexus around the internal carotid artery (Carotid Plexus). Those fibres then leave the plexus as the deep petrosal nerve. The deep petrosal nerve joins the greater petrosal nerve (parasympathetic) to form the Vidian nerve (mixed nerve) (nerve of the Pterygoid canal).

The trigeminal network and the pterygopalatine ganglion (located adjacent to the pterygoid canal) share pathways. As a result, hypertension in the pterygoid muscles can trigger referred pain or autonomic symptoms (such as sinus pressure, nasal congestion, or eye tearing) that mimic Vidian nerve irritation. This can be caused by temporomandibular imbalance. (80, 81, 82) These fibres then pass through the canal where the nerve can be compromised before it reaches the pterygopalatine ganglion. There it continues without synapsing and distributes throughout the nasal cavity to supply blood vessels. The superior alveolar (anterior, middle, and posterior) nerves, branches of the maxillary nerve provide sensory innervation. This system works in opposition to the parasympathetic system, which causes vasodilation and increased secretion. (83)

As we stated, there may be local stimulation in the chronic stage. Sensory neuropeptides and nociceptive type C fibres of the trigeminal nerve contribute to mast cell degranulation and stimulate the itching/sneezing reflexes. (84) Mast cell degranulation causes itching and sneezing due to rapid immune process where mast cells release inflammatory mediators, most notably histamine which is stored in cytoplasmic granules. Triggered by allergens (via IgE antibodies), infections, or physical stimuli, this process causes immediate allergic reactions, inflammation, and symptoms like itching, flushing, and vascular changes. (85) There is also neurogenic dysfunction when the condition involves a malfunction of nerve signals between the brain and nasal tissue, which is sometimes linked to broader autonomic disorders. (86)

There is another mechanism which merits mentioning as it causes brainstem and spinal cord irritation, the myodural bridge. (87) (Figure 2) The myodural bridge (MDB) functions as a mechanical pump that facilitates the circulation and drainage of cerebrospinal fluid (CSF), which directly impacts the clearance of metabolic waste through the cribriform plate. (88) By connecting the suboccipital muscles to the spinal dura mater, the MDB transmits head movements into pressure changes that drive fluid toward major exit routes like the nasal lymphatics. (89)

Figure 2: ANS. Myodural bridge



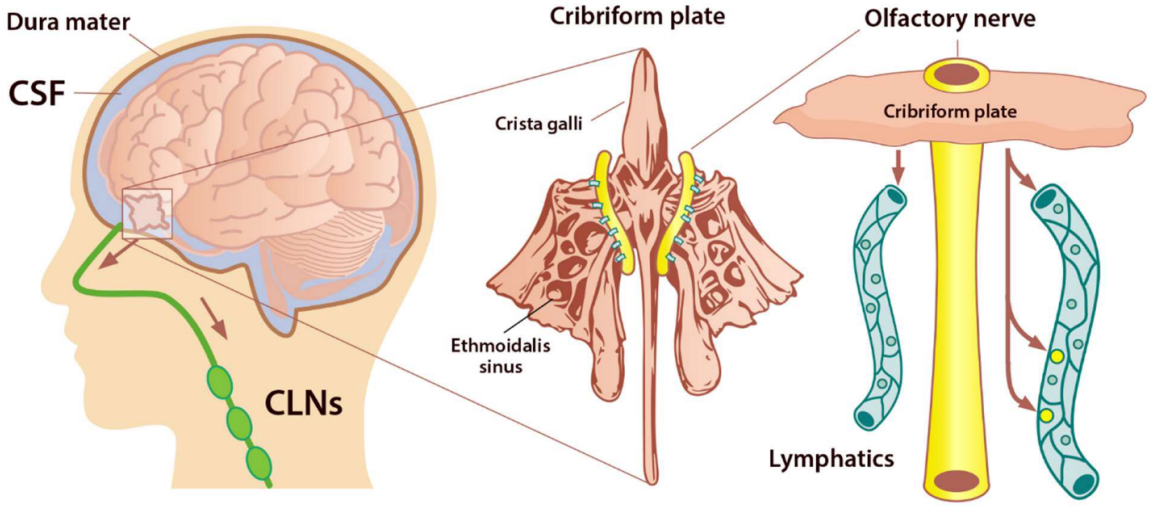
The myodural bridge. Sagittal section of the myodural bridge and its connections to the dura and spine. The posterior atlantooccipital membrane (1) extends from the occiput and coalesces with the dura mater at the cerebrospinal junction. The superior myodural bridge (2) merges with the superior vertebrodural ligament (3) of the atlas and fuses with the PAOM at the level of the atlantooccipital interspace. The inferior myodural bridge comprised of the rectus capitis posterior major fascia (5a) and obliquus capitis inferior fascia (5b) courses between the atlantoaxial ligamentum flavum (4) as bundles of dense fibers. The inferior myodural bridge fuses with the PAOM. The nuchal bridge (6) merges with the inferior vertebrodural bridge (7) and attaches to the PAOM. The PAOM terminates at the level of C3 after this transition point (*). The dura mater (DM) continues as an independent structure after that. O, occiput; C1, atlas; C2, axis; C3, third cervical vertebra; NL, nuchal ligament; PAOM, posterior atlantooccipital membrane. Adapted from Scali et al. .**Source:** Scali F, Ohno A, Enix D, Hassan S. The Posterior Atlantooccipital Membrane: The Anchor for the Myodural Bridge and Meningovertebral Structures. Cureus. 2022;14(5):e25484. Published May 30, 2022. DOI 10.7759/cureus.25484

Glymphatic system and CSF

There is an intimate connection between the nasal lymphatics, congestion via vasomotor rhinitis, foggy brain and dull mental function that so many VMR patients complain of. This brain fog or mental confusion has always been attributed to lack of adequate oxygen to the brain due to respiratory issues. However, a new study utilising neuro-imaging biomarkers such as the diffusion tensor imaging along the perivascular space (DTI-ALPS) index and choroid plexus volume (CPV) has described glymphatic congestion as the aetiology. (90) This connection is now explained via the Glymphatic System which we will see is intertwined with nasal health. (91) The glymphatic system clears metabolic waste (such as amyloid beta) from the brain, moving them into the cervical lymph nodes and to peripheral drainage sites. A major route for this CSF outflow

is alongside olfactory nerves as they pass through the cribriform plate into the nasal cavity. (Figure 3)

Figure 3: CSF flow and the cribriform plate



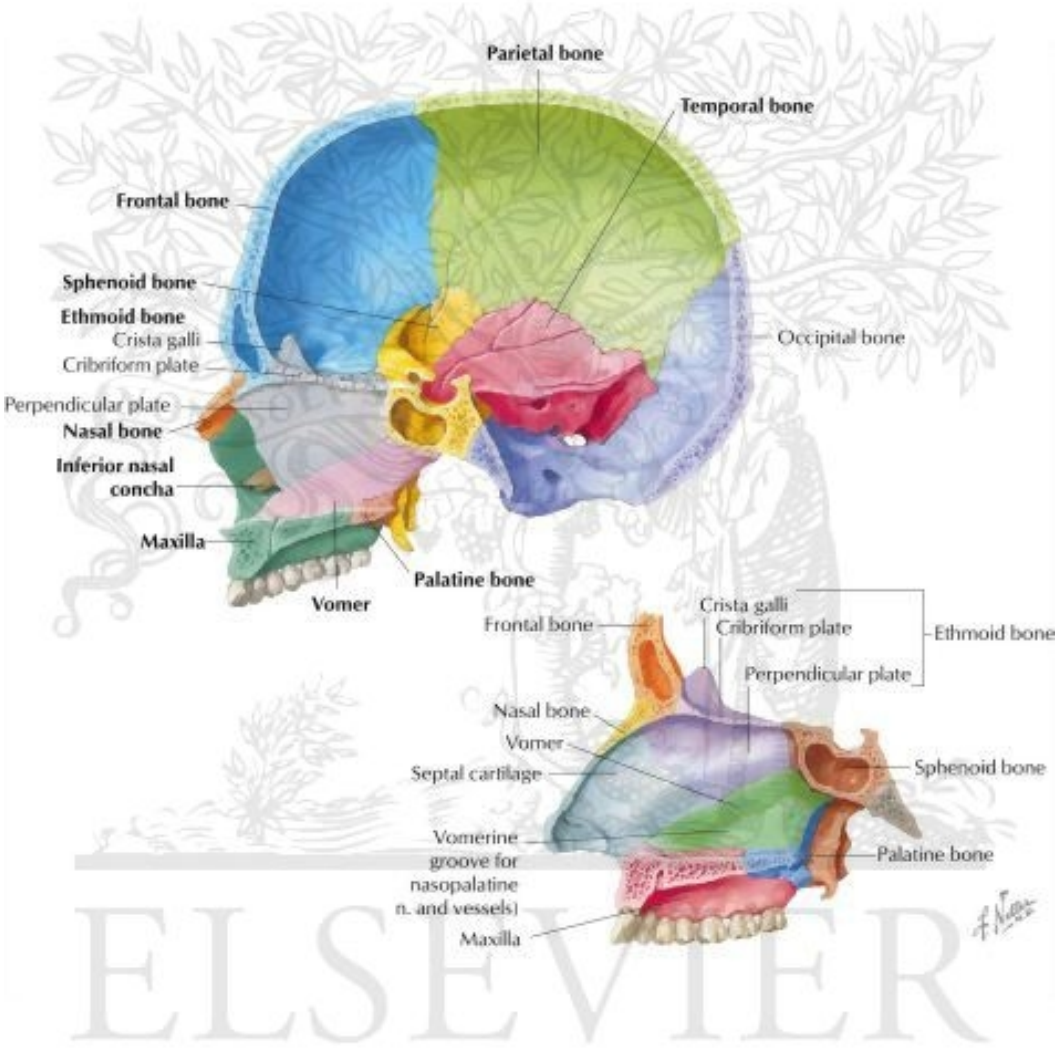
When there exists a naso-lymphatic dysfunction studies show that this drainage pathway is blocked or impaired, it can lead to, or be associated with, chronic rhinitis and olfactory dysfunction. When the CSF cannot drain efficiently through the nasal mucosa (due to obstruction or congestion), it can impair the entire brain's clearance system, potentially contributing to further neuro-inflammation (92, 93, 94, 95, 96, 97). Phillips states that, 'nasal turbinate vasodilation and resultant blood pooling lead to the obstruction of normal nasal lymphatic clearance of cerebrospinal fluid and its waste products from the brain'. (98) The cribriform plate is a vital, sieve-like subcomponent of the ethmoid bone that forms this horizontal boundary. The ethmoid is directly related to the Vomer and the sphenoid bone. In SOT, motion dysfunction of the sphenoid is paramount, and the ethmoid moves in conjunction with the anterior aspect of the sphenoid. (99) (Figure 4) Whedon et al demonstrated that physiologic factors affecting the normal circulation of CSF include cardiovascular, respiratory, and vasomotor influences. Physiologic impedance or cessation of CSF flow may occur commonly in the absence of degenerative changes or pathology and may compromise the normal physiologic functions of the CSF. Their paper found that therapies directed at affecting CSF flow include osteopathic care (especially cranial manipulation), Sacro Occipital Technique, chiropractic adjustment of the spine and cranium, were all successful in restoration of CSF flow. (100, 101)

Considering the glymphatic system, attention must be given to Alpha 7 nicotinic acetylcholine receptors (alpha7nAChR) which we mentioned in relation to VMR, as we observe their affects here as well. The connection with the glymphatic system via neuro-immune regulation is noted through alpha7nAChR activation which reduces neuro-inflammation and maintains the astrocytic function that is so critical for cerebrospinal fluid (CSF) flow and amyloid-beta clearance. By regulating microglia and astrocytes, alpha7nAChR signalling directly supports the efficiency of the glymphatic system in clearing waste from the brain. (98)

The importance of the glymphatic system as a 'pseudo-lymphatic' network in the brain is that it flushes out metabolic waste, such as amyloid beta, primarily during deep sleep. The term glymphatic system was only introduced in 2012 to describe a glia-dependent, perivascular clearance pathway in the brain. And yet, everything it refers to, including perivascular spaces, astrocyte end-feet, cerebrospinal fluid flow, and interstitial clearance, was already well documented. 'They were just not interconnected … this new label did something powerful. It brought anatomy, physiology, sleep biology, and neurodegeneration into a single framework, "Channelopathy" … suddenly, CSF clearance is not just a plumbing issue but a potential key factor affecting health and disease'.

Helbig further stated that 'neurodegeneration research reframed protein accumulation as a drainage problem'. Ion channels do not only set membrane potentials. They also influence development, synaptic release, dendritic integration, and network wiring. (Ingo Helbig neurologist and epilepsy genetics researcher working at the Children's Hospital of Philadelphia (CHOP), USA)

Figure 4: In SOT, motion dysfunction of the sphenoid is paramount, and the ethmoid moves in conjunction with the anterior aspect of the sphenoid.



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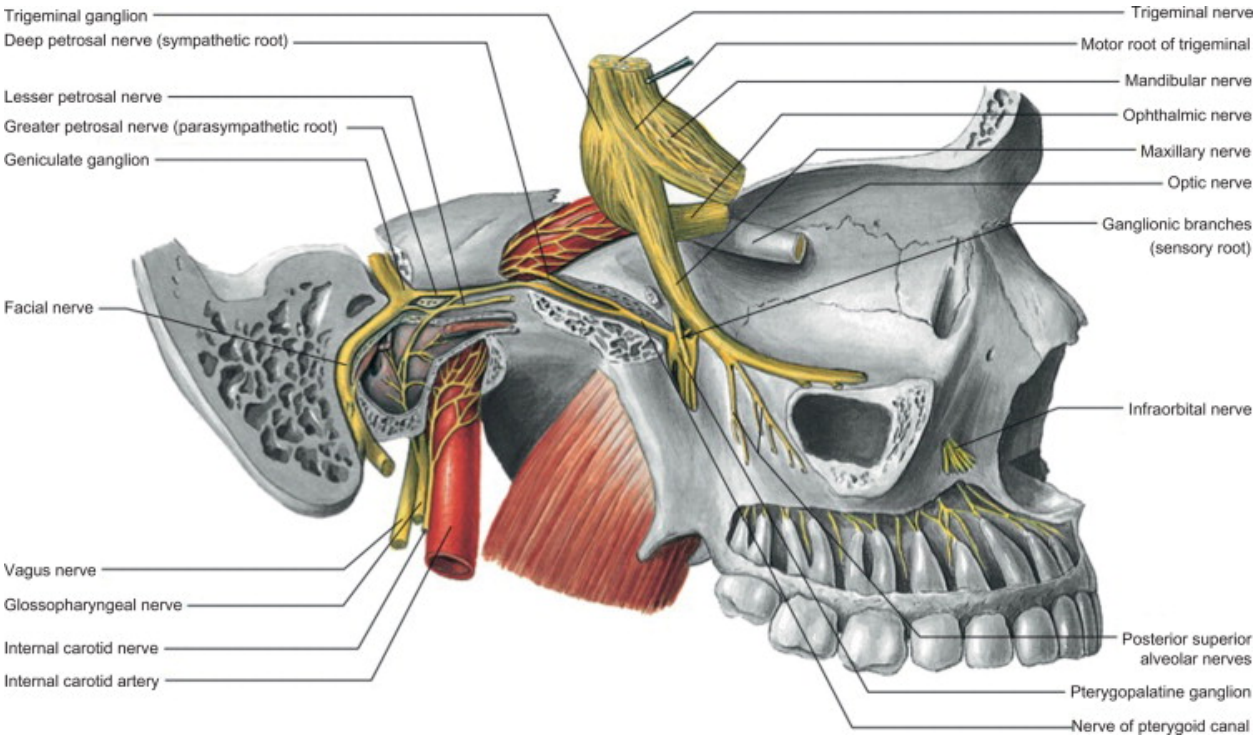
These statements by Helbig has opened a new window for therapies that can affect CSF flow. In dealing with the glymphatic system we recall that the waste-filled fluid exits the brain and must pass through the meningeal lymphatic vessels at the base of the skull before entering the cervical lymph nodes in the neck. (99, 100, 101) The 'Neck Bottleneck' of poor posture causes tension, physical obstruction in the cervical region which will impair this outflow, potentially contributing to 'brain fog' or neurodegenerative decline.

SOT Therapy, as low-force mobilisation of the cranial sutures and suboccipital region (base of the skull), is used to assist the glymphatic-lymphatic continuum. (102) SOT and Chiropractic can affect this entire system as documented by various studies concerning its effect on HRV and hence validating the Chiropractic - ANS -Organ system connection. (103, 104, 105) We know that HRV is an indicator of sympathetic – parasympathetic balance. We further know that the mechanism of VMR is ANS sympathetic-parasympathetic imbalance. Finally, we have shown here that VMR is intimately related to failure of lymphatic and glymphatic drainage and other forms of neurogenic inflammation which are responsible for causative factors in many disease conditions. A case report by Bloink and Blum in the Journal of Chiropractic Education (2020) explored the use of Sacro Occipital Technique (SOT) and cranial procedures for a patient with dysautonomia suspected to be caused by vagus nerve entrapment at the jugular foramen. The intervention aimed to alleviate structural tension between the occiput and temporal bones, it resulted with the patients demonstrating improvement and reduction of the dysautonomia. (106, 107)

In SOT, cranial manipulation exerts an effect on the pterygopalatine plates, part of the sphenoid bone's pterygoid process. This serves as a crucial anatomical anchor and spatial landmarks for the pterygoid muscles of mastication. (Figure 5)

The lateral pterygoid plate, in particular, provides the major site of origin for both the medial and lateral pterygoid muscles. (108) Their primary mechanical effect on these plates is the exertion of continuous tensile and traction forces during mastication, which can influence bone remodelling, temporomandibular joint biomechanics, and long-term skeletal disorders and effect the Vidian nerve (109) Chiropractic and Osteopathic cranial manipulative medicine view the sphenoid bone as the keystone of the cranium. Again, the Vidian nerve passes through the pterygoid canal in the sphenoid body. Chiropractic and Osteopathic theory suggest that restricted mobility or spatial strains of the sphenoid can compress the nerve, potentially impairing autonomic function. (110) Upper Cervical Alignment concentrating on C1 (atlas) and C2 (axis) vertebrae can also ensure the major circulation pathways for brain detox are not restricted. (111, 112) This was shown in various study with asymptomatic control groups.

Figure 5: Cranial relationships of relevance to SOT.



As we place all the focus on the cranium and upper spine we must remember CSF produced in the brain flows through the subarachnoid space and down the spinal canal to the lumbosacral area. Pressure management such as from obstructions in the spinal column, i.e. spinal stenosis, tumors such as Schwannomas, (113) or mechanical tension (subluxation), can impair this flow, leading to metabolic waste buildup. The Sacral Pump utilised in SOT models, acts as a 'pump' for CSF; rhythmic sacral movement during breathing helps drive fluid upward toward the brain and downward for drainage. Sphenoidal movement with respiration provides a similar pumping mechanism. The sphenoid and occipital bones flex and extend at the sphenobasilar junction. (114, 115, 116) (Figure#6)

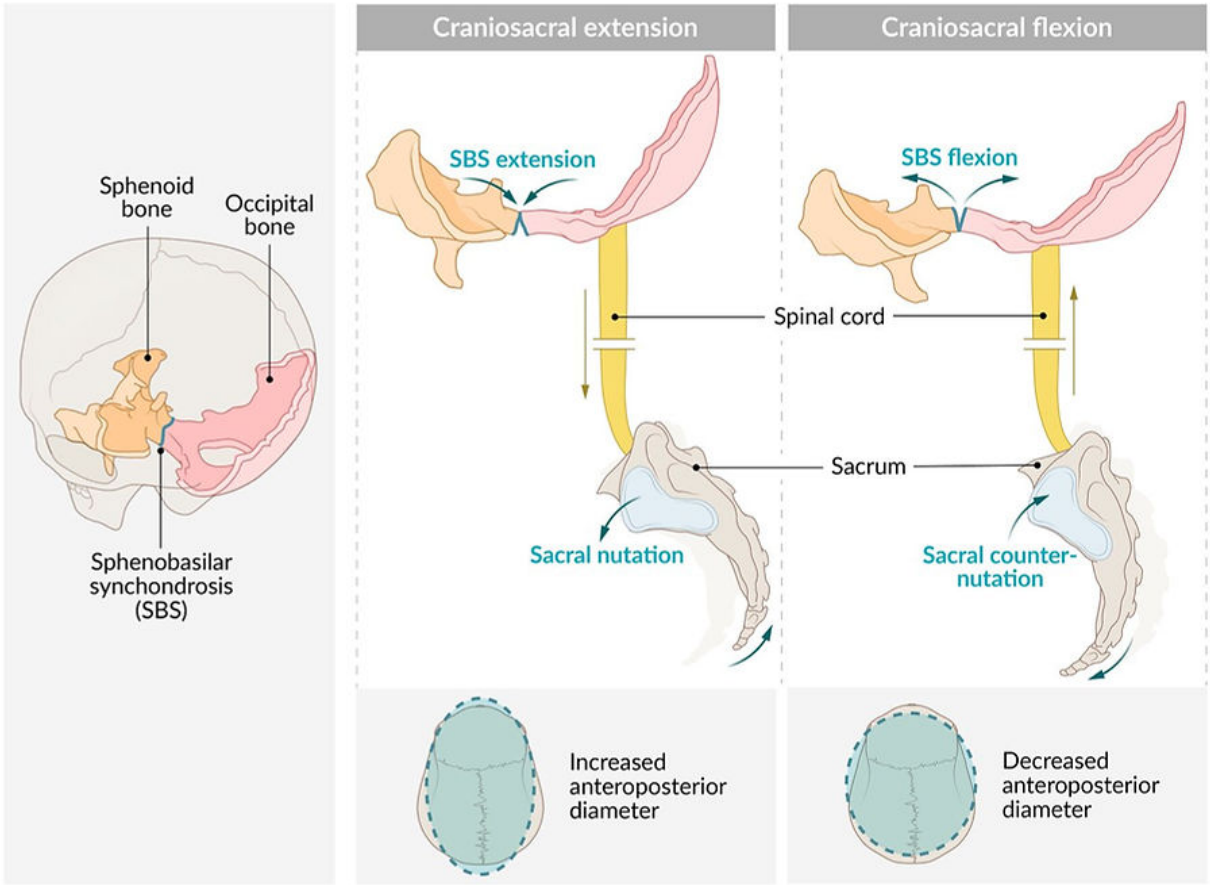
This continuous rocking action acts as a mechanical pump to force CSF down the spine. Rationale and research have been presented in various papers to explain the basis behind Dural Port Therapy. Blum states that 'This Sacro-Occipital Technique (SOT) procedure, which uses the sacrum as a lever to influence and balance the spine and cranium by way of the meningeal system, is an excellent method of affecting the craniospinal system and its relationship to the meninges'. (117, 118) Mechanical Influence is obvious where manipulating the lumbosacral segments can relieve mechanical 'traction' on the dura mater, potentially reducing 'metabolic tension' in the brain's drainage pathways. (119, 120, 121) The therapeutic role of lumbosacral manipulation for meningeal normalisation (122) will aid in glymphatic drainage helping to restore mobility, targeting restricted (hypomobile) segments in the lumbar spine, like L4/L5. (123)

This will normalise the mechanical activity of the spinal cord. There will also be a symbiotic effect with C2 slope unloading the pressure at the brainstem through various fascial and reflexive avenues. (124) Therapeutically SOT enhances CSF outflow even during upright posture and

movement. It enhances CSF outflow in the lumbosacral region via gravitational adaptations. SOT also reduces dural tension, addresses sacral trauma or lumbar stiffness and can transmit positive mechanical changes through the 'fascial continuum' to the base of the skull enhancing the siphoning effect. It has been documented that Manual therapies are observed to create a 'system-wide siphoning effect' that helps move fluids through the brain and body. (125) This has a huge effect on VMR and glymphatic drainage.

The prestigious Cleveland Clinic recommends Chiropractic (SOT) including self-massage, acupressure, and techniques targeting the vomer bone, that can provide immediate, drug-free relief for nasal congestion by promoting sinus drainage, reducing fascial pressure, and relaxing tense muscles often bringing relief within seconds to minutes. (Cleveland Clinic Health-Sinus) (126)

Figure 6: Source, Blum. (116)



Continuing to examine the connections we observe that the sphenoid articulates with the occiput at the sphenobasilar synchondrosis (SBS). During inhalation, the SBS exhibits an upward movement (flexion), and during exhalation, it moves downward (extension). (127) (Figure 6) In the Flexion Phase (Inhalation), The sphenoid body rotates such that the anterior portion moves superiorly and anteriorly, while the greater wings move inferiorly and anteriorly. The midline of the cranium widens. While in the Extension Phase (Exhalation): The movement reverses. The sphenoid rotates in the opposite direction, and the midline narrows. (128) The sphenoid moves in conjunction with the Vomer and Ethmoid bones. As the sphenoid rotates during inhalation, the

ethmoid rotates in the opposite direction to maintain balanced tension on the reciprocal tension membranes (such as the falx cerebri) and promote proper intercranial CSF fluid motion. (129) So, we can see how normalising motion of the occiput and upper spine can contribute to proper CSF flow.

Conclusion

Through this report we believe we have demonstrated the effects that Chiropractic can have on organ function in general and on vasomotor rhinitis in particular. In correcting VMR and restoring normal nasal drainage we have demonstrated the concomitant effect that will have on glymphatic drainage and hence, CSF flow. The information we shared was from a broad spectrum of therapeutic sciences all pointing to an overall aetiology of autonomic nervous system dysfunction, neurogenic inflammation and those biochemical changes that they cause in the human body. We have demonstrated that chiropractic can offer a targeted specific resolution to many organic system malfunctions and with the minimal collateral symptomatology, side effects, or complications. It is the hope of the authors that further clinical studies are performed to complement our conclusions.

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