A personal lived experience. The Quiet Struggle to Breathe again: A Personal Narrative of Persistent Nasal Symptoms Following E-Bike Collision, Septal Fracture and Nasal Surgery

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Abstract

Background: Nasal trauma may result in persistent structural and functional symptoms that can substantially affect breathing, sleep, and quality of life.

However, the lived experience of prolonged nasal obstruction and uncertainty following trauma and subsequent surgery is less frequently described or observed.

Objective: This personal narrative explores the long-term experience of persistent nasal symptoms following an unexpected e-bike collision, septal fracture, delayed surgical management, and subsequent nasal surgery.

Case narrative: In December 2021, the author sustained multiple injuries after being struck from behind by an e-bike while walking on a public footpath; including nasal trauma and septal fracture. Despite having no previous history of significant nasal problems, persistent and alternating nasal obstruction, turbinate swelling, rhinorrhoea, postnasal mucus, crusting, bleeding, sneezing, and sleep disturbance subsequently developed. Following a prolonged wait, septal surgery and inferior turbinate procedures were undertaken, followed by further intervention, without meaningful improvement in symptoms. The absence of clear explanations prompted the author to independently explore nasal anatomy, turbinate physiology, autonomic regulation, and neurogenic mechanisms. Multiple treatments, including intranasal corticosteroids, antihistamines, ipratropium, capsaicin, and non-pharmacological approaches, provided little or no benefit.

Conclusion: This narrative illustrates the substantial physical and emotional burden of persistent breathing difficulty following nasal trauma and surgery. Altered autonomic regulation, neurogenic mechanisms, structural changes, and mucosal dysfunction may contribute; however, the precise mechanism in this individual case remains uncertain. The narrative highlights the importance of communication, explanation, and recognising the lived burden of persistent nasal symptoms.

Keywords:
Nasal trauma; Septal fracture; Persistent nasal obstruction; Inferior turbinate; Septoplasty; Nonallergic rhinitis; Vasomotor rhinitis; Autonomic nervous system; Neurogenic inflammation; Nasal mucosa; Lived experience; Personal narrative; Quality of life.

Introduction

Breathing through the nose is usually an unconscious act. It occurs continuously, without the need to think about airflow, mucosal swelling, secretions, or the changing resistance within each nasal passage. I had never paid attention to my nose or examined my nostrils before December 2021.

The nasal mucosa filters and cleans inhaled air and humidifies, warms, or cools the gas to body temperature. Normal functions of the epithelium, glands, and deep venous sinusoids facilitate this cleansing. This process is highly complex and depends on local mucosal feedback systems, sensory, and autonomic reflexes. Nasal nerves play a central role in regulating nasal patency and secretion production. Stimulation of sensory nerves triggers parasympathetic reflexes, which mediate glandular secretions and obstruct nasal patency, whereas sympathetic influence causes mucosal contraction and thus widens nasal passages; a disorder of which would lead to symptoms of rhinitis. Any insult or trauma conveys messages to the central nervous system via neurons by causing local vascular inflammatory reactions, the release of inflammatory substances, and an increase in blood flow. Each released peptide has its unique functionality, and its binding sites have been mapped in human nasal mucosa.

The actions of the sympathetic and parasympathetic nervous systems are probably integrated at the brainstem level. The coordinated actions of the autonomic systems probably produce the nasal cycle.

Case story

The cost of a split second of recklessness by others: the collision that changed breathing

On 18 December 2021, my life changed within seconds. I was walking forward on a public footpath in Liverpool (UK) when I was unexpectedly struck from behind by an e-bike rider travelling at speed. The force of the collision threw me to the ground. I struck my face, hands, and knees. The rider left the scene.

I experienced immediate nasal bleeding and sustained several injuries, including nasal trauma, a fractured septum, a fractured left little finger, a right knee microfracture, facial swelling and bruising, and grade II whiplash in addition to exacerbation of pre-existing neck problems. I will focus in this paper on the nose section and the aftermath. Woefully, the ENT did nothing immediately, such as closed manipulation within the first two weeks, and the long wait of the NHS added to the hassle and bustle of troubles incurred thereafter. Also, no one explained to me what was going on and what to expect, which left me in limbo and in great despair. I struggled with my breathing, which kept getting worse and is still affecting the quality of my life and my sleep to date.

One of the ENT specialists whom I saw at the very first visit told me I needed nasal septum surgery after three months, which never happened. When I inquired further, he dismissed me harshly without feeling or empathizing with my daily struggles, my situation, and the difficulties I was put through. I felt dismissed and left without an adequate explanation of my symptoms.

I subsequently developed what I understood to be compensatory hypertrophy of my inferior turbinate, which added to the blockage in my nasal passage. I am literally gasping for air and can feel it becomes swollen and seems to adhere to my nasal septum, almost obliterating that nasal passage completely. This is more pronounced when reclining at night (Figures 1 and 2). None of the ENT specialists I saw explained this to me until I started my relentless online journey looking for answers to my odd, unsettling, and upsetting symptoms.

And as none of the ENT specialists explained this alternating blockage, which I kept experiencing and still have to this day, I started examining my nostrils myself. I had lost faith in doctors who never explained why I was experiencing alternating breathing and blockage. I decided to research for myself to find answers to the injuries, pains, and difficulties I was suddenly facing, all of a sudden, all alone, which sadly never left me or returned to normal.

I had never had any problems with my nose before the tragic day of the assault and had never needed to visit an ENT specialist in my life. One ENT specialist said it was an allergy and sent me for an allergy test, which came back negative. I know I don't have any allergy issues with my nose, and I had never experienced nasal problems before the assault.

Figure 1: Notice the extreme narrowing on the right nostril and the widening on the left nostril

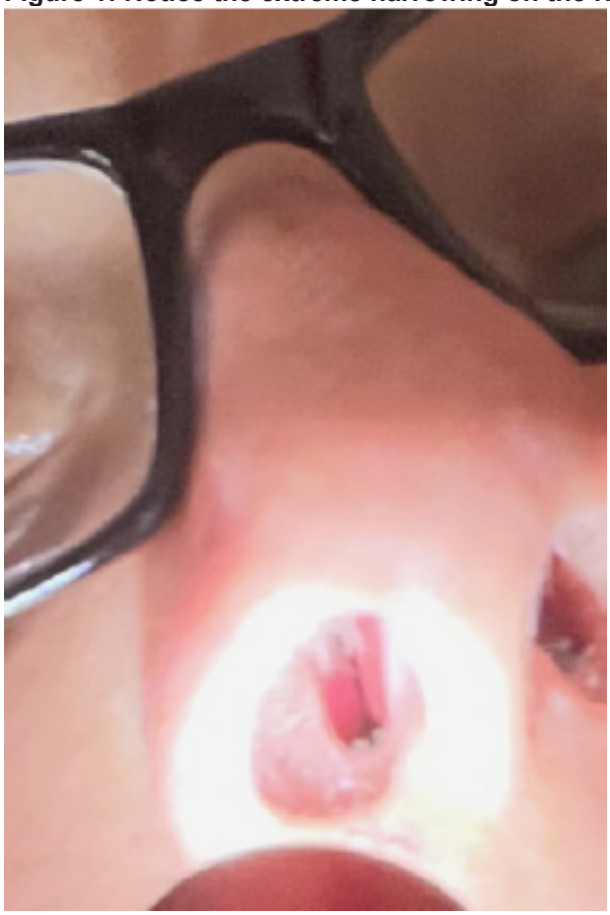
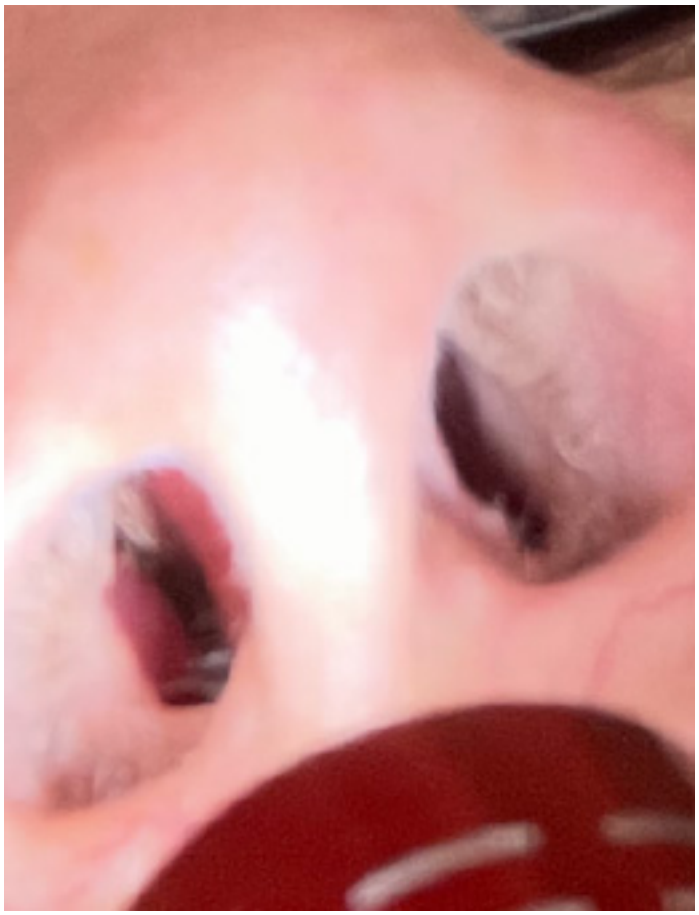


Figure 2: Notice how it started to widen, and it will switch to the other side



Waiting, uncertainty, and delayed treatment and surgery:
I was extremely anxious and deeply afraid of the operation and was very reluctant to undergo it. However, I challenged myself because I could not live like this anymore and eventually underwent the operation after 18 months' wait.

When I came out of the operation, my upper lip was heavy and swollen, with three large, rounded bruises underneath the mucosa. I also had a red, painful left eye that was worsening and had started watering after the first nose operation (Figure 3).

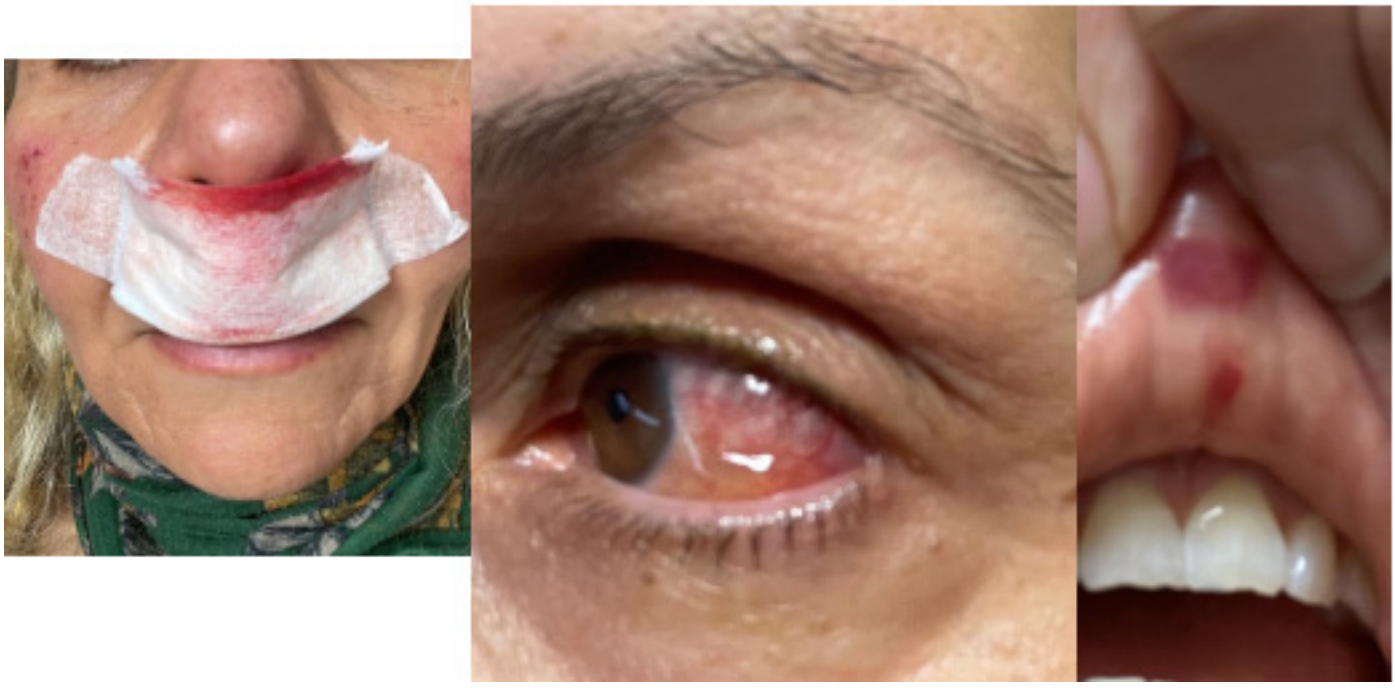
I do not know what happened, as there was no documentation when I contacted the nurse who had carried out the preoperative assessment. I wondered whether it had anything to do with the anaesthesia or handling and draping during the procedure. I had previously undergone three operations back home in Libya under general anaesthesia and had never experienced anything like this.

I managed to attend the eye emergency department, where a laceration/cut to my left conjunctiva was identified. I had to be long-suffering and put up with more pain when I was already in too much pain and agony.

Additionally, I was discharged without being seen or given a follow-up appointment after the nasal correction operation. I am not sure whether that was appropriate, though. I literally could not breathe through my nose, and I kept feeling my inferior turbinate when it became swollen and blocked my nasal passage in that alternating manner. The left turbinate was far larger than the right side. Also from this lived experience I believe that once the turbinates are disturbed they never go back to normal as in my case.

I understand that I had a septoplasty and an outfracture of the inferior turbinate. I therefore decided to write this report to explain the changes and the physiology, in order to understand them myself and make others aware if they need information about a situation similar to mine, to gain knowledge, comprehend what may be happening, and not be left in limbo.

Figure 3: postoperative findings after the first nose operation



Nasal fracture does not only affect appearance but can also affect function. In my situation, it resulted in what I experienced as a predominantly functional disruption and persistent blockage to this date.

Trying to understand the pathophysiology/ physiopathology of sympathetic and parasympathetic systems

Let's start with the inferior turbinate physiology and anatomy

The inferior turbinate has well-recognized respiratory and immune functions to provide the airway with appropriate warmth, humidification, and filtration of the inspired air while sampling the environment for pathogens before reaching the lungs.

Normal functioning of the inferior turbinate relies on an intact, healthy autonomic nervous system (ANS) to maintain homeostasis within the nasal cavity. The autonomic nervous system innervates the submucosal glands and the vasculature within the inferior turbinate, resulting in control of major turbinate functions: nasal secretions, nasal patency, warmth, and humidification (1).

It is made up of a bony frame underneath soft tissue, specifically respiratory epithelium. The bone itself is cancellous, consisting of interwoven trabeculae that accommodate the major arterial supply of the turbinate. The folded structure of the inferior turbinates can increase the mucosal surface area of the nasal cavity up to 200 cm squared (1).

The nasal cavity contains a complex neuronal system that includes parasympathetic and sympathetic nervous supply that maintains turbinate function.

Additionally, the size of the inferior turbinate due to blood flow is correlated with nasal patency as mandated by the nasal cycle. It is recognized that when the nasal septum is deviated toward one side, the excess space in the opposite nasal cavity is occupied by hypertrophic nasal turbinate to compensate (Figure 4). Hypertrophy of the contralateral inferior turbinate is thought to be compensatory to deflection of the nasal septum because the hypertrophy protects the more patent passage from excess airflow, which has drying and crusting effects on nasal mucous membranes (3). However, enlargement of the inferior turbinate significantly increases nasal airway resistance, contributing greatly to symptoms of nasal airway obstruction. In my case, the fracture of the nasal septum caused deflection to the right side, which caused narrowing of the airway, and the left size compensated for by the enlargement of the inferior turbinate on the left side, which to date is quite big and once it enlarges, which I sense, feel and can glance, it impedes the air flow and causes a whistling sound and it vibrate when I force the breathing through it.

Figure 4: Anatomy of the septum and inferior turbinate.



Recent studies showed that any disruptions of the ANS could lead to characteristic symptoms of nasal obstruction, rhinorrhoea, and facial pressure and pain.

Physical nasal obstruction is mediated via a variety of mechanisms, including dilatation of the nasal vasculature, mucosal oedema, the presence of polyps, and physical deformities, which reduce nasal cavity diameter and thus impede airflow.

Superimposed on this is a degree of sensory modulation that contributes to nasal congestion symptoms, with or without the physical obstruction (4).

A major component of nasal obstruction is the engorgement of the nasal vasculature, which is richly innervated by fibres of the ANS.

In my case, the septal fracture was associated with deviation and subsequent asymmetry of the inferior turbinates; however, the precise relationship between these structural changes and my persistent symptoms remains uncertain.

Also, it has been stated that inferior turbinate surgery as an adjunct to septoplasty is associated with increased morbidity, primarily haemorrhage, intranasal adhesions, and atrophic rhinitis (2,3,9).

The present indications for turbinate surgery are based on empirical criteria and have resulted in extensive, unnecessary, or insufficient surgery without objective evaluation. Inappropriate selection of surgery as a therapeutic option and inappropriate choice of surgical modality seems to be the major causes of patient dissatisfaction.

The current literature didn't examine the operation with the evaluation of the nasal cycle postoperatively. It was postulated that the reduction of mucosal oedema after septal surgery is likely the result of diminished submucosal blood circulation.

The effect reported by Illum is probably related to the phenomenon that septoplasty induces a reduction of mucosal thickness even without turbinate surgery.

If, however, septoplasty is successfully performed, the distance between the nasal septum and the inferior turbinate on the concave side of the septum becomes smaller.

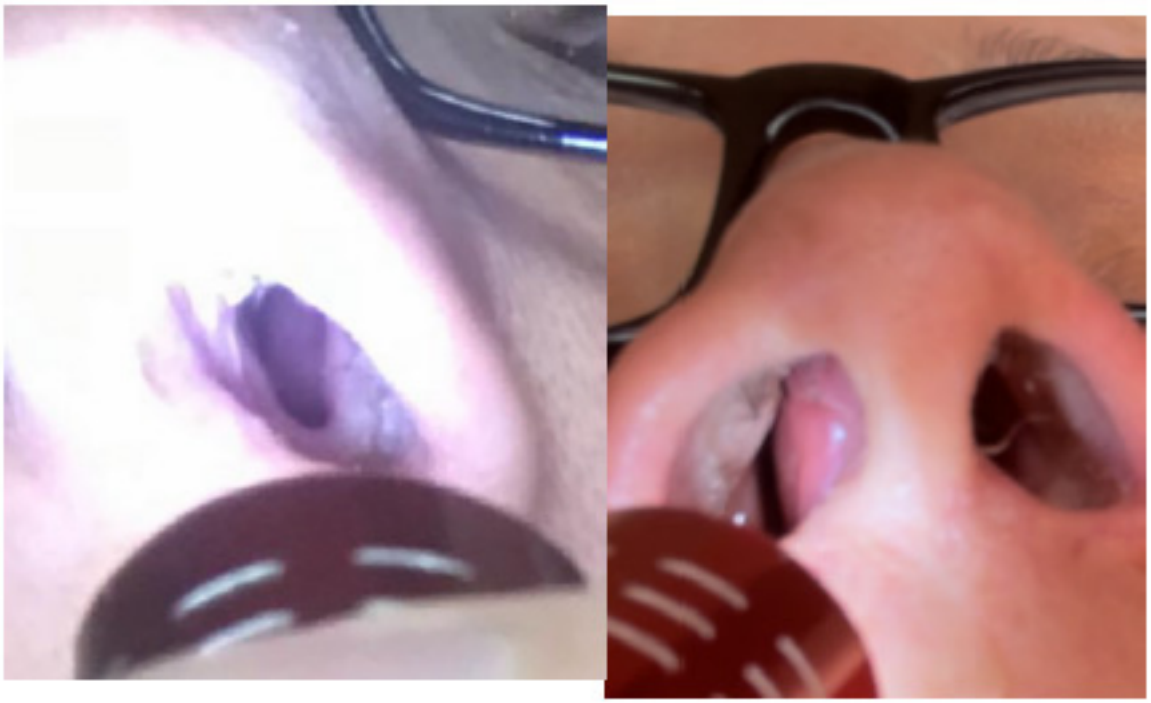
Living with persistent nasal symptoms- How does an enlarged turbinate feel?

It feels literally as if an object, like a soft cotton ball or a small pea, is lodged, trapped, and is merely blocking one side of my nose, and I can feel it once it gets enlarged and touches my nasal septum, which leads to nasal blockage and obstruction, and I can see it with my torch and a mirror. So, I started to examine my nostrils, something I had never done before and never thought to do so, since no one explained what was going on inside my nostrils and why I felt those symptoms of alternate breathing and blockage. It is a nasty sensation by all means to those who know it.

What is septoplasty and turbinectomy?

The doctor, who I only saw for the first time two days before the operation, said he would do a septoplasty and cut the turbinate with a nail-like thing to suck and curate; however, preoperatively, on the day of the surgery, I read debridement and wasn't sure what that was. I believe it is a microdebrider machine used to shave off the bony part and snip it, as I was searching online for some information, as I was never given any information or handed a leaflet of this procedure to read, (Figure 5). The only thing written on the discharge is septoplasty and out fracture of the inferior turbinate (2).

Figure 5: Postoperative image of first operation. Note the hard swelling on the right side of the nasal septum



The nose and the autonomic nervous system

There is a more complex relationship between the nose and the autonomic nervous system (ANS).

It is quite clear now that the nose plays an important role in regulating temperature and in humidifying and protecting the passage of air that enters the lower airways via the rich autonomic innervation of the nasal vasculature and nasal glands (3,9).

Recent studies showed that any disruptions of the ANS could lead to characteristic symptoms of nasal obstruction, rhinorrhoea, and facial pressure and pain.

Physical nasal obstruction is mediated via a variety of mechanisms, including dilatation of the nasal vasculature, mucosal oedema, the presence of polyps, and physical deformities, which reduce nasal cavity diameter and thus impede airflow.

Superimposed on this is a degree of sensory modulation that contributes to nasal congestion symptoms, with or without the physical obstruction (4).

A major component of nasal obstruction is the engorgement of the nasal vasculature, which is richly innervated by fibres of the ANS.

Sympathetic tone, by causing vasoconstriction and emptying of the venous sinusoids via control of arteriovenous anastomoses, is the most important determinant in nasal patency. A reduction in sympathetic tone causes venous sinusoids dilatation and contributes to symptoms of nasal obstruction; whether altered autonomic regulation contributes to my symptoms remains uncertain, but this became one of the mechanisms I explored in trying to understand my persistent nasal obstruction.

I was told I developed chronic vasomotor rhinitis after the second operation, which didn't make any difference or improvement to my breathing and patency (3-4).

Humans exposed to upright posture change, exercise, or temperature changes developed increased sympathetic tone and improved nasal flow.

Whereas parasympathetic nerve stimulation in the nose has the opposite effect on the venous capacitance vessels and causes their dilatation, which seems to be overdominating now and relevant to my relentless existing current symptoms (6).

A small study of 26 patients with vasomotor rhinitis demonstrated heart rate variability (HRV), indicated hyperactive parasympathetic activity compared with controls without rhinitis (4-5).

Both sympathetic and parasympathetic components are suggested to play a role interchangeably in alternating unilateral nasal obstruction symptoms that behave much like an exaggerated nasal cycle. It has been demonstrated that attenuation (reduced force of action) of either sympathetic or parasympathetic innervation to the nose obliterates the nasal cycle phenomenon (6).

Searching for answers; from patient to investigator

What Science Says and attempted treatment and their outcomes:

The nasal mucosa has a sophisticated innervation-sensory, sympathetic, parasympathetic system, that regulates airflow, mucus, and comfort. Any dysfunction or derangement can lead to chronic congestion or altered sensation.

The mucus is relentless and nonstop, always on the back of my throat and nose 24/7, and is an annoying and irritating state to live in, besides the other pains and difficulties along with its limitations. The site of the fracture is always dry with a raw area that never healed along with yellowish crusts that don't go away, and it bleeds from time to time. Also, there are crusts in the right vestibule.

In conditions like empty nose syndrome, nerve and mucosal damage reduce airflow perception even when airflow is sufficient. Restoring moisture, using humidified air, or even tissue implants may help.

In vasomotor rhinitis (a nerve-mediated, non-allergic inflammation), symptoms stem from an autonomic imbalance; too much parasympathetic, too little sympathetic (5). The suggested trials mandate that treatment should focus on avoiding triggers, nasal irrigation, antihistamines, sprays, and sometimes nerve targeting procedures.

Possible mechanisms: what is known and what remains uncertain

Let us understand the nasal discharge

Nasal discharge is the net result of a combination of plasma extravasation from the dilated vessels, inflammation, cell debris, and secretion from submucosal nasal glands and goblet cells. Glandular secretion is controlled by the balance of parasympathetic and sympathetic activity in the nose (6).

Stimulation of parasympathetic activity results in increased nasal secretions rich in lysozyme, immunoglobulin A, and glycoproteins (Figure 7). Now I notice my mucus can be lumpy and hard to expel, and I feel suffocated to get it out, and it is persistent 24/7 (Figure 6).

The Cochrane Library defines neurogenic inflammation as the "injurious stimulus of peripheral neurons and resulting release of neuropeptides which affect vascular permeability and help initiate proinflammatory and immune reactions at the site of injury." Nasal mucosal inflammation potentially contribute to a variety of symptoms, from obstruction to hyposomia. The neuromodulatory role of the ANS has been indicated to be an important component in the response toward potential noxious environmental stimuli (4).

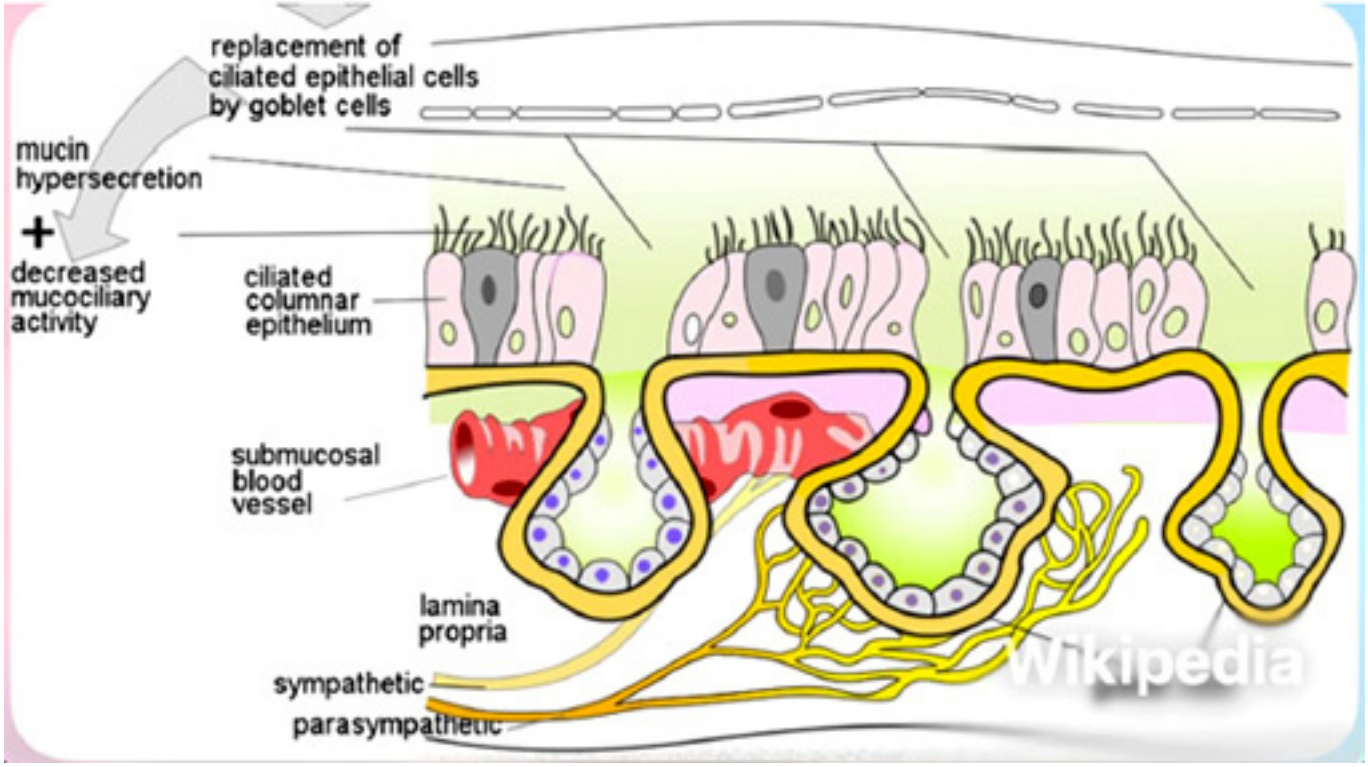
Substance P also seems to be an important mediator of neurogenic inflammation in conditions such as non-allergic rhinitis. Substance P receptors (neurokinin 1 receptor) are expressed in afferent nerves in submucosal glands and airway mucosa (4,7).

A randomized controlled trial that used the substance P-depleting effects of capsaicin demonstrated symptom improvements in patients with non-allergic rhinitis when applied topically for 2 weeks.

Figure 6: Notice the nonstop lumpy, thick, sticky texture of irritating mucus, which can be hard and suffocating to expel



Figure 7: disruption of normal secretions



applied topically for 2 weeks. I bought that online from Holland as it's not available in the UK, and sadly, I tolerated the bad, bitter taste without any improvement or a change in the symptoms I developed (Figure 8). It was costly and didn't improve my state.

Figure 8: Capsinol spray original



Similarly, the topical application of histamine antagonist azelastine is associated with reduced substance P levels and improved vasomotor rhinitis symptom scores, which I tried as well without any improvement, and it has an odd, unbearable taste.

Recent evidence indicates a role of neuropeptides, such as substance P and bradykinin, in immunomodulation. The neuropeptide calcitonin-related gene peptide is a known potent vasodilator whose receptors are found in the nose and paranasal sinuses. Its importance in the autonomic regulation of the vasculature is becoming increasingly recognized (3,7).

The relationship between inflammation and ANS activity is potentially bidirectional because chronic inflammation can upregulate neural hyperresponsiveness and potentiate neurogenic inflammation.

Sneezing and reflexes:
The resultant oedema causes trigeminal stimulation and a positive feedback loop, summating neural impulses into the putative sneezing centre in the medulla. I keep getting bouts of sneezing with excessive dripping from my nostrils, which is another agony (5).

When a threshold is reached, the second phase (respiratory phase) is initiated, which results in a systemic stereotyped and coordinated muscular response that involves abdominal, thoracic, pharyngeal, and facial muscles.

Sinonasal Symptoms and Drug Adverse Effects
Drugs that act on the ANS can have a vasoactive effect on the nose, which causes nasal obstruction and rhinorrhea. For example, nasal obstruction is a recognized adverse effect of blockers, e.g., tamsulosin (commonly used in benign prostatic hypertrophy management) and doxazosin (used in hypertension treatment). Other medications causing nasal obstruction adverse effects include certain psychotropic drugs (e.g., chlorpromazine), peripheral vasodilators (e.g., sildenafil), and hormonal treatments (e.g., estrogen).

A small study of 26 patients with allergic rhinitis found that a 6-week course of high-temperature sauna treatment increased sympathetic activity as measured by HRV (heart rate variability) and increased peak nasal flow, which I tried as well and got some fainting episodes, but didn't notice any betterment as well, despite tolerating it for over 6 weeks trialing myself. Overall, it is possible to hypothesize a complex relationship in which psychological stressors and physical stressors drive mechanisms of ANS dysfunction (3,9).

Neurogenic dysfunction of the nasal mucosa, also known as neurogenic rhinitis, occurs when the nasal nerves are out of balance, causing the sinuses to respond with inflammation when it's not needed. This can lead to congestion and other symptoms without a clear cause (7,10).

Fluticasone propionate and beclomethasone are currently the only topical steroid preparations approved by the FDA for vasomotor rhinitis, which I tried without benefit, along with ipratropium nasal spray.

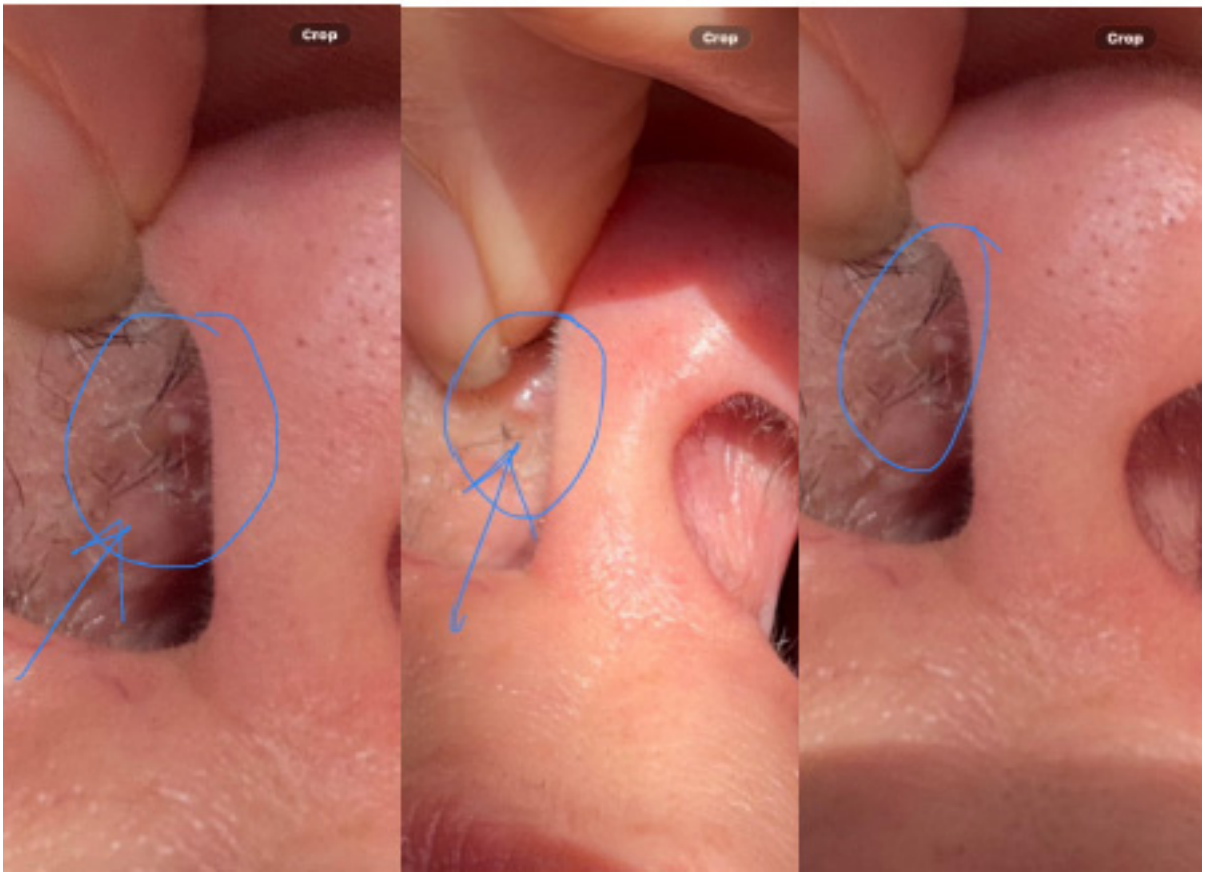
Additionally, I keep getting episodes of painful pustules inside my nostrils lately, and I can't figure out the reasons for that. I had to manage taking photos to prove to the GP, who has no idea whatsoever and keeps saying that they are limited in what they can do or offer (Figure 9).

The lived burden of persistent breathing difficulty
All in all, this is how I live day in, day out. People assume I have the flu and offer tissues and don't understand the relentless new condition I developed, which is affecting and impacting my daily life and the quality of my sleep. My mouth is also dry and becomes very dry when talking, and my saliva has become frothy.

This persistent experience has made me increasingly aware of how much we normally take effortless nasal breathing for granted. What was once an unconscious and automatic process has become something I monitor throughout the day and night.

Possible explanations for developing chronic vasomotor rhinitis after nasal trauma
Developing chronic vasomotor rhinitis (VMR) after a nasal septum fracture may occur due to several interconnected factors related to how trauma affects the nose's nervous system, blood vessels, and mucosal lining. In my case, these are possible explanations rather than established causes.

Figure 9: Notice the painful minute pustule



1. Nerve damage or dysregulation
The nasal mucosa is richly innervated by autonomic nerves, including sympathetic and parasympathetic fibres.

Trauma such as a septal fracture may disrupt normal nerve signalling, potentially causing hypersensitivity or dysregulation in how blood vessels respond to triggers such as temperature, humidity, and strong odours.

This imbalance may contribute to symptoms associated with vasomotor rhinitis, including nasal congestion, rhinorrhoea, and postnasal drip, without an allergic cause.

2. Neurogenic inflammation
Trauma can provoke inflammation, and in some circumstances inflammatory and neural mechanisms may persist after the initial injury.

Neurogenic inflammation involves the release of substances such as substance P and CGRP, which can influence vascular permeability and vascular responses.

These mechanisms may potentially contribute to increased sensitivity of the nose to non-allergic stimuli such as perfumes, smoke, or cold air.

3. Structural changes
A septal fracture may alter nasal airflow patterns, particularly if healing results in persistent deviation.

Abnormal airflow may dry or irritate certain areas of the mucosa and potentially contribute to inflammation and altered mucus production.

These changes may make the nose more reactive and could contribute to symptoms resembling vasomotor rhinitis.

4. Alteration of protective reflexes
The nose has reflex mechanisms that regulate airflow, moisture, and vascular tone.

Injury might potentially affect these neural pathways and alter how the nose responds to environmental changes.

The result could be exaggerated or inappropriate vascular responses, such as excessive constriction or dilation of the nasal blood vessels.

Trauma might therefore have been a trigger that contributed to the development of chronic VMR in my case.

Reflections on early assessment and management of nasal trauma. What I believe should have happened earlier
Preventing vasomotor rhinitis (VMR) after a nasal septum fracture is not always possible. However, early and appropriate assessment and management of nasal trauma may reduce the risk of persistent structural and functional problems.

1. Immediate and appropriate assessment of the fracture
Timely assessment and, where indicated, realignment of a displaced nasal fracture may help restore normal nasal anatomy and airflow.

Delayed or inadequate alignment can result in persistent septal deviation and abnormal airflow patterns and may contribute to ongoing nasal obstruction.

In my case, the prolonged delay left me feeling that I had no understanding of what was happening inside my nose or what I should expect. This was particularly distressing because I had never experienced significant nasal problems before the collision.

Exploring ways of influencing nasal sympathetic activity.
Boosting sympathetic activity in the nasal mucosa has been proposed as one way of reducing congestion because the sympathetic nervous system contributes to vasoconstriction and nasal patency. However, this does not mean that deliberately increasing sympathetic activity is an established treatment for my condition.

1. Topical vasoconstrictors
Sympathomimetics:
These medications mimic sympathetic effects and cause vasoconstriction of the nasal mucosal blood vessels, reducing congestion and improving airflow.

Examples:
Topical decongestants such as xylometazoline and other sympathomimetic agents are commonly used for short-term relief of nasal congestion. Prolonged use can result in rebound congestion and rhinitis medicamentosa.

This is important because prolonged use of topical decongestants can lead to rebound vasodilation and worsening congestion, which could become another source of distress. They therefore need to be used only as directed and for a short period.

2. Temperature changes
Heat exposure:
Some studies have examined sauna treatment and its effects on autonomic activity and nasal airflow. I explored this approach myself but did not experience meaningful improvement.

Temperature extremes:
Changes in temperature, including exposure to cold air, can influence autonomic activity and nasal vascular responses. However, deliberately using temperature extremes is not an established treatment for chronic nasal obstruction.

3. Sphenopalatine ganglion acupuncture
Effectiveness:
Some research suggests that sphenopalatine ganglion (SPG) acupuncture may influence nasal ventilation and nasal patency. However, the evidence remains limited, and this is not something I have established as an effective treatment for my symptoms.

4. Other factors
Exercise:
Physical activity, such as walking or running, can increase sympathetic nervous system activity and may temporarily improve nasal airflow in some people.

Stress management:
Reducing stress may help regulate autonomic nervous system activity, although this should not be interpreted as meaning that my nasal symptoms are psychological. My symptoms began following physical trauma and have persisted despite my attempts to manage stress.

Supportive measures explored
I also explored a number of simple approaches intended to improve nasal comfort and mucus clearance.
- **Hydration:** Drinking adequate fluids may help prevent secretions from becoming excessively thick.
- **Steam:** Steam or a warm shower may temporarily moisten the nasal passages and loosen mucus.
- **Saline nasal irrigation:** Saline irrigation may help clear mucus, crusting, and debris from the nasal passages.
- **Warm compresses:** A warm, moist compress may provide temporary comfort around the face and nasal area.
- **Elevated head:** Sleeping with the head elevated may reduce the sensation of congestion for some people.
- **Humidification:** Maintaining adequate humidity may help reduce dryness and crusting.
- **Essential oils:** I also explored eucalyptus, peppermint, and similar preparations. Evidence for their effectiveness in chronic nasal obstruction is limited, and they may irritate sensitive nasal mucosa.

Natural remedies explored
I also looked extensively online for natural approaches to congestion and mucus production. These included ginger, capsaicin, curcumin, eucalyptus, peppermint, pineapple, and other foods and preparations.

Some of these substances have been investigated for anti-inflammatory or sensory effects, but evidence that they can treat persistent nasal obstruction following trauma is limited. Therefore, I consider these to be part of my personal exploration rather than established treatments.

Ginger
I explored ginger tea because of its reported anti-inflammatory properties. I did not find it to provide a meaningful solution to my symptoms.

Capsaicin
Capsaicin has been investigated in non-allergic rhinitis because of its effects on sensory nerves and TRPV1 receptors. In my case, I tried intranasal capsaicin but experienced no meaningful improvement.

Hydration and warm fluids
I found that maintaining hydration and drinking warm fluids could provide some temporary comfort, particularly when the mucus became thick and difficult to expel, but they did not resolve the underlying problem.

Suggested and attempted treatments

Anticholinergic medications can provide relief from persistent rhinorrhoea. Oral preparations can have systemic side effects such as blurred vision, dry mouth, and thickened secretions. A topical anticholinergic such as ipratropium bromide can be useful for rhinorrhoea. Two separate randomised controlled trials demonstrated its effectiveness in controlling rhinorrhoea (10). Topical anticholinergics act locally and reduce parasympathetic stimulation of the nasal glands (10). Epistaxis and nasal dryness may occur (10).

I tried ipratropium nasal spray because of my persistent rhinorrhoea. However, I did not experience meaningful overall improvement.

Oral antihistamines have a limited role in vasomotor rhinitis and tend to be more useful when sneezing and itching are prominent. Topical antihistamines such as azelastine can be used for both allergic and non-allergic rhinitis.

Azelastine is an H1-receptor antagonist and may have additional antiinflammatory effects. It has demonstrated benefit in some patients with vasomotor rhinitis, including symptoms such as rhinorrhoea and nasal congestion (10).

I also tried azelastine but experienced no meaningful improvement and found the taste unpleasant.

Patients with chronic vasomotor rhinitis may be less responsive to pharmacological treatment than patients with allergic rhinitis. The combination of intranasal corticosteroids and topical antihistamines has been used in chronic non-allergic rhinitis (10).

Topical capsaicin has also demonstrated efficacy as an adjunctive therapy for some patients with rhinorrhoea and nasal congestion (10). Its mechanism is thought to involve modulation of C-fibres associated with nociceptive neurons.

Capsaicin targets transient receptor potential vanilloid type 1 (TRPV1), an ion channel present in the nasal mucosa that is involved in sensory responses.

Repeated intranasal applications of capsaicin may desensitise TRPV1 and reduce sensitivity to certain physical and chemical stimuli. However, its irritant quality can make treatment difficult to tolerate.

I tried capsaicin myself, but despite tolerating the unpleasant taste, I experienced no meaningful improvement.

Sympathomimetic medications

Topical decongestants can provide short-term symptomatic relief. They work primarily through stimulation of alpha-adrenergic receptors in the vasculature of the nasal mucosa. This causes vasoconstriction, decreased blood flow, and subsequently reduced congestion.

However, long-term use can lead to rebound vasodilation and increased congestion. This condition is termed rhinitis medicamentosa, a form of drug induced rhinitis. Therefore, topical decongestants should be limited to shortterm use according to recommended dosing.

Botulinum toxin

Botulinum toxin has also been investigated as a potential treatment for vasomotor rhinitis through its anticholinergic effects (10). Botulinum toxin inhibits acetylcholine release from presynaptic nerve terminals.

Injection into the turbinates has been reported to decrease rhinorrhoea and nasal mucous gland secretion in patients with vasomotor rhinitis. The improvement is generally temporary (10).

Although this is an interesting potential treatment, temporary symptom relief may not be a practical option for every patient.

Surgical treatment

When medical management does not adequately control symptoms of vasomotor rhinitis, surgical interventions may sometimes be considered.

Mucosal-sparing inferior turbinate reduction surgery can improve symptoms of nasal obstruction while attempting to preserve turbinate function (10). I was advised any further operation will worsen things and I already have dryness, crusts and I can't tolerate heat, all of which I noticed myself.

However, my own experience has made me particularly cautious about further nasal surgery. I have already undergone nasal procedures without meaningful improvement and remain uncertain about whether further intervention would help or potentially create additional problems.

Long-term burden

One study by Rondon and colleagues re-examined 180 patients with nonallergic rhinitis three to seven years after initial diagnosis. A substantial proportion experienced worsening or persistence of symptoms, and some developed additional comorbidities (10).

Chronic non-allergic rhinitis can interfere with work, sleep, daily activities, and quality of life. It can also result in frequent healthcare consultations and ongoing treatment. Medical treatments themselves may produce undesirable effects such as nasal dryness, epistaxis, palpitations, and drowsiness. These effects can compound the negative impact of chronic rhinitis on patients (10).

Chronic non-allergic rhinitis may also co-exist with conditions such as headaches, eustachian tube dysfunction, nasal polyps, obstructive sleep apnoea, and chronic cough, further complicating management and affecting quality of life.

My personal experience is summarised below:

Intervention	Reason for trying	Personal experience
Intranasal corticosteroids	Nasal inflammation/congestion	No meaningful improvement
Intranasal antihistamine	Possible non-allergic rhinitis	No meaningful improvement; unpleasant taste
Ipratropium	Persistent rhinorrhoea	No meaningful overall improvement
Capsaicin	Possible neurogenic mechanism	Poor taste/tolerability and no meaningful improvement
Saline and supportive measures	Mucus and nasal comfort	Limited symptomatic benefit
Sauna/heat exposure	Exploration of possible autonomic effects	No meaningful improvement; I experienced fainting episodes and discontinued this approach

Turbinates are considered a functional organ for climatisation, humidification, and cleaning. Before total turbinectomy was performed, which is now considered a nasal crime due to its longterm complications and disabilities such as atrophic rhinitis and ozaena (smelly nasal discharge with atrophy of nasal structures (11).

From my extensive reading the proposed surgery is nerve cutting through the sphenopalatine foramen, which transmits signals for triggering the allergic immune response.

Most proposed operations seem to have observed studies only reaching level 1, as they have too few patients and a too short follow up period (11).

Chronic vasomotor rhinitis symptoms can interfere with work performance and daily activities because of reduced productivity, sleep disturbance, and frequent healthcare visits. Chronic non-allergic rhinitis can therefore place considerable physical, emotional, and economic burdens on sufferers.

Conclusion

To conclude, the unseen burden of persistent breathing difficulty and this new state has added a substantial burden to my existing neck pain. The constant, relentless blockage is overwhelming, unseen, disabling, and incapacitating by all means.

One possible explanation for my persistent symptoms is altered autonomic regulation or neurogenic mechanisms following trauma, altered structure, mucosal dysfunction, altered neural regulation, and subsequent surgery. However, the precise mechanism in my individual case remains uncertain.

From my perspective, the problem feels predominantly functional, although structural changes are also present. It affects my days and nights and disturbs my sleep.

I find myself drained and exhausted all the time and still cannot find a solution.

What was once an unconscious and effortless act, breathing through my nose, has become something I am constantly aware of 24/7. I have had to learn about anatomy, physiology, autonomic regulation, nasal mucosal function, and possible neurogenic mechanisms simply because I wanted to understand what was happening to me.

The greatest difficulty has not only been the physical sensation of obstruction, mucus, dryness, crusting, sneezing, and disturbed sleep. It has also been the uncertainty and the feeling of being left without an explanation.

This is the quiet struggle: breathing is something most people never have to think about. For me, it has become something I notice every day and every night.

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