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Mechanisms and clinical applications of the valsalva maneuver in migraine relief: A mini-review

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Abstract

Background: Migraine headaches (MH) are often managed with pharmacologic treatments, but there is growing interest in non-pharmacologic approaches that can reduce reliance on medications. The Valsalva Maneuver (VM) may offer a novel approach to managing MH.

Methods: We conducted a comprehensive literature review in this regard using Google Scholar and PubMed. The search focused on studies examining the relationship between VM and MH, various aspects of VM, and studies regarding migraine etiology.

Results: Our search reviewed 4,659 articles and included 57 that discussed the relevant topics. The findings suggest that VM may serve as an effective non-pharmacological technique for reducing MH severity. Several potential mechanisms may contribute to this event, including: 1- Autonomic Nervous System (ANS) Modulation: VM influences cardiac function and the trigeminovascular system (TVS), which are dysregulated in migraine patients. 2- Vascular Regulation: Abnormal vascular resistance in the dura mater and cerebral arteries, important in migraine pathophysiology, may be alleviated through VM-induced readjustment of vasodilation and modulation of the vasoconstrictor index (VI). 3- Intracranial Pressure and Neurochemical Modulation: Controversial but intriguing mechanisms suggest that VM regulates internal air pressure within the skull sinuses, manipulates cerebrospinal fluid (CSF) pressure, induces transient hypoxic effects, and triggers the release of endogenous pain modulators, all of which could contribute to shortening MH duration.

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Conclusion: The VM demonstrates potential symptom-relieving benefits in MH, supported by both verified evidence and some unexplained findings. However, large-scale clinical trials on this topic are lacking to clarify the mechanism of the role of VM in MH and to establish a standardized protocol for its application.

Introduction

Migraine is a prevalent neurovascular condition impacting a vast number of individuals globally, exceeding 1 billion in count.[1] Its extensive occurrence and resulting disability have diverse and significant repercussions, not solely on the affected individuals but also their relatives, peers, employers, and the overall society.[2] Traditional migraine treatments encompass pharmaceutical agents, lifestyle adjustments, and behavioral interventions.[3] Given the adverse effects associated with analgesic usage and the aim of curtailing expenses, there is a predisposition towards non-pharmacological migraine management.[4] Most non-pharmacological approaches for migraine headache (MH) relief focus primarily on symptom management, such as avoiding light exposure, resting in a supine position, and maintaining hydration to reduce nausea and subsequent MH. However, there is a notable lack of easily accessible techniques targeting MH based on its underlying pathophysiology.[5,6] The Valsalva maneuver (VM), a physiological phenomenon influencing multiple body systems—including cardiovascular, pulmonary, cerebrovascular, and musculoskeletal functions—emerges as a potential candidate for non-pharmacological migraine relief.[7,8] It involves 4 phases, each associated with specific changes in the cardiovascular system and the autonomic nervous system (ANS). In phase 1, increased intrathoracic pressure leads to initial bradycardia due to vagal activation, followed by baroreflex responses. Early phase 2 sees a drop in venous return and cardiac output (CO), triggering compensatory sympathetic activation and vagal-mediated tachycardia. During late phase 2, sympathetic tone increases further, restoring CO and blood pressure (BP). Phase 3 begins with pressure release, causing a transient drop in BP and pulse pressure, with heightened sympathetic activity to stabilize circulation. Finally, in phase 4, preload and BP normalize, shifting the balance toward parasympathetic tone, leading to heart rate (HR) stabilization. This sequence highlights the dynamic interplay between sympathetic and parasympathetic systems in maintaining hemodynamic stability (Figure 1).[7,9-11]

VM has a definite association with migraine-related mechanisms, including the ANS, vascular wall resistance, cerebral blood flow (CBF), cerebrospinal fluid (CSF) pressure, and endogenous neurochemical secretion, all of which have recognized roles in migraine pathophysiology.[3,12-14]

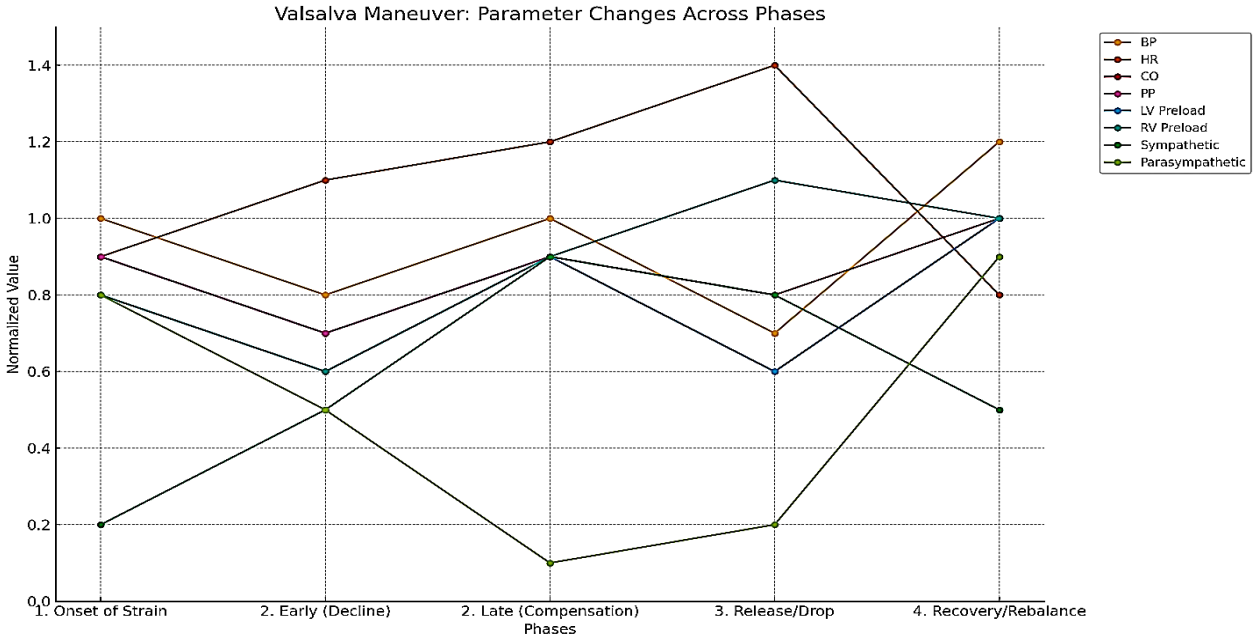

Figure 1. Valsalva maneuver (VM) phases: Physiological and autonomic nervous system (ANS) changes
LV: Left ventricle: RV: Right ventricle: BP: Blood pressure; HR: Heart rate; CO: Cardiac output; PP: Pulse pressure

This is particularly relevant given the promising evidence suggesting a possible mechanistic link between VM and MH alleviation, as well as positive responses reported in the literature.[15] This review critically evaluates the potential role of VM in MH management and explores its therapeutic viability as a non-pharmacological intervention.

Materials and Methods

To investigate the literature examining the effects of VM on MH, we conducted a comprehensive literature review using the Google Scholar and PubMed databases. Our primary search utilized keywords including "Migraine," "Chronic headache," "Valsalva maneuver," "Autonomic nervous system," "Cerebrospinal fluid," "Cardiovascular," and "Cerebrovascular."

To explore the relationship between VM and MH, we screened studies published between 2000 and 2024, based on their titles and abstracts. We selected studies that assessed MH during VM, investigated the physiological effects of VM relevant to MH mechanisms, examined various medical applications of VM, and explored hypotheses regarding the etiology of migraines.

We excluded letters to editors and articles with redundant content, while including all original research articles and reviews. Due to the limited number of studies specifically addressing this topic, we did not perform a formal quality assessment and instead included all relevant studies. Finally, we extracted key findings from the selected studies, focusing on potential mechanisms of VM efficacy in migraine relief, and conducted a critical review to identify correlations across different study results.

Results and discussion: After reviewing 4659 articles and addressing the associated controversies, we ultimately included 57 experimental studies, encompassing in vivo, observational, clinical trials, and review articles. The comprehensive findings of our investigation are organized into 3 main sections: "Migraine Pathophysiology," "Valsalva Maneuver," and "Underlying Processes."

Migraine pathophysiology: Insights from the ANS: A recent systematic review by Pavelić et al. established that autonomic dysfunction (AD) is a well-documented feature in migraine patients. The study further suggests that migraine-specific prophylactic therapies should focus on modulating ANS function as a potential strategy for migraine relief and management.[16] Some investigations have indicated the presence of AD, specifically a reduction in parasympathetic activity, among individuals with migraine.[3,13] Heart rate variability (HRV) denotes the fluctuation in the time gap between successive heartbeats, a phenomenon regulated by the ANS.[17] The utilization of the Standard Deviation of the Average Normal-to-Normal (SDANN) and Root Mean Square of Successive Differences (RMSSD) as parameters to assess HRV implies a plausible association between HRV patterns and the occurrence of migraine episodes.[18] Individuals diagnosed with migraine exhibited notably elevated resting pulse rate and mean HR, alongside significantly diminished mean R-R interval, SDANN, and RMSSD compared to their healthy counterparts.[13]

AD also impacts cardiac function in migraine patients.[19,20] Electrocardiographic changes observed during a migraine attack confirm that ventricular repolarization is influenced by dysregulation of the ANS.[21,22] While most studies have reported an increased cardiovascular risk, including ischemic stroke and myocardial infarction, among migraine patients—particularly in women and those experiencing migraine with aura,[19,23,24] some cohort studies have also identified a higher prevalence of cardiac arrhythmias, such as atrial fibrillation, in this subgroup. This association is likely a consequence of AD.[25]

This dysfunction can extend beyond the cardiac function, affecting the cardiovascular system as well. Postural changes significantly impact physiological indices, with studies showing notable differences in diastolic BP variability between migraine patients in a supine position and the general population. Specifically, findings indicate reduced sympathetic vasomotor activity when patients are lying supine, whereas a standing posture triggers hyperresponsive sympathetic vasomotor activity in individuals with migraine.[20,25]

Concentrating on CNS, an important phenomenon related to ANS is the vasoconstrictor index (VI), a quantitative measure of cerebrovascular constriction used to assess vascular responsiveness and regulation during migraine episodes. An increased VI reflects the initial phase of a migraine attack, where cerebral blood vessels constrict before transitioning into the subsequent vasodilation phase. This disproportionate vascular response in migraine sufferers may be triggered by underlying AD.

Delving deeper into the issue, it is well-established that migraines with aura are associated with more pronounced AD compared to migraines without aura.[13] Additionally, the interictal period is characterized by greater sympathetic impairment, whereas the ictal period shows increased sympathetic responsiveness, indicating potential adrenoreceptor hypersensitivity in migraine patients.[13] This suggests that AD in MH is highly significant, to the extent that its varying severity may influence the presentation and diversity of migraine symptoms.[17,26]

Another important aspect of ANS in migraine pathophysiology is the activation of the trigeminovascular system (TVS), which consists of both somatic and autonomic nerve fibers along with meningeal blood vessels. TVS likely contributes to the initiation of migraine through several mechanisms.[27-29] Some researchers suggest that painful impulses from the trigeminal nerve act as the primary trigger for migraine episodes. The initial phase of vasodilation is often followed by the constriction of densely innervated blood vessels within the dura mater, a region recognized as a key site for the generation of nociceptive signals associated with MH.[30,31] Moreover, a variety of neuropeptides and neurotransmitters, including substance P, calcitonin gene-related peptide (CGRP), and glutamate, participate in the propagation of pain impulses within the TVS.[1] This activation and sensitization of the trigeminal nerve in a migraine attack leads to the release of neuropeptides and inflammatory mediators, causing vasodilation, neurogenic inflammation, and pain.[3,32,33]

These various ANS-related mechanisms in MH highlight the need to explore how VM influences the body, with a particular focus on its effects on the neurological system.

VM: VM entails straining against a closed glottis by forcefully constricting the chest muscles, which as a consequence will lead to a marked elevation of intrathoracic pressure. Such an increase in intrathoracic pressure will largely impede venous return, subsequently reduce CO, and ultimately result in a rapid fall in arterial BP that will seriously challenge the cardiovascular nervous reflex regulatory functions.[34] This breathing technique induces physiological changes across its 4 phases, affecting all the organs influenced by ANS as mentioned earlier.[7,8,12] This approach shows potential as a promising strategy for alleviating migraine symptoms. A clinical trial, conducted in 2022, investigated the efficacy of the VM in alleviating migraines. The findings suggest the potential of the VM as a non-pharmacologic technique for managing headache episodes. This discussion explores the possible mechanisms underlying how the VM may reduce headache symptoms, incorporating both physiological and molecular considerations.[15]

The patient respectively experiences an increase in intrathoracic and intra-abdominal pressure followed by an increase in peripheral resistance in phase 1. The decrease in ventricular end-diastolic volume, CO, and systolic arterial pressure in phase 2 has an inhibitory effect on the vagus nerve center (parasympathetic) and an excitatory effect upon the vasomotor center (sympathetic) and results in reflex tachycardia and peripheral vasoconstriction and raised venous pressure. It causes a further decrease in BP in phase 3 and an "overshoot" of arterial pressure in phase 4 which causes reflex bradycardia. There is a marked transition towards parasympathetic dominance in phase 3, as the abrupt upsurge in cardiac filling instigates a vagal reflex and engenders a transient reduction in HR.[7,8] Our clinical trial demonstrated that these reflexive responses, along with the final deep exhalation, can help alleviate MH in patients who use VM repeatedly at the onset of their symptoms.[15,35] This sets the stage for the exploration of how migraine rehabilitation occurs through VM.

Underlying processes: To investigate how VM alleviates MH, it is notable that no focused study has specifically evaluated its effects on migraine patients performing the maneuver. However, considering AD in migraines, discussed earlier in this paper, manipulating the ANS through VM can have rehabilitative effects on MH.[13,17,36] But how does it work?

Considering the importance of VI, as mentioned before, dysfunction in the CBF regulation is considered a key contributor to MH. Several studies propose that the transient modulation of CBF dynamics and total oxygen delivery, both of which are influenced by the VM, play a probable role in migraine symptom relief by altering vascular tone and cerebral perfusion patterns.[29,37] Preliminary angiographic inquiries corroborate the role of basal artery wall tone dysregulation, such as in the carotid artery, in migraine pathogenesis.[38]

The mechanisms behind this possible association are not known, but some speculative

possibilities include endothelial dysfunction, micro-emboli formation, and compressive excitation of TVS.[2] The significant reduction in vascular resistance observed during the vagal reflex may have a rehabilitative effect on the basilar artery, potentially benefiting MH pathophysiology.[38] During VM, baroreceptor activation influences the cardiovascular system, affecting HRV, BP variability, cardiac function, and vascular resistance. Given that these physiological indices are often dysregulated in MH, VM holds potential as a therapeutic intervention that improves migraine symptoms by enhancing cerebral perfusion and autonomic balance.[8,34,39]

From another perspective, neural activity has become a central focus in recent innovative migraine management techniques, including transcranial magnetic stimulation (TMS) and transcutaneous electrical nerve stimulation (TENS). Both methods have demonstrated encouraging results in clinical trials.[40] One study found that non-invasive vagus nerve stimulation (n-VNS) significantly reduced migraine or headache days, non-invasive cervical vagus nerve stimulation (n-cVNS) for migraine markedly increased (≥ 50%) responder rate, and low-frequency non-invasive auricular vagus nerve stimulation (n-aVNS) significantly reduced headache intensity. The findings support the potential of n-VNS to reduce MH burden and improve the quality of life (QOL) in migraineurs.[41] A similar theory has been proposed to explain the efficacy of acupuncture in MH, suggesting that it works through ANS modulation and its impact on HRV.[42,43] Given that VM facilitates ANS modulation and stimulates the vagus nerve, it may employ a similar mechanism like n-VNS and acupuncture to regulate pain perception in MH.[35]

CSF pressure is a contentious factor in the etiology of MH.[39] Various headache types are associated with CSF pressure abnormalities, including intracranial hypertension (IIH), cough-induced headaches, and intracranial hypotension headaches.[40] Additionally, there exists an overlap between new-onset migraines and IIH-related headaches.[41] These complexities suggest that manipulating CSF pressure may have diverse effects on MH, depending on the underlying pathophysiological mechanisms.[44-46]

Research suggests that alterations in CSF pressure and its dynamics can influence neuroinflammatory processes within CNS, potentially mitigating pain sensitization and neuronal hyperexcitability.[42] During phase II of VM, an increase in CSF pressure can occasionally induce headaches, as seen in primary headache disorders triggered by the VM.[43] However, this transient increase in CSF pressure may also promote enhanced CSF circulation within the CNS. Given the role of inflammatory substances in migraine headaches (MH), as underscored by some studies, this displacement of CSF could theoretically help move inflammatory substances away from trigeminal meningeal nociceptors, thereby reducing the transmission of pain signals to higher brain centers and potentially alleviating MH. Further research is needed to confirm this hypothesis.[27,28,33,47-49]

Based on some studies, there is a compelling association between hypoxia and headaches.[14,50] The potential mechanisms underlying hypoxia-induced headaches include the release of nitric oxide and CGRP. Additionally, some researchers suggest neuroanatomical changes, such as cortical spreading depression and blood-brain barrier leakage, as contributing factors.[51,52] During phase II of VM, transient hypoxia occurs, potentially inducing neural stimulation patterns that, combined with the rapid recovery facilitated by deep exhalation and the return to normal respiration in phase III, may contribute to alleviating MH.[8] This mechanism could form the core of the VM's rehabilitative efficacy in managing migraines through repeated application.[15]

Air pressure within different sections of the airways is another debatable factor in this context. Variations in air pressure, particularly those affecting the sinuses, may trigger or exacerbate MH in susceptible individuals.[3,53,54] Significant fluctuations in atmospheric pressure, such as those experienced during alterations in weather conditions, changes in altitude, engagement in activities like scuba diving, or as a consequence of sinusoidal pathologies, can exert an influence on the pressure within the sinus cavities and the contiguous cranial structures.[54,55] The VM, by adjusting the air pressure within the sinuses, holds promise in addressing this contributory factor.[7,8,54]

Some research suggests that endogenous opioids, including endorphins, enkephalins, and dynorphins, are released during the VM, particularly during the deep breath preceding phase I and the deep exhalation in phase IV.[56,57] Additionally, the release of neurotransmitters such as serotonin and gamma-aminobutyric acid (GABA) has been observed during VM.[13,37]

Table 1. Potential mechanisms of the valsalva maneuver (VM) in migraine headache relief

Affected system	Mechanism	Description
ANS	Regulation of cardiac function	Modulation of HRV and BP variability
	Modulation of VI	Regulation of CBF and oxygen delivery to the brain
	Influence on the TVS	Improvement of nociceptive signal processing, vasodilation of meningeal blood vessels, and regulation of neuropeptides and inflammatory mediators
Respiratory system	Regulation of air pressure in paranasal sinuses	Equalization of atmospheric pressure within the cranial sinuses
	Mitigation of hypoxia-induced headaches	Termination of transient hypoxia and stimulation of the respiratory center through deep exhalation
Craniospinal hydrodynamics system	Regulation of CSF pressure	Enhancement of CSF circulation and clearance of neuroinflammatory mediators
CNS	Neurotransmitter and endogenous pain modulation	Stimulation of the release of endorphins, enkephalins, dynorphins, serotonin, and GABA
	Vagal nerve stimulation	ANS modulation and HRV improvement

ANS: Autonomic nervous system; CNS: Central nervous system; TVS: Trigeminovascular system; CSF: Cerebrospinal fluid; VI: Vasoconstrictor index; HVR: Heart rate variability; BP: Blood pressure; CBF: Cerebral blood flow; GABA: Gamma-aminobutyric acid

This can explain why VM is used as a pain-relieving method in various nursing procedures.[58] These substances collectively can influence pain pathways, ameliorating migraine-related discomfort.[38]

A summary of our findings supporting the efficacy of VM in MH relief is presented in table 1.

In contrast, certain headache types, such as exertional headaches, cough-induced headaches, or laugh-induced headaches, can occasionally be triggered by VM. However, these headaches are typically localized, short-lived, and not widespread.[59] A recent updated study on headaches associated with cough, exercise, and sexual activity reported that these headaches are linked to specific clinical characteristics and structural lesions, indicating that they cannot be generalized across all headache types.[60]

In the aforementioned clinical trial, some patients reported experiencing such headaches post-intervention; however, the overall efficacy in headache relief remained favorable. While in our clinical trial significant alterations were not observed in pain scores following VM, discernible improvements were noted in the "Overall Headache Relief Rate" and the "effectiveness of subsequent use" among participants.[15]

Limitations: One limitation of this review is the scarcity of studies specifically examining the clinical effects of VM on MH. Existing research in this domain often involves small sample sizes, including our own clinical trial, limiting the statistical power and generalizability of the findings. Moreover, many studies investigating the anatomical and physiological aspects of migraine pathophysiology—particularly those employing invasive methods—are outdated, which may reduce their applicability to current clinical practice.

Furthermore, significant controversies persist regarding the pathophysiology of MH, particularly in relation to CSF dynamics, hypoxia-induced headache, and neuroinflammation, making it challenging to draw definitive conclusions. Another limitation is the inability to fully evaluate the potential biases in existing studies due to heterogeneous methodologies and study designs. Some paradoxical findings, such as cases of primary headaches induced by VM, remain unexplained and may reflect individual variations rather than a universal physiological response.

Conclusion

The potential efficacy of VM in mitigating MH has gained increasing interest in headache medicine. A deeper understanding of the physiological and molecular mechanisms underlying the impact of VM on migraine relief can enable healthcare practitioners to refine therapeutic strategies for patients who do not have contraindications for this maneuver. This review indicates the beneficial role of VM in MH relief, primarily through its ability to modulate ANS, influence cardiovascular function, regulate cerebral vascular resistance, balance inter-sinus air pressure, and induce the release of endogenous pain modulators. These mechanisms suggest that VM could serve as a non-pharmacological intervention for migraine management. However, further research is needed to establish its clinical efficacy in large-scale clinical

trials across diverse migraine severities, including mild, moderate, and severe cases, as well as to assess its effectiveness during both chronic and acute headache phases. Moreover, future studies should explore its potential role in relieving tension-type headaches, expanding its scope as a viable therapeutic option in headache management.

Conflict of Interests

The authors declare no conflict of interest in this study.

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