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Review

Galvanic Vestibular Stimulation and Its Effects on Sympathetic Nervous System Activation

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Abstract

Cardiovascular modulation in response to movement and gravitational forces can be influenced by vestibular input or peripheral baroreflex mechanisms. Galvanic vestibular stimulation (GVS) is a widely used, noninvasive method for activating neural pathways within the vestibular system, as well as associated pathways such as vestibulo-spinal, oculomotor, and vestibulo-autonomic circuits. Research on vestibulo-autonomic function via GVS has primarily focused on its effects on cardiovascular modulation and sympathetic muscle and nerve activity. However, inconsistencies in GVS application protocols across studies have made it challenging to reach a consensus regarding its effectiveness in modulating the vestibulo-autonomic pathway. Evidence suggests that GVS induces transient autonomic changes by stimulating a neural pathway sensitive to otolith input. This review collates the parameters used in GVS application and examines their effects on autonomic neural pathways by analyzing variations in amplitude, frequency, and electrode montage to understand their impact on autonomic responses, including changes in heart rate (HR), blood pressure (BP), and sympathetic muscle or nerve activity (MSNA). By analyzing stimulation parameters and experimental protocols, we aim to determine their impact on autonomic activation and evaluate their potential for precise autonomic modulation. Finally, based on the evidence generated in populations with neurological disorders and motion sickness, we discuss the potential of GVS as a complementary neuromodulation strategy to treat autonomic dysregulation.

Keywords: galvanic vestibular stimulation; cardiovascular modulation; muscular sympathetic activity; autonomic function; blood pressure; heart rate

1. Introduction

Over the past two decades, galvanic vestibular stimulation (GVS) has been extensively employed in both basic and clinical research to investigate the vestibular system and its associated neural pathways [1]. While most studies have primarily focused on posture and ocular movements, GVS has also been used to explore other vestibular-related systems in both healthy and diseased populations [2,3]. The vestibulo-sympathetic system is no exception, as it plays a critical role in regulating blood flow following head movement to ensure adequate circulation to vital and peripheral organs. Early studies demonstrated c-Fos activation of the lateral tegmental field, nucleus of the solitary tract (NTS), ventrolateral medulla, and the vestibular nucleus of the rostral medulla, during the elicitation of excitatory cardiac reflexes in bradykinin-treated cats [4]. In humans, numerous studies have used GVS as an analog to mechanical stimuli, measuring heart rate (HR), blood pressure (BP), muscle sympathetic nerve activity (MSNA), and skin sympathetic nerve activity (SSNA) for evaluation parameters. However, the complex interplay between movement, acceleration, and cardiovascular response regulated the vestibular apparatus, vestibular nuclei in the brainstem, chemoreceptive centers in the medulla oblongata, the reticular formation, and the hypothalamus, remains insufficiently understood, as do the neural mechanisms underlying disorders that involve vestibular activity such as motion sickness [5,6], orthostatic hypotension [7], and vasovagal syncope [8].

Given its ability to stimulate the vestibular system without inducing fluid shifts that could interfere with baroreceptor function, GVS has emerged as a valuable tool for investigating autonomic responses under controlled experimental conditions. In this sense, this review provides an overview of cardiovascular and MSNA responses, associated to stimulation parameters, such as current intensity and frequency, exploring the possibility that its configuration may activate vestibular pathways associated to the autonomic activity under study.

2. GVS on Cardiovascular Regulation

GVS has been used to study cardiovascular sympathetic modulation in both animal models and human subjects. Research has primarily focused on understanding the effects of gravitational force or vestibular input, using electrical stimulation, on HR and BP. While findings have

been varied, the approach and specific stimulation settings may influence the observed outcome in each case. A considerable number of studies have utilized the anesthetized rat models to determine the effects of GVS on cardiovascular responses [9,10]. A key distinction between animal models and human studies is the electrode type. In animal models, subdermal electrodes are positioned either over the mastoid process in a bilateral bipolar configuration or implanted in the tympanic bulla. This alters the distance between the source of electrical current and the activated neural tissue, affected by the heterogeneity of the various interfaces' impedance [11].

Regarding stimulation frequency, animal model protocols typically use lower values than those applied in human studies. For instance, one study found that, in rats, sinusoidal Galvanic Vestibular Stimulation (sGVS) at 0.008–0.4 Hz and 1–4 mA led to a decrease in HR and BP, with gradual recovery within approximately six minutes [12]. In human subjects, a 1 Hz and $\pm$2 mA bipolar square pulse was applied bilaterally over the mastoid process for more than seven minutes while participants were tilted 30° in a supine position, resulting in a more pronounced BP decrease compared to the control condition [13]. A later study combining the Head-Up Tilt (HUT) test with $\pm$2 mA sGVS at randomly selected frequencies from 0.2 to 10 Hz reported a significant increase in the high-frequency band, suggesting a parasympathetic predominance in the cardiovascular response of subjects experiencing a BP decrease of more than 5 mmHg during the test [14]. Interestingly, studies with animal models and human cases concur that cardiovascular variables decrease as a result of low-frequency GVS. This may be associated with low-frequency activity of the R-R interval electrocardiography power spectrum associated with baroreflex sensitivity, by decreasing HR and suppressing the amplitude of BP response, likely due to parasympathetic activation [15]. Notably, GVS with Gaussian noise did not alter heart rate variability or BP during the HUT test in healthy elderly individuals [16], highlighting the critical role of selecting optimal stimulation parameters to evoke targeted cardiovascular responses.

Another variable of relevance in the reviewed studies is electrode montage. The most commonly used configurations are unilateral, with both electrodes placed in the periauricular region, and bilateral, with the cathode and anode placed on opposite mastoids [17]. Again, studies in both animal models and human experiments have revealed that vestibular stimulation using monolateral/bilateral GVS induces changes in cardiac modulation. A study comparing monolateral and bilateral stimulation in anesthetized rats using 3 mA sGVS at frequencies of 0.025–0.5 Hz demonstrated that both electrode configurations induced HR and BP oscillations at twice the stimulation frequency, being the most effective range of 0.025–0.05 Hz [18].

Gravitational force has emerged as an interesting input variable in experimental protocols that evaluate cardiovascular regulation using GVS, both in animal models and in human subjects. In rodents, it was demonstrated that bilateral biphasic square-wave GVS at 1 Hz and $\pm$0.5 mA significantly attenuated the pressor response to gravitational changes in unanesthetized rats exposed to microgravity and hypergravity conditions [9]. Consistent with these observations, human studies have also reported vascular modulatory effects. During parabolic flight, the application of a biphasic square GVS ($\pm$1–2 mA) $>$1 Hz with a 0–500 ms interphase interval, effectively suppressed the BP response in the participants [19]. These findings underscore the capacity of vestibular stimulation to modulate autonomic cardiovascular responses under changing gravitational states.

To explore the potential interconnections between the vestibular system and cardiovascular modulation, some studies using GVS should be mentioned. For instance, Holstein *et al*. (2012) [20] examined the distribution of c-Fos-positive neurons in rats following 30 minutes of sGVS at 2 mA and 0.025 Hz. This paradigm resulted in significant c-Fos expression within the spinal, medial, and superior vestibular nuclei, as well as the parasolitary nucleus, all of which are known to receive substantial otolithic input. Notably, the highest density of c-Fos-labeled neurons was observed in the parvocellular region of the medial vestibular nucleus, a key area that projects efferent signals to the rostral ventrolateral medulla (RVLM), which in turn modulates preganglionic sympathetic neurons. Given that GVS activates both the otolith organs and the semicircular canals, it is likely that cardiovascular modulation is provoked by fore and lateral movement [21]. However, otolith input alone may not be the only factor influencing changes in HR and BP. As seen with sGVS, pulsed infrared light beam stimulation at 0.05 Hz targeting specifically the anterior and posterior semicircular canals also resulted in a decrease in cardiovascular variables in rats [22]. A complementary study using FluoroGold c-Fos protein and triple-label immunofluorescence after five cycles of 2 mA sGVS at 0.025 Hz identified glutamatergic and GABAergic populations in both the RVLM and caudal ventrolateral medulla (CVLM). Vestibulo-sympathetic glutamate-immunofluorescent neurons were predominantly found in the spinal and medial vestibular nuclei, projecting to the RVLM and CVLM, whereas vestibulo-sympathetic γ-aminobutyric acid (GABA) mediated projections were mainly localized to the CVLM. The co-localization of glutamate-immunoreactive neurons and GABAergic projections within the CVLM may underlie the complex interplay between excitatory and inhibitory pathways of the vestibulo-sympathetic projections that modulate cardiovascular activity in response to head movement [23].

Other studies have focused on the influence of the medial vestibular nucleus (MVN) on cardiac activity through epinephrine release via the vestibulo-sympathetic reflex. While microinjection of a glutamate receptor agonist into the MVL increased BP, the administration of a glutamate

receptor antagonist reduced epinephrine release in response to hypotension induced by sodium nitroprusside. These findings highlight the role of a vestibulo-spinal-adrenal axis modulating a sympathetic response, particularly in epinephrine-mediated cardiovascular modulation aimed at preventing syncope during hypotensive episodes. Consistently, an increased number of c-Fos-positive neurons were observed in the intermediolateral cell column of the spinal cord, at T4–T7 levels, following administration of glutamate receptor agonists, whereas this increment was not present when glutamate receptor antagonists were administered in combination with the hypotension-inducing agent [24]. Another study demonstrated that direct application of GABA (61 ng or 100 µg) or glutamate (2 µg) into the rat posterodorsal amygdala elicited opposing effects on heart rate variability parameters. While GABA enhanced cardiac parasympathetic activity, glutamate provoked a sympathetic behavior [25]. Still, the neural pathways connecting the amygdala with vestibular inputs remain under investigation, together with the possibility of eliciting sympathetic or parasympathetic outputs by modifying GVS parameters (Fig. 1, Ref. [26–28]).

3. GVS-Induced Modulation of HR and BP as a Vaso-Vagal Analogue

Vaso-vagal syncope is typically triggered by factors such as prolonged upright posture, emotional stress, or impaired thermoregulation. These triggers can induce a sudden drop in HR and BP, leading to nausea, dizziness, pallor, and palpitations, and may ultimately result in transient loss of consciousness [29]. Considering that 49.3% of students susceptible to motion sickness were also prone to vaso-vagal syncope [30], the prevalence of this condition appears to be considerably high within certain populations. As previously mentioned, low-frequency sGVS has been employed in animal models to induce HR and BP reductions. Some research groups have proposed that these signs may resemble those observed during vaso-vagal syncope. If the use of sGVS as a method to induce syncope-like symptoms were validated, it could enable the exploration for both therapeutic and pharmacological strategies to counteract such responses. Several strategies have been employed to induce pathological vaso-vagal responses using sGVS. For instance, $<$0.5 Hz at intensities of 1–4 mA has been shown to produce a reduction in HR by 2–3 beats per second and a decrease in BP by 10–20 mmHg in anesthetized rats [12]. In a subsequent study, sGVS at 3 mA and frequencies of 0.025 and 0.1 Hz applied for 5 minutes elicited a vasovagal response, evidenced by the emergence of Mayer waves, analogous to those observed during a $>$70° head-up tilt or 1 g acceleration [31]. Furthermore, cerebral perfusion using a laser Doppler probe, revealed that bilateral transmastoid sGVS (2–4 mA, 0.025–0.5 Hz, 3 minutes) significantly reduced cerebral blood flow in male rats, with effects persisting up to 30 min post-stimulation [32]. An illustrative example of the utility of considering sGVS as a model for vasovagal syncope, and of exploring interventions to mitigate its symptoms, is the use of remote limb ischemic preconditioning (RLIPC). This method involves applying brief, repeated cycles of ischemia and reperfusion to a limb. RLIPC demonstrated to mitigate the sGVS-induced reductions in BP, HR and cerebral blood flow (4 mA, 0.025 Hz, 3 minutes) [33]. The procedure has potential therapeutic approach that could be readily tested in humans. Nausea, a symptom that frequently precedes vasovagal syncope in humans, can be induced after 20–25 minutes of pseudorandom sGVS ($\pm$5 mA) [34]. Similarly, maneuvers or interventions aimed at reducing this symptom can also be systematically tested.

The cardiovascular response to sGVS is so consistent that it has enabled the development of a computational model, based on experimental data from rats exposed to sGVS (1–4 mA, 0.008–0.5 Hz) [35]. The model replicates the role of BP drops in triggering vasovagal responses. It incorporates physiological parameters such as low-frequency vasovagal oscillations, baroreflex sensitivity, response thresholds, system saturation, and target BP levels. The model concludes that a reduction in the desired BP is the primary driver of vasovagal syncope [36]. Moreover, the model can be employed to manipulate these variables in order to explore potential strategies for mitigating the symptoms that culminate in a vasovagal syncope.

4. Effect of Galvanic Vestibular Stimulation on Muscular and Skin Nerve Sympathetic Activity

To investigate the vestibular projections that influence sympathetic reflexes, sGVS in combination with MSNA and SSNA recordings in humans has been used to study the presence of neural pathways that link the vestibular system to autonomic function. Similar to the modulation of BP and HR, research has focused on the sGVS stimulation parameters that elicit the strongest entrainment of MSNA and SSNA. Using this approach, it was found that the most effective modulation-to-stimulus ratio occurred at low frequencies, specifically between 0.05 and 0.5 Hz, from a range of sGVS frequencies 0.05–5 Hz. This effectiveness was quantified through cross-correlation histograms comparing SSNA with the GVS waveform which revealed two distinct peaks. Some have proposed that the two modulation peaks observed in response to sGVS correspond to the positive and negative phases of the sinusoidal stimulus [37]. It has also been suggested that these two peaks in MSNA modulation are independently associated with cardiac and vestibular activity, respectively, and can be distinguished based on the phase relationship between heart beat and sGVS at $\pm$2 mA and 0.5–0.8 Hz [38]. Supporting this hypothesis, MSNA exhibited the lowest modulation index when $\pm$2 mA sGVS was applied at each participant's HR value compared to a frequency sweep ranging from 0.1

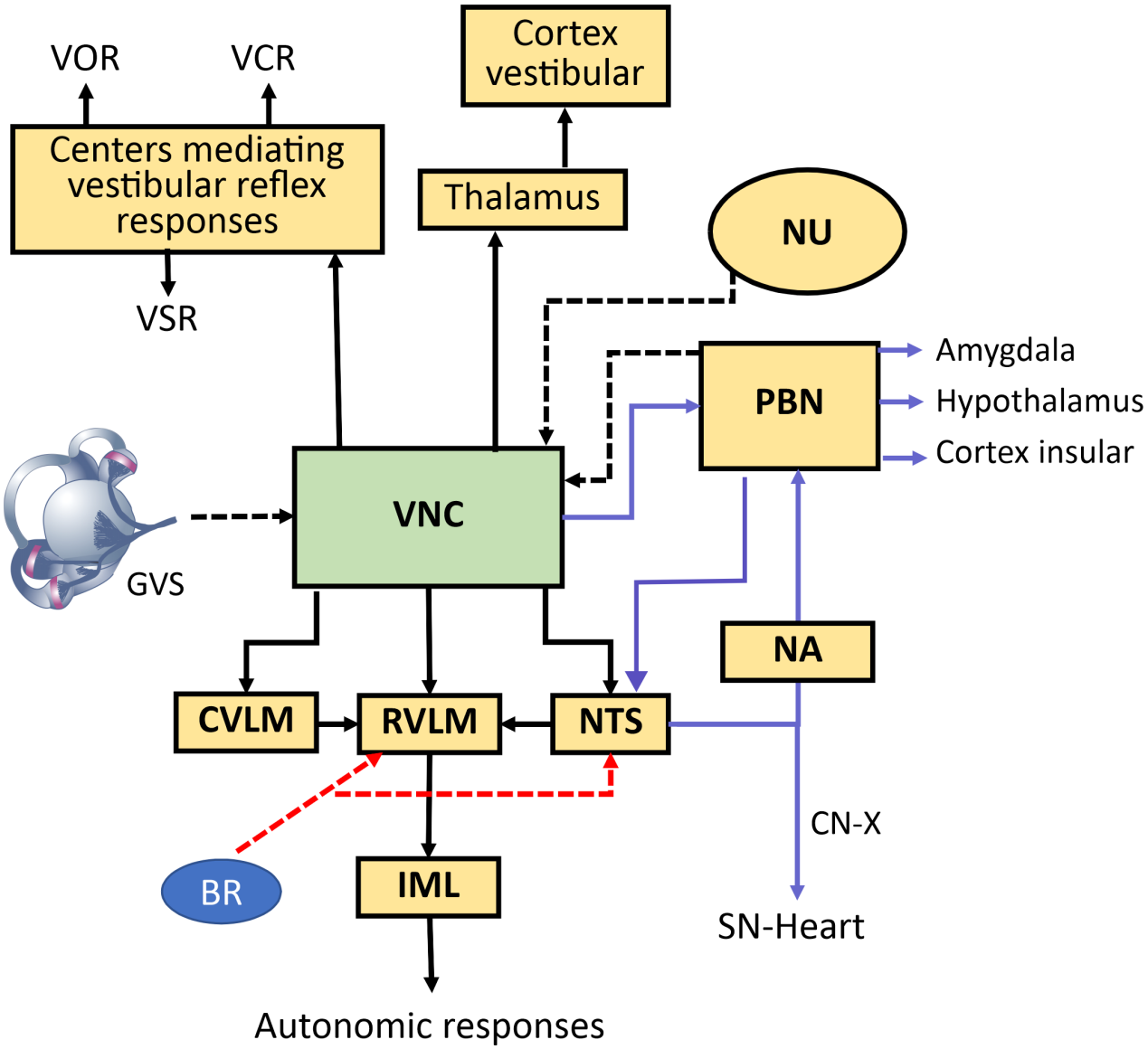

Fig. 1. Neural pathways mediating vestibulo-autonomic responses. Schematic diagram illustrating the central pathways connecting vestibular inputs to autonomic nervous system outputs. Vestibular information from the vestibular nuclei complex (VNC) influences classic vestibulo-ocular (VOR), vestibulocollic reflexes (VCR), and vestibulospinal reflexes (VSR). Simultaneously, the VNC projects to autonomic control centers, primarily via two streams (dashed lines input to vestibular nuclei (VN), solid lines efferent paths from VNC) : (1) a primary reflex pathway to the nucleus tractus solitarius (NTS), rostral ventrolateral medulla (RVLM), and caudal ventrolateral medulla (CVLM) to integrate with baroreceptor (BR) signals and modulate blood pressure and heart rate via the intermediolateral nucleus (IML), influencing sympathetic nerve activity (skin sympathetic nerve activity (SSNA), muscle sympathetic nerve activity(MSNA)) [26–28]; and (2) a parallel pathway (purple lines) via the parabrachial nucleus (PBN) and NTS to the nucleus ambiguous (NA) for parasympathetic control of the sinus node, and to limbic (Amygdala) and homeostatic (Hypothalamus, Insular Cortex) regions. The red arrows show the input from the BRs to the RVLM and NTS. Vestibular signals also reach cortical vestibular areas via the thalamus, contributing to spatial awareness. The solid lines show the main output from vestibular nucleus and other interconnections related to vestibular autonomic responses. VNC, vestibular nuclei complex; VOR, vestibulo-ocular reflex; VCR, vestibulocolic reflex; VSR, vestibulospinal; VN, vestibular nuclei; NTS, nucleus tractus solitarius; RVLM, rostral ventrolateral medulla; CVLM, caudal ventrolateral medulla; BR, baroreceptor; IML, intermediolateral nucleus; SSNA, skin sympathetic nerve activity; MSNA, muscle sympathetic nerve activity; PBN, parabrachial nucleus; NA, nucleus ambiguous; NU, nodulus-uvula; CN-X Cranial nerve X; GVS, galvanic vestibular stim-ulation.

to 0.6 Hz. The reduction in MSNA modulation near the HR frequency suggests that, at this specific frequency, the baroreflex response may interfere with the activation of the vestibulosympathetic pathway, ultimately suppressing its input [39]. Interestingly, with low-frequency stimulation (0.08, 0.13, and 0.18 Hz), the primary modulation peak remains consistent, while the secondary peak progressively diminishes as the sGVS frequency increases [40]. Furthermore, cross-correlation histograms between the electrocardiogram and SSNA, revealed significantly elevated modulation indices at frequencies near HR values, specifically at 0.8, 1.1, 1.4, 1.7, and 2 Hz [41]. This evidence suggests that MSNA and SSNA correspond to distinct components of the sympathetic nervous system, with differing neural origins. The selective response to sGVS frequency range indicates separate central projections, as noted by the differential modulation of sympathetic outflow to muscle and skin activation. The distinction becomes particularly relevant when examining the simultaneous recording of MSNA and SSNA during $\pm$2 mA sGVS at 0.08–0.18 Hz in subjects who experienced nausea. The analysis revealed a significant increase in the modulation index of SSNA, whereas MSNA remained unaffected, underscoring the differential modulation of these two sympathetic outflows [42].

Given that GVS is considered analogous to mechanical stimulation of the vestibular system, it has served as an input that does not induce fluid shifts and, consequently, does not interfere with the baroreceptor activity involved in the orthostatic reflex [43]. This similarity has been corroborated by the MSNA response to sGVS, which was found to be comparable to that elicited by sinusoidal linear acceleration of $\pm$4 mG at 0.08 Hz along the rostro-caudal and medio-lateral axes. The previously described two-peak modulation pattern was observed in 7 out of 11 human subjects, suggesting that both linear acceleration and sGVS activate the vestibulo-sympathetic pathway by stimulating utricular and saccular afferents [44]. Equally, two-peak responses were observed in SSNA with sGVS at $\pm$2 mA and frequencies of 0.08, 0.13, and 0.18 Hz, as well as during linear acceleration delivered by a motorized chair moving along the anteroposterior and mediolateral axes at 0.08 Hz. In this study, the biphasic peak activity was attributed to the directional deflection of utricular hair cells, with each peak corresponding to movement-induced deflection in opposite directions [45]. Moreover, the combination of both inputs, gravitational gradient tilt and sGVS, provoked the highest modulation index recorded at a 75° tilt from the horizontal on a motorized table during stimulation at $\pm$2 mA and 1.4 Hz, compared to the supine position [46].

The differential responses of MSNA and SSNA to sGVS suggest that it is possible to selectively activate distinct sympathetic pathways by adjusting stimulation parameters. It has been proposed that MSNA is primarily associated with peripheral vasoconstriction mediated by otolith input, whereas SSNA is more closely linked to thermoregulatory processes and blood pressure modulation [47]. Although the origins of SSNA remains unclear, evidence indicates that its occurrence is not sensible to body temperature [48]. Both sympathetic outflows are sensitive to vestibular input, presumably via projections from the otolith organs to the RVLM for MSNA and to the medullary raphe for SSNA, respectively [40]. Representative studies that alter autonomic function, provoking a sympathetic or parasympathetic response by activating neural pathways using GVS, are shown in Table 1 (Ref. [10,12,14,16,18,31,32,34,37,39,42,49–53]).

In this regard, the accumulated body of evidence on the sympathetic activation, for instance, linking abdominal obesity, elevated BP, and increased MSNA [54], must be reconsidered in light of incorporating GVS. The combined experimental approach of GVS and MSNA in obese populations, may uncover relevant strategies to enhance body fat loss, profiting from the potential of MSNA to identify individuals who may derive the greatest benefit from weight loss interventions under hypocaloric dietary conditions [55].

5. Applications of GVS in the Study of Vestibulo-Autonomic Interactions

A wide range of neurological pathologies, primarily motor and cardiovascular, have been evaluated during and after GVS, including vestibular and auditory disorders, Parkinson's disease, ischemic lesions, myelopathies, and mental impairments [56]. GVS has also proven valuable in the study of symptoms related to motion sickness [57]. For instance, sGVS at $\pm$8 mA and 0.08 Hz has been employed to study physiological parameters regulated by the autonomic nervous system (ANS), such as HR, BP, skin blood flow, and respiratory rate, comparing them between individuals who reported nausea during stimulation and those who did not. The differences observed in these variables between the two groups supported the utility of sGVS in assessing susceptibility to motion sickness symptomatology [49]. Other related symptomatology using animal models have demonstrated that sGVS can induce a range of autonomic and behavioral responses such as emesis, salivation, licking, retching, panting, and head rolling. Anatomical structures involved in motion sickness were characterized through c-Fos expression using 5 Hz, 3–5 V for 90 minutes sGVS in a feline model, finding five statistically independent neural networks, identified via principal component analysis [58]. In reference to the potential therapeutic applications of GVS, it has been demonstrated that sGVS delivered at 3.5 mA and 1 kHz can enhance movement tolerance during rotary platform exposure. This effect was evaluated using physiological and behavioral indicators, including pupil diameter and gaze stabilization, as objective measures of motion sickness susceptibility [59]. Interestingly, GVS has been applied to either mitigate or exacerbate motion sickness symptoms, by employing a sensory

Table 1. Representative studies on the effects of GVS on autonomic functions and selected stimulation parameters.

Authors	Subject type	Stimulation parameters	Electrode configuration	Autonomic effect
Cohen *et al.* (2011) [12]	Animal model (Long-Evans rat)	1–4 mA, 0.008–0.5 Hz, 15–30 min	Binaural	Instant decrease in HR and BP followed by increment
Cohen *et al.* (2013) [31]	Animal model (Long-Evans rat)	3 mA, 0.025–1 Hz, 15 min	Binaural	Induced vasovagal response
Yakushin *et al.* (2014) [18]	Animal model (Long-Evans rat)	3 mA, 0.025–0.5 Hz, 1–5 min	Binaural and monaural	Monaural and binaural stimulation induced vasovagal oscillations
Abe *et al.* (2009) [10]	Animal model (S-D rat)	±10 μA, 70 min	Binaural	GVS inhibited BP and HR hypergravity response
Tanaka *et al.* (2014) [14]	Human (healthy)	2 mA, 0.2–10 Hz, 250 s	Binaural	R-R electrocardiographic interval increased
Matsugi *et al.* (2021) [16]	Human (healthy elder)	±0.5 mA, 0.1–640 Hz, 660 s	Binaural	Noisy GVS has no effect on BP and HR variability
McBride *et al.* (2016) [32]	Animal model (Long-Evans rat)	2–4 mA, 0.025–0.5 Hz, 3 min	Binaural	Hypotension, bradycardia, decreased cerebral blood flow
Quinn *et al.* (2015) [34]	Human (healthy young)	<±5 mA, 0.16–0.61 Hz, 25 min	Binaural	GVS provokes placebo-induced nausea
Singh *et al.* (2019) [37]	Human (healthy young)	0.05–5.0 Hz, ±2 mA, 500 cycles	Binaural	Modulation is of MSNA is highest at 0.05 Hz and lowest at 5 Hz
James & Macefield (2010) [39]	Human (healthy)	±2 mA, 0.1–0.6 Hz, 200 cycles	Binaural	MSNA was weakest at sGVS cardiac frequency
Klingberg *et al.* (2015) [42]	Human (healthy)	±2 mA, 0.08–0.18 Hz, 200 cycles	Binaural	SSNA was significantly larger in subjects experiencing nausea. MSNA did not increase
Javaid *et al.* (2019) [49]	Human (healthy)	±2 mA, 0.08 Hz, 21 min	Binaural	SSNA, BP, HR, and respiratory rate changed distinctively in subjects experiencing nausea due to sGVS
Allred *et al.* (2025) [50]	Human (healthy elder)	0.01–4.0 mA, 0.275–0.325 Hz, 40 min	Binaural	Motion sickness was increased by Detrimental GVS and reduced by Beneficial GVS
Yamamoto *et al.* (2005) [51]	Human (PD)	0.33 ± 0.2 mA, 0.01–2 Hz, 24 h	Binaural	Noisy GVS increased short time scales HR variability
Lotfi *et al.* (2021) [52]	Human (DM2)	<threshold, 1–30 Hz, 20 min	Binaural	GVS reduced BP and Body Mass Index
Viirre *et al.* (2025) [53]	Human (overweight and obesity)	1 mA, 0.5 Hz, 60 min	Binaural	Visceral adipose tissue loss increased in the active group

DM2, Diabetes mellitus type 2; PD, Parkinson's disease; R-R, R wave–R wave interval; S-D, Sprague-Dawley; sGVS, sinusoidal Galvanic Vestibular Stimulation.

conflict computational model to determine the properties of the stimulation signal. The opposite effects provoked by GVS highlight the critical importance of selecting appropriate stimulation parameters to induce either beneficial or detrimental responses in an individual's motion sickness susceptibility [50]. Another potential application involves modulating cardiovascular autonomic function in patients with impaired heart rate variability due to sympathetic-parasympathetic imbalance. This is supported by findings in patients with autonomic failure caused by multiple system atrophy, where noisy GVS ranging from 0.01 to 2.0 Hz was shown to enhance heart rate variability towards healthier dynamics, presumably through parasympathetic activation [51]. A similar rationale of using GVS as a potential treatment aid, guided a study that investigated whether GVS could influence blood glucose levels in patients with type 2 diabetes mellitus. The study reported a statistically significant reduction in fasting blood glucose levels in the group that received subthreshold pseudorandom GVS at 1–30 Hz for 20 minutes, three times per week on non-consecutive days, when comparing baseline levels to those measured after 12 weeks of intervention, with no significant changes observed in the sham or no-treatment control groups [52]. Moreover, a recent study showed the potential of GVS to influence metabolic activity in complex conditions such as obesity. The study investigated the effects of GVS in conjunction with a hypocaloric diet over a one-month period. Participants were divided into two groups: both received the same dietary regimen, but only one group underwent daily GVS sessions (1 hour per day) using the Modius Lean device. The results revealed a significant reduction in visceral adipose tissue in the GVS group compared to the sham group. It was speculated that this reduction may be attributed to increased hypothalamic activity triggered by vestibular input, which in turn leads to decreased fat storage via the vestibulo-hypothalamic pathway, particularly through the action of the medial vestibular nucleus. The results suggest that GVS could represent a promising adjuvant in obesity management [53].

Another example of a potential application focuses on the modern variant of motion sickness induced by prolonged exposure to video games and virtual reality (VR), cybersickness, GVS has gained attention as a method for both quantifying and attempting to mitigate the symptoms of this technology-induced condition. Typical symptoms of cybersickness such as nausea, headache, dizziness, or salivation, were assessed with and without GVS (sinusoidal, 10 Hz, $\pm$3.7 mA) to evaluate the influence of vestibular input on their intensity. Only cold sweating, an early sign of syncope, was significantly increased with GVS compared to the control condition. No other consistent differences were observed in as measured by the Graybiel Motion Sickness Questionnaire [60]. Another study demonstrated a reduction in cybersickness scores during and immediately after exposure to an intense VR self-motion environment when noisy GVS was applied. The observed improvement was attributed to three potential mechanisms: (1) masking of vestibular input with noisy GVS, facilitating sensory reweighting in favor of visual cues; (2) sensory enhancement via noisy stimulation, consistent with the stochastic resonance phenomenon in the nervous system; and (3) increased sense of presence in the VR environment, potentially augmented by GVS [61]. The effects of GVS on autonomic output have also been compared with other analogous forms of stimulation. One such variant, galvanic cutaneous stimulation, with electrodes placed over the mastoid insertion of the sternocleidomastoid muscle, showed to reduce symptoms of simulator adaptation syndrome, a form of motion sickness induced by virtual simulation, in 28.5% of participants, as measured by the Simulator Sickness Questionnaire [62]. Furthermore, the efficacy of noisy GVS in reducing simulation sickness was compared to that of noisy bone-conducted vibration during a moderate or intense virtual reality task that required to advance through a game or explore the VR environment, respectively. Both, GVS and bone-conducted vibration presumably acted by masking vestibular input, thereby modulating sensory integration and reducing cybersickness symptoms [63]. It is possible that the sensory conflict elicited by GVS, which in some individuals leads to nausea, emesis, and other motion sickness-related symptoms, may diminish in intensity with repeated exposure. This potential desensitization effect has been explored in spaceflight training, where 20 minutes of sGVS at 3.5–5 mA was applied to induce motion sickness symptoms during flight simulation. Given that only 16.7% of participants experienced severe nausea at a stimulation intensity of 5 mA, GVS proved to be a tolerable and viable tool for integration into demanding operational tasks, such as spacecraft docking procedures and long-duration extravehicular activities [64]. Focusing on gastric motility in motion sickness induced by GVS, one study reported a reduction in sympathetic activity following 20 minutes of continuous GVS at 2.5 mA. This reduction was associated with a decrease in tachygastria compared to a control group receiving sham stimulation during a flight simulation task. These findings suggest that GVS may promote normogastric activity, thereby alleviating motion sickness symptoms such as nausea, vomiting, dizziness, and nystagmus [65]. Interestingly, tachygastria induced by caloric vestibular stimulation in a group of 20 young volunteers was counteracted by GVS using a 1 ms squared pulse at 100 Hz and 1–3 mA, suggesting a modulatory effect of GVS on gastric activity [66]. This result implies that the neural pathways activated by caloric vestibular stimulation and galvanic vestibular stimulation may not be identical, potentially engaging distinct vestibulo-autonomic circuits. However, in Parkinson's disease, both caloric and galvanic vestibular stimulation have been employed to alleviate autonomic dysfunction symptoms such as urinary incontinence, abnormal sweating, impaired digestion, ex-

cessive salivation, and sexual dysfunction [67]. These findings highlight the relevance of understanding not only the interaction between vestibular projections to the autonomic control centers, but also the effects of the diverse stimulation approaches (e.g., caloric or electrical), which may define the type of intervention according to the intended autonomic effect.

6. Conclusion

Although GVS and the modulation of autonomic variables remains limited, with most studies focusing on HR, BP, MSNA and SSNA outcomes, its effect on autonomic variables is not negligible. A wide range of stimulation parameters vary considerably among studies: lower frequencies are typically employed to influence cardiovascular variables, while higher frequencies are more effective in modulating muscular and cutaneous sympathetic activity. However, most studies have analyzed either cardiovascular variables or those related to sympathetic and cutaneous muscular activity, but not both simultaneously, making it difficult to determine whether sinusoidal sGVS predominantly enhances sympathetic or parasympathetic activity. Nonetheless, continued exploration of GVS across a broader range of autonomic functions could lead to the development of targeted therapeutic protocols. There is growing evidence that GVS can modulate various autonomic functions and, in certain pathological conditions, may contribute to restoring physiological parameters toward values considered within a healthy range. Among the potential applications, the use of GVS has gained relevance, preventing nausea and motion sickness in sensory-conflict scenarios such as VR video games, normalizing blood glucose levels in individuals with type 2 diabetes mellitus, and promoting abdominal fat loss in individuals with obesity.

It is important to note that GVS is unlikely to produce responses analogous to natural vestibular stimulation. The low specificity of the electrical field may result in heterogeneous activation of neural pathways associated with the otolith organs and semicircular canals, in combination to other neural structures such as the auricular branch of the vagus nerve and cutaneous receptors. This could lead to autonomic responses with no natural equivalent. Moreover, GVS allows for the use of frequency oscillations that would be unachievable with mechanical stimuli, enabling selective activation of structures near the periauricular region without engaging other sensory systems, such as baroreceptors, typically stimulated by physical movement.

In summary, the stimulation parameters of GVS, such as frequency, current intensity, and signal waveform, may play a critical role in determining sympathetic or parasympathetic activation, producing varied effects across autonomic end-organs. Additionally, the potential for long-term therapeutic use of GVS to induce neuroplastic changes in specific autonomic functions remains a promising area for future research.

Abbreviations

ANS, autonomic nervous system; BP, blood pressure; BR, Baroreceptor; CVLM, caudal ventrolateral medulla; DM2, type 2 diabetes mellitus; GABA, γ-aminobutyric acid; GVS, galvanic vestibular stimulation; HR, heart rate; HUT, Head-Up Tilt; IML, intermediate lateral column; MSNA, muscle sympathetic nerve activity; MVN, Medial Vestibular Nucleus; NA, Nucleus ambiguous; NTS, nucleus tractus solitarius; NU, Nodulus-Uvula; PBN, Parabrachial nuclei; PD, Parkinson's disease; RLIPC, remote limb ischemic preconditioning; RVLM, rostral ventrolateral medulla; S-D, Sprague-Dawley; sGVS, sinusoidal Galvanic Vestibular Stimulation, SN, Sinus node; SSNA, skin sympathetic nerve activity; VCR, Vestibulocolic reflex; VNC, Vestibular nuclei complex; VOR, Vestibulo-ocular reflex; VER, Vestibulospinal reflex; VR, virtual reality.

Author Contributions

AP and ES conceived and wrote the manuscript. Both authors contributed to editorial changes in the manuscript. Both authors read and approved the final manuscript. Both authors have participated sufficiently in the work and agreed to be accountable for all aspects of the work.

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Conflict of Interest

The authors declare no conflict of interest.

Declaration of AI and AI-Assisted Technologies in the Writing Process

During the preparation of this work the authors used Microsoft Copilot in order to check spelling and grammar. After using this tool, the authors reviewed and edited the content as needed and takes full responsibility for the content of the publication.

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