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Vasovagal Syncope Following Pulsed Radiofrequency of Cervical Dorsal Root Ganglia and Occipital Nerves in a Patient with Chronic Cervical Radiculopathy and Occipital Neuralgia: A Case Report

Kurnia Hendra Wijaya[1*], Buyung Hartiyo Laksono[2]

[1]Resident, Department of Anesthesiology and Intensive Care, Faculty of Medicine, Universitas Brawijaya/Dr. Saiful Anwar Regional General Hospital, Malang, Indonesia

[2]Staff, Department of Anesthesiology and Intensive Care, Faculty of Medicine, Universitas Brawijaya/Dr. Saiful Anwar Regional General Hospital, Malang, Indonesia

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***Corresponding author:**
Kurnia Hendra Wijaya

E-mail address:
wijayakurniahendra@gmail.com

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A B S T R A C T

Introduction: Pulsed radiofrequency is a minimally invasive, non-destructive neuromodulation technique used to manage chronic cervical radicular pain and occipital neuralgia. It is generally considered safe, with common adverse events limited to mild transient dysaesthesia or local discomfort. Vasovagal syncope following pulsed radiofrequency of the cervical dorsal root ganglia and occipital nerves has not been well documented in the anaesthesia and pain medicine literature. **Case presentation:** A 41-year-old woman with chronic cervical radiculopathy attributable to C5–C6 herniated disc disease, tension-type headache with pericranial tenderness, a history of cluster headache, chronic spontaneous vertigo, and newly diagnosed hypertension underwent bilateral pulsed radiofrequency of the lesser and greater occipital nerves and of the C3 and C4 dorsal root ganglia under fluoroscopic guidance. The procedure was performed under light sedation with intravenous propofol and midazolam and completed without immediate complication. Approximately 20 hours later, the patient developed an acute decrease in consciousness with a nadir Glasgow Coma Scale of 12 and a heart-rate profile consistent with a reflex vasovagal event. Gradual spontaneous recovery of consciousness was documented over seven hours, reaching a Glasgow Coma Scale of 15 without any neurological deficit. Pre- and post-procedural symptom comparison showed clear improvement in cervical paraesthesia, vertigo, tinnitus, and cluster-type headache, while tension-type headache persisted at a similar intensity. **Conclusion:** Vasovagal syncope is a rare but clinically relevant adverse event after pulsed radiofrequency of the cervical dorsal root ganglia and occipital nerves. The likely pathophysiology involves afferent stimulation of the trigeminocervical complex and activation of the Bezold–Jarisch reflex in a susceptible patient. Multimodal monitoring, adequate hydration, careful sedation titration, and structured post-procedural observation are recommended to anticipate and manage this complication.

1. Introduction

Chronic pain is the third most prevalent health problem in adult populations and a leading reason for outpatient referral to multidisciplinary specialty clinics.[1] It affects physical function, sleep, emotional wellbeing, and productivity, and is frequently under-recognised and under-treated, with published estimates suggesting that up to half of patients with

chronic pain receive suboptimal analgesia.[2] Cervical radiculopathy is among the most common neuropathic pain syndromes of the upper extremity, with an approximate annual incidence of 83 cases per 100 000 population; it causes radiating arm pain, sensory disturbance, and functional impairment that is often refractory to first-line pharmacological therapy alone.[3] When conservative strategies fail, a range of minimally invasive interventions can be offered, including transforaminal or interlaminar epidural steroid injection, pulsed radiofrequency of the dorsal root ganglion, and, in selected refractory cases, posterior cervical decompression using endoscopic or open techniques.[4]

The mechanical substrate of cervical radiculopathy is typically degenerative disc disease, with endplate changes of the Modic classification and disc degeneration of the Pfirrmann grading system, sometimes accompanied by foraminal narrowing or segmental stenosis. Progressive disc pathology and facet arthropathy contribute both to local axial pain and to radicular symptoms by producing mechanical compression and a secondary chemical-inflammatory milieu around the nerve root; similar mechanisms drive lumbar disc disorders.[5] Occipital neuralgia is a distinct condition, defined by sharp, electric-shock-like pain within the dermatomal territories of the greater and lesser occipital nerves, and it is frequently associated with chronic cervical pathology through the trigeminocervical complex.

Pulsed radiofrequency (PRF) has emerged as an important neuromodulatory intervention in chronic cervical pain and in occipital neuralgia. Unlike conventional continuous radiofrequency ablation, PRF delivers high-voltage radiofrequency energy in short bursts with intervening cooling periods, keeping the electrode tip temperature at or below 42°C so that neurodestruction is avoided. The landmark double-blind, sham-controlled trial by Van Zundert and colleagues demonstrated clinically meaningful improvement in pain and function at three months when PRF was applied adjacent to the cervical dorsal root ganglion, without significant procedural morbidity.[6] Subsequent reviews have elaborated the mechanism of PRF, including modulation of glutamatergic transmission, upregulation of inhibitory neurotransmitters, and alterations in microglial activation.[7] Preclinical work further suggests epigenetic modulation of pain-related gene expression — including histone acetylation and restoration of K-Cl cotransporter 2 function — as a plausible durable mechanism.[8] PRF may additionally reverse maladaptive plasticity in central nociceptive pathways that propagate chronic neuropathic pain.[9]

Despite its broad adoption, uncertainty remains about safety. National survey data from specialist pain practices confirm that PRF is considered a cornerstone intervention, although practice patterns vary, with some specialists preferring transforaminal steroid injection first and only escalating to PRF when steroid response is incomplete.[10] Reports of adverse events have largely focused on transient dysaesthesia, local pain, and rare cases of ataxia after technical deviation. Vasovagal syncope after cervical or occipital PRF has not been systematically documented in the available literature, despite the proximity of the targets to cranial nerves and autonomic afferents that could plausibly trigger a reflex response. The aim of this report is therefore to describe an episode of acute decrease in consciousness consistent with vasovagal syncope that developed in the 24 hours after bilateral occipital-nerve and cervical dorsal root ganglion PRF, and to situate this event within the existing safety literature so that anaesthetists performing interventional pain procedures may anticipate, monitor, and manage a comparable complication in the future.

2. Case Presentation

A 41-year-old woman presented to the chronic pain clinic with a six-month history of right-dominant neck and occipital pain. The pain was described as throbbing and movement-related, radiating from the cervical spine to the occipital region, with a baseline numerical rating score of 3–4 and flares to 5–6. She also reported a tension-type headache since November 2023 that was described as a band-like sensation from the nape to the occiput, and separate episodes of severe unilateral right periorbital pain of approximately three hours' duration, occurring once per month, accompanied by right conjunctival injection, lacrimation, and nasal

congestion, consistent with cluster-type headache phenotype. Vertigo with right-sided tinnitus and mild right hearing loss had been present since May 2024, and the patient had been newly diagnosed with arterial hypertension in October 2024, for which she received amlodipine 10 mg daily. Other current medications included betahistine, sodium diclofenac, amitriptyline, and low-dose diazepam. There was no personal history of diabetes, cardiac disease, or previous surgery, and no relevant family history. The patient worked at a beauty salon and managed a small catering business. Demographic and baseline clinical parameters are summarised in Table 1.

Table 1. Demographic characteristics, clinical history, medications, and baseline vital signs at the chronic pain clinic.

Parameter	Value
Age/Gender	41 years/Female
Body weight/Height/BMI	50 kg/155 cm/22.9 kg/m²
Chief complaint	Right-dominant neck and occipital pain for 6 months
Baseline pain (NRS)	3–4 at rest, 5–6 on flare
Coexisting headache	Tension-type headache with pericranial tenderness; history of cluster-type episodes
Vertigo/Tinnitus/Hearing	Present since May 2024; right tinnitus; mild right hearing loss
Comorbidity	Newly diagnosed arterial hypertension (Oct 2024)
Medications	Amlodipine 10 mg od; betahistine 3×/day; sodium diclofenac 2×/day; amitriptyline 25 mg; diazepam 5 mg prn
Vital signs on admission	BP 163/80 mmHg; HR 80 bpm; RR 16/min; SpO_2 98% RA; T 36.5°C
Glasgow Coma Scale	E4 V5 M6 = 15
Provocative tests	Lhermitte +/+; Spurling +/+; Pericranial tenderness +; Valsalva −

Physical examination on the day of admission showed a well-oriented woman in moderate discomfort, Glasgow Coma Scale 15, blood pressure 163/80 mmHg, heart rate 80 beats per minute, respiratory rate 16 breaths per minute, peripheral oxygen saturation 98% in room air, temperature 36.5°C, body weight 50 kg, height 155 cm, and body mass index 22.9 kg/m². Head and neck examination revealed no conjunctival pallor, scleral icterus, or cervical lymphadenopathy. The chest was symmetrical with vesicular breath sounds and no adventitious sounds; cardiac auscultation demonstrated regular heart sounds without murmurs. The abdomen was soft and non-distended. Extremities were warm and well perfused with capillary refill under two seconds. Comprehensive neurological examination showed intact higher cortical function and cranial nerves I through XII, with the exception of cranial nerve VIII findings of right-sided tinnitus and mild right hearing loss. Motor power was five out of five in all extremities, with pain reported during myotomal testing of C5 to T1. Deep-tendon reflexes were symmetrical and normoactive. No pathological reflexes were elicited. Provocative manoeuvres were positive bilaterally for Lhermitte and Spurling tests and for pericranial tenderness; Valsalva was negative.

Magnetic resonance imaging of the cervical spine performed on 4th November 2024 demonstrated multilevel spinal canal stenosis with severe narrowing at C5–C6 and mild narrowing at C4–C5 due to bulging discs, mild foraminal neural stenosis at C5–C6 bilaterally, Modic type II endplate changes, Pfirrmann grade III disc degeneration, and a straightened cervical curvature consistent with chronic muscular spasm. These findings, together with the clinical examination, supported a working diagnosis of chronic cervical radiculopathy at C3–C6 secondary to cervical herniated disc disease, coexistent tension-type headache with pericranial tenderness, a prior diagnosis of episodic cluster headache, chronic spontaneous vertigo with suspicion of Ménière's disease, and occipital neuralgia. The imaging findings are summarised in Table 2.

Table 2. Magnetic-resonance imaging findings of the cervical spine performed on 4th November 2024.

MRI parameter	Finding
Spinal canal	Multilevel stenosis — severe C5–C6, mild C4–C5 from bulging disc
Neural foramen	Mild foraminal neural stenosis at C5–C6 bilaterally
Endplate	Degeneration — Modic type II
Disc	Degeneration — Pfirrmann grade III
Cervical curvature	Straight cervicalis (loss of lordosis)
Working diagnosis	Chronic radiculopathy C3–C6 due to cervical herniated disc disease

The care team offered the patient bilateral pulsed radiofrequency of the lesser and greater occipital nerves combined with pulsed radiofrequency of the dorsal root ganglia at C3 and C4. The procedure was performed on 19th November 2024 in the operating theatre under full American Society of Anesthesiologists standard monitoring including electrocardiography, non-invasive blood pressure measurement, pulse oximetry, and continuous verbal contact. Light intravenous sedation was achieved with propofol 0.5 mg/kg and midazolam 0.01 mg/kg to maintain responsiveness during sensory stimulation. Skin infiltration with 2% lidocaine preceded insertion of a 22-gauge, 10 cm radiofrequency electrode with a 10 mm active tip under fluoroscopic C-arm guidance. Electrode impedance was verified within the 300–500 Ω range. Sensory stimulation at 50 Hz reproduced a tingling sensation in the appropriate dermatomes at voltages below 0.8 V, confirming correct position. After dermatomal mapping, 0.5–1 mL of 1% lidocaine was injected through the electrode, and PRF was delivered as two 120-second cycles at 2 Hz and 45 V with tip temperature limited to 42°C. The same protocol was performed at each of the four target sites. The procedure was completed without immediate complication; the patient was monitored in the post-anaesthesia care unit for one hour and transferred to the ward in stable condition. The procedural parameters and the pre- and post-intervention symptom evolution are summarised in Table 3.

Table 3. Pulsed radiofrequency procedure parameters and pre- vs. post-intervention symptom profile (20th November 2024).

Item	Detail
Guidance	Fluoroscopic C-arm
Sedation	IV propofol 0.5 mg/kg + midazolam 0.01 mg/kg
Skin infiltration	2% lidocaine
Electrode	22-gauge, 10 cm, 10 mm active tip
Impedance verified	300–500 Ω
Sensory stim 50 Hz	< 0.8 V — dermatomal tingling
Intraneural injection	0.5–1 mL 1% lidocaine
PRF delivery	Two 120-s cycles, 2 Hz, 45 V, tip ≤ 42°C
Target sites	Lesser occipital N. (bilateral); greater occipital N. (bilateral); DRG C3 and C4
Cervical radicular pain (NRS 5–6)	Improved (non-radicular local pain 5–6, resolved in 48 h)
Cervical paraesthesia/hypoaesthesia C3–C6	Improved
Tension-type headache (NRS 4–5)	Unchanged
Cluster-type episodes	Resolved
Vertigo/tinnitus/hearing	Improved
Lhermitte sign	+/+ → −/−
Spurling sign	+/+ → +/+
Pericranial tenderness	+ → +

***** NRS, numerical rating scale for pain intensity (0–10). DRG, dorsal root ganglion. † Post-procedural comparison recorded on 20th November 2024.

Approximately 20 hours after the PRF procedure, at 16:00 on 20th November 2024, the nursing team observed a sudden decrease in the patient's level of consciousness. The Glasgow Coma Scale was recorded at E3V3M6 (total 12), blood pressure at 102/62 mmHg, heart rate 58 beats per minute, respiratory rate 14 breaths per minute, and peripheral oxygen saturation 96% on room air. There was no focal neurological deficit, no tongue biting, no urinary incontinence, and no post-event confusion. Serial neurological assessments documented gradual spontaneous improvement: E3V4M6 (GCS 13) within the first hour, E3V5M6 (GCS 14) by 23:00, and E4V5M6 (GCS 15) by the following morning, with return of pre-event vital signs. The temporal evolution of the Glasgow Coma Scale is plotted in Figure 1. Neurological examination on full recovery showed no new deficit, no cranial-nerve asymmetry, and no evidence of cerebellar dysfunction. Cranial computed tomography was performed to exclude an intracerebral event; the scan was unremarkable. An electrocardiogram at the time of recovery was in sinus rhythm without ischaemic changes. Laboratory investigations including complete blood count, electrolytes, and random plasma glucose were within normal limits.

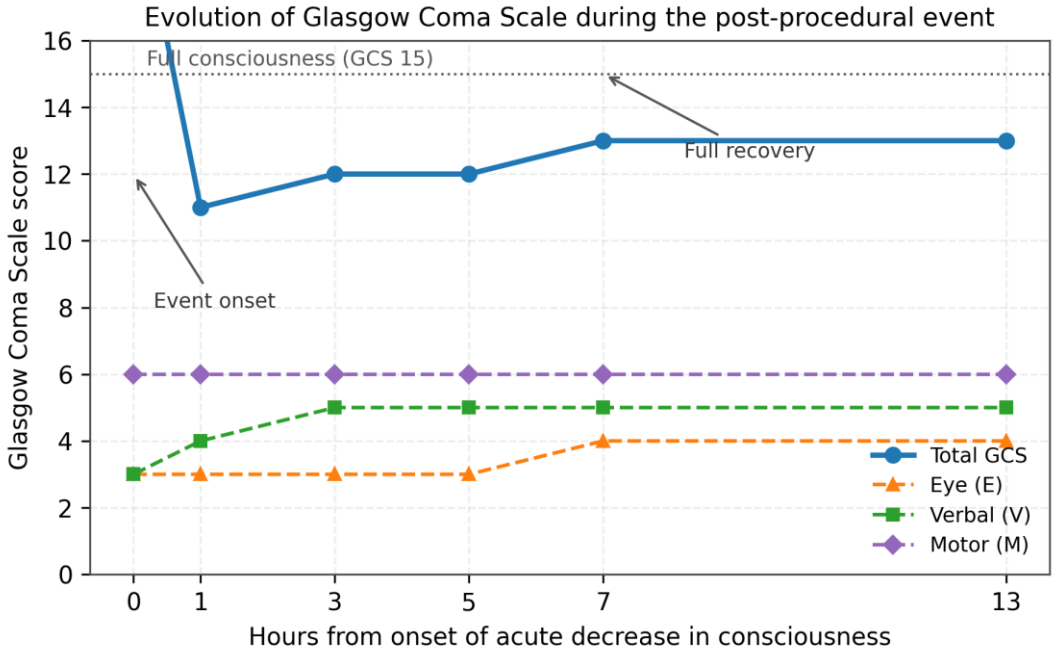

Figure 1. Evolution of the Glasgow Coma Scale (GCS) during the 7–15 hours after the suspected post-procedural reflex syncope event. Individual eye (E), verbal (V), and motor (M) sub-scores are shown; the patient recovered to full consciousness by the morning after admission without any neurological sequelae.

The event was classified as a reflex vasovagal syncope in accordance with published diagnostic frameworks. The differential diagnosis included orthostatic hypotension in the context of amlodipine therapy, cardiac arrhythmia, new-onset cerebrovascular event, procedural sedation carry-over, and a seizure. Each alternative was examined and excluded on the basis of the clinical course, imaging, and investigations. The patient was observed for an additional 24 hours, during which she remained haemodynamically stable. Comparison of symptoms before and after the PRF procedure is summarised in Table 3 and demonstrates clear improvement in cervical paraesthesia and hypoaesthesia along C3–C6, resolution of cluster-type headache episodes, and subjective improvement in vertigo, tinnitus, and hearing. Pericranial tenderness and tension-type headache persisted at similar intensity, and a transient

non-radicular local pain (NRS 5–6) at the PRF site was reported but resolved within 48 hours with paracetamol and cold compresses. The patient was discharged on the third post-procedural day with structured outpatient follow-up at the chronic pain clinic, instructions to increase oral fluid intake and sodium intake within cardiovascular tolerance, and explicit education on prodromal symptoms of vasovagal syncope and physical counter-manoeuvres.

3. Discussion

Chronic cervical radiculopathy frequently coexists with primary headache disorders and with occipital neuralgia, a pattern that reflects convergence of afferent traffic within the trigeminocervical complex. Our patient demonstrated this overlap explicitly: degenerative C5–C6 disc disease with mild foraminal stenosis, tension-type headache with pericranial tenderness, episodic cluster-type headache, and occipital neuralgia were present in parallel. Interventional options for patients who fail multimodal conservative care include physiotherapy, pharmacotherapy, local infiltration, pulsed radiofrequency, and posterior cervical decompression. Contemporary comparative-effectiveness studies are evaluating personalised multimodal physiotherapy against surgical decompression as a non-inferiority strategy in painful cervical radiculopathy, reflecting the ongoing debate about where neuromodulation and surgery should sit in the treatment hierarchy.[3] Minimally invasive posterior cervical biportal endoscopic decompression is a further emerging option, with published meta-analyses reporting high success rates in appropriately selected patients.[4] Within this spectrum, pulsed radiofrequency represents a low-morbidity intermediate step that can provide meaningful analgesia while deferring or obviating surgery.

Pulsed radiofrequency delivers high-voltage, high-frequency electrical pulses to a target neural structure through a cannula tip, producing a strong electrical field while preventing thermal neurodestruction through controlled duty cycling and maintenance of tip temperature at or below 42°C. The landmark randomised trial of cervical dorsal root ganglion PRF demonstrated superiority of PRF over sham at three months in patients with chronic cervical radicular pain, with a favourable safety profile.[6] Subsequent mechanistic reviews have proposed three overlapping mechanisms of effect: first, modulation of synaptic transmission with attenuation of glutamatergic and substance P signalling and upregulation of inhibitory mediators including gamma-aminobutyric acid and met-enkephalin; second, induction of long-term depression of dorsal horn nociceptive circuits; and third, anti-neuroinflammatory effects through reduction of microglial activation and pro-inflammatory cytokines.[7] Preclinical evidence additionally implicates epigenetic modulation of pain-related gene expression, such as restoration of K-Cl cotransporter 2 function and dampening of neuroinflammatory transcriptional programs, as a plausible mechanism of durable effect.[8] Central maladaptive plasticity in the anterior cingulate cortex and other cortical loci contributes to the maintenance of chronic neuropathic pain, and therapies that disrupt that plasticity — including PRF applied to peripheral targets — may indirectly modulate central pain processing.[9]

Occipital neuralgia arises most commonly from irritation or compression of the greater occipital nerve, or less often of the lesser occipital nerve; the greater occipital nerve is implicated in approximately 90% of cases. The nerves derive from the dorsal rami of C2 and C3 and pass through the semispinalis capitis muscle, where entrapment can occur. Surgical series of combined nerve combining and decompression based on neurovascular classification report clinically meaningful improvement in the majority of patients, indicating that mechanical decompression remains effective when less invasive measures fail.[11] Secondary occipital neuralgia can result from central lesions such as dorsal medullary infarction, which can present with coexistent short-lasting unilateral neuralgiform headache attacks, emphasising the need for vigilance toward central causes in atypical presentations.[12]

Cluster headache, one of the trigeminal autonomic cephalalgias, is characterised by strictly unilateral severe orbital or temporal pain accompanied by ipsilateral autonomic features including conjunctival injection, lacrimation, nasal congestion or rhinorrhoea,

and eyelid oedema. Contemporary reviews emphasise the central role of the hypothalamus, the trigeminal autonomic reflex, and pituitary–adrenal interactions in the generation of cluster attacks, and the overlap between cluster headache and cervico-occipital pain through the trigeminocervical complex.[13] Tension-type headache, by contrast, is linked more to autonomic dysregulation of the cervical and pericranial musculature, with measurable differences in parasympathetic tone, sleep quality, and psychological comorbidity compared with asymptomatic controls.[14] The epidemiology of primary headache disorders is substantial even in adolescent populations, underscoring the importance of recognising their contribution to the chronic pain burden.[15] In our patient, the clustering of occipital neuralgia, tension-type headache, and cluster-type episodes on a substrate of cervical radiculopathy created an anatomical and functional context in which cervical and occipital PRF could plausibly modulate both peripheral and central pain circuits and autonomic tone simultaneously.

Syncope is defined as a transient loss of consciousness due to global cerebral hypoperfusion, with rapid onset, short duration, and spontaneous complete recovery. The European Society of Cardiology classification divides syncope into reflex, orthostatic, and cardiac aetiologies, with reflex (neurally mediated) syncope — of which vasovagal syncope is the commonest subtype — dominating the prevalence data across age groups.[16] Contemporary reviews of syncope pathophysiology emphasise multiple overlapping mechanisms, including the Bezold–Jarisch reflex triggered by mechanoreceptors in the inferior-posterior left ventricle, impaired baroreflex sensitivity, autonomic imbalance with sudden parasympathetic dominance and sympathetic withdrawal, and neurohumoral disturbance involving adenosine, catecholamines, and, as increasingly appreciated, serotonergic neurotransmission.[17,18] Mechanistic animal studies of ganglionated plexus ablation support the view that selective attenuation of autonomic ganglia can abolish or blunt the vasovagal reflex, reinforcing the anatomical plausibility that stimulation or modulation of autonomic afferents during cervical and occipital interventional procedures could trigger a reflex response in susceptible individuals.[19] Pain, anxiety, prolonged upright posture, needle procedures, and venepuncture are all recognised triggers in the published literature.

In this case, the temporal association between bilateral occipital-nerve and cervical dorsal root ganglion PRF and the subsequent acute decrease in consciousness strongly suggests a causal link. Several mechanisms are plausible and most likely act in combination. First, direct PRF-induced afferent traffic from the occipital nerves and cervical DRGs converges on second-order neurons within the trigeminocervical complex, which has extensive reciprocal projections to the nucleus tractus solitarii. Sustained or exaggerated afferent input to this nucleus can trigger cardioinhibitory and vasodepressor responses through the Bezold–Jarisch reflex pathway.[17] Second, the pre-existing autonomic context of the patient — hypertensive, on amlodipine, and with a clinical history of vertigo suggestive of autonomic involvement — may have lowered the threshold for a reflex vasovagal response, especially given that anti-hypertensive therapy can accentuate orthostatic tendencies. Third, post-procedural pain at the PRF sites (non-radicular NRS 5–6) likely contributed a pain-triggered component similar to that documented in classical pain-induced vasovagal syncope. Fourth, serotonergic neurotransmission is increasingly implicated in the pathogenesis of vasovagal syncope; patients whose chronic analgesic regimen includes amitriptyline — as in our case — may show altered serotonergic tone that further predisposes them to reflex responses.[18] The 20-hour interval between the procedure and the event argues against an intraoperative sedation carry-over and is consistent with delayed autonomic rebalancing that followed the combined peripheral and central neuromodulatory effect of PRF.

Interventional pain procedures at the cervical level are usually conducted under light sedation to preserve responsiveness for intraprocedural sensory mapping. Propofol and midazolam, used in our protocol, provide a favourable combination of sedation depth, amnesia, and rapid recovery, although both agents carry cardiovascular depressant effects that must be titrated

carefully in older or hypertensive patients on chronic antihypertensive therapy. Variation exists across pain-medicine specialties in sedative choice, dosing, and monitoring standards, as shown in surveys of pain specialists and in analyses of opioid and sedative practice across specialties and geographies.[10,20] From an anaesthesiological standpoint, the key lessons from this case are that continuous electrocardiographic and non-invasive blood-pressure monitoring must extend into the post-procedural period, that peripheral intravenous access must be maintained for at least 24 hours after complex neck-level procedures in susceptible patients, and that adequate analgesia after PRF should be provided to minimise pain-triggered vasovagal events. Our patient received paracetamol and non-opioid analgesia, in keeping with modern opioid-sparing practice, which likely reduced but did not eliminate the residual noxious drive from the PRF sites. Whether alternative patient-selection tools, such as preinjury risk prediction models drawn from complex regional pain syndrome research, could identify patients at risk of amplified nociceptive or autonomic responses remains an open question.[21] Institutional pathways should also specify default fasting times, default peripheral intravenous catheter size, recommended prophylactic anti-emetics, and a standardised discharge criterion set, such as a modified Aldrete score of nine or more; this kind of procedural bundling has been shown in other settings to reduce both late complications and unplanned readmissions.

The safety literature on cervical and occipital PRF is dominated by reports of transient dysaesthesia, local soreness, and rare ataxia. In the Van Zundert trial, adverse events after cervical DRG PRF were minor and self-limiting, with no serious complications reported in the PRF arm.[6] Subsequent narrative reviews by Cohen and colleagues reached the same conclusion regarding the safety profile of PRF across anatomical targets.[7] Survey data from Dutch pain specialists show that more than 80% consider PRF a first- or second-line interventional option and reserve conventional radiofrequency for defined subgroups, and that specialists rarely report serious complications.[10] Reports of occipital neuralgia treated with combined surgical decompression document transient autonomic or sensory disturbances after intervention, but vasovagal syncope as a specific post-procedural entity has not been highlighted.[11] Secondary causes of occipital neuralgia associated with central lesions, such as dorsal medullary infarction, can also produce autonomic instability that may mimic a peripheral post-procedural reflex event.[12] Against this backdrop, the present case adds to the safety corpus in two ways. First, it demonstrates that vasovagal syncope can occur in the 24 hours after cervical and occipital PRF in a patient without a prior syncope history. Second, it exemplifies the importance of extended structured observation after complex neck-level pain procedures, even when intraprocedural vital signs remain stable. The favourable outcome in our patient — full spontaneous recovery, no neurological sequelae, and clinically meaningful improvement in the primary pain complaint — supports the continued use of PRF in appropriately selected patients, provided that a safety pathway for detection and management of reflex syncope is embedded in the care protocol.

The prevention strategy for reflex syncope after interventional pain procedures comprises five practical components. First, preprocedural risk stratification should capture age, antihypertensive therapy, prior syncope events, and chronic analgesic regimens — including tricyclic antidepressants, selective serotonin reuptake inhibitors, and anticonvulsants — that may alter autonomic tone. Second, adequate hydration and electrolyte optimisation should be ensured by a structured fasting and clear-fluid protocol; dehydration, so common before elective interventional procedures, is a potent amplifier of vasovagal susceptibility. Third, careful sedation titration is essential at cervical and occipital levels, where deep sedation can mask prodromal symptoms and delay rescue. Fourth, intraprocedural multimodal monitoring should be extended into the post-procedural ward for at least 24 hours in patients with identified risk factors, using continuous pulse oximetry and intermittent blood-pressure measurement. Fifth, structured post-procedural analgesia and observation protocols should explicitly recognise delayed vasovagal events: ward staff training, clear escalation criteria, and ready availability of intravenous fluids and atropine are the cornerstones

of a safe pathway. Management of an acute reflex syncope event follows standard principles: placement in the supine position with leg elevation, oxygen supplementation, intravenous isotonic fluid resuscitation, exclusion of cardiac, neurological, or metabolic alternatives, and observation until full recovery of consciousness and haemodynamic stability. In the longer term, first-line management of recurrent vasovagal syncope is conservative, with emphasis on hydration, sodium intake (within cardiovascular tolerance), recognition of prodromes, and physical counter-manoeuvres.[16] In refractory cases, selective serotonin reuptake inhibitors, midodrine, or, very rarely, pacemaker implantation may be considered, although evidence specifically supporting these therapies after post-procedural events is limited.[18]

The patient was reviewed in the chronic pain clinic at two weeks and again at six weeks after the procedure. At two weeks, cervical radicular pain was rated at NRS 1–2 at rest and 3 on exertion; cluster-type episodes had not recurred; vertigo, tinnitus, and subjective hearing loss were reported to have improved to approximately 30% of baseline severity; tension-type headache persisted at NRS 3–4. At six weeks, these gains were sustained. Blood pressure readings remained within a controlled range on amlodipine, and no further syncope episodes had occurred despite normal daily activities. The patient reported a marked reduction in the use of sodium diclofenac and amitriptyline and a subjective improvement in sleep. This favourable trajectory supports the conclusion that the adverse event described was transient and did not diminish the therapeutic benefit of the PRF intervention. Systematic follow-up after interventional pain procedures is thus essential both to document durable efficacy and to detect late or recurrent adverse events.

This report describes a single patient and is therefore subject to the inherent limitations of case-based observation. Tilt-table testing was not performed because full spontaneous recovery and absence of recurrence made this investigation non-contributory to immediate care. Continuous ambulatory blood-pressure and electrocardiogram monitoring for 24 hours post-event might have captured transient arrhythmic contributions, and future institutional protocols could consider Holter placement at the end of any cervical or occipital PRF performed in patients with identified autonomic risk markers. We also acknowledge that chronic kidney disease, new diabetes, or other systemic comorbidities can confound post-procedural observation; in our case, these were excluded by baseline laboratory screening.[22] Generalisability is further limited by the fact that the procedure was performed at a single tertiary centre with an experienced team; outcomes may differ in less specialised settings. A further caveat is that the precise contribution of each potential trigger — afferent stimulation, residual sedation, pain-induced parasympathetic surge, chronic tricyclic exposure, antihypertensive therapy — cannot be disentangled from a single observation. A prospective registry of post-procedural adverse events following cervical and occipital PRF, capturing timing, clinical phenotype, and recovery trajectory, would more robustly characterise the incidence, risk factors, and optimal management of reflex syncope in this context and is a logical next step for the specialty.

Several practical learning points emerge from this case. First, vasovagal syncope should be included in the differential diagnosis of an acute decrease in consciousness within 24 hours of cervical and occipital PRF, even in patients without a prior syncope history, and the differential must also include cardiac, neurological, and metabolic aetiologies, each of which has to be systematically excluded before a diagnosis of reflex syncope is accepted. Second, multimodal monitoring should extend beyond the immediate post-anaesthetic care unit to the ward in the first 24 hours after complex cervical interventional procedures, with an explicit nursing responsibility for documenting any change in consciousness or vital signs. Third, hydration, sodium optimisation within cardiovascular limits, and patient education on prodromal symptoms are simple but effective preventive measures that do not require additional resources in most settings. Fourth, the clinical effectiveness of PRF in improving cervical radicular pain, cluster-type headache, tinnitus, and vertigo illustrates its value as an integrated neuromodulatory intervention when appropriately applied, and the favourable outcome in our patient

should not deter future use; clinicians should communicate both benefits and rare risks during informed consent. Fifth, department-level post-procedural safety checklists should explicitly include reflex syncope as a recognised adverse event for cervical and occipital PRF so that the frequency and context of similar events can be captured systematically. Finally, the structured post-procedural evaluation table, illustrated in Table 3, is a practical template for documenting comparative symptom outcomes before and after neuromodulatory interventions and could be adopted as a standard reporting format in interventional pain registries.

4. Conclusion

We describe a reflex vasovagal syncope event occurring 20 hours after bilateral pulsed radiofrequency of the greater and lesser occipital nerves and of the C3 and C4 cervical dorsal root ganglia in a 41-year-old woman with chronic cervical radiculopathy, tension-type headache, a history of cluster-type headache, and occipital neuralgia. The likely mechanism involved convergent afferent traffic to the trigeminocervical complex, activation of the Bezold–Jarisch reflex, pain-triggered autonomic imbalance, and predisposition related to antihypertensive therapy and chronic amitriptyline exposure. Spontaneous complete recovery was achieved within seven hours without neurological sequelae, and pre-procedural pain indices improved substantially. For the anaesthesiology and chronic pain community, the case highlights the importance of structured post-procedural monitoring, proactive hydration, careful sedation titration, and systematic documentation of reflex syncope in departmental safety registries. PRF remains a safe and effective neuromodulatory technique, and recognition of this rare but clinically relevant complication will permit earlier identification and better management in future patients.

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