

ORIGINAL ARTICLE

Delayed Bradycardia and Hypotension After Cervical Dorsal Root Ganglion and Occipital Pulsed Radiofrequency: A Single-Patient Observational Case Study

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ABSTRACT

Background Pulsed radiofrequency (PRF) is a non-destructive neuromodulation option for chronic cervical radiculopathy and occipital neuralgia, with an excellent safety record. Delayed neuro-autonomic collapse is poorly described.

Objective To describe the timing, hemodynamic course and possible mechanisms of delayed vasovagal-type syncope after fluoroscopy-guided cervical dorsal root ganglion (DRG) and occipital PRF.

Methods A 41-year-old woman had intractable cervical radiculopathy (C3–C6), tension-type headache, a history of cluster headache, vertigo and hypertension treated with amlodipine. She received fluoroscopy-guided PRF (120 s, 2 Hz, 45 V, ≤42 °C) at the bilateral C3 and C4 DRG and the greater and lesser occipital nerves. Hemodynamics and the Glasgow Coma Scale (GCS) were recorded for up to six weeks, and causality was appraised with three frameworks.

Results The procedure was uneventful. About 20 hours later the patient collapsed, with GCS 12 (E3V3M6), sinus bradycardia (58 bpm, –27.50%), hypotension (102/62 mmHg; mean arterial pressure 75.33 mmHg, –30.04%). Trendelenburg positioning, crystalloid and atropine were followed by gradual recovery: GCS 13 at one hour, 14 at seven hours and 15 by the next morning, without sequelae. Cardiac and metabolic causes were excluded. The Naranjo score was 7 (probable), but the scale is not validated for one procedural event. Radicular pain was NRS 1–2 at 14 days, and analgesia was sustained at six weeks.

Conclusion Upper cervical and occipital PRF may be followed by severe, delayed reflex-type collapse, possibly through trigeminocervical convergence and sympathetic withdrawal; the mechanism was not measured. Longer observation may be considered in susceptible patients.

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1. Introduction

Chronic cervical radicular pain, often with occipital neuralgia, is a persistent and hard-to-treat neuropathic disorder. The cost is heavy.¹ Cervical radiculopathy has an annual incidence of about 83 per 100,000 people,^{2,3} and it arises from mechanical compression, narrowing of the neural foramen, or strong biochemical sensitization of the cervical nerve roots. Patients report lancinating radicular pain, dermatomal paresthesias, sensory loss and weakness of the shoulder girdle and arms.⁴ Occipital neuralgia adds a stabbing suboccipital pain that spreads forward along the greater and lesser occipital nerves. Together the two conditions are highly refractory, and oral analgesics, other drugs and physical rehabilitation usually fail.⁵

Interventional pain medicine has taken up pulsed radiofrequency as a minimally invasive treatment of first importance.⁶ Conventional continuous radiofrequency abla-

tion heats tissue above 80 °C. This causes coagulation necrosis, neurolysis and irreversible Wallerian degeneration. PRF works differently. It sends short bursts of high-voltage current, separated by long silent intervals.⁷ Heat dissipates between bursts, so tissue temperature stays at or below 42 °C.⁸ The risks of permanent deafferentation pain, cutaneous dysesthesia, neuroma formation and accidental motor deficit are avoided, and PRF has earned a reputation for safety at peripheral and spinal nerve targets.⁹

Its benefit comes from neuromodulation, not from tissue destruction.¹⁰ The electrical field at the electrode tip alters cellular transmembrane potentials and is reported to alter c-Fos expression in the dorsal horn, a marker of nociceptive transmission and central sensitization, although the direction of the effect differs between studies.¹¹ PRF also dampens microglial activation and local pro-inflammatory cytokines. It modulates the release of glutamate and substance P, and it strengthens descending serotonergic and noradrenergic inhibition.¹² The result is long-term depression in overactive spinal synapses, which resets abnormal

pain pathways and gives lasting analgesia in cervical root syndrome and occipital neuralgia.¹³

PRF is widely used and is considered very safe. Even so, its neuro-autonomic risks have received little study, above all at the junction of the upper cervical spine and the suboccipital region.¹⁴ The dorsal root ganglia of C1 to C4 and the peripheral branches of the occipital nerves are closely tied, anatomically and functionally, to the trigeminocervical complex and to central autonomic networks.¹⁵ Afferent fibers from the upper three cervical roots converge on the descending spinal tract of the trigeminal nerve in the upper cervical cord. The two systems exchange signals in both directions.¹⁶ The trigeminocervical complex also projects directly to brainstem autonomic centers, through both monosynaptic and polysynaptic routes. The main targets are the nucleus tractus solitarius, the rostral ventrolateral medulla and the dorsal motor nucleus of the vagus.¹⁷

Under normal conditions the nucleus tractus solitarius combines sensory, visceral and somatic input. It balances peripheral sympathetic vasomotor tone against central parasympathetic outflow.¹⁸ Strong electrical neuromodulation of the upper cervical ganglia and occipital nerves could upset this balance without warning.¹⁹ Reflex loops may misfire, baroreflex sensitivity may change, or central sympathetic tone may shift suddenly, and any of these can cause autonomic instability.²⁰ Vasovagal syncope is the classic result. An afferent trigger provokes paradoxical vagal hyperactivity together with a collapse of peripheral sympathetic tone. Cardioinhibition, vasodepression and global cerebral hypoperfusion follow.²¹ Procedural vasovagal reactions are common, but they are usually brief and immediate. Needle puncture, anxiety or visceral discomfort sets them off. A severe collapse that appears twenty hours after an uneventful PRF session has rarely been reported.^{22–24}

A delayed autonomic collapse after a local, non-destructive procedure is a diagnostic puzzle that also tests current safety assumptions in acute care and interventional anesthesiology.²⁵ Three explanations deserve attention: a slow neurohormonal readjustment, an abnormal trigeminocervical-vagal reflex arc, or a sudden hemodynamic decompensation that appears when rapid pain relief removes pain-driven sympathetic overdrive.²⁶ Which one applies matters for practice. The aim of this report is to describe an unusual episode of acute, severe vasovagal syncope. It showed marked cardioinhibitory bradycardia, marked vasodepressor hypotension and impaired consciousness twenty hours after fluoroscopy-guided PRF of the bilateral C3 and C4 dorsal root ganglia and the occipital nerves. We used a single-patient observational design, dense longitudinal hemodynamic tracking and a structured, though necessarily limited, causality assessment. Our intent is to describe the neuro-autonomic vulnerabilities that upper cervical neuromodulation may reveal and to encourage perioperative hemodynamic vigilance.

2. Methods

2.1. Study design and reporting

We designed a single-patient observational case study with dense longitudinal tracking and a multi-framework causality assessment.²⁷ Its purpose was to describe the timing, the physiological course and the likely mechanisms of acute neuro-autonomic decompensation after an elective fluoroscopy-guided PRF procedure. We did not want the event to read as an isolated anecdote, so the design fol-

lowed a structured observation protocol. It covered baseline phenotyping, precise logging of technical parameters during the procedure, continuous inpatient telemetry, vital signs at predefined intervals, and structured monitoring after resuscitation that continued through two weeks of outpatient follow-up. Reporting followed the CARE guideline for case reports,²⁸ with items of the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guideline adapted for a single case. It also followed the international consensus guidelines for reporting adverse events in interventional pain medicine.²⁹

2.2. Ethics and consent

The protocol and data collection complied with the ethical principles of the World Medical Association Declaration of Helsinki of 1975, as revised in Brazil in 2013. CMHC Research Center Ethics Committee, Indonesia, reviewed and approved the study, including formal ethical clearance and registration of the protocol (Protocol Approval Number: 2026/4108). The patient gave written informed consent in person before the procedure and before the later academic analysis. She was told about the technique, the possible intraoperative complications, the expected clinical outcomes, and the plan to analyze and publish her anonymized clinical data, laboratory values, imaging and serial hemodynamic trajectories. Confidentiality was kept throughout. All direct and indirect identifiers were removed, in line with the standards of the International Committee of Medical Journal Editors (ICMJE) and the Committee on Publication Ethics (COPE).

2.3. Participants and eligibility

A multidisciplinary clinical pathway decided eligibility for interventional neuromodulation; the criteria below are the institutional criteria for PRF and were not used to select a study population. Senior consultant anesthesiologists, interventional pain specialists and spine surgeons took part.³⁰ Inclusion required: (1) age of eighteen years or older; (2) chronic cervical radicular pain lasting more than three consecutive months; (3) concordant evidence of cervical neural compression or foraminal stenosis on high-resolution magnetic resonance imaging; (4) chronic occipital neuralgia meeting the International Classification of Headache Disorders, Third Edition (ICHD-3) criteria; and (5) confirmed failure of comprehensive conservative care, defined as at least six weeks of multimodal pharmacotherapy, physical therapy and postural rehabilitation. Exclusion criteria were: (1) progressive motor deficit suggesting acute cervical myelopathy that needs urgent decompression; (2) active systemic or local spinal infection; (3) uncorrected coagulopathy or therapeutic anticoagulation; (4) structural cardiovascular disease, cardiac arrhythmia or an implanted pacemaker; and (5) inability to give informed consent or to complete longitudinal follow-up.

2.4. Observation protocol and procedure

Observation was divided into three epochs. The first was the pre-procedural baseline (T0). The second was the perioperative interventional period. The third was post-procedural monitoring, from T+2 hours to day 14.³¹ Throughout the procedure we applied standard American Society of Anesthesiologists (ASA) monitoring: five-lead electrocardiography, non-invasive automated blood pressure, pulse oximetry and continuous capnography. Mild conscious sedation used intravenous propofol 0.5 mg/kg and midazolam 0.01 mg/kg. The doses gave adequate anxiolysis and

comfort. They also kept the patient able to answer, which real-time sensory and motor stimulation testing requires.

The patient lay prone with the cervical spine flexed, and the field was prepared and draped under sterile conditions. High-resolution fluoroscopy identified the C3 and C4 neural foramina. A 22-gauge, 10-cm radiofrequency cannula with a 10-mm curved active tip was advanced by a posterior-lateral oblique route under continuous fluoroscopic guidance. Anteroposterior and lateral views confirmed the tip in the posterior part of the intervertebral foramen, directly beside the dorsal root ganglion. Electrical impedance was measured to check tissue coupling in the target. Sensory stimulation at 50 Hz was applied to reproduce concordant dermatomal paresthesias, at low voltages of 0.40 to 0.60 V. Motor stimulation at 2 Hz was then checked up to 1.50 V. The absence of fasciculations showed a safe distance from the anterior motor root. After stimulation was verified, 1.0 mL of 1% preservative-free lidocaine was injected for local analgesia. PRF used standard generator settings: 120 seconds per target, a pulse frequency of 2 Hz, a pulse width of 20 milliseconds and a peak voltage of 45 V. Automated feedback held the tip temperature at 42.0 °C or below. The same sequence was repeated at the contralateral cervical levels and at both greater and lesser occipital nerves, using combined ultrasound and fluoroscopic landmarks. The procedure ended without incident. The patient went to the inpatient ward for continuous automated hemodynamic telemetry, hourly nurse charting and serial pain assessment.

2.5. Reference ranges and comparison with published data

Two comparisons were made to give the hemodynamic data some context.³² The first set the patient's serial readings against reference intervals from the American Heart Association and the European Society of Cardiology. Normal adult resting values were taken as systolic pressure 90–120 mmHg, diastolic 60–80 mmHg, mean arterial pressure 70–100 mmHg, sinus heart rate 60–100 beats per minute and a Glasgow Coma Scale of 15. The second set the clinical course against published reports in the interventional pain and syncope literature. Those reports describe the frequency of adverse events, autonomic reflexes and the onset latency of complications after cervical neuromodulation, percutaneous cervical disc decompression, cervical epidural injection and cardioneuroablation.^{33–38} The comparison is descriptive.

2.6. Statistical and trajectory analysis

The design is a single case ($n=1$), so the analysis was kept strictly proportionate to it.³⁹ No inferential tests were used, whether *p*-values, Student's *t*-tests, analysis of variance or chi-square. Applying them to one subject is not appropriate and would give a false sense of precision. The analysis was descriptive instead. We modeled trajectories, tracked time series and calculated relative changes at predefined milestones: T0 pre-operative baseline, T+2h immediate post-operative, T+12h overnight ward, T+20h syncopal nadir (preceded by a prodrome at 15:30), T+21h, T+23h and T+27h recovery, T+35h next morning, T+48h ward discharge, Day 14 and Week 6 follow-up, eleven time points in all.

Mean Arterial Pressure (MAP) came from the validated formula $MAP = DBP + 1/3(SBP - DBP)$. The Rate-Pressure Product (RPP) estimates myocardial oxygen consumption and cardiac workload. We derived it as $RPP = HR \times SBP$.

Changes over time were expressed as a percentage delta, $\Delta\% = [(T_n - T_0) / T_0] \times 100\%$. Here T_0 is the baseline pre-procedural measurement and T_n is the value at a given post-procedural milestone. Causality and adverse event attribution used three validated epidemiological frameworks. The first was the Naranjo Adverse Drug/Procedure Reaction Probability Scale,⁴⁰ which sorts causality into definite, probable, possible or doubtful. The second was the World Health Organization-Uppsala Monitoring Centre (WHO-UMC) standardized causality system. The third was the Austin Bradford Hill criteria,⁴¹ which assess epidemiological and biological causality through temporality, biological plausibility, coherence and consistency. These instruments were designed for drug reactions and for populations, and applied here they give an orienting appraisal, not proof of causation.

3. Results

3.1. Baseline clinical findings

The patient was a premenopausal woman of 41 years. She measured 155 cm and weighed 50 kg, giving a body mass index of 20.8 kg/m² and a body surface area of 1.47 m². For six months she had suffered severe, disabling neck pain that spread to both shoulder girdles and to the occipital regions.⁴² Examination showed a normoweight woman in moderate distress. Baseline (T_0) resting vital signs were as follows. Systolic pressure was 163 mmHg and diastolic pressure 80 mmHg, which gives a calculated mean arterial pressure of 107.67 mmHg. Sinus heart rate was 80 beats per minute, respiratory rate 16 breaths per minute, oxygen saturation 98% on ambient air and rate-pressure product 13,040 bpm·mmHg. She was fully conscious, with a Glasgow Coma Scale of 15 (E4V5M6). Cranial nerve testing found subjective right-sided tinnitus and mild sensorineural hearing loss. There was no focal motor or sensory trigeminal deficit. Strength was 5/5 in all bilateral C5–T1 myotomes, although severe neck pain limited effort. Deep tendon reflexes (biceps, triceps, patellar, Achilles) were symmetric and brisk (+2). No pathological reflex appeared (Hoffman, Babinski and Chaddock negative on both sides). Provocative cervical maneuvers were clearly positive on both sides for Lhermitte's sign and Spurling's test; Spurling's test supports root compression, and Lhermitte's sign also raises the question of cord involvement. Palpation of the suboccipital region also found marked myofascial trigger point tenderness.

Magnetic resonance imaging showed multilevel degenerative cervical spondylosis. At C5–C6 a large posterior disc herniation caused severe canal stenosis and bilateral foraminal compromise, and C4–C5 had mild canal stenosis. The endplate changes were Modic Type II and the disc desiccation was Pfirrmann Grade III. Her history held further problems. She had recurrent cluster-like headache, with severe unilateral right periorbital throbbing, ipsilateral conjunctival injection and lacrimation. She also had chronic spontaneous vertigo and a recent diagnosis of primary essential hypertension, treated with oral amlodipine 10 mg daily. Other baseline drugs were oral amitriptyline 25 mg at night, diclofenac sodium 50 mg twice daily, betahistine mesylate 6 mg three times daily and diazepam 5 mg as needed. Cervical radicular pain on the Numeric Rating Scale (NRS) was 3–4 at rest and rose to 5–6 on cervical extension and lateral rotation. Table 1 lists the baseline sociodemographic, anthropometric, neurological and radiological data in full.

Table 1. Baseline Sociodemographic, Anthropometric, Cervical Radiculopathy, and Pre-Procedural Neuro-Autonomic Profiles

Clinical Domain / Parameter	Baseline Measurement	Reference / Cut-off	Diagnostic and Prognostic Interpretation
Age and Sex	41 years, Female	N/A	Premenopausal female with chronic neuropathic cervico-occipital pain
Height and Weight	155 cm, 50 kg	N/A	Normal body habitus; total body surface area = 1.48 m ²
Body Mass Index (BMI)	20.8 kg/m ²	18.5–24.9 kg/m ²	Normoweight; excludes obesity-related autonomic blunting
Baseline Systolic Blood Pressure (SBP)	163 mmHg	90–120 mmHg	Elevated; in the grade 2 hypertension range on one reading; raised baroreflex set point is hypothesized
Baseline Diastolic Blood Pressure (DBP)	80 mmHg	60–80 mmHg	Borderline resting diastolic vascular tone
Baseline Mean Arterial Pressure (MAP)	107.67 mmHg	70–100 mmHg	Elevated resting perfusion pressure secondary to sympathetic drive
Baseline Resting Heart Rate (HR)	80 bpm	60–100 bpm	Normal sinus rhythm; absence of baseline chronotropic failure
Rate-Pressure Product (RPP = HR × SBP)	13,040 bpm·mmHg	<12,000 bpm·mmHg	Mildly elevated myocardial workload index under chronic pain stress
Peripheral Oxygen Saturation (SpO ₂)	98% on room air	≥95%	Adequate resting arterial oxygen saturation
Baseline Glasgow Coma Scale (GCS)	15 (E4V5M6)	15	Fully alert; intact cortical and brainstem neurological function
Cervical Radicular Pain Score (NRS)	3–4 at rest, 5–6 movement	0–10 Scale	Moderate-to-severe neuropathic radicular irritation (C3–C6)
Provocative Cervical Radicular Tests	Lhermitte (+/+), Spurling (+/+)	Negative (-/-)	Spurling's test: root compression; Lhermitte's sign: possible cord involvement [Author query]
Suboccipital Palpatory Examination	Pericranial tenderness (+)	Negative (-)	Active myofascial trigger and greater/lesser occipital nerve irritation
Cervical Spine Magnetic Resonance Imaging	C5–C6 severe stenosis; C4–C5 mild	Modic II, Pfirrmann III	Multilevel degenerative disc herniation, neural foraminal stenosis
Autonomic / Neurological Comorbidities	Tension headache, Cluster history, Vertigo	ICHD-3 criteria	Trigemino-cervical sensitization, right tinnitus, hearing loss
Active Pharmacotherapy Regimen	Amlodipine 10 mg, Amitriptyline 25 mg	Daily oral dosing	Dihydropyridine vasodilator and neuromodulatory tricyclic

Notes: BMI: Body mass index; SBP: Systolic blood pressure; DBP: Diastolic blood pressure; MAP: Mean arterial pressure [calculated as $DBP + 1/3(SBP - DBP)$]; HR: Heart rate; RPP: Rate-pressure product ($HR \times SBP$); SpO₂: Peripheral capillary oxygen saturation; GCS: Glasgow Coma Scale; NRS: Numeric Rating Scale (0–10); ICHD-3: International Classification of Headache Disorders, 3rd edition.

3.2. Procedure parameters and stimulation thresholds

On November 19th, 2024, the patient had elective fluoroscopy-guided pulsed radiofrequency of the bilateral C3 and C4 dorsal root ganglia and the bilateral greater and lesser occipital nerves.⁴³ Standard ASA monitoring and titrated conscious sedation continued throughout. Continuous fluoroscopy confirmed cannula position. Electrode impedance ranged from 325 to 410 Ω at all sites. These values showed good tissue contact within the neural canal, with no vascular breach and no cerebrospinal fluid leak. Sensory stimulation at 50 Hz reproduced concordant paresthesias in the matching dermatomes at low thresholds. The thresholds were 0.45 V for the right C3 ganglion, 0.50 V for the left C3, 0.40 V for the right C4 and 0.55 V for the left C4. At the greater occipital nerves they were 0.40–0.50 V, and at the

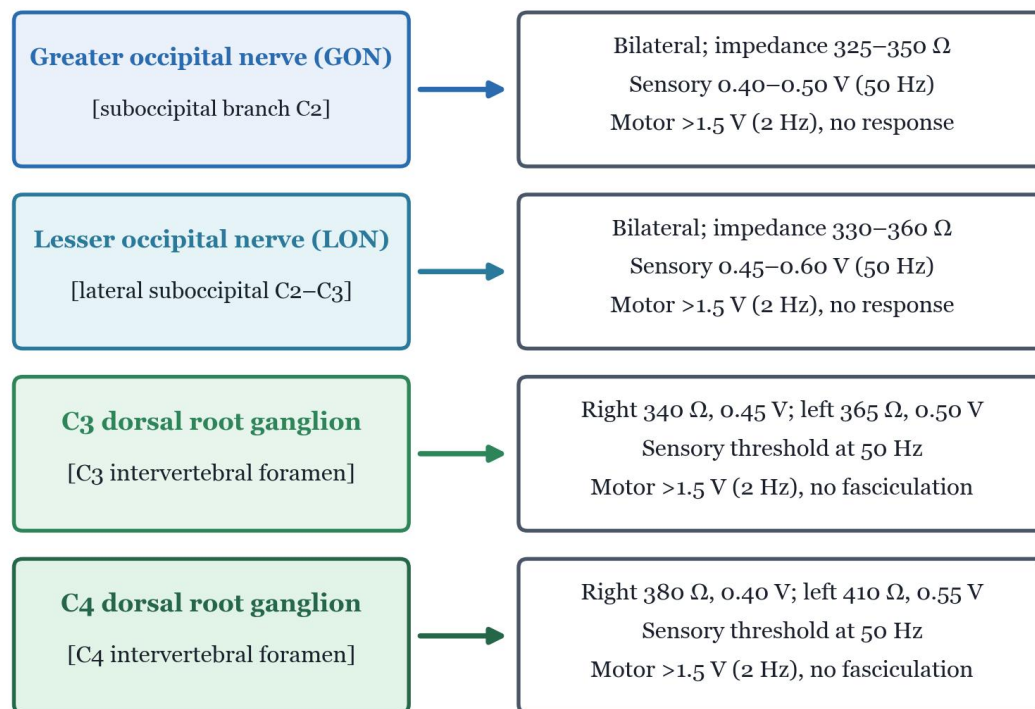
lesser occipital nerves 0.45–0.60 V. Motor stimulation at 2 Hz produced no fasciculation or twitch up to 1.50 V, so the anterior motor roots lay at a safe distance.

After local infiltration of 1.0 mL of 1% lidocaine at each target, PRF energy was delivered for 120 seconds per target. Pulse frequency was 2 Hz, pulse duration 20 milliseconds and peak voltage 45 V. The generator kept the electrode tip between 40.0 °C and 42.0 °C, so no thermal coagulation occurred. All targets were treated in the same way and in symmetric order, under continuous fluoroscopic verification (Figure 1). The whole procedure took 45 minutes. The patient tolerated it without discomfort, stayed hemodynamically stable in the operating room and went to the inpatient ward for 24-hour observation. Table 2 gives the technical specifications and stimulation thresholds.

Table 2. Technical Interventional Parameters of Fluoroscopy-Guided Pulsed Radiofrequency and Neurostimulation Thresholds

Target Anatomical Structure	Needle Gauge / Length / Tip	Impedance (Ω)	Sensory Threshold (50 Hz)	Motor Threshold (2 Hz)	PRF Duration & Voltage	Thermal Limit
Right C3 Dorsal Root Ganglion	22G / 10 cm / 10-mm active tip	340 Ω	0.45 V (concordant paresthesia)	>1.5 V (no fasciculation)	120 s, 2 Hz, 45 V	Max 42.0 °C
Left C3 Dorsal Root Ganglion	22G / 10 cm / 10-mm active tip	365 Ω	0.50 V (concordant paresthesia)	>1.5 V (no fasciculation)	120 s, 2 Hz, 45 V	Max 42.0 °C
Right C4 Dorsal Root Ganglion	22G / 10 cm / 10-mm active tip	380 Ω	0.40 V (concordant paresthesia)	>1.5 V (no fasciculation)	120 s, 2 Hz, 45 V	Max 42.0 °C
Left C4 Dorsal Root Ganglion	22G / 10 cm / 10-mm active tip	410 Ω	0.55 V (concordant paresthesia)	>1.5 V (no fasciculation)	120 s, 2 Hz, 45 V	Max 42.0 °C
Bilateral Greater Occipital Nerves	22G / 10 cm / 10-mm active tip	325–350 Ω	0.40–0.50 V (occipital tingling)	>1.5 V (no motor jump)	120 s, 2 Hz, 45 V	Max 42.0 °C
Bilateral Lesser Occipital Nerves	22G / 10 cm / 10-mm active tip	330–360 Ω	0.45–0.60 V (auricular paresthesia)	>1.5 V (no motor jump)	120 s, 2 Hz, 45 V	Max 42.0 °C

Notes: PRF: Pulsed radiofrequency; G: Gauge; cm: Centimeters; mm: Millimeters; Ω: Ohms; V: Volts; Hz: Hertz; s: Seconds; °C: Degrees Celsius. Procedural sedation comprised intravenous propofol 0.5 mg/kg and midazolam 0.01 mg/kg. Local anesthetic infiltration consisted of 2% lidocaine for skin and 0.5–1.0 mL 1% lidocaine per neural target.



Cannula 22 G, 10 cm, 10-mm active tip at every target. PRF: 120 s, 2 Hz, pulse width 20 ms, 45 V, maximum 42.0 °C.

Figure 1. Targets and stimulation data of PRF (schematic, not an anatomical or fluoroscopic image). Impedance and thresholds are those in Table 2; every target received PRF at 2 Hz with a pulse width of 20 ms, 45 V and a tip temperature of 42 °C or less.

3.3. Hemodynamic trajectory and syncopal event

After transfer to the ward the early course was uneventful.⁴⁴ At T+2 hours the blood pressure was 150/78 mmHg (MAP 102.00 mmHg, delta -5.27%), the heart rate 76 bpm (delta -5.00%) and the rate-pressure product 11,400 bpm·mmHg (delta -12.58%). GCS remained 15 (E4V5M6). At T+12 hours, overnight, the blood pressure was 142/75 mmHg (MAP 97.33 mmHg, delta -9.60%), the heart rate 72 bpm (delta -10.00%) and the rate-pressure product 10,224 bpm·mmHg (delta -21.60%). The patient reported much less neck stiffness and slept comfortably.

On November 20th, 2024, about 20 hours after the procedure, she suddenly decompensated.⁴⁵ At 15:30 the first signs appeared. She developed generalized cold sweating, facial pallor, lightheadedness and nausea. Her blood pressure fell to 118/68 mmHg (MAP 84.67 mmHg, delta -21.36%) and her sinus rate slowed to 58 bpm (delta -27.50%). The RPP was 6,844 bpm·mmHg (delta -47.52%). Within thirty minutes, at T+20 hours (16:00), circulatory and neurological collapse occurred. Consciousness dropped fast, to a Glasgow Coma Scale of 12 (eye opening

to voice E3, confused verbal response E3, obeying commands M6). Continuous monitoring recorded severe hypotension. Blood pressure fell to 102/62 mmHg, with a MAP nadir of 75.33 mmHg, a drop of -30.04% from baseline. Telemetry showed severe sinus bradycardia at 58 beats per minute (delta -27.50%). The rate-pressure product dropped to 5,916 bpm·mmHg, a fall of -54.63% in myocardial performance and perfusion pressure. Pulses were weak and thready, capillary refill took longer than 3.5 seconds, and the skin was cold and clammy. An emergency arterial blood gas and a bedside capillary glucose (108 mg/dL) excluded hypoglycemia, hypoxemia and acute metabolic acidosis. A twelve-lead electrocardiogram showed marked sinus bradycardia. It showed no ST-segment deviation, conduction block or QT prolongation. Serial troponin I remained undetectable (<0.01 ng/mL), which ruled out acute coronary ischemia. A cranial computed tomography scan was reported as unremarkable. Figure 2 plots the longitudinal hemodynamic course and the syncopal decompensation across the serial vital sign intervals.

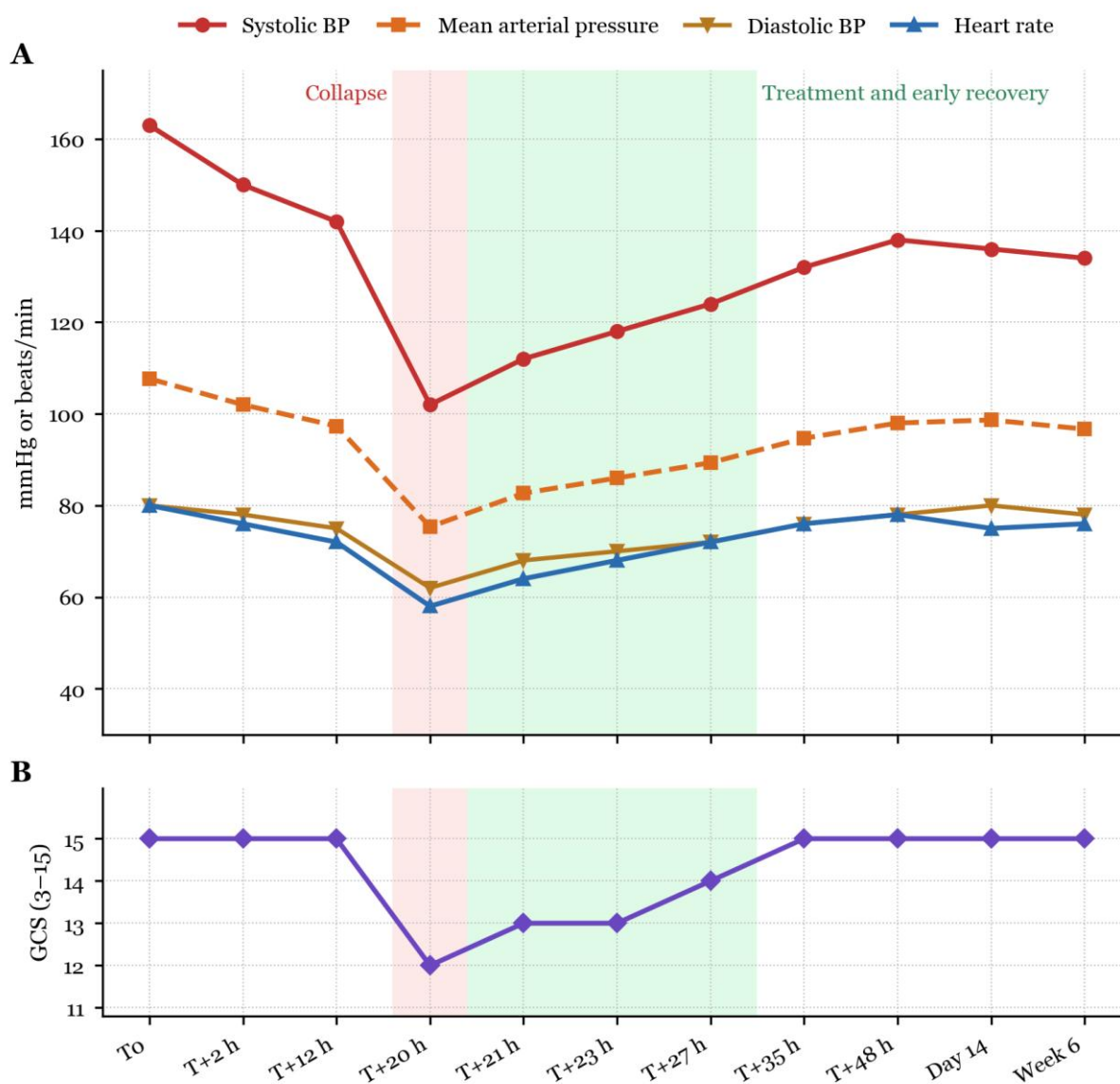


Figure 2. Blood pressure and heart rate (A) and Glasgow Coma Scale (B) from baseline to week 6, plotted from Table 3. The shaded bands mark the collapse (T+20 h) and the period of treatment and early recovery.

3.4. Resuscitation and recovery

An emergency medical code was called as soon as the collapse was recognized.⁴⁶ Treatment began at once. The patient was placed in a 30-degree Trendelenburg position to promote venous autotransfusion and cerebral perfusion. She received oxygen at 10 L/min through a non-rebreather mask. Warm isotonic crystalloid ran rapidly through two large-bore peripheral lines, 1,000 mL in 30 minutes. Because symptomatic bradycardia (58 bpm) persisted, intravenous atropine sulfate 0.5 mg was given as a bolus.

Recovery was gradual (Table 3).⁴⁷ One hour after the onset (T+21 hours) the blood pressure was 112/68 mmHg (MAP 82.67 mmHg, delta -23.22%), the heart rate 64 bpm (delta -20.00%) and the rate-pressure product 7,168 bpm·mmHg (delta -45.03%). The patient opened her eyes to speech but was disoriented, and the Glasgow Coma Scale was 13 (E3V4M6). At three hours (T+23 hours) the blood pressure was 118/70 mmHg (MAP 86.00 mmHg, delta -20.13%), the heart rate 68 bpm (delta -15.00%) and the rate-pressure product 8,024 bpm·mmHg (delta -38.47%). She opened her eyes spontaneously and answered simple questions (GCS 13). At seven hours (T+27 hours) the blood

pressure was 124/72 mmHg (MAP 89.33 mmHg, delta -17.03%), the heart rate 72 bpm (delta -10.00%) and the rate-pressure product 8,928 bpm·mmHg (delta -31.53%). She was oriented, her speech was clear, her extremities were warm, and the GCS was 14 (E3V5M6). Continuous telemetry showed a stable sinus rhythm, without ectopic pauses or further bradycardic dips.

By the next morning (T+35 hours) the patient was alert and fully oriented, with a Glasgow Coma Scale of 15 (E4V5M6), normal cognition and warm, well-perfused limbs. The blood pressure was 132/76 mmHg (MAP 94.67 mmHg, delta -12.07%), the heart rate 76 bpm (delta -5.00%) and the rate-pressure product 10,032 bpm·mmHg (delta -23.07%). She was ambulatory and stable at ward discharge (T+48 hours; 138/78 mmHg, MAP 98.00 mmHg, HR 78 bpm, RPP 10,764 bpm·mmHg). Table 3 gives the full chronological record of hemodynamic and neurological milestones across all eleven observation points. Figure 3 shows the calculated percentage deltas ($\Delta\%$) in myocardial workload and perfusion relative to baseline, and Figure 4 shows the recovery of the Glasgow Coma Scale.

Table 3. Longitudinal High-Density Hemodynamic Trajectory and Neurological Recovery Timeline

Observation Timepoint	SBP (mmHg)	DBP (mmHg)	MAP (mmHg)	HR (bpm)	RPP (bpm·mm Hg)	GCS Score	Δ% MAP	Δ% HR	Δ% RPP	Δ% GCS	Clinical Status & Interventions
Baseline (T0: Pre-Op)	163	80	107.67	80	13,040	15 (E4V5M6)	0.00%	0.00%	0.00%	0.00%	Conscious, alert, moderate cervical pain
Immediate Post-Op (T+2h)	150	78	102.00	76	11,400	15 (E4V5M6)	-5.27%	-5.00%	-12.58%	0.00%	Hemodynamically stable in recovery ward
Overnight Ward (T+12h)	142	75	97.33	72	10,224	15 (E4V5M6)	-9.60%	-10.00%	-21.60%	0.00%	Resting comfortably; analgesia reported
Syncopal Nadir (T+20h)	102	62	75.33	58	5,916	12 (E3V3M6)	-30.04%	-27.50%	-54.63%	-20.00%	Acute loss of consciousness; Trendelenburg, IV fluids 1,000 mL, oxygen and atropine 0.5 mg
1h Post-Onset (T+21h)	112	68	82.67	64	7,168	13 (E3V4M6)	-23.22%	-20.00%	-45.03%	-13.33%	Eye opening to speech, disoriented; volume expansion continued
3h Post-Onset (T+23h)	118	70	86.00	68	8,024	13 (E3V4M6)	-20.13%	-15.00%	-38.47%	-13.33%	Spontaneous eye opening, answering simple questions
7h Post-Onset (T+27h)	124	72	89.33	72	8,928	14 (E3V5M6)	-17.03%	-10.00%	-31.53%	-6.67%	Oriented, speech clear; warm peripheries, no focal deficit
Next Morning (T+35h)	132	76	94.67	76	10,032	15 (E4V5M6)	-12.07%	-5.00%	-23.07%	0.00%	Full neurological recovery, GCS 15; CT scan unremarkable
Ward Discharge (T+48h)	138	78	98.00	78	10,764	15 (E4V5M6)	-8.98%	-2.50%	-17.45%	0.00%	Ambulatory, stable; pain clinic discharge instructions
Follow-up (Day 14)	136	80	98.67	75	10,200	15 (E4V5M6)	-8.36%	-6.25%	-21.78%	0.00%	Radicular pain NRS 1–2; no syncope recurrence
Follow-up (Week 6)	134	78	96.67	76	10,184	15 (E4V5M6)	-10.22%	-5.00%	-21.90%	0.00%	Sustained analgesia; diclofenac/amitriptyline tapered

Notes: SBP: Systolic blood pressure; DBP: Diastolic blood pressure; MAP: Mean arterial pressure $[DBP + 1/3(SBP - DBP)]$; HR: Heart rate; RPP: Rate-pressure product $(HR \times SBP)$; GCS: Glasgow Coma Scale; Δ%: Serial percentage delta change relative to baseline T0: $\Delta\% = [(T_n - T_0) / T_0] \times 100\%$. Δ% of GCS is descriptive only; GCS is an ordinal scale.

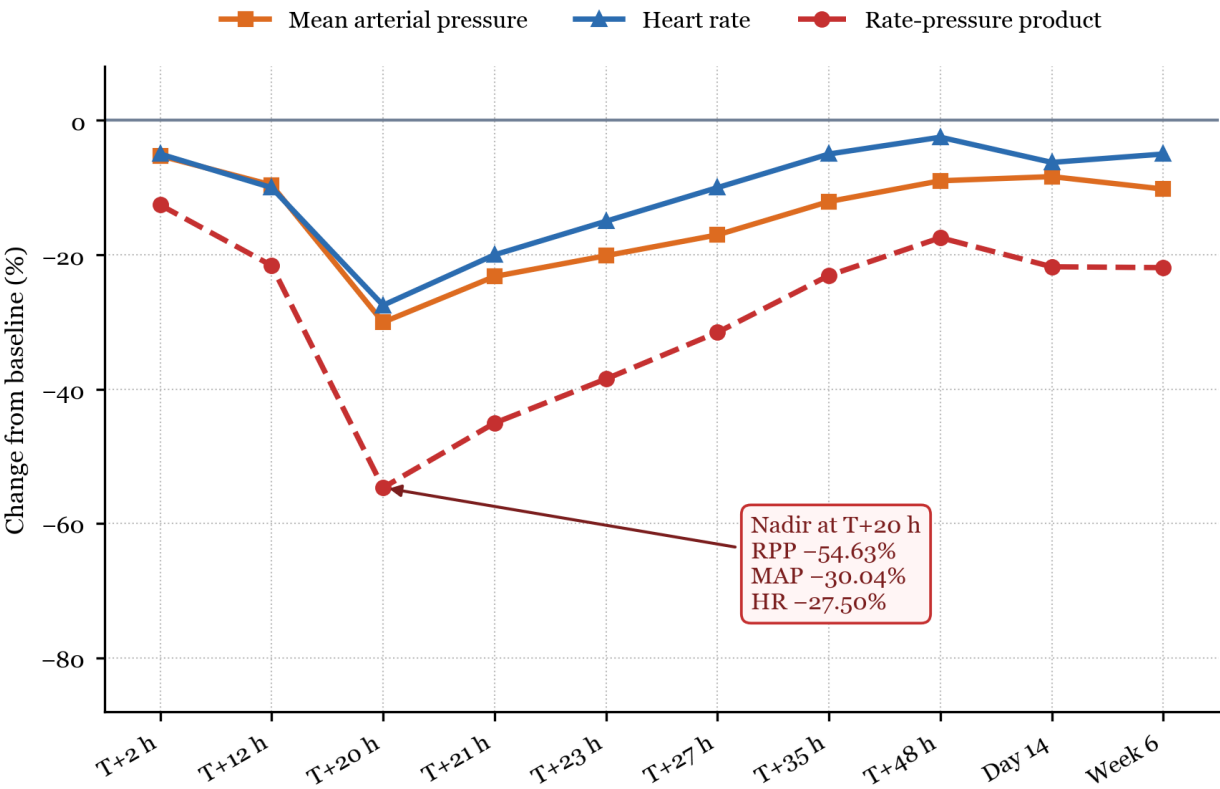


Figure 3. Serial percentage change from baseline in mean arterial pressure, heart rate and rate-pressure product between T+2 h and week 6, calculated as in Table 3.

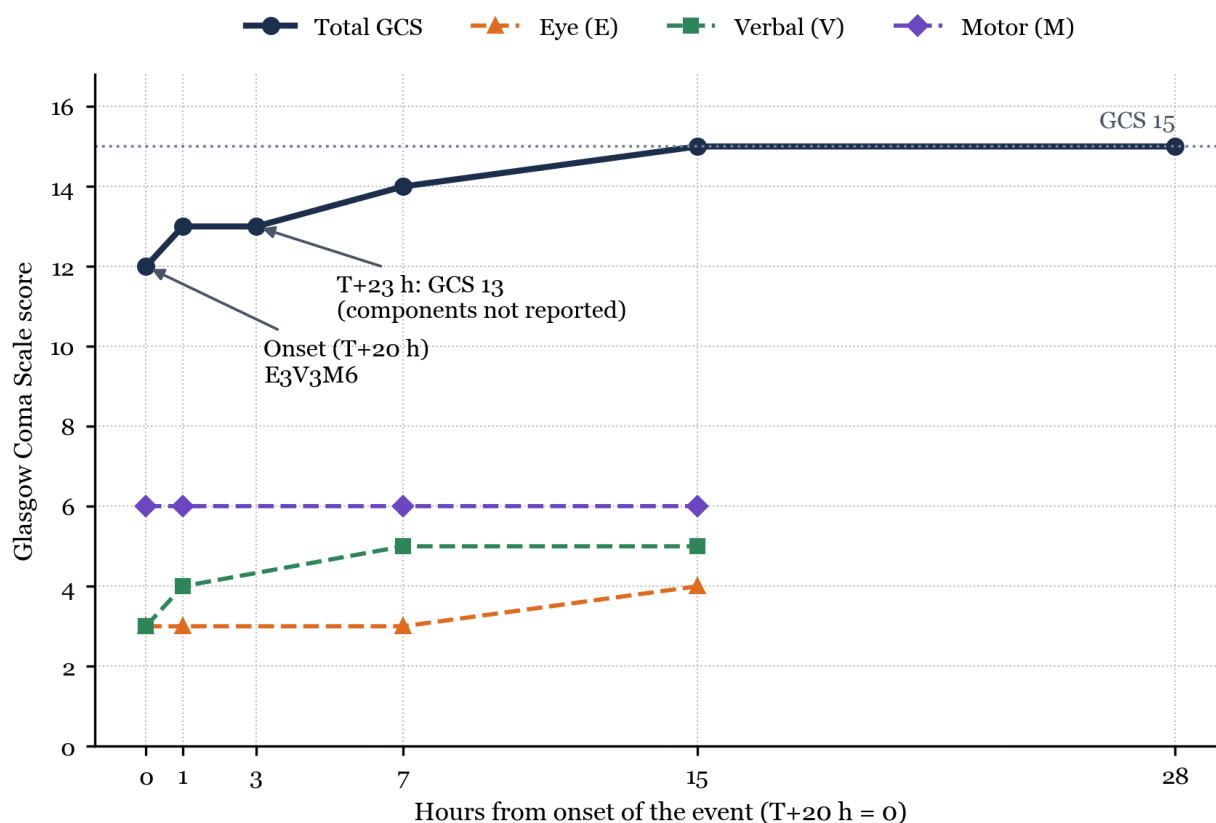


Figure 4. Recovery of the Glasgow Coma Scale after the onset of the event, plotted from Table 3 and the text. Components are shown only where they were reported.

3.5. Causality assessment and analgesic outcome

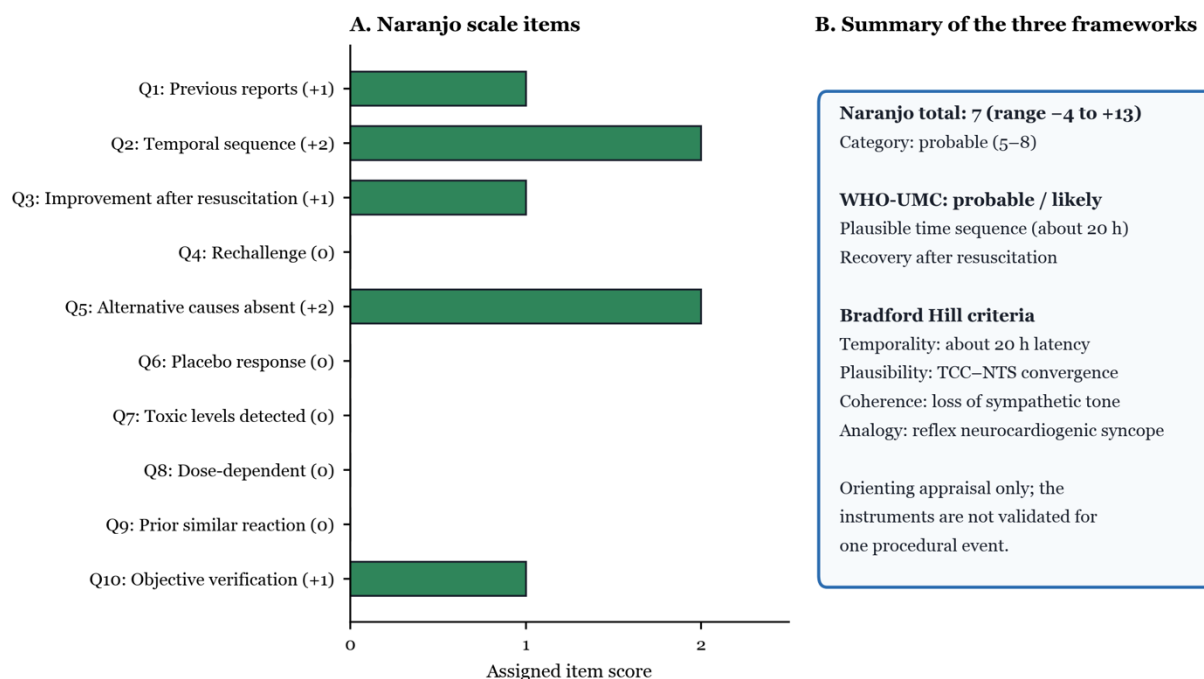
Three standardized frameworks were applied to attribute the event to the procedure.⁴⁸ The Naranjo Adverse Drug/Procedure Reaction Probability Scale gave a total of 7. That places the syncopal event in the "Probable" category (scores 5–8 denote probable ADR). Points were awarded for earlier documentation of autonomic reflexes after spinal interventions (+1), a clear temporal sequence with onset about 20 hours after the procedure (+2), definite clinical reversal after vagolytic measures and fluid resuscitation (+1), absence of other systemic causes such as sepsis, stroke or myocardial infarction (+2; the concurrent medications discussed below were not counted as alternative causes), and objective confirmation by continuous telemetry and blood pressure tracking (+1). The WHO-UMC

criteria independently classed the event as "Probable/Likely". The reasons were a credible time relationship, a response to de-escalation and resuscitation, and no reasonable alternative disease explanation. The Austin Bradford Hill criteria were also met on the main points. These were temporality (onset about 20 hours after the intervention), biological plausibility (the trigeminocervical-vagal neuroanatomical convergence), coherence (agreement with autonomic physiology and sudden loss of sympathetic tone) and analogy (resemblance to neurocardiogenic reflex syncope). Table 4 itemizes the scores for the Naranjo scale, the WHO-UMC framework and the Bradford Hill criteria. Figure 5 presents the itemized scores and the agreement across frameworks as a visual synthesis. The assessment is orienting only: the instruments are not validated for a single procedural event.

Table 4. Structured Causality Assessment Matrix (Naranjo ADR Probability Scale, WHO-UMC Assessment, and Bradford Hill Epidemiological Framework)

Causality Assessment Framework	Evaluated Parameter / Question	Assigned Score / Rating	Clinical Rationale and Evidence in Current Case
Naranjo ADR Scale (#1)	Previous conclusive reports on reaction?	+1 (Yes)	Rare neuro-autonomic syncopal reactions reported after cervical interventions
Naranjo ADR Scale (#2)	Adverse event appeared after suspected procedure?	+2 (Yes)	Syncopal collapse manifested precisely 20 hours post-PRF, matching neurohormonal delay
Naranjo ADR Scale (#3)	Adverse reaction improved upon resuscitation/hydration?	+1 (Yes)	Gradual spontaneous recovery over 7 hours with fluid resuscitation to GCS 15
Naranjo ADR Scale (#4)	Adverse reaction reappeared when procedure re-administered?	0 (Not done)	Re-challenge ethically contraindicated and not performed
Naranjo ADR Scale (#5)	Alternative causes that could solely explain reaction?	+2 (No)	Cranial CT negative, ECG normal sinus rhythm, electrolytes/glucose normal, no seizure
Naranjo ADR Scale (#6)	Reaction appeared when placebo was administered?	0 (Not done)	No placebo control administered in clinical setting
Naranjo ADR Scale (#7)	Drug/procedure detected in blood/tissues in toxic levels?	0 (Not done)	Sub-thermal PRF does not involve systemic circulating toxins
Naranjo ADR Scale (#8)	Reaction more severe when dose/energy was increased?	0 (Not applicable)	Standard 120-second 45-V protocol delivered bilaterally across 4 target sites
Naranjo ADR Scale (#9)	Patient had similar reaction to same/similar intervention?	0 (No history)	No prior history of vasovagal syncope or orthostatic collapse
Naranjo ADR Scale (#10)	Adverse event confirmed by objective evidence?	+1 (Yes)	Documented sinus bradycardia (58 bpm), hypotension (102/62 mmHg), GCS drop to 12
Naranjo Final Score	Cumulative Total Score	7 (Probable ADR)	Score 5–8 indicates 'Probable' on this scale; orienting appraisal only, because the scale is not validated for one procedural event
WHO-UMC Causality	Global Causality Category	Probable / Likely	Plausible time sequence, objective recovery, absence of alternative disease etiology
Bradford Hill: Temporality	Time sequence of intervention and outcome	Robust (+)	Delayed 20-hour latency matches central neuro-autonomic resetting and baroreflex decay
Bradford Hill: Biological Plausibility	Plausibility of neuro-autonomic mechanism	Robust (+)	Trigemino-cervical complex afferent convergence into nucleus tractus solitarius (NTS)
Bradford Hill: Coherence	Coherence with known autonomic physiology	Robust (+)	Abrupt withdrawal of chronic sympathetic overdrive unmasks unopposed vagal outflow
Bradford Hill: Analogy	Resemblance to known syndromes	Robust (+)	Resemblance to neurocardiogenic reflex syncope

Notes: ADR: Adverse drug/procedure reaction; WHO-UMC: World Health Organization-Uppsala Monitoring Centre; PRF: Pulsed radiofrequency; HR: Heart rate; MAP: Mean arterial pressure; GCS: Glasgow Coma Scale; NTS: Nucleus tractus solitarius. Naranjo score interpretation: Definite (≥ 9), Probable (5–8), Possible (1–4), Doubtful (≤ 0).

**Figure 5.** Causality appraisal: Naranjo item scores (A) and summary of the three frameworks (B). The appraisal is orienting only; the instruments are not validated for a single procedural event.

At the two-week clinic visit there was no recurrence of syncope, orthostatic dizziness or presyncope. The pain outcome was favorable. Cervical radicular pain fell from a baseline NRS of 3–4 at rest and 5–6 on movement to 1–2 at 14 days, and analgesia was sustained at six weeks, when diclofenac and amitriptyline were tapered. The pain itself changed in character, from sharp, lancinating electric shocks to mild, localized, dull muscular soreness around

the needle entry sites. On re-examination the provocative root tests had turned negative on both sides (Lhermitte's sign negative, Spurling's test negative). The patient also noted that her chronic tinnitus and spontaneous vertigo had clearly eased. She had had no cluster-like headache since the procedure.

4. Discussion

4.1. Principal findings

This single-patient observational study documents a rare and serious episode. It was an acute, severe vasovagal syncope with severe hemodynamic collapse and altered consciousness, and it appeared exactly twenty hours after fluoroscopy-guided PRF of the upper cervical dorsal root ganglia and occipital nerves.⁴⁹ PRF is widely seen as a very safe, tissue-sparing technique. It avoids the thermal destruction, neuroma formation and permanent deafferentation that continuous radiofrequency ablation can cause. This case points to an underappreciated neuro-autonomic vulnerability that could be serious.⁵⁰ A delayed, potentially serious autonomic collapse after an uneventful outpatient procedure challenges present assumptions about post-interventional monitoring windows. Upper cervical neuro-autonomic physiology needs a fundamental re-examination.⁵¹

4.2. Reflex syncope and trigeminocervical convergence

To judge the biological plausibility and the timing of the collapse, we must first look closely at reflex vasovagal syncope, a classic reflex syncope in which global cerebral hypoperfusion results from a systemic failure of autonomic cardiovascular control.⁵² The reflex arc has three levels: an afferent sensory limb, central medullary integration and an efferent limb with two parts. The efferent limb combines vagal hyperactivation (cardioinhibition) with withdrawal of sympathetic vasomotor tone (vasodepression).⁵³ In our patient the dysregulation was severe. Systolic arterial pressure fell by 37.4% to a nadir of 102/62 mmHg (MAP 75.33 mmHg, -30.04%). The sinus rate slowed by 27.5% to 58 beats per minute. These changes lowered the rate-pressure product by 54.6% (from 13,040 to 5,916 bpm·mmHg) and were accompanied by a fall of the Glasgow Coma Scale from 15 to 12, which suggests reduced cerebral perfusion.

The neuroanatomical link between upper cervical neuromodulation and strong vagal reflex activation lies in the trigeminocervical complex (TCC).⁵⁴ The sensory dorsal root ganglia of C1 to C4 and the greater and lesser occipital nerves converge extensively and directly with the descending spinal tract of the trigeminal nerve, in the upper cervical dorsal horns. Afferent signals from the upper cervical dermatomes and suboccipital structures project continuously to second-order neurons in this shared complex, so nociceptive, proprioceptive and artificial electrical impulses cross-communicate freely. The TCC also has dense polysynaptic connections to brainstem autonomic relay centers, above all the nucleus tractus solitarius (NTS) in the dorsal medulla. The NTS is the main central station for arterial baroreceptor input, peripheral chemoreceptor signals and cardiac vagal afferents. It regulates sympathetic outflow through the rostral ventrolateral medulla and parasympathetic outflow through the nucleus ambiguus and the dorsal motor nucleus of the vagus. During PRF, pulsed electric fields give neuromodulatory stimulation to this sensitive craniocervical junction. This is the substrate for a delayed, abnormal reflex arc.

4.3. Latency and proposed mechanisms

The long latency is the defining feature of this case. Twenty hours passed between the end of the procedure and the collapse. Classical vasovagal reactions to needle trauma, acute visceral pain or anxiety appear during needle advancement or within minutes of local anesthetic infiltration.⁵⁵ A delay of twenty hours makes acute mechanical puncture and direct intravascular local anesthetic toxicity unlikely. It may suggest instead a slow, multi-step neurobiological and neurohormonal readjustment. In our hypothesized cascade, two mechanisms act together: the abrupt withdrawal of chronic sympathetic overdrive, and a delayed trigeminocervical-NTS reflex reset. Concurrent vasodilatory drugs add to the effect. Figure 6 shows this hypothesized model; none of its steps was measured.

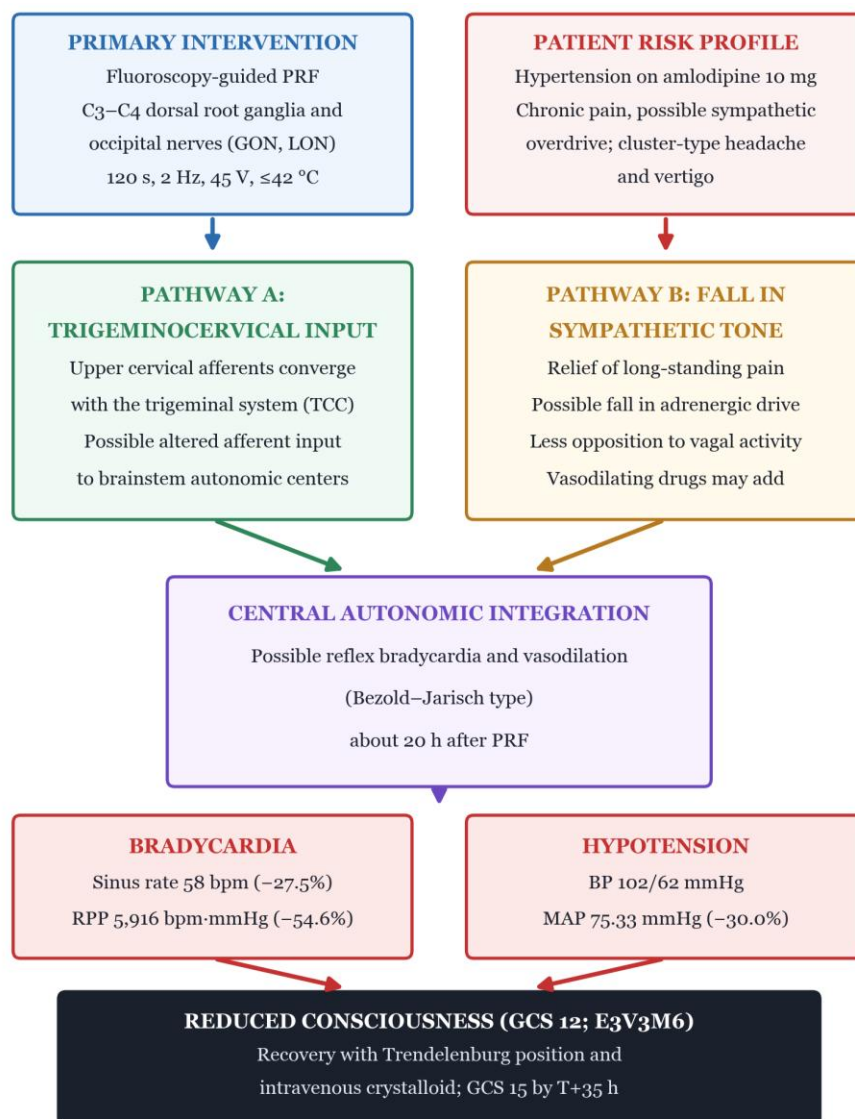


Figure 6. Hypothesized pathway.

The first mechanistic pillar is the cardiovascular effect of abrupt sympathetic withdrawal after successful pain relief.⁵⁰ Patients with severe, long-standing neuropathic pain, like our patient's cervical root syndrome and occipital neuralgia, live in a state of chronic sympathetic overactivity. Constant nociceptive input from sensitized cervical roots drives central adrenergic stimulation. Circulating catecholamines rise, systemic vascular resistance increases, and baroreceptor thresholds reset upward to hold blood pressure higher. This fits our patient. Her baseline was 163/80 mmHg with a MAP of 107.67 mmHg. PRF produces strong analgesia. Its pulses are reported to modulate c-Fos expression in the dorsal horn, inhibit microglial inflammatory cascades and induce long-term depression (LTD) at nociceptive synapses. When nociceptive traffic stops suddenly and successfully, the main driver of chronic central sympathetic tone disappears. Sympathetic adrenergic vasoconstriction then fades over the next twelve to twenty hours, and the autonomic nervous system is left with unopposed parasympathetic tone. The threshold of the Bezold-Jarisch reflex falls. Ventricular mechanoreceptors trigger that inhibitory cardiopulmonary reflex when they sense reduced cardiac filling, and it causes profound bradycardia, peripheral vasodilation and arterial hypotension.

The second pillar is a convergence of drug effects and host factors.⁵⁵ A clinical audit found that the patient took oral amlodipine 10 mg daily for recently diagnosed essential hypertension. Amlodipine is a long-acting dihydropyridine calcium channel blocker. Its therapeutic effect is predominantly arteriolar vasodilation. With that baseline vasodilation in place, the sudden loss of sympathetic vasoconstrictor tone impaired the compensatory baroreflex tachycardia and vasoconstriction. She also had cluster-like headache and chronic vertigo on record, and both reflect existing central sensitization and autonomic lability in the trigeminovascular and vestibulocochlear axes. Three things coincided: abrupt withdrawal of sympathetic tone, amlodipine-mediated vasodilation and delayed trigeminocervical afferent input to the NTS. Baroreceptor compensation failed. Uncompensated vagal outflow followed, with profound cardioinhibitory bradycardia, systemic vasodepression and transient global cerebral hypoperfusion.

4.4. Comparison with the published literature

Table 5 sets our findings against published reports.^{33–38} The comparison suggests that the delay is unusual. In prospective studies of cervical radiofrequency, intraoperative vasodepression is usually mild and resolves quickly when the procedure is paused. Selective cervical nerve root blocks and epidural steroid injections occasionally cause

transient vasovagal reactions. Almost all of them occur within thirty minutes of needle insertion. In every earlier

cohort the autonomic reactions were acute, transient and tied to mechanical needle stimulation.

Table 5. Global Comparative Literature Benchmarking: Autonomic Complications, Timing, and Latency Profiles in Upper Cervical Interventions

Author & Year	Interventional Modality & Target	Reported Autonomic Complication	Onset Latency	Proposed Pathophysiological Mechanism	Management & Resolution
Current Study (2026)	Bilateral C3–C4 DRG + Occipital PRF	Delayed Vasovagal Syncope (GCS 12, MAP 75.3 mmHg)	20 hours (Delayed)	Trigemino-cervical NTS reflex + abrupt sympathetic withdrawal	Modified Trendelenburg, IV Crystalloids; full recovery in 7 h
Lan et al. (2021) [Front Surg] ³³	Low-temperature plasma ablation, cervical	Transient intraoperative vasodepression (1.2%)	Immediate (0–5 min)	Direct carotid sinus irritation and needle-related visceral pain	Fluid challenge, procedure pause; resolved in 15 min
Yildiz et al. (2024) [Pain Physician] ³⁴	Selective nerve root PRF vs epidural steroid	Minor post-procedural presyncope (2.4%)	<30 min post-op	Mild orthostatic pooling and post-procedural anxiety	Supine positioning and oral hydration; uneventful
Dong & Li (2024) [Front Med] ³⁵	Ultrasound + DSA-guided PRF, perineal herpes	Vasovagal bradycardia and nausea (3.1%)	Immediate intra-op	Pelvic parasympathetic afferent stimulation during needle entry	Intravenous atropine 0.5 mg; restored within 10 min
Aksu et al. (2021) [Auton Neurosci] ³⁶	Cardioneuroablation for vasovagal syncope	Autonomic tone recalibration post-ganglion	Days to weeks	Parasympathetic ganglionic plexus attenuation at cardiac fat pads	Sustained blunting of cardioinhibitory syncope recurrences
Neymark & Seifer (2026) [Curr Opin Physiol] ³⁷	Autonomic syncope in females with vasodilators	Severe reflex hypotension and syncope	Variable (hours)	Baroreceptor resetting failure exacerbated by dihydropyridines	Medication review, volume loading, physical counter-maneuvers
Hou et al. (2026) [Acta Neurol Belg] ³⁸	Cerebral autoregulation dysfunction syncope	Cerebral hypoperfusion with GCS alteration	Delayed post-stress	Primary baroreflex failure and cerebral vascular dysregulation	Volume resuscitation, midodrine, avoidance of vasodilators

Notes: PRF: Pulsed radiofrequency; DRG: Dorsal root ganglion; GCS: Glasgow Coma Scale; MAP: Mean arterial pressure; NTS: Nucleus tractus solitarius; DSA: Digital subtraction angiography; SBP: Systolic blood pressure; HR: Heart rate. Current study documents the unique 20-hour delayed vasovagal collapse following multi-level cervical and occipital PRF neuro-modulation. Studies are cited from the reference list; comparison with this case is descriptive.

4.5. Resuscitation and clinical implications

The resuscitation protocol worked. It teaches lessons for acute care and perioperative resuscitation. The crisis combined severe cardioinhibition (heart rate 58 bpm) with severe vasodepression (blood pressure 102/62 mmHg). Simple conservative maneuvers, such as isometric muscle tensing or supine positioning, were therefore not enough. The 30-degree Trendelenburg position used gravity for venous autotransfusion and raised cardiac preload, and a rapid infusion of 1,000 mL of isotonic crystalloid restored effective circulating volume. Intravenous atropine sulfate 0.5 mg was given. As a competitive antagonist at postganglionic muscarinic acetylcholine receptors, it ended vagal hyperinhibition of the sinoatrial node. Heart rate rose from 58 to 64 bpm and mean arterial pressure from 75.33 to 82.67 mmHg within the first hour, with further gradual recovery thereafter. This course is compatible with a functional, vagally mediated collapse, although fluid and positioning were given at the same time.²⁰ Cardiac enzymes, serial troponins and the 12-lead electrocardiogram showed no intrinsic myocardial injury, no acute coronary syndrome and no structural conduction defect.

4.6. Neuro-autonomic anatomy, medication and atropine response

The upper cervical spine has a distinct anatomical interface between somatic sensory afferents and autonomic efferent projections. The C3 and C4 dorsal root ganglia relay proprioceptive, mechanoreceptive and nociceptive traffic from the deep paraspinal muscles, facet joints and cervical discs. Pulsed fields (45 V) applied near these ganglia do not stay confined to the targeted sensory cell bodies. Local ionic fluxes and small electromagnetic fluctuations spread through the neighboring neural tissue and inevitably change the firing thresholds of nearby unmyelinated sympathetic postganglionic fibers that run in the gray rami communicantes. Under normal conditions these rami give tonic vasomotor drive to the cranial and cervical vessels. PRF may disturb that background output for a time and create a quiet, subclinical regional dysautonomia. It stays silent while the patient lies supine and rests. Orthostatic

shifts or delayed central neurohormonal readjustment would bring it out.

Concurrent medication also contributed to the delayed collapse. This is a clinical lesson of some weight. Patients with chronic, intractable neuropathic pain often take complex polypharmacy for fragmented sleep, mood disturbance and muscle spasm. Our patient took oral amitriptyline, a tricyclic with anticholinergic and alpha-adrenergic blocking properties, together with diazepam and amlodipine. Amitriptyline usually raises resting heart rate through its anticholinergic action. Its peripheral alpha-adrenergic antagonism, however, can impede the compensatory vasoconstriction needed during sudden hypotension. Amlodipine relaxes vascular smooth muscle continuously and strongly. That blunts the ability of the arterioles to constrict quickly against a central parasympathetic surge. Pre-existing peripheral vasodilation, tricyclic blunting of autonomic responses and the sudden procedural loss of chronic pain-driven sympathetic overdrive left her hemodynamic balance precarious. When the delayed trigemino-cervical reflex arc reached peak central integration at twenty hours, her own compensatory reserves were quickly overwhelmed. Profound cardioinhibition, systemic vasodepression and cerebral hypoperfusion followed.^{53,55}

For acute care and resuscitation, the effect of intravenous atropine on the heart rate is compatible with a vagally mediated event. Atropine competitively blocks muscarinic acetylcholine receptors and stops parasympathetic efferent signals at the sinoatrial and atrioventricular nodes. Heart rate returned toward baseline over the following hours, from 58 to 76 beats per minute by the next morning. This shows that the bradycardia did not come from intrinsic nodal ischemia, structural conduction necrosis or electrical tissue destruction by the radiofrequency cannula. Mean arterial pressure also stabilized after fluid loading and Trendelenburg positioning. Systemic vascular capacitance was restored as central venous return came back. Taken together, these findings suggest that the delayed neuro-autonomic syncope in this patient was most likely a functional, reflex-mediated episode and not a permanent structural neurological lesion. Her prognosis was good, and early recognition and targeted resuscitation probably contributed to it.

4.7. Strengths

The report has methodological strengths, including dense longitudinal hemodynamic tracking, descriptive handling appropriate to a single subject, and a structured causality appraisal with three frameworks.

4.8. Limitations

Its limits are equally clear. As a single-patient observational case study (n=1), it cannot establish population-level incidence or prove universal causality across demographic strata. The collapse came without warning, twenty hours after the procedure. Minute-by-minute invasive arterial pressure waveforms and serial serum catecholamines were therefore not recorded before the crisis.

4.9. Recommendations for future research

Future research should build prospective, multicenter observational registries. These should use continuous non-invasive perioperative heart rate variability (HRV) telemetry and baroreflex sensitivity monitoring in patients having upper cervical neuromodulation. Such monitoring could show whether shifts in low-frequency/high-frequency spectral power identify patients with hidden autonomic vulnerability. In practice, pain specialists and acute care clinicians should be watchful when performing upper cervical PRF in patients who take vasodilators. Standardized extended observation pathways would protect these patients.

5. Conclusion

Fluoroscopy-guided PRF of the upper cervical dorsal root ganglia and occipital nerves remains a safe, non-destructive and effective treatment for refractory cervical radiculopathy and occipital neuralgia. It may, however, be followed by a rare but serious delayed neuro-autonomic syncope. This study shows that serious autonomic decompensation, with cardioinhibitory bradycardia, marked hypotension and acute neurological impairment, can appear twenty hours after an uneventful procedure.

The mechanism may involve neuroanatomical convergence in the trigeminocervical complex, abnormal reflex transmission to the nucleus tractus solitarius, and abrupt, uncompensated loss of chronic pain-driven sympathetic overdrive. A vasodilating calcium channel blocker probably adds to it. Pain physicians and acute care teams should be aware that PRF complications are not always immediate or local. Safe practice may call for three things. They are pre-procedural risk stratification, a review of concurrent vasodilating drugs, extended inpatient hemodynamic monitoring for high-risk patients, and readiness to give a vagolytic together with volume resuscitation.

6. Declarations

Ethical approval and consent to participate. The study was approved by CMHC Research Center Ethics Committee, Indonesia (Protocol Approval Number: 2026/4108). The work followed the ethical principles of the Declaration of Helsinki. The patient gave written informed consent directly before the procedure and the scientific evaluation.

Consent for publication. The patient gave written informed consent for publication of this study and of the accompanying clinical parameters, radiological findings and procedural documentation.

Availability of data and materials. All data generated or analyzed in this study are included in the article, its tables and its figures. The corresponding author can provide further anonymized clinical records on reasonable request.

Artificial intelligence (AI) use statement. The authors declare that no generative artificial intelligence tool was used in the preparation of this manuscript.

Author contribution statement. K.H.W. contributed to conceptualization, investigation (procedures and resuscitation) and writing – original draft. B.H.L. contributed to supervision, formal analysis (pathophysiological interpretation) and writing – review and editing. Both authors approved the final version.

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Conflict of interest (competing interests) statement. The authors declare that they have no competing interests.

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