

CASE REPORT

Delayed Reflex Bradycardia and Hypotension 24 Hours After Fluoroscopy-Guided Upper Cervical and Occipital Pulsed Radiofrequency: A Case Report

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ABSTRACT

Background. Pulsed radiofrequency (PRF) is a tissue-sparing treatment for refractory cervical radicular pain and occipital neuralgia and is generally regarded as safe. Delayed autonomic complications after upper cervical PRF are rarely reported.

Objective. To describe severe bradycardia, hypotension and reduced consciousness 24 hours after upper cervical and occipital PRF and to discuss possible mechanisms.

Methods. A 41-year-old woman with cervical root syndrome (C3–C6), occipital neuralgia and treated hypertension had fluoroscopy-guided PRF (120 s, 2 Hz, 45 V, maximum 42 °C) of the bilateral C3 and C4 dorsal root ganglia and the occipital nerves. Blood pressure, heart rate and Glasgow Coma Scale (GCS) were recorded at ten time points over 14 days.

Results. The procedure was uneventful, and hemodynamics were stable for 12 hours. At 24 hours the patient collapsed: blood pressure 75/40 mmHg (mean arterial pressure 51.67 mmHg, –52.01% from baseline), heart rate 42 bpm (–47.50%) and GCS 12. Electrocardiogram, troponin I and glucose excluded cardiac and metabolic causes. Trendelenburg positioning, 1,000 mL of crystalloid and atropine 0.5 mg restored heart rate (78 bpm) and mean arterial pressure (83.33 mmHg) within 60 minutes; GCS was 15 at 36 hours. At 14 days there was no recurrence, and neck-movement pain had fallen from 5–6 to 4–5 on the numeric rating scale.

Conclusion. Severe but reversible bradycardia and hypotension can occur 24 hours after upper cervical PRF. A reflex mechanism is plausible but was not measured, and one case cannot establish causation. Longer observation may be considered in patients on vasodilators.

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1. Introduction

Cervical radicular pain with coexisting occipital neuralgia is a common and difficult neuropathic disorder, and neck pain places a heavy burden on health systems.¹ Cervical radiculopathy has an annual incidence of roughly 83 per 100,000 people, and most cases are related to spondylosis, disc disease or both.² It causes radicular pain with sensory and motor deficits in the arm. Occipital neuralgia adds lancinating pain along the greater and lesser occipital nerves.³ Interventional treatment is considered when conservative drug therapy and physical rehabilitation fail.

Pulsed radiofrequency (PRF) is widely used as a minimally invasive option for radicular pain^{4–7} and for occipital neuralgia.^{3,8} Continuous radiofrequency ablation heats tissue above 80 °C and causes coagulation necrosis and Wallerian degeneration. PRF delivers short bursts of current separated by silent intervals instead, so that heat dissipates and tissue temperature stays below 42 °C, at which nerve damage is rare.⁹ PRF therefore avoids permanent deafferentation pain, dysesthesia, neuroma formation and motor deficit, and its safety record is favorable.⁹ Its analgesic effect is

attributed to neuromodulation rather than tissue destruction. Proposed mechanisms include activation of dorsal horn lamina I and II neurons with c-Fos expression,¹⁰ suppression of microglial signaling,¹¹ inhibition of Cav2.2 calcium channels in the dorsal horn and ganglion,¹² and long-term depression of synaptic transmission with enhanced descending noradrenergic and serotonergic inhibition.⁹

The autonomic effects of PRF at the junction of the upper cervical spine and the suboccipital region are rarely studied. Neuromodulation of the trigeminocervical complex combined with vagal stimulation has systemic effects, such as improved hyperglycemia in type 2 diabetes.¹³ The upper cervical dorsal root ganglia and the occipital nerves are linked to the trigeminocervical complex, where occipital and trigeminal afferents converge, and blocking the greater occipital nerve modulates nociceptive signals in this complex.¹⁴ Stimulation of trigeminal afferents can cause reflex parasympathetic dysrhythmia and hypotension (the trigeminocardiac reflex), which shows that trigeminal input reaches brainstem autonomic pathways.¹⁵ The nucleus tractus solitarius integrates baroreceptor and other visceral input and, with the rostral ventrolateral medulla and the

vagal nuclei, sets sympathetic and parasympathetic outflow.¹⁶ Whether neuromodulation of the upper cervical ganglia and occipital nerves can disturb this balance is unknown. Sympathetic fibers contribute to neuropathic pain,¹⁷ and chronic pain is associated with reduced heart rate variability.¹⁸ In vasovagal syncope, an afferent trigger causes vagal activation together with loss of sympathetic vasomotor tone, which leads to bradycardia, hypotension and cerebral hypoperfusion.¹⁹ Vasovagal reactions during spine procedures are reported in 2.6% of fluoroscopically guided procedures and in 8% of cervical translaminar epidural injections and rarely cause serious harm.^{20,21} A severe collapse 24 hours after an uneventful PRF has rarely been described.^{22,23} It is unclear whether such events arise from gradual neurohormonal readjustment, an abnormal trigeminocervical–vagal reflex or hemodynamic decompensation after abrupt relief of pain-driven sympathetic activity.

This report describes a patient who developed severe bradycardia, hypotension and reduced consciousness 24 hours after fluoroscopy-guided PRF of the bilateral C3 and C4 dorsal root ganglia and the occipital nerves, and it discusses possible mechanisms.

2. Methods

2.1. Report design and reporting guideline

This is a single case report prepared in accordance with the CARE guideline.²⁴ Data were taken from the clinical record and from the procedural and monitoring logs.

2.2. Setting

The patient was treated at Dr. Saiful Anwar Regional General Hospital, Malang, Indonesia, in the Pain Management Division of the Department of Anesthesiology and Intensive Therapy. PRF was performed on day 0. The patient was observed on the inpatient ward and followed for 14 days.

2.3. Patient information and case selection

The patient was a 41-year-old premenopausal woman (height 155 cm, weight 50 kg, body mass index 20.8 kg/m², body surface area 1.47 m²) with a six-month history of severe neck pain radiating to both shoulders and the occipital region. PRF was indicated by chronic cervical radicular pain lasting longer than three months, magnetic resonance imaging showing concordant cervical neural compression, coexisting occipital neuralgia that met the International Classification of Headache Disorders, third edition (ICHD-3) criteria, and failure of at least six weeks of multimodal pharmacotherapy, physical therapy and postural rehabilitation. A multidisciplinary team of anesthesiologists, interventional pain specialists and spine surgeons found no contraindication to PRF: no progressive motor deficit, spinal infection, coagulopathy, anticoagulant therapy, structural heart disease, arrhythmia or pacemaker. Her history included recurrent cluster-like headache, chronic spontaneous vertigo, tinnitus and recently diagnosed essential hypertension treated with amlodipine 10 mg daily. Other medicines were amitriptyline 25 mg at night, diclofenac sodium 50 mg twice daily, betahistine mesylate 6 mg three times daily and diazepam 5 mg as needed. The case is reported because of the severity of the event and its delay after a procedure that is generally regarded as safe.

2.4. Case timeline

The timeline is shown in Table 1. Day 0 is the day of PRF. Time points are given in hours after the end of the procedure (T+), and clock times are given for the day of the event. The clock times in the record imply that the procedure ended at about 16:00 on day 0.

2.5. Clinical findings and diagnostic assessment

Before the procedure the patient had a history and neurological examination that covered the cranial nerves, myotomes C5–T1, deep tendon reflexes, pathological reflexes (Hoffman, Babinski and Chaddock signs) and the provocative tests Spurling's test and Lhermitte's sign. Cervical magnetic resonance imaging was reviewed. Pain was rated on the numeric rating scale (NRS, 0–10). When the event occurred, the assessment consisted of vital signs, GCS, peripheral perfusion, arterial blood gas analysis, capillary glucose, a twelve-lead electrocardiogram and serial troponin I.

2.6. Therapeutic intervention

Standard American Society of Anesthesiologists (ASA) monitoring was used: five-lead electrocardiography, non-invasive blood pressure, pulse oximetry and capnography. Light sedation was given with intravenous propofol 0.5 mg/kg and midazolam 0.01 mg/kg, which kept the patient able to answer questions during stimulation testing.

With the patient prone and the cervical spine flexed, fluoroscopy identified the C3 and C4 neural foramina. A 22-gauge, 10-cm radiofrequency cannula with a 10-mm curved active tip was advanced through a posterolateral oblique approach. Anteroposterior and lateral views confirmed placement at the posterior part of the foramen next to the dorsal root ganglion. Impedance was measured. Sensory stimulation at 50 Hz was used to reproduce concordant paresthesias at 0.40–0.60 V. Motor stimulation at 2 Hz was tested up to 1.50 V to confirm that no fasciculation occurred. After 1.0 mL of 1% preservative-free lidocaine was injected, PRF was delivered for 120 seconds at 2 Hz with a pulse width of 20 milliseconds and a peak voltage of 45 V, and the generator limited tip temperature to 42.0 °C. The sequence was repeated at the contralateral levels and at the bilateral greater and lesser occipital nerves, using combined ultrasound and fluoroscopic landmarks. The patient then went to the inpatient ward for continuous automated telemetry, hourly nursing charts and serial pain assessment.

When the event occurred, the patient was placed in 30-degree Trendelenburg position and given oxygen at 10 L/min by non-rebreather mask and warm isotonic crystalloid through two large-bore peripheral lines (1,000 mL over 30 minutes). Atropine sulfate 0.5 mg was given intravenously because symptomatic bradycardia persisted.

2.7. Outcome measures and follow-up

Systolic blood pressure (SBP), diastolic blood pressure (DBP), heart rate (HR) and GCS were recorded at ten time points: before the procedure (T0), at T+2, T+12, T+23.5, T+24, T+25, T+28, T+31 and T+36 hours, and at day 14. Mean arterial pressure (MAP) was calculated as $DBP + 1/3(SBP - DBP)$ and the rate-pressure product (RPP) as $HR \times SBP$. Change from baseline was expressed as $\Delta\% = [(T_n - T_0)/T_0] \times 100\%$. Pain was rated on the NRS at baseline and at day 14. Follow-up was a clinic visit at 14 days. Causality was described with the Naranjo scale,²⁵ the WHO-UMC sys-

tem²⁶ and the Bradford Hill criteria.²⁷ These tools were designed for drug reactions or for population-level inference. They are used here only as a structured description of one event and do not prove causation. The data are described only; no inferential statistics were used, because they do not apply to one patient.

2.8. Ethics and consent

CMHC Research Center Ethics Committee, Indonesia, approved the report (protocol approval number 2026/4182). The work followed the Declaration of Helsinki. The patient gave written informed consent for the procedure and for publication of her anonymized clinical data, laboratory values, imaging and serial hemodynamic records. All direct and indirect identifiers were removed.

3. Results

3.1. Presentation and baseline findings

At baseline (T0) blood pressure was 163/80 mmHg (MAP 107.67 mmHg), heart rate 80 bpm in sinus rhythm, respiratory rate 16 breaths/min, SpO₂ 98% on room air and RPP

13,040 bpm·mmHg. GCS was 15 (E4V5M6). She had subjective right-sided tinnitus and mild sensorineural hearing loss, with no focal motor or sensory trigeminal deficit. Strength was 5/5 in all C5–T1 myotomes bilaterally, although effort was limited by pain. Deep tendon reflexes were symmetric and brisk (+2), and Hoffman, Babinski and Chaddock signs were negative. Spurling's test and Lhermitte's sign were both positive bilaterally, and the suboccipital muscles had prominent tender trigger points. Pain was 3–4 on the NRS at rest and 5–6 on cervical extension and lateral rotation. Magnetic resonance imaging showed multilevel degenerative spondylosis, with severe canal stenosis and bilateral foraminal narrowing at C5–C6 from a posterior disc herniation and mild canal stenosis at C4–C5. Endplate changes were Modic type II, and disc desiccation was Pfirrmann grade III. The baseline data are in Table 2.

Table 1. Clinical timeline of the case.

Time point	Clinical event	Investigation and result	Intervention
Before PRF (T0)	Neck pain with radicular and occipital features	Examination and MRI as in Table 2; NRS 3–4 at rest, 5–6 on movement	—
Day 0, PRF	PRF of the bilateral C3 and C4 DRG and the occipital nerves; 45 min; uneventful	Impedance 325–410 Ω; sensory thresholds 0.40–0.60 V; no motor response up to 1.50 V	Propofol 0.5 mg/kg, midazolam 0.01 mg/kg; lidocaine 1% 1.0 mL per target; PRF 120 s per target
T+2 h, T+12 h	Ward observation; relief of neck stiffness; slept comfortably	Hemodynamics in Table 4; GCS 15	—
T+23.5 h (15:30)	Prodrome: cold sweating, facial pallor, lightheadedness, nausea	Blood pressure 118/68 mmHg, HR 58 bpm (Table 4); GCS 15	—
T+24 h (16:00)	Collapse; weak, thready pulses; capillary refill >3.5 s; cold, clammy skin	BP 75/40 mmHg, HR 42 bpm, GCS 12 (E3V3M6); glucose 108 mg/dL; ECG sinus bradycardia without ST deviation, block or QT prolongation; troponin I <0.01 ng/mL; blood gas without hypoxemia or acidosis	Trendelenburg 30°; oxygen 10 L/min; crystalloid 1,000 mL over 30 min; atropine 0.5 mg IV
T+25 h (17:00)	Heart rate and pressure recovering	BP 110/70 mmHg, HR 78 bpm, GCS 13 (E3V4M6)	—
T+28 h, T+31 h	Stable sinus rhythm on telemetry; speech and perfusion returning	Table 4; GCS 14	—
T+36 h	Alert and oriented; warm extremities; ward discharge	BP 130/78 mmHg, HR 76 bpm, GCS 15 (E4V5M6)	—
Day 14	No syncope, orthostatic dizziness or presyncope	NRS 4–5 on movement; Spurling's test and Lhermitte's sign negative; less tinnitus and vertigo; no cluster-like headache	—

Notes: BP, blood pressure; DRG, dorsal root ganglia; ECG, electrocardiogram; GCS, Glasgow Coma Scale; HR, heart rate; IV, intravenous; MRI, magnetic resonance imaging; NRS, numeric rating scale; PRF, pulsed radiofrequency. T+ is the time after the end of the procedure; clock times are those of the day of the event.

Table 2. Baseline characteristics of the patient.

Parameter	Value	Reference range
Age, sex	41 years, female (premenopausal)	—
Height, weight	155 cm, 50 kg	—
Body mass index	20.8 kg/m ²	18.5–24.9 kg/m ²
Body surface area	1.47 m ²	—
Systolic blood pressure	163 mmHg	90–120 mmHg
Diastolic blood pressure	80 mmHg	60–80 mmHg
Mean arterial pressure	107.67 mmHg	70–100 mmHg
Heart rate	80 bpm, sinus rhythm	60–100 bpm
Rate-pressure product	13,040 bpm·mmHg	<12,000 bpm·mmHg
SpO ₂	98% on room air	≥95%
Glasgow Coma Scale	15 (E4V5M6)	15
Numeric rating scale, neck and radicular pain	3–4 at rest; 5–6 on extension and rotation	0–10
Spurling's test, Lhermitte's sign	Positive bilaterally	Negative
Suboccipital palpation	Tender myofascial trigger points	No tenderness
Cervical MRI	Severe canal stenosis and bilateral foraminal narrowing at C5–C6; mild canal stenosis at C4–C5; Modic type II; Pfirrmann grade III	—

Parameter	Value	Reference range
Other history	Recurrent cluster-like headache, chronic vertigo, tinnitus, essential hypertension	—
Regular medication	Amlodipine 10 mg daily; amitriptyline 25 mg nightly; diclofenac 50 mg twice daily; betahistine 6 mg three times daily; diazepam 5 mg as needed	—

Notes: MRI, magnetic resonance imaging; SpO₂, peripheral oxygen saturation. Mean arterial pressure = DBP + 1/3(SBP – DBP); rate-pressure product = HR × SBP.

3.2. Procedure and diagnostic findings

PRF was performed on day 0 at the bilateral C3 and C4 dorsal root ganglia and the bilateral greater and lesser occipital nerves. The total procedure time was 45 minutes (Figure 1). Impedance ranged from 325 to 410 Ω, and there was no vascular puncture or cerebrospinal fluid leak. Sensory stimulation reproduced concordant paresthesias at 0.40–

0.60 V, and motor stimulation produced no fasciculation up to 1.50 V (Table 3). The generator kept tip temperature between 40.0 and 42.0 °C. The patient tolerated the procedure, remained hemodynamically stable in the operating room and was transferred to the ward for 24-hour observation.

Table 3. PRF targets, impedance and stimulation thresholds.

Target	Impedance (Ω)	Sensory threshold, 50 Hz (V)	Motor threshold, 2 Hz
Right C3 dorsal root ganglion	340	0.45 (concordant paresthesia)	>1.5 V, no fasciculation
Left C3 dorsal root ganglion	365	0.50 (concordant paresthesia)	>1.5 V, no fasciculation
Right C4 dorsal root ganglion	380	0.40 (concordant paresthesia)	>1.5 V, no fasciculation
Left C4 dorsal root ganglion	410	0.55 (concordant paresthesia)	>1.5 V, no fasciculation
Bilateral greater occipital nerves	325–350	0.40–0.50 (occipital tingling)	>1.5 V, no motor response
Bilateral lesser occipital nerves	330–360	0.45–0.60 (auricular paresthesia)	>1.5 V, no motor response

Notes: PRF, pulsed radiofrequency. At every target: 22-gauge, 10-cm cannula with a 10-mm active tip; PRF for 120 s at 2 Hz, pulse width 20 ms, 45 V, tip temperature limited to 42.0 °C; 1.0 mL of 1% lidocaine per target before PRF.

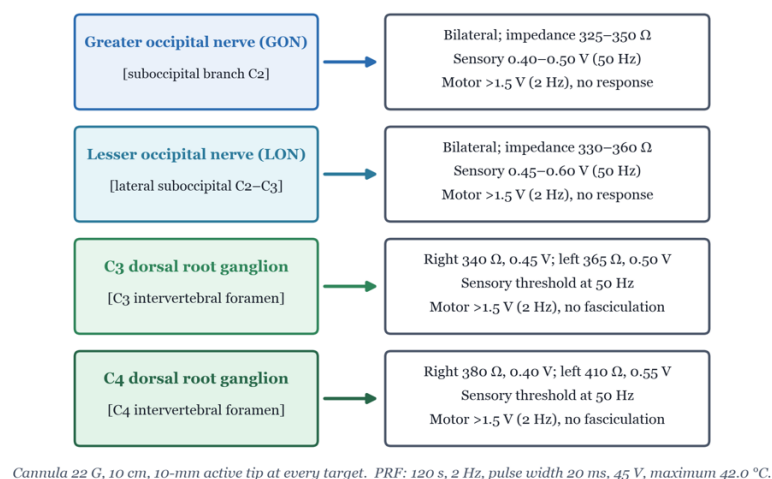


Figure 1. Targets and stimulation data of PRF (schematic, not an anatomical or fluoroscopic image). Impedance and thresholds are those in Table 3. Every target received PRF for 120 s at 2 Hz with a pulse width of 20 ms, 45 V and a maximum of 42.0 °C.

At the time of the collapse, arterial blood gas analysis and capillary glucose (108 mg/dL) excluded hypoglycemia, hypoxemia and acute metabolic acidosis. The twelve-lead electrocardiogram showed sinus bradycardia without ST-segment deviation, conduction block or QT prolongation, and serial troponin I was undetectable (<0.01 ng/mL).

3.3. Treatment course and outcome

For the first 12 hours the course was uneventful (Table 4, Figure 2). At T+2 h blood pressure was 150/78 mmHg and heart rate 76 bpm, and at T+12 h 142/75 mmHg and 72 bpm, with GCS 15 and relief of neck stiffness. At T+23.5 h

(15:30) she developed cold sweating, facial pallor, light-headedness and nausea. Blood pressure was 118/68 mmHg (MAP 84.67 mmHg, –21.36%) and heart rate 58 bpm (–27.50%). At T+24 h (16:00) she collapsed, with GCS 12 (E3V3M6). Blood pressure was 75/40 mmHg (MAP 51.67 mmHg, –52.01%), heart rate 42 bpm (–47.50%) and RPP 3,150 bpm·mmHg (–75.84%). Systolic pressure had fallen by 54.0%. The pulses were weak and thready, capillary refill exceeded 3.5 seconds and the skin was cold and clammy.

Table 4. Hemodynamic variables and Glasgow Coma Scale from baseline to day 14.

Time point	SBP (mmHg)	DBP (mmHg)	MAP (mmHg)	HR (bpm)	RPP (bpm·mmHg)	GCS	Δ% MAP	Δ% HR	Δ% RPP
T0 (baseline)	163	80	107.67	80	13,040	15	0.00	0.00	0.00
T+2 h	150	78	102.00	76	11,400	15	–5.27	–5.00	–12.58
T+12 h	142	75	97.33	72	10,224	15	–9.60	–10.00	–21.60

Time point	SBP (mmHg)	DBP (mmHg)	MAP (mmHg)	HR (bpm)	RPP (bpm·mmHg)	GCS	Δ% MAP	Δ% HR	Δ% RPP
T+23.5 h	118	68	84.67	58	6,844	15	-21.36	-27.50	-47.52
T+24 h (collapse)	75	40	51.67	42	3,150	12	-52.01	-47.50	-75.84
T+25 h	110	70	83.33	78	8,580	13	-22.61	-2.50	-34.20
T+28 h	118	72	87.33	76	8,968	14	-18.89	-5.00	-31.23
T+31 h	124	74	90.67	75	9,300	14	-15.79	-6.25	-28.68
T+36 h	130	78	95.33	76	9,880	15	-11.46	-5.00	-24.23
Day 14	132	80	97.33	74	9,768	15	-9.60	-7.50	-25.09

Notes: DBP, diastolic blood pressure; GCS, Glasgow Coma Scale; HR, heart rate; MAP, mean arterial pressure = $DBP + 1/3(SBP - DBP)$; RPP, rate-pressure product = $HR \times SBP$; SBP, systolic blood pressure. $\Delta\% = (value - T0)/T0 \times 100$.

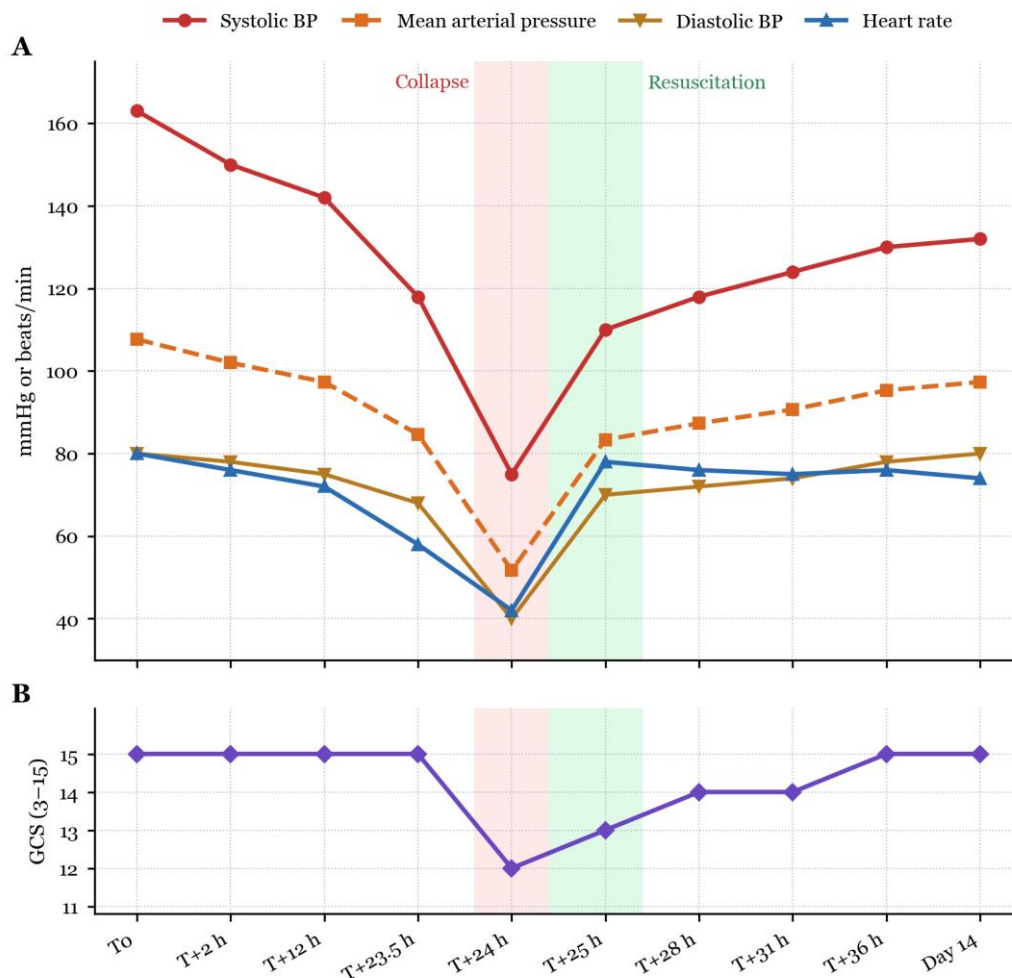


Figure 2. Blood pressure, heart rate (A) and Glasgow Coma Scale (B) from baseline to day 14. The shaded bands mark the collapse (T+24 h) and the resuscitation period. GCS at T+28 h and T+31 h is plotted as reported.

The resuscitation measures are described in section 2.6. At T+25 h (17:00) heart rate was 78 bpm (-2.50%), blood pressure 110/70 mmHg (MAP 83.33 mmHg, -22.61%), RPP 8,580 bpm·mmHg (-34.20%) and GCS 13 (E3V4M6). At T+28 h (20:00) blood pressure was 118/72 mmHg (MAP 87.33 mmHg, -18.89%), heart rate 76 bpm and GCS 14 (E4V4M6). Overnight telemetry showed stable sinus rhythm without pauses or further bradycardia. At T+31 h

(23:00) blood pressure was 124/74 mmHg (MAP 90.67 mmHg, -15.79%), heart rate 75 bpm, RPP 9,300 bpm·mmHg and GCS 14 (E4V4M6). At T+36 h (08:00) she was alert and oriented, with GCS 15 (E4V5M6) and warm extremities. Blood pressure was 130/78 mmHg (MAP 95.33 mmHg, -11.46%), heart rate 76 bpm and RPP 9,880 bpm·mmHg. The percentage changes are shown in Figure 3.

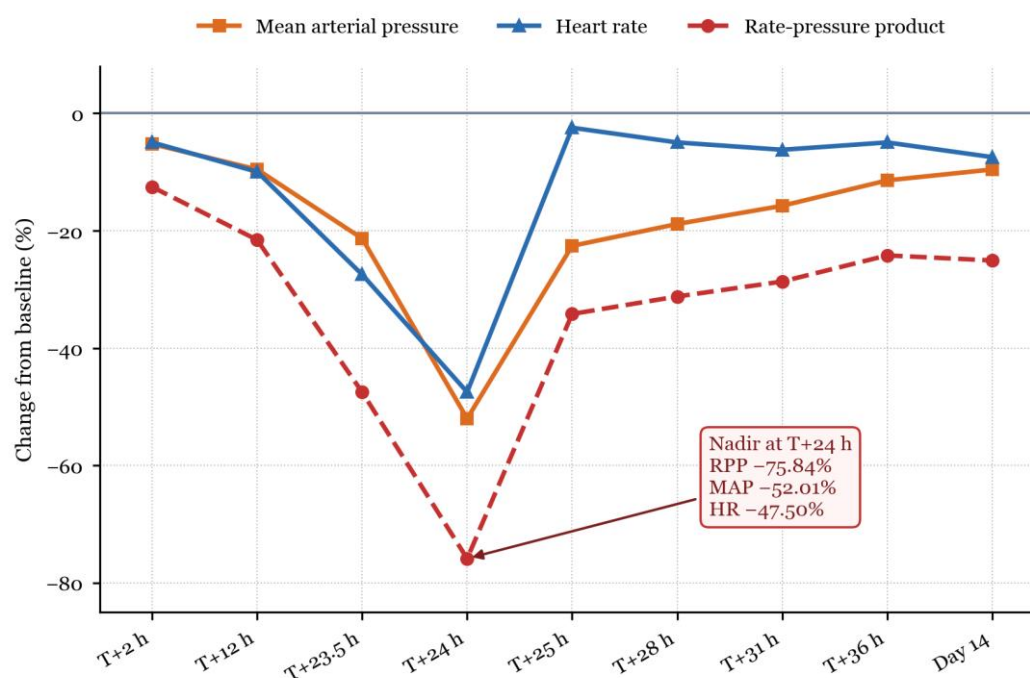


Figure 3. Percentage change from baseline in mean arterial pressure, heart rate and rate-pressure product between T+2 h and day 14.

At 14 days there was no recurrence of syncope, orthostatic dizziness or presyncope. Blood pressure was 132/80 mmHg and heart rate 74 bpm. Pain on cervical extension and rotation was 4–5 on the NRS (baseline 5–6). Its quality had changed from sharp electric shocks to mild, dull soreness around the needle entry sites. Spurling's test and Lhermitte's sign were negative bilaterally. The patient reported less tinnitus and vertigo and no cluster-like headache since the procedure.

3.4. Causality assessment

The Naranjo scale gave a total of 7, which falls in the probable category (5–8 points; Table 5, Figure 4). The WHO-UMC system gave probable or likely, citing a plausible time relationship, response to atropine and fluids, and no reasonable alternative explanation from disease. For the Bradford Hill criteria, temporality (onset 24 hours after the procedure), biological plausibility (trigeminocervical-vagal convergence), coherence (agreement with autonomic physiology) and analogy (similarity to reflex syncope) were met. Consistency cannot be assessed in one patient.

Table 5. Structured causality description (Naranjo scale, WHO-UMC system and Bradford Hill criteria).

Framework	Item or question	Score or rating	Rationale in this case
Naranjo 1	Previous conclusive reports of this reaction?	+1 (yes)	Two earlier case reports of delayed collapse after cervical PRF
Naranjo 2	Did the event follow the suspected procedure?	+2 (yes)	Collapse exactly 24 hours after PRF
Naranjo 3	Did it improve with an antagonist or resuscitation?	+1 (yes)	HR 42 to 78 bpm and MAP 51.67 to 83.33 mmHg after atropine and fluids
Naranjo 4	Did it reappear on re-administration?	0 (not done)	Re-challenge not performed
Naranjo 5	Are there alternative causes that could explain it?	+2 (no)	ECG, troponin, glucose and blood gas normal
Naranjo 6	Did it reappear with placebo?	0 (not done)	No placebo given
Naranjo 7	Was the agent detected at toxic levels?	0 (not done)	Not applicable to PRF
Naranjo 8	Was it more severe with a higher dose?	0 (not applicable)	Same PRF settings at every target
Naranjo 9	Similar reaction to a similar exposure before?	0 (no history)	No earlier vasovagal syncope or orthostatic hypotension
Naranjo 10	Confirmed by objective evidence?	+1 (yes)	Telemetry bradycardia 42 bpm, SBP 75 mmHg, GCS 12
Naranjo total	Cumulative score	7 (probable)	Probable = 5–8 points; possible 1–4; doubtful ≤0; definite ≥9
WHO-UMC	Causality category	Probable / likely	Plausible time relationship, response to atropine and fluids, no reasonable alternative explanation from disease
Bradford Hill	Temporality	Met	Onset 24 hours after the procedure
Bradford Hill	Biological plausibility	Met	Trigeminocervical-vagal convergence
Bradford Hill	Coherence and analogy	Met	Agreement with autonomic physiology; similarity to reflex syncope
Bradford Hill	Consistency	Not assessable	One patient

Notes: ECG, electrocardiogram; GCS, Glasgow Coma Scale; HR, heart rate; MAP, mean arterial pressure; PRF, pulsed radiofrequency; SBP, systolic blood pressure. The Naranjo scale ranges from -4 to +13. The tools are designed for drug reactions or population-level inference and do not prove causation in one patient.

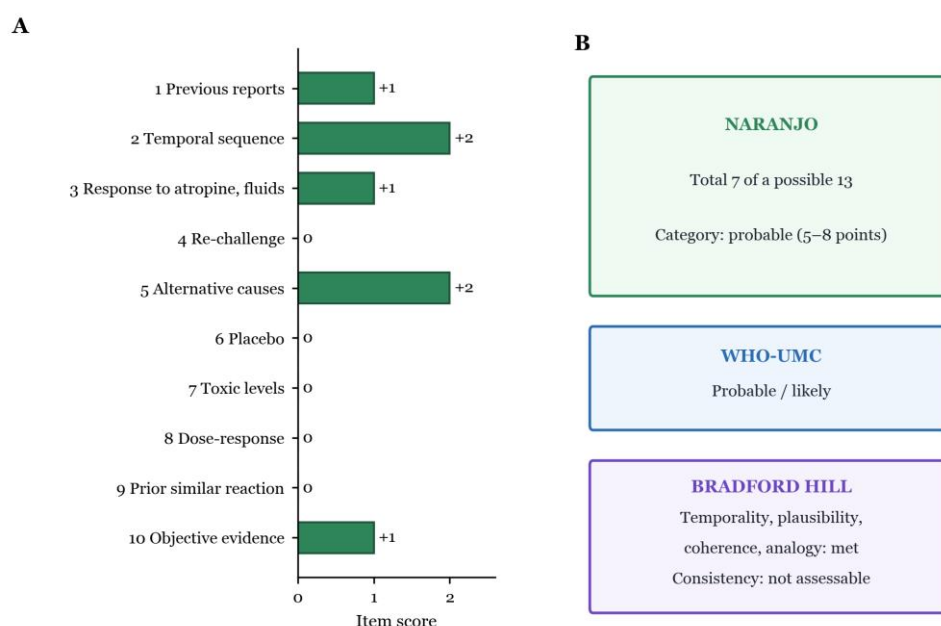


Figure 4. Naranjo item scores (A) and summary of the three causality frameworks (B). The Naranjo total is 7 of a possible 13. The tools do not prove causation in one patient.

4. Discussion

4.1. Principal lesson of the case

This patient developed severe bradycardia, hypotension and reduced consciousness 24 hours after upper cervical and occipital PRF, following an uneventful procedure and 12 hours of stable observation. The event reversed with positioning, fluids and atropine. PRF is generally regarded as a safe, tissue-sparing technique,^{28,29} and the timing of this event is later than that of the vasovagal reactions usually described during spine procedures.^{20,21} It suggests that a delayed hemodynamic event is possible after upper cervical PRF and that standard monitoring windows may not detect it.

4.2. Comparison with the published literature

Vasovagal reactions during interventional pain procedures occur at the time of the procedure. They were seen in 2.6%

of 8,010 fluoroscopically guided procedures, with the highest rate (5.1%) after diagnostic medial branch blocks,²⁰ and in 8% of cervical translaminar epidural steroid injections compared with 1% of lumbar injections (Table 6).²¹ Conservative treatment was usually sufficient, and emergency protocols address these reactions in the procedure room.²⁹ In a randomized trial of PRF for occipital neuralgia, adverse events were similar to those of steroid injection,³ and the complications after PRF for perineal herpes zoster were local (hematoma, infection, numbness) with no vasovagal event listed.³⁰ Reflex bradycardia and cardiac arrest have been described under sedation and after neuraxial anesthesia,^{31,32} but those events also followed the triggering intervention closely. The present report should therefore not be described as the first of its kind unless its relation to those reports is clarified.

Table 6. Published data on vasovagal and autonomic events after spine and cervical procedures, and earlier reports of delayed collapse after cervical PRF.

Study	Design and sample	Procedure	Event reported	Timing and outcome
Trentman 2009 ²¹	Retrospective review; 249 first cervical translaminar injections matched with lumbar injections	Cervical versus lumbar translaminar epidural steroid injection	Vasovagal reaction in 8% (cervical) and 1% (lumbar)	During the procedure; attributed to anxiety, prone position with neck flexed, drapes and neck stimulus
Kennedy 2013 ²⁰	Retrospective analysis of prospective data; 4,183 patients, 8,010 injections	Fluoroscopically guided spine and joint injections, radiofrequency ablation, medial branch blocks	Vasovagal reaction in 2.6% overall and 5.1% after medial branch blocks; 0.8% ended the procedure early	During the procedure; no serious adverse event
Cohen 2015 ³	Randomized trial; 81 patients with occipital neuralgia or migraine with occipital tenderness	PRF (three 120-s cycles per nerve) versus steroid injection	Adverse events similar between groups	Not stated in the abstract
Dong and Li 2024 ³⁰	Cohort; 212 patients (complications reported for 177)	PRF for perineal herpes zoster with ultrasound and DSA or DSA alone	Puncture-site hematoma 4.0–12.8%; infection 1.0–2.6%; local numbness 3.0–5.1%; no vasovagal event listed	Local complications
Present case	Case report; 41-year-old woman	PRF of bilateral C3 and C4 dorsal root ganglia and occipital nerves	Heart rate 42 bpm, blood pressure 75/40 mmHg, GCS 12	24 hours; positioning, fluids and atropine; full recovery

Notes: DSA, digital subtraction angiography; GCS, Glasgow Coma Scale; PRF, pulsed radiofrequency. The studies differ in population, design and outcome definitions and are not directly comparable. Values are taken from the abstracts and tables of the cited articles.

4.3. Pathophysiological or mechanistic interpretation

Reflex vasovagal syncope results from transient global cerebral hypoperfusion after a failure of autonomic cardiovascular control, with vagal activation (cardioinhibition) and sympathetic vasomotor withdrawal (vasodepression) occurring together.¹⁹ In this patient heart rate fell by 47.50% and MAP by 52.01% at the same time, which is compatible with a mixed cardioinhibitory and vasodepressor reflex. Recovery followed atropine, fluids and positioning, which is consistent with this mechanism, but a response to atropine is not specific. A prodrome of sweating, pallor and nausea preceded the collapse, as in most vasovagal syncope (118 of 152 patients with vasovagal syncope in a tilt-table study had prodromal symptoms),³³ and none of the mechanisms below was measured (Figures 5 and 6).

The first possibility is an afferent contribution from the trigeminocervical complex. The upper cervical dorsal root ganglia and the occipital nerves converge with trigeminal afferents in this complex,¹⁴ trigeminal stimulation can produce reflex bradycardia and hypotension,¹⁵ and the nucleus tractus solitarius integrates baroreceptor input and modulates sympathetic and parasympathetic outflow.¹⁶ PRF fields close to the C3 and C4 ganglia might influence nearby sympathetic fibers in the gray rami communicantes, but this is speculative and was not examined. A delay of 24 hours argues against needle trauma or intravascular injection of local anesthetic as the cause.

The second possibility is withdrawal of pain-driven sympathetic activity. Chronic pain is associated with autonomic dysregulation, including reduced heart rate variability,¹⁸ and this patient's baseline blood pressure was 163/80 mmHg. If effective analgesia lowered sympathetic drive

over 12–24 hours, parasympathetic tone could become unopposed and the threshold for the Bezold-Jarisch reflex could fall.³⁴ Blood pressure did decline gradually between baseline and T+23.5 h (MAP -9.60% at 12 hours and -21.36% at 23.5 hours), which fits a gradual process. However, pain relief was modest (NRS 5–6 to 4–5), so this explanation is uncertain. No published measurement of catecholamines or autonomic indices after PRF was found in Europe PMC and Crossref searches, so the idea of a gradual neurohormonal readjustment remains untested.

The third possibility is medication and host factors. The patient took amlodipine, a long-acting dihydropyridine calcium channel blocker with mainly arteriolar vasodilating action, together with amitriptyline and diazepam. Patients taking tricyclic antidepressants such as amitriptyline can develop severe hypotension, for example after induction of anesthesia,³⁵ and benzodiazepines were associated with a larger initial fall in systolic pressure on standing in older people.³⁶ Hypotensive drugs are associated with syncope due to orthostatic hypotension in older adults,³⁷ although in the ALLHAT trial amlodipine did not increase syncope compared with other antihypertensive classes and increased falls only in the first year.³⁸ These drugs may have limited compensatory vasoconstriction in this patient, but the evidence comes from other populations and their contribution is uncertain. A history of cluster-like headache and vertigo may also be relevant, but its role is unknown. The 24-hour delay means that sedation given for the procedure is unlikely to explain the event. Other explanations were not excluded: an orthostatic or postural reflex after mobilization, dehydration or fasting, residual drug effects, and events in the posterior circulation.

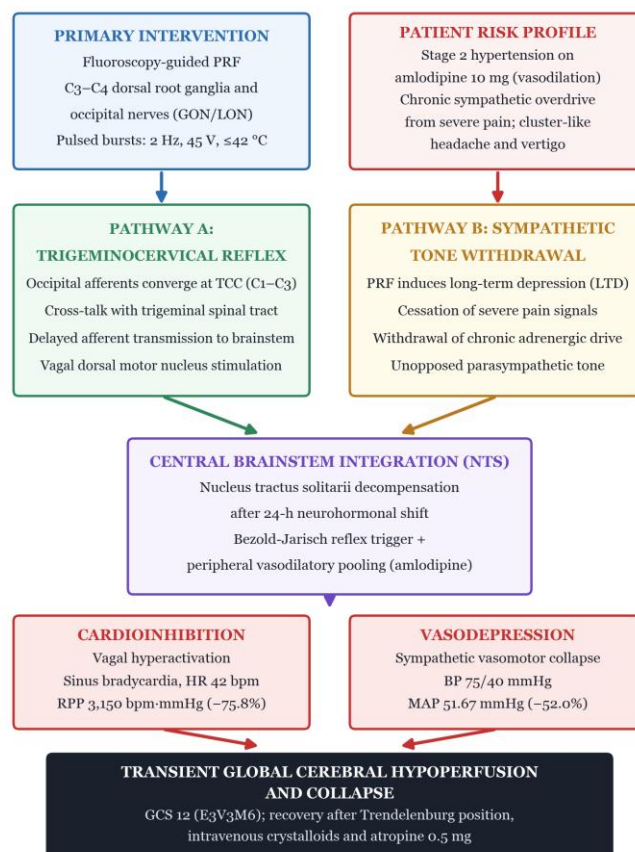


Figure 5. Hypothesized pathway.

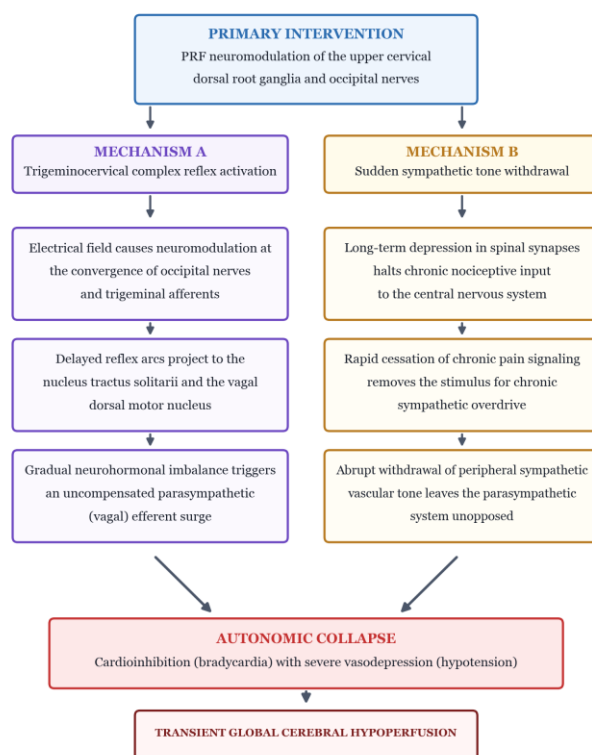


Figure 6. Simplified schema of the two proposed mechanisms leading to reflex bradycardia and hypotension after PRF.

4.4. Implications for practice

In patients who take vasodilating or sedating drugs, clinicians performing upper cervical PRF may consider reviewing these drugs before the procedure, asking about presyncope symptoms after the usual recovery period, and ensuring that ward staff can recognize bradycardia and hypotension and give positioning, fluids and atropine. Applied muscle tension and hydration reduce vasovagal reactions in blood donors,³⁹ but such simple maneuvers were unlikely to be sufficient when both bradycardia and hypotension were severe. Atropine has been tested for vagally mediated bradyarrhythmia and hypotension in inferior myocardial infarction,⁴⁰ and it reversed the bradycardia in this patient; advice drawn from one case is of limited transferability.

4.5. Strengths

The report gives a time-stamped record with ten hemodynamic measurements, the PRF parameters for each target, exclusion of cardiac and metabolic causes by electrocardiogram, troponin I, glucose and blood gas analysis, written informed consent, and 14 days of follow-up.

4.6. Limitations

This is one patient, so incidence and causation cannot be established. The data may have been abstracted retrospectively, and the time points could not have been defined before an unpredictable event. Brain and vertebral artery imaging, serum catecholamines, heart rate variability, orthostatic testing and invasive arterial pressure were not obtained. Reduced consciousness lasted for hours, which is longer than is typical of syncope. The sedation, medication timing, fluid balance and the patient's position at the time of the event are not recorded. Pain relief was modest, and follow-up was 14 days.

4.7. Recommendations for future work

Prospective registries of patients having upper cervical PRF, with scheduled blood pressure and heart rate monitoring beyond 24 hours and measurement of heart rate variability and baroreflex sensitivity, would show how often delayed events occur and whether vulnerable patients can be identified.

5. Conclusion

In one patient, severe but reversible bradycardia, hypotension and reduced consciousness occurred 24 hours after upper cervical and occipital PRF. A reflex mechanism that involves the trigeminocervical complex, withdrawal of pain-driven sympathetic tone and vasodilating medication is plausible, but it was not measured, and a single case cannot establish causation. PRF remains generally well tolerated. Clinicians should be aware that delayed hypotension and bradycardia may occur, particularly in patients who take vasodilating drugs, and further reports and prospective monitoring are needed.

6. Declarations

Ethical approval and consent to participate. CMHC Research Center Ethics Committee, Indonesia, approved this report (protocol approval number 2026/4182). The work followed the Declaration of Helsinki. The patient gave written informed consent before the procedure.

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

Availability of data and materials. All data generated or analyzed in this report are included in the article. Further anonymized clinical records are available from the corresponding author 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. F.R. contributed to conceptualization, investigation (procedures and resuscitation) and writing – original draft. B.H.L. and T.A.S. contributed to supervision, formal analysis (pathophysiological interpretation) and writing – review and editing. All authors approved the final version.

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