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Hypothesis

Gaze-Evoked Orbital Pain (GEOP) After Concussion: A Neurobiological Hypothesis and Clinical Assessment Framework

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Abstract

Visual symptoms following concussion are routinely evaluated using symptom checklists and measures of binocular and ocular motor performance. Although these assessments identify visual fatigue and motor deficits, they fail to distinguish generalized asthenopia from pain mechanosensitively coupled to globe displacement. We propose Gaze-Evoked Orbital Pain (GEOP) as a provisional post-traumatic clinical phenotype. We operationally define GEOP as reproducible ocular or retro-orbital pain provoked or exacerbated by ocular excursion into specific gaze vectors or sustained eccentric globe positioning. This paper presents a dual-component neurobiological hypothesis. First, concussive microstructural strain within orbital connective tissues excites sensitized primary ophthalmic trigeminal (V1) afferents. These afferents converge onto a hyper-responsive trigeminocervical complex (Vc/C1–C2). Second, GEOP is dynamically modulated by sensorimotor context. Identical globe-in-orbit positions achieved via visually guided pursuit versus vestibulo-ocular reflex (VOR) cancellation or passive cervical counter-rotation elicit divergent nociceptive outputs. We attribute this divergence to differences in central motor efference copy, extraocular muscle co-contraction, and brainstem multisensory gating. We delineate an exploratory clinical assessment framework to map directional pain vectors, decouple photic and retinal inputs from mechanical excursion, and evaluate sensorimotor modifiability. Systematically evaluating GEOP may enable clinicians and researchers to parse refractory post-traumatic headache and asthenopia, guiding targeted neuro-rehabilitative and neuromodulatory interventions.

Keywords: concussion; mild traumatic brain injury (mTBI); gaze-evoked orbital pain; trigeminocervical complex; ocular motor; vestibulo-ocular reflex; post-traumatic headache; trochleodynia; sensorimotor gating

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1. Introduction

Visual, binocular, and ocular motor dysfunctions represent some of the most frequent and disabling sequelae of concussion and mild traumatic brain injury (mTBI) [1–4]. Patients regularly report blurred or double vision, photophobia, eye strain, reading difficulty, visual-motion intolerance, and headache intensified by visually demanding environments [5,6]. Objective clinical assessments commonly identify deficits in convergence amplitude and

facility, accommodation, saccadic dynamics, smooth pursuit maintenance, and vestibulo-ocular reflexes [2,4]. Although these assessments quantify functional performance, they do not establish whether retro-orbital pain arises as a nonspecific consequence of prolonged cognitive effort or is mechanosensitively coupled to specific vectors of globe displacement [5,6]. Visual discomfort and visual stress encompass diverse pathophysiological mechanisms [7], yet conventional screening batteries aggregate post-traumatic visual complaints into broad categories such as 'headache,' 'vision problems,' or 'light sensitivity' [8,9].

Contemporary clinical guidelines emphasize phenotype-guided management of post-traumatic headache (PTH) and persistent post-concussive visual complaints [6]. Screening batteries such as the Vestibular/Ocular Motor Screening (VOMS) and the Convergence Insufficiency Symptom Survey (CISS) evaluate symptom provocation during visual tasks [10–12]. However, receded near point of convergence and saccadic dysmetria occur across diverse vergence and accommodative disorders [13,14], and self-reported visual symptoms correlate imperfectly with isolated binocular anomalies or instrumented kinematics [15,16]. Crucially, existing clinical tools do not provide a standardized directional map to determine whether ocular or retro-orbital pain is triggered by specific mechanical excursions of the eye in the orbit [6,12]. Reporting that the eyes hurt during visual tasks does not establish whether moving the eye into a specific spatial vector generates nociceptive input [5,17]. Diffuse discomfort building insidiously during sustained reading and focal retro-orbital pain elicited within seconds by an eccentric gaze vector represent distinct clinical phenomena [5,6]. To define, investigate, and translate this clinical phenomenon, we propose the clinical construct and neurobiological model of Gaze-Evoked Orbital Pain (GEOP) [6].

2. Defining the Proposed Gaze-Evoked Orbital Pain (GEOP) Phenotype

2.1. Operational Definition

For the purposes of this scientific and clinical framework, Gaze-Evoked Orbital Pain (GEOP) is operationally defined as reproducible ocular or retro-orbital pain that is provoked or exacerbated by movement of the globe into a specific gaze direction or by the maintenance of a specific eccentric ocular position [6]. GEOP may be provoked during the dynamic excursion of the eye upon reaching eccentric mechanical limits, during sustained isometric fixation at end-range, or following repeated directional excursions [18]. Preliminary clinical observations reveal that this vector-specific pain frequently persists when identical globe excursions are executed with the eyelids closed, indicating that retinal photic stimulation and visual foveal fixation are not necessary prerequisites for pain provocation [9,19,20]. Provoking vectors may be horizontal, vertical, torsional, or oblique, as well as isolated vergence movements [14]. GEOP is proposed strictly as a descriptive clinical pain phenotype rather than an etiological diagnosis; identifying a painful gaze vector does not specify the precise anatomical source of nociception or central pain mechanism [21,22].

2.2. GEOP Versus Asthenopia and Visual Fatigue

Post-concussive ocular discomfort is commonly labeled as asthenopia, visual fatigue, or eye strain, terms that imply discomfort emerging secondary to sustained visual, accommodative, or vergence load [5,23]. In contrast, GEOP is defined by its immediate, reproducible vector- and movement-dependence [18]. A patient may possess full, comfortable horizontal motility while experiencing sharp retro-orbital pain during elevation in adduction or sustained downgaze [18,24]. Importantly, this distinction must not rely on patient terminology: patients frequently use descriptors such as 'strain,' 'pressure,' 'pulling,' or 'deep eye ache' interchangeably [5,6]. The clinical distinction rests entirely on whether the symptom is reproducibly coupled to specific ocular kinematics and directional vectors rather than cumulative temporal duration [14].

2.3. Coexistence with Established Post-Concussion Impairments

GEOP is not a competing or replacement diagnosis for established post-concussion ocular motor, binocular, or vestibular disorders [2,14]. A patient may concurrently present with convergence insufficiency, accommodative insufficiency, vestibulo-ocular dysfunction, photophobia, cervical spine strain, and chronic post-traumatic migraine [2,4,6,17]. Coexistence does not imply a shared etiology: treating an objective convergence deficit with standard orthoptic vergence therapy may restore normal NPC without ameliorating a coexisting, vector-specific retro-orbital pain [12,15]. GEOP adds an essential, previously unmeasured dimension: mapping whether ocular pain is mechanically coupled to how and where the eyes move [6].

2.4. Differential Diagnosis and Clinical Red Flags

Because pain provoked by ocular movement can herald vision- or life-threatening orbital, neuro-ophthalmic, or intracranial pathology, GEOP must be evaluated within an exhaustive differential framework [21,25]. Pain with ocular excursion is a classic hallmark of optic neuritis, orbital myositis, scleritis, and inflammatory orbitopathies [26–28]. Accordingly, the clinical identification of GEOP is permissible only when established structural, inflammatory, neoplastic, vascular, and infectious causes have been definitively excluded [21,22,25]. Clinical red flags mandating immediate ophthalmologic, neuro-ophthalmic, or emergency neuroimaging evaluation are summarized in Table 1 [18,21,22,24–30].

Table 1. Differential diagnosis of gaze-evoked orbital pain (GEOP), representative clinical hallmarks, and clinical significance [18,21,22,24–30].

Condition	Typical Clinical Hallmarks & Clues	Diagnostic Significance for GEOP
Thyroid Eye Disease (TED)	Lid retraction, proptosis, restrictive motility, diplopia, thyroid dysfunction, extraocular muscle belly enlargement on imaging [21,29].	Established endocrine cause of mechanical gaze-evoked orbital pain; rule out via orbital CT/MRI [21].
Trochleodynia/Trochleitis	Point tenderness over superomedial orbital rim; pain aggravated by SO muscle contraction (downgaze in adduction) or stretch (upgaze in abduction) [18,24].	Key mechanical precedent; demonstrates gaze-evoked orbital pain despite normal motility and imaging [18,24].
Brown Syndrome	Restriction of elevation in adduction; diplopia unusual in children (compensatory posture/suppression) but occurs in adults; audible or palpable click at trochlea [23,31].	Mechanical differential when gaze-provoked pain is accompanied by objective structural restriction [23].
Optic Neuritis	Subacute visual loss, RAPD (absent if bilateral and symmetrical); pain usually mild-to-moderate; pain occurs only in intraorbital optic neuritis [19,27].	Traction of rectus muscle sheaths on inflamed optic nerve sheath; intracranial optic neuritis is painless [19,27].
Orbital Myositis	Acute orbital pain with ocular movement, chemosis, proptosis, diplopia, extraocular muscle and tendon enlargement on MRI [28,29].	Peripheral inflammatory source of extraocular muscle pain requiring high-dose corticosteroids [28].
Posterior Scleritis	Severe deep ocular pain radiating to temple/jaw, scleral thickening ('T-sign' on B-scan ultrasound), visual loss [25,29].	Urgent ophthalmic emergency requiring prompt systemic immunosuppression [25,29].
Orbital Cellulitis	Fever, periorbital erythema/edema, proptosis, chemosis, painful and restricted extraocular motility, leukocytosis [25].	Surgical and medical emergency secondary to paranasal sinus infection; requires emergent IV antibiotics [25].
Orbital Apex/Cavernous Sinus Pathology (Tolosa–Hunt)	Painful ophthalmoplegia with multiple cranial neuropathies (III, IV, VI, V1/V2), retro-orbital pain, Horner syndrome [21,22].	Urgent neurological emergency; granulomatous or neoplastic lesion requiring contrast-enhanced skull base MRI [21].
Post-Concussive GEOP (Proposed)	Quiet, white eye; normal visual acuity and pupillary reflexes; preserved passive motility; reproducible pain in specific gaze vectors [1,6].	Hypothesized post-concussive sensorimotor-trigeminal phenotype; requires systematic directional mapping [1,6].

2.5. Differential Distinction: GEOP Versus Trochleodynia, Ocular Motor Dysfunction, and Cervicogenic Pain

To ensure clinical and research clarity, GEOP must be differentiated from three clinically adjacent entities: trochleodynia, ocular motor dysfunction, and cervicogenic referred pain [6,18,32].

1. Trochleodynia: Trochleodynia involves inflammation or mechanical irritation of the cartilaginous trochlea and superior oblique tendon sheath [30]. Its pathognomonic hallmark is exquisite, localized point tenderness upon palpation of the superomedial orbital rim, aggravated specifically by superior oblique activation (depression in adduction) [18]. In contrast, GEOP manifests across diverse horizontal, vertical, torsional, or vergence vectors without mandatory trochlear tenderness, reflecting mechanical strain across broader extraocular pulleys, peritenon, and orbital connective tissues [6].
2. Ocular Motor Dysfunction: Binocular vision conditions (e.g., convergence insufficiency, accommodative dysfunction, saccadic dysmetria) represent objective performance deficits characterized by fatigue, visual blur, and binocular misalignment [2,13,14]. GEOP is defined strictly by nociceptive mechanosensitivity linked to globe excursion. An individual may demonstrate normal motor performance alongside severe vector pain, or prominent motor fatigue without gaze-evoked pain [15,16].
3. Cervicogenic Referred Pain: Post-traumatic cervical spine injury commonly generates referred retro-orbital pain via convergent cervical (C1–C3) and trigeminal (V1) afferents in the trigeminocervical complex [32–34]. Cervicogenic headache is provoked by cervical movement, sustained head postures, and suboccipital palpation [34]. In contrast, GEOP is elicited by active or passive eye-in-orbit excursion while the cervical spine remains mechanically immobilized in a neutral posture. Although cervical afferent barrage can lower central trigeminocervical thresholds, the immediate trigger in GEOP remains intraorbital mechanical excursion.

3. Central Hypothesis and Testable Propositions

We advance the central hypothesis that Gaze-Evoked Orbital Pain (GEOP) occurs in a distinct subset of post-concussion patients as a consequence of a peripheral–central hybrid dysregulation: subclinical biomechanical shear or tension within orbital connective tissues activates sensitized primary ophthalmic trigeminal (V1) afferents, which converge upon a hyper-excitable trigeminocervical complex (Vc/C1–C2) [1,19,32,35]. Because routine clinical assessments only evaluate aggregate visual strain or performance metrics, this movement-coupled nociceptive source remains obscured within broad categories such as post-traumatic headache, visual-motion sensitivity, or asthenopia [5,6]. We formalize this model into four explicit, testable scientific propositions:

Hypothesis 1 (Biomechanical Extraocular Micro-Strain)**.** *Concussive acceleration–deceleration forces induce microstructural strain, low-grade peritendinous inflammation, or altered tissue compliance within the orbital pulley system, trochlear apparatus, or optic nerve sheath [18,23,31]. When the globe is moved into eccentric vectors, mechanical shear or tensile loading stimulates primary mechanonociceptors innervated by the ophthalmic division of the trigeminal nerve (V1) [18]. This proposal represents a theoretical extrapolation from cranial biomechanics, as direct in vivo histological proof in concussed humans remains to be established.*

Hypothesis 2 (Trigeminocervical Sensitization and Central Amplification)**.** *Post-traumatic neuroinflammation and cervical nociceptive influx produce central sensitization within second-order neurons of the trigeminocervical complex (Vc/C1–C2) [32,33,36]. Due to lowered activation thresholds and expanded receptive fields, normal physiological proprioceptive and mechanoreceptive*

signals generated during ocular excursion are centrally transcoded into conscious retro-orbital pain (mechanically evoked cephalic allodynia) [35–37].

Hypothesis 3 (Sensorimotor Context-Dependent Gating)**.** *GEOP is dynamically modulated by ocular motor efference copy, active motor commands, and vestibular and cervical afferent integration [38,39]. Reaching an identical eccentric orbital position via smooth pursuit versus vestibulo-ocular reflex (VOR) cancellation or passive cervico-ocular rotation produces divergent nociceptive output due to differences in extraocular co-contraction and central multisensory gating within the vestibular nuclei and trigeminal nucleus caudalis [37–39].*

Hypothesis 4 (Non-Photic Trigeminal Autonomic Persistence)**.** *GEOP operates independently of retinal photic stimulation and pupillary dynamics [21,26]. Volitional or attempted eye excursions performed behind closed eyelids reproduce vector-specific orbital pain, which is consistent with the hypothesis that primary optical, cortical, and retinal luminance inputs are not required for pain generation [21,26,36].*

4. Biological and Neuroanatomical Plausibility

4.1. Clinical Precedents: Eye Movement as a Direct Nociceptive Trigger

Pain provoked directly by ocular movement is well documented across neuro-ophthalmic disorders, providing clinical precedent and supporting evidence that ocular motility can generate focal nociception [19,27,28]. In trochleodynia, inflammation or mechanical irritation of the cartilaginous trochlea produces focal pain during superior oblique contraction or stretch [31]. Jarrin et al. observed that while 25% of patients with trochleitis presented with movement-coupled pain, objective motility limitations occurred in only 14% [24]. Thus, ocular motor performance and gaze-evoked pain are clinically dissociable [24].

In optic neuritis, retrobulbar pain provoked by eye movement occurs in approximately 90% of patients with demyelinating, multiple sclerosis-associated presentations [19,27]. Anatomically, this symptom arises because rectus muscle sheaths fuse directly with the optic nerve dural sheath at the orbital apex [19,27]. Crucially, this traction mechanism is restricted to intraorbital optic neuritis; the intracranial optic nerve lacks extraocular muscle connections and possesses only a pial sheath, explaining why intracranial optic neuritis is typically painless [27]. Furthermore, in conditions such as neuromyelitis optica spectrum disorder (NMOSD), which frequently features retrochiasmatic or extensive intracranial involvement, movement-induced ocular pain is less frequent or atypical [19,27]. In orbital myositis, stretch and contraction of inflamed extraocular muscles produce severe movement-provoked pain [28]. While these conditions feature macroscopic pathology, they establish the biological principle that ocular excursion mechanically excites orbital nociceptors [27,28].

4.2. Ocular and Orbital Trigeminal Nociception

Sensory and nociceptive innervation of the globe, cornea, sclera, uvea, extraocular muscle sheaths, trochlear apparatus, and anterior dural reflections is supplied by the ophthalmic division of the trigeminal nerve (V1) [19,31]. Primary V1 afferents pass through the superior orbital fissure and trigeminal ganglion to project to the trigeminal brainstem sensory nuclear complex, concentrating in the subnucleus caudalis and its transition zone into the dorsal horns of the upper cervical spinal cord (Vc/C1–C2) [19,31]. Electrophysiological studies demonstrate that neurons within this region respond vigorously to corneal and orbital nociceptive input, sending ascending projections to the thalamus, parabrachial nucleus, periaqueductal gray, and higher-order cortical pain matrix [20,31].

Importantly, trigeminal nociceptive and retinal visual processing pathways are anatomically coupled [9,20]. Retinal ganglion cell pathways and trigeminal nociceptive signals converge within posterior thalamic nuclei, providing a direct physiological mechanism for photophobia and light-induced headache exacerbation [9,20]. Experimental studies demonstrate that bright light directly activates trigeminal nociceptive neurons in the nucleus caudalis [20], whereas noxious trigeminal stimulation enhances visual cortical excitability [9]. In the context of GEOP, this bidirectional coupling suggests that movement-evoked orbital nociception can heighten visual sensitivity and photophobia, while photic input may lower the threshold for movement-provoked pain [9,19,20].

4.3. Trigeminocervical Convergence and Central Sensitization

The functional overlap between V1 trigeminal afferents and upper cervical sensory nerves (C1–C3) in the trigeminocervical complex is of paramount relevance to concussion [32–34]. Second-order nociceptive neurons in Vc/C1–C2 receive extensive convergent input from both cephalic structures and suboccipital cervical myotomes [32,33]. Because concussive injuries frequently involve concurrent acceleration–deceleration cervical trauma (whiplash strain), continuous nociceptive influx from injured suboccipital muscles and facet joints drives sustained central sensitization of the trigeminocervical pool [32,34]. Consequently, referred pain from the upper cervical spine is commonly projected into the retro-orbital and frontal dermatomes of V1 [34].

Following traumatic brain injury, central pain-processing networks demonstrate enduring hyperexcitability [35,36]. Ashina et al. established that individuals with persistent post-traumatic headache exhibit widespread cephalic cutaneous allodynia and hyperalgesia, reflecting marked central sensitization [17]. Functional neuroimaging by Leung et al. confirmed that patients with chronic mTBI demonstrate significantly impaired descending supraspinal pain modulation and disrupted functional connectivity between the periaqueductal gray and cortical pain centers [36]. In experimental rodent models of concussive impact, Daiutolo et al. demonstrated sustained up-regulation of CGRP and calcitonin-receptor signaling within the trigeminal ganglion and nucleus caudalis, co-occurring with pronounced periorbital allodynia [35]. Within this hyper-sensitized environment, mechanical tensile strain on extraocular connective tissues—which is normally sub-threshold and imperceptible—can readily trigger severe nociceptive output [31,35,36].

4.4. Sensorimotor Context-Dependent Modulation

A cornerstone of the GEOP hypothesis is that ocular nociception is not merely a function of static eye-in-orbit position, but is dynamically modulated by the sensorimotor context through which that position is achieved [37,38]. During active smooth pursuit or volitional saccades, cortical ocular motor commands (frontal eye fields, parietal cortex) generate motor efference copies that pass to the brainstem and cerebellum, recruiting agonist extraocular muscle contraction and antagonist inhibition [37,38]. Conversely, when the head is actively or passively rotated while maintaining visual fixation on a stationary object, the vestibulo-ocular reflex (VOR) drives compensatory globe movement with distinctly different extraocular activation dynamics, absent saccadic motor efference, and concurrent vestibular and cervical proprioceptive inflow [37,38].

If an identical eccentric orbital position produces severe pain during smooth pursuit but negligible pain during VOR cancellation or passive head rotation, the final anatomical position of the globe cannot be the sole determinant of nociception [37,38]. Such context-dependent modulation supports the hypothesis that central motor efference copy, extraocular muscle co-activation, and vestibular/cervical sensory gating dynamically filter incoming trigeminal nociceptive signals [31,37,38].

5. Clinical Observations Motivating the Hypothesis

5.1. Direction- and Vector-Specific Nociception

The clinical observations motivating this hypothesis were collected from an exploratory retrospective review of 13 consecutive adult patient charts evaluated in a tertiary neurorehabilitation clinic for persistent post-concussion symptoms and chronic PTH [6,17]. The cohort comprised three males (age range: 26–56 years; mean age: 40 years) and 10 females (age range: 26–64 years; mean age: 38 years). This retrospective chart review was exempt from Institutional Review Board (IRB) approval; all patients provided informed consent for the use of their anonymized medical data in this research project, and all clinical procedures were conducted in accordance with the guidelines of the Declaration of Helsinki. These preliminary clinical observations served as hypothesis-generating data to establish standardized parameters for gaze-evoked ocular pain (GEOP). A standardized bedside directional excursion protocol evaluated ocular tracking across eight cardinal/diagonal axes and vergence to 30° eccentricity. In these individuals, focal retro-orbital pain was reproducibly elicited by guiding the eyes into specific eccentric vectors [23]. Patients frequently maintained comfortable horizontal motility while experiencing sharp retro-orbital pain during elevation in adduction or depression in adduction [30]. Pain arose during active transit, upon reaching end-range, or during sustained end-range fixation [23]. When patients closed their eyes and volitionally steered their globes into the provocative vector, the pain location and intensity were reproduced, which is consistent with the hypothesis that retinal illumination is not required for pain induction [21,26]. These preliminary, uncontrolled findings serve as hypothesis-generating observations requiring systematic empirical evaluation.

5.2. Standardized Bedside Assessment Protocol

A structured bedside examination was developed to evaluate ocular pain across dynamic and static gaze conditions. The protocol was divided into two consecutive phases:

1. Eyes-Closed Volitional Excursion Protocol:

Patients were instructed to close their eyes and volitionally steer their globes into the eight cardinal and diagonal axes. The presence of strain or pain, its anatomical distribution (unilateral, bilateral, or facial), and its perceived intensity (categorized as none, mild, moderate, or severe) were systematically recorded for each vector. For vectors eliciting discomfort, globe excursions were immediately repeated with eyes closed to verify pain reproducibility.

2. Eyes-Open Tracking and Convergence Protocol:

Patients performed active ocular tracking across the eight cardinal and diagonal axes to 30° eccentricity, followed by a convergence assessment. Focal retro-orbital pain elicited during active transit, upon reaching end-range, or during sustained end-range fixation was documented. The total number of painful gaze vectors was quantified for both eyes-closed and eyes-open conditions, alongside an evaluation of vector overlap between the two states.

5.3. Sensorimotor and Mechanical Modulation Protocols

To investigate potential biomechanical and sensorimotor contributions to GEOP, targeted modulation maneuvers were applied during painful eccentric gaze vectors:

Sensorimotor Strategy Modification: Eccentric globe-in-orbit positions (up to 30° eccentricity) that consistently triggered pain during active smooth pursuit with the head stationary were re-tested by achieving the identical eccentric orbital position via vestibulo-ocular reflex (VOR)-mediated counter-rotation. Specifically, patients maintained visual fixation on a stationary earth-fixed target while the examiner passively rotated the patient's head 30° in the opposite direction, thereby generating a matched 30° compensatory globe

excursion within the orbit without volitional pursuit efference. Alterations in reported pain intensity, including attenuation or complete absence, were documented.

Biomechanical Tissue Displacement: Gentle manual pressure (20–30 g) was applied over the orbital rim using digital palpation during ocular excursion to assess whether localized periorbital tissue displacement modulated retro-orbital pain severity.

5.4. Immediate Contextual and Mechanical Modulation

In preliminary clinical evaluations, altering the sensorimotor strategy modified pain intensity [38]. When an eccentric gaze position that triggered pain during active smooth pursuit was reached by fixating a distant target while the examiner passively rotated the patient's head (VOR-mediated movement), the reported pain was observed to vary in some patients, showing marked attenuation or absence [38,39]. Gentle manual tissue displacement (20–30 g pressure over the orbital rim) during ocular excursion also reduced reported pain intensity in selected patients [23]. While these observations could reflect tactile sensory gating (large-fiber A-beta inhibition of A-delta/C nociceptive inputs), trial order, or examiner expectation, they suggest that movement-provoked orbital pain is subject to sensorimotor modulation [37].

6. Proposed Exploratory Clinical Examination Framework

To transition GEOP from anecdotal observation to rigorous empirical investigation, we propose a standardized exploratory clinical examination framework [14]. This protocol is designed to map directional pain vectors, determine movement versus positional latencies, and isolate sensorimotor variables (Table 2) [14,37].

Table 2. Proposed exploratory clinical examination framework for the characterization of Gaze-Evoked Orbital Pain (GEOP) [14,37].

Candidate Dimension	Standardized Clinical Protocol	Mechanistic/Phenotypic Question
1. Directional Vector Mapping	Patient tracks a 14-point visual target to 30° eccentricity across 8 cardinal/diagonal axes and vergence [14]. Pain rated on NRS 0–10 [1].	Is pain reproducibly coupled to specific horizontal, vertical, or oblique gaze vectors, or isolated vergence? [4]
2. Movement vs. Sustained Position	Distinguish pain during active globe translation (kinetic shear) from pain during 10 s sustained isometric hold at end-range [18].	Does pain arise from dynamic tissue transit/friction or sustained tensile load and venous engorgement? [18]
3. Pain Laterality & Distribution	Record whether pain is unilateral, bilateral, retrobulbar, periorbital, or referred to frontal/suboccipital dermatomes [34].	Are the perceived pain locus and laterality consistent across repeated trials and concordant with vector biomechanics? [34]
4. Pain Severity & Scaling	Quantify pain intensity (NRS 0–10) across eccentric amplitudes (10°, 20°, 30°) along the identified provocative vector [1].	Does pain severity scale proportionally with the angular degree of globe excursion and tissue stretch? [1]
5. Isolated Vergence Provocation	Assess pain during near point of convergence (NPC) and step-vergence testing, independent of conjugate gaze [10,14].	Can isolated medial rectus co-contraction and accommodative convergence evoke GEOP? [10]
6. Photic Decoupling (Eyes Closed)	Patient closes eyelids; examiner instructs volitional movement into the previously identified painful vector [9,20].	Does vector-specific pain persist when retinal illumination, foveal fixation, and pupillary constriction are eliminated? [9,20]
7. Sensorimotor Modulation	Compare pain across three conditions achieving the identical eye-in-orbit angle: (A) pursuit, (B) VOR cancellation, (C) passive VOR [37,38].	Does altering central motor efference copy, vestibular inflow, and cervical afference modulate pain intensity? [37,38]
8. Somatosensory Gating	Apply gentle cutaneous touch/displacement (20–30 g) over the orbital rim during excursion into the painful vector [31].	Does large-diameter A-beta sensory input attenuate gaze-evoked trigeminal nociceptive transmission? [31]

Note on Dimension 7 Methodological Standardization: To isolate ocular motor efference copy and vestibular/cervical afferent gating under standardized kinematics, fixed orbital alignment markers are utilized across three conditions: Condition A (Active Pursuit: head stationary at 0°, eyes pursue a moving target to 30° globe-in-orbit eccentricity); Condition B (VOR Cancellation: head and target rotate synchronously 30° from primary gaze—or from a pre-positioned 30° eccentric offset—suppressing the compensatory VOR while maintaining constant globe-in-orbit position to control for head-motion and VOR-suppression effects); and Condition C (Passive VOR: fixation remains on a stationary center target while the examiner passively rotates the head 30° in the opposite direction, inducing a matched 30° compensatory globe-in-orbit excursion).

7. Potential Contribution of GEOP to Real-World Disability

Human ocular behavior involves continuous saccades, smooth pursuit tracking, and vergence shifts throughout the waking day [37]. Ordinary daily activities—including reading, computer operation, smartphone navigation, driving, and walking through dynamic public environments—require thousands of eye movements across diverse gaze vectors [5,37,39]. Consequently, an individual with GEOP may repeatedly drive the globe into a painful vector dozens or hundreds of times per hour without consciously recognizing that eye movement is the underlying trigger [39]. Because patients lack anatomical insight, they naturally describe the resulting discomfort with broad labels such as 'eye fatigue,' 'screen intolerance,' 'brain fog,' or 'frontal headache' [5,6].

Repeated provocation of GEOP during routine visual behavior creates continuous nociceptive bombardment into the trigeminocervical complex, sustaining central sensitization and lowering the threshold for broader cephalic pain [17,32,35]. D'Silva et al. demonstrated that post-concussion symptoms provoked during driving frequently cause patients to alter driving habits, reduce travel distances, or avoid highway navigation altogether [39]. Characterizing GEOP provides a tangible, vector-specific target: resolving movement-dependent pain may relieve a primary upstream trigger for daily visual intolerance, fatigue, and post-traumatic headache [6,39].

8. Research Agenda: Prospective Testing of the GEOP Hypothesis

To advance GEOP from a conceptual hypothesis to an empirically validated clinical construct, a structured three-phase translational research agenda is essential [6,17].

8.1. Phase 1: Epidemiological Base Rates, Reliability, and Multidimensional Burden

The initial imperative is to establish the base rates and psychometric reliability of GEOP across diverse cohorts [17]. Prospective multi-center studies must compare acute (<2 weeks), subacute (2–12 weeks), and chronic (>3 months) post-concussion patients with orthopedically injured controls (evaluating pain sensitivity without head trauma) and healthy uninjured controls [16,17]. Standardized examinations must determine inter-rater reliability (independent examiner pairs) and test–retest reproducibility across multiple sessions [1]. Furthermore, studies must examine whether GEOP burden correlates with validated instruments, including the Quality of Life after Brain Injury (QOLIBRI) [40], the Rivermead Post-Concussion Symptoms Questionnaire (RPQ) [7], the Convergence Insufficiency Symptom Survey (CISS) [10], and the Vestibular/Ocular Motor Screening (VOMS) [14].

8.2. Phase 2: Neuroimaging and Neurophysiological Biomarkers

The second phase must elucidate the neurobiological and structural correlates of GEOP [30,35,36]:

- High-Resolution Orbital Neuroimaging: Employing 7.0 Tesla MRI with dynamic kinematic orbital sequencing (e.g., CISS/FIESTA sequences) to quantify structural deformation, optic nerve sheath displacement, and extraocular pulley translation during terminal gaze excursions [27,30].
- Trigeminal Neurophysiology: Assessing trigeminocervical reflex (TCR) excitability, blink reflex recovery curves, and quantitative sensory testing (QST) of periorbital V1 dermatomes to measure central trigeminal sensitization and impaired descending inhibition [31,36].
- Infrared Saccadometry & Videonystagmography: Quantifying micro-saccadic square-wave jerks, peak velocities, and gaze fixation stability during painful versus pain-free gaze vectors [16,37].

8.3. Phase 3: Targeted Interventional Clinical Trials

Ultimately, the clinical value of GEOP hinges upon whether treating this phenotype improves patient recovery [6]. Controlled clinical trials should investigate whether vector-specific desensitization paradigms—such as VOR-gated gradual sensorimotor exposure—and non-invasive neuromodulatory interventions (e.g., transcutaneous V1 trigeminal nerve stimulation) alleviate GEOP [6,35]. Critically, trials must evaluate whether sustained reduction in GEOP burden translates into meaningful improvements in headache impact scores, computer screen tolerance, driving capacity, and return-to-work rates [5,39,40].

9. Limitations and Alternative Explanations

Several limitations and alternative interpretations must be considered [6]:

1. Referral Selection Bias: The initial observations motivating this hypothesis originated from a specialized tertiary neuro-rehabilitation clinic evaluating chronic, treatment-refractory post-concussion symptoms and chronic PTH [17]. Prevalence in unselected acute concussion or sports sideline settings remains unknown [12].
2. Nonspecific Habituation and Order Effects: Pain reductions observed during clinical modulation maneuvers could reflect neural habituation, cognitive distraction, trial order effects, or examiner expectation rather than true sensorimotor gating [37]. Controlled paradigms with randomized block presentation and sham controls are required [1].
3. Multi-Morbidity and Phenotypic Overlap: Concussion patients frequently exhibit overlapping cervical, vestibular, visual, and psychological comorbidities [2,4,6]. Differentiating whether retro-orbital pain reflects local extraocular tissue strain, referred cervical nociception, or widespread central sensitization requires careful multifactorial evaluation [17,32,34].
4. Theoretical Nature of Orbital Tissue Strain: Although biomechanically and biologically plausible, direct in vivo evidence of extraocular connective tissue injury in concussed human cohorts has not yet been demonstrated. This mechanism remains a theoretical hypothesis requiring empirical validation via high-resolution kinematic imaging and biomarker analysis [18,31].

10. Conclusions

Following concussion, asking whether a patient's eyes hurt is clinically insufficient; clinicians and researchers must determine whether eye movement itself provokes pain [6]. Gaze-Evoked Orbital Pain (GEOP) introduces a testable theoretical framework linking peripheral extraocular biomechanics, trigeminocervical neurobiology, and sensorimotor integration [23,32]. Systematically evaluating this movement-coupled nociceptive source may enable clinicians to distinguish mechanical ocular pain from generalized asthenopia [5,6]. However, prospective clinical and physiological studies are essential before GEOP can be considered a validated clinical phenotype or adopted into targeted neuro-rehabilitative and therapeutic algorithms [6,40].

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