

# Clinical and Neurophysiological Analysis of Photopsias in the Context of Psychotherapeutic Treatment

## Abstract

This comprehensive research report investigates the phenomenon of perceiving white light, flashes, or luminous artifacts within the eyelids in darkened environments (photopsias), specifically addressing the potential causal link to psychotherapy. The analysis integrates ophthalmological mechanics, neurobiology, psychopharmacology, and somatic psychology. While the primary differentials for photopsias involve vitreoretinal mechanics (e.g., Posterior Vitreous Detachment) and neurological events (e.g., ocular migraine), substantial evidence suggests that psychotherapeutic processes—particularly those involving trauma release, somatic experiencing, and specific eye-movement modalities—can precipitate visual phenomena. These manifestations arise through autonomic nervous system (ANS) discharge, heightened interoception of retinal "dark noise," mechanical stress from ocular-centric therapies, and the pharmacological side effects of concurrent psychiatric medication. This report provides an exhaustive examination of these intersecting pathways.

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## Chapter 1: The Biomechanical and Biophysical Basis of Phosphenes

To determine whether the perception of "white in the eyelids" is a result of psychotherapy, one must first establish the baseline physiological noise of the human visual system. The eye is not a passive camera but a metabolically active, electrically charged organ that generates internal signals even in the absence of external photons.

### 1.1 The Physiology of Phosphenes

Phosphenes are defined as the experience of seeing light without light actually entering the eye.<sup>1</sup> They represent the entoptic (within the eye) translation of non-photic stimulation. The retina, the light-sensitive tissue lining the back of the eye, possesses a limited vocabulary; it translates all stimuli—whether mechanical, electrical, or metabolic—into electrical impulses that the visual cortex interprets as light.<sup>2</sup>

#### 1.1.1 Mechanical Transduction

The most prevalent cause of phosphenes is mechanical pressure. The physical deformation of retinal ganglion cells triggers depolarization. This is commonly experienced when one rubs

their eyes, producing geometric patterns, checkerboards, or sparkles.<sup>1</sup> In the context of a "dark room" scenario described by the user, subtle mechanical pressures become relevant. If an individual is resting their head on a pillow in a manner that exerts pressure on the globe, or if they squeeze their eyelids shut tightly during a moment of emotional distress (common in therapy aftermath), they may induce pressure phosphenes.<sup>4</sup>

### **1.1.2 Saccadic Traction**

The eye is filled with the vitreous humor, a gel-like substance. Rapid eye movements (saccades) cause this gel to swish and tug against the retina. In a lighted room, the visual system suppresses the noise generated by this motion. However, in a dark room, the signal-to-noise ratio shifts. The movement of the eyes, particularly if the vitreous is undergoing age-related liquefaction, can exert microscopic traction on the retina, resulting in a perceptible flash or white streak.<sup>5</sup> This is a critical baseline: simply moving one's eyes in the dark can generate light.

## **1.2 Retinal "Dark Noise" and Biophotons**

Deep research into retinal physiology reveals that the retina is never truly silent. The "white" seen in a dark room may be the brain perceiving the inherent thermal noise of its own sensors.

### **1.2.1 Thermal Isomerization of Rhodopsin**

Rhodopsin is the visual pigment found in rod cells, which are responsible for scotopic (low-light) vision. Rhodopsin molecules are incredibly sensitive, capable of detecting a single photon. However, they are also subject to thermal agitation. Occasionally, the thermal energy in the retina is sufficient to spontaneously isomerize a rhodopsin molecule, triggering the same chemical cascade as a photon hit.<sup>7</sup> This "discrete dark noise" creates a background hum of visual activity.

In daylight, millions of photons hit the retina, drowning out this noise. In the complete darkness of a bedroom, however, this thermal noise becomes the dominant signal. A subject with heightened awareness—such as one undergoing mindfulness training in psychotherapy—may begin to perceive this background thermal activity as a faint, grainy white light or "visual snow".<sup>7</sup>

### **1.2.2 Ultraweak Photon Emissions (Biophotons)**

Beyond thermal noise, biological tissues spontaneously emit light, a phenomenon known as ultraweak photon emission (UPE) or biophotons. These emissions are primarily generated by reactive oxygen species (ROS) during cellular metabolism.<sup>7</sup> The retina has one of the highest metabolic rates of any tissue in the body and consumes vast amounts of oxygen.

Consequently, it is a significant source of biophotons.

Research indicates that under conditions of oxidative stress—which can be induced by high emotional states, crying, or physiological arousal associated with trauma release—the production of ROS and subsequent biophotons may increase.<sup>7</sup> Theoretically, the photoreceptors could detect these internally generated bioluminescent photons, creating a

perception of light that is truly "inner" in origin.

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## Chapter 2: The "Hardware" Pathologies: Ocular and Neurological Differentials

Before attributing visual symptoms to the psychological process of therapy, it is medically imperative to rule out structural pathologies. The user's symptom profile ("white in eyelids," "dark room") overlaps significantly with conditions involving the vitreoretinal interface and cortical processing.

### 2.1 Posterior Vitreous Detachment (PVD)

The most common cause of seeing flashes (photopsia) in the dark is Posterior Vitreous Detachment (PVD).

#### 2.1.1 Pathophysiology of Vitreous Syneresis

The vitreous humor is composed of 99% water and a scaffold of collagen fibrils and hyaluronic acid.<sup>6</sup> As the human body ages, or due to factors like myopia and trauma, the hyaluronic acid depolymerizes, releasing the water it binds. This process, called syneresis, causes the vitreous gel to liquefy and shrink.<sup>6</sup>

Eventually, the vitreous cortex peels away from the internal limiting membrane of the retina. This separation is PVD. As the vitreous pulls away, it may adhere to the retina at points of strong attachment (the optic disc, macula, or blood vessels). This physical tugging stimulates the retina mechanically.<sup>9</sup>

#### 2.1.2 Symptomatology of PVD

- **The Flash:** The brain interprets the mechanical tug as a sudden, brief streak of white light. This is most often described as a "lightning streak" or a "camera flash" in the peripheral vision.<sup>6</sup>
- **Dark Room Sensitivity:** These flashes are most visible in the dark because the contrast is highest and the rod cells are dark-adapted.<sup>5</sup>
- **Movement Trigger:** The flashes often correlate with eye or head movement, as the inertia of the liquefied vitreous causes it to slosh against the retina.<sup>5</sup>

If the user notices that the white light "flashes" when they roll over in bed or move their eyes quickly in the dark, PVD is a leading differential diagnosis that is unrelated to psychotherapy, except perhaps coincidentally by age.

### 2.2 Retinal Tears and Detachment

If the vitreous traction is forceful enough, it can tear the retina. This is a medical emergency.

- **Progression:** A retinal tear allows fluid to pass through the break, lifting the retina off the

- underlying retinal pigment epithelium (detachment).
- **Distinguishing Features:** While PVD flashes are intermittent, a retinal detachment often presents with a "shower of floaters" (blood or pigment cells released into the eye) and a "curtain" or shadow obscuring part of the visual field.<sup>3</sup>
- **Urgency:** Differentiating this from psychogenic light is critical. If the white light is accompanied by a shadow or new black spots, immediate ophthalmological intervention is required.<sup>2</sup>

## 2.3 Visual Snow Syndrome and Thalamic Dysrhythmia

The user mentions seeing "white" which could be interpreted as a static or graininess rather than a distinct flash. This aligns with Visual Snow Syndrome (VSS).

- **The Thalamic Filter:** Sensory information passes through the thalamus before reaching the cortex. The thalamus acts as a gatekeeper, filtering out irrelevant noise (like the thermal retinal noise discussed in 1.2.1).
- **Cortical Hypermetabolism:** In VSS, it is hypothesized that the thalamocortical loop is dysrhythmic, or the visual cortex is hyper-exitable.<sup>8</sup> The filtering mechanism fails, and the conscious mind perceives the internal "noise" of the visual system as a continuous overlay of white or colored dots ("snow").
- **Link to Anxiety:** VSS is often comorbid with anxiety and stress. Psychotherapy patients, who may be in states of high vigilance, might be prone to noticing or exacerbating this sensory filtering issue.<sup>8</sup>

## 2.4 Ocular Migraine (Scintillating Scotoma)

A distinct neurological event is the ocular migraine.

- **Mechanism:** This is caused by Cortical Spreading Depression (CSD)—a wave of depolarization followed by inhibition moving across the visual cortex.<sup>12</sup>
- **Appearance:** Unlike the single flash of PVD, migraine auras are complex. They appear as expanding zigzag lines, shimmering "heat waves," or kaleidoscope patterns of white and colored light.<sup>13</sup>
- **Duration:** They typically last 20 to 60 minutes and resolve spontaneously.<sup>12</sup>
- **Stress Trigger:** Stress relief (the "let-down" effect after a therapy session) is a known trigger for migraines. It is plausible a user sees these lights in the evening after a taxing therapy session.<sup>1</sup>

## Chapter 3: The Psychotherapeutic Bridge: Somatic Release and Autonomic Arousal

Having established the physiological baseline, we address the core query: *Could this be because of psychotherapy?* The evidence suggests a robust affirmative, mediated through

the Autonomic Nervous System (ANS) and the physiological release of trauma.

### 3.1 Somatic Experiencing (SE) and the "Freeze" Discharge

Somatic Experiencing, a modality developed by Dr. Peter Levine, posits that trauma is energy trapped in the nervous system when the "fight, flight, or freeze" response is thwarted.<sup>15</sup>

#### 3.1.1 The Polyvagal Shift

During a traumatic event, the body may enter a "dorsal vagal" shutdown (freeze). In therapy, the goal is to help the client move out of freeze and discharge this survival energy. This process involves a massive shift in autonomic dominance.

- **Sympathetic Arousal:** As the "freeze" thaws, the sympathetic nervous system (fight/flight) briefly activates to complete the defensive response. This results in heart rate acceleration, muscle tension, and pupil dilation.<sup>17</sup>
- **The Discharge Phenomenon:** The release of this energy is often physical. Clients report trembling, heat, cold, and **visual phenomena**.<sup>15</sup>
- **Visual Manifestation:** The brainstem and limbic system activation during this discharge can spill over into sensory processing areas. Users often report seeing "flashes of light" or "white light" as the nervous system resets.<sup>15</sup> This is akin to the "seeing stars" effect after a physical blow, but generated internally by a neurochemical shockwave.

#### 3.1.2 The Anatomy of Emotional Release

Therapy often induces deep emotional release, including crying and shaking.

- **Tears and Chemistry:** Emotional tears have a distinct chemical composition (higher protein, manganese, and hormones like prolactin) compared to basal tears.<sup>21</sup> The act of producing these tears taxes the lacrimal system.
- **Mechanical Pressure:** Crying involves the contraction of the orbicularis oculi muscles, increasing pressure on the globe. As established in Section 1.1.1, pressure on the retina generates phosphenes. A user lying in the dark after a heavy crying session may see lingering white spots or sparkles due to this mechanical stress.<sup>12</sup>
- **Vasovagal Syncope:** Intense emotional relief can trigger a sudden drop in blood pressure (vasovagal response). Retinal perfusion is highly sensitive to blood pressure. A transient drop in oxygen to the retina causes "greying out" or "whitening" of vision.<sup>1</sup>

### 3.2 Interoception and the "Searchlight" of Attention

A critical mechanism linking therapy to visual symptoms is **interoception**—the awareness of internal bodily states.

- **Mindfulness Training:** Many therapies (CBT, SE, Mindfulness-Based Stress Reduction) explicitly train patients to "scan" their bodies and notice subtle sensations.<sup>22</sup>
- **Sensory Amplification:** Normally, the brain uses "gating" mechanisms to ignore

background noise (like heartbeat, digestion, or retinal dark noise). Therapy lowers this gate. The patient becomes hyper-aware.

- **The Dark Room Effect:** In a dark room, external stimuli are absent. The "searchlight" of attention, trained by therapy, turns inward. The patient begins to see the biophotons, the thermal noise, and the blood flow in their own retina—phenomena that were always present but previously filtered out.<sup>23</sup>
  - **Anxiety Loop:** If the patient is anxious about their health (a common reason for therapy), they may fixate on these visual artifacts, creating a feedback loop where attention amplifies the perception of the light.<sup>4</sup>
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## Chapter 4: Eye-Movement Therapies: EMDR and Brainspotting

Specific therapeutic modalities utilize the eyes as the primary mechanism of treatment. These therapies have direct biomechanical and neurological impacts on the visual system that can result in the perception of white light.

### 4.1 Eye Movement Desensitization and Reprocessing (EMDR)

EMDR involves the patient tracking a moving object (therapist's finger or light bar) back and forth while holding a traumatic memory in mind.<sup>26</sup>

#### 4.1.1 Ocular Motor Fatigue and Phosphenes

The lateral eye movements in EMDR are saccadic and repetitive, a pattern rarely performed in daily life.

- **Muscle Strain:** This taxes the extraocular muscles (lateral and medial rectus). Fatigue in these muscles can lead to small tremors or varying tension on the sclera, which transmits pressure to the retina, generating phosphenes.<sup>27</sup>
- **Vitreous Turbulence:** Vigorous side-to-side eye movement creates turbulence in the vitreous humor. If the patient has a vitreous that is partially liquefied (common in adults), this movement can exacerbate vitreoretinal traction, causing flashes (as detailed in Section 2.1).<sup>5</sup>

#### 4.1.2 Neurological "Leakage"

EMDR works by stimulating bilateral brain activation, theoretically facilitating the processing of "stuck" memories between hemispheres.

- **Visual Hallucinations:** Clinical discussions and patient forums report side effects including "flashes of bright light," "shadows," and "visual snow" following sessions.<sup>28</sup>
- **Mechanism:** The intense activation of the memory networks (which often contain visual components) alongside the active stimulation of the visual motor cortex may lead to a

form of sensory "leakage" or persistence (palinopsia), where visual processing continues even after the session ends. In a dark room, this residual activity manifests as light.<sup>28</sup>

## 4.2 Brainspotting (BSP)

Brainspotting is derived from EMDR but focuses on a fixed gaze position ("brainspot") that correlates with somatic activation.<sup>30</sup>

### 4.2.1 The Fixed Gaze and Retinal Bleaching

In BSP, a client may hold their gaze on a specific point for an extended period.

- **Photochemical Depletion:** Staring at a fixed point causes the depletion of photopigments (rhodopsin/cone opsins) in that specific area of the retina. When the eyes eventually move or close (in the dark room), the brain perceives a negative afterimage. If the spot was bright or contrasting, the afterimage may appear as a white or luminous spot.<sup>31</sup>
- **Subcortical Access:** BSP claims to access the "midbrain" and subcortical structures involved in regulation.<sup>32</sup> Deep processing in these primitive brain areas is often accompanied by reflexive physiological signs: blinking, pupil dilation, and *twitching*.
- **Pupillary Flux:** Emotional processing causes the pupil to dilate (fear/arousal) and constrict (relaxation). Rapid changes in pupil size during the integration phase after therapy can alter how light (even ambient dark-room light) is processed, creating halos or starburst effects.<sup>33</sup>

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## Chapter 5: Psychopharmacology: The Chemical Variable

It is impossible to isolate the effects of psychotherapy from the effects of the medications often prescribed concurrently. Several classes of psychiatric drugs are known to cause photopsias.

### 5.1 Selective Serotonin Reuptake Inhibitors (SSRIs)

SSRIs (e.g., Fluoxetine, Sertraline, Paroxetine) are the standard of care for depression and anxiety. Serotonin (5-HT) is a key neurotransmitter not just in the brain, but in the eye.<sup>34</sup>

#### 5.1.1 Serotonergic Retinopathy and Photopsia

- **Receptor Presence:** 5-HT receptors are present in the retina, ciliary body, and cornea.
- **Adverse Events:** A large-scale analysis of FDA data revealed a significant association between SSRIs and "photopsia" (flashing lights). **Paroxetine** (Paxil) showed the highest specific risk for photopsia, while **Fluoxetine** (Prozac) had a 45.9% incidence rate of severe eye disorders in the study cohort.<sup>35</sup>

- **Mechanism:** SSRIs can cause mydriasis (pupil dilation), increasing light sensitivity. They also likely modulate the firing threshold of retinal ganglion cells, making them more prone to spontaneous firing (seeing light in the dark).<sup>35</sup>
- **Timeline:** These visual side effects typically appear within the first 30 days of treatment, correlating with the initiation of therapy.<sup>35</sup>

## 5.2 Benzodiazepines and GABAergic Agents

Benzodiazepines (e.g., Xanax, Valium) are used for acute anxiety.

- **Visual Haze:** They are linked to blurred vision and difficulty with accommodation (focusing) due to their muscle-relaxant effects on the ciliary muscles.<sup>36</sup>
- **Withdrawal:** If a patient is tapering off benzodiazepines (often a goal of psychotherapy), the withdrawal syndrome is characterized by neuronal hyperexcitability. This can manifest as photophobia (light sensitivity) and visual hallucinations or flashes, as the brain lacks its usual inhibitory "brakes".<sup>1</sup>

## 5.3 Antipsychotics

Though less common for mild anxiety, atypical antipsychotics are used as adjuncts.

- **Dopaminergic Effects:** Dopamine plays a major role in the retina (adaptation to light/dark). Modulating dopamine can disrupt night vision and lead to visual dysfunctions.<sup>39</sup>
- **Hallucinations:** Ironically, medications to treat hallucinations can sometimes cause them (paradoxical reaction) or cause "oculogyric crises" where the eyes move uncontrollably, generating mechanical phosphenes.<sup>39</sup>

**Table 1: Psychotropic Medications and Associated Ocular Effects**

Medication Class	Generic Name	Specific Ocular Side Effect	Mechanism
SSRI	Paroxetine	<b>Photopsia</b> (flashing lights)	Serotonergic modulation of retina; Mydriasis.
SSRI	Fluoxetine	Blurred vision, Dry eye	Anticholinergic effects; Pupil dilation.
Benzodiazepine	Alprazolam	Accommodative spasm, Blur	GABAergic muscle relaxation.

<b>Antipsychotic</b>	Quetiapine	Cataracts (long term), Blur	Blockage of histamine/muscarinic receptors.
<b>Tricyclic</b>	Amitriptyline	Mydriasis, Cycloplegia	Strong anticholinergic activity.

## Chapter 6: Transpersonal and Altered States of Consciousness

The user's query ("white in my eyelids") resonates with descriptions found in transpersonal psychology and meditation literature, which are often integrated into modern holistic therapy.

### 6.1 Hypnagogia: The Threshold of Sleep

The "dark room" context implies a pre-sleep state. The transition from wakefulness to sleep (Stage 1) is known as hypnagogia.

- **Visual Hallucinations:** It is neurologically normal to experience sensory hallucinations during this phase, including geometric patterns, speckles, and flashes of light.<sup>25</sup>
- **The Stress Rebound:** Hypnagogic phenomena are significantly more frequent and vivid in individuals with stress, anxiety, or insomnia. Psychotherapy often unearths suppressed material. The "rebound" of these thoughts during the relaxation of sleep onset can manifest as intense visual imagery or bright lights.<sup>41</sup>
- **Thought Suppression:** Research indicates that suppressing a thought during the day (avoidance) leads to its re-emergence during hypnagogia. If therapy is addressing avoided trauma, the visual cortex may "vent" this material as light or imagery at night.<sup>42</sup>

### 6.2 The "Nimitta" and Meditative Light

If the user practices meditation as part of therapy:

- **The Nimitta:** In deep states of concentration (Jhana), practitioners report a "counterpart sign" or *Nimitta*—often described as a bright, steady white light or a "star" in the darkness.<sup>43</sup>
- **Neurological Basis:** This is believed to be a result of sensory deprivation. When external visual input ceases, the visual cortex gains (sensitivity) increase. Spontaneous firing of neurons in the visual cortex (V1) creates the perception of light. This is a sign of a "quieting" mind allowing background activity to be seen.<sup>44</sup>
- **Third Eye Phenomenon:** Subjectively, this light is often felt at the forehead. While biologically centered in the occipital cortex, the perception is projected. This "white light"

is considered a milestone in many contemplative traditions.<sup>44</sup>

### 6.3 Kundalini and Energetic Awakening

In Somatic and Transpersonal therapies, "energy" movement is a core concept.

- **Kundalini Rising:** This framework describes a dormant energy at the base of the spine rising to the brain. Symptoms include tremors (kriyas), heat, and **blinding white lights** or flashes of insight.<sup>47</sup>
- **Therapeutic Correlation:** Patients undergoing intense emotional release (catharsis) often report these phenomena. From a scientific perspective, this maps to the massive autonomic discharge discussed in Section 3.1. The "light" is the subjective experience of a high-energy neurophysiological event.<sup>47</sup>

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## Chapter 7: Differential Diagnosis and Actionable Insights

To provide a safe and useful answer, we must categorize the user's experience to guide their next steps.

### 7.1 Distinguishing Features Table

Feature	Retinal Detachment / PVD (Medical Urgency)	Ocular Migraine (Neurological)	Psychogenic / Somatic (Therapeutic)
<b>Visual Form</b>	Lightning streaks, arcs, camera flash.	Zigzags, fractals, shimmering heat waves.	Diffuse white glow, sparkles, "snow", orbs.
<b>Duration</b>	Split-second (flash), but recurring.	20–60 minutes, then fades.	Variable; can be momentary or sustained in meditation.
<b>Trigger</b>	<b>Eye/Head movement</b> , looking far lateral.	Stress relief, food, light glare.	Crying, deep relaxation, emotional release, anxiety.
<b>Associated Signs</b>	<b>New floaters</b>	Headache (maybe),	Shaking, tingling,

	<b>(black spots),</b> shadow in vision.	nausea.	emotional relief, vivid dreams.
<b>Laterality</b>	Usually Monocular (one eye).	Usually Binocular (both eyes/visual fields).	Often felt centrally or binocularly.

## 7.2 Recommendations

1. **Ophthalmological Clearance:** Because PVD and Retinal Detachment are sight-threatening, **any new onset of flashes (photopsia) requires a dilated eye exam.** The user must rule out the "Hardware" issues first. If they see "black spots" or a "curtain," this is an emergency.<sup>2</sup>
2. **Medication Review:** If the user started an SSRI (like Paxil or Prozac) recently, this is a prime suspect. They should consult their prescribing psychiatrist about "photopsia" as a side effect.<sup>35</sup>
3. **Therapeutic Contextualization:** If the eye exam is normal, the user can reframe the lights as a **benign side effect of somatic release.**
  - o **Normalization:** Understanding that "discharge" causes lights reduces the anxiety loop.
  - o **Grounding:** If the lights are disturbing, use grounding techniques (open eyes, turn on a lamp, touch a textured surface) to bring the brain back to external sensory processing and turn off the "internal searchlight".<sup>26</sup>

## 7.3 Conclusion

The perception of white light in the eyelids in a dark room is a multifaceted phenomenon. While it demands a medical exclusion of retinal pathology, it is a scientifically plausible and documented consequence of psychotherapy.

The mechanisms are threefold:

1. **Physiological:** Mechanical pressure from crying/eye movements and pharmacological side effects of SSRIs.
2. **Neurological:** Autonomic discharge (freeze/thaw response) and visual cortex hyperexcitability (hypnagogia/meditation).
3. **Psychological:** Heightened interoception turning the brain's attention toward its own internal visual noise (biophotons and thermal noise).

Thus, the "white light" is likely the visible signature of a nervous system in the profound act of processing, healing, and reorganizing itself.

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- **Biophotons & Dark Noise:**<sup>7</sup>
- **Somatic Experiencing & Trauma:**<sup>15</sup>
- **EMDR & Brainspotting:**<sup>26</sup>
- **Medications (SSRIs/Benzos):**<sup>34</sup>
- **Visual Snow & Migraine:**<sup>8</sup>
- **Meditation & Transpersonal:**<sup>43</sup>
- **Hypnagogia:**<sup>25</sup>

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