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Perspective

Perceptual Uncertainty and Its Monitoring in Visual Snow Syndrome: A Multimodal Framework

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Abstract: Neurobehavioral signatures, such as cortical hyperexcitability, thalamo-cortical dysrhythmia, and pulvinar dysfunction, appear to drive the persistent visual misperceptions in Visual Snow Syndrome (VSS). We propose that heightened perceptual uncertainty and impaired metacognitive monitoring perpetuate these disturbances and formalize these processes within different frameworks (predictive coding, signal detection theory, and attentional control). By clarifying these mechanisms, we aim to inform targeted interventions that could address this currently untreatable condition.

Keywords: visual snow syndrome; perceptual uncertainty; metacognition; predictive coding; signal detection theory and attentional control

1. Introduction

Patients with Visual Snow Syndrome (VSS) experience a persistent, static-like flickering across the entire visual field, often referred to as "visual snow", alongside additional disturbances such as enhanced entoptic phenomena (e.g., floaters), palinopsia, photophobia, and nyctalopia. These symptoms occur without detectable structural lesions to the visual system [1–4]. Despite clear diagnostic criteria (ICHD-3), the pathophysiological mechanisms of VSS remain poorly understood, and no effective treatment options are available [5].

We propose that perceptual uncertainty, an inability to reliably distinguish relevant signals from sensory noise, underlies the visual misperceptions in VSS. Moreover, we hypothesize that impaired metacognition, disrupted self-monitoring, and confidence assessment may contribute to the chronification of these symptoms. When perceptual processing fails to correct noisy inputs, patients can experience ongoing misinterpretations and a persistent sense of visual overload.

By first describing the neural underpinnings that produce perceptual uncertainty in VSS, applying these formal models, and finally showing how metacognitive dysfunction may perpetuate symptoms, we hope to offer a comprehensive hypothetical perspective on VSS. We conclude by discussing potential clinical implications, including how training metacognitive abilities might alleviate the disorder's hallmark misperceptions.

2. Hypothesis

2.1. Neural Underpinnings of Perceptual Uncertainty in VSS

In healthy perception, subtle neural processes filter out irrelevant noise, maintaining clarity of sensory input. In VSS, however, these processes appear impaired, leaving patients hypersensitive to both internal and external stimuli. Three key disruptions that drive

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this sensory uncertainty are cortical hyperexcitability, thalamo-cortical dysrhythmia, and pulvinar dysfunction. Taken together, they make it difficult for patients to evaluate the reliability of their visual experiences, setting the stage for persistent misinterpretation (see Table 1).

Table 1. Neural underpinnings of perceptual uncertainty in VSS.

Location	Neural Mechanism	Description	Functional Consequence
Pulvinar nucleus (thalamus)	Pulvinar dysfunction	Impaired salience filtering and confidence estimation	Persistent misinterpretation of visual percepts
Thalamo-cortical pathway	Thalamo-cortical dysrhythmia	Disrupted oscillatory communication between thalamus and cortex	Affecting sensory gating and integration
Occipital cortex, higher visual sensory areas	Cortical hyperexcitability	Increased excitatory-inhibitory imbalance and visual cortex	Amplification of irrelevant sensory noise

2.1.1. Cortical Hyperexcitability

Clinically, VSS involves hypersensitivity to internal stimuli (e.g., static and entoptic phenomena) as well as external stimuli (e.g., photophobia), plus an abnormal persistence of visual impressions (e.g., palinopsia and nyctalopia) [1]. These features align with cortical hyperexcitability, wherein an excitatory–inhibitory imbalance in visual cortical networks intensifies incoming signals.

Neuroimaging studies support this notion. Magnetic resonance spectroscopy (MRS) has identified increased glutamate/glutamine (Glx) levels in the occipital cortex of patients with VSS, indicating heightened excitatory drive [6]. Similarly, electroencephalography (EEG) reveals elevated high-frequency oscillations in visual cortices, consistent with a hyperexcitable state [7]. Together, these findings suggest that hyperexcitable cortical networks amplify every stimulus, whether relevant or not, thus escalating perceptual uncertainty.

2.1.2. Thalamo-Cortical Dysrhythmia

Thalamo-cortical dysrhythmia is discussed to be a second critical mechanism in VSS, marked by aberrant oscillatory coupling between the thalamus and cortical regions. Functional connectivity analyses indicate disruptions in these pathways, which may undermine normal sensory gating and integration between the thalamus and occipital cortex [7–9]. For example, certain low-frequency thalamic oscillations have been linked to hypervigilant sensory states [10], potentially explaining the intrusive quality of visual disturbances in VSS.

Thalamo-cortical dysrhythmia often interacts synergistically with cortical hyperexcitability, creating a feedback loop that magnifies noise. Hyperexcitable visual areas boost incoming signals, while dysrhythmic thalamo-cortical circuits fail to filter or integrate them properly, further heightening perceptual uncertainty [11]. Evidence of abnormal functional connectivity between the thalamus and visual cortices [7] supports this dual disruption in patients with VSS.

2.1.3. Pulvinar Function and Confidence Processing

The pulvinar nucleus of the thalamus is pivotal for visual information processing and attentional prioritization, filtering stimuli based on their salience [12]. Resting-state neuroimaging in VSS suggests that reduced pulvinar activity may undermine normal salience gating [2–4], contributing to visual disturbances such as flickering snow and heightened light sensitivity.

Beyond merely relaying sensory information, the pulvinar has been linked to perceptual confidence, i.e., the subjective certainty in one's sensory judgments. Research on

non-human primates shows that changes in pulvinar activity correlate with how confident animals are when making visual decisions [13]. Studies in humans and other species further indicate that the brain integrates confidence signals at multiple stages of visual processing [14]. Hence, reduced activity in the pulvinar can cause irrelevant noise to be treated with the same weight as truly salient stimuli [15,16] with eventually disturbed monitoring of perceptual stimuli in VSS. Thus, pulvinar dysfunction becomes a critical link between heightened perceptual uncertainty and chronic misinterpretation in VSS: if the system fails to gauge the reliability of ambiguous inputs, faulty signals go uncorrected, and visual misperception persists. This mechanism aligns with broader models of metacognition in which subcortical and cortical relays jointly enable self-monitoring [17].

These three disruptions, namely cortical hyperexcitability, thalamo-cortical dysrhythmia, and pulvinar dysfunction, when brought together, might offer a foundation for understanding how VSS arises at the neural level. To further formalize these processes, predictive coding, signal detection theory, and Attentional Control Theory could each help to illuminate how sensory noise becomes entrenched when higher-order regulation is compromised.

2.2. Formal Frameworks Explaining the Mechanisms

2.2.1. Predictive Coding Framework

We adopt a Bayesian perspective [15,18,19] to formalize disrupted sensory updating in VSS. Under predictive coding, the brain continuously generates top-down predictions about incoming signals and compares them with bottom-up input to compute prediction errors. Ordinarily, these errors prompt the revision of internal models. In VSS, heightened perceptual uncertainty may manifest as amplified prediction errors as patients struggle to reconcile noisy sensory data (e.g., flickering dots and static) with stable predictions [1,4].

According to predictive coding, the brain may respond to persistent mismatch by increasing the precision (or "gain") assigned to sensory signals [20]. In VSS, this can lead to hyperexcitability in primary visual cortices [7], creating a vicious cycle with a momentary sharpening of visual perception that may briefly improve detection of subtle stimuli. Over time, however, new set-points may be established that may treat "noise" as relevant, hindering the ability to filter our trivial fluctuations [21], (see Figure 1A).

In line with the literature on predictive coding, chronic or unresolved prediction errors are a hallmark of pathological perception [15]. A core tenet of predictive coding is updating priors in response to errors. However, metacognitive deficits [22,23] can prevent patients from recognizing when these priors are maladaptive. If the system does not "downregulate" noise, symptoms become entrenched. Thus, the fundamental issue may not simply be high prediction errors, but the inability to monitor and revise them [24] (see Figure 1B).

2.2.2. Signal Detection Theory (SDT)

Decision Thresholds and Sensory Noise: Signal detection theory (SDT) [25,26] posits that perception depends on the following:

- Sensitivity (d′), i.e., the ability to distinguish true signals from noise;
- Decision criterion, i.e., a threshold balancing false alarms vs. misses.

In VSS, cortical hyperexcitability may effectively lower the decision threshold [1], causing patients to over-detect minimal fluctuations (e.g., benign flickers) and consistently report "seeing something" even in low-noise situations. Metacognition shapes how individuals set and adjust this criterion based on confidence in their ability to discriminate signals from noise [22]. In VSS, an impaired sense of confidence may lead patients to adopt a liberal criterion to avoid missing any signal, thus increasing false alarms and

reinforcing the conviction that static is ever-present. Without metacognitive recalibration, these misperceptions linger or intensify [24].

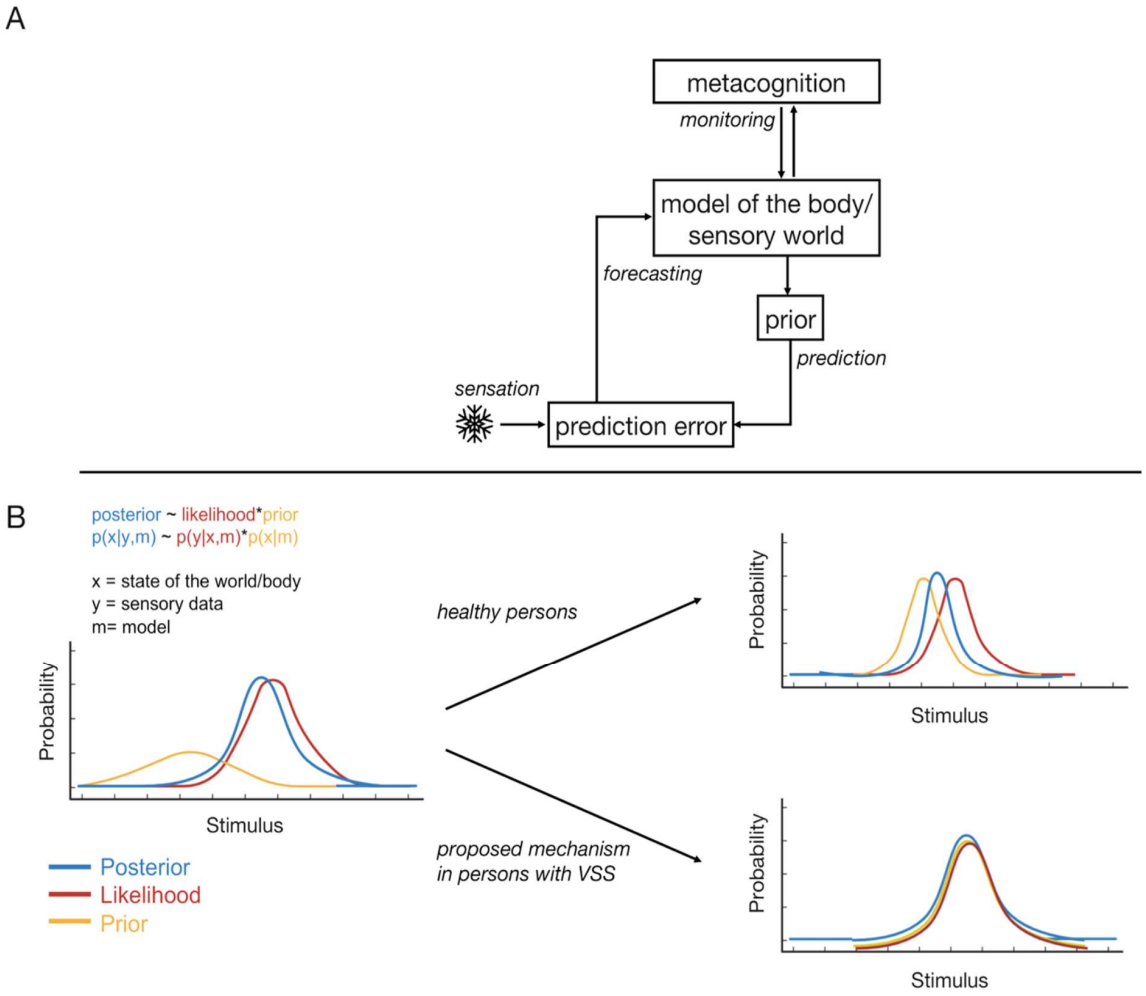

Figure 1. (**A**) Detecting and monitoring sensory stimuli. Predicted and actual sensory data are compared in specialized neural areas of the visual system, yielding to a precision-weighted prediction error that feeds beliefs regarding the internal/external world. Detecting a visual stimulus (e.g., a snowflake) is bound to a threshold, in seeing a stimulus as a signal vs. noise; thus, there is an (un)certainty of seeing the stimulus. Importantly, sensory (un)certainty is reflected by an estimation of confidence in seeing the stimulus properly. That is, the beholder can report their confidence in seeing the snowflake and reflect on the acuity of their perception. This monitoring of perceptual processes can be described as a metacognitive ability. Metacognition can be thought of as a higher-order layer, monitoring beliefs and their amount of precision, thereby impacting expectations that drive priors and self-efficacy. (**B**) A schematic evolution of posterior probability distribution. Shown are the probability distribution of the prior (yellow), the likelihood (red), and the posterior (blue) of a sensory input, i.e., experimental stimuli. Prior beliefs about states of the world and new sensory data (likelihood) are combined to form the actual perception (posterior). The amount of belief update equates to the prediction error, that is, the difference between actual sensory data and the prior beliefs weighted by their relative precision. In case of low and imprecise prior beliefs about a sensory stimulus (yellow), a posterior belief distribution (blue) is closer to the likelihood (red), and both are widely distributed, leading to a higher prediction error and a belief update. In healthy persons, with time, beliefs are updated, and priors increase in precision. In patients with VSS, however, we think that beliefs are not updated in an effective manner, with posterior distribution staying closer to likelihood, resulting in a new set of imprecise priors: the distribution of priors overlies both the likelihood and posterior distribution, eventually leading to the perseverance of non-filtered sensory stimuli. The graphs were formatted with biorender.com.

2.2.3. Attentional Control Theory (ACT)

Over-Vigilance in VSS: The Attentional Control Theory [27] states that heightened arousal or anxiety can bias attention toward irrelevant stimuli. In VSS, an analogous hyperexcitability may lead patients to fixate on flickers, floaters, or "static" dots [4], causing these low-level phenomena to dominate awareness.

In typical cognition, self-monitoring flags when attention is misplaced, redirecting it to more salient tasks [28]. In VSS, this mechanism appears diminished, so patients fail to shift away from visual noise [22].

2.3. Impaired Metacognition Explaining Chronification of Symptoms

2.3.1. Why Perceptual Uncertainty Persists

Each framework—predictive coding, SDT, and ACT—describes a route by which VSS may emerge:

- Predictive Coding: Miscalibrated priors inflate noise and perpetuate prediction errors [15].
- SDT: A liberal decision threshold heightens false positives [25].
- ACT: Over-vigilance locks attention onto minor flickers [27].

However, we propose that the chronic nature of VSS likely stems from deficits in the metacognitive control layer, which normally evaluates and corrects errors, thresholds, or attentional biases [29].

2.3.2. Impaired Metacognitive Control Layer

Hypothesizing that patients with VSS have deficits in confidence and the monitoring of perceptual precision aligns with evidence that higher-order cortical areas (e.g., the prefrontal cortex) and subcortical relays (e.g., the pulvinar) facilitate gating and evaluating sensory input [17,30]. When these processes break down, patients are faced with the following:

- A lack of feedback for recalibration: The system fails to adjust inflated prediction errors or shift a liberal decision criterion [31].
- A persistent hyperexcitability: Abnormal gating in primary visual areas remains uncorrected [4,7].
- Over-detection: Benign flickers are interpreted as significant stimuli, reinforcing the sense that static pervades the visual field [1].

Once new "perceptual set-points" become established under hyperexcitability, weak metacognitive oversight cements these biases, making VSS a chronic rather than transient phenomenon [24].

2.3.3. Clinical Implications: Metacognitive Training

Explicitly recognizing and correcting the tendency to over-detect noise can improve metacognitive awareness [22]. By learning to moderate confidence judgments, i.e., trusting the absence of a signal rather than defaulting to "there must be something", patients may reduce false alarms and reclassify entoptic phenomena as irrelevant.

Self-monitoring is central to metacognition [22]. In VSS, persistent noise becomes self-reinforcing when patients do not recognize they are hyper-focusing or over-weighting prediction errors. Even if some neural hyperexcitability remains, strengthening metacognitive insight may help reduce the subjective impact of these signals.

Indeed, metacognitive training has shown promise in treating conditions with cognitive distortions and anxiety-driven attentional biases [29,32]. CBT-informed approaches combining psychoeducation, confidence calibration, and attentional training are effective in disorders with sensory distortions [33]. Although direct RCTs in VSS are limited, evidence

from related conditions (e.g., persistent migraine aura and tinnitus) suggests that targeting metacognition and attention can relieve chronic perceptual symptoms [34,35].

3. Conclusions

Visual Snow Syndrome (VSS) highlights how perceptual uncertainty can become self-perpetuating when neural and cognitive factors converge. As prior research has shown, cortical hyperexcitability, thalamo-cortical dysrhythmia, and pulvinar dysfunction all amplify and foster the misinterpretation of sensory input, while impaired metacognition impedes the recalibration of these faulty processes. Viewing VSS through different frameworks such as predictive coding, signal detection theory, and Attentional Control Theory reveals that although each model emphasizes different facets—e.g., prediction errors, decision thresholds, and attentional bias—they all point to the crucial role of self-monitoring in mitigating symptom persistence.

By focusing on metacognitive training, clinicians can address both the sensory (hyperexcitability and dysrhythmia) and cognitive (over-vigilance and miscalibrated priors) components of VSS. Even if the underlying excitatory–inhibitory imbalance remains, strengthening patients' ability to evaluate and respond to their own perceptions can lessen the severity of visual disturbances over time. Future research and targeted clinical trials are needed to refine these interventions, but evidence from related conditions suggests that improving metacognitive skills may offer a viable path toward sustained relief in VSS.

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