ARTICLE IN PRESS

Advances in Ophthalmology and Optometry ■ (2024) ■–■

ADVANCES IN OPHTHALMOLOGY AND OPTOMETRY

Visual Snow Syndrome

Visual Consequences, Diagnosis, and Treatment

Kenneth J. Ciuffreda, OD, PhD, FARVO, FCOVD-A, FAAO, Diplomate in Binocular Vision, FNAP[a],
Barry Tannen, OD, FAAO, FCOVD[b,c,*],
Noah Tannen, OD, FCOVD, FAAO[c],
Daniella Rutner, OD, MS, MBA, FAAO, FCOVD, FNAP, Diplomate ABO[d]

[a]Department of Biological and Vision Sciences, Brain Injury Research Unit, Vision Rehabilitation Service, SUNY/College of Optometry, 33 West 42nd Street, New York, NY 10036, USA; [b]Department of Clinical Sciences, Vision Rehabilitation Service, SUNY/College of Optometry, 33 West 42nd Street, New York, NY 10036, USA; [c]Eye Care Professionals, PC, 1777 Kuser Road, Hamilton Square, NJ 08690, USA; [d]Department of Clinical Sciences, Brain Injury Research Unit. Vision Rehabilitation Service, SUNY College of Optometry, 33 West 42nd Street, New York, NY 10036, USA

Keywords

• Chromatic tints • Concussion • Diagnostic criteria
• Neurologic and perceptual mechanisms • Neuro-optometry
• Neurologic substrate(s) • Nonvisual symptoms • Oculomotor-based vision therapy

Key points

- The condition of visual snow syndrome (VSS) is an emerging neurologic/neuro-optometric condition having a wide range of visual and nonvisual symptoms. These are of a sensory, motor, and perceptual nature.
- VSS is estimated to be present in at least 2% of the population, which is a far cry from the early days of stating that it was a ''rare'' condition.
- The key visual symptom of VSS is the presence of a pixelated overlay of ''visual snow'' across and in front of the entire visual field.

*Corresponding author. Eye Care Professionals, PC, 1777 Kuser Road, Hamilton Square, NJ 08690. *E-mail address:* drt@eyecareprofessionals.com

https://doi.org/10.1016/j.yaoo.2024.01.001
2452-1760/24/

Fig. 1. Visual scene with VS superimposed on right side and not on the left side. (*From* Ciuffreda KJ, Rutner D. Visual snow syndrome in traumatic brain injury: effect on driving. NewsBrake 2023; 47:26–28.)

SIGNIFICANCE

Visual snow (VS) is an enigmatic, intriguing, and bothersome, typically nonprogressive [1], visuo-perceptual, and neurologic abnormality [2–5]. It has been characterized as a pixelated array of dynamic visual noise, or "dots," appearing in a "perceptual" plane superimposed on the "physical" plane of the visual scene (Fig. 1) [6]. Thus, there are two depth planes of visual context: the important background visual scene itself and the interfering overlay of VS in the foreground. It has been described as being somewhat similar to the "electronic noise" occurring in a detuned television. The VS typically is constantly present and a chromatic nature (80%), although it may appear to be transiently present and achromatic to some (20%) [6].

In addition to the key symptom of VS, several other primary and secondary, visual and nonvisual symptoms occur that must typically be present for at least 3 months to be diagnosed as "visual snow syndrome" (VSS) (Box 1) [2–6]. Regarding the primary visual symptoms of VSS, the individual must report

Box 1: Visual and nonvisual symptoms in visual snow syndrome

1. *Visual snow*: pixelated, dynamic visual ''noise'' overlaying and in front of the entire visual scene
2. *Palinopsia*: persistence of a visual image (ie, an afterimage), sometimes with a comet-like trail
3. *Photosensitivity*: light sensitivity
4. *Enhanced entoptic imagery*: perception of ocular floaters and other ocular debris not normally visible to others
5. ''*nyctalopia*'': difficulty seeing at night or in very dim illumination
6. *Photopsia*: perception of light arising without an external light stimulus; random flashes of light
7. *Balance problems*: sensation of bodily unsteadiness
8. *Hyperacusis*: a disorder of loudness perception, overly sensitive to sounds, reduced tolerance to sounds, sometimes only very specific sounds
9. *Phonophobia*: unwarranted fear of sound including common ones in the environment and home, may elicit a sympathetic ''fear'' response
10. *Migraine*: severe headache usually unilateral, may cause transient light sensitivity, transient scotomas, and nausea
11. *Tremor*: small, involuntary, rhythmic muscle contractions
12. *Tinnitus*: ringing in the ears
13. *Cutaneous allodynia*: pain sensation from normal touching of the skin

From Ciuffreda KJ, Rutner D. Visual snow syndrome in traumatic brain injury: effect on driving. NewsBrake 2023; 47:26-28.

at least two of the following four: palinopsia (abnormal persistence or recurrence of an image in time: Fig. 2), photosensitivity, enhanced entoptic imagery, and impaired night vision (''nyctalopia''). In addition, they frequently report one or more of the following visual and nonvisual symptoms: photopsia, migraine, phonophobia, hyperacusis, cutaneous allodynia, tinnitus, balance problems, and tremor. Thus, those with VSS report a wide spectrum of visual and nonvisual symptoms of a sensory, motor, and perceptual nature, with many involving the visual system.

INTRODUCTION

VS, in isolation, is an uncommon (3.7% in the UK population) [7] neurologic condition first reported by Frank Carroll related to use of the medication digitalis prescribed for heart conditions [8]. Moreover, one recent sample estimate of the prevalence of the more visually encompassing VSS in the UK population was 2.2% [7]. The conditions of VS/VSS remain an enigma. VSS is a complex entity having a wide and diverse range of visual and nonvisual symptoms, with some complicating comorbid conditions (eg, migraine) [2–6,9]. Until recently, there were few successful therapeutic options for the reduction of the primary symptom of VS, and its other disturbing visuo-perceptual (eg, palinopsia) and

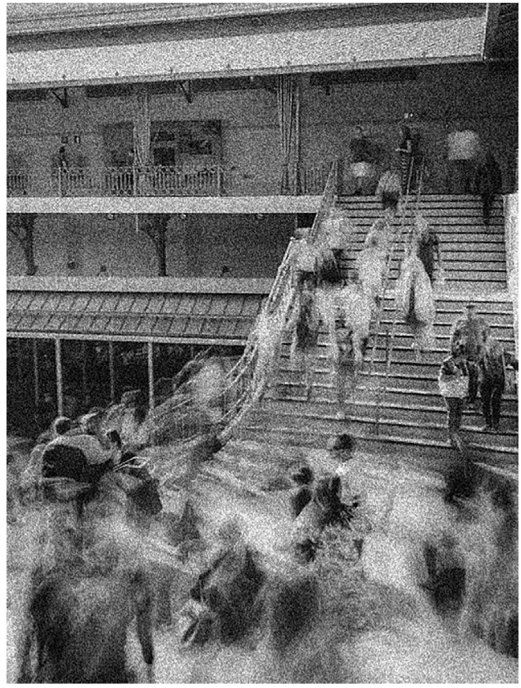

Fig. 2. Complex, naturalistic, dynamic visual scene showing palinopsia with trailing and superimposed VS.

visuo-motor/general motor (eg, balance problems, tremor) phenomena for VSS per se. Unfortunately, in the past, in some cases, these abnormalities were either dismissed or misdiagnosed by some doctors [10,11].

There have been considerable research efforts into ascertaining the patient history, defining characteristics, categorization, and diagnostic aspects of the VSS [2–5]; this was critical to our understanding of this condition. However, a relative paucity of efforts directed at therapeutic aspects exists. In the past 6 years or so, several clinical research reports have occurred describing successful therapies and related protocols using the broad neuro-optometric approach [6,9–19], which primarily includes not only the prescription of chromatic tints and oculomotor-based vision therapy, but also visuomotor integrative aspects [12]. Treatment will be a primary focus of the present article.

Neurophysiology of visual snow/visual snow syndrome

Over the past decade, considerable effort has occurred to uncover the neural substrates and mechanisms underpinning VS/VSS. Several sites have been proposed, yet there remains no consensus. The research indicates a heterogeneous, network-like disorder involving multiple sensory and attentional processing sites. The four most frequently cited neural areas are presented in Table 1.

Table 1
Most commonly proposed active neural sites with regard to visual snow syndrome based on brain imaging and other techniques

Neurologic Site	Neuroimaging Techniques	Findings
Right lingual gyrus	PET, fMRI, VBM, H-MRS	Increased GMV, hyperconnectivity with the thalamus/basal ganglia, hypermetabolism
Visual cortex and higher visual processing areas	MRI, VBM, DTI, VEP	Increased GMV, WM alterations, abnormal VEP responses
Thalamocortical Pathways	MRI, IC	Hypoconnectivity with the basal ganglia, S-cone excitation
Attentional/Salience Networks	fMRI, IOR	Hyper and hypoconnectivity within the dorsal/ventral attention networks and DMN Decreased activity in the ACC and anterior insulae, increased saccadic latency and errors

Abbreviations: ACC, anterior cingulate cortex; DMN, default mode network; DTI, diffusion tensor imaging; fMRI, functional MRI; GMV, gray matter volume; H-MRS, proton magnetic resonance spectroscopy; IC, Intuitive Colorimeter; IOR, inhibition of return; S-cone, short-wavelength cone; VBM, voxel-based morphometry; VEP, visual-evoked potential; WM, white matter.

They include the lingual gyrus, visual cortex, thalamus, and attentional/salience networks [20–30].

Lingual gyrus

The lingual gyrus, located in the occipital lobe, plays a role in visual memory, facial recognition, and the perception of letters, shapes, colors, and motion. Studies have shown that individuals with VS/VSS often exhibit increased gray matter volume (GMV) and hypermetabolism of the lingual gyrus [20–23]. Hypermetabolism of the right lingual gyrus has been demonstrated with PET imaging [20], and increased GMV in the right lingual gyrus has been shown with voxel-based morphometry (VBM) [21] These changes were most prominent in the lingual gyrus–fusiform gyrus junction [20] and in the post-thalamic visual pathways from the right pulvinar to the right lingual gyrus [22]. The magnitude of GMV was associated with disease duration but not severity. The researchers also found increased lactate and glutamate concentrations in the right lingual gyrus of VS patients, thus suggesting metabolic hyperactivation [23].

These changes in GMV likely indicate a dynamic increase in synaptic strength and plasticity over time. This increase in synaptic activity may reflect

chronicity. Given the role of the lingual gyrus in multisensory integration, alterations in connectivity and metabolism could explain the abnormalities beyond vision that individuals with VSS report, including auditory and tactile sensitivities.

Visual cortex

The visual cortex consists of multiple specialized areas that collaboratively form one's conscious visual experience. Individuals with VSS exhibited morphologic changes in GMV of the left primary (V1) and secondary (V2) visual cortices, as well as the left visual motion area (V5), when compared with matched controls [24]. White matter alterations also were evident with diffusion tensor imaging (DTI) in the visual cortex, as well as the frontal and temporal cortex, including the inferior fronto-occipital fascicle, sagittal stratum, and right superior longitudinal fasciculus [25]. Electrophysical studies using VEP indicate possible dysfunction in the primary visual and visual association cortices as well, but the results were mixed [20].

These areas comprise the visual motion network in the dorsal stream. It is plausible that hyperexcitation in these areas may result in the illusion of a moving, full-field, pixelated overlay. These anatomic changes were not associated with clinical features, such as duration or severity, perhaps indicating an inherent trait and not a consequence of VSS per se.

Thalamus

The thalamus plays a crucial role in relaying sensory information to the cortex. Dysregulation in thalamic processing has been proposed as a potential factor in VSS [20].

In contrast to post-thalamic pathways to the lingual gyrus, there was reduced connectivity between the pulvinar and bilateral dorsal aspects of the caudate nuclei [23]. This part of the visual cortico-striatal loop helps *inhibit* general visual noise through a feedforward mechanism. A bottom-up disruption may allow this error to reach higher processing areas, implying a disruption in filtering and integrating visual information versus internal generation.

When neuronal *integration* and *synchronization* of the thalamus is disrupted, thalamocortical dysrhythmia (TCD) may arise. TCD is thought to be a frequency imbalance due to dysregulated neural oscillations between the thalamus and cortex, thus contributing to a host of persistent sensory disturbances. With the Intuitive Colorimeter (IC), VS patients exhibited a yellow–blue color preference and disliked blue–violet wavelengths as compared with controls [26]. This could implicate the koniocellular pathway, which includes cells that transmit short-wave (S-cone) signals related to blue–yellow color vision through the thalamocortical pathway. These findings further support the use of chromatic filters, which may promote thalamocortical synchrony.

Attentional and salience networks

These networks allocate cognitive resources to facilitate directed attention and awareness of relevant stimuli in the environment. Widespread disturbances

were found in those with VSS in the integration of these networks with the ventral attention network (VAN) and dorsal attention network (DAN) using fMRI [22]. Several parts of the VAN were less well integrated with the visual motion network, thus suggesting a reduced capacity to refocus visual attention to the environment.

Altered coupling also was noted between the default mode network (DMN) and DAN, and within the salience network itself. The DMN is active when an individual is at rest or engaged in internally focused thoughts. The anterior cingulate cortex (ACC) and anterior insula (AI) function as part of the salience network to assign significance to sensory and internal stimuli. Any aberrant connectivity between these networks implies a shift from the self-referential cognition of the default mode network (DFM) to the directed attention of the DAN. This dysfunctional salience and attentional modulation may misappropriate irrelevant internal stimuli as external perception.

Further support for an attentional interaction comes from eye movement studies. Patients with VSS had longer prosaccade latencies and more antisaccade errors, as well as a delayed onset of inhibition of return [27]. The investigators suggest that disrupted attentional networks may increase saccade-related activity, as opposed to poor frontally mediated inhibitory control. These findings may provide an oculomotor profile to aid in the measurement of dysfunction and assist in the determination of a therapeutic intervention.

Other brain sites

Many other neurologic sites have been implicated including the frontal eye fields, right middle temporal (MT) gyrus, right parahippocampal gyrus, left superior temporal gyrus, bilateral cuneus, precuneus, supplementary motor cortex, premotor cortex, posterior cingulate cortex, left primary auditory cortex, left fusiform gyrus, and left cerebellum [28]. This list is not exhaustive, and it is important to view this condition as a *multidimensional sensorimotor network disorder*, combining afferent, efferent, and central processing components. Clearly, more research in the area is needed to obtain a more accurate representation of the VS/VSS neural pathways.

It is worth noting that this condition is related to, but distinct from, migraine, thus displaying its own metabolic and structural profile on neuroimaging studies. In addition, neuro-ophthalmic evaluation has shown normal pupillary light reflex and contrast sensitivity in patients with VSS compared with migraineurs further supporting this distinction [29].

Many possibilities have been proposed to be the neurophysiological-based etiology of VS, as discussed earlier. One that is frequently mentioned is hyperexcitability of the angular gyrus [30]. However, more recently, another neural site was proposed, namely the extrastriate visual area of the MT region [19]. It is well-known that MT is active in the generation and perception of visual motion and furthermore has poor overall retinotopic mapping. However, when its small clusters of cells are stimulated, they *are* directionally specific. But, when injured, for example, a mild traumatic brain injury (mTBI), a marked increase

occurs in its cellular spontaneous activity, thus resulting in hyperexcitability of its cells. If sustained, current spread would take place, with the resultant simultaneous stimulation of *all* of the cells. This would give rise to multidirectional signals, that is, dynamic visual noise, that is, VS. Furthermore, because MT has a chromatic input, this could be responsible for the VS being chromatic in most cases. Hence, the use of a precision chromatic filter would reduce the overall luminance of the visual field and in turn decrease the cellular hyperexcitability, as well as its chromatic bias acting to filter out the blue–green end of the spectrum.

DIAGNOSIS OF VISUAL SNOW SYNDROME

The diagnosis of VSS is relatively easy to make for the experienced neuro-optometrist, and other practitioners, having read about, examined, diagnosed, and treated many such patients. However, this is not necessarily so for the inexperienced with this population. For example, the primary, presenting visual symptom of VS itself can be perplexing. And, in conjunction with such seemingly unrelated visual symptoms of photosensitivity and enhanced entoptic imagery, as well as nonvisual symptoms such as tinnitus and cutaneous allodynia, one can be stymied, even though only a few other conditions exist that strictly meet the VSS criteria [31,32] to require a complicated differential diagnosis (eg, occipital epilepsy). However, some conditions (eg, Creutzfeldt-Jakob disease, hallucinogen persisting perception disorder) may have VS without VSS [31,32] and, hence, may require more tests for the correct diagnosis [13,18]. In this section, critical aspects to assist in the diagnosis of VSS will be considered.

A range of tools exist in the clinician armamentarium of the neuro-optometrist, and others, to assist in the diagnosis of VSS [6,9–19]. One early comprehensive approach (Box 2) has been proposed [13] with its emphasis on making the diagnosis based on exclusion. Another approach is proposed here in detail (Box 3) [18]. The latter includes a detailed case history, several questionnaires, the basic vision examination, specialized sensory testing, and an expanded binocular/ocular motility workup.

Case history

A comprehensive case history is critical with any patient, but perhaps more so in those with suspected VS/VSS due to their unusual, and varied visual and nonvisual symptoms. The chronology of events is important to ascertain possible correlates with the first presence of VS (eg, concussion, medications, brain surgery). How the presence of the VS/VSS adversely impacts their vocational and avocational pursuits should be determined, with an eye on priorities and related optimal therapeutic intervention(s). A full description of the basic VS/VSS characteristics and symptoms (eg, constant, chromatic, photosensitivity, palinopsia), provocative/exacerbating conditions (eg, anxiety, high room illumination), and any other prior treatments (eg, phototherapy such

Box 2: Proposed neuro-optometric diagnostic test protocol for visual snow syndrome

Advanced Tests

- Electroretinography (ERG): to assess objectively retinal physiology/integrity
- Visual-evoked potential (VEP): to assess objectively visual cortical physiology/integrity
- Dynamic posturography/gait analysis: to assess objectively balance/postural stability/ambulation
- Dark adaptation: to assess retinal rod/cone physiology/integrity
- Intuitive Colorimetry (IC): to assess quantitatively chromatic filter effects on the perception of VS

Basic Tests

- Baseline, comprehensive vision examination: to assess refractive, binocular, and ocular health aspects
- Corneal topography: to assess for visual distortion
- Optical coherence tomography (OCT): to assess retinal anatomy/integrity
- B-scan ultrasound: to assess retinal and vitreal anatomy/integrity
- Critical flicker frequency (CFF): to assess temporally based, global visual neurosensory integrity
- Visual fields (VF): to assess global visual field integrity
- Contrast sensitivity function (CSF): to assess effects of entoptic phenomena on low-contrast visual perception
- Amsler grid: to assess visuospatial directional integrity
- Dynamic visual acuity (DVA): to assess global visuo-vestibular interactive integrity
- Egocentric localization: to assess the sense of ''straight ahead,'' which may affect balance/gait
- Balance test: to assess globally postural stability
- Tremor test: to assess globally fine visuomotor integrity
- Filter test: to assess the effects of chromatic/achromatic filters on the perception of VS and related VSS phenomena.
- Draw/describe what you see: to assess/confirm the presence of VS and related VSS phenomena

From Ciuffreda KJ, Han MHE, Tannen B. Pediatric visual snow syndrome (VSS): a case series. Vis Dev Rehabil 2019; 5:249-253.

as syntonics, chromatic tints, oculomotor-based vision therapy) and their level of success needs to be detailed, as well as any other possible relevant factors.

Symptom questionnaires

Four specific questionnaires exist that should be administered following the detailed case history described. The first is the Visual Snow Syndrome Symptom Survey (VSSSS) (Fig. 3) developed by the SUNY Visual Snow Group

Box 3: Visual snow syndrome comprehensive diagnostic vision assessment

VSS Comprehensive Vision Assessment

1. Detailed case history
   a. Basic case history
   b. Completion of VSSSS questionnaire
   c. Completion of BIVSS global symptom questionnaire
   d. Completion of VLSQ-8 photosensitivity questionnaire
   e. Visual Snow Handicap Inventory (VSHI)
2. Comprehensive vision examination: refractive, binocular, ocular health
3. Additional sensory testing
4. Expanded binocular vision/oculomotor workup

Adapted from Han MHE, Ciuffreda KJ, Rutner D. Historical, diagnostic, and chromatic treatment in visual snow syndrome: a retrospective analysis. Optom Vis Sci 2023; 100:328-333.

based on earlier defining studies [1–6] incorporating demographic and related symptomatology, with a rating scale of 0 to 2 [9]. The second is the Brain Injury Vision Symptom Survey [33]. It addresses the area more broadly, with an emphasis on binocular/oculomotor concerns (visual comfort, reading, diplopia), with a rating scale of 0 to 4. It has become particularly relevant, as a

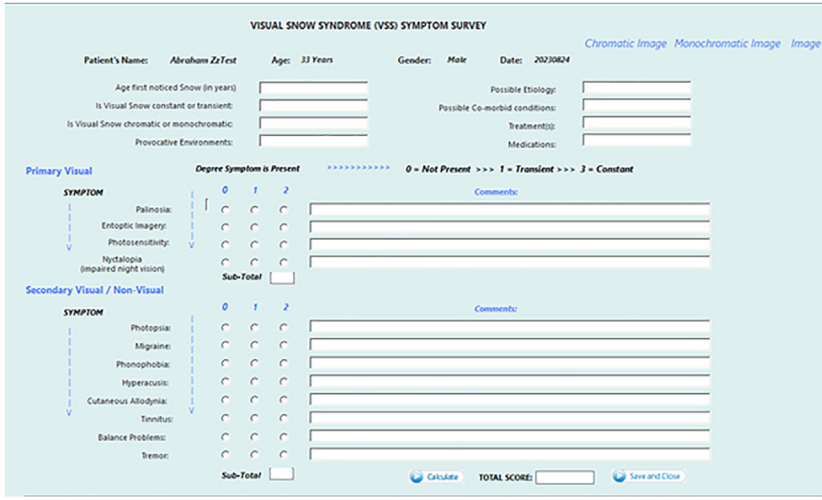

Fig. 3. The SUNY visual snow syndrome symptom survey (VSSSS). (*Adapted from* Ciuffreda KJ, Tannen B, Han MHE. Visual snow syndrome: an evolving neuro-optometric perspective. Vis Dev Rehabil 2019; 5:75-82.)

recent study revealed that up to 60% of those diagnosed with VSS manifest such problems [10]. The third is the Visual Light Sensitivity Questionnaire (VLSQ-8) [34], dealing solely with the symptom of photosensitivity, which is so prevalent in this population (~80%) [19]. It has a 5-point rating scale. The fourth is the Visual Snow Handicap Inventory (VSHI) [35]. It is composed of a 25-point scale, which assesses the patient's *perceived* disability from the presence of the VS/VSS, especially the VS. There are three possible responses: Yes, Sometimes, and No.

Comprehensive vision examination

A comprehensive vision examination is crucial to establish the overall baseline condition and visual status [18]. It includes three areas: refractive, binocular, and ocular health status. Regarding the first, this should include distance and near visual acuity, with and without the most current prescription, autorefraction, distance and near retinoscopy, and keratometry/corneal topography, all followed by the subjective refractive refinement. For the second area, this should include a cover test at distance and near in all positions of gaze and include the Marlow prolonged occlusion test at near in primary position, near point of convergence with repetition to assess fatigue effects, relative accommodation and vergence, accommodative amplitude in each eye and binocularly, stereoacuity, and global ocular motility, that is, fixation, saccades, pursuit, vestibular, and vergence. Last, regarding ocular health status (and related systemic manifestations and medications), this would include gross visual observation of the eyes and anterior segment using a penlight and slit-lamp gonioscopy, pupillary reflexes to light and accommodation, intraocular pressure, direct/indirect ophthalmoscopy, ocular coherence tomography, color vision, and screening visual fields. These would be the minimum tests for each area. Other tests might be included, as deemed informative by the doctor.

Additional special testing

Some additional testing may be helpful to understand the full spectrum of the visual dysfunctions, especially those of a neurosensory nature. This includes static and dynamic contrast sensitivity [29,36], visual fields (as 20% manifest some loss) [37], and the visual-evoked response [38]. However, the deficits may be of very subtle nature and require careful assessment to uncover. This should be explored in future clinical research studies.

Expanded binocular vision/oculomotor workup

As mentioned earlier, a very high prevalence of clinically-based, binocular/oculomotor vision problems, and related visual symptoms, occurs in this population. Some of the more common diagnoses include general oculomotor dysfunction (ie, versional), convergence insufficiency, and accommodative insufficiency. Some of the more common visual symptoms are intermittent diplopia, transient blur, and skipping lines while reading. Thus, additional probing into this important area is warranted (Box 4) [10,18,19].

Box 4: Expanded binocular vision/ocular motility diagnostic workup

- Accommodative facility
  - (± 2D or age-appropriate)
  - monocular and binocular at near
- Vergence facility
  - distance: 2pdBI/4pd BO prism flippers
  - near: 4pd BI/12pd BO prism flippers
  - fusional vergence ranges at near in extended free space with vectograms
- Distance and near phoria
  - Telebinocular in instrument adjunct measure
  - Modified Thorington in free space as an adjunct measure
- Near phoria while performing a visuo-motor task
  - Van Norden Star Test
  - Cheiroscope to assess for subtle central suppression.
- Developmental Eye Movement (DEM) test to assess saccadic sequencing and relation to reading/automaticity
- Horizontal, vertical, and oblique versional eye movements to assess for accuracy and field extent (eg, paresis)
- Objectively-based reading and versional eye movement assessment
  - RightEye
  - ReadAlyzer

From Han MHE, Ciuffreda KJ, Rutner D. Historical, diagnostic, and chromatic treatment in visual snow syndrome: a retrospective analysis. Optom Vis Sci 2023; 100:328-333.

TREATMENT FOR VISUAL SNOW SYNDROME

Several treatments have been proposed and attempted for the remediation of the VS and related perceptual phenomena (Box 5) [6,9–19,28]. Most have had success.

Discussion of the condition with the patient

The *first* treatment approach is the simplest, namely the clinician having a thorough discussion of the condition with the patient [2,10]. Some with VSS, at the

Box 5: Treatment for visual snow syndrome

1. Discussion of the condition with the patient
2. Medications
3. Chromatic tints
4. Oculomotor-based vision therapy
5. Environmental alterations

Table 2
The three most commonly used medications for visual snow syndrome

Most common Medication in order of use	Name of Medication
1.	Lamotrigine
2.	Verapamil
3.	Acetazolamide

first VS presentation, are especially concerned, and rightfully so. For example, they wonder if it will get worse, will they eventually lose their vision, and if is it associated with any other serious medical condition that might negatively impact their quality of life (QOL). Fortunately, in most cases, the answer is no. This response typically alleviates their fears. Such initial fears are not unwarranted, however, as VS is such an unusual and disturbing visual phenomenon of sudden onset. Furthermore, caution should be exercised, as possible pathologic etiology must be ruled out [31,32]. In addition, it has been a little understood, or even misunderstood, neuro-perceptual affliction [10,11,28]. Last, VSS can have a negative and alarming effect on one's QOL. For example, the VS and the frequently associated palinopsia (especially with trailing) can adversely affect reading continuity and comprehension [10], as well as have adverse driving consequences [6].

Medications

The *second* treatment approach involves medications (Table 2). The lack of a well-defined pathophysiological mechanism and related anatomic substrates has hindered the development of targeted treatments for VS/VSS. The breadth of studies investigating pharmacologic management indicates that only a minority of patients benefit from medications. In some trials, however, medications have exacerbated the symptoms [39]. Prospective studies have been rare, with most data coming from case reports, case series, and retrospective cohort studies, which make comparisons difficult [29]. The primary focus has been on anticonvulsants, with lamotrigine showing mixed results. Additional pharmaceutical treatments have included calcium-channel blockers, beta blockers, diuretics, benzodiazepines, antidepressants, triptans, antipsychotics, nonsteroidal anti-inflammatory medications, antiplatelet agents, antibiotics, antifungals, muscle relaxants, and vitamins/nutraceuticals. Patients with VS/VSS may be understandably reluctant to take these medications due to concerns about potential adverse effects.

Anticonvulsants

Anticonvulsants include lamotrigine, topiramate, gabapentin, pregabalin, and valproate. Results have been mixed at best, with lamotrigine and topiramate showing the greatest therapeutic effect. In one study of 26 patients taking lamotrigine, 19.2% reported partial remission of their symptoms [40]. Another systematic review found lamotrigine to be partially effective in 8 of 36 patients,

followed by topiramate in 2 of 13 patients [20]. However, a survey conducted by Puledda and colleagues did not confirm this trend [39]. Although lamotrigine and topiramate had an improvement frequency of 21% and 18%, respectively, up to 35% of patients also reported a *worsening* of symptoms.

Although the mechanisms of action vary between these medications, anticonvulsants modulate various aspects of synaptic transmission, stabilizing neuronal membranes and reducing abnormal synaptic activity in the brain. Lamotrigine down-regulates glutamate, which is known to propagate cortical spreading depression in migraine. It is thought that reduced neuronal hyperexcitability may result in less overactivity in the regions of the brain contributing to VS/VSS.

However, these medications are not without side effects. The most common side effects include nausea, dizziness, drowsiness, fatigue, and headache. More serious side effects, such as Stevens–Johnson syndrome, also can occur [40].

Antihypertensives

Antihypertensives include calcium channel blockers, such as verapamil and flunarizine, and beta-blockers, namely propranolol. Scattered case reports have found these to have a partial benefit for some patients. For example, Puledda and colleagues [39] found the proportion between the total number of reported improvements and the total number of reported worsening (ie, the therapeutic ratio) to be 1.6:1. Support for these medications remains scarce.

Diuretics

Diuretics include acetazolamide and furosemide. This class of drugs has shown some degree of effectiveness in patients with migraine, migraine with aura, and persistent visual symptoms related to migraine in limited case studies [41]. Yet, there remains no evidence for their effectiveness in patients with VS/VSS.

Acetazolamide, a carbonic anhydrase inhibitor, decreases the production of cerebrospinal fluid and reduces intracranial pressure while promoting the dilation of blood vessels. Furosemide inhibits the cellular membrane Na+/K + -ATPase pump, which disrupts extracellular potassium accumulation. Both medications decrease the susceptibility of neurons to cortical spreading depression. Common side effects include extremity paresthesias, lightheadedness, dry mouth, gastrointestinal disturbances, hypotension, hyperglycemia, polyuria, metabolic acidosis, and renal dysfunction [41].

Chromatic tints

The *third* treatment approach involves the application of chromatic tints (Box 6) [18,19]. It is conducted in two sequential phases [18].

The *initial phase* of the assessment is conducted using the IC, if available (Ceriumvistech.com), to obtain a "precision tint" incorporating a well-developed protocol and underlying psychophysical approach (Fig. 4) [42,43]. It takes less than 30 minutes for completion. The clinician obtains a precision tint with respect to hue, saturation, and overall percent luminance transmittance. Basically, the patient views some black-and-white text with surrounding patterns, whereas the

Box 6: Visual snow syndrome tint assessment and prescribing

1. Initial Tint Selection
   a. Intuitive Colorimeter
   b. Full spectrum of general ophthalmic tints
   c. FL-41 and BPI-Omega tints
2. Performance Specific Tint Selection, including vision therapy for any binocular/oculomotor dysfunction, with chromatic tint if deemed beneficial from the above testing.
   a. Computer simulation of various environmental conditions
   b. Natural surrounds (especially provocative ones)
   c. Computer scrolling
   d. Different illuminations
   e. Long hallway with obstacles and signage
   f. Follow-up: 1, 3, and 6 months

From Rutner D, Ciuffreda KJ. A clinical diagnostic and treatment protocol for the patient with visual snow/visual snow syndrome and concurrent binocular dysfunctions. Vis Dev Rehabil 2023; 9:7-14.

above three parameters are adjusted to determine which combination results in the greatest reduction of VS and other related abnormal visual phenomena. Then, these three values are entered into the IC system's computer program to ascertain that a combination of the 42 chromatic trial lenses results in the optimal tint, which the patient can now assess. In addition, one obtains a color plot of wavelength versus percent transmittance for this specific precision tint [18,19]. This plot is helpful for the clinician to obtain direct visualization and better understanding of the precision tint's spectral chromatic bias and range of effectiveness. This precision tint can then be ordered from the company and be incorporated into the patient's spectacle refractive correction.

Following obtaining this optimal chromatic tint reference point, the clinician can try more readily available general ophthalmic tints having similar spectral

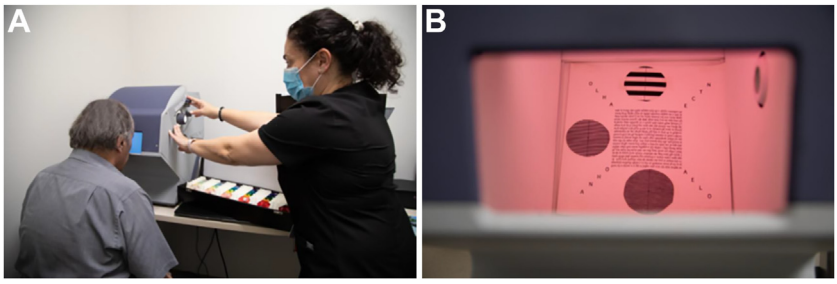

Fig. 4. Intuitive Colorimeter (IC). (*A*) Patient being tested in the instrument with nearby precision tints. (*B*) Patient's view of the instrument and stimuli. (*From* Rutner D, Ciuffreda KJ. A clinical diagnostic and treatment protocol for the patient with visual snow/visual snow syndrome and concurrent binocular dysfunctions. Vis Dev Rehabil 2023; 9:7-14.)

characteristics (chadwickoptical.com). If one does not have the IC, then this would be the starting point for testing. These lenses are usually easier to order and less expensive than those for the IC. If any of these lenses are initially found to be similarly helpful, then the patient is directed to assess their effectiveness (ie, percent perceived reduction of the VS and other related abnormal visual phenomena) for a range of natural conditions, such as a computer screen, as well as what was deemed to be their most provocative/exacerbating viewing situation (eg, in high room illumination) [19]. Last, the patient can assess perceptual effectiveness of two other commonly prescribed and readily available filters: FL-41 (eschenbach.com; chadwickoptical.com) and BPI-Omega (callbpi.com). In one clinical report [10], these two lenses were found to be effective in 90% of the patients tested. However, this study did not have an IC comparison test lens. In general, these filters reduce transmittance in the offensive blue–green end of the visible spectrum. Last, the prescription of a chromatic filter may simultaneously reduce several of the visual symptoms, for example, the VS, palinopsia, and photosensitivity (Fig. 5).

In *phase* 2, there are two aspects. The *first* aspect is an extension of the aforementioned [18] (see Box 6). The patient is now presented with additional possible scenarios to assess tint performance and any changes in VS perception. This would include walking along a long hallway with multiple objects present and also viewing computer-simulated VS scenes (https://visionsimulations.com/visual-snow.htm), and more. Thus, the full tint assessment covers an extensive range of likely visual conditions to be encountered in one's everyday environment. Given the multitude of potential visual scenarios, some patients might require two separate chromatic spectacle prescriptions, such as one for computer viewing and another for general outdoor/indoor conditions [19]. These tints can be prescribed in the form of clip-overs for added convenience and less expense [19]. The *second* aspect involves repetition of the earlier binocular/oculomotor diagnostic workup, but *now* with the optimal chromatic prescription in place to ascertain how the perceived reduced presence of VS, and any other related disturbing visual perceptual phenomena, impact on the findings. Informally, we have found that approximately 20% of the VSS patients exhibited improved binocular/oculomotor performance with the optimal chromatic tint in place; that is, with less perceptual interference of the visual test targets, several of the initial clinical test findings improved. For example, the frontal plane of VS might result in an increased accommodative error per the frontal plane of visual interference (ie, Mandelbaum effect) [6], which the tint would likely reduce. This observation needs to be carefully explored further. Once prescribed, the patient should return for a progress evaluation to assess continued efficacy of the tint, as chromatic adaptation [18,19,26,44] might alter its short- and long-term effectiveness.

Oculomotor-based vision therapy

Because a large percentage of individuals with VSS have common oculomotor deficits (eg, convergence insufficiency, saccadic dysmetria, accommodative

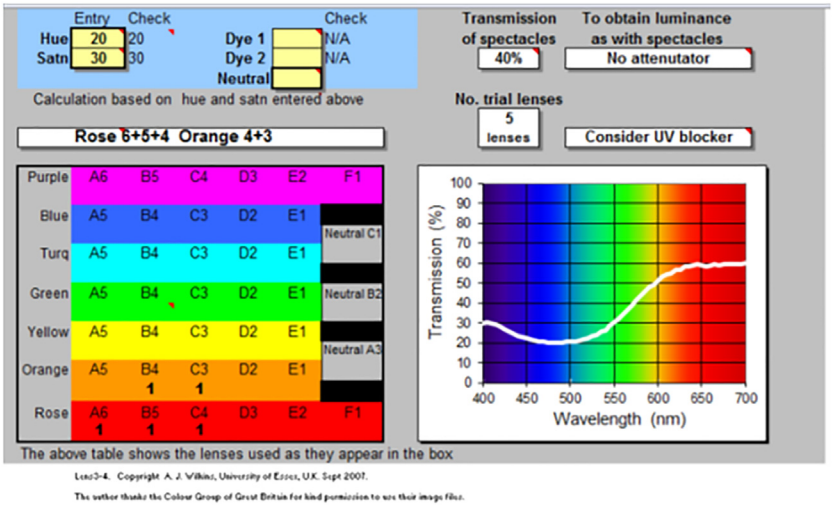

Fig. 5. Intuitive Colorimeter (IC) tint specification (*left* and *top*). Spectral range and magnitude of the effect with respect to transmission as a function of wavelength for a precision tint (*right*). (*From* Han MHE, Ciuffreda KJ, Rutner D. Historical, diagnostic, and chromatic treatment in visual snow syndrome: a retrospective analysis. Optom Vis Sci 2023; 100:328-333.)

insufficiency) [45] and related problems (eg, visuomotor deficits, balance difficulties) [12], active and broad-based therapeutic interventions are warranted [10,12,18,45,46]. Conventional vision therapy for the range of common oculomotor dysfunctions should be implemented (eg, prism/lens flippers, Hart chart saccades) [10,45] as well as specific saccadic therapy for remediation of the palinopsia [10]. In addition, related visuomotor (eg, eye-hand coordination) and global balance therapy [12,46] should be helpful (see Box 2).

Environmental alterations

Some changes in one's visual environment may also prove helpful to the patient with VS/VSS. For example, one can alter their iPhone and computer screen settings to approximate that of their prescribed chromatic tinted spectacles when they are not worn/available [14]. If the overhead illumination in one's home exacerbates the VS and related visual symptoms, then the luminaires can be changed to something less provocative [18]. For example, fluorescent illumination is biased toward the blue/lower end of the visible spectrum, whereas tungsten illumination is biased toward the red/higher end of the spectrum. The same concept is true regarding the paint color scheme of the problematic rooms. Also, fluorescent illumination with its 60-Hz refresh rate approaches the critical flicker fusion frequency of some concussion patients [47,48], who may also have VS/VSS and hence be perceptually troublesome. Thus, several interactive visual effects need to be considered. The patient and doctor need to be creative in this endeavor!

RELEVANCE AND FUTURE AVENUES

VSS is an important, and emerging, neurologic/neuro-optometric condition, which has an estimated prevalence of 2.2%, with the prevalence of VS alone in 3.7% [7]. Hence, it has contemporary public health and related economic ramifications. For example, as VS and VSS have become "new," neurologic conditions, governmental agencies need to consider them in their basic health care statistics dealing with a range of other medical/optometric conditions (eg, amblyopia, strabismus, macular degeneration) prevalent in the United States (eg, the National Center for Health Care Statistics, www.cdc.gov>nchs), including its overall fiscal and societal impact.

Concerning optometry/neuro-optometry per se, the above agencies and others (eg, Medicaid, vision care plans) dealing with the patient's vision care need to incorporate the conditions of VS/VSS into their benefits package (eg, for the specialized IC examination, costs of the precision chromatic tint of a medical nature), rather than simply dismissing the condition and its important care. For example, an insurance company may decide not to reimburse the doctor and patient for a precision chromatic tint by considering it "non-essential, cosmetic," along with the oculomotor-based vision therapy for remediation of the frequently concurrently occurring, and visually disturbing, palinopsia.

Thus, a need exists for governmental lobbying to increase awareness of the condition, and its related disabling consequences, both vocational (eg, job requirements) and avocational (eg, hobbies, pleasure driving), in nature. Groups, such as the American Academy of Optometry, the American Optometric Association, the College of Optometrists in Vision Development, the National Academies of Practice, the Optometric Extension Program, and the Neuro-Optometric Rehabilitation Association, as well as neurologic and ophthalmologic associations, should be actively engaged in the lobbying process.

FUTURE DIRECTIONS

Improved communication is a critical factor. As mentioned earlier, a better and more complete patient–doctor discussion and interaction is important for the patient to understand the condition, and its relatively high prevalence, to alleviate their fear and anxiety, the latter of which tends to exacerbate their attentional awareness of these visual phenomena [49], and thus it produces a vicious cycle. The patient might also be referred to a clinical psychologist (eg, cognitive behavioral therapist, cognitive behavioral therapist [CBT] specialist) to develop coping strategies to reduce their periods of anxiety and its adverse effect on the perception of VS and its related visual disturbances. In conjunction with the abovementioned, the patient also might be referred to a psychiatrist for prescription of an antianxiety medication. Surprisingly, there needs to be more education for many primary care providers (eg, optometrists, ophthalmologists), and others (eg, neurologists, neuropsychologists), who may interact with the patient. Thus, a need for better knowledge exists and in turn more effective communication at all levels. In addition, the notion of improved communication needs to be extended to our widely used social media networks (eg,

Facebook) and organizations/groups involved in VS advocacy (eg, The Visual Snow Initiative, www.visualsnowinitiative.org).

Related to the abovementioned, having unique international, ICD-11-CM diagnostic codes for VS and VSS would help legitimize its status. Currently, the formal diagnostic description is "subjective visual disturbances, 53.1." This vague nomenclature only assists in masking and diluting the specific, and abnormal, visual perceptual disturbances. Given its currently high level of clinical and basic research interests (eg, 144 citations for VS and 89 for VSS in PubMed,10/19/2023), with many being published in the past 5 years, recognizing its uniqueness and importance in the scientific, clinical, and governmental worlds will likely happen shortly.

Further clinical and basic research investigations are needed. The former could involve well-conducted clinical studies (eg, case series, prospective analysis) and formal clinical trials. The resultant information would lead to better understanding of the clinical scenario with respect to refining the diagnosis and improving the treatment. This could include the use of chromatic tints, and oculomotor-based vision therapy, as well as less conventional approaches (eg, photo light therapy, syntonics) [50]. In parallel, laboratory studies should be conducted to investigate further the physiologic and perceptual underpinnings of this enigmatic condition. This could include brain imaging before and after some therapeutic intervention (eg, precision chromatic tints, oculomotor-based vision therapy).

Related to the abovementioned, better optical options would be important. For example, the development of lenses with narrow band ($\pm$ 10 nm), thin-coating filtering ability, with reasonable costs and availability to both the doctor and patient, would be helpful (www.perceptcorp.com) [51,52]. The specific therapeutic, narrow band filter could be set for the peak of the disturbing wavelength following careful, quantitative testing (eg, with the IC). This would provide more efficacious treatment as well as reduce the side effects of altered color perception and reduced overall luminance of the scene. This is critical for certain occupations, such as tailors and policemen, as well as commercial truck drivers, regarding the effective luminous intensity of a tricolor traffic stop light, especially under adverse weather conditions (eg, fog, snow) [6]. Altered and disturbed color perception could produce transient mental confusion and lead to a dangerously increased reaction time to a "stop" traffic signal, especially in the elderly and in those with mTBI [53–55], and the aforementioned would be exacerbated by the presence of any residual VS. [6]. Last, therapeutic chromatic lenses could be developed in the form of contact lenses, or even be changeable from achromatic to chromatic by being triggered with changes in one's environment (eg, room color), as is true for the Photo Sun spectacle lens.

Last, there are two important differential diagnostic aspects that need to be parsed and better defined. First, it is important to differentiate between "latent VS" and "manifest VS" [49]. The former relates to those who either do not realize that they have the VS or for whom it is not bothersome. In contrast, the latter perceives the VS, and it is their primary visual symptom, and very disturbing. Second, and related to the abovementioned, a need exists to

understand better the visual perceptual-based, neural substrates for VS versus VSS in these two diagnostic groups, and their interactions.

SUMMARY

The condition of VSS is an emerging neurologic/neuro-optometric condition having a wide range of visual and nonvisual symptoms. These are of a sensory, motor, and perceptual nature. VSS is estimated to be present in at least 2% of the population, which is a far cry from the early days of stating that it was a "rare" condition. This value is the same as the estimated prevalence of amblyopia, which is a common and important visual abnormality [56].

Until relatively recently, the focus of attention has been on defining the entity, and then developing detailed, diagnostic criteria. This was critical to understand this complex, and somewhat enigmatic, condition. However, more recently, the focus has been on two areas. First, on the therapeutic applications, namely chromatic filters, and basic vision therapy/oculomotor-based vision therapy. Second, on laboratory investigations, namely psychophysical studies to provide insight into the underlying basic neurobiological/visual physiologic causes of the problem, and related brain imaging to determine the site(s) of the anomaly [57,58], with the hope of objectively quantifying the enhanced brain-based effect of some therapeutic intervention.

The increased recognition and diagnosis by professionals and others of this unusual condition, and its treatment, bodes well for the future of the patient with VSS. Their treatment options will expand as our basic and clinical understanding of the condition increases.

CLINICS CARE POINTS

- Visual snow (VS) is found in about 4% of the general population, whereas visual snow syndrome (VSS) is found in about 2% of the general population and is generally benign.
- VSS is found in a range of neurologic conditions, such as concussion and following brain surgery with anesthesia.
- Individuals with VSS present with a constellation of symptoms of a sensory, motor, and/or perceptual nature, with many involving the visual system (eg, palinopsia, photosensitivity, photopsia).
- VSS can be treated primarily with chromatic tints for the VS and palinopsia as well as oculomotor-based vision therapy for the palinopsia and high frequency of occurrence (~60%) of binocular dysfunctions, such as convergence insufficiency and saccadic dysmetria.

References

[1] Graber M, Scutelmic A, Klein A, et al. Natural course of visual snow syndrome: a long-term follow-up study. Brain Commun 2022;4(5):fcoc 230.

[2] White OB, Clough M, McKendrick AM, et al. Visual snow: visual misperceptions. J Neuro Ophthalmol 2018;94:514–21.
[3] Puledda F, Schankin C, Digre K, et al. Visual snow syndrome: what we know so far. Clin Opin Neurol 2018;31:52–8.
[4] Puledda F, Schankin C, Goadsby PJ. Visual snow syndrome: a clinical and phenotypical description of 1100 cases. Neurol 2020;94(6):e1–11.
[5] Fraser CL, White OB. There's something in the air. Sur Ophthal 2018;63:1–5.
[6] Ciuffreda KJ, Rutner D. Visual snow syndrome in traumatic brain injury: effect on driving. NewsBrake 2023;47:26–8.
[7] Kondziella D, Olsen MH, Dreier JP. Prevalence of visual snow syndrome in the UK. Eur J Neurol 2020;27:764–72.
[8] Carroll F. Visual symptoms caused by digitalis. Trans Am Ophthalmol Soc 1944;42:664–8.
[9] Ciuffreda KJ, Tannen B, Han MHE. Visual snow syndrome: an evolving neuro-optometric perspective. Vis Dev Rehabil 2019;5:75–82.
[10] Tannen, Brown J, Ciuffreda KJ, et al. Remediation of visual snow (VS) and related phenomena in a neuro-optometric practice: a retrospective analysis. Vis Dev Rehabil 2022;8:105–13.
[11] White OB, Fielding J, Pelak VS, et al. Editorial: Visual snow: old problem, new understanding. Front Neurol 2022;13:884752.
[12] Tsang T, Shidlofsky C, Mora V. The efficacy of neuro-optometric visual rehabilitation therapy in patients with visual snow syndrome. Front Neurol 2022;13:999336.
[13] Ciuffreda KJ, Han MHE, Tannen B. Pediatric visual snow syndrome (VSS): a case series. Vis Dev Rehabil 2019;5:249–53.
[14] Liu C, Han MHE, Ciuffreda KJ. Primary chromatic filter treatment in a concussion patient: traditional and contemporary approaches. Vis Dev Rehabil 2020;6:26–31.
[15] Ciuffreda KJ, Tannen B. Future directions in neuro-optometry. Concussion 2020;5(4):CNC80.
[16] Ciuffreda KJ, Han HME, Tannen B, et al. Visual snow syndrome: evolving neuro-optometric considerations in concussion/mild traumatic brain injury. Concussion 2021;CNC89.
[17] Ciuffreda KJ, Tannen B, Rutner D, et al. Neuro-optometric treatment for visual snow syndrome: recent advances. Concussion 2023;CNC110.
[18] Rutner D, Ciuffreda KJ. A clinical diagnostic and treatment protocol for the patient with visual snow/visual snow syndrome and concurrent binocular dysfunctions. Vis Dev Rehabil 2023;9:7–14.
[19] Han MHE, Ciuffreda KJ, Rutner D. Historical, diagnostic, and chromatic treatment in visual snow syndrome: a retrospective analysis. Optom Vis Sci 2023;100:328–33.
[20] Eren O, Schankin CJ. Insights into pathophysiology and treatment of visual snow syndrome: A systematic review. Prog Brain Res 2020;255:311–26.
[21] Aldusary N, Traber GL, Freund P, et al. Abnormal Connectivity and Brain Structure in Patients with Visual Snow. Front Hum Neurosci 2020;14:582031.
[22] Puledda F, O'Daly O, Schankin C, et al. Disrupted connectivity within visual, attentional and salience networks in the visual snow syndrome. Hum Brain Mapp 2021;42:2032–44.
[23] Puledda F, Ffytche D, Lythgoe DJ, et al. Insular and occipital changes in visual snow syndrome: A BOLD fMRI and MRS study. Ann. Clin. Transl. Neurol 2020;7:296–306.
[24] Puledda F, Bruchhage M, O'Daly O, et al. Occipital cortex and cerebellum gray matter changes in visual snow syndrome. Neurology 2020;95:e1792–9.
[25] Michels L, Stämpfli P, Aldusary N, et al. Widespread White Matter Alterations in Patients with Visual Snow Syndrome. Front Neurol 2021;12:723805.
[26] Hepschke JL, Martin PR, Fraser CL. Short-Wave Sensitive (''Blue'') Cone Activation Is an Aggravating Factor for Visual Snow Symptoms. Front Neurol 2021;12:697923.
[27] Foletta PJ, Clough M, McKendrick AM, et al. Delayed Onset of Inhibition of Return in Visual Snow Syndrome. Front Neurol 2021;12:738599.

[28] Rusztyn P, Stańska W, Torbus A, et al. Visual Snow: a review on pathophysiology and treatment. J Clin Med 2023;12:3868.
[29] Yoo Y, Yang HK, Choi JY, et al. Neuro-ophthalmological findings in visual snow syndrome. J Clin Neurol 2020;16:646–52.
[30] Werner RN, Gustafson JA. Case report: visual snow syndrome after repetitive mild traumatic brain injury. Optom Vis Sci 2022;99:413–6.
[31] Hang C, Leishangthem L, Yan Y. Not all cases of visual snow are benign: mimics of visual snow syndrome. Neuropsychiatric Dis Treat 2021;17:3293–300.
[32] Chen BS, Lance S, Laullu B, et al. Visual snow: not so benign. J Clin Neurosci 2019;l64: 37–9.
[33] Laukkanen H, Scheiman M, Hayes JR. Brain injury vision symptoms survey (BIVSS) questionnaire. Optom Vis Sci 2017;94:43–50.
[34] Verriotto JD, Gonzalez A, Aquilar ME, et al. New methods for quantification of visual photosensitivity threshold and symptoms. Trans Vis Sci Tech 2017;6:18.
[35] van Dongen RM, Alderliefste GJ, Onderwater GLJ, et al. Migraine prevalence in visual snow with prior drug use (hallucinogen persisting perception disorder) versus without. Eur J Neurol 2021;28:2631–8.
[36] Eren OE, Straube A, Schoberl F, et al. Age and frequency dependent change in dynamic contrast perception in visual snow syndrome. Headache Pain 2021;22:148.
[37] Vaphiades MS, Grondines B, Cooper K, et al. Diagnostic evaluation of visual snow. Front Neurol 2021;12:743608.
[38] Eren O, Rauschel V, Ruscheweyh R, et al. Evidence of dysfunction in the visual association cortex in visual snow syndrome. Ann Neurol 2018;84:946–9.
[39] Puledda F, Vandenbussche N, Moreno-Ajona D, et al. Evaluation of treatment response and symptom progression in 400 patients with visual snow syndrome. Br J Ophthalmol 2021;106:1318–24.
[40] Van Dongen RM, Waaijer LC, Onderwater GL, et al. Treatment effects and comorbid diseases in 58 patients with visual snow. Neurology 2019;93:e398–403.
[41] De Simone R, Marano E, Di Stasio E, et al. Acetazolamide efficacy and tolerability in migraine with aura: a pilot study. Headache 2005;45:385–6.
[42] Willeford KT, Fimreite V, Ciuffreda KJ. The effect of spectral filters on VEP and alpha-wave responses. J Optom 2016;9:110–7.
[43] Fimreite V, Willeford KT, Ciuffreda KJ. Effect of chromatic filters on visual performance in individuals with mild traumatic brain injury (mTBI): a pilot study. J Optom 2016;9:231–9.
[44] Engel SA, Wilkins AJ, Mand S, et al. Habitual wearers of colored lenses adapt more rapidly to the color changes the lenses produce. Vision Res 2016;125:41–8.
[45] Scheiman M, Wick B. Clinical management of binocular vision. 4th edition. Philadelphia: Lippincott Williams and Wilkins; 2014.
[46] Press LJ. Applied concepts in vision therapy. New York: Elsevier; 1996.
[47] Chang TT, Ciuffreda KJ, Kapoor N. Critical flicker frequency and related symptoms in mild traumatic brain injury. Brain Inj 2007;21:1055–62.
[48] Schrupp LE, Ciuffreda KJ, Kapoor N. Foveal versus eccentric retinal critical flicker frequency in mild traumatic brain injury. Optom 2009;80:642–50.
[49] Thompson AC, Goodbourn PT, Forte JD. Perceived severity of visual snow syndrome is associated with visual allodynia. Headache 2023;63:494–505.
[50] Gottlieb RL, Wallace LB. Syntonic phototherapy. Photomed Laser Surg 2010;28:449–52.
[51] Hoggan RN, Subhash A, Blair S, et al. Thin-film optical notch filter spectacle coatings for the treatment of migraine and photophobia. J Clin Neurosci 2016;28:71–6.
[52] Noseda R, Bernstein CA, Rony-Reuven Nir, et al. Migraine photophobia originating in cone-driven retinal pathways. Brain 2016;139:1971–86.
[53] Gould JA, Ciuffreda KJ, Yadav NK, et al. The effect of retinal defocus on simple eye hand and eye-foot reaction time in traumatic brain injury (TBI). Brain Inj 2013;27:1643–8.

[54] Gould JA, Ciuffreda KJ, Arthur B, et al. Retinal defocus and eye dominance effect on eye had reaction time. Optom Vis Perf 2013;1:129–36.
[55] Ciuffreda KJ. Simple eye-hand reaction time in the retinal periphery can be reduced with training. Eye Contact Lens 2011;37:145–6.
[56] Ciuffreda KJ, Levi DM, Selenow A. Amblyopia. Boston: Butterworth; 1991.
[57] Hepschke JL, Seymour RA, He W, et al. Cortical oscillatory dysrhythmias in visual snow syndrome: a magnetoencephalography study. Brain Commum 2022;18(4):fcab296.
[58] Strik M, Clough M, Solly EJ, et al. Micro-structure in patients with visual snow syndrome: an ultra-high field morphological and quantitative MRI study. Brain Commum 2022;23(4):fcac 164.