PERSPECTIVE

Visual Snow Syndrome (VSS): An Evolving Neuro-Optometric Clinical Perspective

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ABSTRACT

Visual snow syndrome (VSS) is a relatively new diagnosis in optometry and in medicine. VSS represents a constellation of visual and non-visual problems, with the hallmark symptom being the perception of visual snow (VS) appearing in a single plane in front of and throughout the visual field. Individuals with VS describe seeing "snow", dots, pixelated fuzz, bubbles, and "static" as found on a poorly-tuned television. In this paper, the evolving area of VSS is briefly reviewed, a VSS symptom survey is proposed, several cases and related information are presented, and current neuro-optometric therapeutic interventions are discussed.

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Figure 1a: Patient depictions of VS throughout the entire visual field. Case One looking at her cat.

INTRODUCTION

One of the most interesting and intriguing, as well as bothersome, visual abnormalities is the presence of "visual snow" (VS), more specifically referred to as the "visual snow syndrome" (VSS).[1,2] These individuals perceive a somewhat pixelated visual world, sometimes fuzzy, with either black-and-white or chromatic dots appearing in a single plane in the foreground superimposed over the entire visual scene (Figure 1a). This is typically reported to be a constant, persistent phenomenon that has been present for years. They frequently describe it as being akin to the "snow" that is present when the primary television signal is reduced, and the residual electronic "noise" predominates on the screen.

Individuals with VSS typically manifest two or more of the following four visual perceptual phenomena: (1) palinopsia; (2) photosensitivity/photophobia; (3) "nyctalopia"; and (4) enhanced entoptic imagery.[1,3] They may also report having other visual and non-visual symptoms.[2] These will be described in more detail in the next section.

As interest and knowledge in this relatively new area (1995)[4] increases and evolves, the topic of its treatment becomes important. Currently, there is a paucity of such information. Treatment in the past has included:[2] (1) chromatic filters; (2) pharmacologic interventions; and (3) simple explanation of the condition to the patient. These too will be considered in more detail later.

In the present paper, several cases of VSS that the authors have examined will be discussed, as well as related information. The emphasis will be placed on the development of a comprehensive VSS patient diagnostic symptom survey, as well as discussion of the neuro-optometric treatment options that were successfully prescribed.

VSS Symptom Survey

We have developed a VSS diagnostic symptom survey, including demographics and VS history, primary VSS-related visual symptoms, and secondary VSS-related visual and non-visual symptoms (Table 1). These questions were based on a summary of several reports in the

Table 1. Visual Snow Syndrome (VSS) Symptom Survey

VISUAL SNOW SYNDROME (VSS) SYMPTOM SURVEY

Patient's Name:_____ Date:_____

DEMOGRAPHICS	
CURRENT AGE (IN YEARS)	
GENDER	
AGE FIRST NOTICED SNOW (IN YEARS)	
IS VISUAL SNOW CONSTANT OR TRANSIENT?	
IS VISUAL SNOW CHROMATIC OR MONOCHROMATIC?	
PROVOKING ENVIRONMENTS?	
POSSIBLE ETIOLOGY?	
POSSIBLE CO-MORBID CONDITIONS?	
TREATMENT(S)?	
MEDICATIONS?	

Please rate each symptom below as: 0=Symptom is not present 1=Symptom is transient 2=Symptom is constant

PRIMARY VISUAL SYMPTOMS	**0**	**1**	**2**	**COMMENTS**
PALINOPSIA				
ENTOPTIC IMAGERY				
PHOTOSENSITIVITY				
"NYCTALOPIA" (IMPAIRED NIGHT VISION)				

SECONDARY VISUAL/NON-VISUAL SYMPTOMS				
PHOTOPSIA				
MIGRAINE				
PHONOPHOBIA				
HYPERACUSIS				
CUTANEOUS ALLODYNIA				
TINNITUS				
BALANCE PROBLEMS				
TREMOR				

literature over the past two decades involving on the clinical testing and formal queries of several hundred individuals with VSS.[1-11]

The first section queries the basic demographics, VS history, and possible related factors. There are also other areas that the clinician may elect to probe based on historical aspects of the field that might yield additional insights:[1-11] (1) Did headaches precede the perception of VS?; (2) Is the VS stable or progressive?; (3) Was the perception of VS of sudden onset?; (4) Are there any family members with VS?; and (5) Has the individual ever taken hallucinogenic drugs? Early literature suggested a link.

The second section queries the presence of the four primary VSS visual symptoms, their provoking environments, and treatment aspects. Based upon the latest clinical thinking,[3] typically the patient must also report at least two of the following four visual symptoms/visual perceptual phenomena to be diagnosed with VSS: (1) *palinopsia:* persistence of an image, at times superimposed on the new image, and frequently with trailing; (2) *enhanced entoptic imagery:* imagery persistence above what many others would normally perceive; (3) *photosensitivity/photophobia:* visual discomfort, perhaps even actual pain, when exposed to light stimuli that are not bothersome to others; and (4) *"nyctalopia":* difficulty seeing well at night, likely due to VS interference/overlay rather than to an actual retinal problem or retinal disease. In addition, determining provoking environments may help the patient emphasize and reinforce such "avoidance". Lastly, treatment is a rapidly evolving and exciting area, especially for the neuro-optometrist. Treatment has fallen into three categories: (1) filters to reduce the perception of the VS intensity (e.g., transmission peaking in the yellow-blue spectrum);[9] (2) drugs to reduce the VS intensity (e.g., lamotrigine);[7] and (3) simply discussing the condition with the patient to explain that VS is a real phenomenon which some individuals experience, as well as to avoid provoking environments, and secondly to assure the patient that it is a relatively benign and not progressive condition, assuming that any possible abnormal neurological or ocular condition has been ruled out.[2] In general, its presence appears not to have any adverse consequences on their activities of daily living (ADLs), although some have reported difficulty driving at night due to VS interference. However, this aspect has not been well documented.

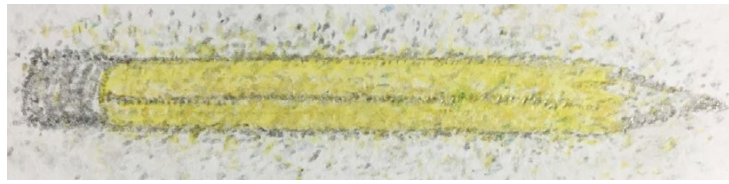

Figure 1b: Patient depictions of VS throughout the entire visual field. Another VSS patient looking at a pencil.

The third section presents a list of several possible secondary visual and non-visual symptoms that the patient with VSS may also experience. These include: (1) *photopsia:* perception of flashes of light, usually very brief and intermittent; (2) *phonophobia:* unwarranted fear of/aversion to specific sounds (e.g., traffic noises, voices); (3) *hyperacusis:* increased sensitivity to certain sounds, at times specific frequencies; (4) *cutaneous allodynia:* pain sensation to the skin/scalp from innocuous stimuli (e.g., hair brush on the scalp, spectacles on the nose/ears), (5) *tinnitus:* perception of noise (e.g., hissing) or "ringing" in the ears; (6) *balance problems:* difficulty maintaining body position/stability; and (7) *tremor:* involuntary, unintentional, rhythmic movements of the body (e.g., hands). In addition, migraine is included as a possible comorbid but not a causative condition that may exacerbate the perception of VS.[8] Since some believe that VSS represents a general neurological "hypersensitivity" phenomenon,[7,12,13] then several of the above conditions would be logical to be present (e.g., hyperacusis, cutaneous allodynia, tinnitus). The last two symptoms of balance problems and tremor above should be carefully probed, especially if there is no past history of head trauma or neurological disease.

Table 2. Patient Demographics

Age tested: 11-35 years
Age VS first noticed: 11-29 years
Gender 7F/3M
VS Constant (C) or Transient (T): 7C/3T
Etiologies of VS • Concussion • Auto-immune disease • Brain cyst surgery • Antibiotic usage • Finesteride usage • Idiopathic

Table 3. Categories of VSS

• Constant idiopathic (n=1)
• Transient idiopathic (n=3)
• Constant non-idiopathic (n=6)

Table 4. Frequency of Primary VSS Visual Symptoms in Our Ten Patients

• Enhanced entoptic imagery	9
• Palinopsia	8
• Photosensitivity/Photophobia	7
• "Nyctalopia"	3

Table 5. Frequency of Secondary VSS Visual and Non-Visual Symptoms in Our Ten Patients

• Migraine	7
• Tinnitis	4
• Photopsia	3
• Phonophobia	1
• Tremor	1
• Balance problems	1
• Cutaneous allodynia	0
• Hyperacusis	0

Table 6. Neuro-Optometric Treatments

• Chromatic/achromatic filters for VS
• Saccadic tracking for palinopsia

Cases

The following brief narrative case summaries are representative of the ten patients with a diagnosis of VSS examined by us over the past six months. The group has been divided into three categories based on the VS and related diagnostic characteristics and case histories. In addition, the group data have been summarized in Tables 2-6.

Case One: Transient Idiopathic VSS

This was a 31-year-old, female professional student. Her comprehensive optometric examination revealed an accommodative insufficiency and versional oculomotor dysfunction, for which she was prescribed conventional oculomotor-based vision therapy. At a later session, she reported having four episodes of monochromatic VS over the past two years (Figure 1a). These occurred at night when fatigued, and each episode lasted for about one minute. She has also experienced one or two migraine headaches each year for the past five years. Other VSS-related primary visual symptoms included palinopsia, enhanced entoptic imagery, and photosensitivity. Other VSS-related visual and non-visual symptoms included photopsia, hyperacusis, and tinnitus. Due to the infrequency and short duration of the VS perception and related symptoms, she did not require prescription of tinted lenses or other testing.

Case Two: Transient/Constant Concussion-Based VSS

This was a 16-year-old male who fainted due to dehydration, hit his head, and sustained a concussion in August 2018, with an undetermined period of loss of consciousness. When his friends found him, he was confused, had head and neck pain, and nausea, all of which persisted. Furthermore, he reported that "everything looked white" for a few minutes after resuming consciousness. Later, he reported blurred vision, intermittent diplopia, and oculomotor-based reading difficulties (e.g., skipping lines of print). He started to wear non-prescription blue-blocker lenses during computer use for additional visual comfort. He and his family have a history of migraine headaches. His physician prescribed amantadine for the head and neck pain, which helped. He was then referred for both physical

and vestibular therapy, which did not reduce any of his other symptoms. He was later referred for a neuro-optometric examination for his residual visual symptoms. Interestingly, four years earlier, he was diagnosed and successfully treated for "amplified musculosketal pain syndrome (AMPS)", which involves neurological "amplification", or hypersensitivity, to pain.[14]

The neuro-optometric examination in December 2018 yielded several important findings. He now reported the perception of monochromatic VS. The initial episode occurred three months *before* the concussion and was transient in nature. Then, four weeks *after* the concussion, the monochromatic VS reappeared and was now constant. His primary VSS-related visual symptoms were enhanced entoptic imagery, photosensitivity, and "nyctalopia". His secondary VSS-related visual and non-visual symptoms included hyperacusis, tinnitus, and balance problems, in addition to the migraines. He was prescribed bluish-green blocking BPI-Omega lenses for reading, which reduced the VS intensity, and he continued use of the blue-blocker lenses for computer viewing. His diagnosed oculomotor-based problems included convergence insufficiency, accommodative insufficiency, and saccadic deficiency, all of which were remediated using conventional oculomotor-based, optometric vision therapy. His symptom of visual motion sensitivity (VMS), reported later, was reduced by visual motion habituation training.[15]

Case Three: Constant Drug-Induced VSS

This was a 30-year-old, female professional violinist. Her comprehensive optometric examination in 2018 was unremarkable, except for the symptom of VS. She gave birth to a healthy baby in July 2017. However, she soon developed bilateral mastitis and breast abscesses for which she was prescribed the antibiotic dicloxacillin for ten days. Soon thereafter, she reported constant VS of either a monochromatic or chromatic (rainbow effect) nature, the latter especially in dimly-lit environments. Her primary VSS-related visual symptoms included palinopsia, enhanced entoptic imagery, and photosensitivity. Her other VSS-related non-visual symptom included balance problems. She was tested to determine which tints, if any, reduced her visual symptoms, especially the VS. A range of tint densities (%) were trialed, and all produced a positive effect to varying extents: 10-60% brown, 10-30% rose, 10-30% gray, and 70% BPI-Omega. In addition, she was examined with the Cerium Intuitive Colorimeter[9,16] and preferred the following tint: (hue 285, saturation 35, blue 5+4, purple 5+3, plastic CR-39). She was prescribed a 20% gray tint for general use until the Cerium tint is fabricated.

DISCUSSION

The literature on VSS indicates that the VS per se is *constantly* present, once it initially becomes manifest, frequently at an early age (e.g., ages 11-14 years).[1-11] Our small sample of individuals with VS (and VSS), which was obtained by "convenience sampling", suggests otherwise with regards to constancy. Three of the ten (30%) reported their VS to be transient, and infrequent. What might account for this discrepancy? We speculate that VS/VSS may be more prevalent than currently believed. Some who noted VS at an early age either thought it was "normal" or were fearful that would be labelled as "crazy" or "weird". Thus, while VS/VSS indeed may not be common, it may not be that rare, per our case scenarios. Furthermore, in discussion with one experienced neuro-optometrist, he reported seeing 30-50 such patients per year. Thus, the neuro-optometrist and others in the field are encouraged to query in that direction at the slightest suspicion of VS/VSS to obtain the proper diagnosis. To this end, we have proposed a neuro-optometric, diagnostic, clinical test protocol for the patient with VSS based on the literature findings[1-11] (Table 7).

Of particular interest to the neuro-optometrist and others (e.g., vision therapist,

Table 7. Proposed Neuro-Optometric Diagnostic Test Protocol for VSS

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 neuro-sensory 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 visuo-spatial 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 visuo-motor 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 presence of VS and related VSS phenomena

ADVANCED TESTS
• Electro-retinography (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

occupational therapist) is the use of neuro-optometric rehabilitation (NOR) to reduce symptomatology in patients with VSS. So far, there have been two primary optometric routes. The use of chromatic and achromatic filters show promise. In our limited experience, this has included the BPI-Omega, FL-41, and custom color filtration using the Intuitive Colorimeter, as well as achromatic gray tints. Thus far, most of the chromatic tints have included a blue-blocking component (e.g., yellow). Their effectiveness may be due to a specific chromatic-based neurological mechanism, which remains unknown, and/or in part simply to luminance reduction which decreases the perceived intensity of the VS.[16] We believe that both are involved. These arguments may also relate to the perception of the other visual symptoms. Our positive findings here are consistent with an earlier medically-based research study.[9] They tested twelve patients with VS/VSS using the Intuitive Colorimeter. Eleven of the twelve (92%) noted VS symptom reduction with some specific filter combination, with most (83%) selecting a filter maximally transmitting light in the yellow-blue color spectrum. Testing in the group was repeated yielding the same results. None were prescribed in this pilot research investigation. Further testing of VSS patients regarding this promising therapeutic intervention will be required to obtain an optimal and long-lasting, tint-based effect.

Related to the above is the condition of palinopsia found in most with VSS. Again, in our limited experience, we have used a range of simple, conventional saccadic tracking protocols (e.g., predictable saccadic tracking), which resulted in reduced palinopsia. We speculate that the underlying mechanism may be related to the well-known and normal visual phenomenon of saccadic suppression.[17] This cortical suppression (i.e., neural inhibition) occurs to prevent the perception of a "smeared" retinal image during the actual saccade itself. Such normal inhibition appears to be lacking

in the patient with palinopsia, thus resulting in neural/perceptual "hypersensitivity" and a sustained afterimage especially with trailing. Thus, the saccadic training appears to "reset" this abnormal "disinhibition" to a more normal inhibitory level. Again, more clinical and laboratory testing is required to optimize this positive oculomotor training effect.

Current thought is that VS and the related abnormal perceptual phenomena may be due to neurological "hypersensitivity"[7,12,13] as mentioned above. This notion is consistent with their primary visual symptoms (e.g., visual snow, palinopsia, enhanced entoptic imagery). It is also consistent with their secondary visual (i.e., photopsia) and non-visual (e.g., tinnitis, hyperacusis, cutaneous allodynia) symptoms. That is, those with VSS appear to experience a global "hypersensitivity" effect, presumably reflecting a generalized, abnormal neurological "disinhibition" mechanism across sensory modalities. Thus, they sense and perceive what others do not. Recent research findings in human brain physiology[13] and psychophysics[12] support this general idea, a *likely unifying hypothesis.*

The underlying neural mechanisms and related anatomical substrates for VS remain incomplete, elusive, and highly speculative, and furthermore have been confounded at times by the presence and effects of comorbid migraine. However, there have been two primary hypotheses. The first proposed that patients with VS had increased, intrinsic visual noise in the brain, which resulted in the perception of VS overlapping the visual scene.[18] Using a psychophysical approach in the laboratory, however, they found that both visually-normal individuals (n=16) and those with VS (n=5) had equivalent amounts of intrinsic visual noise. They then speculated that the VS may be due to be increased perceptual gain, which now seems unlikely, but this was never tested. The second is based on the concept of "hypersensitivity", as discussed earlier.[7,12,13] Using positron emission tomography (PET) brain imaging, hypermetabolism was found in two areas:[8] the right lingual gyrus and the left anterior cerebellum. Interestingly, the former area is associated with visual memory, facial recognition, attention, and color perception, thus consistent with some of the general perceptual aspects of VSS. And, the left anterior cerebellum is involved in coordination, accuracy, and timing of movement, but not their initiation per se, thus being consistent with the motor deficits (e.g, balance problems, tremor) found in some with VS/VSS. More recent VEP data suggested involvement of the visual association cortex.[19] Lastly, and related to the above, is objective VEP and transcranial magnetic stimulation data demonstrating both a lowered cortical excitation threshold and lack of habituation in those with VS to visual stimuli.[20] Taken together, the above experiments suggest that the patient with VS has a two-fold neurological problem: an inability to suppress and also to habituate to visual stimuli in a normal manner. Both the perceptual and motor aspects of the above remain speculative and warrant further careful laboratory testing.

Lastly, there are several possible future directions that should prove to be fruitful. First, the prevalence of VSS must be ascertained. This is currently unknown and likely underestimated. Clinicians and others in the field need to probe the area using the VSS symptom survey we have developed, and other means, to provide detailed information regarding the depth and breadth of the problem. Second, as VSS becomes more widely recognized, further ideas related to its possible mechanism will evolve, and thus improve our understanding of this unusual condition. Third, a randomized clinical trial (RCT) should be conducted to determine the optimal filter-based therapeutic intervention to reduce the VS and VSS symptoms.

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