Curr Pain Headache Rep (2015) 19: 23
DOI 10.1007/s11916-015-0497-9



# Visual Snow—Persistent Positive Visual Phenomenon Distinct from Migraine Aura

**Christoph J. Schankin**[1] · **Peter J. Goadsby**[2]

Published online: 29 May 2015


**Abstract** Patients with visual snow complain of uncountable flickering tiny dots in the entire visual field similar to the view of a badly tuned analogue TV channel (TV snow). The symptoms are often continuous and can persist over years. This condition is grouped among the persistent visual phenomena in migraine, although it clinically presents a unique entity distinct from persistent migraine aura or migraine aura status. Here, we review the recent literature leading to the identification of the visual snow syndrome. The additional visual and non-visual symptoms are described in detail, and criteria are presented for future studies. Using these criteria, the relationship to migraine and typical migraine aura was recently evaluated. Further, patients with visual snow differ from controls in respect of hypermetabolism in the supplementary visual cortex (lingual gyrus). This provides evidence that visual snow, despite being purely subjective in the individual patient, has a clear biological basis. The area of hypermetabolism overlaps with the functional correlates of photophobia in migraine supporting the close relationship of migraine and visual snow.

This article is part of the Topical Collection on *Uncommon and/or Unusual Headaches and Syndromes*

✉ Christoph J. Schankin
christoph.schankin@med.uni-muenchen.de

[1] Department of Neurology, University of Munich Hospital—Großhadern, Marchioninistr. 15, 81377 Munich, Germany

[2] Headache Group, Basic and Clinical Neuroscience, NIHR-Wellcome Trust Clinical Research Facility, King's College London, London, UK

**Keywords** Visual snow · Migraine · Aura · FDG PET · Persistent visual phenomena · Headache

**Abbreviations**

| | |
|---|---|
| VS | Visual snow |
| [$^{18}$F]-FDG | [$^{18}$F]-2-fluoro-2-deoxy-D-glucose |
| PET | Positron emission tomography |

## Introduction

In 2013, Simpson et al. [1•] presented an illustrative case of a 12-year-old girl with visual snow (VS). Having a history of migraine since age seven, she woke up one day at age ten with pure visual symptoms. One symptom resembled the noise of an analogue TV when the channel was not tuned in properly and consisted of millions of tiny flickering dots in the entire visual field. Further, she complained of after-images, photophobia, squiggles, and lines everywhere as well as small moving dots at the sky. Her symptoms were continuous, did not respond to migraine or seizure medication, and had an enormous impact on quality of life preventing her from doing what she wanted or used to do. Two years later, she was still complaining of these visual disturbances. For her complaints, a descriptive diagnosis of 'positive persistent visual symptoms' (or 'persistent positive visual phenomena') in a migraineur could be applied. Such a non-specific term confounds the impressive clinical picture of the TV noise or TV snow-like visual disturbance, i.e. visual snow (VS), with persistent (typical) migraine aura, and other visual disturbances in migraineurs. This commingling does not reflect the phenotype of VS, its high impact on quality of life, and the poor response to various treatment approaches.

In the following, we will delineate the significance of VS among the persistent visual phenomena in migraine. In particular, we will review the recent literature on its phenotype, time course, the relation to migraine, and typical migraine aura as well as objective findings taking it out of the corner of psychogenic or simulated symptoms.

## Persistent Positive Visual Phenomena in Migraine

'Persistent positive visual phenomena' is a pure descriptive term for visual symptoms that are not darkness, which would be instead 'negative', and which are unremitting. In migraineurs, such symptoms have been described occasionally in the past, and the current headache classification of the International Headache Society acknowledges persistent visual symptoms in patients with migraine only for the aura category under the terms 'persistent aura without infarction' (ICHD-3 code 1.4.2) and 'migraine aura status' (in the appendix A1.4.5). Basically, patients with previous migraine aura have a similar aura episode that persists for longer than 1 week to call it 'persistent aura without infarction' or have at least two aura episodes per day for at least 3 days to call it 'migraine aura status' [2]. There have been several case reports or series presenting patients with such symptoms in detail. Visual snow (VS), described as a visual disturbance resembling the noise of a badly tuned analogue television (TV snow), has been mixed with usual persistent aura in some reports.

In an excellent early work, Liu et al. [3] presented ten patients with visual disturbances and tried to group them into three categories depending on the likelihood of being caused by migraine. The first group ('definitely related to migraine') had visual aura with headache prior to the occurrence of the visual disturbance, the second ('probably related to migraine') had headache during, but not prior to the visual disturbance suggesting some association, whereas the third group ('possibly a migraine equivalent') had migraine history, but no association of headache and visual disturbance. All three subjects with VS were put in the third group since the beginning was not associated with migraine, but there was a clear migraine history suggesting some relation. In an attempt to predict the outcome of persistent visual aura without infarction, Wang et al. [4] assessed six of their own patients and 23 patients from the literature using the visual aura rating scale [5] giving points to the presence of a duration of 5–60 min, gradual development over more than 5 min, presence of scotoma, zigzag lines, and unilaterality (homonymous presence in both eyes). They found that the prognosis was better in subjects with a higher score, i.e. closer similarity to typical migraine aura. The six subjects with visual snow, who were included in the study, had a very low score: continuous visual symptoms in the entire visual field without scotoma, zigzag lines, or unilaterality and were accordingly in the group with a worse outcome in contrast to other forms of 'persistent visual aura without infarction'. Chen et al. [6] have studied six patients with 'persistent visual aura without infarction'—two of whom had VS—using magnetoencephalography and found increased visual cortex excitability being inversely correlated to the duration of visual symptoms. The VS patients had very long durations of 5 and 10 years suggesting an electrophysiological difference between VS and the other forms of 'persistent visual aura without infarction'.

These studies suggest VS behaves differently in outcome [4] and electrophysiological behaviour [6] than 'persistent visual aura without infarction'.

## The Defining Symptom—Visual Snow (VS)

A distinction from persistent visual migraine aura is supported from the phenotype in clinical practice. Visual snow was named by patients due to its resemblance to TV snow (Fig. 1a). It thus is a phenomenon, which is present in both eyes in the entire visual field and consists of uncountable tiny dots, which are flickering constantly in front of the background. In contrast, typical migraine aura is very distinct. Lashley [7] has mapped his own aura, and Hansen et al. [8•] has presented a patient who documented his visual auras over many years. Each showed directed movement over the visual field and were in general unilateral (homonymous) supporting the likely underlying pathophysiological mechanism of cortical spreading depression (CSD) in the visual cortex that has been postulated since Leão [9], and that was shown in human using functional brain imaging [10].

The unique clinical presentation of VS, its long duration, and high impact on quality of life of those affected advocates for studying this condition separately. Major unresolved issues include: is VS indeed a clinical condition distinct from migraine aura? Is it a sole symptom or part of a syndrome? What is its relation to migraine and typical migraine aura? Are there objective findings of this otherwise subjective syndrome?

## Clinical Characterization of Patients with Visual Snow

Based on our own clinical experience and with the support of a self-help group, we had the opportunity to identify possible additional symptoms in patients with self-assessed VS [11••]. Using the arbitrary cut-off frequency of one third appearance in the cohort, eight additional symptoms (see below) were identified and added to the main criterion VS to generate preliminary criteria for a 'visual snow syndrome'. One hundred and forty two subjects with a TV snow-like visual disturbance (i.e. VS) were prospectively recruited over the internet. Seventy-eight had confirmed VS and normal

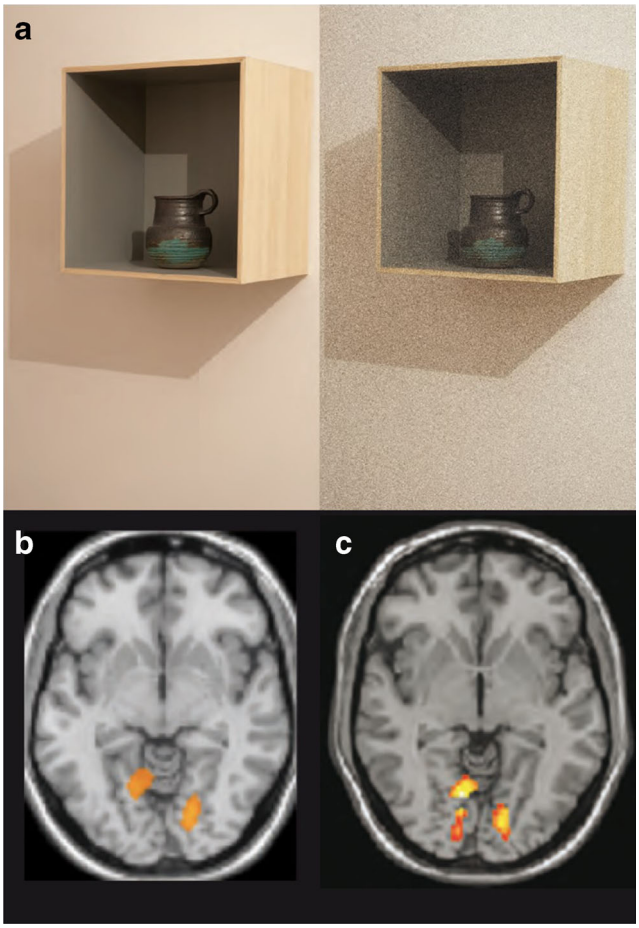


**Fig. 1** **a** Illustration of visual snow (*right*) done by a patient in contrast to the original image on the left. A continuous flickering of the dots has to be envisioned and clearly shows the similarity to TV snow that can persist over decades. **b** Using [$^{18}$F]-FDG positron emission tomography, brain hypermetabolism in the lingual gyrus was demonstrated in visual snow patients confirming that patients do not make up their symptoms (figure taken from [16••]). **c** Comparison with $H_2{}^{15}O$ positron emission tomography after light stimulation during migraine attacks [21••] was used as a model for photophobia in migraineurs. This revealed the same area in the lingual gyrus being hyperperfused suggesting some relevance of this area for visual snow pathophysiology and for the clinical overlap of migraine/aura with visual snow

ophthalmological exams, and of those, 72 or 92 % had at least three of the additional symptoms strongly supporting that VS not only is phenotypically different from typical migraine aura as explained above but also represents a clinical syndrome. The additional visual symptoms found in this study were very similar to the complaints of the young girl presented earlier [1•] and could be grouped into the following categories: palinopsia (including after-images from stationary and from moving objects, i.e. trailing), enhanced entoptic phenomena (floaters, blue field entoptic phenomenon, self-light of the eye, and photopsia), photophobia, and impaired night vision (nyctalopia). These findings were used to generate criteria for the visual snow syndrome (Table 1). The most frequent non-visual symptom was tinnitus in 62 % of patients.

## The Additional Visual and Non-visual Symptoms

As shown, VS is typically associated with a variety of additional symptoms resulting in a clinical syndrome [11••]. Analysing this syndrome in detail might give insights as to its biology. Palinopsia, i.e. the persistence of a visual image after the removal of the exciting stimulus [12], can manifest as 'trailing' or persistence of stationary scenes. Hypersensitivity to light, i.e. photophobia, means that patients perceive light photons as too bright when non-VS subjects would rate them as 'normal'. Entoptic phenomena are defined as 'phenomena arising from…structure[s] of the visual system as a result of specific stimulation' [13]. Typical phenomena are for example floaters, i.e. protein aggregations in the vitreous corpus or the blue field entoptic phenomenon, i.e. the white blood cells in retinal blood vessels [14, 15] that cast shadows on the photoreceptors. The physical stimuli behind these phenomena are identical for everybody, i.e. for VS patients and for healthy individuals. It is however striking that these mechanisms result in profound visual perceptions in patients with VS. The question arising from the high prevalence of these symptoms in patients with VS is probably, why these phenomena are not present in everybody, and the hypothetical consequence would be that a mechanism suppressing these phenomena in healthy individuals might be dysfunctional in patients with VS. In other words, VS itself and the additional visual symptoms might be the consequence of a suppression deficit in normal regulatory pathways for visual input. Understanding the VS syndrome will be crucial for advancing its management and will contribute to broader understanding of visual

**Table 1** Proposed criteria for the visual snow syndrome, modified from Schankin et al. [11••]. Patients compare visual snow often with TV static or TV snow

A. Visual snow: dynamic, continuous, tiny dots in the entire visual field lasting longer than 3 months.

B. Presence of at least two additional visual symptoms of the four following categories:
   i. Palinopsia. At least one of the following: after-images (different from retinal after-images) or trailing of moving objects.
   ii. Enhanced entoptic phenomena.[a] At least one of the following:
   excessive floaters in both eyes,
   excessive blue field entoptic phenomenon, self-light of the eye, or spontaneous photopsia.
   iii. Photophobia.
   iv. Nyctalopia (impaired night vision).

C. Symptoms are not consistent with typical migraine visual aura [2].

D. Symptoms are not better explained by another disorder (especially normal eye exams).

[a] Entoptic phenomena arise from the structures of the visual system. They include blue field entoptic phenomenon (uncountable little grey/white/black dots or rings shooting over visual field in both eyes when looking at homogeneous bright surfaces, such as the blue sky), floaters, spontaneous flashes of light (photopsia), or self-light of the eye (coloured waves or clouds when closing the eyes in the dark)

modulation. Interestingly, about two thirds of patients with VS also had tinnitus [11••], which could be described as 'acoustic noise' in this context. This shows that other sensory systems might also be involved in patients with VS supporting that VS is a disorder of the brain with a suppression deficit of the visual and, in some patients, the auditory system.

## The Time Course of Visual Snow

During the interviews, 64 patients (84 %) either recalled a period in life without VS ($n$=59, 76 %) or had stepwise worsening. The remainder had VS as long as they could remember [11••]. All subjects had continuous VS, 24/7 with eyes closed and open, independent of the outside light level, without periods of remission although it has to be stated that this might be a selection bias due to the high likelihood that patients with very severe symptoms, which would include persistence over years, are more likely to participate in research. Similarly, the mean age at onset in the early 20s is likely to have been biased by the recruitment strategy via internet due to the high familiarity of this generation with computer and internet.

## The Influence of Migraine and Typical Migraine Aura on the Phenotype of Visual Snow

Of the 64 subjects who recalled the onset or worsening of VS, only seven recalled that VS started or worsened with an episode of their typical migraine aura [11••]. In those, the aura was different from the VS they continuously experienced since. Further, only five in total had initially episodic VS and developed continuous VS later. All others had only one persisting and continuous episode of VS. An initial episodic occurrence would be typical for migraine aura [2], and its absence therefore underlines the distinction from migraine aura. The 23 patients (36 %) who had headaches, of whom 16 met the criteria for migraine, in the beginning together with the high prevalence of comorbid migraine (59 %) and typical migraine aura (27 %), however, highlights that VS and both migraine, as well as typical migraine aura, might have some pathophysiological overlap [11••].

To assess this relationship, 120 patients with VS were studied of which 70 had comorbid migraine and 37 had comorbid typical migraine aura [16••]. Comparison of the additional visual symptoms revealed that subjects who had comorbid migraine had a significantly higher chance of having more (mainly) non-entoptic visual symptoms: the odds ratios were 2.8 for palinopsia, 2.6 for trailing, 3.2 for photophobia, 2.9 for photopsia (spontaneous flashes of light), and 2.7 for impaired night vision (nyctalopia). The odds ratio for the non-visual symptom tinnitus was 2.9. This shows that patients who have migraine in addition to VS have a more severe visual snow syndrome than patients who have solely VS. The significance of this is not known, but it could be speculated that migraineurs suffer more when they have VS than non-migraineurs and thus are more likely to come to medical attention. Therefore, the high prevalence of migraine in VS patients might be overestimated due to self-selection bias—and with it the importance of migraine for VS pathophysiology. In contrast, typical migraine aura did not demonstrate such correlation with additional symptoms except for a higher prevalence of photopsia (odds ratio 2.4). Therefore, typical migraine aura does not aggravate the visual snow syndrome in the same way, and the higher prevalence of typical migraine aura might not be biased by selection. This data therefore supports that VS and typical migraine aura might share some pathophysiological background.

## Objective Measures in Patients with Visual Snow

Besides having continuous visual symptoms, patients with VS suffer significantly from being diagnosed as malingerers or as having a psychogenic disorder. This is mainly due to the inability to demonstrate pathologic findings in routine neurological and ophthalmological exams with also unremarkable brain imaging results. Further, the location of the syndrome in neurological terms would be helpful in understanding the mechanism behind this condition. To address this, 17 patients with visual snow were investigated using [$^{18}$F]-2-fluoro-2-deoxy-D-glucose ([$^{18}$F]-FDG) positron emission tomography (PET) and were compared to 17 age- and gender-matched healthy controls [16••]. When adjusted for the presence of typical migraine aura, the group with visual snow showed significant hypermetabolism in the area of the right lingual gyrus and the left anterior lobe of the cerebellum (Fig. 1b). This has several important implications: First, the lingual gyrus is part of the secondary visual cortex that is thought to modulate visual processing. Therefore, we have patients complaining of subjective visual symptoms and, correspondingly, alterations demonstrated with an objective method in an area that processes visual perception. This clearly supports a biological basis for the symptoms. The mechanism underlying this condition seems to be a dysfunction of a supplementary visual area that appears normal in routine brain imaging. Similarly, migraine occurs in subjects with a normal structural brain on the one hand but various dysfunctional areas on the other hand as demonstrated with different methods and during different phases of the migraine attack [17, 18]. By analogy, it can be argued that VS patients should be treated as if having a genuine biologically based problem not as being malingerers or having a psychogenic disorder. Secondly, photophobia is a hallmark of migraine. It is defined by light being perceived either as too bright or as painful and occurs typically during migraine attacks in migraineurs. Even interictally, migraineurs

are more sensitive to light than non-migraineurs [19]. A correlate of this could be that migraineurs, but not healthy controls, demonstrate hyperperfusion in $H_2{}^{15}O$ PET of the cuneus and lingual gyrus when exposed to light interictally [20]. The same area showed hyperperfusion in migraineurs during migraine attacks and light stimulation when compared with the interictal period [21••] (Fig. 1c) suggesting a correlate of migraine photophobia. Similarly, Maniyar et al. demonstrated hyperperfusion also in the lingual gyrus in patients who had photophobia in the premonitory phase of migraine, i.e. prior to any head pain in the beginning of the attack [22]. The similarity of the areas found in VS patients and during migraine attacks might, in part, explain the clinical overlap of both conditions. However, it has to be stated that the primary visual cortex (cuneus) did not show hypermetabolism in VS patients [16••] arguing for VS being a disorder of visual post-processing outside of the retino-ceniculo-cortical pathway. Further, VS patients were injected with [$^{18}$F]-FDG and stayed in the dark during the period prior to the PET scan. In other words, this study did not use light stimulation [16••] and thus did not measure photophobia as done in Denuelle's study [21••]. Thirdly, the lingual gyrus is part of Brodmann area 19 (BA 19), which is defined by the histological structure of the cortex [23]. Hadjikhani et al. [10] have studied patients during migraine aura using functional MRI and found that the blood-oxygen level dependent (BOLD) response to checkerboard stimulation shows an alteration during the course of the aura, which is located in the cortical area that retinotopically represents the aura symptoms in the visual field. This has been interpreted as an equivalent of cortical spreading depression, which has been thought to represent the pathophysiological correlate of typical migraine aura [9]. Interestingly, the authors also looked at the origin of the alteration of BOLD response and identified V3A [10] as being the first area in the brain that exhibits this pattern even before the clinical onset of visual symptoms. Although V3A is distant from the lingual gyrus, its histological structure indicates that it belongs to BA 19. In other words, the origin of typical migraine aura and the area of hypermetabolism in VS share a common histological structure, and this might indicate why typical migraine aura is so common in patients with VS.

## Conclusion

Visual snow (VS) belongs to the group of persistent positive visual phenomena seen in migraine due to the high prevalence of migraine and typical migraine aura in this population, as well as being observed in patients without migraine. Its main symptom is specifically defined as TV static-like vision, and the combination with additional visual symptoms, as well as tinnitus, emphasizes that it is a unique clinical syndrome that should be distinguished from persistent migraine aura or other, more non-specific visual phenomena in migraine. Comorbid migraine seems to worsen the clinical phenotype of the VS syndrome by increasing the risk of having additional visual symptoms and tinnitus. In contrast, typical migraine aura is very common in patients with VS without altering the phenotype suggesting some shared pathophysiology. Criteria for visual snow syndrome have been recently proposed (Table 1). These criteria were used in functional brain imaging, which has provided objective evidence that VS is associated with a dysfunction of the lingual gyrus. This shows that VS and typical migraine aura are both associated with a dysfunction in Brodmann area 19. The common cortical histology might account for the high prevalence of aura in patients with VS. The definition of these criteria will facilitate studies of the mechanism of VS and hopefully will result in treatment options to reduce the suffering of affected patients.

**Acknowledgments** CJS was supported by the German Research Foundation [SCHA 1676/1-1] and the Friedrich Baur Foundation of the University of Munich [58/14].

**Compliance with Ethics Guidelines**

**Conflict of Interest** Christoph J. Schankin: travel grant by Allergan and Boehringer Ingelheim and speaking honorarium by Boehringer Ingelheim and Novartis.

Peter J. Goadsby: Dr. Goadsby reports grants and personal fees from Allergan, grants and personal fees from eNeura, personal fees from Autonomic Technologies Inc, grants and personal fees from Amgen, personal fees from Bristol-Myers Squibb, personal fees from AlderBio, personal fees from Pfizer, personal fees from Zogenix, personal fees from Nevro Corp, personal fees from Impax, personal fees from Dr. Reddy, personal fees from Zosano, personal fees from CoLucid, personal fees from Eli Lilly, personal fees from Medtronic, personal fees from Avanir, personal fees from Gore, personal fees from Ethicon, personal fees from Heptares, personal fees from NuPathe, personal fees from Ajinomoto, personal fees from Teva, outside the submitted work.

**Human and Animal Rights and Informed Consent** This article does not contain any studies with human or animal subjects performed by any of the authors.

## References

Papers of particular interest, published recently, have been highlighted as:
- • Of importance
- •• Of major importance

1.• Simpson JC, Goadsby PJ, Prabhakar P. Positive persistent visual symptoms (visual snow) presenting as a migraine variant in a 12-year-old girl. Pediatr Neurol. 2013;49(5):361–3. **This is a very detailed description of a young girl with visual snow. It shows**

**that even young children can be affected, and it illustrates clearly the high impact on quality of life**.

2. Headache Classification Committee of the International Headache Society (IHS). The international classification of headache disorders: 3rd edition (beta version). Cephalalgia. 2013;33(9):629–808.
3. Liu GT, Schatz NJ, Galetta SL, Volpe NJ, Skobieranda F, Kosmorsky GS. Persistent positive visual phenomena in migraine. Neurology. 1995;45(4):664–8.
4. Wang YF, Fuh JL, Chen WT, Wang SJ. The visual aura rating scale as an outcome predictor for persistent visual aura without infarction. Cephalalgia. 2008;28(12):1298–304.
5. Eriksen MK, Thomsen LL, Olesen J. The Visual Aura Rating Scale (VARS) for migraine aura diagnosis. Cephalalgia. 2005;25(10):801–10.
6. Chen WT, Lin YY, Fuh JL, Hamalainen MS, Ko YC, Wang SJ. Sustained visual cortex hyperexcitability in migraine with persistent visual aura. Brain. 2011;134(Pt 8):2387–95.
7. Lashley KS. Patterns of cerebral integration indicated by the scotoma of migraine. Arch Neurol Psychiatr. 1941;42.
8.• Hansen JM, Baca SM, Vanvalkenburgh P, Charles A. Distinctive anatomical and physiological features of migraine aura revealed by 18 years of recording. Brain. 2013;136(Pt 12):3589–95. **This work shows the variability of typical migraine aura in a sufferer who had experienced and recorded more than 1000 attacks of migraine aura. The attacks were all very similar and clearly different from visual snow**.
9. Leao AAP. Spreading depression of activity in cerebral cortex. J Neurophysiol. 1944;7:359–90.
10. Hadjikhani N, Sanchez Del Rio M, Wu O, Schwartz D, Bakker D, Fischl B, et al. Mechanisms of migraine aura revealed by functional MRI in human visual cortex. Proc Natl Acad Sci U S A. 2001;8:4687–92.
11.•• Schankin CJ, Maniyar FH, Digre KB, Goadsby PJ. 'Visual snow'—a disorder distinct from persistent migraine aura. Brain. 2014;137(Pt 5):1419–28. **This study presents in detail the so far largest number of patients with visual snow. In addition to describing the phenotype of the visual snow syndrome consisting of additional visual symptoms, this work shows that visual snow typically does not start with headache or typical migraine aura and thus is likely different from persistent migraine aura. Criteria for the visual snow syndrome are presented**.
12. Critchley M. Types of visual perseveration: "palinopsia" and "illusory visual spread". Brain. 1951;74(3):267–99.
13. Tyler CW. Some new entoptic phenomena. Vis Res. 1978;12:1633–9.
14. Sinclair SH, Azar-Cavanagh M, Soper KA, Tuma RF, Mayrovitz HN. Investigation of the source of the blue field entoptic phenomenon. Invest Ophthalmol Vis Sci. 1989;30(4):668–73.
15. von Helmholtz H. Verschiedene subjective Erscheinungen. Handbuch der Physiologischen Optik. 2nd ed. Hamburg: Verlag von Leopold Voss; 1896. p. 572.
16.•• Schankin CJ, Maniyar FH, Sprenger T, Chou DE, Eller M, Goadsby PJ. The relation between migraine, typical migraine aura and "visual snow". Headache. 2014;54(6):957–66. **This work investigates the impact of migraine on visual snow. Having migraine results in a worsened visual snow syndrome by being associated with a higher frequency of additional visual symptoms. In contrast, typical migriane aura does not show a correlation suggesting some pathophysiological relevance. Using the criteria for the visual snow syndrome (see reference 11), hypermetabolism in the lingual gyrus could be demonstrated**.
17. Afridi SK, Matharu MS, Lee L, Kaube H, Friston KJ, Frackowiak RS, et al. A PET study exploring the laterality of brainstem activation in migraine using glyceryl trinitrate. Brain. 2005;128(Pt 4):932–9.
18. Weiller C, May A, Limmroth V, Juptner M, Kaube H, Schayck RV, et al. Brain stem activation in spontaneous human migraine attacks. Nat Med. 1995;1(7):658–60.
19. Vanagaite J, Pareja JA, Storen O, White LR, Sand T, Stovner LJ. Light-induced discomfort and pain in migraine. Cephalalgia. 1997;17(7):733–41.
20. Boulloche N, Denuelle M, Payoux P, Fabre N, Trotter Y, Geraud G. Photophobia in migraine: an interictal PET study of cortical hyperexcitability and its modulation by pain. J Neurol Neurosurg Psychiatry. 2010;81(9):978–84.
21.•• Denuelle M, Boulloche N, Payoux P, Fabre N, Trotter Y, Geraud G. A PET study of photophobia during spontaneous migraine attacks. Neurology. 2011;76(3):213–8. **The authors demonstrate that the lingual gyrus and cuneus might be associated with photophobia in migraine. The similarity to the distribution of hypermetabolism in visual snow supports the clinical and pathophysiological overlap of visual snow and migraine**.
22. Maniyar FH, Sprenger T, Schankin C, Goadsby PJ. Photic hypersensitivity in the premonitory phase of migraine—a positron emission tomography study. Eur J Neurol. 2014;21(9):1178–83.
23. Brodmann K. Vergleichende Lokalisationslehre der Großhirnrinde : in ihren Prinzipien dargestellt auf Grund des Zellenbaues. Johann Ambrosius Barth Verlag 1909.