MedCrave
Step into the World of Research

Journal of Neurology & Stroke

Review Article

Open Access

CrossMark

Visual allesthesia: looking through a negative prism? A review

Abstract

Visual Allesthesia is a rare misperception consisting of the visualization of an object, briefly exposed to view, whose resulting image is seen in one hemifield of vision while a duplicated image is viewed simultaneously in the opposite visual hemifield. It is the result of diverse medical or surgical conditions that cause disruption of the ventral visual stream. Surgical procedures that cause damage of that area should be avoided.

Keywords: misperception, stroke, epilepsy, release phenomenon, visual processing

Volume 13 Issue 4 - 2023

Allan J Drapkin MD, FACS(R)
Clinical Associate Professor, Department of Neurosurgery, Rutgers Biomedical and Health Sciences University, USA

Correspondence: Allan J Drapkin, Alejandro Serani Norte 9458 Dept.402, Región Metropolitana. Chile. Postal code: 7640801, Tel +56 233082519, Email ajdrapkin@gmail.com

Received: August 01, 2023 | **Published:** August 14, 2023

Introduction and background

Visual perceptual abnormalities, better defined as positive spontaneous visual phenomena (PSVP),[1] are occasionally encountered in various fields of medicine and may be caused by a variety of pathological conditions. PSVP may be expressed as *illusions,*[2,3] which are the distortion of a visual stimulus that is present in the external environment, or as *hallucinations* where a stimulus is perceived when in reality none is present.[4] Other types of PSVPs include *palinopsia*[1–5] which consist in the persistence of a visual sensation after its visual stimulus has ceased, and *metamorphopsia,* where the view of the objective´s size and/or form is altered. Another quite exceptional form of PSVP is *Visual Allesthesia* (VA), a condition that differs from illusions and hallucinations, in that a visual stimulus, present in the environment, is not distorted but its image is transposed from one homonymous visual field to the opposite one[2,6–13] resulting in the duplication of the viewed image, a duplication that is spatially reversed from the original one. In simple terms, it consists in seen the same half of an apple twice. VA, which at first could be interpreted as an ophthalmological refractive pathology, appears to be the result of damage to the occipito-temporal subcortical white matter[14,15] due to diverse medical conditions,[16] the most common of which is a right-sided, posterior stroke. In the realm of neurosurgery, VA has been rarely reported in cases of intra or extra-axial tumors located in that vicinity[17,18] or, as an exceptional postoperative complication.[19] Such a postoperative case[19] prompted the writing of this review.

Materials and methods

Published VA cases in the literature were collected and additional ones were found by perusing their references list and through Google searches. All the cases gathered were tabulated and analyzed in order to find their commonality and extract well founded conclusions (Table 1).

Table 1 Published cases of Visual Allesthesia

Author	Age	Sex	Cause	Side	Location	Seizures	Palinopsia	Visual Field Defect	Neglect
Murakami H[8]	49	M	Melas + AVM	Rt	O	Yes	Yes	HH	n/s
Ardilla #1[5]	37	M	Cysticercosis	Lt	O	Yes	Yes	HH	n/s
Ardilla #2[5]	50	M	Stroke	Rt	O	No	Yes	HH	Possible
Baumeler[10]	60	M	Hemorrhage	Rt	P	Yes	No	LIQ	Yes
Mendez & Chen[12]	57	M	Gun shot	Rt	P	Yes	Yes	Superior Arcuate	n/s
Gonzalez Mingot[9]	64	F	Hemorrhage	Rt	O	No	No	No	n/s
Kasten & Poggel[7]	61	F	Hemorrhage	Rt	OP	Yes	No	HH	n/s
Eretto et al.[13]	59	M	Dural AVM	Lt	O	Yes	Yes	No	n/s
Arai et al.[17]	63	F	FT Meningioma	Rt	O	Yes	Yes	No	n/s
Nakajima et al.[18]	30	M	AVM	Rt	OP	Yes	Yes	HH	n/s
Repsis et al.[15]	46	M	Glioblastoma	Rt	O	No	No	HH	n/s
Campos & Drapkin[19]	78	M	IV Meningioma	Rt	P	Yes	No	HH	No
Jeanette & Brouchon[11]	64	F	Stroke	Rt	P	Yes	No	No	Yes

F, female; M, male; O, occipital; P, parietal; HH, Homonimous hemianopsia; LIQ, lower inferior quadrantanopsia

Discussion

Review of this data supports the concept that VA´s pathological basis resides in damage to the temporo-occipital white matter with disruption of the ventral visual stream. It also confirms the marked prevalence of the pathology´s location in the non-dominant hemisphere. The ventral visual stream is a pathway through which visual information about the object´s color, size and form is transmitted from the striate cortex to the inferior temporal lobe (Brodman areas 20,37 and 38) for its processing.[20–23] Further analysis of the Table raises attention to the frequent association of VA with palinopsia (52%) and with seizure activity (78%), an association that suggests an ictal basis for VA, as its physio-pathogenic mechanism. This is further supported by the beneficial effect that anticonvulsive medications have over

VA and the exacerbation of VA that results from the discontinuation or reduction in this therapy dosage. Further analysis showed that although visual field defects are frequently associated with VA, these are not an indispensable VA accompaniment. Another surprising finding of this review was the rarity of neglect syndrome associated with VA, an association which should be expected considering the mostly non-dominant temporo-occipital location of VA´s cases.[24] This low frequency is more likely due to a lack of appropriate testing for neglect in the reported cases, rather than to a real scarcity of neglect symptoms in patients with this condition.[25] Although the ultimate cause of misperceptions is in general, is nebulous, various theories have been advanced to explain VA, with varying degrees of acceptance. Among these, the interhemispheric hypotheses model, which seems plausible, is based on two postulates: 1) It sustains that sensory information does not only reach the hemisphere contralateral to the stimulation but it would also reach, in some degree, the ipsilateral one, and 2) It supports the notion that, in normal brain functioning, the hemispheric locus contralateral to the stimulus will inhibit the activity of the ipsilateral one, possibly through callosal interhemispheric transmission. Should this be the case, the presence of a unilateral brain lesion disrupting the ventral visual stream would cause an imbalance that could result in VA[26] by:

a. Eliciting epileptogenic activity in the related cortical area.

b. by the biochemical and molecular changes induced in the deafferented neurons, that result in their hyperexcitability.[27]

c. by shutting-off an inhibitory circuitry, leading to a focal cortical excitation i.e., a release phenomenon.[27]

These hypothetical mechanisms strengthen Jacob´s hypothesis[6] that sustains that the irritated temporo-occipital cortex in the affected hemisphere, could hijack visual information from the normal contralateral homologous locus, attracting it, via callosal interhemispheric conduit,[28] and resulting in the duplicated image seen in VA.

In clinical practice, the type of PSVP encountered seems to depend on the particular cortical area affected by the pathological process.[22] For instance, electrical stimulation of the striate cortex (area 17) results in simple visual forms such as phosphenes, while stimulation of areas 18 and 19 evokes intermediate, more organized types of visual perceptions.[20,21] From a neurosurgical perspective, intra or extra-axial expansive lesions involving the parieto-temporal area may lead not only to neurological deficits but also to SPVPs by local pressure or tissue destruction.[17,18] Moreover, similar deficits can also be induced by damage incurred during cerebral resections for uncontrollable epilepsy or other surgical interventions, depending on the technique and the surgical approach undertaken.[19,29,30]

Considering the age of the patient, (because the outlook for recovery of function seems to be better in the younger age group),[31] the extent and location of the cortical and/or subcortical areas affected, and their inherent plasticity[32] these sequelae, VA among them, may be permanent or recover in a major or minor degree.

Ongoing research is focused on the detection and decoding of the mechanisms involved in generating and/or stimulating neural plasticity along the optic pathways,[33] possibly leading to newer and better rehabilitation methods that may eventually result in a better prognosis for these patients.[34]

Conclusion

VA occurs infrequently in clinical practice and it results from damage to the ventral visual stream circuit within the parietal white matter. Its prognosis depends on the extent of the neural damage and on the inherent plasticity of the tissues affected. To avoid this postoperative deficit, transcortical interventions through this area should be avoided.

Acknowledgments

The author wants to thank Ms. Elizabeth Gonzalez and to Mr. Gabriel Zepeda for their bibliographical support and to Ms. Camila Berner and to Mr. Samuel Abogabir for their technical assistance.

Contribution to the field

This article highlights a neurological symptom (VA) rarely encountered in clinical practice and its possible physio-pathological basis. Further-more it emphasizes the significant neurological deficits incurred by surgical interventions that disrupt the parieto-occipital subcortical white matter, some of them currently still being employed as an approach to the lateral ventricle. Such interventions should only be performed with the intraoperative assistance of diffusion tensor imaging and a frameless navigation system.[30]

Conflicts of interest

The author declares no conflicts of Interest to report for this work.

Funding

This research did not receive any specific grant from funding agencies in the public, commercial or not-for-profit sectors.

References

1. Celesia GG. The mystery of photopsias, visual hallucinations, and distortions. *Suppl Clin Neurophysiol.* 2006;59:97–103.
2. Norton JW, Corbett JJ. Visual perceptual abnormalities:hallucinations and illusions. *Sem in Neurol*. 2000;20:111–121.
3. Coffey E. Cummings J. Textbook of geriatric neuropsychiatry. Washington: APA Press. 1994.
4. Strub R. Black F. The mental status examination in Neurology. 3rd edn. Philadelphia: Davis Comp. 1993.
5. Ardilla A, Botero M, Gomez J. Palinopsia and visual Allesthesia. *Intern J Neurosci*. 1987;32:775–782.
6. Jacobs L. Visual Allesthesia. *Neurology.* 1980;30(1):1059–1063.
7. Kasten E, Poggel DA. Mirror in the mind: A case of Visual Allesthesia in homonymous hemianopia. *Neurocase.* 2006;12(2):98–106.
8. Murakami H, Ichikawa H, Sugimoto A, et al. Perceiving "ghost" Images:a unique case of visual Allesthesia with hemianopsia in mitochondrial disease. *Neuropsychiatric disease and treatment.* 2014;10:999–1002.
9. Gonzalez Mingot C, Velazquez A, et al. Macropsia, micropsia, Allesthesia and dyschromatopsia after occipital hemorrhage. *Neurología.* 2011;26(3):188–189.
10. Baumeler D, Born S, Burra N, et al. When left is one and right id double: An experimental investigation of visual Allesthesia after right parietal damage. *Vision*. 2020;4(1):1–13.
11. Joanette Y, Brouchon M. Visual Allesthesia in manual pointing:Some evidence for a sensorimotor cerebral organization. *Brain and cognition*. 1984;3(2):152–165.
12. Mendez MF, Chen WY. Epilepsia partialis continua with Visual Allesthesia. *Am J Neurol.* 2009;256:1009–1011.

13. Eretto PA, Schoen FS, Krohel GB, et al. Palinoptic Visual Allesthesia. *Am J Ophthalmol.* 1982;93(6):801–803.

14. Ungerleider LG, Mishkin M. Two cortical visual systems In: Ingle DJ, Goodale MA, et al. editors. *Analysis of visual behavior.* Cambridge: Massachusetts. The MIT Press. 1982.

15. Reptsis A, Demitzoglou I, Nikolakopoulos A, et al. Visual Allesthesia in a patient with glioblastoma multiforme. *J Ophthalmol.* 2012;2:97–102.

16. Girkin CA, Miller NR. Central disorders of vision in humans. *Survey of Ophthalmology.* 2001;45(5):379–405.

17. Arai T, Irie K, Akiyama M. et al. A case of falco–tentorial meningioma with visual allesthesia. *No To Shinkei*. 2002;54(3):255–259.

18. Nakajima M, Yasue M, Kaito N, et al. A case of visual Allesthesia. *No to Shinkei.* 1991;43:1081–1085.

19. Campos M, Drapkin AJ. The retro–chiasmal optic pathway: a link in jeopardy. *Front Surg*. 2020.

20. Catani M, Dell Aqua F, Bizzi A et al. Beyond cortical localization in clinical–anatomical correlation. *Cortex*. 2012;48(10):262–287.

21. Wall M. The retrogeniculate sensory visual system and higher cortical function. *J Neuro–Ophthalmol.* 1993;15(1):48–55.

22. Shallice T, Mussoni A, D´Agostino S, et al. Right posterior cortical functions in a tumor patient series. *Cortex.* 2010;46(9):1178–1188.

23. Milner AD, Goodale MA. The visual brain in action. 2nd.ed. New York: Oxford University Press. 2006.

24. Heilman KM, Valenstein E, editors. Clinical Neuropsychology. 2nd edn. New York: Oxford University Press. 1985. pp:243–293.

25. Burke W. The neuronal basis of Charles Bonnet hallucinations:a hypothesis. *J Neurol Neurosurg Psychiat.* 2002;73(5):535–541.

26. Kinsbourne M. A model for the mechanism of unilateral neglect of space. *Trans Am Neurol Assoc.* 1970;95:143–146.

27. Cogan DG. Visual hallucinations as a release phenomena. *Albrecht von Graefes Archiv für klinische und experimentelle ophthalmologie*. 1973;188(2):139–150.

28. Celeghin A, Diano M, Gelder B, et al. Intact hemisphere and corpus callosum compensate visuomotor functions after early visual cortex damage. *PNAS.* 2017;114:e10475–e10483.

29. Engel J, McDermott MP, Wiebe S. et al. Early surgical therapy for drug–resistant temporal lobe epilepsy. JAMA. 2012;307(9):922–930.

30. Agarwal V, Malcolm JG, Padilla G, et al. Tractography for optic radiation preservation In transcortical approaches to intracerebral lesions. 2017;9(9):e1722.

31. Guzzetta A, D´Acunto G, Rose S, et al. Plasticity of the visual system after Early brain damage. *Dev Med Child Neurol*. 2010;52(10):891–900.

32. Nahmani M, Turrigiano GG. Adult cortical plasticity following injury: recapitulation of Critical period mechanisms. *Neuroscience*. 2014;283:4–16.

33. Huxlin KR. Perceptual plasticity in damaged adult visual systems. *Vision Res*. 2008,48:2154–2166.

34. Sabel BA, Kasten E. Restoration of vision by training of residual functions. *Curr Opin Ophthalmol.* 2000;11:430–436.