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Holmes and Horrax (1919) revisited: Impaired binocular fusion as a cause of "flat vision" after right parietal brain damage – A case study

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ABSTRACT

The complete loss of binocular depth perception ("flat vision") was first thoroughly described by Holmes and Horrax (1919), and has been occasionally reported thereafter in patients with bilateral posterior-parietal lesions. Though partial spontaneous recovery occurred in some cases, the precise cause(s) of this condition remained obscure for almost a century. Here, we describe a unique patient (EH) with a large right-sided occipito-parietal hemorrhage showing a complete loss of visual depth perception for several months post-stroke. EH could well simultaneously describe multiple visual objects – hence did not show simultanagnosia – but at the same time was completely unable to estimate their distance from him. In every 3-D visual scene objects appeared equidistant to him, thus experiencing a total loss of depth perception ("flat vision"). Neurovisual assessments revealed normal functions of the eyes. EH showed bilateral lower field loss and a severely impaired binocular convergent fusion, but preserved stereopsis. Perceptual re-training of binocular fusion resulted in a progressive and finally complete recovery of objective binocular fusion values and subjective binocular depth perception in a far-to-near-space, gradient-like manner. In parallel, visual depth estimation of relative distances improved, whereas stereopsis remained unchanged. Our results show that a complete loss of 3-D depth perception can result from an isolated impairment in binocular fusion. On a neuroanatomical level, this connection could be explained by a selective lesion of area V6/V6A in the medial occipito-parietal cortex that has been associated with the integration of visual space coordinates and sustained eye-positions into a cyclopean visual 3-D percept.

1. Introduction

The three-dimensional (3-D) percept we perceive as our visual world requires the continuous unification of the two disparate images provided by our eyes (binocular fusion). Binocular fusion depends on a widespread neural network ranging from the frontal eye fields and brain stem oculomotor nuclei for proper eye alignment (convergence and divergence eye movements) to striate and extrastriate visual areas in order to merge the monocular images into a refined 3-D percept (Skelton and Kertesz, 1991; Alkan et al., 2011; Anzai and DeAngelis, 2010). A loss of single components in this neurocomputational system may cause distinct deficits in visual space perception, like diplopia or astereopsis, among others (Hart, 1969, Kraft et al., 2014). Probably, the most severe sequel is a total loss of binocular depth perception, resulting in a completely "flat" visual world, like on a picture.

This condition was first reported by Holmes and Horrax (1919) in their description of patient WF who suffered from a full loss of 3-D vision following a bilateral posterior head-wound caused by a gunshot in World War I. WF showed a complete lack of stereoscopic vision, manifesting in an inability to estimate and recognize any spatial depth between visual stimuli. Besides his full depth perception loss, he showed additional neurovisual and neuropsychological impairments, e.g. bilateral homonymous scotomas in the lower quadrants, simultanagnosia and reduced visual working memory capacities. With regard to his binocular functions, WF displayed severe difficulties in "bringing [visual stimuli]

Abbreviations: FU, follow up; SD, standard deviation; Mdn, median; IQR, interquartile range

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into central vision […] and in keeping his eyes fixed on a moving point" (Holmes and Horrax, 1919, p. 405), in the absence of any gross oculomotor impairments. This may be interpreted as a qualitative indication of difficulties in the initiation and maintenance of convergence eye movements (see above). Though repetitively examined over several months, WF´s condition remained remarkably stable, neither recovery nor slight improvements were reported.

A few similar cases since WF have been occasionally described in the last decades (see De Renzi, 1982 for review). All of these patients suffered from bilateral, mostly occipito-parietal lesions, usually resulting in homonymous visual field loss and deficits in simultaneous object detection (De Renzi, 1982; Gloning, 1965). Comorbid oculomotor impairments like gaze palsies or exotropia were occasionally reported, too (Michel et al., 1963; Gloning, 1965). A detailed and quantitative analysis of binocular visual capacities including an assessment of binocular fusion, stereopsis and visual depth estimation was however not conducted in the majority of reported patients. Thus, the identification of any "core deficit(s)" within the binocular vision network which may be responsible for the 3-D-vision loss is still missing. Unsurprisingly, little is known about recovery and nothing about potential treatments for this severe condition.

Here, we describe a unique patient (EH) that we studied over 2 years who showed an abrupt loss of visual depth perception after a unilateral right-sided stroke. We assessed several visual and binocular components potentially responsible for this condition and evaluated whether and to which extent this deficit could be modified by repetitive dichoptic training that is effective in less severe conditions of binocular vision impairment (Schaadt et al., 2014a, 2014b). Taken together, we argue that disturbed horizontal convergent fusion caused "flat vision" in patient EH and consequently may be the core deficit for this peculiar disorder.

2. Case description and Methods

Patient EH was a 56-year-old right-handed, successful lawyer with 18 years of education who had suffered from a right-sided parieto-occipital probably hypertensive hemorrhagic stroke 11 months before study entry. He had no history of neurological, ophthalmological or neuropsychological deficits prior to his stroke, in particular no squint. Neurovisual anamnesis revealed bilateral lower visual field loss and transient diplopia after enduring periods of binocular near space activity like reading or PC work. Particularly the latter prevented him from pursuing his regular vocation. However, as his most severe problem he described the complete loss of 3-D depth perception, subjectively manifesting in "flat vision": All visual stimuli in 3-D-space appeared equidistant to him, "like coulisses with no information about the space between the objects" and their distance to him. Though EH was independent in basic daily activities (e.g. personal hygiene, dressing), he described plain difficulties in all visuomotor activities involving depth judgments, e.g. taking staircases or walking on uneven ground, grasping, perception of motion in depth, despite proper sensorimotor functions of the lower and upper limbs.

2.1. Magnetic resonance imaging (MRI) of brain lesion

EH underwent MRI 3 weeks after the hemorrhage. To screen for extra lesions and white matter lesions, distinct T1 and T2-weighted data sets (FLAIR; TR/TE 10000/90 ms, TI = 2500 ms, FOV = 165 × 220 mm²; 28 transversal slices; T2*; TR/TE 700/15 ms, FOV = 192 × 220 mm²; 28 transversal slices) were analyzed. There was no evidence for additional subcortical, cortical or white matter lesions.

Table 1
Characteristics and affected areas of EH's brain lesion.

Lesion size	20375 voxels
Automatic anatomical labeling (AAL)	Angular gyrus Calcarine Cuneus Middle occipital Precuneus Superior occipital
Brodmann areas (BA)	BA 7 BA 17 BA 18 BA 19 BA 31 BA 30 BA 39
Center of mass	$X = 22$, $Y = -71$, $Z = 26$

For lesion reconstruction we used a high-resolution T1 MR data set (MPRAGE; TR/TE 1300/3.5 ms, TI = 650 ms, FOV = 256 × 240 mm²; 176 sagittal slices). The data set was normalized with MATLAB (The MathWorks, Natick, MA, USA) and SPM8 software (Wellcome Trust Centre for Neuroimaging, University College London, UK) using Chris Rorden et al.'s Clinical Toolbox (http://www.mccauslandcenter.sc.edu/CRNL/clinical-toolbox).

Two experienced raters (neurologist and psychologist) manually delineated the lesions to MRI images using MRICron software (http://www.sph.sc.edu/comd/rorden/mricron/install.html).

After normalization and lesion delineation, the lesion was registered to MNI space. This allows a standardized classification of which areas are affected in the patient brain using Automatic anatomical labeling toolbox (see Table 1 and Fig. 1). The lesion had an approximate volume of 20.38 cm^3, and was centered around the grey and white matter of the right parieto-occipital sulcus (see Fig. 1). Here it affected the calcarine fissure, middle and superior occipital gyrus, the cuneus and precuneus (see Table 1 for Brodmann areas). Only the most rostral part of the angular gyrus was lesioned. Moreover, the white matter was affected and the lesion extended to the posterior horn of the lateral right ventricle.

2.2. Neuropsychological profile

EH had no visual neglect, as he did not show left- or right-sided omissions in a digit cancellation task nor a neglect-typical shift towards the right side in horizontal line bisection (Hesse et al., 2012; Machner et al., 2009; Reinhart et al., 2013; Kuhn et al., 2012; see Table 2, below). He also showed no abnormalities in figure copying (star, daisy, face), nor omissions in an indented reading test (180 words; see Table 2). Furthermore, neglect-associated behavioral symptoms in everyday life situations like dressing, washing or eating were negated by himself and his wife and were never observed by us during the 2 years of investigations.

EH did not show simultanagnosia, as he could simultaneously detect and name more than 6 visual objects in a 3-D topographical scenery (in a room or outside). He was also well able to recognize up to 5 geometric forms drawn over each other, in order to assess simultaneous visual perception (cf. Finke et al., 2007). He showed no motor (i.e. hemiparesis), nor somatosensory impairments. His speech was fluent and educated; he answered promptly and appropriately to questions, thus showed no signs of aphasia. His memory capacities were unimpaired by the stroke. He performed above average in all visual and verbal memory tests (see Table 2 below). This finding is also congruent with Kraft et al. (2014)