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IMAGERY WITHOUT PERCEPTION—A CASE STUDY OF ANOSOGNOSIA FOR CORTICAL BLINDNESS

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Abstract—A patient with complete cortical blindness after bilateral posterior cerebral artery infarctions denied her blindness. Her pretended visual experiences could frequently be traced back to synaesthetic translations of acoustic or tactile perceptions into mental visual images. Possibly, the belief to see resulted from a confusion of mental visual images with real percepts. The patient manifested preserved visual imagery also by correct responses to questions concerning the shapes of letters and the shapes and colours of objects. MRI showed an almost complete destruction of primary visual cortex with sparing of only small remainders of cortex at the occipital tip of the left upper calcarine lip. In the literature there are a few cases of denial of blindness with similarly severe damage to primary visual cortex but none with unequivocal evidence of complete destruction of primary visual cortex. We conclude that severe damage to primary visual cortex is compatible with visual imagery but that there is a possibility that islands of visual cortex must be spared to permit the generation of mental visual images.

Key Words: imagery; blindness; anosognosia; visual cortex.

INTRODUCTION

The idea that similarities between visual imagery and visual perception are due to the involvement of identical cerebral structures found support in physiological studies which demonstrated activation of occipital cortex in healthy subjects engaged in visual imagery [6, 9, 11, 16–19, 21, 22, 30]. Recent advantages in PET and functional MRI imaging make it possible to distinguish the contributions of different cortical areas to occipital brain activity. Studies with both of these techniques showed activation of primary visual cortex in mental imagery [21, 22] in addition to activity in higher order visual cortex. The significance of these findings has, however, been called into question. On the one hand, other PET studies failed to find activation of primary visual cortex in mental visual imagery [31]. On the other hand, it has been argued that activation of an area during visual imagery does not necessarily prove that this area is critical to imagery [24]. The activation may reflect concomitant cognitive activities or shifts of attention which do not contribute to the generation of mental visual images.

If primary visual cortex contributes to the mental experience of visual imagery, loss of it should impair or abolish imagery. Clinical data on this issue are scarce as case

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studies of disorders of mental imagery were largely conducted on patients in whom the lesions affected secondary visual areas rather than the primary visual cortex [see 14 for review].

A condition which might allow insight into the role of primary visual cortex in visual imagery is anosognosia for blindness or Anton's syndrome [2]. Patients with Anton's syndrome believe that they are able to see despite absolute blindness. The patients do not simply disregard their blindness but positively report visual perceptions [2-4, 8, 12, 20, 23, 27, 29, 33]. Possibly, these descriptions refer to mental visual images. Anton's syndrome can occur with ocular diseases or optic nerve atrophy [7, 12, 26, 27, 29] as well as with cortical blindness [1-5, 8, 25-28, 34-36]. Preserved visual imagery in patients with blindness due to destruction of the primary visual cortex would be a strong argument against a critical involvement of primary visual cortex in imagery.

We recently observed a patient with transient cortical blindness and Anton's syndrome. This gave us the opportunity to investigate the role of mental visual images in Anton's syndrome and to assess the extent of damage to primary visual cortex.

CASE REPORT

H.S., a 46-year-old woman with 8 years of schooling suffered bilateral posterior cerebral infarction in November 1992. Ultrasonography showed occlusion of the tip of the basilar artery. Cardiac embolism was suspected but could not be proven. When taken to a rehabilitation unit 1 month after the accident, H.S. was cortically blind. Pupillar reaction to light was preserved but she could not discriminate between light and dark. Mobility was severely restricted because of tetraparesis and ataxia, the latter being most marked on the left limbs and the trunk.

In contrast to her repeatedly proven absolute blindness the patient spontaneously reported that sometimes she could see. She said that her sight was unreliable. Sometimes things around her would suddenly appear very clearly but after a few minutes they would vanish again. She never saw things which she believed to be unreal or unlikely to be visible, but when she grasped for a thing she had seen, for example a cup on the table, she could not find it where she expected it to be. She considered her visual abilities as being of little value in daily life, and when asked what her greatest problems were, she responded that these were her immobility and her 'bad sight'. She said that she had little hope of ever regaining normal vision.

While mobility improved, her blindness remained unchanged during the following 3 months. Neuropsychological assessment, examination of visual evoked potentials and three video-taped explorations of her visual behaviour were performed during that time (see below). MRI was performed near the end of this period.

The patient then returned in care of her family. When seen again 2 months later, her visual behaviour had changed. Her husband reported that she now actually saw things. She could describe them and reach them with her hands. The patient herself confirmed that her sight had improved and that now she could see things which previously she had not seen. On examination it was found that sight had indeed recovered within a narrow area of the right lower visual field. On Goldmann perimetry, the visual fields were restricted to the central 5 deg of the right lower quadrant being slightly wider on the right eye than on the left. Within the intact field, visual acuity was 1.25 with the right, and 1.0 with the left eye. A second MRI was done, visual evoked potentials and the neuropsychological assessment were repeated, and two explorations of her visual behaviour were video-taped (see below).

General neuropsychological findings

Only verbal tests could be given. Their results are summarized in Table 1. Her verbal IQ was 85 at the first examination in the period of anosognosia and 86 at the second examination after recovery of vision. At the first examination there was a marked memory deficit with delayed recall being nil for a list of 15 words as well as for the paragraphs of the Wechsler Memory Scale. However, recognition memory for words was better, and the patient was oriented to place, situation and approximate time and could remember day-to-day events. At the second examination there was only a slight improvement of episodic memory.

Semantic memory, as assessed by sentence verification, was defective for both knowledge about non-visual and visual properties of objects. On the first examination, H.S.'s score was below the range of controls (13, 15) on low-imagery sentences and shape imagery sentences, and in the lower range on colour imagery sentences. On the second examinations, all scores were at the lower margin of the normal range.

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