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# Neurological and neuropsychological characteristics of occipital, occipito-temporal and occipito-parietal infarction

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ABSTRACT

Neuropsychological deficits after occipital infarction are most often described in case studies and only a small sample of studies has attempted to exactly correlate the anatomical localization of lesions with associated neuropsychological symptoms. The present study investigated a large number of patients ($N = 128$) in order to provide an overview of neurological and neuropsychological deficits after occipital, occipito-temporal and occipito-parietal infarction. A particular approach of the study was to define exact anatomical correlates of neuropsychological dysfunction by using voxel-based lesion-symptom mapping (VLSM) in 61 patients. In addition to a visual field defect and phosphenes, patients often reported anomia, difficulties in reading and memory deficits. Visual disorders, such as achromatopsia, akinetopsia or prosopagnosia, were rarely reported by the patients. Memory and visual disorders were diagnosed efficiently using simple clinical screening tests, such as the Rey–Osterrieth Complex Figure Test for immediate recall, the Demtect and the Lang Stereo Test. Visual field defects, reading disorders and the perception of phosphenes were associated primarily with lesions of the calcarine sulcus. Anomia and memory deficits were related to lesions of the occipital inferior gyrus, the lingual gyrus and hippocampus, as well as to lesions of principal white matter tracts.



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## 1. Introduction

In the clinical context, occipital infarction is often perceived as being associated with visual field defects only (see Brandt et al., 2000 for a review; Steinke et al., 1997; Yamamoto et al., 1999; Ntaios et al., 2011). However, several studies have shown that occipital, occipito-temporal and occipito-parietal infarction lead to additional clinical and neuropsychological symptoms, such as deficits in reading, writing, memory and executive function (Cals et al., 2002; Kumral et al., 2004; Pessin et al., 1987; Gloning et al., 1966; Maulaz et al., 2005).

Visual disorders after cerebral lesions in the occipital, occipito-temporal or occipito-parietal cortex were described carefully in several studies in large samples of patients (e.g., Zihl and von Cramon, 1986; Gloning et al., 1966; Rowe et al., 2009). These studies did not distinguish between distinct etiologies such as stroke, tumors, craniocerebral injury or hemorrhage. Notably, they did not seek to relate the exact anatomical localization of lesions to associated clinical or neuropsychological symptoms in a systematic manner. This attempt was made by more recent reviews (e.g., Girkin and Miller, 2001; ffytche et al., 2010) that tried to allocate certain visual syndromes to specific cortical visual areas or to a disconnection between them. But these reviews still did not differentiate between distinct brain lesion etiologies.

Focusing on stroke, there is a limited number of reports which give systematic overviews of neuropsychological deficits after occipital, occipito-temporal and occipito-parietal infarction (Cals et al., 2002; Kumral et al., 2004; Pessin et al., 1987; Gloning et al., 1966). Some larger sample studies estimated a frequency of neuropsychological deficits such as visual agnosia, dyslexia, dysphasia or memory impairment between 32% and 50% of these patients (Cals et al., 2002; Milandre et al., 1994; Steinke et al., 1997). However, these studies did not allocate visual deficits or neuropsychological symptoms to specific anatomical locations within the infarct territory. Such an approach was taken by numerous single case reports or small sample accounts describing neuropsychological deficits after posterior strokes (Pessin et al., 1987; Holt and Anderson, 2000), applying the mini-mental state examination (Park et al., 2009), or focusing on disorders of reading and writing (Pillon et al., 1987; Habekost and Starrfelt, 2006; Pflugshaupt et al., 2009), memory (Benson et al., 1974; von Cramon et al., 1988; Machner et al., 2009), executive functions (Park et al., 2011), neglect (Park et al., 2006; Tomaiuolo et al., 2010) or on specific visual disorders such as visual hallucinations (Baier et al., 2010a; Tombini et al., 2012) and visual agnosia (Landis et al., 1986; Carlesimo et al., 1998; Ohtake et al., 2001; Konen et al., 2011). In summary, these case reports offer a good overview about the diversity of visual and neuropsychological deficits after occipital, occipito-temporal or occipito-parietal infarction, but cannot contribute to a statistically valid clinico-anatomical correlation between neuropsychological deficits and anatomical areas within the occipito-temporo-parietal region.

Conversely, there is only a small sample of studies that attempt to exactly correlate the anatomical localization of stroke lesions with associated clinical symptoms using a relatively new method called "voxel-based lesion-symptom mapping" (VLSM, e.g., Karnath et al., 2004). The emphasis of these studies was not to give a systematic overview about neuropsychological deficits. Instead, they focused on single clinical aspects, such as phosphenes and complex hallucinations (Baier et al., 2010a), motion processing (Billino et al., 2011), neglect (Karnath et al., 2004, 2011; Bird et al., 2006; Molenberghs and Sale, 2011), extinction (Chechlacz et al., 2013), dyslexia (Ptak et al., 2012), alexia (Pflugshaupt et al., 2009), picture naming (Baldo et al., 2013) or action recognition (Kalénine et al., 2010). To date there is no study using the VLSM approach in a large sample of stroke patients which tries to exactly correlate a broad array of neuropsychological deficits with anatomical lesions after occipital, occipito-temporal or occipito-parietal infarction.

The aim of the present study was to realize both: (a) a systematic description of subacute clinical and neuropsychological deficits after occipital, occipito-temporal and occipito-parietal infarction in a large sample of patients; and (b) an exact clinico-anatomical correlation for these deficits using a VLSM approach (Rorden et al., 2007).

## 2. Methods

### 2.1. Patients

An overall number of $N = 128$ patients participated in this study. Patients were recruited between 2007 and 2011 at the Department of Neurology, Charité Universitätsmedizin Berlin (Berlin, Germany) and at the Stroke Unit of the Medical Hospital Gesundheit Nord Klinikum Bremen-Mitte (Bremen, Germany). After receiving the approval of the local ethics committees, written informed consent was obtained from all patients and the examination was conducted in conformity with the Declaration of Helsinki.

Patients with ischemic cerebral infarction, as evidenced by magnetic resonance imaging (MRI) or computer tomography (CT), affecting the occipital gyri and sulci were included in this study. Apart from this primary criterion, lesions additionally extending into the temporal and/or parietal gyri and sulci were included as well. Both lesions involving the territories of the middle cerebral artery (MCA) or posterior cerebral artery (PCA) qualified for inclusion, if the above stated criteria were fulfilled. Exclusion criteria were severe aphasia, ophthalmological disorders such as glaucoma, cataract or macula degeneration in an advanced stage, or any other severe neurological or psychiatric disorder (e.g., epilepsy or schizophrenia).

### 2.2. Neurological testing and neuro-ophthalmological testing

Patients underwent a clinical neurological examination including finger perimetry, oculomotor function, reaching, motor function, extinction, neglect and reading. The basic neurological examination was carried out at least 24 h after a patient received a stroke.

Visual acuity (near and far, using Landolt rings) and static perimetry (30°) were conducted for the ipsilesional and contralesional eye. The Ishihara Test of color vision (Ishihara,