



Clinical neuroanatomy

Dissociable spatial and non-spatial attentional deficits after circumscribed thalamic stroke

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ABSTRACT

Thalamic nuclei act as sensory, motor and cognitive relays between multiple subcortical areas and the cerebral cortex. They play a crucial role in cognitive functions such as executive functioning, memory and attention. In the acute period after thalamic stroke attentional deficits are common. The precise functional relevance of specific nuclei or vascular sub regions of the thalamus for attentional sub functions is still unclear. The theory of visual attention (TVA) allows the measurement of four independent attentional parameters (visual short term memory storage capacity (VSTM), visual perceptual processing speed, selective control and spatial weighting). We combined parameter-based assessment based on TVA with lesion symptom mapping in standard stereotactic space in sixteen patients (mean age 41.2 ± 11.0 SD, 6 females), with focal thalamic lesions in the medial ($N=9$), lateral ($N=5$), anterior ($N=1$) or posterior ($N=1$) vascular territories of the thalamus. Compared with an age-matched control group of 52 subjects (mean age 40.1 ± 6.4 , 35 females), the patients with thalamic lesions were, on the group level, mildly impaired in visual processing speed and VSTM. Patients with lateral thalamic lesions showed a deficit in processing speed while all other TVA parameters were within the normal range. Medial thalamic lesions can be associated with a spatial bias and extinction of targets either in the ipsilesional or the contralesional field. A posterior case with a thalamic lesion of the pulvinar replicated a finding of Habekost and Rostrup (2006), demonstrating a spatial bias to the ipsilesional field, as suggested by the neural theory of visual attention (NTVA) (Bundesen, Habekost, & Kyllingsbæk, 2011). A case with

Abbreviations: ACC, anterior cingulate cortex; DLPFC, dorsolateral prefrontal cortex; FEF, frontal eye field; MR, magnetic resonance; LGN, lateral geniculate nucleus; MWT-B, Mehrfachwahl-Wortschatz-Intelligenz-Test; NTVA, neural theory of visual attention; PPC, posterior parietal cortex; ROCF, Rey-Osterrieth Complex Figure Test; RWT, Regensburger-Wortflüssigkeits-Test; SD, standard deviation; TPJ, temporal parietal junction; TVA, theory of visual attention; VBM, voxel-based morphometry; VOI, volume of interest; VSTM, visual short term memory; WCST, Wisconsin Card Sorting Test.

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an anterior-medial thalamic lesion showed reduced selective attentional control. We conclude that lesions in distinct vascular sub regions of the thalamus are associated with distinct attentional syndromes (medial = spatial bias, lateral = processing speed).

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1. Introduction

The thalamus is thought to be the gate to consciousness and is involved in nearly all behavioral functions. It is a critical component of cortical-basal ganglia-thalamic circuits that mediate planning and cognition, motivation, and emotional drive and the expression of goal-directed behaviors (Haber & McFarland, 2001). The thalamus consists of a large number of nuclei, which can be well displayed with the digital line drawings of Anne Morel's stereotactic atlas (Morel, 2007). The nuclei are mainly supplied by four distinct arteries: tuberothalamic artery, paramedian artery, inferolateral artery and posterior choroidal artery. However, it is difficult to define which nuclei in the human thalamus mediate specific cognitive functions, as lesions of the thalamus are typically large and encompass multiple nuclei (Van der Werf et al., 1999). So far, few researchers have investigated a sufficient number of patients with small thalamic lesions to make it possible to ascribe specific behavioral changes to lesions in circumscribed vascular territories or nuclei within the human thalamus (e.g., Lieberman, Ploner, Kraft, Kopp, & Ostendorf, 2013). Recent studies (e.g., Carrera & Bogousslavsky, 2006; Nolte, Endres, & Jungehülsing, 2011; Schmähmann, 2003) which made comparisons of cases with different lesion localisations, suggest that characteristic behavioral changes after thalamic lesions can be delineated on the basis of the four vascular territories (i.e., vascular syndromes of the thalamus). In the acute period the rare cases with isolated anterior (tubero) thalamic infarction showed an “anterior behavioral syndrome” with apathy, amnesia and perseveration or executive dysfunction. Paramedian infarction is associated with a decreased level of consciousness, gaze paresis and cognitive impairment, such as disinhibited behavior or amnesia, while inferolateral infarction results in ataxia and hemihypesthesia. Isolated posterior choroidal infarction is also extremely rare. For the few cases with posterior choroidal infarction, visual field defects and neglect were observed (Habekost & Rostrup, 2006; Karnath, Himmelbach, & Rorden, 2002; Nolte et al., 2011).

These distinct vascular syndromes of the thalamus occur because specific regions of the thalamus are connected with specific areas of each basal ganglia structure and specific cortical regions. For example, the frontal eye field (FEF) and the dorsolateral prefrontal cortex (DLPFC) are connected with the corpus of the caudate nucleus and the ventral lateral and mesial part of the mediodorsal nucleus of the thalamus, thus resulting in gaze paresis and cognitive impairment after paramedian infarction. However, recent data indicate that the thalamus not only relays information back to the cortex, but may also serve as an important center of integration of networks that underlie the ability to modulate behavior (Haber & McFarland, 2001). For example, the “attention network”,

involves multiple cortical areas [DLPFC, FEF, anterior cingulate cortex (ACC), posterior parietal cortex (PPC), temporo-parietal junction (TPJ)] and structures of the basal ganglia (caudate nucleus, putamen) that are connected with specific nuclei of the thalamus (ventral anterior nucleus, mediodorsal nucleus, ventral lateral nucleus, pulvinar). It has not yet been clarified whether distinct attentional deficits (e.g., bias in spatial weighting, reduced/enhanced selective control, reduced processing speed) can be observed if specific nuclei/vascular territories of the thalamus are lesioned.

Bundesen and colleagues have proposed a thalamic model for the theory of attention (TVA, Bundesen, 1990) called “neural theory of attention” (NTVA) which is mainly built upon neurophysiological findings in macaque monkeys (Bundesen, Habekost, & Kyllingsbæk, 2005; Bundesen et al., 2011). According to NTVA, visual information is first processed in an unselective wave from the retina to the lateral geniculate nucleus (LGN) of the thalamus and then to the striate and extrastriate visual cortex. In the cortex, individual perceptual values for each object are computed and multiplied by pertinence values. The information then enters the pulvinar nucleus of the thalamus where a saliency map is located and products are summed up as attentional weights. Weighted information is then processed in a second selective wave from the pulvinar to higher level visual processing areas in the cortex. The resulting perceptual values are multiplied by bias values, and the products are then transmitted from the cortex to the thalamic reticular nucleus (TRN) where a visual short term memory storage capacity (VSTM) map of locations is localized.

TVA is a mathematical model which allows disentangling four independent attention parameters: Using whole and partial report paradigm allows measuring of the perceptual processing speed, VSTM, selective control and the spatial distribution of attentional weights reliably in normal participants, as well as in neurological patients, such as patients suffering from stroke (Duncan, et al., 1999) or Huntington's disease (Finke, Bublak, Dose, Müller, & Schneider, 2006). In recent years the TVA-based methodology has been used to investigate selective attention deficits after persistent and transient lesions of cortical sub regions, revealing that lesions in the entire cortical attention network consisting of the FEF, the middle frontal gyrus, the DLPFC, the TPJ, posterior PPC and inferior parietal regions lead to selective attention deficits in TVA parameters (Bublak et al., 2005; Duncan et al., 1999; Finke et al., 2006; Habekost & Bundesen, 2003; Habekost & Rostrup, 2006, 2007; Peers et al., 2005, see Habekost & Starrfelt, 2009 for a review).

So far, distinct thalamic lesions have not been investigated with the TVA-based methodology. However, a number of studies report attentional deficits after thalamic or basal

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