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Review

Neurosurgical targets for compulsivity: What can we learn from acquired brain lesions?

Martijn Figee[a,*], Ilse Wielaard[b], Ali Mazaheri[a], Damiaan Denys[a,c,**]

[a] Department of Psychiatry, Academic Medical Center, University of Amsterdam, Amsterdam, The Netherlands
[b] Utrecht University, Utrecht, The Netherlands
[c] The Netherlands Institute for Neuroscience, an institute of the Royal Netherlands Academy of Arts and Sciences, Amsterdam, The Netherlands

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ABSTRACT

Treatment efficacy of deep brain stimulation (DBS) and other neurosurgical techniques in refractory obsessive-compulsive disorder (OCD) is greatly dependent on the targeting of relevant brain regions. Over the years, several case reports have been published on either the emergence or resolution of obsessive-compulsive symptoms due to neurological lesions. These reports can potentially serve as an important source of insight into the neuroanatomy of compulsivity and have implications for targets of DBS. For this purpose, we have reviewed all published case reports of patients with acquired or resolved obsessive-compulsive symptoms after brain lesions. We found a total of 37 case reports describing 71 patients with acquired and 6 with resolved obsessive-compulsive symptoms as a result of hemorrhaging, infarctions or removal of tumors. Behavioral symptoms following brain lesions consisted of typical obsessive-compulsive symptoms, but also symptoms within the compulsivity spectrum. These data suggests that lesions in the cortico-striato-thalamic circuit, parietal and temporal cortex, cerebellum and brainstem may induce compulsivity. Moreover, the resolution of obsessive-compulsive symptoms has been reported following lesions in the putamen, internal capsule and fronto-parietal lobe. These case reports provide strong evidence supporting the rationale for DBS in the ventral striatum and internal capsule for treatment of compulsivity and reveal the putamen and fronto-parietal cortex as promising new targets.

Contents

* Corresponding author. Tel.: +31 20 891 3500; fax: +31 20 891 3701.
** Corresponding author. Tel.: +31 20 891 3899; fax: +31 20 891 3701.
E-mail addresses: m.figee@amc.nl (M. Figee), ddenys@gmail.com (D. Denys).

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1. Introduction

Compulsivity encompasses the repetitive, irresistible urge to perform a behavior, the experience of loss of voluntary control over this intense urge, the diminished ability to delay or inhibit thoughts or behaviors, and the tendency to perform repetitive acts in a habitual or stereotyped manner (Denys, 2011). Compulsivity occurs in a broad range of psychiatric disorders, such as drug addiction, Tourette's syndrome, impulse control disorders and eating disorders, but it is epitomized by obsessive-compulsive disorder (OCD). OCD is a chronic psychiatric illness in which the person suffers from recurrent and disturbing thoughts (obsessions) that cause distress and ritualistic acts (compulsions) that are usually performed to neutralize the distress (Diagnostic and Statistical Manual of mental disorders (DSM-IV-TR, 2000)). Previous research estimated that pharmacotherapy and behavioral therapy provide on an average a 40–60% symptom reduction in half of OCD patients, however, approximately 10% of patients remain severely affected and suffer from treatment-refractory OCD (Denys, 2006). For a small proportion of treatment-refractory patients, ablative neurosurgery or deep brain stimulation (DBS) may be appropriate (de Koning et al., 2011). DBS involves the implantation of electrodes that send electrical impulses to specific locations in the brain. In contrast to ablative neurosurgery, DBS is reversible and adjustable. The location in the brain is chosen according to the type of disorder to be addressed and its putative neuroanatomical correlates (Rauch, 2003). Numerous neuroimaging studies have related OCD to pathology in the cortical-striatal-pallidal-thalamic-cortical (CSTC) network (Saxena et al., 1998; Menzies et al., 2008). In the CSTC network, information from limbic and associative cortices is integrated in the striatum, projected on to the globus pallidus, subthalamic nucleus and substantia nigra and then returned to the cortex through a thalamic relay (Alexander, 1986). The CSTC network integrates cortical with subcortical information for the selection of automatic motor and cognitive programs that maximize rewarding outcomes, and these features are dysfunctional in OCD (e.g. Figee et al., 2011). Accordingly, targets that have been used for DBS treatment in over 100 patients with OCD are all located within the CSTC-circuit: the anterior limb of the internal capsula (ALIC), ventral capsule/ventral striatum (VC/VS), nucleus accumbens, subthalamic nucleus and the inferior thalamic peduncle (de Koning et al., 2011). However, stimulation of these different targets has resulted in variable efficacy, ranging from a minimal response to almost complete remission of symptoms.

We argue that neuroimaging data by itself may be inadequate as a theoretical basis for choosing DBS targets, given that any hyper/hypo activation in a region could be a symptom of the illness, rather than its cause. In this paper, we will explore a new avenue to achieve reliable information on potential targets for DBS in OCD. We will review available neuroanatomical information from case reports of patients with acquired or remitted OCD due to infarctions or other brain lesions. We believe that lesion studies provide a very strong foundation for a possible link between a brain region and a behavior (in this case compulsivity). Furthermore, circumscribed brain lesions that induce or resolve obsessive-compulsive symptoms may reflect important brain network nodes that can be inhibited or excited with DBS. Ultimately, the goal of our endeavor is to use the information yielded by lesion studies as a potential guide for defining optimal brain targets for therapeutic DBS in OCD and other compulsive disorders.

2. Methods

Published studies of case reports with acquired OCD were identified through searches of electronic databases (e.g. PsycINFO, PubMed) and reference lists of scientific articles. We searched for: OCD, obsessive-compulsive behavior, obsession, compulsion, stereotyped activities, lesion, injuries, damage, CVA, hemorrhage, tumor, infarction, stroke, disease, Parkinson's, Huntington's, Wilson's, Tourette's, Sydenham chorea, basal ganglia, lenticular (nuclei), striatum, caudate, putamen, nucleus accumbens, globus pallidus, internal capsule, limbic system, amygdala, brainstem, hypothalamus, pineal region, cerebellum, frontal, parietal, temporal and occipital lobe or cortex. These words were entered in different combinations in the electronic databases. We included all studies that contained cases of patients with acquired obsessive-compulsive symptoms due to a neurological lesion, or due to a disease that affected brain tissue or closed head injuries. We only included case reports that also reported neuroanatomical data using computed tomography (CT) or structural magnetic resonance imaging (MRI), or using functional imaging such as positron emission tomography (PET) or functional MRI. This pursuit yielded an initial sample of 42 articles that reported a total of 87 cases, published between 1954 and 2012. From this sample, we excluded three articles because of the lack of individual patient information (Alegret et al., 2001; Giroud et al., 1997; Berthier et al., 2001). Additionally, two studies were excluded for methodological reasons: one study did not provide detailed neuroimaging information (McKeon et al., 1984), while the other did not describe an acquired