



# An unusual presentation of convergence insufficiency in a patient with Parkinson's disease stimulated by deep brain stimulation

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## ABSTRACT

**Purpose:** To report convergence insufficiency in a patient with Parkinson's' disease stimulated by turning on the deep brain stimulator.

**Observations:** 72-year-old male with Parkinson's disease and hypertension presenting for the evaluation of blurry vision at near and mid distance that started after activation of an implanted Deep brain stimulator.

Baseline ophthalmologic evaluation prior to deep brain stimulator implantation surgery and with the deep brain stimulator turned off demonstrated a full motility, centered eyes for distance and near and a best corrected visual acuity of 20/20, normal pupil exam, confrontational visual fields and dilated fundus exam. Following this examination, the Deep brain stimulator was turned on and re-evaluation few minutes later demonstrated the same findings except for a 6-prism diopter exotropia at near consistent with convergence insufficiency.

Following our evaluation a set of +3 diopters base-in prisms were added to near glasses with total relief of symptoms. The patient did not require surgical adjustment of the deep brain stimulator leads.

**Conclusions and importance:** Given the therapeutic effects of deep brain stimulation on convergence insufficiency reported in several studies, in addition to the influence of deep brain stimulation as Parkinson's Disease treatment in areas possibly associated with vergence control, convergence insufficiency secondary to deep brain stimulation does not seem very unlikely, although not often reported. Further studies are needed to optimize deep brain stimulation surgery to maximize benefits and limit adverse events, as well as being aware of convergence insufficiency as a possible cause for visual disturbance.

## 1. Introduction

Vergence and ocular alignment involve the coordination of complex neural networks distributed in different areas of the central nervous system (CNS). Hence, abnormal eye movements and subsequent diplopia may be seen in multiple neurodegenerative diseases, including Parkinson's Disease (PD), a progressive neurodegenerative disease characterized by the loss of dopaminergic neurons in the substantia nigra, leading to disturbance of the basal ganglia signaling pathways that control motor function.<sup>1</sup> Convergence insufficiency (CI) is an eye movement abnormality commonly seen in patients with PD, presenting as blurry vision or diplopia when performing tasks at near, and it significantly reduces vision-related quality of life.<sup>2</sup> Fortunately, convergence ability was shown to improve on dopaminergic therapy or deep brain stimulation (DBS), a neurosurgical therapy used in patients

with symptoms refractory to medications (see [Table 1](#)).<sup>3,4</sup>

In the following case, we discuss visual abnormalities reported by a PD patient, diagnosed as convergence insufficiency, that appeared after the activation of an implanted deep brain stimulator.

## 2. Case report

A 72-year-old male patient known to have Parkinson's Disease and hypertension presented to our Neuro-Ophthalmology clinic for evaluation of blurry vision at near and intermediate distances that started after the activation of the implanted deep brain stimulator.

The patient had been examined before DBS surgery at our clinic as a baseline and was found to have: a best corrected visual acuity (BCVA) of 20/20 in both eyes (OU), normal pupillary reaction to light and to near accommodation, normal ductions and versions, normal saccades and

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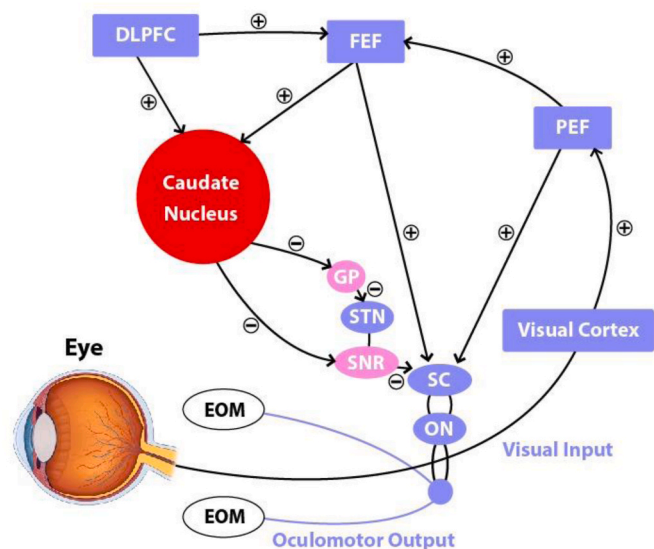
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**Table 1**  
Deep brain stimulator-Induced neurologic complications with proposed underlying pathways.<sup>1</sup>

Complication	Deep brain stimulator Target	Proposed region underlying manifestation of side effects
Weight gain	STN, GPI	Unknown, but thought to result from normalization of energy metabolism
Verbal fluency, working memory	GPI, STN	Associative circuits of basal ganglia
Sadness, depression	STN	Ventromedial STN, SNr, regions around SNr
Phosphenes	GPI	Optic tract
Parasthesias	VIM, STN, VOP, PPN	Lemniscal fibers
Oculomotor disturbances	GPI, STN	<b>Internal capsule for conjugate eye deviation, third nerve medial to STN for ipsilateral eye movements</b>
Muscle contraction	STN, GPI, VOP	Corticospinal tract of internal capsule
Mood changes, risky behavior	GPI, STN	Associative and limbic circuits of basal ganglia
Dysphonia, dysarthria	STN, GPI	Internal capsule and associative circuits of basal ganglia
Dyskinesias	GPe, GPI, STN	Excessive modulation of indirect pathway, including GPe and STN

Deep brain stimulator, deep brain stimulation; GPe, external globus pallidus; GPI, internal globus pallidus; PPN, pedunculo pontine nucleus; SNr, substantia nigra pars reticularis; STN, subthalamic nucleus; VIM, ventral intermediate nucleus; VOP, posterior ventral oral nucleus.



**Figure 1.** Saccadic eye movements are supported by a distributed network of cortical and subcortical regions. Saccades are initiated by direct signals sent from the parietal or frontal eye fields (PEFs or FEFs) to the superior colliculus (SC), which drives the oculomotor network (ON) in the brainstem. An indirect “gating” circuit arising from the FEFs and dorsolateral prefrontal cortex (DLPFC) projects via the basal ganglia (caudate nucleus, globus pallidus [GP], and subthalamic nucleus [STN]) to the substantia nigra pars reticulata (SNr). The SNr inhibits the SC, preventing saccade generation. To switch off this inhibition, when the FEFs and other frontal structures are activated before a saccade, the caudate nucleus is activated, which, in turn, inhibits the SNr via an inhibitory pathway.

pursuit gaze movements, full visual fields on confrontational testing, and normal anterior segment exam and dilated fundus exam OU. The patient was re-examined after the deep brain stimulator implantation surgery before its activation, and his ophthalmic exam was identical to baseline. The deep brain stimulator was then activated, and the patient was re-evaluated 30 minutes later: the exam showed comparable

findings, except for a 6 prism-diopter ( $\Delta$ ) exotropia at near, with otherwise full motility and normal ocular alignment in both eyes, consistent with convergence insufficiency. The patient would return to baseline when DBS was turned off.

Given the need for DBS to treat parkinsonism, the patient was prescribed spectacles with +3.00  $\Delta$  base-in OU for near vision as a treatment of CI, with total relief of his visual symptoms. Hence, the surgical manipulation of the deep brain stimulator was not required.

### 3. Discussion

Parkinson’s Disease is associated with many motor and non-motor ocular signs and symptoms: a recent prevalence study based on the Visual Impairment in Parkinson’s Disease Questionnaire (VIPD-Q) showed that 82% of patients with PD have one or more ophthalmologic complaints, more frequently than healthy controls, and interfering with their daily activities and reducing their quality of life.<sup>5</sup> These include ocular surface symptoms such as dry eyes and epiphora, oculomotor symptoms such as blepharospasm, apraxia of eyelid opening, abnormal saccades and pursuit, diplopia, and convergence insufficiency, and neural problems such as visual hallucinations, decreased visual acuity, color vision and contrast sensitivity.<sup>2,3,5,6</sup> Convergence Insufficiency is particularly debilitating as it affects the patient’s ability to perform daily tasks at near.<sup>7</sup> Fortunately, CI was shown to improve on PD treatment, both dopaminergic treatment and DBS.<sup>3</sup>

DBS is a neurosurgical intervention used to relieve the motor symptoms of movement disorders (essential tremor, Parkinson’s disease, dystonia), and other neurological (epilepsy, cluster headache, chronic neuralgia) and psychiatric diseases (Tourette Syndrome, obsessive-compulsive disorder, depression).<sup>8</sup> DBS is being used more frequently in PD especially in patients with severe symptoms or those who have experienced unacceptable dopaminergic medication side effects, namely, severe motor fluctuations (“wear-off” effect) and dyskinesias.<sup>9</sup>

DBS consists of implanting programmable electrodes in specific targets in the brain, connected to a neurostimulator placed typically below the clavicle. In PD, DBS targets the Sub-Thalamic Nucleus (STN) or the Globus Pallidus internus (GPI), either unilaterally or bilaterally.<sup>10</sup> The effect of DBS on neuronal tissue surrounding the electrodes is not fully elucidated. Recent studies have suggested that the high-frequency regular pattern of axonal activation via DBS abolishes pathological bursting activity, leading to reduction in disease symptoms, rather than inhibiting neuronal activity at the site of stimulation.<sup>11</sup>

As any surgical intervention, DBS is not without risks, which may include cerebral hemorrhage, stroke, and infection. Most importantly, undesirable sensorimotor side effects, including paresthesia, speech difficulties, dystonias, dyskinesias, and contractile movements may occur either due to the alteration of basal ganglia-mediated signaling pathways, or due to the stimulation current spreading into regions adjacent to the DBS target.<sup>11</sup> In our case, CI was triggered by DBS activation: it was not present before DBS surgery and hence, was not secondary to PD, nor after DBS surgery before the activation of the stimulator and hence, was not secondary to the implantation surgery itself.

Oculomotor disturbances have been previously reported upon the activation of DBS targeting the STN and the GPI. Shields et al. have reported reduced ipsilateral gaze and a contralateral conjugate eye deviation after the activation of DBS targeting the STN in a patient with PD, which was possibly due to the activation of the ipsilateral frontal eye field cortical regions via the spread of the electric current to nearby frontal eye field axons coursing lateral to the STN within the internal capsule.<sup>12,13</sup> Friedrich et al. described an ipsilateral ocular tilt reaction and body lateropulsion and a contralateral torsional nystagmus post-DBS activation at the STN, attributed to the irritation of a possible pathway between the interstitial nucleus of Cajal, the integrator of vertico-torsional eye and head movements, and the zona incerta, a subthalamic area involved in locomotion, posture and arousal.<sup>14</sup> Miyagi

et al. reported a case of paroxysmal diplopia and convergence insufficiency after DBS lead replacement surgery, possibly due to improper oculomotor fibers running medial to the STN stimulation.<sup>15</sup>

An association between vergence and the basal ganglia must exist, given the therapeutic effects that DBS targeted at the basal ganglia has on convergence insufficiency and related eye movements, which can also explain why CI could occur as an adverse effect to DBS. CI was shown to be responsive to STN-DBS possibly through stimulation of supranuclear inputs to midbrain near response neurons or superior colliculus disinhibition by normalizing STN input to the substantia nigra pars reticulata.<sup>4</sup> The aberrant stimulation of this network (i.e. inadequate frequency) may explain DBS-induced CI. DBS was shown to improve pro-saccadic latencies and velocity, and increase smooth pursuit velocity and accuracy, which involve not only the basal ganglia but also higher cortical functions.<sup>16</sup> This could be possible because the basal ganglia modulate the extended cortical network possible for pursuit and saccades, notably, the frontal eye fields, supplementary eye fields, dorsolateral prefrontal cortex, and parietal eye fields, areas that seem to be partly involved in vergence control.<sup>16–18</sup> Vergence control is also mediated by thalamic networks, the targets of DBS in PD, as loss of control and vergence deficiency is observed secondary to thalamic hemorrhages.<sup>18</sup> A direct clear pathway connecting the basal ganglia and vergence centers might not be elucidated, but the presence of such network is certain for the reasons mentioned above, which is why the effects of DBS on CI, whether therapeutic or adverse, are not surprising.

#### 4. Conclusion

As the effects of DBS on neuronal tissues are not fully elucidated and given that they are affected by multiple factors related to the surgery itself, the pathology and the programming of the stimulating leads, DBS may have a wide range of therapeutic or adverse effects linked to electrostimulation. In PD treatment, DBS may positively or negatively affect convergence, as multiple studies denote the presence of networks connecting vergence control and the basal ganglia. Further studies are needed to optimize DBS localization and programming to maximize benefits, minimize side effects and to discover the therapeutic spectrum of basal ganglia-targeted DBS.

#### Patient consent

The patient consented to publication of the case in writing.

#### Conflicts of interest

The following authors have no conflict of interest to declare: Rayan Abou Khzam, Nahia Dib El Jalbout, Roland Seif, Ama Sadaka.

All authors attest that they meet the current ICMJE criteria for Authorship.

#### Conflicts of interest

Potential conflict of interest exists:

We wish to draw the attention of the Editor to the following facts, which may be considered as potential conflicts of interest, and to significant financial contributions to this work:

The nature of potential conflict of interest is described below:

No conflict of interest exists.

We wish to confirm that there are no known conflicts of interest associated with this publication and there has been no significant financial support for this work that could have influenced its outcome.

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#### Intellectual property

We confirm that we have given due consideration to the protection of intellectual property associated with this work and that there are no impediments to publication, including the timing of publication, with respect to intellectual property. In so doing we confirm that we have followed the regulations of our institutions concerning intellectual property.

#### Research ethics

We further confirm that any aspect of the work covered in this manuscript that has involved human patients has been conducted with the ethical approval of all relevant bodies and that such approvals are acknowledged within the manuscript.

IRB approval was obtained (required for studies and series of 3 or more cases).

Written consent to publish potentially identifying information, such as details or the case and photographs, was obtained from the patient(s) or their legal guardian(s).

#### Authorship

The International Committee of Medical Journal Editors (ICMJE) recommends that authorship be based on the following four criteria:

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2. Drafting the work or revising it critically for important intellectual content; AND
3. Final approval of the version to be published; AND
4. Agreement to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

#### Contact with the editorial office

The Corresponding Author declared on the title page of the manuscript is: Ama Sadaka.

#### Declaration of competing interest

None.

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