Disputes & Debates: Editors' Choice

Steven Galetta, MD, FAAN, Section Editor

Editors' note: Deep brain stimulation improves restless legs syndrome in patients with Parkinson disease

In the article "Deep brain stimulation improves restless legs syndrome in patients with Parkinson disease," Dr. Klepitskaya et al. reported sustained improvement in symptoms of restless legs syndrome (RLS) over 2 years in 22 patients with Parkinson disease (PD) who underwent subthalamic nucleus (STN) deep brain stimulation (DBS), despite a decrease in dopaminergic treatment. Drs. Marques et al. point out that the article misinterpreted the results of their previous study, which showed an emergence of RLS in patients with PD with a higher dose of dopamine agonists and a lower decrease in dopaminergic treatment after STN DBS. They discuss the paradoxical findings of STN DBS apparently improving preexisting RLS but causing emergence of RLS in some patients with PD and the need for further research. In response, Dr. Klepitskaya acknowledges these comments including the misinterpretation. A correction appears on page 871.

Aravind Ganesh, MD, and Steven Galetta, MD
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Reader response: Deep brain stimulation improves restless legs syndrome in patients with Parkinson disease

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Several studies assessed the effect of deep brain stimulation (DBS) of the subthalamic nucleus (STN) on restless legs syndrome (RLS) symptoms in patients with Parkinson disease (PD) with conflicting results.[1–5] As underlined by Klepitskaya et al.,[1] one crucial variable is dopaminergic therapy, as emergence of RLS after STN DBS in patients with PD has been reported after abrupt decrease of dopaminergic treatment.[3] Klepitskaya et al. argued that, in a previous study we conducted,[5] patients with emergence of RLS after STN DBS took a higher dose and had a more significant reduction of dopamine agonists compared to those without emergence with RLS. Yet, we actually reported the opposite, showing an emergence of RLS in patients with PD with a higher dose of dopamine agonists postoperatively and with a lesser decrease of dopamine agonists after STN DBS.[5]

The results from these apparently controversial studies suggest a paradoxical effect of STN DBS that could improve preexisting RLS,[2,4] but could cause the emergence of RLS in preoperatively RLS-free patients with PD via different reported mechanisms (abrupt decrease of dopaminergic treatment, which could unmask previously controlled RLS[3]; or insufficient decrease of dopamine agonists, which could lead to dopaminergic overstimulation,[5] resembling augmentation syndrome). Further studies, taking into account the localization of contacts, are needed to clarify the role of STN DBS on RLS in PD.

Author disclosures are available upon request (journal@neurology.org).

1. Klepitskaya O, Liu Y, Sharma S, et al. Deep brain stimulation improves restless legs syndrome in patients with Parkinson disease. Neurology 2018;91:e1013–e1021.
2. Chahine LM, Ahmed A, Sun Z. Effects of STN DBS for Parkinson's disease on restless legs syndrome and other sleep-related measures. Parkinsonism Relat Disord 2011;17:208–211.
3. Kedia S, Moro E, Tagliati M, Lang AE, Kumar R. Emergence of restless legs syndrome during subthalamic stimulation for Parkinson disease. Neurology 2004;63:2410–2412.
4. Driver-Dunckley E, Evidente VG, Adler CH, et al. Restless legs syndrome in Parkinson's disease patients may improve with subthalamic stimulation. Mov Disord 2006;21:1287–1289.
5. Marques A, Fantini ML, Morand D, et al. Emergence of restless legs syndrome after subthalamic stimulation in Parkinson's disease: a dopaminergic overstimulation? Sleep Med 2015;16:583–588.

Author response: Deep brain stimulation improves restless legs syndrome in patients with Parkinson disease

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I thank Marques et al. for the interest in our study,[1] the clarification, and the thoughtful comments. Their study reported that patients with Parkinson disease (PD) with postoperative emergence of restless legs syndrome (RLS) had higher preoperative and postoperative dopamine agonist (DA) doses, and had a lower percentage of DA reduction after deep brain stimulation (DBS),[2] not "more significant reduction" as misinterpreted from our article.[1] This underscores the controversy surrounding this issue since there are reports of RLS emergence after DBS in patients with PD with greater reduction of dopaminergic therapy,[3] less reductions,[2] and no correlation with degree of medication reduction.[1] We thank Marques et al. for bringing attention to the complexity of this topic, and the call for further research in this area that might shed light on the effects of DBS on RLS symptoms and the mechanisms of RLS in general.

1. Klepitskaya O, Liu Y, Sharma S, et al. Deep brain stimulation improves restless legs syndrome in patients with Parkinson disease. Neurology 2018;91:e1013–e1021.
2. Marques A, Fantini ML, Morand D, et al. Emergence of restless legs syndrome after subthalamic stimulation in Parkinson's disease: a dopaminergic overstimulation? Sleep Med 2015;16:583–588.
3. Driver-Dunckley E, Evidente VG, Adler CH, et al. Restless legs syndrome in Parkinson's disease patients may improve with subthalamic stimulation. Mov Disord 2006;21:1287–1289.

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Editors' note: Mystery Case: Bilateral temporal crescent sparing after cardiac arrest

In their Mystery Case, Lindegger et al. presented the unique visual fields of a middle-aged man who complained of tunnel vision 6 months following a cardiac arrest. One challenge to the case was the appearance of the Goldmann perimetry findings, which are nearly the opposite of what would be seen in a lesion affecting the optic chiasm. Instead of having bitemporal loss of vision, the patient had sparing of the most peripheral temporal visual fields with additional sparing of the binocular central fields. Among the 394 survey responses to the case, only 22% of readers correctly localized the lesion to the bilateral calcarine cortex (38% localized the lesion incorrectly to the chiasm). Dr. Rosenberg, a neuro-ophthalmologist, appropriately considers these Goldmann visual field findings controversial, and even contrary to our current understanding of visual pathways. Sparing of the far peripheral nasal fields and crossing of the vertical meridian peripherally would be untenable for bilateral occipital lobe lesions and probably explains why the survey participants had great difficulty localizing the lesion. Lindegger et al. respond that the manual perimetry was performed by an experienced technician, but that some of these atypical visual field findings may have resulted from the fixation losses and cognitive impairment that follows cardiac arrest.

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Reader response: Mystery Case: Bilateral temporal crescent sparing after cardiac arrest

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I was surprised that the Mystery Case by Lindegger et al.[1] was published in the Resident & Fellow Section. Although there is clear temporal crescent sparing, there are also smaller—but equally clear—residual nasal crescents in both the superior and inferior fields that cross the vertical meridian. The case seems to teach that the anterior most portion of the calcarine cortex has not only peripheral temporal fibers, but also fibers serving a peripheral nasal crescent.

A critically important teaching point regarding postchiasmal deficits is the presence of a disparity in the deficit on one side of the vertical meridian compared to the other. There was no "notch" in either the peripheral or central aspects of the field in either eye. Another important teaching point is that lesions involving the occipital pole, as opposed to those involving the radiations, result in either central scotomas or central sparing that is typically very congruous. The central changes shown are clearly noncongruous in their relationship to the vertical.

This field goes against major teaching points of hemianopsia: the presence of a notch at the vertical meridian, the congruity of occipital pole lesions, and the idea of a spared temporal not-nasal crescent.

1. Lindegger DJ, Helfenstein M, Job O, Pless M. Mystery Case: Bilateral temporal crescent sparing after cardiac arrest. Neurology 2018;90:1035–1036.

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Author response: Mystery Case: Bilateral temporal crescent sparing after cardiac arrest

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My coauthors and I appreciate Dr. Rosenberg's comments on our article,[1] and we agree in principle. The scope of the case report did not allow for the elaboration of the additional teaching points that Dr. Rosenberg elucidates, namely that there may be a contribution by the so-called peripheral nasal crescent, and that central sparing is typically very congruous. The visual field published is a copy of the exact visual field as it was obtained in the acute clinical setting.[1] It depicts precisely what was extracted from the patient shortly after the original CNS insult by an experienced perimetrist. The patient was not cognitively entirely fit at the time due to the insult itself, which limits the precision of perimetry. However, it demonstrates the main principles offered as teaching points in the short communication. Regarding the comment pertaining to the notch, it is difficult to demonstrate such a finding even by experienced perimetrists, let alone in patients coming out of the intensive care unit.

1. Lindegger DJ, Helfenstein M, Job O, Pless M. Mystery Case: Bilateral temporal crescent sparing after cardiac arrest. Neurology 2018;90:1035–1036.

CORRECTION

Deep brain stimulation improves restless legs syndrome in patients with Parkinson disease

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In the article "Deep brain stimulation improves restless legs syndrome in patients with Parkinson disease" by Klepitskaya et al.,[1] first published online August 15, 2018, the article included a misinterpretation of the reference to Marques et al.[2] The Marques et al.[2] study reported that patients with Parkinson disease with postoperative emergence of restless legs syndrome had higher preoperative and postoperative dopamine agonists (DA) doses, and had a lower percentage of DA reduction after deep brain stimulation, not "more significant reduction," as misinterpreted in the Klepitskaya et al.[1] article. Misinterpretation of the reference did not change the data, the interpretation of the data, or the conclusions of the article. The authors regret the error.

References

1. Klepitskaya O, Liu Y, Sharma S, et al. Deep brain stimulation improves restless legs syndrome in patients with Parkinson disease. Neurology 2018;91:e1013–e1021.
2. Marques A, Fantini ML, Morand D, et al. Emergence of restless legs syndrome after subthalamic stimulation in Parkinson's disease: a dopaminergic overstimulation? Sleep Med 2015;16:583–588.

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Deep brain stimulation improves restless legs syndrome in patients with Parkinson disease

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