Letters to the Editor

NEUROSURGICAL FORUM

Transfalcine approach

TO THE EDITOR: I read with interest the article by Bohnstedt et al.¹ (Bohnstedt BN, Kulwin CG, Shah MV, et al: Posterior interhemispheric transfalcine transprecuneus approach for microsurgical resection of peritrial lesions: indications, technique, and outcomes. *J Neurosurg* [epub ahead of print May 1, 2015. DOI: 10.3171/2015.3.JNS14847]). The authors used a transfalcine approach for a variety of paratrigonal lesions that included meningiomas and arteriovenous malformations. The success that they achieved in treating these relatively complex lesions by the discussed approach is a compliment to their microsurgical skills. I wish to inform the authors and the readers of the *Journal* that my initial description of the transfalcine approach in 1995 included a paratrigonal tumor.⁵ I also discussed in another publication the use and advantages of a transfalcine approach for a contralateral hemispheric arteriovenous malformation.^{2–4} The authors have erroneously mentioned that my technical description included only a frontal brain tumor.¹

The article and commentary by Dr. Heros with the response by Dr. Cohen-Gadol summarize the various positive and negative issues about the approach.^{1,6} As discussed, the transfalcine approach to contralateral hemispheric lesions has the advantage of retracting a normal hemisphere over a tumor-affected brain. The approach to the deep-seated lesion is also direct and in cases with trigonal meningiomas, the tumor vascularity that arises from the depth of the lesion can be approached early in the operation. The feeding vessels of the arteriovenous malformation can sometimes be relatively more easily exposed by the transfalcine corridor, avoiding the draining veins that may be encountered early in an ipsilateral interhemispheric approach. However, as my experience in treating such lesions has grown over the years, I have realized that large lesions, hypervascular tumors, and arteriovenous malformations that will need prolonged microsurgical dissection can be more effectively approached by an ipsilateral interhemispheric approach. Retraction of the normal brain with visual pathways can sometimes affect the function of these fibers. Surgical manipulation of the trigonal region can by itself affect the visual pathways primarily or following surgery. As cautioned by Dr. Heros, although rare, danger to visual pathways on both sides must be kept in mind when using a transfalcine approach. More importantly, the distance of the trigonal region increases by a

few critical and probably defining millimeters by a contralateral approach. Considering these negative issues, I have used this approach only infrequently after my description of the technique 20 years ago.

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DISCLOSURE

The author reports no conflict of interest.

References

1. Bohnstedt BN, Kulwin CG, Shah MV, Cohen-Gadol AA: Posterior interhemispheric transfalcine transprecuneus approach for microsurgical resection of peritrial lesions: indications, technique, and outcomes. *J Neurosurg* [epub ahead of print May 1, 2015. DOI: 10.3171/2015.3.JNS14847]
2. Goel A: Bilateral parafalcine approach for arteriovenous malformations located in the interhemispheric fissure, in Kobayashi S, Goel A, Hongo K (eds): *Neurosurgery of Complex Tumours and Vascular Lesions*. New York: Churchill Livingstone, 1997
3. Goel A: Commentary on “The contralateral transfalcine transprecuneus approach to the atrium of the lateral ventricle: operative technique and surgical results.” *Neurosurgery* **11** (Suppl 2):110–118, 2015
4. Goel A: Transfalcine approach. *J Neurosurg* **113**:1126, 2010 (Letter)
5. Goel A: Transfalcine approach to contralateral hemispheric tumor. *Acta Neurochir (Wien)* **135**:210–212, 1995
6. Heros RC: Editorial. Contralateral transfalcine approach for peritrigonal lesions. *J Neurosurg* [epub ahead of print May 1, 2015. DOI: 10.3171/2014.10.JNS142002]

Response

We appreciate the clarifications and corrections mentioned by Professor Goel. We also apologize for inadvertently not mentioning his numerous contributions in our article.

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Is one large target better than two?

TO THE EDITOR: We have read the article by Kilbane et al.³ (Kilbane C, Ramirez-Zamora A, Ryapolova-Webb E, et al: Pallidal stimulation for Holmes tremor: clinical outcomes and single-unit recordings in 4 cases. *J Neurosurg* 122:1306–1314, June 2015) with great interest. Treatment of Holmes tremor (HT) is challenging, and there have been a series of reports concerning the outcomes of stereotactic surgery for this debilitating disorder. As the authors mentioned, single-lead thalamic deep brain stimulation (DBS) may not sufficiently resolve HT. Hirai et al. first reported that a larger lesion of the thalamotomy was more effective than a smaller lesion,² and given that finding, Yamamoto et al. reported the first case in which the unilateral dual-lead technique was successfully used for severe essential tremor.⁵ Subsequently, Foote and colleagues applied this technique for the treatment of debilitating tremors due to multiple sclerosis and traumatic brain injury.¹

The advantage of the unilateral dual-lead thalamic stimulation technique is that dual DBS leads can modulate a larger network of the basal ganglia circuits than single-lead DBS. Since the ventralis oralis (VO) nucleus is a pallidal-receiving area, DBS of the VO thalamic nucleus may have an effect similar to that of globus pallidus internus (GPi) DBS.⁴ However, Kilbane et al. did not observe an additional benefit of thalamic DBS beyond single GPi stimulation in 2 cases, even though previous reports have demonstrated an “add-on” effect of the second lead in cases treated with dual-lead DBS. Thus, one may suspect that the thalamic DBS leads were suboptimally placed, as the second DBS lead trajectory is limited by the first lead placement in rescue DBS cases, particularly in those with an ex vacuo change due to structural brain damage or severe atrophy.

The authors showed that the firing rate of the GPi cells in patients with HT was lower than that in the Parkinson's disease cohort, and they mentioned that this finding was consistent with “nonparkinsonian disorders.” Nevertheless, this does not mean that HT and nonparkinsonian disorders such as dystonia are characterized by similar problems in the GPi. In fact, there is a critical difference between HT and other nonparkinsonian disorders with respect to responsiveness to DBS. For example, both HT and secondary dystonia are associated with structural damage to the brain, but various studies have shown that DBS is ineffective in most secondary dystonia cases. Moreover, the movement disorder phenomenology from the same lesion may be variable among cases. Therefore, although the authors' data are interesting, it may be difficult to specifically underpin the etiology of HT based on single-unit recording data.

The outcomes of the presented case series are promising, and there is no doubt that this paper is an important contribution to the field. However, the etiology of HT remains unclear, and further accumulation of clinical cases is warranted to test the reproducibility of the favorable outcomes of GPi DBS for the treatment of HT. We advocate for further studies to find biomarkers to predict which HT patients are most likely to show a favorable response to DBS. Such data may help clinicians to select the best DBS target for each HT case.

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DISCLOSURE

The authors report no conflict of interest.

References

1. Foote KD, Seignourel P, Fernandez HH, Romrell J, Whidden E, Jacobson C, et al: Dual electrode thalamic deep brain stimulation for the treatment of posttraumatic and multiple sclerosis tremor. *Neurosurgery* 58 (4 Suppl 2):ONS-280–ONS-286, 2006
2. Hirai T, Miyazaki M, Nakajima H, Shibasaki T, Ohye C: The correlation between tremor characteristics and the predicted volume of effective lesions in stereotaxic nucleus ventralis intermedius thalamotomy. *Brain* 106:1001–1018, 1983
3. Kilbane C, Ramirez-Zamora A, Ryapolova-Webb E, Qasim S, Glass GA, Starr PA, et al: Pallidal stimulation for Holmes tremor: clinical outcomes and single-unit recordings in 4 cases. *J Neurosurg* 122:1306–1314, 2015
4. Morishita T, Foote KD, Haq IU, Zeilman P, Jacobson CE, Okun MS: Should we consider vim thalamic deep brain stimulation for select cases of severe refractory dystonic tremor. *Stereotact Funct Neurosurg* 88:98–104, 2010
5. Yamamoto T, Katayama Y, Fukaya C, Oshima H, Kasai M, Kobayashi K: New method of deep brain stimulation therapy with two electrodes implanted in parallel and side by side. *J Neurosurg* 95:1075–1078, 2001

Response

We appreciate the comments of Dr. Morishita and colleagues on our recent paper describing clinical outcomes and the analysis of intraoperative single-unit recordings in 4 patients with HT successfully treated with pallidal DBS. Given only a handful of published cases, with various underlying pathophysiologies, it is not possible to determine the best treatment for all patients suffering from HT. Future studies may help shed light on optimal target selection in this challenging disorder, but our outcomes are certainly promising.

Dr. Morishita and colleagues bring up the important issue of how to best modulate the motor circuit to improve HT, a condition with various etiologies that is often difficult to treat. Our experience suggests that single-lead unilateral ventralis intermedius (VIM) nucleus stimulation seems inadequate to control the hyperkinetic movements of HT, whereas unilateral GPi stimulation provides better relief. We only evaluated the effect of VO nucleus stimulation in 1 patient, and in that case the patient did not obtain additional clinical benefit, as has been reported in post-traumatic tremor.² It is interesting to hypothesize that thalamic VO stimulation may have an effect similar to that of GPi DBS since they are part of the same circuit; however, this was not observed in our 1 case.

Certainly, if a condition can be adequately treated with 1 target, then the surgical risk, programming, device complications risk, and battery drain are simplified. In our series, 1 GPi target was better than 2 and resulted in greater clinical efficacy than most others have shown using single

VIM or dual VIM, VO anterior, or VO posterior leads.^{1,3–5} Dr. Morishita and colleagues suggest that implanting rescue leads can be less precise and may help explain poor outcomes; however, in our series, a single GPi lead produced substantial benefit in 3 patients and did not require the placement of rescue leads. One lead is certainly better than 2 if that is all that is required, but we agree that it would be very helpful to be able to identify which type of HT patients might benefit from various target choices using imaging or physiological biomarkers rather than relying purely on phenotype. Our pallidal single-unit recordings cannot be generalized to all nonparkinsonian movement disorders, but they do help support the role of this circuit in HT, which was readily modulated with DBS.

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References

1. Diederich NJ, Verhagen Metman L, Bakay RA, Alesch F: Ventral intermediate thalamic stimulation in complex tremor syndromes. **Stereotact Funct Neurosurg** **86**:167–172, 2008
2. Foote KD, Seignourel P, Fernandez HH, Romrell J, Whidden E, Jacobson C, et al: Dual electrode thalamic deep brain stimulation for the treatment of posttraumatic and multiple sclerosis tremor. **Neurosurgery** **58** (4 Suppl 2):ONS-280–ONS-286, 2006
3. Kudo M, Goto S, Nishikawa S, Hamasaki T, Soyama N, Ushio Y, et al: Bilateral thalamic stimulation for Holmes' tremor caused by unilateral brainstem lesion. **Mov Disord** **16**:170–174, 2001
4. Pahwa R, Lyons KE, Kempf L, Wilkinson SB, Koller WC: Thalamic stimulation for midbrain tremor after partial hemangioma resection. **Mov Disord** **17**:404–407, 2002
5. Samadani U, Umemura A, Jaggi JL, Colcher A, Zager EL, Baltuch GH: Thalamic deep brain stimulation for disabling tremor after excision of a midbrain cavernous angioma. Case report. **J Neurosurg** **98**:888–890, 2003

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