Neurosurgical forum

Letters to the editor

Shaving

TO THE EDITOR: It is sad to see an academic neurosurgeon abandoning scientific standards in support of his conclusions in the pages of this prestigious journal. Recently, Dr. Ken Winston⁸ (Winston KR: Editorial. Neurosurgery and shaving. *J Neurosurg* 115:669, October 2011) advocated abandoning the shaving of neurosurgical sites based on the work published by Broekman et al.² (Broekman MLD, van Beijnum J, Peul WC, et al: Neurosurgery and shaving: what's the evidence? A review. *J Neurosurg* 115:670–678, October 2011), analyzing infection rates associated with shaving. It was also discouraging to see these latter authors, in their editorial response, change their original, sound conclusion calling for careful study to one that advises neurosurgeons “to stop this routine.” There are no data given to support any danger from routine shaving of cranial sites. In fact, Broekman and colleagues cite proven concerns with increased surgical time in patients without shaving. They cite a single Class I study showing a decrease in the infection rate for spinal cases only (which cannot be generalized to cranial cases). That study did not show a statistically significant difference and was biased in that the unshaved group had more “complex cases.”¹ The rate of antibiotic use or additional care received because of that complexity was not accounted for by the authors, and the results have yet to be reproduced. The only Class II or better studies evaluated by Broekman et al. and showing possible increased cranial infection with shaving were those comparing patients shaved “preop on ward” with patients unshaved in the operating room. This is similar to the faulty studies used by panels to justify a shaving ban everywhere based on comparisons between clipping the morning of surgery and shaving on the ward the night before surgery.⁴ Clearly, a valid study requires a comparison between shaving at the moment of surgery and unshaven surgery in specific and defined groups.

Unfortunately, neither Winston nor Broekman and colleagues mention the results of compliance with measures of the Surgical Care Improvement Project (SCIP) in which infection rates increased as compliance with the SCIP guidelines rose.⁷ A similar study by the Agency for Healthcare Research and Quality (AHRQ) found that compliance with SCIP guidelines on shaving, among other things, was not beneficial to patients.⁶ After banning razors from the operating room to ensure “appropriate hair removal” (and other measures), infection rates were up nearly 20% in 2 years just as compliance was also up about 15%–20% (Fig. 2 of the *JAMA* article⁷).⁶ Somehow Dr. Winston found the need to “gently” chastise Broekman et al. for using “mollifying language” in their analysis to indicate that “uncertainty” remained and

that “properly designed studies are needed.” Dr. Winston properly points out that such a study would need 3000–5000 patients for acceptable statistical power. Yet, he then argues that the type of data provided by Broekman et al. for “any other subject” would induce doctors to abandon a practice. I do not see that I would abandon, for instance, treating vasospasm based on the very limited data presented by Dr. Broekman and colleagues.

Sadly, all of this is a result of continued government and corporate intrusion into the practice of medicine and our unwillingness, as a profession, to reject faulty assertions and substandard studies as a basis for interference in our profession—this as even more published studies are showing that compliance with such standards does not benefit patients in any way³ and that harm is likely.⁵ As accountable care organizations, capitation, pay for performance, bundling, shared savings programs, value-based purchasing, and similar programs grow, doctors will be coerced to lower their standards and provide substandard care to serve the interests of politicians and corporations—often with their own colleagues supporting this agenda.

As a surgeon, I like shaving hair. I like having a naked scalp that gives me more options for an incision and better visualization for planning the surgery. I like removing hair quickly with cheap and new razors so that I can focus on the important parts of the case. I like to avoid slow removal of hair with an expensive electric clipper that may not have been cleaned well, may need batteries, and may not work that day. I like closing quickly without hair getting in the wound or the drapes coming off and creeping into unclean areas. I like being able to rapidly remove hair in the middle of the night on a trauma patient's head matted with bloody hair. I like knowing that all the staples or stitches will be removed and not hidden by some hair and that the dressing will remain postoperatively. In short, I like my razor. If my patients want a surgeon who doesn't shave with a razor, there are others in the community who will satisfy that need. There are no data showing that I am hurting my patients by shaving, and there are sound data showing that I may be hurting them by not shaving.⁷ There is only one thing that will make me stop insisting on the razor: sound clinical data from a valid study demonstrating a benefit to my patients. Until then, let's not lower our standards and cajole our colleagues to change their valid scientific conclusions. Congratulations to Dr. Broekman and associates for calling for more studies; you should stick with your first and sound conclusion.

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Disclosure

The author reports no conflict of interest.

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RESPONSE: We respect Dr. McKalip's preference for using a razor, as it is ultimately the individual surgeon's duty to weigh all pros and cons to provide optimal care to his or her patients. However, we disagree with the claim that there are sound data showing we may be hurting patients by not shaving. The study by Stulberg and colleagues¹ that Dr. McKalip is referring to demonstrates that compliance with SCIP measures is associated with a lower risk of postoperative infection when these SCIP measures are aggregated into a single composite score (OR 0.85, 95% CI 0.76–0.95). The observed beneficial effect appears to be entirely mediated by SCIP measures infection (Inf)-1 through Inf-4, all of which have point estimates of an OR < 1 (although they are not significant on their own). Importantly, SCIP measure Inf-6 (for appropriate hair removal) is entirely consistent with the null hypothesis of no effect (OR 1.00, 95% CI 0.85–1.19). Without commenting on the fact that cranial surgeries were not even included (according to their Table 2), this large study of more than 400,000 patients provides no compelling evidence that shaving confers a protective effect or, conversely, that not shaving may be harmful. We stick to our original recommendation that, until a carefully designed, well-powered randomized controlled trial demonstrates a significant benefit, shaving should be considered a clinical intervention and as such—all other things being equal—should be avoided. We realize this may not always be feasible in clinical practice.

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RESPONSE: I stand by the excellent review published by Broekman et al. on the safety of not shaving for neurosurgical operations, by my comments on that article, and by the many publications from around the world on the subject. The supporting evidence and the logic are widely published and, in my opinion, compelling. Listed in the top Centers for Disease Control and Prevention recommendations to prevent surgical site infections is the following: “Do not remove hair at the operative site unless it will interfere with the operation; do not use razors.”¹

Neurosurgeons, nurse practitioners, nurses, and infection preventionists, when wondering why some surgeons continue to shave their patients, can now remember the primal scream in defense of that tradition so very clearly explained in Dr. McKalip's final paragraph.

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Incidental low-grade gliomas

TO THE EDITOR: We enjoyed reading the recent article by Potts et al.⁹ (Potts MB, Smith JS, Molinaro AM, et al: Natural history and surgical management of incidentally discovered low-grade gliomas. Clinical article. *J Neurosurg* 116:365–372, February 2012).

Supratentorial diffuse low-grade gliomas (LGGs) in adults are rarely discovered incidentally on brain imaging, although their detection will probably increase as access to brain imaging broadens worldwide.⁶ In the present study, Potts and colleagues analyzed the effects of early resection on incidental LGGs. Their results agreed with those of an earlier study focusing on long-term outcomes in patients harboring an incidental LGG, which was conducted by our French glioma study group (Réseau d'Étude

des Gliomes, France). These 2 studies provided evidence that incidental LGGs grow, become symptomatic, and progress to malignancy, supporting the idea that they can be managed as one would a symptomatic LGG. Moreover, Potts and colleagues elegantly demonstrate with volumetric measurements that incidental LGG removal is associated with a greater extent of resection (possibly explained by a smaller volume and predominant noneloquent location) and better subsequent overall survival.

Based on these results, we agree with the authors that maximal safe resection with functional intraoperative corticosubcortical brain mapping must be proposed to each patient with an incidental LGG, before any clinical signs appear, as soon as radiological progression is demonstrated on MRI follow-up. This early surgical management may help to optimize the resection and provide the ideal situation to achieve a functionally based “supratotal” resection.¹¹ Indeed, because of the shorter tumor history and reduced tumor volume, the cerebral infiltration by isolated tumor cells that exists beyond MRI-defined abnormalities is possibly less than in a symptomatic LGG.⁸

As suggested by the authors, we would also recommend demonstrating radiological progression on repeated control MRI studies before any treatment. Potts and colleagues quantified the radiological tumor growth in 8 patients and presented volumetric growth (Fig. 1). We wonder why the authors did not convert volumetric growth rates in terms of diameter growth rates, as it was previously proposed for LGG.^{2–4,7} If we agree that 3D volume segmentation offers better measurement reproducibility¹⁰ and that it must be performed when analyzing the postoperative residual tumor,¹ we think that volumes should then be converted to diameters (see below). Volumetric growth rate is indeed a combination of 2 independent factors: initial tumor volume and tumor growth rate. Thus, the same volume increase may represent both large tumors with low growth rates and small tumors with higher growth rates.⁵ We have converted the volumetric growth rates in terms of diameter growth rates (the so-called velocity of diametric expansion [VDE]) by using the formula $D = (2 \times V)^{1/3}$, where V is the volume given by the authors from the 3D tumor segmentation. All LGGs under study present a positive VDE, demonstrating a linear radiological growth at a mean VDE of about 3 mm/year, which is in the known range for LGG.^{1,5} As an example, we clearly see that Patients 1 and 7 pres-

ent the same VDE, suggesting the same behavior (Fig. 1, right graph), although the tumor in Patient 1 was diagnosed at a later stage and with a larger size.

Thus, we propose, for each patient harboring an LGG, monitoring radiological growth rates by using the diameter derived from the tumor volume. As soon as radiological tumor growth is reliably estimated during 3–9 months of follow-up, as illustrated in the present examples, a maximal safe resection should be proposed to patients harboring an incidental LGG.

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Disclosure

The authors report no conflict of interest.

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Fig. 1. Graphs demonstrating tumor volume progression with time. Pt. = patient.

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RESPONSE: We thank Drs. Pallud and Mandonnet for both their comments on our study and their contributions to the field of glioma surgery. The main objective of our study was to compare the characteristics of incidental LGGs with those of symptomatic LGGs. Our results confirmed earlier findings by Drs. Pallud, Mandonnet, and others that patients with incidental gliomas have smaller tumor volumes and improved extents of resection, which are associated with improved survival. As with authors of previous studies from our institution, we were interested in examining the extent of resection among our incidental LGG cohort and therefore performed volumetric analysis in these patients. Our cohort included a small subset of patients with multiple preoperative MR images, and we believed it would be of general interest to glioma surgeons to report on the natural growth of these incidental gliomas. We therefore included Fig. 1 in our study, which described the volumetric growth of this subset of gliomas.

While we are not aware of studies directly comparing volumetric measurements with diametric measurements in gliomas, we believe that volumetric analysis remains the most accurate and reliable method for analyzing the extent of resection, which was our primary interest in this study. However, we agree that diametric expansion may be a better way to follow tumor growth and is certainly easier for practitioners to perform. It is therefore reasonable to propose that future studies of glioma growth utilize diametric measurements.

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Cerebrospinal fluid hypovolemia

TO THE EDITOR: We read with great interest the article by Takeuchi et al.⁶ (Takeuchi N, Horikoshi T, Kinouchi H, et al: Diagnostic value of the optic nerve sheath subarachnoid space in patients with intracranial hypotension syndrome. Clinical article. *J Neurosurg* **117**:372–377, August 2012) and would like to address some comments.

The results of the current study are consistent with ours, which was published last year.¹ In a cohort of 10 patients suffering from post-dural puncture headache (PDPH), we showed an increase in optic nerve sheath diameter (ONSD) measured by ultrasonography after epidural blood patch (EBP) success in 9 patients. In the patient with failure of EBP, we observed only a small increase in ONSD over time. Whereas Takeuchi et al. found a decrease in ONSD before treatment, we haven't been able to show a decrease before EBP. This may be explained by the different pathophysiological characteristics between PDPH and spontaneous intracranial hypotension syndrome. Patients suffering from spontaneous intracranial hypotension are likely to present with more severe intracranial hypotension than patients suffering from PDPH, probably in relation to difficulties in diagnosing intracranial hypotension syndrome, leading to long-term progression of the disease.

In the recently published studies in which ONSD has been used as an estimate of intracranial pressure, the ONSD has always been measured 3 mm behind the globe.^{2,3} This allows precise and stable measurement. The method used in Takeuchi's study (the mean of the two measurements of ONSD, one just behind the eyeball and the other 3.5 mm behind the first slice) is questionable, because it has been clearly shown that the architecture and distribution of arachnoid trabeculae, pillars, and septa in the subarachnoid space of the human optic nerve is heterogeneous along the nerve.⁴

Finally, we insist on the fact that specialized ONSD MRI should include calibrated MRI devices such as high magnetic field (at least a 3-T magnet) and ultrafast single-shot T2-weighted sequences to allow shortened recording time, attenuated motion artifacts, and higher spatial resolution.⁵

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Disclosure

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RESPONSE: We appreciate Drs. Dubost et al. for their great interest to our article. They found an increase of ONSD measured by ultrasonography after EBP and speculated that this might be caused by an immediate increase of epidural pressure due to the temporary tamponade effect of EBP. This is a feasible finding; however, we cannot comment on this issue because our study evaluated the ONSD from 1 to 6 months after treatment. In this duration no tamponade effect on the increase of ONSD could be expected, and therefore we concluded that normalization due to the sealing effect of EBP would be the main cause. In other words, a decrease of ONSD before treatment is pathological, and indeed many of our patients showed disappearance of CSF in the optic nerve sheath, whereas it is usually found in healthy individuals.^{1,2}

In our study, the mean value of two slices was used to reduce measurement variation, because the MRI slices were not consistently located among the patients with respect to the eyeballs. We agree that fast MRI sequences obtained using a high magnetic field system shorten recording time and provide better images without motion artifact; however, eye position is still a problem. The optic nerve close to the eyeball bends and obliquely passes the imaging plane when the eye position is upward or downward during examination. We hope that accumulation of knowledge in this field will clarify the nature of CSF hypovolemia syndrome.

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Diffusion tensor imaging and traumatic brain injury

TO THE EDITOR: We read with great interest the article by Matsushita et al.² (Matsushita M, Hosoda K, Naitoh Y, et al: Utility of diffusion tensor imaging in the acute stage of mild to moderate traumatic brain injury for detecting white matter lesions and predicting long-term cognitive function in adults. Clinical article. **J Neurosurg** 115:130–139, July 2011). The authors investigated where white matter injury following mild to moderate traumatic brain injury (TBI) is specifically located using diffusion tensor imaging (DTI) in the acute disease stage, and they examined the relationship between the severity of the white matter lesion on DTI in the acute stage of TBI and future cognitive function in the chronic disease stage. They compared fractional anisotropies (FAs) between patients and controls using the regions of interest method in 8 different areas. Their analysis revealed that there were significant relationships between cognitive dysfunction and FA reductions in the splenium and frontal white matter in the acute stage of mild to moderate TBI. We agree with the findings of the authors and wish to provide further comment on this issue. The cingulum bundle (CB) is a white matter tract that underlies the cingulate cortex, and all connections entering and exiting the cingulate gyrus pass through this bundle. These pathways include projections between prefrontal and parahippocampal cortices and projections to the median raphe nucleus that terminate in the dorsal hippocampus. The CB is involved in attention, emotions, spatial orientation, and memory. Recently, several authors reported that the CB is susceptible to TBI because of its long coursing nature, and this injury is detectable by DTI in studies involving mild to severe TBI in both an acute and chronic phase.^{1,3,4} However, Matsushita et al. did not investigate the CB. Further investigation of the relationship between cognitive function and DTI findings, especially in the CB during an acute stage of TBI, may provide additional insight into determining a better prognostic factor for long-term cognitive dysfunction.

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RESPONSE: We thank Dr. Takeuchi et al. for their interest in our paper and for giving us the opportunity to continue the debate.

As they indicated, we investigated FA of the CB in 11 patients and analyzed the relationship between FA values of the CB in the acute stage and the results of neuropsychological testing in the chronic stage. Simple linear regression analysis demonstrated a statistically significant relationship between FA in the CB and the value of P300 latency ($r = 0.71$, $p = 0.016$ [Fig. 1]). Regarding the total intelligence quotient, Wisconsin Card Sorting Test–Keio version, and Trail Making Test, however, no significant relationship was found. The P300 component of event-related potential was reported to indicate information processing speeds, attentional capacity, and attention.¹ Considering that the CB is involved in attention, emotions, spatial orientation, and memory, as pointed out by Dr. Takeuchi et al., these findings seem to be reasonable.

However, there are several confounding factors such as age, education, severity of trauma, and FA values of other regions of white matter. A larger number of patients and further investigation with multivariate analysis are needed to clarify these points.

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Fig. 1. P300 latency in the chronic stage is significantly associated with FA of the CB in the acute stage of 11 patients with TBI. A significant negative linear relationship is observed between FA of the CB and P300 latency in TBI patients, with the line defined by $P300 \text{ latency (msec)} = -1976.2 \times FA + 1317.3$ (correlation coefficient $r = 0.71$, $p = 0.016$).

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Muscle and nerve transfer in tetraplegia

TO THE EDITOR: With astonishment we read in your journal the recent case report by Mackinnon et al.⁹ (Mackinnon SE, Yee A, Ray WZ: Nerve transfers for the restoration of hand function after spinal cord injury. Case report. *J Neurosurg* 117:176–185, July 2012).

A 71-year-old male tetraplegic patient classified as International Classification for Surgery of the Hand in Tetraplegia (ICSHT) Group 5 with severe finger joint stiffness received a selective bilateral brachialis to anterior interosseous nerve transfer 22 months after spinal cord injury (SCI). After 15 months of intensive physiotherapy this produced “some” hand function. The authors declare this “the first reported case of thumb and finger flexor reinnervation after an SCI” and “[w]hile the results in this patient are usually modest, due to the severe joint stiffness, his function has improved significantly with his ability to feed himself.” This conclusion deserves some comments and we would not recommend this approach to improve hand function in similar tetraplegic patients.

The brachialis branch has been used by Benassy and Robart¹ before 1966 as an axon donor to restore median nerve function in tetraplegia, and proved “very useful for the patient to type with the left hand, eat nearly alone, light a match and drive his wheelchair.” Krasuski and Kiwerski⁸ in 1991 reported 32 good results in 42 patients with improved grasp, mostly in young patients (younger than 25 years) who underwent operation early (3–6 months after their accident). Notably, all results in patients older than 40 years of age and in 5 of 7 patients operated beyond 10 months after trauma were classified as “bad” (no improvement). Those authors transferred the brachialis branch onto the whole median nerve, with likely reinnervation of sensory by motor fibers and thus noteworthy axon loss. In contrast, Mackinnon et al. applied selective neurotization of the anterior interosseous nerve by using intraoperative stimulation of donor and recipient fascicles. However, their outcome seems regrettably weak—the patient is only able to grab a cookie and hold a (hollow) ball, but lacks strong pinch or grasp, which is critical to regain independence from other help.

A tendon transfer in this elderly patient is likely to have produced a much stronger and more useful grip within a few months.⁶ The average grip strength was 2 kg in a recent meta-analysis of 377 pinch reconstructions in 23 studies,⁷ including many patients with fewer potentially transferable muscles than this Group 5 patient, who has at least the brachioradialis, extensor carpi radialis longus, and pronator teres muscles ready for transfer, plus the

brachialis muscle if prolonged with tendon graft.² Why leave so many transferable muscles completely unutilized and not transfer them to reliably and rapidly enhance the patient's feeble grip?

Instead, the authors envision a further 2-stage nerve transfer of the supinator or brachioradialis branches to reinnervate ulnar intrinsic hand muscles, anticipating at least an additional 12 months. Should a nerve transfer performed using long nerve grafts in a 71-year-old patient with final delay of not less than 49 (22 + 15 + 12) months really be preferred over established methods for immediate intrinsic reconstruction, such as House or Zancolli plasty, which could be further activated by remaining motor muscles, for example the pronator teres?

We agree that selective axon transfers promise fascinating prospects in tetraplegia and compare favorably to muscle transfers; for example, if they shorten or facilitate functional recovery in elbow extension restoration as suggested by Bertelli and colleagues^{3,4} by using the teres minor or brachialis branch for triceps neurotization. New options may arise in cases not amenable to conventional tendon transfers, such as by restoring wrist extension-driven grip function in patients with ICSHT Group 0 by transfer of the brachialis onto the extensor carpi radialis longus muscle branch.⁵ It is still unclear whether such neural reconstructions remain reasonable even years later, yet regarding the few published cases it seems advisable that they should be done preferably in young patients, as early as possible, and only over short regeneration distances.

In conclusion, we should combine nerve and muscle transfers into new algorithms depending on a careful evaluation of individual factors, such as age, extent of paralysis, functional needs, or time delay since SCI (Table 1). If we use only one method, the patient loses the benefits of the other. Uniting the advantages of both techniques will enable us to maximize arm and hand function in these severely handicapped patients as fast as possible.

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Disclosure

The authors report no conflict of interest.

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RESPONSE: We appreciate the opportunity to respond to the comments by Drs. Fridén and Gohritz. As they point out, tendon transfer procedures are well established to improve hand function in tetraplegic patients. These procedures are time tested and produce reliable results that are realized more quickly than with nerve transfers. Although we agree that tendon transfers have a limited established role in the management of patients with SCI and tetraplegia, a growing body of literature and personal experience with nerve transfers suggests that nerve transfers are useful in patients with tetraplegia not only for

TABLE 1: Comparison of tendon and nerve transfer to restore upper-extremity function in patients with tetraplegia

Parameter	Tendon Transfer	Nerve Transfer
incisions	2–3 (donor & recipient site, harvest of interposition tendon graft)	only 1
immobilization	up to 3 mos	approximately 10 days (like any nerve suture)
motor muscle	weaker substitution, inferior muscle architecture	original muscle (or part of it; for example 1 triceps head only)
donor site morbidity	acceptable	same (or less if innervation partly remains)
interposition graft	tendon graft sometimes required	optimally none (if direct donor-to-recipient coaptation)
time until recovery of function	after splinting to secure suture, immediate activation possible	until muscle reinnervation after nerve regrowth from suture site (1 mm/day); usually many mos
impact of patient age	similar chances to obtain good outcomes	nerve regeneration severely impaired in elderly individuals
impact of time delay	none	theoretically none (but worse results reported in late transfers)

hand function, but also for improving elbow extension and thus their ability to self-transfer.¹⁻⁴ Nerve transfers offer a number of advantages over tendon transfers. They offer restoration of muscle groups without altering their biomechanics. Nerve transfers do not require prolonged immobilization and offer a much greater than 1:1 functional exchange. That is, in contrast to tendon transfers, the sacrifice of a single nerve function can provide for the reinnervation and functional recovery of multiple target muscles. Use of the brachialis nerve, as described in our case report, does not eliminate any future tendon transfer options because the brachialis is not a traditional donor for hand function.

Although Drs. Fridén and Gohritz correctly point out that this patient had only Grade 3 motor recovery at the time of publication and when examination videos were made, it is worth noting that the patient was only 15 months out from surgery. Since the time of publication and video assessment the patient has continued to gain both strength and functional control. This procedure was done in anticipation of later tendon transfers to augment any functional improvements gained by the nerve transfer. Given his continued improvement the patient has elected not to undergo further tendon transfer surgery, and has since written a book with his “feeble grip” hand. We agree with Drs. Fridén and Gohritz that nerve transfers should be offered in combination with tendon transfers, and given our growing experience in the treatment of tetraplegic patients, we offer a multidisciplinary

approach in which both nerve and tendon transfers are used. Similar to the case reports by Bertelli et al.,^{1,2,3} we report a novel option to augment the existing treatment strategies for tetraplegic patients, and we hope that this article serves to make readers of the *Journal of Neurosurgery* aware of this treatment option.

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