Letters to the Editor

NEUROSURGICAL FORUM

Trigeminal neuralgia

TO THE EDITOR: We are very interested in the article by Monteith et al.1 (Monteith SJ, Medel R, Kassell NF, et al: Transcranial magnetic resonance–guided focused ultrasound surgery for trigeminal neuralgia: a cadaveric and laboratory feasibility study. J Neurosurg 118:319–328, February 2013). Trigeminal neuralgia often induces severe pain and impairs quality of life, even with multimodal therapies. The trigeminal pain often results from offending artery compression to the trigeminal nerve. Monteith et al.1 performed a laboratory investigation in cadaveric specimens to clarify the feasibility of transcranial MR-guided focused ultrasound therapy for trigeminal neuralgia. They found that real-time MR thermometry demonstrated the heating effect of focused ultrasound on the trigeminal nerve with 10°C increments in temperature. Moreover, the heating effect may collaterally spread to the internal acoustic canal (IAC).

Their study provided solid evidence that MR-guided focused ultrasound surgery (MRgFUS) is capable of increasing focal heating of up to 18°C in the trigeminal nerve of a cadaveric specimen at the root entry zone. Importantly, MRgFUS did not produce a significant heating effect on the skull base and surrounding neural structures in no-pass regions. However, there are some minor concerns. First, MRgFUS cannot avoid an off-target effect producing a local temperature effect on adjacent crucial neurovascular structures, such as the brainstem and cranial nerves, as the authors observed with the vestibular nerve in the region of the IAC. Their study leads readers to ask about the damaging heating effects on vascular structures, which can induce thrombus formation and lead to ischemic injury of the brainstem. Their study delivered results in cadaveric human specimens, but not in living animal models. Therefore, the pain reduction effect still cannot be estimated from their study. We fully agree that in vivo studies are warranted to ensure the safety and efficacy of MRgFUS in treating trigeminal neuralgia.

DiSCLoSurE
The authors report no conflict of interest.

Reference

Response
We greatly appreciate the thoughtful comments from Dr. Zheng and colleagues. In our study, we investigated the potential use of transcranial MRgFUS treatment for trigeminal neuralgia. As Zheng et al. point out, there is concern regarding the collateral heating of adjacent structures, namely surrounding bone of the acoustic canal. Indeed, it was this concern that prompted specific design aspects of our investigation and the development of strategies to minimize these potential heating effects. From in vivo experimentation in swine4 and the results of thermal lesioning in the ventral intermediate nucleus of the thalamus in humans for the treatment of essential tremor,1 we have learned much in terms of local temperature effects. The lesion size is highly reproducible with an extremely sharp gradient between normal and necrotic tissue that is similar to that in other thermal lesioning modalities, such as radiofrequency, and is probably even more distinct than the gradient produced from ionizing radiation.4 This sharp lesion drop-off means that there is no damage to surrounding brain parenchyma. Experience in 30 patients treated for chronic pain and movement disorders, as described by Jeanmonod et al., confirms this sharp drop-off in a lesion created by FUS.2,3 No cases of thrombus formation or stroke in the region surrounding the lesion, as determined by diffusion-weighted imaging, were reported.

In trigeminal neuralgia, larger vessels near the trigeminal nerve would not be directly targeted, and this sharp energy drop-off would be utilized to target the nerve distant to the vascular structure. In addition, the heat-sink effect from rapid vascular flow helps defray heating of the vessel. It should also be noted that in the setting of trigeminal neuralgia, a higher “lesional” temperature—as currently utilized in FUS lesioning procedures for pain and movement disorders—would not be used since the goal is not to cause necrosis of the entire nerve, but to demyelinate and interrupt the pain fibers only.

Concern regarding the IAC is valid and was of particular interest in these experiments. The geometry of the re-
required positioning of the ultrasound transducer to target the trigeminal nerve is such that the ultrasound “shadow” falls onto the IAC. Bony absorption of ultrasound energy and subsequent heat generation were significantly mitigated by turning off transducer elements pointing at the IAC. Further optimization of this process will continue to address this issue and improve this collateral heating.

We agree with Zheng et al. as regards the determination of the pain-relieving effect. The cadaveric targeting model is clearly not helpful in determining this. Given the subjective nature of pain and the complex nature of trigeminal neuralgia, we suspect that a clinical trial is required to determine efficacy. Intraoperative assessments of the therapeutic effect following sequential increases in temperature application to the nerve, similar in technique to that performed with radiofrequency rhizotomy, may be a reasonable paradigm to follow. In vivo studies of somatosensory evoked potential signal attenuation and histological analysis of cranial nerves in animal models of FUS may be of some value in parameter optimization prior to a clinical trial.

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Diagnostic strategy in patients with subarachnoid hemorrhage

TO THE EDITOR: With interest I read the article by Delgado Almazooz et al. about the diagnostic yield of CT angiography (CTA) and MR angiography (MRA) in angiographically negative subarachnoid hemorrhage (SAH) (Delgado Almazooz JE, Jagadeesan BD, Refai D, et al: Diagnostic yield of computed tomography angiography and magnetic resonance angiography in patients with catheter angiography–negative subarachnoid hemorrhage. J Neurosurg 117:309–315, August 2012). Computed tomography angiography demonstrated a cerebral aneurysm in 4 (9.3%) of 43 patients with aneurysmal SAH. Magnetic resonance angiography demonstrated only 1 of these aneurysms. No causative cerebral aneurysms were found in patients with perimesencephalic SAH or CSF xanthochromia. Three of these 4 aneurysms were, in retrospect, visible on angiography but were not recognized or interpreted as the cause of SAH.

In another article about the same patient group, diagnostic yield of repeat catheter angiography was assessed in patients with SAH and negative catheter angiography and CTA. The first repeat catheter angiography performed 7 days after presentation demonstrated a causative vascular abnormality in 3 (4.4%) of 68 patients, 2 with aneurysmal SAH and 1 with perimesencephalic SAH. The second repeat catheter angiogram obtained in 43 patients (59.7%) did not demonstrate any causative vascular abnormalities. In this scenario, a patient with an SAH without an aneurysm eventually undergoes 3 catheter angiography sessions, a CTA, and a MRA to confirm that there is no aneurysm.

This study again demonstrates that aneurysms can be missed fairly easily on catheter angiography as well as on CTA and MRA. This is not surprising since all 3 imaging modalities are no longer the best way to detect an aneurysm. In the late 1990s 3D rotational angiography became available for clinical practice. This technique has evolved into a quick and easy to perform procedure. In our hospital we have used 3D angiography as a primary imaging tool in patients with aneurysmal SAH for more than a decade (we reserve CTA for patients with perimesencephalic SAH only). A rotational run takes 6 seconds with manual injections of 12–18 ml of contrast material, which is much quicker and cheaper than a multiple projection 2D angiogram with 10 ml of contrast per projection. Reconstruction of the 3D images takes a few seconds.

In various studies we demonstrated the superior image quality and diagnostic yield of 3D angiography in comparison to 2D angiography. In 19 (83%) of 23 patients with aneurysmal SAH and negative 2D catheter angiography, 3D angiography demonstrated an aneurysm as the cause of SAH. Of 94 additional aneurysms detected with 3D angiography in 350 patients with a target aneurysm, 27 (29%) were missed on 2D angiography. Perhaps the best demonstration of the superior image quality of 3D angiography is in the detection of fenestrations in cerebral arteries in almost 40% of patients; most of these anomalies were, even in retrospect, not visible on 2D angiography.

References
An SAH is a serious disease, and the patient has the right to a quick and definitive diagnosis followed by treatment. We use the following imaging strategy: CTA is used as the exclusive imaging modality in patients with a perimesencephalic SAH pattern to exclude a basilar tip aneurysm. These patients have the same low a priori chance of an aneurysm as every other healthy individual (1%–2%). In patients with aneurysmal SAH, we start with 3D angiography studies of all vessels. In cooperative patients this is done under local anesthesia, and when an aneurysm that can be coiled is detected, we immediately proceed with the induction of general anesthesia followed by coiling. In uncooperative or intubated patients we perform 3D angiography directly under general anesthesia, and when possible, the aneurysm is coiled. When no aneurysm is found on 3D angiography studies of all vessels, we do not routinely repeat the angiography but do so only on a case-by-case basis, for example, in young patients. Magnetic resonance angiography is reserved for the follow-up of coiled aneurysms only and has no place in the setting of acute SAH.

As Delgado Almandoz et al. rightly point out, the most important drawback of their study is “the lack of rotational angiography with 3D reconstructions in the initial catheter angiogram in all patients.” There is a simple solution to this problem: use 3D angiography more liberally.

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DISCLOSURE
The author reports no conflict of interest.

References

Response
No response was received from the authors of the original article.

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Correlation of diffusion tensor imaging and intraoperative macrostimulation

TO THE EDITOR: We read with great interest the paper by Said et al. (Said N, Elias WJ, Raghavan P, et al: Correlation of diffusion tensor tractography and intraoperative macrostimulation during deep brain stimulation for Parkinson disease. J Neurosurg 121:929–935, October 2014). On deeper inspection we find that the conclusions drawn from the results of this work are based on scientific methods that are not consistent with the current state-of-the-art methods in the burgeoning field of “connectomic neurosurgery.” It is our belief that the authors’ conclusions need to be put into a more explicit context. 1) There cannot be a direct correlation between voltage (U) amplitude (applied during macrostimulation in deep brain stimulation [DBS] surgery) and the distance to the medial corticospinal tract (CST). There is some correlation that might be found between stimulation current (I) and distance, as we have used in subthalamic nucleus surgery previously. However, according to Ohm’s law (I = U/R), resistance (tissue impedance and others = R) needs to be correlation-evaluated. This in itself is difficult, and there might be ways to bring impedance, electrode geometry, and stimulation voltage into perspective in an attempt to draw conclusions on the distance to the medial CST border. In the currently performed fashion, a pure correlation between voltage and distance is not the most relevant measure. 2) The rendition of the CST with the diffusion tensor imaging (DTI) technology as shown in this work (Fig. 2) is not constructed using state-of-the-art methods and underestimates the spatial distribution of the pathway in the subthalamic region. This has implications for the measurements and, in turn, for the definition of the medial border of the CST, which are known limitations of deterministic tractography in this application. However, we agree that it is difficult to correctly judge the size and shape of the internal capsule and explicitly define the medial border of the CST on a patient-specific basis under any circumstances. This issue is compounded by the diversity of tracking algorithms that are available to the surgeon by commercial software systems, albeit not originally designed for this purpose, but rather to provide a gross estimate of structural connectivity. 3) The inherent desire to trust software that the user may not fully understand represents a dangerous proposition when subsequently used for scientific analysis or clinical decision support, exemplified in Fig. 1 of Said et al.’s article, which depicts the CST in the wrong anatomical location relative to the patient anatomy (i.e., it should have been recognized...
on simple visual inspection that the CST should be situated lateral to the DBS electrode).

Several groups around the world, including both of our groups, are researching the application of diffusion-weighted imaging and tractography technology for the use in planning and performing DBS surgery in its various indications.5,10 We have shown in our previous work that tractography can be applied with a high enough accuracy to directly assist DBS surgery. It is not our intention to suppress results that might contradict our own work. However, conclusions should be based on proper scientific methods, and these should be debated openly. Our analysis suggests that this study represents an attempt to apply advanced imaging concepts with typical clinical imaging data sets whose collection were likely not appropriately designed for such a purpose, coupled with the use of overly simplified “push button” tractography provided in commercial software. We propose that an important basic goal for our field should be to establish a best-practices approach to preoperative MRI data collection and tractography analysis for future studies, for example, definition of minimum resolutions and signal-to-noise ratios for imaging data sets worthy of inclusion in scientific publications, as well as consensus-based identification of the most accurate and robust tractography metrics, specifically designed for the task at hand.

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DISCLOSURE
Dr. Coenen has been a consultant for Medtronic and is a consultant for Precisis AG and received research support from Medtronic Europe and Boston Scientific. Dr. McIntyre reports that he is a consultant for Boston Scientific Neuromodulation and Surgical Information Sciences.

References

Response
We thank Drs. Coenen and McIntyre for sharing their thoughts about our article. We must admit that we too were surprised by the negative results of our study, as we are also believers in the potential of DTI and advanced neuroimaging in general. However, we thought it important to publish these negative results to warn the neurosurgical community about the pitfalls of a simplistic application of DTI to neurosurgical guidance. As demonstrated by our results, the traditional macrostimulation in DBS surgery cannot be merely replaced by measuring a distance from electrodes to tracts on DTI. There are multiple reasons for this, including some related to physiology and physics, or others related to the variation in terms of the shape and size of the CST in individual patients. Another important source of potential error in the clinical setting is the diversity of DTI acquisition techniques and DTI processing algorithms. These multiple methods cannot be considered as being interchangeable, and the degree of specialization required to master the DTI technology challenges the widespread, safe utilization of DTI for neuronavigation in individual patients. Definitions of best practices are a desired goal, but they are extremely difficult to achieve considering the number of stakeholders involved (imaging manufacturers, processing software companies, users, and regulatory authorities). An easier alternative is for individual institutions to develop their expertise in the specific DTI tool that they select and to develop a detailed understanding of the strengths, limitations, and pitfalls of this tool.

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Nickel allergy and aneurysm clips

TO THE EDITOR: In the article by Tan et al.3 (Tan T, Tee JW, Han TF: Cell-mediated allergy to cerebral aneurysm clip causing extensive cerebral edema. J Neurosurg
The authors highlight the first case of vasogenic cerebral edema caused by a cell-mediated hypersensitivity reaction 4 years after aneurysm clipping in a 60-year-old woman with a history of severe nickel contact dermatitis. They discuss the possibility of nickel hypersensitivity, speculating that the intermittent nature of their patient’s cerebral edema may have resulted from the sporadic leaching of nickel from the Phynox clip. This then produced the cycle of acute-on-chronic cell-mediated hypersensitivity response that was visualized on imaging studies.

In contrast with their findings of an acute-chronic cycle of hypersensitivity reaction in a patient with a known nickel allergy, we recently reported on an acute case in a 33-year-old woman who developed a life-threatening allergic vasculitis following clipping of her brain aneurysm several weeks earlier with a nickel-containing clip. Our patient progressively declined, facing deteriorating clinical status (i.e., seizures) and additional infarctions. Repeatedly, neither she nor her family affirmed any contact dermatitis or nickel allergy. Finally, during decompressive surgery, biopsy findings raised the possibility of lymphocytic vasculitis, the nickel allergy was confirmed, and the clip was replaced with a titanium one.

Like the one reported by Tan et al., our case reminds the neurosurgical community of the possibility of nickel sensitivity—acute or chronic. When possible, screening can be used. Given the 10%–15% rates of nickel allergy, which are higher in women, we also advocate for the routine use of medical-grade titanium clips for aneurysm clipping. We agree with the authors that follow-up cerebral MRI can detect development of cerebral edema, but we wouldn’t advocate for its routine use in asymptomatic patients given the rarity of this reaction after aneurysm clipping. We reported the findings of this case in our Mayfield Clinic blog. The blog addresses the public health issue of nickel allergy, speculating that the intermittent nature of their patient’s cerebral edema may have resulted from the sporadic leaching of nickel from the Phynox clip.

Response

We read with interest the case report by Grande et al. and their attendant Letter to the Editor.

Key histopathological and clinical differences exist between our original report and that of Grande et al. In our paper, a local Type IV hypersensitivity reaction represented by intraparenchymal histiocytic infiltrates occurred in the area adjacent to where the Phynox aneurysm clip was placed. This presented as increased intracranial pressure and headaches. In contrast, the case reported by Grande et al. demonstrated mainly perivascular lymphocytic cuffing. This presented with what the authors have appropriately termed an allergic vasculitis of the CNS, with seizures and ischemic infarctions secondary to multifocal stenosis of the major intracranial arteries surrounding the nickel-containing clip. The multidisciplinary diagnosis of allergic vasculitis is supported by the temporal relationship between clipping and clinical re-presentation and deterioration, histopathological findings, and clinical stabilization after clip replacement with an Aesculap titanium clip.

The report by Grande et al. supports the fact that immunological reactions to nickel-containing aneurysm clips can result in a spectrum of clinical presentations, from low-grade, relapsing-remitting symptoms to life-threatening conditions. A documented allergy to nickel is helpful for diagnosis, but given the relatively high prevalence of nickel contact dermatitis and the rarity of allergic reactions to nickel-containing clips, a high degree of clinical suspicion remains of paramount importance. As mentioned previously, the increasing use of titanium-based clips can nullify the risk of nickel allergy in aneurysm clipping.

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Reference


Vertebral artery pexy for microvascular decompression

TO THE EDITOR: I read the article by Ferreira et al. with great interest (Ferreira M Jr, Walcott BP, Nahed BV, et al: Vertebral artery pexy for microvascular decompression of the facial nerve in the treatment of hemifacial spasm. J Neurosurg 114:1800–1804, June 2011). The offending vessels in hemifacial spasm vary. Compression by an anterior inferior cerebellar artery (AICA) or posterior inferior cerebellar artery (PICA) is common, but compres-

References


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sion by a vertebral artery (VA) is not rare, although in most cases there is a PICA between the VA and the facial nerve. In my personal experience of 500 cases of hemifacial spasm, the frequency of VA-related compression has been around 20%. Thus, how to decompress this big vessel is an important issue in microvascular decompression for hemifacial spasm.

Ferreira et al. described being able to treat VA compression by transposing the VA and suturing it to the clival or petrous dura safely. The procedure seemed to be effective in all 6 cases, but I feel uneasiness regarding the safety. The authors reported 1 case of “transient” vocal cord paralysis. In general, the neurological recovery from palsy of the lower cranial nerves (especially CN IX and X) is not easy. In most patients, swallowing and hoarseness improves to an almost normal functional level, but their vocal cord movement remains still paralytic. Compensation with the other vocal cord makes the symptoms better. Without a laryngeal fiberscope, it cannot be concluded that the lower cranial nerve damage was fully resolved. Thus I want to know if the authors examined the patient’s vocal cord with a fiberscope or asked any otolaryngologist to evaluate it.

In addition to the vocal cord paralysis, the authors reported a case of hearing loss (16.7%). The reported incidences of hearing loss are around 2%–3% and 1 case in 6 patients is an exceedingly high rate. In the literature, hemifacial spasm with VA compression is described as being surgically treated with less morbidity. In summary, I have concerns about the safety of this procedure and want to hear from the authors regarding this point as well as the status of the lower cranial nerves of the patient with “transient” palsy.

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DISCLOSURE
The author reports no conflict of interest.

References

Response
We thank Dr. Fujimaki for his very insightful comments regarding our manuscript. Most large series include all offending vessels (AICA, PICA, and VA) causing hemifacial spasm. The rate of VA compression has been found to be 24% in a series of 782 patients, which is in line with Dr. Fujimaki’s personal experience. It cannot be understated that our series of 6 cases treated with VA pexy were most severe, with impressive compression. The majority of our own patients undergo microvascular decompression of the AICA, PICA, or veins compressing the facial nerve with the use of Teflon felt pieces, with an excellent outcome. The senior author has not observed any cases of hearing loss in such patients in the last 10 years. The senior author has also observed the failure of microvascular decompression of the enlarged VAs when only Teflon felt or other padding was used. Looking at the physics of vascular compression of a cranial nerve, when the compressive artery is large, atherosclerotic, or calcified, it is not likely that any amount of padding will “decompress” the nerve, or even dampen the pulsations. Therefore, one needs to mobilize the artery completely away from the nerve, or vice versa.

Neurophysiological monitoring using lateral spread as an indication of resolution of spasm is applied in all of our cases. In addition, we employ the monitoring of hearing function, and of lower cranial nerve function, in patients with large-vessel compression. That being said, we agree with Dr. Fujimaki’s comments that both lower cranial nerve dysfunction and hearing loss are distressing complications. We attribute the lower cranial nerve dysfunction and hearing loss to increased manipulation of the offending vessel. In the patient described with vocal cord paralysis, the condition had improved at 1 year following the procedure (as observed by direct laryngoscopy), and subsequently had resolved completely at 2 years after surgery. Hearing loss was seen in 1 of 6 cases. In this case the patient had trigeminal neuralgia and hemifacial spasm due to severe vascular compression by the vertebral artery. The rate of hearing loss after microvascular decompression procedures has been found to be 2.7% in larger series. It is unknown whether those cases of hearing loss were due to severe compression due to VA ectasia or involved compression from smaller vessels. The small size of our case series will inflate any complication rates. The pexy of the VA that we have described is a technically more involved procedure and must be weighed against the potential hazards in the treatment algorithm.

Since our article was published, an additional 5 cases of hemifacial spasm caused by VA compression have been treated with a pexy procedure (most of these patients had other arteries or veins compressing the facial nerve in addition to the VA) have been treated with a pexy procedure. One of these 5 patients had a transient lower cranial nerve weakness, which resolved within 2 months. None of these 5 patients had hearing loss as a complication. All of the patients were cured of hemifacial spasm.

It should also be noted that, increasingly, most patients with hemifacial spasm are being referred to us after receiving Botulinum toxin injections for treatment of the condition over the course of many years. The longer duration of the hemifacial spasm may accentuate the compression of the facial nerve by the VA due to the progression of atherosclerotic change. It may also make the cure of the hemifacial spasm by simple microvascular decompression more difficult.
Neoadjuvant chemotherapy to maximize glioblastoma resection in the elderly

TO THE EDITOR: We read with great interest the article from Oszvald et al. (Oszvald Á, Güresir E, Setzer M, et al: Glioblastoma therapy in the elderly and the importance of the extent of resection regardless of age. J Neurosurg 116:357–364, February 2012). For almost a century, treatment of most high-grade gliomas has started with maximum safe resection. In this setting, this study may not seem to be a novel observation. However, while the impact of the extent of resection on the survival of patients with glioblastoma (GBM) is still questionable, the authors’ take-home message could very well change our approach to malignant gliomas, especially in elderly patients. The number of elderly patients with GBM can be expected to continue to increase as the proportion of elderly patients continues to grow, and the challenges of treating these patients are increased by the lack of randomized controlled studies involving this population. The lesson we learned from supratentorial low-grade gliomas in older patients is similar to the conclusion of Oszvald et al., strongly suggesting that treatment decisions in the elderly should follow the same criteria as in younger patients, with complete or extensive surgery as the goal. However, despite the innovative microsurgical armamentarium that is currently available, complete resection of GBM is often impossible due to the highly infiltrative nature of these tumors, their multiplicity, and their location within deep-seated or eloquent areas.

How can we enhance the chances for a complete surgery?

Here we present the case of a 66-year-old patient harboring a GBM within the whole left frontal lobe (Fig. 1A). Extensive involvement of this eloquent area prevented us from performing a complete resection and nonsurgical treatments were thought to represent a valuable therapeutic alternative. Thus, we considered a combination of temozolomide (TMZ) and bevacizumab (BV) as initial treatment. Eight weeks later (after 2 monthly cycles of TMZ and 2 biweekly cycles of BV), MRI showed partial regression of peritumoral edema and mass effect and moderate reduction in the size of the tumor (Fig. 1B). No toxicity or side effects were evident. This tumor shrinkage allowed us to perform a gross-total resection. No excessive hemor-
removal of a previously “unresectable” high-grade tumor.

benefit of reducing the tumor, allowing for safe gross-total resection beyond the apparent tumor boundaries, the so-called “supratotal” resection, if possible.5 In this way, we might be able to prevent tumor recurrence.

However, further studies and longer follow-up are necessary in order to provide evidence of whether this therapeutic strategy satisfies our expectations and improves the survival of patients with GBM. Since older patients are systematically excluded from multimodal treatment trials, these remain the main indications for the technique today. Its use for endovascular rescue following coil perforation arose a few years later, and is of course another good indication.

TO THE EDITOR: I congratulate Durst et al.2 on their experience and results with the dual (or double) microcatheter technique for the coiling of wide-necked intracranial aneurysms (Durst CR, Starke RM, Gaughen JR Jr, et al: Single-center experience with a dual microcatheter technique for the endovascular treatment of wide-necked aneurysms. J Neurosurg 121:1093–1101, November 2014). I personally appreciated their acknowledgment of my first conception of the method.1 I wish to clarify that in our original report, the technique was not used for “rescue” as the authors have suggested. Rather, we observed coil instability in the sac, and/or coil herniation through the neck, during deployment from a single microcatheter. I believe these remain the main indications for the technique today. Its use for endovascular rescue following coil perforation arose a few years later, and is of course another good indication.

The author reports no conflict of interest.

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3. Willinsky R, ter Brugge K: Use of a second microcatheter technique for the coiling of wide-necked intracranial aneurysms (Durst CR, Starke RM, Gaughen JR Jr, et al: Single-center experience with a dual microcatheter technique for the endovascular treatment of wide-necked aneurysms. J Neurosurg 121:1093–1101, November 2014). I personally appreciated their acknowledgment of my first conception of the method. I wish to clarify that in our original report, the technique was not used for “rescue” as the authors have suggested. Rather, we observed coil instability in the sac, and/or coil herniation through the neck, during deployment from a single microcatheter. I believe these remain the main indications for the technique today. Its use for endovascular rescue following coil perforation arose a few years later, and is of course another good indication.

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ENDOVASCULAR TREATMENT OF WIDE-NECKED ANEURYSMS

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DISCLOSURE
The authors report no conflict of interest.

Response
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