Creative use of endovascular devices in cerebral aneurysm treatment

To The Editor: The “balloon bounce” technique by Wolfe et al. (Wolfe SQ, Farhat H, Moftakhar R, et al: Intraaneurysmal balloon assistance for navigation across a wide-necked aneurysm. Technical note. J Neurosurg 112:1222–1226, June 2010) is a creative solution to the problem of how to navigate a device across the neck of a wide-necked cerebral aneurysm when tortuosity of the afferent artery or a sharp angle of the efferent artery becomes a challenge to all available guidewires and microcatheters. The authors inflated a HyperForm balloon (ev3) within the aneurysm sac to provide a contact surface to “bounce” another remodeling balloon across the aneurysm neck to a stable position. The HyperForm balloon was then deflated and removed, and a microcatheter was placed in the aneurysm for embolization. Three patients had aneurysms successfully occluded using embolization with Onyx 500 (ev3), but endosaccular coiling could surely have been done instead, and the same technique could have been used as a tool for “bouncing” a stent catheter as well. Some years ago I described one technical variant of intraaneurysmal balloon assistance for endovascular coiling of a wide-necked, unruptured aneurysm of the middle cerebral artery.1 In that case, after exhausting all available technical possibilities of navigating a remodeling balloon across the aneurysm neck, I placed a HyperForm balloon into the aneurysm neck and a microcatheter into the aneurysm fundus distally for coiling. The aneurysm was occluded using platinum coils, with the balloon inflated as necessary at the neck and into the aneurysm to keep the coils inside the aneurysm sac and the balloon withdrawn as the coils were deployed. The procedure was uncomplicated, and aneurysm occlusion was satisfactory on angiographic follow-up.

I would like to address two technical aspects of intraaneurysmal balloon assistance that were not mentioned by Wolfe et al.4 First, blood flow tends to move the balloon forward against the aneurysm fundus, and some length of the balloon catheter must be pulled out as the balloon is inflated to keep it in optimal position. The HyperForm balloon has a hard, 5-mm-long translucent tip that is positioned inside the aneurysm and that can be pushed against the aneurysm wall by the blood flow. Thus, the aneurysm sac must be large enough to accommodate the balloon, the balloon tip, and some length of the balloon system’s guidewire. This inconvenience can be avoided by using a rounded, guidewire-free, flow-directed balloon for intraaneurysmal assistance. Second, Wolfe et al. warned against balloon overinflation to avoid aneurysm rupture, but a dangerous situation is also theoretically possible if the HyperForm balloon occludes the aneurysm ostium partially, resulting in a siphon effect, with inflow of blood into the aneurysm during systole and outflow hindrance in diastole, which could cause aneurysm expansion with the risk of rupture.

Other reports on intraaneurysmal techniques permitting the endovascular treatment of complex aneurysms when more conventional techniques have failed describe the “neck bypass technique,”1 also mentioned by Wolfe et al., in which a balloon catheter is navigated over a guidewire into the aneurysm until it makes a complete loop along the dome wall and exits into the afferent artery. Another is the “waffle cone technique”2 in which one deploys a stent into the aneurysm sac and afferent artery. These techniques have in common the fact that devices designed to bridge the aneurysm neck were instead used inside the aneurysm sac, and the procedures were successful and uncomplicated. It is important that such unconventional, “creative” techniques are subjected to peer review and description in the literature. It is also important to point out that the decision to use such a technique for the first time should be based on the operator’s skills, experience in treating the actual pathology, and extensive experience with the devices involved.

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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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The effect of carotid endarterectomy

To The Editor: I read with interest the article by Nagaki et al.1 (Nagaki T, Sato K, Yoshida T, et al: Benefit of carotid endarterectomy for symptomatic and asymptomatic severe carotid artery stenosis: a Markov model based on data from randomized controlled trials. Clinical article. J Neurosurg 111:970–977, November 2009). The purpose of this letter was to determine whether the effect of endarterectomy can be assessed only observationally or only experimentally.

For patients with both symptomatic and asymptomatic carotid artery stenosis, several major randomized controlled trials of carotid endarterectomy (CEA) have previously tackled the remaining effects of CEA on the risk of stroke. Nevertheless, because the risk of stroke among patients with asymptomatic carotid artery stenosis is quite low, whether to treat their stenosis with CEA continues to be an important public health concern.

The authors established a Markov model in order to assess the efficacy of CEA. There were 4 different health states modeled. The authors did consider the probability of transition from one health state to another state and calculated such a probability approximately by using data from major randomized controlled trials.

With regard to their baseline analyses, they adopted 3 comorbidity index values. They stated outcomes via the use of the anticipated number of quality-adjusted life years (QALYs) for a theoretical cohort treated with CEA and another without CEA.

In fact, CEA for asymptomatic stenosis had little benefit (0.07 QALY) for normal-risk CEA candidates who were older than 70 years of age, not to mention those who were even much older, according to the authors’ baseline analysis.

It is understood that some ethical and practical issues may hinder randomized trials in medicine and surgery.2 Therefore, despite the fact that the authors established a Markov model in order to assess the efficacy of CEA, of importance in this context is the other fact that in order to determine whether a study is truly experimental, it will be necessary to ascertain at least some comparisons between two groups that differ on the basis of an intervention of interest. Endarterectomy, for instance, is the intervention in the context of current discussion.

In fact, if the endarterectomy treatment is not properly (i.e., with randomized controls) compared to a conservative treatment or condition, the effect of endarterectomy can then be interpreted only as observational, not experimental at all.

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Disclosure

The author reports no conflict of interest.

References


Response: We appreciate the author’s interest in our study. Carotid endarterectomy for asymptomatic carotid artery stenosis has been the focus of many reports and trials during the past decade. Although large randomized control trials have demonstrated a net benefit of CEA beyond that of the best medical treatment,1,4 the initial increase in events associated with perioperative mortality and morbidity required a few years for a benefit in event-free ratio to emerge. Therefore, long-term survival after CEA for asymptomatic stenosis is an important consideration when deciding whether to perform this prophylactic procedure. However, several natural history studies of patients with carotid artery stenosis have demonstrated decreased survival rates compared with the general population.2,3 These findings caused some concern about the long-term overall benefit from CEA.

Moreover, the clinical situation is more complicated than for simple stroke-risk versus treatment-risk analysis. It is undoubtedly important that CEA would be properly compared with a conservative treatment. We used a Markov model to address this problem. Although in order to construct the mathematical model, several simplifications are inevitable, Markov modeling permits an increase in complexity of the model, such as incorporating stroke rate in patients with or without CEA, surgical risk, or the long-term survival rate of “CEA candidates.” In our baseline analysis for symptomatic stenosis, CEA nonetheless might be beneficial in the long term. On the contrary, in our baseline analysis, treatment for asymptomatic carotid stenosis yielded very little QALY gain, and surgery is not likely to have a large impact upon the burden of stroke. The benefit of CEA for such patients is only marginal and is rapidly lost to be negative because of the increased rate of surgical complications, advanced age, and comorbidities. Our results suggest that only carefully selected conditions, such as very low treatment risks in relatively young patients without comorbidities, could justify CEA for asymptomatic stenosis.

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Neuritis ossificans

**To The Editor:** We read with great interest the paper by Kemper et al.¹ (Kemper CM, Rojas JC, Bauserman S: Neuritis ossificans of a cranial nerve. Case report. *J Neurosurg* 113:1112–1114, November 2010). We also observed a somewhat similar finding in a 56-year-old woman who presented with progressive dysphagia and dysphonia that was the result of an intradural calcified lesion of the lower cranial nerves. Our preoperative diagnosis in this case, based largely on the finding of the patient’s clinical history and imaging, was calcified meningioma (Fig. 1). Laryngoscopic examination revealed the presence of a total preoperative left vocal cord palsy. Using a left suboccipital retromastoid approach, we were able to expose the lesion, which turned out to be inseparable from the lower cranial nerves, the anterior inferior cerebellar artery, and the posterior inferior cerebellar artery, and it was inseparable from both the dura mater and the brainstem. For these reasons, we could do no more than a partial resection. When the patient recovered from anesthesia, she reported worsened dysphagia and respiratory difficulties, which prompted a tracheostomy to be performed. An MRI examination ruled out both ischemia and/or infarction, but it did reveal an increase in the edema surrounding the surgical cavity. The ensuing course of action was then further complicated by systemic sepsis, which required prolonged

This article contains some figures that are displayed in color online but in black-and-white in the print edition.

![Fig. 1. Preoperative CT scans and MR images showing the right C-1 and the left jugular tubercle calcified lesions. A: Preoperative CT scans without intravenous contrast administration. B: Preoperative axial T2-weighted MR images. C: Preoperative coronal T1-weighted MR image. D: Preoperative axial T1-weighted MR image with intravenous contrast administration.](image-url)
assistance in the intensive care unit. Histopathological examination disclosed calcified tissue fragments within a granulomatous reaction with inflammatory infiltrates, giant cells, and fibrosis. Ziehl-Neelsen staining ruled out the possibility of a specific granuloma (Fig. 2).

The course of the disease we observed (progressive cranial nerve impairment) and the results of imaging seem to be strikingly similar to the report by Kemper et al. Although histological diagnosis does provide for a differentiation made possible due to the lack of nerve fibers, there does not appear to be enough evidence to rule out the possibility of a diagnosis of ossificans neuritis. We say this because, in our case, the deliberate choice of the surgeon (G.B.) was to avoid resection of cranial nerves along with the pathological mass. Our sense is that, perhaps, we may have actually found a second case of ossificans neuritis of cranial nerves.

These considerations led us to ask 2 questions that in our opinion remain unanswered. The first regards the epidemiology of this disease. Is ossificans neuritis of the cranial nerves really as rare as was first reported in 2010, or is this yet another example of a misdiagnosis that goes unreported? The second consideration involves the putative etiopathogenesis of this disease. Ossificans neuritis involving the peripheral nerves is a rare entity that, given its pathological resemblance to myositis ossificans, is currently thought to be to be secondary to a traumatic event. With that said, however, it needs to be emphasized that even in cases involving the peripheral nervous system, the disease’s etiology continues to remain unclear. Although Kemper and colleagues’ idea that neuritis ossificans “is caused by a repetitive mechanical trauma or friction of the nerve near the porous hypoglossus” does seem to be quite original, we wonder why the disease should be so uncommon when it is known that the hypoglossal nerve is, indeed, quite prone to mechanical trauma or friction. Could the anatomy of their patient be significantly different from the normal anatomical pattern? If this is the case, what then are we to conclude about the findings in our patient? Are we to conclude that the lower cranial nerves are also prone to mechanical trauma or friction? We know that the environment surrounding the cranial nerves is quite different from that surrounding peripheral nerves. The cranial nerves are bathed in a liquid medium of CSF, whereas peripheral nerves are surrounded by a more solidified fatty tissue. Such a dramatically different kind of surrounding should be expected to affect the nature of the susceptibility of these different nerves to mechanical trauma. It certainly seems reasonable to expect that such dramatically different environments should call into question any kind of simplistic trauma hypothesis that treats peripheral and cranial nerves in the same way. On the contrary, it seems much more realistic and plausible to recognize that the etiology of this disease, at the level of cranial nerves, continues to be unknown and is probably unrelated to trauma.

We invite the neurosurgical community to share the experience of such a fascinating, puzzling, and seemingly unusual disease. It should also be noted that, since the lesion in question is, in our experience, unresectable, we recommend the avoidance of any kind of aggressive surgical approach before the course of this disease is clarified.

**Disclosure**

The authors report no conflict of interest.

**Reference**


**RESPONSE:** We thank Ferroli and colleagues for their interest and comments regarding our paper with the first report of neuritis ossificans of a central nerve. The definitive diagnosis was made by pathological analysis showing zonal heterotopic calcification and observation of Haversian canals confirming ossification. Positive staining for neurofilament protein, S100, and epithelial membrane by immunohistochemical analysis confirmed the presence of nerve fiber. We believe that the description of intracranial neuritis ossificans will increase with the addition of this entity to the differential diagnosis.

The occurrence of neuritis ossificans is in fact a rare entity itself. The reporting of this phenomenon is uncommon even in the general literature. With the quality of current imaging we find ever-increasing ability to resolve or identify intracranial pathology radiographically. We suspect that the report by Ferroli et al. and ours will prompt neuroscientists to further investigate the incidence, natural history and response to treatment of this pathology. We
believe that this entity will be described in the future or in retrospect. The suggestion that this entity is either under-reported or misdiagnosed is supported.

The source of this lesion was suggested to be from localized trauma at the entrance to the hypoglossal foramen. For example, the theory of syrinx formation relies on local physiological trauma at the foramen magnum to produce deformation of its contents. Additionally, tension, as a source of local trauma, is a known factor in cranial nerve damage. We maintain that it is conceivable for localized trauma by tension of the nerve at the hypoglossal foramen to be a possible explanation for pathogenesis in central neurositis ossificans. No abnormal anatomy of this patient was observed intraoperatively or radiographically.

The treatment for neurositis ossificans, as always, should be based on a risk-benefit analysis. The case presented in the original article described a patient’s fixed deficit and end organ damage as demonstrated by electromyography and allowed a total resection of this focal lesion under evoked potential monitoring with little risk to additional structures. If vital viable tissue were involved with this lesion, we would concur with Ferroli et al. that a measured surgical response would be appropriate. Based on the comments by Dr. Ferroli, as well as findings in our own patient, it appears that this lesion can result in significant neurological morbidity. Due to the uncommon incidence of this entity, further observations would be helpful in establishing its natural history.

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Minute-by-minute monitoring of autoregulation

TO THE EDITOR: We read with great interest the recently published paper by Depreitere and colleagues1 (Depreitere B, Güiza F, Van den Berghe G, et al: Pressure autoregulation monitoring and cerebral perfusion pressure target recommendation in patients with severe traumatic brain injury based on minute-by-minute monitoring data. Clinical article. J Neurosurg [2014]20:1451–1457, June 2014), in which they report the use of minute-by-minute intracranial pressure (ICP) and mean arterial blood pressure (MABP) data to assess the vascular pressure reactivity of cerebral autoregulation with an algorithm called the low-frequency autoregulation index (LAX). This index is similar to the pressure reactivity index (PRx) proposed by Czosnyka et al.,2 nevertheless, PRx requires second-by-second ICP and MABP data for its calculation. The hypothetically optimal cerebral perfusion pressure (CPPopt) can be individually assessed from the minima of the U-shaped plot of PRx values at different CPPs. The novelty of the algorithm used here lies in the “dynamic adaptive target of active cerebral autoregulation” (DATAcar), which considers historical data up to 24 hours preceding the time point of interest and allows the calculation of a dynamic CPPopt during 95% of the time. The time that the patients were close to this individualized CPPopt correlated with the outcome.

These results are encouraging since many neurocritical care units around the world have the ability to obtain by-the-minute data samples, where these findings can be easily reproduced. As mentioned in the authors’ paper, in 2011, in a small study with 18 patients suffering from nontraumatic intracerebral hemorrhage, we reported the use of minute-by-minute ICP and MABP data with an index called low-frequency pressure reactivity index (L-PRx) to assess autoregulation.3 In that study we found a good correlation with PRx (p = 0.846, p < 0.001) and were able to obtain CPPopt in 66.6% of the patients. Moreover, L-PRx correlated and PRx correlated comparably with the outcome as assessed using the National Institutes of Health Stroke Scale. In a later study of 29 patients with traumatic brain injury, we also monitored cerebral autoregulation using L-PRx. We found a significant negative correlation with the 6-month Glasgow Outcome Scale (r = -0.556, p = 0.002),3 giving more support to the hypothesis that the minute-by-minute data contain important autoregulation information.

One of the reasons why LAx works fine in critical patients (only the first 48 hours were used in this study) may be the high variability in the MABP curves due to the use of fluids and catecholamines to reach certain MABP or CPP goals. In centers where the MABP is forced to reach higher thresholds, the variability of MABP would be artificially increased, and autoregulation would be more challenged in a wave band that can be obtained using minute-by-minute data and having fewer artifacts than those in high-frequency data. Also, the nature of the DATAcar algorithm (that is, weighted contribution of 45 time-window calculations) allows for a higher frequency of successful CPPopt calculations.

The results are promising given the steadily increasing number of neurocritical care units using minute-by-minute sampling with commercial monitoring devices capable of obtaining this information for direct analysis.

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Disclosure
The authors report no conflict of interest.

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RESPONSE: We thank Dr. Sánchez-Porras and colleagues for their positive comments on our paper on autoregulation monitoring and CPP target recommendation based on minute-by-minute data. Sánchez-Porras et al. and Santos et al. have already reported on their expertise with the use of minute-by-minute ICP and MABP data for the monitoring of cerebrovascular pressure reactivity.2,3 Our basic methodology, as well as theirs, follows the same principles as those for the calculation of the PRx, and we are therefore indebted to Czosnyka et al.1 Our paper does not at all question the established value of PRx but instead represents a pragmatic exercise on the optimization of the information on pressure reactivity present in monitoring data with standard resolution. By dynamically widening the window of data under investigation, we significantly increased the percentage of monitoring time for which a CPPopt recommendation could be given. The wider range of blood pressures that are consequently incorporated into the calculation of CPPopt might have contributed to this, as is suggested by Dr. Sánchez-Porras. The use of routine ICP and MABP data in combination with the almost permanent CPPopt recommendation makes the method applicable in most intensive care units. The DATACAR calculation method is now being programmed and tested. We agree with the conclusions made in the Letter to the Editor and thank the authors for their support.

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Supraorbital and transorbital minicraniotomies

TO THE EDITOR: We read with interest the article by Beretta et al.1 (Beretta F, Andaluz N, Chalaala C, et al: Image-guided anatomical and morphometric study of supraorbital and transorbital minicraniotomies to the sellar and perisellar regions: comparison with standard techniques. Laboratory investigation. J Neurosurg 113:975–981, November 2010). In this cadaveric study, to quantify and compare the surgical exposure afforded by the pterional, supraorbital keyhole, and transorbital keyhole approaches, the authors defined 3 quantitative parameters for comparison. First, a “working area,” relative to the target deep in the surgical field, was defined as a sum of 6 adjacent triangles with the anterior communicating artery (ACoA) at the center. The other 2 parameters were “depth of the surgical window” and “angle of basal view.” Using frameless stereotactic navigation, they found that the “working area” increased progressively from the pterional approach (425.73 ± 85.85 mm²) to the supraorbital keyhole approach (460.24 ± 160.19 mm²), and then to the transorbital keyhole approach (568.78 ± 70.09 mm²). When comparing the surgical exposure provided by different minicraniotomies, there are several useful concepts such as the working area at the level of the target, the angle of attack on different anatomical structures around the target, and the intracranial optical field in the keyhole concept.2,3 The intracranial optical field increases with the depth of the targeted pathology; this concept is used in the keyhole approach, by which a small cranioectomy is fashioned after thorough understanding of the location of the lesion and its relationship with the surrounding normal anatomical structures. In this way even a small cranioectomy can provide adequate exposure for deep-seated pathological entities. However, because of the limited angle of attack, for example, in doing a keyhole supraorbital cranioectomy, the cranioectomy may need to be placed slightly medially or laterally in different patients.

On the other hand, all large-scale approaches in microneurosurgery can be considered to consist of a side-by-side combination of several small keyhole approaches.2,3 Therefore, provided a keyhole cranioectomy falls within the boundary of a standard cranioectomy (e.g., a supraorbital keyhole approach and a pterional approach), the surgical exposure affordable through the keyhole cranioectomy will be attainable via the standard cranioectomy if the center of the view of the microscope is shifted to align with that of the keyhole cranioectomy (Fig. 1). However, when part of a keyhole cranioectomy lies outside the boundary of a stan-
standard craniotomy (e.g., a transorbital keyhole approach and a pterional approach), then in that particular direction the keyhole approach can provide certain surgical exposure that is not attainable by the standard craniotomy. However, there exists also a standard craniotomy encompassing the transorbital keyhole approach. In summary, we think by that simple deduction, the supraorbital keyhole approach should not be able to provide a greater “working area” than the pterional approach.

There are certainly other characteristics of a neurosurgical approach that are difficult to quantify for comparison, relevant to this study, such as the advantage of a widely split sylvian fissure available via the pterional approach. We think that to fully evaluate the surgical exposure afforded by a craniotomy, the surgical window at the surface, which is related to the angle of attack, should be taken into consideration as well. One also needs to bear in mind that throughout an operation, the view of the microscope is constantly shifted and a retractor blade is repeatedly adjusted. Last, although the title of the article is about the sellar and perisellar region, the “working area” was defined as centered at the ACoA. We think the information derived from this study cannot be directly generalized to the whole region but should be limited to around the ACoA complex, as mentioned in the article (the last paragraph in Discussion): “When using the mid-ACoA as the main target, we noted the transorbital keyhole craniotomy afforded the best visualization of the entire ACoA complex.”

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Fig. 1. Sketch diagram showing a pterional craniotomy (dashed line), a supraorbital keyhole craniotomy (square-dotted line), and an additional orbital osteotomy (shaded area). The arrows that radiate from point A show directions in which to move a microscope to align with the view of a supraorbital keyhole craniotomy, at point B. Point C marks an additional viewing window after an orbital osteotomy.

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Disclosure
The authors report no conflict of interest.

References

RESPONSE: Thank you for the opportunity to respond to Drs. Wong and Fong.

The authors of the letter question the clinical validity of the results of our study and state that the same results could be accomplished by changing the position of the microscope, the use of retractors, and the consistent use of a pterional craniotomy. They provide no anatomical, morphometric, radiological, or clinical data to support their claim.

While we respect and value their opinion, we beg to differ with their statements based not only on our surgical experience, but also the results of nearly a decade of anatomical, morphometric, and clinically proven data in multiple peer-reviewed publications that we have produced during that period. Furthermore, our results have been confirmed by others who have used both similar and different methodologies.

The authors claim that similar views can be afforded by changing the position of the microscope. Although intuitively correct, this is a limited resource; in a deep surgical field, sooner or later, the vectors of vision will be blocked by either the edges of the craniotomy (including the base of the skull), or the brain itself. We have previously shown these facts in a cadaveric study as well as in the work in question. The authors later suggest the use of retractors and their repositioning, and opening of the sylvian fissure as additional measures to improve surgical exposure. These maneuvers, while helpful, are known to
be associated with increased risk of brain damage and operating times.\textsuperscript{3–7,12,16,17,21,22,29,30,33,36} In summary, it was precisely because of the potential (and certainly avoidable) consequences associated with the use the maneuvers proposed by the authors of the Letter to the Editor that we have been conducting our studies on the topic for the last 10 years.

Ever since the introduction of endovascular coiling, the bar has been raised for microvascular surgery, and outcomes that were previously irrelevant, such as cognitive function, independent living, employability, and even cosmesis have attained a more prevalent role in modern neurosurgery. We have found the techniques described in our article to be helpful toward the accomplishment of those goals, as documented throughout our clinical peer-reviewed reports. We challenge the authors of the Letter to present (as we have) compelling anatomical, morphometric, radiographic, and long-term clinical data to support their statements before issuing any further judgment based on intuition, subjective observations, and “simple deduction.”

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