Neurosurgical Forum

LETTERS TO THE EDITOR

Keyhole and standard subtemporal approaches

TO THE EDITOR: Recently, Ercan and colleagues¹ published their cadaveric laboratory investigation of keyhole versus a traditionally sized craniotomy with and without zygomatic osteotomy to assess temporal lobe retraction and surgical exposure via a subtemporal corridor (Ercan S, Scerrati A, Wu P, et al: Is less always better? Keyhole and standard subtemporal approaches: evaluation of temporal lobe retraction and surgical volume with and without zygomatic osteotomy in a cadaveric model. *J Neurosurg* [epub ahead of print September 16, 2016. DOI: 10.3171/2016.6.JNS16663]). The authors' cadaveric specimens were assessed radiographically with CT-based navigation and volumetrically with computer software and a measured resin mold of the surgical corridor.

In this comparison, the keyhole and traditional craniotomy approaches exposed a similar anatomical volume with no difference in temporal lobe retraction. The keyhole approach was limited in its surgical exposure around the area of interest and in its vertical attack angle. Ercan and colleagues¹ warned of only partial venous visualization of basal temporal veins in a keyhole subtemporal approach. This visualization was attributed to decreased angles and reduced surgical exposure due to the smaller keyhole craniotomy that limit the space available for execution of surgical movements. The use of an endoscope to enhance visualization in the keyhole approach was only briefly mentioned. The authors acknowledged that maneuvers for brain relaxation (mannitol, head positioning, and CSF drainage) were missing in this comparison but did not comment on the role of single-shaft instruments or dynamic movement of the microscope to facilitate visualization in a smaller corridor.² Lastly, their use of fixed retraction may not recreate the surgical approach used in the operating room.³

The interpeduncular and ambient cisterns lie in the plane of the tentorial incisura. The tentorium leading up to the incisura extends from the petrous ridge and curves upward toward the free edge of the tentorium. Therefore, the area of interest in a subtemporal approach is actually above the plane of the lateral skull base and middle fossa floor. The ideal lateral approach that minimizes brain manipulation would utilize an inferior-to-superior trajec-

tory—in fact, the more inferior the starting point, the less theoretical brain retraction and manipulation would be required. With this anatomical maxim in place defining the inferior aspect of the craniotomy, the superior aspect of the craniotomy is dictated only by what is necessary to create sufficient working space. In our experience, this has been 1.5–2.5 cm depending on the pathology. With this exposure in place, the amount of brain retraction is dependent almost exclusively on brain relaxation maneuvers. Again, in our experience, with proper brain relaxation techniques, even with very small keyhole approaches, fixed retractors are rarely, if ever, used.

Ultimately, the question of “is less always better” perhaps misses the point altogether. The principles that guide successful keyhole surgery are the same as those that guide a “conventional” craniotomy. Sufficient brain relaxation that obviates the need for retractors is essential regardless of the approach taken. The need for surgical maneuverability to implement safe microsurgical technique is also indisputable. However, the advent of specially designed keyhole instruments and growing surgical experience with the use of the endoscope has made the area necessary for safe surgical maneuverability substantially smaller. Once these elements are taken into consideration, the question becomes the following: Why perform a 6-cm craniotomy when a 2-cm one is sufficient? Furthermore, do these smaller surgeries translate into improved outcomes—shorter exposure time, lower infection rates, less muscle atrophy, improved cosmesis, less postoperative pain, shorter length of stay? It should be noted that keyhole approaches, given their smaller exposures, have little margin for error. Detailed anatomical knowledge and careful patient selection, operative planning, and placement of the opening are required. Keyhole techniques, much like endoscopic endonasal techniques, have a steep learning curve. Effective utilization of keyhole approaches requires brain relaxation, proper instrumentation (single shaft), and adept use of both the microscope and endoscope.

As a comparative anatomical study to provide basic information regarding the subtemporal approach and its minimally invasive modification, this work by Ercan and colleagues¹ finds similar anatomical exposure with equal temporal lobe retraction; this underscores that a keyhole subtemporal approach, perhaps with some technical modifications, provides the same access as a traditional craniotomy. True comparisons between keyhole and larger-

sized craniotomies must consider all available modalities to achieve success in a more precise corridor.

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Disclosures

The authors report no conflict of interest.

Response

We appreciated the interest generated by our paper as reflected in the comments by Drs. Stamates and Wong.

As we clearly stated, our study is a comparative one and the results are what they are, given the constraints of the study. Obviously we changed the microscope angle during the execution of the procedure: this is a standard tenet of microsurgical techniques. The results, as recorded in the study, are that “the keyhole subtemporal craniotomy was not associated with less temporal lobe retraction than the standard subtemporal approaches. While the anatomical space was the same in both craniotomies, the surgical space, the space in which one can execute surgical maneuvers, was larger in the standard subtemporal craniotomy than in the keyhole craniotomy. In addition, the standard craniotomy allowed visualization of the stretched temporal lobe basal vein so that maneuvers to control/limit this stretching may be undertaken. This venous visualization was limited in the keyhole approaches, where it was only achieved partially by using the endoscope.”

Perhaps the results are not surprising (a larger bony and dural opening allows you more usable angles of attack on the target). However, we felt it was important to share the findings with our colleagues, particularly in light of the somehow unverified proposal/acceptance of different techniques that oftentimes are thought to be preferable to well-established ones just because they have the hallmark of being minimally invasive and having a smaller incision or the like.

As has been stated before, there are tools to evaluate and prove/disprove new surgical techniques.^{1,2} The phrase “in our experience” is somehow of a circular self-referential nature that, unless pointing to quantum leap improvement in measurable outcome, has very little substance.

We are clearly in favor of a learning curve (a paraphrase to indicate suboptimal outcome) if it takes us to a better place in terms of a reproducible and measurable better outcome for our patients. If we are able to prove that

we achieve a better outcome with a smaller incision, different surgical techniques, or no surgery at all, then all the better for our patients and our field.

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Blunt cerebrovascular injuries in severe TBI

TO THE EDITOR: We thank Esnault et al.¹ for their study, which is a valuable effort to look at blunt cerebrovascular injury (BCVI) in patients with severe traumatic brain injury (Esnault P, Cardinale M, Boret H, et al: Blunt cerebrovascular injuries in severe traumatic brain injury: incidence, risk factors, and evolution. *J Neurosurg* [epub ahead of print July 29, 2016. DOI: 10.3171/2016.4.JNS152600]). The only intracranial hemorrhage complication in their study was an epidural hematoma following an iatrogenic procedure—insertion of an intracranial pressure monitoring device. We do have a few questions for the authors.

They mention that the incidence of a poor outcome (Glasgow Outcome Scale Score 1–3) was similar in patients with or without BCVI. However, among the 14 patients with BCVI who received systemic anticoagulation, was there any hematoma expansion after initiating therapy?

Of the 4 patients with BCVI who suffered an ischemic stroke, 2 had a stroke despite early anticoagulation therapy. Assuming these strokes occurred in the ipsilateral vascular territory (left internal carotid artery), do the authors have any thoughts on the failure of anticoagulation to prevent stroke in these patients?

We also ask the authors to specify, if possible, how many patients underwent digital subtraction angiography (DSA) and in what situations. The practice seems to be variable, with some people advocating DSA in CT angiography (CTA)–positive patients, while most others reserve DSA for CTA-negative patients who have focal neurological deficits.⁴

The authors mention a recent paper describing the sensitivity of CTA as rather low.³ It would be interesting to know the incidence of ischemic stroke in the CTA-negative patients in the study.

It may also be important to mention the type of cervical spine injury. Historically, C1–2 fractures associated with subluxation and fractures extending to the neural foramen have been considered to be high risk, but recent papers have also described BCVI with subaxial fractures.

Finally, was any follow-up imaging performed, and if so, at what time?

The current study is significant in showing that systemic anticoagulation may be safe to use in patients with BCVI and severe traumatic brain injury. Answers to the questions mentioned above will help to determine the cost-effectiveness of screening for BCVI, especially given the significant heterogeneity in the literature.²

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Disclosures

The authors report no conflict of interest.

Response

We thank authors Wu and Malhotra for the interest shown in our study and here address the interesting issues they raised.

During the study period, we noticed no hematoma expansion despite the use of early anticoagulation therapy. However, the relatively small number of patients with BCVI included in our study must counterbalance this result. The decision to start systemic anticoagulation soon after severe traumatic brain injury (TBI) is difficult and must be made while considering the benefit/risk ratio.

Effectively, despite early initiation of anticoagulation therapy, 2 patients with Grade II left internal carotid artery injury both developed stroke in the left middle cerebral artery territory. Both of these events seemed to have an embolic mechanism. One possible explanation is that these ischemic lesions were present at arrival but were not detected by initial CTA.

At our institution, DSA is performed in CTA-negative patients presenting with focal neurological deficits.⁴ Unfortunately, these data were not collected in the prospective database. We recorded only DSA performed in CTA-

positive patients. In the present study, 2 patients benefited from DSA for endovascular interventions.

Likewise, we did not record the incidence of ischemic stroke in CTA-negative patients. However, we believe it would be very low. Indeed, there is some evidence that a majority of false-negative CTA studies involved low-grade injury (that is, Grade I injury).³ These low-grade injuries probably have a lower impact in patients and are rarely complicated by neurovascular events, as demonstrated by Biffi and colleagues.¹

The classic screening criteria for BCVI include complex cervical fractures involving subluxations, fractures extending into the transverse foramen, and C-1 to C-3 fractures.² In our work, 9 patients with BCVI had cervical spine fractures. We noted 5 patients (56%) with single-level cervical lesions and 4 patients (44%) with multiple-level injuries. Two patients (22%) had subluxation fractures (atlantooccipital and C-7 dislocations, respectively), 4 patients (44%) presented with lesions involving and/or adjacent to the foramen transversaria, and 6 patients (67%) suffered from C-1 to C-3 fractures. Only 1 patient (11%) with severe thoracic injuries had no historically retrieved lesions (spinous process fracture of C-5 and C-6).

Finally, the follow-up imaging was not codified in this study and was performed at the discretion of the treating neurosurgeon or radiologist. Fourteen patients with BCVI survived until hospital discharge. Among them, 8 (57%) had follow-up imaging with CTA or MRI. The median delay between trauma and first follow-up imaging was 6 months (range 3–21 months). The initial injuries evolved as follows: 1 residual stenosis, 3 complete occlusions, 2 developed aneurysms, and 2 complete recoveries.

To conclude, we hope that this response will eventually convince practitioners to detect BCVI in all patients with severe TBI.

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Seasonal subarachnoid hemorrhage: temperature or daylight?

TO THE EDITOR: We read with interest the article by Backes et al.¹ (Backes D, Rinkel GJE, Algra A, et al: Increased incidence of subarachnoid hemorrhage during cold temperatures and influenza epidemics. *J Neurosurg* 125:737–745, September 2016). The authors analyzed a nationwide cohort of Dutch patients with subarachnoid hemorrhage (SAH) along with data on influenza epidemics and average daily temperatures to determine if these 2 factors contribute to the seasonal variation in the incidence of SAH. The authors found an increased incidence of SAH during epidemic influenza, with gradual declines in this association in the weeks after an influenza epidemic. The authors also found an independent relationship between SAH and cold temperatures.

With limited direct exposure to the elements in our modern lifestyle, the association of SAH with temperature is puzzling, and we were surprised that there was no discussion of other synchronous factors during cold weather. For example, as the daylight hours decrease in northern latitudes, vitamin D levels will follow in some patients. A previous report found an association of low levels of vitamin D with hours of daylight, but at a somewhat more northern latitude (i.e., Sweden).³ This may be relevant to the findings in the Backes paper because vitamin D receptors play an important role in the expression of vascular endothelial growth factor and metalloprotease enzymes that affect development and remodeling of vessels, and that have antiproliferative effects on smooth-muscle cells in the walls of arteries, in addition to potent antiinflammatory effects.⁴

Wong and colleagues evaluated the association between low vitamin D levels and the presence of abdominal aortic aneurysms. They found that in older male participants whose abdominal aortas were larger than 30 mm in diameter there was a significant relationship between these individuals' 25-hydroxycholecalciferol vitamin D levels and their aortic artery diameters.⁵ The authors went so far as to suggest a dose-dependent relationship (i.e., a 10-nmol/L decrease in vitamin D concentration was associated with a 0.49-mm increase in mean aortic diameter).

More relevant to this issue was the report of hypovitaminosis D among patients who were treated for cerebral aneurysms.² In a retrospective analysis, Guan et al. compared the vitamin D levels in patients treated for cerebral aneurysms with patients who had no documentation of a cerebral aneurysm. The authors acknowledged a seasonal variation in vitamin D levels but found no significant difference in the season of the blood draw between their aneurysm and control groups. This study focused broadly on patients who required any treatment for cerebral aneurysm; it was not limited to patients who had ruptured aneurysms, because some patients underwent treatment secondary to

development of neurological symptoms, increase in aneurysm size, a dysplastic aneurysm morphology, or worrisome family history. Nevertheless, these authors found a higher incidence of hypovitaminosis D in their patients with treated cerebral aneurysm, which held even after controlling for normal cardiovascular and smoking risk factors. This study simply provided additional support for the concept that a vitamin D deficiency is an important factor to consider with regard to the risk for growth and rupture of cerebral aneurysms.

We congratulate Backes et al. for their contribution to the literature by providing additional evidence for the observed seasonal variation in SAH. Based on the literature as well as the expected short winter days in the northern latitude in which the study subjects lived, we wonder if the higher incidence of SAH in winter could be secondary to the short hours of daylight with a corresponding decrease in vitamin D levels, rather than the low temperatures.

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Disclosures

The authors report no conflict of interest.

Response

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

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