Letters to the Editor

NEUROSURGICAL FORUM

Predictors of outcome for gunshot wounds

TO THE EDITOR: I have read with great interest the article by Gressot et al.¹ (Gressot LV, Chamoun RB, Patel AJ, et al: Predictors of outcome in civilians with gunshot wounds to the head upon presentation. *J Neurosurg* 121:645–652, September 2014). The authors concluded that several factors, including patient age, Glasgow Coma Scale score, nonreactive pupils, and the path of the bullet and its fragments on CT scans, have predictive value for patient survival, and they created a scoring system based on these parameters. In their series of 119 patients 19% had good functional survival. We published an article in 1979 dealing with the same issues.² In our study of 82 patients with gunshot wounds sustained in civilian life, we evaluated the same presentation parameters as the authors and we assessed functional outcome. We also evaluated surgical findings, specifically the incidence of hematomas either at surgery or at autopsy in all patients who did not survive. Although we evaluated the importance of the same criteria, we concluded that the only truly meaningful parameter predicting patient survival was the state of consciousness on admission. While all the other parameters had some value, they were of secondary importance in predicting survival and its quality. There were 4 groups of patients based on the state of consciousness at the time of presentation: I) alert, awake; II) obtundation with or without neurological deficit; III) unresponsive to all but noxious stimulation in appropriate or semi-appropriate fashion; and IV) comatose inappropriate or no response at all.

We have found that this system provides as useful information as the point system proposed by the authors, although it is substantially simpler. Adding additional information did not increase the accuracy or reliability of our basic scale. For instance, while a bihemispheric bullet path generally indicates a poor prognosis, if the examination results fall into Group I or II, it is the state of consciousness that determines the prognosis rather than the path of the bullet. Similarly, if a patient has fixed, dilated pupils, the prognosis is poor, but patients in this case will undoubtedly be comatose with, at best, a decerebrate posture, which would place them in Group IV in which there are no survivors. Again, it is the state of consciousness that determines outcome. This simplified neurological examination makes it easier to rapidly assess patients with

gunshot wounds, particularly if non-neurosurgeons in the emergency department triage the patients. It provides rapid and accurate early information for patients and their families.

I was intrigued by the fact that in our study, 39% of patients achieved a functional survival status compared to 19% in the authors' study. The extent of injury caused by a bullet is determined to the greatest degree at the time of impact and is dependent on bullet mass and exit muzzle velocity squared. Passage of the bullet through brain tissue creates waves of massive increases in intracranial pressure in the wake of the bullet. Based on the above formula, the damage is greater with a greater bullet mass and greater muzzle exit velocity such as that seen in military grade weapons. Thus, the degree of neurological deficit is determined at time of impact in most instances. The development of mass lesions, such as hematomas, is rare and was seen in only 10% of our cases. Admitting that neither the authors nor we have studied the ballistic profile of the guns used, it is tempting to think that the difference in functional survival between our studies most likely reflects the currently greater availability of better quality weapons with larger bullet sizes and greater muzzle exit velocities. These cause much more damage than the "Saturday night specials" commonly used at the time of our study and could explain this discrepancy.

The authors are to be congratulated for continuing to study the problem of gunshot injuries, which are becoming more and more serious, particularly with easier access to higher-grade weapons in the civilian population.

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DISCLOSURE

The author reports no conflict of interest.

References

1. Gressot LV, Chamoun RB, Patel AJ, Valadka AB, Suki D, Robertson CS, et al: Predictors of outcome in civilians with gunshot wounds to the head upon presentation. *J Neurosurg* 121:645–652, 2014
2. Hubschmann O, Shapiro K, Baden M, Shulman K: Cranio-cerebral gunshot injuries in civilian practice—prognostic criteria and surgical management: experience with 82 cases. *J Trauma* 19:6–12, 1979

Response

We recognize Dr. Hubschmann and colleagues for their pioneering work in this area. Their study was performed between 1973 and 1975, and they did not have the benefit of routine CT scanning in evaluating patients with gunshot wounds to the head. While they found that the state of consciousness upon arrival was the only meaningful predictive measure of outcome, we found that the Glasgow Coma Scale score upon arrival was less important than pupillary reactivity and bullet trajectory as seen on CT scan upon arrival. As imaging technology continues to improve, we expect our understanding and evaluation of these patients to evolve as well.

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The blood-hammer effect and aneurysmal basilar artery bifurcation angles

TO THE EDITOR: The interesting study by Tütüncü et al.³ (Tütüncü F, Schimansky S, Baharoglu MI, et al: Widening of the basilar bifurcation angle: association with presence of intracranial aneurysm, age, and female sex. *J Neurosurg* 121:1401–1410, December 2014) seems to support the idea that unfavorable vessel design in arterial bifurcations may be a risk factor in intracranial aneurysm formation and progression. The hypothesis that aneurysmal basilar artery (BA) bifurcations present increased angles compared with nonaneurysmal basilar bifurcations was scrutinized. The BA bifurcation (α) angle, estimated in age-matched cohorts, was found to be significantly wider in patients with a BA aneurysm ($146.7^\circ \pm 20.5^\circ$) than in patients with an aneurysm in another cerebral artery (non-BA) ($111.7^\circ \pm 18^\circ$) and in a nonaneurysmal control group ($103^\circ \pm 20.6^\circ$). Also in the non-BA aneurysm group, the α was significantly wider than in the control group.

Aneurysm size, height, and neck presented a significant positive correlation with the α angle, which was also independently predicted by the size of the aneurysm neck in multivariate analysis. In nonaneurysmal controls, α grew wider with increasing age, with a steeper dependence in female than in male patients. In computational fluid dynamics (CFD) simulations, done on parametric BA models (with radii of mother and symmetrical daughter vessels following the vascular optimality principle) as well as on patient-derived models of the BA bifurcation, low wall shear stress (WSS) at the area of flow impingement at the bifurcation apex was spatially contiguous with an acceleration area along the daughter branches (where WSS

increased abruptly to maximal values) that was followed by a deceleration area (where WSS gradually decreased). Both the flow impingement area and the acceleration area increased with increasing α angles on CFD simulations. The vertebrobasilar junction angles were also evaluated, but no significant geometrical changes were observed in any patient group.

It is appealing to speculate that the observed strong correlation between the BA α angle and aneurysm presence, and the finding that in patients with aneurysms at sites other than the BA the α angle was significantly wider than in the control group (the authors suggested that this denotes a global arterial weakening at bifurcation sites throughout the cerebral circulation in patients with aneurysms), indicates that an increased α angle can precede aneurysm formation. The authors cautiously and correctly dismissed such a causal relation, but this hypothesis surely is worth keeping in mind in future observations (prospective observations would be necessary to ascertain temporal priority). Moreover, the difference in α angles in the BA aneurysm group and in the non-BA aneurysm group (35° on average) was much larger than the difference between the non-BA cases and the controls (8.7°), and can reasonably result from aneurysm formation and progression.

Apart from the intuitively understandable fact that a saccular aneurysm is a focus of vessel wall rupture and that a lumen herniation in a pouch of vessel wall remnants usually located just at the apex of the α angle would increase this angle even more, in terminal aneurysms every systolic blood pressure wave exposes the aneurysm sac to the “blood-hammer” effect.^{1,2} This phenomenon, known in hydraulic engineering as the “water hammer,” occurs when flow of fluid in a pipe is stopped by abrupt closure of a valve, reducing the kinetic energy of the upstream fluid to zero, briefly creating an overpressure at the valve that adds to the steady pressure, and causing a pressure wave to move upstream at the speed of sound that is followed by secondary (“bouncing”) waves until the fluid comes to rest. Ahlqvist¹ provided a simple equation to estimate the overpressure (ΔP): $\Delta P = v_p [\rho (K + d/Eh)]^{1/2}$.

Assuming physiological or nearly analogous values for blood flow velocity (v), blood density (ρ), blood compressibility (K), vessel wall thickness (h), and elastic modulus (E) as provided by Ahlqvist¹ in his hypothetical considerations about embolic occlusion of the middle cerebral artery, but adjusting the internal arterial diameter (d) to 3.5 mm for the BA and assuming the presence of a BA tip aneurysm whose neck is as wide as the BA, the overpressure provoked by the impact of the blood flow at the domus of the aneurysm, that is reflected upstream in the BA bifurcation, is approximately 55 mm Hg.

In other words, at the apex of the BA aneurysm sac a sudden arterial pressure increase of 55 mm Hg is added to the blood pressure at the parent artery at every heartbeat, and this pressure wave is transmitted upstream along the aneurysm wall to the aneurysm ostium and parent artery. The overpressure is added to and does not depend on the baseline arterial blood pressure. Compared to abrupt embolic occlusion of an artery, the primary wave probably spreads along a shorter distance (the posterior cerebral

arteries may function as a shunting system that damps the primary overpressure wave) and thus with less intense secondary waves, if any, but a new primary wave is produced after the next heartbeat.

This estimate, even if admittedly rough, indicates an amplification of the hemodynamic stresses in terminal BA aneurysms that could drive the BA tip upward, and increase the α angle over time. The blood-hammer effect is intuitively similar to the impingement force, because both express the transformation of kinetic energy in pressure load at the aneurysm domus. The blood-hammer effect can also be related to the propensity of wide-necked terminal BA and middle cerebral artery aneurysms to recur after simple coiling procedures, and the necessity to complement the endovascular treatment of many of these aneurysms with stents.

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DISCLOSURE

The author reports no conflict of interest.

References

1. Ahlqvist J: Stress-related intracerebral hemorrhage and the water-hammer effect. **Stroke** **32**:277–278, 2001 (Letter)
2. Damsa T, Appel E, Cristidis V: “Blood-hammer” phenomenon in cerebral hemodynamics. **Math Biosci** **29**:193–202, 1976
3. Tütüncü F, Schimansky S, Baharoglu MI, Gao B, Calnan D, Hippelheuser J, et al: Widening of the basilar bifurcation angle: association with presence of intracranial aneurysm, age, and female sex. **J Neurosurg** **121**:1401–1410, 2014

Response

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

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Minimally invasive technique for epilepsy surgery

TO THE EDITOR: We read with great interest the article by Quigg and Harden⁷ (Quigg M, Harden C: Minimally invasive techniques for epilepsy surgery: stereotactic radiosurgery and other technologies. *J Neurosurg* **121** (Suppl 2):232–240, December 2014). Surprisingly, the authors completely missed the recent progress of stereotactic radiofrequency amygdalohippocampectomy for mesial temporal lobe epilepsy (MTLE), a recently modified method that is the only one having seizure control results comparable with anterior temporal resection. Only less effective stereotactic treatments for MTLE published by Parrent and Blume⁶ in 1999 and by Guénot et al.¹ are mentioned.

We would like to focus readers' attention on our stereotactic radiofrequency method, which has been used since 2004. For radiofrequency amygdalohippocampectomy the occipital access is used and thermolesions are administered by an electrode with a 10-mm bold active tip; usually 24 lesions are made in 8 segments. Thermocoagulative lesions are made in the long axis of the amygdalohippocampal complex (AHC).² After surgery, thermocoagulative necrosis affects nearly the whole AHC. One year after surgery an irregular pseudocyst developed in the AHC, which caused partial destruction of AHC, the entorhinal and perirhinal cortices included.⁵ As of this writing we have treated 63 patients with MTLE, and we recently published long-term clinical seizure outcomes in the group of 61 patients. The mean clinical seizure follow-up was 5.3 years, and 70.5% of our treated patients have been seizure free since surgery (Engel I).⁸ Neuropsychological outcomes were very good; patients did not decline in any memory parameters 1 and 2 years after therapy, despite the fact that two-thirds of patients were treated on the left side.^{3,4} We are not aware of any other stereotactic method that achieves similar clinical outcomes that are fully comparable with open surgery procedures.

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DISCLOSURE

The authors report no conflict of interest.

References

1. Guénot M, Isnard J, Ryvlin P, Fischer C, Mauguière F, Sindou M: SEEG-guided RF thermocoagulation of epileptic foci: feasibility, safety, and preliminary results. **Epilepsia** **45**:1368–1374, 2004
2. Liscak R, Malikova H, Kalina M, Vojtech Z, Prochazka T, Marusic P, et al: Stereotactic radiofrequency amygdalohippocampectomy in the treatment of mesial temporal lobe epilepsy. **Acta Neurochir (Wien)** **152**:1291–1298, 2010
3. Malikova H, Kramská L, Vojtech Z, Liscak R, Sroubek J, Lukavsky J, et al: Different surgical approaches for mesial temporal epilepsy: resection extent, seizure, and neuropsychological outcomes. **Stereotact Funct Neurosurg** **92**:372–380, 2014
4. Malikova H, Kramská L, Vojtech Z, Lukavsky J, Liscak R: Stereotactic radiofrequency amygdalohippocampectomy: two years of good neuropsychological outcomes. **Epilepsy Res** **106**:423–432, 2013
5. Malikova H, Liscak R, Vojtech Z, Prochazka T, Vymazal J, Vladyka V, et al: Stereotactic radiofrequency amygdalohippocampectomy: does reduction of entorhinal and perirhinal cortices influence good clinical seizure outcome? **Epilepsia** **52**:932–940, 2011
6. Parrent AG, Blume WT: Stereotactic amygdalohippocampectomy for the treatment of medial temporal lobe epilepsy. **Epilepsia** **40**:1408–1416, 1999
7. Quigg M, Harden C: Minimally invasive techniques for epilepsy surgery: stereotactic radiosurgery and other technologies. **J Neurosurg** **121** (Suppl 2):232–240, 2014
8. Vojtěch Z, Malíková H, Krámská L, Anýž J, Syruček M,

Zámečník J, et al: Long-term seizure outcome after stereotactic amygdalohippocampectomy. *Acta Neurochirurgica (Wien)* 156:1529–1537, 2014

Response

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

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Malignant meningiomas

TO THE EDITOR: I read with great interest the paper by Sughrue et al.¹⁴ (Sughrue ME, Sanai N, Shangari G, et al: Outcome and survival following primary and repeat surgery for World Health Organization Grade III meningiomas. *J Neurosurg* 113:202–209, August 2010), and I would like to ask the authors some questions.

1. In the *Abstract* the authors wrote they studied all malignant meningioma cases operated on over a 16-year period while subsequently, in the *Patient Demographics* paragraph, it is stated that the 63 cases of malignant meningioma included in the study were taken from a total of 1228 meningiomas evaluated over a 22-year period. Does this mean that the authors didn't observe malignant meningioma cases over a 6-year period?

2. The sex ratio of malignant meningioma patients studied by Sughrue et al. shows a female predominance. This comes as a bit of a surprise to me, since previous studies on malignant meningiomas point to the opposite (i.e., a definite male predominance).^{7–9,15} At the same time, it should be noted that an equal male-to-female ratio^{7,9,15} or even a slight female predominance^{2,3} has occasionally been found for patients with atypical meningiomas.

3. One third of the malignant meningioma patients who were surgically treated by the authors were asymptomatic. Presumably, these lesions were incidentally discovered. How many incidental/asymptomatic meningiomas did the authors observe during the study period, and what was the global incidence of malignant forms among them? How long was the mean observation period before the discovery of malignant progression? In the literature on incidental/asymptomatic meningiomas,^{5,16,17} the incidence of malignant meningiomas among the small number of patients who needed surgery was of 1.5%, and that of atypical meningiomas was of 4.5%—similar to what is commonly observed in primary symptomatic meningiomas.¹⁵ Since 29 of 63 patients had only the second surgery at the University of California, San Francisco (UCSF), it can be assumed that the incidence of asymptomatic/incidental cases among patients with malignant meningiomas having initial surgery at UCSF was surprisingly high, at 62% (21/34). Is this so? In my opinion, a possible partial explanation could

be the overgrading of cases of atypical meningioma submitted to preoperative embolization. How many patients had undergone preoperative embolization in Sughrue and colleagues' case series of malignant meningiomas? In the literature there has been some concern about the possibility of overgrading of preoperatively embolized benign meningiomas.^{4,8,10,11} Can the authors exclude the possibility of having classified as malignant cases of atypical meningioma that were embolized before resection? Given such doubt, did the authors confirm the malignancy of their cases by means of a cytogenetic analysis?¹

4. Perhaps the most surprising result of this study is that patients who had subtotal resection (STR) fared better than those who underwent gross-total resection (GTR), a finding not explained by the authors. Here again, I think preoperative embolization might matter. Such a procedure not only could have led to histological overgrading of an otherwise atypical meningioma as proposed above but, as hypothesized by the authors themselves in their paper on benign meningiomas,¹³ also could have induced better control of residual tumor after STR through its involution following devascularization and partial infarction. Sughrue et al.¹⁴ wrote: "Preoperative embolization was performed for larger tumors...." What actually was the respective incidence of such procedures in the GTR and STR groups? Another potential confounding variable leading to the suggested benefit of STR over GTR in the series studied by Sughrue et al. lies in the concept of grouping together Simpson Grade I, II, and III resections. It cannot be denied that a true total resection of a meningioma, classically defined as Simpson Grade I, can be obtained with confidence in convexity tumors only.⁶ Accordingly, a paper from the same San Francisco group¹² showed recurrence rates of 0% and 17% after Simpson Grade 0–I and Grade II resection, respectively, in their series of benign convexity meningiomas. In our series of 29 malignant meningiomas,⁹ we found that Simpson Grade I resection was successful only in convexity tumors. Simpson Grade II or III actually means incomplete resection, even if less incomplete than Simpson Grade IV. In a case of malignant meningioma it is hard for me to imagine what a difference it could make to have many cells inside the dura mater (Simpson Grade II–III) or a small cluster (Simpson IV) left behind. In addition it should be considered that the biology of meningiomas, whether benign or malignant, is so far largely unknown and highly variable. So it cannot reasonably be excluded that patients with malignant meningioma undergoing Simpson Grade II–III resection and included in the GTR group had biologically more malignant lesions and then had a more downhill clinical course than patients included in STR group.

In conclusion, before reversing the intuitive benefit of radical resection on survival in their series of malignant meningiomas, I suggest that Sughrue et al. should separate the cases of convexity tumors with Simpson Grade I resection to be compared to all the others. Notwithstanding all the above observations, I think this is an excellent paper and I have enjoyed reading it very much.

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DISCLOSURE

The author reports no conflict of interest.

References

1. Carvalho LH, Smirnov I, Baia GS, Modrusan Z, Smith JS, Jun P, et al: Molecular signatures define two main classes of meningiomas. *Mol Cancer* **6**:64, 2007
2. Hufmann BC, Reinacher PC, Gilsbach JM: Gamma knife surgery for atypical meningiomas. *J Neurosurg* **102 Suppl**:283–286, 2005
3. Hug EB, Devries A, Thornton AF, Munzenride JE, Pardo FS, Hedley-Whyte ET, et al: Management of atypical and malignant meningiomas: role of high-dose, 3D-conformal radiation therapy. *J Neurooncol* **48**:151–160, 2000
4. Jiménez-Heffernan JA, Corbacho C, Canizal JM, Pérez-Campos A, Vicandi B, López-Ibor L, et al: Cytological changes induced by embolization in meningiomas. *Cytopathology* **23**:57–60, 2012
5. Jo KW, Kim CH, Kong DS, Seol HJ, Nam DH, Park K, et al: Treatment modalities and outcomes for asymptomatic meningiomas. *Acta Neurochir (Wien)* **153**:62–67, 2011
6. Kinjo T, al-Mefty O, Kanaan I: Grade zero removal of supratentorial convexity meningiomas. *Neurosurgery* **33**:394–399, 1993
7. Krayenbühl N, Pravdenkova S, Al-Mefty O: De novo versus transformed atypical and anaplastic meningiomas: comparison of clinical course, cytogenetics, cytokinetics, and outcome. *Neurosurgery* **61**:495–504, 2007
8. Mohda A, Gutin PH: Diagnosis and treatment of atypical and anaplastic meningiomas: a review. *Neurosurgery* **57**:538–550, 2005
9. Palma L, Celli P, Franco C, Cervoni L, Cantore G: Long-term prognosis for atypical and malignant meningiomas: a study of 71 surgical cases. *J Neurosurg* **86**:793–800, 1997
10. Paulus W, Meixensberger J, Hofmann E, Roggendorf W: Effects of embolisation of meningioma on Ki-67 proliferation index. *J Clin Pathol* **46**:876–877, 1993
11. Perry A, Chicoine MR, Filiput E, Miller JP, Cross DT: Clinicopathologic assessment and grading of embolized meningiomas: a correlative study of 64 patients. *Cancer* **92**:701–711, 2001
12. Sanai N, Sughrue ME, Shangari G, Chung K, Berger MS, McDermott M: Risk profile associated with convexity meningioma resection in the modern neurosurgical era. *J Neurosurg* **112**:913–919, 2010
13. Sughrue ME, Kane AJ, Shangari G, Rutkowski MJ, McDermott MW, Berger MS, et al: The relevance of Simpson Grade I and II resection in modern neurosurgical treatment of World Health Organization Grade I meningiomas. *J Neurosurg* **113**:1029–1035, 2010
14. Sughrue ME, Sanai N, Shangari G, Parsa AT, Berger MS, McDermott MW: Outcome and survival following primary and repeat surgery for World Health Organization Grade III meningiomas. *J Neurosurg* **113**:202–209, 2010
15. World Health Organization: **World Health Organization Classification of Tumors: Tumors of the Central Nervous System: Pathology and Genetics**. Lyons: IARC Press, 2000
16. Yano S, Kuratsu J, Kumamoto Brain Tumor Research Group: Indications for surgery in patients with asymptomatic meningiomas based on an extensive experience. *J Neurosurg* **105**:538–543, 2006
17. Yoneoka Y, Fujii Y, Tanaka R: Growth of incidental meningiomas. *Acta Neurochir (Wien)* **142**:507–511, 2000

Endoscopic transnasal transcribriiform approach

TO THE EDITOR: The article by Banu et al.² is an important contribution to the analysis of predictive factors of postoperative CSF leaks after endoscopic skull base surgery (Banu MA, Szentirmai O, Mascarenhas L, et al: Pneumocephalus patterns following endonasal endoscopic skull base surgery as predictors of postoperative CSF leaks. *J Neurosurg* **121**:961–975, October 2014). However, we think that the term “transcribriiform” has been misused in this article.

Although the endoscopic approach to the anterior skull base had been already described in several case series,^{4,5,11,13,17,21,28,29} it was definitely systematized in 2004 by Jho and Ha,¹⁵ in 2005 by Har-El and Casiano,¹⁴ and, in the same year, by Kassam et al.¹⁸ These authors introduced in their article the term “transcribriiform” to refer to the endoscopic approach to the anterior skull base. In the following years, the indication of the endoscopic route for the anterior skull base lesions had been progressively expanded, and it became common for authors to start using the term “transcribriiform” to refer to the demolition of the cribriform plate and fovea ethmoidalis, thus describing an approach that implied the demolition of the anterior part of the ethmoidal roof. Using the term “transcribriiform” in these cases, in our opinion, is not correct because it describes only the removal of the cribriform plate and therefore, it is not sufficient, in several cases, for creating a surgical corridor that is useful to approach the anterior skull base lesions, with the exception of CSF leaks.

An accurate description of the anatomy of the cribriform plate and anterior skull base has been given in the literature,^{19,26} in particular, the analysis of dimensions and asymmetries, proposing various classifications on the basis of form¹⁹ and height of the lateral lamina.²⁰ According to the article by Vasvari et al.,²⁶ the dimensions of the cribriform plate are on average 20.7 mm in length (range 14.1–28.4 mm), 3.1 mm in width at the tip of the crista galli (0.1–4.2 mm), and 5.7 mm in width at the tip of the ethmoidal spine (2.6–9.3 mm). Recent neuroradiological studies^{1,9,10,16,30} have confirmed the descriptions of Vasvari et al. As we mentioned earlier, Jho and Ha¹⁵ and Har-El and Casiano¹⁴ were the first to describe the approach to the anterior skull base more systematically. The milestone description regarding the endoscopic approach to the anterior skull base is provided in the article by Kassam et al.¹⁸ and consists of the following surgical steps: resection of the nasal septum, exposure and demucosization of the cribriform plate with cutting of the olfactory filament and branches of the ethmoidal arteries, and drilling of the crista galli and the olfactory sulcus until exposure of the dura mater. According to Kassam et al., the approach to the anterior skull base was thus divided into 2 procedures: transcribriiform and transplanum.

Since then, in our opinion, the term “transcribriiform” has been commonly used incorrectly, indicating indeed the approach to the anterior part of the skull base and usually being associated with “transplanum” to indicate the approach to the entire anterior skull base.^{3,6,7,8,22–25,27}

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We think that using the term “transcribriiform” to indicate the endoscopic approach to the anterior skull base is quite synecdochic and misleading. In our opinion, a descriptive name has to correctly represent the approach, reflecting the real surgical procedure and not only a part of it. When a skull base team deals with tumors of the anterior skull base, the demolition steps involve of course not only the cribriform plate but possibly reach laterally to the superomedial margins of the orbit.

Is the “transcribriiform-transplanum approach” really representative of the anterior skull base approaches? Anatomically speaking, does the so-called transcribriiform approach only involve the cribriform plate, or is the resection extended to the fovea ethmoidalis of the frontal bone?

The anatomical measures of the cribriform plate are sufficient to clarify that the approach can be purely transcribriiform, and sometimes unilateral, only in select cases (CSF leak repair) and will have to be extended bilaterally to the ethmoidal fovea in cases of intracranial lesions.

The uniformity of nomenclature is an important feature to improve the communication among different groups, and we support the idea that representative and descriptive definitions could be a strong starting point to share and improve our knowledge.

We think that the term “transcribriiform transfovea ethmoidalis” (used only once in the literature¹²) is to be preferred to indicate the endoscopic approach to the anterior skull base in cases of dural exposure from orbit to orbit.

On the basis of our analysis, we think that the endoscopic endonasal approaches to the anterior skull base could be classified as follows:

Transethmoidal: when a partial or complete ethmoidectomy and only extracranial procedure has been performed;

Transcribriiform: when only the cribriform plate is resected and some intracranial procedure has been performed;

Transfovea: when only the fovea is resected and some intracranial procedure has been performed;

Transcribriiform-transfovea: when the anterior part of the ethmoidal roof is resected from orbit to orbit;

Transcribriiform-transfovea-transplanum: when the entire ethmoidal roof is resected to access the anterior cranial base.

Sometimes, the most common scientific terms are semantically incorrect and it is mandatory to correct any evident error in order to create a common and shared lexicon that is accepted by the entire scientific community. The term “transcribriiform” has been commonly used incorrectly when it indicates the standard endoscopic approach to the anterior skull base with the complete demolition of the ethmoidal roof from orbit to orbit. Actually, the anatomically appropriate term to indicate this approach is “transcribriiform transfovea,” whereas only the term “transcribriiform” should be used when the bone demolition involves the cribriform plate.

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DISCLOSURE

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References

1. Arikan OK, Unal B, Kazkayasi M, Koc C: The analysis of anterior skull base from two different perspectives: coronal and reconstructed sagittal computed tomography. **Rhinology** 43:115–120, 2005
2. Banu MA, Szentirmai O, Mascarenhas L, Salek AA, Anand VK, Schwartz TH: Pneumocephalus patterns following endonasal endoscopic skull base surgery as predictors of postoperative CSF leaks. **J Neurosurg** 121:961–975, 2014
3. Batra PS, Lee J, Barnett SL, Senior BA, Setzen M, Kraus DH: Endoscopic skull base surgery practice patterns: survey of the North American Skull Base Society. **Int Forum Allergy Rhinol** 3:659–663, 2013
4. Boseley ME, Tami TA: Endoscopic management of anterior skull base encephaloceles. **Ann Otol Rhinol Laryngol** 113:30–33, 2004
5. Casiano RR, Numa WA, Falquez AM: Endoscopic resection of esthesioneuroblastoma. **Am J Rhinol** 15:271–279, 2001
6. Eloy JA, Choudhry OJ, Shukla PA, Kuperan AB, Friedel ME, Liu JK: Nasoseptal flap repair after endoscopic transsellar versus expanded endonasal approaches: is there an increased risk of postoperative cerebrospinal fluid leak? **Laryngoscope** 122:1219–1225, 2012
7. Eloy JA, Patel SK, Shukla PA, Smith ML, Choudhry OJ, Liu JK: Triple-layer reconstruction technique for large cribriform defects after endoscopic endonasal resection of anterior skull base tumors. **Int Forum Allergy Rhinol** 3:204–211, 2013
8. Eloy JA, Shukla PA, Choudhry OJ, Singh R, Liu JK: Assessment of frontal lobe sagging after endoscopic endonasal transcribriiform resection of anterior skull base tumors: is rigid structural reconstruction of the cranial base defect necessary? **Laryngoscope** 122:2652–2657, 2012
9. Elwany S, Medanni A, Eid M, Aly A, El-Daly A, Ammar SR: Radiological observations on the olfactory fossa and ethmoid roof. **J Laryngol Otol** 124:1251–1256, 2010
10. Erdem G, Erdem T, Miman MC, Ozturan O: A radiological anatomic study of the cribriform plate compared with constant structures. **Rhinology** 42:225–229, 2004
11. Gjuric M, Goede U, Keimer H, Wigand ME: Endonasal endoscopic closure of cerebrospinal fluid fistulas at the anterior cranial base. **Ann Otol Rhinol Laryngol** 105:620–623, 1996
12. Greenfield JP, Anand VK, Kacker A, Seibert MJ, Singh A, Brown SM, et al: Endoscopic endonasal transethmoidal transcribriiform transfovea ethmoidalis approach to the anterior cranial fossa and skull base. **Neurosurgery** 66:883–892, 2010
13. Handley GH, Goodson MA, Real TH: Transnasal endoscopic closure of anterior fossa cerebrospinal fluid fistula. **South Med J** 86:217–219, 1993
14. Har-El G, Casiano RR: Endoscopic management of anterior skull base tumors. **Otolaryngol Clin North Am** 38:133–144, ix, 2005

15. Jho HD, Ha HG: Endoscopic endonasal skull base surgery: Part 1—The midline anterior fossa skull base. **Minim Invasive Neurosurg** 47:16–23, 2004
16. Jones TM, Almahdi JM, Bhalla RK, Lewis-Jones H, Swift AC: The radiological anatomy of the anterior skull base. **Clin Otolaryngol Allied Sci** 27:101–105, 2002
17. Kaptain GJ, Vincent DA, Sheehan JP, Laws ER Jr: Trans-sphenoidal approaches for the extracapsular resection of midline suprasellar and anterior cranial base lesions. **Neurosurgery** 49:94–101, 2001
18. Kassam A, Snyderman CH, Mintz A, Gardner P, Carrau RL: Expanded endonasal approach: the rostrocaudal axis. Part I. Crista galli to the sella turcica. **Neurosurg Focus** 19(1):E3, 2005
19. Kawahara G, Matsuda M, Sugiyama K, Nakazawa R, Shima K: [Studies on the Japanese lamina cribrosa—statistical observation on its shape, number of pores and area.] **Zasshi Tokyo Ika Daigaku** 26:185–194, 1968 (Jpn)
20. Keros P: [On the practical value of differences in the level of the lamina cribrosa of the ethmoid.] **Z Laryngol Rhinol Otol** 41:809–13, 1962 (Ger)
21. Kuhn FA, Javer AR: Low-grade fibrosarcoma of the anterior skull base: endoscopic resection and repair. **Am J Rhinol** 17:347–350, 2003
22. Lee JM, Ransom E, Lee JY, Palmer JN, Chiu AG: Endoscopic anterior skull base surgery: intraoperative considerations of the crista galli. **Skull Base** 21:83–86, 2011
23. Lee JT, Kingdom TT, Smith TL, Setzen M, Brown S, Batra PS: Practice patterns in endoscopic skull base surgery: survey of the American Rhinologic Society. **Int Forum Allergy Rhinol** 4:124–131, 2014
24. Liu JK, Christiano LD, Patel SK, Tubbs RS, Eloy JA: Surgical nuances for removal of olfactory groove meningiomas using the endoscopic endonasal transcribriform approach. **Neurosurg Focus** 30(5):E3, 2011
25. Liu JK, Eloy JA: Expanded endoscopic endonasal transcribriform approach for resection of anterior skull base olfactory schwannoma. **Neurosurg Focus (Suppl)** 32(1):Video 3, 2012
26. Vasvari G, Reisch R, Patonay L: Surgical anatomy of the cribriform plate and adjacent areas. **Minim Invasive Neurosurg** 48:25–33, 2005
27. Verillaud B, Bresson D, Sauvaget E, Mandonnet E, Georges B, Kania R, et al: Transcribriform and transplanum endoscopic approach for skull-base tumors. **Eur Ann Otorhinolaryngol Head Neck Dis** 130:233–236, 2013
28. Weiss DD, Robson CD, Mulliken JB: Transnasal endoscopic excision of midline nasal dermoid from the anterior cranial base. **Plast Reconstr Surg** 102:2119–2123, 1998
29. Wigand ME, Hosemann WG: Results of endoscopic surgery of the paranasal sinuses and anterior skull base. **J Otolaryngol** 20:385–390, 1991
30. Zacharek MA, Han JK, Allen R, Weissman JL, Hwang PH: Sagittal and coronal dimensions of the ethmoid roof: a radio-anatomic study. **Am J Rhinol** 19:348–352, 2005

Response

We thank the authors for providing this thoughtful discussion of the nomenclature and anatomy of the endonasal approach to the anterior fossa. The authors are correct to point out that the term “transcribriform approach” is often used incorrectly to refer to either the actual transcribriform approach or the transethmoidal-transfovea-ethmoidalis approach, or a combination of the two.¹ As the authors mention, our group at Cornell discussed this distinction in our paper on the subject. We agree with the authors that it is critically important to differentiate the two approaches

since the transcribriform approach by itself does not, in principle, require one to open the ethmoid air cells. However, in practice it is almost impossible to appropriately address pathology of the cribriform plate without also opening the ethmoid air cells since the corridor is so narrow. In the paper in question, we used the terminology “transcribriform-transethmoidal” to refer to the combination of approaches that included the “transcribriform,” the “transethmoidal-transfovea-ethmoidalis,” and the combination of the two approaches. The approaches were grouped together so that we could have high enough numbers of cases to try and achieve statistical significance with regard to patterns of pneumocephalus. Nevertheless, we should have been more clear about our definitions and very much appreciate the opportunity to clarify our meaning.

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Reference

1. Greenfield JP, Anand VK, Kacker A, Seibert MJ, Singh A, Brown SM, et al: Endoscopic endonasal transethmoidal transcribriform transfovea ethmoidalis approach to the anterior skull base and anterior cranial fossa. **Neurosurgery** 66:883–892, 2010

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Perianeurysmal edema

TO THE EDITOR: We read with great interest the article by Pahl et al.⁴ (Pahl FH, de Oliveira MF, Ferreira NPFD, et al: Perianeurysmal edema as a predictive sign of aneurysmal rupture. *J Neurosurg* 121:1112–1114, November 2014). The authors reported on 2 cases of middle-aged women with previous histories of headaches in which MRI examinations revealed the presence of surrounding parenchymal edema related to aneurysms. These findings were attributed to a progressive inflammatory process possibly triggered by enlargement of the bleb formation.⁴ In a recent report by Nussbaum et al., the authors (one of whom is an author of this letter) described 13 patients with hemosiderin staining of the pial surface immediately adjacent to the aneurysm dome, suggesting a remote and unrecognized history of microbleeding from an aneurysm.³ We identified that those patients presented with a recurrent history of episodic, unusual type of headaches, mimicking flu-like symptoms lasting for several days. In all cases, a diagnosis of a sentinel bleed or subarachnoid hemorrhage had never been suggested, and no patient had been admitted to the hospital for formal evaluation of prior headache episodes. In addition, the intervals between these headaches shortened between clinical presentations.³

Similar to those reported by Pahl et al., most cases in our series were female patients; 53.8% of these patients had a history of smoking, 30.8% had hypertension, and 23.1% had a history of alcohol abuse. Dyslipidemia and a family history of aneurysms were present in 15.4% of the patients, and hypercholesterolemia was noted in 1 patient (8%). The aneurysms were considered particularly “thin-walled” in 8 cases.³

Some authors have highlighted the role of surrounding brain edema as an early manifestation in the course to aneurysm rupture.¹ They have also suggested that the edema might be a result of a progressive inflammatory process culminating with rupture.⁴ We are very aware of the fact that inflammation and apoptosis occur intraluminally, from the intima-internal elastic lamina layer (triggered by hemodynamic stress) and spread toward the media and adventitia.⁵ A description by Pahl et al. of perianeurysmal edema portrays it as basically intraparenchymal in location. The possibility exists that perianeurysmal edema is nothing more than a progressive inflammatory process caused by a microbleed at the aneurysm site.

Finally, based on prior reports, it may also be possible to identify radiological evidence of minor hemorrhages by performing thin-cut studies utilizing T2 gradient recall echo or MRI-susceptibility weighted imaging (SWI) techniques. MRI-SWI is 3–6 times more sensitive than conventional T2-weighted gradient echo sequences for hemosiderin and perianeurysmal edema detection.² We are currently investigating the use of this technology when evaluating patients with “unruptured” aneurysms who present with a suspicious history of remote atypical headache.²

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DISCLOSURE

The authors report no conflict of interest.

References

1. Hiu T, Tsutsumi K, Kitagawa N, Hayashi K, Ujifuku K, Yasunaga A, et al: Progressive perianeurysmal edema preceding the rupture of a small basilar artery aneurysm. *Clin Neurol Neurosurg* **111**:216–219, 2009
2. Mittal S, Wu Z, Neelavalli J, Haacke EM: Susceptibility-weighted imaging: technical aspect and clinical applications, part 2. *AJNR Am J Neuroradiol* **30**:232–52, 2009
3. Nussbaum ES, Defillo A, Zelensky A, Pulivarthi S, Nussbaum L: “Microbleeding” from intracranial aneurysms: Local hemosiderin deposition identified during microsurgical treatment of unruptured intracranial aneurysms. *Sur Neurol Int* **5**:28, 2014
4. Pahl FH, de Oliveira MF, Ferreira NPFD, de Macedo LL, Brock RS, de Souza VC: Perianeurysmal edema as a predictive sign of aneurysmal rupture. *J Neurosurg* **121**:1112–1114, 2014
5. Turjman AS, Turjman F, Edelman ER: Role of fluid dynamics and inflammation in intracranial aneurysm formation. *Circulation* **129**:373–382, 2014

Response

We completely agree with points stated by Defillo and Kennedy in their letter. Subarachnoid hemorrhage (SAH) following intracranial aneurysmal rupture is a major cause of morbidity and mortality. Much effort has been made toward avoiding aneurysmal rupture and establishing prediction patterns. Although several factors may interfere with the probability of rupture, such as smoking; use of alcohol; size, shape, and location of the aneurysm; presence of intraluminal thrombus; and even the sex of the patient, there are still scarce data to correlate such findings with the timing of aneurysmal rupture.

In this light, after a pooled analysis of 6 prospective cohort studies, Greving et al.¹ proposed a score to estimate the 5-year aneurysm rupture risk (PHASES score). They evaluated ruptures that occurred in 230 patients during 29,166 person-years of follow-up. The mean observed 1-year risk of aneurysm rupture was 1.4% (95% CI 1.1%–1.6%) and the 5-year risk was 3.4% (95% CI 2.9%–4.0%).

Predictors were age, hypertension, history of SAH, aneurysm size, aneurysm location, and geographic region. According to the given score, which may vary from 0 to 22 points, 5-year rupture risk may be between 0.4% and 17.8%.¹

Additionally, Korja et al.² published their observations after following 118 Finnish patients with unruptured aneurysms until their death or SAH. Twenty-nine percent of patients presented with SAH during lifelong follow-up. The annual rupture rate per patient was 1.6%. They found that female sex, current smoking, and aneurysm size of 7 mm or greater in diameter were risk factors for a lifetime SAH, and, depending on the risk factor burden, the lifetime risk of an aneurysmal SAH varied from 0% to 100%. The still intriguing finding was that even among the 96 patients with small (< 7 mm) unruptured aneurysms, 24 (25%) had an aneurysmal SAH during the follow-up.²

Those reports, as well as other studies, have identified factors clearly related to aneurysmal bleeding. However, such factors were still linked to demographic and epidemiological data and anatomical characteristics of the aneurysm. Nevertheless, the finding that even small aneurysms bleed together with the failure to prevent aneurysmal bleeding in low-risk patients has brought attention to microstructural and chemical environment involved in the development of aneurysms and their rupture.

The intraoperative finding of microbleeding adjacent to incidental aneurysms submitted to microsurgery was reported by Nussbaum et al.⁴ It reinforces the underlying microstructural pathophysiology of aneurysmal rupture, which probably involves regional blood flow disturbances and the presence of an inflammatory process, allowing for bleb formation, enlargement of a cerebral aneurysm, and microbleeding before definitive bleeding.^{4,5}

The marker of such a microscopic phenomenon is the surrounding tissue edema, revealing inflammatory status near the aneurysm. Such edema may probably be the origin of oligosymptomatic headaches in patients harboring such findings, mimicking flu-like symptoms and happening especially in middle-aged women.⁴ Such edema probably has a temporal link with risk of rupture, including risk of immediate rupture.

Magnetic resonance imaging utilizing T2 gradient recalled echo or MRI-SWI techniques to evaluate perianeurysmal edema and perianeurysmal hemosiderin deposits become essential tools in the routine assessment of unruptured aneurysms.³ Magnetic resonance spectroscopy with evaluation of chemical environment surrounding aneurysms may also be a potential target for future discussions.

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References

1. Greving JP, Wermer MJ, Brown RD Jr, Morita A, Juvela S, Yonekura M, et al: Development of the PHASES score for prediction of risk of rupture of intracranial aneurysms: a

pooled analysis of six prospective cohort studies. **Lancet Neurol** **13**:59–66, 2014

2. Korja M, Lehto H, Juvela S: Lifelong rupture risk of intracranial aneurysms depends on risk factors: a prospective Finnish cohort study. **Stroke** **45**:1958–1963, 2014
3. Mittal S, Wu Z, Neelavalli J, Haacke EM: Susceptibility-weighted imaging: technical aspect and clinical applications, part 2. **AJNR Am J Neuroradiol** **30**:232–252, 2009
4. Nussbaum ES, Defillo A, Zelensky A, Pulivarthi S, Nussbaum L: “Microbleeding” from intracranial aneurysms: Local hemosiderin deposition identified during microsurgical treatment of unruptured intracranial aneurysms. **Surg Neurol Int** **5**:28, 2014
5. Turjman AS, Turjman F, Edelman ER: Role of fluid dynamics and inflammation in intracranial aneurysm formation. **Circulation** **129**:373–382, 2014

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