Neurosurgical Forum

LETTERS TO THE EDITOR

Shunt dependency following aSAH

TO THE EDITOR: We read with great interest the article by Winkler et al.⁸ (Winkler EA, Burkhardt JK, Rutledge WC, et al: Reduction of shunt dependency rates following aneurysmal subarachnoid hemorrhage by tandem fenestration of the lamina terminalis and membrane of Lilliequist during microsurgical aneurysm repair. *J Neurosurg* [epub ahead of print December 15, 2017. DOI: 10.3171/2017.5.JNS163271]). Hydrocephalus following subarachnoid hemorrhage (SAH) is still a significant problem. In their article, Winkler and colleagues report the results of a retrospective review of 663 consecutive cases of aneurysmal SAH (aSAH) in which patients were treated by open microsurgery. They found that tandem fenestration of the lamina terminalis (LT) and membrane of Lilliequist (MoL) at the time of open microsurgical clipping and/or bypass to secure ruptured anterior and posterior circulation aneurysms was associated with reductions in shunt-dependent hydrocephalus following aSAH.

We have some concerns about their results. In SAH, it is assumed that aSAH changes CSF dynamics through 2 major pathways: 1) anatomic obstruction in ventricles and/or cisterns blocked by blood products and 2) reduced absorption from impaired arachnoid granulations.⁴ The authors demonstrated a marked and statistically significant 82% reduction in shunt dependency with application of this tandem fenestration technique.⁸ The study, however, is a retrospective analysis of a case series.⁸ In such studies, it is impossible to completely clarify the exact morphological pathogenetic mechanism of shunt-dependent hydrocephalus following SAH, and the histopathological data from human studies are still limited,⁴ because it is not possible to detect histopathological changes of post-SAHA hydrocephalus in patients in vivo or through autopsy.

In the last 5 years, several studies have shown the histopathological basis of post-SAHA hydrocephalus.^{1,6,10} Two of these studies^{1,6} demonstrated the water-filled vesicles as a cause of hypersecretion of CSF from choroid plexus. In the third study,¹⁰ it was shown that choroidal artery vasospasm-related aqueductal stenosis¹⁰ and the caliber of the cerebral aqueduct differ in early, middle, and late phases of hydrocephalus depending on the vasospasm of the choroidal artery. These histopathological studies have enhanced the neurologist's and neurosurgeon's view of SAH.

Recent advances in endovascular approaches have resulted in good outcomes with low rates of mortality and

morbidity,⁹ so it has been suggested that coiling should be considered a first-line treatment in aSAH.⁵ However, in patients with SAH, clot removal, which can be accomplished only by conventional microsurgery, may be important for preventing post-SAHA hydrocephalus. In a single-center clinical trial, Erixon et al. found that patients undergoing endovascular treatment were more likely to receive shunts than were patients undergoing surgical aneurysm repair.³ In another prospective study, however, Dehdashti et al. found no statistically significant benefit in terms of reduction of shunt-dependent hydrocephalus for microsurgical aneurysm treatment alone compared with endovascular treatment.² In a recent review, it was reported that there is no association between fenestration of the LT and reduced incidence of post-aSAH shunt-dependent hydrocephalus.⁷

In summary, there are conflicting results of studies on this subject, and more research is warranted.

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Disclosures

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Response

We thank Dr. Kanat for his astute comments regarding our recent manuscript describing a reduction in shunt-dependent hydrocephalus with tandem fenestration of the LT and the MoL at the time of microsurgical treatment of ruptured aneurysms. As he stated, the etiology of hydrocephalus following aSAH is complex and multifactorial, including anatomic obstruction of CSF flow with clotted blood, increases in CSF production by the choroid plexus, decreases in CSF reabsorption by arachnoid granulations, and maybe even alterations in the diameter of the cerebral aqueduct. However, the relative contributions and mechanisms governing each of these factors remain poorly understood, and hydrocephalus remains an important source of morbidity following aSAH. We agree that a better understanding of hydrocephalus after aSAH is badly needed, as are innovations to prevent and treat hydrocephalus without resorting to ventriculoperitoneal shunting.

Recent work identified that posthemorrhagic inflammation may play a role in the development of hydrocephalus through activation of toll-like receptors on the choroid plexus epithelium in experimental models.¹ A reduction in clot burden with tandem fenestration may reduce inflammation and oxidative stress induced by subarachnoid blood products, such as fibrin, thrombin, and hemoglobin.³ As we and others have described,⁴ the MoL often becomes thickened and inflamed following aSAH, leading to loculation of CSF in the prepontine and interpeduncular cisterns, which we informally refer to as the “fifth ventricle.” Restoration of CSF flow through tandem fenestration may

therefore further promote clearance of subarachnoid blood products by reopening the natural circulatory pathway of CSF and improving reabsorption of CSF by the arachnoid granulations along the brain's convexities.

As noted by Dr. Kanat, tandem fenestration of the LT and MoL requires arachnoid dissection and subarachnoid clot removal that is only possible with open surgery. Clinical experience with surgical techniques to alter CSF hydrodynamics following aSAH has been mixed. Isolated fenestration of the LT is one such technique,² but this alters CSF flow exclusively within the supratentorial compartment and does not re-establish the complete CSF circulation. We view our results as compelling but acknowledge that our analysis was both retrospective and limited to a single surgeon's experience. We were encouraged by the magnitude of benefit observed and by the low morbidity associated with these extra maneuvers, with no complications in this series. We hope that others will consider validating our findings with future prospective multi-institutional trials.

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“Burned out” at work but satisfied with one's job: anatomy of a false paradox

TO THE EDITOR: In a recent study of 346 US neurosurgery residents, Attenello et al.¹ reported that 67% of their participants suffered from burnout (Attenello FJ, Buchanan IA, Wen T, et al: Factors associated with burnout among US neurosurgery residents: a nationwide survey. *J Neurosurg* [epub ahead of print February 9, 2018. DOI: 10.3171/2017.9.JNS17996]). The authors also found that

81% of surveyed residents were satisfied with their career and that 79% would choose neurosurgery again. Attenello et al.¹ described their results—involving a coexistence of burnout and job satisfaction—as paradoxical. The authors' questioning is understandable. Based on the view that burnout reflects “a crisis in one's relationship with work,”⁹ marked by occupational frustration and vocational disappointment, one could expect burnout to be antithetical to job satisfaction.³ In our estimation, the apparent paradox attached to Attenello and colleagues' findings is accounted for by persistently ignored problems in burnout's conceptualization and measurement.

Attenello et al.¹ used the Maslach Burnout Inventory (MBI)⁹ and the cutoff scores presented in the MBI manual for identifying “cases” of burnout. The MBI, however, is not a diagnostic instrument.^{5,6,10} The cutoff scores presented in the MBI manual have no clinical underpinning; they merely reflect tercile-based splits of convenience samples of workers.^{5,9} The developers of the MBI themselves emphasized that “such arbitrary ‘cut-off’ scores do not have any diagnostic validity” and that “there is no definitive score that ‘proves’ a person is ‘burned out.’”⁹ Attenello et al.¹ thus relied on identification criteria that fail to discriminate clinically relevant symptoms of exhaustion and withdrawal from harmless variations in mood or stress and motivation levels—remarkably, experiencing detachment once per month was sufficient to be classed as “burned out” in the authors' study. This state of affairs sheds a different light on the apparently contradictory findings of the authors. That a vast majority of residents were satisfied with their job may reflect the fact that many (if not most) residents categorized as “burned out” were only experiencing benign, transitory fluctuations in their well-being at work.^{3,4}

Furthermore, although burnout has been defined as a work-induced condition,⁹ the determinants of the symptoms assessed by measures such as the MBI are far from clear.^{3,5,8} In a recent study of 468 Swiss health professionals, only 44% of the individuals presenting with burnout symptoms attributed these symptoms to their job.² Such results are consistent with Attenello and colleagues' finding that stress outside of work is a significant predictor of burnout. Many individuals reporting burnout symptoms may simultaneously be satisfied with their job for the basic reason that their symptoms are not caused by work-related difficulties.

The burnout construct is plagued by definitional problems.⁵ In the current context, relying on “burnout” as an index of occupational ill-health 1) diverts our attention from professionals who truly need help by drowning them in a sea of heterogeneous health conditions and 2) impedes our ability to identify depressogenic workplaces and managerial patterns for the purpose of implementing organizational reforms. The use of loose, catch-all categorization criteria is probably advantageous to individuals attempting to create new consulting and pharmaceutical markets, but it is highly questionable from both a methodological and a public health standpoint.^{5,7} Researchers should stop making claims about burnout's prevalence based on arbitrary criteria.

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Response

We acknowledge and appreciate the observations by Laurent et al., noting that they have published several very similar critiques in recent “Letter to the Editor” commentaries in response to clinicians employing the MBI.^{1–4,6,10} Discourse on surgeon burnout is critical to improving care in the neurosurgical community. We are therefore pleased to address the authors' points that the MBI is not a definitive diagnostic instrument, that non-workplace factors may affect resident well-being, and that burnout should not detract from consideration of psychiatric diagnoses in the workplace.

We agree that the MBI does not provide definitive psychological diagnosis. However, the MBI is widely employed throughout the physician-burnout literature as an easily administered screening tool addressing emotional exhaustion, depersonalization, and lack of accomplishment, each limiting success of health care workers.^{7,8} Our survey was

intended to shed light on a burnout phenomenon and associated modifiable factors, a goal recently espoused by MBI creator Christina Maslach.⁷ Predictably, interventions based on the MBI show positive results in residents.⁹ Though we agree that the MBI cannot provide the definitive clinical diagnosis of a more thorough battery, the 22-item questionnaire was a reasonable length for the 346 neurosurgical residents who complied and provided data.

Laurent et al. further emphasize that burnout is traditionally defined as a workplace-induced phenomenon. We agree, although we must emphasize that neurosurgical residents invariably see a blurring of home and workplace. Residents dedicate 80 hours per week to patient care, often voluntarily allocating their “non-work” time to prepare for cases, follow up on patient imaging and laboratory studies, and pursue academic research. Neurosurgical residents, like many other surgical and medical residents, do not have the traditional exclusive workplace. Not surprisingly, stressors outside of work were associated with burnout in our study.

Finally, regarding “arbitrary criteria” of the MBI diverting attention from workplace depression, we strongly disagree. The MBI is employed in hundreds of studies precisely because it is well-designed, reproducible, and validated, repeatedly showing association with personal, social, and organizational outcomes.^{5,7} Our goal was not to define psychological disease prevalence—our goal was to evaluate modifiable burnout factors, such as mentorship, shown to associate with patient care and outcomes.⁵ The authors, in their title, cite the “false paradox” of 81% resident career satisfaction despite a 67% burnout rate to highlight inadequacy of the MBI. Residents, within survey text (and later, offering response following publication) elaborated upon long-term satisfaction with improving patient outcome, developing surgical skills, and enjoying neuroscience study, although they felt patient-disconnect (depersonalized) and emotionally exhausted on a day-to-day level.

For a neurosurgeon, the Hippocratic oath espouses “do no harm,” and our study aimed to highlight a factor (burnout, via MBI) traditionally associated with poor workplace performance.⁵ Laurent et al. do not offer a practical alternative. Whether the MBI measures transient phenomena or does not truly correlate with depression is not relevant to our goal. MBI-measured burnout shows association with workplace performance, and if there is any factor associated with negative outcome in our residents and critically ill patients, we must make the neurosurgical community aware of it and explore every avenue to improve these outcomes.

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Carotid atherosclerotic plaque instability and cognition: collecting additional data

TO THE EDITOR: We have read with great attention the article by Dempsey and colleagues³ (Dempsey RJ, Varghese T, Jackson DC, et al: Carotid atherosclerotic plaque instability and cognition determined by ultrasound-measured plaque strain in asymptomatic patients with significant stenosis. *J Neurosurg* **128**:111–119, January 2018). The authors presented a stimulating study that describes “the use of ultrasound measurements of physical strain within carotid atherosclerotic plaques as a measure of instability and the potential for vascular cognitive decline, microemboli, and white matter hyperintensities (WMHs).” Subsequently, the same group demonstrated that atherosclerotic vascular cognitive decline may be modified by removal of the unstable plaque.² In that earlier study,³ the authors stated in their results that “the degree of strain instability measured within the atherosclerotic plaque directly predicted vascular cognitive decline in these patients thought previously to be asymptomatic according to classic criteria. Furthermore, 26% of patients showed microemboli, and patients had twice as much white matter hyperintensity as controls.”

As our lead author (A.N.) is involved in studying the natural course of ischemic WMHs and explaining the relationship between brain revascularization and the course of ischemic WMHs,^{5,6} he has uncovered some interesting points that have raised several questions, and we would like to obtain answers from experts³ that may be helpful in our current and future research. Based on a thorough

survey of the English literature,¹⁻⁷ evidently WMHs have been related to cognitive decline, and the changes in ischemic WMHs over time can be dynamic⁴⁻⁶ or partially reversible in patients with carotid artery stenosis.⁷

Previously, we established a relationship between brain revascularization and the course of ischemic WMHs.^{5,6} Therefore, we recommended further prospective studies to search for a relationship between WMH changes and functional neurological or neurocognitive data.⁶ Herein, we would like to ask the authors³ the following questions.

1. Have they³ done follow-up neuroimaging to assess the course of WMHs (improvement, unchanged, worsened, or fluctuating)? What about the time factor?
2. Did the authors³ find remarkable WMH changes in patients without evidence of microemboli? We would like to know the details of these WMHs.
3. Are the degree of strain instability and the surprising amount of WMH changes considered as direct predictors for the vascular cognitive decline in their cases?
4. What about the distribution of the WMHs (periventricular, deep WMHs, cortical)?
5. Did the authors³ have a fluctuating cognitive readout in their population?
6. We completely agree with the authors in their belief that “the presence of some emboli in other distributions and the symmetry of the WMHs showed that the presence of plaque instability not only suggests ipsilateral emboli but also represents a marker of advanced systemic large- and small-vessel disease.”³ Can we correlate that with the WMH improvement following direct extracranial-intracranial bypass surgery?^{5,6}

Finally, we appreciate the authors' contributions to neurosurgery, and we consider the above-mentioned study³ as a fundamental reference in our current research. Thus, we are looking forward to receiving their valuable comments and additional data.

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Disclosures

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Response

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Ultra-early aneurysm treatment

TO THE EDITOR: We read with interest the article by Linzey and colleagues² (Linzey JR, Williamson C, Rajajee V, et al: Twenty-four-hour emergency intervention versus early intervention in aneurysmal subarachnoid hemorrhage. *J Neurosurg* 128:1297–1303, May 2018), which is an important contribution to the debate whether around-the-clock aneurysm treatment should be performed. We have some qualms regarding the authors' conclusion.

The authors reported an overall recurrent bleeding rate of 7.6% for patients presenting with an aneurysmal subarachnoid hemorrhage (SAH). Although this rate seems quite comparable with those in the literature, with reported incidences between 9% and 17% in a meta-analysis by Starke and Connolly,³ these figures are very difficult to interpret as time intervals to recurrent bleedings and location of the patients at the time of recurrent bleeding are seldom reported accurately. If one wishes to substantially lower the recurrent bleeding rate, one must also take into account the recurrent bleeding before admission to the treatment center and clearly define specific time frames; a recurrent bleeding rate in the first 24 hours after admission to a treatment center is completely different from the recurrent bleeding rate in the first 24 hours after initial hemorrhage, especially as transfer times vary considerably between countries. Based on the results from this retrospective study, we think that the statement “the benefit of ultra-early aneurysm repair may be outweighed by the additional risks and costs of emergency overnight aneurysm clipping or coiling” is a premature conclusion.

When transfer times to the treatment center after initial hemorrhage are very short, a majority of recurrent bleedings will occur in the treatment center, as most recurrent

bleedings occur within 24 hours after the initial hemorrhage. Improvement of in-hospital logistics, analogous to the “time is brain” concept in ischemic stroke, should make it possible to reduce recurrent bleeding rates and subsequently improve clinical outcome.

To minimize the occurrence of recurrent bleedings before admission to a treatment center, regardless of transfer times, antifibrinolytic therapy may be a possibility, but the results of the ongoing randomized controlled trial (ULTRA trial [Ultra-Early Tranexamic Acid After Subarachnoid Hemorrhage]) on clinical outcome must be awaited.¹

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Disclosures

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Response

We appreciate the thoughtful comments by Dr. Post et al. Transfer systems and availability of critical care services during patient transfer vary from country to country, and thus our results and perspectives may not apply to all healthcare systems worldwide. Our statement, “the benefit of ultra-early aneurysm repair may be outweighed by the additional risks and costs of emergency overnight aneurysm clipping or coiling,” cannot be a premature conclusion, as it has the phrase “may be” within it. Our statement is not a conclusion but rather a hypothesis for future studies. Within the *Discussion*, we mentioned that there are limitations of retrospective studies, and thus no conclusion can be drawn from a single retrospective analysis. Nonetheless, we do report an extremely low rate of rehemorrhage in those SAH patients awaiting treatment, and this could be attributed to the advancements in the critical care and preoperative management of SAH patients. Nonetheless, future studies are necessary to further elucidate the role of ultra-early surgery in managing subarachnoid hemorrhage patients.

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