Delayed ipsilateral hemorrhage following aneurysm treatment with flow diverter


Over the last few years, endovascular treatment has been the firstline treatment for ruptured and unruptured intracranial aneurysms, with satisfying anatomical and clinical results. Nevertheless, treatment of wide-necked and fusiform aneurysms remains difficult with standard endovascular treatment (coil embolization), and recanalization remains an issue after coiling of large and giant aneurysms. Although the use of flow diveters in these subgroups of aneurysms has demonstrated encouraging results, cases of delayed IPH after flow-diverter placement have been described with potential serious clinical worsening.

In this context, Hu et al. performed 3 post mortem histopathological analyses of brain sections from 3 patients who suffered fatal delayed IPH. Microscopic and spectroscopic analyses revealed the presence of polyvinylpyrrolidone (PVP) occluding the small vessels in the area of bleeding in 2 of the 3 patients. As outlined by the authors, this finding is a cause for serious concern not only with respect to aneurysm treatment with flow diverters but for the entire field of neurointerventional procedures, as PVP is a commonly used coating on a variety of interventional devices. Thus, the etiopathogenesis of delayed IPH has to be analyzed in light of these new findings.

The authors report an incidence of delayed IPH of 4.1%, which is very close to what we reported in our recent case series (5.7%). As suggested by the authors, it is difficult to consider delayed IPH as hemorrhagic transformation of a thromboembolic ischemic lesion in the absence of large territories of acute stroke on pathological examination of the brain tissue. We recently reported similar findings in 2 cases of delayed IPH in which post-flow-diverter treatment MRI studies (a few hours or days before IPH) showing no ischemic lesion in the area where the IPH occurred (Fig. 1). In fact, as outlined by the authors, the occurrence of small silent thromboembolic events is usual after aneurysm coiling, however no cases of delayed IPH have been reported. PVP is known to compose the outer coat of many devices, but the relation between the presence of PVP in the small vessels of the hemorrhagic area and the occurrence of delayed IPH is not clear. Endovascular procedures are usually performed with devices made with PVP, but delayed IPH occurred only with flow-diverter utilization.

The histological findings reported by the authors are relatively disappointing, revealing filamentous, nonbiological material occluding the lumen of vessels of about 100 μm in diameter, which was depicted as PVP by Fourier transform infrared (FTIR) spectroscopy. Neither inflammatory reaction nor granulomas were observed in the histological specimens. However, as granulomas have been reported after PVP emboli in the cardiology literature, the authors suggest a potential role for the weakening and disruption of the vasculature created by the foreign body reaction in the arterial wall in the occurrence of delayed IPH. Also histological specimens revealed attenuation of the tunica media of postcapillary venules with extravasated erythrocytes suggesting a rise in venule pressure and a potential venous mechanism for delayed IPH.

Other potential mechanisms have been advocated, in-

Fig. 1. Pretreatment angiography (lateral view angiogram, A) revealed a left saccular aneurysm. Axial FLAIR MRI (B) performed 1 day after the procedure showed no postprocedural complications (no intracranial bleeding or ischemic complications). Two days later, the patient presented with hemiparesis and mydriasis. CT (C) revealed a left frontal hematoma with subarachnoid hemorrhage, inducing a mass effect treated surgically. CT was performed after surgery (D). Lateral view angiogram (E) obtained 10 months after the surgery showed a complete aneurysm occlusion with no stenosis of the parent artery.
including the deleterious effect of dual antiplatelet therapy and flow modification due to flow-diverter placement. Dual antiplatelet therapy is usually administered for stent-assisted coiling, but the rate of hemorrhagic complications is not as high as for flow-diverter placement. In their comprehensive literature survey on stent-supported coiling, Shapiro et al. observed a 2.2% rate of hemorrhagic complications, but these complications were not precisely described and are probably not all delayed IPH.2, Cruz et al. hypothesize that the reconstruction of arteries with the flow-diverter device could reduce vascular compliance and change the blood pressure waveform, generating a larger pulse pressure and increasing the pressure transmitted to the aneurysm and the distal cerebral arteries, which might lead to hemorrhagic complications; this is a potential explanation, but it has not been demonstrated by experimental evidence.1

Surprisingly, Hu et al. did not report any surgical evacuation for the 3 patients with delayed IPH.2 In our series, all patients had surgical evacuation of their hematoma (combined with platelet transfusion) with satisfying clinical outcomes (modified Rankin Scale score 1) for all patients.8 This underscores the value of surgical evacuation of the hematoma in this specific situation even in patients receiving dual antiplatelet therapy.

Delayed IPH is a serious but poorly understood complication after flow-diverter treatment. The pathological series published by Hu et al. suggests new potential mechanisms, including weakening of the arterial wall due to a foreign-body reaction or venous phenomenon. However, neither pathological findings nor previous imaging findings confirm the hypothesis of the hemorrhagic transformation of an ischemic stroke. Dual antiplatelet therapy probably also plays a role in the occurrence of delayed IPH. Given that no single mechanism may explain delayed IPH, a study of a larger series of cases, analyzing the risk factors, should be performed to explain and prevent this kind of complication.

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Disclosure

Dr. Pierot is a consultant for Codman, Covidien, MicroVention, Penumbra, and Sequent. Dr. Benaiissa reports no conflict of interest.

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RESPONSE: We thank Dr. Benaissa and Prof. Pierot for their thoughtful comments. They rightly highlight the fact that our report raises more questions than it answers and that the precise mechanism(s) of delayed ipsilateral hemorrhages after flow-diverter placement remain uncertain. As a practical matter, the pathological finding of PVP in association with the hemorrhages does not prove causality, but measures to reduce PVP emboli would seem prudent.

The commentators note that none of the 3 patients underwent surgical evacuation of their hematomas; this unfortunately was a function of the condition in which these 3 patients presented to the emergency room after their hemorrhages. In all instances, the patients were clinically moribund. Their statuses were sufficiently deteriorated so that hematoma evacuation was judged to be futile by the neurosurgeons evaluating them.

Finally, as Dr. Benaiassa and Prof. Pierot suggest, we hope that we can continue work with our colleagues to help understand and minimize the risk of delayed hemorrhage after flow-diverter placement.

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Football helmet design and concussion

To The Editor: We find it astonishing that JNSPG continues to publish articles on football concussion that only serve to retard scientific progress (Rowson S, Duma SM, Greenwald RM, et al: Can helmet design reduce the risk of concussion in football? Technical note. J Neurosurg 120:919–922, April 2014).1 It is obvious that the reviewers were more impressed by statistics than inspecting the data that produced these findings.

The notion that Division I college football programs
are roughly equivalent is laughable. The two Ivy League schools, Brown and Dartmouth, play 25.83% fewer games than Virginia Tech or Oklahoma. The latter two schools would be better compared to NFL teams, as each had/has a style and intensity of play that is consistent with professional teams.

In that light, the lack of recorded concussions should have been a red flag. The teams studied reported 1.33 concussions per team per year and averaged 0.11 concussions per game. While the authors suggest the rate of concussion is consistent with that of Division I schools, it simply betrays that most NCAA schools fail to report concussions, not that this sample had any validity. During the same period, the NFL, an organization not noted for its willingness to record concussions, documented more than 3 times the incidence of concussion than this sample.

The authors also represent that this is not an epidemiological study but rather an unbiased retrospective product comparison. In this light, the authors claimed the data were controlled for exposure. Given that star players and starters are more likely to be provisioned more modern equipment, and coaches promote policies intended to keep these highly valuable players on the field (especially in revenue-generating programs), it would clearly bias the results toward the Revolution helmet. Stars and starters have more exposure but would be less likely to record a concussion.

One must also consider the differences in product design with respect to the accommodation of sensors. The VSR4 was designed in the 1980s, long before sensors were considered by helmet manufacturers as a product extension. The Revolution was designed concurrently with the Head Impact Telemetry System (HITS) and might have been conceived to allow the introduction of sensors without affecting the helmet’s fit. Torg and colleagues have reported that a helmet’s fit, as opposed to its design or age, explains differences in the number of recorded concussions (JS Torg et al., presented at the American Academy of Orthopedic Surgeons Specialty Day, February 11, 2012).

Furthermore, we find it disturbing that engineers, who are well represented among the authors, would claim that simply thickening the outer shell of the helmet would reduce the forces generated by tackling. It is reminiscent of 1950s and 1960s automobile designers who increased the weight of vehicles and stiffened car bodies in the mistaken belief that these measures would enhance safety. Instead, the efforts resulted in more and more serious injuries from traffic accidents. Might we expect athletes to engage in more aggressive behavior, based upon the belief that a particular helmet mitigates the risk of a brain injury, whether true or not? The force needs to be dissipated. Is it transmitted into the head, neck, and spine? What are the long-term health consequences? Should we expect more long-term neurological afflictions based on poor quality studies endorsing so-called concussion mitigation technologies?

Finally, it is distressing that the authors suggest that this study confirms the findings of two earlier studies. Confirming results of two poorly conducted experiments merely demonstrates some degree of precision, not accuracy. In fact, the Collins paper referenced in the article has been subject to inquiry by the members of the US Senate.1 Years after Riddell promoted the sale of the Revolution helmet based on this study, governmental scrutiny compelled coauthor Joseph Maroon and UPMC to state that the findings were misrepresented and to distance themselves from the results.

We find it difficult to believe that the authors can make any claims regarding a difference in the risk of concussion between these two products. Given this study’s obvious lack of rigor, JNSPG reviewers should have been far more critical of this study, as it will be used as a means of promoting the sale of expensive new products.

**Disclosure**

The authors report no conflict of interest.

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**RESPONSE:** We appreciate Mr. Comrie’s and Mr. Morey’s interest in our study and their thoughts on the role helmets play in reducing concussion risk in football players. With this response, we simply aim to address inaccurate statements in their letter.

Mr. Comrie and Mr. Morey are wrong in their various statements suggesting the data set is not valid for addressing this and other important scientific questions. This research has been ongoing for over a decade, has been extensively published in peer-reviewed journals, and is both consistent and complementary to historical biomechanical studies evaluating protective equipment and head injury.1–5,9–11,13,14,16,17,19,24 The medical staff at each institution actively participated in this research, and the concussion rates reported are supported by epidemiological studies on athletes with similar demographic characteristics.15 It is simply not accurate to suggest that the concussion rate in collegiate football should be the same as the NFL.

Mr. Comrie and Mr. Morey are wrong in their descriptions of the design process and timeline of the two helmet types. The VSR4 was introduced in 1993 and the Revolution was introduced in 2002. The initial HITS design was first implemented in 2003 and was compatible with VSR4 helmets.11 As Revolution helmets became widely available, a new HITS model was created and made available for use in 2005. Neither helmet was initially designed with the intention to incorporate helmet
instrumentation; rather, HITS was developed to fit within existing helmet space without affecting helmet fitment or performance. Our study period (2005–2010) spanned the time when both instrumented helmet types were regularly in use.

Mr. Comrie and Mr. Morey are wrong in pointing to a helmet’s fit explaining differences in the number of recorded concussions. Not a single study in a peer-reviewed journal has found a helmet’s fit to influence concussion rates. However, over 60 years of research within the injury biomechanics community has shown that as head acceleration decreases, the risk of brain injury decreases.12,16–22 The risk of concussion and head acceleration also correlate strongly in our on-field head-impact data.20,21,23

Mr. Comrie and Mr. Morey are wrong in implying that we reported any data regarding a thicker shell and that the Revolution has a thicker shell than the VSR4. In fact, the Revolution shell is thinner than the VSR4. Moreover, the key design differences are the increased padding and the optimized shell geometry in the Revolution compared to the VSR4. Laboratory tests and on-field head-impact data clearly demonstrate differences in the ability of helmets to reduce head acceleration resulting from impact. Helmets that better modulate impact energy transfer to the head, reduce head acceleration, and, as a result, reduce concussion risk.

Mr. Comrie and Mr. Morey are wrong in their automotive analogy suggesting that design cannot be optimized to reduce concussion risk. It is ironic that they bring up this analogy given our extensive background in automobile safety. Contrary to what Mr. Comrie and Mr. Morey claim, stiffening vehicle structures and optimizing design have led to steady reductions in fatality rates associated with motor vehicle crashes for almost 50 years.18

Mr. Comrie and Mr. Morey are wrong in their interpretation of the US Senate inquiry into the Collins et al. 2006 study.7 The Federal Trade Commission (FTC) did open an investigation into Riddell’s promotional materials based on that study; however, after extensively reviewing and analyzing the underlying science, the FTC closed the investigation and took no action. We can only interpret this to mean that the FTC found no reason to refute the science in the Collins et al. 2006 study.

In summary, the purpose of our study was not to identify any specific helmet as being superior, but to answer the general question of whether or not helmet design can influence concussion rates in football. This question was addressed by performing a retrospective analysis on a preexisting dataset that consisted of head-impact data paired with clinical data on diagnosed concussions from 8 separate institutions. Like all scientific studies, there are of course limitations and we clearly acknowledge these in our article. However, our study presents the best available data for answering this timely and important question. Accounting for the number of head impacts that players experienced in each helmet is critical to accurately assessing concussion rates by helmet type, and it addresses major limitations of previous and emerging studies investigating this topic. The data presented in our study clearly indicate that advancing helmet design plays a role in reducing concussion rates in football.

Helmet design is just one part of a multipronged approach to minimizing concussions in football. Rule and regulation changes are perhaps most important, as they will limit the number of head impacts in football.14 When incidental head impacts do occur, having the best head protection available will further reduce risk. Going back to the automobile safety analogy, this is similar to requiring seat belt use and mandating airbags in all vehicles, in addition to optimizing vehicle structure. Together, efforts like these have continued to reduce traffic-related fatalities in the US. The same can be done for concussions in football.

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The cerebellum and Parkinson’s disease

To The Editor: We have found the article by Sweet et al.8 to be a remarkable work (Sweet JA, Walter BL, Gunalan K, et al: Fiber tractography of the axonal pathways linking the basal ganglia and cerebellum in Parkinson disease: implications for targeting in deep brain stimulation. Clinical article. J Neurosurg 120:988–996, April 2014), as it represents one of the major current trends in functional neurosurgery at the levels of both research and surgical technique—explicitly, the use of dynamic and functional imaging studies when approaching the brain to perform more precise surgeries in terms of increasing efficacy and patient safety.

The general theme of this article provides further evidence of the rising awareness of the cerebellum’s involvement in what we can call the “pathological infrastructures” of Parkinson’s disease (PD). The cerebellum is usually neglected in the classic illustration of the pathological neurocircuit of PD, and its contribution to PD is generally ignored. In another recently published article,10 Wu and Hallett reviewed the growing body of evidence that the cerebellum may have certain roles in the pathophysiology of PD. However, they focused on motor symptoms, that is, akinesia and/or rigidity, tremor, gait disturbance, and dyskinesia; their coverage of nonmotor symptoms was based mainly on connectivity and metabolic studies without moving toward clinical correlations or definitive conclusions.

We believe that including the cerebellum in the pathological neurocircuit of PD may provide even more fundamental insights into the nonmotor symptoms of PD—to be precise, the cognitive and emotional symptoms. In fact, over the last couple of years, the cerebellum has been shown to be involved in emotion and cognition.1,5 In this respect, cerebellar dysfunction is beginning to be considered a significant contributor to nonmotor conditions such as autism spectrum disorders.2 Likewise, several articles have indicated important roles for the cerebellum in the evolution of the capacity to plan, execute, and understand complex tasks, such as tool use and language.4 The idea that the cerebellum contributes to cognition as well as motor control is supported by studies that illustrate connections between the cerebellum and both the prefrontal and motor territories, as well as by functional neuroimaging studies that show cerebellar activations evoked during the performance of cognitive and motor tasks.3 In this respect, two distinct neurocircuits that connect the cerebellum with the cerebral neocortex were found, and both of these circuits relate to distinct functions. Specifically, prefrontal-parietal-cerebellar circuits are more active during cognitive and emotional tasks, whereas motor

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cortico-cerebellar circuits are more active during action execution tasks.2

Further support for the relationship between the cerebellum and PD is the fact that facial emotion processing is impaired in PD patients, with a disproportionate deficit involving fear and sadness.3 Interestingly, Adamaszek et al. showed impairment in facial emotion processing and prosody discrimination following ischemic cerebellar lesions in 15 patients.4 On the same subject, the patterns of cognitive decline—that is, those affecting executive function, attention, processing speed, and visuospatial learning and memory—are to some extent similar to those in patients harboring cerebellar pathologies, specifically, the cerebellar cognitive affective syndrome.5

We believe it is time to clearly include the cerebellum in the pathological neurocircuit model of PD given the following: fundamental acknowledgment of the cerebellum’s participation in sensory-motor matching and learning as well as the processing of cognition and emotion,2 the results of hodological studies conducted using virus tracing or functional imaging techniques (as in the study by Sweet et al.), and the accumulated data of functional connectivity research. Moreover, we believe that this inclusion could positively affect our understanding of the nonmotor symptoms of PD even more than the motor symptoms (as most of us would think), for example, by giving us more clues about that special pattern of cognitive decline and the pattern of emotional impairment in PD.

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Disclosure

The authors report no conflict of interest.

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RESPONSE: We appreciate the thoughtful comments of Drs. Salma and Tracy. Our data suggest that activation of cerebellar axonal tracts may play a major role in the mechanism of action of the clinical effects of deep brain stimulation. It stands to reason that a similar process may be responsible for side effects: just as the spread of current into the adjacent internal capsule or medial lemniscus is known to be responsible for sensorimotor side effects, nonmotor effects may be mediated by tracts that are associated with cognitive or emotional functions. The dentatothalamic tract has been implicated in tremor, but the function of other tracts, such as the subthalamoponto-cerebellar, is less clear. If cerebellar pathology is indeed related to the Parkinson’s phenotype, improved visualization of these pathways may provide insight into the disease and allow for improved neuromodulation strategies. Confirmation of this hypothesis will require verification that imaging of axonal pathways can predict clinical benefit as well as side effects.

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Value-based neurosurgery and microvascular decompression


First, we would like to congratulate the authors on tackling this important and relatively new health care issue and introducing what is conceivably the first scientific article about value-based health care in neurosurgery. The authors examined the hypothesis that specific quality improvement projects will lead to improved quality of product, which in this case is “microvascular de-
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The authors report no conflict of interest.

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RESPONSE: We thank Drs. Raslan and Burchiel for their thoughtful comments.

In our view, the emerging concept of focusing on health care value as the key goal of medical reform has great potential to improve care delivery. It is also our impression that our article is the first to plan, implement, and critically assess care redesign of this type in the field of neurosurgery. We believe that the specialty of neurosurgery, given its history of innovation and orientation to action, is optimally positioned to lead health care in transformative improvement and positive reform from the inside.

The article in reference reviews a series of clinical interventions and their resultant clinical outcomes. Following the implementation of improvement processes throughout the continuum of care associated with MVD surgery, patients benefited from a reduction in the mean total operating room time (22%, from 455 minutes to 357 minutes), a decrease in the mean postoperative length of hospital stay (15%, from 3.01 days to 2.59 days), a decrease in the mean length of stay on the floor (41%, from 41 hours to 24 hours), and a reduction in the rates of complications and readmissions.

We fully agree that a complete assessment of the value equation requires integration of cost of care. This component of the value equation is dealt with in detail in the manuscript entitled “Value-based neurosurgery: measuring and reducing the cost of microvascular decompression surgery,” which is in press in the Journal of Neurosurgery, as of this writing. In this follow-up article, we assessed how process improvement and strategic cost containment efforts could optimize the value of MVD surgery. After targeting the 3 most expensive cost activities, the average total cost of the surgical care episode for a patient undergoing MVD decreased 25%. In addition to reviewing in this work our contemporary activity-based costing data for this patient population, we have subsequently gone further to explore improving the accuracy of measuring the cost of delivering care using the time-driven activity-based costing (TDABC) model popularized by Robert S. Kaplan (TDABC manuscript currently in revision).

The initial phase of value-based care redesign is enhanced by process mapping—the careful study and illustration of the flow of the individual steps of the care deliv-
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every process. We have described the details of this element of care redesign specifically for neurosurgical care in the article entitled “Demystifying process mapping: A key step in neurosurgical quality improvement initiatives.” An additional key activity is quantitative measurement of process and outcomes, enabling data-driven monitoring and management of care redesign strategies. The use of metrics dashboards in our value redesign management meetings to visualize and track and compare performance data over time are described in the article entitled “Tracking and sustaining improvement initiatives: leveraging quality dashboards to lead change in a neurosurgical department.”

We agree that patient centeredness, patient satisfaction, and preserving and restoring quality of life are all of essential importance in value-based care delivery. This dimension has been the cornerstone in our current care redesign initiatives, which consider the patient and family’s voice at every step of the redesign. A consideration of meaningful measurements of the outcomes that matter most to patients is critical, and we are actively working to understand and develop and integrate these into value-based neurosurgery redesign.

We look forward to a rapid embrace of creative value-based care redesign throughout neurosurgery, and we anticipate that our specialty will play a key leadership role in pioneering effective patient-oriented reform of American health care—from the inside by the frontline care providers.

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