Delayed ipsilateral hemorrhage following aneurysm treatment with flow diverter

TO THE EDITOR: The article by Hu et al.² is an important contribution to the analysis of delayed intraparenchymal hemorrhage (IPH) after flow-diverter treatment (Hu YC, Deshmukh VR, Albuquerque FC, et al: Histopathological assessment of fatal ipsilateral intraparenchymal hemorrhages after the treatment of supraclinoid aneurysms with the Pipeline Embolization Device. Report of 3 cases. *J Neurosurg* 120:365–374, February 2014).

Over the last few years, endovascular treatment has been the firstline treatment for ruptured and unruptured intracranial aneurysms, with satisfying anatomical and clinical results.^{4,5} 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 diverters 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.^{1,3,8}

In this context, Hu et al. performed 3 post mortem histopathological analyses of brain sections from 3 patients who suffered fatal delayed IPH.² 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%).⁸ 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).⁸ 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.⁶ 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 μ m 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.

cluding 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.⁷ 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. Benaïssa reports no conflict of interest.

References

1. Cruz JP, Chow M, O'Kelly C, Marotta B, Spears J, Montanera W, et al: Delayed ipsilateral parenchymal hemorrhage following flow diversion for the treatment of anterior circulation aneurysms. *AJNR Am J Neuroradiol* **33**:603–608, 2012
2. Hu YC, Deshmukh VR, Albuquerque FC, Fiorella D, Nixon RR, Heck DV, et al: Histopathological assessment of fatal ipsilateral intraparenchymal hemorrhages after the treatment of supraclinoid aneurysms with the Pipeline Embolization Device. Report of 3 cases. *J Neurosurg* **120**:365–374, 2014
3. Kulcsár Z, Szikora I: The ESMINT Retrospective Analysis of Delayed Aneurysm Ruptures after flow diversion (RADAR) study. *EJMINT*:1244000088, 2012 (<http://www.ejmint.org/original-article/1244000088>) [Accessed April 30, 2014]
4. Pierot L, Cognard C, Ricolfi F, Anxionnat R: Midterm anatom-

ic results after endovascular treatment of ruptured intracranial aneurysms with GDC and Matrix coils: analysis of the CLARITY series. *AJNR Am J Neuroradiol* **33**:469–473, 2012

5. Pierot L, Spelle L, Vitry F: Immediate anatomic results after the endovascular treatment of unruptured intracranial aneurysms: analysis of the ATENA series. *AJNR Am J Neuroradiol* **31**:140–144, 2010
6. Rordorf G, Bellon RJ, Budzik RF Jr, Farkas J, Reinking GF, Pergolizzi RS, et al: Silent thromboembolic events associated with the treatment of unruptured cerebral aneurysms by use of Guglielmi detachable coils: prospective study applying diffusion-weighted imaging. *AJNR Am J Neuroradiol* **22**:5–10, 2001
7. Shapiro M, Becske T, Sahlein D, Babb J, Nelson PK: Stent-supported aneurysm coiling: a literature survey of treatment and follow-up. *AJNR Am J Neuroradiol* **33**:159–163, 2012
8. Tomas C, Benaïssa A, Herbreteau D, Kadziolka K, Pierot L: Delayed ipsilateral parenchymal hemorrhage following treatment of intracranial aneurysms with flow diverter. *Neuroradiology* **56**:155–161, 2014

RESPONSE: We thank Dr. Benaïssa 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. Benaïssa 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).² 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/had 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

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References

1. Collins M, Lovell MR, Iverson GL, Ide T, Maroon J: Examining concussion rates and return to play in high school football players wearing newer helmet technology: a three-year prospective cohort study. *Neurosurgery* **58**:275–286, 2006
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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 ap-

proach to minimizing concussions in football. Rule and regulation changes are perhaps most important, as they will limit the number of head impacts in football.^{6,8} 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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References

1. Beckwith JG, Greenwald RM, Chu JJ, Crisco JJ, Rowson S, Duma SM, et al: Head impact exposure sustained by football players on days of diagnosed concussion. **Med Sci Sports Exerc** 45:737–746, 2013
2. Beckwith JG, Greenwald RM, Chu JJ, Crisco JJ, Rowson S, Duma SM, et al: Timing of concussion diagnosis is related to head impact exposure prior to injury. **Med Sci Sports Exerc** 45:747–754, 2013
3. Broglio SP, Schnebel B, Sosnoff JJ, Shin S, Fend X, He X, et al: Biomechanical properties of concussions in high school football. **Med Sci Sports Exerc** 42:2064–2071, 2010

4. Broglio SP, Surma T, Ashton-Miller JA: High school and collegiate football athlete concussions: a biomechanical review. **Ann Biomed Eng** 40:37–46, 2012
5. Brolinson PG, Manoogian S, McNeely D, Goforth M, Greenwald R, Duma S: Analysis of linear head accelerations from collegiate football impacts. **Curr Sports Med Rep** 5:23–28, 2006
6. Cobb BR, Urban JE, Davenport EM, Rowson S, Duma SM, Maldjian JA, et al: Head impact exposure in youth football: elementary school ages 9–12 years and the effect of practice structure. **Ann Biomed Eng** 41:2463–2473, 2013
7. Collins M, Lovell MR, Iverson GL, Ide T, Maroon J: Examining concussion rates and return to play in high school football players wearing newer helmet technology: a three-year prospective cohort study. **Neurosurgery** 58:275–286, 2006
8. Crisco JJ, Greenwald RM: Let's get the head further out of the game: a proposal for reducing brain injuries in helmeted contact sports. **Curr Sports Med Rep** 10:7–9, 2011
9. Crisco JJ, Wilcox BJ, Beckwith JG, Chu JJ, Duhaime AC, Rowson S, et al: Head impact exposure in collegiate football players. **J Biomech** 44:2673–2678, 2011
10. Crisco JJ, Wilcox BJ, Machan JT, McAllister TW, Duhaime AC, Duma SM, et al: Magnitude of head impact exposures in individual collegiate football players. **J Appl Biomech** 28:174–183, 2012
11. Duma SM, Manoogian SJ, Bussone WR, Brolinson PG, Goforth MW, Donnenwerth JJ, et al: Analysis of real-time head accelerations in collegiate football players. **Clin J Sport Med** 15:3–8, 2005
12. Duma SM, Rowson S: The biomechanics of concussion: 60 years of experimental research, in Slobounov SM, Sebastianelli WJ (eds): **Concussions in Athletics**. New York: Springer, 2014, pp 115–137
13. Duma SM, Rowson S: Past, present, and future of head injury research. **Exerc Sport Sci Rev** 39:2–3, 2011 (Letter)
14. Guskiewicz KM, Mihalik JP, Shankar V, Marshall SW, Crowell DH, Oliaro SM, et al: Measurement of head impacts in collegiate football players: relationship between head impact biomechanics and acute clinical outcome after concussion. **Neurosurgery** 61:1244–1253, 2007
15. Guskiewicz KM, Weaver NL, Padua DA, Garrett WE Jr: Epidemiology of concussion in collegiate and high school football players. **Am J Sports Med** 28:643–650, 2000
16. Hardy WN, Khalil TB, King AI: Literature review of head injury biomechanics. **Int J Impact Engng** 15:561–586, 1994
17. Mihalik JP, Bell DR, Marshall SW, Guskiewicz KM: Measurement of head impacts in collegiate football players: an investigation of positional and event-type differences. **Neurosurgery** 61:1229–1235, 2007
18. National Highway Traffic Safety Administration: **Traffic Safety Facts 2010**. Washington, DC: US Department of Transportation (<http://www.nrd.nhtsa.dot.gov/Pubs/811659.pdf>) [Accessed May 15, 2014]
19. Rowson S, Brolinson G, Goforth M, Dietter D, Duma SM: Linear and angular head acceleration measurements in collegiate football. **J Biomech Eng** 131:061016, 2009
20. Rowson S, Duma SM: Brain injury prediction: assessing the combined probability of concussion using linear and rotational head acceleration. **Ann Biomed Eng** 41:873–882, 2013
21. Rowson S, Duma SM: Development of the STAR evaluation system for football helmets: integrating player head impact exposure and risk of concussion. **Ann Biomed Eng** 39:2130–2140, 2011
22. Rowson S, Duma SM: The Virginia Tech response. **Ann Biomed Eng** 40:2512–2518, 2012
23. Rowson S, Duma SM, Beckwith JG, Chu JJ, Greenwald RM, Crisco JJ, et al: Rotational head kinematics in football impacts: an injury risk function for concussion. **Ann Biomed Eng** 40:1–13, 2012
24. Schnebel B, Gwin JT, Anderson S, Gatlin R: In vivo study of

head impacts in football: a comparison of National Collegiate Athletic Association Division I versus high school impacts. **Neurosurgery** 60:490–496, 2007

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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 context, cerebellar dysfunction is beginning to be considered a significant contributor to nonmotor conditions such as autism spectrum disorders.⁷ 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

cortico-cerebellar circuits are more active during action execution tasks.³

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.² Interestingly, Adamaszek et al. showed impairment in facial emotion expression and prosody discrimination following ischemic cerebellar lesions in 15 patients.¹ On the same subject, the patterns of cognitive decline—that is, those affecting executive function, attention, processing speed, and visuospatial learning and memory⁶—are to some extent similar to those in patients harboring cerebellar pathologies, specifically, the cerebellar cognitive affective syndrome.⁹

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,⁵ 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

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References

1. Adamaszek M, D'Agata F, Kirkby KC, Trenner MU, Sehm B, Steele CJ, et al: Impairment of emotional facial expression and prosody discrimination due to ischemic cerebellar lesions. **Cerebellum** 13:338–345, 2013
2. Ariatti A, Benuzzi F, Nichelli P: Recognition of emotions from visual and prosodic cues in Parkinson's disease. **Neurol Sci** 29:219–227, 2008
3. Balsters JH, Laird AR, Fox PT, Eickhoff SB: Bridging the gap between functional and anatomical features of cortico-cerebellar circuits using meta-analytic connectivity modeling. **Hum Brain Mapp** [epub ahead of print], 2013
4. Barton RA: Embodied cognitive evolution and the cerebellum. **Philos Trans R Soc Lond B Biol Sci** 367:2097–2107, 2012
5. D'Angelo E, Casali S: Seeking a unified framework for cerebellar function and dysfunction: from circuit operations to cognition. **Front Neural Circuits** 6:116, 2012
6. Palavra NC, Naismith SL, Lewis SJ: Mild cognitive impairment in Parkinson's disease: a review of current concepts. **Neurol Res Int** 2013:576091, 2013
7. Reeber SL, Otis TS, Sillitoe RV: New roles for the cerebellum in health and disease. **Front Syst Neurosci** 7:83, 2013
8. Sweet JA, Walter BL, Gunalan K, Chaturvedi A, McIntyre CC, Miller JP: 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, 2014
9. Tedesco AM, Chiricozzi FR, Clausi S, Lupo M, Molinari M, Leggio MG: The cerebellar cognitive profile. **Brain** 134:3672–3686, 2011
10. Wu T, Hallett M: The cerebellum in Parkinson's disease. **Brain** 136:696–709, 2013

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 subthalamopontocerebellar, 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

TO THE EDITOR: We read with great interest the article by McLaughlin and colleagues² (McLaughlin N, Buxey F, Chaw K, et al: Value-based neurosurgery: the example of microvascular decompression surgery. Clinical article. **J Neurosurg** 120:462–472, February 2014).

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-

compression" (MVD). This is a valuable endeavor and a complex task, and while the article introduces the concept and tackles it from an investigative perspective, it also illustrates the challenges associated with demonstrating higher value in health care:

1. Defining cost: Although the authors selected the title "value-based neurosurgery," there was no discussion of "cost." Value in health care is defined as quality of health care per dollar spent. The authors could have compared the cost of MVD before and after the intervention, adjusting for health care inflation. Defining and estimating costs is complex because cost means different things to different entities—for example, direct cost and total cost concern the hospital, charges concern the payer, and out-of-pocket expenses concern the patient. In this specific case, direct cost or total cost adjusted for health care inflation would have sufficed.

2. The interchange of quality and outcome: The authors adopted Porter's 3-tier Outcome Measures Hierarchy³ model to estimate health care outcome; however, quality comprises more than just outcomes. Quality measures today are patient-centered and encompass 4 main pillars: access, safety, outcome, and respect. Patient satisfaction is major component of the respect pillar, and presentation of a case for improved value without discussion of patient satisfaction scores is suboptimal.

3. Patient-centeredness: Granted, the issue is bigger than this specific article. The outcome measures the authors used were known validated measures, such as the Barrow Neurological Institute pain inventory. Patient-centeredness would take into account things such as missed days of work or lack of pleasure due to pain, weaning off medication, and the like. As physicians strive to improve value for their patients, patient-centeredness is a challenge to define and include in discussions.

4. Quality improvement collides with evidence-based medicine: The authors at the University of California, Los Angeles introduced a comprehensive quality-improvement project that included preoperative, operative, and postoperative processes intended to improve quality. Operative processes included but were not limited to decreasing craniotomy size, specific closure technique, and hemostasis. No individual process has proven to improve quality for MVD or has ever been studied as such; however, the collective processes could perhaps show statistically significant improvement if the sample size was large enough. As physicians, we need to consider a threshold for adopting quality-improvement processes as a best practice from an evidence-based point of view. There will come a time when a group of sequential processes will collectively show improved outcome, while it will be difficult for an individual process to show effectiveness.

In closing, this is a valuable effort and a leading initiative in neurosurgery and we commend the authors on their article. We expect the issues of value, quality, and health care outcomes^{1,4} to be the centerpiece of our discussions for the next decade.

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Disclosure

The authors report no conflict of interest.

References

1. Healthcare Financial Management Association Educational Foundation: **Value in Health Care: Current State and Future Directions**. Westchester, IL: HFMA, 2011 (<https://www.hfma.org/Content.aspx?id=1126>) [Accessed May 22, 2014]
2. McLaughlin N, Buxey F, Chaw K, Martin NA: Value-based neurosurgery: the example of microvascular decompression surgery. Clinical article. **J Neurosurg** 120:462–472, 2014
3. Porter ME: What is value in health care? **N Engl J Med** 363:2477–2481, 2010
4. Porter ME, Lee T: **The Strategy That Will Fix Health Care**. Watertown, MA: Harvard Business Review, 2013 (<http://hbr.org/2013/10/the-strategy-that-will-fix-health-care/ar/pr>) [Accessed May 22, 2014]

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.⁷ 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.⁶ 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).^{2,3}

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-

ery 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.”⁵ 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.”⁴

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.¹

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References

1. Afsar-Manesh N, Martin NA: Healthcare reform from the inside: a neurosurgical clinical quality program. **Surg Neurol Int** 3:128, 2012
2. Kaplan RS, Anderson SR: Time-driven activity-based costing. **Harv Bus Rev** 82:131–138, 150, 2004
3. Kaplan RS, Porter ME: How to solve the cost crisis in health care. **Harv Bus Rev** 89:46–52, 54, 56–61 passim, 2011
4. McLaughlin N, Afsar-Manesh N, Ragland V, Buxey F, Martin NA: Tracking and sustaining improvement initiatives: leveraging quality dashboards to lead change in a neurosurgical department. **Neurosurgery** 74:235–244, 2014
5. McLaughlin N, Rodstein J, Burke MA, Martin NA: Demystifying process mapping: a key step in neurosurgical quality improvement initiatives. **Neurosurgery** [epub ahead of print], 2014
6. McLaughlin N, Upadhyaya P, Buxey F, Martin NA: Value-based neurosurgery: measuring and reducing the cost of microvascular decompression surgery. Clinical article. **J Neurosurg** [in press], 2014
7. Porter ME: A strategy for health care reform—toward a value-based system. **N Engl J Med** 361:109–112, 2009

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