Survival after cerebral herniation

To the Editor: I would like to thank Dr. Stiver and colleagues for their recent case report (Stiver SI, Gean AD, Manley GT: Survival with good outcome after cerebral herniation and Duret hemorrhage caused by traumatic brain injury. Case report. J Neurosurg 110:1242–1246, June, 2009).1 While I am in agreement that the patient received extraordinary care and excellent management in the hospital, I would like to address several key points in the early management and care of this patient with traumatic brain injury (TBI).

It is noted that the patient’s initial Glasgow Coma Scale (GCS) score was M1 E1 V1 at the time of her TBI, but upon presentation to the emergency department, the patient’s score improved to M4 E1 V1. This does not describe a “hopeless situation” as indicated. It is well known that the best predictive value when dichotomizing the GCS is the motor response component, in this case a 4.2,3 In addition, it is important to mention that the patient’s GCS score was possibly compromised by the presence of an important secondary brain insult, such as hypotension related to the left humeral fracture. It is very unlikely that the intracranial lesion can be the source of such severe secondary brain insult in the absence of ongoing scalp hemorrhage.

Additionally, the airway management provided prior to admission at the receiving hospital warrants discussion. Further information regarding her ventilation and respiratory condition in the field, during transport, and on arrival to the receiving hospital would illustrate if hypoxia contributed to the patient’s initially lower GCS score. This is an important factor as the combination of hypoxia and hypotension can increase the mortality rate by 70% after TBI, and may contribute to increased intracranial pressure, cerebral edema, and herniation syndrome.2,3,6,8–11 It may be possible with early interventions to control and prevent these insults. Early airway management and intubation in the field, control of the hemorrhage, stabilizing the fracture, and intravenous fluid administration provided by emergency personnel could have contributed to a reduction of secondary brain insults during the so-called “golden hour.” Perhaps this sequence of events reduced the risk for uncontrollable cerebral edema that can lead to herniation syndrome.

The centered ventral pons location of the secondary Duret hemorrhage without involvement of the pontine and medullary reticular formation in the dorsal pontine tegmentum was definitely an important factor in promoting this patient’s survival. I thank Drs. Stiver, Gean, and Manley for such descriptive information about the brainstem displacement during herniation syndrome in patients with TBI.

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References

Response: We thank Dr. Defillo for his comments and critique and for the kind remarks on the excellent care provided to this patient at our hospital. As pointed out by Dr. Defillo, it is important to remember that the GCS score of record should be obtained after resuscitation. Many patients with TBI, including those with a mild head injury, incur a transient episode of unresponsiveness at the time of impact. Secondary insults such as hypoxia and hypotension do suppress the GCS score and may lead one to overestimate the severity of the primary impact. Dr. Defillo raises 2 important questions. First, the patient’s GCS score on arrival to the emergency department was 6. He notes that this is not a “hopeless situation,” and questions the attention drawn to her survival. The second question he asks is whether continued resuscitation efforts would have allowed her GCS score to improve even further. Both points suggest that perhaps this was not a devastating injury, but rather was compounded by sec-
ondary injury, and that the patient’s eventual good recovery was not as remarkable as we have indicated.

Details of the patient’s course in the emergency department corroborate that the patient’s neurological condition was deteriorating and that her condition was, in fact, consistent with extremis and life-threatening cerebral herniation in the emergency department. The mechanism of injury was a high-speed, rollover motor vehicle accident with a 10-minute extrication. Her blood pressure in the field was 80/palp with a pulse of 80 beats per minute. An 

\[ \text{O}_2 \] \text{saturation was not recorded in the field. However, as Dr. Defillo points out, given the difficult extrication one can presume that she may have incurred hypotension and hypoxic secondary injuries due to the delayed resuscitation. She arrived at the emergency department with blood pressure 100/palp, pulse of 106, and with agonal respirations. Her pupils were bilaterally blown. She was noted to have an initial GCS score of 6 with withdrawal of all 4 extremities to pain, but her condition quickly deteriorated. She was intubated within the first 5 minutes of arrival. Just prior to intubation, her GCS score had dropped to 4. Her condition declined from a motor examination of withdrawal to extension within this short time. Upon suctioning for the intubation, cough and gag responses were noted to be absent. As is common during resuscitation in the emergency department, intubation is a very early step and the pharmacological agents used obscure further neurological testing.

In the interests of brevity for publication, we did not detail her emergency department course and deterioration. It has been argued that the GCS score that immediately precedes operative intervention is more important than that on admission. The patient’s last neurological examination prior to paralysis showed a GCS score of 4, with fixed and dilated pupils and absent cough and gag responses. With expeditious transfer to the operating room, it is impossible to know whether the patient’s GCS score would have improved with ongoing efforts to resuscitate her hypotension and hypoxia. Given the precipitous decline in her GCS score prior to intubation and the ominous appearance of her CT scan, we think this was unlikely. Thus, we do not believe that the patient’s survival was predictable based on her initial GCS score of 6.

Independent of the initial GCS score and whether or not further resuscitation might have improved that score, the patient did go on to show radiological signs of brainstem herniation. Thus, one important point of the report is that the patient did sustain a Duret hemorrhage from which she not only survived but that she did so with a good outcome. Many regard a Duret hemorrhage as an irreversible sign of medical futility with cause to withdraw medical care. We agree that the patient’s initial GCS score of 6 may have contributed to our motivation to persist with aggressive care in this case. Differentiating patients with low GCS scores due to primary impact injuries who may be at high risk for persistent vegetative outcomes from those with potentially reversible secondary insults is extremely difficult in the acute setting. As Dr. Defillo points out, it is critically important to remember the role of resuscitation. Patients with TBI can present with GCS scores of 3 or 4 as a result of secondary injury that is both treatable and reversible. Ultimately, this is what makes TBI care in the emergency department both challenging and rewarding. (DOI: 10.3171/2009.6.JNS091009)

To The Editor: We read with great interest the article by Walsh et al. (Walsh M, Parmar H, Mukherji SK, Mamourian A: Developmental venous anomaly with symptomatic thrombosis of the draining vein. Report of 2 cases. J Neurosurg 109:1119–1122, December, 2008).2 We would like to take this opportunity to comment on the first case that presented with seizures and imaging features consistent with thrombotic occlusion of the collecting vein of a large frontal lobe developmental venous anomaly (DVA). Remarkably, thrombosis of the collecting vein did not result in hemorrhagic or ischemic infarction within the drainage territory of the DVA. As the authors mention in the discussion, the essential role played by DVAs in the normal cerebral venous drainage has been well illustrated in the literature by cases of venous ischemic and hemorrhagic complications secondary to either surgical removal or spontaneous thrombosis of a DVA. It is not clear in the article by Walsh and colleagues how the confirmed thrombosis of the collecting of the DVA did not result in venous infarction.

Cerebral DVAs represent a purely venous entity, an anomalous venous disposition due to the absence of normal pial or subependymal veins. They may be understood as a variation of the venous angioarchitecture. When the DVA serves as an anomalous pathway for a deep venous territory, it can either drain centrifugally toward the cortical veins or directly into a dural venous sinus.1 Alternatively, where a DVA serves as an anomalous pathway for the cortical and subcortical venous territory, it drains centripetally toward the network of deep subependymal veins, tributaries of the deep venous system. In some cases, however, the venous system that the DVAs are supposed to compensate for remains patent although hypoplastic, and connections between the two may be observed. The result is a dual drainage toward the deep and superficial venous systems, with either the superficial or the deep venous system being clearly dominant over the other (personal observations; Fig. 1). Walsh et al. likely refer to this in their statement “the drainage vein can empty into the superficial venous system such as the cortical veins, the dural venous sinus, the subependymal veins, or some combination.”

Though it is not possible to ascertain from the figures that illustrate Case 1, we suppose that a connection similar to the one demonstrated in Fig. 1 between the superficially draining collecting vein of the DVA with the subependymal veins of the left frontal horn could have been retained during intrauterine life, allowing for additional centripetal drainage of the caput medusa of the DVA. Hence, the existence of this connection could account for
...major collecting vein becomes occluded. Routine imaging studies, but may act as an important collateral pathway. Dual superficial and deep drainage of DVAs is often overlooked during cerebral vein (ICV) by way of a direct lateral vein (SSS), though it has retained a connection preferentially drains superficially towards the superior sagittal sinus. The positive outcome of the patient could have also resulted from incomplete thrombosis of the draining vein and the early recognition and therapeutic intervention rendered, preventing the disastrous complications demonstrated in the second case we presented. Though we may never be able to completely understand the circumstances contributing to the favorable outcome in the first case, an increased awareness of this entity with prompt recognition and treatment will hopefully result in more favorable outcomes in the future. (DOI: 10.3171/2009.2.JNS081595)

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### Traumatic brain injury outcome

To The Editor: We read with great interest the recent article by Vik et al. (Vik A, Nag T, Fredriksli OA, et al: Relationship of “dose” of intracranial hypertension to outcome in severe traumatic brain injury. Clinical article. J Neurosurg 109:678–684, October, 2008). In their well-organized study the authors calculated the “dose” of intracranial hypertension in head-injured patients by measuring the area under the curve (AUC) for intracranial pressure (ICP) whenever ICP was > 20 mm Hg. This parameter takes into account not only the elevated ICP but also the duration of the intracranial hypertension. These authors concluded that there is a significant relationship between the ICP-AUC, the observed mortality rate, and the 6-month clinical outcome. They also postulated that this ICP-AUC methodology may improve the treatment of ICP in patients with traumatic brain injury (TBI). We agree with Vik et al. that this ICP-AUC concept may well predict the occurrence of secondary injuries due to intracranial hypertension after TBI. However, the calculation of the ICP-AUC parameter cannot predict the possibility of forthcoming ICP increases. We are wondering if the authors have implemented the so-called slope of the curve (ΔICP/Δt) parameter in their study, and if they have examined its role in the ICP profile in their cohort.

In TBI cases, intracranial hypertension (defined as ICP > 20 mm Hg) measured on admission or at the time of clinically observed worsening is a strong predictor of outcome. Intracranial hypertension is also one of the leading causes of secondary brain injuries, which exert a worsening effect on the overall clinical outcome. The same data indicate that the early recognition and prevention of episodes of intracranial hypertension could possibly result in a more favorable outcome in patients with TBI. The proposed “dose” model of Vik et al. reflects the...
level and duration of intracranial hypertension and possibly its role in inducing secondary brain insults, but does not provide us with a tool of early detection and timely prevention of these episodes.

Critical information regarding the ICP behavior could be potentially extracted if we could measure the slope of the mean ICP graph at a specific time interval (for example, every 30 or 60 minutes). The corresponding slope of the graph, represented by the formula of ΔICP/Δt and calculated as the difference of mean IC Ps measured between time 2 (t2) and time 1 (t1), divided by the time interval (t2 − t1 = Δt), can provide us with valuable additional data regarding the tendency of the mean ICP to rise or fall. If the ΔICP/Δt is positive, close to 90° (or above a chosen threshold, for example 60°), and the measured ICP is in the proximity of the 20 mm Hg threshold, we could predict the rise of the ICP above the threshold in our next measurements. This prediction could give to the involved clinician the opportunity to intervene in a timely fashion and prevent a secondary insult, and to alleviate the detrimental effect of intracranial hypertension.

We should mention that the proposed method is different from the methodology proposed by Westhout et al., who implement the measurement of the slope of ICP waveform during inspiration and expiration phases. A meta-analysis by Sorani and Manley showed that the mean time of maximum ICP response was approximately 60 minutes after mannitol administration, which is a significant time interval for the brain to be stressed under an unfavorable environment. Therefore, it would be beneficial if this period of stress for the brain could be prevented, or its duration could be somehow shortened by predicting it and appropriately treating it in a timely fashion. The study of the ΔICP/Δt parameter may lend itself to the early detection of such forthcoming ICP episodes. Thus, we should try to find additional algorithms to act in a preventive way apart from the posthypertensive therapeutic maneuvers.

In conclusion, the “dose” model presented by Vik et al. reflects the level and duration of the secondary insults in relation to the outcome, but does not provide us with early recognition and prevention algorithms that could possibly produce a more favorable outcome. The authors should be congratulated on their well-executed study. However, further research is necessary to assess the role of ΔICP/Δt measurements in the outcome of TBI.

References

Response: We thank Drs. Filippidis and Fountas for their interest and general appraisal of our recent work. We also appreciate that they agree that the ICP-AUC method can assess the extent and duration of secondary injuries due to intracranial hypertension after TBI. Our suggestion that “the AUC method may be useful in refining and improving the treatment of ICP in patients with TBI” refers more to the utility of the method in retrospective clinical trials than in clinical practice for treating individual patients. Thus, we agree that the calculation of ICP-AUC cannot predict the possibility of forthcoming ICP increases. The method is most useful for postprocessing performed to evaluate the amount or burden of ICP above a certain level from data sets in which ICP is only recorded at 1-hour intervals. We do believe, however, that this method has clinical utility if the data are collected at more frequent intervals (once per minute or more) and are readily accessible for acquisition and processing to give “real-time” assessments of the aggregate “dose” of intracranial hypertension.

Drs. Filippidis and Fountas introduce in their letter another method, which they call the slope of the mean ICP graph, or ΔICP/Δt. This is essentially a rate of change in ICP and would be expected to increase as the patient’s intracranial compliance worsens over time. This is an interesting concept, but because our ICP values were only recorded hourly, they are not suitable for such calculations. There are a number of methods that have been proposed to evaluate ICP data for prediction and clinical care. Balestreri et al. used indices derived from ICP waveform analysis for the interpretation of progressive intracranial hypertension. More recently, Eide et al. have used the ICP waveform amplitude as an indicator of intracranial compliance. However, as we previously discussed in our paper, there are practical issues for the implementation and interpretation of these methods that must be addressed.

Despite our ability to monitor and measure a vast array of physiological data, our ability to routinely collect and process this information is limited. In fact, in the majority of intensive care units today this information is still recorded by hand, using paper and pencil. At San Francisco General Hospital we have demonstrated that the recording of high-frequency, multivariate physiological data across multiple patients is feasible. We are currently using these data to calculate the ICP-AUC and are transitioning to using this as part of daily clinical care, along with indices of fever burden and hyperglycemia. We firmly believe that broad advances must be made in neurocritical care informatics to implement the analytical tools suggested by our group, Drs. Filippidis and Fountas, and others. Such systems and tools will allow us to understand more fully the information we already have, and to use this to reduce secondary brain injury, disability, and death. (DOI: 10.3171/2009.2.JNS09153)
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References

Multidrug-resistant Acinetobacter baumannii brain abscess

TO THE EDITOR: We have read with great concern the article by Guinand Vives and colleagues (Guinand Vives CH, Monsalve Duarte GA, Valderrama Beltrán S, et al: Brain abscess caused by multidrug-resistant Acinetobacter baumannii. Case report. J Neurosurg 111:306–310, August, 2009). The authors described the case of a patient with multidrug-resistant Acinetobacter baumannii (MDRAB) brain abscess. We congratulate their success in the difficult situation. However, the following issues require further clarification.

The authors state, “there have been no reported cases in the worldwide literature of brain abscess by this infectious agent.” In 2007, Young and coauthors1 reported a series of 67 patients with MDRAB infection in the surgical intensive care unit. A brain abscess occurred in 1 of these patients. In 2000, Jiménez-Mejías et al.2 reported on a patient with frontal epidural and frontal brain abscesses. Multidrug-resistant A. baumannii menigitis developed 20 days after operation. In 1999, Fernández-Viladrich et al.3 described 5 cases of catheter-associated ventriculitis due to carbapenem-resistant Acinetobacter baumannii with intraventricular colistin sulfomethate sodium. Clin Infect Dis 28:916–917, 1999

RESPONSE: We thank Dr. Liu et al. for their interest in our recently published article.3 They brought to our attention the prior reports of multidrug-resistant Acinetobacter baumannii (MDRAB) brain abscesses in the literature.1,3–5 We mentioned our search strategy in the introduction of the work, and we did not find any article related to brain abscess caused by MDRAB. However, we are thankful to Dr. Liu et al. who noted that a brain abscess was caused by MDRAB in a series of 67 patients in an ICU,3 a case reported by Jiménez-Mejías et al.3 during the postoperative period of a 55-year-old woman who had undergone a craniotomy to resect a relapse of a frontal meningioma, and 6 cases of brain abscesses in the course of MDRAB meningitis in patients with intraventricular catheters.4 Unfortunately, we do not find mention of brain abscess in the article by Fernández-Viladrich et al.3 as suggested by Dr. Liu et al. Nevertheless, we apologize for not having taken into account those previous works, and thanks to Dr. Liu and colleagues, we now know that there were other reports of this uncommon entity in the worldwide literature. We aimed our case report to raise attention of this serious disease to the world medical community. (DOI: 10.3171/2009.8.JNS09608)

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References
**History of detachable coils: the backdrop**

To The Editor: In the July 2009 issue of the *Journal of Neurosurgery*, I published an article on the history of detachable coils (Guglielmi G: History of the genesis of detachable coils. A review. *J Neurosurg* 111:1–8, July, 2009). In this article I described all the steps that eventually led to the invention of detachable coils in 1989. While looking at the article after publication, I had the sense that something was missing. I then realized that the description of an invention should not only consider the scientific aspect, it should also describe the human factor.

In fact, there is something that goes beyond the rational scientific aspect of an invention. During the creative and delicate moments of the conception and also during the research, the interaction with other knowledgeable, humane, and positive persons becomes of utmost importance. Encouragement and scientific advice as well as the injection of enthusiasm can become decisive factors. Without considering these factors, the mere scientific, rational description can somehow be cold.

During the development of the detachable coils, I was helped by 3 pivotal persons: Fernando Viñuela (Director of the Division of Interventional Neuroradiology at the University of California, Los Angeles [UCLA]), Ivan Sepetka (mechanical engineer at Target Therapeutics), and John Robert (Research Associate at the UCLA radiology experimental laboratory). They were a living part of the research that eventually led to the detachable coils. Their intelligence and knowledge as well as their outstanding technical and scientific background were all important factors for the success of the invention. Additionally, the encouragement I received from these 3 people was fundamental to the entire process of the creation of the new technique. I believe that it could be of interest to show the persons who participated in the process that led to the materialization of my original idea (Fig. 1). These photographs were taken in the early 1990s.

Fernando Viñuela was a living part of the entire project. Overall, his role was crucial. I will be forever grateful to him for his advice, suggestions, judgment, and for his genuine support. Furthermore, his vast knowledge, experience, and determination were decisive factors in the implementation and in the clinical application of the detachable coils. He was also emotionally encouraging and enthusiastic, even in the difficult phases of the research, when the enthusiasm may fade. Believing in the potential of my idea, he made all the efforts to bring me to UCLA: since I had no personal funds whatsoever, he succeeded in providing a salary for me and my family. On a more personal note, on my arrival at UCLA in January 1989, since I did not have an office, he immediately gave me a part of his office to work in and to store the materials I had brought from Italy!

Ivan Sepetka constructed the first prototypes of the coils, gave important technical advice, and was always acute in improving the mechanical properties of the coils. I always had the sense that everything was technically possible in his hands. Ivan Sepetka rapidly deciphered and solved all the problems with acumen and technical elegance. On a more personal note, we both liked “unofficial” settings; most fundamental and decisive advancements (like the radiopaque markers and the smaller microcatheters) were made while having lunch, always in the same restaurant, at the same table, drawing the concepts on paper napkins!

John Robert was a practical collaborator with me during all the sessions of surgical construction of experimental aneurysms, and during the sessions of endovascular embolization in the laboratory. He gave me important technical advice based on his longstanding and vast experience and knowledge in the field of experimental animal procedures. On a more personal note, on one occasion he...
stood up to the chairman of our department on my behalf. In fact, he loudly complained that, ever since my arrival in Los Angeles 1 year before, I had been sleeping on a sofa bed, and therefore asked for an increase of my salary so that I could rent a larger apartment!

Putting aside for a moment the scientific contribution, some of the forms of human support I received from these 3 people may sound somehow “collateral.” Instead it was truly decisive, in that it allowed me to serenely focus on the materialization of the original idea and was a key factor for the success of the research. The help I received from them was truly invaluable.

Fernando, Ivan, and John had a definite impact in the success of the detachable coils. I was very fortunate to encounter them in my life. It is my honor to continue my friendship with these 3 special and wonderful persons.

Until now I did not mention the determinant impact my family had on the detachable coils. My wife Nella and my daughters Marta and Silvia constantly and adorably gave me the warmth, tranquility, and spiritual and emotional support I needed most. (DOI: 10.3171/2009.10.JNS091557)

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Reference