Neurosurgical forum
Letters to the editor

Aneurysm Size

TO THE EDITOR: I read with interest the article on the association of aneurysm size and the extent of subarachnoid hemorrhage (SAH) (Russell SM, Lin K, Hahn SA, et al: Smaller cerebral aneurysms producing more extensive subarachnoid hemorrhage following rupture: a radiological investigation and discussion of theoretical determinants. *J Neurosurg 99:* 248–253, August, 2003).

Abstract

Object. The goal of this study was to determine the relationship between aneurysm size and the volume of subarachnoid hemorrhage (SAH).

Methods. One hundred consecutive patients who presented with acute SAH, which was diagnosed on the basis of a computerized tomography (CT) scan within 24 hours postictus and, subsequently, confirmed to be aneurysmal in origin by catheter angiography, were included in this study. The data were collected prospectively in 32 patients and retrospectively in 68. The volume of SAH on the admission CT scan was scored in a semiquantitative manner from 0 to 30, according to a previously published method.

The mean aneurysm size was 8.3 mm (range 1–25 mm). The mean SAH volume score was 15 (range 0–30). Regression analysis revealed that a smaller aneurysm size correlated with a more extensive SAH ($r^2 = 0.23$, $p < 0.0001$). Other variables including patient sex and age, intraparenchymal or intraventricular hemorrhage, multiple aneurysms, history of hypertension, and aneurysm location were not statistically associated with a larger volume of SAH.

Conclusions. Smaller cerebral aneurysm size is associated with a larger volume of SAH. The pathophysiological basis for this correlation remains speculative.

Data on mean SAH volume scores for aneurysms grouped by size (as presented in Table 5 and Fig. 3 in the article) seem to be inconsistent. As shown in Table 5, an aneurysm size of 6 to 10 mm was associated with a mean SAH volume score of 10.7, whereas a mean score of about 13 is depicted in Fig. 3. Similarly, data on larger aneurysms (11–15 mm, mean SAH volume score 5.1 compared with ~10) and greater than 15 mm (mean SAH volume score 1.6 compared with ~8) are not corresponding. Given the high variability of the SAH volume scores, I wonder if the presentation of the data is erroneous.

MARTIN HASSELBLATT, M.D.
Institute of Neuropathology
Münster, Germany

RESPONSE: The data presented in Table 5 and Fig. 3 were reviewed using our statistical analysis program. The means graphically depicted in Fig. 3 were correct, but the means reported in Table 5 were incorrect. The probability value remained highly significant, however. We have provided a revised version of Table 5. We apologize for the inaccuracy, and thank Dr. Hasselblatt for highlighting this error to the readership.

STEPHEN M. RUSSELL, M.D.
JAFAR J. JAFAR, M.D.
New York University School of Medicine
New York, New York

TABLE 5

*Mean SAH volume scores for aneurysms grouped by size**

Aneurysm Size (mm)	No. of Lesions	Mean SAH Vol Score†	SEM
1–5	35	20.2	1.3
6–10	39	14.0	1.3
11–15	18	9.9	1.9
>15	8	8.8	2.8

* SEM = standard error of the mean.

† The mean volume of SAH decreases with increasing aneurysm size ($p < 0.0001$).

Rotational Injury

TO THE EDITOR: This article (Prange MT, Coats B, Duhaime AC, et al: Anthropomorphic simulations of falls, shakes, and inflicted impacts in infants. *J Neurosurg 99:* 143–150, July, 2003) extends the work of two of the authors (Duhaime and Margulies) published by this journal 16 years ago.[1] The methodology used in the most recent paper, although more complex, seems to have produced results similar to the original work in one important aspect.

Abstract

Object. Rotational loading conditions have been shown to produce subdural hemorrhage and diffuse axonal injury. No experimental data are available with which to compare the rotational response of the head of an infant during accidental and inflicted head injuries. The authors sought to compare rotational deceleration sustained by the head among free falls, from different heights onto different surfaces, with those sustained during shaking and inflicted impact.

Methods. An anthropomorphic surrogate of a 1.5-month-old human infant was constructed and used to simulate falls from 0.3 m (1 ft), 0.9 m (3 ft), and 1.5 m (5 ft), as well as vigorous shaking and inflicted head impact. During falls, the surrogate experienced occipital contact against a concrete surface, carpet pad, or foam mattress. For shakes, investigators repeatedly shook the surrogate in an anteroposterior plane; inflicted impact was defined as the terminal portion of a vigorous shake, in which the surrogate's occiput made contact with a rigid or padded surface. Rotational velocity was recorded directly and the maximum (peak–peak) change in angular velocity ($\Delta\dot{\Theta}_{max}$) and the peak angular acceleration ($\ddot{\Theta}_{max}$) were calculated.

Analysis of variance revealed significant increases in the $\Delta\dot{\Theta}_{max}$ and $\ddot{\Theta}_{max}$ associated with falls onto harder surfaces and from higher heights. During inflicted impacts against rigid surfaces, the $\Delta\dot{\Theta}_{max}$ and $\ddot{\Theta}_{max}$ were significantly greater than those measured under all other conditions.

Conclusions. Vigorous shakes of this infant model produced rotational responses similar to those resulting from minor falls, but inflicted impacts produced responses that were significantly higher than even a 1.5-m fall onto concrete. Because larger accelerations are associated with an increasing likelihood of injury, the findings indicate that inflicted impacts against hard surfaces are more likely to be associated with inertial brain injuries than falls from a height less than 1.5 m or from shaking.

One of the conclusions of the original paper is ". . . the shaken baby syndrome, at least in its most severe acute form, is not usually caused by shaking alone. Although

shaking may, in fact, be a part of the process, it is more likely that such infants suffer blunt impact." In the current work, the authors state: "In addition, there are no data showing that the $\Delta\dot{\Theta}_{max}$ and the $\dot{\Theta}_{max}$ of the head experienced during shaking and inflicted impact against unencased foam is sufficient to cause SDHs [subdural hematomas] or primary transient ischemic attacks in an infant."

Given the above conclusion by the same authors who conducted the same experiments with differing methodologies, published in the same journal 16 years apart, it would seem appropriate at this point to pose the question, "Why, then, need shaking be invoked as a causative mechanism in intracranial injury in infants at all, since according to both series of experiments, it does not seem to be a factor in causing such injury?"

Ronald H. Uscinski, M.D.
Lawrence E. Thibault, Sc.D.
Ayub K. Ommaya, M.D., F.R.C.S.
Neurological Surgery
Fairfax, Virginia

Reference

1. Duhaime AC, Gennarelli TA, Thibault LE, et al: The shaken baby syndrome. A clinical, pathological, and biomechanical study. **J Neurosurg 66:**409–415, 1987

Response: Whereas our recent paper has a considerably broader focus that emphasizes falls and the influence of contact surface, Uscinski, Thibault, and Ommaya are correct in pointing out consistency with results reported in our earlier paper.[1] In both papers we measure relatively small rotational velocities and/or accelerations during vigorous shaking, and low height falls are below the thresholds for severe primary brain injuries. At these levels, however, there is a paucity of data in humans, animals, and cadavers, and therefore we cannot postulate whether brain injury would be associated with these events. To summarize, new research is needed to determine if injuries can occur in the brain, cervicomedullary junction, or cervical spinal cord as a result of a single or series of head rotations at these low magnitudes, and if these injuries are primary or secondary in nature. Therefore, we cannot yet answer if shaking can cause intracranial injury in infants, and use of terminology that includes this mechanism should be avoided.

Michael T. Prange, M.D.
Brittany Coats, B.S.
Ann-Christine Duhaime, M.D.
Susan S. Margulies, Ph.D.
University of Pennsylvania
Philadelphia, Pennsylvania

Reference

1. Duhaime AC, Gennarelli TA, Thibault LE, et al: The shaken baby syndrome. A clinical, pathological, and biomechanical study. **J Neurosurg 66:**409–415, 1987

Cerebral Metabolism

To The Editor: I read with interest the article by Zauner, et al. (Zauner A, Clausen T, Alves OL, et al: Cerebral metabolism after fluid-percussion injury and hypoxia in a feline model. *J Neurosurg 97:*643–649, September, 2002).

Abstract

Object. Currently, there are no good clinical tools to identify the onset of secondary brain injury and/or hypoxia after traumatic brain injury (TBI). The aim of this study was to evaluate simultaneously early changes of cerebral metabolism, acid–base homeostasis, and oxygenation, as well as their interrelationship after TBI and arterial hypoxia.

Methods. Cerebral biochemistry and O_2 supply were measured simultaneously in a feline model of fluid-percussion injury (FPI) and secondary hypoxic injury. After FPI, brain tissue PO_2 decreased from 33 ± 5 mm Hg to 10 ± 4 mm Hg and brain tissue PCO_2 increased from 55 ± 2 mm Hg to 81 ± 9 mm Hg, whereas cerebral pH fell from 7.1 ± 0.06 to 6.84 ± 0.14 ($p < 0.05$ for all three measures). After 40 minutes of hypoxia, brain tissue PO_2 and pH decreased further to 0 mm Hg and 6.48 ± 0.28, respectively ($p < 0.05$), whereas brain tissue PCO_2 remained high at 83 ± 13 mm Hg. Secondary hypoxic injury caused a drastic increase in cerebral lactate from 513 ± 69 μM/L to 3219 ± 490 μM/L ($p < 0.05$). The lactate/glucose ratio increased from 0.7 ± 0.1 to 9.1 ± 2 after hypoxia was introduced. The O_2 consumption decreased significantly from 18.5 ± 1.1 μl/mg/hr to 13.2 ± 2.1 μl/mg/hr after hypoxia was induced.

Conclusions. Cerebral metabolism, O_2 supply, and acid–base balance were severely compromised ultra-early after TBI, and they declined further if arterial hypoxia was present. The complexity of pathophysiological changes and their interactions after TBI might explain why specific therapeutic attempts that are aimed at the normalization of only one component have failed to improve outcome in severely head injured patients.

In this study of experimental brain trauma the authors monitor the cerebral energy metabolism and O_2 consumption rate and arrive at conclusions with direct clinical implications. The experimental approach of using a technique for cerebral microdialysis that is available also during neurointensive care is of particular interest. Unfortunately, the study is burdened with severe methodological problems, which render the conclusions questionable.

Evaluation of extracellular or whole tissue lactate/pyruvate ratio is a generally accepted decades-long way of describing cerebral cytoplasmic redox states.[1] The lactate/pyruvate ratio reflects the equilibrium of the lactate dehydrogenase (LDH) reaction and, irrespective of the species studied, the cerebral lactate/pyruvate ratio is close to 20 under physiological conditions. During ischemia and hypoxia there is a rapid shift in the LDH equilibrium and the ratio increases to very high levels due either to a simultaneous increase in lactate and decrease in pyruvate (ischemia, hypoxia with relative ischemia) or to a pronounced increase in lactate and a moderate increase in pyruvate (pure hypoxia). In studies using microdialysis the relative recoveries of lactate and pyruvate are similar and the lactate/pyruvate ratio is accordingly not affected by technical variations such as length of dialysis membrane, perfusion rate, and so on. In the study by Zauner, et al., the lactate/pyruvate ratio was 306 ± 176 in control animals. This value is an obvious technical artifact. It is also difficult to understand how the measured mean levels of lactate (513 ± 69 μM/L) and pyruvate (8 ± 2.8 μM/L) can result in this mean lactate/pyruvate ratio. Since the given lactate/pyruvate baseline value does not represent a control level, it is not possible to interpret the (nonexisting) effects of FPI or subsequent hypoxia. In consideration of the experimental conditions (general anesthesia, 10 mm dialysis membrane, perfusion rate 2 μl/