Letters to the Editor

NEUROSURGICAL FORUM

Flying with a shunt

TO THE EDITOR: The report by Zrinzo et al.¹⁷ (Zrinzo LU, Crocker M, Zrinzo LV, et al: Commercial flight and patients with intracranial mass lesions: a caveat. Report of two cases. *J Neurosurg* 105:627–630, October 2006) on intracranial mass lesions during commercial flight prompted us to consider the effects of flying on children with hydrocephalus and ventriculoperitoneal shunts.

We performed an informal survey, in which a parent active in the local hydrocephalus association queried other parents and children via 4 social media outlets, asking if shunt malfunction symptoms occurred during or immediately after commercial airplane travel. Eighteen of 323 parents (5.6%) responded, 72% of whom reported the occurrence of transient symptoms. The most common symptom was headache, followed by nausea, vomiting, and seizures. Symptoms resolved 24 hours to 2 weeks after flying. These complaints raise the following question: can air travel cause shunt malfunction–like symptoms in patients with hydrocephalus, and if so, why might this occur?

The US Federal Aviation Regulations require occupied cabins to be pressurized to 632–552 mm Hg (8000 ft).^{1,4} At cabin pressures of 575 mm Hg, gas expands to 132% of its volume when at sea level, leading to an elevation of intra-abdominal pressure,⁶ which may cause underdrainage and raised intracranial pressure (ICP) in shunt-treated patients.¹³ It is also reasonable to suggest that hypoxemia may contribute to a mild increase in ICP during commercial flights, especially in children with reduced cerebral compliance,⁶ by increasing cerebral blood flow and triggering the upregulation of vascular endothelial growth factor with subsequent cerebral edema.^{8,9,12,16} Finally, increasing levels of inspired CO₂ during commercial flights may lead to cerebral vasodilation and increased ICP.^{7,17}

In contrast, an overdrainage mechanism may also be responsible for producing symptoms. Many contemporary valves use Delta Chamber (Medtronic PS Medical) antisi-phon devices (ASDs), which have a mobile membrane that moves toward or away from the control orifice when the pressure inside the shunt falls below or rises above atmospheric pressure, respectively.⁵ When atmospheric pres-

sure is lower, less pressure inside the shunt may be needed to move the ASD membrane away from the control orifice, theoretically facilitating overdrainage. Of course, whether these changes actually impact CSF physiology or shunt function remains to be determined. In fact, the Delta valve has been shown to function adequately in hyperbaric conditions, possibly because the pressure is equally distributed along the entire system, resulting in no actual change in pressure gradient.¹¹

Three parents reported changes in their child's adjustable valve's settings. The Sophy, Strata, and Codman-Hakim adjustable valves can be readjusted by relatively weak fields (400,000 mG).³ Magnetic field levels in aircrafts oscillate from 8 to 17 mG.^{10,14,15} Airport walk-through and handheld metal security detectors produce maximum field strengths of 3741 mG and 30 mG, respectively,² making it unlikely that programmable valves are directly reset by these magnetic fields.

One-third of parents who responded did not report shunt-related symptoms during air travel. There is the distinct possibility that these symptoms reported in children with shunt-treated hydrocephalus and attributed to their shunt may simply reflect the pains of modern air travel, which are shared by all of us.

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DISCLOSURE

The authors report no conflict of interest.

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Response

No response was received from the authors of the original article.

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Out-of-body experience and epilepsy

TO THE EDITOR: We read the article of Fang et al.³ with interest (Fang T, Yan R, Fang F: Spontaneous out-of-body experience in a child with refractory right temporoparietal epilepsy. *J Neurosurg Pediatr* **14**:396–399, October 2014). Fang et al. present the case of a 15-year-

old boy with an out-of-body experience (OBE) of epileptic origin. Due to the unfamiliarity of physicians with autoscopic phenomena (AP), the patient had been judged as mentally ill prior to correct diagnosis. The paper includes many important details: the broad differential diagnosis, the frequent misinterpretation of epileptic AP as a sign of a psychiatric disorder, and also the important role of the temporoparietal junction for AP. Our group reported a similar case in an adult patient a couple of years ago.¹

Nevertheless some important aspects of ictal OBE are missing. The authors assume that up to now the frequency of OBE or other AP in epilepsy has not been studied. Devinsky et al.,² however, found already in 1989, that ictal AP were reported in 6.3% of the 158 epilepsy patients when they were interviewed by means of a standardized interview. Therefore, the assumed frequency of ictal AP (1/1500) according to Fang et al.³ represents an underestimation.

The cerebral pathology causing the ictal OBE reported by Fang et al.³ was a focal cortical dysplasia. Unfortunately, the lateralization of the autoscopic sensation was not mentioned. This feature can be used as a valuable lateralization sign,⁴ which is of special interest for neurosurgeons and epileptologists in the context of a presurgical work-up.⁵

In conclusion, ictal OBE is a frequently overlooked seizure symptom that should be explicitly asked for and that may be useful in presurgical diagnostics.

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DISCLOSURE

The authors report no conflict of interest.

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Response

We appreciate the comments of Drs. Brandt and Hoepner very much. We totally agree with them about the frequent misinterpretation of epileptic AP as a sign of a psychiatric disorder, and also the important role of the temporoparietal junction for AP.

In 1500 consecutive epilepsy surgery cases, we identi-

fied 31 patients whose epileptogenic zones were located in the temporoparietal junction. However, only one of these 31 patients had an ictal OBE. Definitely, we admit we probably omitted some patents with AP.

The epileptogenic zone of the right-handed patient we reported on was in the right temporoparietal cortex, which is different from the left temporoparietal lesion in a previous case report.¹ So, the lateralization of the autoscopic sensation needs to be studied more carefully.

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