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Expert Opinions

A Motion Simulator Ride Associated With Headache and Subdural Hematoma: First Case Report

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We report the first case report of symptomatic bilateral subdural hematomas (SDH) associated with riding a centrifugal motion simulator ride. A previously healthy 55-year-old male developed new onset daily headaches 1 week after going on the ride that were due to symptomatic bilateral SDH requiring operative intervention with a full recovery. There was no history of other trauma or other systemic or intracranial abnormality to account for the development of the SDH.
We review the headaches and other clinical features associated with chronic SDH. Twelve cases of roller coaster headaches due to SDH associated with riding roller coasters have been reported. The pathophysiology is reviewed, which we believe is the same mechanism that may be responsible in this case. Although it is possible that this neurovascular injury is truly rare, it is also possible that this injury is underreported as patients and physicians may not make the association or physicians have not reported additional cases. The risk of this injury likely increases with age, as the size of the subdural space increases, and may support the maxim that "roller coasters and simulators are for kids."

Key words: roller coaster, motion simulator, subdural hematoma, headache

Abbreviations: MRI magnetic resonance imaging, msec millisecond, rad radian, SDH subdural hematoma

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INTRODUCTION

Neurological injury in relation to amusement park rides is an infrequent occurrence. These include spinal column and cord damage as well as vascular injuries such as vessel dissections, and subarachnoid and subdural hemorrhage, and hematoma. Precise epidemiological data relating to such injuries are lacking. While the Consumer Product Safety Commission monitors amusement ride-related deaths and injuries throughout the United States, the true incidence is undeterminable.[1] Loder and Feinberg estimated that approximately 9200 children per year receive emergency department care for amusement ride related injuries, although "catastrophic injury and death are rare."[2] Another study by Pelletier and Gilchrist examined this topic and estimated that there are approximately 4 deaths per year in the United States associated with roller coasters.[3]

Subdural hematomas (SDH) associated with blunt trauma can result from multiple sources. These include rupture of a bridging vein, laceration of a cortical vein or artery, hemorrhagic contusion, or rupture of an intracerebral hemorrhage through the arachnoid plane.[4] Approximately one third of acute SDH are the result of bridging vein rupture as

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reported in an autopsy series by Maxeiner.[5] The mechanism most often cited is through rotational acceleration rather than translational motion as hypothesized by Holbourn.[6] This is based on work by Gennarelli and Ommaya in primate models that concludes that purely translation motion is incapable of creating SDHs and a rotational component is necessary.[7–10] Roller coaster rides produce many vector forces with rotational components, and thus are potential generators of this type of injury.

We report the first case to our knowledge of an SDH associated with riding a motion simulator at a theme park.

CASE HISTORY

This is a 55-year-old male seen on October 2, 2014 with a prior history of mild headaches every 2-3 months lasting a few hours without medication. Since June, 2014, he was having daily headaches, daily since onset and initially mild, described as a generalized aching and sometimes throbbing with an intensity of 4-7/10 without associated nausea, light or noise sensitivity. He took ibuprofen with help. He had been going to acupuncture, which he thought helped. The headache was present about 8 hours a day. Butalbital/acetaminophen/caffeine combination and rizatriptan did not help.

He and his wife denied a history of head trauma. However, about 1 week prior to the onset of the headaches, he had been on a centrifuge motion simulator ride, which exposes riders to forces up to 2.5 G.

Since 7/14, he had been sleeping 13 hours a night and was sleepy during the day. Previously, he would sleep 5-6 hours a night without excessive daytime drowsiness. His wife had noticed mild memory problems for a few days.

He was taking only ibuprofen as necessary. Past medical history was negative. No history of cocaine use.

Neurological examination was normal except for a Mini Mental State Examination score of 24/30.

A MRI of the brain on July 24, 2014 and CT of the brain on August 11, 2014 were reported as showing bilateral chronic SDH measuring up to 10 mm in width bilaterally.

Blood tests including a comprehensive metabolic profile, TSH, free T4, erythrocyte sedimentation rate, complete blood count, and coagulopathy testing were normal. An MRI of the brain without contrast on October 2, 2014 showed chronic bilateral hemispheric SDH 12-14 mm in greatest thickness with mass effect on the cerebral hemispheres (Fig. 1).

He was then referred for neurosurgical evaluation. Four-vessel diagnostic cerebral angiography demonstrated the bilateral subdural hemorrhages without any underlying brain or vascular abnormality.

The patient presented for surgical evacuation of the hematomas after serial imaging indicated recurrent hemorrhage and enlarging SDH. This was associated with a clinical decline, with deteriorating mentation and increasing somnolence. On October 10, 2014, drainage was accomplished via bilateral craniotomies. Subdural fluid was encountered under high pressure on the left side. The right side was under low pressure, but had dense and thick blood clots. The collections were evacuated, and no vascular abnormality or acute hemorrhage was encountered. The patient remained at his neurological baseline immediately following the procedure with marked improvement in his headache. He continued to improve, and was discharged. Follow-up imaging demonstrated resolution of the hematomas. On subsequent office visits and telephone follow-up on August 30, 2015, he was back to work at full activities with no residual deficits.

Questions.—What are the features of headaches due to chronic SDH? What is the pathophysiology of SDH? Have SDH been associated with riding roller coasters and simulator rides previously?

EXPERT OPINION

Features of SDH and Headaches.—Swiss physician Wepfer was the first to report chronic SDH in his 1675 book[11] in two patients who he treated and then performed autopsies on.[12] One was an about 50-year-old male who, "A few weeks before his death, a most cruel headache began to affect his entire head, especially the front and the occiput, from which he was given neither relief nor rest. . . .Because of this pain he was at times out of his mind so that he often did not remember whatever he said or did." The second was a

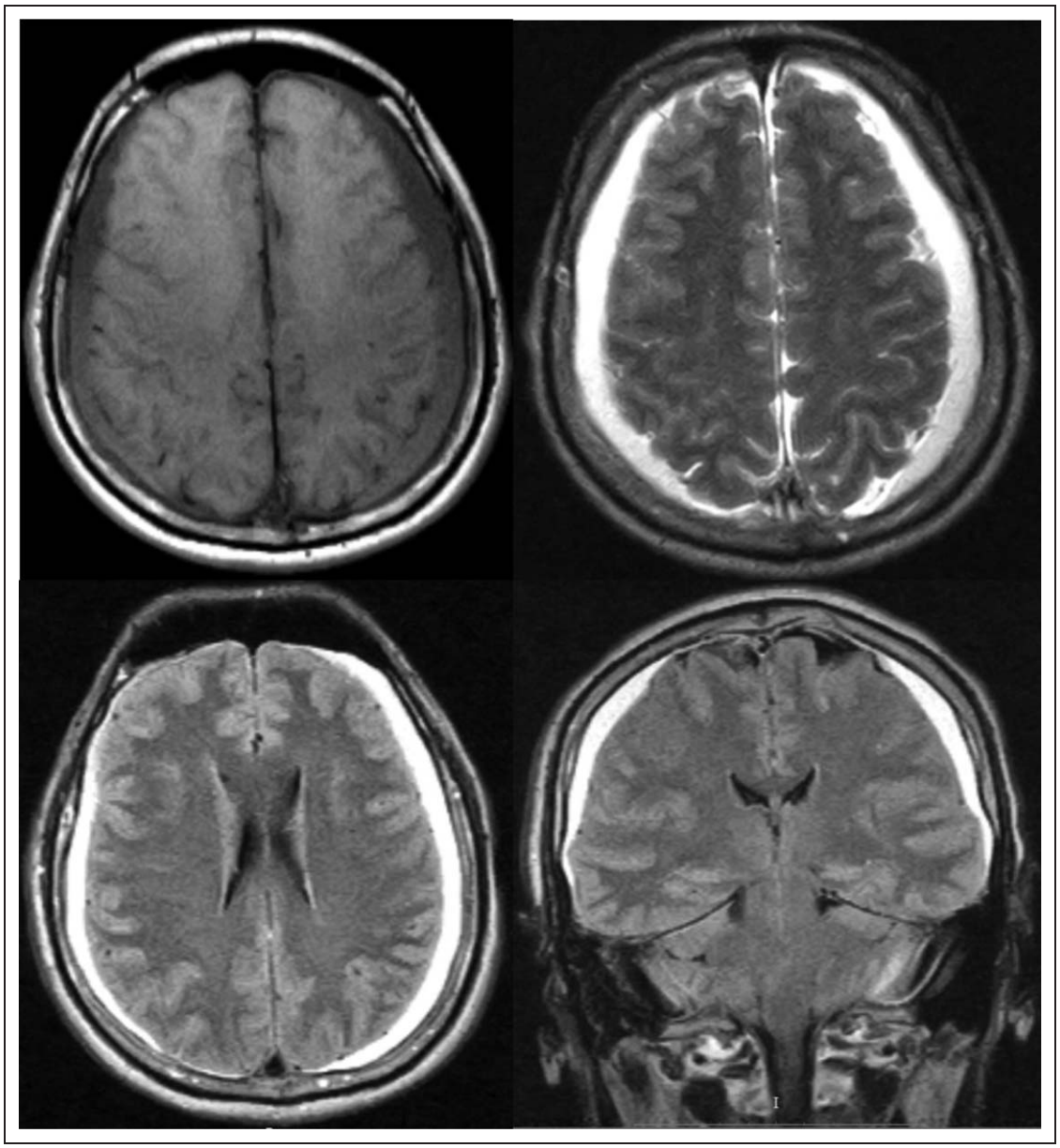

Fig. 1.—Preoperative axial MRI brain T1 (top left), T2 (top right), T2 flair (bottom left), and coronal T2 flair (bottom right) showing bilateral chronic subdural hematomas with mass effect on the cerebral hemispheres.

man in his seventies who had a "steady headache" with occasional vertigo and sometimes vomiting in the morning. Hulke published the first case report on successful surgical treatment of a chronic SDH in 1883 in London.[13]

SDH are usually located over the hemispheres, although other locations, such as between the occipital lobe and tentorium cerebelli or between the temporal lobe and skull base, can occur. An SDH becomes subacute between 2 and 14 days after the injury when there is a mixture of clotted and fluid blood, and becomes chronic when the hematoma is filled with fluid more than 14 days after the injury. Rebleeding can occur in the chronic phase. Most patients who have chronic SDH are late middle aged or elderly. SDH can be present with a normal neurologic examination.

A review of a recent surgical series is illustrative of the clinical features of chronic SDH. In a series of 778 patients who underwent surgical

drainage of chronic SDH,[14] symptoms reported, similar to other studies, were as follows: headache, 58.9%; hemiparesis; 34.8%; cognitive disturbances, 22.6%; altered behavior, 35.0%; and seizure, 2.6%. Headache was more likely the presenting symptom in younger patients while behavioral changes were more likely the presenting symptom in those age 65 or older. Similar to other series, the mean age was 64 years (range 14-93 years) with 56.8% 65 years or older, predominantly males (82.6%), and usually occurred after trivial or mild head injury (69.4%) but unclear origin in 36.1%. Convexity hematomas were left sided in 37.7%, right sided in 40.0%, and bilateral in 22.3%, with bilateral SDH more common in younger than older patients.

Headaches associated with SDH are nonspecific, ranging from mild to severe and paroxysmal to constant.[15] Unilateral headaches usually are the result of ipsilateral SDH. Headaches associated with chronic SDH have at least one of the following features in 75% of cases: sudden onset; severe pain; exacerbation with coughing, straining, or exercise; and vomiting and or nausea.

Similar to many cases of chronic SDH, this patient presented with a new onset generalized headache associated with cognitive abnormalities as well as hypersomnia.

Pathophysiology of SDH.—Post-traumatic SDH can be attributed to three causes: extension of a hemorrhagic contusion or intracerebral hemorrhage, tearing of a bridging vein or, rarely, laceration of a cortical vessel. Classically, most SDHs result from tear or rupture of a bridging vein. Trotter first presented the concept that an anterior–posterior blow is the critical event for vein hemorrhage, as the falx is unable to protect the brain from an anterior–posterior traction injury, in contrast to a lateral vector force.[16] This theory was later refined by Leary, who focused on the microanatomy describing the bridging portion of the cortical veins as thin walled without an elastic or muscular layer and lacking the reinforcing protection of the arachnoid enjoyed by the subarachnoid veins.[17] Four decades passed before Yamashima objectively described some of these characteristics where subarachnoid veins are relatively uniform in wall diameter ranging from 50-200 μm thick with dense reinforcement by the arachnoid trabecular cells. In contrast, the subdural portion of the veins are variable in wall thickness from 10-600 μm with a loose arrangement of fibers in a circumferential manner rather than longitudinal, making them more prone to a traction injury.[18]

Depreitere et al studied the biomechanics of SDH in a cadaver study using cortical impact while perfusing the venous structures with barium sulfate.[4] The heads were examined with fluoroscopy at autopsy. This study is an "approximation of reality," but the authors were able to produce SDHs in the Rolandic or post-Rolandic region with a critical threshold of 10,000 rad/second^2 with a pulse duration less than 10 milliseconds, which decreased with increased pulse duration. Another author found 4500 rad/second^2 as the minimum threshold for disruption of bridging veins.[19] Bridging veins can rupture with significant pure translational motion; however, rotational acceleration is much more efficacious and is estimated to create 2-2.5 times the force on the forward draining veins.[7,20]

A significant weakness associated with models used to study head motion, impact, and strain tolerances is the variability introduced by the materials (cadaver age, length of time to harvesting, animal models). Correlation with living state is further complicated by individual variances such as age, brain atrophy, condition of the vessels, and neck length and vessel strength that may alter the rotational acceleration calculations.[7,21–25] Smith and Meaney used G-force data from three popular roller coasters, applied to a mathematical model to estimate maximal rotational acceleration, and found a potential maximum of 502 rad/second^2.[26] This is below estimated thresholds for bridging vein rupture in the models cited previously. Pfister et al studied three-dimensional head motions during three different roller coaster rides, a pillow fight, and in car crash simulations.[27] They found rotational acceleration values as high as 1847 rad/second^2. The reported linear and rotational components of head acceleration during roller coaster rides were within the range of many common activities. As such, they concluded that there appears to be a low risk of traumatic brain injury (TBI) due to the head motions induced by roller coaster rides, but they did not specifically consider SDH.

While much progress has been made in the study of head injuries, comparison between approximations generated by cadaver labs for bridging vein rupture thresholds at peak values of rotational acceleration and those measured on a roller coaster are inadequate for definitive statements of causality. Common models used are the head injury criterion and head impact power which report head motion as a function of time. Individual peak values of acceleration alone are not adequate to predict the risk of a brain injury."[27] The head injury criterion model, based on the Wayne State Tolerance curve, was first developed in the 1960s and has undergone continuous revision.[28] It is based on translational accelerations and not rotational forces, and may not be an appropriate or at the very least an incomplete model for the assessment of head injuries.[29] Recent studies and models use a rotational brain injury criterion and devices with accelerometers such as the six degree of freedom football helmet and Hybrid III system used in studying sports injuries.[30–34] It will be interesting to see what the results will be when newer models and technology can be applied to the question of roller coaster and motion simulator safety and head injury including concussion, axonal injury, and SDH.

SDH, Roller Coasters, and Motion Simulators.—SDH formation following roller coaster rides has been reported 12 times previously and is more than just a chance occurrence.[21,35–45]

We are not aware of any prior reports of SDH associated with riding a motion simulator. However, it is plausible that the rapid change in acceleration could cause bridging vein rupture.

As one ages, the brain atrophies, and the size of the subdural and subarachnoid spaces increase. This, in turn, exposes a larger portion of subdural bridging veins to the subdural space, where they are more prone to tear. The addition of other medical conditions in which the vasculature is affected or for which anticoagulants are administered likely further increases the risk of developing a SDH.

CONCLUSION

We hypothesize that, in our case, this 55-year-old man was more susceptible to developing an SDH because of his older age and a larger subdural space. The normal atrophy of the brain that occurs with age may predispose older individuals to a higher risk of developing subdural hemorrhages from a variety of sports activities where cranial rotational vector forces occur, including a centrifugal motion simulator and roller coaster rides. Although it is possible that this neurovascular injury is truly rare, it is also possible that this injury is underreported as patients and physicians may not make the association or physicians have not reported additional cases. We believe that the maxim that "roller coasters and simulators are for kids" may have a scientific basis, and is worth emphasizing.

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