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# Short Communication

# Roller Coasters, G Forces, and Brain Trauma: On the Wrong Track?

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## ABSTRACT

**There has been enormous attention in the general press on the possibility that high G force roller coasters are inducing brain injury in riders. Armed with a handful of anecdotal case reports of brain injuries, the U.S. Congress has recently proposed legislation to regulate the level of G forces of roller coasters. However, high G forces are well tolerated during many activities and, therefore, are a poor measure for the risk of brain injury. Rather, accelerations of the head that can be caused by G forces are the key to producing injury. To determine the extent of head accelerations during roller coaster rides, we acquired G force data from three popular high G roller coasters. We used the highest recorded G forces in a simple mathematical model of head rotational acceleration, with the head rigidly pivoting from the base of the skull at a radius representing typical men and women. With this model, we calculated peak head rotational accelerations in three directions. Even for a conservative worst-case scenario, we found that the highest estimated peak head accelerations induced by roller coasters were far below conventional levels that are predicted for head injuries. Accordingly, our findings do not support the contention that current roller coaster rides produce high enough forces to mechanically deform and injure the brain.**

**Key words:** G force; head rotational acceleration; roller coaster; traumatic brain injury

RECENTLY, THERE HAS BEEN MUCH ATTENTION focused on the possibility that larger and faster roller coasters with high G forces (G's) are inducing brain injury in riders. A series of case reports appearing in medical journals have described hemorrhage in the brains of some roller coaster riders (for review, see Braksiek and Roberts, 2002), calling into question whether these injuries could have resulted from the forces experienced during the rides. In the general press, news reports have described the perils of riding high-powered roller coasters, such as stories from the *Los Angeles Times*, "As thrills increase, risks to brain rise" (6/5/01), and the *Washington Post*, "The thrill is . . . Deadly" (5/21/02). Through efforts spearheaded by Representative Edward Markey of Massachusetts, the U.S. Congress is turning its attention to roller coaster safety, and legislation is being proposed to regulate G forces induced by roller coaster rides. However, absent from all of this fanfare is any sound evidence or analysis directly linking roller coasters with brain injury. Here, we examine if roller coasters actually pose a risk and point out a fundamental misunderstanding of how G forces play a role in the biomechanics of brain injury.

Clearly, as new roller coaster designs incorporate greater vertical drops, the G's increase, as do the visceral sensations of the riders. The current upper range of peak

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G forces on the more powerful rides is 4–6 G's, as listed on Rep. Markey's Amusement Park Ride Safety website (www.house.gov/markey/iss_parkrides.htm) and from the Roller Coaster DataBase (www.rcdb.com). However, it is far too simplistic to use the G's alone as a measure for the risk of brain injury. For example, 5–9 G's is thought to be the maximum exposure limit for a human based on the tolerance of fighter pilots exposed to high G's (Whinnery and Whinnery, 1990). Yet, this threshold is for sustained G's over many seconds (mean of 43 sec), which will cause unconsciousness from reduced blood flow to the brain. Roller coasters apply only brief accelerations (<3 sec) in different directions throughout the ride, with little chance of inducing unconsciousness from pooling of the blood in the extremities. Furthermore, high G's of short duration are common and well tolerated in many daily activities, such as hoping off a step or "plopping" into a chair, where 8–10 G's have been measured in volunteers (Allen et al., 1994). Accordingly, G force alone is not a good measure for the risk of brain injury. Rather, *head accelerations* that can be caused by G forces are the key to producing injury (Gennarelli, 1993).

To induce nonimpact brain trauma, several reports have demonstrated the importance of head rotational acceleration caused from loading applied elsewhere on the body (Gennarelli, 1993; Meaney et al., 1995). The indirect loading can occur from the seat belts that restrain an occupant during a motor vehicle crash or, in the case of roller coasters, the acceleration delivered through the seat of the occupant. Brain injury due to rotational acceleration is dependent on very rapid deformations of the brain, typically within a time span of less than 50 msec (Metz et al., 1970). It is now well recognized that rapid head rotational acceleration can initiate several significant brain injuries, including diffuse axonal injury throughout the white matter, and, at very high levels of acceleration, tissue tears and vascular disruption (Adams et al., 1982; Gennarelli et al., 1982; Smith et al., 2000). Recent studies from humans, animal models, physical models, and emerging computational simulations provide guides for the human tolerance to rotational accelerations of the head (Pincemaille et al., 1988; Lowenhielm, 1974; Margulies and Thibault, 1992; Zhang et al., 2001; Meaney et al., 1995; Gennarelli et al., 1982; Smith et al., 2000). Although each roller coaster will expose its riders to unique G profiles and, in turn, unique head rotational accelerations, it is possible to approximate the rotational accelerations and place the loading in the context of known criteria for traumatic brain injury. Three basic features of G forces experienced by riders contribute to the resulting head acceleration: (a) the acceleration magnitude, (b) the principal acceleration direction, and (c) the time interval over which each significant acceleration occurs. It is necessary to account for all these features in order to truly understand how different environments can pose risks to humans.

To estimate noncontact head accelerations induced from roller coasters, we acquired G-force data from "high G" rides at three parks: (1) "Rock 'n' Roller Coaster" at the Disney-MGM Studios in Lake Buena Vista, FL, (2) "Speed—The Ride" at the Nascar Café, Sahara Hotel in Las Vegas, NV, and (3) "Face-Off" at Kings Island, OH. We used this data in a mathematical model to determine a worst-case scenario of head accelerations. Analysis of the temporal G force data during the course of the rides revealed that the accelerations in a roller coaster vary from side-to side, fore-to-aft, and in the up-down direction throughout the ride. Typically, the accelerations experienced along the up/down direction are higher than accelerations applied in the fore/aft and side-to-side direction. During different segments of the ride, the relative contribution of each acceleration component can change. Moreover, the time over which these accelerations are applied can vary considerably. As a first approach, we related G forces at the seat level to head accelerations of the occupant. For the worst-case condition from measured data, we used the maximum peak acceleration over the shortest duration. We approximated the head as pivoting stiffly about the base of the skull with the acceleration at the seat transferred directly to the junction between the head and the neck. Over the range of neck and head sizes (5th to 95th percentile) that correspond to the male and female population, we estimated the head rotational accelerations ($\ddot{\theta}$) that occur during the ride:

$$\ddot{\theta}_{\text{peak}} = \frac{a(t)}{r}$$

where $a(t)$ is the acceleration at the base of the skull and $r$ is the radius (female, 10.56–11.33 cm; male, 11.15–11.63 cm), from the head center of gravity to the pivot point. For the maximum side-to-side (coronal plane) acceleration during the rides with maximum G's of 1.2–4.2, head rotational accelerations are 111–387 rad/sec$^2$. Similarly, for the fore/aft (sagittal plane) accelerations measured in the rides with maximum G's of 1.65–5.4, the most significant estimated head rotational acceleration was 139–502 rad/sec$^2$. In the vertical direction towards the seat, peak high G's of greater than 5 were produced. However, vertical accelerations are transmitted along the axis of the spine and would induce only modest head rotational accelerations. It is important to note that our calculated rotational accelerations are highly conservative estimates. Actual head accelerations of human riders are likely to be lower than peak estimates due to dissipation of the G's through the body and by cervical spine articulation.

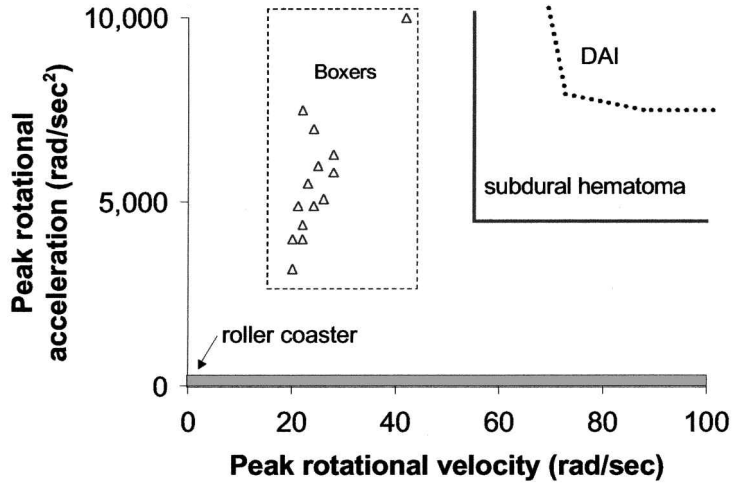


**FIG. 1.** Relative comparison of the predicted head accelerations experienced by roller coaster riders to thresholds proposed for brain injury. Thresholds depend on the magnitude and duration of applied acceleration. Therefore, criteria are defined using peak rotational acceleration and peak rotational velocity limits. Thresholds have been proposed separately for subdural hematoma from the tearing of parasagittal bridging veins and diffuse axonal injury (DAI). The significant head accelerations experienced by human volunteers in a three-round boxing match are also shown (symbols). None of the volunteer boxers experienced any signs of injury. The maximum predicted head accelerations of roller coaster riders (gray shaded region) are well below the proposed tolerance limits, as well as the measured safe accelerations in the volunteers.



Even for a conservative worst-case scenario, we found that the estimated head rotational accelerations experienced by roller coaster riders are nowhere near the range of established injury thresholds for severe forms of brain injury. For tearing of parasagittal bridging veins that can cause subdural bleeding, a minimum head rotational acceleration of 4,500 rad/sec$^2$ has been determined for human subjects (Lowenhielm 1974), which is over nine times our highest predicted accelerations during roller coaster rides. Using an alternative analysis of the kinematics of brain tissue deformation during head rotational acceleration, the threshold for diffuse axonal injury in the white matter was determined as 9,000 rad/sec$^2$ (Margulies and Thibault, 1992), 18 times higher than the highest predicted maximum we calculated for roller coaster riders (Fig. 1). Recent studies measuring the head accelerations experienced by human volunteers in a three-round amateur boxing match (Pincemaille et al., 1988) are also well above the predicted head accelerations during roller coaster kinematics (Fig. 1). None of the boxers showed any signs of significant brain injury or even concussive-type symptoms.

It should be noted that the thresholds we have cited for brain injury apply to normal, healthy individuals. Although some case reports have described rupture of preexisting vascular malformations in the brains of roller coaster riders (Braksiek and Roberts, 2002), it is unknown whether these individuals had a reduced tolerance to head accelerations. However, it is well recognized that hemorrhage from vascular malformations can occur during many activities that do not mechanically deform the brain. Factors other than head accelerations should also be considered in these cases, such as hypertension from the excitement of the ride.

In the general press, there seems to be confusion between increased reporting of brain injuries following roller coaster rides and an actual increased incidence. To our knowledge, no peer-reviewed studies have found a risk of brain injury by riding newer, more powerful roller coasters, let alone measuring the possible increase risk factors that could occur with preexisting vascular malformations. While waiting for this issue to resolve, we highly recommend that all roller coaster riders use a proven method to reduce the risk of brain injury: make sure your seatbelts are buckled at all times when driving to an amusement park.

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