Ow! I Hit My Head: The Sheer Force of it All

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Abstract

This study investigated the efficacy of a simplified model of brain damage that was presented at the Kids Judge competition in Pullman, WA. Children participating in the Kids Judge competition were introduced to the concepts and mechanisms of brain damage using an interactive model that identified three areas of trauma: (1) axonal shearing, (2) cerebral swelling, and (3) chemotoxicity. The children were asked to rate the presentation in four areas: (1) could they understand the project, (2) were the presenters friendly, (3) was the project fun, and (4) would they like to learn more about the topic. The children were also required to rank the presentations they participated in. The model of brain damage that was presented received high marks in understanding, friendliness, and fun, but received a comparatively low mark in the desire to learn more. The model was also ranked as second out of four presenters in the pool it was placed. There was a positive correlation between the mark received for the desire to learn more and the project rankings. These results suggest that the model of brain damage was effective in presenting brain damage to the children and further modification designed to increase the desire to learn more about the subject is the only necessary change the model requires.

Introduction

People fall, get in car accidents, ride without helmets, and generally slam their heads around on a daily basis. In fact, there is even a popular dance style, termed head banging, which enjoys popularity as self-inflicted head trauma. The simple conclusion; people take their brain for granted. What is generally believed to be a fairly well protected organ, even in most medical textbooks, has a potential much greater then any other organ for traumatic damage.

Though the brain is generally protected due to a layer of fluid surrounding it, impact forces and damage to blood vessels can cause major problems to arise. As with any organ, damage causes inflammation as fluid rushes to the site of trauma, unlike most other organs the brain is encased in a hard thick and unmovable cage of bone that does not allow for very much swelling. When the brain begins to swell, blood vessels can be restricted limiting blood flow and killing tissue, mechanical damage from the force of the brain pushing against the bony projections of the skull causes further cell death, and swelling in ventricles can lead to cell death within the brain as well (Fishman, 1975). However, swelling is only the beginning.

The first true characterization of forces involved in brain damage as reported by Denny-Brown and Russel (1941), examined what might really happen when we allow our cranial cavity to move around unhindered. It turns out that the cortex is fairly resilient to damage, more recent studies have linked this particular resilience to the ability of the cortex to compensate by using other pathways to convey the same information (Hammoud & Wasserman, 2001). This resilience, unfortunately, does not carry into the axons that run throughout the “white matter” of the brain. Studies involving car accidents, bicycle falls, and other trauma that involved intense acceleration and deceleration forces noted that the twisting and pulling of axons causes shearing of both the related blood vessels and the axons themselves (Shaw, 2002). This shearing effect is most prominent in the brain stem and can cause profound difficulties even if the cortex remains generally intact.

Those who have experienced a traumatic brain injury (TBI) generally have a number of cognitive and motor deficits including memory loss, coma, difficulty learning, reduced speed of processing, loss of hearing/vision, and weakness in limbs. This gross damage is most likely due to the fact that large numbers of axons have been torn, twisted, or cut and there is simply no more pathways to carry the necessary information between cells. Even without significant impact, acceleration and deceleration forces can cause tremendous damage to brain tissue.

Recently, as techniques have become more precise and allowed for examination of more minute details, a third type of brain damage resulting from trauma has been discovered. As axons twist and pull under the forces responsible for brain damage they begin to swell. The exact mechanisms of this swelling are still being examined. Accompanying axonal swelling is the increased release of glutamate at synapses. Glutamate, a neurotransmitter normally found in the brain, is toxic to nerve cells in large concentrations, thus as glutamate is released cells begin to die. This progressive cell death means that individuals with brain trauma often do not reach the full extent of damage until up to a few days following injury. In children, this pattern of damage can continue even longer, and the extent of damage may not be apparent until a few weeks have passed. The reason for glutamate release is that axonal swelling is accompanied by an influx of calcium, which stimulates neurotransmitter release and is potentially causing glutamate release. Further chemotoxic death can occur amongst the “gray matter” itself as sodium-potassium pumps become disrupted leading to osmotic pressure build up and cell destruction (Bullock, et al., 1991; Choi, 1988).

A combination of these three mechanisms of brain damage is usually seen in all individuals with traumatic brain injury. The extent of damage is linked to the force of impact, the amount of acceleration and deceleration involved, and the final extent of chemotoxic damage that occurs at the synapses (Gaetz, 2003). Unfortunately, the brain is simply not a resilient instrument and is less able to repair itself then most other tissues, this means that damage within the brainstem and cortex can endure for a lifetime leading to extended difficulties and complications.

It is important that the need for brain safety is clearly understood; yet the mechanisms of brain injury consist of difficult concepts centered in both anatomy and physiology. In order to elucidate brain damage for a younger audience it is necessary to simplify the mechanistic explanation. To do this, we adopted a simple model demonstration that we believe effectively illustrates brain damage mechanisms to a younger audience. The success of the model we designed was tested at the Washington State University Kids Judge Neuroscience presentation.

We believe that our model of brain damage was effective in engaging the children, presenting the information in an understandable manner, and teaching them about the mechanisms of brain damage.

Method

Participants

Elementary school children from Pullman, WA who participated in the Kids Judge Neuroscience fair as part of Brain Awareness Week.

Materials

To construct the model we required one large piece of poster board to place pictures of the brain and spinal cord to show the children how the brain was connected to the rest of the body and what the inside of the skull looked like. One bicycle helmet and one chair were used to demonstrate a bicycle rider. 6-10 Styrofoam cups (depending upon how many kids) and 10 plastic, hollow practice golf balls were used to demonstrate the chemical messengers passed across the synapse and chemotoxicity. One Styrofoam cup with a tack in it and one balloon were used to show how brain swelling could cause damage. We also required an adequate amount of space to have the children stand up and hold hands in a line to demonstrate axonal connections and shearing. 10 signs, 5 reading brain and 5 reading spinal cord, were given to the kids when they first arrived.

Methods

When the children came to our station we split them into two equal groups (when possible) and assigned brain signs to one-group and spinal cord signs to the other group. We had the kids remain standing and form a chain with the brain group located on one end and the spinal cord group on the other. We then explained how an action potential is passed from the brain down the spinal cord to the muscles to tell them to move. To facilitate the explanation, one of us sat on the chair and acted like we wanted to ride our bike, we did not wear our helmet. The first brain child was told to tell the last spinal cord child to move the feet of the bike rider by sending an impulse, by way of squeezing hands, to the spinal cord. Upon completion by the children, the rider began to move his legs. While “riding” the bike the other presenter threw a small plastic golf ball at our head to simulate an impact and the bike rider reacted as if they had been struck. We explained to the children that such an impact would cause three things to happen.

First we explained that outside force against the skull can cause the brain to whip around inside of the cranial cavity and we asked if any of the kids had heard of whiplash. We then had the kids act out what would happen to the brain in this situation by continuing to hold hands and having the brain group run back and forth to simulate the whiplash effect. Naturally, during the course of their movement it became more difficult for the children to continue holding hands and we explained the breaking of these bonds as axonal shearing. The children were then asked to try again to pass a signal to the spinal cord, but with broken axons this was not possible.

Secondly we addressed the issue of brain tissue swelling that can occur due to trauma to the blood vessels in the brain. One of us used the Styrofoam cup with the pin in it as the skull and a balloon as the brain. The balloon was blown up a little to show how the brain sits inside the protective skull. To demonstrate swelling, the balloon was filled with more air until it came in contact with the pin and popped (you may have to force the pin into the balloon to have it pop). We explained to the children that, though your brain does not pop, it is still damaged when it swells and is pressed against the skull.

Finally we had the children sit down in two rows facing each other. One row was made up of the brains and the other of the spinal cords. The spinal cords were given Styrofoam cups and the brains were each given a small, plastic practice golf ball. We explained to the children how at each one of those connections that they made with their hands there was actually a chemical transmission that occurred and we had them demonstrate this by rolling the golf ball into the Styrofoam cup to simulate neurotransmitter release and uptake. We had them do this a couple of times to get the hang of it. Then we explained that when the brain is damaged, there could be chemical leakage due to cellular tearing, blood, and trauma to tissue. One last time we had the kids roll the "neurotransmitters" across the floor, but this time one of us threw in a large handful of more plastic golf balls from the side to simulate toxic chemicals. The toxic chemicals knocked some of the neurotransmitters aside and crowded the synapse, which made it impossible for a signal to get transmitted.

At the end of our demonstration we gave each kid one balloon and told them to protect their brain, we then had them tell us what the three types of brain damage that could occur were. As a last point, we asked them how the bike rider could have avoided such injury with the intent that they would respond "he should wear his helmet."

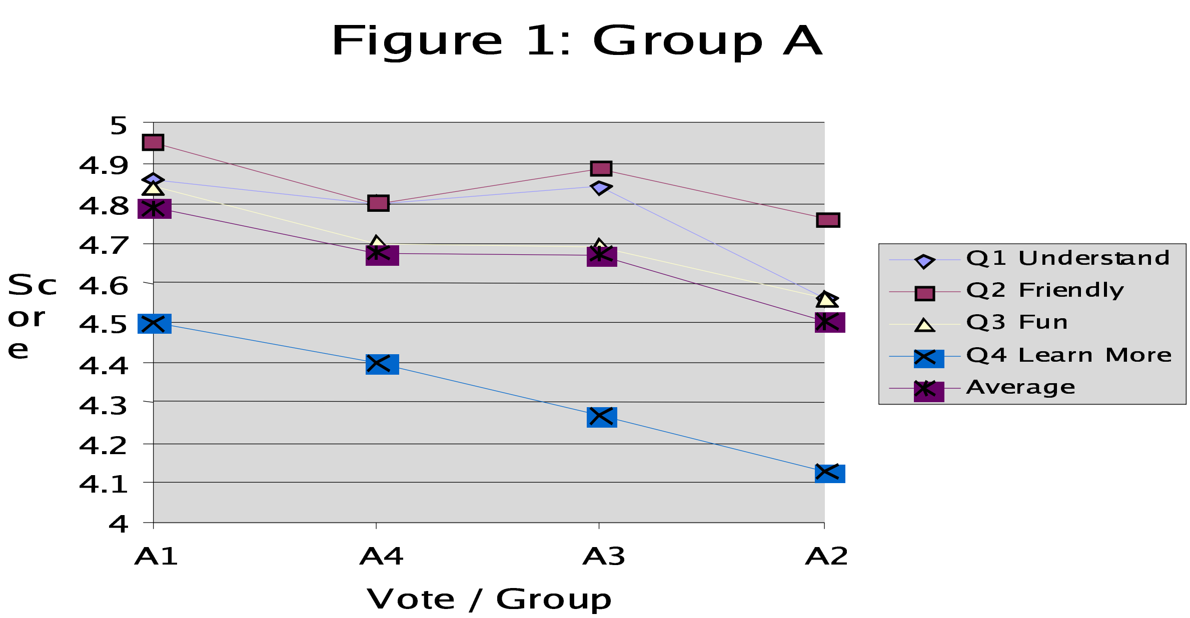
Results

Our project was ranked second place by the kids who attended the Kids Judge competition. The averages of the scores we received are shown in table 1 below and a comparison of the projects in our judging pool is shown in the corresponding figure 1.

Table 1: Average Kids Judge Scores

Measure:	Understand	Friendly	Fun	Learn More	Overall
Average Score:	4.8	4.8	4.7	4.4	4.68

Note: Scores are out of 5



The graph shows that there was a positive correlation between the children's desire to learn more about the subject and the placement of the project.

Analysis of the judging sheets provided by the children upon the completion of the Kids Judge competition allowed us to see what part of our project they most enjoyed. A large number of kids indicated in the further comments section that rolling the golf balls was a favorite activity of theirs.

Observation during the kids judge event indicated, to us, that the children enjoyed our presentation. They did not attend as well when we were trying to explain certain aspects of the model, particularly when discussing how chemicals affect the brain, as compared to the activity portion of our presentation. However, at no time did the children become unmanageable and we were able to run through the entire model successfully each time. The children were able to tell us what whiplash was when we had them simulate the head jerk and many asked if damage to the connections that we discussed could be the cause of paralysis. We felt the model was successful in conveying the serious consequences of brain trauma.

Discussion

The second place ranking and overall score of 4.68 indicates that our presentation was a success in conveying the idea of brain damage to the children. We scored particularly high on understanding, meaning that the model was not too complicated for the children to understand which was one of our major concerns with presenting the mechanisms of brain trauma. At the finality of each presentation, we were also able to elicit the "he should wear a helmet" response further indicating that the children understood the importance of protecting their brain.

The average score we received on fun was slightly lower then that of understanding, but it was still relatively high and suggests to us that the model was engaging and entertaining enough to keep the children interested. However, our score for the desire to learn more was quite low compared to the rest of the scores we received. It can be seen by figure 1 that this score was relatively low for the entire group, but since it showed an important correlation to the ranking of the projects it is a necessary score to attend to. In order to increase the children's desire to learn more about brain damage, it would be advantageous to include a summarizing remark and small demo related to brain regeneration or long-term motor and cognitive deficits that are a result of traumatic brain damage. A very simplistic anatomy demo that explains how damage in certain areas can have vastly different physical effects may be an effective modification of the model. By including a final word that points out these possibilities it may help to increase their desire to find out more about how brain damage can affect their lives.

The model we presented was certainly effective for use with a younger audience, however it does sacrifice accuracy in order to be simpler to understand. For instance, we presented axonal shearing as the simple breaking of axons at junctions, but in reality axonal shearing can be caused by small tears in the membrane that cause axonal death, it can also be caused by rotation strains as the brain twists inside the skull. Swelling of the brain is also much more complicated then we presented, and it involves a number of intermediate steps including damage to blood vessels and inflammation within the cortex as fluid rushes to the sight of injury. Finally, we had to sacrifice quite a bit of accuracy to explain chemotoxic effects of brain damage. Chemotoxicity was indicated, by us, to be the rushing of "toxic" chemicals like blood into the synapses causing the cell signal to become interrupted, however the actual mechanism of chemotoxicity is much more complex. The majority of damage due to chemotoxicity is actually due to the neurotransmitter glutamate. When the axons are stretched and torn it causes mechanical calcium channels to open on the surface of the cell allowing calcium ions to rush into the post and pre-synaptic buttons. Calcium stimulates the over release of glutamate and glutamate, in high concentrations, is quite toxic to the neuronal cells.

Though we had to sacrifice accuracy to present the model of brain damage, overall we feel that the model was successful in presenting the general concepts of brain damage, keeping the children entertained, and giving them a greater appreciation for their brains.

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