Concussions in Baseball from Non-Linear Oscillations of the Brain

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INTRODUCTION

It has been found that head injuries in baseball may comprise up to 18.5% of all competitive sports-related injuries (Beyer). In baseball, a traumatic brain injury (TBI)—any injury that causes damage to the brain as a result of a blow, bump, or jolt (U.S. National Library of Medicine)—can occur following a head impact with a ball that is thrown, pitched, or batted. The most dangerous of these—by far—comes following a pitch hit directly back at a pitcher. The bat exit velocity (BEV), the velocity at which the baseball comes off the bat, has been found to be approximately 45 m/s (100 mph) for metal bats, which are used in competitive baseball up until the professional level (Dainty). This value is more than enough to cause a TBI. The danger in these situations are the impacts which the seriousness is not as apparent and can cause a mild traumatic brain injury (mTBI).

These mTBIs have been defined as a traumatic brain injury in which a number of different things may happen: any loss of consciousness, any memory loss of what happened immediately prior to the event, any altered mind state (i.e. confusion or feeling dazed), or neurological deficits which may not be visible. All of these, with the exception of the lack of visible neurological deficits, are also characteristic of more serious TBIs. The standard is as long as the severity does not exceed a loss of consciousness for more than 30 minutes, post-traumatic amnesia of more than 24 hours, and does not go below a 13 on the Glasgow Coma Scale (Laker) then no TBI has occured. Zhang et al found that head injuries occurred following peak brain accelerations of 61-144 G, while non-injury cases following impact ranged from 32-102 G (Zhang). Some players returned immediately into play following an mTBI despite the risk of worsening the injury they have already endured as little to no symptoms were displayed.

The danger with these players returning is recent findings which pin the cause of Chronic Traumatic Encephalopathy (CTE) to the suffering of multiple mTBIs (Turner et al., Guskievicz et al., Gavett et al., Daneshvar et al.) CTE is a degenerative brain disease which is typically found in athletes, military veterans, and others who have a history of repetitive head trauma, which has been well known to result in progressive neurological deterioration. These repetitive traumas can be a result of either/both blast-induced (a result of close proximity to explosion described by McKee and Robinson) traumatic brain injury (TBI) and sports related concussions (Chen et. al). This deterioration was first associated with boxers in the 1920s (McKee et al.). —Previously nicknamed “punch-drunk”, there is widespread literature of CTE associated with boxing, but due to more recent findings in association with CTE in former football players, more studies have emerged (Mez et al Mez et al. found (“Clinicopathological Evaluation of Chronic Traumatic Encephalopathy in Players of American Football”) that out of 202 deceased American football players, CTE was neuropathologically diagnosed in 177 of them (87%). Of these 177, 111 of these players were former NFL players, of which CTE was discovered in an astounding 110 of them (99%) (Emanuel). Another common injury is a result of a line-drive being hit directly at a pitcher following a pitch. This can happen at varying velocities, but it is clear that lower velocity impacts, which may not display the serious symptoms of those higher velocity impacts, are important to observe.

Due to the little amount of knowledge of sports-related mTBIs and its association with CTE, it seems prudent that more must be discovered on the topic to prevent further degeneration after suffering a head injury which may not be discovered at the time of the incident. A brain movement model was generated using non-human primate brains by Matlack et al. Another model was generated by John Hopkins’ Whiting School of Engineering as a method of determining areas damaged following impacts, but none have been used to attempt to treat the brain as a non-linear oscillator which can stretch and compress to tear the brain tissue from the inside. We have found no research attempting to link the stretching and compressing of the brain resulting in shearing as a possibility for CTE. With the knowledge that CTE is a degenerative tissue disease, it would seem that this method of damage could be cause the initial start of tissue degradation, which is then only intensified as more impacts occur. With this possibility in mind, this experiment was performed to observe the brains movements following very light impacts. This was accomplished by firing baseballs from a pitching machine at a model skull that has a 10% ballistics gelatin brain implanted with an accelerometer and then observing the brains movements following impact. Assuming the brain can stretch and compress to resist movements following impacts, the belief is that initial movement of the brain will not be the brain’s peak acceleration, rather it will be after rebounding off of the skull when the system rebounds that the brain will maximize acceleration.

MATERIALS AND METHODS

Stand Apparatus Height

8 collegiate pitchers' heights from the Hampden-Sydney College baseball team were taken. They were then filmed while being instructed to stand tall (as if being measured by the doctor) and then to go through their regular pitching motion (n=5). The video was then used to take screenshots at their full height and immediately following release of the baseball. A line was drawn on the image of each pitcher at their full height in ImageJ. The line was then designated as the height taken for each individual pitcher. Another line was drawn to the forehead of each pitcher and the height was then determined through ImageJ. The average of each pitchers' forehead heights were then taken and used as the height for the stand.

10% Ballistics Gel Brain

In a beaker, 1300 mL of water was mixed with 130 g of Gellita USA brand ballistics gelatin were stirred until all of the gel mix was mostly dissolved. The solution was incubated for around 4 hours at 37° C until the entire mixture was liquid (some stirring and breaking of the clumps was necessary). The mixture was then poured into a plastic model human skull lined with a plastic bag and placed in the fridge for 24-48 hours to set. The accelerometer was then inserted into the brain by slicing the brain horizontally and cutting out an area the size of the accelerometer to place inside. Some of the leftover gelatin solution was then brushed onto both the top and bottom halves of the brain and then replaced together and allowed to set in the fridge until hard.

Stand/Skull Apparatus

A spring, to allow the head to oscillate when impacted, was placed onto a long metal stand. The spring was then put into an oil funnel and then taped to place the forehead of the skull at 147.2cm (from stand height experiment). The model skull was then taped onto the funnel to hold in place.

mTBI Velocity Determination

The gelatin brain was placed into the skull and the whole system was placed onto the stand apparatus. Baseballs were fired to impact the forehead (n=3) of the skull starting at setting 2.5 (8.48m/s) on the pitching machine and going up to setting 6 (22.08m/s) by 0.5 increments (Table 2). The resulting brain/skull movements were recorded by the CEM DT-178A 3-axis vibration datalogger accelerometer with a threshold value of 1g to record any movements. The accelerometer was removed and the data downloaded into the Vibration Datalogger program. These data points were imported into Excel for analysis of the x and y-axes.

RESULTS

The recorded data for pitcher heights are in Table 1. The average forehead height immediately following a pitch was 147.2cm.

Forehead				**After Pitching Height (cm)**					
Pitcher	*Height (in.)*	*Height (cm)*	*Age (yr)*	*1*	*2*	*3*	*4*	*5*	*Average*
A1	72.1	183.134	19	151.721	163.083	133.675	138.354	138.354	145.0374
A2	71.7	182.118	22	138.709	134.489	145.565	143.982	127.633	138.0756
A3	72.35	183.769	20	150.234	135.478	155.599	145.54	143.527	146.0756
A4	73.27	186.1058	18	130.94	130.94	124.957	132.047	137.587	131.2942
A5	71.1	180.594	19	165.824	160.453	160.453	161.126	160.455	161.6622
A6	71.1	180.594	18	138.452	137.783	129.757	128.42	138.452	134.5728
A7	73	185.42	20	170.023	157.974	167.345	167.345	166.676	165.8726
A8	74	187.96	21	150.064	156.127	144.758	167.495	157.643	155.2174
								Average:	147.226

Setting	Speed 1 (m/s)	Speed 2	Speed 3	Speed 4	Speed 5	Avg.
2.5	8.94	8.49	8.49	8.05	8.49	8.49
3	10.28	10.28	9.83	9.83	9.83	10.01
3.5	12.52	12.07	12.96	12.96	12.07	12.52
4	13.86	13.41	13.86	13.41	13.41	13.59
4.5	14.75	14.75	13.86	13.86	12.52	13.95
5	14.31	14.75	15.20	15.20	14.75	14.84
5.5	18.33	18.33	16.99	18.33	18.78	18.15
6	20.12	22.80	22.35	23.25	21.90	22.08

Figures 1 and 2 shows the brains behavior following various impacts. The time-related index is how the Vibration Datalogger program recorded the data, in which typically 1 time point = 50ms. The interesting figure is figure 1 as it shows the brain behaving as hypothesized, in which the initial acceleration is not the maximum but rather the acceleration when the apparatus snapped back. It also, interestingly, shows quite a large amount of brain movement orthogonally, which would also result in even more shearing of the internal tissue as the brain is moving non-linearly. Figure 2 shows how the brain behaved following the majority of impacts, which the initial acceleration after the impact was the maximum and then dampened out over time.

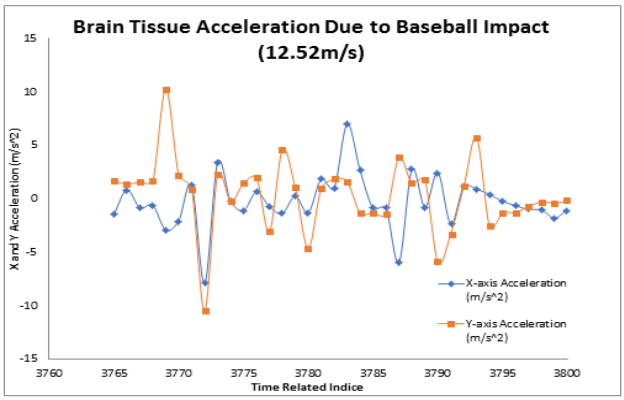

Figure 1: Example of Brain Movement Behaving as Hypothesized.

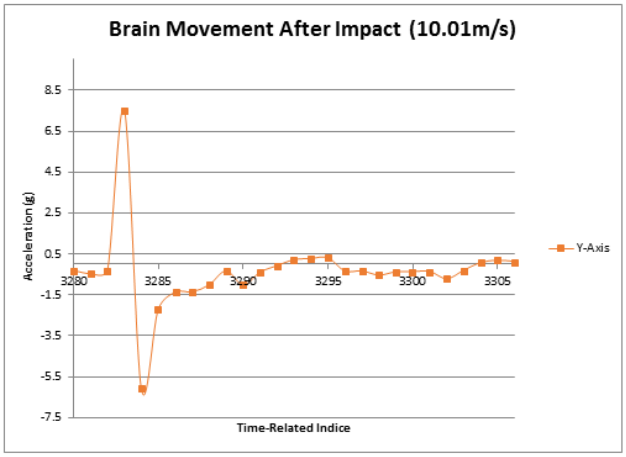

Figure 2: Example of Brain Movement Following Most Impacts as a Dampening Oscillator

DISCUSSION

The resulting graph in Figure 1 shows that what was hypothesized could be observed, but the fact that most impacts followed a similar behavior to that in Figure 2 makes a correlation difficult to prove. There were multiple sources for error that may have caused issues with both data collection and analysis that may fix this issue if corrected. The first was an issue with the accelerometer which, when inserted, the accelerometer read a constant 1g reading and continually took data as this was the threshold value. This resulted in many data points being taken and caused spreading out of data points from impacts when otherwise they may have been more easily distinguishable. Another issue was the pitching machine. It was difficult to precisely aim the baseballs which exited the machine directly at the forehead and, therefore, some of the impacts were not perfect. The exit velocities of the baseballs also greatly varied, visible in Table 2, which could have caused variations in the location of impact of the ball and also the movement of the brain. The cap of the skull also became loose towards the end of data collection and could have possibly altered brain movement.

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