Mechanism of Traumatic Brain Injury Resulting from Rear-End Car Crashes[1]

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Introduction and Purpose

It is well known and well documented that a person can suffer a traumatic brain injury with cognitive impairments in a rear-end car crash, regardless of the speed of impact or the severity of the damage to the cars. Unfortunately, all too often, the medical, legal, and insurance communities get so focused on things like speed of impact, damage to the cars, or whether there was an obvious blow to the head that no one seems to notice the suffering of the brain-injured person.

It has been almost 5,000 years since the Egyptians first started writing about head injury, and it has been over 2,300 years since Hippocrates said that no head injury should be ignored; but cars have only been around for 100 years, and people are still struggling to understand how the brain can be injured in a rear-end car crash.

The purpose of this paper is to examine and put together the research, literature, and experience to help better understand the movement and forces of the brain within the skull during a rear-end car crash to help raise the level of awareness about the possibility of a traumatic brain injury.

Of course, and most fortunately, not every person involved in a rear-end car crash suffers a traumatic brain injury, but this does not lessen the suffering or consequences endured by those who do. In those circumstances where a real person's life has been disrupted, altered, or ruined following a rear-end car crash, we must respect the possibility that these changes may be related to something that happened to the two-and-one-half pounds of differing density gel-like substance, encased within a bony skull, resting on a column of bone, muscle, and tissue, when it was unexpectedly hit from behind by a 2,000 or 3,000 pound car.

The following citations from the literature are offered to help the reader understand that these are not just theoretical considerations, but rather, issues that have been discussed in the literature for many years.

In their article, *Common Whiplash Injuries of the Neck*,[2] Drs James R. Gay and Kenneth H. Abbot conducted a study of 50 persons who suffered whiplash injury between 1948 and 1952. This study is particularly interesting because persons who suffered a direct blow to the head or neck were excluded.

> In 31 patients (62%) there was historical and symptomatic evidence of a cerebral concussion. These persons suffered a momentary lapse of consciousness (from seconds to one-half hour) ... some recovered consciousness after the car was driven ahead several hundred feet by the impact and sometimes the car was still in motion when they recovered consciousness.
>
> The mechanics of the concussion were interpreted as sudden mechanical deformity and pressure on the frontal and temporal lobes of the brain, which occurred when the forward movement of the brain was arrested against the anterior walls of the skull when the head and neck were whipped backward immediately after the initial acute flexion of the neck.
>
> It was possible that secondary and lesser concussions occurred to the frontal, temporal and occipital lobes of the brain in some instances when multiple oscillations of the head and neck occurred in alternating flexion and extension.
>
> Another phenomena in the production of a concussion in these cases, which may be more important than the mechanical factors already described, is acceleration or deceleration influence on brain tissue, which has been described in the experiments of Denny, Brown and Russell.

In their 1989 study for the Michigan Catastrophic Claims Fund, Drs Kenneth M. Adams and Stephen H. Putnam wrote[3]:

> Deceleration injuries occur when the head itself is moving rapidly, sometimes striking a stationary object (maybe the headrest) causing a rapid deceleration, which usually thrusts the brain forward in the cranium (parenthesis added). A common example of this would be whiplash injury where the brain rapidly decelerates, but without actually striking another object.
>
> Because the brain is suspended in cerebral spinal fluid and surrounded by several layers of tissue, it is normally protected from the bony prominences and dural ridges on the inner suffer of the skull. However, in virtually all types of closed head injury there are sharp linear or

> rotational gradients of force that may, in effect, throw the brain against these areas, resulting in contusions of the cortical surface.
>
> The areas most vulnerable to this type of injury are in the frontal and temporal poles, along with the orbital surfaces of the frontal lobes. This is due to the bony ridges and uneven surfaces present in the inner surface of the skull in these anterior areas.

They further wrote that[4]:

> In virtually all forms of sudden head trauma resulting from an automobile accident, variable numbers of cerebral neurons and axons are irreversibly damaged. The nature, direction and magnitude of the forces applied to the skull and brain at impact will largely determine the neurobehavioral consequences.

In the article, *Physiological Response to Angular Acceleration of the Head*, Dr Thomas Gennarelli wrote[5]:

> The causes of human head injury are not only extremely complex but vary considerably from patient to patient ...
>
> Although the mechanisms of head injury are complex, they can be reduced to two categories of input: contact phenomena and acceleration ... Acceleration results from head movement after an injury and results in pressure gradients within the skull and brain as well as sheer, tensile and compression strains.
>
> These strains are the primary injurious factors to the brain and their magnitude and thus their influence on the brain depend not only on the amount of acceleration, but also on its direction. Thus, the way in which the head moves is an important determinant of what structures in the head will suffer from acceleration.

We must study this issue, because we must be ever mindful about the possibility that a person who suffers continuing cognitive, emotional, and other problems following a rear-end car crash might be suffering from the after-effects of a traumatic brain injury.

Background

In a typical post-accident course of events, the person is taken to a hospital, may be given a skull X-ray or CT scan whose results are deemed normal, and then sent home without a diagnosis. The person might be told that he or she has a cervical strain, and the overwhelming medical problem at that point is often the headache.

As the days and weeks go by, little problems - maybe with memory, emotional changes, problems getting organized, sleep, or other behavioral areas - start to appear. A trip to the family doctor may yield a prescription for medication but no diagnosis or understanding of traumatic brain injury.

Some of the problems that may occur following a traumatic brain injury, are listed below; and it is important to remember that depending on degree, any which of these alone can be disabling:

1. Headaches;
2. Memory problems;
3. Attention and concentration problems;
4. Personality change;
5. Irritability, anger, and frustration;
6. Difficulty with organizing tasks, planning the day, preparing meals, planning a work day or planning and organising activities;
7. Fatigue or inability to get going;
8. Inability to fall asleep or remain asleep;
9. Balance and dizziness problems;
10. Difficulty with reading or watching television;
11. Speech and communication problems with inability to find the right words, inability to express thoughts, and misunderstanding of what others are saying;
12. Difficulty in noisy environments, like the grocery store, restaurants, kids watching television in the next room or work environment, which difficulty might not have been present before;
13. Difficulty in social settings, like parties, church, and such, following multiple conversations;
14. Depression, which can be related to the traumatic brain injury itself, or as a reaction to the changed abilities.

The following statements are widely known and well documented in the literature to the extent that even the most hardened defence medical experts will agree they are absolutely true:

1. A person need not strike his or her head to sustain a traumatic brain injury.
2. A person need not be knocked out or in a coma following a traumatic event to sustain a traumatic brain injury.
3. That a diagnosis of traumatic brain injury or

closed head injury is not made in the emergency room, or in the weeks or months following the trauma, does not in any way mean the injury was not there.

4. The word "mild," in describing a traumatic brain injury does not mean that the injury is not serious.

5. A person need not suffer cuts, bruises, or broken bones to suffer a traumatic brain injury.

6. The amount of damage to the cars is not always relevant in determining the presence or severity of a traumatic brain injury.

7. That a neurological examination may be normal does not rule out the presence of traumatic brain injury with cognitive impairments.

8. That a skull X-ray, CT scan, or brain MRI may be normal does not rule out the presence of a traumatic brain injury.

9. Whether it is called a concussion, post-concussion syndrome, post-traumatic complex, or others, it all boils down to the fact that something happened to the brain.

Defence lawyers are always fond of asking treating physicians if they think that everyone gets a traumatic brain injury from a rear-end car crash, and it brings to mind a tragedy that occurred in a running race several years ago. The 15 mile race had about 300 participants, and most who finished it were exhausted, but one poor fellow suffered a heart attack and died. That he was the only one to suffer a heart attack and die from the 300 participants did not make his death any less real. That not everyone suffers a traumatic brain injury in a rear-end car crash does not make the tragedy and its far-reaching consequences any less real for the injured party.

While examining the issue of traumatic brain injury, it is important to bear in mind the following words from Dr Bryan Jennette[6]:

> ... For a severe injury may be followed by a complete recovery, or a mild one may have serious consequences.

In discussing minor head injury in her article, *Cumulative and Persisting Effects of Concussion on Attention and Cognition*, Dr Dorothy Gronwall wrote[7]:

> After minor head injury, patients have difficulty in all areas that require them to analyse more items of information than they can handle simultaneously. They present as slow because it takes longer for smaller than normal chunks of information to be processed. They present as distractible because they do not have the spare capacity to monitor irrelevant stimuli at the same time as they are attending to the relevant stimulus. They present as forgetful because while they are concentrating on point A, they do not have the processing space to think about point B simultaneously. They present as inattentive because when the amount of information that they are given exceeds their capacities, they cannot take it all in.

She further wrote[8]:

> Even those patients who appear to have made a full functional recovery, who record normal scores on all neuropsychological tests, and who have returned to their pre-injury social and work life, may demonstrate persistent impairment when subjected to another stress.

The conclusion is that minor head injury produces long term damage perhaps in the nature of neuronal fall out, which may not be apparent in normal circumstances, but which is evident when the system is stressed.

In her article, *Subtle Sequelae of Brain Damage, Perplexity, Distractibility and Fatigue*, Dr Muriel Lezak wrote[9]:

> Perplexity, distractibility and fatigue are among the most common and troublesome problems consequent to brain damage in adults ... The occurrence of these problems with every kind of brain injury suggests that they are not associated with damage to specific areas of the brain, nor, short of mental functioning so devastated that the patient loses self awareness, do they appear to be related to the severity of the injury. Rather, they seem to result from disruption of accustomed pathways and loss of change of any mental function.

She further provided[10]:

> Subtle problems of perplexity, distractibility, and fatigue accompany all kinds of brain injury for they appear to result from disruption of accustomed neural pathways and loss or change of mental function.

In his article, *Neuropsychological Recovery in Head Injury*, Dr Ralph M. Reitan wrote[11]:

> Cognitive, intellectual and emotional problems also appeared to be more persistent and socially and vocationally disabling than physical or sensory and motor disabilities.

Dr Lawrence M. Binder, in his article entitled *Persisting Symptoms After Mild Head Injury: A Review of the Post-Concussion Syndrome*, wrote[12]:

> Most of the scientific controversy about minor head injury surrounds the post-concussion syndrome (PCS), a term reserved for patients who have persisting subjective symptomology resulting from cerebral concussion. The common symptoms include headache, dizziness, irritability, anxiety, blurred vision, insomnia, easy fatigability, and concentration and memory difficulty. These and other symptoms occur frequently after concussion.
>
> In one series of patients with concussion of varying lengths, only 49% of the patients were asymptomatic six weeks after injury. In a study of brief concussion only 16% had no subjective complaints three months after their injury. Of those who had been employed at the time of their injury, 34% were unemployed at follow up. *Follow up studies have shown that these symptoms often persist for months or for years.*

Now that we have an understanding of the type of problems that a person can experience following even what is called a 'mild' head injury resulting from a rear-end car crash, it is important to understand the movement of the brain within the skull during this sequence of events.

The Make up of the Brain and its Placement within the Skull

The composition of the human brain has been likened to two-and-one-half pounds of gel like substance that is of varying densities. The connections in the brain are extremely delicate, and in brain trauma-inducing situations, cerebral neurons, axons, nerve fibres, and blood vessels can be stretched and even torn, but such occurrences may not be detectable on CT scan, MRIS, or PET scan. Such negative readings do not mean that the brain is not injured but, rather, that current medical technology is not able to detect such microscopic shearing, tearing, and bleeding in the brain.

The inside of the skull has areas of bony ridges and dural prominences, and parts of the brain actually rest in the rough bony area. As the skull and brain are snapping back and forth during a rear-end car crash, they do not move in perfect synchronisation. The skull moves first, followed by the brain, and as the skull and brain are moving, the brain can actually slide back and forth over these bony ridges and dural prominences, which is one of the mechanisms of injury to the brain in a rear-end car crash.

While many people, sometimes defence experts, talk about expected patterns of deficits resulting from traumatic brain injury, whether rear-end car crash or otherwise, the following passage, written by Dr Muriel D. Lezak, is particularly informative and supportive of the proposition that all traumatic brain injuries, just like all human beings, are unique[13]:

> Not only is the pattern of deficit displayed by one brain damaged person likely to differ from the pattern displayed by another with damage involving anatomically and functionally different areas, but impairments patterns of patients with similar lesions may also differ.

The Movement of the Car, Human Body, Head, and Neck in a Rear-End Crash and how the Brain can be Injured

Breaking the sequence of events in a rear-end car crash into milliseconds, the sequence of movement during a rear-end car crash is as follows:

1. The car body moves forward.
2. The trunk of the occupant body moves forward.
3. The head snaps backwards, sometimes striking a headrest that is moving forward in the opposite direction, with the forward movement of the car.
4. The brain moves in the same direction of the skull, but slightly out of synchronisation.

As long ago as 1955, Severy, Mathewson, and Bechtol wrote[14]:

> It was found that a car struck in the rear by another car moving at 20 mph or less very narrowly requires its resultant peak acceleration before the motorist's head and upper torso have accelerated a significant amount. This pronounced lag in acceleration is attributed to inertia, seat back compressibility and flexure. The relatively high velocity differential developed is almost immediately followed by excessive acceleration of the head and upper torso.
>
> The seat back is generally strong enough to at least partially support the motorist's back so that the upper torso was found to accelerate at a rate only slightly greater than the car. The head has the only flexible and somewhat extensible neck through which the forces of acceleration of the torso may be transmitted.
>
> The delay in acceleration of the upper torso with respect to the acceleration of the car body is further amplified as these forces are transmitted to the head through the neck so that the head has an even shorter time to overcome the pronounced velocity differential which has developed. This can only be accomplished by a greater rate of acceleration for the head.

> The body posture and state of preparedness of the motorist at the time of impact was found to have an influence on the acceleration pattern of the body components and hence the injury potential of a given collision.
>
> The load to the head may exceed 100 pounds for collisions not exceeding 15 mph.

The following discussion by Dr Mason Hohl in *The Cervical Spine, Second Edition*, is particularly informative in understanding the movement of the head and brain in rear-end car crashes[15]:

> It has been established conclusively through documented experimental rear end collisions, using volunteers for low velocity impact and anthropomorphic dummies for higher velocities, that the unsupported head and neck rapidly hyperextend until the back of the head strikes the seat, well beyond the normal range of allowable movement.
>
> This violent motion occurs in the first quarter second after impact and is followed by a less rapid flexion of the neck as tissue elasticity and slowing of acceleration take effect. Thus, the actual injury to soft tissues occur so rapidly that normal protective muscle reflexes cannot respond.

Further on, Dr Hohl wrote:

> MacNab, Ommaya and Associates, and Wilkstrom and Associates created experimental hyperextension injuries to animals. They found a variety of anterior neck injuries, including muscle hemorrhages and tears, ruptures of the anterior longitudinal ligament, intervertebral disc disruption and esophageal muscle hemorrhages.
>
> Brain injury, long suspected was substantiated by findings of superficial brain hemorrhages and electroencephalographic abnormalities. Much earlier clinicians had noted frequent electroencephalographic changes in human victims of rear end collisions.

Dr Ian MacNab, of whom Dr Hohl wrote, has said this[16]:

> In rear end collisions, injury results from the relative acceleration of the head and the trunk of the occupant, and the degree of injury is independent on the rate of acceleration. Many factors influence the rate of acceleration and must be specifically sought for when assessing the severity of injury.
>
> Acceleration depends on the force applied and the inertia of the vehicle that has been struck. The force is dependent upon the weight and speed of the striking vehicle, so that a street car travelling at 3 mph can apply as much force and initiate the same degree of acceleration as a compact car travelling at 40 mph. The inertia of the car that has been struck will depend not only on its weight but also on factors that will allow it to roll evenly; for example, slippery road conditions, whether the brakes were on, and whether the car had automatic or standard transmission. A car that is moving slowly will accelerate more rapidly than one that is stationary.
>
> The amount of damage sustained by the car bears little relationship to the force applied. To take an extreme example: if the car was stuck in concrete, the damage sustained might be very great but the occupants would not be injured because the car could not move forward, whereas, on ice, the damage to the car could be slight but the injuries sustained might be severe because of the rapid acceleration permitted.

In an article, *Whiplash Injury and Brain Damage*, Drs Ayub K. Ommaya, Fred Faas, and Philip Yamell wrote[17]:

> Experimental whiplash injury in Rhesus monkeys has demonstrated that experimental cerebral concussion, as well as gross haemorrhages and contusions over the service of the brain and upper cervical cord, can be produced by rotational displacement of the head on the neck alone, without significant direct head impact. These experimental observations have been studied in the light of published reports of cerebral concussion and other evidence for central nervous system involvement after whiplash injury in man.

They further wrote[18]:

> It is a matter of crucial importance that we investigate and manage the clinical problems of whiplash injuries in our patients not only with regard to the musculoskeletal and peripheral nervous systems, but also with greater attention to the finer details of behavioural and neurological deficits and to the results of our special investigations.
>
> Furthermore, we should search for points of clinical-pathological resemblance between patients with head injury and others with whiplash injury. This recommendation is based on our experimental evidence which suggests that the whiplash type of injury mechanism may be of significant importance in producing the effects of closed head injuries under conditions when the head is free to move.

Conclusion

When working with someone who has cognitive, intellectual, or other complaints of a continuing nature following a rear-end car crash, be ever mindful to the possibility of a traumatic brain injury. Look at the person and then backward to the traumatic event. In a society where it is estimated that some two million persons a year suffer a traumatic brain injury, the more people know about how these injuries occur, the more effectively it can be prevented.

As people work together, whether doctor, lawyer, or insurance company representative, to get others on the right path of diagnosis, treatment, and therapy, it is hoped that understanding something of the mechanics of the movement of the brain during a rear-end car crash will assist in that endeavor.

It is hoped that this paper will assist lawyers, doctors, and insurance company representatives in gaining some insight into how a traumatic brain injury may occur in a rear-end car crash, regardless of the speed of impact or extent of damage to the cars. This will hopefully help raise the level of consciousness regarding the seriousness and significance of these types of injuries so that this population of injured people can receive proper medical and emotional treatment, as well as legal representation.

Footnotes:

1. Adapted from the author's chapter of the same title in "National Head Injury Foundation, Analysis, Understanding and Presentation of Cases Involving Traumatic Brain Injuries" (Charles N. Simkins ed.)
2. *JAMA* 1698 (Aug. 29, 1953)
3. At 11.
4. *Ibid.* at 13.
5. "Head Injury, Basic and Clinical Aspects" 129.
6. "Severity of Brain Damage, Altered Consciousness and Other Indicators" 215.
7. At 154.
8. *Ibid.* at 161.
9. 57 (1) *Am. J. Phys. Med.* (1978).
10. *Ibid.* at 14.
11. 40 *Archives of Neurology* 333 (June 1983)
12. 8 (4) *J. Clin. & Expt'l Neuropsychol.* 323-46 (1986).
13. *Neuropsychological Assessment* 17 (2d ed.)
14. "Controlled Automobile Rear End Collisions, an Investigation of Related Engineering and Medical Phenomenon", *Canadian Serv. Med. J.* 727 (1955).
15. "Soft Tissue Injuries", in *The Cervical Spine* 436 (Cervical Spine Res. Soc'y, ed., 2d ed.).
16. "Acceleration Extension Injuries of the Cervical Spine", in *The Spine* 648 (Roth & Simeone, eds.).
17. 204(4) *JAMA* 285 (Apr. 22, 1968)
18. *Ibid.* at 289.