

HEAD ACCELERATION AND ASSOCIATED PAIN FELT IN THE NECK REGION
DURING A SIMULATED AUTOMOBILE LOW VELOCITY REAR-END COLLISION AS A
FUNCTION OF SEAT BACK ANGLE

By

MARTIN CHARLES HILLIER

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To all those who supported me throughout my ventures, including my wife, family, advisors, and financial supporters.

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LIST OF ABBREVIATIONS

AIS	Abbreviated injury scale
cm	Centimeter
$\Delta v / \Delta t$	Change in velocity divided by change in time
°	Degrees
ft	Feet
Hz	Hertz
in	Inch
kg	Kilogram
kph	Kilometers per hour
MRI	Magnetic resonance imaging
m	Meter
m/s^2	Meters per second squared
mph	Miles per hour
mm	Millimeter
ms	Milliseconds
lbs	Pounds
SCM	Sternocleidomastoid muscle
V	Volts
WAD	Whiplash associated disorder

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Martin Charles Hillier

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The angle of the seat back is an important factor in head acceleration and pain felt during a low velocity rear-end collision. Ten male volunteers with a mean age of 22.40 ± 2.37 years, mean height of 1.79 ± 0.035 m, and mean mass of 81.92 ± 11.51 kg were exposed to impacts designed to replicate this type of collision in a lab setting. The change in velocity for each impact was approximately 8 kph (5 mph). The variable during the study was the angle of the seat back. Three seat back angles consisting of 100° , 115° , and 130° from horizontal were tested. In general, as the angle of the seat back increased, the peak horizontal, vertical, and resultant accelerations decreased. More specifically, significant differences were detected for the horizontal accelerations with increased values for 100° when compared to 115° and 130° . There was no significance difference observed when comparing the vertical accelerations across the three seat angles. The resultant acceleration showed significant differences when comparing 130° to 100° and 115° , with the lower acceleration occurring in the 130° seat position in both cases. Conversely, pain increased significantly as the angle increased. The 130° seat position had significantly higher pain ratings when compared to the 100° and 115° seat positions.

Therefore, it can be inferred that there is an inverse relationship between seat back angle and acceleration while there is a direct relationship between seat back angle and pain. Based on further analysis of the data collected during this study, it may be possible that the increase in pain is due to the larger contribution of the muscles and the decreased contribution of the headrest to stop the head during the trials at 130°. To minimize risk, it is recommended to maintain a 100° seat back angle when in the front row of an automobile.

CHAPTER 1 INTRODUCTION

Low velocity rear-end collisions are considered to be minor in severity, however they are very common and they represent a financial burden to society.¹ The injuries caused by these collisions can be classified under one common disorder known as whiplash associated disorder (WAD). These injuries include vertebral and spinal injuries and muscle strains.² Although the injuries related to rear-end collisions are known, the causes are still under investigation. “Whiplash” is characterized by a sudden and forceful extension of the neck followed by a less severe flexion of the neck. The motion associated with whiplash injuries has been broken into five separate phases. Included are the initial response, principal forward acceleration, torso recovery, head deceleration, and restitution phases.³ Phases that involve extension of the neck typically cause injuries to the anterior musculature of the neck, intervertebral discs, posterior vertebral structures, and the anterior longitudinal ligament. Extension occurs during the first two phases. Flexion of the neck causes less severe injuries including posterior muscle strains and herniated discs and occurs during the third and fourth phases of the whiplash motion.²

Previous research in this area has focused on factors such as change in velocity, headrest position, occupant awareness, and gender. However, research in the area of seat orientation has been limited. Based on the geometry of an automobile seat and the natural seated position of the occupant, changing the angle of the seat back could create a change in body position and occupant kinematics, specifically the acceleration that the head undergoes. It is also possible that a change in acceleration could create a variation in the risk of injury during a low velocity rear-end collision. The purpose of the current study is to determine whether the angle of the seat back will contribute to the peak acceleration experienced by the head during a low velocity rear-end collision. In addition, pain felt by the participant after the collision will be assessed and

compared across seat angles. The findings of this study could provide valuable information to automotive safety engineers and could assist in the design of a safer automobile seat.

CHAPTER 2 REVIEW OF LITERATURE

Whiplash

Whiplash Stages

Generally, whiplash injuries occur much more frequently in rear-end collisions than in any other crash configuration,⁴ with rear-end collisions accounting for 85% of all crashes reporting whiplash injuries.⁵ More specifically, during a low speed, rear-end collision there are five phases of the motion associated with whiplash injuries. These five phases begin shortly after the collision between the two automobiles. During the first 100 ms of the collision there is no movement of the body (Figure 2-1A). Therefore, this initial period is not included in the sequence of events that causes the injuries associated with whiplash.²

The first phase involving movement of the body is called the initial response. This phase can last anywhere from 50 to 100 ms.^{3,6,7} During this phase the seat of the vehicle moves forward relative to the body due to the rear impact (Figure 2-1B). This applies pressure to the pelvis and the lower back and also causes the seat back to begin deflecting backwards due to the forces the body applies to the chair.³ This deflection can be simply explained by Newton's third law of motion. As the inertial forces of the body accelerate the seat backwards, the seat back applies an equal force that acts to decelerate the body. The head does not undergo any motion during this phase nor do the muscles of the neck exhibit any response. However, towards the end of the first phase, the thoracic vertebrae begin to move forward and slightly upward.⁶ Finally, the body undergoes the beginning stages of ramping up. Ramping up is defined as the body sliding up the seat due to the forces encountered during impact. The degree of severity of this phenomenon changes with the severity of the crash and the inclination of the seat back.⁷ During low speed collisions (Five mph or less) this motion is typically not severe enough to

cause the occupant's head to rise above the headrest, assuming that the headrest is present and raised to the appropriate height for the occupant.³

The second phase of the motion is called the principal forward acceleration.³ This phase lasts approximately 100 ms.^{3,6,7} At the beginning of this phase, the seat back has reached maximum deflection^{3,6} and the head and neck undergo an extension motion.^{3,6,7} The head moves backward relative to the torso due to its inertia while accelerating and rotating in the rearward direction.⁷ This rotation can be stopped by the head restraint or by the anatomical structures of the spine and the neck, if the head restraint is ineffective. In addition, the body continues the ramping up motion and there is an arching of the torso due to the extension of the spine. The spine reaches maximum extension during this phase (Figure 2-1C).⁶ Compression injuries to the posterior structures of the cervical spine, if present, are a result of this phase.² The next significant occurrence in this phase is the activation of the anterior musculature of the neck. This musculature, specifically the SCM, is actively resisting the abrupt backward motion of the head.^{6,7} Due to the time it takes for the nerve impulse to reach the muscle, this peak muscle activation occurs approximately 70-100 ms after the rearward motion of the head begins. It is also worth noting that the muscles may not reach peak activity before this phase ends. If the muscles never reach peak activity, the risk for injury to the muscles goes down.⁷ Factors including crash severity and horizontal distance between the headrest and the back of the head can significantly alter the amount of time and the risk of injury during this phase. The angle of the seat back is directly related to the horizontal distance between the back of the head and the headrest. More specifically, as the angle of inclination increases so does the horizontal distance. Therefore, as the angle of the seat increases, the time before contact with the headrest also increases.

The third phase of the whiplash motion, or the torso recovery and forward head acceleration phase also lasts approximately 100 ms.^{3,6,7} During this phase, the head and neck undergo flexion and the torso and the seat back begin to return to their pre-impact positions. The torso achieves a velocity that exceeds the velocity of the vehicle, thus creating the forward motion of the torso (Figure 2-1D).^{3,6} However, the velocity of the head is significantly higher than the velocity of the vehicle and the acceleration is usually two to three times that of the vehicle.^{3,7-12} This relative increase of velocity and acceleration creates a flexion motion of the neck. During this phase, the muscle activity of the neck continues, however the posterior neck muscles are placed under more stress due to their resistance of the forward flexion motion. The most involved muscle is the trapezius.^{3,6,7}

The fourth phase also lasts approximately 100 ms,^{3,6,7} and is called the head deceleration phase.³ In this phase, the head finishes the forward flexion motion while the musculature decelerates the head, and the head begins to return to the pre-impact position.^{3,6} During this phase, the upper cervical vertebrae are in extension while the lower cervical vertebrae are in flexion (Figure 2-1E).⁶ If the impact is severe enough, the head will be stopped by the chest through contact with the anterior portion of the mandible, or chin. Otherwise, the posterior musculature of the neck will be primarily responsible for the end of the forward movement. If injuries are present in the posterior musculature of the neck, it is due to phases three and four. Finally, the torso continues its return to its pre-impact position.^{3,6}

The fifth and final phase, or the restitution phase,³ consists of the body and head finishing their progression to the pre-impact position and is characterized by the ceasing of muscle activity in the neck (Figure 2-1F). This phase is generally the longest phase lasting 150 to 200 ms.^{3,6,13}

This five phase motion is representative of all whiplash injuries, however the severity of these injuries can change due to the characteristics and circumstances related to the individual crash. Some of these factors include change in velocity and occupant orientation. The most significant factor concerning occupant orientation is the angle of the head relative to the torso.⁷ In the current study, this angle will change based on the angle of the seatback. As the angle of the seatback increases, the angle of flexion between the neck and the head must increase in order to maintain a head position that is perpendicular to the surface of the road.

Injury Mechanism

The motion previously described is capable of causing a variety of injuries. However, separate injuries occur during the different phases of motion of the head and neck. During extension, compressive forces are applied to the posterior structures and tensile forces are applied to the anterior structures. The most common anterior structures involved during extension are the esophagus, anterior longitudinal ligament, anterior cervical muscles, the odontoid process of the second cervical vertebrae, and the intervertebral discs. The posterior structures at risk during extension are the facet joints and the spinous processes of the vertebrae.² During normal extension, the structures of the vertebrae and the muscles of the anterior neck are within their physiological and anatomical limits. In this case, no injury will occur. However, during low velocity rear-end collisions, these structures may be subjected to forces which exceed healthy limits. In this scenario, the facet joints between the superior articular process of the lower vertebra and the inferior articular process of the higher vertebra are the first anatomical structures to be stressed, thus they become the point about which the vertebrae rotate. If the forces of rotation become too great for the facet joints to withstand, one of three things is possible. First, the facet joints could experience a crush fracture. Second, the spinous processes of the vertebrae could come in contact due to the forced extension and fracture as a result.

Finally, the anterior structures of the vertebrae and neck could stretch beyond their elastic limit or the vertebral body could fracture. These anterior structures include muscles, the anterior longitudinal ligament of the cervical vertebrae, and the intervertebral discs.²

During the flexion phase the opposite occurs. Compressive forces are now applied to the anterior anatomy and tensile forces are applied to the posterior anatomy of the cervical spine. The main structures involved in this phase are the intervertebral discs and vertebral bodies in the anterior portion and the facet joint capsules along with the posterior neck muscles in the posterior portion. The most common injuries during the flexion phase are ruptured discs, vertebral body fractures, and muscle and ligament strains.²

The final and least likely source of injury during low velocity rear-end collisions are shear forces resulting from the horizontal motion of the body during the crash. Assuming a near-vertical position of the spine before the collision, horizontal forces will be transmitted parallel to the intervertebral discs. These forces can cause injury to the facet joint capsules and the anterior portion of the intervertebral discs. Based on the anatomy of the cervical vertebrae, the facet joint capsules experience compression from shear forces and the anterior portion of the intervertebral discs experiences a tensile force due to the backward motion of the superior vertebra.²

Specific Injuries Caused by Whiplash and Associated Symptoms

As previously mentioned, the body parts most affected by whiplash injuries are the musculature of the neck and cervical vertebrae and surrounding structures. Injuries involving these structures can either be short lived or chronic, and are generally classified as WAD.¹⁴ However, because these injuries are non-fatal, no formal pathological studies can be done to determine exact causes or locations of injury. As medical technology becomes more advanced, accuracy of diagnosis is becoming better, but these injuries will probably never be as well understood as more serious, fatal injuries. To date, many types of studies have been performed

to better understand these injuries; however each method has its own limitations. These methods include cadaver experiments, animal experiments, and radiographic studies. Cadaver experiments utilize the same anatomy, but do not demonstrate the same muscular response as a live human being. Animal experiments provide a live subject, but differences in anatomy and body size provide limiting factors. Finally, radiographic studies show injuries in live human beings, but they do not allow for first-hand examination of the affected structures. Even if the patient requires surgery for the injury sustained, it is generally much later and the anatomy during surgery may be different than it was directly after the crash.²

Generally, injuries to the musculature of the neck are short lived, while injuries to intervertebral discs, facet joints, and cervical ligaments usually cause chronic neck pain. Injuries to the facet joints, including fractures and tears of the joint capsules, are fairly common and can cause chronic pain. Many times, this pain is due to a missed diagnosis resulting from an inability or difficulty to see the injury on a typical X-ray or MRI. Disc injuries are also common, but are easier to diagnose, as they are generally more common in the medical community and are easier to detect from an MRI. The two most common varieties of disc injuries are a separation of the disc from the vertebra and an anterior rupture due to compression of the posterior spine during extension.² Muscle injuries, specifically tears and strains, are the most common injuries suffered by individuals with whiplash. This is due to the sequence of events of a whiplash injury. The muscles act as a protective mechanism to the other structures that could potentially be involved. They are the first structure that resists the motion of the head, thus they undergo the greatest stress. Muscles will undergo mild injury in order to keep the spine aligned and free of injury. It is only when the muscles can no longer withstand the acceleration and resultant forces of the head that the other structures become susceptible to injury.⁶ Muscle injuries, while being the

most common, are also the shortest lasting, with a maximum healing time of two to three weeks, while many times resolving in just a few days.² The final major structures contributing to whiplash injuries are the ligaments, specifically the anterior longitudinal ligament. Anatomical studies have shown that the anterior longitudinal ligament and the annulus fibrosis of the anterior intervertebral discs in the cervical vertebrae merge together, thus anterior longitudinal ligament injuries and disc injuries are often associated together. Injuries to this ligament occur only during the extension phase of the whiplash motion, and are characterized by a partial tear or complete rupture of the ligament.² In summary, although there are other sources of injury during whiplash, the vast majority of whiplash injuries are directly related to the facet joints, intervertebral discs, and anterior ligaments of the cervical vertebrae.

With these very specific injuries come various symptoms. Symptoms of WAD include pain in the neck or shoulder region, dizziness, headaches, blurred vision, concentration and memory disturbances, and paraesthesia, or tingling and numbness in the hands.^{2,14-16} Neck pain is the most reported complaint among individuals who report whiplash injuries. This pain is reported in the anterior and posterior portion of the neck, although posterior pain is the dominant complaint. This pain can come in several forms. The two most common forms are dull and aching and sharp pain. Both forms are exacerbated with movement and many times movement is restricted due to stiffness.² Previous studies using similar speeds to the current study have shown that neck pain, if any is present, has resolved within four days.^{6,7,12,13,17-19} Due to the complexity and multitude of possible causes of injury, the exact correlation between types of neck pain and type of crash is not completely understood. The second most frequently reported symptom reported is headaches. The most likely causes of these headaches are the upper three cervical nerves originating from the first, second, and third cervical vertebrae. Afferents from these three

nerves terminate in the cervical portion of the trigeminal nucleus, located in the caudal region. Because the ophthalmic portion of the trigeminal nucleus is also in the caudal region, pain from C1-C3 is usually referred to the orbital and temporal regions. This referred pain is the most likely cause for headaches related to the whiplash motion.² Previous studies at or near the speed used in the current study show that headaches, if present, resolved within two days.^{4,19} Other headaches that could originate would result from concussion or intracranial hemorrhage, however during a low velocity collision these types of injuries are extremely unlikely. Paraesthesia, another symptom reported by whiplash patients, is caused by compression of the nerves of the brachial plexus by the surrounding anatomical structures including muscles. The other symptoms relating to whiplash, including dizziness, blurred vision, and concentration and memory disturbances, have not been sufficiently explained in the current research.²

Prevalence of Whiplash Injuries in Society

Whiplash injuries, although minor in severity, are a major health problem around the world and they create a huge economic burden to insurance companies, hospitals, and the patients affected by these injuries. In general, whiplash injuries are most likely to occur when the vehicle is involved in a rear-end impact with speeds less than 12.5 mph.^{4,16,20} The injuries are usually classified as minor on the abbreviated injury scale, otherwise known as AIS 1 injuries. However, the frequency of permanent disability is about 10%. This is extremely high when compared to other AIS 1 classified injuries, which have a permanent disability rate of only 0.1%.¹ Thus, injuries related to whiplash are 100 times more likely to produce permanent disability than other injuries of similar severity.

In addition to having a relatively high rate of disability, these injuries are also very common, as neck strains and sprains associated with whiplash are the most serious injuries reported by 40 % of automobile accident claimants in the United States.¹¹ This percentage

coincides well with statistics from two studies in Japan during the years 1996 and 1997. Watanabe et al. state that 44.0 % of 547,654 injuries suffered during a car accident in 1996 were neck injuries resulting from a rear-end impact and 44.7% of 563,121 injuries from 1997 were neck injuries resulting from a rear-end impact.²¹ In Western countries, about 25% of all people reporting an injury related to whiplash develop a chronic condition and 20% of those people suffer serious pain.⁵ Finally, in 2002 Croft et al. estimated that the cost associated with whiplash injuries exceeds \$19 billion every year, which is sure to be much higher currently.²² All of these statistics show that there is a great need to better comprehend the causes of these injuries and to reduce the risk of whiplash injuries throughout the world.

Automobile Characteristics Contributing to Injury

Seat Angle

The angle of the seatback is an important controllable factor related to the motion of the neck and the forces felt by the muscles of the posterior and anterior portions of the neck; however it has received little attention when discussing whiplash research. Ergonomics of products such as office chairs have been studied extensively, and there have been several studies on the ergonomics of automobile seats, yet an optimal position to reduce the risk of whiplash injury for a driver in an automobile has been more or less ignored, thus there is extremely limited research in this area.¹⁷ The previous studies in automobile seat ergonomics have shown a minimal myoelectric activity of the anterior and posterior muscles of the neck at a seat back inclination of 120°. ^{17,23} However, these studies had no relation to whiplash injuries.

The inclination of the seat back in an automobile affects the loads applied to the ligaments and the intervertebral discs of the cervical spine.⁷ When the seatback angle is altered, the angle between the neck and the torso must change, assuming that the driver's head is always perpendicular with the surface of the road. The change in the angle between the neck and the

torso may be an important factor contributing to the differences in force felt by the anatomical structures of the neck. When the seat is reclined past 90°, the neck is forced into flexion, subsequently creating a larger angle through which the head must move, and possibly creating a larger end velocity of the third phase of the whiplash motion. Therefore, when the muscles activate to decelerate the head during this motion, it is also possible that the inertial forces exerted by the muscles of the neck are greater from a flexed position when compared to an upright position. If this increase in force exceeds the injury threshold, the increase in seat back angle would create injury where there would be no injury from a more upright-seated position. In addition, if the seat is more upright, the distance between the back of the head and the headrest reduces. Therefore, the time between backward cervical rotation and contact between the head and headrest will be reduced. This topic will be discussed further in a later section.

The final factor that is affected by the position of the seatback is an aforementioned motion referred to as ramping up. This motion is characterized by an upward movement of the body along the seatback during a rear-end collision. If the motion is significant enough, the head will move above the headrest, thus eliminating the effectiveness of the headrest and increasing the risk of injury due to hyperextension. However, having a more upright seat position decreases this motion and should also decrease the risk of pain or injury. The variable manipulated in the present study was the angle of the seat back and how it contributed to the acceleration of the head during the whiplash motion and the pain felt in the musculature of the neck.

Headrest Function and Position

The headrest of a car seat has been proven to be effective in the reduction of injuries during a low velocity rear-end collision when used in the correct manner.^{1,24-26} The primary function of the head restraint is to limit the amount of rearward motion, or extension, that the neck undergoes during a rear-end collision. If used properly, the head restraint is designed to

terminate the head motion before hyperextension of the neck occurs. During hyperextension the more serious injuries such as intervertebral disc rupture, anterior longitudinal ligament rupture, and vertebral fractures occur. Although the headrest should eliminate these types of injuries, it is still very possible to have less severe soft tissue injuries such as muscle strains, even if the headrest is used properly. Hyperextension is one of the leading causes of injury to the neck, thus if the head restraint is not used properly the likelihood of injury increases.^{27,28}

Head restraint height is the only adjustable variable in the majority of modern head restraints. It has been reported that a minimum of 27.5 inches from the seat to the top of the headrest should be present to reduce injury, and the top of the headrest should be at least as high as the vertical midpoint of the head.^{24,26} In addition, Nygren et al. have shown that complaints of whiplash injuries nearly doubled when occupants reported a low headrest position instead of a high headrest position.²⁹ Additionally, 83% of drivers and passengers do not adjust their headrest to the proper height.⁹ In order to help prevent injuries during this study, the headrest will be placed in the highest position allowed by the structure of the car seat.

The final factor related to the headrest that contributes to the forces felt by the head and neck is the horizontal headrest distance, defined as the distance from the occipital protuberance of the head to the front of the headrest. While the headrest height stays constant, the change in seat back angle will also create a change in horizontal headrest distance, which in turn will cause a larger degree of neck flexion and a greater angular displacement of the head during the whiplash motion. Ideally, the gap between the occipital protuberance and the headrest should be no greater than half of the anteroposterior diameter of the head.²⁶ Many studies have shown that the greater this distance is, the greater the demands are on the muscles of the neck.^{1,14,19,22,24,26,27} However, none of these studies have related this phenomenon to the angle of the seatback.

Seat Belt

Previous studies have shown that an anthropomorphic dummy was adequately retained by the seat in rear-end impacts up to 14.3 mph.^{30,31} In order for a seat belt to be required and effective, the forward excursion of the torso must be large enough to lock the seatbelt. In the current study, the movement of the torso would not have caused this to happen. Therefore, it was determined that a seat belt would have little to no effect on the kinematics of the head or the torso during the collision.

Crash Factors Affecting Injury

Comparison of Body Kinematics during Rear-End Collisions and Frontal Impacts

The motion of the body during rear end and frontal impacts has similarities, however the motion during rear end impacts is responsible for the vast majority of recorded whiplash injuries.^{16,20,21} Frontal impacts are defined as having a direction of force between eleven o'clock and one o'clock with direct contact to the front of the car. During frontal impacts, the accepted motion of whiplash is not present, thus the injuries are generally less severe and less frequent. The first motion of the neck during a frontal impact is flexion, which is followed by a less severe extension motion. Furthermore, at a velocity of five mph, the likelihood of a hyperextension injury becomes very small. This is due to the dissipation of force from the posterior musculature of the neck and possible contact between the sternum and chin. As the first motion is the most severe in terms of acceleration, extension injuries during frontal impacts occur with much less regularity and generally occur in higher velocity crashes.³² This is opposite to the motion that takes place during a rear end collision. In this type of collision, extension of the neck is the first motion and it is followed by flexion.^{3,6,7} Because the first motion is more severe than the second, the anatomical structures involved with hyperextension injuries become much more susceptible, and because hyperextension injuries such as ligament ruptures, herniated discs, and vertebral

fractures are more serious than flexion injuries, it is fair to conclude that low velocity rear end collisions create a much higher probability for whiplash injuries than low velocity frontal impacts.

Relationship Between Selected Velocity and Injury Tolerance

Most WAD injuries occur when there is a change in velocity between 6.2 mph and 12.5 mph.²⁷ In previous studies testing human subjects at a velocity of five mph, no subject experienced WAD symptoms lasting more than three days, and there was no recurrence of pain at any point in the future. Primary complaints consisted of muscle pain, headache, and other cervical symptoms.^{4,12,13,16} In addition, the Insurance Institute for Highway Safety, the primary government crash test agency, uses a velocity of five mph to test vehicles for resistance to low speed crash damage. Therefore, in order to protect the participants as much as possible, five mph was chosen as the test velocity. The second reason for selecting five mph as the velocity was to ensure that the defined motion of whiplash occurred. If the change in velocity of the vehicle were too low, the forces required for the head to undergo the five phases of motion previously described would not be sufficient. Previously, West et al. tested at three mph or less and reported that the rearward displacement of the head following impact was not large enough to classify the motion as a typical whiplash movement.¹²

Influence of Occupant Awareness during a Crash

Occupant awareness, or the anticipation of an impending impact, not only changes the motion of the head and neck during a rear-end collision, but it can also change the likelihood of a whiplash injury. The main factors that create awareness during a rear-end collision are the screeching of the tires from the vehicle behind and seeing the car behind in the mirrors of the vehicle. The most common reaction of drivers when they anticipate a collision is to contract the muscles in the neck. Participants in studies who were instructed to anticipate the impending

crash and tense their neck muscles prior to impact have reported less severe pain and injuries and state that the crash seemed less severe when comparing it to a crash with the same change in velocity and an unaware state.^{15,33,38} Contraction of the muscles prior to impact creates an ability to resist extension beyond normal physiological and anatomical limits and consequently reduces the incidence of hyperextension injuries and symptoms.^{7,10,15,33-38} However, muscle tension before impact could also contribute to muscle strain injuries if the muscles involved eccentrically contract to resist head motion that has already begun.^{6,39-41} The kinematics of the whiplash motion are also changed when the occupant is aware. Kumar et al. state that the initial acceleration of the head in aware conditions was delayed by 25-40 ms.³³ This delay is due to the resistance of the backward rotation created by the anterior musculature of the neck. This will create a decrease in the extension of the neck by approximately 30-40% when compared to a relaxed state.⁷

Conversely, if the subject is in a relaxed state prior to impact, the muscles of the neck do not affect the head-neck-torso kinematics during a rear-end impact and the motion is as described previously.⁷ Because of the muscle activity onset delay, much or all of the movement in the extension phase of the whiplash motion is already completed before the anterior muscles, specifically the SCM, are maximally activated.

Demographics of Selected Participants

Human Subjects Compared to Anthropomorphic Dummies and Cadavers

In many studies involving automobile crashes, anthropomorphic dummies, or crash test dummies, are used for safety reasons. However, these tests are generally at much higher speeds than five mph. In addition, anthropomorphic dummies are designed to replicate the motions observed in a human during high-speed crashes and they do not exhibit the correct response when subjected to a low velocity rear-end collision.^{4,11,16,18} More specifically, the complicated

intersegmental dynamics of the neck, including vertebral interaction and muscle responses, cannot be reproduced in anthropomorphic dummies.¹¹ The final reason these dummies cannot be used for this type of research is the lack of a pain response. When studying whiplash, analysis of pain is an extremely vital part in analyzing what has happened to the anatomical structures affected.⁴ Because the dummies are unable to give verbal feedback about pain, injuries associated with whiplash are very difficult to study when using dummies as subjects.

Another substitute for in vivo human testing has been the use of human cadavers. This method also has very serious drawbacks when applied to low velocity impacts. As opposed to high-speed research where cause of death is usually the desired information, low speed impacts present much more subtle and complicated questions such as the role of muscle activity in the neck in preventing injury.^{4,11} Obviously, a cadaver has no muscle response and therefore cannot produce accurate results.

Likelihood of Injury in Males and Females

Although whiplash injuries affect both males and females, it has been widely reported that women are more susceptible to whiplash injuries, and the associated WAD symptoms last for a longer period of time when compared to men.^{7,11,14,24,26,42-55} In fact, women have a 40% higher risk than men of sustaining these injuries.²⁶ This increased risk can be attributed to several factors. The first factor is a greater ratio of head mass to neck circumference. This ratio, represented by the cube of the head circumference divided by the square of the neck circumference, places a greater demand on the muscles of the neck for female occupants.⁴ The increased muscular demand causes the incidence of whiplash injuries to increase. The next factor contributing to the higher incidence of injury in females is the difference in the alignment of the cervical spine between male and female occupants. The female spine is more prone to kyphosis, or a curvature of the upper spine.⁷ Because the spine is not aligned in a straight

position, the muscles of the neck are already under more strain than they would be in men.

When placed in a situation such as a rear-end collision, the muscles could be fatigued and less able to perform the demands necessary to prevent hyperextension. The final factor contributing to the higher risk in females is muscle strength. On average, females have weaker muscles in the neck region than men.²⁵ When combining this with the higher relative head mass, injuries are much more likely to happen. Because the muscles cannot resist the rearward rotation as well as men, the horizontal acceleration of the head is also higher in women.^{11,55}

Effect of Age on Injury Susceptibility

The effect of the whiplash motion on the body changes as an individual ages. Not only are elderly people less physically prepared to withstand the forces created by the whiplash motion, but the injuries suffered are generally more severe, last longer, and have increased symptoms.^{2,14,51} As people age, more effort is required to maintain muscle mass. If muscle mass is not maintained, the likelihood of severe injury increases because the muscles can no longer protect the ligaments and vertebrae. In addition, weakening of the bones, or osteoporosis, can occur as one ages. If the cervical vertebrae are weakened, when they are placed under the stresses caused by whiplash, they are more likely to fracture.

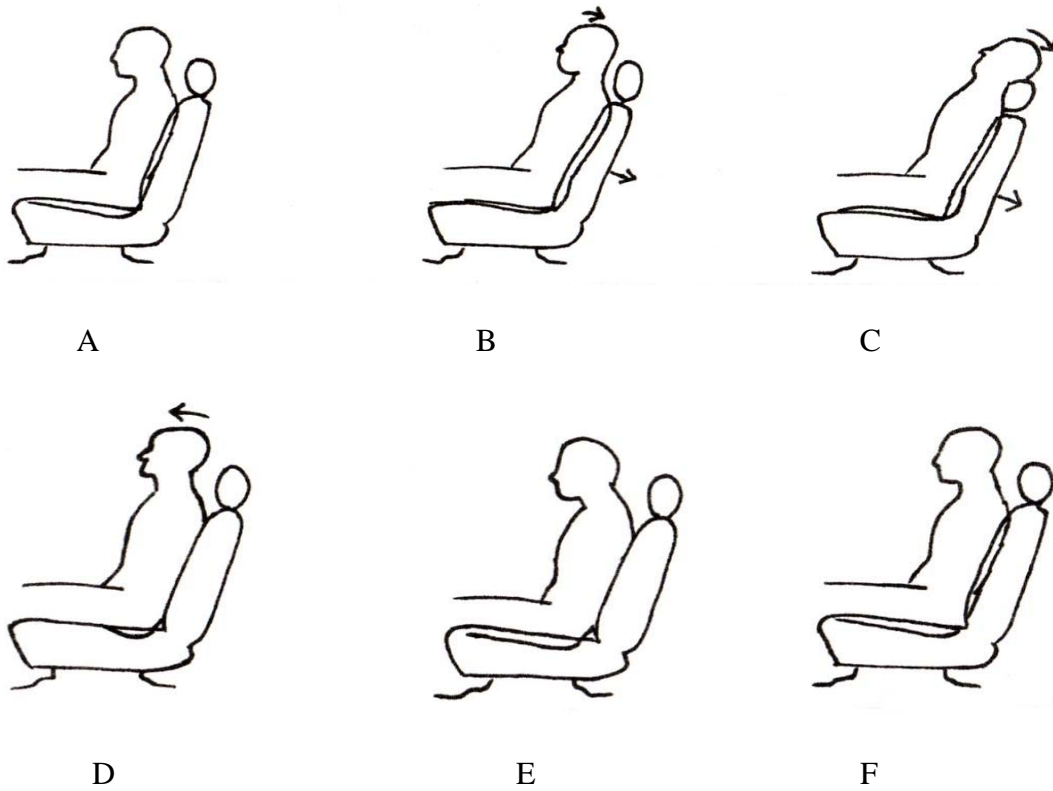


Figure 2-1. Stages of whiplash motion. Arrows indicate the direction of motion of the head and seat back. A) Pre-impact position. B) End of phase one. C) End of phase two. D) End of phase three. E) End of phase four. F) End of phase five.

CHAPTER 3 METHODS

Participants

Ten participants were tested. All participants were males between the ages of 18 and 35. Participants were recruited from the student population of the University of Florida. Each participant was required to read and sign an informed consent agreement approved by the Institutional Review Board of the University of Florida prior to participation. In addition, each participant was required to fill out a medical health questionnaire (Appendix A). Each participant was involved in three testing sessions, spaced at least two weeks apart in order to rule out any potential discomfort from the previous session. During each testing session, each participant was subjected to three trials that involved one rear-end impact per trial at a velocity of five mph. The only difference between testing sessions was the angle of the seat back. Exclusion criteria included female gender, previous spinal injury, history of severe headaches or migraines, current injuries to the neck or back, previous whiplash injuries, previous diagnosis of a herniated disc or any other disc injury, and history of dizziness.

Experimental Apparatus

Ramp

In order to simulate a low velocity rear end collision each participant sat on a car seat mounted to a rolling platform (Figure 3-1) that was released and allowed to roll backwards down a wooden ramp until colliding with a fixed wooden barrier (Figure 3-2). The ramp was inclined at 10° from horizontal and was 4.6 m (15 ft) long. The height of the ramp was 0.8 m (2.6 ft). In addition, there was a flat 2.44 m (8 ft) long segment attached to the bottom of the ramp that served as a final runway for the seat (Figure 3-2). The ramp was constructed from wooden beams (2 in x 4 in) to create the framework and 1.9 cm (3/4 in) plywood to create the upper

rolling surface. On the surface of the ramp, there were 1.9 cm (3/4 in) tall rails that served to keep the platform for the seat securely in place while it traveled down the ramp. In addition, there were 7.6 cm (3 in) tall guards attached to each rail in order to provide an extra level of safety (Figure 3-3). The ramp was comprised of two segments. The final 2.44 m (8 ft) section of the ramp was held in place with four steel cables attached to the floor in order to prevent rearward movement of the ramp during impact. At the end of the flat segment was an immovable stopping apparatus, consisting of a wooden barrier (Figure 3-2).

Seat Platform

The car seat, extracted from a 2001 Toyota Corolla, was securely bolted to a platform that measured 0.9144 m (3 ft) wide by 0.9144 m (3 ft) long by 0.305 m (1 ft) deep. There was also an extension on the platform so the participants could rest their feet (Figure 3-1). The extension was the same height relative to the seat as the floor of an automobile. The platform utilized two rows of four Rollerblade 76 mm wheels with SG5 bearings (Nordica, Treviso, Italy), placed on the outside of the platform (Figure 3-1). In addition, there was an interior compartment that allowed for an addition of weight to the platform. This functioned to lower the center of gravity of the platform and prevent backward rotation upon impact. The compartment also allowed the testers to normalize the weight of the platform across subjects.

Instrumentation

A tri-axial accelerometer (Model CXL10GP3, Crossbow, San Jose, CA) was used to capture acceleration measurements of the head. The accelerometer was connected to EVa Real-Time software (Motion Analysis Corporation, Santa Rosa, California). All acceleration data was sampled at 1000 Hz. Data was exported into Microsoft Office Excel 2003 (Microsoft, Redmond, WA) for reduction. Statistica version 5 (Statsoft Inc., Tulsa, OK) was used for statistical analyses.

Procedure

The experimental procedures were the same for all participants and all trials took place in the Biomechanics Lab in the College of Health and Human Performance at the University of Florida. Before the participant arrived, the seat back was set to the appropriate angle. The three angles that were used in this study were 100, 115, and 130° from horizontal, in that order. The participants were not told which angle was being tested. Only one angle was tested per day. After signing the informed consent form and filling out the medical health questionnaire, the participant was weighed. The forms were only filled out prior to the first visit, however the participant was weighed during each visit. Next, the participant was fitted with the accelerometer that was securely fastened to a mouthpiece (Figure 3-4). In order to normalize the weight of the platform and the test participant, weight was added to the compartment below the seat until the combined weight of the participant, rolling platform, and added weight reached 113.6 kg (250 lbs). This constant weight helped to ensure that each trial was as close to five mph as possible. The proper release point that produced the desired speed was determined during pilot testing using motion analysis software.

Each participant was then instructed to sit in the seat at the top of the ramp while looking straight ahead keeping his head parallel with the ramp's surface and his arms relaxed on his lap throughout the trial. The participant was told to remain relaxed throughout the trial, meaning that he would not tense his muscles before impact. The participant was also instructed to keep his scapulas against the seat. This served to normalize the seating position for all participants. During all trials, the headrest was in the highest position to decrease the chance of neck hyperextension. Upon verification that the participant is ready, the rolling platform was released from the pre-determined point and the seat began to move down the ramp. After impact with the fixed wooden barrier, the platform came to rest. Throughout the trial, acceleration data was

recorded. Three trials were performed during each testing session. Finally, a follow-up phone call was made at one day, three days, one week, and two weeks post-test. These calls served to monitor the participant's health and to quantify soreness, if present, on a graded scale. The pain scale appears in Appendix B⁵⁶. In the two subsequent sessions, the data collection method was the same, however the angle of the seat was changed. The data was analyzed after each session.

Data Analysis

Acceleration of the Head

Acceleration of the head was calculated in g's, where one g equals an acceleration of 9.81 m/s², the acceleration on gravity. The peak acceleration in the anterior-posterior and vertical directions was recorded and the resultant acceleration was calculated. The third direction was ignored due to the minimal motion in the lateral direction during the whiplash motion.^{6,19} Initial output from eVart 4.3 Motion Analysis Software was measured in millivolts. This raw data was exported to Microsoft Excel 2003 and graphed. The graph was used to find peak acceleration in volts. A sample graph has been provided (Figure 3-5). The baseline voltage was then subtracted from the peak voltage in order to quantify the true peak acceleration. After finding this peak acceleration in volts, a conversion factor supplied by Crossbow allowed for a conversion from volts to g's. The conversion factors for horizontal and vertical acceleration were 0.098 and 0.101 volts per one g, respectively. Peak horizontal acceleration measured in g's (a(g)) was calculated using Equation 3-1:

$$a(g) = (\text{voltage}_{\text{max}}(\text{V}) - \text{voltage}_{\text{baseline}}(\text{V})) / 0.098 \quad (3-1)$$

Peak vertical acceleration was calculated using equation 3-2:

$$a(g) = (\text{voltage}_{\text{max}}(\text{V}) - \text{voltage}_{\text{baseline}}(\text{V})) / 0.101 \quad (3-2)$$

This process was repeated for each trial completed. The peak mean horizontal and vertical accelerations for each seat angle were calculated. In addition, the resultant peak accelerations

were calculated and averaged for each seat angle. Finally, one trial was selected for time-motion analysis. During this process, key moments were picked from the trial and the resultant acceleration, magnitude and direction, were plotted on a graph. This graph shows the severity and direction of acceleration throughout the trial.

Pain Scale

Participants rated their pain one day, three days, one week, and two weeks post-test. This process was completed for each of the three test sessions. Data was written into Microsoft Excel. Mean pain ratings were calculated for each time interval at each seat angle.

Statistical Analysis

Acceleration of the Head

A repeated measures one-way ANOVA with three levels was used to determine if there was any significant difference in the mean peak acceleration of the head across seat angle. This was done for the horizontal, vertical, and resultant acceleration values. A traditional level of significance was used ($\alpha = 0.05$). Tukey HSD post hoc analyses were performed when necessary.

Pain Scale

A repeated measures two-way ANOVA (3:seat angle x 4:time interval) was used to determine if there was a significant pain difference among the three seat angles and as time passed post testing. A traditional level of significance was used ($\alpha = 0.05$). In addition, Tukey HSD post hoc analyses were performed when necessary.

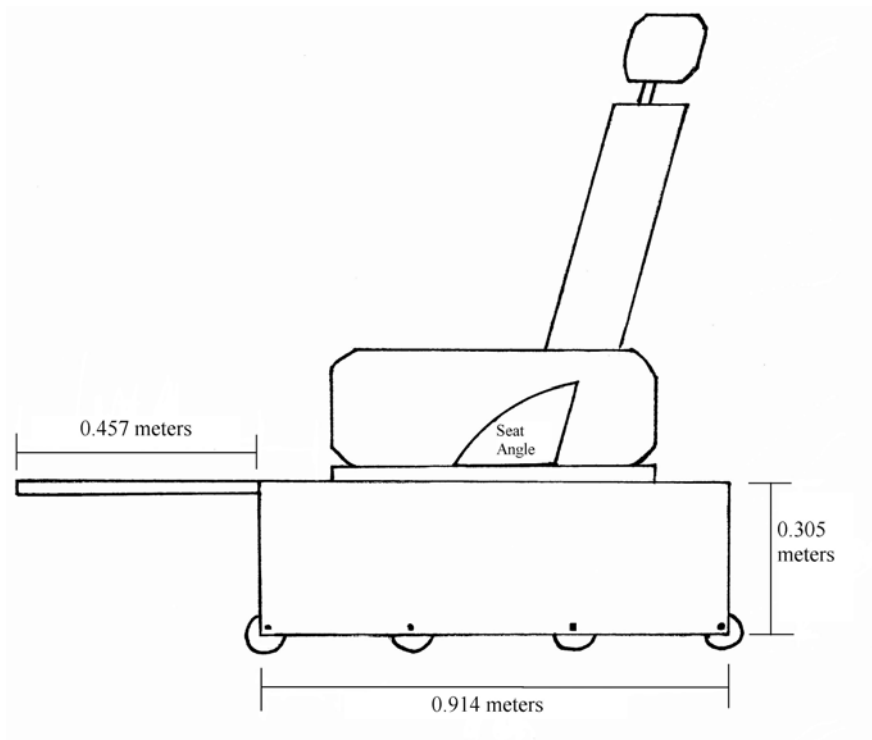


Figure 3-1. Automobile seat mounted on a rolling platform.

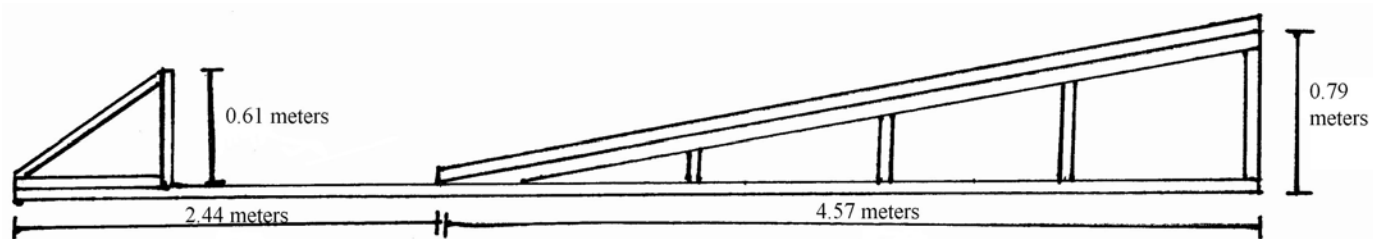


Figure 3-2. Ramp and stopping apparatus.

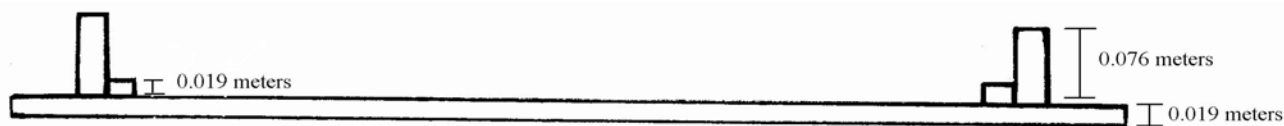


Figure 3-3. Guard rails on ramp.

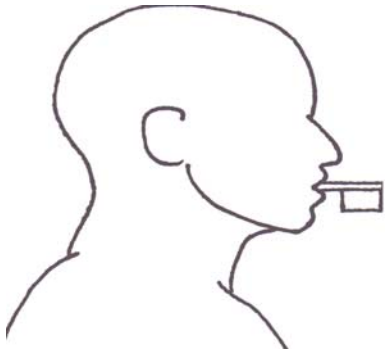


Figure 3-4. Accelerometer and mouthpiece.

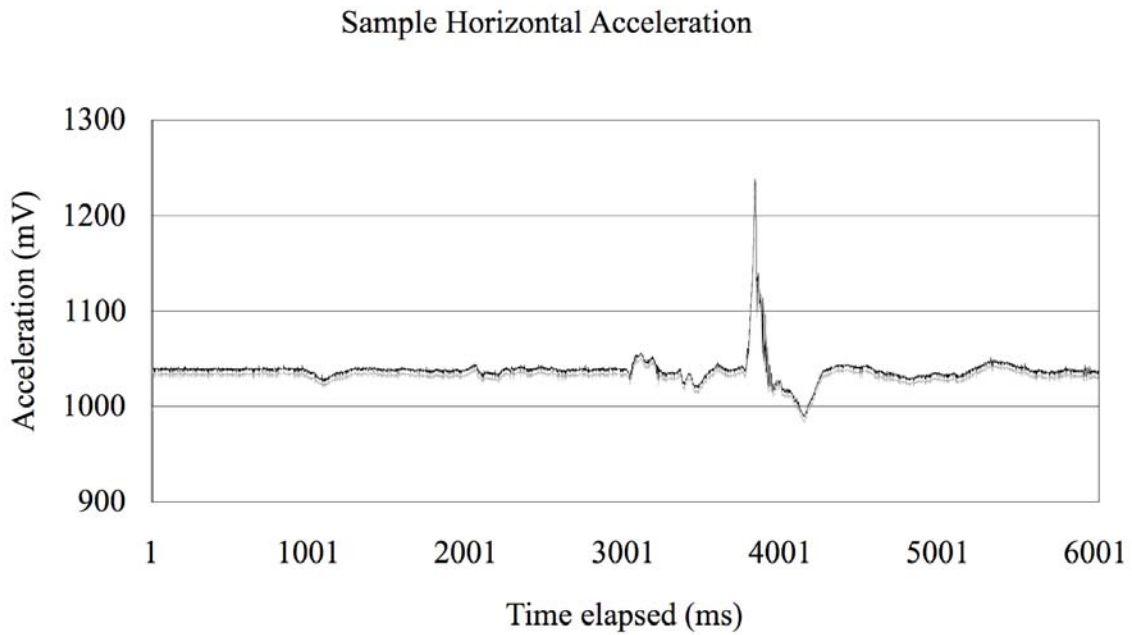


Figure 3-5. Sample horizontal acceleration graph- the vertical axis is measured in millivolts and the horizontal axis is measured in milliseconds.

CHAPTER 4 RESULTS

Participants

Nine of the ten participants completed testing at all three seat angles. The horizontal acceleration data for the third trial at 130° for participant five were not recorded due to equipment errors. Mass, height, and age were recorded for all ten participants (Table 4-1).

Horizontal Acceleration

Following testing, mean peak acceleration values for all three seat angles were calculated (Figure 4-1). A repeated measures one way ANOVA revealed a significant difference when comparing peak horizontal acceleration across the three levels of seat angle ($F(2,18) = 27.39, p = 0.000003$). Tukey's HSD post-hoc procedure indicated that horizontal acceleration was decreased for the 130° seat position when compared to the 100° position ($p = 0.000151$). In addition, the horizontal acceleration was also decreased for the 115° seat position when compared to the 100° position ($p = 0.000552$).

Vertical Acceleration

The same statistical methods previously mentioned were used for the vertical acceleration data. Mean peak acceleration values can be found in Figure 4-1. No differences in vertical acceleration across seat position were detected ($F(2,18) = 3.48, p = 0.0529$).

Resultant Acceleration

Resultant accelerations were calculated using the horizontal and vertical components and applying the Pythagorean theorem. Mean peak resultant acceleration values for each seat angle were then calculated (Figure 4-1). Statistical analyses were the same as described for the horizontal and vertical accelerations. Resultant acceleration varied with seat position ($F(2,18) = 4.51, p = 0.0258$). The mean acceleration values decreased as the seat angle increased

(Figure 4-1). Tukey's post hoc analysis revealed that the 100° seat position had a higher resultant acceleration than the 130° position ($p = 0.0425$). The 115° seat position also had a significantly higher resultant acceleration than the 130° seat position ($p = 0.0488$).

Time Motion Analysis

A time motion analysis was performed on one selected trial (Participant 3, 100°, trial 1). The horizontal and vertical acceleration graphs corresponding to this trial can be found in Figure 4-2. To create the time motion analysis, seven significant points of motion were chosen and the magnitude and direction of the resultant acceleration were calculated and plotted (Figure 4-3). Horizontal and vertical components of the resultant accelerations can be found in Table 4-2. There are no data points for the first phase because there is no movement of the head during the first phase, as defined by the previously described five phase whiplash motion. The first two points selected were 50 and 75 ms after the initial horizontal acceleration of the head, respectively, and were during the middle portion of the second phase of motion. Both of these points have a forward and downward acceleration. In addition, the acceleration is increasing in both directions across the 25 ms time span. At 100 ms, peak horizontal acceleration occurs and phase two is complete. During phase three, the horizontal acceleration is beginning to approach zero g's at 125 ms. 153 ms after impact, the horizontal acceleration has changed from forward to backward. Phase three is over 180 ms after impact and the participant is at the peak horizontal acceleration for phase three. This acceleration is directed backwards and down. Finally, at 269 ms, the peak horizontal acceleration during phase four was recorded. The direction of the acceleration is forward and up. At this point in the motion, all relevant accelerations have been completed. Due to this, the fifth phase, or restitution phase, was not included.

Pain Scale

The participants subjectively reported pain at four different time intervals that included one day, three days, one week, and two weeks post-test (Appendix B). All ten participants completed the pain ratings at all time intervals for all three seat positions. There were no ratings above a one (no pain) for the last two time intervals. Therefore, only the pain ratings given at one day and three days post-test were used in the statistical analysis. The highest pain rating given by any participant was a four (Participant 7, 130°, 1 day post-test) and no subject had any long term effects from the impacts. Generally speaking, the mean pain rating increased as the seat angle increased (Figure 4-4). In addition, when comparing pain ratings from day one to day three, pain decreased as time passed for the 115° and 130° seat positions while it stayed the same for the 100° seat position (Figure 4-5). Statistical analysis revealed significance when comparing overall pain ratings across time ($F(1,9) = 6.43, p = 0.032$), with pain decreasing from one day post-test to three days post-test. Pain ratings also varied with seat position ($F(2,18) = 6.55, p = 0.0073$). Tukey's HSD post hoc test revealed that there were significant increases among the mean pain ratings when comparing the 130° seat position to the 100° position ($p = 0.0076$) and when comparing the 130° position to the 115° position ($p = 0.041$), with the 130° position having the higher pain rating in both cases (Figure 4-4). A time x seat position interaction was also detected ($F(2,18) = 6.28, p = 0.0085$). Tukey's HSD post hoc test demonstrated that only the 130° seat position showed significance when comparing the pain ratings across both time intervals (Figure 4-5), meaning that pain ratings were significantly worse one day post test when compared to three days post test for that seat position ($p = 0.000094$).

Table 4-1. Participant characteristics

Participant	Age (years)	Height (m)	Mass (kg)
1		21	1.85
2		21	1.83
3		21	1.78
4		25	1.83
5		23	1.75
6		22	1.80
7		22	1.78
8		19	1.75
9		28	1.78
10		22	1.75
Mean \pm SD	22.40 \pm 2.37	1.79 \pm 0.035	81.92 \pm 11.51

Table 4-2. Acceleration data for time motion analysis

Time (ms)	Phase of motion	Horizontal acceleration (g)	Vertical acceleration (g)	Resultant acceleration (g)	Angle (°)
50	2	-1.1122	-0.3465	1.1649	17.3 below negative x-axis
75	2	-1.2755	-0.9406	1.5848	36.4 below negative x-axis
100	End of 2	-2.4490	1.1485	2.7049	25.1 above negative x-axis
125	3	-0.5306	1.9010	1.9737	74.4 above negative x-axis
153	3	0.0102	-0.3861	0.3862	88.5 below positive x-axis
180	End of 3	0.5816	-0.3564	0.6821	31.5 below positive x-axis
269	4	-0.3980	0.5644	0.6906	54.8 above negative x-axis

Note: For horizontal data, negative was defined as forward acceleration. For vertical data, negative was defined as downward acceleration.

Mean Peak Acceleration

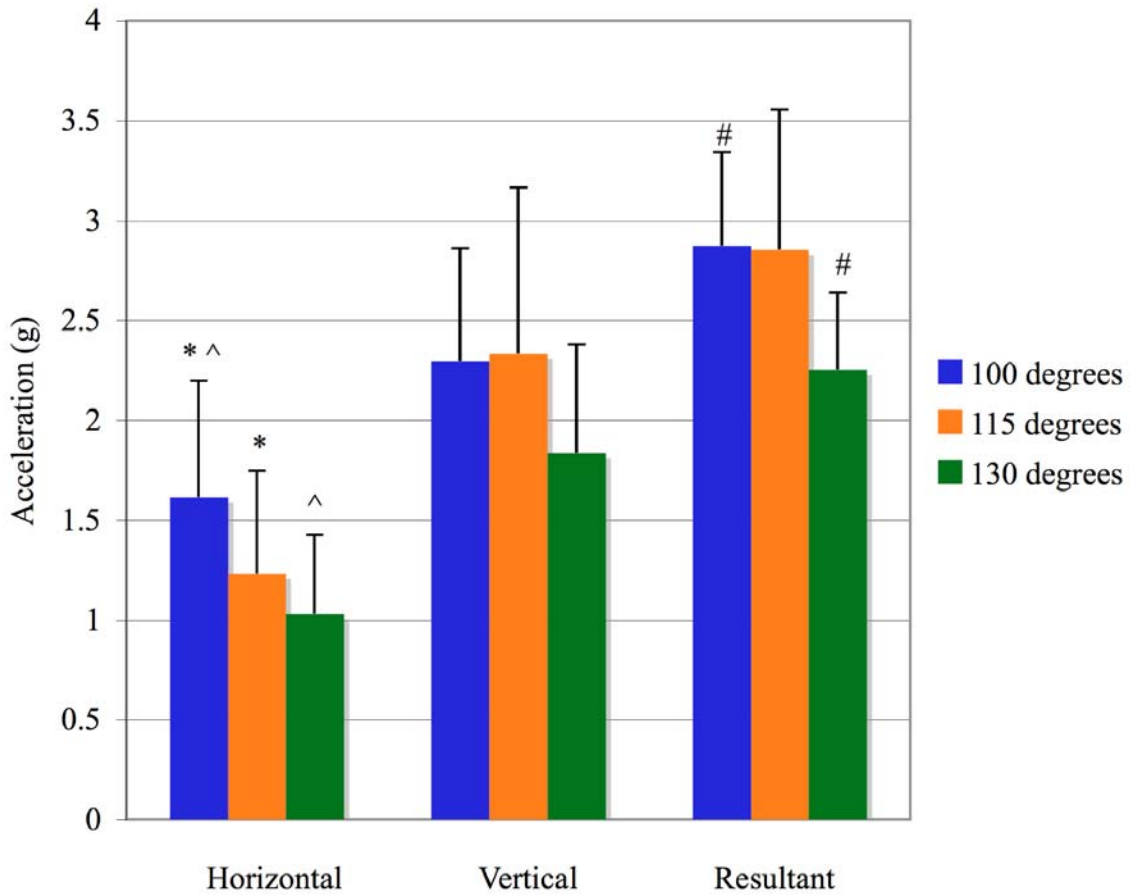


Figure 4-1. Mean horizontal, vertical, and resultant acceleration for all angles. * indicates a significant difference between peak horizontal acceleration values when comparing 100° and 115° ($p < 0.05$). ^ indicates a significant difference between peak horizontal acceleration values when comparing 100° and 130° ($p < 0.05$). # indicates a significant difference between peak resultant acceleration values when comparing 100° and 130° ($p < 0.05$).

Horizontal and Vertical Acceleration

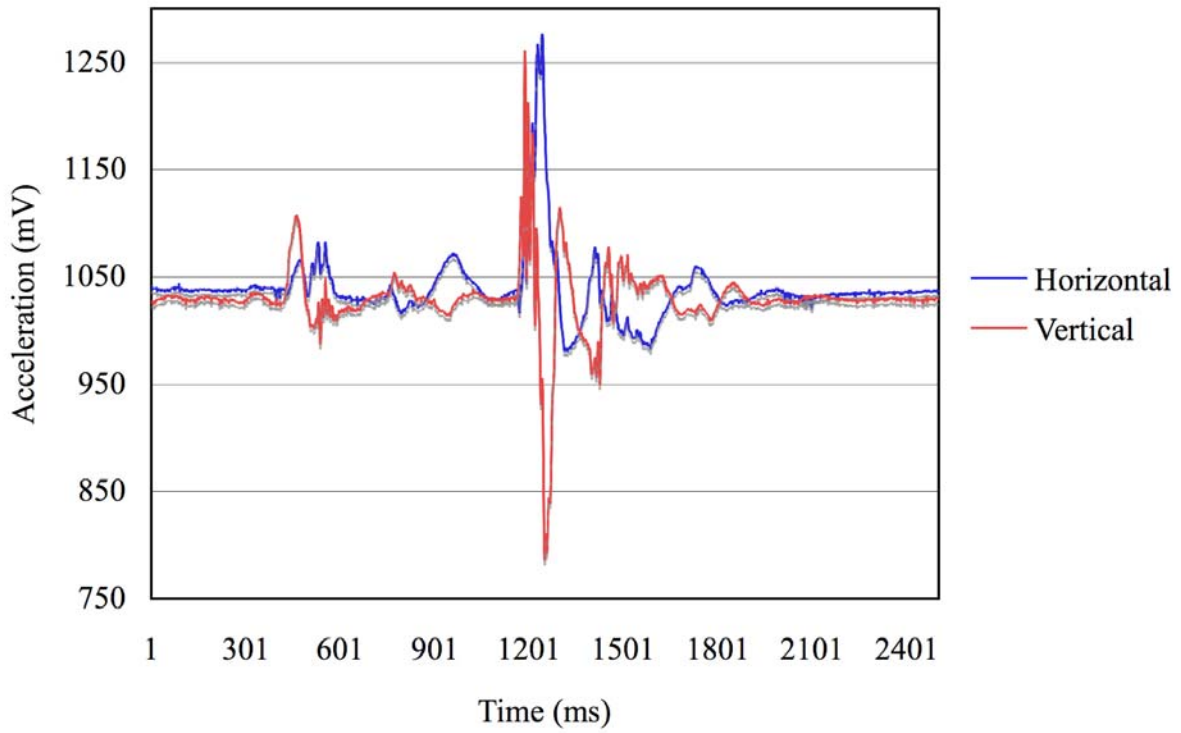


Figure 4-2. Horizontal and vertical acceleration for participant three, 100°, trial one. For horizontal acceleration, values above and below the baseline represent anterior and posterior acceleration of the head, respectively. For vertical acceleration, values above and below the baseline represent downward and upward acceleration, respectively.

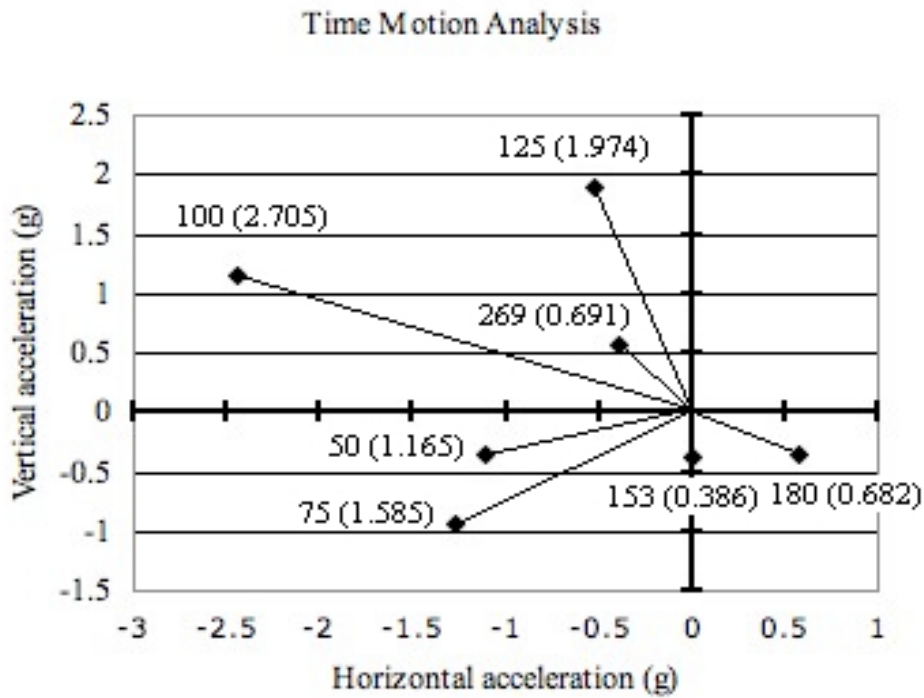


Figure 4-3. Time-motion analysis for participant three, 100°, trial one. The first number represents time in milliseconds after impact. The number in parentheses is the resultant acceleration value at that time.

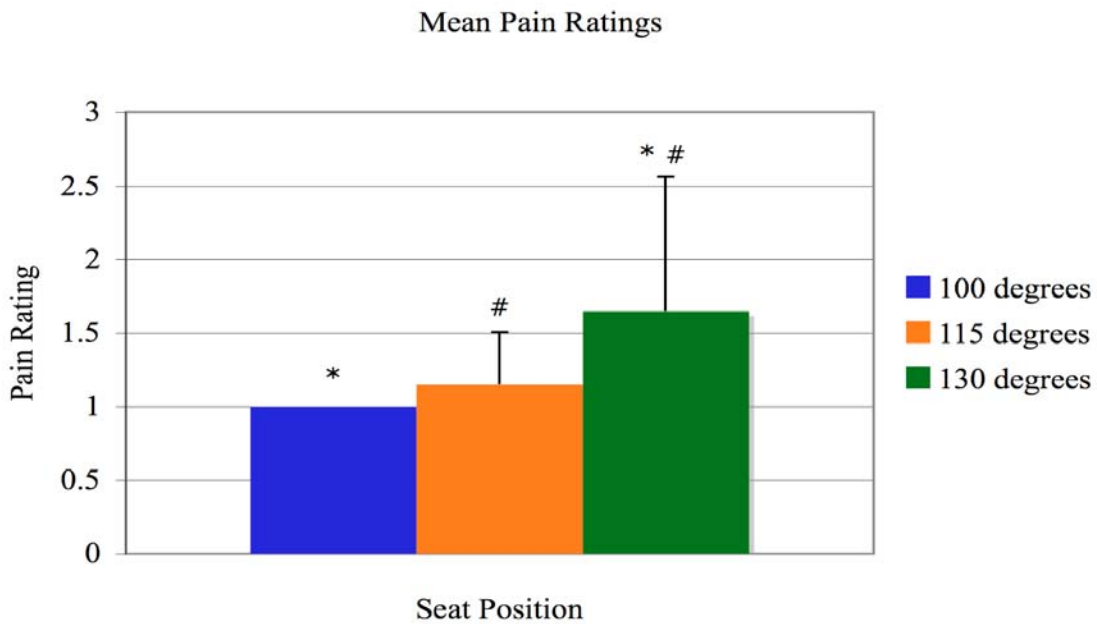


Figure 4-4. Mean pain ratings at each seat position. * indicates a significant difference between 100° and 130° ($p < 0.05$). # indicates a significant difference between 115° and 130° ($p < 0.05$).

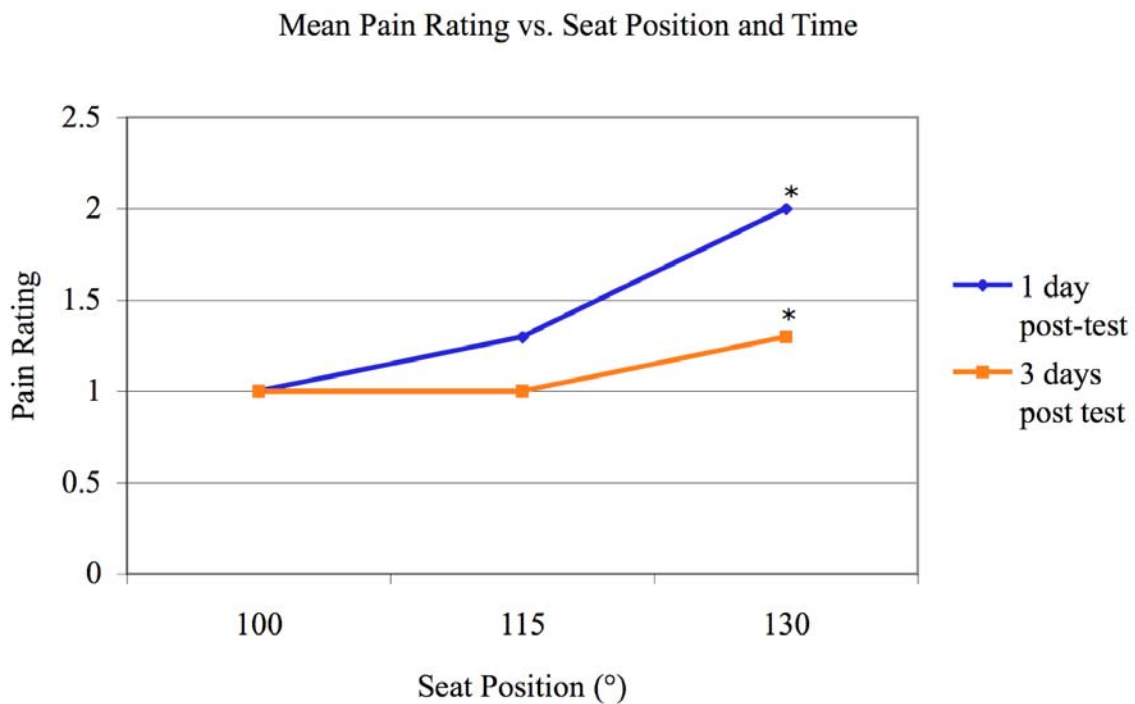


Figure 4-5. Mean pain rating interaction with seat position and time. * indicates a significant difference between 1 day and 3 days post-test at a seat position of 130° ($p < 0.05$).

CHAPTER 5 DISCUSSION

Horizontal Acceleration

In comparison to other studies at similar impact speeds, the peak horizontal accelerations during this study were slightly lower.^{12,19} However, because there are no comparative data regarding the kinematics of the head and the contribution of seat back angle to the acceleration of the head, this portion of the discussion is intended to explain these novel data.

The data collected during this study suggest that altering the angle of the seat back can create changes in the horizontal acceleration of the head during a low velocity rear-end collision. This is shown by the decrease in mean acceleration values as the seat angle was increased (Figure 4-1). When studying the kinematics of the head during a selected trial, the horizontal portion of Figure 4-2 shows only one peak. This peak coincides with the larger of the two vertical peaks and represents the maximum forward acceleration of the head while it is in contact with the headrest. While the head does accelerate downwards after impact, evidenced by the first vertical peak, it does not accelerate horizontally unless the musculature of the neck is activated to resist the rearward motion. During this study, the seat was rolling backwards at approximately five mph until impact. Upon impact the body continues to move horizontally at that velocity without accelerating until it hits the headrest or until the muscles fire, whichever comes first. This can be compared to a true rear-end collision where the car moves forward until the seat interacts with a stationary body. This phenomenon explains why the vertical acceleration begins just before the horizontal acceleration. The main difference between the vertical and horizontal motions is that the head was already moving horizontally, but had no initial velocity in the vertical direction, thus producing the kinematic differences. During the analysis of this trial it can be estimated that the muscles fired approximately fifteen ms after

impact with the wooden barrier. Although there was no electromyographic data collected, the initiation of the horizontal and vertical acceleration can be approximated from the time motion analysis (Figure 4-2). This indicates that the participant was anticipating the impending impact, as this delay is insufficient for a reflex response.⁷

When discussing differences in acceleration among seat positions, the horizontal acceleration of the head should be determined by the time it takes for the stopping forces of the muscles and headrest to arrest all horizontal motion, assuming the same Δv of five mph (acceleration = $\Delta v / \Delta t$). Because the 100° seat back angle creates less horizontal distance from the back of the head to the headrest, when compared to a 130° seat back angle, it is fair to conclude that the time it takes for the head to make contact with the headrest is shorter. Because of this, the muscles have less time to react and all or most of the forces created by the impact must be dissipated by the headrest in a shorter time, thus creating higher peak acceleration values. However, a higher peak acceleration value does not necessarily mean that there will be a higher risk of WAD injuries. A key factor that determines injury is the mechanism used to decelerate the head. The muscles and headrest both act to stop the motion of the head, but the contributions of each cannot be calculated from the acceleration data collected during this study. However, the contribution of pain in conjunction with the acceleration data may provide some insight and will be discussed later.

Vertical Acceleration

The vertical acceleration data showed no significance when comparing peak acceleration across the three seat positions. During the trial chosen for analysis, the temporal presentation of the vertical acceleration was very different than the horizontal acceleration and was most likely representative of the other data collected. According to Figure 4-2, the vertical acceleration

began fifteen ms before the horizontal acceleration. In addition, the vertical acceleration had two peaks while the horizontal acceleration only had one true peak. The first, smaller peak in the vertical acceleration represents the initial downward motion of the head after the seatback has pushed the torso forward and the cervical spine is forced into extension. The anterior neck muscles are responsible for attempting to counteract the force of the impact. This peak is prior to contact with the headrest. The net vertical acceleration during this time period is downward for one primary reason. Although the anterior musculature of the neck is attempting to slow the head down by producing an upward acceleration, the downward force created by the impact is greater in magnitude, thus the overall acceleration is still downward. The second, larger peak represents the net upward acceleration imparted by the combination of the headrest and the continuing action of the anterior musculature. During this portion, the resultant upward force is greater than the downward force remaining from the impact, thus the acceleration vector points upward. This peak is larger because the headrest and muscles are providing resistant forces, as opposed to just the muscles. However, although the second peak is larger, I believe the first peak is where an injury is more likely to occur, due to the higher initial load on the muscles. Finally, because there has been no significant difference found when comparing peak vertical accelerations across the three seat positions, it can be concluded that vertical motion during whiplash is not influenced by seat position. In comparison with other studies, peak vertical accelerations in this study were slightly lower, with previous studies showing peak vertical accelerations ranging from 2.4 to 3.0 g's.^{12,13}

Resultant Acceleration

As shown by the data in Figure 4-1, the resultant acceleration of the head does change as the angle of the seat back is altered. This difference may be attributed to the following. During a rear-end collision, the head may undergo two sub-phases during the initial rearward rotation, or

phase two of the whiplash motion. The first sub-phase is prior to contact with the headrest and the second is initiated with contact between the head and headrest and terminated when rearward rotation ceases. What occurs during the first sub-phase may be crucial to the injury risk encountered by the driver and the front row passenger. During this study, the overall Δv was the same for all trials. However, the final velocity before impact with the headrest, or the initial velocity upon impact with the headrest, was most likely different across the seat positions. As the head was moving backwards and downwards relative to the body, the 130° seat angle could have allowed the muscles to reach higher activity levels due to the increased horizontal distance and time required, and thus would create a lower final velocity before impact when comparing to the 100° seat angle. The lower final velocity is simply due to the increased deceleration caused by the muscle force. Because the initial velocity on impact with the headrest is lower at the 130° seat angle, the deceleration created by the headrest will be reduced while the deceleration created by the musculature is increased. Conversely, if the seat is in the 100° position, the resultant peak acceleration is higher, but the headrest contributes more retarding force and the muscles contribute a lower retarding force. Therefore, in accordance with previous work, the headrest should be as close to the back of the head as possible in order to avoid hyperextension injuries of the neck.^{1,14,19,22,24,26,27}

Time Motion Analysis

The main function of the time motion analysis was to verify the motion of the head throughout the whiplash motion during this study. As seen in Figure 4-3, the direction and magnitude of acceleration was constantly changing throughout the motion. The first point selected was 65 and 50 ms after initiation of the vertical and horizontal accelerations, respectively, and occurred during phase two. At this time, the head had a forward acceleration of

1.11 g's in combination with a downward acceleration of 0.35 g's (Table 4-2). The anterior musculature of the neck, specifically the SCM, was responsible for slowing the head down. Although the net vertical acceleration was downwards, the musculature was acting to accelerate the head upward, against the motion of the head, however the force of the impact was larger than the muscle force. This combination created a net downward force and acceleration. The next point of interest is at 87 ms after initiation of horizontal acceleration and is also in phase two. It is at this moment that the head made initial contact with the headrest. Notable occurrences include a transition from downward to upward vertical acceleration and a continuing increase in the horizontal acceleration. The third major point during phase two was 100 ms after initiation of the horizontal acceleration and represented the peak horizontal acceleration throughout the entire trial. This point also represents the end of phase two. Perhaps the most important time span during this phase was the 13 ms in which the head was in contact with the headrest. It was during this short time that the head experienced the most severe g forces in all dimensions. However, the headrest is primarily responsible for these high g forces, thus WAD injuries during contact with the headrest are unlikely. In addition, the g forces are not high enough to cause significant intracranial injury.

Phase three was characterized by far less severe g forces. The first notable point in phase three occurs at 153 ms. Horizontal acceleration has just crossed zero g's and is now 0.01 g's in a backward direction, meaning that the posterior musculature has been activated and is attempting to terminate the forward motion of the head created by the rebound off of the headrest, thus producing a backward acceleration. Vertical acceleration at this point is 0.3861 g's downward. The peak horizontal acceleration occurred 180 ms after the initial horizontal acceleration, or 80

ms after phase three began and had a value of 0.5816 g's. It is also at this time that phase three was completed.

The final point of interest occurs 269 ms after initial horizontal acceleration and is in phase four. The head had passed its equilibrium, or upright, point and was forced into slight extension. Peak horizontal acceleration was 0.40 g's forward and peak vertical acceleration was 0.56 g's upward.

Overall, the results of this time motion analysis were comparable to previous work done by McConnell, et al.¹³ The impact speed of both trials was very similar, and the overall motion of the head during this study seemed to follow the previously defined five stage motion associated with whiplash.^{3,6,7}

Pain

Using the pain data in conjunction with the horizontal and resultant acceleration data, one can infer that the muscles were contributing more during the trials with the seat reclined to 130°. This is because there were significant differences in overall pain ratings when comparing 100° with 130° and when comparing 115° with 130° (Figure 4-4), with the higher pain rating means corresponding to the higher seat angles. Therefore, the increased time allowed the muscle to increase its contribution and spread the force more equally among the muscles and headrest, creating pain in some participants, while decreasing the headrest's contribution. The pain data also show that the 130° seat position was the only position that demonstrated a significant difference in pain from one day post-test to three days post-test, which would suggest a more significant muscle injury and increased muscle contribution during the testing at 130°. Other structures, such as the vertebrae, intervertebral discs, and ligaments are also at increased risk when the seat is more reclined. If the muscles are unable to absorb the complete force of the

impact prior to contact with the headrest, the force will be transmitted to other anatomical structures. The muscles will no longer be able to resist the extension motion of the cervical spine and more serious hyperextension injuries become more likely.

Limitations

This project had limitations that should be addressed while interpreting the results and for designing future studies. Due to injury risk, only males between the ages of 18 and 35 were approved to participate by the Institutional Review Board of the University of Florida, thus it cannot be assumed that the results apply to the whole population. Second, there was no plausible way to ensure that the participants were relaxed prior to impact. Because the platform was rolling backwards into a fixed barrier, the participant could predict when the impact would occur. This is supported by the time motion analysis of the selected trial. There are several ways to correct this in the future. Electromyographic recording devices for the musculature of the neck could be used to determine exactly when the muscles fired relative to impact. In addition, the barrier could move forward into a stationary seat platform with all visual and auditory cues removed from the environment. This technique could be combined with random dummy trials, or trials in which there is no contact between the barrier and the platform. Third, sample size is a concern. A larger sample size would produce more reliable results. Fourth, only one seat model was used. For better results, seats from all major car manufacturers could have been used. Fifth, only three angles were tested during this study. To truly determine the best combination of comfort and safety, seat positions in increments of 5° should be tested in further studies. Sixth, the headrest was at the maximum height for all subjects. Due to differences in height among the participants it cannot be assumed that all participants made contact with the same part of the headrest. Finally, the apparatus could have been substituted with real automobiles to create a true bumper to bumper impact.

Conclusions

The data collected during testing show that horizontal and resultant acceleration are significantly altered when the orientation of the seat back is changed from 100° to 130°. In addition, the pain felt by the participants increases when comparing 100° and 130° and when comparing 115° to 130°. These results support the current trend in automobile manufacturing to produce active head restraints. If the head restraint moves forward as the seat is reclined backwards, it is possible to maintain the same horizontal distance from the back of the head to the front of the headrest, thus eliminating much of the muscle force required to arrest the motion of the head during a low velocity rear-end collision. However, much of the population does not own an automobile with this capability. Therefore, it is recommended that a seat position of 100° is used in order to minimize risk of injury from a rear-end collision when operating an automobile. Finally, I believe that the previously defined five stage whiplash motion^{3,6,7} should be modified to a six stage motion including the two sub-phases previously discussed. Although the rearward rotation of the head is characteristic of both sub-phases, the injury risk and decelerating mechanisms are different.

APPENDIX A
MEDICAL HEALTH QUESTIONNAIRE

Subject Number: _____

Age: _____ (must be between 18 and 35 to be eligible)

Height: _____

Weight: _____

Sex: M / F

Please answer yes or no to questions #1-11. If you answer “yes” to any of the following questions (1-7,10), you will not be eligible for this study. If you answered “no” to questions 8 or 9, you will not be eligible for this study.

1. Have you ever had a spinal injury of any kind?
2. Do you suffer from severe headaches or migraines?
3. Do you currently have any injury to your neck or back?
4. Have you ever had “whiplash” before?
5. Have you ever had a herniated disc or any other disc injury?
6. Do you suffer form dizziness?
7. Are you currently taking any pain medications?
8. Do you have a pain-free range of motion of your cervical spine?
9. Can you move your neck freely in all directions?
10. Do you suffer from any long standing neck pain?
11. Please list any other medical problems that you think would prevent you from participating.

APPENDIX B
SORENESS SCALE

Subject Number: _____ Session #: _____ Date: _____

Directions: Rate your level of soreness on a 1-10 scale.

1 = no pain 3 = slightly painful 5 = moderately painful
8 = very painful 10 = extremely painful

1 day post-test

Date: _____

Level of Soreness: 1 2 3 4 5 6 7 8 9 10

3 days post-test

Date: _____

Level of Soreness: 1 2 3 4 5 6 7 8 9 10

1 week post-test

Date: _____

Level of Soreness: 1 2 3 4 5 6 7 8 9 10

2 weeks post-test

Date: _____

Level of soreness: 1 2 3 4 5 6 7 8 9 10

LIST OF REFERENCES

1. Svensson M, Lovsund P, Haland Y, Larsson S. The influence of seat-back and head-restraint properties on the head-neck motion during rear impact. Proceedings of the International IRCOBI Conference on the Biomechanics of Impacts 1993; 395-406.
2. Barnsley L, Lord S, Bogduk N. Clinical review: whiplash injury. *Pain* 1994; 58: 283-307.
3. McConnell WE, Howard RP, Poppel JV. Human head and neck kinematic after low velocity rear-end impacts: understanding "whiplash". In: 39th Stapp Car Crash Conference Proceedings: SAE No 952724 1995: 215-238.
4. Brault J, Wheeler J, Siegmund G, Brault E. Clinical response of human subjects to rear-end automobile collisions. *Archives of Physical Medicine and Rehabilitation* 1998; 79: 72-80.
5. Yoganandan N, Pintar F, Cusick J. Biomechanical analyses of whiplash injuries using an experimental model. *Accident Analysis and Prevention* 2002; 34: 663-671.
6. Howard R, Bowles A, Guzman H, Krenrich S. Head, neck, and mandible dynamics generated by 'whiplash'. *Accident Analysis and Prevention* 1998; 30(4): 525-534.
7. Ono K, Kaneoka K, Wittek A, Kajzer J. Cervical injury mechanism based on the analysis of human cervical vertebral motion and head-neck-torso kinematics during low speed rear impacts. In: 41st Stapp Car Crash Conference Proceedings: SAE No 973340 1997: 339-356.
8. Harrison DD, Harrison SO, Croft AC, Harrison DE, Troyanovich SJ. Sitting Biomechanics, Part II: Optimal Car Driver's Seat and Optimal Driver's Spinal Model. *Journal of Manipulative and Physiological Therapeutics* 2000; 23(1): 37-47.
9. Severy DM, Mathewson JH. Automobile barrier and rear-end collision performance. Society of Automotive Engineers summer meeting, Atlantic City, NJ, 1958: June 8-13.
10. Severy DM, Mathewson JH, Bechtol CO. Controlled automobile rear-end collisions, an investigation of related engineering and mechanical phenomenon. *Canadian Services Medical Journal* 1955; 11: 727-758.
11. Siegmund GP, King DJ, Lawrence JM, Wheeler JB, Brault JR, Smith TA. Head/neck kinematic response of human subjects in low-speed rear-end collisions. SAE technical paper 973341 1997: 357-385.
12. West DH, Gough JP, Harper TK. Low speed rear-end collision testing using human subjects. *Accident Reconstruction Journal* 1993; 5(3): 22-26.
13. McConnell WE, Howard RP, Guzman HM, Bomar JB, Raddian JH, Bendict V, Smith HL, Hatsell CP. Analysis of human test subject kinematic responses to low velocity rear end impacts. SAE paper No 930889 1993.

14. Minton R, Murray P, Stephenson W, Galasko CSB. Whiplash injury – are current head restraints doing their job?. *Accident Analysis and Prevention* 2000; 32: 177-185.
15. Ryan GA, Taylor GW, Moore VM, Dolinis J. Neck strain in car occupants: the influence of crash-related factors on initial severity. *Medical Journal of Australia* 1993; 159: 651-656.
16. Zuby DS, Vann T, Lund AK, Morris CR. Crash test evaluation of whiplash injury risk. Society of Automotive Engineers SAE No 99SC17 1999.
17. Anderson R, Welcher J, Szabo T, Eubanks J, Haight WR. Effect of braking on human occupant and vehicle kinematics in low speed rear-end collisions. Society of Automotive Engineers SAE No 980298 1998.
18. Szabo TJ, Welcher JB. Human subject kinematics and electromyographic activity during low speed rear impacts. Society of Automotive Engineers SAE No 962432 1996.
19. Welcher JB, Szabo TJ. Relationships between seat properties and human subject kinematics in rear impact tests. *Accident Analysis and Prevention* 2001; 33: 289-304.
20. Lundell B, Jakobsson L, Alfredsson B, Jernström, Isaksson-Hellman I. Guidelines for and the design of a car seat concept for improved protection against neck injuries in rear end car impacts. Society of Automotive Engineers SAE No 980301 1998.
21. Watanabe Y, Ichikawa H, Kayama O. Relationships between occupant motion and seat characteristics in low-speed rear impacts. Society of Automotive Engineers SAE No 1999-01-0635 1999.
22. Croft AC, Herring P, Freeman MD, Haneline MT. The neck injury criterion: future considerations. *Accident Analysis and Prevention* 2002; 34: 247-255.
23. Hosea TM, Simon SR, Delatizky J, Wong MA, Hsieh CC. Myoelectric analysis of the paraspinal musculature in relation to automobile driving. *Spine* 1986; 11: 928-936.
24. Farmer CM, Wells JK, Werner JV. Relationship of head restraint positioning to driver neck injury in rear-end crashes. *Accident Analysis and Prevention* 1999; 31: 719-728.
25. Siegmund GP, Heinrichs BE, Wheeler JB. The influence of head restraint and occupant factors on peak head/neck kinematics in low-speed rear-end collisions. *Accident Analysis and Prevention* 1999; 31: 393-407.
26. Viano D, Gargan M. Headrest position during normal driving: implication to neck injury risk in rear crashes. *Accident Analysis and Prevention* 1996; 28(6): 665-674.
27. Maher J. Report investigating the importance of head restraint positioning in reducing neck injury in rear impact. *Accident Analysis and Prevention* 2000; 32: 299-305.

28. Severy D. Engineering studies of motorist injury exposures from rear-end collisions. *Trauma* 1970; 12(2): 81-106.
29. Nygren A, Gustafsson H, Tingvall C. Effects of different types of head restraints in rear-end collisions. 10th International Conference on Experimental Safety Vehicles, Oxford, England, 1985: 85-90.
30. Lawrence JM, Siegmund GP. Seat back and head restraint response during low-speed rear-end automobile collisions. *Accident Analysis and Prevention* 2000; 32: 219-232.
31. Viano D. Influence of seatback angle on occupant dynamics in simulated rear-end impacts. Society of Automotive Engineers SAE No 922521 1992.
32. Thomas P, Bradford M. The nature and source of the head injuries sustained by restrained front-seat car occupants in frontal collisions. *Accident Analysis and Prevention* 1995; 27(4): 561-570.
33. Kumar S, Narayan Y, Amell T. Role of awareness in head-neck acceleration in low velocity rear-end impacts. *Accident Analysis and Prevention* 2000; 32: 233-241.
34. Emori R, Horiguchi J. Whiplash in low speed vehicle collisions. Society of Automotive Engineers SAE No 900542 1990.
35. Mertz HJ, Patrick LM. Strength and response of the human neck. In: Proceedings of the 15th Stapp Car Crash Conference. Coronado, California: Society of Automotive Engineers SAE No 710855 1971: 207-255.
36. Ryan GA, Taylor GW, Moore VM, Dolinis J. Neck strain in car occupants: injury status after 6 months and crash-related factors. *Injury* 1994; 25: 533-537.
37. States J, Korn M, Masengill J. The enigma of whiplash injuries. Proceedings of the 13th Annual Conference of the American Association for Automotive Medicine 1969: 83-108.
38. Sturzenegger M, DiStefano G, Radanov BP, Schnidrig A. Presenting symptoms and signs after whiplash injury: the influence of accident mechanisms. *Neurology* 1994; 44: 688-693.
39. Brault J, Siegmund G, Wheeler J. Whiplash injury biomechanics: cervical electromyographic response and influence on whiplash dynamics. *Whiplash: Compendium of Abstracts* 1998; SP-1406: 25.
40. Elson L. The jolt syndrome – muscle dysfunction following low-velocity impact. *Pain Management* 1990; November/December: 317-326.
41. Szabo TJ, Welcher JB. Human subject responses to various acceleration fields. Low Speed Collision TOPTEC (SAE), Richmond, BC, August 1996.

42. Balla JI. The late whiplash syndrome. *Aust. N.Z. Journal of Surgery* 1980; 50(6): 610-614.
43. Bring G, Bjornstig U, Westman G. Gender patterns in minor head and neck injuries: an analysis of casualty register data. *Accident Analysis and Prevention* 1996; 28: 359-370.
44. Hohl M. Soft-tissue injuries of the neck in automobile accidents. *Journal of Bone and Joint Surgery* 1974; 56A: 1675-1682.
45. Kahane CJ. An evaluation of head restraints- federal motor vehicle safety standard 202 (DOT HS-806 108). US Department of Transportation, National Highway Traffic Safety Administration, February 1982.
46. Krafft M, Thomas A, Nygren A, Lie A, Tingvall C. Whiplash associated disorder- Factors influencing the incidence of rear-end collisions. In: *Proceedings of the 15th ESV Conference*. Melbourne, Australia: Paper No 96-S9-O-09 1996: 319-326.
47. Lövsund P. Neck injuries in rear end collisions among front and rear seat occupants. *Proceedings of International IRCOBI Conference Biomechanics of Impacts* 1988: 319-325.
48. Minton R, Murray P, Pitcher M, Galasko CSB. Causative factors in whiplash injury: implications for current seat and head restraint design. *Proceedings of International IRCOBI Conference on Biomechanics of Impacts* 1997: 207-222.
49. Morris A, Thomas P. A study of soft tissue neck injuries in the UK. *Proceedings of the 15th ESV Conference* May 1996; Paper No. 96-S9-O-08: 1412-1425.
50. O'Neill B, Haddon W, Kelley AB, Sorenson WW. Automobile head restraints: Frequency of neck injury insurance claims in relation to the presence of head restraints. *American Journal of Public Health* 1972; March: 399-406.
51. Otremski I, Marsh JL, Wilde BR, McLardy Smith PD, Newman RJ. Soft tissue cervical spinal injuries in motor vehicle accidents. *Injury* 1989; 20: 349-351.
52. Otte D, Pohlemann T, Blauth M. Significance of soft tissue neck injuries AIS1 in the accident scene, and deformation, characteristics of cars with delta-V up to 10 km/h. *Proceedings of the International IRCOBI Conference on the Biomechanics of Impacts* 1997: 265-83.
53. Spitzer WO, Skovron ML, Salmi LR, Cassidy JD, Duranceau J, Suissa S, Zeiss E. Scientific monograph of the Quebec task force on whiplash associated disorders: redefining 'whiplash' and its management. *Spine* 1995; 20: 8.
54. States J, Balcerak J, Williams J. Injury frequency and head restraint effectiveness in rear end impacts. *Society of Automotive Engineers SAE No 720967* 1972.

55. Van den Kroonenberg A, Philippens M, Cappon H, Wismans J. Human head-neck response during low-speed rear end impacts. Society of Automotive Engineers SAE No 983158 1998.
56. Cassidy J, Carroll L, Cote P, Frank J. Does multidisciplinary rehabilitation benefit whiplash recovery? Results of a population-based incidence cohort study. Spine 2007; 32: 126-31.

BIOGRAPHICAL SKETCH

Martin Charles Hillier was born in 1981, in Abbekerk, Netherlands. He was raised by English parents and lived in Holland until the age of five, when he moved to New Jersey. Martin immediately became involved in soccer, a life-long love. At the age of 10 Martin moved to Boca Raton, Florida, where he spent the next eight years of his life. While there, he attended Spanish River High School where he excelled in athletics and academics. Martin was later accepted into the University of Florida, where he spent his college years, without a doubt the years that defined him as a person. While there he graduated with a B.S. in exercise and sport sciences and an M.S. in applied physiology and kinesiology. Martin also has an older sister and a younger sister who attends or has attended the University of Florida. His other sibling, Robert, is training to be a law enforcement officer. Martin's years in Gainesville were most memorable because of his wife. He met Jill Megan Binkley on July 4, 2002, and they were married on July 8, 2006. Martin and Jill currently live in Saint Augustine, Florida. They are both teachers and they are avid travelers. Jill teaches 4th grade and Martin teaches IB physics. Jill plans to return to Gainesville to earn her doctorate degree and Martin plans to become involved in the accident reconstruction field in the near future.