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Hour 1
Section 1: Soft Tissue Injury – Acceleration/Deceleration Kinematics

In 1928, the first definition of Whiplash was recognized by Crowe, HE, in his paper *Injuries to the Cervical Spine*, presented in San Francisco at the Western Orthopedic Association. Defined as a syndrome, its symptoms included neck pain, headache, blurred vision, tinnitus, dizziness, nausea, paresthesias, numbness and back pain. Over time, due to third party liability payers, sufferers after acute trauma with these symptoms were accused of being hypochondriacs or malingering. Only more recently this stigma has been reduced as research allowed for better injury documentation and analysis of impact mechanisms and affects on the human frame. Although more is understood about the phenomena of movement impact on the body, there still remains a battle for evidence in each individual case as third parties continue to fight to minimize their financial responsibility. As practitioners, it is our responsibility to thoroughly evaluate, treat, educate and sometimes advocate for our acceleration/deceleration impact injury patients who may face a lifetime disability.

The term Whiplash was originally used to describe the mechanism of injury as the head whips around on the neck when it is subjected to abnormal speed and force. Over the years, it has been used to describe the syndrome of symptoms which can vary from patient to patient and is dependent on a variety of factors that include but not limited to the mechanics of the actual impact. Because of the wide variety of sequela and circumstance, this vague description of a complicated medical situation has been devalued making it easy fodder for third party payers to minimize their responsibility in paying for the patient’s recovery and/or disability.
In an attempt to avoid using Whiplash as a descriptive term to define the syndrome of injuries that occur in an acute traumatic event, hyperflexion/hyperextension impact was coined to better define the condition. The problem with this term is that not all impacts result solely in the flexion/extension of the cervical spine. This term was later abandoned for the more currently used term acceleration/deceleration impact. This allows for the high acceleration force and traumatic rotation of the head during impact and effects on soft tissues and bones. Descriptions within the framework of this nomenclature also take into account the direction of traumatic force. For example, a frontal collision would be described as a cervical deceleration injury; a lateral collision would be a lateral cervical acceleration/deceleration impact.

**Terms of movement dynamics:**

To best understand the phenomena of acceleration/deceleration injuries, it is necessary to learn some important terms used in describing the physics of the crash. These terms tend to be purposefully vague and confusing to allow for the doubt that is usually cast with a 3rd party payer who wishes to limit liability. They are also misused within the insurance profession by individuals who do not have a clear understanding of the differences between the terms and use them interchangeably. Having the knowledge of crash dynamics and terminology will allow for better reporting, documentation, clarification and determination for necessity of care. It should be noted that the definition of low speed rear impact crashes (LOSRIC) is that the change in velocity is less than 10 mph.

**Velocity:**

In defining the events of a crash, velocity or speed is usually referred in terms that would indicate a change.

*Impact velocity* is the speed of the striking vehicle. Therefore, if a truck is going E at 10 mph (V1) and is hit by a car going E at 15 mph (V2), the impact velocity can be considered to be 15 mph.

*Closing velocity* (relative speed of approach) is the difference between the two vehicle velocities (Vc). Therefore, using the above example, the closing velocity (V2 - V1) would be 5 mph. This is commonly referred to when attempting to discern the potential for injury. This is not used to determine the forces on the patient’s body during the crash.

*Change in velocity* is the difference between the initial velocity and the velocity after the crash. So, if the initial speed of the impacted vehicle was 10 mph and after being hit by a vehicle going 15 mph, the difference in speed between the two which is 5 mph is considered the change in velocity. This information usually determines the forces on the patient’s body during the crash or the kinematic response to the action.

Change in velocity is used to determine if the impact should be considered low or high. The definition of LOSRIC is that the change in velocity is less than 10 mph. The use of the term “low speed” has nothing to do with the actual speed but the difference in speed between the two vehicles at the time of the crash. If a vehicle was traveling 70 mph and was hit by a vehicle
traveling 80 mph, the difference of 10 mph would be the change in velocity. This impact would be considered a low speed rear impact even though the vehicles were traveling fast.

Guidelines have been determined by crash dummy tests to determine the forces on the human frame. The Federal Motor Vehicle Safety Standards (FMVSS) requires crash tests to be performed against a concrete barrier at the speed of 30 mph. The crush to the vehicle is also measured. These are front end crashes as opposed to the rear end crashes that calculate the change in velocity equations. The equations are used for these determinations are called Barrier Equivalent Velocities (BEV), Energy Equivalent Speeds (EES) or Equivalent Barrier Speeds (EBS). All of these terms are interchangeable and refer to a vehicle crashing into a fixed, rigid barrier. They do not refer to a vehicle crashing into a more flexible or moving target. Confusion arises when auto crash reconstructionists confuse the term change in velocity with BEV.

**Acceleration:**

Acceleration (a) of the vehicle is determined by comparing the change in velocity over a period of time.

\[
a = \frac{\text{change in velocity}}{\text{change in time}}
\]

*Velocity* is calculated in units of feet per second (fps). It is a measurement of the displacement during a fixed time. Time is considered to be the difference between the start of the movement at the time of initial impact and the end of the movement when the vehicle comes to rest.

*Acceleration* is determined in linear units of feet per second squared (fps/s or fps²) or meters per second squared (ms²). It is a displacement in relation to time. It can also be represented by gravitational forces or g forces. One g is equal to 32.2 fps² or 9.81 ms².

*Jerk* is another measurement of displacement during a period of time and is measured is fps/s/s.

*Deceleration* describes the slowing down of the object after impact. It is described in mathematical terms as negative acceleration or \( -a \).

*Angular acceleration* (noted by the symbol alpha) is the acceleration of a body moving through an arc. This occurs when the head is rotated about the neck on an arc in flexion, extension and lateral flexion. It is measured as radians per seconds squared or (rad/s²)

**Moments:**

Previous definitions of whiplash injuries included bending moments (violent flexion and/or extension of the neck) of impact. The terms hyperflexion and hyperextension lack in defining the variety of actions that take place on the cervical spine during impact. Also, they suggest the injuries occur when the normal physiological ranges of motion of the vertebral joints are exceeded and that preventing such motion will prevent injury. This is incorrect. It is also lacking in the rotary component that is common with impacts. Moments is used to describe
acceleration/deceleration impact in a variety of directions and is measured in Newton meters as Nm. One Nm is 0.7376 pound-force-foot (lbf-ft).

**Torque:**

This is the angular acceleration proportional to the force times the lever arm. The product of this equation is called the moment of force about the axis or torque. The angular acceleration is directly proportional to the torque.

**Compression:**

This is the load on the spine during the movement of forces during and as a result of impact. In the cervical spine, there is vertical loading which causes compression.

**Tension:**

Tension is the opposite of compression. This measures the forces on the spine when the segments are pulled apart.

**Shear:**

Shear measures the forces on the spine when one segment moves and the one above or below it does not. This is measured in Newtons (N).

**Section 2: Soft Tissue Injury – Acceleration/Deceleration Industry Guidelines and Models**

**Commonly Used Guidelines of Injury:**

Guidelines used to determine if a particular mechanism of injury caused a particular condition. Many of these guidelines do not translate into the real world and are used to perpetuate a malingering theory. In short, 3rd party payers do not want to pay so they minimize their responsibility by using guidelines that indicate a particular injury could not have come from a particular incident. Having knowledge of the guidelines will assist the practitioner to advocate for their patient and justify payment for services rendered.

One particular set of guidelines is the Ferrari et al known as “criteria for establishing the probability of a causal relationship between acute whiplash (excluding grades 3 and 4) and chronic symptoms”. If their particular set of criteria is not met, the authors determined that “it is highly improbable that the chronic injury following the accident is the basis for the chronic symptoms”. These criteria have no record of credible scientific basis and can be viewed as merely opinion.
The criteria are as follows:

1. For rear end collision between similar sized vehicles, the guidelines use the criteria of speed change – a striking speed of at least 16.1 mph or a change of velocity of 10.6 mph.

2. For front end collision or side impact the striking speed must be 21 mph.

3. There should be an onset of significant or pronounced neck pain within 48 hours.

4. Symptoms must follow “traumatologic principles” in which symptoms peak in 48 hours and continue to improve after. If not the patient is responsible for explaining what factors have perpetuated their pain and what relationship it has to the accident.

5. There should be no significant pre-accident history of similar symptoms.

6. There should be no Waddell’s signs. These are defined as no significant signs of non-organic features on examination characterized as “non-dermatomal factors”.

Regarding criteria #1: In truth, it has been determined that the majority of injuries with rear impact crashes occur at or below a change in velocity of 10 mph. With frontal crashes, these criteria do not consider seat belt usage. Statistics show that 50% of serious injuries occur with a change in velocity of 16 mph. Also shown is that 50% of fatalities occurred with a change of velocity of 25 mph. Regarding side impacts, it is common that the change in velocity often exceeds the speed of the vehicle. This can cause confusion in determining liability.

Regarding criteria #2: A speed change of 21 mph is necessary to justify a neck injury. This is a very high number and injuries can occur at much lower speeds and changes of speed. It only takes a speed of 29.8 mph for fatality.

Regarding criteria #3: Symptoms onset is commonly delayed with an average of 72 hours, not the 48 hours that is mentioned in the criteria.

Regarding criteria #4: This is not characteristic in the real world when a multitude of factors are actually considered with acceleration/deceleration impact.

Regarding criteria #5: Any pre-accident history of complicating factors or similar symptoms should be separated from the objective and subjective findings after the impact and should not be confused with the clear cut symptoms and conditions caused by the impact.

Regarding criteria #6: Waddell’s signs are often vague and usually subject to interpretation. It is common for them to be used to deny treatment by those unfamiliar with referred pain patterns or who wish to use whatever they can to deny care. Paresthesias and myofascial pain radiations are common with acceleration/deceleration trauma but can be excused away as “non-dermatomal pain patterns” and used to deny care or accuse the patient of malingering.
Whiplash Models:

The search for the causation of pain after acceleration/deceleration impact has taken many decades to unfold. The first Whiplash model was developed in the 1960s by the surgeon, Dr. Ian Macnab. There were no headrests, seat belt restraints or air bags so the kinematics of body motion during impact was dramatically different than it is today. His initial theory was based on rear impact and the movement that caused the most injury was neck hyperextension. He believed neck hyperflexion was not as injurious because the chin hitting the chest limited head forward movement. His studies focusing on hyperextension were done on primates with seat backs parallel to the ground. Dissection after impact showed torn SCMs, esophageal and retropharyngeal space hemorrhages and damage to cervical sympathetic nerves. His hypothesis was severely lacking in explanation for injury to the posterior structures from hyperflexion rebound. However, he is responsible for finding and defining disc avulsions from vertebrae or rim lesion.

Muscle straining theory was another early attempt to describe injuries sustained in an acceleration/deceleration impact. Acute pain was explained as muscle tearing and fascial irritation and was supported by the symptom delay. Common with muscle overuse is the feeling of soreness the next day or so and contributed to the validation of this theory. It was refuted because even though the anterior cervical muscles are the most vulnerable to tear, combining this theory with Macnab’s theory, there is commonly less pain in the anterior muscles than posterior muscles after impact.

Applying the theory of referred pain developed in the 1930s, it was theorized that the facet joints were responsible for the cervical spine pain after trauma. This helped explain why the pain was posterior when the theories available defined the injury to be of the anterior components.

In the 1960s and 1970s Wickstrom et al. did primate whiplash experiments detecting tears to the ALL associated with rim lesions through dissection. Rim lesions can lead to DJD and spurs.

Around the same time, pigs were subjected to whiplash studies where pressure changes within the spinal canal were measured during hyperflexion/hyperextension. Angular accelerations consistent with moderate acceleration/deceleration were measured with peak head acceleration of 25 g, peak displacement of 75 degrees. Capsular bleeding in the cervical ganglia was evident and using dye the authors determined that ganglion nerve cells (mostly between C4-C7) had lost their normal blood nerve barrier. This study hypothesized that pain from a whiplash injury could be from bleeding within nerve cells leading to increased CSF pressure. The study has been used for years despite the physical and biomechanical differences between a pig and human head and neck.

Ellertsson et al. found that whiplash patients had erythrocytes and elevated protein in their CSF. Using the CSF bleeding and excessive pressure theory, in vitro volume of human cadavers was measured during hyperflexion/hyperextension. Volume change corresponded with the space in the canal. Theorized to explain the symptoms experienced from whiplash was that rapid extension/flexion movement of the neck may cause hydraulic overpressure. Questionable,
because recent shearing studies at the onset of neck movement can also result in changes to CSF pressure. Pressure changes as a cause of pain has not been proved.

Primates and pigs prove insufficient for models so studies used human cadavers subjected to harsher conditions than living humans. The disadvantage is that there is no muscle tone or reflex reaction during impact nor is there movement in the abdominal wall and thoracic cavity as would be during respiration for living subjects. Some models had pressurized vascular and nervous systems, some cadavers were heated to normal body temperature. But all of the preparation could not allow for testing closest to real life situations.

Although not the most reliable, tests continued on cadavers into the 1980s. In crash tests averaging 9-15 mph, besides documenting damage to the ALL and ligamentum flavum, facet joint hematoma, capsular tears, C5 chip fractures and disc disruption; it was noted that the vehicle itself could contribute to injury severity. Vehicles made before 1990 can result in catastrophic seat back failure leading to rearward ejection of the occupants into the rear seats. Dynamic crush defines deformation to the vehicle that can be measured. Since the vehicle’s body offers some flexibility, it should be distinguished from residual crush measured after the crash is over. Dynamic crush is usually greater than residual crush as the vehicle’s body rebounds some. These factors need to be considered in determining if an impact justifies an injury.

Section 3: Soft Tissue Injury – Acceleration/Deceleration Industry Guidelines and Models (continued)

Whiplash Models (continued):

As acceleration/deceleration impact studies continued, more attention was paid to the variety of factors affecting results. Position of the patient, angle of the seat, headrest, restraints, rotary factors, speed, direction of impact; all came into consideration. A cadaver study by Deng et al. using sleds coupled to a semi-rigid seat equipped with headrests was performed using high speed biplanar X-ray imaging to record kinematic motion at the crash moment. Facet sprains were recorded. Extension angles for the head and upper cervical spine were less than the lower cervical spine. Rotation of the lower vertebrae increased more rapidly than the upper segments.

The study determined that the kinematics of movement during an impact occurs in the following sequence: compression, tension, shearing force, flexion and extension. Compression occurs immediately after impact in which thoracic kyphosis is decreased. This sets the head into vertical acceleration (vertical ramping). Studies have shown up to 3 inches of additional vertical height
has been reached within the cervical spine during impact. After vertical acceleration, the weight of the torso and seat belt pulls down on the torso rapidly reversing the compression in the spine to tension. The torso is pushed forward in relation to the head (head lag) which adds to the tension. A shearing force transmitted from one vertebra to the one above it - going up one segment at a time straightening the cervical curve causing flexion of the entire neck. This moment is greater at the upper cervical spine.

Over time, studies determined extent of injury with the head turned at the time of rear impact. It was concluded that cervical spine facet joint capsules were more vulnerable to severe injury when rotated. Due to the ligament tautness during head rotation, incidence of tear was dramatically increased from the combination of shear, bending and compression. Only 35 degrees rotation was necessary to produce enough tension to increase injury. Alar ligaments are the most vulnerable. The ipsilateral side of head turn was injured less than the contralateral side consistent with greater tautness of ligaments on the contralateral side. Further studies determined injury can occur to the whole joint capsule under combined extension, shear and compression. Muscular attachments have yet to be studied.

Panjabi et al conducted cadaver sled experiments and reported the s-shaped cervical curvature occurring in the initial phases of an acceleration/deceleration impact. They noted the upper cervical spine flexed during maximum extension of the lower cervical spine. According to the 1997 study during the 41st Stapp Car Crash Conference Proceedings, initially flexion of the upper cervical spine occurs during hyperextension of the lower segments followed immediately by the s-shaped curve. The cervical spine will then flex into a C shape in which the lower cervical spine experiences more flexion than the upper cervical spine.

Rotations of C6-C7 and C7-T1 experienced maximum extension well before full extension of the entire cervical spine indicating vulnerability. Studies measured stretch of individual capsular ligaments and injury increased with the higher level of trauma and lower cervical capsular ligaments were more likely to be strained especially if the head was turned contralaterally. The vertebral artery can stretch up to 6mm.

Crash dummies provided the industry with the most accurate information in the movement of the human frame during rear, front, and side impacts. Dummies placed in various real life positions determined the effects of body position on injury. Also termed anthropometric test dummies (ATD), they were designed with weight, weight distribution, joint flexibility, anatomical proportionality, gender and age specification. The pediatric dummy for airbag deployment studies is called the CRABI or child restraint airbag interaction dummy; and a dummy specifically designed to study side impact is called BioSID. These models were fit with instruments to record loads to the head, thorax, pelvis and lower extremities in higher speed crash tests. Videography is also used to study actual body movement during an impact. Some dummies were fit with jaws to study impact to the TMJ. Although the ATD make the best models for these studies, they are not perfect as there is no thoracic kyphosis so vertical ramping effects on the thoracic spine have yet to be fully appreciated.

Two mathematical models analyze injuries from vehicle crashes; finite element analysis (FEA) and the mathematic dynamic model (MADYMO).
FEA uses a computer to predict stress, strain, thermal effects and areas of potential failure in a two or three dimensional model. It can predict structural failure and combined with MRI and CT allows researchers to understand the effects to the human body and soft tissues. For Yang et al. at Duke University, FEA correlated findings of head lag to the cervical spine during rear end impact. They also confirmed that T1 has an upward movement during compression which led to laxicity of the neck ligaments during impact. This laxicity decreases the neck’s resistance to shearing forces and up to 27% of capsular stretch was predicted. This model is more detailed and can be combined with other equations. It has recently has been used to predict pediatric and liver injury in frontal crashes.

The MADYMO is considered the most accurate mathematical model to evaluate the newest rear impact dummy neck known as BioRID. It takes into account linear displacement, angular displacement and head acceleration in relation to the upper torso at a velocity change of 4.3 mph. This model is better suited to determine the kinematic response of the body.

Since 1996, European auto manufacturers have been studying real world crashes with crash pulse recorders (CPRs), black box of automobiles. A spring mass system triggered by 3g acceleration, results determined that in impacts of vehicle acceleration 6g or less, 40% of subjects sustained no injury. In impacts of 10g or less, 50% sustained short term injury. In impacts where peak acceleration was 15 g, 3 individuals experienced long term disabling injuries. In these cases, change in velocity was not very high. In 44% of the recordings, there were initial symptoms, 7% had long term symptoms. The lowest vehicle acceleration with injuries was 1.7 g and the highest was 14.7 g. The overall impression of these recordings is that approximately 50% of those involved in an impact will suffer injury, 7% will suffer disabling injury.

Human subject crash testing collected data regarding differences between males and females, between restrained and non-restrained subjects, between frontal and rear impact collisions, to observe how subjects interact with the seat back and head restraints of different designs, and to observe crash vectors and a variety of speeds. Images are recorded using high speed video and filmed at different angles. Criteria must meet rigid standards of the Human Subject Committee as to not harm the test subjects. This requires individuals with complicating factors to be at the lowest risk for injury which omits a large section of the population. It also cannot remove the knowledge from the participant that an impact is impending which provides results not representative of real world scenarios. There is no follow-up for long term affects. Although not a perfect method of study, it has provided valuable information for the potential for injury and provides information for variety such as seat back heights, head rest positions, etc.

In the early 1980s, West et al. conducted crash tests using healthy male subjects ages 25-43. Speeds were maximized to 7 mph. Results determined that crashes up to 3mph expose the occupants to forces that can be experienced normally in daily life. In crashes up to 5 mph, they determined there is no injury due to the head restraints. They also determined that low back and TMJ injuries were unlikely in LOSRIC. The opinion of these researchers comparing the impact to that of daily life has no scientific basis. Another downside was that volunteers wore helmets with accelerometers attached. Helmets slip under this type of loading especially if it strikes the head restraint or seat back. This results in artificial findings and unreliable data. The small
sample of healthy of test subjects also make this study limited. Low back and jaw data were not collected. Test subjects were not followed up over time which is not representative of real life situations. Yet, these studies are commonly used to deny treatment.

Section 4: Soft Tissue Injury – Acceleration/Deceleration Kinematic Effects on the Human Frame

Whiplash Models (continued):

Szabo et al. conducted 10mph vehicle crash tests using female subjects. The change in velocity was 5mph and was determined to be the threshold for minor cervical soft tissue injury. The TMJ was not studied but concluded not to be harmed in low speed impact. Concluded was that the TMJ does have some movement during impact but the type was not determined. Szabo also performed studies using heavier padded Volvo head restraints where the distance between the head and restraint 2 inches less and determined there was a decrease in injury with the improved head rest.

McConnell et al performed human crash tests up to 6.8 mph change in velocity. They determined injury can occur with change in velocity of 3.6 mph in subjects with their head turned. With the head straight, injury occurred up to 6.8 mph change in velocity.

Ono et al. performed studies on human subjects focusing on seat back movement. There were no head restraints. The more rigid the seat cushion, the greater the axial compression force to the cervical spine. With softer seat cushions, torso rebounding increased the shearing force to the upper cervical spine resulting in a serious compression injury to the facets during extension. It was also discovered that there is a disadvantage to having the neck in flexion during impact, the loads on the facets and discs were increased. When the neck was in extension at the time of impact loads were decreased.

Siegmund et al. performed human crash tests and determined that the kinematics of the head relative to the C7-T1 joint are more important than those of the C7-T1 joint in relation to the ground. Females experienced greater and earlier acceleration of their head and C7/T1 region mostly due to height difference. Therefore, injury can be experienced at lower changes in velocity due to the differing factors. Peak head acceleration was in the 5 mph range from 6.2-12.0g. These studies determined that 29-38% of individuals in rear end impacts at 2.5 mph and 5 mph experienced mild, transient symptoms mostly of neck and head pain. This puts into question the 5mph change in velocity threshold for injuries that was determined in earlier studies. In fact, injury can occur at very low speeds.

Rear end collision:

Phase I:

Immediately after impact, the target vehicle accelerates. Collision energy dissipates throughout the bumper system and as heat and sound waves. The majority of energy is transferred to the occupants through the seat. Upon impact, the seatback moves into the occupant with the springs and cushions compressing. This compression absorbs and stores the force as potential energy. There is a counter-reaction from the occupant’s low back and pelvis in which the upper seat back is bent rearward. This causes the seatback to be further from the patient’s neck, an increase in distance from the patient’s head to the headrest, seatback stores potential energy, and vertical ramping is increased through a violent interaction with the seatback.

4-1

Lumbar lordosis and thoracic kyphosis flatten as the seatback continues to move forward against the occupant. This increases the vertical height of the torso by inducing a vertical acceleration of the neck and head and compresses the entire spine. A stiffer lumbar seat support can increase lumbar spine injury. Sequential shearing forces move up the spine from the cervicothoracic region as a response to curve straightening which is accentuated by the now greater distance of the seat back.

4-2

Flexion of the upper cervical spine occurs at the same time as hyperextension of the lower cervical spine creating an S curve. Facet imbrication and shearing forces subject the facet capsule to high strain loads.
Once ramping has peaked, the torso restrained from a seat belt drops back into the seat. Since the neck and head are in motion vertically and in extension, cervical spine compression is replaced by tension. The torso then moves forward as the seat continues to move forward. The cervical spine now under stress in an S curve, is dragged forward by the torso which increases the shearing forces and tension. This head lag begins phase II.

In comparing the effects of rear impacts in men and women, heavier males load the seat back more than women. This makes the interaction with the seat back and head restraint more violent leading to greater extension angles. Due to the greater size, the time it takes for males to complete the cycle of changes to the spine is longer thus decreasing the acceleration they experience. With women, the load is less but because there is less weight there is less resistance and women are accelerated sooner and faster. This gives women greater accelerations than men in the same crash set up. Women will experience less head and neck extension but from greater acceleration they tend to experience a more violent Phase III and IV than males. Women are usually more injured in the latter phases of the crash whereas males are injured usually in the earlier phases of the crash.

Phase II:

Head strike on the headrest is the motion that occurs in phase II. Seat and headrest position, type, height of patient, and restraint position and quality all affect head strike. Often, the occupant’s head rides over the top of the headrest accentuating hyperextension. Some poorly designed headrests drop down on impact as the head rides over it further increasing extension of the occupant’s neck. Vertical ramping of the spine can sometimes bring the cervical spine over the top of the headrest and is forced downward accentuating the drop. Once the head has impacted the headrest, the seatback begins to rebound (seat bounce) and moves forward once again. Seat bounce adds to the head forward motion as it rebounds into flexion.
Phase III:

Forward movement begins phase III which can lead to greater injury for larger and taller individuals. Once this phase begins the seatbelt is triggered and pulls back on the individual.

Phase IV:

The function of the seatbelt is to limit forward torso movement. The seatbelt may have been shortened by the brief period of the torso being thrown back into the seat in Phase III. As the torso is restrained, the cervical spine continues to move into flexion with most of the force directed to the cervicothoracic spine.

The above responses occur faster than the stretch reflexes allow muscles to contract in order to prevent ligamentous or muscular tear. Significant injury already occurred prior to the head hitting the headrest. Injury depends on the responsiveness of the seat and headrest. Males are injured more in this phase due to their mass.

Awareness of motion when hit in the rear is a question for researchers and a trap by litigators. It is common for a medico legal team from a third party payer to ask the patient if their head hit the head restraint or if their head went forward or backward first. In many cases the answer is “no” or “I don’t know”. Studies using cameras and questions asked of post-crash test subjects indicated that in fact, movements are so fast test subjects are unaware if their head hit the restraint or went forward or backward first. Also, observed that upon impact, the eyes blink shut as a reflex. This adds to the difficulty of subjects knowing if they hit the head rest or which direction their body moved first. Although this is common, it is not commonly known. It is used to cast doubt on the patient when questioned by a legal expert who will make the case since the patient did not recall, it could not have been that bad of an impact.

*Front end collision*:

Even though front end collisions are considered the most common, injuries seem less severe. Approximately 1/3 of injury and disability found with acceleration/deceleration impact occur from a front end trauma. The key difference is patient awareness of the impending impact and they can brace with their feet against the toe pan. They can also brace their hands on the steering wheel which serves to focus the spinal bending to the lower cervical spine. Seat belt configurations, bracing and air bags are largely responsible. A front impact forces the patient’s neck into flexion with a rebound into extension limited by the restraint system.
In studies comparing injuries sustained by females, drivers tend to be injured more than passengers. This could be due to the steering wheel position and that women clutch the steering wheel to brace for impact. This can make the thoracic spine rigid and decreases the bending moment in the lower cervical spine thus increasing injury. Size and position of the steering wheel and head rest could account for the increase in injury of females over males.

Section 5: Soft Tissue Injury – Acceleration/Deceleration Kinematic Effects on the Human Frame (continued)

*Front end collision* (continued):

Studies determined the effectiveness of seat belts and airbags on front end crashes. At a change of velocity of 9 mph and a force of 5 g, risk for injury was 20%. It rose to 40% with a change in velocity of 25 mph and a force of 9g. This 5 fold change in velocity increased the risk 2 fold. At higher speeds and acceleration levels, injury risk decreased. It has been hypothesized that air bags and seat belt restraint systems function differently at higher speeds. Also, various other more severe injuries may decrease the reporting of neck injury. A recent study in Italy focusing on high speed impacts determined that the neck is injured severely and is the most prominent symptom in hospitalized and unhospitalized persons.

Recent studies using MADYMO divided crashes into three time components and reviewed the information affecting risk for short and long term neck injury. Time intervals were determined by change in velocity at 1-33 msec., 34-66 msecs., and 67-100 msecs. It was correlated that if the largest velocity change occurs in the 34-66 msec. segment and a smaller change occurs in the 67-100 msec. period there will be the highest risk for long term injury. Breaking the impact into sections of time and comparing the changes in velocity provided greater injury than if the overall change in velocity was greater for the entire 100 msec. This confirms that the type of impact is an important factor and that the bulk of the injury occurs at 34-66 msec. During this time, the neck and head loading was affected the greatest by the seat belt restraint.

*Side collisions:*

Injury from side collisions is about the same as for front end collisions as the level of awareness is similar. Restraint systems and seats are not designed for this type of impact. Most studies evaluate impacts into the occupant compartment (or safety cage). Few studies on the cervical spine have been done to determine injury outside of the safety cage or where severe injuries to other regions of the body are also reported.
During a side collision, there is a greater risk of head impact on the side door creating brain injury, and cervical spine injury in a lateral flexion motion. There is a risk of other occupants colliding with each other especially if they are not restrained. Coupled with lateral flexion is forward movement of the cervical spine since most vehicles that are hit on the side are also traveling forward. Rotary effects are seen as the vehicle may spin. Low back injury is more common with side impact collisions. Foreman and Croft hypothesized that the unrestrained lateral torso moves while the pelvis is stabilized from the lap belt concentrating the bending moment to the lumbosacral region. Also, the impact is slower because the softer sheet metal of the vehicle deforms more easily. All of this creates a more complicated injury.

Not all injuries are pure rear, front or side impacts but can incorporate several directions of force. One of the most serious impacts is a side impact when two vehicles are traveling in the same relative direction which leads to a spin. Any rotational component creates complications because the rotational structures of the cervical spine are weak and limited to the upper region. Rupture of the alar ligament can occur with rotation and lead to dislocation of the atlas and spinal cord compression from the dens. For this reason, it is vital to collect all data of the mechanism of injury, direction the vehicles were traveling, position of the patient, speed, type of vehicle and effectiveness of the restraint.

**Rear vs. front end collisions:**

In evaluating the most recent and cumulative studies of motor vehicle crashes, statistics show that there is a 35% risk of injury with rear end collisions, a 24% chance of injury in frontal crashes and a 26% chance with side impact crashes. This reflects that rear end collisions are more common than front or side impact crashes. In fact, risk and vector of impact is greater with front end collisions.

The Spine Research Institute of San Diego performed a series of human subject crash tests to compare rear and front end collisions. Three crashes were evaluated for each direction, each with different volunteers. The first crash was low speed from 2.5 -4.5 closing velocity, the second and third at closing velocities were 9.9 mph. For the rear end impact, the first and second crashes were done with the volunteers distracted, the 3rd crash was the volunteers braced. In the frontal end impact, volunteers were aware of all three crashes and were told to brace. Variables were removed such as staggering the order of the crashes and being consistent with the size of the target and bullet vehicles.
Studies determined that the resultant accelerations were greater in the rear impact crash tests. Subjects experienced rearward retraction and extension followed by a forward protraction and flexion. The seat back is almost immediately ramped up. In frontal crashes, the occupant first glides into the seat belt webbing, then loads the seat with compression creating less movement of the cervical spine. For the frontal crash, there was forward movement of the head limited by the seat belt.

**Factor Analysis:**

Factor analysis is a statistical procedure that explores a situation allowing for subjectivity and judgment. This method is used to assess acceleration/deceleration impact reviewing the deficit that exists in documenting and confirming soft tissue injury, pain levels, complicating factors, etc.

**Vehicular and external factors:**

**Relative Mass:**

Sizes of the vehicles in an acceleration/deceleration impact influence the severity of injury. A smaller target vehicle increases the chance and severity of injury; a larger bullet vehicle also increases the severity of injury. Mass, has a double effect on risk because as a target vehicle, a small vehicle undergoes a larger change in velocity whereas a larger vehicle undergoes a lower change in velocity.

Kinetic energy determines the risk for fatality in a motor vehicle crash. The equation takes into consideration half the mass times velocity squared. As the speed of a vehicle doubles, kinetic energy quadruples increasing a risk greater than the linear rate of speed. In a smaller vehicle, injury risk at higher speeds is greater. Studies show more severe injuries and higher fatality rate with smaller vehicles. The fatality risk is greater for vehicles built from 1990 to current than in vehicles built from 1984 -1989. Those built from 1966 - 1980 vary from less risk to greater risk. Many factors determine this; seat restraint, air bags, flexibility of bumper mechanisms and cage metals. The most recent trend is to lighten the weight of the vehicles to assure less fuel use. This increases injury risk. Most recently, use of ultra light steel has provided for safety as well as fuel efficiency.

**Stiffness:**

Larger vehicles have more space around the engine block, trunk and passenger compartments for engine and vehicle sections to move during impact. The force is absorbed by the collapsing space and less force is applied on the passenger compartment during a crash. With smaller vehicles, there is less space so the vehicles are designed with stiffer compartments for protection. Acceleration to the driver in a smaller vehicle striking a rigid barrier is higher than the acceleration in a larger vehicle with softer metals striking the same barrier. The greater the stiffness, the greater the acceleration to the passenger. Unfortunately, newer vehicles tend to have stiffer rear bumpers which increase the risk of injury at lower speeds but decrease the risk of injury at higher speeds.
Different vehicles deform to the load of an impact differently. **Elasticity** is when the vehicle body springs back to its original form after a crash. **Plasticity** is when the vehicle body does not spring back to its original form after crash. “Spring back” of vehicle metals is not necessarily related to vehicle size even though smaller vehicles tend to be stiffer. In low speed crashes with little property damage the vehicles were more elastic. A lack of damage does not represent a lack of impact or injury but is more a reflection of materials that compose the vehicle.

There is an inverse relationship between vehicle damage and occupant risk in LOSRIC. In a recent study by Chapline et al., 38% of vehicles tested had what was considered “no damage”, however, 38% of females and 19% of males were injured. This included crashes of a variety of speeds. This study is a fair representation that speed is not necessarily the greatest factor in the advent of whether someone will be injured and that stiffness and elasticity must be considered. There was a 40% chance of injury at 25 mph and a 40% risk at 6 mph. This study is in direct conflict with the common argument that low speed crashes do not cause injury.

Hour 2  
Section 6: Soft Tissue Injury – Acceleration/Deceleration Kinematic Factors as Vehicle Variables

**Factor Analysis** (continued):

**Vehicular and external factors** (continued):

**Variation in impact direction:**

Ascertain body position from the patient at the time of impact as well as what positions the vehicles were in and what part of the vehicle was struck. Have an idea of the vectors of force placed upon the vehicle or which direction the vehicle moved when struck. For instance, the vehicle could have spun upon a side impact that was not centered to the vehicle. In this case, the patient’s neck and body also spun and that is important to know. Whether the patient was looking in the rear view mirror or changing a radio station or turning their head for an instance to speak to a passenger; all of these factors will be important in determining the type of forces that were placed on the patient’s body. Injuries from a side or front or rear impact will be complicated by a turned head.
Studies have been done with oblique rear end impacts where the striking zone was off center causing the vehicle to alter its course to a spin. In this type of strike, including linear and rotational acceleration there will always be asymmetric facet loading. This injury is common at intersections as the target vehicle is in the process of turning.

Secondary collisions:

This is the scenario where more than one force affects the patient’s body. This can occur when a target vehicle makes secondary contact with another vehicle or obstacle as a result of the initial strike; when the patient’s body strikes the inside of the vehicle; when internal organs strike the inside of the patient such as the heart, abdominal organs and brain.
With rear impacts, in second impact with another vehicle stopped directly in front, the additional deceleration can increase the Phase IV forward rebound phase and increase injury.

Be wary of contact with the steering wheel, dashboard, windshield, door, headrest, airbag, brake pedal, floor board, and/or roof. Be wary of any item that broke away from its secured place and flung around the vehicle that may have hit the patient such GPS systems. A secondary collision with the windshield, could increase the hyperextension and/or compression of the cervical spine. Rim lesions, anterior longitudinal ligament tears and compression fractures can result. Any impact that includes the head should be assessed for brain injury.

With side impacts, lateral bending of the cervical spine allows for more diffuse distribution of forces on the cervical and thoracic spines. But if an impact with the door panel prevents the thoracic spine from absorbing force, the cervical spine will absorb all of the energy and results in increased forward bending moment increasing injury.

**Plastic vs. elastic crash behavior:**

The wide variation of bumper systems and sizes of vehicles can allow for a broad difference in damage between the vehicles in similar crashes. This factor is most important for LOSRIC because there can be little or no damage to the vehicle however the patient suffers from injury. Understanding this “gotcha phenomena” can assist you and your patient a great deal when documenting and justifying injury that has occurred with LOSRIC.

A formula has been developed to understand the energy transferred from the striking vehicle to the target vehicle; ratio of pre impact and post impact speeds. The closer the difference or smaller ratio, the closer energy is toward 0. Closer to 0, the collision is plastic - the vehicular body’s deformation and energy are not restored. If closer to 1, the collision is elastic - the vehicle body’s deformation and energy are restored. The range in between determines the flexibility in the vehicle’s body and its ability to absorb energy and rebound. There will be more damage to a plastic vehicle than elastic. However, there will be more injury to the occupant in an elastic vehicle as the collision energy is transferred to the occupant increasing injury.

The only difference in this equation is that very low impacts have a low speed differences between the striking and target vehicles making the energy value closer toward 1 and elasticity. At higher speed differences, the equation favors lower differences in energy transfer and the number may be closer to 0 and plasticity. In either case, this may not adequately represent the ability of the vehicle’s body to alter upon impact. However, these numbers are guides and usually reliable.

**Restraint systems:**

Seat belts and air bags save lives. Seat restraints reduce the risk of fatal injury by 50%. However, in LOSRIC, risk for spinal injuries increases with restraints. For most vehicles, the shoulder and lap belt are one unit and used simultaneously. Usage is about 60% in states that have seat belt laws. With vehicles possessing separate lap belts and automatic shoulder harnesses, use of the
harness is greater than that of the lap belt. Injuries differ if the lap belt is not used to anchor the pelvis. The loads to the chest and abdomen increase and rib fractures can be seen. When the shoulder harness is used exclusively, cervical dislocation and occupant ejection can occur. When the lap belt is used exclusively, injury is greater in the lumbar spine.

Neck injuries can be accentuated by seat belt shoulder and lap harness combination. Abrupt restraint of the shoulder harness on the torso while decelerating accentuates forward moment on the cervicothoracic spine. This creates a more exaggerated flexion injury. Another problem occurs when the lap belt harness may not retract fast enough to protect the patient from exaggerated force into the seat back or from being thrust forward.

Several unintended events may happen during an impact. The patient may receive injury to their cervical vascular structures from acute contact with the shoulder harness. There may be abdominal, pelvis or lumbar injuries from slipping below the lap belt. The head rest may be at the wrong height, the patient may be slumped, the shoulder harness may be anchored too high on the door, seat cushions may be too soft to absorb the blow, or they may place the lap belt too high (above the ASIS). This is especially dangerous in pregnant women. Another problem occurs when the patient was seated forward and slammed on the brake. If this occurs, the seat restraint has a greater slack to recover and the patient has a greater distance between their back and the seat back in Phase I. All of these situations must be ascertained from the patient to best understand the forces on their body at the time of impact.

Seat belt injury is more common in children because the standards conform to an average male in the 50% range. The most serious injuries for children caused by seat belts are trauma to the lumbar spine and spinal cord injury. This is extremely dangerous because pediatric spinal trauma may be missed since it is difficult to detect with ordinary testing methods. Internal injuries are also noted. There may be a classic “seat belt sign” where the seat belt leaves a bruise mark across the child’s chest and abdomen. If this is evident or the patient is complaining of symptoms of the extremities, then MRI for the cervical spine and internal organ evaluation would be warranted.

Seat backs:

Rigid or pliable seat backs both fail to absorb energy during acceleration/deceleration impact. Research shows that the best type of seat back is somewhere in the middle. More recently, car makers are moving toward more rigid seat backs and cervical spinal injuries with crashes has increased.

Severy et al. determined that in 20 mph rear impacts, the seat back and torso move forward from the seat back before the occupant’s head had fully extended. This increases shear through the neck and is called the “diving board effect”. It is possible incline angle of the seat can be a contributing factor. Ono and Kanno found that when the seatback was reclined, forces on the cervical spine increased by 30%. However, when the seatback is more forward, shearing on the cervical spine is exaggerated. When the seatback fails, there is less injury to the occupant. The best position for the seatback is mid range between rigid and elastic and reclines somewhere in the middle.
**Brakes:**

In some impacts brakes are applied in others they are not. Studies show there is greater acceleration and higher risk for secondary collision when the brakes are not applied. With a rear end collision and brakes are applied, the impact exceeds the pressure the driver is applying to the brakes and brake pressure is lost, similar to when there are no brakes. To compound the problem, the occupant is thrown forward in Phases III and IV when the foot reestablishes contact with the brakes creating a secondary collision of the foot with the brake pedal. This is most likely when the occupant is unaware of the impending crash.

Section 7: Soft Tissue Injury – Acceleration/Deceleration Kinematic Factors as Human Variables

**Factor Analysis** (continued):

**Human factors:**

**Out of position occupant:**

Ideally, the occupant should be upright, facing forward and restrained. Crash tests with crash dummies only provide information on optimal seating. Occupants in the rear seats of a vehicle are injured less than those in the front. Many factors such as lack of head restraints can accentuate cervical extension during impact, most rear passengers are children and do not reach the head restraint. Passengers may also not use the shoulder harness or seat belts and although dangerous in high speed accidents where fatality risk is high, in lower speed impacts, injuries that occur from seat belts or rigid seat backs are avoided in the rear of the vehicle.

**Stature:**

The most important factor in stature is the height of the head rest. Occupants usually sit with the head rest too low which increases cervical extension injury. Taller individuals (6’2” or over) are at greater risk for cervical spine injury in LOSRIC. This also compounded by the probability that they have reclined their seat back to make room for their legs which increases injury. Shorter individuals (5’ or less) will have less cervical spine injury as their head is lower on the head rest, but they risk greater injury from the seat belt riding too high and compressing structures of the neck.

**Body mass:**

Persons with larger body masses are at less risk of injury as there is greater force into the seat back and head restraint decreasing acceleration. However, there may be greater extension if the head rest is too low. Persons with lower body masses may experience greater forward movement from less force into the seat back and head rest. This causes greater flexion and increases bending moments.
**Gender:**

Women are more likely to be injured in an impact from smaller mass. However, their bodies usually have less mass for their size and more vulnerable to the effects of a crash.

**Age:**

The most common age for injury from acceleration/deceleration impact is 20-40. This age group has the highest number of drivers. Incidence of injury from crashes increases after 30 most likely from decreased ROM, slower reflexes, less muscle strength and slower healing times. There is also the probability of complicating factors. Children have 60% risk of injury than adults.

**Strength:**

Greater muscle tone which accompanies greater strength can be a limiting factor in injury as the muscles will better protect the facets. However, greater muscle mass is more susceptible to muscle strain.

**Awareness and bracing:**

Being aware of an impending impact can decrease injury. This is most common with front end impacts but studies with rear impacts demonstrated that those individuals tend to press themselves deeper into the seat bracing for impact. Their injuries are less severe than those not aware of the potential crash. However, those that tend to lean forward to brace themselves have greater injury.

**Auto crash reconstruction:**

A common practice after acceleration/deceleration impact is an auto crash reconstruction. Its purpose is to determine what occurred just before and just after impact in order to assign blame. Insurance agents use this information to minimize liability. Here the patient and their physicians are assumed to be telling falsehoods about the events and injuries. Parameters used are distance of skid marks, which wheels produced those marks, distances between vehicles or between vehicle and obstacle, coefficient of friction of a particular road surface, rate of acceleration of the vehicle as it begins to move forward from a stop, etc. It helps make determinations of who was speeding, who failed to stop at a signal, etc. Most of this information is guess work gleaned from witnesses, occupants, police reports, etc. This information is then plugged into specific calculations which determine speed, distance, spin and other factors in a crash. Many variables are not included in these calculations. Their results can be inaccurate, incomplete and vague.

Regarding LOSRIC, the following methods are used to determine events that occurred in a crash for the auto crash reconstruction:
Conservation of Momentum:

Momentum or quantity of motion is expressed by “p” (the product of mass x velocity). The law of conservation of momentum states that for any group of objects that act on one another, the total momentum before the impact must equal the total momentum after the event. In actual crash situations, most of the energy is conserved but some is dissipated as metal crushing, sound waves, heat, microfractures and transfer to pavement. In actuality with highly plastic vehicles, the loss of energy can be up to 50%. Also, there are factors which are not in play for the equations such as angulations of the vehicle with the road, friction factors of the tires and the road, and the effectiveness of the vehicle’s suspension system which varies considerably between vehicles. Factors not considered and vulnerability of the equations brings the accuracy of this method into question.

Computer Analysis:

Two means computer programs analyze a situation are simulation programs or data programs. Simulation programs tend to determine the hypothetical situation. Data programs tend to determine the actual situation based on facts gathered at the scene and eyewitness statements. These programs go under the name of CRASH, CRASHEX or EDCRASH. They are based on the 1970 era model that the depth of the indentation of the target vehicle is directly related to the speed of the bullet vehicle. Most programs were designed with crashes of 30 mph. In the real world, the variables such as speed and plasticity are so varied they do not fit in the formula created by the computer. This is most evident in LOSRIC where most crashes do not result in a crush and indentation of bumpers. These models have a very narrow reliability but are used in a wide range of scenarios for which they were not designed.

Momentum, Energy and Restitution Method:

This method was devised by Siegmund et al. and Szabo et al. and is based on determining speed changed from barrier data combined with momentum, energy and restitution. Calculations take into account the elasticity and/or plasticity of the vehicles or obstacles, vehicle weight, velocity change and elasticity (standardized figures are available for specific vehicles) to determine the closing speed (how fast the striking vehicle was going when it hit). Once the closing speed is determined, the target vehicle’s change in velocity can be calculated. Calculations do not consider that the tire contact with the ground and forces absorbed by the vehicle’s suspension can have a distinct impact on the vehicle’s mass which would change the data in the formula. Also, effects of braking need to be considered. This was done by Siegmund in his formula but he used locked brakes or parking brakes to determine his calculations. The use of a locked brake in an impact cannot adequately define the effects of human braking during a crash.

Change in velocity is a favorite value for accident evaluations. It is however, incomplete. So many factors contribute to injury that any report or explanation of events should be discussed in detail. For instance, the bumper standards for elasticity in crashes are values that have been precalculated. In fact, these standards are not always accurate. Some vehicles with specific pliability standards can demonstrate little or no damage in real world situations where the change
in velocity or speeds were up to twice what the standards state the vehicle’s bumper can handle before it dents.

Witness statements are also inaccurate as events during a crash can be unclear. Many times, the advent of bruising, for instance, on the arm is the only indication the patient has that they hit the car door with their shoulder. Hitting the head against the restraint can go unnoticed immediately after the crash. Witnesses stating speeds of the vehicles cannot be reliable for calculations because they could be off by enough to change the type of accident from LOSRIC to high speed.

Photographs of the vehicle are a common practice but be wary of incomplete studies. Also, know that only one vehicle may be photographed which provides incomplete information. Most photographs do not take into account the underside of the vehicle where bumper brackets, isolators, frame stability, etc are not evaluated but can have a tremendous effect on understanding the full scope of the impact.

**Neck Injury Criteria:**

The newest calculations are neck injury criteria. They consider the relative velocity and horizontal acceleration between the top and bottom of the spine. It is a closer evaluation of the moment where the head is retracted during the 1st 100 milliseconds of the crash, the head lag phase. These criteria are in flux and research is changing the parameters but serious studies are underway to understand forces on the cervical spine during this process. Siegmund et al. determined that the peak linear head acceleration is not sufficient by itself to predict injury. No one factor is sufficient to predict injury.

Section 8: Soft Tissue Injury – Acceleration/Deceleration Risk Evaluation

*Factor Analysis* (continued):

**Current Research:**

Seat design in Volvo and Saab has taken the absorption of acceleration into consideration by allowing for a more limited controlled collapse of the seat back. However, the majority of car makers focus on comfort for seat design not injury prevention.

Bumper design in which the bumper collapses upon impact has been considered by some car makers but they are resistant to making anything that is costly to replace once damaged. It would however, decrease the forces transferred to the passenger compartment if it is more elastic.

Black boxes have been installed on Volvos and Saabs. These provide more detailed real life information that can assist with future vehicle design.

Intelligent vehicle highway systems are being tested on road surfaces such as a 17 mile stretch of highway in San Diego. The preventative measures are passive crash avoidance systems that possess forward and rearward scanning sensors to alert drivers to threats based on computer...
calculations of closing velocities of other vehicles. There is the potential of these systems to take control of the vehicles to help avoid crashes. Cars that drive themselves are also being tested.

Whatever the newest technology may present, car makers and researchers are constantly trying to understand crash dynamics and injury prevention. Practitioners need to understand how to advocate for their patient in a complicated and mostly biased insurance system designed to minimize the truth. Information provided will help you understand the evidence being used by third party payers. It will also assist you in providing the third party payers with research and evidence that may be more accurate than that of their own. Advocating and documenting accurate reliable information for patients will aid in their fight for justifiable care.

**Incidence:**

Approximately 3 million motor vehicle accidents occur each year in the United States. Rear impacts account for 25% of all auto accidents but 40% of injuries sustained in those accidents. Approximately 60% of motor vehicle accidents that produce minor neck injuries are responsible for permanent impairment claims. In Sweden, studies found cervical strain from motor vehicle accidents account for 55% of injuries and 82% of sick leave within 2 ½ years after injury. Rear impacts account for 64% of sick leave costing about $1.4-2.5 million per year.

In British Columbia, $600 million was spent for whiplash claims in 1996. In this study, whiplash injuries accounted for more than 70% of all bodily injury claims. In the U.S., acceleration/deceleration claims value $19 billion per year. This figure does not consider lost productivity and other social costs. Recently, the cost of motor vehicle crashes in the US was measured as a percentage of gross national product including the value of loss of quality of life. Elvik reported this portion to be about $5.7 percent of GNP or about $332 billion annually.

**Prevalence:**

Studies to determine how long symptoms last after impact vary greatly. Outcome/prognostic studies indicate that 12% - 100% of whiplash sufferers continue to be symptomatic years after the injury. Most of these studies followed patients for 6 months to 2 years. Rear impact injuries carry a worse prognosis than side or frontal impact injuries. About 35% - 50% of patients had not recovered completely at follow-up with 10% rating their problems as disabling or severe. Arthur C. Croft, DC, determined that with 2 million injuries per year and 25% of nonresolution of symptoms, after 25 years of whiplash trauma, the prevalence of chronic pain in the US would be 6.7% of the population. Also, assuming a 50% nonresolution of symptoms after 25 years of whiplash trauma, the prevalence of chronic pain in the US would be 9.6% of the population.

In a case control study performed by Croft et al., 45% of individuals with chronic pain reported being in a motor vehicle accident. Using the figure of 45% and combining with the prevalence of chronic neck pain in the US, the estimate of late effects of whiplash affect 6.2% of the population. Findings in literature increase the probability closer to 9.6%. These findings indicate a large percentage of the population are added to the disability rosters each year and a high percentage of individuals suffer from chronic headaches after motor vehicle accident.
Comorbidities after whiplash injury also include allergies, breathing disorders, hypertension, cardiovascular disorders, digestive disorder and lower back pain. These conditions have developed at twice the normal rate for those individuals who experienced cervical spine injury after acceleration/deceleration impact.

**Risk:**

Auto crash reconstructionists are hired by insurance companies to compile information similar to the crash that was experienced by your patient. If they determine crash study statistics show there should be no injury because a crash dummy was not showing evidence of injury they will use this information to deny care or that injury ever occurred. There are so many variables that must be taken into account regarding the results of an impact that their information would be vague at best. Understanding the specifics and some of what these risk analysts utilize in their opinions will assist you and your patient in proving their injuries have indeed occurred and are directly related to the crash.

It is common for auto crash reconstructionists to use unreliable witness accounts, photos to determine speed and acceleration figures and a variety of “tricks “ to minimize liability. Crash studies may be reliable in determining insight into occupant kinematics under crash conditions but they cannot be used to assess risk or probability of injury for a particular case. This is, however, what is done. Study flaws have been identified by Croft et al. are the use of a nonrepresentative study sample, inadequate study size, nonrepresentative crash conditions, inappropriate study design, unsupported conclusions, unsubstantiated unreferenced claims, misquoted literature, improper use of terminology and misleading illustrations.

It is common to use the excuse of a low velocity crash (under 10mph) to deny that an injury has taken place. In fact, occupants of these crash vehicles can experience high head acceleration of 7-14 g and at times violent kinematic responses. These do not take into account complicating factors.

It is also common to correlate property damage to injury potential in which if there was no or little damage to the car, there would be no injury. Within the range of low speed crashes, this correlation cannot be made in studies due to variables such as seat backs, head rests, change in velocity, bumper crush, etc. In truth about 25-50% of occupants are injured when all types of impact are taken into consideration. A Japanese study has shown about 44% of occupant injury from rear impact. About 22% of occupants suffer neck injuries from a front end impact. Croft et al. has determined that between 30 and 60% of car occupants exposed to LOSRIC of 5-10 mph change in velocity sustain some injury ranging from short lived to disability.

Croft has compiled two tables to determine risk of injury from acceleration/deceleration impact in order of severity; *Risk for Acute Injury* and *Risk for Late Whiplash*.

**Risk for Acute Injury**

1. Female gender
2. History of neck injury
3. Poor head restraint geometry/tall occupant
4. Rear versus other vector impacts
5. Use of seat belts/shoulder harness
6. Body mass index/head neck index
7. Out of position occupant
8. Nonfailure of seat back
9. Having the head turned at impact
10. Nonawareness of impending impact
11. Increasing age
12. Front versus rear seat position
13. Impact by vehicle of greater mass
14. Crash speed under 10 mph

**Risk for Late Whiplash**
1. Female gender
2. Body mass index in females only
3. Immediate/early onset of symptoms and/or severe initial symptoms
4. Ligamentous instability
5. Initial back pain
6. Greater subjective cognitive impairment
7. Greater number of initial symptoms
8. Use of seat belt shoulder harness
9. Initial physical findings of limited range of motion
10. Initial neurological symptoms
11. Initial loss of range of motion
12. Past history of neck pain or headache
13. Initial degenerative changes seen on radiographs
14. Loss or reversal of cervical lordosis
15. Increasing age
16. Front seat position

Neck pain after whiplash can continue well after injury. This is caused by stretched and torn ligaments which allow for abnormal range of motion and can be an ongoing source of pain. Muscles may compensate for laxity by contracting which also contributes to pain. Muscles may heal with scar tissue perpetuating pain. Myofascitis and trigger points can develop and adhesions and scar tissue can lead to nerve impingement or elongation. All of these factors must be taken into consideration when determining risk of injury from impact.

Section 9: Normal Biomechanics of the Cervical Spine

*Cervical Spine:*

For a more detailed understanding of the biomechanics of the cervical spine please refer to Soft Tissue Injury 106- The Cervical Spine.
Structural and Functional Anatomy:

**Bones:**

There are two sections to the cervical column; *superior segment* (C1-2) and *inferior segment* (C3-C7). Each cervical vertebra has two sections; anterior vertebral body and posterior vertebral arch. The body, a supporting structure, absorbs 75% of compressive forces, the arch 25%. Compression strengths of the cervical bodies are ¼ that of lumbar vertebral bodies leaving the endplates and discs vulnerable.

**Joints:**

The cervical spine is dynamic with up to 8 joints per articulation allowing for rotation, lateral flexion, flexion and extension. Rotation and lateral flexion are coupled. Excessive translation is common with acceleration/deceleration injury.

Ranges of motion vary due to age and gender. According to Ferlic, D, *Johns Hopkins Hospital Bulletin*, there is greater range in females than males and it decreases with age.

The ranges of motion for the cervical spine in the full plane of movement are as follows:

- Flexion/extension 127 degrees +/- 19.5
- Lateral flexion (left and right) 73 degrees +/- 15.6
- Rotation (left and right) 142 degrees +/- 17.1

Synovial facet joints and uncovertebral joints are bilateral with discs between them excluding the atlanto-occipital, atlanto-axial articulation, and atlanto-odontoid articulations. C1 has 5 articular surfaces, C2 has 8, C3-C7 each have 7. Annular disc fibers insert into the bony rim of the vertebral body. Nuclei contact the articular hyaline plates associated with the body endplates.

Together with the occiput, C2-C6 comprise movements in three axes perpendicular to one another; flexion/extension, lateral flexion and rotation (at the atlanto-axial joint). C7/T1 allows for flexion/extension and lateral flexion with rotation. The superior and inferior segments function almost as separate units due to the variance in the planes of motion but respond to one another. The upper cervical spine allows for 50 percent of rotation to turn the neck by turning the
head, mostly at C1-C2. Further rotation occurs with lateral flexion coupled in the lower cervical spine. During this movement, flexion is an unwanted response of the suboccipital spine. This is compensated for by suboccipital extension and lateral flexion in the opposite direction.

**Superior Segments:**

**Atlanto-occipital Joint:**

For *flexion* and *extension*, there is a convex surface on the atlas and concave surface on the occipital condyle. The head rests on the atlas lateral masses angled obliquely, anteriorly and medially allowing for motion in three planes;

- **Flexion/extension**: 13 degrees
- **Lateral flexion**: 8 degrees
- **Rotation**: 0 degrees

*Flexion* creates a receding of the occipital condyles on the atlas and a moving away of the occipital bone from the atlas posterior arch. It is limited by the contact between the dens and anterior foramen magnum, and tension of the articular capsular and nuchal ligaments.
9-3

*Extension* demonstrates an anterior slide of the occipital condyles on the atlas approximating the occipital bone to the posterior arch, and the atlas to the axis. Extension is limited by these bony abutments. Hyperextension can fracture the atlas posterior arch.

9-4

*Lateral flexion* exhibits a slipping of the occipital condyles on the opposite side; therefore, left lateral flexion exhibits a right lateral occipital condyle shift and vice versa.

9-5

*Rotation* is a result of movement at the atlanto-axial joint. When the head rotates to the left, the right occipital condyle moves anteriorly. The left atlanto-occipital ligament wraps itself around the odontoid becoming taught forcing the right occiput condyle to laterally shift to the left. A linear displacement of the occiput to the left occurs with lateral flexion to the right. The central
pivot point during rotation is now lateral and slightly right of the midline affecting the anatomical vertical axis of the brain stem.

Atlanto-axial Joint:

Movement occurs at three joints; two atlanto-axial lateral masses and atlanto-odontoid joint. Anteriorly, on the dens is a facet for the atlas anterior arch. Posteriorly, there is a cartilage lined gutter for the transverse ligament.

- **Flexion/extension**: 10 degrees
- **Lateral flexion**: 0
- **Rotation**: 47 degrees

The atlanto-axial joint presents with concave surfaces on the atlas and axis allowing for flexion in a rolling and sliding motion. The axis is stabilized by the transverse ligament. The dens is shaped with a slight curvature for tracking.

For rotation, the dens is a stationary pivot and 50% of cervical rotation occurs at this joint. The transverse ligament becomes contracted on one side and relaxed on the other; one lateral mass of the atlas moves anteriorly and the other posteriorly. As the one side moves anteriorly, it drops inferiorly by 2-3 mm creating a spiral motion. Rotation is coupled with lateral flexion in a ratio of 2:3 or 2 degrees of rotation for every 3 degrees of lateral flexion. In the lower cervical spine this coupling ratio is 1:7.5. When the cervical spine laterally flexes coupled with rotation, the atlanto-axial joint compensates with a counter-balance of lateral flexion to the opposite side.
**Inferior Segments:**

**Intervertebral Discs:**

Discs of the cervical spine are higher than thoracic and lumbar discs allowing for greater range of motion. Their nuclei pulposi have a higher capacity to imbibe with fluid for better shock absorption and load distribution. However, with degeneration, this ability diminishes and extension is mostly affected as laxity allows for increased translation. During the day, constant loading causes water to seep into the vertebral bodies decreasing the height of the disc. At rest, this fluid returns to the nucleus allowing it to resume its preloaded state.

**Lateral Masses:**

<table>
<thead>
<tr>
<th></th>
<th>flexion/extension</th>
<th>lateral flexion</th>
<th>rotation</th>
</tr>
</thead>
<tbody>
<tr>
<td>C2-C3</td>
<td>5-23 degrees</td>
<td>11-20 degrees</td>
<td>6-28 degrees</td>
</tr>
<tr>
<td>C3-C4</td>
<td>7-38 “</td>
<td>9-15 “</td>
<td>10-28 “</td>
</tr>
<tr>
<td>C4-C5</td>
<td>8-39 “</td>
<td>0-16 “</td>
<td>10-26 “</td>
</tr>
<tr>
<td>C5-C6</td>
<td>4-34 “</td>
<td>0-16 “</td>
<td>8-34 “</td>
</tr>
<tr>
<td>C6-C7</td>
<td>1-29 “</td>
<td>0-17 “</td>
<td>6-15 “</td>
</tr>
<tr>
<td>C7-T1</td>
<td>4-17 “</td>
<td>0-17 “</td>
<td>5-13 “</td>
</tr>
</tbody>
</table>

*Flexion* is accomplished by the superior body tilting and translating anteriorly. This compresses the intervertebral space anteriorly driving the nucleus posteriorly stretching the posterior annulus fibers. Flexion is limited by the PLL, posterior capsule, ligamentum flavum, ligamentum nuchae and posterior cervical ligament.
Extension is accomplished by the superior body tilting and translating posteriorly. This compresses the intervertebral space posteriorly and the nucleus is driven anteriorly stretching the anterior annulus. Extension is limited by the ALL, the abutment of the superior articular process on the transverse process of the upper vertebra, and the impact of the posterior arches upon one another.

Rotation is always coupled with lateral flexion in the inferior cervical spine. Inconsistency in angle obliqueness exists throughout the cervical spine due to cervical lordosis. This leads to differences in ranges of motion between the vertebral levels. Inferiorly, the angle allows for more rotation than lateral flexion whereas higher up, rotation and lateral flexion are equal.

The greater the degree of flexion/extension at C5-7 can account for a greater incidence of injuries in this region as well as the development of spondylosis. Within 7 years of acceleration/deceleration injury there is a 39% incidence of spondylosis at C5-7 compared to 7% in the normal population. For those with a history of losing consciousness at presumably higher impact speeds, the incidence increases to 60%.
Joints of Luschka:

These sagittal cartilage lined joints present on the cervical bodies lateral to the discs. Capsules are continuous with the disc annulus. Flexion and extension is assisted by these joints. Lateral flexion is stabilized as the joints approximate the side of lateral flexion and separate on the contralateral side.

**Ligaments:**

Ligaments exhibit greater strength and distensibility than those of the thoracic spine which allows for the intricacies of movement and coupled motion. The atlanto-occipital ligaments are part of the rotational system of the upper cervical spine. Ligaments farthest from the axis of rotation demonstrate the greatest stretch. Longitudinal ligaments degenerate with age.

**Anterior ligaments:**

_Cruciate ligament_ wraps around the dens from the postero-lateral atlas with a synovial joint between them. _Apical ligament_ extends between the dens tip and basi-occiput to center the vertebral column with the skull. _Alar ligaments_ from the superior dens to the foramen magnum stabilize the head with the center of the cervical spine allowing the cranium to balance properly on the spine and prevent hyperrotation. _Antero-lateral atlanto-occipital ligament_ runs from the basi-occiput to the TP of the atlas and is involved in upper cervical rotation. _Occipital-axial ligaments_ run from the occiput base to the body of C2 and limits rotation and lateral flexion. _Anterior atlanto-axial ligament_ runs from the anterior of C1 to the dens and stabilizes the suboccipital region in extension. _Anterior longitudinal ligament_ extends from the occiput and
C2 anterior vertebral body to the sacrum and limits extension and possible dens fracture. *Posterior longitudinal ligament* runs posterior of the vertebral body from the occiput to the sacral canal and limits flexion.

Section 10: Normal Biomechanics of the Cervical Spine

**Structural and Functional Anatomy** (continued):

**Ligaments**: (continued)

**Posterior Ligaments**:

*Posterior atlanto-occipital ligament* runs from the occiput to C1 and is continuous with the *lateral atlanto-occipital ligament* which stabilizes the occiput-atlas connection and limits flexion. *Posterior atlanto-axial ligament* runs between the posterior arches of C1 and C2 and limits flexion. *Ligamentum nuchae* connects spinous processes of C2-C7 and posterior arch of C1 and limits hyperflexion. It is the most commonly injured ligament in acceleration/deceleration impact. *Interspinous ligaments* are between each spinous process. *Posterior cervical ligament* descends the midline and divides the musculature into right and left compartments. *Ligamentum flavum* connects posterior arches of C2 and C3. *Capsular ligaments* support the joint capsules.

**Muscles**:

The head and neck are part of a lever system in which the center of gravity is balanced anterior to the spinal canal and bodies. Extensor muscles counterbalance this by maintaining constant tone. Flexor muscles allow for forward flexion of the cervical spine and contribute to extension due to the biomechanics of cervical lordosis. The muscles criss-cross the ipsilateral vertebral arches and bodies creating a pulley system for movement. The balance between long and short muscles determines if an anterior muscle creates flexion or extension. Posterior suboccipital muscles fine tune by acting as levers, stabilizers responding to each other synergistically. Biomechanics depends on the tone of the opposing muscles or rigidity of joint play. In patients
with fixated joints, anterior muscles flatten the lordosis. Although it would seem this would create instability, it is, stability being created for a cervical mechanics that differs from the norm. This is most commonly seen with late effects of acceleration/deceleration injury. However, if the correct biomechanics are not restored, DJD can occur.

**Anterior musculature:**

*Sternocleidomastoid* allows for head rotation contra-laterally, lateral flexion ipsilaterally and extension. Bilateral contraction creates extension in a normal spine and flexion in an inflexible spine reversing of lordosis. *Longus cervicis* runs from bodies to the TPs of several segments above. Unilateral contraction allows for forward and lateral flexion. Bilateral contraction allows for a flattening of the curve and flexion. *Rectus capitus anterior* runs from the occiput to the TPs of C1, 3-6. Bilateral contraction causes flexion of the head on the spine with flattening of the lordosis. Unilateral contraction leads to forward and lateral flexion of the head and ipsilateral rotation. *Rectus capitus lateralis* runs from the occiput to the TP of C1. Bilateral contraction flexes the head on the spine at the atlanto-occipital joint. Unilateral contraction laterally flexes the head. *Intertransverse* attaches from TP to TP and with the scalene group, flexes the cervical column ipsilaterally.

*Scaleneus anterior* runs from the TPs of C3-C6 to the first rib. *Scaleneus medius* runs from the TPs of C2-C7 to the first rib. *Scaleneus posterior* runs from TPs of C4-C6 to the second rib. Bilateral contraction allows for flexion increasing lordosis. However with spasm, lordosis is not increased. Unilateral contraction causes ipsilateral lateral flexion and rotation. When vertebral attachments remain steady, scalenes are accessory breathing muscles elevating ribs 1,2 during inspiration. Between the anterior and medial scalenes runs the brachial plexus and subclavian artery.

The following muscles are accessory: *suprahyoid* attaches the mandible and hyoid; *infrahyoid* attaches hyoid to the sternum, thyroid bone and clavicle; *platysma* runs from the fascia over the
pectoralis major and deltoïd to the mandible and skin of the face. Accessory muscles act in unison to lower the mandible. However, when the jaw is fixed by the muscles of mastication, hyoid muscles flex the head. They also flex the cervical column on the thoracic spine decreasing lordosis.

**Posterior Musculature:**

Suboccipital muscles: *rectus capitis posterior major* runs from SP of C2 to the occiput; *rectus capitus posterior minor* runs from C1 to the occiput; *obliquus capitis superior* runs from C2 to the TP of C1; *obliquus capitis inferior* runs from SP of C2 to TP of C1. The obliquus capitis inferior maintains the integrity of the atlanto-axial joint. Bilateral contraction allows C1 to be retracted and extended on C2 relieving tension on the transverse ligament. When the rectus capitus and obliquus capitus muscles contract unilaterally, lateral flexion of the head occurs at the ipsilateral atlanto-occipital joint. Bilateral contraction of the four muscles creates extension of the head on the cervical spine. Head rotation is created at the atlanto-occipital joint by the contralateral superior oblique and at the atlanto-axial joint by the ipsilateral posterior rectus capitus major. Unilateral contraction of the superior oblique will move one side of the atlas anteriorly thus creating a 10 degree rotation of the head to the opposite side.
Interspinous runs between C3-C7 and is homologous to the rectus capitus posterior. Intertransverse runs from TP to TP. Cervical transverso-spinalis attach the SPs to the TPs of one or two vertebra below. Bilateral contraction extends the cervical spine increasing lordosis. Unilateral contraction creates ipsilateral lateral flexion and contralateral rotation. Semispinalis capitis arises from the TP of T1-T6, C3-C7 and the SP of C7, T1 and inserts into the occiput. Bilateral contraction extends the head and increases lordosis. Unilateral contraction extends with ipsilateral lateral flexion. Semispinalis cervicis runs from the TPs of T1-T5 to the TPs of C3-C7. Bilateral contraction extends the lower cervical spine. Unilateral contraction creates ipsilateral lateral flexion. Longissimus thoracic runs from the first six ribs to the posterior tubercles of C3-C7. It tightens the lower cervical column and elevates the first six ribs. Splenius capitus runs from the SPs of C2-T4 to the nuchal line and mastoid and splenius cervicus runs from the SPs of C2-T4 to the TPs of C1-C3. Contraction of these muscles bilaterally extends the head and increases lordosis. Unilateral contraction creates extension, ipsilateral lateral flexion and rotation of the lower cervical column. Levator scapula runs from the TPs of C1-C4 to the supero-medial scapula. Function is determined by fixed attachments; fixed cervical attachment allows for elevation of the scapula, fixed scapula attachment allows for movement of the cervical spine. Bilateral contraction causes extension with increased lordosis. Unilateral contraction causes ipsilateral extension, lateral flexion and rotation of the lower cervical spine. Trapezius runs from the occiput, posterior cervical ligament and SP of C1-T10 to the clavicle, acromion and scapula spine. It acts on the scapula, however, when the scapula is stabilized bilateral contraction extends the cervical column increasing lordosis, unilateral contraction creates head extension increasing lordosis while it ipsilaterally laterally flexes and rotates the cervical spine.
Nerves:

The spinal cord’s greatest vulnerability is between the inferior articular facet of C5 and superior facet of C6 with anterior dislocations being the most common cause of injury. Because of this, any attempt to place the head and neck into flexion after trauma will further compress the spinal cord. Stabilization of the cervical spine relies on extension.

Dorsal and ventral nerve roots; dorsal nerve root carries sensory nerves, ventral root holds motor nerves. The roots converge prior to exiting the vertebral column minus the meningeal branch and are reorganized laterally into anterior and posterior primary divisions. Once outside the spinal column, they form trunks of specific nerves. The anterior primary divisions form the cervical and brachial plexii. Branches of the cervical plexus from C2-C4 combine to form the superior cervical sympathetic ganglion relaying information to and from the autonomic nervous system. It is located on the antero-lateral surface of the cervical spine at the level of C2 and can be injured during acceleration/deceleration trauma. Symptoms include pain, skin changes and motor problems and it is termed Reflex Sympathetic Dystrophy Syndrome.
The *cervical plexus* consists of nerves from C1-C4. After dividing as sensory and motor nerves, some reorganize and descend the cervical spine. Swallowing, breathing, neck flexion, extension, lateral flexion and rotation are functions of the cervical plexus.

The *brachial plexus* consists of nerves from C5-T1. Neck flexion, extension, lateral flexion, rotation; shoulder abduction, adduction, internal rotation, external rotation, flexion, extension; scapula retraction, protraction, upward rotation, elevation; elbow flexion, extension; wrist flexion, extension, adduction, abduction; finger and hand flexion, extension, adduction, abduction are functions of the brachial plexus.

The muscles innervated by the cervical and brachial plexii are as follows:

- **C1,2,3**
  - sternocleidomastoid (also CN 11)
  - suprathyroid muscles
  - infrathyroid muscles

- **C3,4**
  - trapezius
  - neck extensors and suboccipital

- **C5,6,7**
  - serratus anterior
  - pectoralis major
  - pectoralis minor

- **C5,6,7,8**
  - latissimus dorsi
  - teres major
C3,4,5 (phrenic nerve)  
diaphragm  

C4,5  
rhomboids  
teres minor  
levator scapulae  

C4,5,6  
supraspinatus  
infraspinatus  
subclavius  
subscapularis  

C5,6 (axillary nerve)  
deltoid  

C5,6 (musculocutaneous nerve)  
biceps  
coracobrachialis  
brachialis  

C 6,7,8 (radial nerve)  
triceps  
brachioradialis  
long extensors or carpi and digits  
abductor longus  

C6,7,8,T1 (median nerve)  
pronator teres  
palmaris longus  
long flexors of carpi and digits  
pronator quadratus  
abductor pollicis brevis  
opponens pollicis  
lumbricles I, II  

C8,T1 (ulnar nerve)  
interossei  
lumbricles III, IV  
hyposthenar  
flexor carpi ulnaris  
flexor profundus  
adductor pollicis  

Sensory nerves innervate the neck and upper extremity as follows:  
C1,2 – occipital region  
C2,3,4 – neck region  
C4,5 - shoulder region  
C5,6 – axillary region  
C6,7,8 – radial region  
C6,7,8 – median region  
C8,T1 – ulnar region
**Blood vessels:**

The carotids ascend the neck outside the spinal column with branches joining those of the vertebral artery to form the Circle of Willis. Vertebral arteries ascend the vertebral column through openings in the TPs of C6-C1 and wrap around the lateral masses of the atlas to enter the cranium. A cervical spine in extension compresses the vertebral arteries in the upper cervical region increasing risk of stroke.

**Fascia:**

Anterior fascial components are continuous with the platysma and anterior chest musculature. Posterior layers are continuous with the fascia of the upper extremity. Fascial adhesions and scar tissue are responsible for trigger points, decreased stretch of muscle fibers and limited range of motion. Nerves become entrapped and can mimic symptoms of neuropathy or neuritis. The pain pattern associated with fascial disturbances can mimic muscle or nerve pain but do not follow a dermatome or neurological pattern.
Upper Cervical Spine:

Fracture:

Up to 80% of cervical fractures are a result of acceleration/deceleration impact. Severe hyperflexion or hyperextension and/or head strike on a hard surface such as the dashboard is responsible for these injuries. Complicating factors that increase the probability of fracture is DJD, osteoporosis, RA, DISH, ankylosing spondylitis, etc. Those with osteoporosis can develop anterior and posterior fractures simultaneously. Those with ankylosing spondylitis will fracture along the line of the disc. Those with DISH tend to fracture at the body. Any individual who has been in an acceleration/deceleration impact where there is cervical pain especially if their head hit an object should be X-rayed even if the impact was considered minor. Fracture or minor dislocation can be missed in standard examination.

Occipital condyles can fracture from high speed incidents of extreme hyperextension with distraction. The alar ligaments can tear. However, because this is a strong articulation, fracture is rare. Usually, the atlas will tend to fracture first. When it does occur, it is usually associated with severe brain and spinal cord injury.

The atlas can fracture from hyperextension through the posterior arch behind the lateral masses, the weakest part of the arch. There can also be a burst or Jefferson fracture of the atlas. This is common with rollovers where the occupant’s head strikes the vehicle’s roof, windshield or dash. This can also occur to a passenger in the rear when the seatback fails and their body becomes unrestrained and rides over the seat back. Here, the vertical forces on the atlas lateral masses between the articulating surfaces of the occiput and axis will burst the atlas ring. A Jefferson fracture does not usually cause neurological deficits but cranial nerve involvement has been recorded. The atlas can also fracture at the lateral masses, TPs and the anterior arch horizontally.
The axis can break at the posterior arch causing a hangman fracture or bilateral pedicle fracture also known as traumatic spondylolisthesis. It is measured in three types of severity:

Type 1 - bilateral fractures through the pars interarticularis of 3mm or less.
Type 2 - over 3mm and a compression fracture of the antero-superior corner of C3.
Type 2a - as above with anterior angulation of the dens.
Type 3 - As above and includes a dislocation of C2 on C3.

With acceleration/deceleration impact, this fracture may not cause death because the forces on the anterior dens are not vertical but horizontal. The dens is compressed toward the vertebral arch avoiding fatal injury to the spinal cord. This can occur if the occupant is too close to an inflating airbag and the force pushes their cervical spine into extreme extension.

Dens fractures can be classified into 3 categories:

Type 1 - avulsion of the tip from excessive tension on the alar ligaments (heals with bracing).
Type 2 - at the base with an angulation anterior displacement (less likely for cord symptoms).
Type 3 - at the neural ring of the body. This is the most serious.

There can also be fractures to the transverse processes, the body and spinous processes of the axis.

Dislocation:

Occiput – C1 dislocations are rare and usually fatal causing death by respiratory paralysis. Those that do survive the initial trauma suffer from quadraparesis. Extreme hyperextension with a distractive force can rupture the alar ligaments causing the occiput and atlas to separate.

Between C1 and C2, there can be an anterior dislocation of the atlas from a ruptured transverse ligament. These fractures are highly unstable and usually fatal. RA patients can have weak or
deteriorated ligaments; Down’s syndrome patients can have minimal or no transverse ligament. MRI would be the best to determine the patency of the transverse ligament. A posterior dislocation of the atlas can occur if the patient’s chin hits the dash, steering wheel or seat back or is victim to an air bag. Dislocations between this joint usually encompass dens fracture but does not cause severe neurological deficits. However, anterior dislocations of the atlas on the axis can cause compression of the spinal cord between the posterior arch of C1 and posterior axis body. This can be due to a transverse ligament tear and lead to spinal cord damage. If survived, these patients will need fusion of C1 and C2 because the transverse ligament cannot be sufficiently repaired due to its inherent weakness. Rheumatoid arthritis and Downs syndrome patients are vulnerable to this injury. Rotational dislocations can also occur from overrotation which can tear the articular capsule. Here the patient can rotate their head to the side the atlas has overrotated but they cannot turn their head the other way.

Instability:

Clinical instability is defined by Punjabi and White as “the loss of the ability of the spine under physiologic loads to maintain relationships between vertebrae in such as way that there is neither damage nor subsequent irritation to the spinal cord or nerve roots, and, in addition, there is no development of incapacitating deformity or pain due to structural changes”. However, Scher defines instability as “abnormal mobility between any pair of vertebrae with or without pain or other clinical manifestation” thereby conflicting with Punjabi and White in the need for incapacitating deformity or pain. Subluxation or vertebral dysrelationship/dysfunction would be included in this definition.

Instability is usually the result of injured or torn ligaments or fracture, not muscular injury. There have been several methods to measure ligamentous instability. These include X-ray and videoflouroscopy. X-ray is most commonly used but can vary in its reliability due to size distortion as the image projected is formed from particles fanning out across the film. It is however, the most commonly used method to determine instability.

Powers et al. uses the ratio of distances between specific bony projections on the atlas posterior arch and anterior arch to determine atlanto-occipital dislocations. A ratio of less than 1 is normal. Fracture will void this test.

Swischuk described a line from the inside surfaces of the posterior arch of C1 and spinous processes of C2,3. A posterior displacement of C2 of more than 2mm can indicate a hangman’s fracture in adults or dislocation in children.

The atlanto-dens interval or ADI has been used to determine tear to the transverse ligament. Less than 3mm is normal in children, less than 2mm is normal in adults.

Rotary subluxation of C1 on C2 is more common with acceleration/deceleration impacts when the patient’s head was turned. A patient that presents with torticollis after impact should be suspected and evaluated. APOM X-ray will show a dysrelationship between the lateral masses of the atlas and dens. The space will be wider on the side of posterior atlas rotation and the shadow of the lateral mass will be narrower on that side.
Fielding and Hawkins described several types of subluxations of C1 on C2:

Type 1: rotary fixation with no anterior displacement (the odontoid is the pivot).
Type 2: rotary fixation with anterior displacement of 3-5 mm. (1 articular process is the pivot).
Type 3: rotary fixation with anterior displacement of more than 5 mm.
Type 4: rotary fixation with posterior displacement usually from dens fracture.

The level of instability will determine the course of treatment. Manipulation is contraindicated in Type 4 and possibly Type 3 depending upon the extent of ligament injury. CT combined with MRI can better clarify the level of ligament damage and assists in determining diagnosis and treatment.

Lower Cervical Spine:

Forces on the lower cervical spine differ than those of the upper cervical spine and are defined as major vectors which produce initial strain on the tissues and minor vectors which produce strain from a second direction. Major vectors can be flexion or extension and are produced by acceleration or deceleration of a vehicle. In the spine, both directions of force are produced simultaneously. Hyperflexion produces traction along the ligamentum nuchae and compression is along the anterior edges of the bodies. For hyperextension, traction is at the anterior bodies and compression is to the posterior structures. The region of the spine that experiences neither is considered the neutral zone and is usually the region of the PLL. The minor vector accentuates the major vector and determines if the injury is from compression or traction forces to the spine.
Fracture:  

Wedge fracture of the body can occur after a severe hyperflexion. X-ray detects decrease in height of the anterior body. It is considered a stable fracture because there is no neurological sequela and can be supported by a halo. Fracture sites put undue stress on the discs, alter lengths and stability of ligaments and muscles in about 22% of patients.

Clay Shoveler’s fracture found at the SPs usually occurs at C6,7, T1. Severe posterior muscular contraction can avulse. It is considered a stable fracture but ligament damage is common. The fragment is usually surgically removed.

Articular pillar can fracture from severe hyperextension with rotation. This compression type injury can occur in up to 21% of those with cervical spine fractures. These can be difficult to diagnose and specific pillar radiographic views may be ordered as well as CT. There will be radicular symptoms but this is considered a stable fracture. Lamina fractures are common with articular pillar breaks.
Transverse processes can fracture and can present with radiculopathy and/or vertebral artery occlusion or dissection. Be wary of patients with vertebrobasilar symptoms such as posterior headache, dizziness, imbalance, etc.

Section 12: Traumatic Effects on the Cervical Spine after Acceleration/Deceleration Impact (continued)

**Lower Cervical Spine:**

* Bones (continued):

* Fracture (continued):

Compression fracture can occur from a vertical force. It can accompany a Jefferson fracture of the atlas ring as well as injury to the IVDs. The force may cause the nucleus to push through the endplates into the body forcing the bony material posteriorly into the spinal canal. The cord may be bruised causing neurological damage. The bony material may recede making the neurological symptoms more severe than what appears on X-ray. This is less severe than the flexion teardrop fracture.

**Compression flexion fracture:** there are 5 stages:

Stage 1- compression of anterior-superior vertebral border. Cervical pain and decreased ROM.
Stage 2 - as Stage 1 but with compression of anterior-inferior border. A loss of 3mm or more of vertebral height. Nucleus is pushed through the end plate causing vertical fracture lines.

Stage 3 - as Stage 2 but with an oblique fracture from the inferior to the superior end plate. Possible damage to posterior ligamentous structures.

Stage 4 - as Stage 3 but with posterior displacements of vertebral body fragments into the neural canal.
Stage 5 – Further displacement of vertebral body fragments into the canal.

**Distraction flexion fracture:**

Posterior damage to the ligamentum nuchae and interspinous ligaments occurs during hyperflexion from a minor vector of compression when the occiput hits the head rest. It provides a distraction force on the posterior cervical spine.
Stage 1 - widening of facets with body compression.

Stage 2 - unilateral facet dislocation with rotation, anterior body displacement, widening of posterior disc height.
Stage 3 - bilateral dislocation of a posterior facet with body displacement of 50%; tearing of PLL, annulus of IVD, interspinous ligament, facet capsules.

Stage 4 - total dislocation with tearing to posterior ligaments.
Compression extension fracture:

Hyperextension produces traction along the ALL and compression between the SPs. Here the posterior facets become fulcrums and receive most of the damage.

Stage 1 - unilateral fracture through lamina or facet without displacement.
Stage 2 - multiple levels of stage 1 fractures. Possible rotary component.
Stage 3 - anterior displacement of the vertebral segment
Stage 4 - further anterior displacement of the vertebral segment.

Stage 5 - anterior dislocation of vertebral segment
Distraction extension fracture:

This is usually due to a blow to the face or chin. There can be injuries to the trachea and esophagus.

Stage 1 - tear to ALL and disc, possible avulsion of inferior body.
Stage 2 – posterior displacement of the body.

Distraction flexion fracture:

Complex comminuted fractures can be associated with tears to the posterior longitudinal ligament and spinal cord.

Fractures of the facets and joints of Luschka can occur. These are relatively stable.

Dislocation:

Dislocation is usually associated with fracture and can be missed during initial evaluation. X-ray has been insufficient; CT/MRI is recommended. It is also possible that dislocation occurred during impact but spontaneously reduced from the actions of the cervical spine. Inflammation, ligament tear, neurological sequela can all be symptoms. Again, CT/MRI can identify effects of soft tissue injury.

Unilateral facet dislocation can be recognized on plain film as one vertebra riding over another. On an oblique film, there would be a disruption of the stacked appearance of facets. There would usually be a flexion and rotary factor in the impact.

Bilateral facet dislocation results from severe hyperflexion of the cervical spine. Both facets, PLL, interspinous ligament and IVD can be involved. This is highly unstable and requires surgery.

Instability:

A disruption in the normal mechanics associated with pain, spasm and/or neurological effects indicates instability of the lower cervical spine. The most common instability in the lower cervical spine after acceleration/deceleration is anterior vertebral subluxation. Detection can be a
delayed from muscle splinting. Once spasms reduced, this flexion fixation can become more evident as instability increases. Greater instability is determined if the segment moves further anterior upon cervical flexion. X-rays of anterior flexion fixations can show fanning of the SP from posterior damage, narrowing of the anterior IVD space, widening of the posterior disc space, loss of lordosis or development of kyphosis greater than 11 degrees, loss of normal facet association, and anterolisthesis. A 1-2 mm change in movement can indicate clinical instability from vertebral subluxation. Anterior flexion fixations can lead to thoracic outlet syndrome and increase in anxiety due to inflammation compression of the sympathetic chain. Left unchecked, it can lead to DJD.

**Joints:**

**Disc:**

Injury to the disc is usually due to a tear in the annulus and can occur anteriorly from hyperextension and/or posteriorly from hyperflexion. It may be accompanied by an anterior subluxation of the vertebral segment from a disruption in the posterior elements. There may be an endplate fracture with seepage of the nuclear material into the body of the vertebra above or below. The nucleus can also seep into the annulus which if torn can lead to a herniation of material outside of the disc. The disc may also swell without tear. Studies have shown that it is the combination of compression and horizontal sheer that accounts for most disc injuries.

Acute disc injury can occur to a patient with the complicating factor of chronic disc disease. The symptomatology may be difficult to discern. Chronic disc disease can produce varying levels of usually intermittent local pain, and/or headaches; shoulder, elbow, wrist or hand pain. Examination and thorough history will assist in discerning the two conditions from one another.
**Ligaments:**

Hyperextension phase of the acceleration/deceleration impact can injure the ALL. Injury extent depends on severity and vectors of impact; position, size and age of patient and any complicating factors that may leave the anterior components of the cervical spine more vulnerable. This can be accompanied by a rim lesion where the disc separates from the vertebral body. Also common are facet capsule sprains and bleeding beneath the ALL and can be found with MRI.

Hyperflexion phase of the acceleration/deceleration impact has damaged the posterior capsule, interspinous ligament and ligamentum flavum. Green at al. has studied anterior subluxation of the cervical spine and determined that there is a 20% incidence of delayed instability due to abnormal ligamentous healing. This indicates that the instability, secondary to improper healing, can become apparent weeks or months after the initial impact. The transverse ligament can tear. When this occurs, the alar ligament becomes the primary stabilizing structure in the cervical spine. If the patient’s head was turned during impact, the alar ligament can tear as well. The apical ligament can also tear in severe trauma.

**Muscles:**
In a rear impact, the patient’s torso is accelerated and their head and neck lag behind. When the head begins to move into extension, there is a stretch of the cervical spine. This causes the patient’s anterior spinal musculature to stretch beyond their capacity. The remaining force is taken up by the anterior spinal ligaments and annulus. The inability to adapt to the rapid changes could lead to rupture of muscle fibers and damage to fascial structures, blood and lymph vessels, nerves, ligaments and discs. When the head alters direction and heads into flexion, there may be a violent contraction of the flexors and excessive stretch of the posterior cervical elements causing the posterior cervical muscles to contract and spasm.

Szabo and Welcher used surface EMG and human crash tests to determine that the posterior cervical muscle spindles begin to contract before the cervical spine begins to move. This could be due to the reaction of the lumbar spine as the torso is forced into the seat back. This hypothesis needs further testing but is a viable explanation for injury.

Wickstrom et al. and Unterharnscheidt studied the deceleration or flexion phase of the impact. The flexion injury can be worse if a patient hits another vehicle that is in front of them. When this occurs, assuming there is a shoulder harness seat belt apparatus, there is a linear traction injury at the end of forward rotation of the cervical spine. This observation and studies of the cervical spine in hyperflexion has lead to the conclusion that the flexion phase of the injury is more harmful than the extension phase. This would explain the level of high injuries that are associated with the suboccipital muscle group. A lateral and/or a rotary component would increase damage.


**Neurological:**

**Brain Injury:**

The brain can be traumatized in acceleration/deceleration impact from direct head strike to the dash or door, or from excessive hyperextension/hyperflexion where the brain bounces off the internal of the bony cranium. This would be considered a secondary impact. EEG is usually used to determine brain injury, however, it has been shown that it is less accurate than previously thought. Approximately 24% of brain injured test subjects had normal EEG. Test should be used as an adjunct to examination findings and symptomatology.

Most brain injuries will be suffered from skull fractures. These will be assessed in an emergency setting and is out of the scope of this course. Any injury to the brain that would be seen in a walking patient is brain contusions. Symptoms are loss of consciousness, nausea or vertigo. Here, there may be bleeding into the subdural space or the development of an extradural hematoma. Neurological deficits may be present depending where on the head the injury was sustained.
There are several signs of head injury. These include anosmia or loss of smell, double vision, subdural hemorrhage, extradural hemorrhage. Injury to the brain stem is rare because they tend to be fatal. These usually occur from dislocation of the occiput or C1 or severe hyperextension of the neck.

Spinal Cord Compression:

Compression, stretch or torquing can occur to the spinal cord and may be due to excessive movement between two vertebrae. These injuries are usually due to tearing in the ligaments or in patients with spinal stenosis. Other conditions that may lead to a compression of the spinal cord at impact are ossification of the PLL, herniation of the nucleus pulposi or infolding of the ligamentum flavum. During rapid deceleration of the spine, the cord may strike the vertebrae as with dislocations or fragments of vertebrae as in the case of fracture. Full transaction may also occur, being fatal.

Nerve Root Compression:

A nerve root can become injured after acceleration/deceleration impact from a spondylophyte or injured disc material, from compression fracture at the IVF, due to a cerebral spinal fluid wave injury during the movement of the trauma. Late effects of trauma such as scar tissue and adhesions in muscle and fascia can also compress nerve roots and nerves. Symptoms of nerve compression at the root are neck pain relieved by cervical collar and aggravated by movement, muscle spasm, possible atrophy, paresthesias that follow a dermatome path, possible reflex changes and fasciculations, positive compression tests. Pain is also noted at the trapezius and scapula.

Reflex Sympathetic Dystrophy Syndrome:

This condition can result from trauma to the sympathetic plexuses that are located along the anterior of the SCMs and anterior scalenes bilaterally. Direct trauma is rare but nerve elongation and compression can occur secondary to muscle and fascial injury. Symptoms are burning pain usually in an extremity, edema, muscle spasm, atrophy or dryness of the skin, changes in hair growth nails and temperature, Raynaud’s phenomena, vasomotor hyperesthesia, hypoesthesia, muscular dyscoordination, tremor, joint pain and swelling, bone changes such as localized osteoporosis, and palmar fascitis. It is common for injury to the sympathetic chain to cause anxiety and depression or exacerbate an underlying condition in patients that have experienced cervical spine trauma.

Peripheral Nerve Damage:

Vulnerable to crush injuries at the cervical spine, they are also susceptible to stretch injuries at the IVF. Nerves can be entrapped by soft tissue anywhere along their path. Compression and/or elongation of the nerve can lead to partial or total ischemia. Complete ischemia occurs at 15% stretch. Local ischemia may lead to numbness along the dermatome or an entire limb. There will be no pain.
According to Seddon, there are 3 categories of nerve injuries:

*Neuropraxia* - Mild neural compression resolves in 3-6 weeks.

*Axonotmesis* - Severe crush takes up to 6 months to heal. It causes Wallerian degeneration (part of the axon is separated from the neuron cell body and degenerates distally).

*Neurotmesis* - Complete disruption of the neural and fibrous structures around the nerve. Recovery is guarded even after surgical repair.

According to Seddon, there are 5 categories of nerve injury:

*Stage 1* - Initial stretch tears the axon.

*Stage 2* - Torn axon has its distal aspect degenerate.

*Stage 3* - Axon, sheath and endoneurium are disrupted.

*Stage 4* - Separation of perineurium in addition to Stage 3.

*Stage 5* - Complete disruption of nerve and cessation of function.

Double Crush Phenomenon:

In this condition, a nerve is compressed in two places. Two syndromes in this phenomenon are Carpal Tunnel Syndrome and Thoracic Outlet Syndrome. In Carpal Tunnel Syndrome, the double crush is at the carpal tunnel and complicated with a direct insult to nerve roots at the cervical spine related to the medial nerve. With Thoracic Outlet Syndrome, some patients were treated successfully by decompressing the carpal tunnel. This would suggest that there was a compression at the cervical spine as well as wrist.

Brachial Plexus Injuries:

Most injuries to the brachial plexus are due to high speed tractional forces such as a fall from a motorcycle when the head compresses the plexus between the spine and the clavicle. There are supraclavicular and infraclavicular injuries.

Supraclavicular injuries depend upon the position of the patient’s arm at the time of impact. When their arm is at their side, the greatest tension on the nerve roots is at the upper portion of the brachial plexus. The 1st rib acts as a pulley and accentuates the upping on the upper nerve roots. When the arm is extended and parallel to the floor as in holding the steering wheel, traction of the arm can injure any of the nerve roots in the plexus. When the arm is raised above the head, there is a slack of the upper nerve roots and the main tension is on the lower aspects of the brachial plexus. Here the coracoid process acts as the pulley and allows the force to be on the lower region of the plexus.

Injury to C5 and C6 Nerve Roots:

Loss of shoulder control and elbow flexion can be expected in an injury to C5 and/or C6 nerve root. There would be atrophy of deltoid, infraspinatus and supraspinatus muscles causing strength loss leaving the patient’s shoulder in adduction and medial rotation. There would be an inability to abduct, lateral rotate and flex their arm with only partial control of the scapula but not
much else. Biceps, brachialis, brachioradialis would be nonfunctional. Shoulder atrophy would eventually lead to an inferior subluxation of the glenohumeral joint due to the weight of the arm.

Injury to C5, C6 and C7 Nerve Roots:

These patients will have the same symptoms as above with additional issues from C7 injury; loss of elbow, wrist and finger extension. Injury to the C7 nerve root alone is rare due to its position of it in the midst of the brachial plexus. If injured, it would be along with injuries to other nerve roots as well.

Injury to C8 and T1 Nerve Roots:

This is the least likely injury to occur due to the position of the nerve roots and the slack the nerves have when the arm is hanging to the side of the patient. Injuries to this region are most likely when the arm is raised creating traction on the lower region of the brachial plexus. Injury at this level will produce a Horner’s Syndrome and possible medial and ulnar nerve palsy.

Injury to the Entire Brachial Plexus:

It takes a serious neck injury for there to be damage to the entire brachial plexus. One of the first signs that this has occurred is a Horner’s Syndrome. There would be a flail arm and sensory exam will reveal total anesthesia below the elbow and major anesthesia above the elbow since T2 does have some innervation to the upper arm and is not part of the brachial plexus. Although the patient’s injury will render their arms anesthetic to external stimulus, they will experience severe pain from the neurological damage.

Section 14: Traumatic Effects to the Soft Tissue of the Cervical Spine after Acceleration/Deceleration Impact

Nerves (continued):

Injury to Individual Nerves:
Median Nerve:

This nerve can be injured in several sections creating several conditions:

**Infraclavicular median nerve entrapment** occurs after arm hyperabduction which stretches the median nerve around the clavicle or traps it between the pectoralis minor and coracoid process.

**Anterior Interosseous Syndrome** is the peripheral entrapment of the anterior interosseous nerve (median nerve branch). There would be motor involvement, the patient is unable to flex the distal joints of their index finger and thumb or pinch sign and weakness in pronation.

**Pronator syndrome** occurs when the nerve is entrapped in the pronator muscle producing weakness in the hand muscles and decreased sensory function.

**Carpal Tunnel Syndrome** has been recorded after acceleration/deceleration impact. Double crush may occur at the nerve roots or there may be local wrist trauma from the steering wheel or air bag.
**Ulnar Nerve:**

Compression leads to motor weakness of the intrinsic hand muscles. Sensory deficits lead to numbness of the dorsal and palmar aspects of the 4th and 5th digits. The most common point of entrapment is in the cubital tunnel where the nerve travels between the medial epicondyle and aponeurosis of the flexor carpi ulnaris. The nerve can also be trapped in the Guyon’s canal which is between the pisiform and hook of the hamate. Although rare, it is still clinically significant.

**Radial Nerve:**
Three conditions can occur when the radial nerve is compressed:

**Saturday Night Palsy** occurs when there is pressure on the proximal medial portion of the arm. There is usually a neuropraxia that responds to conservative care.

**Posterior Interosseous Syndrome** occurs by compression of the radial nerve distal to the elbow limiting the patient’s ability to extend both the thumb and metacarpal-phalangeal joints. This is usually a nerve entrapment from a fibrous band within the arm.

**Superficial Radial Nerve Entrapment** occurs from an external source of compression. There will be pain and paresthesia of the web of the thumb.

**Vascular:**

Neurovascular Compression Syndrome:

Thoracic Outlet Syndrome is a condition of compression of neurological components. The neurological structures are responsible for vascular dilation and constriction in the region of the cervical spine and upper extremity. When these structures are compressed, the symptoms are numbness and tingling in the upper extremity. The compression can be due to costoclavicular syndrome where the structures are compressed between the 1st rib and clavicle, scaleneus anticus syndrome where the anterior scalene muscle compresses these structures, cervicobrachial syndrome where C7 TP can be elongated or there is a cervical rib that either stretches or compresses the brachial plexus, pectoralis minor syndrome when the pectoralis muscle is short or spastic and is compressing these structures, hyperabduction syndrome when the symptoms occur with hyperabduction of the arm and can be due to a structural anomaly or swollen soft tissue, cervical ribs can also cause this condition. Approximately 86% of this condition has been caused by cervical trauma due to acceleration/deceleration impact from violent stretch of the anterior and/or medial scalene has lead to this condition. It has been hypothesized that the neurovascular bundle is compromised by injured muscle tissue.

Vertebral Artery Insufficiency:

This condition has been documented after acceleration/deceleration impact. Increased tissue pressure from spasm, edema or hemorrhage can alter blood flow. Osteophytes from the lateral disc, facet or compression of the inferior facet from sheering by the superior facet can also lead to vertebral artery insufficiency. Arterial spasm can occur in the suboccipital region.
There are 3 regions of the vertebral artery that are most susceptible. These are the posterior atlanto-occipital membrane which is firmly attached to the artery and can become calcified, the space between the occiput and posterior arch of atlas (mostly during hyperextension), and the region between the lateral mass of the atlas and the transverse process of the axis (mostly from lateral flexion and rotation).

Vertebral Artery Syndrome (aka vertebrobasilar insufficiency):

This condition is due to thrombosis from hyperextension and rotation of the cervical spine and is more common in older patients who have atherosclerosis. Symptoms are vertigo, nausea, vomiting, dysarthria, nystagmus, and partial facial paralysis. If a plaque or thrombus is released, the result could be an occlusion of the posterior inferior cerebellar artery leading to Wallenberg’s Syndrome. Symptoms include ipsilateral loss of CN 5, 9, 10 and 11, cerebellar ataxia, Horner’s syndrome and contralateral loss of pain and temperature sensation.

Visceral:

Esophageal and hypopharyngeal perforations:

Injury to the anterior structures of the throat can occur from severe hyperflexion trauma. Perforations have been reported in severe injuries and hemorrhage into the esophagus has been noted in less severe injury. Older patients with spondylophytes are at a greater risk. Perforation of the esophagus can be fatal as it can lead to meningitis, bronchopneumonia or mediastinitis. Fever, neck swelling, dysphagia, leukocytosis, hematemesis and hemoptyysis are all symptoms.

Restraint Injury:

Restraints were devised to save lives by decreasing facial and chest injury. However, their design and function has increased spinal injury as well as abdominal and vascular injuries. The most vulnerable of the population are children due to improper fitting of the harness. The incidence of spinal injury is threefold in those wearing seat belts with twice the risk of symptoms after one year.

The cervical spine can be injured when the torso is restrained and snapped back on the decelerating trunk allowing the head to snap forward further injuring the neck. Also, the seat belt does not snap back fast enough in rear impacts creating a gap between the patient and the seat back. This slack allows for further shearing of the decelerating body and the accelerating head.

A fulcrum fracture (horizontal break through the spinous and body) can occur at the cervical spine from hyperflexion and a seat belt snap. Here, the superior segment flexes forward pulling the vertebral components apart. The spinous can also avulse from hyperflexion.

The TMJ can be injured by applied forces, movement of the head on the cervical spine and/or jaw on the head. Several theories to the mechanism of injury are inconclusive as testing
procedures are flawed. Most valid theories point to opening and closing of the jaw during movement of the head.

The shoulder harness can cause vascular injury with direct contact on the brachial plexus and subclavian structures.

The occupant can slip under a lap belt causing abdominal and pelvic visceral injury. Known as submarining, this can occur from slouching posture, poorly designed seat, bad placement of the seat belt at the door.

Breast tissue can be injured with evidence of fat necrosis from a seat belt during impact. Some cases evolved into eventual breast carcinoma. In breasts with implants, there is injury to the capsule that develops around the implant which can alter appearance.

Low back pain has been found in 57% of injury cases and 71% from a broad side collision. Factors affecting the causes of low back pain from acceleration/deceleration impact to those with restraints are:

- Occupant position
- Use of seat belts
- Air bag deployment
- Method of restraint (with or without pretensioners)
- Stiffness and inclination of the seat back
- Type of seat back padding
- Degree of ramping
- Vector and severity of the collisions
- Second collisions
- Speed of the snap back and fit of the restraint
- Position of restraint on occupant and on door
- Size, weight age, fitness level, complicating factors of occupant
- Awareness of the impending collision

Side impacts have shown to be most injurious to the lower back. Restraint systems offer little support to the lateral shearing forces that occur with lateral impact. When an individual is hit laterally, the initial reaction of the torso is to bend toward the side of impact compressing the spinal lateral structures on that side and stretching the lateral structures on the opposite side. The seat belt anchors the pelvis and protects those structures but that increases the lateral bend on the lumbar and thoracolumbar spine increasing the advent of disc, ligament and muscle injury. When the occupant’s vehicle is stationary the vector is perpendicular. When the occupant’s vehicle is moving, the vector is perpendicular and forward creating an oblique movement. The patient’s body will bend laterally as well as forward in an oblique direction toward the striking vehicle. This creates excess shearing to the lumbar spine and discs.

Other conditions that have occurred from the seat belt during acceleration/deceleration impact are compression fracture of the cervical spine, fracture dislocation at the cervical spine, posterior
arch fracture of the atlas, pelvic fracture (usually when there is a pelvic fracture there is more than one break), rib fractures and flexion compression fracture at the cervicothoracic region.

Mechanics of the impact vectors, speed, type of vehicle, etc combined with the body type, torso and extremity position, complicating factors, and age of the patient are necessary for a complete understanding of what the patient’s body went through as their vehicle moved in space from the impact. Gathering this information is vital in understanding their injuries in order to provide the best methods of evaluation and treatment.

Section 15: Symptoms of Cervical Acceleration/Deceleration Impact

An acceleration/deceleration impact event is varied by size, age, posture, gender, etc. The combination of injury type and mechanics determine which tissues are injured. Each tissue produces symptoms, some more obvious than others. These vary from patient to patient as some have more tolerance or experience with pain than others. Initially, particular symptoms will be more prevalent than others due to the fact that the brain will express the most offending region of inflammation and injury. Injury may be present that is not consciously apparent to the patient immediately. Also, symptoms may present up to one or more months after the impact. Symptoms are a guide to what can be wrong. They do not indicate the only issues that may exist. This is the reason for a thorough examination and history and periodic reexams.

**Symptoms:**

*Neck pain:*

The most common of symptoms is neck pain and can occur immediately after impact or be delayed hours, days, weeks or even months after the incident. In approximately 78% of acceleration/deceleration cases, the pain begins within hours. For 13% of patients, the pain begins between 12 and 24 hours with 7% of pain cases occurring after 48 hours. Theirry Ettlin has reported the greatest intensity and occurrence of neck pain after impact is at 72 hours. It has been hypothesized that delays in pain for months or years can be due to secondary biomechanical or myofascial issues that continue to change throughout a prolonged healing process.

Be wary of studies describing pain as an indicator of injury within the first minutes after an impact. These are usually derived from police reports, emergency room staff or individuals who can be in shock reporting to their insurance agents that they are not in pain at that moment. An Australian study by Dolinis using police reports found a small percentage of individuals went to the emergency room after an injury. It did not take into account that the majority of individuals tend to see their private doctors after a serious incident.

Another phenomenon is delayed instability with a delayed symptomatology. Immediate pain can be related to injury of the ALL, anterior annulus, end plate fracture, disc herniation. These lead to instability in which onset can be delayed as inflammation retreats.
A 24 hour delay in symptoms is common with severe acute cervical sprain/strain injury. TMJ symptoms can take weeks or months to develop. This could be due to the secondary spasms and resultant biomechanical dysfunction of regional cervical and temporal structures affecting CN 5,7,9-12 and spinal nerves C1-4.

**Headache:**

The second most common symptom in acceleration/deceleration impact is headache occurring in 66-92% of trauma cases. The most common injured regions of the head are the occipital and frontal with temporal and parietal pain usually accompanying TMJ injury. Headaches from acceleration/deceleration impact are extremely varied in pattern, frequency and region from patient to patient. Some headaches are from direct impact of the head on the head rest, door, steering wheel or dash. Some are from muscle spasm and direct force trauma to the muscles around the skull. Other headaches can be cervicogenic.

Of all the headache types, tension headache is most common with acceleration/deceleration impact. Characterized by frontal and occipital pain from muscular contraction around the cranium, pain can be felt between the eyes. Tension headache can be caused by aggravation to cervical spondylosis or from inflammation of the soft tissue structures.

Cervical muscle spasms with inflammation and microtears can cause cervicogenic headaches. Inflammation of myofascial attachments to the cranial periosteum can cause headaches after impact. It has been hypothesized that the greater and lesser occipital nerves are being trapped between the myofascial attachments and periosteum causing pain along their innervation.

Muscle, ligamentous and myofascial structures can tear, produce inflammation and release substance P and bradykinins leading to pain. Ischemia from muscle spasm can cause pain and impede the repair process.

Posture can affect cervicogenic headaches. A goose neck increases the load on the cervicothoracic spine and posterior muscles. Chronic goose neck leads to weak posterior cervical spine muscles with less endurance. It is common for individuals with osteoarthritis to develop contracted flexors and weak extensors. The reversal of the cervical curve commonly found with acceleration/deceleration impact leads to forward goose neck posture and eventually weakened posterior structures.

Vascular headaches can occur after acceleration/deceleration impact but are rarer than tension and cervicogenic headaches. Vascular headaches tend to be throbbing and unilateral in nature.

Posttraumatic headache occurs from a direct head strike or secondary acceleration injury from the brain to the skull. Response to rate of acceleration, duration of acceleration, and velocity change to the brain define headaches from brain injury.

In 1988 posttraumatic headache was defined by the International Headache Society. It differentiates between posttraumatic headache and acute traumatic headache. Acute traumatic


headache is characterized by any loss of consciousness or by posttraumatic amnesia lasting more than 15 minutes and relative abnormalities in at least two of the following areas:

1. Neurological exam
2. Skull radiographs
3. Neuroimaging
4. Evoked potentials
5. CSF examination
6. Vestibular function tests
7. Neuropsychological tests

These headaches occur less than 14 days after impact and are alleviated in less than 8 weeks after the patient regains consciousness. Posttraumatic amnesia has an established time frame which begins at impact and ends when the patient has regained full memory.

Posttraumatic headache is defined as above except they last more than 10 minutes and occur less than 14 days after impact but are not alleviated in less than 8 weeks after injury.

In 1991, the Head Injury Interdisciplinary Special Interest Group to the American Congress of Rehabilitation Medicine defined mild traumatic brain injury. This definition is based on the subjective responses of a patient after injury in their ability to communicate. The problem with the gathering of criteria is that the patient may or may not be aware of whether they lost consciousness. The patient needs to meet one of these guidelines:

1. Any period of loss of consciousness.
2. Any loss of memory for events immediately before or after the injury.
3. Any alteration in mental state at the time of the injury (e.g. Feeling dazed, disoriented, or confused).
4. Focal neurological deficit(s) that may or may not be transient but in which the severity of the injury does not exceed the following:
   a) Loss of consciousness of approximately 30 minutes or less.
   b) After 30 minutes, a Glasgow Coma Scale of 13-15.
   c) Posttraumatic amnesia not greater than 24 hours.

Concussion:

Postconcussion syndrome is brain injury that leaves patients temporarily or permanently impaired. Two million cases occur each year in the USA with acceleration/deceleration impact being the greatest cause followed by direct trauma to a hard object and internal brain strike against the cranium.

Posttraumatic headache can result from a postconcussion syndrome with an incidence of 40-60%. The following tables differentiate between posttraumatic headache syndrome and posttraumatic concussion syndrome. They are not mutually exclusive but can occur together.

Posttraumatic headache:

1. Headache
2. Irritability
3. Insomnia
4. Anxiety
5. Memory issues
6. Pain
7. Concentration issues
8. Depression
9. Dizziness
10. Confusion
11. Loss of emotional control
12. Loss of libido
13. Tinnitus
14. Difficulty carrying out plans
15. Difficulty planning
16. Flashbacks
17. Does not enjoy sex
18. Nightmares
19. Problems doing math

Postconcussion syndrome:
1. Lightheadedness
2. Vertigo/dizziness
3. Neck pain
4. Headache
5. Photophobia
6. Phonophobia
7. Tinnitus
8. Impaired memory
9. Easily distracted
10. Impaired comprehension
11. Forgetfulness
12. Impaired logical thought
13. Difficulty with new concepts
14. Difficulty with abstract concepts
15. Insomnia
16. Irritability
17. Easily fatigued
18. Apathy
19. Anger outbursts
20. Mood swings
21. Depression
22. Loss of libido
23. Change in personality

In studies of soft tissue injuries to the neck after acceleration/deceleration impact, Radanov et al. describes two separate conditions. These are cervicoencephalic syndrome and lower cervical spine syndrome.
In cervicoencephalic syndrome, symptoms are headache, fatigue, dizziness, poor concentration, disturbed visual accommodation, impaired adaption to light intensity, cognitive impairment in divided attention, and speed of processing information.

In lower cervical spine syndrome, symptoms are neck and upper extremity pain, impaired divided attention. Loss of consciousness was found to be unrelated with divided attention testing results.

Cognitive skill impairment studies of posttraumatic headache after acceleration/deceleration impact determined significant injury to the cervical spine is necessary for posttraumatic headache to produce cognitive alteration. Headache sufferers prior to impact were twice as likely to develop posttraumatic headaches. Studies demonstrated that at 6 months 68% of patients suffering from this condition were asymptomatic. Those 32% still suffering from cognitive impairment were usually older and displayed greater cognitive impairment and reported more pain than those asymptomatic. Cognitive functions affected were memory, information processing, hearing, divided and selection attention.

These statistics differ with children. A 23 year study of children affected by this condition, physical, emotional and intellectual problems were still evident in 31% of cases. In preschool children who suffered from this type of trauma, incidence was 3.3 times the norm for behavioral issues and lower IQ.

Adult females are more likely to develop cognitive impairment with posttraumatic headache after acceleration/deceleration injury. This is because the female brain accelerates twice as fast in the cranium as does males. Other cognitive dysfunctions noted were difficulty with pursuits, ocular posturing, stereopsis, extraocular movements, near and far sightedness, dissociation of reflex and intentional tasks, all problems of the frontal lobe.

A high number of patients with mild traumatic brain disorder from acceleration/deceleration impact suffer from neck, low back and shoulder pain.

Depression can develop from the brain injury directly or from having to live with a brain injury. Also noted were posttraumatic stress disorder, conversion reaction, anxiety reaction mixed neurologic reaction and affective disorder.

Mechanism of trauma with brain injuries is hypothesized to be a shearing of axons from the acceleration and deceleration of brain tissue. The outer part of the brain moves at a different pace than the inside. Primate studies determined that shear is worse for rear and frontal impacts than for side impacts. Subdural hematomas can be produced with microhemorrhages developing between 12 and 96 hours after injury.