

Sprain / Strain Recovery Cycle

(This does not account for micro-trauma or connective tissue injuries)

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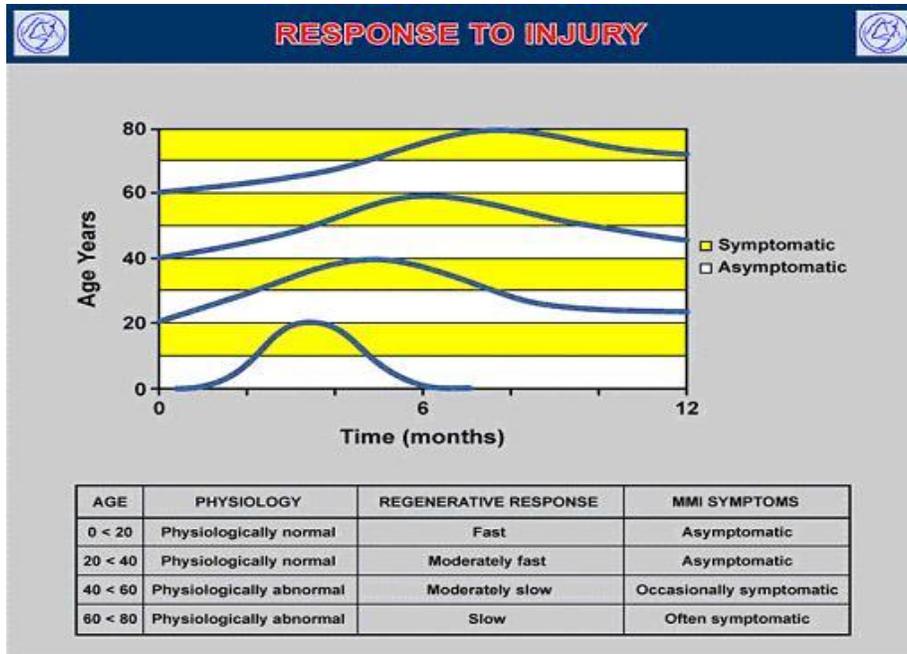
THE MECHANISMS OF SOFT TISSUE HEALING FOLLOWING WHIPLASH

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I was recently retained as an expert for a personal-injury case, in which a vehicle was struck from the driver's side and forced the victim's car to hit the curb. This occurred while the vehicle was moving; this collision would be defined as a double impact.

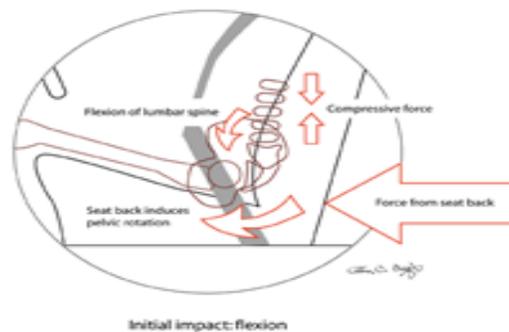
While the property damage was considered "minor", the victim a 37 year old male (Front seat passenger) was ejected from his seat and caused his head to strike the windshield thus, throwing back into the passenger seat.

The peer review doctor stated that this victim should have only received limited treatment because the patient's acute phase of injury repair was 48 hours and did not require more treatment; he recommended 6 treatments max.



RANGES OF RECOVERY ON A 12 MONTH SCALE

The victim sustained an L5/S1 disc herniation; this would obviously require more than 6 visits. Flattening of the spine with compression would occur when the victim slammed back into the seat.



Not only was the peer review wrong, crash tests at Spine Research Institute of San Diego, as well as those of others, have demonstrated a strong vertical acceleration of the lumbar and thoracic spine during the initial phase of a side-impact crash test (i.e., the so-called vertical ramping effect). This produces a large axial compressive force in the lumbar region. This would produce a horizontal (extensile) line of force, and would coincide with ramping and compression, thus creating injury to the lumbar facet joints and soft tissue.

My argument was based on the fact that the human body will not heal in the proposed time the peer review suggested. I provide the studies and references as consideration for your cases and peer reviews you may face.

Phases of Healing

The article I have seen referenced (and personally used in numerous contexts) most often was published in 1986 in the journal *Medicine and Science in Sports and Exercise* by Australian physician John Kellett, MD, and titled (6):

Acute soft tissue injuries—a review of the literature

In this article, Dr. Kellett describes the pathological processes of soft tissue healing as following three distinct phases:

- Phase 1: the acute inflammatory phase
- Phase 2: the repair phase
- Phase 3: remodeling phase

The phases of soft tissue injury repair are:

Phase 1: The Acute Inflammatory or Reaction Phase

This phase of healing lasts up to about 72 hours. It is characterized by vasodilation, immune system activation of phagocytosis to remove debris, the release of prostaglandins and inflammation.

The prostaglandins play a prominent part in pain production and increased capillary permeability (swelling).

The wound is hypoxic because the blood vessels have been disrupted, but immune system macrophages perform their phagocytosis duties anaerobically.

Phase 2: The Repair or Regeneration Phase

This phase begins at about 48 hours and continues for about 6 weeks. This phase is characterized by the synthesis and deposition of collagen, which literally glue the margins of the healing breach together.

The collagen that is deposited in this phase is not fully oriented in the direction of tensile strength. Rather, is laid down in an irregular, non-physiological pattern. Dr. Kellett states:

This phase is “largely one of increasing the quantity of the collagen” but this collagen is not laid down in the direction of stress.

Phase 3: The Remodeling Phase

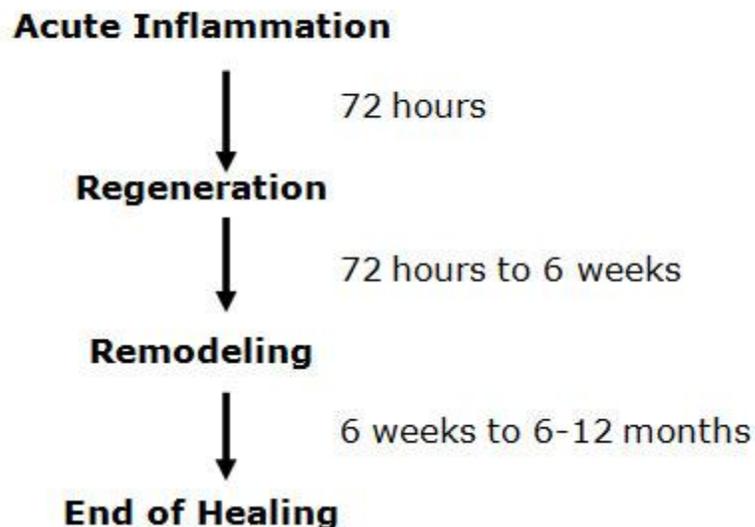
This phase may last up to “12 months or more.” Dr. Kellett stresses that the remodeling phase of healing is critical for establishing the ultimate quality and functional capabilities of the healed tissues. He states:

“The collagen is remodeled to increase the functional capabilities of the tendon or ligament to withstand the stresses imposed upon it.”

“It appears that the tensile strength of the collagen is quite specific to the forces imposed on it during the remodeling phase: i.e. the maximum strength will be in the direction of the forces imposed on the ligament.” [This could argue for the need for specific line-of-drive joint adjustments.]

This phase is largely “an improvement of the quality” (orientation and tensile strength) of the collagen.

Stages of Healing Following Soft Tissue Injury



In support of the healing steps detailed by Dr. John Kellett, Physician Kevin Hildebrand, MD, and colleagues, from the University of Calgary, Alberta, Canada published a study in the journal Sports Medicine Arthroscopic Review, in 2005 titled (14):

The Basics of Soft Tissue Healing and General Factors that Influence Such Healing

Dr. Hildebrand and colleagues point out that wound healing following overt injury to a tissue follows general “rules” irrespective of the tissue involved. These “rules” are similar but somewhat more detailed than those outlined by Dr. Kellett.

They include:

Phase 1: The Inflammatory

Following acute injury there is bleeding into the area of injury and pain. “Hemostasis is restored by the formation of a fibrin clot, which prevents further bleeding and serves as a provisional matrix for migrating cells.” This clotting cascade results in the release of inflammatory molecules and inflammatory cytokines from cells such as platelets. There is an influx of fibroblasts, which sets the stage for the second phase of the repair process.

Phase 2: The Matrix Deposition Phase

The fibroblasts produce collagen proteins that bridge the damaged area with the residual endogenous ligament tissue. “The tissue deposited early after injury appears to be an attempt to bridge the damaged area without regard to what was present before injury.”

Phase 3: The Remodeling Phase

“The remodeling phase is a slow process and is accompanied by alterations not only in matrix remodeling, but also gene expression, cellularity, vascularity, and innervation.” The scar tissue in a ligament “undergoes a protracted process where the initially deposited material seems to be turning over and the organization of collagen fibrils become more oriented along the long axis of the ligament.” “Because the remodeling phase occurs slowly, and may take months (i.e., skin) or years (i.e., tendon and ligament).” [Important]

References

1. Kandel ER, Schwartz JH, Jessell TM; Principles of Neural Science, Elsevier; 2000.
2. Bogduk N; On Cervical Zygapophysial Joint Pain After Whiplash; Spine December 1, 2011; Volume 36, Number 25S, pp. S194–S199.
3. Bogduk N, Aprill C; On the nature of neck pain, discography and cervical zygapophysial joint blocks; Pain; 54; 1993, pp. 213-217.
4. Wallis, BJ, Lord, SM and Bogduk, N (1997). “Resolution of psychological distress of whiplash patients following treatment by radiofrequency neurotomy: a randomized, double-blind, placebo-controlled trial.” Pain; 73: pp. 15-22.
5. Spearing NM, Connelly LB; Is compensation “bad for health”? A systematic meta-review; Injury; January 2011; Vol. 42; No. 1; pp. 15-24.
6. Scholten-Peeters GGM, Verhagen AP, Bekkering GE, van der Windt DAWM, Barnsley L, Oostendorp RAB, Hendriks EJM; Prognostic factors of whiplash-associated disorders: A systematic review of prospective cohort studies; Pain ; July 2003, Vol. 104, pp. 303–322.
7. Spearing NM, Connelly LB, Gargett S, Sterling M; Does injury compensation lead to worse health after whiplash? A systematic review; Pain; June 2012; Vol.153; No. 6; pp.1274-82.
8. Cassidy JD, Carroll LJ, Côté P, Lemstra M, Berglund A, Åke Nygren A; Effect of Eliminating Compensation for Pain and Suffering on the Outcome of Insurance Claims for Whiplash Injury; New England Journal of Medicine; April 20, 2000; Vol. 342; No. 16; pp. 1179-1186.

9. Spearing NM, Connelly LB, Nghiem HS, Pobereskin L; *Journal of Clinical Epidemiology*; November 2012; Vol. 65; No. 11; pp. 1219-1226.
10. Augenstein J, Perdeck E, Bowen J, Stratton J, Horton T, Singer M, Digges K, Malliaris A, Steps J: Dummy measurement of chest injuries induced by two-point shoulder belts. 44th Annual Proceedings of the
11. Association for the Advancement of Automotive Medicine, Chicago, IL, Oct 2-4, 2000, 1-15.
12. Klinghoffer K, Murdock MG. Spondylosis following trauma. *Clin Orth Rel Res*, 1982;166:72-4.
13. Ono K, Kaneoka K, Wittek A, Kajzer J: Cervical injury mechanism based on the analysis of human cervical vertebral motion and head-neck-torso kinematics during low speed rear impacts. 41st Stapp Car Crash Conference Proceedings. SAE paper 973340, 339-356, 1997.
14. Ono K, Kanno M: Influences of the physical parameters on the risk to neck injuries in low impact speed rear-end collisions. International IRCOBI Conference on the Biomechanics of Impact, Eindhoven, Netherlands, 201-212, 1993.
15. Kaneoka K, Ono K, Inami S, Hayashi K. Motion analysis of cervical vertebrae during whiplash loading. *Spine* 24:763-769, 1999.
16. Schulte TL, Lerner T, Hackenberg L, Liljenqvist U, Bullmann V. Acquired spondylolysis after implantation of a lumbar ProDisc II prosthesis: case report and review of the literature. *Spine*, Oct. 15, 2007;32(22):E645-8.
17. Green TP, Allvey JC, Adams MA. Spondylolysis: bending of the inferior articular processes of lumbar vertebrae during simulated spinal movements. *Spine*, 1994;19(23):2683-91.
18. Cyron BM, Hutton WC, Troup JD. Spondylolytic fractures. *J Bone Joint Surg (British)*, November 1976;58-B(4):462-6.
19. Cyron BM, Hutton WC. The fatigue strength of the lumbar neural arch in spondylolysis. *J Bone Joint Surg (British)*, May 1978;60-B(2):234-8.
20. Kitagawa Y, Yasuki T, Hasegawa J. Consideration of possible indicators for whiplash injury assessment and examination of seat design parameters using human FE model. 20th International Conference of the Enhanced Safety of Vehicles (ESV), Lyon, France, 2007.
21. Croft AC. *Whiplash and Mild Traumatic Brain Injuries: A Guide for Patients and Practitioners*. Coronado: SRISD Press; 2009.
22. Viano DC. Seat properties affecting neck responses in rear crashes: a reason why whiplash has increased. *Traffic Inj Prev*, September 2003;4(3):214-27