

## Connective Tissue Repair and Residuals

Last week I was preparing a doctor for a deposition. We were discussing the science surrounding wound healing and the importance of educating a fact finder about the likely sequelae. The case happened to be on a patient of ours that was seriously injured in a significant impact MVA but was discharged at one year without an impairment because she had a negative exam and was essentially asymptomatic for the prior 8 months with the exception of an occasional brief flareup. There was no ratable condition according to the AMA Guides and the patient insisted she was doing fine during their final evaluation. However, we knew that the underlying injury would result in complications in the future due to the fundamental nature of connective tissue repair.

I decided to write a primer for attorneys with information that could be used to examine or cross-examine a witness. As I started writing, I had a *deja'vu* moment, and I found a newsletter that I had written in 2013 on the topic which was already a good primer. I encourage you to go back to our website and read that newsletter first ([link](#)).

In this newsletter I will review some of the prior topics and hopefully give you some greater insight into the importance of wound repair in the prognosis of your clients' connective tissue injuries, with or without a numerical rating according to the AMA Guides.

A healing wound is an expression of an intricate sequence of cellular and biochemical responses directed toward restoring tissue integrity and functional capacity following injury. Wound healing has four overlapping phases: (a) Hemostasis, (b) Inflammation, (c) Proliferation and (d) Remodeling/Maturation. To avoid confusion, Hemostasis and Inflammation phases are often considered phase I rather than two distinct phases resulting in either or 4 phases in the literature.

### **HEMOSTASIS – Begins immediately after injury**

Hemostasis literally means the stopping of a flow of blood. During this stage, the injury site is being closed by clotting. Hemostasis starts with blood vessels constriction followed by deposition of platelets to seal the wound. Coagulation continues using threads of fibrin (connective tissue).

### **ACUTE INFLAMMATION PHASE – First 72 hours**

The inflammation phase proceeds concurrently with hemostasis. In this phase the injured tissues leak water, salt, and protein called transudate which causes swelling and triggers a chemical and cellular response. White blood cells, growth factors, nutrients and enzymes infiltrate the wound and create pain, redness, swelling and heat (*dolor, rubor, tumor, and calor*)

### **PROLIFERATION/REPAIR PHASE - 48 hours to 6 months**

During proliferation, the wound is rebuilt with new connective tissue, including mostly collagen and additional cells called extracellular matrix. During this phase, the wound contracts as new tissues are built. In addition, a new network of nerve fibers and blood vessels must be constructed.

### **REMODELING / MATURATION PHASE – 3 Weeks to 12 months**

The remodeling phase is when collagen matures, and the wound fully closes. The collagen laid down during the proliferation stage is disorganized, dense and thick. As implied by the name, remodeling is when the body attempts to better organize and align the collagen fibers to adapt to the stresses applied during normal biomechanics.

Tissue types:	Range of time for healing:
Muscle	Grade I: 2-4 weeks Grade II: 8-16 week Grade III: 9-12 Months
Tendon	Acute: 2-6 weeks Sub-acute: 2-4 Months Chronic: 3-12 Months
Bone	6-12 weeks
Ligaments	Grade I: 2-8 weeks Grade II: 2-6 Months Grade III: 6-12 Months
Cartilage	9-24 months
Nerve	Regrowth rate: 2-4 mm/day

There are many factors that affect the healing process including age, sex hormones, stress, systemic issues (i.e. Diabetes or Thyroid disease), obesity, medications such as steroids and NSAIDS (both which interfere with healing by suppressing cellular response), alcohol, smoking, nutrition and more. Many of these can have an adverse effect on healing but some recent advances in improving wound healing include collagen supplementation, hyperbaric oxygen, Class III and Class IV lasers, various forms of electrical stimulation and more.

As discussed in my 2013 newsletter, there are some critical concepts to understand. Connective tissues heal through a sequence of cellular and chemical events that occur through consecutive phases. The whole process can occur over many months, and despite advances in therapeutics, many tissues do not regain their normal tensile strength. Connective tissue injuries create disruptions in the balance between joint mobility and joint stability, which can lead to abnormal transmission of forces throughout the joint, resulting in damage to other structures in and around the joint.

Connective tissues such as ligaments are composed of collagen which represents 75% of the dry weight. In uninjured tissues the dominant collagen is Type I representing approximately 85% of the total collagen. During tissue repair, the collagen deposited is predominantly Type III and to a lesser extent Type II. These latter types of collagens are of lesser strength and quality and do not have the elasticity or cross bridging capabilities of the replaced Type I collagen. As a result, at the completion of tissue repair the “repaired” tissues are not as functional as the original tissues and have morphological changes that result in the following:

1. Increased likelihood of reinjury
2. Increased likelihood of flare-up while doing routine activities
3. Lowered pain threshold (denervation supersensitivity)

At the completion of their rehabilitation, many patients are asymptomatic and do not have positive exam findings. Assigning a permanent impairment using the AMA Guides is not possible. However, the underlying integrity of the “repaired” tissues remains a concern and should be addressed in some manner.

Many years ago, I would add a statement like the one below to my final reports so that it was understood that the lack of an AMA Guides impairment does not mean that the patient is 100% back to normal

*“At this time, the patient is reporting reduced symptoms and is being discharged from active care to an independent home exercise program. However, it should be noted that connective tissues injured in this traumatic event heal with scar tissues that are composed of lower quality Type II and Type III collagen. This replacement tissue is disorganized, less elastic, and denser than the original tissues. The loss of elasticity and fiber disorganization of the scarred repair tissues leaves this patient more prone towards re-injury and flare-ups. Complicating this further is the increased scar density which results in the healing body overcompensating in its attempt to re-populate the area with new nerve innervation. The reinnervation is deterred by the mature scar which results in augmentation of the body to sprout excessive afferent (sensory) fibers for pain and position. The resulting hyperinnervation lowers the pain threshold of the damaged tissues and increases the likelihood of altered body mechanics increasing the likelihood of reinjury..”*

Years ago, attorneys found statements like this to be too complex and asked for more brevity. Therefore, we now simply report that “the nature of these types of injuries increases the likelihood of future flareups” However, I think the concepts in the above statement and the data I provided in this and the prior newsletter should be known to the attorney. By being familiar with these concepts the attorney can more effectively examine the doctor and bring out the important facts that a jury needs to hear to properly assess the magnitude of their clients’ injuries and their future prognosis.