Prolotherapy: What is it and how can it help my athlete?

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61st EATA
BOSTON, MA
JANUARY 11, 2009
My Goals Today

- Provide Better Appreciation of the use of Non-Surgical Musculoskeletal Physicians
- Introduce Prolotherapy
- Understanding the basics of healing
- Role of Prolotherapy in care of athletes
- Introduce thought that NSAIDS may not be good for treating injuries
Sports Medicine Is NOT ALL SURGICAL

Only a fraction of Musculoskeletal sports medicine Injuries require surgery...

By Walter R. Fontera (Editor), Lyle J. Micheli (Editor), Stanley A. Herring (Editor), Julie K. Silver (Editor), 2005
Does the Injury need Surgery?

- About 95% of sports injuries are minor soft tissue traumas.
- Sports injuries result from acute trauma or repetitive stress associated with athletic activities. Sports injuries can affect bones or soft tissue such as ligaments, muscles, and tendons.
- Sprains account for one-third of all sports injuries. A sprain is a partial or complete tear of a ligament, a strong band of tissue that connects bones to one another and stabilizes joints.
Does the Injury need Surgery?

☑ It had been estimated that only 1 of 10 individuals that are seen by an Orthopedic Surgeon require Surgery.

☑ It has also been extibalte that 95% of all Sports Related Injuries are “minor” soft tissue (Tendon, Ligament or Muscle) Injuries.
“Conventional” Treatment

① R-Rest
② I-Ice
③ C-Compression
④ E-Elevation
⑤ M-MRI
“Conventional” Treatment
“Conventional” Treatment

Rest is good

Rest is not always good.

Acute and Chronic pain in the back and joints mostly involves the ligaments, tendons, and muscles which become stretched, torn, and weakened.

Resting them while they are injured encourages stagnation of blood in damaged tissue resulting in increased swelling and scar tissue formation.

The longer movement is restricted, the longer it will take to heal. For each day of non-movement, two days are added to the length of rehabilitation...

...BUT WE KNOW THIS!
“Conventional” Treatment

Inflammation is bad

Wrong, inflammation is good!
The human body heals through inflammation.

For example, inflammation is necessary to build muscle when the muscle tissue breaks down during exercise. This is why exercising hurts.

No pain, no gain would be better said no pain, no inflammation, no gain."

The pain during a tough workout is a result of the muscles becoming inflamed and it is inflammation that makes the muscles healthier and stronger.

BEST EXAMPLE-A FINGER LACERATION
“Conventional” Treatment

Ice is nice.

Nothing will stop the inflammatory process quicker than ice. Ice **DECREASES CIRCULATION** to the area of injury thereby allowing **FEWER** immune cells to clean up the injured site and lay down new collagen tissue needed to make new ligaments, tendons, and muscle.

OK so why iced saline for presumed spinal cord injuries?
“Conventional” Treatment

Anti-inflammatory medication is good.

Anti-inflammatory medications, by retarding the healing process, make re-injury and chronic pain much more likely in the future.

Not to mention-elevated blood pressure, renal failure, delayed bone healing* and acute gastrointestinal bleeding
### Table 5.4 Type A reactions to NSAIDs

<table>
<thead>
<tr>
<th>Organ/system</th>
<th>Clinical reaction</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood</td>
<td>Decreased platelet adhesiveness</td>
</tr>
<tr>
<td>Gastro-intestinal tract</td>
<td>Dyspepsia</td>
</tr>
<tr>
<td></td>
<td>Haemorrhage</td>
</tr>
<tr>
<td></td>
<td>Peptic ulceration</td>
</tr>
<tr>
<td></td>
<td>Perforation</td>
</tr>
<tr>
<td>Kidney</td>
<td>Salt and water retention</td>
</tr>
<tr>
<td></td>
<td>Interstitial nephritis</td>
</tr>
<tr>
<td>Lung</td>
<td>Bronchospasm</td>
</tr>
</tbody>
</table>

### Table 5.5 Type B reactions to NSAIDs

<table>
<thead>
<tr>
<th>Organ/system</th>
<th>Clinical reaction</th>
<th>NSAIDs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Immuneological</td>
<td>Anaphylaxis</td>
<td>Most NSAIDs</td>
</tr>
<tr>
<td>Skin</td>
<td>Morbilliform rash</td>
<td>Fenbufen</td>
</tr>
<tr>
<td></td>
<td>Angioedema</td>
<td>Ibuprofen</td>
</tr>
<tr>
<td>Blood</td>
<td>Thrombocytopenia</td>
<td>Azapropazone</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Piroxicam</td>
</tr>
<tr>
<td></td>
<td>Haemolytic anaemia</td>
<td>Mefenamic acid</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Diclofenac</td>
</tr>
<tr>
<td></td>
<td>Agranulocytosis</td>
<td>Phenylbutazone</td>
</tr>
<tr>
<td>Gastro-intestinal</td>
<td>Diarrhoea</td>
<td>Phenylbutazone</td>
</tr>
<tr>
<td>Liver</td>
<td>Reye’s syndrome</td>
<td>Mefenamic acid</td>
</tr>
<tr>
<td></td>
<td>Hepatitis</td>
<td>Diclofenac</td>
</tr>
<tr>
<td>CNS</td>
<td>Aseptic meningitis</td>
<td>Aspirin</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Diclofenac</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Piroxicam</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Ibuprofen</td>
</tr>
</tbody>
</table>
Some painkilling drugs may delay or even prevent the healing of fractures, researchers have warned.

"It's time to tell the public," says Thomas Einhorn, an orthopaedic surgeon at Boston University Medical Center "It would seem that a prudent approach is to temporarily avoid the use of these drugs during bone healing."

The main concern is the new generation of non-steroidal anti-inflammatory drugs (NSAIDs). When Patrick O'Connor's team at the University of New Jersey gave these newer painkillers to rats their broken bones did not fully heal. Both drugs, rofecoxib (sold as Vioxx) and celecoxib (Celebrex), are often used to ease the pain of broken bones.
NSAIDS Delay or Block Healing?

But for over 20 years there have been occasional reports of impaired bone healing in patients taking NSAIDs. The issue may have escaped attention because the older generation of NSAIDS, such as ibuprofen and indomethacin, only appear to delay healing by a few weeks instead of blocking it. Aspirin is one of the few NSAIDs that appears to kill pain without this side effect.

Ibuprofen and indomethacin delay bone healing by about one to two weeks in rats, which is the equivalent to slowing it down by 25 to 50 per cent in humans," says O'Connor. None of the rats treated with rofecoxib managed to heal their bones. In those treated with celecoxib, none managed to completely heal their bones but about half had some form of bone regrowth.

Traditional NSAIDs inhibit the enzymes cox-1 and cox-2. Cox-2 catalyses the production of hormone-like chemicals known as prostaglandins involved in inflammation, while cox-1 has a variety of roles not specific to the inflammatory response. Since the new generation of NSAIDs such as rofecoxib block cox-2 almost exclusively, it was hoped they would have fewer side effects.

But it now seems that cox-2 may be crucial in helping bone-forming stem cells and growth factors do their work. This area now needs to be investigated urgently, says Jeremy Saklatvala of the Kennedy Institute of Rheumatology in London. "In the meantime, people with healing fractures should steer clear of these drugs."

Journal reference: *Journal of Bone and Mineral Research* (vol 17, p 963)
“Conventional” Treatment

So are we wrong in what we do?
“Conventional” Treatment

Are we Inhibiting or Delaying Healing?
QUICK LOOK AT THE TISSUE HEALING PROCESS
Wound healing is a complex scheme. Coordinated by a sequential & temporal cascade of events. Healing cascade follows the same sequence of repair irrespective of the initial insult type.
Tissue Healing

Three phases of wound healing

- INFLAMMATION: homeostasis/matrix
- GRANULATION: angiogenesis/collagenesis
- MATURATION: collagen remodeling

Cellular orchestration of stimulation, inhibition, proliferation, communication through chemotaxis, mitogenosis, phagocytosis...
Quick Look at Inflammation

Tissue injury → Phospholipids → Arachidonic acid

Leukotrienes
- Bronchoconstriction

COX-1 (Constitutional)

COX-2 (Inducible)

Indicators
- Cytokines
- Growth factors

Inhibitors
- COX-2 inhibitors
- NSAIDS (non–COX-2)
- Aspirin

Inhibitors
- NSAIDS (non–COX-2)
- Aspirin

Cytoprotective prostaglandins
- Protect gastric mucosa
- Aid platelet aggregation

Inflammatory prostaglandins
- Recruit inflammatory cells
- Sensitize skin pain receptors
- Regulate hypothalamic temperature control

FIGURE 1. Algorithm of the biochemical pathway shows that the formation of prostaglandins occurs via both cyclooxygenase enzymes (COX-1 and COX-2).
Cells Involved in Wound Healing

- Platelets
- Neutrophils
- Macrophages
- Lymphocytes
- Fibroblasts
- Capillaries

Days
<table>
<thead>
<tr>
<th>Cytokine</th>
<th>Cell of Origin</th>
<th>Function</th>
</tr>
</thead>
<tbody>
<tr>
<td>PDGF</td>
<td>Platelets, Macrophages, Endothelial cells</td>
<td>Cell chemotaxis, Mitogenic for fibroblasts, Stimulates angiogenesis, Stimulates wound contraction</td>
</tr>
<tr>
<td>TGF-alpha</td>
<td>Macrophages, T lymphocytes, Keratinocytes</td>
<td>Mitogenic for keratinocytes and fibroblasts, Stimulates keratinocyte migration</td>
</tr>
<tr>
<td>TGF-beta</td>
<td>Platelets, T lymphocytes, Macrophages, Endothelial cells, Keratinocytes</td>
<td>Cell chemotaxis stimulates angiogenesis and fibroplasia</td>
</tr>
<tr>
<td>EGF</td>
<td>Platelets, Macrophages</td>
<td>Mitogenic for keratinocytes and fibroblasts, Stimulates keratinocyte migration</td>
</tr>
<tr>
<td>Fibroblast growth factor</td>
<td>Macrophages, Mast cells, T lymphocytes, Endothelial cells</td>
<td>Chemotactastic and mitogenic for fibroblasts and keratinocytes, Stimulates angiogenesis</td>
</tr>
<tr>
<td>Keratinocyte growth factor</td>
<td>Fibroblasts</td>
<td>Stimulates keratinocyte migration, differentiation, and proliferation</td>
</tr>
<tr>
<td>TNF</td>
<td>Macrophages, Mast cells, T lymphocytes</td>
<td>Activates macrophages, Mitogenic for fibroblasts, Stimulates angiogenesis</td>
</tr>
<tr>
<td>Interleukin (IL)-1, IL-2, IL-6, and IL-8</td>
<td>Macrophages, Mast cells, Keratinocytes, Lymphocytes</td>
<td>IL-1 - Induces fever and adrenocorticotropic hormone release, enhances TNF-alpha and interferon (IFN)-gamma, activates granulocytes and endothelial cells, and stimulates hematopoiesis IL-2 - Activates macrophages, T cells, natural killer cells, and lymphokine-activated killer cells; stimulates differentiation of activated B cells; stimulates proliferation of activated B and T cells; and induces fever IL-6 - Induces fever and enhances release of acute-phase reactants by the liver IL-8 - Enhances neutrophil adherence, chemotaxis, and granule release</td>
</tr>
<tr>
<td>INFs (IFN-alpha, -beta, and -delta)</td>
<td>Lymphocytes, Fibroblasts</td>
<td>Activate macrophages, Inhibit fibroblast proliferation</td>
</tr>
<tr>
<td>Thromboxane A2</td>
<td>Destroyed wound cells</td>
<td>Potent vasoconstrictor</td>
</tr>
</tbody>
</table>
**PHASE ONE**

Injury $\rightarrow$ Inflammation $\rightarrow$ Damage Cell Membranes/Vessels release:
- Thromboxane A2 (from platelets)
- Prostaglandin 2a $\rightarrow$ vasodilatation $\rightarrow$ membrane permeability
- Epinephrine $\rightarrow$ boost blood supply and glucose

Platelets $\rightarrow$ alpha particles from degranulated platelets $\rightarrow$ initial clot $\rightarrow$
- Fibrinogen
- Fibronectin
- Thrombospondin
- Von Willebrand factor

$\rightarrow$ Platelets $\rightarrow$ release PAF (platelet activating factor)
- PDGF-platelet derived growth factor
- TGF beta (transforming growth factor)
- FGF beta (Fibroblast growth factor)
- IGF) Insulin like growth factor

$\rightarrow$ Platelets chemotactic for neutrophils then monocytes
- PAF $\rightarrow$ Neutrophils $\rightarrow$ elastase and collagenase
- Neutrophils attract macrophages
- Monocytes $\rightarrow$ TNF (Tumor necrosis factor) & PDGF
- Angiogenesis and fibroblastic activity

INTEGRIN-cellular adhesion receptors $\rightarrow$ provisional matrix
PHASE TWO-Granulation-Cell Migration, Activation and proliferation

Granulation Tissue consists of
* NEW VESSEL FORMATION
* MATRIX FORMATION
* COLLAGENESIS via Fibroblasts

Macrophages→Monocytes

Macrophage is primary source of PDGF, TGF Beat, IL-8 and FGF
→ Granulation milieu→fibroblast produces collagen
→ Fibroblasts come from EXTRA CELLULAR MATRIX

This is done via:
+ Intergens
+ Fibronectin
+ Hyaluronic Acid (Sound familiar—Synvisc?)
+ Protyoglycans
+ Fibrin

→ Once fibroblast in the site→matrix builder to protein synthesizer→Collagen

ALL DOWN HILL FROM THERE...

...Complete Ligament and Tendon Repair
**FIGURE 1. Prostaglandin and thromboxane biosynthesis.**

COX = cyclooxygenase; coxibs = COX-2 inhibitors; PG = prostaglandin; TxA₂ = thromboxane A₂; NSAID = nonsteroidal anti-inflammatory drug; ASA = aspirin.

![Prostaglandin and thromboxane biosynthesis diagram]

**FIGURE 7. Effects of NSAIDs on platelets and endothelium.**

Platelet

- COX-1
- Non specific NSAIDs/ASA
- Coxibs
- Thromboxane (TxA₂)
- Vasoconstrictor
- Promotes platelet aggregation
- Hemostasis
- Thrombosis

Endothelial Cell

- COX-1
- COX-2
- Prostacyclin (PGI₂)
- Vasodilator
- Inhibitor of platelet aggregation
SO DO ICE AND NSAIDS HELP?
NOW LETS GET TO WHY I AM HERE!
PROLO THERAPY
What is Prolotherapy?

First, it is important to understand what the word **prolotherapy** itself means. "Prolo" is short for **proliferation**, because the treatment causes the proliferation (growth, formation) of new ligament tissue in areas where it has become weak.
Injecting an otherwise non-pharmacological and non-active irritant solution into the body, generally in the region of tendons or ligaments for the purpose of strengthening weakened connective tissue.
Historical review shows that a version of this technique was first used by Hippocrates on soldiers with dislocated, torn shoulder joints. He would stick a hot poker into the joint, and it would then miraculously heal normally.
History of Prolotherapy

Injections of irritant solutions were performed in the late 1800’s to repair hernias and in the early 1900’s for jaw pain due to joint laxity. George S. Hackett developed the technique of prolotherapy in the 1940’s. Gustav Hemwall was a pioneer, beginning his studies and treatments in the 1950s and continuing until the mid 1990s. In his study of almost 10,000 prolotherapy cases, Dr. Hackett found that over 99 percent of the patients found relief from their pain.
How does Prolotherapy Work?

Prolotherapy works by exactly the same process that the human body naturally uses to stimulate the body's healing system, a process called inflammation. The technique involves the injection of a proliferant (a mild irritant solution) that causes an inflammatory response which "turns on" the healing process.
How does Prolotherapy Work?

The growth of new ligament and tendon tissue is then stimulated. The ligaments and tendons produced after Prolotherapy appear much the same as normal tissues, except that they are thicker, stronger, and contain fibers of varying thickness, testifying to the new and ongoing creation of tissue. The ligament and tendon tissue which forms as a result of Prolotherapy is thicker and stronger than normal tissue, up to 40% stronger in some cases!
Left, controls. Right, proliferated. The tendons on the right reveal an increase in diameter of 40 percent, which is estimated to double the strength of the tendon. The upper portion reveals the attachment of the ligament to the bone, which has increased 30 percent in diameter. The proliferating solution stimulates the production of new fibrous connective tissue cells, which become organized into permanent non-elastic fibrous tissue.
How does Prolotherapy Work?

Prolotherapy works by raising growth factor levels to promote tissue repair and growth of new tissue. Growth factors typically act as signaling molecules between cells. Examples are cytokines and hormones that bind to specific receptors on the surface of their target cells.

What are Growth Factors?
The term growth factor refers to a naturally occurring protein capable of stimulating cellular growth, proliferation and cellular differentiation. Growth factors are important for regulating a variety of cellular processes. They often promote cell differentiation and maturation, which varies between growth factors. For example, bone morphogenic proteins stimulate bone cell differentiation, while fibroblast growth factors and vascular endothelial growth factors stimulate blood vessel differentiation (angiogenesis).
Several Well Known Growth Factors

- Transforming growth factor beta (TGF-β)
- Granulocyte-colony stimulating factor (G-CSF)
- Granulocyte-macrophage colony stimulating factor (GM-CSF)
- Nerve growth factor (NGF)
- Neurotrophins
- Platelet-derived growth factor (PDGF)
- Erythropoietin (EPO)
- Thrombopoietin (TPO)
- Myostatin (GDF-8)
- Growth differentiation factor-9 (GDF9)
- Acidic fibroblast growth factor (aFGF or FGF-1)
- Basic fibroblast growth factor (bFGF or FGF-2)
- Epidermal growth factor (EGF)
- Hepatocyte growth factor (HGF)
FIGURE 1. Prostaglandin and thromboxane biosynthesis.
COX = cyclooxygenase; coxibs = COX-2 inhibitors; PG = prostaglandin; TxA<sub>2</sub> = thromboxane A<sub>2</sub>; NSAID = nonsteroidal anti-inflammatory drug; ASA = aspirin.
Back to Inflammation and Healing

FIGURE 1. Algorithm of the biochemical pathway shows that the formation of prostaglandins occurs via both cyclooxygenase enzymes (COX-1 and COX-2).
Cortisone-Most powerful anti-inflammatory versus Prolotherapy

<table>
<thead>
<tr>
<th>PROLOThERAPY VS CORTISONE</th>
<th>PROLOThERAPY</th>
<th>CORTISONE</th>
</tr>
</thead>
<tbody>
<tr>
<td>EFFECT ON HEALING</td>
<td>Enhanced</td>
<td>Inhibited</td>
</tr>
<tr>
<td>EFFECT ON REPAIR</td>
<td>Enhanced</td>
<td>Inhibited</td>
</tr>
<tr>
<td>EFFECT ON COLLAGEN GROWTH</td>
<td>Enhanced</td>
<td>Inhibited</td>
</tr>
<tr>
<td>EFFECT ON TENDON STRENGTH</td>
<td>Enhanced</td>
<td>Inhibited</td>
</tr>
<tr>
<td>EFFECT ON LIGAMENT STRENGTH</td>
<td>Enhanced</td>
<td>Inhibited</td>
</tr>
<tr>
<td>EFFECT ON CARTILAGE GROWTH</td>
<td>Enhanced</td>
<td>Inhibited</td>
</tr>
</tbody>
</table>

To heal an injury, a person needs to receive Prolotherapy.
Proliferants

Irritants

- Directly alter cell surface proteins
- Attach to cell surface at injection site
- Cells rendered reactive to immune system

⇒ INFLAMMATION CASCADE!
What is used in Prolotherapy?

**Anesthetic**
- Lidocaine
- Marcaine
- Procaine

**Proliferants**
- Osmotic
- Irritants
- Sarapin
- Particulates
- Chemotactatics
- Growth Factors
Proliferants

OSMOTIC

- Most commonly used
- **Dextrose and glycerin.**
- Cause a higher osmotic gradient outside of the cells
- Cells to lose water and break.
- Cell particles stimulate an influx of inflammatory cells and initiate the wound-healing cascade to the specific area.
- Water-soluble and thus, very safe.

Other proliferants in this class
- minerals zinc, calcium, and manganese. The minerals are also cofactors for various enzymes. For instance, manganese is needed for the enzyme superoxide dismutase, which helps the antioxidant status of the body. Since some people believe arthritis is from oxidative damage, some Prolotherapists, use manganese in the solution for arthritic patients.
Proliferants

Sarapin

- Alkaline Extract of Pitcher Plant
- How it works → Who knows
- Felt to be due to the Ammonium Sulfate Concentration in the plant or some unidentified biological agent in the plant causing gentle proliferent effect
Proliferants

Particulates

- Most common: Pumice
- Particles attract macrophages
- Macrophages phagocytize the particle → secretion of polypeptide growth factor → collagen tissue growth
Proliferants

Chemotatics

- Direct attraction of immune cells
- Most common: Sodium morrhuate
- Fatty acid component from cod liver oil
- Polyunsaturated fatty acid-
  - Example is Arachidonic Acid
- Direct initiation of immune system
Proliferants

- Polypeptide growth factors act directly upon fibroblasts → Collagen

- Growth hormones being used already
Prolotherapy in Sports

- Majority of Sports Related Injuries are soft tissue
- Soft Tissues are Muscle, Tendon and Ligament
- There is a lack of blood flow to the soft tissues
- Best Blood Flow at the insertion of the tendon or ligament to the periostium of bone or the belly of the muscle
Blood Supply is Key to all Healing and all human function
Blood Supply is Key to all Healing and all Human Function

“The Rule of the Artery Reigns Supreme”
(One would include in this venous (vein) & lymph drainage.)

For healing to occur there needs to be a good blood supply to be able to provide the nutrients and immune cells to the area. Equally, there needs to be effective drainage via the veins and lymph vessels from the area. This is to remove the waste materials and by products of the healing process, such as inflammatory fluids. Inflammation is the source of the pain in most painful conditions

Andrew Taylor Still, M.D., D.O.
OK

So now we want to get to the Meat and Potatoes
Ankle Osteology

- Tibia
- Fibula
- Talus
Foot Osteology

- Talus
- Calcaneous
- Navicular
- Cubid
- Cuneiform
- Metatarsals
- Phalanges
Lateral Ankle Ligaments

- ATF (anterior tibio fibular ligament)
- CF (calcaneofibular ligament)
- PTF (posterior tibio fibular ligament)
Medial Ankle Ligament
Lateral Muscles
Medial Muscles
“Typical” Ankle Sprain

- Inversion
- Injury to ATF-CF-PTF
- Syndesmosis (Distal Tib/Fib-Interosseous Ligament
- Deltoid Medial

Figure 1: Lateral (A) and syndesmotic (B) ligaments of the ankle.
Areas to Treat

- ATF, CF, PTF, deltoid
- Medial and lateral posterior capsules
- Tibiofibular syndesmosis complex
- Interoseous membrane
Plantar Fascitis

- Inflammation of the plantar fascia
- Overuse injury
- Loss of arch
- Posterior Tibial Tendinosis
- Spring Ligament
Areas Treated

- Spring Ligament
- Sinus Tarsi
- Long Plantar Ligament
Multiple studies show that systemic steroids hinder wound (soft tissue) repair:

1. Systemic steroids decrease the number of macrophages by 66% which halted wound repair [Liebovitch & Ross 1975; Stewart 1981; Oates 1988].

2. Klingbeil in 1991 injected mice with systemic steroid five hours prior to wounding which significantly reduced repair, however, was reversed with FGF on the same day.

3. Wahl in 1989 showed that oral steroids reduced the inflammatory response, angiogenesis and fibroblastic synthesis of collagen resulting in a marked wound healing deficit.
Wound (Soft Tissue) Healing Summary

- Wound repair is a complex, well coordinated and multifaceted cellular process that begins with some type of insult that leads to inflammation, granulation and maturation of which these three phases ultimately regenerate healthy, viable collagen at the site of injury.

- Prolotherapy sets the above into action by creating a wound like environment that initiates the healing cascade which effectively treats those patients with musculoskeletal pain, and injuries.
THAT'S GOTTA HURT...
Pittsburgh Steelers running back Derry Wight lies injured on the field Sunday night after he dislocated his right ankle and broke his right leg.