The Therapeutic Management of Healing Tissues

YANSHAN LU

ACKNOWLEDGING SARAH WALDIN & JULIE ROUSE

Introduction

- Knowledge of healing process the foundation for treatment
- an expected reparative process
- skill of the therapist in recognising healing phase and appropriate treatment
- windows of opportunity

windows of opportunity

- windows of opportunity are often missed, they cannot be regained easily since the inevitable sequelae of ever increasing joint stiffness or loss of glide becomes more and more devastating with the passage of time (Fess 1998)

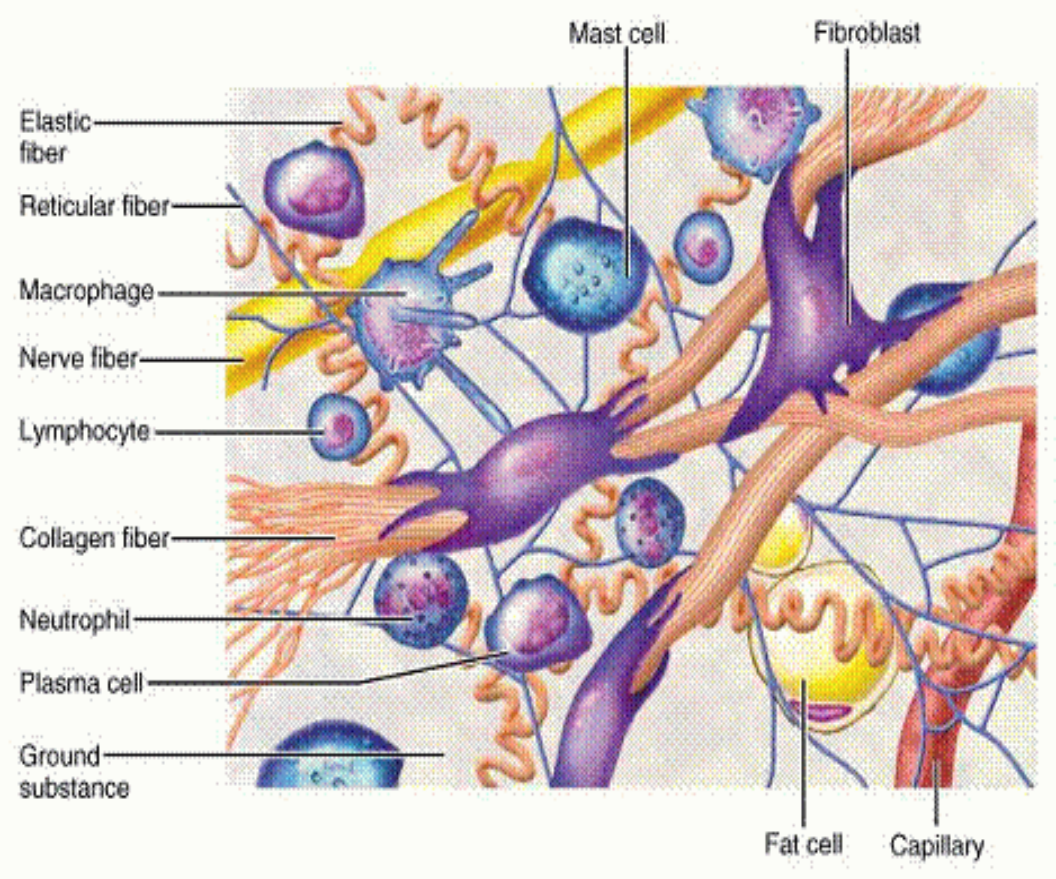
Physiology of Healing

Review and application of basic science

Connective Tissue

- Highly specialised, diverse roles.
- Properties

  determined largely by composition of extracellular matrix (ECM)
  - Fibres: collagen & elastin
  - Proteoglycans(PGs)
  - Glycoproteins



CT Fibres

- Collagen: ability to resist loads, less than 10% elongation under load.
  - 19 types.
  - Triple helix chains of amino acids
  - Aggregate to form fibrils (electron microscope) which aggregate to form fibres (naked eye)
  - Fibril forming collagen (types 1,2,3,5,11)
    - Type 1: bones tendons , ligaments
    - Type 2: cartilage, discs
    - Type 3:extensible tissues, healing tissues

Collagen :triple helix

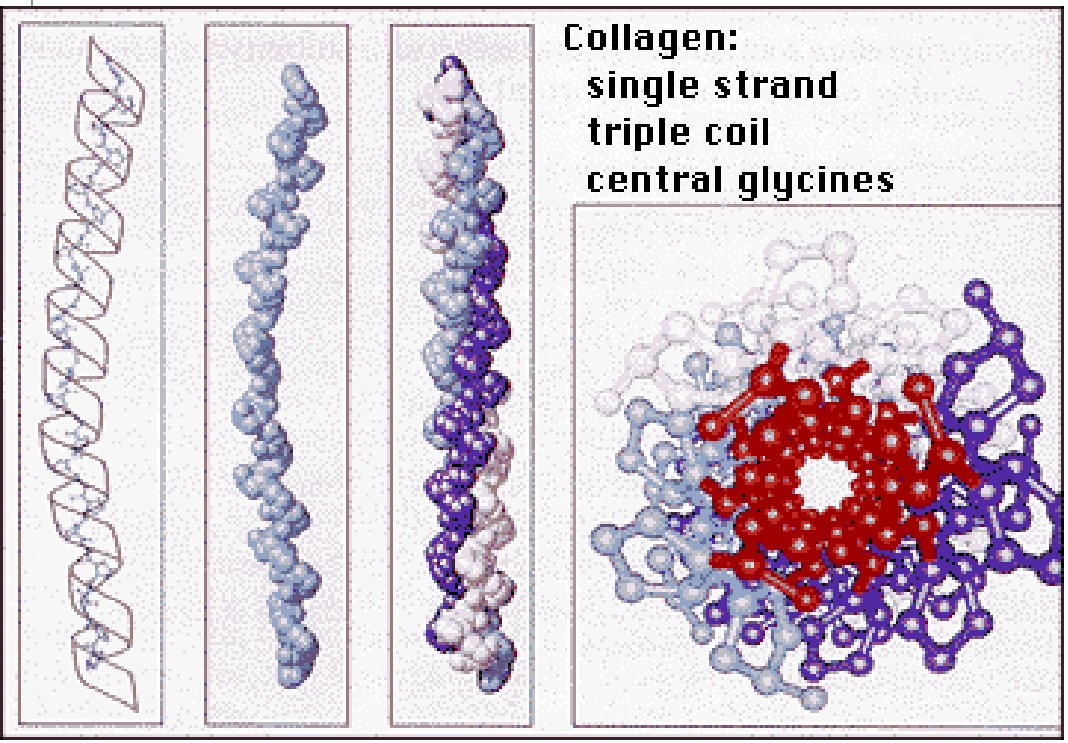
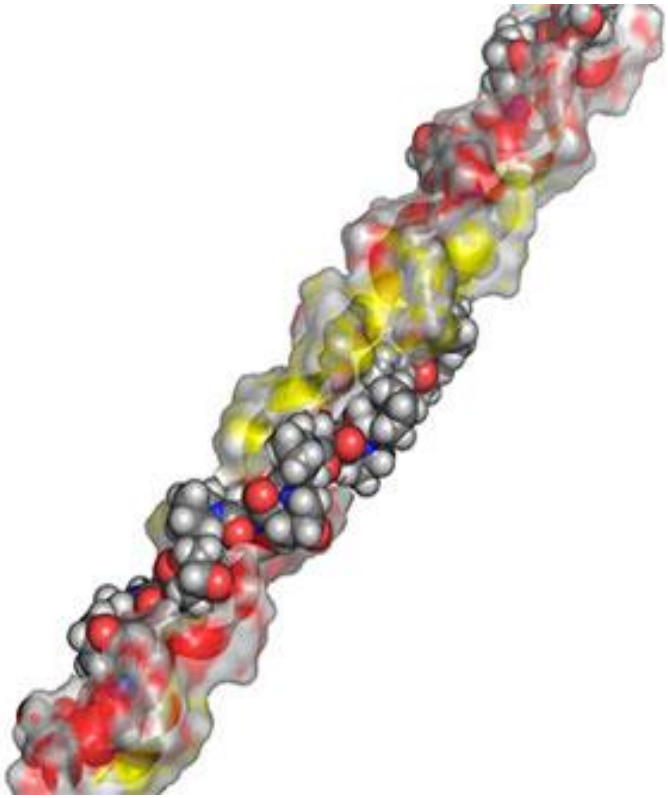


Image found on Web identical to Lehninger textbook image, creator unknown.



collagen crosslinkage

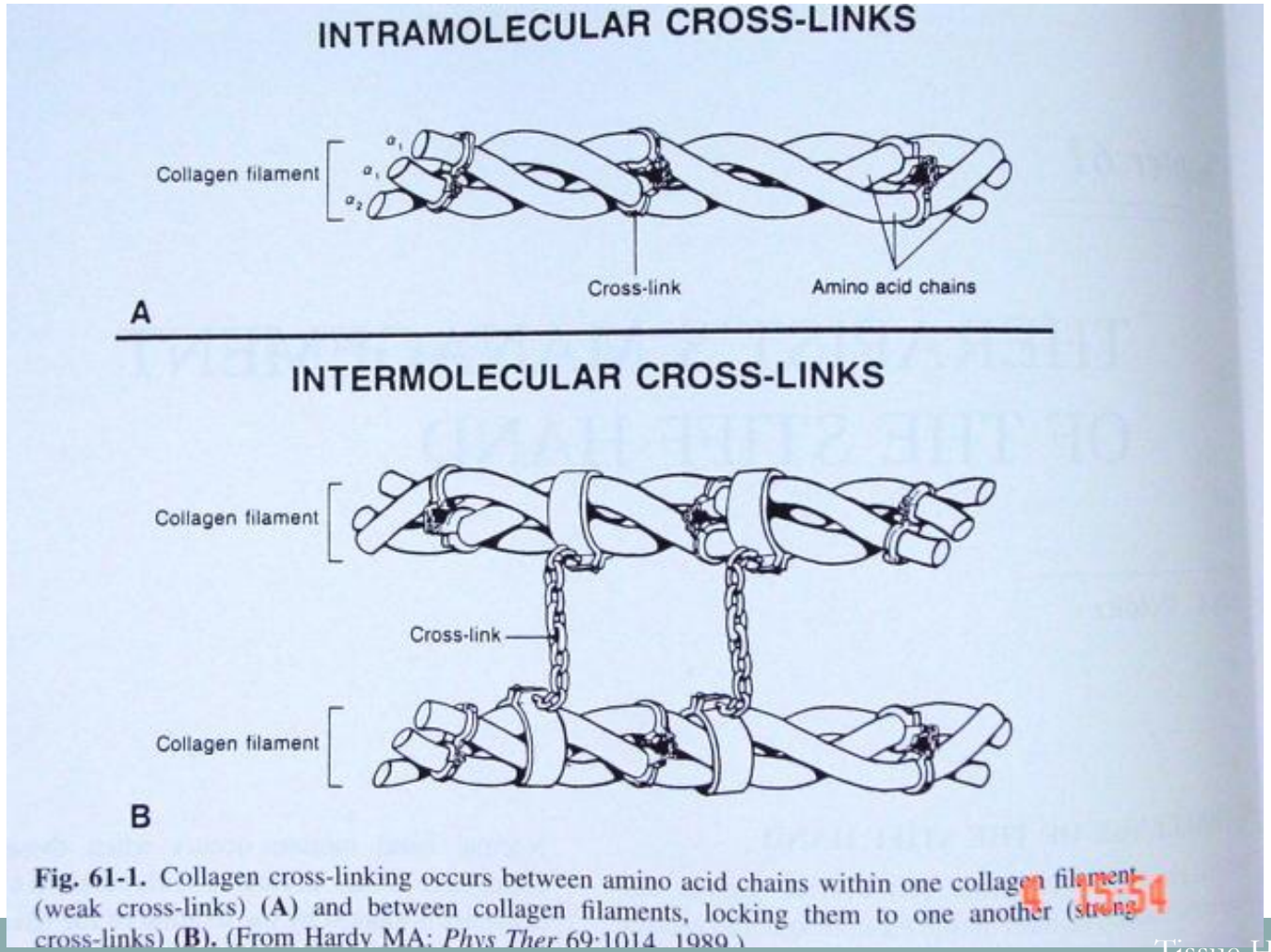
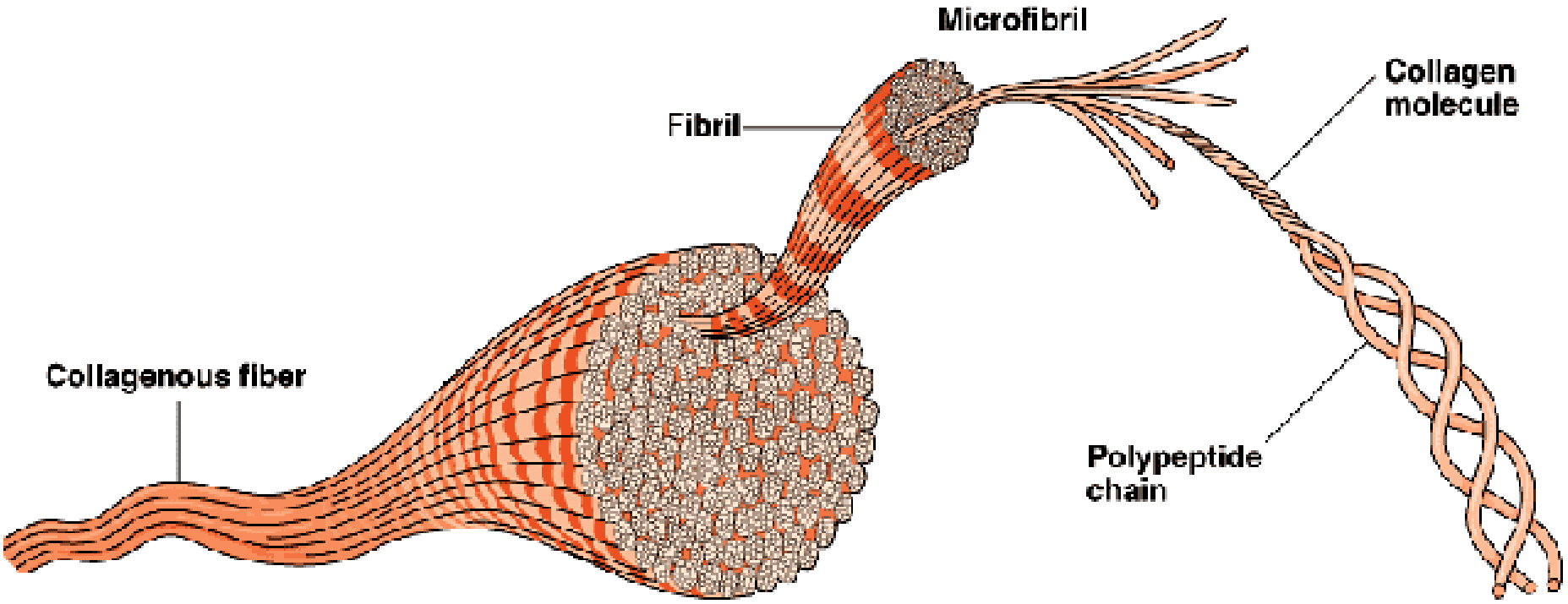
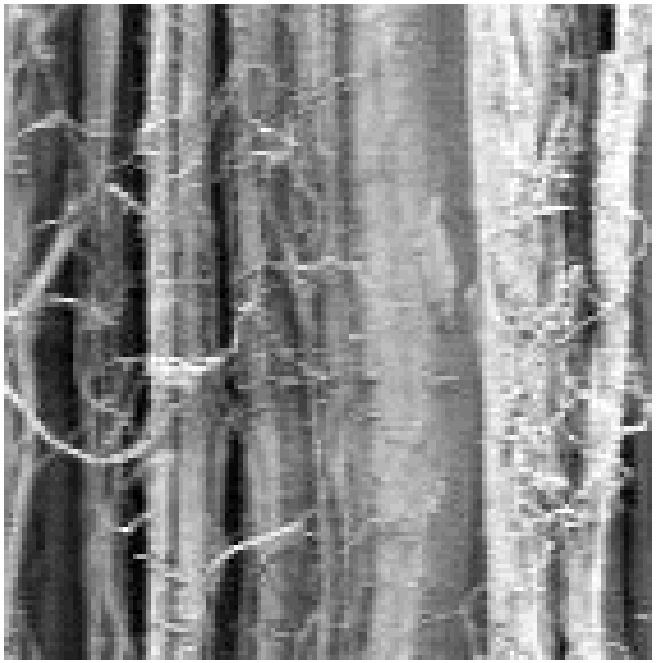


Fig. 61-1. Collagen cross-linking occurs between amino acid chains within one collagen filament (weak cross-links) **(A)** and between collagen filaments, locking them to one another (strong cross-links) **(B).** (From Hardy MA: *Phys Ther* 69:1014, 1989.)

Collagen Fibre



Collagen fibres : ligament



Collagen/Function

- Functions: determined by arrangement and alignment of fibres
  - Limit joint range
  - Transmit forces
  - Impart tensile strength
- Tendons: parallel, unidirectional force
- Ligaments: less parallel, multidirectional forces.
- Insertions: 3 dimensional, distribute load.
- Bone: orthogonal (ply wood), cylinders (osteons)
- Cartilage: type 2, network of bands, between cells

Elastin

- Allows stretch: skin, vessels
  - Elastin core with microfibrils around periphery.
  - Lack of functional regeneration (age probs!)
  - Is synthesised in response to cyclic stretching, injury and some pathology

Proteoglycans (PGs)

- Hydrators, stabilisers and space fillers.
  - Sulphated Glycosominoglycan chains (GAGS) +protein. Ie chondroitin, dermatan.
  - Hyaluronan: atypical structure. H20 absorbing.
- Absorb water through (-) charge.
- %GAG relates to function: high in cartliage (compressive force), low in tendons (tensile)
- CTs respond to changes in applied stress by altering their PG content and type.
- Importance of joint motion +/- loading. (eg tendon over bone)

Connective Tissue responses.

- Normal physiology maintained by balance between synthesis and degradation.
- Pathology: ie OA increase in net degradation.
- Injury: loss of normal tensile loading leads to degradation, repair with type 3 slow progression to type 1.
- exercise/stress loading/ wound tension: increased synthesis.

Connective Tissues: matrix composition and its relevance to Physical therapy. Culav, Clarke, Merrilees .Phys Ther. 1999

the repair cascade

- a continuous process that can be divided into discrete sets of overlapping cellular and biochemical events.
- reparative process of living tissue is both predictable and sequential.
- Each tissue and organ has its own characteristic pattern of response to injury.
- Knowledge of normal connective tissue structure important.

response to tissue injury

FIGURE 1. Consequences of tissue injury. Following injury, a broad spectrum of potential responses, both normal and pathologic, may result. Excess repair may take the form of fibrosis, hyperplasia, or neoplasia, while insufficient repair may affect many tissues.

The Repair cascade

- **Injury / cell death /thrombus/ clot.**
- **Inflammatory or exudative**
- **Proliferative or fibroblastic.**
- **Remodelling**

the repair ladder

.*Injury:cell death*

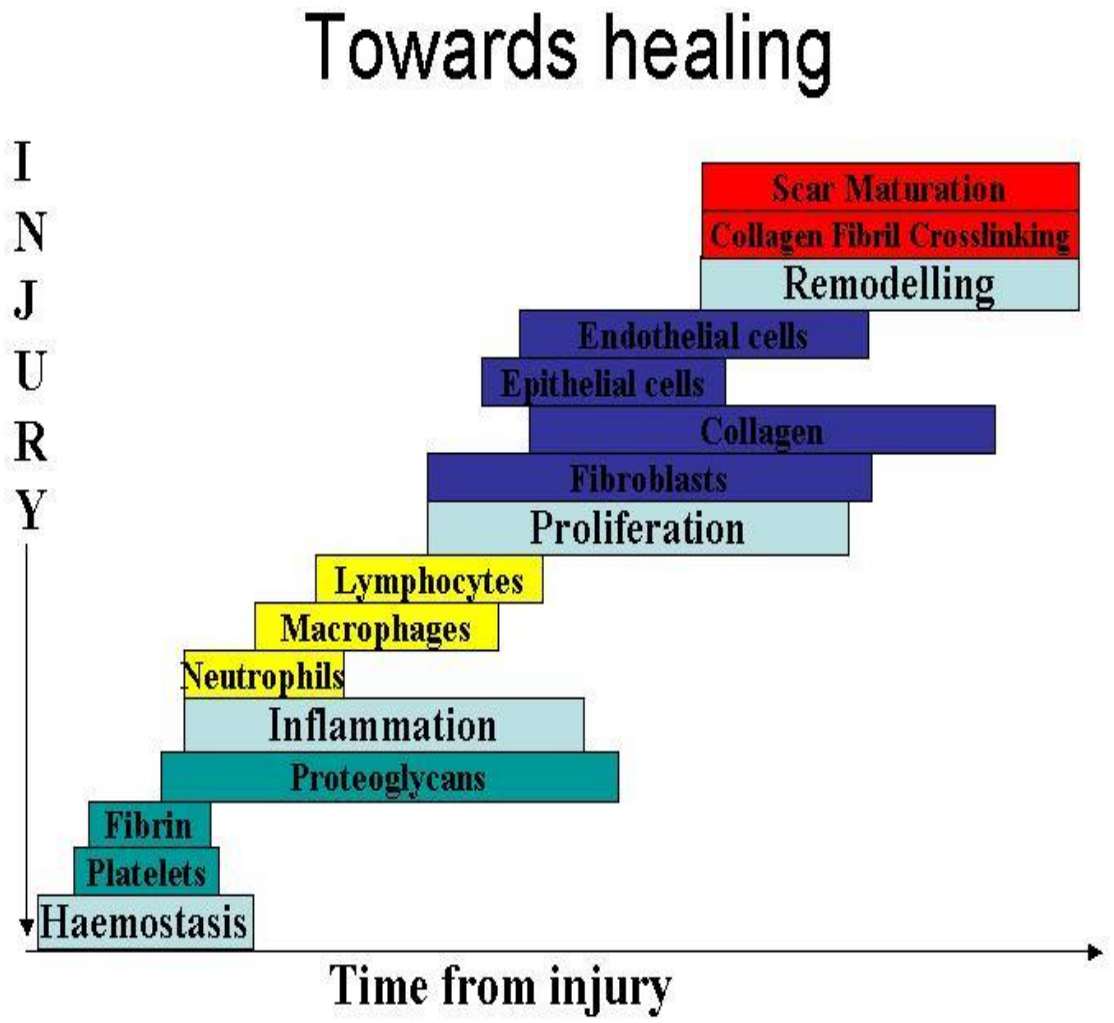
Haemostasis

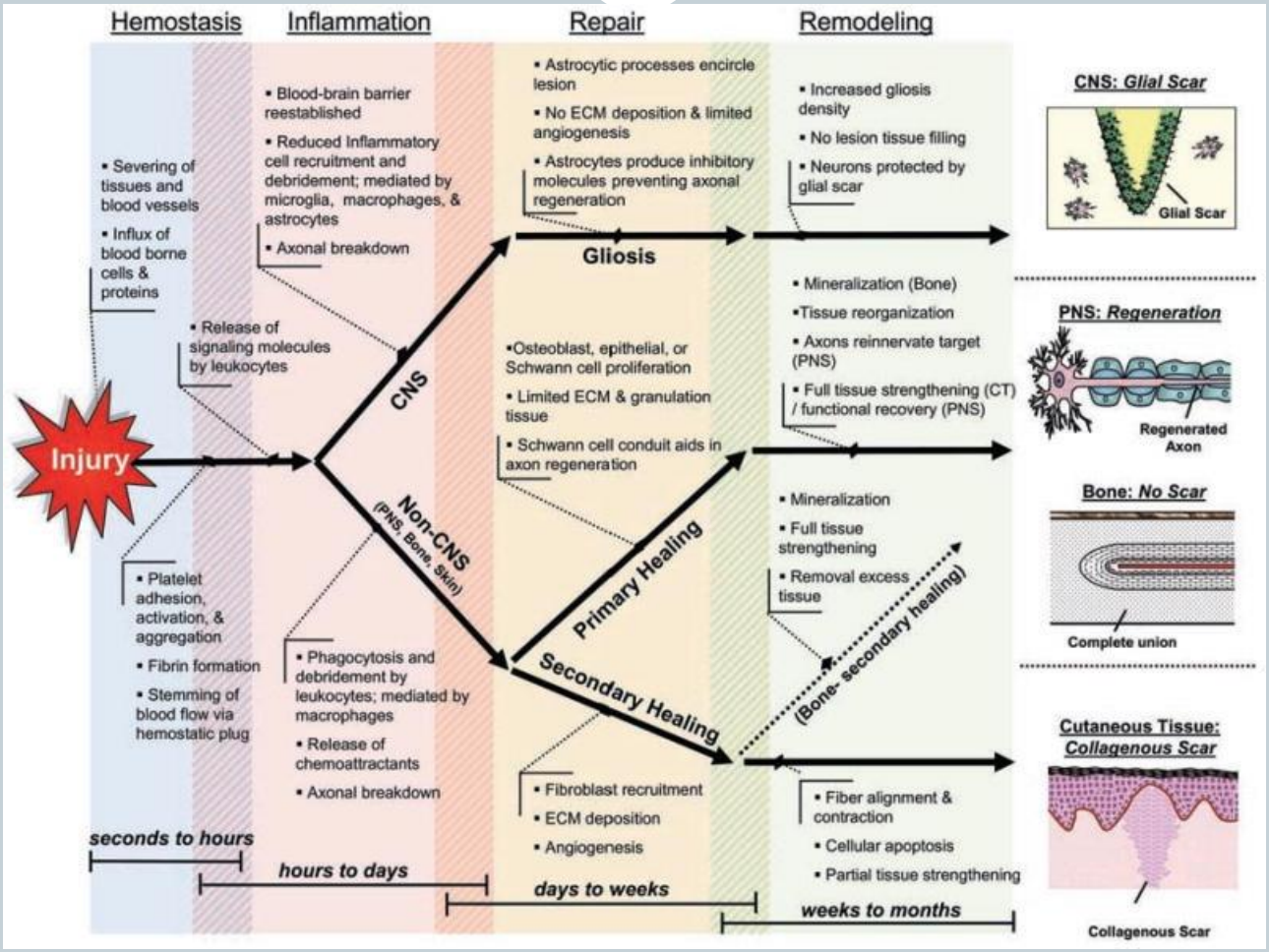
thrombus/ clots.

Inflammatory or exudative

.Proliferative or fibroblastic.

.Remodelling




Hemostasis
Inflammation
Repair
Remodeling
Injury
CNS
Non-CNS
(PNS, Bone, Skin)
Primary Healing
Secondary Healing
(Bone- secondary healing)
Gliosis
• Severing of tissues and blood vessels
• Influx of blood borne cells & proteins
• Release of signaling molecules by leukocytes
• Platelet adhesion, activation, & aggregation
• Fibrin formation
• Stemming of blood flow via hemostatic plug
• Blood-brain barrier reestablished
• Reduced Inflammatory cell recruitment and debridement; mediated by microglia, macrophages, & astrocytes
• Axonal breakdown
• Phagocytosis and debridement by leukocytes; mediated by macrophages
• Release of chemoattractants
• Axonal breakdown
• Astrocytic processes encircle lesion
• No ECM deposition & limited angiogenesis
• Astrocytes produce inhibitory molecules preventing axonal regeneration
•Osteoblast, epithelial, or Schwann cell proliferation
• Limited ECM & granulation tissue
• Schwann cell conduit aids in axon regeneration
• Fibroblast recruitment
• ECM deposition
• Angiogenesis
• Increased gliosis density
• No lesion tissue filling
• Neurons protected by glial scar
• Mineralization (Bone)
•Tissue reorganization
• Axons reinnervate target (PNS)
• Full tissue strengthening (CT) / functional recovery (PNS)
• Mineralization
• Full tissue strengthening
• Removal excess tissue
• Fiber alignment & contraction
• Cellular apoptosis
• Partial tissue strengthening
CNS: Glial Scar
Glial Scar
PNS: Regeneration
Regenerated Axon
Bone: No Scar
Complete union
Cutaneous Tissue: Collagenous Scar
Collagenous Scar
seconds to hours
hours to days
days to weeks
weeks to months

Inflammation -signs

- CALOR & RUBOR- Heat + redness
  - opening up of capillary bed in response to chemical mediators
- DOLOR-Pain
  - chemical excitation of pain fibres (sub P)
- TUMOR-Swelling
  - immediate suggests extensive bleed
  - delayed (ie few hours) = inflammatory exudate caused by increased capillary permeability

Inflammatory Exudate -oedema

- Exudate is protein rich
  - albumins
  - globulins
  - gammaglobulins(antibodies)
  - fibrinogens
- high protein conc. raises OP and draws more fluid out ie swelling can increase for up to 4/7
- inflammatory cells
  - neutrophils
  - macrophages
  - fibronectin

Fibrous Repair

- Begins around 3rd day whilst inflammation still in progress
  - Involves Angiogenesis
  - Granulation Tissue
  - Fibroplasia

Angiogenesis

- Ingress of new BV
- Stimulated by angiogenic factors released from macrophages.
- Characteristic of redness granulation tissue and immature scar.

Granulation Tissue

- Invasion of clot by fibroblastic cells
- deposition of extracellular matrix of
  - collagen types 3 initially then 1
  - proteoglycans
  - glycoproteins
- transient structure associated with repair
- histological random appearance

Fibroplasia (- proliferation)

- 7-14 days post injury
- leads to the greatest accumulation of new connective tissue in the wound site
- early phase of healing scar is a single unit invading all areas of the wound.
- "One wound one scar" Peacock

Factors Influencing Fibrous Healing

- Poor blood supply, disrupted wound, oedema
- continued inflammation and infection
- immobility= weak repair poor collagen engineering

Good blood supply, 02, wound care

minimise inflammation and infection

reduce oedema

controlled motion and appropriate stress (Mason & Allen 1941

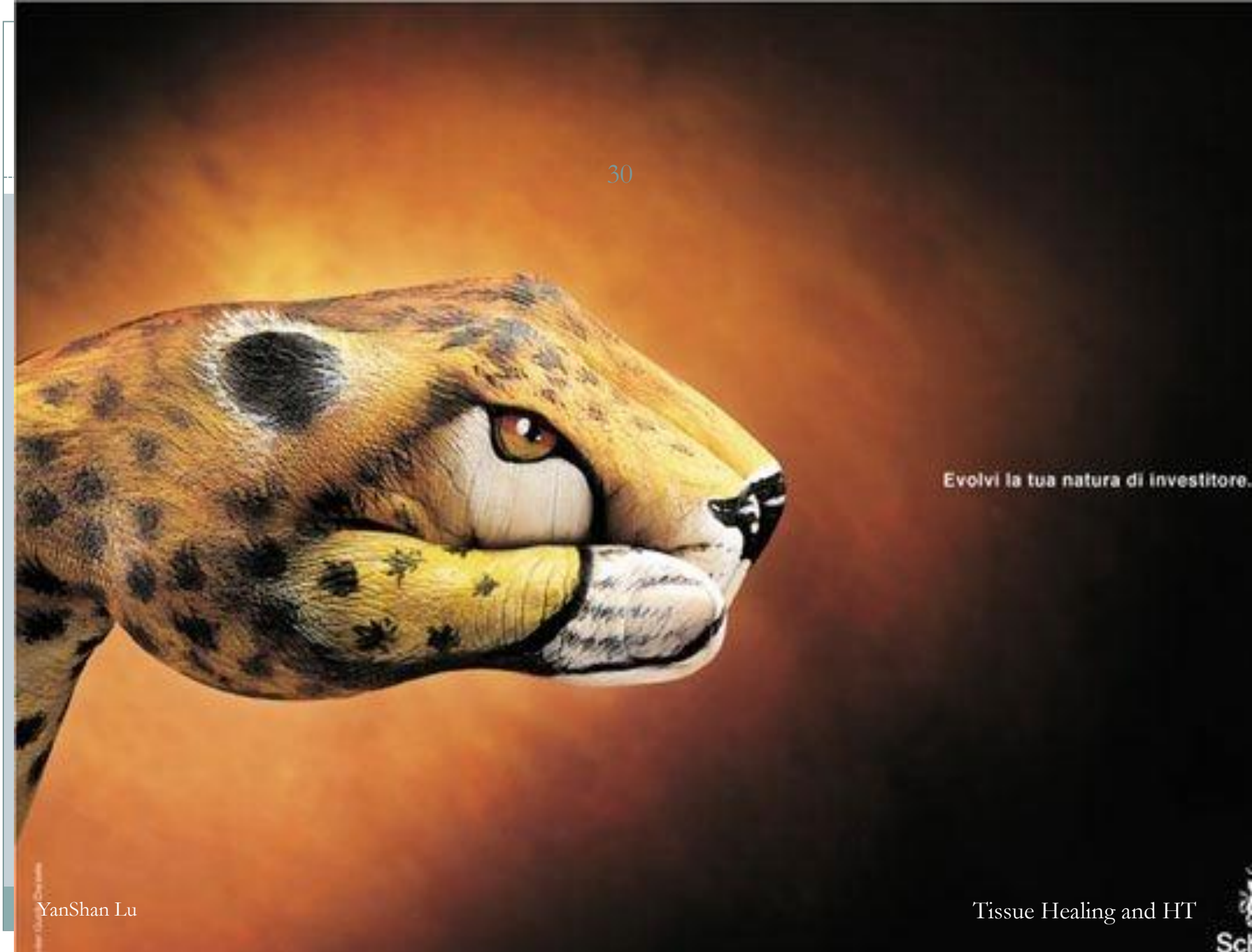
Scar Maturation/Remodelling

- 3/52-6/12 collagen has the property of gradually shortening.
- Increased deposition of collagen, bundles become thicker, covalent bonds between peptide chains
- responsive to lines of stress through orientation (months to years)
- vascularity subsides.

Extent of Scar

- dependent on
- site
  - superficial /deep
  - type of tissue
  - multitissue
- nature of injury
  - clean /compound
  - laceration / crush
- genetic background
  - scarers / non scarers

- wound/scar management
  - blood supply(smoking)
  - infection
  - swelling
  - disturbance
  - stress
  - vit C


Evolvi la tua natura di investitore.

How Different Tissues Heal

Overview and literature

Ligament Healing

- Refer articles:

Scar formation and ligament healing. Kevin Hildebrand, Cyril B frank.Can J Surg. 1998: 41:425-9

Ligament healing: current knowledge and clinical applications. Cyril. B Frank JAOS 4:2 1996

Present summary:

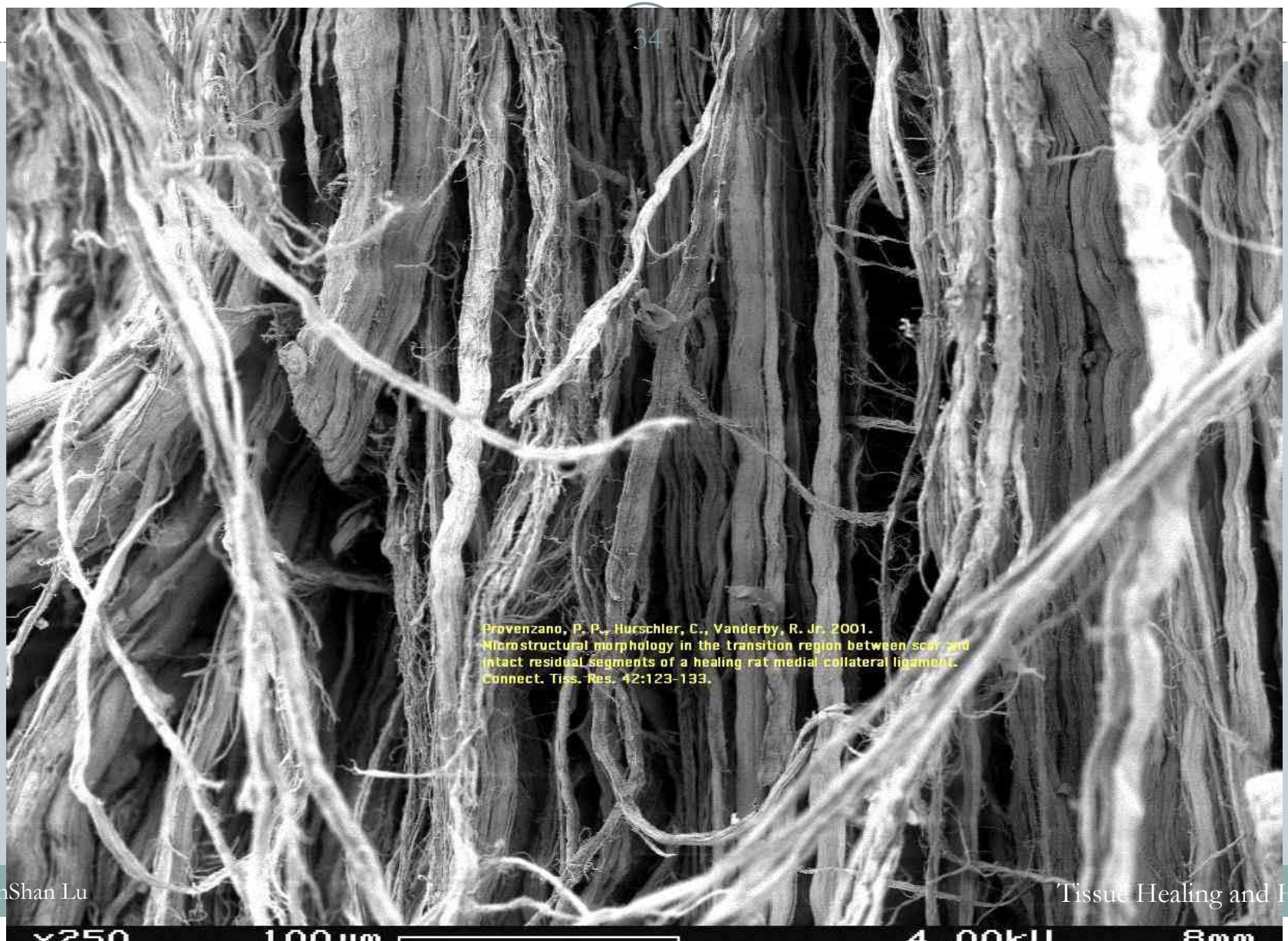
1. Critical functions of the tissue related to structure?
2. How does it heal ?
3. What factors influence healing?

Ligament Healing (Frank 1996)

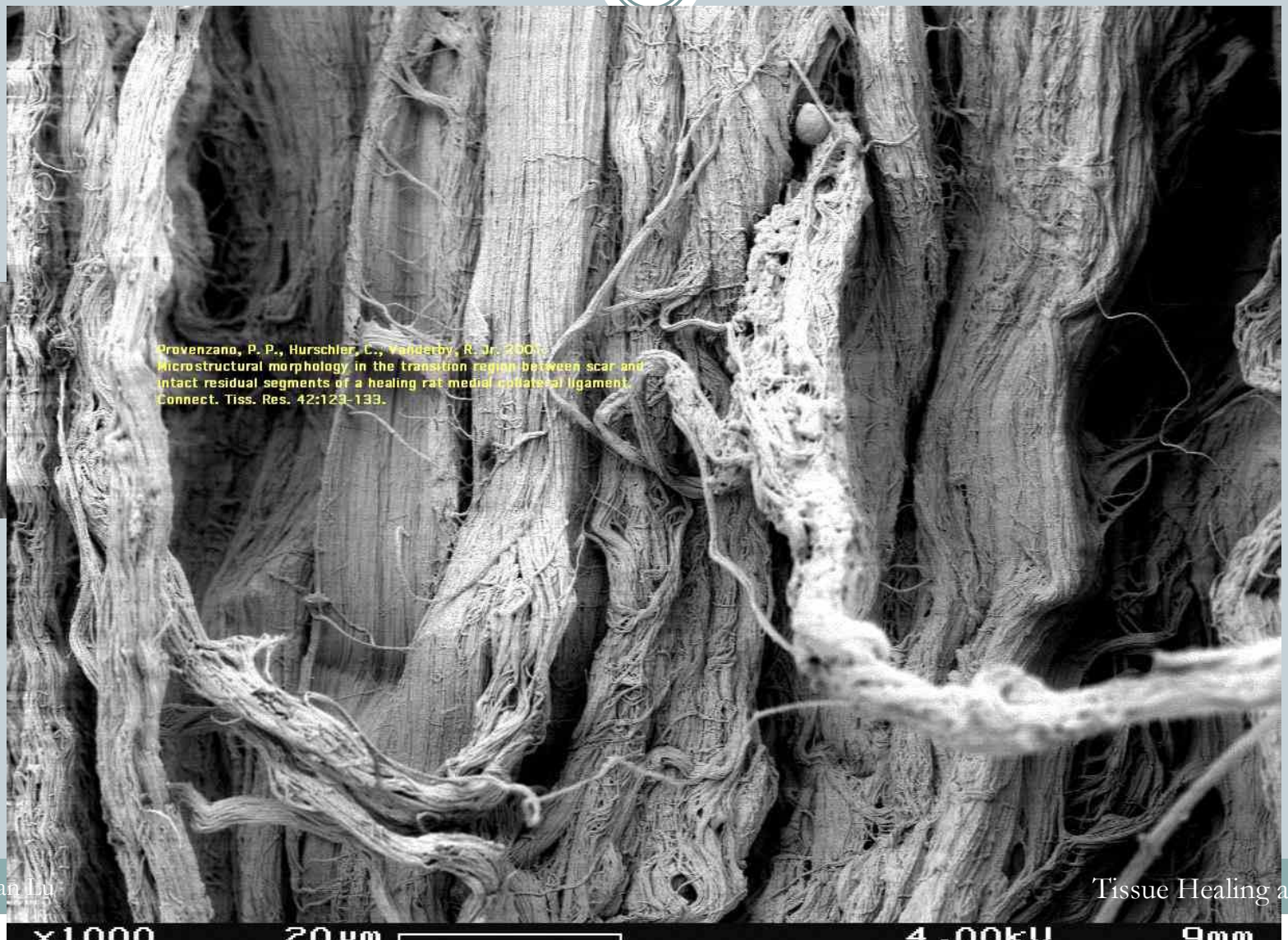
Table 1
Differences Between Normal Ligaments and Scars

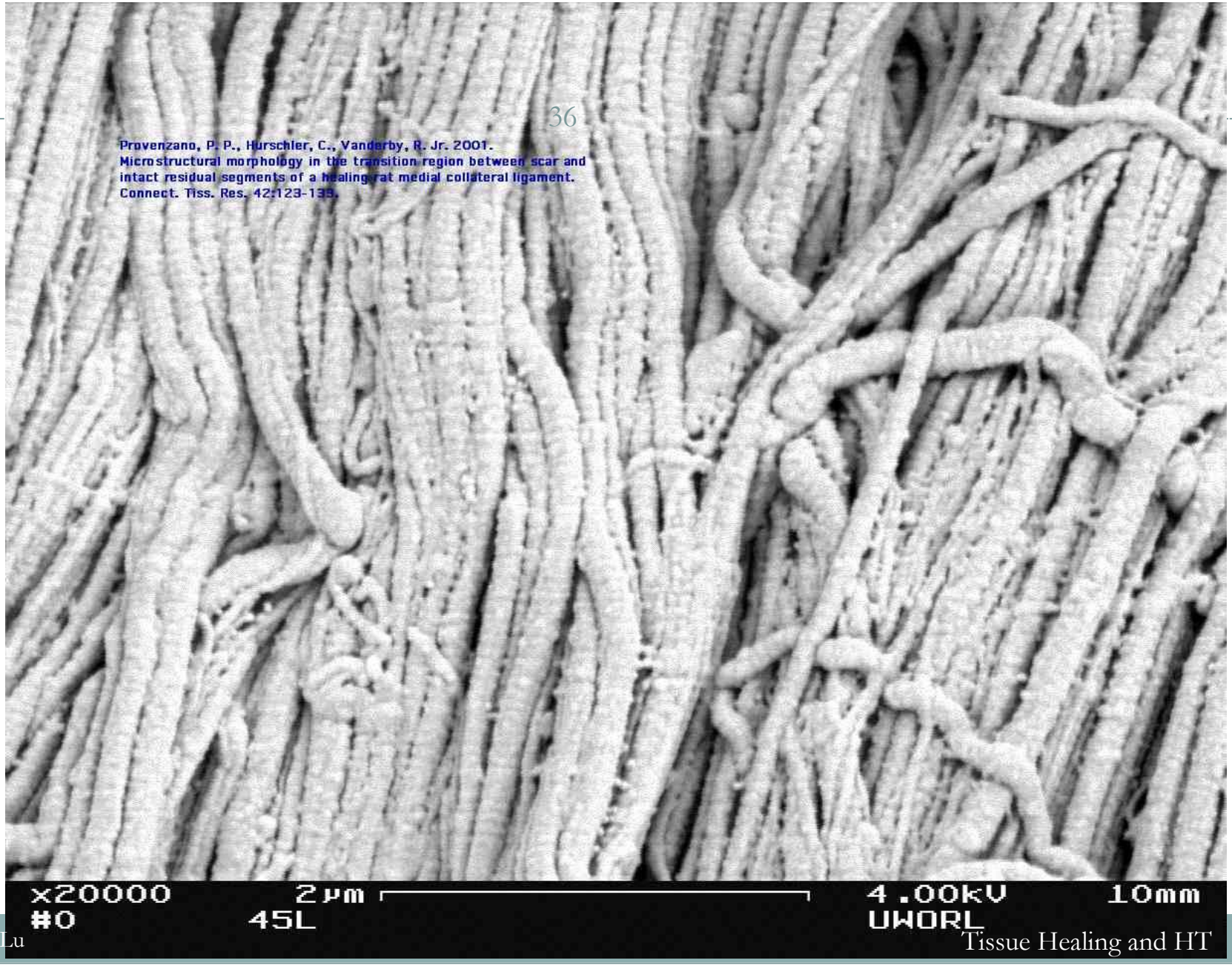
Normal (Uninjured) Ligaments	Ligament Scars
Collagen aligned	Collagen disorganized
Collagen densely packed	Defects between collagen fibers
Large collagen fibrils	Small collagen fibrils
Mature fiber cross-links	Immature cross-links
Primarily collagen type I (<10% type III)	More collagen type III
Small proteoglycans	Some large proteoglycans
Other components minor	Excesses of other components
Cell metabolism low	Cell metabolism high
Low cell density	Increased cell density
Low vascularity	Increased vascularity

Ligament Healing



Ligament Healing: Transition




Provenzano, P. P., Hurschler, C., Vanderby, R. Jr. 2001.
Microstructural morphology in the transition region between scar and
intact residual segments of a healing rat medial collateral ligament.
Connect. Tiss. Res. 42:123-133.
x20000 2μm 4.00kV 10mm
#0 45L UWORL

Controlled Stress: Ligament (Frank 1996)

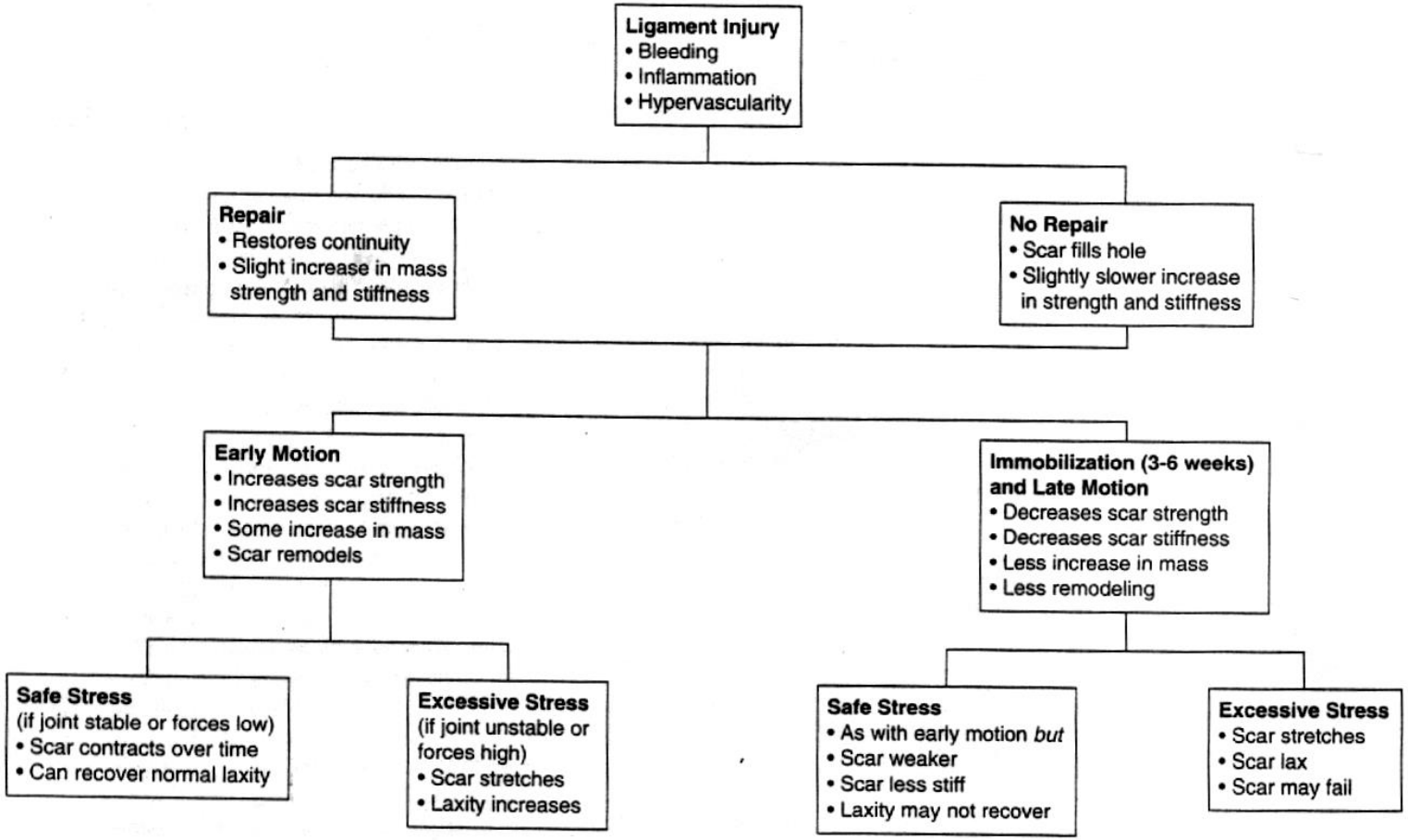


Fig. 4 Algorithm depicts the gross advantages and disadvantages of various treatment modalities.

Tendon Healing

- Refer articles:

Present summary:

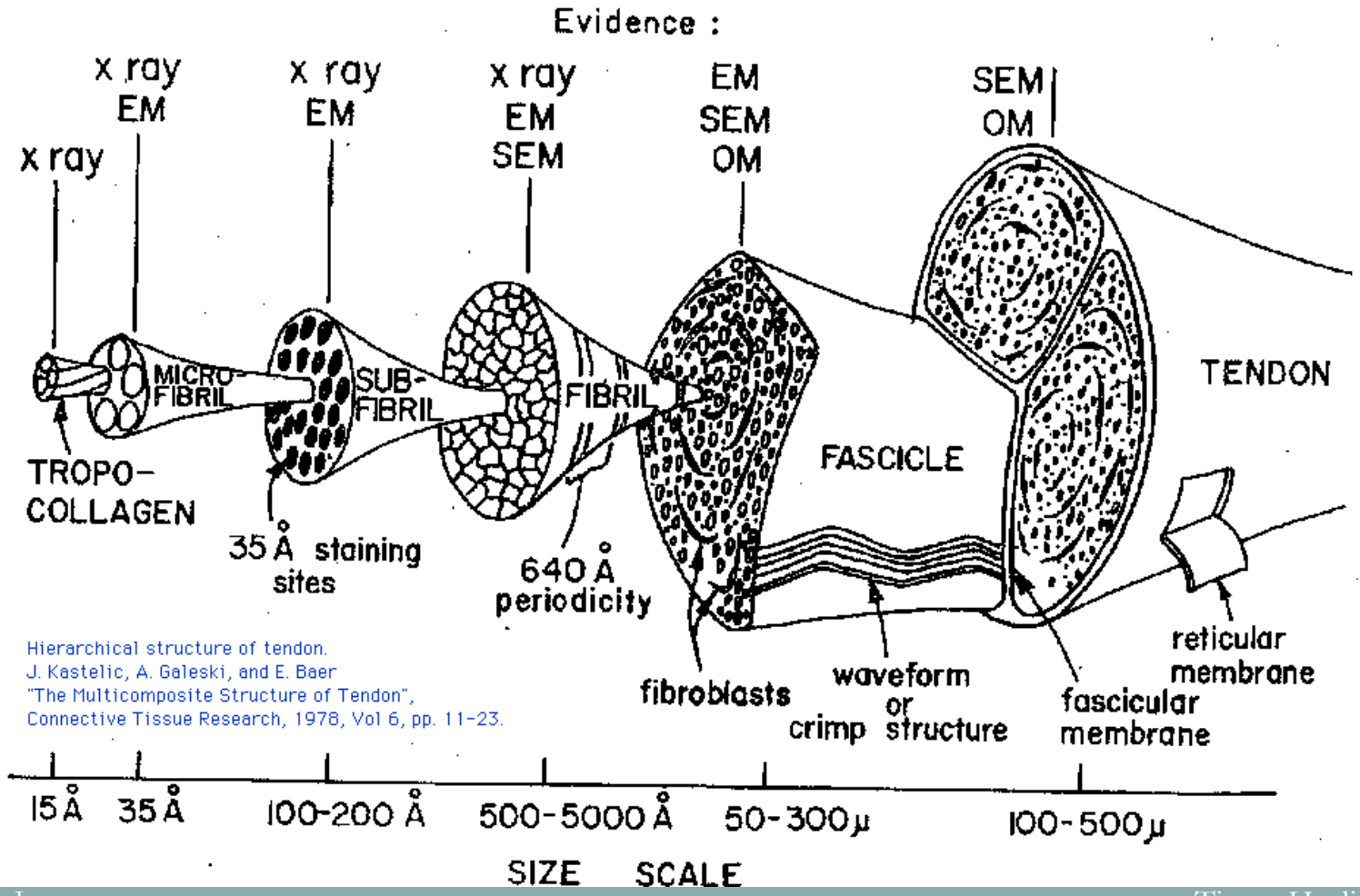
1. Critical functions of the tissue related to structure?
2. How does it heal ?
3. What factors influence healing?

Effect of motion and tension on injured flexor tendons in chickens Kubota et al JHS 21A.3 1996.

Effects of early intermittent passive motion on healing canine flexor tendons Gelberman et al JHS1982

Biologic Rationale, clinical application and results of early motion following flexor tendon repair. Strickland J .JHT1989

Tendon Structure



Hierarchical structure of tendon.
J. Kastelic, A. Galeski, and E. Baer
"The Multicomposite Structure of Tendon",
Connective Tissue Research, 1978, Vol 6, pp. 11-23.

Tendon Healing.

Mechanism

- Intrinsic
- Extrinsic.

Phases

- Inflammatory 1-3/7
- Fibroblastic 3-21/7
- Remodelling 21-112+/7

Factors influencing

- inflammatory state
- controlled stress
- suture technique
- timing

Tendon Rehab

a. Physiological response of tendon to controlled stress

- improved tensile strength
- improved tendon excursion
- increased DNA at repair site
- increased peritendon vessel proliferation
- increased synovial diffusion
- increased fibronectin conc.
- Reorganisation of extrinsic scar.

b. Tendons - Rehabilitation Guidelines.

"Rehabilitation of the healing tendon is simply re establishing its ability to glide and transmit force without creating gapping or rupture."

PROTECTION

V

MOTION

Bone Healing

- Refer articles:

Present summary:

1. Critical functions of the tissue related to structure?
2. How does it heal ?
3. What factors influence healing?

Bony Tissue repair. JHT 1998

Fracture Healing.Le Stayo et al JHT 2003

Bone Healing

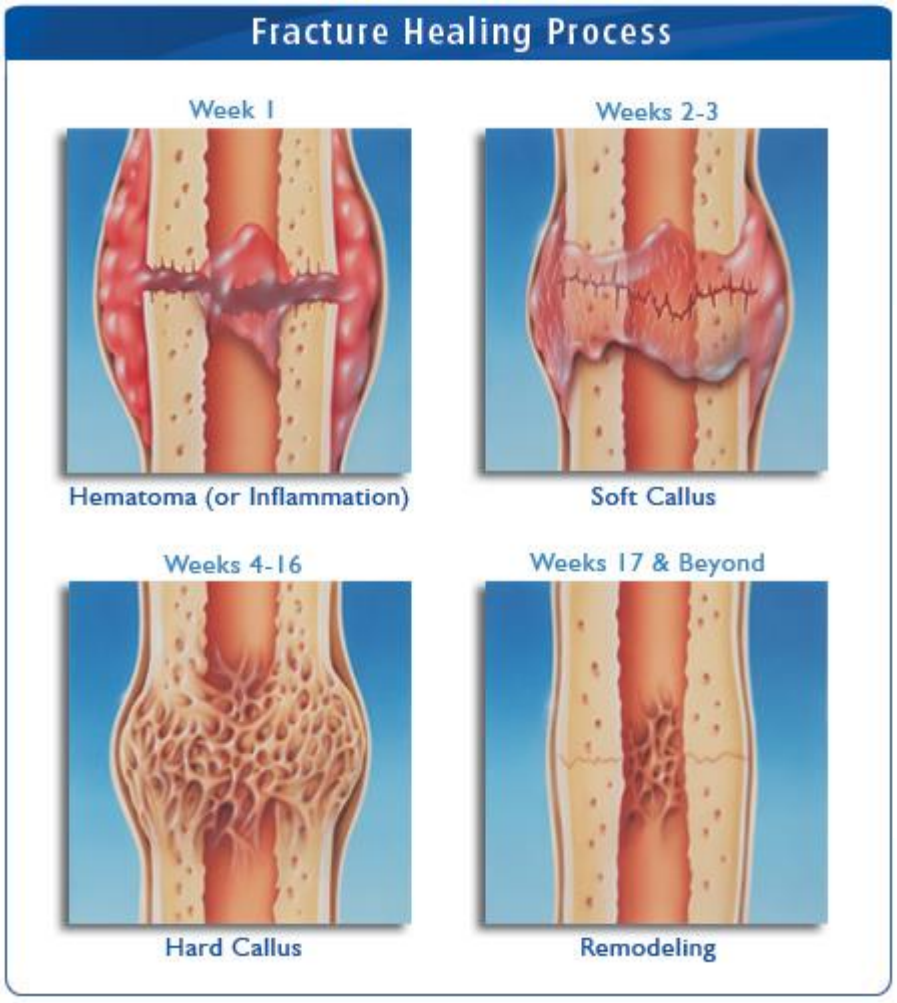
Initiation

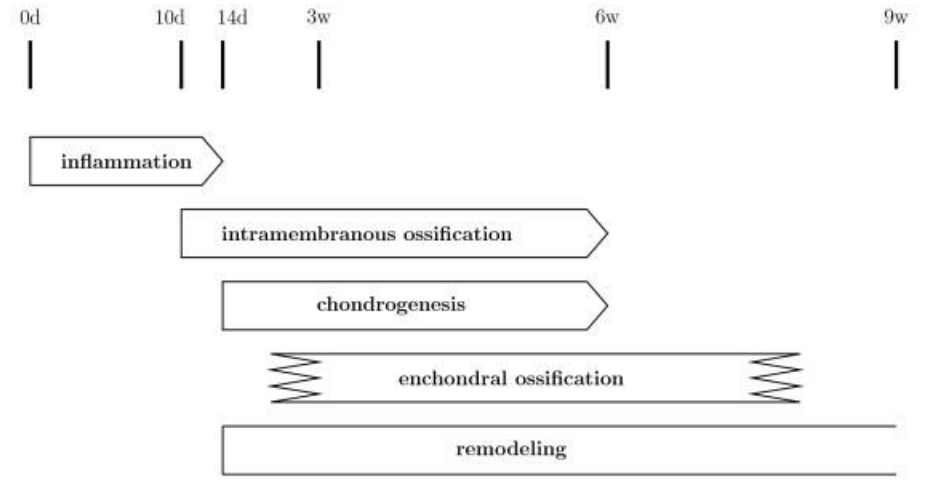
- recruitment
- modulation
- osteoconduction

Stages

- impact
- induction
- inflammation (3-10/7)
- soft callus (3-6/52)
- hard callus (6-12/52)
- remodelling(12-52/52

Fracture healing





Factors Affecting Bone Healing

Systemic

- Protein and Ca intake
- Age
- Tobacco use,
- Hormones.
- Diabetes.

Local

- Bone type
- type of fixation
- configuration of fracture
- accuracy of reduction
- Loading
- Motion
- soft tissues
- Blood supply
- Growth factors
- Electric potentials,
- Local pathology ie malignancy, metabolic bone disease.

Nerve Repair

Repair & Regeneration

- Dependant on degree of injury
- capacity of Schwann cells to myelinate
- governed by properties of
  - neurotropism
  - neurotrophy
  - specificity

Classification (Sunderland)

neuropraxia

axonotmesis

" +endoneurium

" +perineurium

neurotmesis

Nerve Healing

Process

- cell body
- distal stump
  - Wallerian degeneration
- proximal stump
  - Wallerian degeneration one node
  - axonal filopodia
  - growth cone

- Factors affecting

- age
- site
- nature
- timing
- inflammation

Nerve Regeneration and Repair

- Refer articles:

Present summary:

1. Critical functions of the tissue related to structure?
2. How does it heal ?
3. What factors influence healing?

Peripheral Nerve regeneration, repair and grafting
Bee Dagum JHT 1998
Structure and Biomechanics of peripheral nerve:ff..
Topp & Boyd Physical Therapy 2006

Summary

Summary

summary

summary

How can we Influence Healing/ Remodelling Tissues ?

BRAINSTORM

Specific Treatment Strategies

- Wound care & scar management.
- Oedema management.
- Application of controlled stress
  - Literature
  - Use of LLPS……
  - ….. through splinting.
- Principles of Splinting/

2. Scar Management

Hand therapists = scar therapists

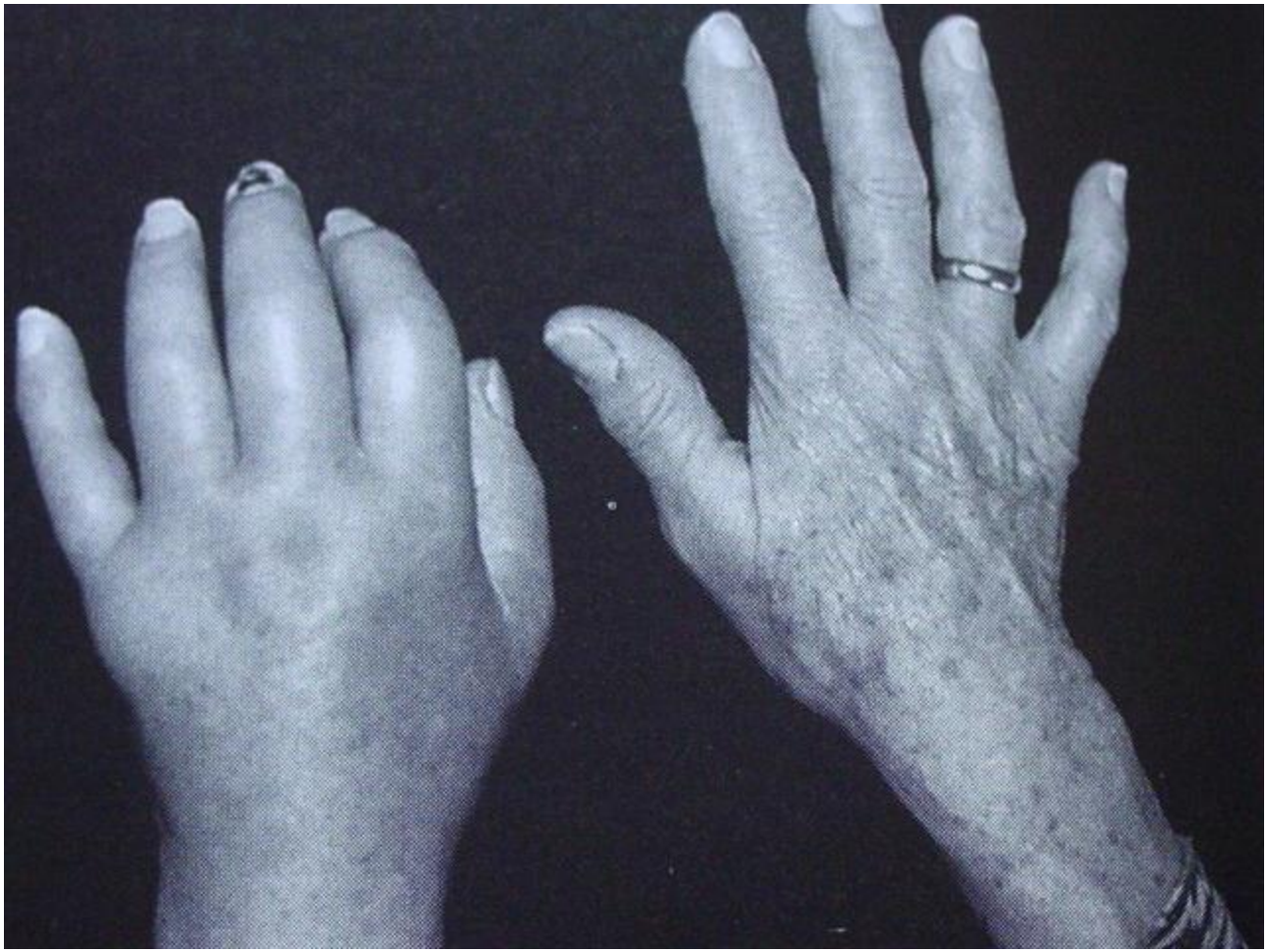
- “recognising the importance of careful soft tissue management to control and diminish scar formation is crucial to rehabilitating healing hands. Hand therapist are in truth scar therapists
- “Fess 1998.

3. Oedema Management

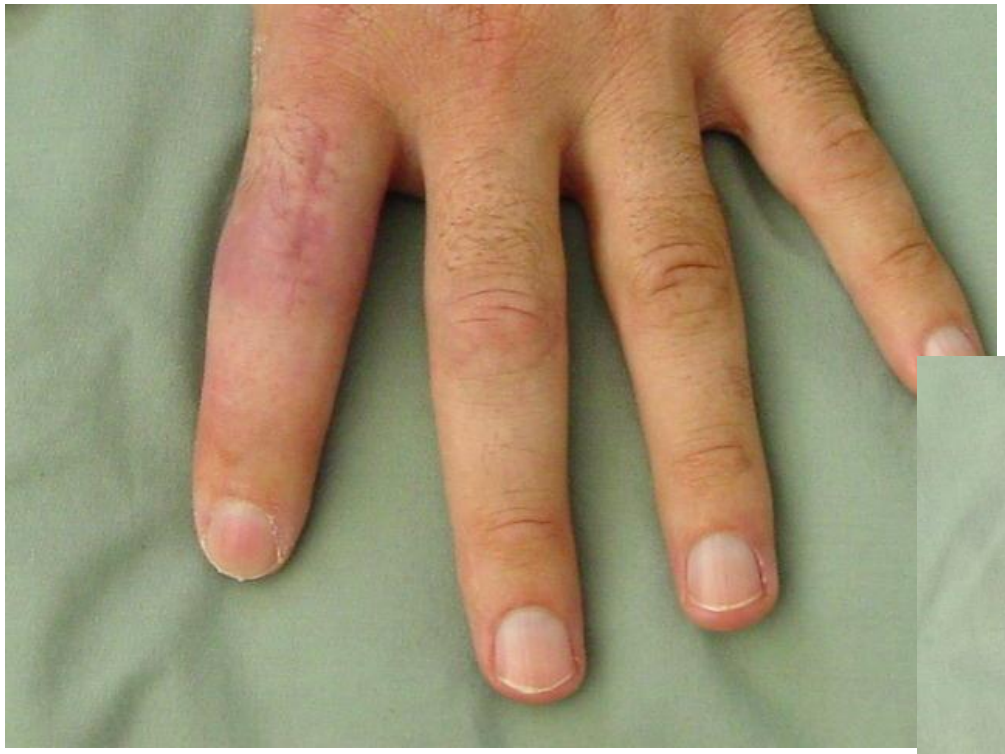
The prevention and treatment of oedema are of paramount importance during all phases of management of the injured hand.

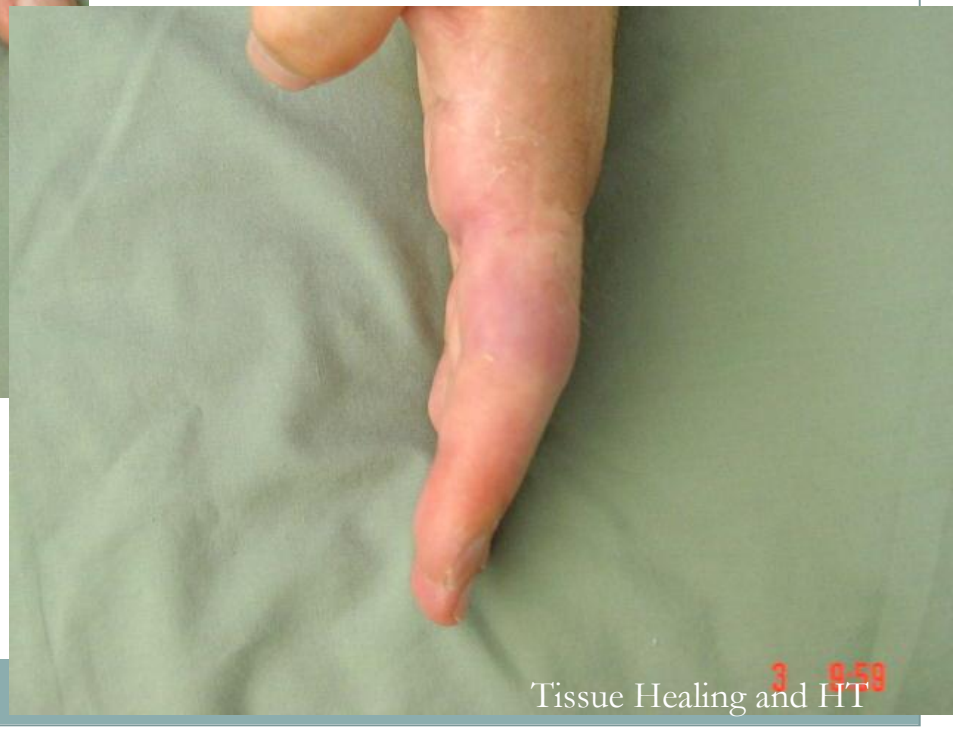
Effective management and control of oedema through all stages of rehabilitation is the foundation of treatment on which to build the restoration of motion, glide and tissue length, essential for a functional hand

Clinical Oedema



Clinical Oedema: Digital





Oedema: Normal Fluid Mechanics

- Arterial pressure 35mm/Hg will push fluid into interstitial spaces
- Normally 90% of interstitial fluid reabsorbed by venous system 10% by lymphatics.
- Lymphatics absorb the larger molecules ie fat cells, hormones, waste products, excess plasma proteins

Hand Vascularity

- Afferent blood flow mainly volar
- Efferent (venous and lymphatic) dorsal, deep palmar and superficial palmar.
- Act in synergy(Simons et al 1996). Perforators drain blood from palm to dorsum.
- Intrinsic mm contraction combined with tension in the dorsal skin

Lymphatic Structure

- Small lymph capillaries , blind ended vessels that sit in all interstitial spaces
- Lymphagions deeper collectors, smooth muscle respond to stretch reflex as they fill, valves prevent back flow. Rate 6-10 contracts per minute, increased 10x with exercise.
- Lymph nodes-sites of lymph/venous anastamosis.
- 4 lymphatic Quadrants.

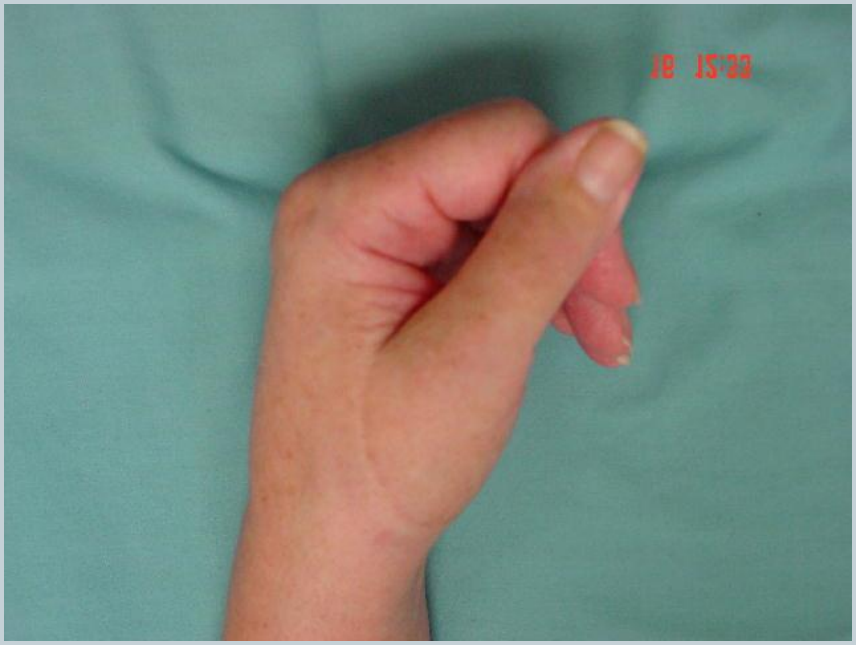
Causes of Oedema

Trauma/ Infection /pathology

- tissue damage - inflammation
- increased permeability endothelium
  - due to direct trauma or inflammatory chemicals ie histamine
  - protein rich exudate increases tissue Colloid osmotic pressure COP. Therefore fluid drawn out into tissues.
  - loss of balance of “flow out” vs “reabsorption”
- lymphatics blocked by fibrin plugs - loss of movt/muscle contraction

Pathomechanics of Oedema

- Delays healing, causes pain and loss of motion.
- Serous exudation leads to a stiffening of all structures, tendons joints ligaments.



Oedema & the Stiff Hand

- Sorensen(1989)protein rich fluid ***causes*** fibrosis and thickening of tissues which leads to stiffness and contracture.
- Brand (1985) direct effect on movement by changing moment arms and obstruction. “Scar in evolution”
- Flowers -persistent oedema ***leads*** to stiffness and then unwanted patterns of fibrosis

stiffness (Flowers)

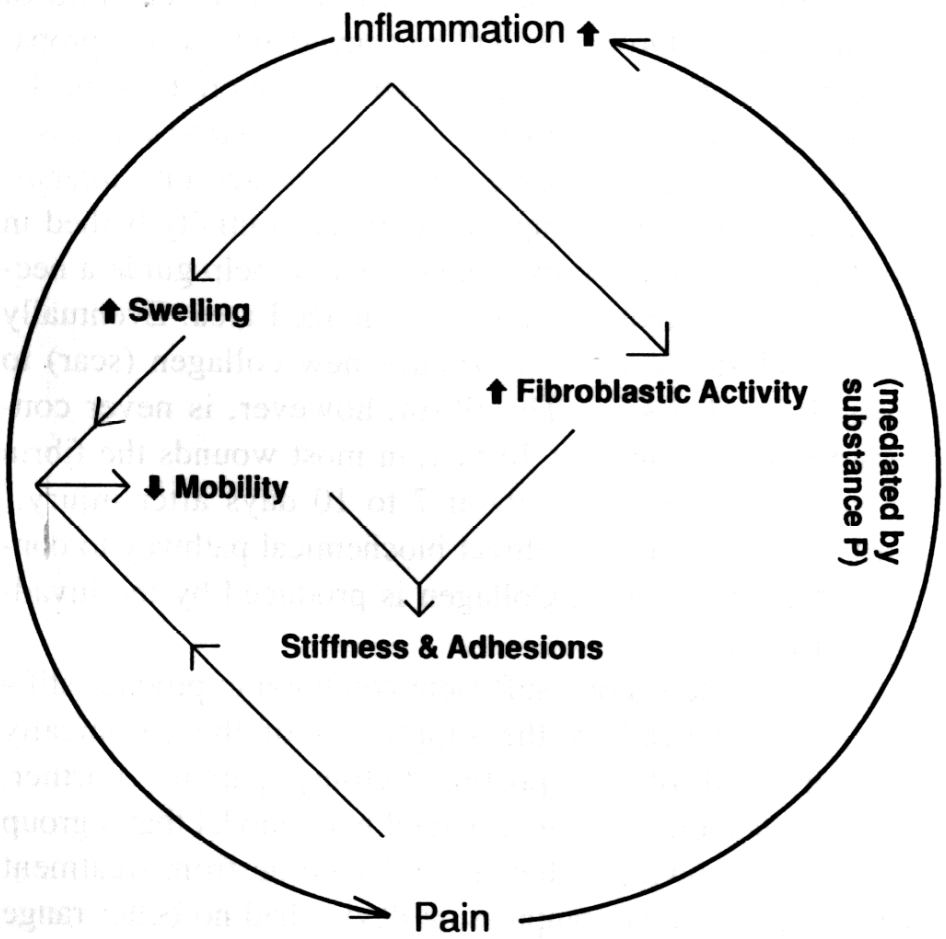


Fig. 7-1. Cycle of pain and inflammation that can result in stiffness.

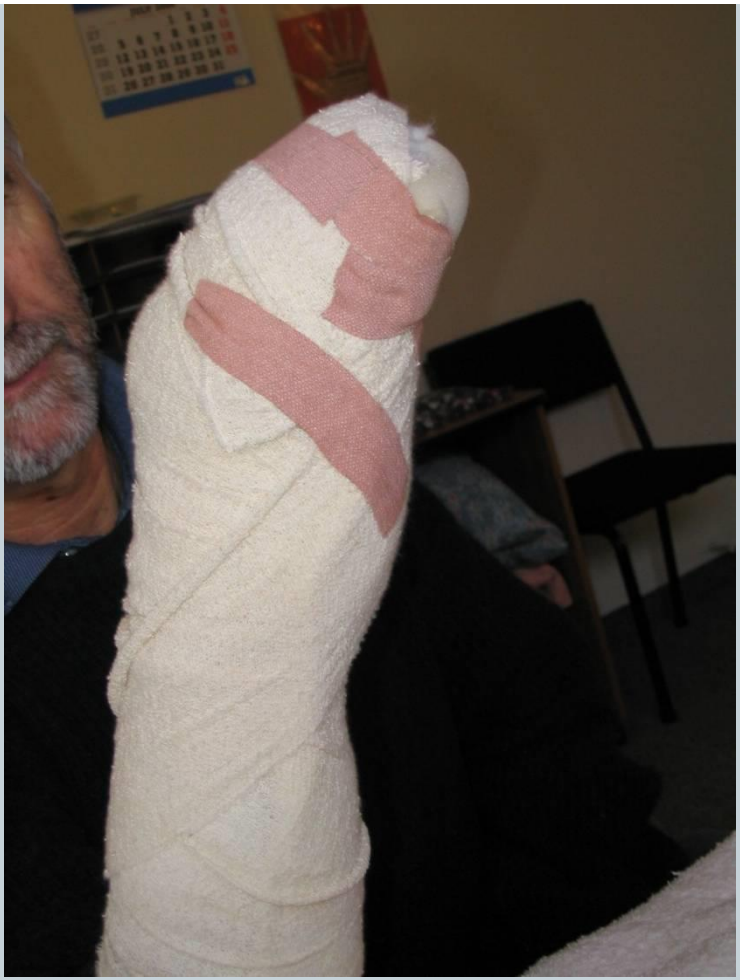
Oedema: Mechanisms of Control

- decreased hydrostatic pressure
- improved venous drainage
- improved lymphatic drainage
- improved limb blood flow.

Oedema: Clinical Considerations

- Heavy compression/massage will collapse the lymphatic system.Pressures of 60mmHg have been shown to collapse the system(Miller & Searle 1981)
- Effect of high compression is to push fluid back into venous system but proteins remain and cause a refilling post compression.
- Multi layered and low compression bandages indicated
- Temperatures 22-41 improve lymph flow.
- Exercise increase lymph flow up to 10x(Weissleder& Schuchhardt 1997)

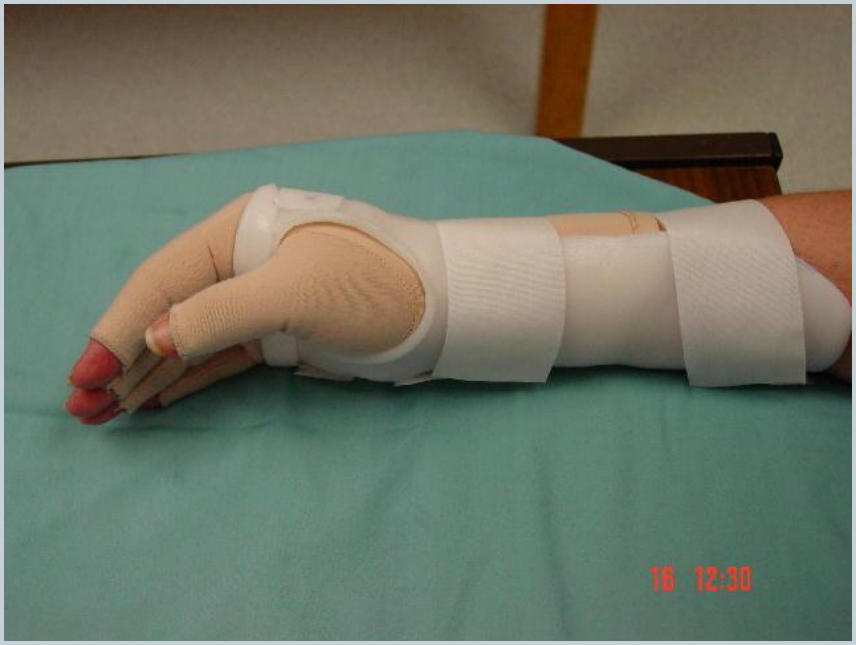
Oedema: Early Control



- Correct position of immobilisation and support
- bulky compressive dressing – Robert Jones
- elevation
- Ice (unproven scientifically more likely pain relief)

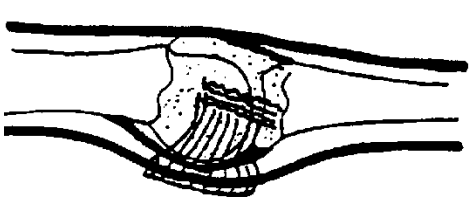
Reduction of oedema

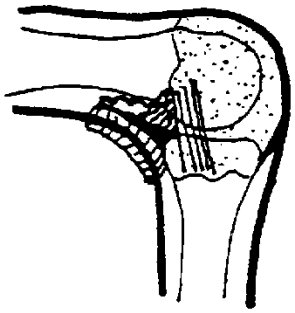
- Elevation
- Rest/splintage
- active ROM
- retrograde massage
- intermittent compression pump
- compression

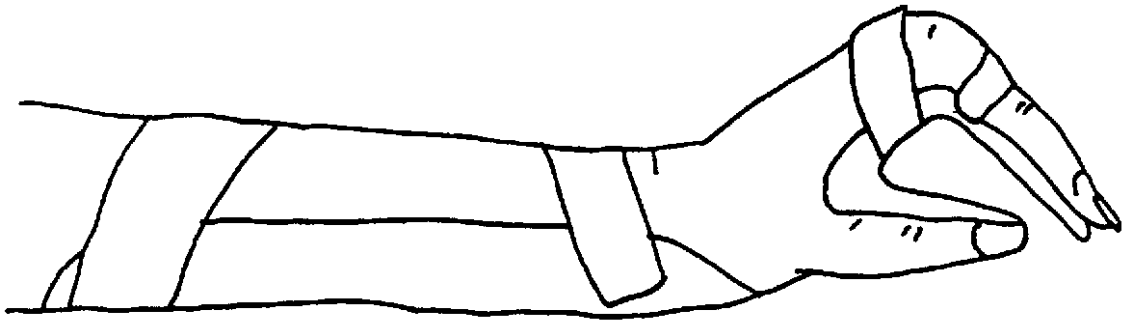


Oedema: Rest

- The importance of
  - Balance
  - Safe positions.







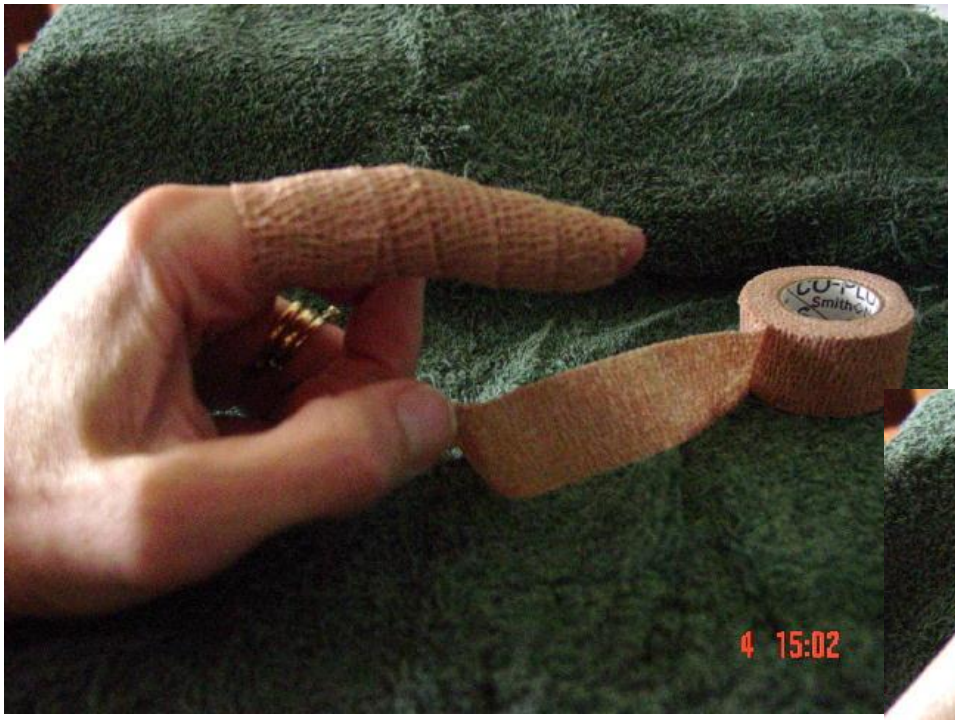
Oedema: compression

- Cohesive bandage
- gloves
  - lycra
  - isotoner
  - jobst
- Digisleeves
- tubigrip
- elastic bandage
- POP

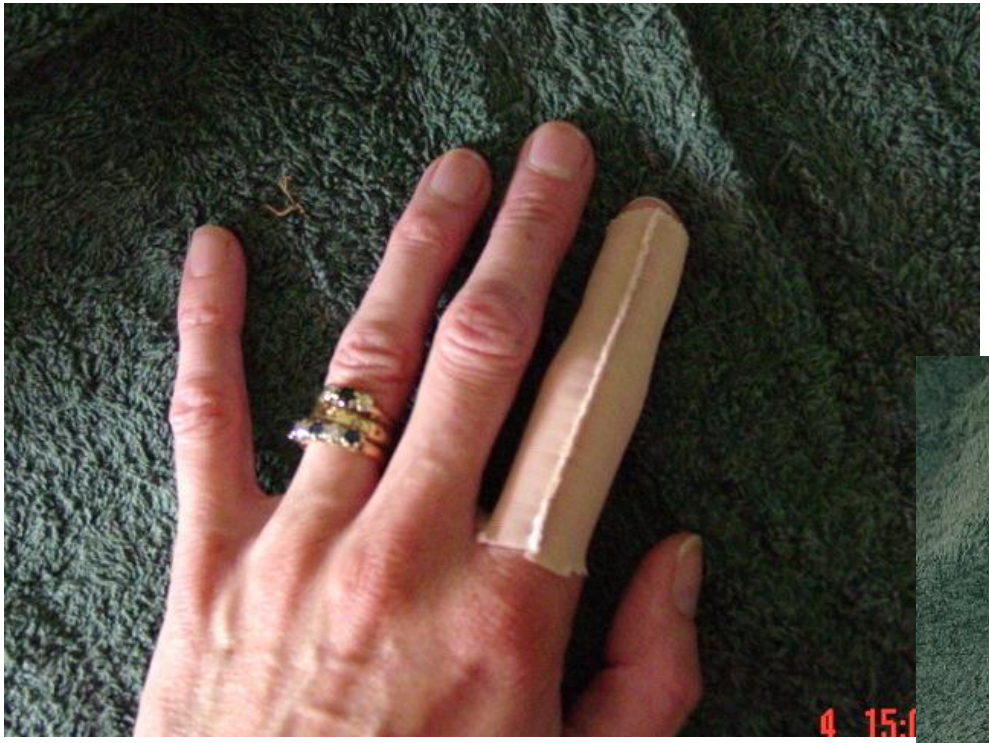
- Pressures variable
  - 3-25mmHg
  - 18mmHg
  - 30mmHg at tips

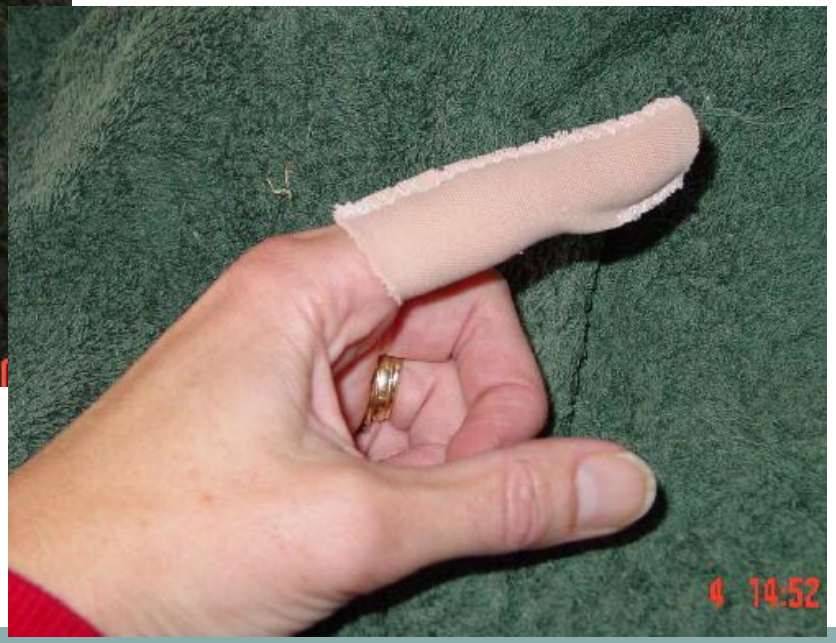
Coban wrap and pinch

Coban 1’ wrap



Compression sleeves





Oedema: Cryotherapy

- Direct effect theoretical but unsubstantiated (Knight 1995)
  - circulatory theory vasoconstriction, dec. permeability, decreased bleeding.
  - Metabolism theory:less tissue damage, less free protein, lower oncotic pressure.
  - Best effect within first hour.
- Indirect effect.
  - Decrease pain and muscle spasm and therefore improve motion.
- Stiffening effect on connective tissue

Oedema: Active Motion

- should be to end ranges to maintain tissue length and tighten dorsal skin
- intrinsics very important, can be isometric (Simons 1996)
- no overpressure or passive
- incorporate elevation
- include shoulder & elbow
- frequent ie hourly appropriate rest between
- electrical stim(Faghiri 1997) CPM (Guidice1990)

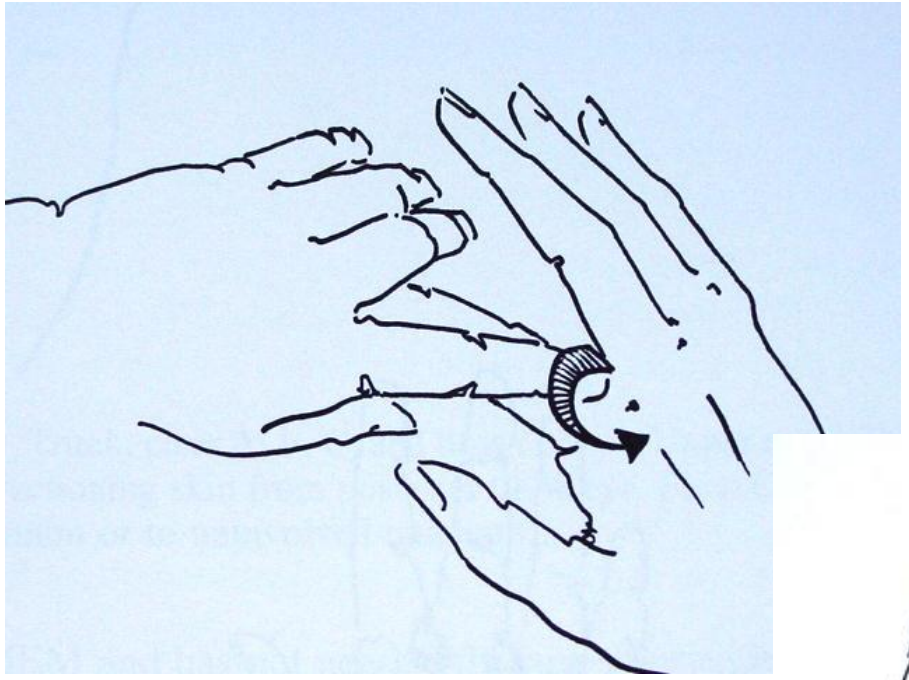
Oedema: Retrograde Massage

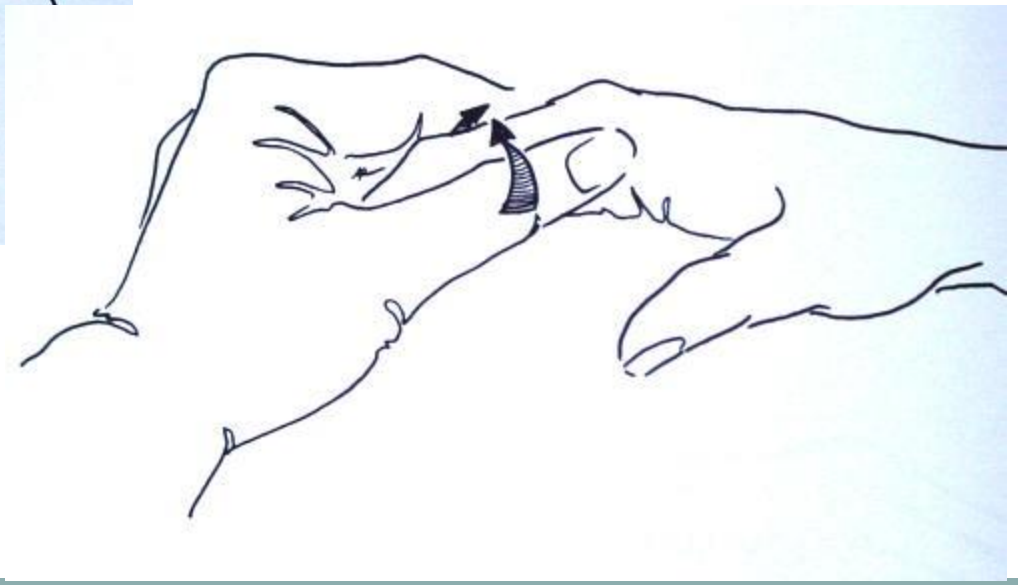
- Centripetal direction
- avoid over stimulation
- important to clear proximal segment
- with elevation
- follow with compression

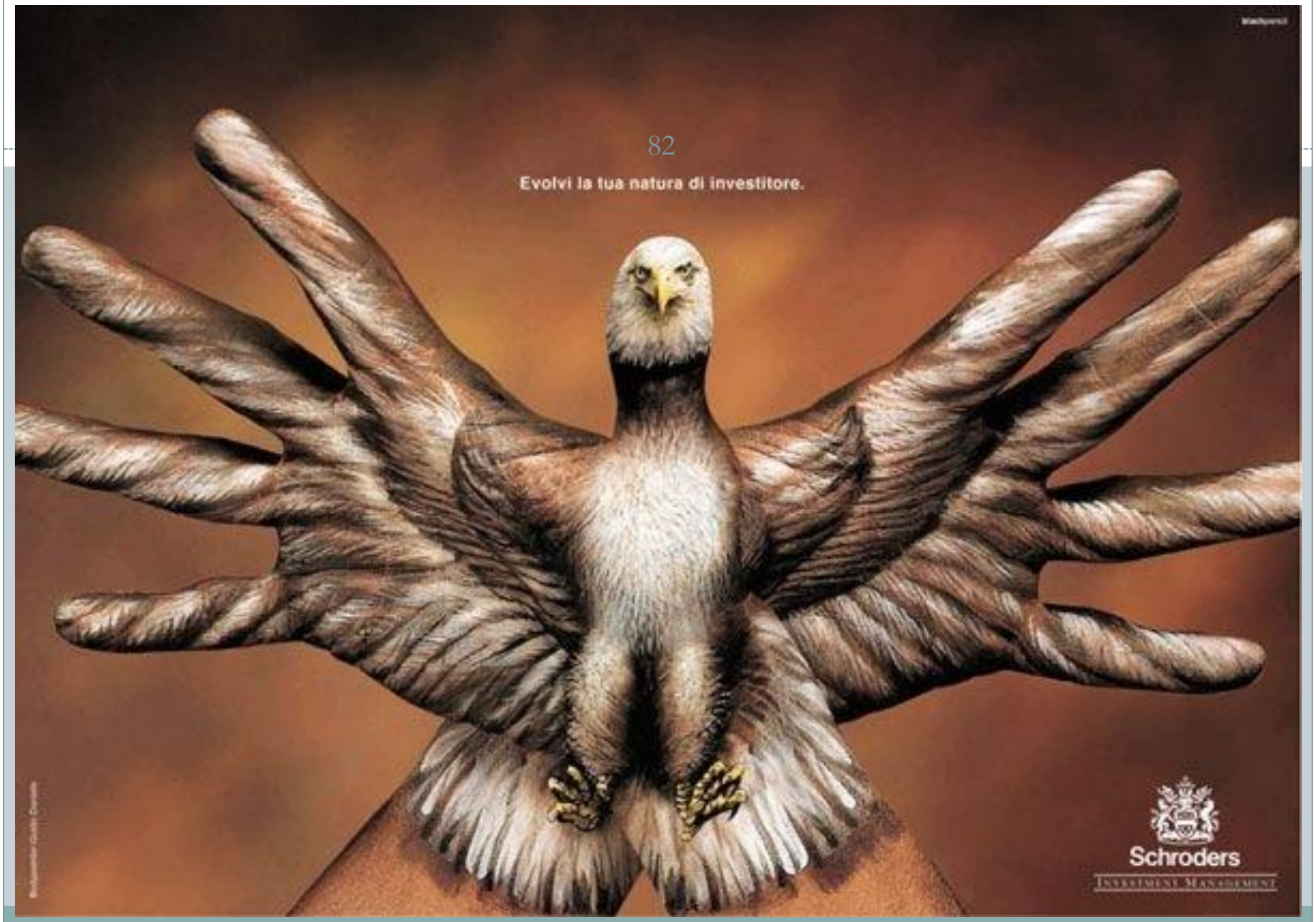
Manual (O)Edema Massage MEM (S Arzburger)

- Not indicated in all cases but of use for recalcitrant subacute or chronic oedema.
- Light massage less than 20-30mmHg
- segmental massage follows lymphatic drainage reroutes around scars
- sequential pre and post exercises. Proximal.
- Home programme – “stroking the cat.”
- low stretch compression

MEM - digital






Evolvi la tua natura di investitore.
Schroders
Investment Management

The Application of Controlled Stress to Healing Tissue

Connective Tissue Changes: STIFFNESS

C yr & Ross (JHT 1998)

- Restoration of a patient's functional ROM and strength are our primary goal.
- Immobilisation of normal connective tissue leads to biomechanical , biochemical and physiological changes within a week.
- These changes are magnified in presence of trauma and oedema, and may create permanent damage if not addressed swiftly and properly

on behalf of the stiff finger…..

- Non specific tissue remodelling
- “the hand is an organ, when one part is injured, the entire hand responds”

  WH Merritt & EE Peacock (1975)

Multi-tissue interface

The complex biomechanics of movement in the hand are highly dependent on the ability of one tissue to **GLIDE** over another.

Injury or pathology involving connective tissue can result in a greater acceleration of collagen syntheses and the resultant **fibrosis or scarring** that can continue beyond 3 months.

If this scar or **adhesion** binds structures together, in the Hand this prevents normal glide and flexibility, limits function and causes pain.

Oedema & Stiffness (Brand)

- Decreased tissue glide
- Increased tissue pressure
- Decreased skin mobility
- Increased work of movement
- Reduced mm efficacy.

Immobilisation: Stiffness

- Prolonged immobilisation
- CT assume shortened position
  - Changes in length and structure occur within 1 week
  - Decrease H20 & glycosaminoglycans
- More resistance to joint motion
- Altered response to load , leads to joint contracture, adhesions and loss of glide.

How to address the problem

- Apply specific types of stress to the involved and associated structures at optimal intervals.
- Load must be applied at adequate intensity and duration to successfully affect the viscous (plastic) property of connective tissue

Cyr & Ross JHT 1998.

Stress modalities

Controlled motion

- Active
  - Assisted
  - Place &hold
  - Range specific.
- Passive
  - Manual
  - Massage
  - Splinting

Evaluating Joint/Tissue Stiffness

- Is there an elastic limitation
- Are the tissue too short or too stiff
- Is there a viscous problem (oedema)
- Is there a frictional problem within joint

- Brand & Colditz 1995

Connective Tissue Properties

- Collagen Fibres in an intercellular matrix
- Demonstrates "viscoleasticity"
  - Elasticity: temporary elongation, recover original position
  - Plasticity/Viscosity: non recovering permanent change.
    - Linear deformation produced by load remains after load removed
    - Collagen separation (creep)
    - Collagen degradation and synthesis (growth)
- Amount and type of change (transient or permanent) dependent on intensity& duration of applied load and temperature of the tissues.
- Optimal plastic change with LLPS (cf dentistry, pregnancy)

Cyr & Ross JHT 1998

CT Biomechanics: Creep

- If tissue stretched beyond its normal elastic limit, “creep” will occur.
- “creep” = gradual irreversible lengthening caused by slippage of short collagen fibres on one another within tissue matrix.
- This is termed “plastic” behaviour. i.e. due to microscopic changes the tissue fails to return to its original length when unloaded.

CT Biomechanics: Growth

- If tissue is held in a slightly lengthened position, within its elastic limit, for a sufficient period of time, the collagen fibres will be actively reabsorbed and laid down again with modified bonding patterns, with no reversible creep and no inflammation
- This is tissue "growth"

Brand JHT 1995

The plasticity of connective tissue

Viscous Stiffness.

- Fluid mechanics (Brand)
- Oedema in finger or joint creates drag and friction, causing stiffness
- Need to remove the fluid to reduce the drag, ie push, elevate, massage.
- Slower response than elastic stretch.
- Important to recognise viscous stiffness and treat accordingly.

Controlled Stress

- Research to support with different tissues
  - Tendons
  - Ligaments
  - Bone.

Controlled Stress: Tendon

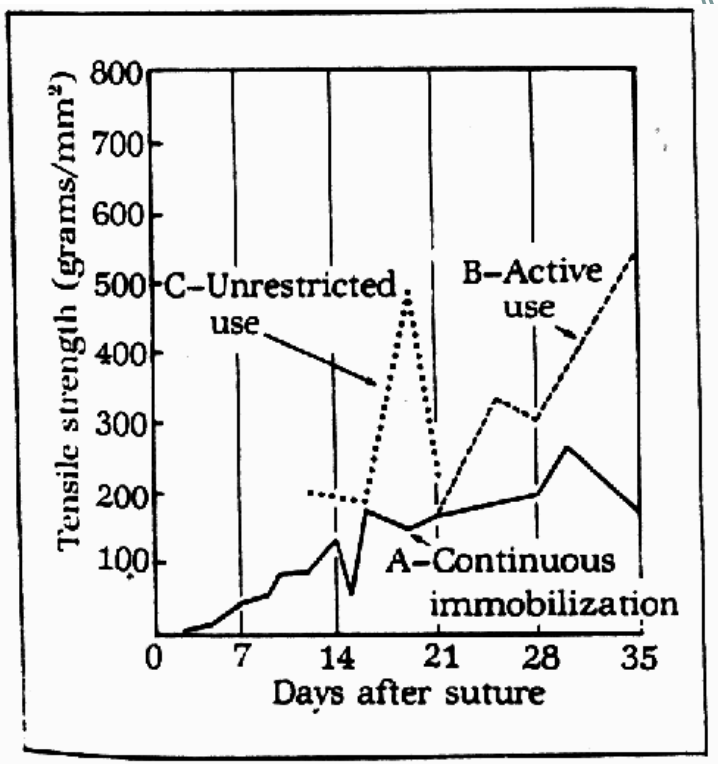


FIGURE 3-2. *Rate of gain of tensile strength in healing canine profundus tendon anastomoses: Group A animals continuously immobilized; group B permitted active use after 3 weeks; and group C permitted unrestricted use after 5 to 14 days of immobilization. (Adapted from M. L. Mason and H. S. Allen, The rate of healing of tendons, Ann. Surg. 113:424, 1941.)*

a. Physiological response of tendon to controlled stress

- improved tensile strength
- improved tendon excursion
- increased DNA at repair site
- increased peritendon vessel proliferation
- increased synovial diffusion
- increased fibronectin conc.
- Reorganisation of extrinsic scar.

Controlled Stress: Ligament (Frank 1996)

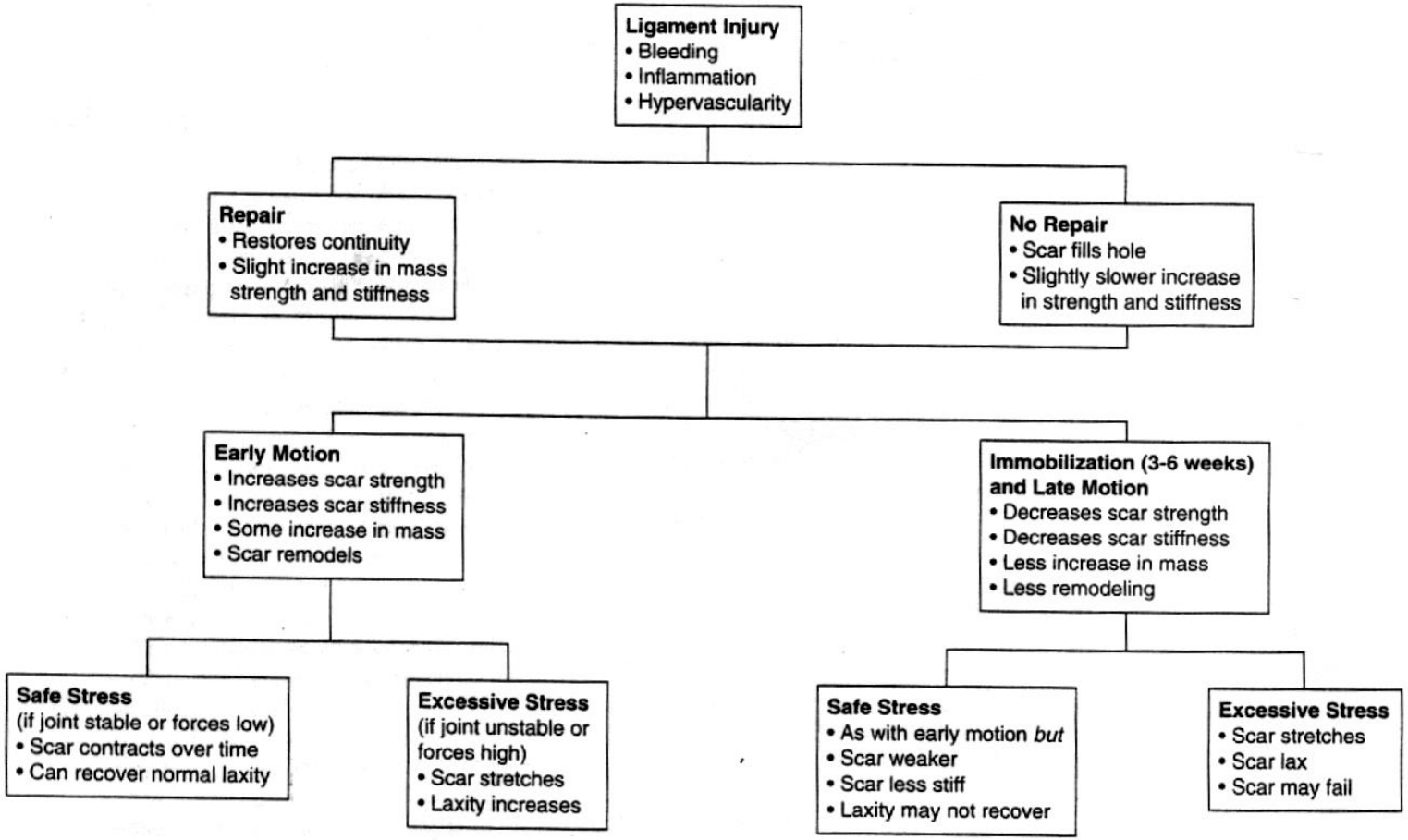


Fig. 4 Algorithm depicts the gross advantages and disadvantages of various treatment modalities.

Controlled Stress Strategies - Healing tissues

1. Mobilisation:
   - a)Active:
     - Early controlled / short arc or restricted motion
       - Wounds ; Arem & Madden JSR 1976
       - Tendons: Strickland & Glogovac JHS 1980, Amiel & Gelberman et al 1976
       - Ligaments: Akeson& Amiel et al Clin. orthop. 1987 Buckwalter JA , HC 1996
   - B) Passive: with care.
   - C) Place and Hold: End range/ control

2. Oedema control: to decrease viscous drag
3. Balance with Rest : important

Controlled Stress - Remodelling

Problem = Adhesion and Shortening

Solution = Glide + Growth.

- Living tissue responds to stress (Mason & Allen 1941)
- Stress triggers increase tissue length (Arem & Madden1976)

Controlled Stress Strategies - Remodelling Tissues

- **Active:** sometimes not enough force to influence tissues, alone, but important element to retain gained motion. End range control
- **Passive:** need to consider effect on CT
  - Physiological.
  - Accessory.
  - Assisted. (splinting)
- **Resisted.** Increases forces on tissues/ tensile strength Increasing strength provides protection from tissue overload
  - When, how much, how often ?
- **Functional.** Consider Hand a functional tool
  - Neural patterning.

Controlled Stress

- **HLPS** aggressive tissue loading=tissue failure
- **HLBS** (manipulation) ; efficacy in small joints not substantiated
- **LLBS** (accessory PJM) can be "look good feels great after therapy syndrome" , ie elastic response easily lost.

  Useful as adjunct or to to condition pre splinting.
- **LLPS** **(splinting)** stress to max length and hold for significant period of time

Controlled Stress Strategies

Low Load Prolonged Stress (LLPS)

Most effective stress application in hand

"Creates permanent growth in tissue" (Brand 1993)

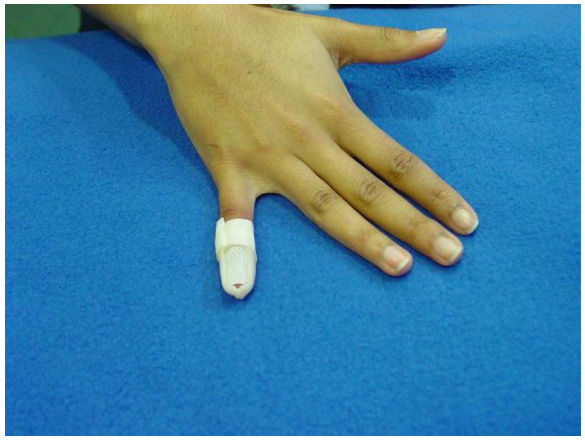
- Cultural cosmetics/ orthodontic evidence to support
- Literature: Arem & Madden.(1976) Light et al (1984)

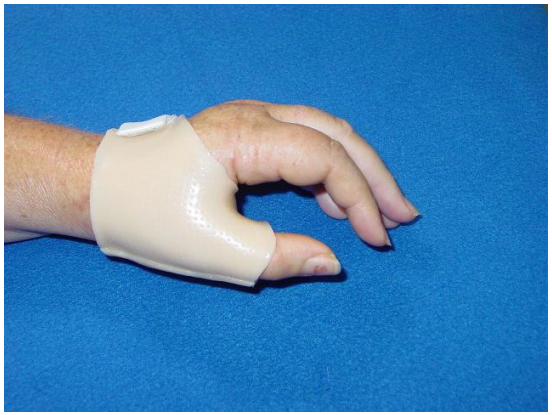
- Plasticity / lasting change. Increase collagen synthesis, linear orientation, inc tensile strength.
  (Le Stayo & Jaffe 1994. Cyr and Ross 1998
- TERT. Improvement proportional to time.
  (Flowers and le Stayo 1994, Prosser 1996)
- CMS (casting motion for stiffness) Colditz
  ? Neural modulation

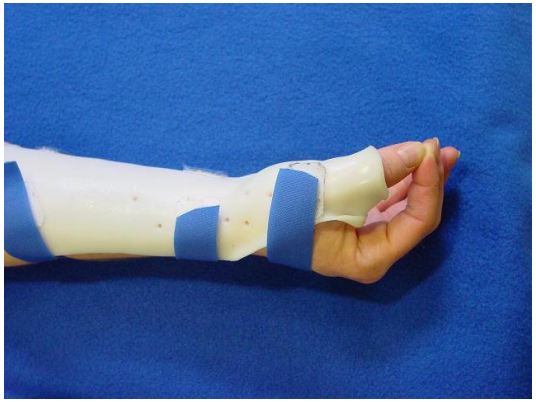
Adverse Stress

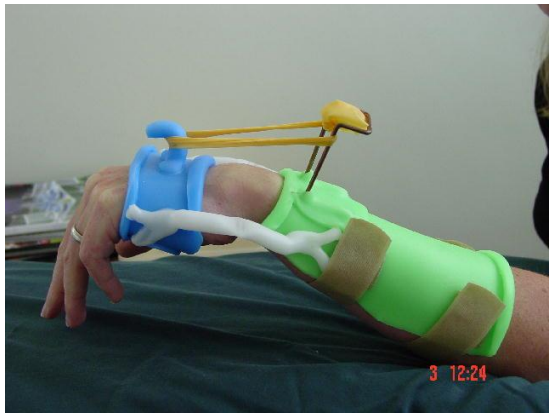
- Too much stress will lead to tissue damage/failure
  - Inflammatory response
  - New repair /scar cycle.
- Inflamatory signs: swelling, pain, redness, heat, dysfunction
- Regulate: force ,time, pressure, recovery.

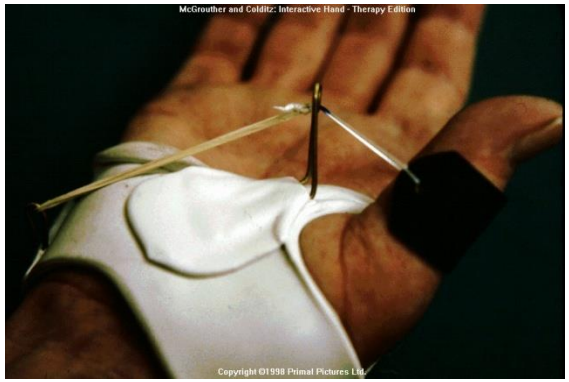
Splints

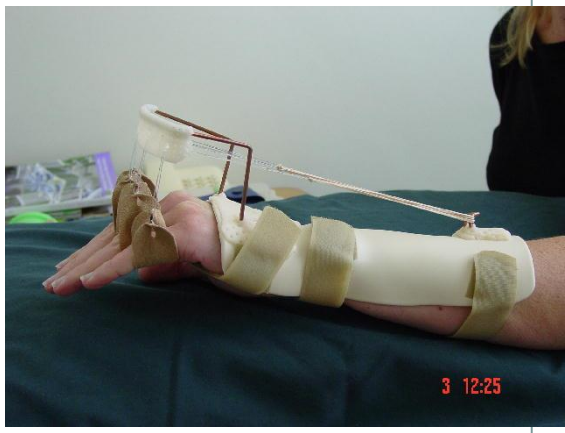







3 12:24

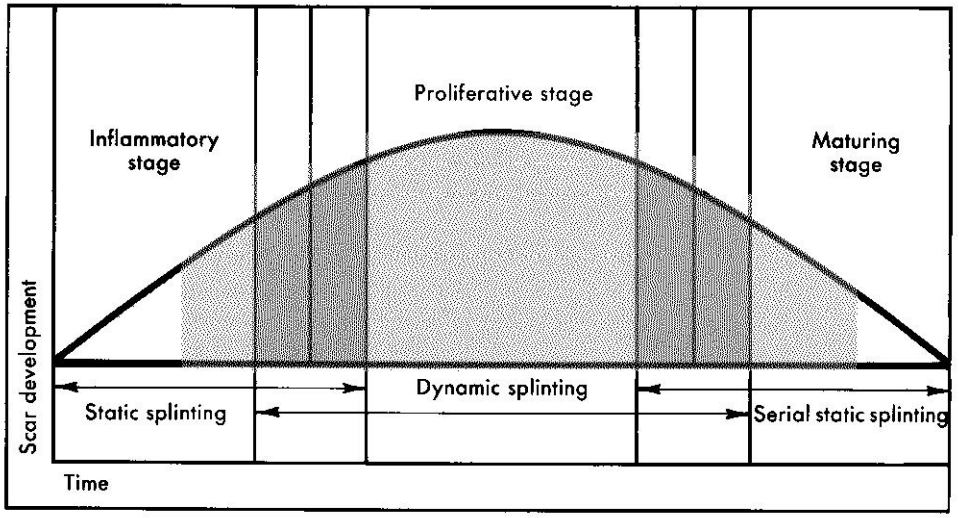

McGrouther and Colditz: Interactive Hand - Therapy Edition
Copyright ©1998 Primal Pictures Ltd


3 12:25

Splint use related to healing

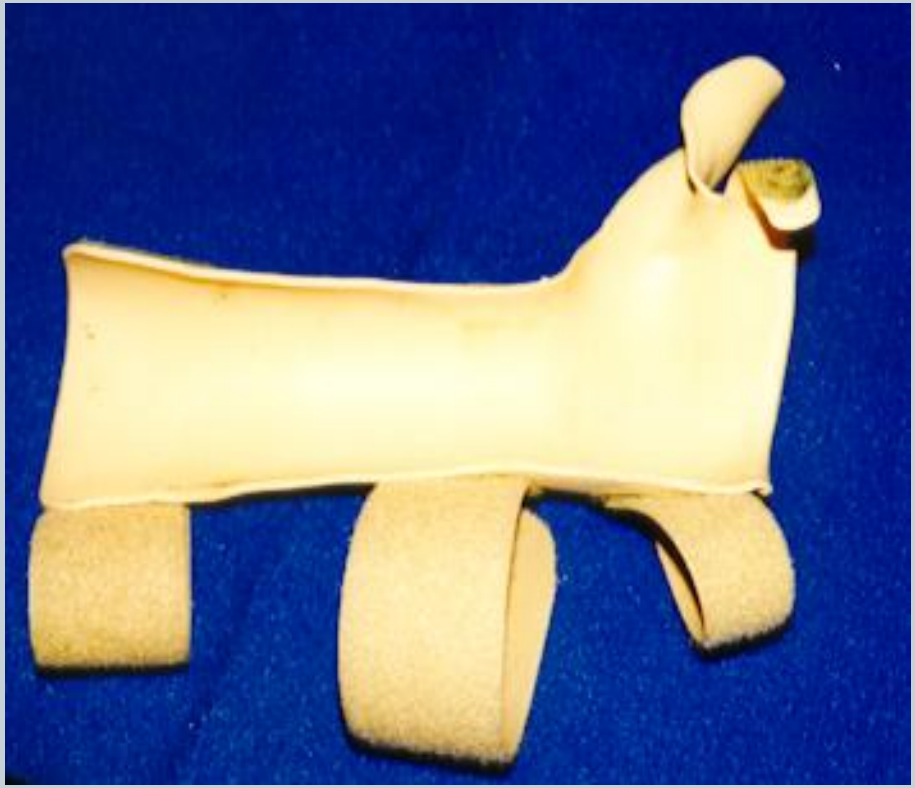
- Inflamatory:
  - Rest and positional splint
  - Protection
- Fibroplastic:
  - Inter
  - intermittent rest.
  - Protection of healing structures.
  - Limited motion.
  - Assisted motion –dynamic.
- Re-modelling:
  - LLPS:
    - Serial static
    - Dynamic
    - Static progressive.

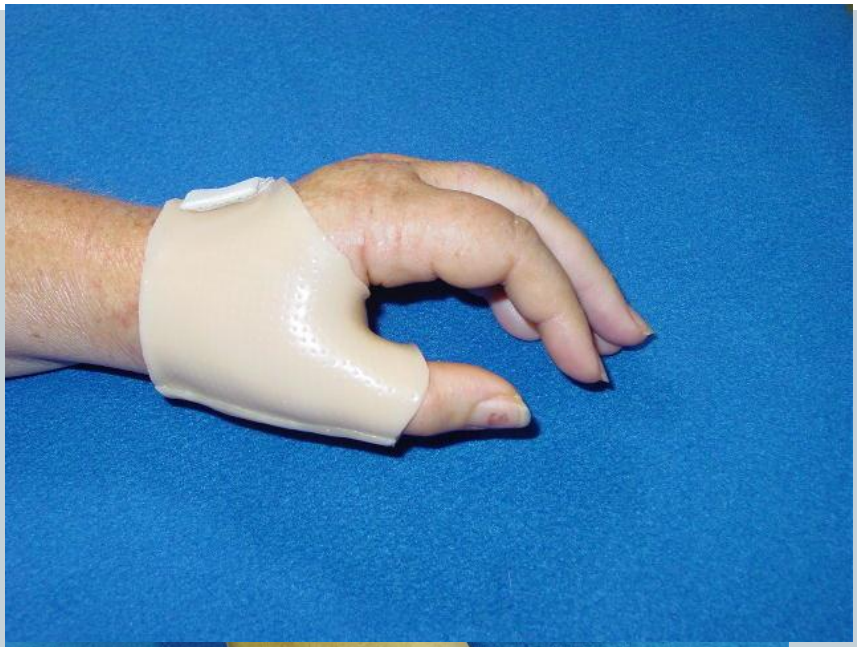
Splints



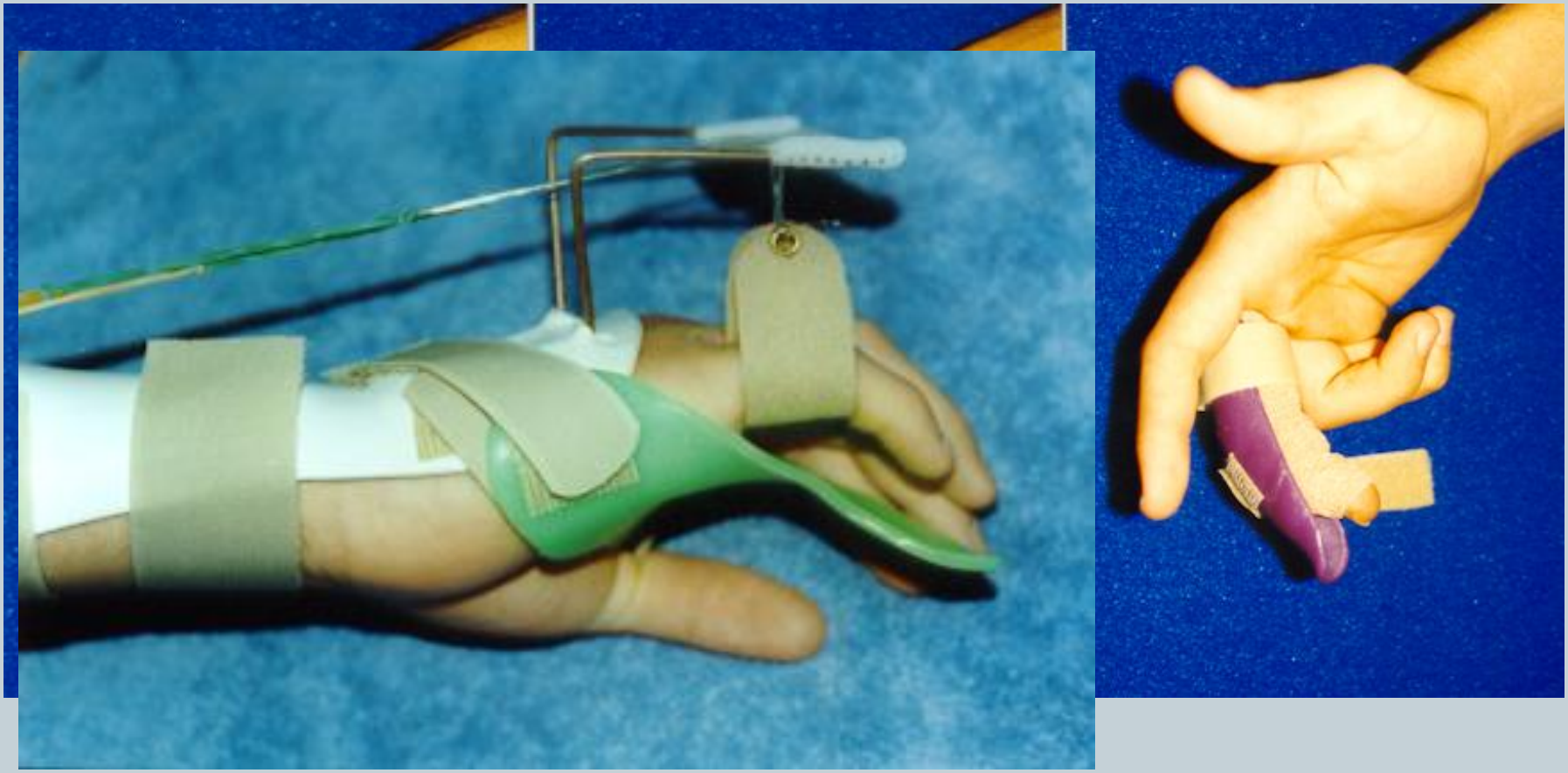
Stages of scar maturation determine appropriate type of splinting.

Inflammation: resting splints





Fibroplasia: Rest and ECM

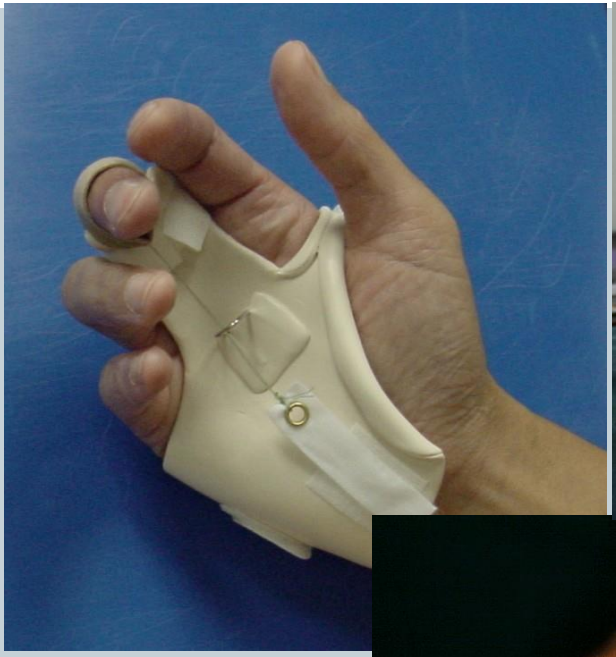


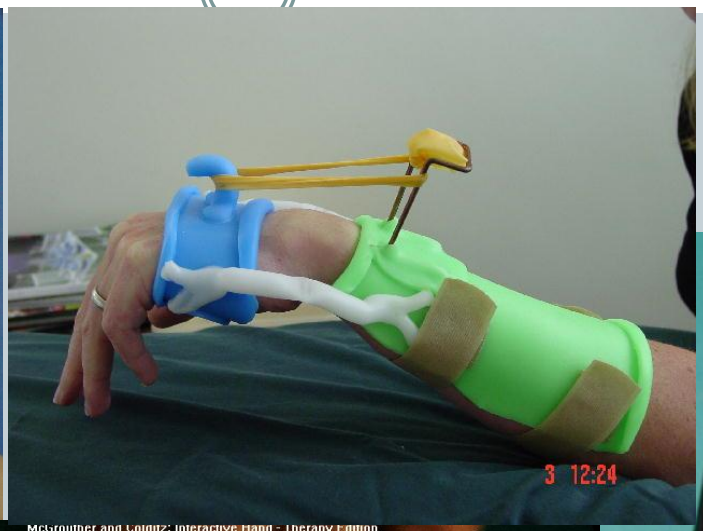
Splinting for LLPS

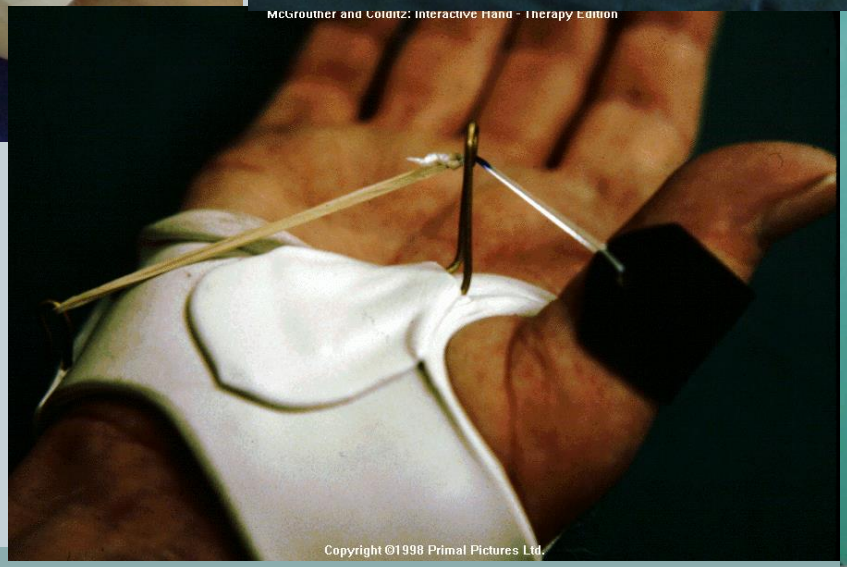
"….is the only available therapeutic modality that applies controlled gentle forces to soft tissues for sufficient length of time to induce soft tissue remodelling without causing microscopic disruption of cellular structures "

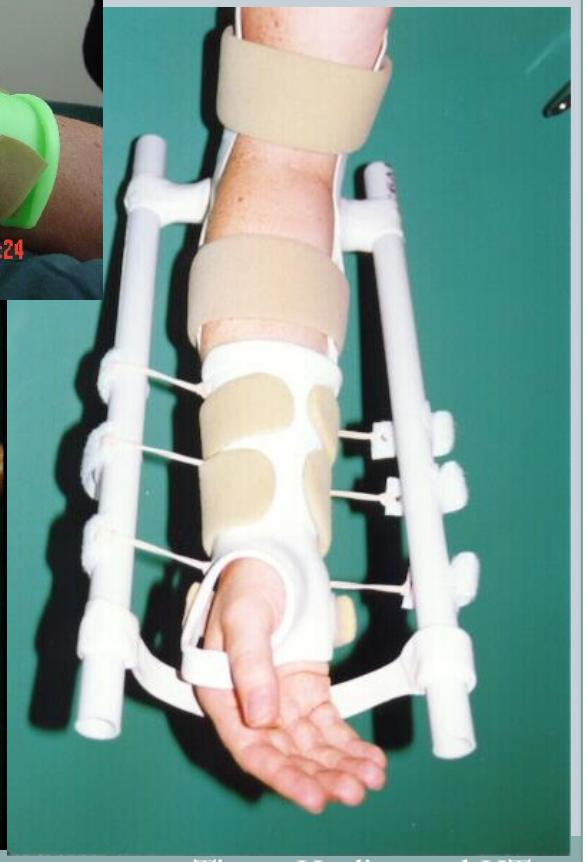
Fess & McCollum 1998

Remodelling: SPS & dynamic









Casting for motion (J Colditz)

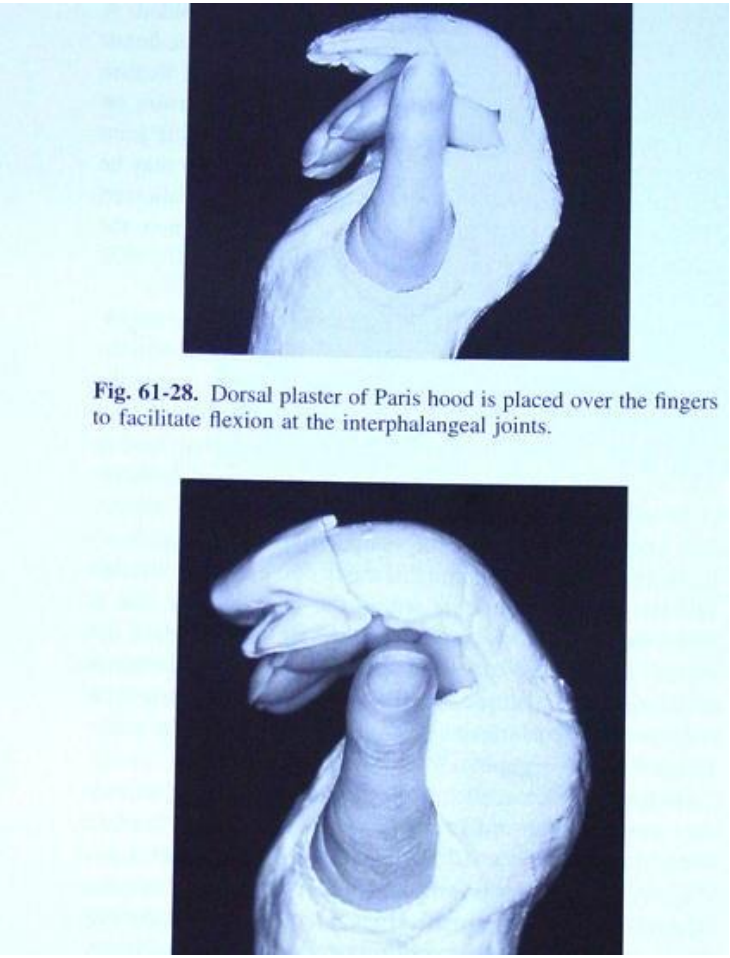


Fig. 61-28. Dorsal plaster of Paris hood is placed over the fingers to facilitate flexion at the interphalangeal joints.

Chronic oedema:inability to pump

↕

Multiple joint stiffness and extensive tissue adherence

↕

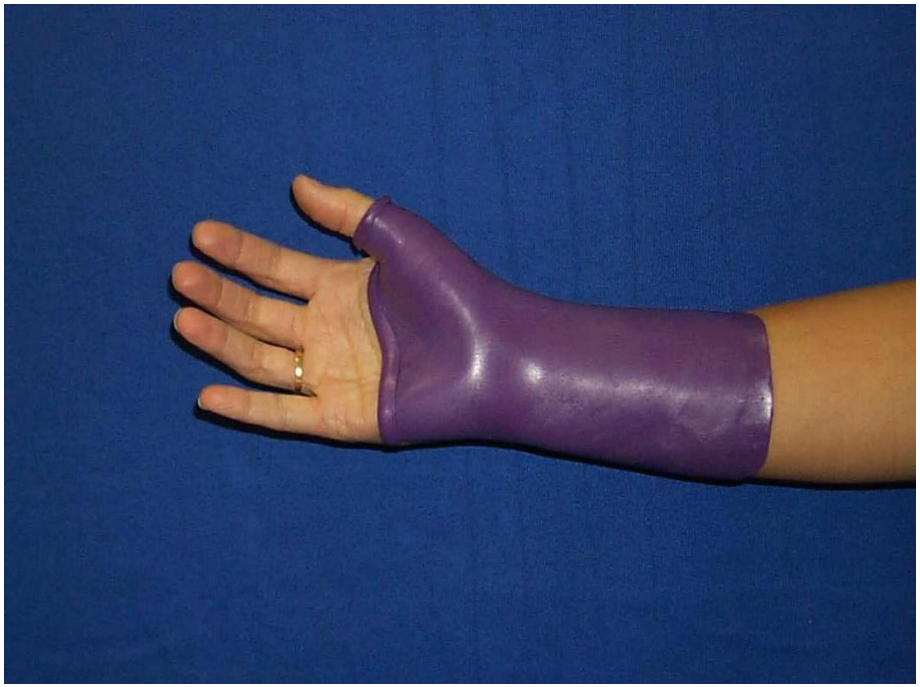
Ineffectual pattern of motion: repatterning of the motor cortex

Applying Stress: Splint mechanics

- "Splinting is both science and art, but the ultimate criterium is "does it work mechanicaly"
- Astute manipulation of mechanical concepts increases splint efficiency, enhances comfort and function.
- Creation of splint designs should be based on mechanical fact rather than on "mode-of-the-day (Fess JHT 1995)

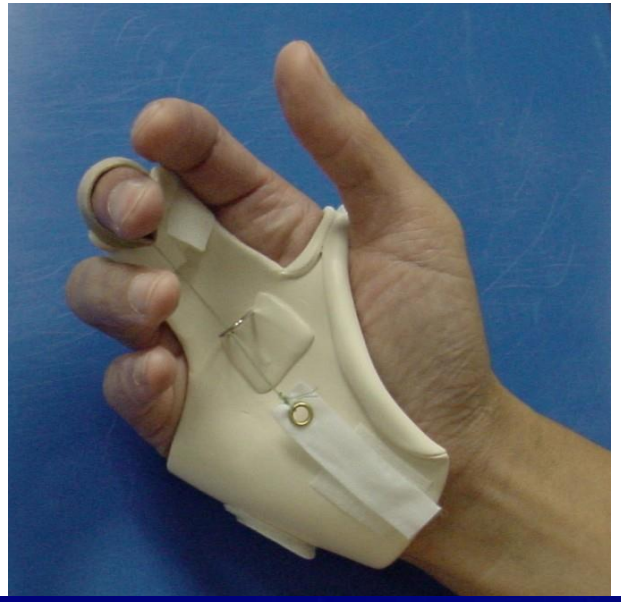
Principles of splinting

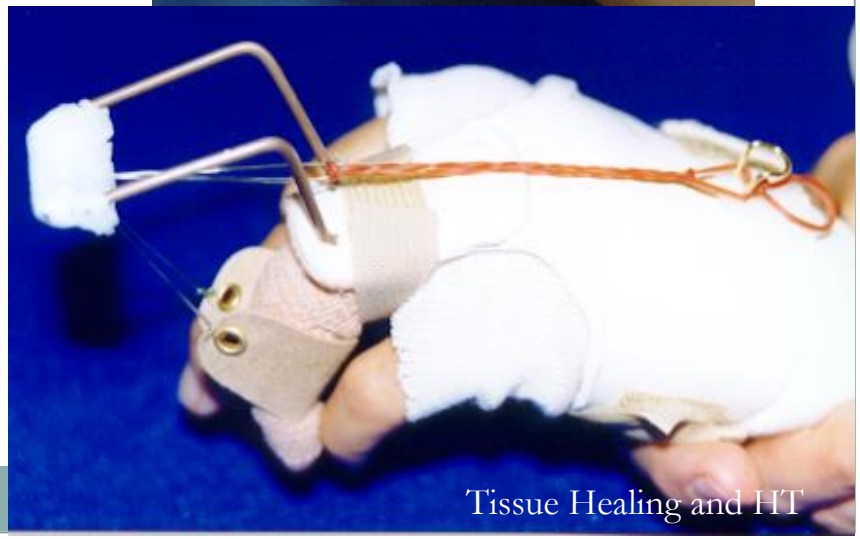
- Minimise pressure/ stress /shear
  - contour moulded : pressure distribution over wide area
  - Balanced splint length (lever principle and MA)
  - Rolled/rounded edges: tangent to edge of sphere
  - Pre pad over boney areas.
  - Width of straps



Principles of Splinting; Application of Forces

- understanding moments of force
  - line of pull at 90 to perpendicular.
    - Avoid distraction or compression on joint
  - How much force/torque.
    - Enough to move or reach end range, ie to counter soft tissue resistance
    - 100-300g recommended small joints of hand
    - Pain free.
  - Differences between high and low profile outriggers.





Principles of splinting

- Education (= compliance.)
  - Purpose of splint
  - Application and use: constant intermittent
  - precautions: pressure, force, pain, skin care.
  - Adjunctive exercises
- Regular review / modification
  - patient tolerance: skin, pain etc
  - Achievement of goals; compliance.

What makes a Good splint?

A. C. E

AESTHETICS: would YOU wear it ?

APPLICATION: easy to Don and Doff.

COMFORT: pressure minimised

EFECTIVE: achieves its goals

When do we apply what ??

Clinical reasoning based on subjective and objective assessment, applying knowledge of the expected reparative process for tissues involved and the normal time frames, including variables that might influence the norm.