Early Active Short Arc Motion
For the Zone III, IV Extensor Injury
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I. Historical Perspective
II. Rationale for Immediate Active Tension
  A. The effects of immobilization
    1. loss of glycosaminoglycan concentration
    2. loss of water
    3. decreased fibronectin concentration
    4. decreased endotenon healing
    5. loss of gliding function
    6. imposed injury to uninvolved tissues (cartilage and ligament) by immobilization
  B. The effects of controlled stress; biochemical/biomechanical benefits
    1. improved tensile strength
    2. increased fibronectin concentration/ fibroblast chemotaxis
    3. increased repair site DNA
    4. improved cellularity with both *motion* and *tension*
    6. maintain homeostasis in uninvolved tissues
  C. The effect of timing
III. Indications/Contraindications for Active Tension
  A. Not limited to any specific repair technique
  B. Appropriate for simple and especially complex injury if associated osseous injury will tolerate controlled motion and if the patient can understand treatment protocols
  C. Optimum time to start motion: 24-48 hours post-operative
  D. Caution if patients started late (>10 days)
  E. Patients need to be monitored in therapy 2/3 times per week for first 3 to 4 weeks
IV. General Considerations
  A. Variables of clinical decision making
    1. Level and complexity of injury (adjacent tissue injury)
    2. Biologic state of host/ personality and age of patient
    3. Repair technique/tensile strength of repair/tension on repair
    4. Drag which will determine resistance to tendon gliding
    5. Timing of referral to therapy: Early referral by day 3 preferred, and supported by basic science studies; referral at day 10 or greater will have associated problems of tendon to bone adherence which will elevate internal tension at the repair site with applied stress to the repair.
  B. Controlling inflammation
  C. The position of immobilization
  D. Duration of exercise
  E. Physiologic excursion

F. Application of force
1. Internal tendon tension transmitted to the repair site with specific joint angles and external loads (splinting configurations/exercise positions)

G. Resistance to tendon gliding

V. Guidelines for the open and repaired Zones III/IV (central slip) injury

A. Zones III and IV: Open or closed injury to the extensor tendon over the PIP joint or proximal phalanx may result in a boutonniere deformity.
1. Splint position
a. traditional management: Uninterrupted splinting of the PIP joint at absolute 0 degrees extension for 4 to 6 weeks with digital cylinder casting or volar thermoplastic splinting
b. SAM protocol (see Evans J Hand Surg 19A:991-997, 1994) Volar digital static extension splint for PIP and DIP joints holding both at 0 degrees of extension between exercises

2. Timing: SAM protocol for immediate active *short arc motion* preferably 24 hours post-operative (for open injury and repaired tendon)

3. Exercise: template splint 1 allows 30 degrees PIP flexion and extension (DIP is allowed to flex simultaneously to 25 degrees). Template splint 2 positions the PIP at O degrees and is cut away at the DIP level. Full DIP flexion is allowed if the lateral bands were not repaired, but only 25 degrees flexion is allowed if the lateral bands were repaired. *If no lag* develops 40 degrees PIP flexion is allowed by week 3, and 60 to 70 by the end of week 4; 90 by week 6. Exercise position is wrist 30 degrees flexion, MP at 0 degrees to slight flexion and IP joints as described within the template splints. (see Evans RB, Thompson DE. J Hand Ther 6:4. 266-284, 1993)

4. Clinical results: 64 digits in 55 patients with open and repaired zone III extensor tendon injuries compared as two groups; 76%/77% complex in groups I (3-6 weeks immobilization) and II (immediate active short arc motion) . Early motion group (SAM) experienced less extensor lag (-13 vs.-3), improved flexion (PIP and DIP, shorter treatment time (76 vs. 51days to discharge), and no boutonniere deformity.

B. Discussion
1. Problems associated with zones III and IV associated with the often complex nature of the injury, unfavorable ratio of tendon-to-bone interface in zone IV, improper postoperative splinting, and the effect of stress deprivation on associated connective tissue.

2. SAM program allows 4mm of excursion Zones III, IV calculated by radians.

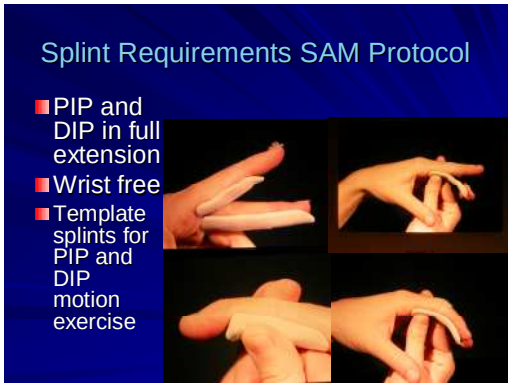
3. Isolated DIP motion creates lateral band excursion

4. Active extension from 30 degrees flexion to 0=300gm internal tendon tension

5. The position of wrist flexion decreases workload of EDC and facilitates interossei which extend the PIP when the wrist is flexed; the position of

MP extension transmits force to the EDC in zones III and IV because the sagittal bands glide proximal with MP extension; some active tension desired to create true proximal migration of the repair site and tostimulate cellular events of healing; the position of MP extension reduces the workload of the EDC through the action of the lumbricals and interossei. The lumbricals assist IP extension both directly through the action on the PIP joint, and indirectly by neutralizing the viscoelastic resistance of the flexor digitorum profundus. The interossei work to assist IP extension with the MP in extension

* It is not necessary to splint more than the PIP and DIP joints
* Early motion at this level is supported by a number of clinical studies (Saldana et al 1991; Pratt et al 2002; Kalb et al 2008; Kayalar M et al 2009; McAuliffe 2011)



VI. **SAM for the closed, non-operated boutonnière deformity**

A. **Serial casting 2-3 weeks combined with SAM exercise in therapy sessions; cast changes 2x per week as edema is reduced.**
B. **3-6 weeks digital splinting as described for open and repaired CS injuries, slow progression to flexion angles, emphasis on PIP extension and isolated distal joint flexion to stretch the ORL and balance LB.**
C. **Results: report on 36 cases, PIP average 6-92°; within 3 weeks of injury with passively correctable PIP extension.**

Recent advances in tendon management include surgical technique, manipulation of the biologic and biomechanical environments to improve intrinsic repair response and diminish extrinsic healing, and efforts to reduce frictional resistance to tendon excursion. But for alterations in surgical technique and the application of mechanical stress with splint geometry and patterns of motion these new biologic therapies do not yet have clinical application. (Boyer et al 2005; Huang et al 2006; Shearn et al 2011; Amadio 2011; Sun et al 201)

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