

Rehabilitation of Distal Radius Fractures: A Biomechanical Guide

David J. Slutsky, MD, FRCS(C)*, Mojca Herman, MA, OTR/L, CHT

3475 Torrance Blvd., Ste. F, Torrance, CA 90503, USA

Watson-Jones pointed out that a fracture is a soft tissue injury that happens to involve the bone [1]. One must keep in mind that the soft tissue envelope greatly influences the final functional result, even though all of the initial attention may be focused on the fracture position. The inflammatory cascade that results in edema, pain, and joint stiffness must be treated aggressively and concomitantly with the bony injury. Distal radius fractures range from simple extra-articular fractures to daunting complex multi-fragmented fracture dislocations. Extra-articular and minimally displaced intra-articular fractures often can be treated with closed reduction and cast application. Comminuted extra-articular and displaced intra-articular fractures often require more rigid fixation. The basic science behind fracture healing and the inflammatory response is reviewed in this article, with a mind to the rehabilitation forces that can be applied during various stages of the healing process.

Basic fracture healing

The major factors determining the mechanical environment of a healing fracture include the rigidity of the selected fixation device, the fracture configuration, the accuracy of fracture reduction, and the amount and type of loading at the fracture gap [2]. The fracture site stability may be enhanced artificially by a variety of external or internal means that includes cast treatment, pins, external fixation, and plates. Fracture healing under unstable or flexible fixation typically occurs

by callus formation. This applies to cast treatment with or without supplemental pin fixation and external fixation. The sequence of callus healing can be divided into four stages [3]. The stages overlap and are determined arbitrarily as follows.

Inflammation (1–7 days)

Immediately after a fracture there is hematoma formation and an inflammatory exudate from ruptured vessels. The fracture fragments are freely movable at this point.

Soft callus (3 weeks)

This corresponds roughly to the time that the fragments are no longer freely moving. By the end of this stage there is enough stability to prevent shortening, although angulation at the fracture site still can occur.

Hard callus (3–4 months)

The soft callus is converted by enchondral ossification and intramembranous bone formation into a rigid calcified tissue. This phase lasts until the fragments are united firmly by new bone.

Remodeling

This stage begins once the fracture has united solidly and may take from a few months to several years.

Four biomechanical stages of fracture healing also have been defined: stage I, failure through original fracture site, with low stiffness; stage II, failure through original fracture site, with high stiffness; stage III, failure partially through original fracture site and partially through intact

* Corresponding author.

E-mail address: d-slutsky@msn.com (D.J. Slutsky).

bone, with high stiffness; and stage IV, failure entirely through intact bone, with high stiffness. These data help determine the level of activity that is safe for patients with a healing fracture [4].

The distal radius is composed largely of cancellous metaphyseal bone. Bone healing in cortical and cancellous bone is qualitatively similar, but the speed and reliability of healing is generally better in cancellous bone because of the comparatively large fracture surface [5]. Most extra-articular fractures heal by 3–5 weeks after injury [6].

For distal radius fractures, stage I would correspond roughly to the initial 4 weeks or the soft callus phase. Protection of the fracture from excessive force is needed to prevent shortening and angulation. Stage II would coincide with the 4–8-week time period. The period beyond 8 weeks would represent stages III and IV in which the fracture has united clinically and can tolerate progressive loading.

Fracture site forces

Movement of the bone fragments depends on the amount of external loading, stiffness of the fixation device, and stiffness of the tissue bridging the fracture. The initial mechanical stability of the bone fixation should be considered an important factor in clinical fracture treatment [7].

The physiologic forces with wrist motion have been estimated to lie between 88–135 Newtons (N) [8,9]. Eighty-two percent of the loads across the wrist are transmitted through the distal radius [10]. Cadaver studies have demonstrated that for every 10 N of grip force, 26 N is transmitted through the distal radius metaphysis. Given that the average male grip force is 463 N [11] or 105 psi (1 lb of force = 4.48 N), this would imply that up to 2410 N of force could be applied to the distal radius during power gripping [12]. Previous studies of radius osteotomies showed that plates fail at 830 N [13]. External fixators compress as much as 3 mm under a 729 N load [14]. To prevent a failure of fixation the grip forces during therapy should remain less than 159 N (36 psi) for plates and less than 140 N (31 psi) for external fixators during the initial 4 weeks [13,14]. Gripping and strengthening exercises should be delayed until there is some fracture site healing.

When a bone fractures, the stored energy is released. At low loading speeds the energy can dissipate through a single crack. At high loading speed the energy cannot dissipate rapidly enough through a single crack. Comminution

and extensive soft tissue damage occur [15]. Fractures that exhibit multiple fracture lines are thus inherently more unstable because of the greater energy absorption at the time of injury. The difference in stability between an undisplaced fracture and a displaced fracture with comminution is significant and dictates a slower pace of fracture site loading during rehabilitation.

Biochemical response to injury

The basic response to injury at the tissue level is well known. It consists of overlapping stages, including an inflammatory phase (1–5 days), a fibroblastic phase (2–6 weeks), and a maturation phase (6–24 months) [16]. Following a fracture there is bleeding from disrupted vessels, which leads to hematoma formation. Several chemical mediators, including histamine, prostaglandins, and various cytokines are released from damaged cells at the injury site, inciting the inflammatory cascade [17,18]. The resultant extravasation of fluid from intact vessels causes tissue swelling [19].

Edema fluid

Simple hand edema is a collection of water and electrolytes. It is precipitated by myriad events, such as limb immobilization or paralysis, axillary lymph node disorders, and thoracic outlet compression. Edema restricts finger motion by increasing the moment arms of skin on the extensor side and by direct obstruction on the flexor side. Since the work that is needed to effect a joint angle change is dependent upon the product of the tissue pressure and the volumetric change during angulation, there is an increase in the muscular force that is necessary to bend a swollen finger. Compression, repeated finger flexion, and dynamic splinting redistribute this fluid to areas with lower tissue pressure. This allows the skin to lie closer to the joint axis, which decreases the effort needed for finger flexion [20].

Inflammatory hand edema has the same mechanical effects as simple edema and is treated in a similar fashion. The consequences of neglect, however, are dire. The swelling that occurs after wrist trauma as a part of the inflammatory response consists of a highly viscous protein laden exudate. This exudate leaks from capillaries and contains fibrinogen. In many instances the fibrin network is resorbed by approximately 7–10 days. Other times the fibrinogen is polymerized into fibrin, which becomes a lattice work for invading

fibroblasts. The fibroblasts produce collagen, which, if the part is immobilized, forms a randomly oriented, dense interstitial scar that obliterates the normal gliding surfaces [21]. The excessive fibrosis also impedes the flow of lymphatic fluid [22], which perpetuates the edema (Box 1).

Tendon gliding

Much of the work on tendon gliding has been applied to tendon repairs. The information gleaned from this work, however, has therapeutic implications with regard to distal radius fractures (Box 2). The dorsal connective tissue of the thumb and phalanges forms a peritendinous system of collagen lamellae that provides gliding spaces for the extensor apparatus [24–26]. The extensor retinaculum is divided into six to eight separate osteofibrous gliding compartments. Within the tunnels and proximal and distal to it, the extensor tendons are surrounded by a synovial sheath [27]. The flexor tendons are surrounded similarly by a synovial bursa and pass through a clearly defined pulley system. Hyaluronic acid is secreted from cells lining the inner gliding surfaces of the extensor retinaculum and the annular pulleys [28,29]. The hyaluronate serves to decrease the friction force or gliding resistance at the tendon pulley interface through boundary lubrication [30]. This in turn influences the total work of finger flexion [31].

Fracture hematoma can interfere with this boundary lubrication. Injury to the gliding surfaces by fracture fragments or surgical hardware can affect tendon excursion and can lead to adhesions. Adhesions also can occur in nonsynovial regions such as the flexor mass of the forearm and can restrict the muscle's gliding and lengthening properties [32]. Differential tendon gliding and active finger flexion are necessary to restore range of motion.

Box 1. Edema management

Acute edema

- Compression, elevation
- Active/passive finger motion
- Icing
- Retrograde massage

Chronic edema

- Jobst intermittent compression unit (Jobst Co.; Toledo, OH); ratio of inflation to deflation time is 3:1 [23]

Box 2. Tendon gliding exercises

Immobilized wrist

- Straight position (MP, PIP, and DIP joints extended)
- Platform position (MP joints flexed, PIP and DIP joints extended)
- Straight fist (MP and PIP joints flexed, DIP joints extended)
- Hook fist (MP joints extended, PIP and DIP joints flexed)
- Full fist (MP, PIP, and DIP joints flexed)

Mobile wrist

- Synergistic wrist flexion and finger extension
- Synergistic wrist extension and finger flexion
- Active and passive finger extension with wrist extended $>21^\circ$
- Active and passive thumb extension with wrist neutral in ulnar deviation

Tendon excursion

Wehbe and Hunter studied the in vivo flexor tendon excursion in the hand. With the wrist in neutral, the superficialis tendon achieved an excursion of 24 mm and the profundus tendon 32 mm. The flexor pollicis longus excursion was 27 mm. When wrist motion was added, the amplitude of the superficialis became 49 mm, the profundus tendon, 50 mm, and the flexor pollicis longus tendon, 35 mm [33,34]. Passive proximal interphalangeal (PIP) flexion results in more flexor tendon excursion than distal interphalangeal (DIP) flexion [35]. This knowledge formed the basis for an exercise program including three basic fist positions: hook, fist, and straight fist, which allows the flexor tendons to glide to their maximum potential [36]. Synergistic wrist extension and finger flexion increase passive flexor tendon excursion by generating forces that pull the tendon through the pulley system [37].

Extensor tendon gliding can be facilitated by extending the wrist more than 21° . This allows the extensor tendons to glide with little or no tension in zones 5 and 6 [38]. Similarly, positioning the wrist close to neutral with some ulnar deviation minimizes friction in the extensor pollicis longus sheath [39].

Immobilization

There is a constant turnover and remodeling of tissue components. Collagen in particular is absorbed and then laid down again with updated length, strength, and new bonding patterns in response to stress. The periarticular tissue adaptively shortens if immobilized in a shortened position, which leads to clinical joint stiffness [40]. This tissue includes the skin, ligaments, capsule, and the neurovascular structures [41]. To restore the length of the shortened tissue, one must hold the tissue in a moderately lengthened position for significant time so that it grows. Growth takes a matter of days, and the stimulus (ie, splinting) needs to be continuous for hours at a time to be most effective.

Tissue biomechanics

Stress is the load per unit area that develops in a structure in response to an externally applied load. *Strain* is the deformation or change in length that occurs at a point in a structure under loading [42]. Various materials have an elastic region whereby there is no permanent deformation of the material after the load is removed, eg, a rubber band. When the point of no return is exceeded (the yield point), there is permanent deformation of the material, eg, bending a paper clip until it deforms.

Collagen contributes up to 77% of the dry weight of connective tissue. The fibers are brittle and can elongate only 6%–8% before rupturing [43]. Elastin comprises only 5% of the soft tissue weight, but it can elongate 200% without deformity [44].

Viscosity is the property of a material that causes it to resist motion in an amount proportional to the rate of deformation. Slower lengthening generates less resistance. Any tissue whose mechanical properties depend on the loading rate is said to be viscoelastic. Biologic tissue is viscoelastic in that it has elastic properties but also demonstrates viscosity at the same time.

Skin and connective tissue is a polymer of loosely woven strands of elastin and coiled collagen chains. With the initial application of tension, little force is needed for skin elongation. The elastin and the collagen chains are unfolding and aligning with the direction of the stress rather than stretching per se. When all of the fibers are lined up parallel to the line of pull, the tissue becomes stiff. Each fiber is uncoiled and can elongate only 6%–8%. A much greater force now produces minimal additional

gains in length. Further attempts at rapid lengthening exceed the fiber's elastic limit, causing microscopic tearing, bleeding, and inflammation. This leads to fibrin deposition with secondary interstitial fibrosis, which may result in further contracture [20].

If the stretching force is applied slowly, the collagen microfibrils have time to slide past one another. This slippage (or creep) allows the polymer chains to recoil. The tissue now has been lengthened permanently (plastic behavior) together with lessening of the tension over time (stress relaxation). If the same tissue is held in a slightly lengthened position for a period of hours or days, the collagen fibers are absorbed then laid down again with modified bonding patterns, without creep or inflammation. Brand refers to this as growth rather than stretch [20].

Types of splints

The principles of splinting exploit the biomechanical properties of tissue to overcome contracture and regain joint motion following injury. The types of splints may be grouped as follows:

- **Static:** Rigid splints used for immobilization. Restrict unwanted arcs of motion (Fig. 1A,B).
- **Serial static:** Serial application of plaster casts. Relies on tissue growth [45].
- **Dynamic:** Continuous load applied through elastic bands or springs. Relies on time-dependent material property creep. The dynamic force continues as long as the elastic component can contract, even beyond the elastic limit of the tissue (Fig. 2A,B).
- **Static-progressive:** Static progressive stretch. Relies on the principle of stress-relaxation [46]. Construction is similar to dynamic splints except these splints use nonelastic components, such as nylon fishing line, turnbuckles, and splint tuners. Once the joint position and tension are set, the splint does not continue to stress the tissue beyond its elastic limit [47]. As the tissue lengthens, the wearer adjusts the joint position to the new maximum tolerable length (Fig. 3A–C).

Fracture rehabilitation

For the purposes of rehabilitation it is useful to consider the stability of the distal radius fracture site in three phases, which in turn guides the therapist as to the loads that can be placed across the fracture site. When internal or external fixation is used, the loads placed on the fracture site

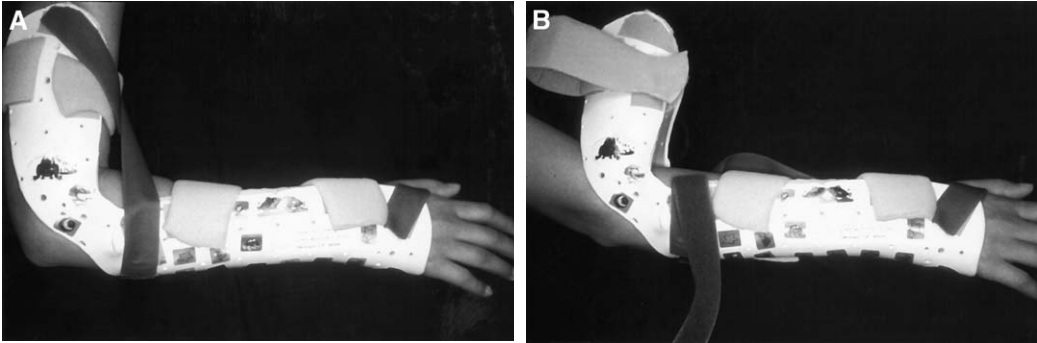


Fig. 1. Restrictive splint. (A) Above elbow splint restricts wrist motion and forearm pronation and supination. (B) Splint does allow elbow flexion and extension.

may be adjusted accordingly. A rough knowledge of the intrinsic or augmented fracture site stability and the expected forces that are generated during therapy are necessary to minimize fracture site deformity (see section on fracture site forces).

Phase I

This phase is defined by low fracture site stiffness (stage I; see section on basic fracture healing). The wrist splints used at this stage are static and are used for immobilization to limit unwanted motion, to prevent displacement at the fracture site, and to prevent or correct joint contractures. Protected wrist motion is initiated in this phase.

Phase II

This phase is characterized by increasing fracture site stiffness that should be able to withstand the forces generated with light strengthening and dynamic/static progressive wrist splinting (stage II).

Phase III

In this phase there is sufficient fracture site stability to tolerate the loads generated during gripping and lifting (stages III and IV). Dynamic/static progressive wrist splinting continues until motion plateaus.

South Bay Hand Surgery Center protocol

Controlled and progressive joint mobilization following trauma has been shown to give superior results to immobilization [48]. The biochemical and biomechanical events that occur during fracture healing provide the underlying foundation for the rehabilitation program following a distal radius fracture. The therapy protocol for regaining finger motion is tiered and instituted immediately in all patients (Box 3). Tendon gliding exercises and passive finger motion with the wrist neutral are started immediately, because there are no

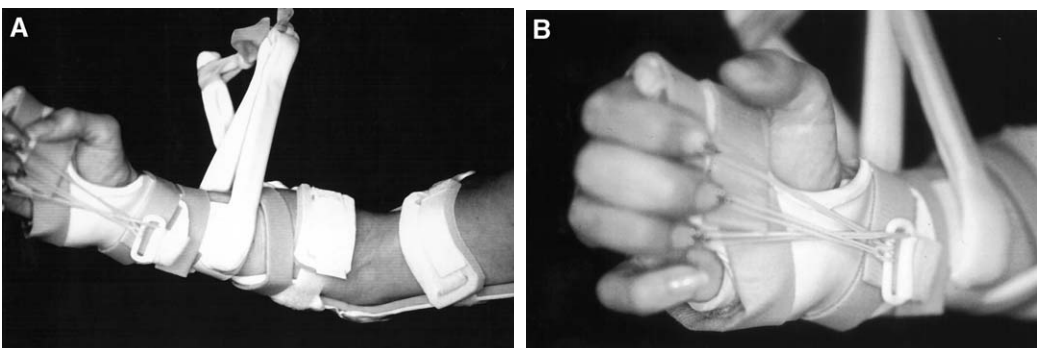


Fig. 2. Dynamic splints. (A) Dynamic supination splint relies on elastic band tension. (From Kleinman WB, Graham TJ. The distal radioulnar joint capsule: clinical anatomy and role in posttraumatic limitation of forearm rotation. *J Hand Surg [Am]* 1998;23:588-99; with permission.) (B) Dynamic PIP flexion splint added to allow simultaneous finger and forearm splinting.

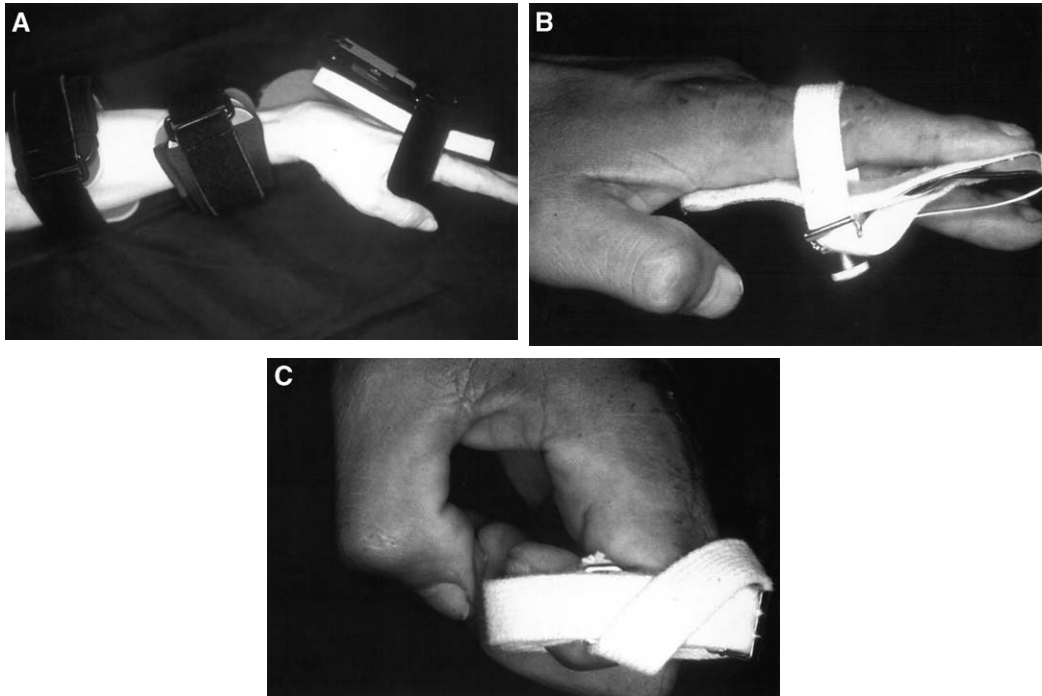


Fig. 3. Static progressive splints. (A) Static progressive wrist flexion splint. (B) Static progressive PIP extension splint. (C) PIP and DIP flexion strap used to regain DIP motion. Note that it cannot increase PIP motion beyond 90° because of its vector force.

biomechanical concerns regarding phalangeal stability. Dynamic and static progressive splinting are instituted early if necessary, based on the observation that the total active finger motion typically plateaus by 3 months [49]. In the authors' experience, static progressive splinting of the fingers is more painful and hence is instituted only after no further gains are seen with dynamic splinting.

Wrist motion is initiated at different times depending on the fracture site stability and the type of splinting or fixation (Box 4). Patient factors, such as age, bone density, pain tolerance, and systemic disease may influence significantly the pace of therapy, which should be adjusted accordingly. Synergistic wrist and finger motion for tendon excursion are started in tandem with wrist motion (see Box 2). Forceful gripping is delayed until there is some fracture site healing.

Procedure-specific treatment

Cast treatment

Cast treatment is nonrigid fixation: it reduces fracture site mobility but does not abolish it

because of the intervening soft tissue. A cast relies on three-point fixation to maintain the fracture position. If the wrist is casted in a flexed and ulnar deviated position, a component of ligamentotaxis is also in play. The initial focus of therapy is directed toward reestablishing finger motion. Active finger motion should be gentle and not pushed early on, because the flexed and ulnar deviated wrist position relaxes the flexor tendons and tightens the extensors, making it painful to make a fist.

Displaced fractures often are associated with more soft tissue trauma, which leads to more swelling and slower healing. The loss of the immobilizing soft tissue envelope around the bones also leads to greater fracture site instability. In these cases it may be necessary to delay strengthening exercise as well as dynamic splinting of the wrist.

Rehabilitation

- Week 1–6: finger rehabilitation protocol
- Week 6–8 (after cast removal): phase I wrist exercises
- Week 8–10: phase II wrist exercise
- >10 weeks: phase III wrist exercises

Box 3. Finger rehabilitation protocol*Day 1–7*

- Individual passive and active finger and thumb motion
- Thumb opposition exercises
- Intrinsic muscle stretching exercises
- Aggressive edema management (see [Box 1](#))
- Tendon gliding exercises (see [Box 2](#))

Week 2–4

- Dynamic PIP flexion splint if passive PIP flexion $<60^\circ$
- Switch to PIP flexion strap after $>80^\circ$ of passive PIP flexion achieved
- Dynamic MP flexion splint if passive MP flexion $<40^\circ$
- Dynamic PIP and DIP flexion strap if passive DIP flexion $<40^\circ$
- Intrinsic muscle tightness: dynamic PIP flexion splint with MP blocked in full extension

Week 4–8

- Switch to static progressive PIP splint if flexion is still $<60^\circ$
- Switch to static progressive MP splint if flexion is still $<40^\circ$
- Dynamic/static progressive PIP extension splint if PIP flexion contracture $>30^\circ$
- Dynamic MP extension splint if MP flexion contracture $>30^\circ$
- Dynamic/static progressive thumb opposition splint if opposition >2 cm from fifth MPJ

After week 8

- Home splinting until motion plateaus

Box 4. Wrist rehabilitation protocol*Phase I: low fracture site rigidity*

- Custom or noncustom below-elbow splint
- Gentle active and passive wrist flexion/extension, pronation/supination

Phase II: intermediate fracture site rigidity

- Add dynamic/static progressive splinting if wrist flexion $<30^\circ$
- Add dynamic/static progressive splinting if wrist extension $<30^\circ$
- Dynamic/static progressive supination splinting if $<60^\circ$
- Dynamic/static progressive pronation splinting if $<60^\circ$
- Address functional activities, light strengthening

Phase III: high fracture site rigidity

- Progressive strengthening exercises
- Home splinting until motion plateaus

static progressive elbow extension splints if elbow flexion contracture is $>30^\circ$ at 8 weeks.

- Satisfactory results can be achieved with a home program in uncomplicated Colles fractures [50].

Intrafocal pinning

Intrafocal pinning is indicated in unstable extra-articular distal radius fractures. Intrafocal pinning, however, does not provide rigid fixation. Supplemental cast or splint immobilization is necessary for 4–6 weeks; otherwise, early wrist motion may produce pain and dystrophy. The therapy protocol differs little from cast treatment alone, but there are added requirements for pin site care. Typically K-wires are introduced through the snuffbox, where injury and irritation of the superficial radial nerve branches (SRN) are common [51]. Pin site interference with thumb or finger extensors requires added emphasis on thumb opposition and extensor tendon gliding exercises (see [Box 2](#)). If comminution involves more than two cortices or if the patient is older than 55 years of age, there is a high likelihood of subsequent fracture collapse [52]. In these cases supplemental external fixation or a spanning bridge plate may be used. Wrist motion therefore is delayed until after fixator/plate removal.

Special considerations

- Ensure the cast does not block thumb and finger metacarpophalangeal (MP) flexion creases to minimize collateral ligament contracture/intrinsic tightness.
- Avoid wrist hyperflexion in the cast. Bivalve/remove cast with any signs of acute carpal tunnel syndrome (may require additional fixation).
- If an above-elbow cast is used, regaining forearm rotation is more difficult. Institute

Rehabilitation

- Week 1–6: finger rehabilitation protocol
- Week 6–8 (after pin removal): phase I exercises
- Week 8–10: phase II exercises
- >10 weeks: phase III wrist exercises

Special considerations

- Pin site care
- After pin removal
 - Superficial radial nerve (SRN) desensitization
 - Ulnar deviation exercises (with radial sided pins)

External fixation

External fixation may provide improved wrist motion through less interference with the soft tissue envelope [53]. External fixation is considered flexible fixation. Regardless of the type of external fixator, callus development is the overriding element providing the rigidity of the fixator–bone system [37]. The stability of fixation can be enhanced significantly through the addition of 0.62 percutaneous K-wires, which approaches the rigidity of a 3.5-mm dorsal AO plate (Synthes, Inc.; Paoli, PA) [54,55]. With intra-articular fractures, increasing the rigidity of the fixator does not increase appreciably the rigidity of fixation of the individual fragments [56]. Augmentation with percutaneous K-wire fixation reduces the dependence on ligamentotaxis to position the fragment and significantly increases the stability of the construct, especially when the K-wire is attached to an outrigger [9].

Pitfalls of ligamentotaxis

External fixators may be applied in a bridging or nonbridging manner. Bridging external fixation relies on ligamentotaxis. Wrist distraction combined with hand swelling predisposes toward extensor tightness, which mandates an emphasis on MP to DIP flexion exercises. If necessary a dynamic MP flexion splint is applied while the fixator is still in place (Fig. 4). Added extensor tendon stretch is accomplished by strapping the PIP and DIP joints in full flexion while using the dynamic MP flexion splint. Overdistraction of the wrist leads to intrinsic tightness and subsequent clawing of the fingers [57]. The index finger extensor tendons are especially sensitive to this and act as an early sentinel warning device.

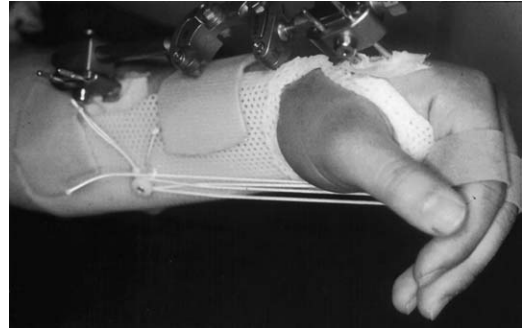


Fig. 4. Dynamic MP flexion splint. This splint is converted easily to a static progressive splint by using a nonelastic nylon line and substituting a splint tuner for the post.

Patients with external fixators often keep their forearms in pronation, which may lead to contractures of the distal radioulnar joint [58]. Distraction, flexion, and locked ulnar deviation of the external fixator should be avoided, because they encourage pronation contractures and may predispose to acute carpal tunnel syndrome (Fig. 5A). Ideally the wrist should be positioned in mild extension, which relaxes the extensor tendons and facilitates finger flexion [57]. This often requires augmentation with percutaneous K-wire fixation of the fracture (Fig. 5B). Dynamic or static progressive supination splinting can be effective and should be instituted soon after fixator removal [59].

Because ligaments are viscoelastic, there is a gradual loss of the initial distraction force applied to the fracture site. The initial immediate improvement in radial height, inclination, and volar tilt are decreased significantly by the time of fixator removal [60]. For this reason, light gripping exercises or using the hand for activities of daily living (ADL) should not be encouraged in the initial 4 weeks, with fracture site loading being limited to <31 psi [12].

Nonbridging fixators allow the institution of early wrist motion (Fig. 6A,B). In these cases therapy includes the addition of early wrist flexion and extension in addition to the finger exercises. Radial deviation usually is blocked by the fixator itself, and ulnar deviation exerts traction on the fixator pin sites, which is painful. Simple extra-articular fractures can tolerate the earlier onset of loading as compared with comminuted extra-articular fractures. When nonbridging external fixation is used for complex intra-articular

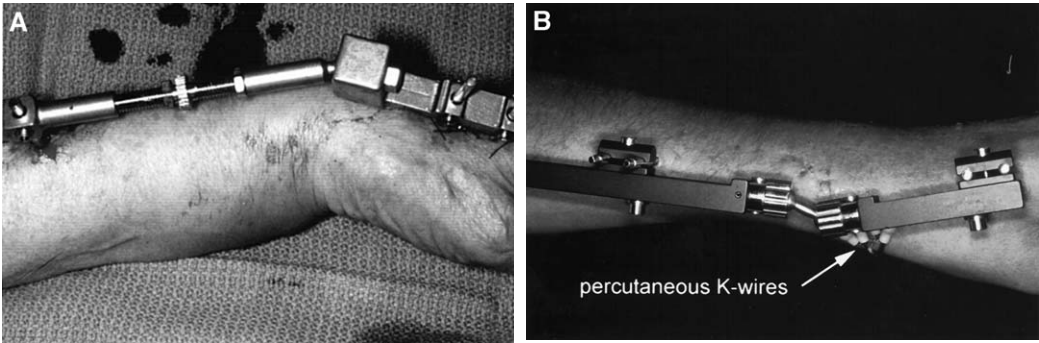


Fig. 5. External fixation. (A) Note the marked wrist flexion, which should be avoided. (B) Augmentation with K-wires allows external fixation with mild wrist extension.

fractures, articular incongruity is common [61]. This may be prevented by use of custom designed fixators with dorsal outriggers (Fig. 7).

Rehabilitation

Bridging external fixator

- Week 1–6: finger rehabilitation protocol
- Week 6–8 (after fixator removal): phase I wrist exercises
- Week 8–10: phase II wrist exercises
- >10 weeks: phase III wrist exercises

Special considerations

- Pin site care
- Aggressive MP flexion; add dynamic MP flexion splint if MP flexion $<40^\circ$ by 2 weeks
- Intrinsic tightness stretching; add dynamic intrinsic tightness splint as needed (MP extended, PIP/DIP flexed)
- After pin removal
 - SRN desensitization
 - Ulnar deviation exercises (with radial sided pins)



Fig. 6. Nonbridging external fixator. (A) Nonbridging application of an external fixator. (B) Fracture position is maintained without spanning the joint.

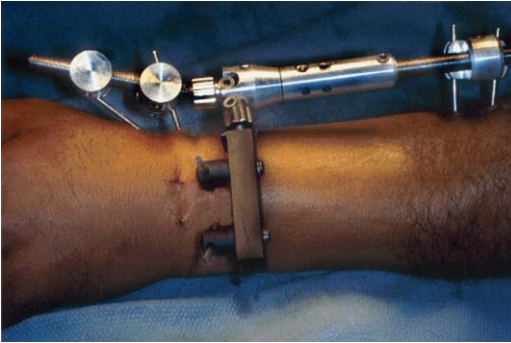


Fig. 7. Custom nonbridging external fixator with dorsal outrigger bar.

Nonbridging external fixator

- Week 1–6: finger rehabilitation protocol; phase I wrist exercises
- Week 6–10 (after fixator removal): phase II wrist exercises
- >10 weeks: phase III exercises

Plate fixation

Rigid fixation of fractures by plating alters the biology of fracture healing. When motion is abolished completely between fracture fragments, no callus forms [62]. This has been termed direct healing, whereby osteons directly bridge the fracture gap to regenerate bone, and the fracture heals by remodeling [63]. Conventional plate fixation relies on friction between the plate and bone interface for stability and the dynamic compression properties of the plate, which preload the fracture site [64]. In general, plate fixation allows earlier loading of the fracture site.

Newer locking plates act by splinting the fracture site without compression, resulting in flexible elastic fixation and stimulation of callus formation. Locking plates do not require friction to secure the plate to the bone. Comminuted diaphyseal or metaphyseal fractures are suited particularly to bridging fixation using locked plates [65]. Fragment-specific fixation (TriMed, Inc.; Valencia, CA) relies on a combination of low profile pin plates with a variety of flexible wire form buttress plates. The pin plates usually are applied dorsoradially underneath the first extensor compartment and dorsoulnarly between the finger extensors and the extensor digiti minimi, although volar applications are not uncommon.

Initially the plate bears all the stress; hence, the rehabilitation forces must not exceed their tolerance. As healing progresses the plate load shares until the fracture is healed and bears almost the entire stress [66]. Feedback from the operating surgeon is necessary as to the stability of fixation before instituting wrist motion and gripping, especially when there is significant intra-articular comminution.

Dorsal plating

Newer low profile dorsal plate designs were greeted with much enthusiasm [67,68]. Extensor tendon irritation is still a problem [69,70]. Rapid tendon acceleration through preload has been proposed as one method to maximize extensor tendon excursion [71].

Rehabilitation

- Week 1–4: finger rehabilitation protocol; phase I wrist exercises
- Week 4–8: phase II wrist exercises
- >8 weeks: phase III exercises

Special considerations

- Emphasize extensor tendon gliding (see Box 2)
- Emphasize wrist flexion
- Suspect extensor pollicis longus (EPL) impingement/entrapment with resistant thumb extensor tightness

Volar plating

Volar fixed angle plating is currently in vogue [72]. Proponents of this procedure cite improved fracture stability and better soft tissue coverage of the implant. Normal wrist extension may be difficult to regain, which has led some to recommend splinting the patient's wrist in 30° of extension between therapy sessions [71]. Dynamic or static progressive splints may be used if needed. Flexor tendon tightness may occur. This should be treated with dynamic MP extension splinting combined with static extension splinting of the PIP and DIP joints, while the wrist is incrementally brought from neutral to extension.

Rehabilitation

- Week 1–4: finger rehabilitation protocol; phase I wrist exercises
- Week 4–8: phase II wrist exercises
- Late (>8 weeks): phase III exercises

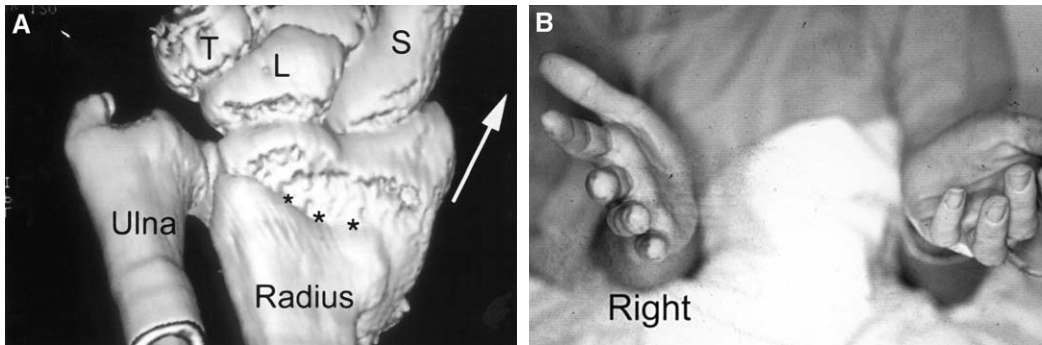


Fig. 8. Volar perspective, 3-D CT reconstruction of a right distal radius malunion. (A) The distal fragment is pronated (arrow) at the fracture site (*), which blocks supination at the distal radioulnar joint. S, scaphoid; L, lunate; T, triquetrum. (B) Clinical photograph showing attempted supination on the right.

Special considerations

- Suspect FPL entrapment with resistant thumb flexor tightness

Fragment-specific fixation

Same as for dorsal and volar plate fixation: hardware irritation of the first extensor compartment tendons and the finger extensors may occur from pins backing out. Emphasize thumb/finger extension with dynamic splinting as necessary.

Combined fixation

Some intra-articular fractures are so inherently unstable that combined internal and external fixation is necessary for the initial 6 weeks. In these instances strengthening exercises may need to be delayed longer than usual because of the risk for displacing the articular fragments. Combined volar and dorsal plating may devascularize bone fragments, which also may contribute to these delays. In these complex fractures it is important to have frequent communication with the treating surgeon together with a review of the radiographs before loading the fracture site.

Rehabilitation

- Same as for plate or external fixation, although fracture site loading may be delayed as necessary

Combined radius and scaphoid fixation

- Same as for plate or external fixation

Special considerations

- Variable delay in implementing wrist motion with carpal fracture-dislocation

- Delay strengthening until there is evidence of scaphoid union by CT scan

Causes of treatment failures

There are a large number of extrinsic tendons crossing the fracture site. Dorsal angulation of $>30^\circ$ and radial angulation $>10^\circ$ greatly affects the moment arms and subsequently the excursion and strength of these tendons [73].

If joint malalignment is the etiology of loss of forearm rotation, then continued therapy is of no benefit (Fig. 8A,B). Biomechanical studies have demonstrated that radial shortening ≥ 10 mm caused a 47% pronation loss and a 27% supination loss [74]. More than 10° of dorsal tilt leads to a dorsal carpal shift with compressive forces. This leads to feelings of pain and insecurity with gripping and difficulties with ADL [75]. More



Fig. 9. Malunited Colle's fracture. Note the marked dorsal tilt of the joint surface with dorsal migration of the carpus resulting in a secondary dorsal intercalated segmental instability pattern.

severe dorsal tilt leads to a dynamic dorsal intercalated segmental instability (Fig. 9) [76].

Summary

Fracture healing and surgical decision making are not always predictable. The suggested protocols are intended to be flexible rather than rigid to be responsive to patient progress and the fracture site stability. A methodologic approach to the rehabilitation following a distal radius fracture, based on a knowledge of the biology of fracture healing and biomechanics of fixation, may preempt some of the pitfalls associated with distal radius fracture healing.

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