The Stiff Finger

F. Thomas D. Kaplan, MD

KEYWORDS

- Finger • Contracture • Stiffness
- Proximal interphalangeal joint
- Metacarpophalangeal joint • Tendon adhesions

The stiff finger is a frequently encountered entity in hand surgical practice. It stems from a myriad of causes, may have multiple components, and requires a variety of solutions. Simple stiff fingers may result from a minor proximal interphalangeal (PIP) joint sprain in which the patient overprotects and self-limits motion, whereas a patient with a table saw injury requiring bone, tendon, and nerve repair presents a more complex problem. A true understanding of the ideal treatments for the stiff finger requires a basic understanding of the local milieu that arises from injury and the anatomic features that are at risk for pathologic changes. Hand surgeons must be able to help patients understand the various factors at play and the time course of wound healing and injury-induced inflammation, because an educated and motivated patient is the best ally in the battle against the stiff finger.

BIOLOGY OF WOUND HEALING

After a traumatic event, infection, or surgical insult, a predictable series of events is set into motion. The magnitude of the response and the duration depends on multiple factors. Local tissue injury triggers the onset of the healing response, which is comprised of three phases: (1) inflammatory, (2) tissue-producing, and (3) tissue remodeling. These phases overlap in time, and the ongoing events in each phase determine the elements that contribute to finger stiffness, and the opportunities available to mitigate those factors.

Beginning with initial injury and tissue disruption, vasoactive and chemotactic factors are produced by injured parenchymal cells and the activated coagulation and complement pathways.[1] The inflammatory cascade results in increased capillary permeability, which allows protein-rich exudate to leak out into the interstitial space. There is also a decreased ability to transport the fluid back out by way of lymphatics, resulting in edema. This extracellular exudative fluid contributes to the stiff finger through an increased resistance to movement, joint capsular distention, and swelling of the articular capsule and ligaments.[2] The exudates also contain fibrinogen, which can turn into fibrin, leading to interstitial scar.[3]

The chemotactic factors produced by injured cells and activated platelets recruit inflammatory leukocytes to the site of injury. These monocytes, neutrophils, and macrophages act to remove foreign debris and necrotic tissue from the wound and secrete matrix metalloproteinases (including collagenase) clearing the injured area and creating the void that is filled by scar tissue.[4] Approximately 4 days after injury new stroma (granulation tissue) begins to fill the void as fibroblasts proliferate and migrate into the wound. New capillaries form and invade the wound by angiogenesis, while macrophages continue to aid in remodeling.

Once the wound has closed and the dermis healed, wound contracture ceases, but scar contraction may continue. Collagen cross-linking both within and between tissues promotes adhesion formation and joint stiffness.[5] The interstitial scar attaches to, and becomes part of, the normal tissues it is in contact with, obliterating normal tissue planes. Fibroblasts differentiate into myofibroblasts, which are the cells primarily responsible for scar contracture.[3] The net result is a wound in which normal gliding structures have more resistance to motion, and are surrounded by exudate and interstitial scar, which connects them to adjacent, nonmobile structures.

EARLY INTERVENTIONS

In the early stages of injury, stiffness can be limited and corrected with appropriate management. The

Indiana Hand to Shoulder Center, 8501 Harcourt Road, Indianapolis, IN 46260, USA
E-mail address: tdk@hand.md

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factors that lead to stiff fingers, including edema, pain, immobilization, and patient participation, should be assessed and appropriate interventions begun. Often, patients come to the office after injury poorly splinted and poorly educated on edema control. The first step is to identify the patient's injury and decide the appropriate treatment. It is then known what needs to be immobilized, what can move, and what may need surgical treatment, planning treatment not only on the injury, but also the patient's entire arm. For example, patients presenting with a distal radius fracture often keep their arms in a sling. They allow their fingers to become swollen, and do not move their shoulder, elbow, or fingers well. What results is all too familiar: not only does the patient have a wrist that needs therapy, but they must also work out their frozen shoulder and stiff hand, decreasing the amount of time they are able to spend on their wrist.

The educated patient is a strong ally. Once the treatment for the injury has been determined, the clinician then needs to ensure the patient understands the plan and what their role is to promote optimal healing. This begins with aggressive edema control including elevation and compressive dressings. Patients should be taught to keep the hand higher than heart level until the swelling has resolved. Early motion can also help control swelling, while limiting stiffness. Joints not needing protection should not be blocked by the splint or cast, and should be kept moving through their full range of motion using both active and passive modalities.

Treatment of the injured area can either set the stage for an excellent outcome or provide a recipe for disaster. Fracture malunion can lead to tendon imbalance, alteration of joint biomechanics, and bony obstruction to motion. Fracture stabilization may allow early motion (rigid fixation); preclude motion (poor construct strength, persistent instability); allow protected partial motion (dorsal block k-wire); and promote tendon adhesions (bulk and location of implants, piercing of tendons with k-wires, transarticular fixation). Tendon and ligament repair may allow full or partial arc active motion, passive motion, or necessitate immobilization. Ideal treatment optimizes fracture stability, tendon and ligament repair strength, and restoration of the soft tissue envelope.

Following the initial phases of protected motion, and once the injured and repaired structures have gained sufficient structural integrity, rehabilitation can advance. In patients with simple injuries, often the progression to full active and passive motion followed by strengthening is all that is necessary to regain normal function. In patients with more complex injuries, and those showing a tendency toward development of a stiff finger, more aggressive interventions are required. This typically involves the use of dynamic or static-progressive splinting to restore the soft tissues back to their normal lengths and elasticity. The goal is to restore a supple finger with full passive motion. Limited active motion can then be addressed surgically if necessary.

LATE TREATMENT

Once the inflammatory cascade has ended, the soft tissues reach equilibrium. This point varies in each patient and may occur within the first 2 to 3 months, or may take up to 6 months. Patients at this point have frequently reached a plateau in their range of motion, no longer having fluctuating edema, and timing is now appropriate to assess their finger for further surgical intervention. The surgeon should keep in mind the motivation the patient exhibited during the early stages of healing, in addition to the objective findings, when determining the appropriateness of a surgical procedure. On physical examination, the hand and fingers should have minimal or no edema or induration. Scars should be mature and hypersensitivity absent. Each joint should be carefully measured for both active and passive range of motion. Note should be made if measurements are limited by patient pain or effort. The presence of obvious deformity may alert the clinician to an underlying tendon imbalance, malunion, or nonunion.

The pathology responsible for the stiff finger can now be determined. It may be caused by tendon adhesions, myotendonous contracture, joint capsular contracture, arthrofibrosis, or a combination of these. Underlying causes, such as malunion, nonunion, or a poor soft tissue envelope, should be excluded or treated first if present. The treatment must be tailored to the pathology, and the clinician must be systematic in designing not only the appropriate procedures, but also the best sequence of procedures. A classification system based on six possible combinations of limited active or passive motion was described by Jupiter and colleagues[6] in 2007. I have added two additional categories, Type 7 and Type 8, to complete the system such that all possible combinations of limited motion are included (**Table 1**). By determining the appropriate category into which the patient's finger falls, one can better understand the pathologic structures that are responsible for the stiffness, and design appropriate treatment.