**Rehabilitation Principals for Interventional Orthopedics and Orthobiologics**

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**Background**

Key Points

* The effects of prehabilitation in preparation for and rehabilitation after interventional orthopedic and orthobiologic procedures have received little attention in the literature.
* Exercise is a cornerstone of management prior to and following interventional orthopedic and orthobiologic procedures.

**Pre-Procedure Considerations**

Prehabilitation is the practice of training and enhancing the patient’s functional capacity to prepare for a major procedure. Postprocedural inactivity can lead to a decline in function, and the objective of prehabilitation is to help the patient withstand the stress of this inactivity.1 Prehabilitation has received little attention in the interventional orthopedic and orthobiologic literature.

In total hip and knee arthroplasty, preoperative functional status is a significant predictor of postoperative function.2-5 In fact, one study found that baseline pain and function was the single best predictor of pain and function at 6-months after a total hip or knee replacement.3 Patients may not report a perceived benefit from prehabilitation programs,2,4 but studies have shown functional benefit.2 Even a 3-week preoperative strengthening program can increase lower extremity muscle strength prior to surgery, and improve the immediate post-operative course.2 The significant strength gains achieved in the 3 to 6-week pre-operative period suggests that the benefit of these short prehabilitation strength training programs is in increased neuromuscular coordination and not necessarily strength.6,7 While there may be short-term benefits to prehabilitation after arthroplasty, the longer-term benefits are unclear.8

The literature on prehabilitation is more limited in other areas of orthopedics, but prehabilitation has been studied in anterior cruciate ligament (ACL) tears. Preoperative quadriceps strength is a significant predictor of knee function 2-years after ACL reconstruction,9 and enhancing quadriceps strength and function preoperatively has been shown to improve surgical outcomes.10-13 In spinal surgery, prehabilitation showed no statistically significant difference in pain or disability, but patients reported feeling more prepared for surgery.14 These principles have not been studied in orthobiologics or interventional orthopedic procedures, but it is reasonable to believe that preconditioning could also have a beneficial effect after the varied procedures outlined in this text.

In addition to rehabilitation, nutrition and the metabolic response to injury has been important to recovery. In the trauma literature,15-17 poor nutrition can contribute to poor wound healing.18 Proper nutrition is an essential parameter in musculoskeletal health including the prevention and treatment of diseases.19 This is likely underrecognized in elderly patients, and older adults may not have the same physiologic reserves of younger adults.20 Given the prolonged wound healing process, nutrition should be considered as part of the presurgical or pre-procedure assessment. Diagnostic markers of malnutrition remain elusive.21 Attention should be focused on basic nutrition and a healthy diet, including adequate caloric and protein intake to support the increased energy demands of collagen synthesis, angiogenesis, fibroblast proliferation, tissue remodeling and wound contraction during the healing process.22 Protein deficiency has been associated with impaired fibroblast proliferation and collagen synthesis.21 Adequate hydration can assist in promoting tissue perfusion, oxygenation, waste removal,23 and macro- and micronutrients may assist in enhancing the healing process.21 In the wound literature, there is evidence for supplementing vitamins A and C, and – if there is a deficiency – supplementing arginine, glutamine and zinc.21 However, literature is limited in orthobiologics.

Malnutrition can take different forms. In addition to specific nutrient deficits, malnutrition can include inadequate intake and overconsumption.24 There is evidence that obesity and associated disorders can effect stem cell function. Obesity and diabetes have been associated with abnormal cytokine signaling, impaired tissue repair and delayed wound closure.25 In rodent models of type 2 diabetes, endogenous mesenchymal stem cells (MSCs) were less effective at mobilizing to a site of injury than those in the nondiabetic controls,26 and high levels of glucose showed reduced mesenchymal progenitor cell growth. 27 High-glucose concentrations have been shown to reduce the osteogenic and chondrogenic potential of adipose-derived mesenchymal stem cells (ADSC),28 and ADSC had a reduced differentiation potential and had a lower capacity for spontaneous or therapeutic repair in patients who were obese and had metabolic syndrome.29 In contrast, caloric restriction is known to reduce inflammation and has been shown to increase proliferation of MSCs in mouse models;30,31 moreover, the restriction of glucose improved the self-renewal and antisenescence abilities of the MSCs.32 Although the role of obesity or calorie restriction on clinical outcomes in orthobiologics is unclear, the literature suggests that there is a fine line between the beneficial effects of caloric restriction and the consequences of malnourishment.

Education has also been shown to positively impact outcomes of total knee and hip arthroplasty.33,34 Being able to counsel patients regarding the expected post-procedural course can help them prepare for the procedure and post-procedure course, which in theory could impact the outcome after an orthobiologic procedure as well. The following section outlines the expected post-procedure course for many orthobiologic procedures.

**Phases of Rehabilitation**

The healing potential following orthobiologic injections will vary depending on the tissue type (e.g. tendon, ligament, muscle, bone), underlying pathology (e.g. tendinopathy vs tear) and anatomic location (e.g. Achilles vs rotator cuff). In general, healing of tendon, ligament, muscle, and bone injuries follow the normal wound healing cascade. The wound-healing cascade is a complex series of events and is typically divided into three phases: (1) inflammatory phase; (2) proliferative phase; and (3) maturation or remodeling phase. Some authors may describe a fourth phase, the hemostasis phase, characterized by vasoconstriction and formation of a blood clot immediately following an injury (Figure 1: Phases of Wound Healing).



Figure 1. Phases of wound healing

Kumar V, Abbas A, Fausto N. Tissue renewal and repair: regeneration, healing, and fibrosis. In: Robbins and Cotran Pathologic Basis of Disease (7th Edition). Elsevier Saunders, PA, USA, 87–118 (2005).

There is limited evidence-based literature on the role of rehabilitation following orthobiologic or regenerative procedures.35-37 While rehabilitation is often encouraged for the management of many orthopedic conditions to improve range of motion, strength and functional activities, rehabilitation may offer a more specific role after orthobiologic procedures. The objective of many regenerative procedures is to trigger a healing response and stimulate the body’s own repair mechanism. Animal models of injury and repair are the primary means of understanding the fundamental process of healing in tendon, ligament, and muscle tissue. In one study by Virchenko and Aspenberg, rats with iatrogenically injured Achilles tendons were injected with platelet rich plasma (PRP). Half of the rats had an intra-muscular injection of Botulinum toxin A (Botox) into the calf muscles to unload the tendon. The rats who received the Botox injection had no effect from the PRP injection; in comparison, the rats that were not treated with Botox showed neotendon development indicating a positive response to the PRP injection combined with activity.38 The failure of the rats treated with Botox to develop neotendon suggests that mechanical stimulation is vital in the early phases of tendon regeneration. Ambrosio et al., in a mouse model, demonstrated that stem cell transplantation into injured skeletal muscle proliferated and terminally differentiated toward a myogenic lineage with daily treadmill running, while the transplanted stem cells failed to rapidly divide in the absence of loading.39 Clinical studies have also supported the role of rehabilitation. In a pilot study of PRP for chronic patellar tendinopathy, Kon et al. found that subjects who did not follow the post-procedure stretching and strengthening program had poorer outcomes.40

Further insight is needed into the molecular and cellular response to therapeutic exercises and stress after regenerative therapies. Based on basic science studies showing that the effects of PRP are lost when tendons are unloaded mechanically, one conclusion is that tendon healing may require a combination of biologic and mechanical factors.37 Mechanotransduction is often used to describe the physiologic responses by which cells convert mechanical stimuli into structural adaptation.41-43 Mechanical stimuli or loads on a tendon are sensed by various cell surface receptors, integrins, stretch-activated ion channels and other mechanisms. This triggers cell-cell communication and changes cellular biology within the cell nucleus.44 Eccentric exercises, with slow lengthening of the muscle-tendon unit while under load, have been shown to stimulate a cellular response, including activation and proliferation of satellite stem cells.45 Heavy-slow resistance (HSR) training in which each repetition is performed slowly for >6 seconds for both the eccentric and concentric phases has shown similar results to eccentric strengthening in long-term pain reduction. HSR training demonstrated normalization of tendon fibril morphology.46 There is also interest in low-load resistance training with blood flow restriction (BFR), and recent studies have shown increased muscle protein synthesis and proliferation of myogenic stem cells after BFR training.47,48

Many translational questions exist about how to apply these principles to orthobiologic procedures. The topic of tissue repair and rehabilitation is vast, and this chapter is by no means exhaustive. The goal is to understand the basic tenants of the healing process to help clinicians design a rehabilitation program, which takes into consideration the patient and pathology (e.g. mechanism of injury, tissue injured, severity, age of patient), the treatment (e.g. intra-articular vs intra-osseous or intra-tendinous vs paratendinous), and the different phase of healing.

Rehabilitation recommendations can vary depending on the tissue treated. For example, tendons require loading in an earlier phase of rehabilitation to help support the development of tensile strength. Overall, the literature suggests that mechanical stress on the tendon is needed to optimize outcomes.35,37 The severity of pathology should also be considered. For example, strengthening exercises after platelet rich plasma injection (PRP) may be started later in high-grade partial tendon tears compared to tendinopathy.

Guidance following a procedure must also be tailored to the injectate or treatment. For example, rehabilitation following an injection around a tendon (e.g. a high-volume injection between the Achilles tendon-Kegar fat pad) will have a different expected post-procedural course than an intra-tendinous procedure (e.g. platelet rich plasma injection into the Achilles tendon). Likewise, an intra-articular knee injection of cortisone or ketorolac will have a different course than an orthobiologic injection.

**The Healing Cascade**

*Phase I of Healing – The Inflammatory Phase (0-5 days)*

The inflammatory phase is the initial response to tissue damage and generally occurs in the first one to five days after an injury.49-52 This phase is initially characterized by hemostasis and “walling off” of the injured site, increased vascular permeability, and an influx of inflammatory cells. Platelets are among the first cells to respond to an injury and form the hemostatic plug and secrete chemokines (e.g. epidermal growth factor (EGF), fibronectin, fibrinogen, histamine, platelet-derived growth factor (PDGF), serotonin, and von Willebrand factor). These factors recruit macrophages to the healing site, resulting in scavenging of debris and phagocytosis to debride the wound bed. Fibroblasts also migrate to the wound in this phase, initiating the formation of granulation tissue and the transition into the proliferative phase.49 The specific cellular events will differ depending on the anatomy and physiology of the given tissue.53 During this phase, patients may experience varying degrees of pain, swelling, redness, and limited range of motion.

*Phase II of Healing – The Repair Phase (5 days – 21 days)*

The proliferative (granulation) phase is characterized by “rebuilding” the local tissue, and generally lasts a few weeks after an injury.50-52 Granulation tissue formation, collagen deposition and angiogenesis create an extracellular matrix and network of blood vessels to supply the area. Neovascularization helps supply the wound bed with nutrients.49 Macrophages continue to supply growth factors and fibroblasts differentiate and produce collagen, depositing and remodeling the extracellular matrix.49

Initially, migrating fibroblasts will begin to synthesize collagen around day 5, but by the fourth week, there is a noticeable increase in the intrinsic proliferation of fibroblasts from the endotenon. Initially the collagen fibers are randomly oriented, but as the tissue starts to mature, the collagen fibers are increasingly oriented along the direction of force through the tendon, increasing the tensile strength of the tendon. By week 5, tenocytes become the main cell type. 54

During this phase, patients should feel a decrease in the initial post-injury/procedural pain. Pre-procedural symptoms may persist, slowly improve, or wax and wane.

*Phase III of Healing – The Maturation Phase (21 days – 12 months)*

The maturation or remodeling phase starts three weeks after an injury and lasts up to 12 months or longer with collagen deposition by fibroblasts continuing for this entire period.51 Increased stability is acquired during the remodeling phase and is stimulated by continued use of the tendon. Mechanical stress influences cell signaling, and contributes to collagen matrix remodeling and increases the tensile strength of the tissue.55 As the maturation process continues, the collagen fibers continue to be reabsorbed and synthesized along the direction of force and cross linking of the collagen fibrils occurs, increasing the tendon’s tensile strength. Moreover, there is increased deposition of type I collagen in preference to type III collagen.54,56 The tensile strength is estimated to reach its maximum strength at 3 months,57,58 but never completely regains its preinjury strength.59 During this period, patients usually feel consistent improvement in symptoms, though their condition will still often wax and wane as tissue transitions from granulation tissue to scar formation.

**Tissue Specific Considerations**

*Tendons and Ligaments*

Healing of tendons and ligaments follow the three phases of wound healing detailed above. In the initial phase, blood clot and granulation tissue fill the gap between the tendon and ligament fibers. In tendons, fibroblasts and tenocytes in the epitenon and paratenon are recruited and proliferate, bridging the injured gap and forming a stable scar. In the early stages, the matrix is composed of increased amounts of type III collagen.60 After 10 weeks, a higher proportion of type I collagen is synthesized and type III collagen decreases, forming scar-like tendon tissue, a process that will continue for years.61,62

*Muscles*

Muscle strains that result in a rupture of the myofibers go through three phases of healing. Satellite cells begin to proliferate and form new myoblasts, which fuse into myotubes within a couple of days.63 Similar to tendons and ligaments, fibroblasts produce collagen and form scar tissue to bridge the gap between muscle fibers.64

*Cartilage*

Articular cartilage has poor intrinsic healing capacity, and generally does not heal or only partial heals under certain conditions.65 In most cases, surgical or biologic interventions may induce a repair response, and treatments include debridement, microfracture and autologous tissue transplantation (e.g. autologous chondrocytes or mesenchymal stem cells). Little is known about the histologic effects of cartilage healing after these procedures. *In vitro* studies, have demonstrated thatmechanical stress can stimulate differentiation of MSCs into chondrocytes, extracellular matrix synthesis, and cytokine secretion,66 while excessive stress may cause cell death and matrix degeneration.67

*Bone*

Injured bone (e.g. fracture or necrotic bone) is resorbed and replaced by new bone following the three phases of healing. Macrophages and osteoclasts remove injured calcified bone, and osteoblasts fill the fracture gap forming granulation tissue. Delayed bone formation and nonunion have been attributed to variations in the local inflammatory environment, as well as the recruitment of muscle-derived stromal cells and osteogenesis early in the healing process.68 Bone repair follows two phases of healing: (1) the initial cartilaginous soft callus followed by (2) remodeling and formation of a bony hard callus. Initially, the soft callus is formed when adjacent soft tissue and periosteum bridge the fracture site stabilizing the fracture.69 Osteons travel along the cortical bone (by the Haversian system); they bridge the fracture gap,70,71 and osteoblasts synthesize woven bone resulting in a hard callus.68,72,73 This irregular woven bone callus remodels over months to years through continuous osteoclast resorption, while osteoblasts replace the matrix with lamellar bone to bring the bone back to its original shape, size and strength/stability.74

**Overview of Rehabilitation Guidelines Per Phase of Healing**

The objective of many traditional orthopedic injections, such as corticosteroids or ketorolac injections, is to address inflammation. The objective of orthobiologic procedures in interventional orthopedics is different, and the aim is to heal the tissue. This is often achieved by stimulating a healing response, through delivery of growth factors to the target tissue (e.g. PRP, MSC’s, etc) or controlled microtrauma to convert a chronic injury into an acute injury with healing potential (e.g. percutaneous tenotomy or vacuum debridement).

The literature on rehabilitation after orthobiologic or interventional orthopedic procedures is limited, and many of the rehabilitation protocols proposed in the literature are an attempt at translating these would-be would healing principals into clinical practice.44,75-78 There is some variability in the duration and overlap of the phases of wound healing in the literature,49-52 and progression through the rehabilitation process should be individualized and informed by clinical progress through the program. The rehabilitation process should reflect the type of tissue undergoing recovery (e.g. tendon, bone, ligament, muscle), the severity of the underlying pathology, the patient’s pre-procedure fitness level, the patient’s physical abilities, and any existing comorbidities.

***Rehabilitation in the Inflammatory Phase of Healing (0 - 5 days)***

The goal of rehabilitation in the inflammatory phase is typically one of protection, and care usually focuses on pain management and managing post-procedural swelling. Management may vary depending on the procedure, but is typically achieved with immobilization or bracing, ice, elevation, medications, and gentle range of motion exercises to activate the vasomotor pump.75

*Immobilization/Bracing*

In most cases, protected weight bearing is recommended for pain control in the first 1-3 days. Most randomized controlled or prospective studies on orthobiologic procedures have prescribe a period of absolute or relative rest during the acute inflammatory phase, although no prospective studies have specifically studied these rehabilitation protocols.35 ­Immobilization is sometimes recommended due to the concern that the orthobiologic may disperse to other locations with movement. However, In a cadaveric model, Achilles tendons that were injected with blue dye to simulate a PRP injection were manipulated through 100 cycles of ankle dorsiflexion and plantar flexion, and there was no significant difference in the spread of the dye compared to control specimens that were kept in a prone resting position for 15 minutes after the injection.79 This suggests that early motion does not increase the spread or clearance of PRP from the target site and thus we recommend early mobilization of tendons following these procedures.

Limiting joint motion can help reduce pain in the associated area, and weight-bearing restriction or bracing can be used for this objective. Avoiding any pain-provoking activities is a common recommendation, but recommendations have varied across the literature from avoiding all physical activity to limiting only repetitive movements. Crutches, a controlled ankle motion (CAM) walking boot, or unloader braces can be used to limit motion or decrease stress on a joint, bone or tendon/ligament after treatment. Any period of immobilization should be extremely limited as discussed previously. In most randomized controlled or prospective studies that detailed post-procedure protocols, most patients were instructed to restrict weight-bearing or immobilize the joint for 3 days to 2 weeks.35 Prolonged immobilization has been associated with joint contractures and functional impairment in the surgical literature, and early gentle active range of motion is considered safe.75

*Pain Management*

Post-procedure pain can vary among patients and procedures. For example, intra-articular injections are often less painful than intra-tendinous or intra-osseous injections. Periprocedural nerve blocks can help with acute post-procedural pain management when possible. Longer acting anesthetics, such as ropivacaine, can extend the efficacy of these blocks. Alternative approaches to pain management include cryotherapy and analgesic medications other than nonsteroidal anti-inflammatory drugs (NSAIDs).

There are different methods of cryotherapy, including crushed ice, ice bags, chemical or gel packs, and circulating commercially available cryotherapy devices that provide continuous circulation of ice water. However, there is evidence that some of the methods are more effective than others.80-82 There is some debate in the literature on limiting cryotherapy in the acute phase after an orthobiologic procedure.35,83 Cryotherapy has been shown to be effective for managing pain in certain situations,84 and is often accepted as an integral part of the treatment of acute soft tissue injuries despite a lack of robust evidence.85 In one review of regenerative procedures for tendinopathy, 20% of prospective and randomized controlled studies prescribed cryotherapy for pain management.35 The theoretical concern is that cryotherapy may reduce blood flow important for healing. However, the literature is limited and the effect of cryotherapy depends on a number of factors, including the temperature of the cooling device, the depth of the subcutaneous tissue, and the frequency and duration of treatment.81,86-88 Studies have shown that cryotherapy can temporarily decrease microcirculatory perfusion when measured at a depth of 2mm to 8mm,89,90 but vary in whether this decrease in blood flow persists following active cooling.90,91 There is limited literature that the superficial effects of cooling impact the perfusion of deeper structures, and skin temperature has been shown to be a poor predictor of perfusion to deeper structures.92 In one study evaluating the effect of cryotherapy on the microcirculation of the midportion Achilles tendon, the authors showed reduced blood flow within the first minute of cryotherapy and return of capillary blood flow during recovery.91 Another theoretical concern is that cryotherapy may decrease platelet activation, but *in vitro* studies have shown temperature did not affect platelet adhesion when tested at 0 and 37 degrees Celsius.93

Limiting NSAIDs before and after an orthobiologic procedure is widely accepted across the literature. NSAIDs have been shown to inhibit platelet function and reduce the release of growth factors.94-96 In a study using a rat animal model of a surgically repaired rotator cuff tendon, even initiating NSAIDs in the proliferative stage of healing decreased the biomechanical strength of the repaired tendon.97 When comparing different classes of oral NSAIDs and their influence on clinical outcomes post-PRP for knee osteoarthritis, patients taking a non-selective cyclooxygenase (COX) inhibitor had lower functional scores and higher pain scores at 4- and 8-weeks post-PRP compared to patients taking a selective COX inhibitor.98

Non-NSAID pain medications and prescription narcotics are often prescribed for the first 72 hours post-procedure. Over the counter acetaminophen is often acceptable for pain control after most procedures, and does not demonstrate any anti-inflammatory activity.96 However, acetaminophen has been shown to inhibit platelet aggregation. 99 Although the impact of impaired platelet aggregation on clinical outcomes post-PRP is unclear, some have recommended that clinicians consider suspending acetaminophen prior to a PRP injection.99 Narcotics can be used for breakthrough pain following opioid risk mitigation strategies, including patient education about opioids, clear instructions about when to use opioids (e.g. for moderate or severe pain only), and prescribing short-acting opioids at the lowest dose necessary. In geriatric patients, doses should be decreased by at least 50% if there is concern for cognitive impairment, risk of falls, respiratory dysfunction or renal insuficiency.100

***Rehabilitation in the Proliferative Phase of Healing (5 days – 6 weeks)***

The objective of rehabilitation in the proliferative phase is to gradually increase activity. There is no consensus on the optimal timing of a stretching or strengthening program, and most published study protocols do not specify the type of strengthening recommended.35 Rehabilitation in this phase is progressive, and is typically governed by the patient’s tolerance. A good guideline is to limit any activity that increases the patient’s pain to greater than a 3/10 during or after that activity.

Intraosseous, intra-articular, intratendinous, intraligamentous and axial injections may all require different periods of rest and activity modification.101,102 The literature on tendon healing clearly suggests early controlled loading influences the early phases of tendon healing after orthobiologic injection or microtrauma (i.e. needle tenotomy),38,103 and that the appropriate mechanical loading induces differentiation of tendon stem cells (TSCs) into tenocytes.104 However, excessive loading can induce differentiation of TSCs into adipocytes, chondrocytes and osteocytes.104 It is possible in theory that the concerns about heterotopic ossification after PRP105 may stem from the failure of rehabilitation rather than the orthobiologic injection.

*Stretching*

In the literature, the timing of a stretching program has ranged from 24 hours to 1 week post-procedure.35 Studies have been performed to determine if dynamic stretching (DS), static stretching (SS), or proprioceptive neuromuscular facilitation (PNF) were superior in aiding recovery; however, there is no consensus on the best form of stretching in the early phases of healing. The literature on DS, SS or PNF in sport shows a small to moderate effect on performance and a similar improvement in range of motion (ROM) with all the stretching approaches. There is no overall effect on injuries with SS and PNF, but no data is available for DS on injury prevention.106 For tendinopathies, specifically, it is inferred that static stretching is the “safest” form of stretching due to its slow and steady nature.75,106

*Strengthening*

Strengthening during the proliferative phase has also been evaluated after regenerative procedures in tendinopathy.40 This is typically started at 2 weeks in most published post-procedure protocols,44,75-78 but timing varies across the literature.35 For joint injections, the strengthening program likely can be started sooner than with tendons or ligaments. Most recommendations are to have a progressive strengthening program. This is based on the progression of healing in tendons, where initially newly synthesized collagen fibers are oriented randomly, but as the proliferative phase progresses, the collagen fibers are increasingly oriented along the direction of force, thus increasing the tensile strength of the tendon.54

The safest type of contraction in this phase of healing may be isometric since joint motion is limited. Isometric contractions can decrease blood flow to the active tissues, but the effect is likely temporal.75 Isometric exercises have also been shown to be effective for short-term pain relief, and the authors will typically start with isometric strengthening exercises after a regenerative procedure.107-109

Eccentric contraction or heavy slow resistance training may be the most beneficial type of exercise for long-term pain reduction and functional improvement in tendinopathy, but it has not specifically been studied after regenerative injections.110 Limited literature guides the ideal timing to initiate eccentric strengthening.111-113 There is some concern that if eccentric strengthening is started too soon, it may have a hypovascular effect attenuating the healing cascade,83 and it has been suggested eccentric exercises should be reserved for the late proliferative or remodeling phase.75 There is also limited literature on open-chain versus closed-chain exercises after regenerative procedures.35,75

***Rehabilitation in the Remodeling Phase of Healing (6 weeks – 12 months)***

The goal of rehabilitation in the remodeling, or maturation, phase is the safe return to higher level activities and sport. At this stage, patients should have completed the early rehabilitation protocols and have full range of motion across the joint. Eccentric strengthening should be started in this phase, if not initiated earlier during the late proliferative stage, and proprioceptive exercises should be added to the rehabilitation program.75

The literature is limited to guide return to play decisions, and timing should be individualized to the athlete. Initially, there should be a focus on the reintroduction of functional activities specific to the athlete’s sport. Patients with lower extremity procedures can start jogging and progress activity as tolerated. Sport specific movements and activities should be included in a controlled setting before progressing to a practice or game/competition setting.

**Conclusion**

Post-procedure recommendations are based on the underlying physiology of the healing cascade and the relative timeframes at each stage of healing. Suggested rehabilitations protocols are general guidelines (Table 1). Ultimately, rehabilitation protocols will have to be individualized to best benefit each particular patient in terms of their specific injury. Tables 2-10 provide examples of post-procedure rehabilitation protocols for ten of the most common conditions that are treated with tenotomy or orthobiologic injections (Achilles tendinopathy, plantar fasciitis, patellar tendinopathy, quadriceps tendinopathy, hamstring tendinopathy, gluteal tendinopathy, lower extremity osteoarthritis, elbow tendinopathy, shoulder tendinopathy, and shoulder osteoarthritis).

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