Rehabilitation of sports injuries

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Introduction

Rehabilitation in a sports-medicine environment is considerably different than in most other settings. The nature of sport necessitates an aggressive approach, as the primary goal is to return the athlete to the intense activity of training and competition as soon and as is safely possible. Consequently, the sports therapist who is supervising a rehabilitation programme usually performs a balancing act between not pushing the athlete hard or fast enough and being overly aggressive. A mistake in judgement in either direction can hinder the athlete's return to activity (BUSCHBACHER & BRADDOM, 1994; PRENTICE, 1999). In addition to the expertise of the therapist, there is a need for coaches and other non-medical personnel supporting an athlete to have a certain level of understanding in this area in order to ensure the rehabilitation process is as effective and efficient as possible.

The purpose of this paper is to provide an overview of the main aspects and discussion points in this field as a general guide and a starting point for further study. The points covered are:

- Key features of sports injuries,
- The healing process,
- Specific characteristics of damage and repair of different tissues,
- Aims and objectives of rehabilitation,
- The role of mobility during the healing process,
- The phases of rehabilitation in sport injuries,
- Training of biomotor abilities within the rehabilitation process,
- Using medications and other modalities to maximise recovery from injury.

Key Features of Sports Injuries

According to PETERSON & RENSTRÖM (2001), sports injuries can be divided into a) traumatic injuries, caused by large forces (macrotrauma), and b) overuse syndromes, caused by repetitive microtrauma. In athletics, most injuries fall into the second category.

Overuse syndromes are generally caused by repetitive overloading, resulting in microscopic injuries to the musculoskeletal system. Tissues can withstand great loads but there is a critical limit to this capacity, which varies greatly between individuals and according to the frequency of load. Tissues may be made more susceptible to injury by a) intrinsic factors e. g. leg malalignement or muscle imbalance, and b) extrinsic factors e. g. training errors, faulty technique, incorrect equipment and surfaces, and poor conditions. Overuse injuries can result from various combinations of frequency and loading, such as:

- normal load at high frequency/many repetitions,
- heavy load at normal frequency,
- heavy load at high frequency.

Overuse injuries are often difficult to diagnose and treat. In most cases, the underlying problem is an inflammatory process.

Inflammation

In the context of sport, inflammation is the body's response to tissue injury caused by repeated load or overload. Very early after injury, vascular flow is diminished due to vasoconstriction. This is usually fleeting and followed by vasodilation, which increases the amount of blood in the injured area, resulting in local warmth and redness. Some degree of transudation may occur, but the majority of fluid transudating from the intravascular compartment to the extravascular space occurs from increased vascular permeability, and thus the clinical sign of swelling is revealed. As these processes occur, leukocytes, which escape from the vascular space, invade the injured area. They begin to release enzymes that degrade cellular material and debris. Blood plasma entering the area brings other chemical mediators of inflammation (SKERKER & SCHULZ, 1995). Symptoms typical of inflammation can be summarised as follows:

- swelling caused by fluid accumulation,
- redness and local rise of temperature caused by increased blood flow,
- tenderness on touching the affected area,
- impaired function of the affected part due to swelling and tenderness.

Inflammation often begins insidiously, and initially pain and stiffness may decrease or even disappear after warm-up. Usually, however, the pain returns and intensifies during continued activity. Unless a rest break is taken, there is a great danger of entering the 'pain cycle' where continued activity leads to further injury, inflammation and pain. Unless the cycle is interrupted, chronic pain, which can be extremely difficult to treat, will result.

The most important step in the management of inflammation is the removal or reversal of its cause. Next in importance is the reduction of swelling so as to relieve pain, improve mobility, and encourage healing.

Pain

The sensation of pain originates in free nerve endings, which end blindly between the tissue cells. These pain receptors are especially numerous in the skin. The most common types of pain encountered in sports injuries are acute pain, and the chronic, dull ache experienced following activity or during the night due to chronic inflammatory problems.

Pain can often be effectively treated with medication, but it will not go away completely until its cause has been removed. It should be interpreted as a warning sign of tissue injury and should lead to modification of activity or resting the injured tissue

The Healing Process

Inflammation stimulates healing. The inflammatory response is in fact the first phase of the healing process, followed by the fibroblastic-repair phase and the maturation-remodelling phase (PRENTICE, 1999).

The inflammatory response phase

The inflammatory response is critical to the entire healing process. If it does not accomplish what it is supposed to or if it does not subside, normal healing cannot take place (PRENTICE, 1999). It is initiated by an altered metabolism and the liberation of materials as a result of the cellular injury.

In this process, the deliverance of leukocytes and other phagocytic cells and exudates to the injured tissue is generally protective, tending to localise or dispose of the injury by-products (e. g. blood and damaged cells) through phagocytosis, thus setting the stage for repair. Local vascular effects, disturbances of fluid exchange, and migration of leukocytes from the blood to the tissues occur. As a result of a combination of these factors, the injured area becomes walled off during the inflammatory stage of healing. The leukocytes phagocytise most of the foreign debris toward the end of the inflammatory phase, setting the stage for the fibroblastic phase (SKERKER & SCHULZ, 1995).

Normally, the inflammatory response lasts for approximately 2-4 days after the initial injury. However, chronic inflammation some-

times occurs. This is the case if the acute inflammatory response does not eliminate the injuring agent and restore tissue to its normal physiological state. Chronic inflammation involves the replacement of leukocytes with macrophages, lymphocytes, and plasma cells. These cells accumulate in a highly vascularised and innervated loose connective tissue (LEADBETTER, 1990). The specific mechanisms that convert an acute inflammatory response to a chronic inflammatory response seem to be associated with situations that involve overuse or overload with cumulative microtrauma to a particular structure (FANTONE, 1990; LEADBETTER, 1990).

While inflammation is a part of the body's natural defence system against injury and disease, chronic inflammation, on the other hand, is a disease. The system has gotten hung up, as it were, and instead of protecting the organism it starts to slowly kill it. Chronic inflammation, which appears to be resistant to both physical and pharmacological treatments (PRENTICE, 2007), is the main contributing factor to all chronic degenerative diseases.

The fibroblastic repair phase

During the fibroblastic repair phase of healing, proliferative and regenerative activity leading to scar formation and repair of the injured tissue follows the vascular and exudative phenomena of inflammation. The period of scar formation (fibroplasia) begins within the first few hours after injury and can last as long as four to six weeks. During this period, many of the signs and symptoms associated with the inflammatory response subside. As scar formation progresses, complaints of tenderness or pain gradually disappear. During this phase, growth of endothelial capillary buds into the wound is stimulated by lack of oxygen, after which the wound is capable of healing aerobically. Along with increased oxygen delivery there is an increase in blood flow, which delivers nutrients essential for tissue regeneration in the area.

A delicate connective tissue, the so-called granulation tissue, is formed. It appears as a reddish granular mass of fibroblasts, collagen and capillaries that fills in the gaps during the healing process. As the capillaries continue to grow into the area, fibroblasts accumulate at the wound site, arranging themselves parallel to the capillaries. Fibroblastic cells begin to synthesise an extracellular matrix that contains protein fibres of collagen and elastin. On about the sixth or seventh day, fibroblasts also begin producing collagen fibres that are deposited in a random fashion throughout the forming scar. As the collagen continues to proliferate, the tensile strength of the wound rapidly increases in proportion to the rate of collagen synthesis. With the increase of tensile strength, the number of fibroblasts diminishes, signalling the beginning of the maturation phase (PRENTICE, 1999).

Maturation-remodelling phase

The maturation-remodelling phase of healing is a long-term process that features a realignment or remodelling of the collagen fibres that make up scar tissue according to the tensile forces to which the scar is subjected. Ongoing breakdown and synthesis of collagen occur with a steady increase in the tensile strength of the scar matrix. With increased stress and strain, the collagen fibres realign in a position of maximum efficiency parallel to the lines of tension. The tissue gradually assumes normal appearance and function. However, a scar is rarely as strong as the normal tissue. Although a firm, strong, contracted, nonvascular scar will exist by the end of approximately three weeks, the maturation-remodelling phase of healing might require several years to be totally complete (PRENTICE, 1999).

Specific Characteristics of Damage and Repair of Different Tissues

Muscle fibre

Clinically, muscle injuries are called strains. As with other tissues, muscle has the capacity to heal by regeneration (LIEBER, 1992). Healing of muscle fibres takes place in the following steps:

1. Damaged cells leak endogenous proteases that auto-digest damaged cells.

2. Inflammatory mediator chemicals soon attract or induce macrophages that infiltrate the area of damage and clean up cellular debris.

3. Satellite cells begin to proliferate at the damaged areas to align themselves along the basal lamina and fuse into myotubes.

4. The myotubes mature and differentiate into muscle cells, generating myofibrillar proteins.

5. The proteins assemble into disorganised myofibres, which begin to align in the direction of the mature muscle.

6. Maturity brings further organisation, recovery of contractile abilities, and increasing tensile strength.

Lengthening (i. e., eccentric) muscle contractions are damaging to muscle fibres, which in part explains why eccentric contractions produce more post-exercise muscle soreness than concentric contractions.

Stretching may be helpful to prevent muscle injuries because it leads to increased length of the muscle-tendon unit and therefore contributes to greater flexibility. Repetitive stretching results in a reduction in the peak load or tensile stress placed on a muscle-tendon unit, which may lessen the chances of injury (TAYLOR et al., 1990).

Ligament

Clinically, ligament injuries are called sprains. The healing process can be described as follows: One week after mild injury, edema and fibroblastic proliferation occur. The surrounding synovium is also inflamed, and the inflammatory process continues into the second and third weeks. During this period, subcutaneous haemorrhage begins to resolve and fibroblastic proliferation continues. By six weeks, the cellular response has subsided, and the healing process with fibrous tissue has begun. More severe sprains follow the same healing progression, but the repair process may take eight to ten weeks, depending on the maturity and strength of the tissue. Generally, by six to eight weeks individuals can return to athletic activities (SKERKER & SCHULZ, 1995).

Many investigators believe that joint range of motion exercises result in stronger ligament healing, although it is agreed that early immobilisation to protect the area during the initial healing stage may be important (GOLSSTEIN & BARMADA, 1984; O'DONOGHUE et al., 1971; VAILAS et al., 1981).

Tendon

Tendon healing occurs through a combination of the following intrinsic and extrinsic mechanisms:

1. Inflammation occurs one to three days after injury or surgery and is marked by the appearance of cells originating from the extrinsic peritenon (connective-tissue sheath of a tendon) and intrinsic epitenon (outer or parietal layers of a tendon) and endotenon (fine connective tissue between the strands in a tendon).

2. Collagen synthesis begins within the first week following repair, with the fibroblasts becoming the predominant cells.

3. During days 5-15, the athlete is at increased risk for tendon re-rupture and additional injury due to the decreased tensile strength, which during the first five days was provided by the now weakening suture used for the repair.

4 Tendon strength begins to increase after 15 days, as the network of collagen fibres becomes more organised.

5. Providing controlled stress to the healing tendon will further increase the tensile strength of the tendon.

6. At eight weeks the tendon is generally strong enough so that the athlete can begin resistive exercise. A return to athletic activities is normally possible 12 weeks after tendon repair (SKERKER & SCHULZ, 1995).

Bone

A fractured bone initiates a sequence of inflammation, repair, and remodelling that is similar to that of other tissue:

1. As the inflammatory phase wanes, the reparative process begins angiogenesis, bringing in new capillary buds and a new blood supply to the region.

2. With the new blood supply, pluripotential mesenchymal cells develop that form fibrous tissue, cartilage, and eventually new bone, which slowly mineralises.

3. Clinically, fracture union occurs with the fracture becoming stabilised by callus.

4. The fracture site becomes pain-free and radiographs show bone crossing the fracture site. At this stage (8-12 weeks), the fracture site is still weaker than surrounding normal bone.

5. During the final stages of healing, the fracture site continues to gain strength.

6. Remodelling of the bone occurs with the replacement of woven bone (callus) with lamellar bone and absorption of unneeded callus. This metabolic stage continues for years after clinical fracture union (SKERKER & SCHULZ, 1995).

Aims and Objectives of Rehabilitation

The aim of rehabilitation is to limit the amount of scarring, and to preserve strength, elasticity, and contractility of the tissue's components. The objective in training muscles, tendons and joints after injury is to:

- regain normal mobility (range of motion) of the joints,
- stretch connective tissue fibres of the tendons and muscles to an optimal length,
- increase muscle strength and endurance,
- increase the strength of muscle and tendon attachments,
- improve coordination and proprioception (PETERSON & RENSTRÖM, 2001).

According to PERRIN & GIECK (1999), the objectives of rehabilitation are to regain range of motion, strength, proprioception, muscular endurance, power, cardiovascular endurance, speed, agility, and sport-specific skills. The likelihood of reinjury will only be reduced if the following criteria for return to activity are adhered to:

- full pain-free range of motion of the injured part,
- pre-injury strength and power in comparison to the uninjured state,
- pre-injury muscular endurance in comparison to the uninjured state,
- pre-injury levels of cardiovascular fitness and endurance,
- pre-injury levels of speed and agility for lower extremity injuries according to the following tests:
  - running full speed straight ahead without a limp
  - 90º cuts to the left and right from full speed without a limp
  - figure of eight running with acceleration and deceleration around pylons without a limp
  - one-leg timed hops for 30 secs equal to the uninjured side
  - long jump and vertical jump equal to the uninjured side,
- pre-injury size of muscular mass in comparison to the uninjured side,
- reabsorption of acute edema,
- ability to perform all exercises free of pain,
- ability to dynamically control joint instability,
- return to pre-injury levels of pain threshold,
- cerebromuscular rehabilitation complete,
- ability to perform tests appropriate to physical demands of the sport,
- presence of a strong desire to return to competition.

The Role of Mobility During the Healing Process

In animal models, controlled mobilisation is superior to immobilisation for revascularisation, muscle regeneration, and reorientation of muscle fibres and tensile properties. However, a brief period of immobilisation of the injured tissue during the inflammatory response phase is recommended and will likely facilitate the process of healing by controlling inflammation, thus reducing clinical symptoms. As healing progresses to the repair phase, controlled activity directed toward return to normal flexibility and strength should be combined with protective support or bracing (KIBLER, 1990). Generally, clinical signs and symptoms disappear at the end of this phase.

As the remodelling phase begins, aggressive active range-of-motion and strengthening exercises should be incorporated to facilitate tissue remodelling and realignment. To a great extent, pain will dictate the rate of progression. Although with initial injury, pain is intense, it tends to decrease and eventually subside altogether as healing progresses. Any exacerbation of pain, swelling, or other clinical symptoms during or after a particular exercise or activity indicates that the load is too great for the level of tissue repair or remodelling. The sports therapist must be aware of the time required for the healing process and realise that being overly aggressive can interfere with that progress (PRENTICE, 1999).

The Phases of Rehabilitation in Sport Injuries

The rehabilitation process following any type of soft tissue or bone injury can be divided into a series of progressive phases, which should be seen as a continuum. Different aspects of rehabilitation are emphasised in each phase. As the athlete progresses through the phases, the injured structures are simultaneously moving through the repair process. The main phases of sport injury rehabilitation are:

- acute injury,
- initial rehabilitation,
- progressive rehabilitation,
- integrated functions,
- return to sport.

Acute injury (1 to 4 days)

- Pathologic process: Tissue injury (haematoma, oedema, inflammation, necrosis);
- Functional goals: Preparation of the damaged area for healing, reduction of swelling, and decreasing pain;
- Rehabilitation treatment: Protection + Rest + Ice + Compression + Elevation = PRICE. Rest to the injured area should be complete. Local anaesthetics should not be injected into the acutely painful area because pain offers a component of protection that helps the athlete avoid further injury. Instead oral analgesics and anti-inflammatory medication should be used (SKERKER & SCHULZ, 1995).

Initial rehabilitation

- Pathologic process: Fibroblastic stage (decreasing inflammation, waning oedema, minimal tensile strength, i.e. 0-15%);
- Functional goals: Pain-free recovery of range of motion;
- Rehabilitation treatment: Continuing cold application, active or active-assisted range of motion exercises, limited short-arc resistance exercises, gentle isometric strengthening exercises, light aerobic activities.

Progressive rehabilitation

- Pathologic process: Early tissue repair (primitive collagen and early tissue maturation, moderate tensile strength, i. e. 15-50%);
- Functional goals: Improved range of motion, increased strength, limited activity skills, increased range of motion to the full arc;
- Rehabilitation treatment: Passive/active range of motion stretching, progressive resistance exercise with isotonic and/or isokinetic exercises, increased aerobic activities as tolerated by the athlete. During this stage, the athlete should switch

from cold to heat application if the following criteria have been attained:
- Oedema has stabilised
- There is almost a full range of motion
- The range of motion is pain-free
- There is no hyperaemia at the injury site
- Progress with ice has plateaued (PERRIN & GIECK, 1999).

Integrated functions

- Pathologic process: Integrated functions (mature collagen, tissue characteristics now evident, better tensile strength, i.e. 50-90%);
- Functional goals: Increased specific athletic skills, increased strength, enhancement of flexibility to pre-injury conditions, improvement of exercise tolerance;
- Rehabilitation treatment: Advanced progressive resistance exercise, flexibility exercises, coordination training, proprioceptive training.

Return to sport

- Pathologic process: Tissue remodelling (maturation of tissue characteristics, increased tensile strength, i.e. 90-99%);
- Functional goals: Maximised skills, simulated participation, prevention of injury;
- Rehabilitation treatment: Maintenance of strength and flexibility, advanced coordination activities, protection of previously injured area from reinjury.

Training of Biomotor Abilities Within the Rehabilitation Process

Exercise is the ultimate therapeutic modality in sports rehabilitation. Although cold, heat, and other treatments are useful in facilitating pain-free exercise, they alone are inadequate for the successful rehabilitation of an athlete. The application and correct timing of rehabilitation exercises are essential to the rehabilitation process.

If the injury is of mild or moderate severity, exercise should begin the day following injury. In this manner loss of strength can be retarded and normal motion can be regained as soon as possible. Exercise should progress in a pain-free manner from the onset of injury to complete recovery. Although some exercises may produce some discomfort, a key element is that the athlete should experience no undue pain or swelling the day following the exercise routine. The application of ice may be useful in preventing this residual discomfort following rehabilitative exercises.

In all rehabilitation programmes the athlete should aim at exercising just below the point of pain. The philosophy of no pain, no gain is extremely inappropriate in acute rehabilitation because there is no gain with pain in acute rehabilitation (PERRIN & GIECK, 1999).

Strength and flexibility

The foundation of a rehabilitation programme begins with the strength and flexibility phase. It is important that strength and flexibility exercises occur simultaneously because residual deficits in flexibility can predispose athletes to reinjury (WORRELL et. al., 1991). Full range of motion should not be a prerequisite for strengthening exercises, as strength can actually help improve flexibility. Strength of muscular and connective tissue is the first line of defence in providing joint stability. For example, hamstring strength can increase knee flexion in the injured knee.

A certain amount of neural learning takes place in the early strength-training phase of rehabilitation. The athlete who can lift 25kg with the quadriceps and the next day after a mild medial collateral ligament sprain can lift only 2kg has not lost his strength. Pain and effusion from soft tissue injury causes a reflex inhibition of strength to prevent further injury by limiting the athlete's functional activity. The return to strength must be relearned. This is accomplished by using pain-free resistive exercise performed in multiple, i. e. three to eight sets of usually ten repetitions per set. In general, an outside stimulus of at least a 70% overload is recommended for strength gains.

First, the athlete's ten-repetition maximum (10 RM) is determined by the maximal amount

that can be lifted through a complete range of motion ten times. Then, 40 repetitions are performed according to the following protocol:

1. 10 reps at $^1/_2$ of 10 RM
2. 10 reps at $^3/_4$ of 10 RM
3. 10 reps at 10 RM
4. 10 reps at 10 RM + 2.5kg

Initially, the athlete will not be able to perform the last set of ten repetitions. When the athlete can perform the fourth set, the 40 repetitions are recalculated with the weight of the fourth set now becoming the third set. As strength progresses, exercises are performed three times per week with a muscular endurance routine on alternate days.

Isometric resistance is the exercise of choice early in rehabilitation, especially when joint movement may be limited or contraindicated. Increases in strength resulting from isometric exercise are limited to a 20º range of motion around the position of exercise. Therefore, several isometrics within the available range of motion are indicated.

Initially, the athlete needs to contract only 40-50% of maximal effort for a few seconds to gain strength. As strength returns, however, a more forceful contraction is needed to produce further increases in strength. These maximal contractions should be held for a duration of six seconds. From the isometric exercise, a slow pain-free pattern should be used to develop a good base for which neural learning can occur.

Isotonic exercise with free weights is started as soon as possible to address the deficiencies left by isometric exercise. In regular strength training, repetitions are usually done with maximum weight. In rehabilitation, however, the athlete should go through a full range of motion with submaximal weight to prevent the oedema and soreness that would develop with three sets of maximum lifts. The part to be exercised should be isolated to prevent accessory input from other muscles. Momentum and inertia should be minimised by performing the exercises slowly during the early phases.

At this point, the strengthening programme should incorporate a combination of weight-bearing (closed-chain or functional) and non-weight-bearing (open-chain) exercises. Weight-bearing exercises require muscular cocontraction at multiple joints, which can be advantageous in reducing joint shearing forces and in attaining a more functional progression through rehabilitation. However, weight-bearing exercises also permit compensation of weak muscle groups by stronger muscles (GAUFFIN & TROPP, 1992; KOWALK et al., 1997). For example, the step exercise can be performed by the hip extensors and the gastrocnemius with a weak quadriceps. Non-weight-bearing exercises enable isolation of specific muscle groups and are useful in preventing the avoidance patterns that can result from weight-bearing exercise. It is important to establish a foundation of strength in the extremity before the functional exercise programme is initiated.

The value of a combination of weight-bearing and non-weight-bearing exercises for the lower extremity is not only obvious for the lower extremity. For example, weight-bearing exercise for the shoulder can facilitate development of strength of the muscles acting on the scapula (proximal stabilisers), which is essential to the complete rehabilitation of the upper extremity.

Early in the rehabilitation programme, strength increases are optimised from daily training sessions. However, as the increases of strength begin to peak, the weight-training programme should be performed on an every-other-day basis. A minimum of three sets of repetitions is needed for optimal gains in strength and muscle size. The repetitions should be performed slowly with a two or three count through as range of pain-free motion. A four count should also be used while returning the weight to the starting position.

As eccentric contraction is an essential component of virtually every athletic ability,

the rehabilitation programme should use exercise equipment that includes both a concentric and eccentric component of resistance. Eccentric loading is especially important in overuse injuries (PERRIN & GIECK, 1999).

Joint degeneration can be retarded if the athlete regains strength and flexibility while recovering function. Slow static stretching (WEBRIGHT et. al., 1997), proprioceptive neuromuscular facilitation (PNF), and joint mobilisation techniques (QUILLEN & GIECK, 1988) are methods that may be used to help gain normal flexibility and joint mobility. A corresponding gain in strength provides joint stability so that a normal range of motion may be achieved without producing abnormal motion. An increase in strength will facilitate range of motion and, vice versa, an increase in motion requires an accompanying increase in strength within the newly gained range. The full range of functional motion is necessary for the successful return to competition.

Endurance

Rehabilitation for muscular endurance should not be neglected, although it takes time and can be monotonous. Endurance training may be done during the intermediate phases of rehabilitation in the intervals between strength exercises. Strength training and endurance training can complement each other by incorporating strength training three days a week and endurance training in between. Repetitions of 20-30 are in the endurance range. A 30-minute a day low-load endurance exercise programme will enhance bone, ligament, and connective tissue strength and is recommended to facilitate successful return to function.

An injured athlete should also strive for a better cardiovascular endurance upon return to competition than was present at the time of injury. One of the goals of cardiovascular endurance is to delay fatigue, which can result in reinjury to the athlete. A 35-50% effort is required for endurance training to be effective. A pulse rate above 120 bpm must be attained for at least 20 minutes to achieve a training effect. One of the most effective means of functional sport training is circuit training whereby the athlete trains from a pulse rate of 120 bpm at rest to 180 bpm – according to the individual demands of the sport. However, elite athletes need a 95% heart rate training to increase and maintain their reserves.

As endurance exercise overtraining leads to breakdown of tissue and a decrease of power, athletes should not exceed a 10% increase in endurance training a week (PERRIN & GIECK, 1999).

Power

According to PERRIN & GIECK (1999), power is perhaps the most neglected aspect of rehabilitation training. Often the injured athlete works on strength and flexibility, begins with endurance training, and then with functional training in the absence of power training. However, an athlete must regain power before returning to competition, as this is a significant functional component. If training occurs only with slow repetitions, the athlete will regain strength but will not be ready for the rapid bursts of energy necessary for competition. Therefore, the injured athlete should start high-speed power training as soon as strength is adequately developed. A 50% effort is required to establish the power phase.

Circuit training is often used in power training. A weekly 1-RM is first determined for each exercise. Then, the athlete trains three times a week with three sets of 30 sec repetitions with one-half of this 1-RM, with 20 secs between sets. These sets are done explosively with proper technique. Usually eight to 10 exercises comprise the circuit. Weights are adjusted once per week as determined by another 1-RM lift by the athlete.

Plyometrics, a rapid eccentric contraction followed by a quick concentric motion, is another effective training technique. Lower extremity exercises consist of double and

single leg hops, vertical jumps, bounding, box and depth jumping. Upper extremity exercises can include push-ups with a clap, using a weighted ball against a rebounder, and medicine ball routines.

It is important to plan to introduce power training, and especially plyometric training, toward the terminal phase of rehabilitation. Plyometrics should not begin until the athlete has participated in at least eight to 12 weeks of supervised strength training. As far as the lower extremities are concerned, the athlete should be able to squat twice his or her body weight before starting with plyometrics. Care should be taken not to overload the athlete, which means that a two to three day interval between plyometrics should be observed (PERRIN & GIECK, 1999).

Speed

Speed is one of the final phases of returning to functional activity after lower extremity injury. Speed training occurs with a maximal effort over a short period of time, usually for six to 10 seconds. A 10% improvement in speed may be possible with proper mechanics, technique and training. Increasing stride length, frequency, flexibility, and strength are all contributors. Proper mechanics of running consist of a relaxed and erect posture, high knee activity with straight-ahead arm swing. Work on starts will improve speed more than that in full stride.

Bench steps with weight, plyometrics, and weight control will also assist in the development of speed. Downhill running on a 5-10% grade emphasises stride frequency and length. Instead of 40m sprint, a 20m sprint may be a better predictor of speed (PERRIN & GIECK, 1999).

Agility and coordination

Agility and coordination exercises comprise the final phase of the rehabilitation process. Agility is the ability to change direction without the loss of motion while maintaining proper body control. In essence, this phase necessitates return of timing for the athlete. The athlete learns to make cuts rather than to round corners, which is even for track-and-field athletes essential to regaining pre-injury levels of quickness and confidence in performing sport skills. During this phase, confidence is restored and the athlete can visualise successful return to competition. Running obstacle courses or games such as basketball can aid in the development of agility and coordination (PERRIN & GIECK, 1999).

Aquatic training

Rehabilitation professionals have long used exercise in water with patients who have physically debilitating conditions, as it provides a medium for even those with limited mobility to gently exercise and relax their muscles (SCHIFFER, 1997). A common means is aqua running, which consists of simulated running in the deep end of a pool aided by a flotation device (vest or belt) that maintains the head above water. The form of aqua running follows closely the patterns used on land. Because no contact is made with the bottom of the pool, impact is eliminated. The elimination of weight-bearing makes aqua running an ideal method for rehabilitating or conditioning injured athletes, particularly those with foot, ankle, or knee injuries, for whom running on land is contraindicated (WILDER & BRENNAN, 1994). The qualities that make aqua running a natural method for rehabilitation are less pain, muscle spasm, soft tissue swell, joint swelling, and less overall soreness as compared to land exercise (PERRIN & GIECK, 1999).

Using Medications and Other Modalities to Maximise Recovery from Injury

Medications

In acute pain due to injury, the first line of treatment is often pain control. This is achieved through the use of medications that also control the inflammatory process. Corticosteroids have a powerful anti-inflammatory effect but they may disrupt the initial phases of the healing process and therefore should be avoided in the first few days after injury. However, in the later proliferative phases, cor-

ticosteroids may help to control oedema and decrease a prolonged inflammatory response. Local injection into a tendon sheath, bursa, or joint may be very helpful. However, infiltration of the tendon itself should be avoided because it weakens the tendon, predisposing it to rupture (KENNEDY & BAXTER-WILLIS, 1976).

Nonsteroidal anti-inflammatory drugs (NSAIDs, e.g. aspirin, ibuprofen, diclofenac, etc.) block the production and release of prostaglandins. NSAIDs have been shown to decrease pain and inflammation in the treatment of ankle sprains when given during the first seven days after injury (BAHAMONDE & SAAVEDRA, 1990; MORAN, 1990). Topical anti-inflammatory gels have also been shown to decrease pain and swelling in acute ankle sprains without systemic side effects (DIEBSCHLAG et al., 1990; LEE et al., 1991).

There are conflicting views regarding the question whether NSAIDs, like corticosteroids, delay the healing process (HUBBEL & BUSHBACHER, 1994; HERTEL, 1997). There is, however, no doubt that NSAIDs can be beneficial in the early days after acute injury, during rehabilitation, and in chronic overuse syndromes (SAAL, 1987). Although acetylsalicylate (aspirin) should be avoided during the early phases of injury because of its antiplatelet activity, which can predispose to bleeding, it may be helpful for pain and oedema control in the later stages of healing (KAWAHARA & SPUNT, 1991). Patients using NSAIDs should be carefully monitored for side effects, especially gastrointestinal irritation (HUBBEL & BUSHBACHER, 1994).

Physical agents

In acute musculoskeletal trauma, cryotherapy can be very helpful. Cold decreases blood flow, metabolic activity, muscle tone, oedema and haemorrhage, and provides analgesia (HUBBEL & BUSHBACHER, 1994; KOTTKE & LEHMANN, 1990). Nerve conductivity is decreased, and stretch reflexes are inhibited with deep cooling. Sensory transmissions are blocked, and painful muscle splinting is decreased. Cold therapy is indicated for at least the first 48 hours after injury and should be continued after that time if persistent oedema is noted and skin temperature is increased. Ice should be applied in 12- to 15-minute time blocks followed by a rest period (DUNCOMBE & HOPP, 1991; SAAL, 1987). Prolonged use of cold should be avoided because of the potential for cold-induced vasodilation (hyperaemia) (McMASTER & LIDDLE, 1980).

Heat treatments are contraindicated in the presence of acute inflammation, trauma, haemorrhage, oedema, or ischemia. This means that heat modalities are not appropriate in the early stages of the healing process. In later stages, however, heat can be used to reduce pain sensation, promote blood flow, and decrease muscle tone. Further positive effects of heat application are: increased collagen extensibility, decreased joint stiffness, and general relaxation and sedation. Superficial heating modalities such as hot packs, paraffin bath, fluidotherapy, and whirlpool may be used for treatment of injuries to the extremities after the acute phase (HUBBEL & BUSHBACHER, 1994; KOTTKE & LEHMANN, 1990).

Ultrasound heats deep tissues by sending acoustical vibrations through them, causing vibration of molecules, which in turn produces heat. Ultrasound is relatively ineffective at heating surfaces but is the most effective modality at heating deep tissues and may penetrate to depths of 4-6 cm. It is, therefore, the method of choice for heating deep tissues and joints and is the most common form of diathermy currently used in sports rehabilitation. It may be helpful for treating joint contractures and scar formation, to reduce pain and muscle spasm, and is commonly used in the management of bursitis, tendonitis, and periarticular calcium deposits (SKERKER & SCHULZ, 1995). Ultrasound is also useful in the treatment of subacute tendinitis and bursitis (LEHMANN, 1982). Generally, ultrasound is indicated in the later phases of healing to enhance local circulation and to increase soft-

tissue extensibility (DUNCOMBE & HOPP, 1991; SAAL, 1987).

Phonophoresis

Phonophoresis utilises therapeutic ultrasound in conjunction with a medicine-laden ultrasound coupling media to drive the medication across the skin and into subcutaneous tissues (DAVICK et al., 1988). There is clinical evidence that the use of hydrocortisone enhances the therapeutic benefit of ultrasound.

Patients may experience beneficial effects from the use of phonophoresis for rehabilitating sports injuries such as epicondylitis, tendonitits, and bursitis. Generally, the individual is given a daily treatment of five to 10 minutes for a total of 10-14 treatments (SKERKER & SCHULZ, 1995).

Electrical stimulation

Neuromuscular electrical stimulation (NMES) has been clinically used to activate electrically excitable tissues (muscle and nerve), and is reported to modulate pain, control edema, improve strength, aid tissue repair, and help drive ions across living tissues (LAKE, 1992; SKERKER & SCHULZ, 1995).

Transcutaneous electrical nerve stimulation (TENS) utilises short duration, high-frequency, low-intensity galvanic pulses delivered above the sensory nerve threshold and below the motor nerve threshold. It is thought that pain fibre transmission is blocked peripherally or there is an activation of central inhibiting fibres – similar to the original gate control theory of pain proposed by MELZACK & WALL (1965) – that modulates pain. The clinical utility of TENS has been shown by ERSEK (1976) among others and it is also well accepted for the management of postoperative pain. SKERKER & SCHULZ (1995) recommend its use for pain control when other standard approaches (e. g. analgesics, ice, compression) fail.

NMES can also be used to facilitate strength gain in weakened muscles, along with other standard approaches to building strength (SKERKER & SCHULZ, 1995). One example of this benefit has been demonstrated using NMES after anterior cruciate ligament reconstruction. Patients who had NMES applied to the quadriceps muscle demonstrated significantly greater quadriceps strength gains when compared to controls. These strength gains could be correlated with functional improvements in gait kinematics and mechanics versus the control group (SNYDER-MACKLER et. al., 1991).

Iontophoresis

Iontophoresis, which is the application of electricity to drive ions across living tissues, has been used for more than a half-century. The electricity employed for ion transfer is continuous low-voltage galvanic (direct) current provided by a low-voltage generator or a battery-powered unit. The basis for ion transfer is that like charges repel each other. Therefore, a positively charged ion will travel away from the positively charged electrode (anode) and toward the negatively charged electrode (cathode). While doing so, a therapeutically active ion will enter the dermis and ideally the subcutaneous tissues. However, the majority of active ions are deposited directly beneath the active electrode with further penetration depending on local blood flow for transport to deeper tissue.

Iontophoresis has become a common treatment regimen in sports rehabilitation. Typically, medications such as dexamethasone disodium phosphate, salicylic acid, or lidocaine are delivered to the skin to relieve inflammation and pain.For athletes who have trouble with injections, the application of pain relief and anti-inflammatory medications without injection via phonophoresis or iontophoresis may be useful. In general, however, they should not be recommended as a first line of treatment (SKERKER & SCHULZ, 1995).

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