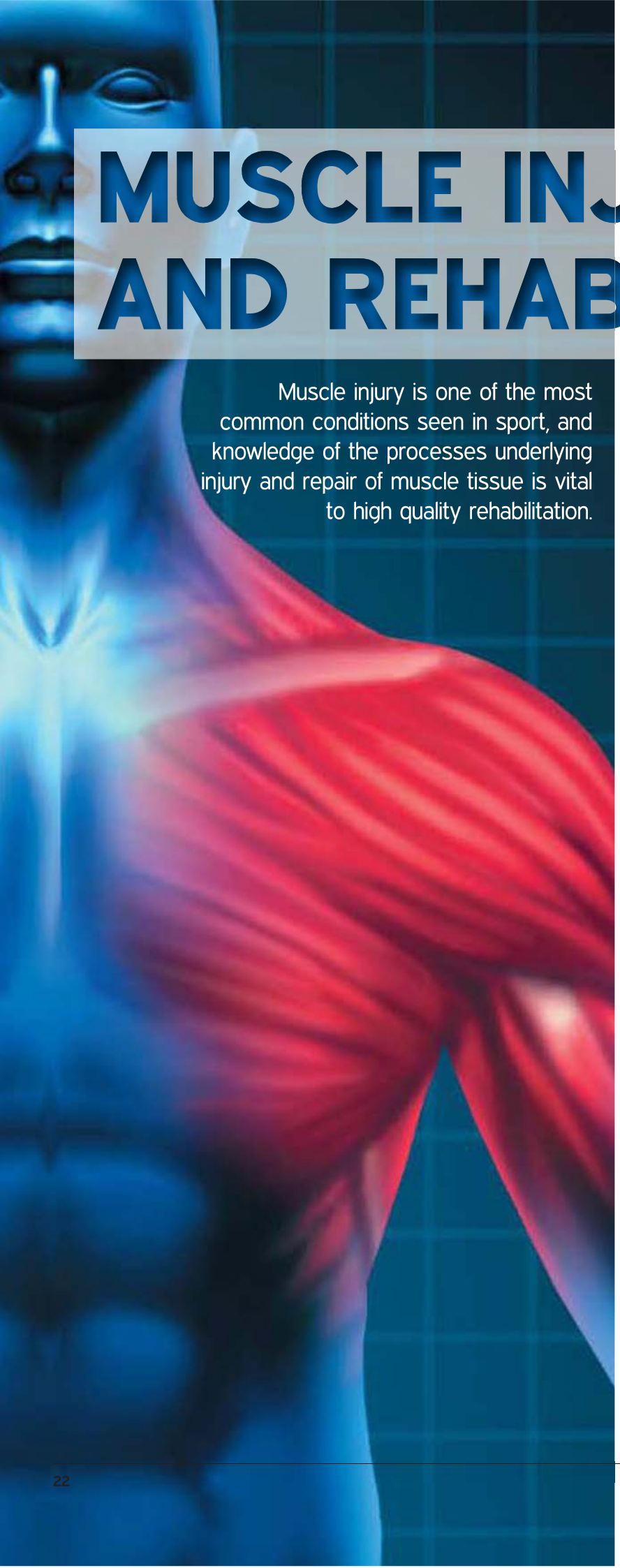


MUSCLE INJURY AND REHABILITATION

Muscle injury is one of the most common conditions seen in sport, and knowledge of the processes underlying injury and repair of muscle tissue is vital to high quality rehabilitation.

BY DR CHRIS NORRIS, MCSP, PHD

STRUCTURE

Muscle consists of both contractile and non-contractile elements (figure 1). Each individual muscle fibre (myofibre) is a long multinucleated cell, which may be up to 50cm long, for example in the long Sartorius muscle. The myofibres are surrounded by a thin membrane (endomysium or basement membrane), and in turn the individual fibres are grouped together in bundles covered by the perimysium. Finally, the whole muscle structure is encased further by the epimysium, the thickest and strongest of the three sheaths. The membranes constitute the non-contractile portion of the muscle and stretch the whole muscle length from tendon to tendon. In so doing, they intimately link the contractile and non-contractile portions of the muscle with the total structure, making up the musculo-tendinous unit. The non-contractile elements form a skeleton for the myofibres which transmits the force created by contraction to a single point of application at the musculotendinous junction (MTJ).

A further membrane, the sarcolemma, surrounds the individual muscle cells. The sarcolemma is important because it is electrically conductive; it has within it sarcoplasm containing fuel stores (glycogen) and enzymes important to muscle contraction. Within the sarcoplasm is an intricate membrane, the sarcoplasmic reticulum. This membrane contains transverse tubules, each of which end on the muscle cell surface as a lateral sac for the transmission of calcium into the muscle to instigate the process of contraction. The sarcolemma contains two important molecules, integrins at the ends of the MTJ, and dystrophin (dystrophin-glycoprotein complex), focused mostly at the MTJ but also spread throughout the sarcolemma. These molecules give the sarcomere its high tensile strength, with the MTJ being able to withstand forces up to 1000kg during maximum muscle work (1).

Looking closely at each fibre, we see alternating light and dark bands, corresponding to different muscle proteins. The light area is composed of a thin actin filament which binds to dystrophin at the MTJ, while the dark area consists of a thicker myosin filament. The myosin filament has projections called crossbridges coming from it much like the oars of a boat, and the movement of one muscle protein upon the other constitutes the sliding filament hypothesis of muscle contraction (see animation online).

INJURY CLASSIFICATION

Muscle injury may be either direct or indirect. Direct injury

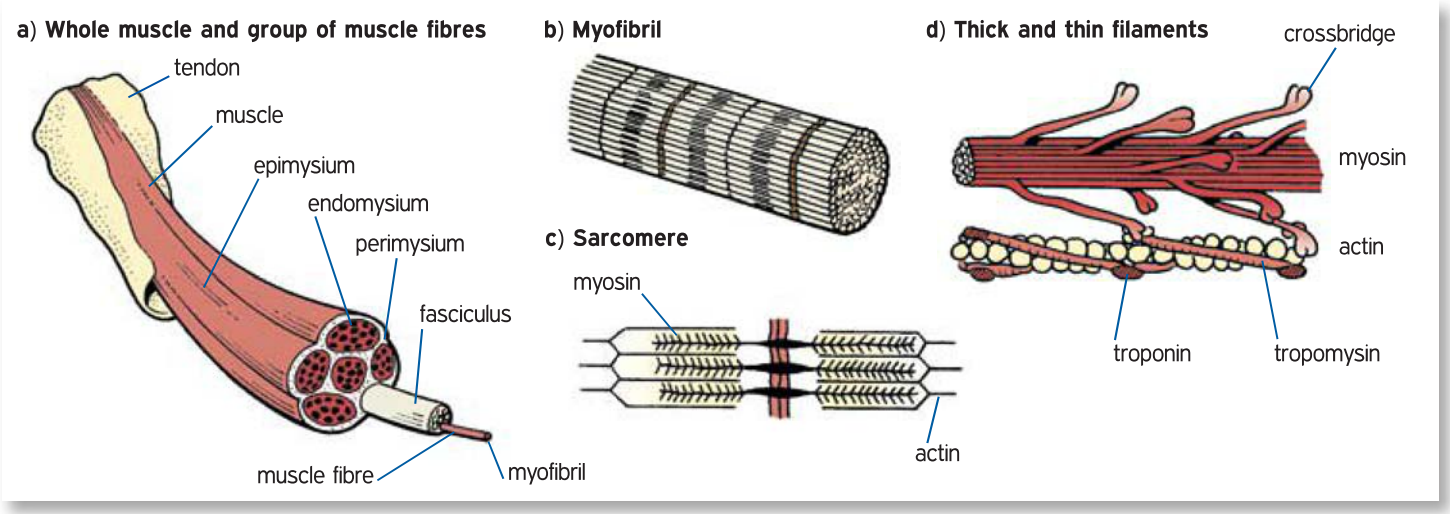


Figure 1: Structure of a muscle (from Complete Guide to Sports Injuries (2011) reproduced with permission from A&C Black)

(applied externally) may result in contusion (bruising) or less commonly laceration (cutting). Indirect injury results in strain of varying categories (table 1). Mild injuries (grade I) involve contusion or tearing of only a few muscle fibres. There is minimal loss of function, slight swelling and discomfort and a feeling of muscle stiffness over and above that of delayed onset muscle soreness (DOMS). Where the muscle is relaxed at the time of a direct injury a contusion occurs deeper within the muscle as the tissue is compressed between the externally applied force (for example a knee in rugby) and the bone. Where the muscle is contracted when struck, the contusion is more superficial as the muscle tension absorbs (attenuates) some of the externally applied force. A moderate (grade II) injury results in greater tissue damage and some loss of function. Tearing across the whole muscle cross section is a grade III (severe) injury, and is associated with complete loss of function. Grade IV injury is sometimes reserved for muscle rupture (2).

Release of blood from broken blood capillaries (extravasated blood) will spread beyond the local area. Where the muscle fascia remains intact, the haematoma formed is contained (intramuscular haematoma). Pressure of the developing haematoma eventually compresses the local blood vessels limiting further blood loss. Where the muscle fascia ruptures however, (intermuscular haematoma) blood will escape through the tear and pressure does not limit bleeding. Blood loss is greater (limited by clotting rather than pressure) and bruising will track between the fascia of two neighbouring muscles within the interfacial spaces to emerge in the limb lower down, or move into the interstitial space to be reabsorbed.

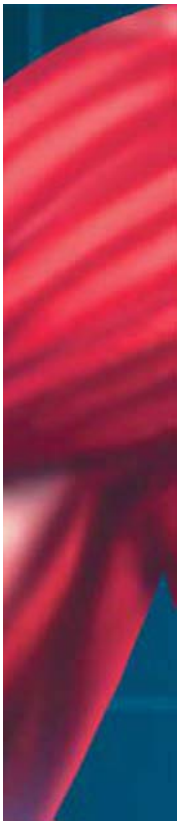


TABLE 1: CLASSIFICATION OF MUSCLE INJURY

Direct	Indirect
Contusion – bruising (haematoma) within muscle	Sprain
■ Intramuscular – muscle fascia remains intact, pressure builds to restrict blood loss	■ Grade I – minor tissue damage, minimal loss of function
■ Intermuscular – muscle fascia tears and blood escapes into interfascial and interstitial spaces	■ Grade II – greater tissue damage and loss of function.
■ Laceration – muscle cut from outside.	■ Grade III – fibre damage across whole muscle cross section
	■ Grade IV – muscle rupture

MUSCLE HEALING

After injury, muscle healing proceeds in three overlapping phases: destruction, repair, and remodelling (table 2, figure 2). During the destruction phase muscle fibres and blood vessels are ruptured, a haematoma formed and inflammation begins. Repair involves removal of dead material and the development of a scar which begins to shrink. Remodelling see tissue maturation and the progressive recovery of muscle function (3).

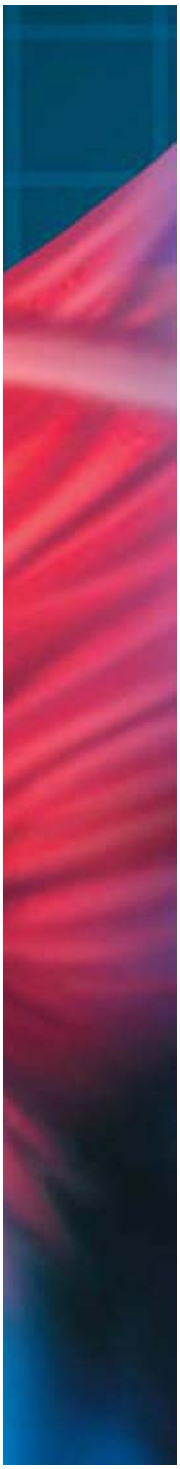
At the time of injury blood vessels have ruptured and released blood into the local area. This blood now represents foreign material which must be cleared, and tissues that have been starved of oxygenated blood (hypoxia) begin to die (necrosis). During the destruction phase the muscle haematoma is forming and inflammation begins. Phagocytes gradually destroy the clot, and fibrin from the clotting blood begins to form granulation tissue. This, in turn, traps fibroblasts to increase the density and strength of the clot structure to begin to withstand the forces created by muscle contraction. The granulation tissue contains proteins (fibronectin and tenascin-C), which, as well as adding strength, are actually elastic. These early proteins are followed by type III collagen (a fibrous scleroprotein) around day 1 after injury and type I collagen (the most abundant type in mammals) 2 days post injury (1).

The increasingly dense granulation tissue forms into a scar, which marks the change from the destruction phase to the repair phase of healing. The repair phase consists of two processes, scar development and myofibre regeneration.

When individual myofibres are torn, as they are long, necrosis could travel the whole cell length, spreading out from the injury site. To limit this, a contraction band is formed as the muscle cytoskeleton shrinks (4). This process of cellular resealing is mediated by calcium ions entering

TABLE 2: HEALING PHASES OF TISSUE INJURY

Destruction	Repair	Remodelling
■ Tissue rupture and local bleeding	■ Phagacytosis	■ New myofibres start to mature
■ Tissue necrosis	■ Scar forms and shrinks	■ Scar reorganises
■ Haematoma formation	■ Regeneration of myofibres	■ Return to full function
■ Inflammatory reaction	■ Tissue revascularisation	
■ Function loss	■ Function begins to return	



the injured cell causing lysosomal vesicles within the cell cytoplasm to fuse (5). Local blood vessel damage causes release of inflammatory products including histamine and prostaglandins. Satellite cells (normally dormant) are activated within the necrosing fibres and in turn release wound hormones and growth factors (6). The growth hormones normally remain inactive and adhered to the extracellular matrix (ECM). Tissue damage causes release of these factors and they amplify the inflammatory reaction (1). Macrophages and fibroblasts are activated and release chemicals to attract other circulating inflammatory cells, a process called chemotaxis. Growth factors, cytokines, and tumour necrosis factor (TNF-α) have all been identified as having this role in skeletal muscle regeneration.

The satellite cells lying at the end of the myofibres provide a source of undifferentiated cells as a reserve, which may be activated through injury. Two types exist, committed satellite cells (CSC) and stem satellite cells (SSC). Activation during the destruction phase of injury causes the satellite cells to proliferate and then to differentiate into myofibres. The CSC react immediately post injury whereas the SSC divide first to increase their number before differentiation. The ability of the SSC to divide means that the satellite cells are not all used up, stocks being replenished to await future injury. Satellite cells seem to be switched on through activation (up regulation) of a potassium ion channel (Kir2.1), which in turn causes the cell membrane to hyperpolarise resulting in calcium influx to switch on the muscle growth (myogenic) activity (7).

The regenerating myofibres of each side of the injured area grow towards each other. The cylinder that contained the old (now necrotic) myofibre forms the casing for the regenerating fibre. The stumps of this new fibre grow outwards, forming several branches that pierce the scar as they approach each other. The ends of the stumps actually adhere to the scar forming small, definite MTJs (4). As a result the shrinking of the scar pulls the stumps together. Adherence to the scar is initially via the stump of the injured myofibre. However this attachment is weak, and is reinforced by lateral adhesion. The lateral adhesion peaks around 5–7 days post injury, and functions to both strengthen the myofibre attachment to the scar and limit myofibre stump movement. Importantly this process requires mechanical stimulation and does not occur as effectively with total immobilisation. By day 14 the lateral adhesive site disappears as the terminal site is now strong enough. Ultimately the two approaching myofibres interlace and contract synchronously but may never completely unite (1).

The young myofibres have very few mitochondria and largely rely on anaerobic metabolism. As they grow, their need for aerobic metabolism increases. This need is fulfilled by the ingrowth of blood capillaries into the scar. Capillary sprouts form from the ends of the old ruptured vessels and grow into the scar. Where fibrosis is excessive and capillary ingrowth is limited, myofibre regeneration will in turn be limited. Reinnervation is also required if myofibre development is to continue. In cases of nerve axon damage (axonotmesis) muscle wasting remains until nerve regeneration is complete.

CLINICAL GUIDANCE

For convenience we can divide the management of muscle injury into three phases (figure 2). Phase one is the immediate post injury care from first aid to day 2–3 post injury. Phase two takes us from day 2–3 to day 10–12 post injury – a point at which the scar (formed within the injury as part of the healing process) reaches its peak strength (1). Phase three continues from 10–12 days post injury until the return to full function and sporting competition (table 3).

Phase one

The question an athlete often asks following a muscle injury is should they rest or should they train? From a physiological standpoint both have value, but at different times. As healing progresses through repair and remodelling, the scar shrinks with little formation of fibrosis unless the limb is immobilised for too long. Immobilisation (no movement) within the destructive phase of injury encourages capillary ingrowth to the scar, and allows the granulation tissue time to develop. If mobilisation (movement) is begun immediately post injury a larger scar will result as the granulation tissue is pulled apart, and additionally penetration of the myofibres into the scar is delayed (8).

In phase one (injury to 2–3 days post injury) the P.R.I.C.E

TABLE 3: CLINICAL GUIDELINES FOR MUSCLE INJURY RECOVERY

Phase 1	**0-2 days**	■ PRICE (protect, rest, ice, compression, elevation)
Phase 2	**2-12 days**	■ Controlled and closely supervised exercise therapy ■ Progressive exercise therapy to stimulate tissue overload
Phase 3	**12-α days**	■ Begin to build fitness relevant to athletes sporting activity ■ Functional training

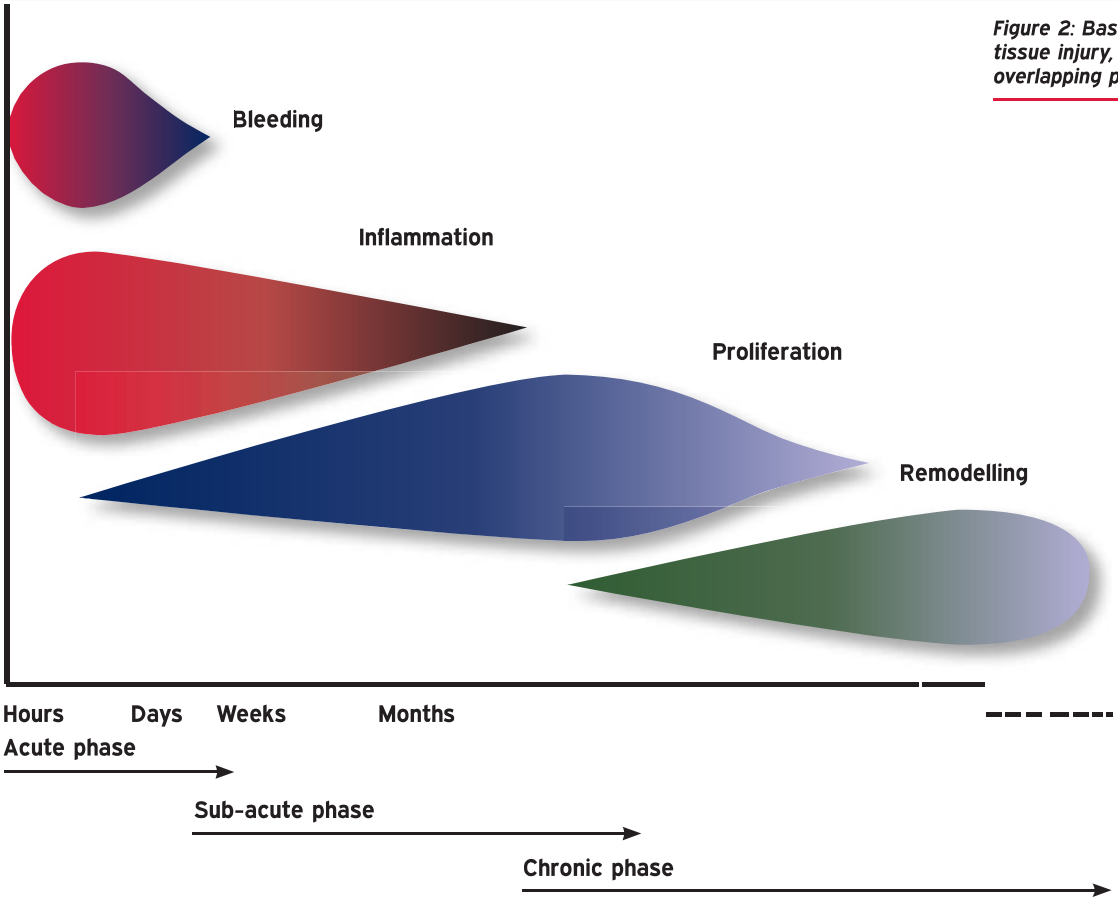


Figure 2: Basic response to tissue injury, and the subsequent overlapping phases of healing

mnemonic (Protect, Rest, Ice, Compression, Elevation) should be used for guidance. The injury should be protected using an external support such as a splint or compression taping to unload the tissues and prevent disturbance of the consolidating haematoma. The athlete should rest from any activity which may stress the injured area. In some cases this may mean total rest, but in others the injured area may be protected sufficiently to allow exercise to the rest of the body. An injured calf or ankle for example may be placed in an inflatable splint allowing full use of the upper limbs and trunk and partial use of the legs.

The use of ice or cold should be combined with compression, to reduce the haematoma size, slow the tissue metabolic rate and reduce secondary tissue necrosis through local hypoxia. Increasing the metabolic rate of the injured muscle through exercise immediately following injury may result in greater damage to the surrounding tissues through hypoxia than that which occurred at the time of injury. Ice or cold will also have the effect of inducing some local analgesia and therefore facilitating the rehabilitation process by reducing muscle inhibition and/or the development of movement dysfunction through pain. Ice is traditionally applied for 5–10 minutes each hour (9,10). Clinically, treatment results support this action but evidence of its effectiveness is lacking (11), and longer periods of cryotherapy have been shown to be effective in the laboratory situation (12).

Compression is applied using external mechanical pressure (typically 15–35mm/Hg) which may be supplied by an elastic tubular bandage (double Tubigrip) or a proprietary cold compression unit (e.g. a cryocuff). To prevent fluid pooling in the case of combined muscle and joint injury orthopaedic felt padding may be used (e.g. horseshoe shape beneath the lateral malleolus).

> "MUSCLE HEALING PROCEEDS IN THREE OVERLAPPING PHASES: DESTRUCTION, REPAIR, AND REMODELLING"

Where swelling develops in the lower limb elevation is used to reduce fluid pooling through the pull of gravity, and enforce rest. The limb is raised to a level above the heart to reduce vascular hydrostatic pressure within the lower limb and minimise resistance to fluid flow through the lymphatic system (13). Reduction in swelling is temporary and upon standing a rebound effect may occur if volume quickly returns to pre-elevation levels. A gradual reduction in elevation (legs up the wall, legs on stool, lying, sitting, standing) is also recommended.

PHASE TWO

Continued immobilisation risks muscle atrophy, excessive scar deposition and reduced strength if it is maintained beyond the acute phase of inflammation, which usually finishes by day 3–4 post injury. In phase two (2–3 days post injury) exercise is begun to assist the healing process, with mobilisation now causing a more parallel orientation

of the developing myofibres (fig 3). Stimulation of the healing tissue through controlled exercise/loading results in mechanotransduction (14). Tissue overload leads to the increased release of mechanogrowth factor (MGF), a member of the insulin-like growth factor (IGF) family. MGF has been shown to activate satellite cells within muscle (6), and is important in both muscle injury and age related muscle wasting or sacropenia (15). Isometric muscle action may be used to assist the muscle pump and facilitate fluid clearance. Where muscle inhibition is noted, isometric contraction of both limbs (ipsilateral and contralateral) can lead to neural overflow and assist contraction of the injured muscle. Where isotonic exercise is used it should be practised within the pain free range. Eccentric action creates greater force than concentric and often athletes will be able to perform controlled lowering (eccentric) movements before they are able to lift their limb (concentric) against gravity. A gravity-eliminated position, involving horizontal (transverse plane) rather than vertical (sagittal or frontal plane) movements, is normally used prior to gravity-loaded (limb weight alone) or resistance-loaded (weight or bands) actions. Free movement within water has the advantage of buoyancy which effectively reduces the weight of the limb being moved.

Initially specific movement of the injured limb is used to target the affected muscle remembering that the majority of indirect muscle injuries occur to two-joint (biarticular) muscles such as the hamstrings, rectus femoris and gastrocnemius in the lower limb, and biceps and triceps in the upper limb. These structures require exercise which moves both joints either separately or together. General actions at the start of phase two are used to the non-affected parts of the body to maintain fitness. As phase two progresses to phase three, we should begin to link movements together to reflect normal function both on the field and off.

Muscle lengthening (to full available range) and stretching (aiming to increase range) is of value within the limits of pain.

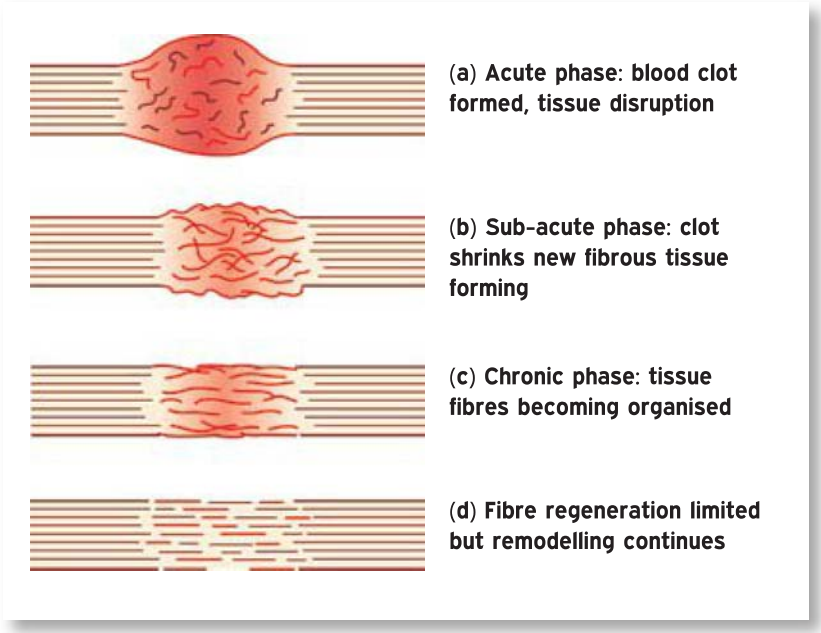


Figure 3: Phases of tissue healing (reproduced with permission from The Complete Guide to Sports Injuries, published by A&C Black 2011

General warm up prior to stretching is useful to prepare the tissues through reduction of tissue resistance, and stretching (static stretch and hold) should also be used. Holding times of 10–20 seconds should progress to 20–30 seconds and movement range should increase progressively (16).

Phase three

Phase three (from 10–12 days post injury) sees an increase in training volume with a gradual switch towards more functional training. For clinical guidance the F.I.T.T mnemonic (Frequency, Intensity, Time and Type) is useful. Muscle must be overloaded to stimulate adaptation, but the overload must match the stage of healing and the state of the tissues. Continual feedback from the athlete is used to monitor and modify exercise therapy. Generally for skill-based movement higher frequencies are used to facilitate practice and improvement of motor skill. Simple leg straightening actions or rehearsal of correct gait patterns for example may be practised three times each day. For force-based actions such as high resistance (strength training) or end range stretching (developmental rather than maintenance stretching) because the intensity is higher, the frequency of exercise practice should reduce. Static stretching for 40–60 seconds, PNF stretching (use of muscle reflexes; proprioceptive neuromuscular facilitation), and dynamic stretching (stretching with movement) are appropriate to phase three but are all intense actions. Recovery is required from these activities so training in these areas may be practised every other day for example.

Timing of exercise reflects both a single repetition and multiple reps. For normal resistance training concentric, isometric and eccentric actions may be used in a typical 2/1/3 ratio. Where practising eccentric loading of the hamstrings (e.g. Nordic hamstring exercise), the emphasis is on the eccentric phase with timing 2/1/10 where as for stability training (e.g. abdominal hollowing) the emphasis would be 1/10/1. Timing of multiple repetitions reflects the need to use both strength and endurance activities (differing energy systems) and to recognise that actions require acceleration (force production) and deceleration (force acceptance).

Exercise type must reflect the requirement of a particular sport, and here remembering the S.A.I.D mnemonic (Specific Adaptation to Imposed Demand) is useful. Prior to reintroduction back into the sports environment athletes need to have challenged their injured muscle in similar situations to those that they will use in competitive sport. Exercise therapy progresses from single- to multi-joint actions using all types of muscle contraction, energy systems, range of motion, and skill levels. Restricting rehabilitation to the muscle which has been injured alone (specific training) has been shown to produce poorer results than a whole body approach (generalised training) involving limbs and trunk combined (17).

CONCLUSION

Correct treatment of muscle injury requires knowledge of the muscle structures and an understanding of the events that occur following the injury. Recovery can be divided into

three well-defined phases, and the appropriate treatment depends on the recovery phase. For example, immobilisation is necessary immediately but is detrimental if maintained for too long, and phase two healing benefits from the careful reintroduction of exercise. Finally, sport-specific exercises are needed, as well as whole-body training, for the best rehabilitation outcome.

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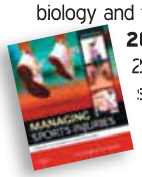

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Christopher Norris is a physiotherapist specialising in musculoskeletal treatment. He is an external lecturer and external examiner to several universities and runs postgraduate continuing professional development courses (listed on his website at www.norrisassociates.co.uk). He is the author of several therapy books.

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