



RECOVERY TECHNIQUES AND PRACTICAL GUIDELINES

Mike I. Lambert and David Van Wyk

¹MRC/UCT Research Unit for Exercise Science and Sports Medicine,
Department of Human Biology, Faculty of Health Sciences, University of Cape Town
South Africa

² David van Wyk, Registered Physiotherapist,
862 Barnard Street, Erasmuskloof, 0048

Address for correspondence:

Associate Professor Mike Lambert

MRC/UCT Research Unit for Exercise Science and Sports Medicine,
PO Box 115, Newlands 7725, Cape Town, South Africa

Phone: +27 21 6854558, Fax: +27 21 6867530, Email: Mike.Lambert@uct.ac.za



*Providing coaches, referees, players, and administrators with the
knowledge, skills, and leadership abilities to ensure that safety and best
practice principles are incorporated into all aspects of contact rugby.*

OUTLINE

It is well understood that recovery forms an integral part of the whole training process. High volumes of training with insufficient recovery lead to symptoms of fatigue with an accompanying high risk of injury. Recovery after exercise can be passive or active. Passive recovery allows the body to recuperate without any the intervention. Active recovery, which can take many forms, attempts to accelerate the rate of recovery so that the player is better prepared for the next training session. A review of the literature on the popular strategies which are used to accelerate recovery in rugby (cryotherapy, massage, stretching, compression garments, active recovery, nutrition, sleep and non-steroidal anti-inflammatories) shows that there is not overwhelming evidence supporting any of these techniques. This can be attributed to the fact that many of the studies are laboratory-based and the protocols which are used to simulate training or competition may lack specificity. Another potential problem is that the markers which are used to define the state of recovery are indirect and also lack specificity. Despite the lack of scientific support, there is fairly strong anecdotal evidence to suggest that “something is better than nothing”. Furthermore, practical experience shows that once a recovery strategy is designed, it should become ritualistic and habitual for the players within the team. They need to be educated about the importance of recovery and be expected to take some responsibility for their own recovery. With this as background, some practical examples of various strategies are provided.

INTRODUCTION

Training for high-performance sport involves a systematic application of a training load, followed by a period during which recovery and adaptation can occur. If there is any imbalance between training load and recovery, the athlete will either incur symptoms of overtraining or underperformance. It has been known for a number of years that each training session imposes a physiological stress, which causes a disturbance of the intracellular homeostasis as well as transient physiological and metabolic changes⁽²²⁾. The nature of these changes depends on the type, duration and intensity of exercise⁽³⁾. This is the primary signal for stimulating adaptation of the muscle and other organs in the body which are associated with improved performance.

For competitors at the elite level, there is a fine line between doing too little or too much training^(45;48;56). Insufficient training does not induce adequate adaptations and results in suboptimal performance. In contrast, too much training results in maladaptations, or the failure to adapt, causing symptoms of chronic fatigue and poor performance^(11;24). Chronic fatigue can also be caused by inadequate recovery. In a review on the basic errors of training, neglecting recovery was regarded as one of the main causes of chronic fatigue.

The other factors listed in the review follow below ⁽⁷⁰⁾:

- demands on the athletes are made too quickly
- after a break in training because of illness or injury, the training load is increased too quickly
- high volume of maximal and submaximal training
- overall volume of intense training is too high when the athlete is training for endurance events
- excessive time is devoted to technical or mental aspects, without adequate recovery
- excessive number of competitions – this includes frequent disturbances of the daily routine and insufficient training time which accompanies competition
- bias of training methodology
- the athlete has a lack of trust in the coach because of inaccurate goal setting

Whilst recovery may be interpreted as a passive process, strategies have been developed which attempt to accelerate the rate of recovery. Data from studies on soccer players showed that optimising recovery after training and matches depend on a combination of factors that consider individual differences between players and lifestyle factors. The procedures to accelerate the recovery should start immediately after the end of the match or training session ⁽⁶⁴⁾. Without these procedures, it is unlikely that a player will be able to maintain a high level of performance throughout the season. This will result in a loss of form and confidence, which will spiral out of control unless there is a positive intervention. Recovery should be viewed as an action orientated, multidimensional process involving a number of systems, all of which might have a different time course ⁽⁴⁰⁾. Furthermore, recovery strategies can be discussed in the short term, (i.e. immediately after training and competition), and also in the long term (i.e. throughout the season). Ensuring long-term recovery requires an ongoing monitoring programme to prevent the levels of fatigue from becoming unacceptably high. The short-term strategies for accelerating recovery between training sessions or after a match will be discussed in this chapter, and the long-term strategies for monitoring recovery will be discussed in the section on overtraining, which appears elsewhere in this manual.

THE PHYSIOLOGY OF RECOVERY

During exercise the metabolic rate increases. This is measured as increased oxygen consumption. Immediately after exercise the oxygen consumption declines. First there is a rapid decline, which lasts about five minutes, followed by a slower decline lasting for up to an hour. Circulating lactate, which may

increase up to 10-fold during a rugby match ⁽²⁵⁾, takes about 60 minutes to return to pre-exercise concentrations ⁽¹⁰⁾.

During a rugby match, forwards lose between 1 and 4 kg and backline players lose about 0.8 kg of body mass ⁽⁵²⁾. This is a transient change in body mass with a return to pre-match weight soon after the game, providing there is adequate fluid ingestion.

Muscle glycogen concentrations decrease with exercise. The concentrations continue to decrease even after exercise has ended, particularly when muscle damage occurs. Glycogen levels return to their pre-match level within 24 hours providing there is no muscle damage. In the presence of muscle damage, glycogen restoration takes about 24 hours longer ⁽⁶⁸⁾. The only study that has been done on muscle glycogen depletion in rugby players showed that a rugby match did not cause significant depletion of muscle glycogen ⁽³⁹⁾. However, studies done on soccer players showed that glycogen stores were reduced to near depletion at the end of a soccer game ⁽⁶⁴⁾. An explanation for the differing results (soccer versus rugby) could be that the subjects were club rugby players whereas the soccer players competed at a higher level. It is reasonable to assume that had rugby players of a higher level been used that more glycogen depletion would have occurred, similar to the findings in soccer players.

Whilst the metabolic rate might decrease down to pre-match levels within an hour, the metabolism associated with muscle damage occurs for longer. There is convincing evidence, as shown by the increased levels of circulating creatine kinase activity, to show that rugby players incur muscle damage after a match, and also as a result of contact at practice ^(72;73). The amount of muscle damage appears to be associated with tackling, as the circulating creatine kinase activity increases in proportion to the number of tackles either delivered, or received ⁽⁷³⁾. The symptoms associated with muscle damage usually develop and reach a peak within 24 to 48 hours after the bout of exercise and then slowly subside, disappearing within five to seven days ⁽¹⁸⁾. Other clinical symptoms associated with muscle damage are inflammation and muscle shortening. After all the symptoms disappear (usually within five to seven days), the muscle continues to regenerate, with signs of regeneration still being present two weeks after the injury. After severe muscle injury, such as that which occurs after a marathon, signs of muscle regeneration are still evident for up to 12 weeks ⁽⁸³⁾.

The goal of recovery is to restore cellular function to pre-exercise levels, without bypassing any of the biological steps that are important for complete regeneration. By excluding or blocking these biological factors, there is a risk of compromising regeneration and the restoration of the cellular function.

Recovery after exercise does not only involve muscle tissue. Following high-intensity exercise, there are aspects of elevated metabolism that have to recover. For many years a conventional understanding was

that increases in muscle and blood lactate and the associated hydrogen ions was the cause of fatigue after high-intensity exercise ⁽¹⁵⁾. This understanding arose from experiments which examined the decline of force during fatiguing exercise and blood lactate concentrations. The erroneous conclusions arising from these studies were that there was a cause-and-effect type relationship between the lactate and fatigue. This led to misplaced speculation that a reduction in circulating lactate would delay fatigue, and extending this argument, that clearing blood lactate after exercise would improve recovery. Therefore the studies which have used blood lactate as a marker of whether recovery has occurred or not, should be interpreted with caution.

A more contemporary understanding of fatigue arising from high-intensity exercise is that impaired muscle function is a consequence of down-regulation by the central nervous system, rather than as a result of fatigue caused by metabolites in the muscle. In particular, after exercise that has repetitive stretch shortening cycle actions, the pre-activation of the muscle decreases, resulting in impaired control of the stiffness of the muscle. This results in a reduced force output, which is proportional to the degree of exercise-induced muscle damage ^(29;38).

Recovery after exercise is dependent on the physical demands of the bout of exercise. It is unrealistic to expect all players to be affected similarly by a match. There are distinct differences in the physiological demands of playing rugby according to the playing positions ⁽²⁵⁾. Forwards spend more time competing for the ball and are involved in physical contact, whereas the backs spend more time running. A study on Vodacom Cup players show that there were 382 (range 306 – 535) impact contacts per match, with the forwards being involved in 68% of these (257 per match (range 199 – 389), while the backline players were involved in 125 (range 93 - 148) impact contacts per match ⁽⁸⁰⁾. The evidence suggesting that rugby forwards have more physical demands placed on them compared to the backline players is supported by a study which showed that forwards had a marker of muscle damage (interstitial creatine kinase concentration) which was nearly threefold higher compared to the backline players ⁽⁶⁹⁾. Studies have also shown, based on heart rate that the backs do less work during a match than the forwards. About 95% of the bouts of activity during a rugby match last less than 30 seconds. The rest periods in between these bouts are generally greater than the preceding bout of exercise ⁽²⁵⁾.

Muscle pain is a poor marker of the state of recovery after exercise that causes muscle damage ⁽⁶⁰⁾. Other indirect markers of muscle damage such as circulating creatine kinase and inflammation are also not necessarily related to the amount of muscle damage. Some studies, which have examined the efficacy of modalities to accelerate recovery, have used pain and indirect markers of muscle damage as their main outcome measures. These studies may have come to the wrong conclusions because of the poor relationships between the “state of the muscle” and markers of muscle damage. This has

unfortunately confounded the evidence-based approach to managing recovery, which will be discussed in more detail later.

In summary, it may be concluded that rugby players have varying degrees of muscle damage after training and matches, ranging from very marginal (backline players) to the more serious damage (loose forwards). There will be accompanying symptoms, such as inflammation and impaired muscle function, with the more serious muscle damage. In most cases, players will have lost intracellular fluid and muscle glycogen after a match. Recovery can be defined in many different ways, but from the perspective of a rugby player, recovery should be defined as the point at which the player is able to train without constraints of sore muscles or an increased risk of injury. This physiological definition does not exclude the fact that there are cognitive processes which also need to recover following a stressful match or period of traveling across time zones, which interferes in sleep patterns. Optimal recovery requires a multidimensional approach that addresses all aspects of the rugby player's lifestyle, such as sleep, nutrition, overall stress exposure and physiological recovery ⁽⁴¹⁾. The next section will examine the experimental evidence supporting or refuting the efficacy of the often-used recovery strategies in rugby. This will be followed by guidelines, which have been established using evidence-based principles (where available) and anecdotal experience.

TYPES OF RECOVERY TECHNIQUES

Recovery needs to be a proactive process and is an integral part of the entire training programme ⁽⁴⁰⁾. Whilst there are several laboratory studies which have attempted to provide evidence for the efficacy of these interventions, many of these studies are limited because they either use untrained subjects, they use non-specific modalities to induce fatigue/muscle damage, or they define their outcome measures of recovery rather poorly. Taking all these factors into account, there are only a few studies which can be used to make evidence-based decisions about techniques for accelerating recovery. The next section will include a discussion on cryotherapy (including cold-water immersion and contrast-temperature therapy), massage, stretching, compression, active recovery, nutrition, sleep, and non steroidal anti-inflammatory drugs, as these are the recovery modalities which are commonly used in rugby.

Cryotherapy

Cryotherapy is a term which describes a range of therapeutic treatments aimed at lowering tissue temperature by the withdrawal of heat from the body ⁽¹⁷⁾. Treatment can be applied either in the form of ice packs, ice massage, ethyl chloride, cold air, or cold water immersion ⁽⁵¹⁾. These treatments have a long history in medicine, with their use ranging from the removal of warts, to more conventional applications aimed to reduce swelling after tissue trauma, and the treatment of pain. More recently,

cryotherapy has been used as an ergogenic aid, as whole-body immersion in cold water before exercise in the heat has been associated with an improvement in performance ^(9,49).

Various forms of cryotherapy are used as an intervention for post-exercise recovery, particularly after exercise which raises body temperature, and causes inflammation of muscles ⁽⁷¹⁾. The basis for using cryotherapy is on the assumption that it is effective in decreasing metabolic rate, inflammation, blood flow, and skin, muscle and intra-articular temperatures ⁽⁵³⁾. Cryotherapy increases pain threshold and pain tolerance possibly as a consequence of a significant decrease in nerve conduction velocity (1). However, prolonged exposure to cold may have negative effects, from a recovery perspective, because blood flow increases in the muscles when the muscle temperature reaches about 10° C. Furthermore, the permeability of the lymph vessels increases at prolonged low temperatures, resulting in increased subcutaneous swelling ⁽⁵¹⁾.

The wide range of cryotherapy modalities, differences in application, and duration of treatment all contribute to varied results in the treatment lowering skin temperature. Chesterton (2002) has summarised a range of studies which showed that the mean skin temperature reduction ranged from 7°C (15 seconds application of ice massage) to 26.6° C (10 minutes of ice massage) ⁽¹⁷⁾. Therefore to achieve the required clinical response, it is important that the modalities are clearly understood. For example, localised analgesia requires a skin temperature of below 13.6° C ⁽¹⁷⁾.

The amount of subcutaneous fat in the area being cooled influences the rate of intramuscular cooling with which cryotherapy has an effect. For example, a study was done on subjects who were assigned to groups dependent on their calf skinfold thicknesses (less than 8 mm, 10 to 18 mm, and greater than 20 mm) ⁽⁵⁹⁾. Intramuscular temperature was monitored every 10 seconds over a 20-minute treatment at 1 cm and 3 cm below the subcutaneous fat in the left medial calf during and immediately after a 1.8 kg crushed-ice pack treatment and again 30 minutes after the treatment. The amount of adipose over the therapy site had a significant effect on the decrease in intramuscular temperature that occurs during and after cryotherapy. This study has practical applications for treating rugby players with cryotherapy, as the amount of subcutaneous fat will vary between players ⁽²⁶⁾.

Whether cryotherapy improves recovery remains open to debate. Relevant studies on cold-water immersion and contrast temperature therapy will be discussed below.

Cold-water immersion

A study reviewed the effectiveness of cold-water immersion and active recovery in 10 well trained cyclists following high-intensity exercise conducted in hot conditions (34°C) ⁽⁷⁹⁾. The therapies consisted of intermittent cold-water immersion for 15 minutes at either 10°C, 15°C, 20°C; or a 15-minute continuous

immersion at 20°, or active recovery. Recovery was assessed as a function of the total work performed after the treatment. The cold-water immersion protocols all reduced the thermal heat strain of the subjects after the high-intensity exercise, compared to the active recovery protocol. There were no differences in total work performed between any of the cold-water immersion protocols. However, the subjects were able to maintain their repeat performance in the heat following all of these interventions in contrast to treatment with active recovery. The authors concluded that cold-water immersion may be a useful strategy in sports where there are going to be two training sessions a day performed in hot conditions. This study was not designed to investigate the effect on inflammation and muscle damage. This question was examined in another study where healthy active subjects were exposed to exercise which caused severe muscle soreness and dysfunction and elevated serum markers of muscle damage (myoglobin and creatine kinase activity). Subjects were then assigned to treatment which consisted of 10 minutes of cold-water immersion (10°C) ⁽⁶⁾. Subjects had reduced muscle pain at 1, 24 and 48 hours after the exercise. The treatment had no effect on creatine kinase activity; however myoglobin was reduced one hour after the exercise. These results suggest that cold-water immersion therapy reduces some of the symptoms of exercise-induced muscle damage. The authors postulated that the effect was manifested either through a decreasing intramuscular temperature with a reduced inflammatory response, or an alternative explanation for the findings is that immersion caused haemodynamic changes as a result of increased hydrostatic pressure.

In another study, subjects stood in either cold water (8°C) or hot water (44°C) up to their gluteal folds for 10 minutes ⁽¹²⁾. All subjects (n = 45) exercised only the right lower limb using a modified proprioceptive neuromuscular facilitation flexibility protocol, consisting of 1 set of 4 repetitions. This procedure was followed for 5 consecutive days. The maximum active hip flexion was measured on the first and fifth days. Both groups had significant improvements in hamstring length (pretest to posttest); however, there were no significant differences between groups. The authors concluded that there was no advantage in using either hot or cold immersion to increase hamstring length in healthy subjects ⁽¹²⁾.

Regular exposure to cold therapy during training may compromise the adaptation of the muscle. This was shown in a study which examined the effects of regular cold application to exercised muscles after training ⁽⁸⁵⁾. Training occurred either by a cycle ergometer or a handgrip dynamometer and cold therapy consisted of cold-water immersion (5°C for 20 minutes, short rest and then repeated). After six weeks, significant training effects were more frequent in the control than in the cold group, including increases in artery diameters in the control but not in the cold group. The authors concluded that training-induced molecular and humoral adjustments during the bout of exercise, including muscle hyperthermia, are physiological signals which are important for inducing training effects (myofiber regeneration, muscle hypertrophy and improved blood supply). These data suggest that cooling generally reduces the signals

associated with increased muscle temperature, and which may have a negative effect on training adaptations. This however should not be confused with the potential benefit effect of cold therapy in the treatment of tissue trauma, i.e. as part of the RICE treatment ⁽⁶⁵⁾. The so-called “negative” effects of cryotherapy were also shown in a study which examined cold-water immersion in 40 untrained subjects who performed eccentric loading on one leg to induce muscle soreness ⁽⁶⁶⁾. Immediately after exercise, subjects were randomly assigned to either a cold-water immersion group (5°C) or tepid water (24°C) for three minutes. Response to these interventions was measured at 24, 48 and 72 hours after the exercise. There were no differences between treatments with regards to swelling, muscle function or circulating creatine kinase activity. However, at 24 hours the cold-water immersion group had more pain when doing a specific movement compared to the control group. This was a paradoxical finding which argued against the use of cold-water therapy as a recovery strategy. As mentioned earlier, a distinction should be drawn between the responses of untrained subjects versus trained subjects. As this study used untrained subjects, it would be interesting to see whether similar findings were obtained with this protocol in a group of more highly trained subjects.

Several studies have used protocols consisting of predominantly eccentric actions to induce muscle damage to determine the efficacy of cryotherapy treatment. For example, Eston and Peters (1999) used an immersion protocol (15° C for 15 minutes after exercise and thereafter same treatment every 12 hours for seven sessions) following eccentric exercise that caused muscle damage. Although they showed that muscle stiffness was reduced, there were no other signs of enhanced muscle repair ⁽²⁷⁾. Another laboratory study, which also used eccentric exercise as a way of inducing muscle damage, also showed that cold-water immersion (20 minute immersions in a 5.5° water bath, interspersed by 60 minute rest periods, was ineffective in reducing symptoms of muscle soreness ⁽⁶³⁾.

In another study with a similar design, a group of subjects completed 100 drop jumps to induce muscle soreness, followed by 12 minutes of cold water immersion (15°C). This treatment was repeated every 24 hours thereafter for three days. Although there were measurable symptoms of muscle damage after the drop jumps, the subjects who had the cold water therapy did not have any advantage over the control subjects ⁽³⁰⁾.

The data from these laboratory studies ^(27;30;63) should however be interpreted with caution because they used eccentric actions to induce muscle damage, and therefore the association with tissue trauma arising from this intervention compared to the physiological trauma of a rugby match may be limited. For meaningful comparisons between studies, a number of factors need to be controlled, such as the intervention causing the fatigue and muscle damage, the type of cold-water immersion protocol, the duration of exposure, the body area immersed, and the temperature of the water. Given these limitations,

the available data are rather sparse, making it difficult to formulate an evidence-based decision about the efficacy of cold-water immersion after rugby. However, physiological evidence aside, there does seem to be anecdotal evidence for using cold-water immersion after a match or hard training session. Perhaps the ritual of undergoing the cold-water therapy focuses attention on recovery, and the increased attention indirectly enhances the recovery process? Further research is needed to answer this question.

Contrast temperature therapy

Contrast temperature therapy consists of alternative cold and hot treatment through contrast temperature baths or warm and cold packs ⁽¹⁹⁾. A reduction in oedema and bruising, vasodilation and vasoconstriction of blood vessels, blood flow changes, and influences on the inflammatory responses have been attributed to this modality ⁽³⁷⁾. The mechanism of action is however unclear as studies have shown that contrast therapy had little effect on deep muscle temperature ^(37;58). Therefore the theory that the effects of contrast therapy can be attributed to fluctuations in tissue temperature is not founded on experimental evidence.

The data supporting the efficacy of this therapy is equivocal. For example, 20 rugby players performed a repeated sprint test and then were either allocated to a contrast temperature water therapy or active recovery ⁽³³⁾. The therapy consisted of three 1-minute immersions in cold water (8 - 10°C) up to hip height, alternated with three 1-minute hot water (38°C) showers. The active recovery consisted of six minutes of slow jogging. The contrast temperature group had a decrease in blood lactate concentration three minutes after the procedure and also had lower heart rates after the procedure and later when the subjects did a further set of exercise. There were no meaningful differences in sprinting performance one hour after either recovery treatment. Another study has also shown a reduction in plasma lactate after intense exercise, following contrast water immersion ⁽⁵⁷⁾. In this study, the lower bodies of the subjects were immersed in hot- (36°C) and cold- (12° C) water baths, after a bout of high-intensity exercise. On average, the exposure time was four minutes hot, one minute cold (5 repetitions). Although the subjects exposed to the contrast water immersion had a marginal decrease in lactate concentration, it is questionable whether this small difference had any practical significance.

Although recommendations have been made about the ratio of warm to cold exposure and duration of treatment, there is a lack of scientific evidence to support the efficacy of any of these combinations ⁽¹⁹⁾. At best it can be said that any protocol involving contrast temperature therapy is based on anecdotal experience.

Massage

Massage is widely used by athletes to prepare for exercise and accelerate recovery from training and competition ⁽⁸⁴⁾. Data collected from 12 major national and international athletic events between 1987 and 1998 showed that physiotherapists spend between 24% and 52% of their time using massage. The premise upon which massage treatment exerts its effects are thought to be through decreasing oedema and reducing pain, enhancing blood lactate removal, and promoting healing by increasing muscle blood flow. These proposed mechanisms are not always supported by the scientific evidence ^(36;62;78), suggesting that the effects may be psychological. Some studies have found that massage does not reduce symptoms of muscle pain ⁽³⁴⁾. Other studies have found to the contrary. For example, ten healthy subjects performed eccentric exercise of the elbow flexors designed to induce muscle soreness (86). The arm which underwent exercise received 10 minutes of massage therapy three hours after the exercise. Massage was effective in reducing inflammation and the symptoms of pain by 30% but it had no effect on muscle function. These results show once again that pain and muscle function are disassociated and that muscle pain should be used with caution as a clinical marker of how a muscle has recovered.

It is understandable why the results of the studies that have used massage to alleviate symptoms of muscle damage are quite varied. This can be explained because there are many different types of massage therapy, while therapy duration and frequency can also influence the results. Furthermore, it is quite difficult to do a completely blinded study using massage as an intervention therapy, making the results difficult to interpret.

A review of all the published studies on massage showed that most studies contain methodological limitations, including inadequate training of the massage therapists, insufficient duration of treatment, too few subjects in the experiment, or over- or under-working of muscles that limits the practical conclusions which can be derived ⁽⁵⁴⁾. However, it may be concluded from all these studies that generally muscle soreness arising from DOMS is reduced with massage. Whether impaired muscle function also recovers after massage remains less clear. Furthermore, whether recovery is enhanced after massage is also unresolved. In conclusion, there are sufficient positive results from the peer-reviewed literature to support the application of massage as part of the recovery process; however there is need for further research to fully understand the mechanism of action.

Stretching

The main goal of stretching is to increase the range of motion around joints. The data showing that this indeed occurs is convincing ⁽⁷⁶⁾. Stretching is commonly advocated as a technique for reducing the risk of injury ⁽³⁵⁾ although the research does not necessarily support this ^(4;76). The evidence supporting stretching as part of a recovery protocol is less convincing. A mechanism by which stretching may enhance the

recovery process has yet to be identified. Furthermore, there do not appear to be any studies that have investigated the effect of stretching between exercise sessions/matches on performance during post-recovery exercise/competition⁽⁶⁾. Stretching exercises have been shown to be ineffective in reducing the symptoms of muscle damage^(18;20;32;54). A comprehensive review of studies which had used stretching after exercise (total stretching time ranging from 300 to 600 seconds) with the goal of reducing muscle soreness, showed that 72 hours after exercise, pain had only reduced by 2%, which was not regarded as meaningful⁽⁴⁾.

Compression

Compression is a therapeutic technique whereby external compression is applied following exercise or an injury. The theory behind this modality suggests that the external pressure reduces oedema by creating an external pressure gradient, thereby reducing the efflux of fluid from capillaries. Furthermore, the space available for fluid leakage is reduced, minimising haemorrhage and haematoma formation. Certain types of compression treatments involve a dynamic immobilisation that reduces movement during the recovery process. Although the evidence for the efficacy of this treatment was largely anecdotal, recent studies suggest that compression can be effective in minimising swelling, improving the alignment and mobility of soft tissue, and improving proprioception in an injured joint^(43;44). The only study on compression garments and recovery from rugby showed that the players who wore lower-body compression garments for 12 hours after the match showed similar signs of recovery (defined by clearance of creatine kinase from transdermal exudate) to players treated with active recovery and contrast temperature water immersion. All these treatments were better than no treatment at all⁽²⁸⁾.

Unlike cold therapy, which should be applied intermittently, compression treatment should be applied constantly for at least 72 hours⁽⁴⁴⁾. Furthermore, it is important that the pressure of the compression garment does not exceed diastolic pressure, which is about 40 to 60 mmHg for the upper limbs and 60 to 100 mmHg for the lower limbs. If the pressure exceeds these values, blood flow will be impeded. Ideally, the garment should create a distal to proximal pressure gradient to facilitate the removal of metabolites from the periphery towards the central circulation. This encourages fluid to move away from the high-pressure areas (site of injury) to the lower pressure areas⁽⁴⁴⁾. The commercial production of garments with these characteristics, designed to fit both the upper and lower body, has popularised this form of recovery treatment among rugby players.

Active recovery

Active recovery enhances the removal of high levels of circulating lactate⁽¹⁰⁾. However, as discussed previously, the link between high levels of circulating lactate and impaired muscle function is dubious. It

follows then that if active recovery has beneficial effects, the mechanism of action is then through other mechanisms.

Despite the lack of understanding of the mechanisms of active recovery, there are several studies which show that this method has some positive effects. For example, a recent study on rugby players showed that recovery rates (using creatine kinase in transdermal exudate as a marker) were similar for active recovery, contrast temperature water immersion, and wearing lower-body compression garments – and were significantly better than passive recovery ⁽²⁸⁾. Another study also showed that after high-intensity training a 15-minute treatment of either (i) active recovery (cycling at 30% VO₂max), (ii) massage or (iii) cold-water immersion (15° C) improved recovery (measured by work performed) in contrast to a group which did not use any of these strategies ⁽⁴⁶⁾.

However, not all studies show that active recovery has a beneficial physiological effect. Suzuki et al (2004) found that active recovery did not have any effect on recovery (measured by circulating creatine kinase and neutrophils) after a rugby match, although the players seemed to have better mental recovery after being exposed to an active recovery protocol consisting of low-intensity exercise ⁽⁷²⁾. Another study showed that low-intensity exercise had a temporary analgesic effect on sore muscles, but no effect on recovery from muscle damage ⁽⁸⁷⁾.

Active recovery needs to be an integral part of the training programme and implemented immediately after a training session (cool down), or after a match. Active recovery can also be structured into the programme on days of “easy” training. Active recovery needs to incorporate aerobic-type activity with stretching exercises included ⁽⁴⁰⁾. The activity should be of a sufficiently low intensity as to not induce further fatigue, but also assist with a psychological recovery, particularly after a tense match. Active recovery should always be performed in a non-competitive environment. A popular form of active recovery, particularly the day after a match, is a pool session ⁽⁴⁰⁾.

Nutrition

There are compelling reasons for embarking on a rehydration and refuelling strategy immediately after a training session or match. The basis for this started over 40 years ago when it was shown that exercise performance (moderate to high intensity) is related to muscle glycogen availability ⁽⁷⁾ and that fatigue during such an activity is often associated with a depletion of muscle glycogen. Furthermore, it can be assumed that muscle glycogen decreases during a rugby match, and that for complete recovery these stores need to be replenished.

There is evidence to suggest that ingesting carbohydrates immediately after exercise results in higher glycogen levels six hours later compared to if the carbohydrate was only ingested two hours after

exercise⁽⁶⁶⁾. Muscles that are damaged from the exercise do not restore their glycogen as efficiently as undamaged muscles⁽²¹⁾, possibly as a result of transient insulin resistance⁽⁴²⁾. In a study using magnetic resonance spectroscopy, two groups of subjects exercised and reduced their muscle glycogen by about 50%⁽⁶⁸⁾. One group of subjects incurred muscle damage whereas the other group did not. During the first two hours after exercise, glycogen concentrations decreased even further in the group that had damaged muscles whereas the glycogen concentrations increased in the group that did not have muscle damage. The group that incurred muscle damage needed more than one day to increase their glycogen stores to the pre-exercise levels, even though they ingested a diet high in carbohydrate. This was in contrast to the group that did not incur damage and who were able to restore their glycogen levels within 24 hours. Another study which analysed muscle biopsy samples showed that severe muscle damage reduced the ability to restore muscle glycogen fully for as long as 10 days⁽⁶¹⁾. The contrasting results of these two studies can perhaps be attributed to the severity of muscle damage in the latter study⁽⁶¹⁾. The magnitude of muscle damage which occurs in rugby players is likely to be much less severe than damage which occurred in the laboratory study of O'Reilly (1987)⁽⁶¹⁾, therefore it is reasonable to assume that their muscle glycogen concentrations will take 24 hours or slightly more to replenish.

There is evidence to suggest that exercise capacity will be restored more effectively when a mixture of carbohydrate and protein is ingested during recovery, compared to the same amount of carbohydrate alone⁽⁶⁾. However, in this study, the inclusion of protein in the solution did not offer more benefits than when a more concentrated carbohydrate solution of similar energy content to the carbohydrate plus protein supplement was ingested. However, another study found that consuming a carbohydrate plus protein or carbohydrate drink immediately after a bout of eccentric exercise that caused muscle damage, failed to enhance recovery of the muscle injury differently than occurred with the placebo drink⁽³¹⁾. This suggests that further studies are needed to develop more definitive guidelines on the recommendations of ingesting carbohydrate and protein immediately after exercise, as the results seem to be influenced by the magnitude of muscle damage.

It is well-known that alcohol reduces glycogen restoration after exercise⁽¹⁴⁾. The reason for this is that alcohol either has a direct effect on glycogen synthesis, or indirectly by reducing the carbohydrate intake immediately after exercise. Another reason for avoiding alcohol in the acute period after a match or training session is that alcohol promotes diuresis, which will delay rehydration⁽¹³⁾.

Until further information becomes available, prudent recommendations are that the goal should be to ingest 1 g carbohydrate per kilogram body mass in the first two hours after exercise, particularly of high glycemic index carbohydrate foods. This goal should extend to 7-10 g carbohydrate per kilogram body

mass over 24 hours ⁽¹³⁾. There are obviously practical and logistical considerations, which should include palatability of the fluid and gastrointestinal comfort ⁽¹³⁾.

Sleep

The relationship between sleep and recovery after exercise, particularly relating to performance, is receiving more attention as the link between sleep cognitive function and metabolic function becomes better understood. To fully understand the role that sleep has in the training process, and in particular during recovery, one needs to understand the different phases of sleep ⁽⁶¹⁾. There are five distinct states of consciousness associated with sleep - stages 1, 2, 3, 4 and rapid eye movement (REM). Stages 1 to 4 are often grouped together and referred to as non rapid eye movement sleep (NREM) ⁽⁶¹⁾. During the day, beta brainwaves dominate and reflect a mental state that is actively aware of the surroundings. As one lies down in preparation for sleep, beta waves are replaced by alpha waves. These are associated with a mental state of being awake yet relaxed. After about five to 20 minutes of alpha brainwave activity, the mind is prepared to enter stage 1 of sleep. This first stage can last from 10 seconds up to 10 minutes and is defined by theta brainwaves. During this stage, respiration becomes shallow and muscle relaxation occurs. The stage is also associated with the feeling of falling, and accompanied by a reflex response such as jerking of the arms or legs. As the person progresses into stage two, the theta waves become intermingled with sleep spindles and K-complex waves. Sleep spindles, which can be measuring by EEG signals, arguably define the beginning of actual sleep since the person is oblivious to most external stimuli. This stage lasts from 10 to 20 minutes. Stage 3 is defined by a combination of theta and delta brainwaves, with the delta brainwaves becoming more dominant. Stage 4, which is the deepest stage of sleep, is defined as the period when the theta waves disappear. Stage 3 and stage 4 together are called slow wave sleep. During slow wave sleep, metabolic activity is at its lowest. Growth hormone is secreted during this phase so muscle repair and growth can be maximised ⁽⁶¹⁾.

After about 30 to 40 minutes of the delta sleep, the stage is reversed, reverting back to stage 3 and stage 4. REM sleep begins after this step. During REM sleep there is an increase in blood pressure, body temperature, breathing rate, heart rate and blood flow. Even though the eyelids are closed the eyes move backwards and forwards. Dreams usually occur during REM sleep. There is also a return of beta brainwaves, suggesting that the brain is more active. The cycling between the stages of sleep is repeated between four and six times a night with each cycle lasting about 90 minutes. As the duration of the sleep increases (i.e. after a few cycles) the duration of stage 3 and stage 4 decreases, while REM sleep increases ⁽⁶¹⁾. If sleep is disturbed before slow wave and REM sleep is achieved, the whole process restarts. It is known that disturbances in sleep (insufficient and poor quality, circadian rhythm disturbance) are the main factors that affect the restorative ability of sleep ⁽⁶⁵⁾.

It has been recommended that athletes should have at least seven to nine hours of sleep a night ⁽¹⁶⁾. This recommendation is far more than the average 6.1 hours per night established in a survey of 15,000 students (18 to 22 years) ⁽⁸¹⁾. If sleep is compromised in any way, the ability to adapt to training will be negatively affected. A study of competitive swimmers through a season showed that slow wave sleep (stages 3 and 4) formed a high percentage of total sleep in the onset (26%) and peak (31%) training periods, but was significantly reduced following a pre-competition taper (16%) ⁽⁷⁴⁾. This suggests that slow wave sleep, which is associated with restoration and recovery, is reduced as the physical demands reduce. Another finding in this study is that the number of body movements during sleep was significantly higher when the swimmers were training high volume, suggesting some sleep disruption ⁽⁷⁴⁾.

Based on the understanding of sleep and how it contributes to recovery and restoration, there is reason to believe that “power naps” during the day will be beneficial for a rugby player. Research has shown that “power naps”, defined as a brief period of daytime sleep lasting less than an hour, improves alertness, productivity and mood, and may contribute to consolidating learning and improved performance of tasks involving visual discrimination ⁽⁵⁰⁾.

Practical guidelines for enhancing sleep, adapted from Jeffreys, (2005) ⁽⁴⁰⁾ are shown below. These guidelines become more relevant with travelling, as maintaining good quality sleep becomes a particular challenge with touring teams, particularly when time zones are crossed.

Table 1: Practical guidelines for enhancing quality of sleep ⁽⁴⁰⁾.

- identify your sleep requirements and try to get this amount daily
- develop a pattern of sleeping and waking times
- practice relaxation techniques before going to bed
- try to avoid worrying about anything before going to bed
- make the bedroom as dark as possible (use a mask if necessary)
- try to maintain a quiet environment (use ear plugs if necessary)
- use a bed that is at least 15 cm longer than the body
- maintain a cool environment within the bedroom
- keep your head cooler than your body
- if you do not fall asleep within 30 minutes, get out and do some relaxation work

- avoid ingesting high-protein meals, caffeine or alcohol in the few hours before going to bed

Non steroidal anti-inflammatory drugs (NSAIDS)

NSAIDS relieve pain and have anti-inflammatory properties ⁽⁴⁷⁾. These properties make them an attractive modality for the treatment of athletes after training and competition to possibly enhance recovery ⁽⁶⁾, and are the most widely used medications for treating muscle injury ⁽⁴⁷⁾. Studies on strains and contusions suggest that the use of NSAIDS can result in a modest inhibition of the initial inflammatory response and the associated symptoms ⁽²⁾. However, the inhibition of the biological steps may cause negative effects later in the healing phase. Many studies have examined the acute affects of NSAIDS on muscle injury and the diverse findings suggest that NSAIDS have a dosage-dependent effect that may also be influenced by the time of administration ⁽¹⁸⁾. Animal studies suggest that whilst NSAIDS may have a short-term positive effect on muscle repair, the long-term effects (four weeks) may be negative and associated with ineffectual or delayed muscle regeneration ⁽⁵⁵⁾. As a follow-up to this study, the effects of anti-inflammatory on satellite cell and fibroblast proliferation were studied in rats in an attempt to identify the mechanism of the delayed regeneration ⁽⁷⁷⁾. The authors found that both satellite cell and fibroblast proliferation were unaffected by the anti-inflammatory treatment and there were no significant differences in myotube or capillary production between treated and control animals. Therefore, the mechanism explaining the findings of Mishra (1995) are not known and are possibly associated with reducing inflammation, a biological process which is part of muscle regeneration ⁽⁶⁷⁾. Such studies have not been performed in humans, and therefore the long-term effects of treatment with NSAIDS in humans are not well understood. It is beyond the scope of this review to go into any more detail so interested readers are therefore referred to other review papers ^(47;82).

Field studies on recovery

It has been mentioned before that one of the reasons for the varying results of many of the studies on recovery is that the laboratory-derived protocols to induce fatigue or muscle damage may lack the specificity of replicating fatigue or muscle damage as a result of training or competition. In an attempt to overcome this deficiency, two studies have been conducted in the “field”.

The first study examined the most effective recovery intervention during a 21-day pre-season soccer training camp ⁽⁷⁵⁾. Twelve 18-year-old elite soccer players were recruited for the study. The recovery modalities that were used in the study were passive, dry aerobic exercises, water aerobic exercises, and electrostimulation. Each intervention strategy lasted for 20 minutes. The performance outcome measures in the study were muscle power (squat jump, countermovement jump, bounce jumping) and 10 m sprinting speed, which were recorded on four occasions, two days apart. The subjective ratings (perceived exertion and muscle pain) were also recorded before, and five hours after, the intervention.

There were no significant effects on recovery intervention in any of the performance measures. Dry aerobic exercise and electrostimulation were slightly more beneficial than water aerobic exercises and passive rest in reducing muscle pain ⁽⁷⁵⁾.

The second field study investigated whether immediate (15 to 20 minutes after the match) post-game recovery strategies enhanced the rate of recovery in Australian football players ⁽²³⁾. The treatments evaluated were stretching, pool walking and contrast temperature. For the stretching protocol, the players had a supervised 15-minute session in which they did gentle static stretching of the legs and back. The stretches were held for 30 seconds and each stretch was repeated two to three times. For the pool walking, protocol subjects underwent 15 minutes of easy walking (moving forwards, backwards and sideways) in the shallow end of a 28°C swimming pool. For the contrast temperature protocol, the players alternated between standing in a hot (45 °C) shower for two minutes, then standing in waist deep icy water (12° C) for one minute. This was repeated until five hot and four cold exposes had been completed. Additional ice was added to ensure that the water remained cold. In addition to these treatments, all players had a normal 25-minute pool session the day after the match. For the control trial the subjects only performed the 25-minute pool session the day after the match. Muscle soreness was rated higher two days after the match compared to the rating before the match in all conditions. There were no differences in the rating of soreness between treatments, including the control. Furthermore, the recovery strategies did not enhance flexibility and muscle power. These results suggest that the additional recovery strategies performed immediately after the match did not enhance recovery any better than just doing the 25 minutes of pool exercise the day after the match.

TRANSLATING RESEARCH ON RECOVERY STRATEGIES INTO PRACTICE

A review of the studies shows clearly that there are varied responses to all the treatments. This can be attributed to the different protocols for inducing fatigue/muscle damage and the outcome measures which are indirect markers of recovery. Many aspects of recovery are very difficult to measure in the laboratory, and the indirect markers of various aspects of recovery lack the required precision to detect small, yet meaningful changes. Where does that leave the practitioner, driven by the desire to have an evidence-based approach to the management of the players? It would be foolish to conclude that recovery strategies do not have an effect, although a strict analysis of the studies might lead one to this conclusion. This approach would fail to acknowledge the anecdotal evidence which has “stood the test of time” and which suggests that there is a role for recovery strategies after training and competition. Whilst the specific details are not known, a logical interpretation of the available knowledge suggests that under certain circumstances there is a role for cryotherapy, active recovery, nutrition, compression garments, massage, stretching and “power” naps as part of a recovery strategy. The studies which have

examined a combination of the strategies together, suggest that “something is better than nothing”, so the details of the protocol should be custom-made based on the circumstances and equipment available to the support staff and team. It has been suggested that the recovery strategies should follow a pattern and become a habit ⁽⁴¹⁾. Prior to embarking on a strategy for recovery, it is important that the rugby players are educated about the process so that they are fully informed about the protocol and assume some personal responsibility. It is important that within a team setting the recovery strategies adopted by the support staff become routine and almost ritualistic ⁽⁴⁰⁾. It is also prudent to individualise the recovery strategies for various players. For example, older players with a history of joint injuries might be handled differently to younger players. Also, players in different positions, who are faced with different physical demands, might also have different strategies after a match. The strategies that are going to be used should be customised for each player, discussed with the player and then implemented in a systematic way. Ambitious strategies that cannot be used when the team is travelling should be avoided, as this may cause psychological problems for the players when they cannot use a procedure to which they are accustomed. It is with this as background that the next section will discuss some practical examples for implementing the different protocols.

PRACTICAL EXAMPLES

The next section will discuss examples of recovery strategies which can be used after training and after a match. Examples are also given for cold-water immersion, contrast water therapy and a pool session. The reader is reminded that these are merely examples, which can be adjusted to suit the circumstances.

Post-training recovery strategies (adapted from Calder, 2004) ⁽¹⁶⁾

Immediately after training

- rehydrate and refuel
- stretch lightly, using both active and short-held static stretches (10 seconds maximum) while the muscles are warm
- walk or move lightly to prevent venous pooling and promote removal of circulating lactate
- check for fluid loss by comparing body mass to body mass before training
- listen to relaxing music on your way home
- wear a compression garment (lower body or upper body depending on the type of training).

When you get home after training

- continue to rehydrate and refuel
- shower as soon as possible
- continue to do some light static stretches in the warm shower
- light self-massage strokes on chest and upper body in the shower
- alternate between heat (30 seconds) and cold (30 seconds) in the shower, repeating three to five times
- within 60 minutes of training have a well-balanced meal, including carbohydrate and protein and continue rehydrating
- use a relaxation technique or music to unwind.

In the evening

- shower, spa or bath to relax muscles
- relax (for example, listen to music, watch TV)
- static stretching, holding the stretch for about 60 seconds
- self massage, especially legs, feet and hips.

Prepare for sleep

- five to 10 minutes before bed, switch off from the day
- use relaxation skills such as visualisation, breathing exercises or music
- get out of bed if you cannot sleep; do not lie awake worrying.

Next morning

- monitor your response to training
- check body mass and record subjective state of fatigue and quality of sleep
- prepare for training.

Post-match recovery strategies (adapted from Jeffreys, 2007) ⁽⁴¹⁾

Within the first five minutes

Rehydrate and refuel. Eat/drink carbohydrate and protein. A recovery sports drink is adequate. Players need to be reminded that thirst is a poor guide to hydration status.

5 to 20 minutes

Cooldown - Move lightly for five to eight minutes, then stretch for eight to 10 minutes.

15 to 20 minutes

Use a hydrotherapy modality, for example contrast showers* or cold bath* (see examples below). Self massage, using predominantly shaking techniques to stimulate the nervous system. The players should continue to hydrate. Body mass can be checked to ensure that hydration is complete.

Within the first 60 minutes

Continue to hydrate. Ingest more food. Carry out a performance review. Start to relax, use music if appropriate. Wear a compression garment.

In the evening

Relax as appropriate, for example read or go to a movie or socialise. Continue to hydrate and refuel.

Prior to bed.

Use relaxation skills to switch off. Follow routine sleep guidelines.

Next day

Active recovery session (i.e. pool session) (see example below).

Contrast shower

Alternate one minute of hot (as hot as tolerable) with 30 seconds of cold (as cold as tolerable). Repeat three times ⁽⁴¹⁾.

Cold bath

Use a temperature of five to 15° centigrade. Immerse for five to seven minutes. Move body parts during the immersion ⁽⁴¹⁾.

Pool session (adapted from Jeffreys, 2005 ⁽⁴⁰⁾)

The goal of a recovery-based pool session is to promote recovery with low-intensity aerobic exercise coupled with stretching activities that utilise all the main joints involved in rugby performance. The structures and the swimming strokes should focus on the full range of movement. The water depth of the swimming pool will determine the degree of weight supported and the amount of ground contact. The intensity of the session should be at a low level, and players who are unable to swim need to reduce the duration of the workout, particularly if the pool is deep. All sessions should last about 20 to 30 minutes.

Sample session

- swim three lengths while alternating the backstroke, breaststroke and front crawl
- walk for three minutes in waist-deep water with a range of upper body activities (for example, sprint action, reaching up, side bends, rotations)
- walk two widths with full high-knee action in shoulder-deep water
- walk two faster widths with high-knee running action in shoulder-deep water
- walk two widths, emphasising ankle action in waist-deep water
- swim two lengths with a sidestroke (alternating side)
- walk two widths sideways with a side lunge action in waist-deep water
- swim three lengths while alternating backstroke, front crawl, and backstroke
- walk two widths, cross oversteps style, in waist-deep water
- walk two widths with a hip circling action in shoulder-deep water (width one forward, width two backwards)
- walk two widths, lunging in waist-deep water, with a variety of lunging combinations
- walk two widths backwards, taking large steps, in waist-deep water
- swim two lengths sidestroke (alternating side)
- walk two widths with a sprinter's paw-back action in waist-deep water
- walk two widths with a high-kick action in waist-deep water

- jog two widths in waist-deep water
- spend five minutes statically stretching key muscles in the water.

AUTHORS' BIOGRAPHY

Mike Lambert is an Associate Professor in the MRC/UCT Research Unit for Exercise Science and Sports Medicine, Sports Science Institute of South Africa, and the Editor-in-Chief of the South African Journal of Sports Medicine.

David van Wyk is a physiotherapist specialising in sports injuries and owns a private sport and orthopaedic practice in Pretoria East. He has worked with many types of sport disciplines and especially rugby at school, club, university and provincial level.

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