

Inflammation, Repair and the Sports Massage Practitioner

Introduction

The process of inflammation and repair is described by Anderson and Hall (1997) as *'Healing of soft tissues is a three-phase process involving acute response, repair and regeneration and remodelling.'*

'Pain is caused by chemicals that are released by the dead and dying cells, acting on the bare nerve ending of pain fibres. It can also be caused by an increase in pressure.'

Evans (1980)

'Swelling takes a few hours to develop. It consists of fluid called the inflammatory exudates. It occurs because the chemicals released by the damaged tissues affect local capillaries and make them more permeable. The inflammatory exudate contains a large number of inflammatory cells and a very high concentration of protein. A network of fibrin formed from fibrogen becomes organised into scar tissue. The resulting scar tissue contracts. In synovial joints, the end result is a thickened contracted joint capsule.

Therefore...a large inflammatory exudate must be prevented if at all possible.' Evans (1980)

'In a strain, bruise or crush, the local network of blood vessels is damaged. Damaged vessels bleed. Oxygenated blood can no longer reach some tissues- so the cells die. When a cell dies, the cell membrane does not remain intact and the contents are released. Extracellular substances including fibrous tissue, elastic fibres and fat. Its architecture is disrupted by the injury. Extravasated blood, blood released from damaged blood vessels consists of cells, platelets and plasma. Within 10 or 15 minutes of an injury the damaged soft tissue contains disrupted extracellular tissues, dead and dying cells which release powerful digestive enzymes, and a variable amount of blood.' Evans

(1980)

Throughout the inflammation process heat and redness evolve *'The general method of healing soft tissue injuries is by fibrous repair.'* Evans (1980)

Stages of Healing

- Stage 1 Bleeding/acute inflammation
- Stage 2 Proliferation and repair phase
- Stage 3 Remodelling

The Acute Response Stage

'The immediate response to injury is the acute inflammatory phase, also known as the reaction phase, which lasts for the first several days following an injury. The characteristics include redness, local heat, swelling, pain and in severe cases loss of function.' Anderson and Hall (1997) Heat and redness is caused *'...by the opening of thousands of tiny local blood vessels, forming an increased vascular bed. The opening of the capillaries is caused by the enzymes that are released by the dead cells.'* Evans (1980) The pain is caused by the *'...chemicals that are released by the dead and dying cells, acting on the bare nerve ending of pain fibres.'* Evans (1980)

'The beginning of the acute phase involves local blood vessel constriction lasting a few seconds to as long as ten minutes. This vasoconstriction curtails loss of blood and initiation of clotting. However the same vasoconstriction can also result in hypoxia and tissue necrosis caused by lack of oxygen in the area. Following the vasoconstriction phase, vasodilation is brought on by the presence of heparin and other chemical mediators. In conjunction with this, there is an increased blood flow to the region, causing swelling. Blood from the broken vessels and damaged local tissues form a haematoma, which in conjunction with necrotic tissue, forms the zone of primary injury.' Anderson and Hall (1997)

'Swelling or edema, occurs as the vascular walls become permeable and increased pressure within the vessels forces plasma out into the interstitial tissues. These processes speed the arrival of several types of specialised cells that ingest dead cells and any foreign material or infectious agents, provide an anticoagulant, dilate the blood vessels and increase blood vessel permeability, and stimulate nerve endings to cause

pain. This chain of chemical activity produces the zone of secondary injury which includes all of the tissues affected by inflammation, edema and hypoxia. ' Anderson and Hall (1997)

'The processes of inflammation may continue for five days or more. The stage of repair begins when nearby cells, previously dormant, begin to actively divide. On the periphery of the dead tissue, macrophages are actively digesting the tissue debris. They are able to work because they can tolerate low levels of oxygen. This process starts about 12 hours after the injury.' Evans (1980)

- Acute inflammatory phase, can last for several days.
- Includes redness, local heat, swelling, pain and in severe cases loss of function caused by opening of blood vessels and nerve exposure
- Local blood vessel constriction lasts a few seconds to as long as ten minutes curtailing loss of blood and initiation of clotting.
- Vasodilation brought on by the presence of heparin and other chemical mediators along with an increased blood flow to the region, causes swelling.
- Blood from the broken vessels and damaged local tissues form a haematoma, which in conjunction with necrotic tissue, forms the zone of primary injury.
- Swelling or edema, occurs due to increased permeability and pressure increases speeding up to transport of specialist cells
- Repair begins when nearby cells, previously dormant, begin to actively divide. Macrophages start digesting the tissue debris about 12 hours after the injury.

The Repair and Regeneration Stage

'Repair and regeneration of injured tissue takes place approximately two days following the injury through the next six to eight weeks, overlapping the later part of the acute inflammation phase. This stage begins when the haematoma is sufficient in diminished size to allow room for the growth of new tissue. Although the skin has the ability to regenerate new skin tissue, the other soft tissues replace damaged cells with scar tissue.' Anderson and Hall (1997) *Over the next three days the cell division*

continues. Cells canalise and from loops, and blood begins to flow round the loops which approach the injured area. Thus all around the injured area there develops a wall of capillary loops filled with blood. ' Evans (1980)

'Healing through scar formation beings with accumulation of excluded fluid containing a large concentration of protein and damaged cellular tissues. This accumulation forms the foundation for a highly vascularised mass of immature connective tissues that include fibroblasts, cells capable of generating collagen.'

Anderson and Hall (1997)

'At about 12-24hours fibrocytes begin to fatten up and move towards the injury.' Evans (1980) *'The fibroblasts begin to produce immature collagen through a process known as fibroplasias'.* Anderson and Hall (1997) *'Soon they start to multiply and by the fifth day they begin to lay down fibrils of collagen. This process cannot occur if there is a serious deficiency of vitamin c.'* Evans (1980)

'By the fourth or fifth day following an injury a weak vascular connective tissue has been produced over the injury. Over the next two to four weeks, this scar tissue increases in tensile strength and decreases in vascularity. As less new collagen is required, the number of fibroblasts at the site is reduced.' Anderson and Hall (1997)

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- Cells canalise and blood begins to approach the injured area. Accumulation of excluded fluid forms the foundation for a highly vascularised mass of immature connective tissues that include fibroblasts, cells capable of generating collagen.
- 12-24hours fibrocytes start fibroplasias and by the fifth day they begin to lay down fibrils of collagen.
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The Remodelling Phase

'The final phase of injury recovery is known as the remodelling phase. This period involves maturation of the newly formed tissue, decreased fibroblast activity, increased organisation of extra cellular matrix and a return to normal histochemical activity. In soft tissue the process begins about three weeks post injury, overlapping the repair and regeneration phase. It continues for a year or more as collagen fibres becomes orientated along the lines of mechanical stress to which the tissue is usually subjected.' Anderson and Hall (1997)

'Muscle fibres are permanent cells that do not reproduce in response to injury or training. However there are reserve cells in the basement membrane of each muscle fibre that are able to regenerate muscle fibre following injury. Severe muscle injury can resulting scarring or the formation of adhesions within the muscle, which inhibits muscle, may regain only about 50% of its pre-injury strength. This factor has major implication toward early return to competition prior to completing a full rehabilitation program. Since tendons and ligaments have few reparative cells, healing of these structures may take more than a year. If these tissues undergo abnormally high tensile stress before scar tissue formation is complete, the newly formed tissues can be elongated. In ligaments this may result joint instability'. Anderson and Hall (1997)

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Application to the Sports Massage Practitioner

'Because scar tissue is fibrous, inelastic and nonvascular, it is less strong and less functional than the original tissues. Scar formation can reduce the structure's tensile strength by as much as 30% compared with pre-injury strength. The development of the scar tissue typically causes the wound to shrink in size, resulting in decreased flexibility of the affected tissues following the injury.' Anderson and Hall (1997) Sports massage practitioners can use their techniques and knowledge to increase flexibility of scar tissue.

'Collagen has the property of shortening when it is fully formed. The contraction continues from the third week to the sixth month.' Evans (1980) Sports massage practitioner's can use their knowledge to explain to clients the importance of this statement and promote stretching routines.

'If the tissue being healed is kept immobile, the resultant repair is weak. Collagen tends to be laid down haphazardly- plentiful but poorly engineered. Fibrous healing is stronger if natural movements are encouraged.' Evans (1980) Practitioners should seek to make their clients aware of the dangers of immobilising joints. Should the collagen fibres be laid down inappropriately and the resultant healing is inappropriate, the practitioner may use friction to re-injure the area and start the process and again.

'Repair is hindered by low serum protein, low levels of vitamin c and poor blood supply.' Evans (1980) Sports massage practitioners can promote a healthy diet while athletes are going through rehabilitation

'With a good understanding of the mechanisms of inflammation and repair, professionals can be sure of the advice and treatment that they offer to patients who have soft tissue injuries.' Evans (1980) In a sport massage practitioner's case, knowledge of an injury will ensure that the client is referred to the best healthcare professional.

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'It is quite possible for as much damage to be done as was caused by the original injury. Healing cannot be accelerated. If we know the basic mechanisms of tissue repair we can promote an optimal healing environment and avoid delayed or poor repair.'

Evans (1980)

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