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Getting Started with Physiotherapy Mini Series

Session 1: Under the Skin of Rehabilitation

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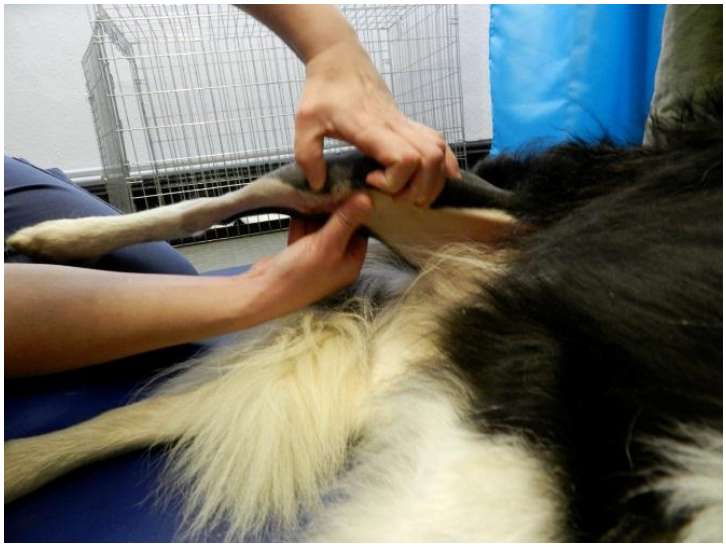

Under the Skin of Veterinary Rehabilitation

INTRODUCTION
Successfully rehabilitation depends on a number of factors, namely:

- Good Clinical Reasoning
- The ability to asses and measure outcomes
- An understanding of the inflammatory response
- An understanding of how tissues respond to injury
- An understanding of muscle biochemistry and exercise physiology

For optimum results a comprehensive pain management strategy coupled with an appropriate and therefore individually tailored rehabilitation programme is desirable.

The role of rehabilitation.
To achieve this goal numerous factors must be considered. Firstly an understanding of the term rehabilitation is required. In essence rehabilitation involves restoration of function in conjunction with appropriate pain management. The two must be addressed simultaneously for a rehabilitation programme to be effective as appropriate pain management without restoring function will not only be difficult to achieve but also difficult to sustain in the long term. Equally it is impossible to restore function without appropriate pain management. Rehabilitation is underpinned by the principles of physiotherapy and relies on moderating the body's own responses for better or for worse. A lack of understanding with respect to these basic principles is likely to make the patient worse, e.g. if a heat pack is applied to inflamed tissue.

Rehabilitation relies on a team approach with veterinary surgeon, physiotherapist, owner and patient working together. Communication and feedback between members of the team is critical and integral to developing good clinical reasoning.

Assessing and measuring outcomes
Assessing and measuring outcomes is again an essential part of clinical reasoning. Since verbal communication cannot be utilised then subtle changes in the patients behaviour become of paramount importance when assessing and measuring outcomes. Data should be objective wherever possible e.g. through goniometric measurements or the use of pedometers and girth tapes but often the clinician has to rely on subjective measures such as:

The ability to perform daily functions such as independent rising, standing to eat food, independent toileting

Improved demeanour and willingness to play

Return to performance

Gait analysis can provide valuable information regarding the efficacy or otherwise of a rehabilitation programme. Any deterioration in lameness should be immediately taken as a red flag. At rest, the patients weight distribution should be assessed. Ideally each forelimb should take 30% of body weight and each hind limb 20%. An attempt to evaluate and score the lameness (usually between 1-5 or 1-10) should also be made.

Joint motion should be evaluated in terms of total range of motion available, the quality of the motion and the nature of the endstop. Joints undergo two distinct types of motion:

Primary or osteokinematic

Secondary or arthrokinematic

Muscle should be evaluated in terms of mass, tone and excitability. Although thigh girth measurements are often advocated as a way of assessing muscle function they are a very crude measure of muscle function.

Body condition scores should be carried out at regular intervals and coupled with neck, chest and girth measurements are often a far more accurate indicator of whether a patient is overweight rather than relying on weight alone. Many patients change their BMI considerably as they tone up and condition without this being reflected in Kilos lost.

The subjective impression of the owner and veterinary surgeon can provide valuable feedback on an individual's progress or lack thereof. With performance animals, the ultimate test is whether they can return to athletic activity and remain sound throughout competition.

Tissue Healing

Tissue injury whether surgical or traumatic initiates a series of cellular and biochemical reactions that aim to bring about tissue healing. The extent of the reaction is generally proportional to the severity of the injury. Three stages of tissue repair are recognised:

- Acute Inflammatory Stage - Immediate post injury - 7days
- Subacute/Reparative Stage - From 6 hours
- Remodelling/Maturation stage - From day 14

These stages are not distinct nor necessarily follow the precise time table set out above.

Acute Inflammatory Phase

In this stage an acute vascular response occurs which results in cell infiltration and activation of the coagulation cascade. A fibrin network is formed which supports the clot and provides the structure for subsequent cellular infiltration. Cytokines released from the clot and changes in vascular permeability stimulates local cellular chemotaxis. At 6 hours post injury, neutrophil numbers start to increase and peak at 2-3 days. These facilitate the process of phagocytosis and wound debridement and minimize the potential for infection. Between 24-48hrs, macrophage numbers peak and a transition from acute inflammation to the reparative phase is seen. Macrophages are critical for wound debridement, phagocytosis, matrix synthesis regulation, cell recruitment and angiogenesis. From the macrophage, cytokines, growth factors and prostaglandins are released and together with the neutrophils they facilitate conditions for transition into the reparative phase.

Reparative phase.

Fibroblast proliferation and migration predominates at this stage. They first start to appear within the wound some 3 days post injury, however collagen synthesis does not start until 2-3 days later and is maximal at 2 weeks. As matrix synthesis of collagen, elastin and proteoglycans increases so does the tensile strength of the fibrin clot.

Endothelial cells adjacent to the wound begin to proliferate and form new capillaries and these migrate in behind the fibroblasts. Proliferation and migration continues until normal oxygen tension is restored. This dense tissue network is normally referred to as granulation tissue.

Remodelling/MaturationPhase

The main feature of the remodelling phase is the deposition and re-orientation of collagen. Collagen fibers should reorientate along the lines of stress and strengthen the repair through cross linkage formation. At this time it is important that collagen deposition and alignment occurs correctly and not randomly in order to facilitate restoration of tensile strength. Although collagen deposition is maximal 2 weeks post injury, tensile strength will continue to increase over a twelve month period. Type I collagen will gradually replace Type 3 and as

proteoglycan concentrations reduce, water content also reduces which facilitates compression of collagen fibers. As the fibers become closer, the surface area for cross linkage formation increases and tensile strength increases.

The rehabilitation programme should assist the timely transition from one stage of tissue healing to the next and within the remodelling phase, ensure that an appropriate level of challenge is applied in the correct plane to ensure organised and functional remodeling.

Factors Which Delay Healing

Severity of the injury

Wound contamination

Extensive Haemorrhage and tissue odema

Systemic disease and altered immune response

Inappropriate inflammatory response

Inadequate pain management

Inappropriate intervention

Overuse of anti-inflammatory therapy

How can rehabilitation help

Primary objective:

- Manage pain
- Restore function

In terms of the inflammatory process this can include:

Minimising cell death

Promoting tissue oxygenation

Promoting appropriate wound debridement and collagen deposition

Minimising adhesion formation

Appropriate techniques may include:

Ice packs

Cryokinetic devices

Hands – massage

Range of motion exercises
Cold Laser

Therapeutic ultrasound

Rehabilitation and tissue remodelling

Rehabilitation requires the application of controlled challenges to enhance the tissue's healing response. The ultimate aim is to improve the strength and function of the tissue long term. In order to achieve this the challenges must be controlled and applicable to the stage of tissue healing. Sustained inappropriate challenge may delay healing or permanently impair function

Tissue	Effects of immobilization	Effects of mechanical loading and remodelling
Bone	Decreased bone mineral content within 3 weeks Decreased bone mineral density within 3 weeks Decreased cross-sectional area Decreased stress to failure for cancellous bone Decreased mechanical properties Inhibition of longitudinal growth Decreased osteoblast numbers Increased apoptosis of osteocytes	After immobilization, efforts to increase bone mineral content and bone mineral density require greater than normal levels of activity Benefits are lost if activity is terminated early Mechanical properties are restored within 11-16 weeks of exericse
Cartilage and joint	Articular cartilage degeneration and resorbtion Increased collagen crosslinks and stiffness within periarticular soft tissues Synovial membrane atrophy Intra-articular fibrofatty proliferation, adhesions and obliteration of joint cavity increased joint contracture, irreparable damage to hyaline cartilage, eventual ankylosis	Cartilage preservation, but questionable recovery of cartilage thickness after remobilization Maintained extensibility of periarticular tissues Facilitation of healing Decreased odema in intra articular and periarticular tissues

Tissue	Effects of immobilization	Effects of mechanical loading and remodelling
Muscle	Decreased cross-sectional area, muscle atrophy Altered sarcoplasmic reticulum Decreased calcium uptake in slow twitch fibres Decreased neuromuscular control and voluntary recruitment Decreased force production, shortened twitch duration, decreased peak force Immobilization effects greater within slow twitch fibres Non-weight bearing induces greater atrophy than does immobilization with weightbearing	Recovery of contractile properties in 60-90 days Recovery of muscle mass takes longer than 90 days Intermittent standing weight bearing attenuates fiber atrophy and partially restores peak isometric force Increased cross sectional area and muscle fibre numbers Increased oxidative capacity and fatty acid utilization Decreased reliance on carbohydrate metabolism Increased fatigue resistance Increased neuromuscular control and voluntary recruitment
Ligament and tendon	Decreased maximum load to failure Decreased energy stored prior to failure Increased collagen fibre crosslinking and random fibre orientation Decreased structural integrity at insertion sites Insertion sites remain weaker for a longer duration than the ligament proper	Increased collagen fibre diameter Conversion of Type III to Type I collagen Increased linear orientation of fibers Minimal stress can maintain 80%-90% of baseline mechanical properties Mechanical deficits are not completely reversed even after 12 months of rehabilitation

Exercise Physiology and its effect on skeletal muscle

It is easy to forget that for the sick or injured patient even short bursts of simple activity such as maintaining sternal recumbency might present a significant challenge.

Skeletal muscle is excitable, extensible and elastic. It is primarily composed of two types of muscle fibre, namely Type I or slow twitch and Type II of fast twitch. Type II muscle fibres can further be subdivided into Type IIa and IIb.

Energy is required to sustain basal muscle tone through maintenance of ion gradients. ATP is also required to maintain normal excitability of nerve and muscle cells through the action of the Sodium/Potassium pump. Energy is derived in the form of ATP either though aerobic or anaerobic means. The ability to exercise aerobically confers a significant athletic advantage on the individual.

Type of metabolism	Aerobic	Anaerobic
Location	Mitochondria	Cytosol
Molecules Employed	Oxygen, fat,protein, glucose	Carbohydrate in the form of intracellular glycogen
When	Low to moderate exercise,lasting more than a few minutes	High intensity activity, at the start of activity
Type of muscle fibre	Type1, Type 11a	Type 11a
ATP production	High	Low

Adaptations to training

Adaptations to training include:

Increased capillary density

Increased mitochondiral volume

Reduced capillary diffusion index

Increased activity of enzymes associated with aerobic metabolism

Increased muscle buffering capacity

Increased muscle glycogen content

Increased muscle fibre diameter and some alteration in muscle fibre ratios