

Animal Physiotherapy

Assessment, Treatment and Rehabilitation of Animals



Edited by Catherine McGowan, Lesley Goff & Narelle Stubbs



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1

Introduction

Catherine McGowan

The aim of this book is to provide physiotherapists and interested others with a broad base of information on aspects of animal physiotherapy. It begins with essential applied background information on animal behaviour, nutrition, biomechanics and exercise physiology.

Following this are three chapters focusing on the assessment of the musculoskeletal and neurological systems in animals from both a veterinary and physiotherapy perspective. The next section reviews physiotherapy techniques, drawing from both the human and animal literature in their discussion. The final two chapters apply this information to an evidence-based clinical reasoning model describing the physiotherapy approaches to treatment and rehabilitation of animals, giving case examples.

Physiotherapy is an established, independent profession with an excellent reputation for evidence-based practice. In the medical field, physiotherapists form an essential part of musculoskeletal, neurological and cardiorespiratory care from paediatrics to geriatrics and sports medicine. Physiotherapy research has led human medical advancement in areas such as back and pelvic pain, whiplash and women's health. The positive perception of physiotherapy in the human sphere, together with an increased awareness of options and expertise available for animals has resulted in a demand for physiotherapy for animals.

Animal physiotherapy is an emerging profession, representing qualified human physiotherapists who are using their skills on animals. Physiotherapists provide a functional assessment to identify pain or loss of function caused by a physical injury, disorder or disability and they use techniques to reduce pain, improve movement and restore normal muscle control for better motor performance and function. Physiotherapists can provide equivalent levels of care and follow-up treatment for their animal patients as they can for people. In small animal surgery the demand for postoperative physiotherapy has paralleled the increase in surgical options for small animal patients. Elite equine athletes and their riders now access a team of professionals including the veterinarian–animal-physiotherapist team. More and more people prefer to opt for treatments where they can see progressive results, professional teamwork and high levels of care and expertise.

Interestingly, despite the very real need for physiotherapy in animals, up until very recently there has been a lack of postgraduate-trained professionals for the application of physiotherapy to animals.

The issues are simple:

- Physiotherapy is not in veterinary curricula and is not commonly a part of veterinary medicine or surgery.
- Physiotherapy and physical therapy are protected by The Physiotherapists Registration Act (Australia)¹ or equivalent.
- Veterinary diagnosis (pathoanatomical) and treatment (i.e. medical or surgical) in animals are protected by The Veterinary Surgeon's Act (Australia)² or equivalent.

The solution the professions have come to in many countries is also simple and relies on both veterinarians and physiotherapists continuing to practise within, and be regulated by their own profession. Physiotherapists, when working with animal patients, work on referral from a veterinary surgeon rather than autonomous first contact practice as with human patients. This new area of expertise has been embraced both by physiotherapy professional bodies and registration boards, as well as educational institutions. Leading universities in the United Kingdom and Australia have led the way in providing postgraduate university-based training for physiotherapists to specialise in treating animals. Formalised, special interest groups (SIGs) of animal physiotherapy have been established by many physiotherapy professional groups around the world; for example, the Animal Physiotherapy Group of the Australian Physiotherapy Association is one of only 12 special interest groups of the Australian Physiotherapists Association. Other SIGs have been formed in the UK, Netherlands, South Africa, Canada, USA, Sweden, Finland, Spain and other European countries. This has predominantly been occurring in the last one to two decades and numbers in these special interest groups are rapidly rising.

¹ <http://www.legislation.qld.gov.au/LEGISLTN/CURRENT/P/PhysiothRegA01.pdf>

² <http://www.legislation.qld.gov.au/LEGISLTN/CURRENT/V/VetSurgA36.pdf>

2 Animal Physiotherapy

This textbook is based on the teachings in the physiotherapy programmes in Australia and the UK. It is not a handbook of physiotherapy, rather a text aiming to cover the science behind animal or veterinary physiotherapy. For

animal physiotherapists it will be a valuable reference text in their profession. For veterinarians and others who work with animals, it will be a valuable insight into the profession of physiotherapy and what it can achieve.

2

Applied animal behaviour: assessment, pain and aggression

Daniel Mills, Suzanne Millman and Emily Levine

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2.1 Introduction

Understanding animal behaviour is important for animal physiotherapists both to ensure safe handling of animals who may be in pain and therefore aggressive, and to facilitate a more complete and accurate assessment of the animal's pain, which may be important both diagnostically and therapeutically. Often, we only know that an animal is in need of physiotherapeutic intervention because of his or her behaviour. The behaviour may be overt such as a non-weight bearing lameness or more subtle such as a decline in activity or in the vigour of the activity. In either case, the challenge may be to distinguish pain from a pain-free loss of physical function or mobility.

In horses, pain may manifest as training problems or poor performance. If we wish to address the cause of this behaviour (rather than simply contain the problem), then we need to be aware of the full range of potential factors, which interact with and influence behaviour. This involves at least some appreciation of the many diverse branches of zoology as well as various branches of psychology, veterinary medicine, animal management and nutrition. This might seem a bit daunting, and is why it is often most effective to work as part of a multidisciplinary team, with everyone respecting each other's expertise. For example, Martin and colleagues (1998) report that by using an interdisciplinary team approach on stallions, that presented with breeding problems owing to primary musculoskeletal or neurological problems, 92% could successfully return to long-term breeding.

The animal physiotherapist is a critical member of the multidisciplinary team for animal health and well-being and will become an even more important member of the team as awareness of the role of chronic pain in many behaviour problems increases. As some pain models highlight, there are underlying neurophysiological pathways involved in both the sensory-discriminative components of

pain (i.e. nature of the aversive stimulus and bodily location) as well as the affective-motivational components of pain (i.e. emotional and behavioural response to pain or anticipation of pain) (Craig 1999).

Therefore, the animal physiotherapist should be aware that some patients might need behavioural therapy in order to treat the affective-motivational aspects of pain before the sensory-discriminative component of pain can be addressed. Although animal physiotherapists are not expected to be behaviour specialists and should not be tempted to practise beyond their own knowledge base and skill, a solid grounding and appreciation of the subject are essential to avoid putting themselves and others at risk of harm and to avoid threatening the well-being of their patients. Animal physiotherapists, who have moved into the field from the human discipline, may have a substantial awareness of the psychological effects of chronic pain, but it is important to appreciate the biological and cognitive differences that exist between humans and non-human animals and not assume that what applies to one species necessarily applies to another. Anthropomorphic tendencies may lead to superficial and/or inaccurate assessments with consequently inappropriate treatment. It is therefore important to always be thorough and assess all of the available information objectively in the light of the biology of the species being considered.

In this chapter we begin with an initial guide to the principles that underpin the assessment of animal behaviour. Behaviour, like physiology, is a mechanism and expression of an animal's attempt to adapt to or cope with his or her environment. To survive and be successful within an evolutionary context, animals must be as efficient as possible, since those able to adapt most appropriately will out-compete those less efficient. Accordingly, the behaviour of a given individual should be viewed as an attempt by the animal to behave most appropriately in the current circumstances given previous experience.

There are therefore three major considerations to the evaluation of an animal's behaviour:

- the nature of the individual concerned;
- his or her previous experience; and
- his or her current circumstance.

Only when all of these are appreciated can we fully understand why an animal is behaving in a particular way. After discussing these three considerations, we move on to discuss the concepts of pain, pain assessment, pain management and aggression within a context that is relevant to the animal physiotherapist.

2.1.1 Assessment of animal behaviour

As previously mentioned, there are three major principles that should be included in one's thought process when trying to evaluate an animal's behaviour.

1. The nature of the individual is influenced genetically at many levels.
2. Previous experience has both general and specific effects on behaviour.
3. The current circumstance of the individual refers to both its general motivational state and the internal and external factors, which cause this state to dominate the animal's behaviour.

Genetic influence

The first consideration is that the nature of the individual is influenced genetically at many levels. Species-typical behaviour refers to those activities that define a dog as a dog and a horse as a horse. One species is a predator-scavenger and the other a prey species. In order to reduce the risk of predation, natural selection is likely to have favoured a greater capacity to mask, where possible, the signs of pain, injury and disease in horses compared with dogs. In other words, by the time a horse appears overtly sick or lame its welfare is often already seriously compromised. Similarly, during treatment and rehabilitation, a horse might be expected to stop showing these signs before it has fully recovered, increasing the risk of relapse if the animal is returned to an inappropriate level of work too rapidly or too abruptly. The animal physiotherapist plays an essential role in ensuring that this does not happen and that the build-up to full fitness is appropriately managed.

It is also essential to be aware of the normal behaviours of the species in order to appreciate if something is genuinely disease related; for example, an inexperienced owner might mistakenly think that their cat is in pain because she is intermittently meowing with great intensity and rolling around on the floor, when in fact this is normal behaviour for a female cat in oestrus. It is not possible to go into detail here about species-typical behaviour patterns of companion animals, so the reader is referred to the many texts available on the different species and breeds.

Although genetics influence typical behaviours of species, such that there is great difference between species, there is also enormous variation within a species (i.e. between breeds) and within a breed itself. So, although some generalisations about breeds may be easy to argue, such as selection favouring greater stoicism in breeds which are used to fight live game (e.g. terriers), it is important to appreciate that genetic variation of certain traits may be greater within a breed than between breeds. Expressions of individual variation arise as a result of the interaction of different genetic and environmental factors throughout life, but during development such interaction may particularly shape the temperament of the individual (Scott & Fuller 1965) and its appraisal of the event (Weisenberg 1977). So whilst it is important to appreciate breed characteristics, they should not be rigid points of reference. One of the characteristics for which there is varied individual responses which is particularly relevant to the animal physiotherapist, is an individual's response to pain. This is perhaps one of the main challenges faced by those trying to devise generic guides to the recognition of pain in animals. It is perhaps not surprising that in many cases the owner is believed to be the best assessor, since they recognise what is normal for that individual, and how it behaved before any change arose (Wiseman *et al.* 2001).

It is therefore important that records of behaviour relevant to the individual are kept and that each subject acts as its own point of reference when trying to evaluate response to treatment. This kind of record keeping is essential for the physiotherapist to be able to identify therapeutic progress and/or identify early signs of relapse, which may not have been noticed by the owner or the veterinarian. In addition, if progress reports show a steady improvement and then the physiotherapist identifies subtle changes during therapy, such as the dog resisting a bit more, or seemingly more tense or painful than normal for that individual animal, this should be relayed to the referring or supervising veterinarian.

Previous experience

The second major consideration is that previous experience has both general and specific effects on behaviour. It has already been mentioned that a large part of temperament arises as a result of interactions between the genetics of the patient and its early experience, and temperament might be considered a general factor reflecting the animal's behavioural predispositions in a wide variety of environments. For example, dogs that are poorly socialised are likely to be more fearful and aggressive towards items that may be unfamiliar to them (Appleby *et al.* 2002), but these unfamiliar items may be very 'normal', such as a man wearing a hat or facial hair. Specific effects include individual learned responses, such as the particular response shown by the fearful dog described before. If a fearful dog growls at someone who approaches him or her and the

person (understandably) leaves the dog alone as a result, then the dog will learn that growling helps achieve his or her goal and may use this strategy in other contexts. The sensible thing to do is to recognise the early signs of unease such as turning the head away, yawning and blinking and avoid an unnecessary escalation to overt aggression (Shepherd 2002), assess why this has occurred and take appropriate remedial steps. Within a clinical context it is obviously important to be able to differentiate an animal that is generally (i.e. temperamentally) fearful and does not like being approached by strangers, from one which is perhaps protecting a painful body region (specific response). Both may threaten aggressively when initially approached for assessment, but in only one of them is the behaviour related to a potential physiotherapeutic problem. Similarly, horses are often generally predisposed to behave fearfully towards any novelty they encounter, which might be a new individual or an unfamiliar form of handling and this does not necessarily mean they are in pain. However, if the animal is not handled sensitively on this first encounter it will create stronger aversion in future similar circumstances, which may be reflected in a general irritability and specific aversive behaviour. There are many horses that become protective of a particular region of the body as a result of insensitive handling, when that region has been irritated by another process. For example, harsh handling to put a bridle on when a horse has a mouth or ear irritation may soon produce a head-shy animal. In these situations the animal learns that the safest response is to always avoid handling even when there is no longer any pain, perhaps because the handling is likely to be rough and unpleasant. With time this will also lead to more general changes in irritability. The inappropriate use of a twitch or painful restraint like a lip chain, or physical punishment at any time, might also result in head shyness or protective avoidance in relation to any body part. It is also important to identify and acknowledge the possible role of any condition in the animal's history, which might result in general irritability, including episodes of low grade general pain, such as sub-clinical rhabdomyolysis in the horse, and any history of a change in temperament in adult life should be viewed with a concern for the possible role of underlying disease. As already mentioned, more than one factor may of course occur concurrently, and temperamentally fearful individuals who are being treated for painful lesions may require considerable training beforehand to allow effective handling. The animal physiotherapist should not be afraid to point this out to the owner, following an initial assessment, and refer to a qualified behaviourist if necessary; although the procedures involved in desensitising animals to being approached are relatively straightforward and easily learned (Box 2.1). This procedure can be applied to overtly aggressive animals and any that are tense in response to initial examination. A relaxed animal is both easier and safer to examine.

A brief behavioural history will help determine how the animal might be expected to behave and should review a range of external and internal factors that can influence behaviour (see Askew 2002, for details of more extensive behavioural history taking). External factors include the general management and any specific triggers of aggression or known fears of the animal. Internal factors include the signalment of the individual (age, breed, sex, etc.), which might be of relevance.

In some cases, animals learn particular behaviours as a result of sustaining an injury. These learned behaviours range from aggression in order to prevent contact to the injured area to attention-gaining behaviours such as sham lameness. The latter can be quite problematic in some dogs, but can usually be recognised by its disappearance in the absence of the owner when the animal is relaxed. Horses, on the other hand are far less likely to produce such vestigial behaviour since the expression of lameness for psychological reasons is likely to have been heavily selected against in evolution as it is likely to result in a greater risk of predation. However, previous poor experience during, for example, shoeing, may manifest as very poor behaviour on the picking up of hind limbs, which may need to be differentiated from a hind limb pain process. Or, a horse may learn behaviour to avoid being saddled or ridden resulting in it appearing 'cold backed' or demonstrating adverse reaction to the tightening of the girth.

Current circumstance

The third consideration is that the current circumstance of the individual refers to both its motivational state and the internal and external factors which cause this state to dominate the animal's behaviour. Motivational states may be thought of as general predispositions for behaviour towards a certain goal. For example, an animal that is hungry is motivated to seek and consume food. Low blood sugar and the presence of food are internal and external factors, which would encourage the animal to start eating in such circumstances, but the presence of a predator might intervene and cause a switch in motivation towards self-preservation. It may be that a given goal (self-preservation) can be achieved in many different ways behaviourally (fight, flight or freeze response) or that a given behaviour (biting) may be associated with achieving different goals (eating or self-preservation). Therefore, there is not necessarily a perfect relationship between behaviour and motivational state. When trying to understand behaviour, it is important to be able to justify the inferred motivational state on the basis of the available information and not assume that one is necessarily associated with the other. An animal's priorities and motivational predispositions may also vary due to seasonal factors, since both bitches and mares may become more irritable around the time they become sexually receptive.

It is also important to recognise that behaviour does not happen independently of environment, and animals are

Box 2.1 Desensitisation and counter-conditioning a dog that is fearful of an approaching stranger (including the physiotherapist)

1. Identify the 'safe distance'

The safe distance is the distance at which the stranger can stand in front of the animal (but not looking directly at the animal) without causing the animal to show any behavioural signs of anxiety, fear, or aggression. Common behavioural signs shown by dogs that are anxious include yawning, lip licking, lifting a paw and panting. In addition, body postures such as ear and tail position can provide information about the animal's underlying emotional state.

2. Counter-condition the dog at the safe distance

As long as the animal is showing no signs of anxiety, fear, or aggression, it is possible to change his or her perception of the stranger by associating the stranger's presence with something positive (e.g. a highly valued treat that the animal does not normally receive). If the animal is not food motivated, toys or attention/praise provided by the owner may be used. Once the animal is willing to take the treats, make sure the owner asks him or her to 'sit' or 'down' before getting any more treats in the presence of a stranger.

3. Desensitise and counter-condition to the stranger getting closer

Once the dog is willing to take treats in a 'sit' or 'down' position in the presence of the stranger, the next stage may be started. The stranger may take one small step closer to the animal and the animal's reaction should be carefully observed. It is expected that the animal may now show some signs of anxiety. The animal needs time to learn that the stranger getting closer is not associated with anything negative. It is important *not* to punish any anxious or aggressive behaviour at this stage. The animal may be distracted with a command and treat. The owner may show the treat (or toy) but it should not be given until he or she sits. Once the animal sits the reward is given (counter-conditioning). *It is important that the stranger should not be making direct eye contact with the dog or raising arms up, as both of these can be seen as threatening gestures.*

4. Small steps forwards

Step 3 should be with the stranger getting steadily closer to the dog, without the dog showing any sign of anxiety or fear. It is important that very small steps are made and the progress is made at the dog's pace. Too often these exercises are done too fast and the dog is not given a sufficient amount of time to learn. For some dogs it may be possible to do this relatively swiftly; however, for others several sessions attending to the behavioural issues may have to be scheduled before actually doing any physiotherapy work. Particular attention should be paid at getting to within 1–2 metres of the dog, as this is when the dog's personal space is being entered.

5. Make the stranger a source of good things

Before taking the final steps, the stranger should offer a highly valued reward, which can be rolled to the dog at a comfortable distance. The dog should start making the association that not only is the stranger nothing to be afraid of but also that the stranger has something positive to offer. This should make the animal willing to approach the stranger. (It is better to allow a nervous animal to do the approaching than being approached.) *When the stranger is giving the treat, he/she should kneel down and look away as he or she is rolling the treat at first as this is a less threatening posture. The stranger can then progress to a more normal position as long as the dog is comfortable.*

6. Stranger approaches dog

Once the dog has learned to approach the stranger, the stranger can try to approach the dog. He/she should show the dog that he/she has a treat to offer, give a relevant obedience command and pay attention to the dog's body language. If, at any time, the dog appears anxious, earlier stages of the programme need repeating first.

7. Stranger touching the dog

It is obviously important for a physiotherapist to not only get within the dog's personal space, but also to be able to touch it (another reactive point). It is important to realise that just because the dog may be okay with a stranger being in close physical proximity does not mean that he/she will be okay with being touched. In order to desensitise and counter-condition the dog to being touched, the same principles are involved as described above, with everybody part that is to be examined or manipulated. Always give the command first and then the treat, as this helps to relax the animal.

8. 'Over-learning'

Once this is successfully done, the final steps should be repeated so the dog 'over learns' that this stranger is a source of pleasure.

rarely aggressive without good reason. Although it might be obvious why a horse attempts to kick you when you touch its painful leg, defensive behaviours may be inadvertently triggered in a number of other contexts, which, if they are not recognised, can result in serious injury.

For example, entering into the animal's personal space or moving into a blind spot are all commonly perceived as potentially dangerous situations and so trigger defensive behaviour. If the animal cannot retreat, it will often resort to an attempt to repel the perceived threat as it has few other options. Defensive behaviour, because it is associated with self-preservation in the face of a perceived threat, will quickly dominate behavioural output regardless of the potential alternatives or competing motivations. It is therefore essential to make sure that your presence is recognised and acknowledged by the patient before intervening too closely. A horse is likely to kick out or a dog snap if it is spooked for any reason, regardless of any pain it may or may not be experiencing. For humans, the natural way to greet each other in a friendly way is directly, while making eye contact, but this can appear very threatening to dogs and horses. This is another example of the danger of anthropomorphism when dealing with non-human animals. Sudden movement of the arms vertically, such as to put your arms around a horse or to withdraw them from a sniffing dog, and looming over an animal can provoke a fear response, and so it is important to consider carefully your own initial approach behaviour towards the patient. It is generally advisable to encourage and allow the patient to approach you in the first instance rather than the reverse, and give them time to investigate so they can establish for themselves that you are not a direct threat. If an animal has made this appraisal of the situation, it is far more likely to be tolerant of you than one that is still uncertain when initial physical assessment is undertaken (Chapter 8). Initial contact should also be structured similarly to give the animal confidence. Just as insensitive handling can provoke aggression, so can indecisive handling. If the therapist is nervous for any reason, then there will be changes in behaviour, which the animal will detect. The animal is most likely to interpret the uncertainty in the behaviour of others in its environment as a sign of potential danger and not realise that nervousness may be caused by the physiotherapist's fear of the animal itself. The patient may then, at best, try to avoid contact with the physiotherapist and at worst seek to repel the physiotherapist by whatever means it deems appropriate! Unfortunately, if the cause is not recognised, the interaction becomes a self-fulfilling prophecy for the handler, which impacts on future attempts at interaction. Initial contact before commencing any palpation or treatment techniques should therefore help to reassure both parties. The physiotherapist may utilise soft tissue techniques such as stroking, kneading, skin rolling, and/or circular finger/hand motion in a region away from the region of pain or lesion. The physiotherapist must adjust their 'touch' to the

behaviour accordingly, making sure hand pressure is not ticklish but definite using a mild to moderate depth of pressure and where possible, preferably with both hands.

Understanding some of these basic tenets that influence how an animal will behave will help the physiotherapist to make a more accurate and thorough assessment of the patient's behaviour. The main reason why an understanding of behaviour is so important to the animal physiotherapist is because many patients may be influenced by any pain associated with their medical condition and the associated physiotherapy treatment and may respond aggressively. Therefore, the next section of this chapter will discuss various aspects of pain and aggression.

2.2 Pain

The International Association for the Study of Pain defines pain as 'an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage' (Paul-Murphy *et al.* 2004). Pain is a potent negative affective state that focuses an animal's attention and biases its behaviour.

One of the problems with assessing pain in animals is that pain can only be measured indirectly; while humans can self-assess their levels of pain and verbally report pain scores, the subjective experiences of animals are particularly difficult to assess. An animal in pain will withdraw from the source of the insult if it can be identified, protect the area affected both through immobilisation and active defensive aggression and may communicate the pain to others through changes in facial expression, body postures and vocalisations. By contrast, health and happiness are identifiable by an open and relaxed posture, facial expressions of contentment and production of chemicals that are associated with pleasure, such as endorphins.

The ability to recognise and to respond to painful stimuli has evolved to protect individuals against tissue damage and provides information to safeguard against dangerous or threatening stimuli in the future (Nesse & Williams 1994). Pain may be associated with suffering at many different levels, depending on both the circumstance and the cognitive ability of the animal concerned. At its simplest it may be a temporary negative state, which guides the animal's withdrawal from a noxious stimulus. A variety of animals may be able to anticipate pain and generate feelings of anxiety when faced with a predictably painful stimulus and will take avoidance action as appropriate. This will cause the activation of the hypothalamic-pituitary axis and behaviourally might include threatening behaviour or attempts at escape (fight or flight response). The intensity of the response is usually directly related to the intensity of the perceived threat. It is important to realise that the perceived threat arises from a combination of factors (e.g. previous experience, sensitivity to pain, emotional state)

without a single cause; and as a result of the accumulation of several risk factors within the three levels of behaviour assessment discussed in the previous section. Therefore, simply approaching the animal may not seem threatening from the person's perspective, but very threatening from the animal's perspective. It is also thought by some that certain species such as horses and dogs may be capable of a pain phobia; this involves the generation of an ungraded and extreme reaction in response to even the most low-grade sign of any pain. While pain phobias may exist, they should be distinguished from extreme responses that have been conditioned and allodynia. This is an exaggerated pain response to normally innocuous stimuli, and although mechanisms are unknown, allodynia probably arises in the structures of the limbic system of the brain, such as the amygdala and periaqueductal grey, which are associated with the processing of emotions (Craig 1999). Animals showing an extreme response for whatever reason are potentially very dangerous and require specialist intervention in consultation with a veterinary behaviourist. An even higher cognitive level of response to pain is pain empathy, i.e. responding to the pain of others and many owners may report that their pets are capable of this, although it remains to be demonstrated scientifically.

Pain is also often classified according to its temporal pattern and this is associated with different psychological impacts and behavioural tendencies, which might be apparent in a range of species. In humans, individual painful episodes may be referred to as peracute pain episodes and are behaviourally characterised by vocalisation and withdrawal of the painful area. Acute pain refers to episodes that last up to about 3 weeks and are associated with fear and anxiety, reduced activity and care-soliciting behaviour. Subacute pain lasts for between 3 and 12 weeks and is characterised by oscillating bouts of activity and inactivity, signs of frustration (including irritability) and the development of coping strategies associated with longer term adaptation to the pain. Early signs of depression may also become apparent at this time. Beyond this, the pain may be considered chronic and depression, together with other passive coping strategies, is more likely. Often subacute episodes may occur against a background of chronic pain in individuals with longstanding musculoskeletal lesions, and in the horse this may present as periodic bucking set against a 'loss of spirit'. While the changes over time may partly reflect natural adaptive developmental changes to an unresolved lesion, it is important to recognise that learning will also occur as a consequence of the responses made over time and affect the response that is shown.

2.2.1 Mechanisms of pain

Pain sensation is a dynamic process with highly organised neural and chemical circuits (Watkins & Maier 2000). Sensory information is transmitted to the central nervous system from afferent neurones, a process termed 'nocicep-

tion'. These incoming pain signals are processed within the dorsal horn of the spinal cord and result in reflexive actions, such as withdrawal from the source of injury. Reflexive actions facilitate a rapid response, while, concurrently, pain signals are transmitted to the brain to produce an emotional response and memory. The motivational responses to pain, which provoke a goal-directed action of avoidance, results from activity within the hypothalamus, periaqueductal grey area and thalamus, whereas the anterior cingulate cortex evaluates the hedonistic value of pleasure and of pain (Sewards & Sewards 2002). Within the mid-brain, the pain system interacts closely with the fear system at several locations, such as within the amygdala and periaqueductal grey (Panksepp 1998), facilitating consolidation of memories that will be important for recognising potentially dangerous stimuli in the future and developing flexible responses of avoidance.

Pain signals are suppressed or amplified by coordinated neural connections between the brain and spinal cord (Watkins & Maier 2000). During sympathetic nervous system activation or the fight-flight-freeze response when animals may be scared, pain sensations are suppressed – a phenomenon referred to as 'stress-induced analgesia'. Conversely, conditioned safety signals can increase pain sensation, through the release of peptides, such as cholecystekinin, in the cerebrospinal fluid, which can suppress pain control mechanisms, including opioid analgesic drugs, acupuncture and placebo effects. The regulation of pain sensation is discussed further below.

During the fight-flight-freeze response, suppression of pain serves an adaptive function, allowing the animal to escape from or resolve the conflict. The 'gate control theory' suggests that sensory inputs of pain are modulated through ascending and descending pathways in the central nervous system (Melzak & Wall 1965). Descending neural pathways potentiate or attenuate pain signals influencing the amount of neurotransmitter released by the incoming neurones or by changing the sensitivity of the ascending nerves in the spinal cord to these neurotransmitters. Analgesia is not just a response to pain but can also be classically conditioned to avoid painful sensation. When stimuli are perceived that are predictive of pain from past experience, descending signals may be sent to inhibit pain sensation (anti-nociception). Conversely, safety signals can result in the release of peptides such as cholecystekinin in the cerebrospinal fluid surrounding the spinal cord, which suppress pain-controlling mechanisms (anti-analgesia). Thus administering painful physiotherapeutic interventions to an animal in the presence of a safety signal (most often the owner) may actually exacerbate the pain of the procedure.

Hyperalgesia refers to exaggerated pain states with increased responsiveness to signals within the spinal cord (Watkins & Maier 2000). The pain threshold is lowered, and sensory nerve fibres release large quantities of

neurotransmitter in the spinal cord in response to afferent signals. It may arise for many reasons, but chronic compression of pain fibres within the spinal cord due to a back lesion are a common cause in animals. In these cases the pain may be sensed as arising from the point of compression or the area served by the nerve. Neuropathic pain refers to a pain that arises as a result of nerve damage and can be extremely painful. Causalgia is a particular form of hyperalgesia associated with nerve damage (neuropathy) particularly stretching (Gregory 2004). It is sensed as a burning pain following trauma local to the nerve. It is therefore an important differential in cases presenting with attempts at self-mutilation. A history of trauma to the region and exacerbation by warmth with remission in response to cooling of the affected area may help identify the problem, which often resolves within a year. Infection may also result in hyperalgesia, both with and without neuropathy. For example, it has been suggested that herpes virus infection of the trigeminal nerve in horses may be a cause of headshaking, a severe, involuntary tossing of the head by the ridden horse (see Mills *et al.* 2005 for a review of this and other repetitive behaviours in the horse). It is also known that two types of glial cells, astrocytes and microglial cells, that act as immune cells within the central nervous system, specifically recognise and bind to bacteria and viruses, and when activated they release nitric oxide, prostaglandins, and pro-inflammatory cytokines, such as interleukin-1 and tumour necrosis factor. These chemicals excite neurones and are key mediators within the spinal cord of exaggerated pain states (Milligan *et al.* 2003; Weiseler-Frank *et al.* 2005).

Phantom-limb pain is a common sequel to limb amputation in humans and usually develops several days following surgery. It is reportedly more common in individuals who experienced pain in the limb before amputation (Codere *et al.* 1986). An animal experiencing phantom limb pain might be expected to present with self-mutilation of the wound site and this must be differentiated from direct wound site problems such as irritation from sutures; alternatively, the animal may show a more general pain response.

Pain sensation may be suppressed by competing motivational systems. For example, in poultry it has been found that expression of feeding and of pre-laying behaviour produces a degree of analgesia (Gentle & Corr 1995). While there are no scientific reports known to the authors of this being tested experimentally in a physiotherapeutic context, this is often applied in practice by feeding or distracting an animal during examination. It would also be interesting to examine the effects of enriched environments on rehabilitation, especially in horses that often undergo box rest in very barren environments. The processing of pain is also affected by background mood. For example, pain reports are lower in human subjects when stimuli are paired with positive or pleasant odours (Marchand & Arsenault 2002). Therapeutically, the creation of a relaxing environment for treatment is therefore to be advised for many reasons.

Suppression of pain also occurs during and following intense aerobic activity, and is likely mediated by endogenous opioids. This may be one of the benefits of hydrotherapy. However, not all interventions producing analgesia are necessarily positive and it is important to be aware that when an animal is faced with inescapable aversion, as might occur as a result of intense restraint during painful manipulation, learned helplessness may result (Seligman & Maier 1967). This results in emotional biasing of behaviour towards passivity, active inhibition of skeletal muscles and opioid-mediated analgesia (Maier 1993). Thus, if an animal initially struggles and is then overzealously restrained, it may be harder to identify the source of pain.

2.2.2 Assessing pain in animals

Pain assessment involves the integration of measurements of behaviour and physiology together with knowledge of the bi-directional mechanisms that control pain. Morton and Griffiths (1985) proposed a framework for the recognition of pain, distress and discomfort based on a combined assessment of appearance, food and water intake, behaviour, cardiovascular functioning, digestive system activity and neurological/musculoskeletal signs. This provides a useful framework, but, the correlation between physiological measures such as heart rate, respiratory rate and pupil dilation versus subjective pain scores may be poor (Holton *et al.* 1998) and there is a need for greater validation of pain scales. These are beginning to appear in the literature in relation to specific problems, for example Wiseman-Orr *et al.* (2004) have developed and validated a scale for the assessment of chronic pain from chronic degenerative joint disease in dogs, and others, which are similarly rigorous in their development, are likely to be published in the near future. It is now being increasingly well recognised that as pain is a subjective experience, animals vary enormously in their individual responses and so it is essential that assessment is focused around an assessor who is very familiar with the animal's normal behaviour, such as the owner or caretaker/groom.

Given the enormous range of individual factors that can affect pain perception in a given context discussed above, it should be apparent that it is difficult to accurately assess the pain of an individual without a thorough history, including baseline assessments of behaviour and temperament (Sanford *et al.* 1986). In addition, given the differences that inevitably exist between assessors (Mathews 2000), it is also important that assessment is repeated by the same assessor on all possible occasions, in order to reduce this possible source of error. Laboratory methods to assess pain in domesticated animals might be thought of as being more objective and are increasingly sophisticated (Table 2.1). However, these techniques are not necessarily practical for clinical situations, and further research is needed to determine how these measures may be integrated for a more complete assessment and how to interpret conflicting results.

Table 2.1 A selection of laboratory techniques used to assess pain responses in animals

Technique	Parameter measured	Species (reference)
Algometer	Pressure sensitivity	Equine (Haussler & Erb 2006)
Sonogram	Frequency and pitch of distress vocalisations	Swine (Weary <i>et al.</i> 1996) Cattle (Watts & Stookey 2000)
Thermal threshold assay	Foot lift response	Cattle (Machado-Filho <i>et al.</i> 1998; Veissier <i>et al.</i> 2000)
Operant tasks	Self-administration of analgesia	Chicken (Danbury <i>et al.</i> 2000)

Clinical assessment generally relies on evaluating a range of behavioural signs of pain (Table 2.2), and these may be integrated into subjective scoring systems. Verbal rating scales involve qualitative description of behaviour observed, and simple quantitative scales involve subjectively rating pain as *No Pain, Mild, Moderate or Severe*. These assessment protocols have been criticised not only for the large variation between different observers, but also for their lack of sensitivity (Mathews 2000). Numeric scales rating pain between 0 and 10, and visual analogue scales marking pain on a ruler on which 0 = No Pain Present and 100 = Worst Pain Imaginable, are generally considered to provide better sensitivity and reliability (Mathews 2000; Paul-Murphy *et al.* 2004). However, the validity of these systems may be questioned owing to a lack of transparency regarding pain parameters considered by observers, and these are weighted in the final score. As Mathews (2000) points out, observers may reliably weight vocalisations heavily because of ease of measurement and anthropomorphism, but these vocalisations may not correlate well with pain experiences since dogs occasionally vocalise while under anaesthesia when pain is presumably prevented. In a survey of equine practitioners, respondents cited personal experience to be the most important source of information about pain in horses, but respondents varied in how they rated pain associated with various procedures (Price *et al.* 2002).

Although the science of valid pain assessment in animals is in its infancy, this does not negate the responsibility of those that work with animals in pain to institute and apply pain assessment criteria within their practice. Given current knowledge, the physiotherapist should at the very least use some form of pain scale that both the owner and the physiotherapist can complete and keep a behavioural diary of therapy sessions to monitor pain responses. Should there be any doubt that a certain condition is painful, it is good practice to assume that what would be painful for a person is painful for that animal (IRAC 1985).

Further information on the recognition and assessment of animal pain is hosted by the University of Edinburgh at: www.vet.ed.ac.uk/animalpain/, and readers may wish to refer to this for further detail of some of the principles that have been discussed in this chapter.

Table 2.2 A selection of behavioural signs of acute pain (Morton and Griffiths 1985; Sanford *et al.* 1986; Molony & Kent 1997; Dobromylskyj *et al.* 2000; Mathews 2000; Mills *et al.* 2002; Hansen 2003; Price *et al.* 2003; Rietmann *et al.* 2004)

Source of pain	Behavioural response
General responses	Lethargy Reduction in grooming Depression Reduced feeding, drinking Protection of painful site Vocalisation (dog: whining, growling; equine: groaning) Aggression Hanging tail Ear position (equine: pinned ears) Facial expression (canine: furrowed brow; equine: clenched jaw, wrinkled muzzle) Restlessness/weight shifting between all limbs
Limb	Avoidance or reduction in weight bearing Abnormal gait Head bobbing during locomotion Rubbing, licking wound site Weight shifting away from painful limb
Abdominal/Spinal/ Visceral pain	Tucked up posture Glancing or nosing abdomen Abnormal stance, stretching of hind limbs Restlessness Sweating Trembling
Head pain	Headshaking and facial rubbing Head shyness Grimacing Signs often exacerbated by exercise Intranasal pain Snorting and sneezing Turning of the upper lip Intra-oral pain Reduced appetite and/or dropping of food being chewed Teeth grinding

2.2.3 Management of pain

It is sometimes suggested that if pain is an evolved response to minimise damage to injured tissues, analgesia may not be in the animal's interest. However, Flecknell (2000) points out that in situations where we take responsibility for an animal's injury and provide therapeutic treatment, the evolved pain responses are not necessary and more benefit is derived from providing pain relief. Pain slows recovery from surgery and the associated reductions in feeding, drinking, and self-maintenance behaviours cause increased risks of mortality resulting from dehydration and catabolism. Furthermore, analgesics reduce, but do not eliminate pain sensations. Pain management is therefore in the animal's interest.

In addition to obvious pharmaceutical and physical interventions designed to reduce pain, social intervention may be important, especially grooming and other physical

contact therapies. Social support has been shown to reduce physical pain in humans (Eisenberger & Lieberman 2004), and it seems reasonable to suggest that a similar mechanism may be in place in social domestic species. If an animal is in pain as a result of a non-infectious agent, unless there is risk of bullying, there may be little need to isolate the individual, as the stress imposed by social isolation of an animal such as a horse can have very detrimental effects. In addition, from what has already been discussed above, encouraging other behaviours though a stimulating environment matched to the animal's comfortable mobility, may also be considered as part of a pain management and rehabilitation strategy. Diet may also play a role, not only in encouraging another motivational system, but also more directly and is discussed in the next chapter.

2.3 Aggression

Aggression has been referred to several times in this chapter in relation to pain and an understanding of aggression is important for those working with animals in distress. Aggression is not a unitary phenomenon. Clearly the emotion underlying predatory behaviour (sometimes referred to as *predatory aggression*) is quite different from that underlying defence of a resource from conspecific (*affective aggression*), or bouts of 'apparent aggression' arising during acts of play. These three types of activity belong to functionally different behavioural systems and are directed towards very different goals. While they might all (in the case of carnivores in particular) share the potential to cause harm to another individual, it is potentially confusing to link them with each other through the use of the term aggression in their description. Injury that arises during play might be a result of *aggressive* play, but that does not make it a form of aggression, it is first and foremost a form of play.

The further subdivision of affective aggression is of questionable value. It may be divided according to descriptive context, such as 'owner directed aggression', or according to motivation/mechanism, such as 'defensive aggression'. Both have their advantages and disadvantages. For example, contextual labels have both the advantage and disadvantage of not implying anything about motivation and so might be quite reliable terms, but do not link with underlying mechanism or treatments aimed at addressing the cause in a reliable way. This is something that is frequently overlooked in the literature. The main problem with motivational descriptions is knowing with confidence what the precise motivation is.

Aggressive displays should be distinguished from aggressivity, which can be used to describe both the mood and temperamental trait relating to the propensity to show aggression when environmental circumstances dictate it might be used. Animals may become temperamentally more aggressive if they are in chronic pain. This may resolve once the pain is eliminated, but the animal may also learn to

use displays of aggression in a wider range of contexts as a result of this episode. In this situation specialist assistance should be sought to help resolve the problem.

The expression of aggression depends on a range of underlying external contingencies as well as internal predispositions. Historically, psychology has focused on the external factors producing aggression and these are well summarised by Archer (1976).

Namely aggression *may* occur when:

1. A territorial boundary is crossed.
2. The personal space is entered.
3. The body is touched.
4. The animal is faced by uncertainty/novelty in the environment.
5. An expected reward is absent or withdrawn.
6. An expected reward is reduced.
7. Behaviour is frustrated from being executed – this includes the application of intended punishment to an animal that is already nervous.

These situations may all occur when a physiotherapist is trying to treat a patient and are perceived at a time of potentially aversive change (i.e. an unpleasant near-future). A number of individual factors determine whether overt aggression rather than freezing, flight or some form of appeasement is offered.

These include the following:

1. *The emotional state (mood) of the animal* – Fearfulness in the absence of an easy route for escape, greatly increases the probability that aggression will be used, but more generally there are a wide range of factors which can increase irritability (an enhanced predisposition towards aggression), including low grade chronic/subclinical pain. This is particularly worth investigating when the pattern is not entirely predictable, and probably underestimated in veterinary practice.
2. *The animal's appraisal of the situation* – This depends on the animal's perceived ability to win the contest, the value of any resource that is being disputed and the expected cost of defence. Learning can be very important in this, as an owner who always gives way to their dog will be perceived both as an inferior competitor, and as an individual who does not put up much of a fight. It is perhaps for this reason that clinicians and therapists are often able to handle an animal in a way that would be impossible for the owner. This can obviously be to the physiotherapist's advantage, but must also be taken into consideration when making recommendations for treatment. Owners may not only lack the skill to undertake certain procedures in the home, but also the necessary authority.

While handouts, such as those by Landsberg *et al.* (2001), can be very useful in the management of such problems, they should not be used without understanding the fundamental nature of the problem faced. Therapists should also

consider the potential need for specialist intervention in handling aggression, and ensure the risks to others of injury from an aggressive episode are minimised.

This involves:

- Informing owners of their responsibility to prevent injury to others.
- Advising owners to avoid situations that are likely to exacerbate the problem. This may include identifiable trigger stimuli, such as approach towards a particularly painful area, uncertainty in handling the animal, frustrating or fearful situations.
- The animal should not be approached when it has no opportunity to retreat.
- If it is safe to do so, the owner should be encouraged to muzzle-train an aggressive dog away from arousing or dangerous environments. A basket muzzle is preferable to a nylon one, as it allows the dog to pant and drink but not bite while it is on. The most common problem with muzzles is that they are only used when the dog is already showing aggression and will resent restraint. So training should begin away from distractions and associated with rewards placed in the muzzle. Once trained, the dog should be muzzled before the problem arises, i.e. before arriving at the treatment centre.

2.4 Conclusion

For behaviours caused by underlying medical factors for which physiotherapy is needed, the physiotherapist should have an understanding of how pain or the anticipation of pain can affect an animal's behaviour and how this behaviour may compromise therapeutic progress. The science of identifying and assessing pain is in its infancy and much more research is needed to answer many unanswered questions. Yet with a basic and sound understanding of behaviour and the factors that influence behaviour; acknowledgement that individuals differ in both their physical and emotional response to pain; and the tools that can be used to assess pain by both the owner and the physiotherapist throughout therapy, the physiotherapist can be confident that appropriate steps are being taken to maximise his or her patient health and well-being.

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3

Applied animal nutrition

Linda M. Fleeman and Elizabeth Owens

- 3.1 Small animal nutrition
- 3.2 Applied equine nutrition
- References

3.1 Small animal nutrition

3.1.1 Introduction and basic nutritional considerations for the clinical animal physiotherapist

Domestication of dogs first occurred 12 000 to 14 000 years ago (Clutton-Brock 1985). The ancestors of dogs and the ancestors of human beings came together to form mixed-species communities, which had an evolutionary advantage over both wolf-only and human-only communities (Newby 1997). Since that time, dogs have played a variety of roles in human communities. They have acted as scavengers of waste, detection and alarm systems against intruders, crucial members of hunting parties, herders of livestock and companions or pets. Domestication of cats occurred some time later, with the earliest evidence dating 4000 years ago (Malek 1993). Cats were likely welcomed as predators of the rodents that infested human grain stores, yet their role did not remain confined to that of an opportunistic predator. Unlike their non-feline predatory contemporaries, cats became pets as well (Newby 1997).

All over the world, there has been a population shift from rural to urban communities and the majority of people now live in cities. This has resulted in a corresponding shift in the roles that dogs and cats play within the community. There has been a change from a peripheral role as scavengers of waste and hunters of vermin to a much more central role as companions and valued 'family members'. More than ever, the relationship between people and their pets is one of interdependence (Becker 2002). Pet ownership is recognised as a non-human form of social support that helps to reduce stress (Bridger 1976) and improve health (Friedmann & Thomas 1995; Dembicki & Anderson 1996). This is associated with an increased responsibility of dog and cat owners for the health care and nutrition of their pets.

Owners' attitudes to feeding dogs and cats

Feeding is a major part of the human–animal bond. For many dog and cat owners, feeding is one of the most important

methods of demonstrating a caring and loving relationship with the pet. Their pet's nutrition is very important to owners and they seek information from friends, family, breeders, pet shops, the Internet, pet food manufacturers and veterinary clinics. Owners may see their pet as a reflection of their own identity. They often see their pets as reflections of their canine and feline wild ancestors. Owners may extrapolate from what they know of human nutrition, which is often irrelevant to dogs and cats.

Everyone understands food, and pet owners generally like to discuss nutrition. It is likely to be a safe and comforting topic when owners are confronted with the news of a serious health problem in their pet. They may prefer to focus on nutrition rather than more time-consuming or costly therapies.

'Performance' of domestic dogs and cats

For the majority of domestic dogs and cats, 'performance' simply means that they must be healthy, long-lived pets. Some dogs and cats are required to perform as breeding or show animals, although for many this is a function that is secondary to their role as pets and companions. There are many working dogs, including guard dogs, dogs that work with livestock, dogs that are trained to assist people with disabilities and dogs trained for service in government organisations such as the armed forces and the quarantine inspection service. Dogs also perform as competitive athletes in a wide variety of high intensity and endurance activities (Figure 3.1).

Comparison with wild species

Wild dogs and cats are not long-lived; the average life span is 5–7 years. Every individual endeavours to breed and all must 'work' to obtain food. In contrast, most pet dogs and cats do not breed, have all of their food provided by their owners, and live considerably longer lives (Kraft 1998). Dogs are omnivores, which means that their diet naturally consists of foods of both animal and plant origin. Wild dogs will hunt and kill prey, in addition to seeking carrion, plant material, especially grass, and other foods (Stahler 2005).



Figure 3.1 Dog in a flyball competition (Syke, courtesy of Natalie Kirkwood).

They tend to feed in a pack, which means that the dominance hierarchy influences food intake. Cats are carnivores and their diet consists entirely of small prey. They tend to be solitary hunters and usually do not share food.

3.1.2 Nutritional requirements of dogs and cats and evaluation of diets

Differences in nutritional needs

Unlike dogs, cats are carnivores and so are not adapted to a diet that includes substantial quantities of food of plant origin. The molars at the back of a dog's mouth have a small grinding surface, whereas this is not the case for cats. Dogs can taste sweet substances and so can distinguish ripe from unripe fruit, whereas cats lack the ability to identify sweet taste (Li *et al.* 2005).

Dog food should never be fed to cats

Cats require a higher proportion of protein in their diet than dogs and have additional amino acid, fatty acid, and vitamin requirements (Kirk *et al.* 2000; Buffington *et al.* 2004). Food that is formulated for dogs is deficient in a number of nutrients if fed to cats, particularly protein, taurine, arginine, B vitamins, vitamin A, linoleic acid and arachidonic acid.

General nutritional requirements and assessment of commercial feeds

Pet dogs and cats have become almost exclusively dependent on their owners for food. It is now much less acceptable for dogs to supplement their nutrition by scavenging and for cats to supplement by hunting. Owners have the responsibility of providing all of the nutrients for their pet over its entire lifespan. Consequently, pet dogs and cats have become more vulnerable to diseases of nutritional origin and great care must be taken to provide them with complete and balanced nutrition during all stages of their lives (Debraekeleer *et al.* 2000; Kirk *et al.* 2000). If attention is given to ensure that pet dogs and cats are fed a complete

and balanced diet throughout their lives, performance and longevity will be optimised. A *complete* food for dogs or cats is one that contains all of the required nutrients in adequate quantities. A *balanced* diet requires that all of the nutrients are present in the correct proportions.

The Association of American Feed Control Officials (AAFCO) is internationally recognised as the organisation that sets the most rigorous and comprehensive standard for pet food labelling claims (Roudebush *et al.* 2000b). AAFCO dog and cat nutrient profiles define the minimum requirements of all nutrients for each species and life stage, as well as the maximum requirements of selected nutrients. The labelling term 'nutritionally complete' indicates that all required nutrients are present in the food in adequate quantities, while 'complete and balanced' indicates that all required nutrients are present in the proper proportions as well as in adequate quantities. The pet food labelling term 'formulated to AAFCO standards' indicates that the food has been formulated to meet the AAFCO nutrient profile for that species and life stage. It is important to realise that if a label claims that a dog food is 'complete and balanced for adult maintenance', then that food will not necessarily be complete and balanced for dogs at other life stages, such as growing pups, breeding bitches or performance dogs.

If a diet contains all required nutrients, it does not automatically mean that those nutrients are available to the animal when the diet is consumed and digested. Pet food formulations that meet AAFCO standards can be additionally tested by AAFCO digestibility feeding trials. This is the preferred method for substantiating a nutritional adequacy claim. To meet these requirements, diets must be tested by long feeding trials where animals at the required life stages are fed only the test food and water while being monitored for nutrition-related disorders. When a dog or cat food meets this high standard, the following statement may be included on the product label: 'Animal feeding tests using AAFCO procedures substantiate that (Name of Product) provides complete and balanced nutrition for (life stage) of dogs/cats'. In certain circumstances, pet foods with very similar formulations may be considered comparable to those that have been tested by AAFCO digestibility feeding trials and this statement may appear on the label: '(Name of Product) provides complete and balanced nutrition for (life stage) and is comparable in nutritional adequacy to a product which has been substantiated using AAFCO feeding tests'.

In some countries, it is a requirement that pet food manufacturers include a brief nutrient profile on their product labels, which outlines percentages of crude protein, crude fat, crude fibre and moisture. This might take the form of a 'guaranteed analysis' of the *minimum* percentages for crude protein and crude fat and *maximum* for crude fibre and moisture. Importantly, this represents the 'worst case scenario' for levels of nutrients and does not reflect the exact or typical amounts of these nutrients. Alternatively,

a 'typical analysis' might be supplied, indicating the *average* of the nutrient levels calculated from several samples. Actual nutrient levels might be within 10% (above or below) the stated 'typical' level. Knowledge of the moisture content of pet foods is important for calculation of the dry matter content of individual nutrients. A common mistake is to confuse the percentage of crude protein, fat, or fibre listed on the nutrient profile on the product label with percent dry matter (%DM) content for those nutrients.

Most pet food product labels also contain an 'ingredient list'. For products that meet AAFCO standards, all ingredients are listed in descending order by weight, and ingredient names conform to the AAFCO name (e.g. poultry by-product meal, corn gluten meal, powdered cellulose) or are identified by the common name (e.g. beef, lamb, chicken). These rules do not necessarily apply to ingredient lists of products that are not formulated to AAFCO standards.

As label information on the nutritive value of a pet food product may be quite limited, it is particularly useful if the full contact details for the manufacturer are supplied on the label. Ideally, a local telephone number should be included for each country in which the food is sold. This allows consumers to easily request additional information on the nutritive value of the product from the manufacturer.

Lists of nutritional requirements for dogs and cats refer to the lower limit of adequacy for each nutrient. Diets that contain nutrient concentrations that are close to the 'recommended' level should be considered marginally adequate.

Specific life stage and performance dietary requirements

Commercial dog and cat foods are available in different formulations to suit the varying nutritional requirements of individual animals. The majority of products are designed for long-term maintenance of adult dogs or cats. Specific formulations are also widely available for growing puppies or kittens. Many of these growth formulations are suitable for feeding bitches or queens during the last part of gestation and during lactation.

Dogs that are involved in high intensity or endurance exercise require food with greater calorie density than sedentary dogs. Fat provides more than twice the calories provided by either protein or carbohydrate, therefore the most effective means of significantly increasing the calorie density of dog food is to increase the dietary fat content (Toll & Reynolds 2000). Commercial products formulated for working and performance dogs typically have higher dietary fat content than other adult maintenance diets. 'Part-time' or 'weekend' canine athletes usually do not require these specific formulations, and increased calorie requirements can usually be met by increased consumption of the usual diet (Buffington *et al.* 2004). The products that are specifically designed for working and performance dogs provide benefit for dogs that habitually have a high activity

level, and can promote obesity in more sedentary dogs. For competitive sprint and endurance canine athletes, optimal nutrition is required for dogs to perform at their peak. Dietary recommendations are specific for sprint (Hill *et al.* 2000) and endurance athletes (Reynolds *et al.* 1999; Toll & Reynolds 2000), although general recommendations for both activities include high digestibility, low bulk, higher fat and protein, and lower carbohydrate dietary content, compared with an adult maintenance diet.

Home-prepared food

Food for pet dogs and cats is often prepared in the home. Ingredients include meat, fish, eggs, milk, other foods suitable for the human table, bones and offal. Home-prepared food might be either raw or cooked when fed. The meals may be carefully formulated to provide a high-quality diet for dogs or cats, or may comprise 'left-overs' of the owner's meals. In some cases, home-prepared food provides 100% of the animal's diet, while in others it supplements a commercial diet.

One of the most common problems with feeding home-prepared food to dogs and cats is that the diet provided is not complete and balanced (Freeman & Michel 2001; Streiff *et al.* 2002). The result is that animals fed these foods are at risk of nutrient deficiencies or excesses. This is a particular concern for growing puppies and kittens. Typical home-prepared meals for pet dogs and cats contain excessive protein, but are deficient in calcium, other minerals and vitamins (Remillard *et al.* 2000). They often contain excessive quantities of meat, which has low calcium and high phosphorus and protein content.

It is especially difficult to provide an adequate balance of vitamins and minerals in home-prepared dog and cat diets because there are no complete and balanced, veterinary, vitamin-mineral supplements. Some human vitamin-mineral supplements are suitable but must not be cooked, heated, or stored with the food as this may cause destruction of the vitamins (Remillard *et al.* 2000). In addition, home-prepared diets will usually also require a specific calcium supplement, such as calcium carbonate. Information on correct supplement types and doses, as well as recipes for home-prepared dog and cat diets, are available in nutrition textbooks and manuals (Remillard *et al.* 2000; Buffington *et al.* 2004).

Even when owners take great care to research and formulate a complete and balanced home-prepared diet, there is a tendency for their recipe to slowly change over time, resulting in a gradual decline of the nutritional value of the pet's diet. Owners often decide to make ingredient substitutions, or they may omit ingredients because of personal preferences or convenience. A common error made by owners who prepare food for their pets is to eliminate the vitamin-mineral supplement because of its inconvenience, expense, or a failure to understand its importance (Remillard *et al.* 2000).

Bacterial contamination is a common problem when food is fed raw or incompletely cooked (Joffe & Schlesinger 2002). This not only presents a health risk for dogs and cats, but also for the people who formulate and mix the raw food. Exposure to bacteria and other potential pathogens can occur from handling raw meat or from cross-contamination of dishes and utensils used to prepare the food. It is therefore strongly recommended that only cooked food be fed to dogs and cats.

Several problems are associated with feeding bones to dogs and cats. Although the actual incidence of these problems is unknown, oesophageal and intestinal obstruction, colonic impaction, gastrointestinal perforation, gastroenteritis, and fractured teeth are all recognised complications to feeding bones to dogs and cats (Robinson & Gorrell 1997). Bones contain a very high amount of fat, particularly in the marrow cavity, and so contribute to an increased risk of obesity. It is a common perception that feeding raw bones to dogs and cats will provide some protection against the development of periodontal disease; however, comparison of dental disease in pet cats eating commercial foods and in free-roaming, feral cats found that a 'natural' diet based on live prey does not protect cats from developing periodontal disease (Clarke & Cameron 1998).

If home-prepared food is to comprise 100% of a dog's or cat's diet for an extended period of time, owners require knowledge, motivation, additional financial resources and careful, consistent attention to recipe detail to ensure a consistent, balanced intake of nutrients.

Health problems associated with nutritionally inadequate diets

Health problems that might result when diets with poor mineral and vitamin balance are fed include: secondary nutritional hyperparathyroidism (dietary calcium deficiency) (Buffington *et al.* 2004); dermatoses (dietary zinc deficiency) (Roudebush *et al.* 2000a); developmental orthopaedic conditions (incorrect dietary proportions of calcium, phosphorus, and/or vitamin D) (Hazelwinkel *et al.* 1991; Hazelwinkel & Tryfonidou 2002); hypercalcaemia (excess dietary vitamin D) (Mellanby *et al.* 2005); excessive periosteal bone formation (vitamin A toxicity associated with excess feeding of liver or cod liver oil) (Goldman 1992); pansteatitis (vitamin E deficiency associated with feeding all-fish diets) (Niza *et al.* 2003); and neurological signs due to thiamine deficiency (all-fish diets and diets containing sulphite preservatives) (Studdert & Labuc 1991; Koutinas *et al.* 1993; Singh *et al.* 2005). The lens and the retina are particularly sensitive to nutritional problems and eye lesions may be seen in dogs and cats fed inadequate diets.

3.1.3 Obesity in dogs and cats

Obesity is a major health issue for people and their pets. The World Health Organization has classified human obesity as

an epidemic. In the USA, 65% of adults are overweight or obese. In Europe, one in 13 deaths can be attributed to people being overweight (Banegas *et al.* 2003), a figure that is six times higher than the death toll due to road accidents. People are getting fatter at an alarming rate all over the world (Mokdad *et al.* 2003). One of the major reasons for this is the trend towards a more sedentary lifestyle. Not surprisingly, similar trends are affecting our pets.

Obesity is now the most common form of malnutrition in dogs and cats. The lifestyles of our pets are becoming more sedentary, resulting in a high incidence of excessive weight gain. It is estimated that 25–35% of dogs and cats are overweight (Edney & Smith 1986; Lund *et al.* 1999; Robertson, 2003; Armstrong *et al.* 2004) and, in some parts of the world, the prevalence has risen to 40% in middle-aged pets (Lund *et al.* 1999; Armstrong *et al.* 2004). Excess body weight has negative effects on health and evidence is mounting for strong associations between body fat content and numerous small animal diseases. Overweight dogs have an increased risk of osteoarthritis (Edney & Smith 1986; Kealy *et al.* 1997; Kealy *et al.* 2002), pancreatitis (Hess *et al.* 1999), bladder (Glickman *et al.* 1989) and mammary cancer (Alenza *et al.* 1998). Fat cats are prone to diabetes mellitus (Rand *et al.* 2004), musculoskeletal problems and lameness (Scarlett & Donoghue 1998; Craig 2001), non-allergic dermatitis (Scarlett & Donoghue 1998), lower urine tract disease (Willeberg 1984; Blanco & Bartges 2001) and hepatic lipidosis (Center 2005).

Of particular note is the strong association between excess body fat and osteoarthritis in dogs. A 14-year life-span study that evaluated the effect of calorie restriction on development and progression of hip osteoarthritis was performed in dogs predisposed to develop hip dysplasia (Kealy *et al.* 1997; Kealy *et al.* 2000; Kealy *et al.* 2002). The results provide strong evidence of an association between obesity and osteoarthritis in dogs. The dogs in the calorie-restriction group had significantly less hip dysplasia, significantly less osteoarthritis of the hips and of other joints, lived for a significantly longer period before requiring medical treatment for the osteoarthritis, and lived significantly longer than the dogs in the control-fed group. The results are summarised in Table 3.1.

Dogs maintained in lean body condition have reduced prevalence and severity of osteoarthritis. The effect of weight loss in overweight dogs with existing radiographic evidence of osteoarthritis has also been found to be associated with significant reduction of lameness (Impellizzeri *et al.* 2000). This indicates that improvement of the clinical signs of osteoarthritis can be achieved with weight loss alone.

Prevention of obesity is the most important goal of feeding dogs and cats. Owners require clear guidelines on how to feed their pets to achieve and maintain ideal, lean body condition. They need to understand how to calculate just how much food is enough food.

Table 3.1 Evidence of an association between obesity and osteoarthritis in dogs

	Control-fed dogs	Restricted-fed dogs
Dogs with radiographic evidence of hip dysplasia at 5 years of age	50%	13%
Dogs with >2 joints affected at 8 years of age	86%	24%
Age at which 50% of dogs required treatment of osteoarthritis	10.3 years	13.3 years
Median lifespan	11.2 years	13.0 years

Calculation of the ideal daily calorie requirement

Unfortunately, it is not possible to easily predict the daily calorie requirement for an individual dog or cat. The Daily Energy Requirement (DER) is a measure of the daily calorie requirement for maintenance of body weight and condition, and is known to vary widely among individual animals.

Basal or resting energy requirements are similar for all animals and are not markedly influenced by age, breed, gender, neuter status, or activity level. The Resting Energy Requirement (RER) is the amount of calories required by an animal at rest in a thermoneutral environment and does not support any exercise, growth, or reproduction. It is a function of metabolic body weight and can be reliably calculated using the formula:

$$\text{RER (Kcal/day)} = 70 \times (\text{BW}_{\text{kg}})^{0.75}$$

However, because RER does not take into account age, breed, gender, neuter status, or daily activity, it does not give a reliable indication of the actual calorie requirement of an individual animal. Formulae used to calculate DER give a rough estimate based on body weight of the calorie requirements of an *average* individual (Thatcher *et al.* 2000). In reality, graphical representation of the actual DER for a population of animals with the same body weight produces a wide, bell-shaped curve. Approximately 50% of the animals will have daily calorie requirements below the average DER and 50% will have requirements above the average DER. If the population has a tendency to be more sedentary than average, then more than half of the animals will have calorie requirements below the calculated DER.

Thus, it can be seen that it is very difficult to accurately determine the daily calorie needs of individual dogs and cats. It is common for one animal to have a daily calorie requirement that is 100% more than another animal of the same body weight, breed, gender, and neuter status. If the two animals are fed exactly the same amount of food, the latter will become obese while the former will remain lean. This can be quite confusing for pet owners.

The following is a rough guide to calculation of DER for

$$\text{Canine DER} = 1.0\text{--}1.8 \times \text{RER}$$

$$\text{Intact adult} = 1.8 \times \text{RER}$$

$$\begin{aligned} \text{Neutered adult} &= 1.6 \times \text{RER} \leftarrow \text{DER (Kcal/day)} \\ &= 110 \times (\text{BW}_{\text{kg}})^{0.75} \end{aligned}$$

$$\text{Obese prone} = 1.4 \times \text{RER}$$

$$\text{Weight loss} = 1.0 \times \text{RER}^*$$

(*RER at ideal weight)

For active and working dogs, the following calculations can be used:

$$\text{Light work} = 2 \times \text{RER}$$

$$\text{Moderate work} = 3 \times \text{RER}$$

$$\text{Heavy work} = 4\text{--}8 \times \text{RER}$$

For growing dogs:

$$\text{Weaning to 4 months of age} = 3 \times \text{RER}$$

$$\text{Four months to adult size} = 2 \times \text{RER}$$

For bitches during pregnancy and lactation:

$$\text{First 42 days of gestation} = 1.8 \times \text{RER}$$

$$\text{Last 21 days of gestation} = 3 \times \text{RER}$$

$$\text{Lactation} = 4\text{--}8 \times \text{RER}$$

Feeding recommendations in textbooks and on pet food packages indicate the calorie requirement for the *average* dog or cat. They are a rough guide only and are not a reliable indication of individual dog or cat calorie requirements. As there is so much variation among individuals, feeding recommendations represent either a marked under- or over-recommendation of the calorie requirement for the majority of animals. Thus, feeding recommendations provide a useful starting point, but it is essential that the calorie intake is regularly reassessed and adjusted to ensure that the animal is not being underfed or overfed. The key is to feed dogs and cats to achieve an ideal, lean body condition, rather than to follow label guidelines strictly.

To summarise: how much food is enough food for dogs and cats? Owners should begin by following the label recommendations for dog and cat food, yet should understand that adjustment of the quantity of food fed will usually be required.

Important nutritional factors for large-breed puppies

The goal of feeding large- and giant-breed puppies is to achieve moderate calorie restriction and sub-maximal growth. Feeding controlled meals, rather than allowing free choice or ad libitum feeding, is the best method to achieve this. Low protein diets should be avoided because adequate protein is important during growth.

Calorie intake should be based on need. Owners should evaluate growth and body condition and decrease calorie intake as growth decreases (usually at 4–6 months). Rapidly growing, large- and giant-breed dogs have a very steep growth curve and their food requirements can change

dramatically in a short time. These puppies should be weighed, their body condition evaluated, and their daily feeding amount adjusted at least every 2 weeks.

Large- and giant-breed puppies are more susceptible to developmental orthopaedic disease than small breeds, particularly when there is rapid weight gain, or dietary calcium levels are either too low or too high (Hazelwinkel *et al.* 1991; Richardson and Toll 1997; Richardson *et al.* 2000; Hazelwinkel & Tryfonidou 2002). Developmental orthopaedic disease includes a diverse group of musculoskeletal disorders that occur in growing animals. Hip dysplasia and osteochondrosis make up the overwhelming majority of diseases in this group. Large- and giant-breed dogs are most at risk and the most critical period for development of these diseases occurs during the growth phase, before epiphyseal closure. Excessive dietary intake of calories causes rapid weight gain during growth and stress on developing bones. Excessive dietary calcium causes hypercalcaionism and influences bone calcium metabolism. In the face of adequate levels of calcium in the food, the absolute level of calcium, rather than an imbalance in the calcium:phosphorus (Ca:P) ratio, influences skeletal development.

Low dietary calcium is usually associated with feeding homemade foods containing meat. All meats are very low in calcium and have a Ca:P ratio in the range of 1:15 to 1:20. High dietary calcium is usually associated with feeding vitamin or mineral supplements to dogs that are fed complete and balanced foods. *If a nutritionally adequate food is being fed, supplementation is contraindicated.* Particular care must be taken to avoid supplements containing calcium, phosphorus, vitamin D, and vitamin A.

If a complete and balanced diet is fed to meet the pet's energy requirement (i.e. total calorie requirement), all requirements for non-energy nutrients will automatically be met.

Evaluation of body condition

It is important to always record both body weight and body condition. Body weight does not correlate well with either body composition or body condition. Body composition refers to percentage of body fat and lean body mass. Body condition is a subjective evaluation of body composition.

Body condition scoring is a practical method for subjectively assessing the animal's body fat stores and muscle mass (Thatcher *et al.* 2000; Buffington *et al.* 2004) that has been validated against a 'gold standard' method of measurement of body composition (Laflamme 1997a and b). It involves viewing the cat or dog from the side and from the top to evaluate the *waistline*. It is also necessary to palpate skeletal structures, particularly the ribs, in addition to palpation of the muscle mass and fat deposits (Buffington *et al.* 2004). Dogs and cats in ideal body condition will have negligible subcutaneous fat covering over the lateral ribs

and, in short-coated animals, the outline of the most caudal 1–2 ribs may be visible. Excess fat is usually deposited over the trunk of dogs and in the abdominal fat pad of cats.

Dog and cat owners should be taught to evaluate their pet's body condition so that they can assess their pet's response to feeding and adjust food intake accordingly. The goal is to feed dogs and cats to achieve and maintain ideal body condition. Owners should be encouraged to monitor their pets' body condition continuously. If the animal starts to become fat, then the amount of food fed each day should be reduced. If the animal becomes thin, then more food should be fed.

Whenever an animal starts to become *overweight*, the *food intake should be reduced* to allow a return to lean body condition.

Management of obesity in dogs and cats

The concept underlying obesity management is simple; weight loss occurs whenever daily energy expenditure exceeds daily consumption of calories. Yet it can be very challenging to implement successful weight-loss programmes for pet dogs and cats. Owners frequently find it difficult to maintain compliance and motivation, even when they believe that their pet's health will be improved by reduction of excess body fat. The key is to use detailed evaluation of diet history and lifestyle to first identify the entire spectrum of specific owner and animal constraints that will affect implementation of a weight-loss programme, and then to develop practical solutions that work within these constraints (Burkholder & Toll 2000; Buffington *et al.* 2004). The aim is to make it as easy as possible for owners to comply with their pet's obesity management regimen. It is important to consider the individual animal–human bond and to provide ongoing support and guidance so that owner motivation and compliance are optimised.

The primary goal is always to reduce the animal's daily consumption of calories and/or increase its daily energy expenditure. Monitoring of progress is crucial and regular reassessment must be scheduled. Aim for loss of approximately 1% (range 0.5–2.0%) of the animal's initial body weight per week and then, more importantly, maintenance of the lost weight (Burkholder & Toll 2000; Buffington *et al.* 2004).

It is recommended that foods that are specifically formulated for obesity management of dogs and cats be fed to animals during weight loss. These products generally have reduced dietary fat content and increased fibre, air, or moisture content (Burkholder & Toll 2000). Importantly, they also have increased protein, essential fatty acids, vitamins, and minerals per calorie compared with standard adult maintenance diets (Burkholder & Toll 2000). Attempting weight loss in a dog or cat by feeding a reduced portion of the usual diet will result in reduced intake of these essential nutrients and might result in deficiencies.

Recommendations for increasing the activity level of the dog or cat as part of an obesity management programme must be individually tailored to suit both the pet and the owner. The most appropriate advice will vary from case to case. In addition to increasing daily energy expenditure, benefits of incorporating activity into the daily routine include the introduction of a form of owner–pet interaction that does not involve feeding, and the perception by the owner that their pet’s quality of life is improved. This is particularly important for owners who are concerned that calorie restriction will be an important welfare issue because they believe that they will be depriving their pet if they do not give the animal food when it appears to be hungry (Kienzle *et al.* 1998).

Although most people are familiar with means of providing exercise for dogs, many do not know how to exercise a cat. Recent research indicates that daily exercise is an effective means of managing obesity in cats, and can be used alone or in conjunction with calorie restriction (Giles *et al.* 2003; Trippany *et al.* 2003; & Clarke *et al.* 2005). The key for increasing activity in cats is to encourage playful, kitten-like behaviour. Cats tend to like to stalk, ambush, and pounce when playing. Devices that resemble a fishing rod with a toy dangling at the end are popular for exercising cats, as are items secured at the end of a piece of wire that flexes in an erratic and unpredictable manner. Another simple method involves blowing bubbles for the cat to chase using a child’s bubble-ring and soapy water. Some cats can be easily encouraged to follow their owner around the house for 5–15 minutes per day. It is not necessary for the cat to be constantly moving throughout the exercise period. Physical activity in cats naturally occurs in sporadic stops and starts. Children often enjoy being given the task of exercising a cat.

3.1.4 Summary of important points

- Feeding is major part of human-animal bond. For many dog and cat owners, feeding is one of the most important methods of demonstrating a caring and loving relationship with the pet. In addition, pet owners like to discuss nutrition.
- *Dogs are omnivores; cats are carnivores.*
- A *complete* food for dogs or cats is one that contains all of the required nutrients in adequate quantities.
- A *balanced* diet requires that all of the nutrients are present in the correct proportions.
- The Association of American Feed Control Officials (AAFCO) is internationally recognised as the organisation that sets the most rigorous and comprehensive standard for pet food labelling claims.
- Commercial dog and cat foods are available in different formulations to suit the varying nutritional requirements of individual animals.
- Commercial products formulated for working and performance dogs typically have higher dietary fat content than adult maintenance diets.
- One of the most common problems with feeding home-prepared food to dogs and cats is that the diet provided is not complete and balanced. If home-prepared food is to comprise 100% of a dog or cats diet for an extended period of time, owners require knowledge, motivation, additional financial resources and careful, consistent attention to recipe detail to ensure a consistent, balanced intake of nutrients.
- *Obesity is now the most common form of malnutrition in dogs and cats.* Approximately 30% of dogs and cats are overweight and, in some parts of the world, the prevalence has risen to 40% in middle-aged pets.
- There is strong evidence of an association between obesity and osteoarthritis in dogs. Dogs maintained in lean body condition have reduced prevalence and severity of osteoarthritis. Excess body fat is associated with greater prevalence and severity of osteoarthritis in dogs.
- *Prevention of obesity is the most important goal of feeding dogs and cats.*
- It is not possible to easily predict the daily calorie requirement for an individual dog or cat. Feeding recommendations provide a useful starting point, but it is essential that the calorie intake is regularly reassessed and adjusted to ensure that the animal is not being underfed or overfed.
- The goal of feeding large- and giant-breed puppies is to achieve moderate calorie restriction and sub-maximal growth. Owners should evaluate growth and body condition at least every 2 weeks.
- Large- and giant-breed puppies are more susceptible to developmental orthopaedic disease than small breeds, particularly when there is rapid weight gain, or dietary calcium levels are either too low or too high.
- Body weight does not correlate well with either body composition or body condition.
- *Body composition* refers to percentage of body fat and lean body mass.
- *Body condition* is a subjective evaluation of body composition.
- *Body condition scoring* is a practical method for subjectively assessing the animal’s body fat stores and muscle mass that has been validated against a ‘gold standard’ method of measurement of body composition.
- Whenever an animal starts to become overweight, the food intake should be reduced to allow a return to lean body condition.
- The primary goals of obesity management in dogs and cats are to reduce the animal’s daily consumption of calories and/or increase its daily energy expenditure. Monitoring of progress is crucial and regular reassessment must be scheduled.
- It is recommended that foods that are specifically formulated for obesity management of dogs and cats be fed to animals during weight loss.

3.2 Applied equine nutrition

Whether an animal is used as a source of income or as a companion, there is an inescapable and desired responsibility to ensure the well-being of the animal and, in the case of performance horses, optimise its performance via training, equipment, management, appropriate therapies and of course, nutrition. The following section will provide an overview of applied equine nutrition as it relates to conditions likely to be encountered by a clinical physiotherapist.

3.2.1 Digestive physiology and function

The horse is classified as a monogastric herbivore and its digestive system (Figure 3.2) is designed to cater to continuous grazing – nibbling and chewing on a variety of grasses while continuously walking, normally for upwards of 17 hours per day. Confinement of horses and meal-feeding high energy, high-grain diets create a series of assaults with which the digestive system was never designed to cope. Developing an understanding of the digestive system, its processes and limitations is essential for the successful feeding of a modern performance or breeding horse.

The stomach

The equine stomach is unique in that it is relatively small in volume for the size of the horse, accounting for only 8% of the total gut capacity. A 500 kg horse would only have a

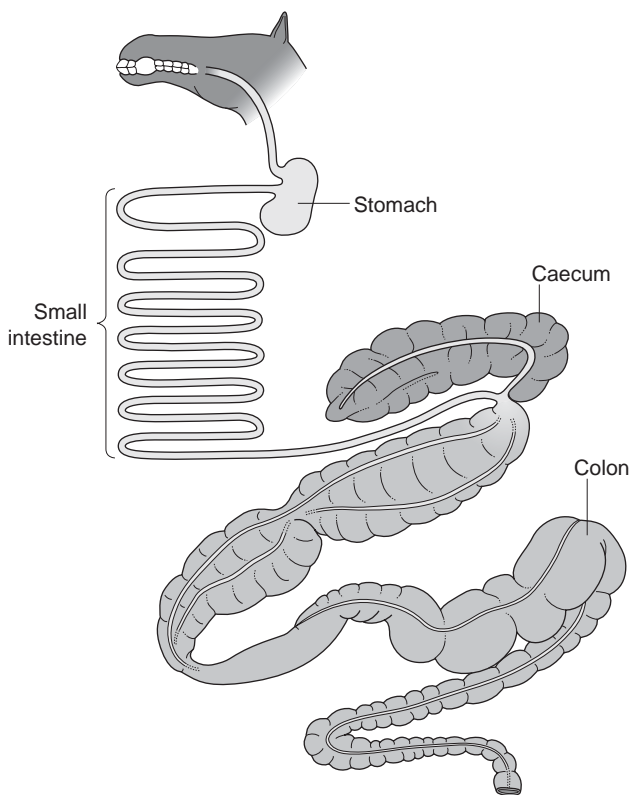


Figure 3.2 The digestive system of the horse.

stomach capacity of between 12 and 17 litres. Feed moves through the stomach in approximately 20 minutes.

Acid secretion in the equine stomach is continuous, with parts of the stomach highly acidic (pH as low as 1.5). This acid is buffered by both the presence of food within the stomach and the production of saliva in response to chewing. The high mucous content of saliva also helps lubricate feed for swallowing. A horse chewing long-stem roughage (such as hay or pasture) will produce 400–480 g of saliva per 100 g of dry matter consumed; while a horse on a grain-based ration will produce about half as much or 206 g of saliva per 100 g of dry matter. Therefore, horses fed predominantly short-stem roughage, such as chaff, grain and or premixed feeds, will produce less saliva with the result of increased acidity in the stomach. This can impact directly on the incidence and occurrence of gastric ulcers.

Small intestine

The small intestine is the principal site of digestion and absorption of protein, fats, starches and sugars. Horses lack a gall bladder and so bile is secreted continuously into the duodenum (approximately 1.5 l/h) along with the pancreatic enzymes for digestion. The majority of vitamins are also absorbed in the small intestine along with a number of micro- and macro-minerals. Enzyme production for starch in the equine small intestine is limited compared with other monogastric species. If a diet is fed with a starch component that exceeds this capacity, the starch will be fermented in the large intestine, with the potential to cause serious digestive disturbances. This limitation has implications discussed later with regard to meeting the energy requirements of performance horses. Food will pass through the small intestine within 2 to 8 hours. The rate of passage is influenced by ration form (pellet, loose mix or extruded nut), quantity, particle size and composition. Cellulose, a principle component of the fibrous portion of the ration, cannot be broken down by pancreatic enzymes and so this, along with plant lignin and undigested food, is passed through the small intestine to the hindgut.

Caecum and colon

The caecum and colon (hindgut) make up a large voluminous fermentative vat containing billions of bacteria and protozoa which ferment fibre and the remaining soluble carbohydrates into volatile fatty acids which are then absorbed into the bloodstream and utilised as a source of energy by the horse. Bacterial fermentation also produces certain B-group vitamins that are absorbed and utilised to a limited degree by the horse and some proteins that are not utilised but passed in the manure. The hindgut is also the major site of absorption of water, as well as phosphorus and certain electrolytes. Bacterial fermentation releases a lot of heat (think of the heat of compost as it ferments) and is important in thermoregulation of horses. This is why a horse can quite happily stand outside in temperatures

around 10°C without requiring a rug (i.e. this temperature is still within the thermoneutral zone for a horse).

The caecum and the colon combined account for over 65% of the gastrointestinal tract capacity and in a grazing horse of 500 kg may contain between 90 and 110 litres in volume. Note how much this contributes to body weight. A 500-kg horse will have at least 150 kg of its body weight from its intestinal tract. Fibrous material may be held in the hindgut for between 50 and 60 h, although rate of passage is greatly reduced in horses on high-grain, low-roughage diets. The environment within the hindgut is sensitive to reductions in pH that can occur when excess starch (or fermentable sugars from grasses) is consumed. Overload of the small intestinal enzymatic digestion allows excessive amounts of starch to pass into the caecum, where it is subject to fermentation and the production of D-lactic acid. This acid suppresses pH below 6.4 pH units, upsets the delicate balance, and can result in death of cellulolytic bacteria. The damage to the large intestinal mucosal allows absorption of endotoxin (the cell wall of the dead Gram-negative bacteria) and other bacterial toxins, stimulating an inflammatory cascade that can result in severe systemic illness and laminitis.

In stabled horses on high-grain, low-fibre diets, chronic reduction in hindgut pH has been implicated in an increase in the incidence of stereotypic behaviours such as wind-sucking and weaving, as well as increasing the prevalence of friskiness or 'hot-headedness'. However, horses on this type of diet and managed in this way may have gastric ulceration, behavioural changes unrelated to hindgut changes and any behaviour change should be interpreted with caution.

The key to maintaining efficient digestive function in a horse is to mimic, as closely as possible, his 'natural' diet by following a few simple rules:

- Base diet on forage: ideally not less than 50% of total feed intake by weight
- Keep a steady supply of forage available
- Balance diet around forage
- Make feed changes gradually i.e. over a 7–10 day period
- Do not feed more than 2.5 kg of grain or concentrate in any one meal

3.2.2 Condition scoring of horses

Knowing the body weight of a horse is important when administering drugs, monitoring growth, for tracking training progress and for diet management. From a musculoskeletal point of view, management to avoid obesity is also very important. Many veterinary practices specialising in horses will have horse scales installed and this provides the most accurate method of weight estimation. Weight tapes are available that have varying degrees of accuracy.

Weight can also be estimated using the following equation:

$$\text{Weight (kg)} = \frac{\text{girth (cm}^2\text{)} \times \text{length (cm)}}{11\,900}$$

Where length (cm) = point of shoulder to point of buttocks (tuber ischii).

In addition to knowing the weight of the horse and being able to estimate the change in weight, it is also important to recognise appropriate body condition in horses. The following system was proposed by Carroll and Huntington (1988) (Figure 3.3.).

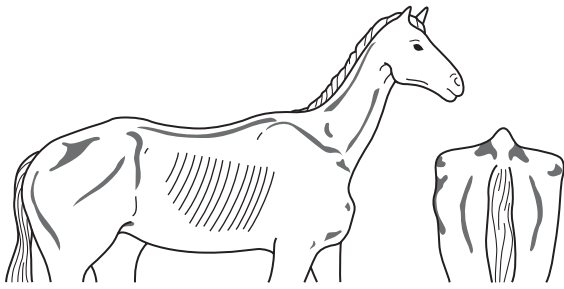
The desired body condition does vary according to the discipline or equestrian endeavour in which the horse and rider are involved. Horses used for showing tend to carry more body condition (Condition Score (CS) 4–5) than does a horse used for show jumping (CS 2–3). An endurance horse will be lighter again – usually CS of 2. Ideally a working horse should be maintained at CS 3 while a late pregnant broodmare maybe closer to 4 to enable her to sustain the demands of early lactation.

3.2.3 Feeding growing and breeding stock

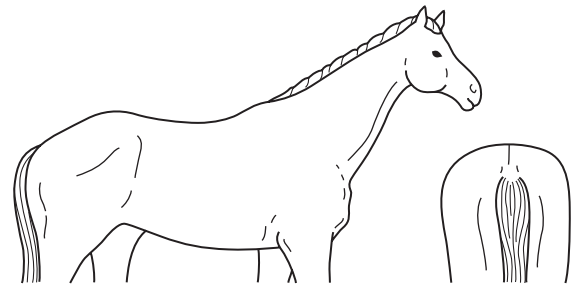
Breeding and young stock, particularly within the Thoroughbred racing industry, represent a large and vital component of the industry. It is common for horses to be at their most valuable before commencing their competition careers. Once they commence racing or performing, most will fall well short of their promised potential and fail to return the original investment. The reasons for this are multitude, but commercial producers recognise the importance of realising optimum prices for young stock and protecting their investment in breeding horses. For this reason, many commercial studs quite rightly pay close attention to the nutrition of their breeding stock and look to feeding as an important factor in the growth performance and sales preparation of their young stock. This focus has been amplified since the introduction of radiographs for Thoroughbred yearlings at major sales.

The following tables (Tables 3.2–3.4) summarise the requirements as listed by the National Research Council (NRC) publication, last updated in 1989. This is a useful reference, but it is unlikely that physiotherapists will be calculating the dietary requirements of equine patients every day. Therefore, it is important to know a good source of information to which clients can be referred. Large nutrition companies are excellent sources of dietary advice, e.g.: in the UK, Dodson and Horrell, Waltham Animal Nutrition; in the USA Kentucky Equine Research (KER); and in Australia there is KER Australasia. Most of these companies will offer dietary analysis, specific feed analysis and advice on feeding for specific problems.

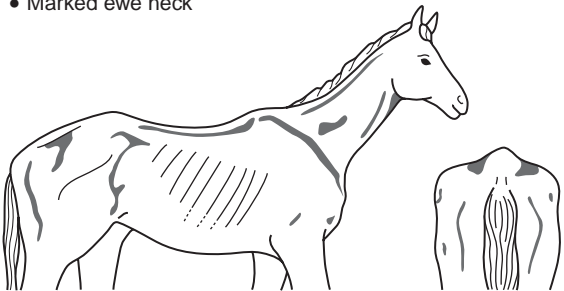
Since the NRC was published in 1989 there has been considerable research on the requirements of all classes of horse, not least of all breeding and growing stock. For this reason, most commercial diets will include higher levels

**0 Very poor**

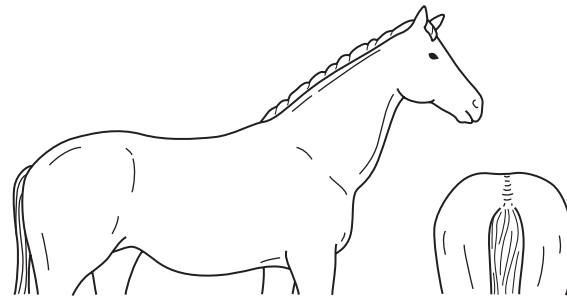
- Very sunken rump
- Deep cavity under tail
- Skin tight over bones
- Very prominent backbone and pelvis
- Marked ewe neck

**3 Good**

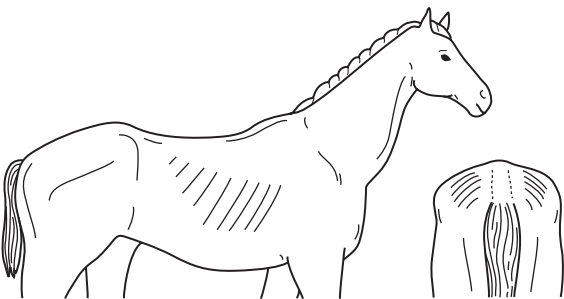
- Rounded rump
- Ribs just covered but easily felt
- No crest, firm neck

**1 Poor**

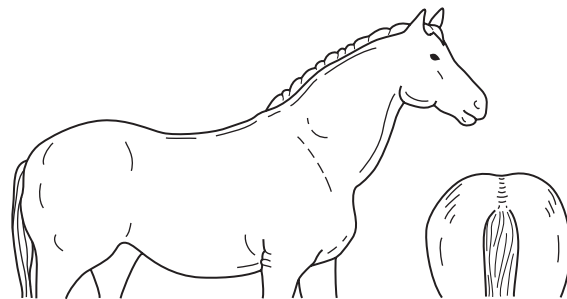
- Sunken rump
- Cavity under tail
- Ribs easily visible
- Prominent backbone and croup
- Ewe neck, narrow and slack

**4 Fat**

- Well-rounded rump
- Gutter along back
- Ribs and pelvis hard to feel
- Slight crest

**2 Moderate**

- Flat rump either side of backbone
- Ribs just visible
- Narrow but firm neck
- Backbone well covered

**5 Very fat**

- Very bulging rump
- Deep gutter along back
- Ribs buried
- Marked crest
- Folds and lumps of fat

Figure 3.3 Condition scoring system for horses (Carroll & Huntington 1988).

of certain nutrients than are contained in the NRC recommendations. Kentucky Equine Research (KER) is a research-based consultancy that has done extensive research on the requirements of growing horses and formulates commercial diets for leading studs worldwide. A comparison of KER recommendations and those of the NRC are listed in Table 3.5.

While there is reasonable agreement between energy and protein requirements, the major difference occurs in the region of macro- and micro-mineral requirements. This is due to recognition of the role improved mineral nutrition plays in the growth and development of young horses and the benefits in prevention of expression of a number of developmental orthopaedic diseases that plague growing

Table 3.2 Recommendation for daily nutrient requirements of broodmares (500 kg mature weight)

	Pregnant mares			Lactating mares	
	9 months	10 months	11 months	3 mths – weaning	
Weight (kg)	500	500	500	500	500
Digestible energy (Mcal)	18.2	18.5	19.7	28.3	24.3
Crude protein (g)	801	815	866	1427	1048
Lysine (g)	28	29	30	50	37
Calcium (g)	35	35	37	56	36
Phosphorus (g)	26	26	28	36	22
Magnesium (g)	8.7	8.9	9.4	10.9	8.6
Potassium (g)	29.1	29.7	31.5	46	33

Table 3.3 Recommendation for daily nutrient requirements of growing horses (500 kg mature weight)

Growth rate	Weanling (4 mths)	Weanling (6 mths)		Yearling	
		Moderate	Rapid	Moderate	Rapid
Weight (kg)	175	215	215	265	265
Daily gain (kg)	0.85	0.65	0.85	0.4	0.5
Digestible energy (Mcal)	14.4	15.3	17.2	15.6	17.1
Crude protein (g)	720	750	860	700	770
Lysine (g)	30	32	36	30	33
Calcium (g)	34	29	36	23	27
Phosphorus (g)	19	16	20	13	15
Magnesium (g)	3.7	4	4.3	4.5	4.6
Potassium (g)	11.3	12.7	13.3	14.5	14.8

Table 3.4 Micro-mineral requirements for different classes of horse

	Adequate concentrations in total rations					
	Sodium (%)	Fe (mg/kg)	Mn (mg/kg)	Cu (mg/kg)	Zn (mg/kg)	Se (mg/kg)
Maintenance	0.1	40	40	10	40	0.1
Pregnant and lactating mares	0.1	50	40	10	40	0.1
Growing horses	0.1	50	40	10	40	0.1
Working horses	0.3	40	40	10	40	0.1
Maximum tolerance levels	—	1000	1000	800	500	2

horses. A young growing horse will achieve 90% of its mature size by the end of its first year of life. Early in the first 3 months the mare's milk will provide the bulk of nutrition (assuming she is well fed) but the foal also requires access to quality roughage and some hard (concentrate) feed during this time. This can be achieved by allowing the foal access to

Table 3.5 Comparison of National Research Council (NRC) and Kentucky Equine Research (KER) nutrient recommendations

Animal	Source	DE	CP	Ca (g)	P (g)	Cu (mg)	Zn (mg)
		(Mcal)	(g/day)				
Weaning (0.85 kg/day)	KER	17.3	864	44	29.3	168	504
	NRC	18.1	906.6	37	20.1	62.5	250
Yearling (0.65 kg/day)	KER	22.4	1008	52.6	35.1	168	504
	NRC	22.1	995	35	18.7	79	316

DE, digestible energy; Cu, copper; Fe, iron; Se, selenium; Zn, zinc; Mn, manganese; CP, crude protein; Ca, calcium; P, phosphorus (Lawrence 2003)

the mare's food or by feeding the foals in a dedicated creep area. The ration selected needs to be high in quality protein, such as from soybean meal, and roughage – preferably of a legume base. Additional energy concentrates may be appropriate to maintain body condition, but supplementation with macro- and micro-minerals is essential – particularly in areas of known mineral deficiency.

3.2.4 Nutrition-related disorders of growing horses

In an attempt to produce very well-grown yearlings, some breeders have been guilty of overfeeding (over nutrition) their young stock without consideration of the implications on bone growth. Increasing incidence of bone disorders in young horses has focused research and management attention on possible causes. Orthopaedic diseases in Thoroughbred horses cost the industry millions each year and up to 10% of all Thoroughbred foals born will require surgery to correct angular limb deformities.

Developmental orthopaedic disease

Developmental orthopaedic disease (DOD) is a general term used for a number of orthopaedic problems that may develop in the juvenile horse. Although not exclusive to DOD, the diseases generally recognised as being part of the DOD syndrome include:

- Osteochondritis dissecans
- Physisitis
- Osteochondrosis
- Angular limb deformities
- Flexural deformities
- Subchondral cystic lesions
- Cervical vertebral malformation (wobbler syndrome)
- Cuboidal bone malformation

Most research has concentrated on reducing the incidence of osteochondrosis. Osteochondrosis occurs when the cells in growing cartilage do not undergo normal differentiation. This occurs at the normal growth locations

including the metaphyseal growth plate and in the articular cartilage in the growth zone of the epiphyseal plate. Calcification of the matrix does not occur because vessels from the bone marrow do not penetrate the cartilage (Stromberg 1979; Jeffcott 1991). The thickened cartilage, which still has potential for growth, is the site where necrosis may occur. From within this layer of cartilage, fissures may develop that extend into the joint surface. The fissures cause damage to cartilage and the normally smooth, hard articular surface. Intra-articular fragments may then occur due to the detachment of endochondral or cartilaginous fragments from the bone. An inflammatory response within the joint may then occur and the condition is then referred to as osteochondritis desiccans (OCD) (Stromberg 1979). Synovitis within the joint may develop and this is associated with pain, loss of joint range of movement and lameness. Removal of any resulting 'chips' or detached pieces of subchondral cysts requires surgery and is most commonly performed on the stifle, hock or pastern joints.

DOD is regarded as a multifactorial problem with possible contributing causes including nutrition of the brood mare, foal, weanling and yearling.

Certainly it is known that faster growing, heavier foals have a higher incidence of the condition than do their slower-growing, lighter counterparts (Jeffcott 1991; Pool 1993). One study of Warmblood foals (Van Weeren *et al.* 1999) showed that osteochondrosis developed in the patellar femoral joint of the foals which had the greatest weight gains. The final measurements of these foals showed they had a higher body weight and grew to a greater height compared with the foals in the study which did not develop osteochondritis.

The formation of the collagen and elastin matrix is dependent on the presence of a normal level of copper and the enzymes associated with this process. Insufficient levels of copper are associated with more friable collagen and osteochondrosis. Normal absorption of copper by foals can be affected by zinc toxicosis. Burton & Hurtig (1991) reported a significant increase in the incidence of DOD lesions in foals fed 8 mg/kg of copper versus foals fed 25 mg/kg.

Nutritional management to reduce the incidence of DOD involves:

- Ensuring the mare's diet is fortified with required minerals in the last trimester, since milk is a poor source of certain minerals and so the foal must rely on liver stores during the first months of life when milk is the principal nutrition source.
- Providing a well-fortified (with minerals and protein) creep or lactation ration that will encourage intake by the foal.
- Monitoring body weight of the foal to prevent excessive weight gain or body condition. Ideally, this is done with the regular use of electronic weigh scales.

Genetics

It has not been definitely established that there is a genetic link for osteochondrosis, although work in Sweden has shown a preponderance of osteochondrosis in foals of a number of stallions (Jeffcott 1991).

Biomechanical factors

Exercise (lack of): Exercise is important to provide some concussion to stimulate bone growth and strength. Van Weeren & Barneveld (1999) found that exercise did not influence the severity of osteochondrosis in the foal's first few months of life but, for foals confined to a stall, osteochondrotic lesions were more severe.

Biomechanics – abnormal limb loading: Abnormal conformation might result in excessive or abnormal forces to a joint or multiple joints. Intervention may assist, for example the farrier may be able to trim the young horse's feet to improve alignment in the lower limb. In some cases special shoes may be fitted to correct alignment. In the foal or young horse these will be glued to the foot rather than nailed to minimise trauma and damage to the developing foot (Jeffcott 1991; Whitton 1998).

When tension and compression limits on a physis are exceeded, physal growth may be significantly decreased or stopped. Furthermore, direct trauma, traction, circulatory loss or shearing forces can lead to premature cessation of growth or asynchronous growth. The end result of these abnormal processes is angular limb deformity, physisitis or bony malformation. Unrestricted exercise may exacerbate excessive physal compression and asymmetric loading of the limb and prevent auto correction. Therefore exercise of foals is restricted to box walking only.

Endocrine factors

- Administration of external steroids
- The glycaemic index of feeds (see below)

The impact of glycaemic index on the incidence of DOD

In recent years, research has been performed on the impact of glycaemic index on both working and breeding horses. The glycaemic index of a feed or feed ingredient refers to the extent of increase in blood glucose (and the hormone insulin) concentration following its consumption. Insulin is thought to have a role in the development of bone from cartilage. Insulin may be responsible for the survival of chondrocytes or suppress the differentiation of chondrocytes (Jeffcott & Henson 1998).

In general, feeds high in starch and/or simple sugars have a high glycaemic index. For example, a meal of oats (which are about 50% starch) will result in a substantial increase in blood sugar, whereas blood glucose is largely unchanged after a meal of grass hay (which is very low in sugar and

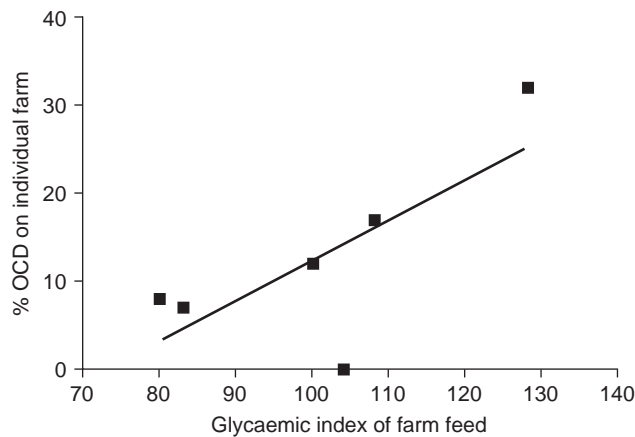


Figure 3.4 Relationship between feed glycaemic index and incidence of osteochondritis dessicans (OCD).

starch). Research by Glade *et al.* (1984) and Ralston (1995) suggest that foals that experience a continued exposure to high levels of circulating glucose or insulin in response to a high-grain meal may be predisposed to development of OCD. Research suggests a link between glycaemic index and the incidence of OCD in growing horses (Pagan *et al.* 2001; Pagan 2003) (Figure 3.4). This research suggests that reducing the level of starch, slowing feed intake and lowering the glycaemic index of the feed may help reduce the incidence of OCD in growing horses.

3.2.5 Feeding the performance horse

One of the greatest challenges in equine feeding comes from meeting the very high energy demands of elite performance horses without compromising digestive function or producing colic, gastric ulceration or other disorders. In order to sustain athletic effort, the digestive system has to cope with a level of energy intake for which it was never designed.

Meeting this challenge involves careful attention to feeding management as well as strategic use of processed feeds, oils and supplements. The following section will describe some of these strategies and some feed-related disorders of the performance horse.

First of all it is important to understand what defines performance in a horse, since riding a pleasure hack twice a week does not qualify that animal as a 'performance' horse.

The definition of performance is (per day):

- Endurance – 2 hours or more – low intensity exercise
- Middle distance – 800–3200 m for several minutes at 75–95% max. intensity exertion
- Sprint – >400 m in <1 minute at 100% intensity exertion (Chapter 5)

A horse in light work has energy requirements approx. 25% above maintenance. A horse in moderate work has

energy requirements 50% above maintenance and a horse in intense work has requirements 100% above maintenance. Energy requirements depend upon:

- Body weight of horse
- Type and duration of work
- Combined weight of rider + gear
- Age, rate of growth and estimated mature body weight
- Ambient temperature
- Condition and training of the horse
- Individual variation

The consequences of excessive starch intake on the digestive function of horses have already been discussed. For working horses it is important that their increased energy requirement is met in a safe and sustainable (and palatable) manner.

There are a number of ways of increasing the safety of energy intake for horses:

- Increase feeding frequency. Split the concentrate portion of the diet over more meals and never exceed more than 2.5 kg of concentrate per meal.
- Use grains that have been processed to reduce the amount of fermentable starch per meal. This can be achieved via steam flaking, extruding, micronising and pelleting. Soaking is not sufficient. Oat starch is the exception and does not require processing since oat starch is preferentially digested in the small intestine.
- Replace some of the starch with oil. Oil has approximately three times the energy concentration of oats and is absorbed in the first metre of the small intestine. It can take the muscles up to 4 weeks to become conditioned to using free fatty acids from oil as an energy source for aerobic work and at least 250 ml per day are required for a 500 kg horse, although much higher levels (up to 20% daily intake) have been used with success.

The NRC estimated the energy requirements by activity (Table 3.6).

Horses, if allowed, will consume between 1.5 and 2.5% of their body weight in dry matter of feed each day. This translates to an intake of between 7.5 and 12 kg for a 500-kg horse. Horses are individuals and they can vary widely in their feed intake and energy requirements depending on workload, climate, breed and temperament. The NRC proposes other nutrient requirements for performance horses (Table 3.7).

From Table 3.7 it would appear as though an increased concentration of protein would be required for horses in hard work. This is not the case. A horse of 500 kg on a 10% crude protein diet would be consuming 1000 g of protein per day – more than adequate to meet requirements. As workload increases, so should intake of energy (otherwise the horse would lose weight). A 500-kg horse in intense work may easily consume 13 kg of feed. Even at the same protein concentration, 10%, the horse will now receive

Table 3.6 Energy requirements for performance horses at various levels of work

Weight (kg)	400	500	600
Energy (MJ) required for maintenance per day	58	68	79
Additional energy (MJ) required per hour for:			
Walking	0.84	1.05	1.26
Slow trotting, some cantering	8.4	10.5	12.6
Fast trotting, cantering, some jumping	21.0	26.2	31.4
Cantering, galloping, jumping	38.5	48.1	57.8
Strenuous activity (polo, racing at full speed)	65.3	81.6	97.9

Table 3.7 Requirements for working horses

	Light work	Moderate work	Intense work
Weight (kg)	500	500	500
Digestible energy (Mcal)	20.5	24.6	32.8
Crude protein (g)	820	984	131.2
Lysine (g)	29	34	46
Calcium (g)	25	30	40
Phosphorous (g)	18	21	29
Magnesium (g)	9.4	11.3	15.1
Potassium (g)	31.2	37.4	49.9

Table 3.8 Estimated intake – a % body weight of forage and concentrate for different levels of work

Category	Forage	Concentrate	Total
Maintenance	1.5 to 2.0	0.0 to 0.5	1.5 to 2.0
Light work	1.0 to 2.0	0.5 to 1.0	1.5 to 2.5
Medium work	1.0 to 2.0	0.75 to 1.5	1.75 to 2.5
Heavy work	0.75 to 1.5	1.0 to 2.0	2.0 to 3.0

1300 g of protein per day by virtue of the increased intake. In reality, horses on part lucerne and prepared feed rations are likely to be consuming a ration of >10% protein and so fortification with high protein supplements to meet increased needs of intense work is not justified. Excess protein is converted to energy, but carries with it an increase in circulating urea and increase in ammonia output. The latter compromises air quality which can predispose horses to inflammatory airway disease and compromise their lung function.

As workload increases, so must intake as shown in Table 3.8. The increased energy requirements of work cannot be met by roughage alone and so an increasing proportion of the diet is composed of concentrate. Ideally the proportion of roughage should not decline below 50% of the total diet by weight or 1–1.5% of body weight. In practice, this does not always happen and many elite-level performance horses receive only 30% of their diet by weight as roughage or less than 0.75% of body weight.

3.2.6 Feeding-related disorders of performance horses

The sub-optimal intake of long-stem roughage (and corresponding increase in starch intake) commonly seen in performance horses has a number of undesirable consequences such as:

- Increased likelihood of developing gastric ulcers – this is due to reduced chewing and therefore saliva production, further exacerbated by greater time without feed present in the stomach
- Reduced production of B-group vitamins due to reduced hindgut fermentation
- Increased likelihood of colic
- Reduced pH in large intestine (due to altered volatile fatty acid content) with resulting increase in stereotypic behaviours
- Higher insulin and blood glucose levels that can compromise behaviour
- Exacerbate condition of horses prone to tying-up (Chapter 7)

It is outside the scope of this text to cover each of these in detail, but the feeding management and predisposing causes for some of these disorders need to be understood by anyone involved in the treatment and rehabilitation of horses thus affected.

Gastric ulcers

It is only in the last decade that endoscopes with the length required to evaluate the equine stomach have been readily available. As a result, more performance horses are being checked and now there is mounting evidence to suggest that the incidence of gastric ulcers in horses is very high – especially among elite performance horses on high-grain diets. Some studies put the incidence in Thoroughbreds at close to 90% (Begg & O'Sullivan 2003). Some horses with gastric ulcers may have no outward signs, while others will show vague signs including poor or 'picky' appetite (often a preference for the roughage portion of their diet), poor performance, poor hair coat and colic (Buchanan & Andrews 2003).

Studies have shown that a horse previously on pasture can develop bleeding gastric ulcers within 3 days of being confined to a stable on hay and concentrates (Vatistas *et al.* 1999). Conversely, it may take up to a month of paddock rest without access to hard feed to heal gastric ulcers. This is not always practical and so most treatments aim at reducing gastric acid secretion or neutralising the acid produced.

Feeding management aims at increasing access to long-stem roughage, including lucerne hay as part of the roughage since lucerne has antacid properties (Nadeau *et al.* 2000) and including oil in the diet, which will slow gastric emptying and decrease gastric acid production.

Feed-induced muscle problems

Muscle problems and diseases (myopathies) that are related to feeding/exercise go by a number of names. Tying-up is the most commonly used name to describe symptoms that can be attributed to a number of specific disorders, such as recurrent exertional rhabdomyolysis (RER) or polysaccharide storage myopathy (PSSM). Other names include azoturia, paralytic myoglobinuria and myositis. Differentiation of these disorders will be covered elsewhere in the text. Feeding is just one factor that can contribute to expression of these problems and usually stems from a combination of the following:

- Overfeeding of carbohydrates (MacLeay *et al.* 1999)
- Electrolyte or mineral imbalances, especially with potassium (Harris 1991)
- Deficiency of selenium and/or Vitamin E

If elite-level performance is to be maintained in horses predisposed to these muscle problems it is crucial to reduce the amount of carbohydrate in the diet as much as possible. This is done by increasing the use of non-starch energy sources such as fibre and oil. The use of highly digestible fibres such as legume hulls, copra meal and sugar beet (without added molasses) is recommended in conjunction with a high fat (>8%) diet.

Results by Williams *et al.* (2003) show that a higher vitamin E intake (2000–4000 IU/d) reduces muscle leakage and oxidative stress. These levels are considerably higher than the 1000 IU/d recommended by the NRC (1989).

Sodium and chloride are the two electrolytes required in the greatest amount by working horses so the addition of salt to the diet is recommended along with a proprietary electrolyte replacer that contains additional potassium. Alternatively a mixture of 2 parts sodium chloride and 1 part potassium chloride can be used.

Feeding older/geriatric performance horses

Horses are competing at international level in Olympic disciplines past 15 years of age and so our view on what constitutes an 'old' horse needs to change. From a nutritional standpoint, a horse is classified as geriatric at 20 years of age, but some horses may require closer nutritional management from 17 years of age. Obviously the presence of particular disorders, such as Cushing's syndrome, insulin resistance, history of laminitis or poor dentition, will influence feeding management.

Typically, geriatric horses do not hold weight as well as their younger paddock mates. While much of this can often be traced back to underlying dental disease, there is also some evidence that digestion can be impaired. Protein and phosphorus absorption may be compromised in older horses but calcium absorption is not, unlike dogs and humans (Ralston *et al.* 1989). Note: horses have an entirely different calcium regulation than man with control being in renal excretion rather than in the amount of absorption

from the gastrointestinal tract, so this is not unexpected. (This is why a horse's urine is cloudy in appearance – due to the appearance of calcium carbonate crystals.) Also, aged horses (horses over the age of 20 years) that were fed a high protein (14%) pelleted/extruded feed improved in body condition scores and haematological variables more than those fed a non-processed concentrate (Ralston & Breuer 1996).

For aged horses not maintaining weight, an increase in the allowance of concentrates is suggested with addition of oil recommended where a history of founder or tying-up is present. It is recommended to feed aged horses a high quality protein such as soybean meal and the use of some yeast cultures can improve digestibility. Vegetable oils are also recommended as a source of concentrated energy. Typically available oils would be soybean oil, canola oil and corn oil. The author suggests a maximum of 480 ml/day, as higher amounts are considered to be unpalatable (Sicilano 2002).

Ponies and geriatric horses with Cushing's syndrome or recurrent laminitis often have insulin resistance and so feeds containing molasses and high levels of starch should be avoided.

Nutraceuticals

A nutraceutical is a term used to describe a non-toxic dietary supplement with demonstrated health benefits. Some nutraceuticals are vitamins and minerals routinely included in feed to counter deficiencies in base ingredients or to provide specific health benefits such as biotin to promote hoof growth or vitamin E as an antioxidant. A nutraceutical may also be a herb such as garlic or valerian that may not be natural components of a horse's diet, but which, based on evidence in humans, have perceived benefits for a horse.

When selecting any supplement for a horse, it is important to be confident of the following:

- *What is the composition?* The ingredients – both active and carrier must be clearly printed on the outside of the container.
- *What will it do?* Vague statements such as 'contains liver salts' give no indication of mode of action and should be avoided. Clear indication of site of activity and likely response is required. A biotin supplement would state for example 'Contains 15 mg biotin per daily dose to promote growth and integrity of horses' hooves.'
- *What do you expect it to achieve?* This relates to realistic expectations of action. If you expect the product to calm a horse down, decide how quickly you expect this to happen and make sure you do not make any other major changes to your horse's regime so you can evaluate if the supplement has worked.
- *Will it be detected on a urine or blood test ('swab')?* Many of the positive swabs received by racing authorities are to herbal supplements that trainers wrongly believed would

not 'swab' because they perceived them as being 'natural'. Remember most commercial drugs are derived from plant materials so it needs to be clearly stated by the company selling a product that its use will not contravene the rules of racing.

- *Is its efficacy backed by some independent research?* This is where the Internet comes in. Search for some university-backed research that demonstrates efficacy of the ingredient you are feeding so that you have some realistic expectations of the outcome. Joint supplements are an excellent example and a short tour of the recent research will confirm if the use of an expensive supplement is likely to be of benefit in managing your horse's lameness. Nutraceuticals labelled for use in other species should be avoided.

3.2.7 Common diet problems and simple feeding rules

Feeding problems with horses arise when owners fail to recognise that they are in charge of a grazing herbivore – an animal designed to walk, nibble and chew 17 h per day. Confining that animal to a small stable or yard, without access to long-stem roughage and offering two meals per day loaded with fermentable starch creates a situation where colic and/or laminitis is an inevitability.

Other problems arise when trainers try to find an instant solution within a bag of feed or a bucket of supplement and do not consider the impact on the rest of the diet. Making sudden changes to feed, over supplementing or feeding insufficient of an appropriate feed/supplement only serves to unbalance a ration and prevents the horse from obtaining the benefit he might from the feeds available.

3.2.8 Summary: Feeding hints for all horses

- Base the diet on roughage – preferably long-stem roughage and no less than 50% of the total diet by weight.
- Know the weight of food given.
- Horses are individuals – feed them accordingly.
- Use quality feeds – no weeds, dust or moulds.
- Feed the horse according to work.
- Do not feed more than 2.5 kg of concentrate in any one meal.
- Feed small meals often (at least three times per day) and at regular times.
- Make changes gradually, ideally over 10–14 days.
- Observe dung for changes.
- Regularly review feed programme – seek advice.
- Reduce grain levels on the night before and on the days of rest.

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4

Applied animal biomechanics

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4.1 Introduction

The aim of this chapter is to introduce key concepts of applied biomechanics in the dog and horse based on the limited research into animal functional biomechanics and kinematics. This chapter does not address pure biomechanics, nor is it intended to replicate the theoretical basis of biomechanics or summarise current biomechanics literature and texts. The authors aim to direct the reader towards some of the applied principles of functional biomechanics in relation to animal physiotherapy assessment and treatment, based on evidence where possible.

An understanding of concepts of neuromotor control, musculoskeletal physiotherapy and rehabilitation are vital to the application of physiotherapy to animals. To enable the animal to achieve a complete functional sports-specific outcome through rehabilitation, the animal physiotherapist must be able to apply treatment techniques and management strategies based on an understanding of the mechanisms behind the cause of musculoskeletal injury in the cursorial mammal. Thus, animal physiotherapists require an understanding of each species' anatomy and biomechanics, as well as the requirements of the animal's sport. A working knowledge of the mechanical demands and constraints that animals operate under during locomotion is essential to enable the clinician to assess the compensations; secondary neuromuscular and skeletal problems and pathology that ensue following failure of one or more elements. The reader is urged to review additional literature and texts referenced.

Biomechanics refers to the application of mechanical principles in the study of living organisms. Kinematics is the measurement and description of motion, including considerations of space and time, without looking at the forces, whereas kinetics is the measurement of force and the relationship of force and mass (Hall 1995; Wilson *et al.*

2003). Locomotion is a result of a force (e.g. via the limbs, particularly hind limb in quadrupeds) being applied to a mass (trunk) that it accelerates. This chapter will cover canine and equine joint biomechanics, species-specific biomechanics of locomotion, and give examples of applied sports-specific biomechanics in the equine and biomechanics of the equine distal limb related to pathology. For species-specific biomechanics of locomotion in animals, the greatest amount of research and knowledge has centred on the athletic horse, with research regarding the same in the canine notably lacking.

4.2 Joint biomechanics

This section focuses on biomechanics related to individual joint motion in the canine and equine, as joint movements indicate how the musculoskeletal system is working. Knowledge of the normal pattern and amount of movement allows the physiotherapist to detect abnormalities of movement (Lee 1995).

According to Lee (1995) there are three parameters required for a full definition of movement at a joint:

- location of the axis of motion including information about how this location changes during the movement;
- amount of rotation – the angle through which the joint rotation occurs about the axis during movement; and
- amount of translation – the displacement that occurs along the axis during the movement.

The way we describe joint motion can only be an approximation, as many aspects of joint dynamics such as torques and forces acting at the joints are not readily available to us. *Axis of rotation* or *axis of motion* is a term for a central imaginary line that is orientated perpendicular to the plane in which the rotation occurs (Hall 1995). It is difficult to know

the axis of joint rotation at any instant in a movement (the instantaneous axis, or instantaneous centre of rotation (ICR)). This is because very few joints have a fixed axis of motion, as biological joint movements are complex (Denoix 1999). Denoix (1999) states that the establishment of the ICR reveals several functional aspects of vertebral structures and associated ligaments during dorsoventral movements of the vertebral column. When a canine stifle is flexed, the instantaneous centre of rotation of the joint moves caudally and then cranially when the joint extends (Ireland *et al.* 1986). In the horse, Denoix (1999) has shown the ICR of thoracolumbar vertebrae to be variable in different dorsoventral positions of the thoracolumbar spine and cervical spine. For example, with cervical flexion the ICR of each thoracolumbar vertebral body tends to move cranially and ventrally, and during thoracolumbar flexion the ICR is centred in the vertebral body. In the lumbosacral joint during flexion, the ICR moves to a more ventral position than during extension.

In three-dimensional joint kinematics, coupled movements are known to occur (Lee 1995; Denoix 1999). To deal with the complexity of three-dimensional movement it is useful to consider the joint motion as consisting of rotations in three orthogonal planes: sagittal, frontal and horizontal (or yaw, pitch and roll) and three components of translation – caudocranial, mediolateral and dorsoventral.

For physiological motion, where rotation is the desired motion, one of the rotations is designated as the main movement. All other movements including rotations in other planes and translations in any direction are called coupled movements (Lee 1995). For example, in dogs and humans, axial rotation of the cervical spine between C3 and C7 is coupled with ipsilateral lateral flexion (Breit & Kunzel 2002). Axial rotation is the main movement and lateral flexion is the coupled or accessory movement.

4.2.1 Joint stiffness

The range of motion of a joint is defined as its entire range of physiological movement, measured from the neutral position. It is divided into the neutral zone and the elastic zone (Panjabi 1992b). The neutral zone is the region of low stiffness, where joint motion is produced with minimal internal resistance, to allow physiological movements to occur freely within a certain range. The elastic zone is measured from the end of the neutral zone up to the physiological limit and is the zone of higher stiffness. When a joint is translated as in a passive accessory movement, the low stiffness zone is smaller than for a physiological movement, and resistance is likely through the entire movement, increasing linearly with degree of translation (Lee 1995). There is little information as to which anatomical structures are responsible for the tissue resistance perceived by the physiotherapist in either type of passive joint movement. The tissues involved in resistance may be muscles/fascia, ligaments, neuromeningeal structures and joint capsule.

4.2.2 Joint instability

It is worth noting here the difference between ‘mechanical instability’, ‘clinical instability’ and ‘functional instability’. In physical terms, an unstable structure is one that is not in an optimal state of equilibrium (Pope & Panjabi 1985). Where there is mechanical joint instability, small initial movements will result in further movement until a position of stability is reached and potential energy is at a minimum (Lee 1995). In a physiological situation, muscles and other restraining soft tissues provide resistance and tend to restore the joint to its original position. In veterinary terminology, luxation is synonymous with dislocation, which is defined as displacement of a bone from a joint, compared with subluxation, which is defined as a partial dislocation (Blood & Studdert 1999). This may be the case in humans with a glenohumeral joint prone to subluxation or dog with a patella that tends to subluxate, or where segmental spinal muscles do not perform the action of maintaining intervertebral motion (Panjabi *et al.* 1989).

4.2.3 Clinical instability

Clinical instability has been suggested by Lee (1995) to be alternatively named pathological hypermobility, where there may be damage to structures restraining a joint’s movement. An example of a clinical instability is excessive motion at a vertebral level that may compromise the spinal cord, such as a Doberman with cervical vertebral malformation requiring surgical stabilisation (Chapter 7). However, clinical instability of a spinal motion segment as defined by Panjabi (1992b); is ‘a significant decrease in the capacity of the stabilising system of the spine to maintain the intervertebral neutral zone within the physiological limit’ such that there may not be altered neurological function, or no major deformity and no incapacitating pain. Therefore size of the neutral zone of a given vertebral motion segment is a better indicator of clinical or functional instability than is the overall range of motion or current clinical symptoms alone, i.e. sensorimotor disturbances. Methods in which to measure the neutral zone *in vivo* are yet to be well established (Panjabi 1992b).

Thus the neutral zone, at least in the vertebral column, is a clinically important measure of spinal stability and overall *functional instability*. Human *in vitro* studies, animal *in vivo* studies and mathematical simulations have shown that the neutral zone is a parameter that correlates well with other parameters indicative of instability of the spinal system (Panjabi 1992b). In the vertebral column, the neutral zone has been shown to increase with injury or secondary weakness of the spinal muscles, due to a decrease in intersegmental dynamic muscle stability. For example, lumbosacral intervertebral motion has been extensively investigated in man via a series of biomechanics and neuromotor control studies, in normal and low back pain subjects (Panjabi *et al.* 1989, 1992a & b; Hides *et al.* 1992, 1994, 1996; Moseley *et al.* 2002; Hodges 2003; Lee 2004). In human

(Panjabi *et al.* 1989, 1992a & b; Hodges & Richardson 1996; Hides *et al.* 2001; Hodges *et al.* 2001) and porcine (Kaigle *et al.* 1995; Kaigle *et al.* 1998; Hodges *et al.* 2003) studies, the role of the deep stabilising muscles of the vertebral column has been investigated. These are the anteriorly situated *transversus abdominus* (hypaxial muscle in the quadruped), and the posteriorly located *multifidus* (epaxial muscle). The activity of the deep stabilising muscles of the vertebral column and pelvis muscle appear to be both preparatory and dynamic, in the way they limit the neutral zone, and have been shown to affect both motion and stiffness of the intervertebral segments of the spine and pelvis. The multifidus muscle is directly associated with dysfunction and atrophy of this muscle has been shown to be closely linked with thoracolumbar and lumbosacral pathology in human back pain (Hides *et al.* 1994, 1996; Moseley *et al.* 2002; Lee 2004). Further, poor dynamic control of these muscles has been shown to be a predictor for lower back pain in humans (Cholewicki *et al.* 2005).

There is limited neuromotor control data for the equine and canine vertebral column, and the role of muscle in dynamic control of stability is virtually unknown (Peham *et al.* 2001). Equine electromyography studies have focused on the large trunk and epaxial muscles only, such as *longissimus dorsi* in relation to trotting on a treadmill (Peham *et al.* 2001; Robert *et al.* 2002; Licka *et al.* 2004).

The activity of the human multifidus muscle has been shown to increase intervertebral stiffness at L4–5 in multiple directions owing to the principal agonist muscles, the abdominal oblique muscles, simultaneously flexing the lumbar spine (Wilke *et al.* 1995). Kaigle *et al.*'s (1995, 1998) work shows that electrical stimulation of multifidus improves the quality of control of intervertebral motion around the neutral position during movement in the sagittal and frontal planes. It is hypothesised that the multifidus may provide a similar role in control of intervertebral stability in horses, as there is a similarity in muscle morphology and architecture in the thoracolumbar and lumbosacral and pelvic regions compared with the human (Stubbs *et al.* 2006).

4.3 Biomechanics of the vertebral joints

The aim of the section that follows is to summarise the functional anatomy of the articulations of the vertebral column in both the dog and the horse, so that the physiotherapist may identify alterations in patterns of joint movement from the normal. In understanding the normal directions and planes of joint motion, the physiotherapist can apply passive movement tests during assessment, with a reasonable amount of accuracy. Understanding the physiological loads experienced by any individual structure is also difficult because the associated moments and forces cannot be measured with any degree of accuracy.

The biomechanical function of the vertebral column is to allow movements between vertebral bodies, carry and

transmit loads and protect the spinal cord and nerve roots (Panjabi 1992a). Panjabi (1992a) classifies the vertebral stabilising system into three parts:

- The passive musculoskeletal subsystem
- The active musculoskeletal subsystem
- The neural and feedback subsystem

The passive musculoskeletal subsystem includes the vertebrae, facet articulations, intervertebral discs, spinal ligaments and joint capsules, plus the passive mechanical properties of muscle.

The mechanical properties of facet joints are determined by the inclination of angle of the facets, and also by any hypertrophy or degeneration due to dysfunction (Panjabi 1992a). Ligamentous components of the passive subsystem do not contribute significantly to stability near the neutral position of the joint. Ligaments develop reactive forces towards the end of physiological range of motion – their role in the neutral position becomes an active feedback function, thus they become part of the neural and feedback systems (Panjabi 1992a).

The passive range of motion varies along the length of the vertebral column within individuals and also between species, with higher flexibility in dogs as compared with horses. This is partly because intervertebral discs influence the extent of the motion available at any given level of the vertebral column (Breit *et al.* 2002). The thickness of the equine intervertebral disc is considerably less than that of the dog and the human. The intervertebral discs in the horse account for 10–11% of the length of the vertebral column, whereas in the dog, the intervertebral discs contribute to up to 20% of vertebral column length (Dyce *et al.* 2002).

Following unilateral transection of the anterior longitudinal ligament, *nucleus pulposus* and *annulus fibrosus* of the T6/7 intervertebral discs in dogs, a significant increase in range of motion (ROM) in flexion–extension, lateral flexion and rotation occurred (Takeuchi *et al.* 1999). This suggests the disc and the anterior longitudinal ligament together may have a role in limiting movement in the thoracic spine.

The greatest combined passive and active vertebral range of motion occurs in cats, then the dog. In comparison, the equine spine is extremely limited and is often referred to as being a balance of stability and mobility (Jeffcott and Dalin 1980). In dogs, sagittal flexion and extension of the lumbar spine is used to increase stride length during gallop, but the horse is unable to apply this mechanism to any substantial level because of its larger musculoskeletal scaling (Dyce *et al.* 2002).

4.4 Canine vertebral column

Many of the studies regarding the biomechanics of the canine vertebral column are based on imaging techniques (Breit *et al.* 2002; Breit & Kunzel 2002; Benninger *et al.*

2006). There is some limited *in vivo* kinematic vertebral column research during gait (Schendel *et al.* 1995). This section pertains to biomechanics of the vertebral column of the dog, based mainly on anatomical dissection and imaging techniques in specimens both normal and with varying conditions of the vertebral column. Before the application of these biomechanical principles to the examination and treatment of a dog, the physiotherapist is encouraged to refer to the chapters on manual therapy, orthopaedic and neurological examination, for information regarding contraindications and precautions for the canine vertebral column.

4.4.1 Cervical spine (O/C1–C7)

Atlanto-occipital joint

In dogs the atlanto-occipital joint is formed by the convex condyles of the occiput and the corresponding concave articulating surfaces of the atlas (C1). It allows nodding motion to occur. In humans it is suggested that there is lateral flexion (lateral tilt) and contralateral conjunct rotation, or an oblique tilt due to sliding of the occipital condyles (Kapandji 1974; Penning & Wilmlink 1987). Information regarding such movement at the canine condyles has not yet been confirmed.

Atlantoaxial joint

The atlantoaxial joint is a pivot joint, which primarily allows rotation of the head around the axis (C2) of the spine. Movement of the atlas (C1) occurs around the dens, or odontoid process of the axis. Human studies report there may be some degree of flexion–extension available at C1/2 (Worth 1995), but this has not been documented in the dog. Mechanical instability of the atlantoaxial joint can result from loss of ligamentous support of the dorsal atlantoaxial ligament, due to excess stress from abnormality or absence of the dens. This may result in dorsal displacement of the axis into the spinal canal.

C3–C7

The spinous processes of the caudal cervical vertebral column (C3–7) increase in height and cranial inclination (Dyce *et al.* 2002). The caudal cervical vertebrae have large, oval planar caudal articular processes, which face ventrolaterally, and are angled at approximately 45° and less to the horizontal plane. The planar nature of the caudal articular processes varies slightly between breeds of dog and with age, and joint surfaces have been described as planar, slightly concave, severely concave, convex and sigmoid. Larger dogs tend to have steeper angles of inclination and more concave caudal articular processes in this region of the vertebral column (Breit & Kunzel 2002). The relative horizontal orientation of the caudal cervical facet joint suggests a weight-bearing function, as well as providing movement in sagittal rotation and lateral bending directions. It is suggested also that dogs with more concave facets

have more ability for axial rotation to occur concurrently with lateral bending (Breit & Kunzel 2002). It is thought that a high degree of concavity is a risk factor for relative or absolute stenosis of the vertebral foramen and may be associated with instability, misalignment and degenerative changes in the facet joints and discs. C6 and C7 were found to have the most axial rotation and this correlates with the vertebral levels most commonly associated with neurological compromise.

4.4.2 Thoracic spine (T1–T13)

The bodies of thoracic vertebrae are short, but increase in length caudally from T10 (Dyce *et al.* 2002). In the upper to mid thoracic spine the spinous processes overlap the body of the next most caudal vertebrae. The orientation of facet joints changes from the lower cervical spine to the thoracic spine. In the cranial thoracic spine the facet joints are orientated in a frontal plane, with the cranial articular processes facing dorsally and the caudal processes facing ventrally. This tends to allow lateral movement to occur (Dyce *et al.* 2002). At T11 the spinous process is vertical (the anticlinal vertebrae) and vertebrae caudal to T11 tend to have spinous processes directed cranially. At roughly the anticlinal vertebrae the orientation of the articular facets changes to a more sagittal alignment where the caudal processes face more laterally and the cranial process face more medially, allowing sagittal flexion and extension to occur (Evans 1993; Dyce *et al.* 2002).

There are variations in the degree of sagittal alignment at facet joints in the caudal thoracic spine – some of the caudal articular facets are directed in a truly sagittal alignment, whereas in some specimens it was found that facets have a greater ventral or caudal component (Breit 2002). In small dogs the alignment tends to be more sagittal and in larger dogs, more oblique towards a transverse plane. This occurs most frequently at L3–4 (Breit 2002).

Costovertebral and costotransverse joints

Costovertebral joints are formed by the head of each rib and the costal facets on the vertebrae. They are described as spheroid joints (Budras *et al.* 2002). The coupled movement of rotation and lateral flexion in the thoracic spine showed an increase in motion after resection of the rib-head joint, suggesting the costovertebral joint also has a role in limiting movement in the thoracic spine.

Costotransverse joints are a planar joint between the tubercle of each rib and the transverse process of the vertebrae.

4.4.3 Lumbar spine (L1–L7)

The lumbar vertebral bodies are the longest in the vertebral column, increasing in length caudally. They have long transverse processes that project cranioventrally alongside the preceding vertebral body (Dyce *et al.* 2002). The lumbar spine facet joints display mostly sagittal alignment, with

interlocking of the caudal and cranial articular processes. The caudal articular processes face laterally and the cranial articular processes face medially. In the caudal lumbar spine, Benninger *et al.* (2006); found there are four variations of shapes of facet joint observed on CT scan – straight (28%); angled (14%); arcuate (29%) and round (14%). Some 15% of facet joints were asymmetrical contralaterally. The difference in shape was found to vary with breed. Facet joint angle tended to be more in the transverse plane in caudal segments compared with the more cranial levels of the lumbar spine. Intervertebral disc height increased from L4–5 to L7–S1. The L7–S1 level had a significantly more wedge-shaped disc, thicker ventrally.

According to Benninger *et al.* (2006), there are four major influences on motion pattern in the lumbar spine: height of intervertebral disc, facet joint angle in the transverse plane, facet joint angle difference between levels in the transverse plane, and length of lever arm (distance between the centre of facet joint and dorsal rim of intervertebral disc). Flexion–extension increased with disc height. Flexion–extension also increased with greater facet joint angle in the transverse plane, despite the motion guiding and limiting function of the facet joint. Differences in facet joint angle in the transverse plane between levels affected all motion in all planes. The short lever arm was associated with increased flexion–extension. In summary, the amount of flexion–extension as the major movement present, increased caudally in the lumbar spine.

An *in vivo* kinematic study of canine lumbar intervertebral joints revealed the following values during gait (Schendel *et al.* 1995): axial rotation 1.3°, lateral flexion 4.25°, flexion–extension 1.8°. During ambulation, axial rotation was coupled with contralateral lateral bending.

A more recent *in vitro* study revealed that flexion–extension was variable throughout the lumbar spine, increasing from 5–10° at L4–5, to 40° at L7–S1. The greatest amount of lateral bend was at L4–5, and very little axial rotation was observed at all lumbar segments. Flexion–extension was coupled with slight axial rotation, which increased from cranial to caudal. During lateral flexion and axial rotation the coupling of motion was greatest in the lumbosacral segment, followed by L4–5 (Benninger *et al.* 2006).

4.4.4 Lumbosacral and sacroiliac joint

Lumbosacral joint

The caudal facet joints face mediadorsally and cranial facets face lateroventrally – they are more angled to the transverse plane than the more cranial lumbar joints (Benninger *et al.* 2006) (Figure 4.1). Flexion–extension is significant at this articulation.

Sacroiliac joint (SIJ)

The canine SIJ has a synovial part and an interosseous part. The synovial part of the joint is planar, and crescent shaped on the sacral and iliac surfaces (Gregory *et al.* 1986).



Figure 4.1 Orientation of canine caudal lumbosacral articular facets – cranio-oblique view.



Figure 4.2 Iliac articular surface of canine sacroiliac joint.

Alignment is basically sagittal, with variations in obliquity of alignment between breeds. Dorsal to the synovial part of the joint is a roughened area, the sacral tuberosity, at which an interosseous ligament unites the wings of ilium and sacrum (Evans 1993) (Figure 4.2).

The main movements available at the SIJ are flexion–extension, with a total of range of 7° thought to be available (Gregory *et al.* 1986). The sagittally aligned wings of ilium and sacrum permit very little lateral translation (Breit & Kunzel 2001); however there may be some varying amounts of craniocaudal translation as accessory motion, depending on the conformation of the dog. Loading forces are transmitted through the coxofemoral joint, acetabulum and ilium to sacrum and lumbar spine thus the SIJ is significant in load transfer (Breit & Kunzel 2001).

The canine (SIJ) is affected by conformation, body weight and activity (Breit & Kunzel 2001). A group of researchers have described the orientation of joint surfaces and the variation in orientation that exists between large and small dogs. Using 1093 radiographs of German Shepherds (GSD), Rottweilers and Golden Retrievers, these researchers discovered a more oblique alignment of the sacroiliac joints in Rottweilers and a more sagittal alignment in GSDs and Golden Retrievers (Breit *et al.* 2002). In large dogs, inclination of wings of sacrum was more vertical (lower than 3.2° in 43% GSD), leading to an increased potential for craniocaudal translation at the SIJ. Large dogs, especially Rottweilers, had greater concavity of articular surface to improve interlock – this is thought to be related to high body weight. Relative to body weight, disproportionately low values of the size of SIJ contact area present, especially in large dogs, resulting in higher forces exerted on their SIJ. Forces in large breeds are approximately twice as high as in toy breeds (Breit & Kunzel 2001). A smaller proportion of sacral tuberosity area with respect to auricular surface was found in Bernese Mountain Dogs and Rottweilers compared with Dachshunds and Collies, thus less interosseus ligament area. Due to less interosseus ligament area, the SIJ is less rigid in these breeds and during locomotion, may place more strain on other SIJ ligaments (Breit & Kunzel 2001).

4.5 Equine vertebral column

Knowledge regarding the morphology and kinematics of intervertebral joints or region of joints has been determined in the horse via a series of *in vivo* research papers utilising reflective markers and Steinman pins implanted into vertebral levels to assess kinematics (Audigie *et al.* 1999; Faber *et al.* 2000). A number of *in vitro* studies on kinematics have also provided some insight into equine vertebral and sacroiliac joint kinematics (Townsend & Leach 1984; Denoix 1999; Deguerce *et al.* 2004; Goff *et al.* 2006). Studies using dissection and imaging have documented some of the facet joint orientation in the vertebral column, and anatomic variations affecting vertebral bodies, articulations and spinous processes (Haussler *et al.* 1997; Stubbs *et al.* 2006).

4.5.1 Cervical spine (O/C1–C7)

Atlanto-occipital joint

This joint is a ginglymus formed by the concave articular surfaces of the atlas (C1) and the convex condyles of occiput (Getty 1975). The main movement is flexion–extension, with small amounts of accessory axial rotation and lateral glide (Clayton & Townsend 1989; Getty 1975). The flexion–extension accounts for 32% of total dorsoventral movements of the cervical spine (Clayton & Townsend 1989). Clinical findings in the anaesthetised horse suggest that when the atlanto-occipital joint is in extension, there

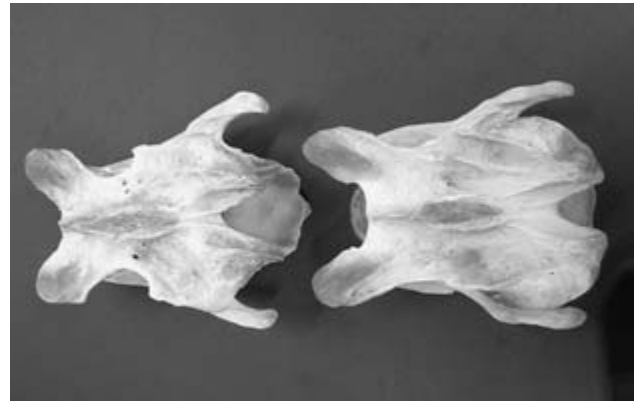


Figure 4.3 Equine cervical spine C3–C4: dorsal view.

may be a greater degree of accessory lateral flexion and rotation movements than when it is in flexion or neutral. This has not yet been documented.

Atlantoaxial joint

This joint is a pivot or trochoid joint formed between the articular surfaces of the atlas (C1) and corresponding saddle-shaped surfaces on the axis (C2), which extend upon the dens, or odontoid process (Getty 1975). The main movement is rotation of the atlas and head upon the axis, with a small amount of accessory lateral flexion. Axial rotation here provides up to 73% of the total axial rotation of the cervical spine (Clayton & Townsend 1989).

C3–C7

The articular surfaces of the cervical spine at these levels are planar, extensive and oval shaped, and oriented obliquely in the transverse plane. The orientation tends to be more transverse more caudally. The cranial articular processes face dorsomedially and the caudal articular processes face ventrolaterally (Mattoon *et al.* 2004) (Figure 4.3). Spinous process height increases caudally from C6. The main movements occurring here are lateral flexion, with mean values of $25\text{--}45^\circ$ for each joint, except C1–2 which had mean lateral flexion of 3.9° (Clayton & Townsend 1989).

4.5.2 Cervicothoracic junction (C7/T1)

Although there is a paucity of information owing to the inaccessibility of the articulation, this junction is a key area of neuromuscular and skeletal anatomy and function for locomotion. This area will be discussed further in species-specific locomotion.

4.5.3 Thoracic spine (T1–T18)

The caudal articular processes of the thoracic spine face ventrally and are placed at the base of the spinous process. The cranial articular processes are oval facets on the arch of the vertebra, which face dorsally. Each thoracic vertebra has a pair of costal facets on the dorsal body, except for the last

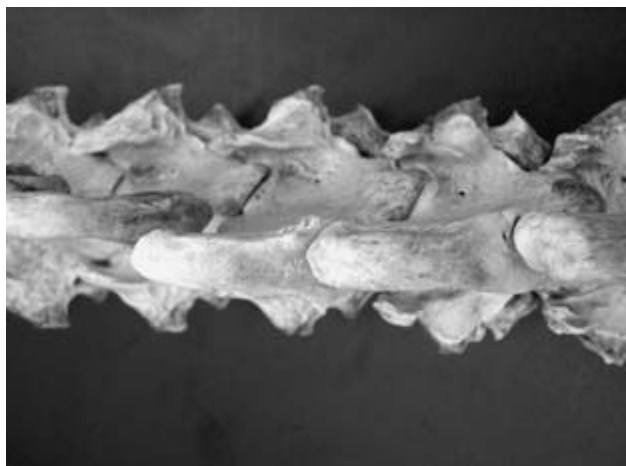


Figure 4.4 Orientation of equine thoracic spine articular facet, dorsal view.

thoracic vertebra, which only has cranial costal facets (Getty 1975) (Figure 4.4). The greatest amount of flexion in the thoracic spine occurs between T17 and T18 (and T18 and L1), with the least amount occurring in the region T3–T9. The greatest extension occurs between T14 and T18, with the least between T2 and T9 (Denoix 1999).

Costovertebral and costotransverse joints

The head of the rib has two convex facets (cranial and caudal) that articulate with the two adjacent thoracic vertebrae. The first rib articulates with C7, T1 and the intervertebral disc. The tubercle of the rib articulates on the transverse process of the caudal vertebra of the adjacent thoracic pair. The movement at these joints is rotation of the rib around an axis that connects the centre of the head and tubercle, and is greater in the caudal ribs (Getty 1975).

4.5.4 Lumbar spine (L1–L6)

The cranial articular processes are fused with the mamillary processes and are concave dorsally, in a mostly sagittal alignment. The caudal articular processes are convex ventrally and correspond to the convexity of the cranial articular surfaces (Getty 1975). The main movements at the lumbar spine are flexion–extension, with lateral flexion and rotation almost non-existent, especially between L4–L6, due to the presence of intertransverse joints (Denoix 1999).

Anatomical variation between equine thoracic and lumbar vertebrae

The bodies of thoracic vertebrae are short and are constricted in the middle (Getty 1975). The length of the tall, narrow thoracic spinous processes increases dramatically from T1 caudally to approximately T4, (the highest point of withers), then diminishes gradually to approximately the anticlinal vertebrae, around T12/13. This is where the spinous processes become more vertically oriented before becoming angled in a cranial direction. At T15 the spinous



Figure 4.5 Equine caudal lumbar sacral spine disarticulated at the lumbosacral joint, dorsal view.

processes remain the same height. The transverse processes of the thoracic vertebrae are short and thick, and reduce in size and are placed more ventrally in the more caudal thoracic vertebrae (Getty 1975; Dyce *et al.* 2002).

The bodies of the lumbar vertebrae become wider and flatter caudally (Getty 1975). Lumbar transverse processes are dorsoventrally flattened and plate like, and project the most laterally to L3, diminishing in lateral projection caudally. The two most caudal transverse processes curve cranially. L4, 5 and 6 (and S1) have articular facets on the transverse processes – the intertransverse joints and these are thought to limit lateral flexion (Haussler *et al.* 1997). Haussler *et al.* (1997) have found that distribution of the intertransverse joints may be asymmetrical in some horses.

4.5.5 Lumbosacral and sacroiliac joint

Lumbosacral joint

The cranial articular processes of the first sacral vertebra are concave and face dorsomedially, for articulation with the caudal articular surfaces of the last lumbar vertebra (Getty 1975) (Figure 4.5). Few studies have investigated the control of intervertebral mobility in the horse. Due to ICR location, lumbosacral (LS) dorsoventral motion is suggested to be an assimilated rotation around the centre of the more caudal vertebral body as with the human (Panjabi *et al.* 1989; Denoix 1999). Lumbosacral dorsoventral motion is guided by intervertebral translation in the lateral part of the left and right intertransverse joints due to the cranial orientation of the L6–S1 transverse process (Denoix 1999). The vertebral body displacement is a result of coupled and accessory intervertebral motion including translation and shearing movements within the intervertebral disc and greater tensile and compressive strain due to the thickness of the disc (Denoix 1987).

The largest motion in the equine thoracolumbar spine occurs at the LS junction in a sagittal plane (flexion–

extension, *in vitro*) of up to 23.4° (Degueurce *et al.* 2004), however Denoix (1987, 1999) reports measurements of $\pm 9\text{--}32^\circ$ from L5 to S1 inclusive. This is thought to be due to: the wide divergence of spinous processes; relative sagittal alignment of the facet joints; thickness and decreased height of the intervertebral disc compared with other vertebral levels (Denoix 1999); poorly developed interspinous ligament and absence of supraspinous ligament (Jeffcott & Dalin 1980); and the vertical orientation of the articular facets (Townsend & Leach 1984) (Figure 4.5).

Variations in the numbers of thoracolumbar and lumbosacral vertebrae (vertebral formula) have been widely reported (Rooney 1969; Getty 1975; Jeffcott 1979; Townsend 1987; Haussler *et al.* 1997). In Thoroughbreds it has been reported that only 61% have the normal vertebral formula (cervical 7, thoracic 18, lumbar 6, sacral 5, caudal vertebrae 15–21) (Haussler *et al.* 1997). Similarly Stubbs *et al.* (2006) found that normal lumbosacral vertebral formula only existed in 67% of 120 horses examined. This occurred in 60% of Thoroughbreds (TB), 100% Standardbreds (SB) and 55% others (OB). In a study of 36 racehorses, Haussler (1997) also reported that a transitional vertebra existed in over 20% horses in the thoracolumbar region and in over a third of horses in the sacrocaudal regions. The divergence of the spinous processes generally takes place between L6 and S1, but may occur between L5 and L6 (Haussler *et al.* 1997; Denoix 1998). A quantitative LS variation of the spinous process orientation relative to the vertebral body and relationship with breed in 120 horses has been reported by Stubbs *et al.* (2006). LS variations were found in a third of horses. In 8% of horses there were only five lumbar vertebrae and maximum dorsoventral motion was at L5–S1. Over all, 25% had the conventional L6–S1 formula, but with spinous process/vertebral orientation divergence of L5 cranially and L6 caudally, and interspinous muscle between L5 and L6 (Figure 4.6). The divergence of the spinous processes between L5–S1 may influence spinal mobility at the point of greatest dorsoventral motion and therefore affect performance and development of pathology in the LS region.

Lumbosacral movement is greater when the spine is in flexion rather than extension. Lateral flexion and rotation accessory movements are very small and are more likely to occur when the segment is in relative flexion (Denoix 1999). The high prevalence of L5–L6 spinal variations may have an effect on the mobility in the LS region that may lead to altered function, performance and pathology.

Sacroiliac joint

As in the dog, the sacroiliac joint (SIJ) is responsible for transmission of forces from the hindlimb to the thoracolumbar vertebral column and forequarter. The equine SIJ has a synovial part and an interosseus part. The synovial part consists of L-shaped articular surfaces on the ilium and the sacrum. The interosseus part lies dorsocaudally to the synovial part and here the interosseous ligament connects



Figure 4.6 Variations in the number and orientation of the equine lumbar vertebrae exist, particularly at the lumbosacral junction. This horse demonstrates the common L5–L6 divergence where L6 is orientated with the sacrum. Note the interspinous muscle present only between the level of greatest divergence of spinous processes (here L5–L6).

the wings of the ilium to the sacrum. The plane of synovial part of the joint is 30° to horizontal (Dalin & Jeffcott 1986).

Preliminary *in vitro* studies have revealed that the largest movement available at the equine SIJ is in the coronal plane, that is, a lateral movement of $2.56 \pm 0.29^\circ$ (Goff *et al.* 2006). This was measured during a lateral rotation, or movement of the pelvis on the fixed sacrum. Previously, Degueurce *et al.* (2004) had measured an average of just less than 1° of nutation at sacrum, which is a rotational movement in the sagittal plane, however these authors had not tested motion in lateral directions.

Note: rotation refers to the movement of the ventral aspect of the vertebral body, that is, left rotation involves movement of vertebral body to the left (relative movement of the spinous process to the right) (Denoix 1999).

4.5.6 Summary

There are many assumptions made regarding the contribution of facet joints, intervertebral discs and other structures to stability and/or movement of the vertebral column, based on morphology and observation. As some studies have shown, a given motion segment of the spine does not necessarily behave in the manner predicted by morphology. We can only be guided by the current knowledge of anatomy and morphology and the growing field of kinematics and motion analysis in the canine and equine vertebral columns.

4.6 Canine peripheral joints

Compared with the canine vertebral column, there has been very little kinematic research in the canine peripheral joints. Table 4.1 provides a summary of the peripheral joints, noting the type of articulation, the main direction of motion available at the joint, as well as conjunct motions available.

Table 4.1 A summary of the canine extremity joints: joint type, articular surfaces, the primary motion of the joint and the accessory movements that occur at each complex. Adapted from Budras *et al.* (2002) and Evans (1993)

Joint	Joint type and articular surfaces	Main movement	Accessory movement
Glenohumeral	Spheroid, between the glenoid cavity of the scapula and the head of humerus, with the glenoid fossa on the scapula	Flexion and extension	Rotation
Elbow	Composite joint formed by the humeral condyle and the head of the radius (humeroradial joint) and the semilunar notch of the ulna (humeroulnar joint) – ginglymus joints. Proximal radioulnar joint communicates with the main elbow joint – trochoid joint	Flexion–extension. Rotation occurs at the radioulnar joint (and carpal joints) so that about 90° of supination of the forepaws can be achieved	Lateral translations minimal due to strong collateral ligaments and the anconeus of the ulna
Carpus	Composite articulation, which involves the proximal, middle, carpometacarpal and intercarpal joints. Proximal carpal joint is a ginglymus between the distal end of the radius and ulna and the proximal carpal row. The middle carpal joint is a compound condylar joint between the proximal and distal rows of carpals. The carpometacarpal joint is a compound plane joint between the distal carpals and the metacarpus. The intercarpal joints are compound planar joints between the carpal bones of each row	As a whole joint, flexion–extension. The majority of the movement occurs at the proximal and middle carpal joints	Lateral movement
Metacarpophalangeal joint	Compound articulation between proximal phalanges, proximal sesamoid bones, dorsal sesamoid bones and metacarpals	Flexion and extension	Ab/adduction and axial rotation
Proximal interphalangeal joint	Saddle joint between proximal and middle phalanges (Forelimb and hindlimb)	Flexion and extension	Axial rotation and lateral movements
Distal interphalangeal joint	Saddle joint between middle and distal phalanges (Forelimb and hindlimb)	Flexion and extension	Axial rotation and lateral movements
Coxofemoral joint (Hip)	Spheroid joint, articulation between the femoral head and the acetabulum of the ilium, ischium and pubis. The acetabulum is deepened by a band of fibrocartilage on the rim of the acetabulum	Flexion and extension are the main movements	Abduction and adduction, multidirectional
Stifle	Complex joint comprising the tibiofemoral joint (condylar) and the patellofemoral joint (simple, sliding joint). At the tibiofemoral joint, the convex femoral condyles articulate with the planar tibial plateau. The incongruity of this joint is improved by the two menisci, into which each condyle fits. The patellofemoral joint is between the patella and the trochlea of the femur	The main movements at the stifle are flexion–extension at the tibiofemoral joint, with the patella gliding in the trochlea during the movement	Accessory craniocaudal movements are limited by the cruciate ligaments, the collateral ligaments and the concave nature of the menisci
Proximal tibiofibular	Simple plane joint between tibia and head of fibula	Minimal gliding movement	
Distal tibiofibular	Simple plane joint between distal tibia and fibula	Minimal gliding movement	
Tarsal joint (Hock)	The hock complex includes the talocrural joint (cochlear joint) proximal and distal intertarsal joints, tarsometatarsal joint (compound plane joints) and intertarsal joints (perpendicular tight joints). The greatest amount of movement occurs at the talocrural joint	At the talocrural joint, flexion–extension, in a plane that deviates about 25° from the sagittal plane. This allows the hindpaws to pass the forepaws laterally in full gallop. Slight flexion–extension available at the proximal intertarsal joint. Little mobility at distal intertarsal, tarsometatarsal and intertarsal joints	Slight rotation at talocrural and proximal intertarsal joint
Metatarsophalangeal joint	See forelimb		
Proximal and distal interphalangeal joints	See forelimb		
Temporomandibular	Is a simple condylar joint which allows translatory movement, with an articular disc	Hinge – opening and closing	Lateromedial excursion. Increased opening is associated with upper cervical extension

Table 4.2 A summary of the equine peripheral joints: joint type, articular surfaces, the primary motion of the joint and the accessory movements that occur at each complex. Adapted from Budras *et al.* (2002)

Joint	Joint type and articular surfaces	Main movement	Accessory movement
Glenohumeral	Spheroid, between the glenoid cavity of the scapula and the head of humerus, with the glenoid fossa on the scapula deepened by the glenoid labrum	Flexion and extension	Rotation and minimal ab/adduction
Elbow	Composite joint formed by the humeral condyle and the head of the radius (humeroradial joint) and the semilunar notch of the ulna (humeroulnar joint) – both simple hinge joints. Proximal radioulnar joint communicates with the main elbow joint – a simple pivot joint	Flexion–extension. No movement at proximal radioulnar joint	Minimal
Carpus	Composite joint made up of radiocarpal joint involving trochlea of radius and carpals (condylar); midcarpal joint involving proximal and distal carpal rows (condylar); carpometacarpal joint involving carpal bones II–IV and metacarpals II–IV (plane) and intercarpal joints involving carpals of the same row (plane)	Flexion–extension at radiocarpal (up to 90°); flexion–extension at midcarpal (up to 45°); carpometacarpal joint little planar motion; intercarpal joint little planar movements	Slight accessory rotation and lateral glide at radiocarpal joint
Metacarpophalangeal joint (Fetlock)	Compound articulation between third metacarpal, proximal phalanx and proximal sesamoid bones – composite hinge joint	Flexion and extension	During flexion, small amounts of ab/adduction and axial rotation
Proximal interphalangeal joint (Pastern)	Simple saddle joint between proximal and middle phalanx. (Forelimb and hindlimb)	Flexion and extension	Axial rotation and lateral movements
Distal interphalangeal joint (Coffin joint)	Composite saddle joint between middle phalanx, distal phalanx, with hoof cartilage and navicular bone (Forelimb and hindlimb)	Flexion and extension	Axial rotation and lateral movements
Coxofemoral joint (Hip)	Composite spheroid joint, articulation between the femoral head and the acetabulum of the ilium, ischium and pubis. The acetabulum is deepened by a band of fibrocartilage on the rim of the acetabulum	Flexion and extension are the main movements	Multidirectional minimal abduction/adduction
Stifle	Complex joint, comprising the tibiofemoral joint (simple condylar) and the patellofemoral joint (simple, gliding joint). At the tibiofemoral joint, the convex femoral condyles articulate with the tibial condyles. The patellofemoral joint is between the patella and the trochlea of the femur	The main movements at the stifle are flexion–extension at the tibiofemoral joint, with the patella gliding in the trochlea during the movement	Tibiofemoral joint – at extreme extension there is accessory external rotation, and with flexion, accessory internal rotation
Tarsal joint (Hock)	The hock complex includes the tarsocrural joint (simple cochlear joint) proximal and distal intertarsal joints, tarsometatarsal joint (composite plane joints) and intertarsal joints (perpendicular tight joints). The greatest amount of movement occurs at the tarsocrural joint	At the tarsocrural joint, flexion–extension. The intertarsal, proximal and distal tarsal (tarsometatarsal) joints undergo small amounts of translatory and rotatory movements during locomotion	At the tarsocrural joint, lateral and rotatory accessory movements
Metatarsophalangeal joint	See forelimb		
Proximal and distal interphalangeal joints	See forelimb		
Temporomandibular	Is a simple condylar joint which allows translatory movement, with an articular disc	Hinge – opening and closing	Lateromedial excursion; rostral glide of mandible with opening

4.7 Equine peripheral joints

Due to the interest in equine locomotion, there is a significantly larger bank of biomechanics research regarding the equine peripheral joints than for those of the canine; both relating to kinematics and forces about the joints

during gait. Table 4.2 summarises the equine peripheral joints, but there are notes in the text, particularly regarding joint forces during gait. Two very comprehensive texts summarising a large portion of the current literature related to equine locomotion and biomechanics are; Back & Clayton (2001) *Equine Locomotion*, and Hodgson & Rose

(1994) *The Athletic Horse: Principles and practice of equine sports medicine*. These books may be a useful adjunct for those physiotherapists working with horses.

Scapulothoracic joint

The horse has no clavicle, so the thoracic limb is attached to the trunk via muscles – a *synsarcosis* (Budras *et al.* 2001), and also the dorsal scapular ligament. The movement of the shoulder on the thorax is rotation around a transverse axis passing through the scapula caudal to the dorsal part of the scapular spine (Getty 1975).

Glenohumeral joint

The glenohumeral articulation is formed between the distal end of the scapula (glenoid cavity) and the head of the humerus (Getty 1975). The main movement at the shoulder joint is flexion and extension. In stance, the angle between scapula and humerus is approximately 120°. There are some accessory rotatory movements, which have been noted when the stabilising muscles are removed. When the horse is not weight bearing on the limb, rotation can be achieved manually, however no motion measurements have been found in the literature (Getty 1975). This may implicate soft tissues such as the *lacertus fibrosus*, which may have a similar role to the dynamic stabilising muscles in the human.

The shoulder joint extends during most of swing phase of walk, to ground contact and early stance phase (Hodson *et al.* 2000). During early stance phase the shoulder flexes and then tends to maintain a constant angle during periods of bipedal support, and flexes slightly during tripod support phase. At breakover the shoulder flexes further. The shoulder has been described as acting as an energy damper during stance phase of the walk, and also shows absorption of power during swing phase (Clayton *et al.* 2000).

Elbow

The elbow is a ginglymus between the distal trochlear surface of the humerus and the fovea of the proximal radius plus trochlear notch of the ulna (Getty 1975). The movements available are flexion and extension. In stance, the articular angle is 150°. There is little appreciable movement at the radioulnar joint, with the forearm being fixed in pronation (Getty 1975).

The elbow remains at a constant angle throughout the first 7% of walking stride, then, during breakover, which occurs between heel off (55% of stride) and lift off (64% of stride) it moves into flexion. The elbow shows a single flexion cycle during swing that elevates the distal limb during that phase. It reaches peak flexion at 84% of stride during swing phase (Hodson *et al.* 2000). The elbow shows net generation of energy to maintain the limb in extension during early stance phase and is the main joint of energy generation during walk gait in the forelimb (Clayton *et al.* 2000).

Carpus

There are three joints of the carpus:

- *antebrachio-carpal* (radiocarpal) joint (between the distal radius and ulna and proximal carpal row);
- *intercarpal* joint (between proximal and distal carpal rows); and
- *carpometacarpal* joint (between distal carpal row and proximal ends of metacarpals).

The proximal and middle joints are ginglymi, but the distal joint is planar. The joints formed between the adjacent carpal bones of each row are also planar (Getty 1975). Main movement of the carpus as a whole is flexion–extension. With flexion there is slight accessory rotation and lateral glide available. These movements occur mostly at the radio-carpal joint and intercarpal joints.

Just after initial ground contact during walking gait, the carpus rapidly assumes its close packed position between 7 and 12% of stride, to allow the limb to act like a propulsive strut through stance phase (Hodson *et al.* 2000; Clayton *et al.* 2001). The carpus then does not flex until breakover, with peak flexion occurring at 76% of stride. The carpus does not play an important role in energy absorption or generation during walking gait, but plays an active role in initiating breakover (Clayton *et al.* 2000).

Metacarpophalangeal joint

The fetlock, or metacarpophalangeal joint, is a ginglymus formed between the distal third metacarpal and the proximal end of the proximal phalanx. In stance the joint is an extension angle of 140° (approximately 150° in the hind fetlock). The main movements at the fetlock are flexion–extension. During flexion, accessory movements of abduction, adduction and rotation can occur (Getty 1975).

The fetlock extends through early stance phase of walk. Maximal extension occurs at around 34% of stride, when forces during gait change from braking to propulsive (Hodson *et al.* 2000). After this point the fetlock flexes for the remainder of stance phase. It continues to flex during breakover, with peak flexion occurring at 82% of stride, during swing. The fetlock has been described as functioning elastically, as there is an initial absorption of energy during early stance and bursts of energy generation in late stance and during breakover. It shows bursts of energy absorption also during swing phase, at 86% of stride (Clayton *et al.* 2000).

Pastern joint and coffin joint (Fore)

The pastern joint is the articulation of the proximal and middle phalanges and is classified as a ginglymus (Getty 1975). The joint is extended in stance. The main movement at the pastern joint is flexion–extension, which moves through 35° during the stance phase (Clayton *et al.* 2000). Accessory movements of medial and lateral flexion are available when the joint is flexed (Getty 1975).

The coffin joint is the articulation between the middle and distal phalanges and is in contact on the palmar aspect with the navicular (distal sesamoid) bone (Getty 1975). In stance the joint is extended, and the main movements at the joint are flexion–extension. Accessory movements of lateral and medial flexion and rotation are available when the joint is in relative flexion (Getty 1975). Flexion–extension patterns in the pastern joint appear to mirror that of the coffin joint (Clayton *et al.* 2000). The pastern joint flexes for up to 10% of the stride (early stance) then reverses direction after this point. Flexion then occurs again during breakover and shows peak flexion during swing, at 84% of total stride (Hodson *et al.* 2000). The coffin joint has been described as an energy damper during stance, with a small amount of energy generation at the beginning of breakover (Clayton *et al.* 2000).

Coxofemoral (Hip) joint

The coxofemoral joint is the articulation formed by the head of the femur and the deep ilial acetabulum bounded by a rim of fibrocartilage. Two ligaments, the ligament of the femoral head and the accessory ligament limit internal rotation and abduction of the hip joint. Thus the main movements are primarily flexion and extension, which are responsible for protraction and retraction of the entire hind limb during walking gait (Hodson *et al.* 2001). Maximal protraction occurs just before the end of swing phase and maximal retraction occurs at breakover. The hip joint is the main source of energy generation during stride, at the walk (Clayton 2001a).

Tibiofemoral and patellofemoral articulation (Stifle)

The stifle is made up of the tibiofemoral and patellofemoral joints. The congruence of this tibiofemoral joint is enhanced by the menisci. The patella glides proximally and distally on the trochlea during tibiofemoral extension and flexion, respectively (Getty 1975).

In the standing position the articular angle is 150° (Getty 1975). The main movements at the tibiofemoral joint are flexion and extension, with the accessory translation of the tibia in a craniocaudal direction restricted by the cruciate ligaments (Clayton 2001a). At extreme extension there is accessory external rotation, and with flexion, accessory internal rotation (Getty 1975).

At walk, during the initial 10% of stride, which is a period of rapid loading, the stifle joint flexes (Hodson *et al.* 2001). The stifle begins to flex further when the hind limb is retracted beyond the midstance position, and flexion of stifle occurs with the swing phase and protraction of the limb, with the hock, which raises the distal limb. The stifle begins to extend in preparation for ground contact at about 80% of total stride.

The stifle joint absorbs equal amounts of energy in the stance and the swing phase of walk (Clayton 2001a).

Tarsocrural and tarsometatarsal joint (Hock)

The hock is a composition of articulations, with most of the movement occurring at the most proximal joint, the tarsocrural joint, which is classified as a ginglymus (Getty 1975). In the standing position the angle of the hock is approximately 150° (Getty 1975). The distal tibia rotates around the trochlea of the talus, allowing the main movement of flexion–extension to occur, along with lateral and rotatory accessory movements. These articular surfaces are directed obliquely dorsal and laterally at an angle of 12–15° (Getty 1975).

The intertarsal and distal tarsal (tarsometatarsal) joints undergo small amounts of translatory and rotatory movements during locomotion. Clayton (2001b) presents some kinematic data on the movement at the distal tarsal joints, as this is most often the site of bone spavin. During the stance phase of walk, the cannon bone internally rotates at the distal joints and then slides cranially. This cranial slide becomes ‘de-coupled’ in swing by the time the hock is flexed to 50°, and re-couples later in swing as the joint reaches the same angle. During swing phase, at about 80% of stride, the hock reaches peak flexion along with the stifle (Hodson *et al.* 2001). After this, the hock extends in preparation for ground contact.

The hock joint assists the hips in generation of energy of stride during both stance and swing phases of walk (Clayton 2001b).

Metatarsophalangeal joint (Hind)

During the initial 10% of walking stride, a period of rapid loading, the fetlock joint extends (Hodson *et al.* 2001).

Pastern joint/coffin joint (Hind)

At 5% of stride (early stance phase) the coffin joint shows a peak in flexion. The coffin joint shows a peak in flexion at 80% of stride during swing phase. After this point it extends in preparation for ground contact (Hodson *et al.* 2001).

The biomechanics of the fetlock, pastern and coffin joints in the hindlimb have been likened to those of the forelimb (Getty 1975).

Temporomandibular joint

The temporomandibular joint (TMJ) is a complex diarthrodial joint formed between the articular tubercles of the temporal bone and the condylar processes of the mandible. A fibrocartilagenous disc improves the congruency between the articular surfaces, and divides the joint into a dorsal and a ventral compartment (Maierl *et al.* 2000; Moll & May 2002; Baker 2002). The mandibular condyles are at an angle of 15° in a plane that runs laterocaudal to ventromedial and a plane that runs mediocaudal to laterorostral. TMJ movements are around a transverse axis. When the mouth opens, the mandibular condyle moves slightly in a rostral direction (Baker 2002).

4.7.1 Summary

Compared with the vertebral column there has been little kinematic research carried out in the peripheral joints, particularly in the canine. In the equine there has been some data developed regarding forces and torques acting about the peripheral joints. As with the vertebral column, physiotherapists can only be guided by the current knowledge of anatomy and morphology and the growing field of kinematics and motion analysis in the canine and equine peripheral joints.

4.8 Biomechanics of locomotion: the dog

Kinematic analysis of gait in the dog has been limited to date, but there is a growing interest in the area (Hottinger *et al.* 1996) motivated by responses of dogs to surgical procedures (Robinson *et al.* 2006), orthopaedic conditions (Evans *et al.* 2005), and breed differences (Colborne *et al.* 2004; Besancon *et al.* 2005).

Establishment of gait analysis in the normal subject has been attempted to be carried out, using force-plate analysis and skin-mounted markers to describe flexion–extension movements in the joints of healthy Greyhounds, at the trot (DeCamp *et al.* 1993). These authors point out that the shape of the joint angle/time curve is intrinsic to an animal's limb conformation. Thus, in dogs, analysis of gait will be specific for the breed of dog.

Despite choosing the Greyhound, because of uniformity of body conformation and temperament, DeCamp *et al.* (1993) discovered variances in joint angles during the swing phase, attributable to trial repetition, with the carpus showing most variance with a mean trial repetition variance. In stance phase, each joint was characterised by peaks of extension – the coxofemoral joint had a single peak towards the end of stance phase; the femorotibial joint had two peaks of extension with maximal extension preceding stance phase; the tarsal and elbow joints had two peaks of extension, as did the scapulohumeral joint. The carpal joint had one peak of extension early in the stance phase and then rapid flexion initiated to the end of stance (DeCamp *et al.* 1993). Movement of skin markers due to skin movement, muscle contraction, and other soft tissue movement may have contributed to the variance in measurement of joint angles during swing. It is as a result of such problems that McLaughlin (2001) reports that data for the swing phase in dogs is minimal.

Force-plate data collection

The dog is led across a force plate by a handler; in a consistent manner, with no interference from the handler. Trials consist of ipsilateral forefoot and hindfoot strikes, and for each valid trial three orthogonal ground reaction forces are recorded (McLaughlin 2001). Velocity of the dog is measured and sometimes accelerometers are used as acceleration and deceleration affect force values. Ground reaction force

data are presented in a force–time curve, or numerically (McLaughlin 2001). Vertical force is usually the largest of the orthogonal forces, with mediolateral and craniocaudal braking forces generally smaller. Force data are also normalised with respect to the dog's body weight, (McLaughlin 2001) which means dogs of different breeds can be compared.

Walking data

Walking in the quadruped involves a cyclic exchange of gravitational potential energy and kinetic energy of the centre of mass. In a study by Griffin *et al.* (2004) kinematic and ground reaction force data were collected from dogs walking over a range of speeds. The authors found that the forequarters and hindquarters of dogs behaved like two independent bipeds, with the centre of mass moving up and down twice per stride. Up to 70% of the mechanical energy required to lift and accelerate the centre of mass was recovered via a mechanism likened to an inverted pendulum (Griffin *et al.* 2004). Using a model of two inverted pendulums, these authors concluded that there are two reasons why dogs did not walk with a flat trajectory of the centre of mass:

1. Each forelimb lagged its ipsilateral hindlimb by only 15% of the stride time – this produced time periods when the forequarters and hindquarters moved up or down simultaneously.
2. Forelimbs supported 63% of body mass during gait. (This is consistent with during static four-legged weight bearing.)

The model proposed here predicts that the centre of mass of a dog will undergo two fluctuations per stride cycle.

In an attempt to establish some normative data in large-breed dogs, Hottinger *et al.* (1996) have presented data on the stance and swing phase of gait at the walk, pertaining to the joint angles, total time of stance and swing phase of each limb. It is beyond the scope of this chapter to reproduce the author's data, but the reader is directed to this research as a useful resource.

Lameness data

A population of adult Labrador Retrievers – 17 subjects free of orthopaedic and neurologic abnormalities, 100 with unilateral cranial cruciate ligament (CCL) rupture, and 131 studied 6 months after surgery for unilateral CCL injury, 15 with observable lameness – were walked over a force platform, with ground reaction force (GRF) recorded during the stance phase (Evans *et al.* 2005). The probability of visual observation detecting a gait abnormality was compared with that of force platform gait analysis. During the stance phase, it was determined that a combination of peak vertical force (PVF) and falling slope were optimal for discriminating sound and lame Labradors. After surgery, 75% of subjects with no observable lameness failed to achieve GRFs

consistent with sound Labradors. The authors conclude that a force platform is an accurate method of assessing lameness in Labradors with CCL rupture and is more sensitive than visual observation. This has clinical relevance for animal physiotherapists as interventions for stifle lameness can be accurately and objectively evaluated using two vertical ground reaction forces obtained from a force platform.

Another group of researchers assessed the relationship between post-operative tibial plateau angle (TPA) and GRFs in Labrador Retrievers at least 4 months after tibial plateau levelling osteotomy (TPLO) surgery (Robinson *et al.* 2006). Thirty-two Labrador Retrievers with unilateral cranial cruciate ligament disease that had TPLO and concurrent meniscal surgery were studied. Both TPA and GRFs were measured before surgery and a time greater than or equal to 4 months after surgery. The GRFs, TPA, duration of injury preoperatively, post-operative TPA and degree of rotation were each compared with post-operative GRFs. No significant relationship was found between pre-operative GRFs, pre-operative TPA, duration of injury, post-operative TPA, degree of rotation, or meniscal release/meniscectomy and post-operative function, suggesting limb function in Labrador Retrievers was not affected by post-operative TPA.

Breed and sports-specific data

Comparisons of breeds have revealed some consistencies between breeds regarding PVF and vertical impulse in the pads of Greyhounds and Labrador Retrievers. Besancon *et al.* (2005) compared eight Greyhounds and eight Labrador Retrievers to discover that digital pads 3 and 4 are the major weight-bearing pads in dogs. The loads were found to be fairly evenly distributed between breeds, and digital pad 5 and the metacarpal or metatarsal pad were found to bear a substantial amount of load in both breeds.

Colborne *et al.* (2004) investigated the angular excursions, net joint moments and powers across the stifle, tarsal, and metatarsophalangeal (MTP) joints in Labrador Retrievers and Greyhounds to investigate differences in joint mechanics between these two breeds of dogs. Not surprisingly, there were gross differences in kinematic patterns between Greyhounds and Labrador Retrievers. At the stifle and tarsal joints, moment and power patterns were similar in shape, but amplitudes were larger for the Greyhounds. The MTP joint was found to be a net absorber of energy, and this was greater in the Greyhounds. Greyhounds had a positive phase across the stifle, tarsal, and MTP joints at the end of stance for an active push-off, whereas for the Labrador Retrievers, the only positive phase was across the tarsus, and this was small, compared with values for the Greyhounds. This is clinically significant for animal physiotherapists, to take into consideration the conformation of the dog when considering biomechanics of locomotion, and the potential for certain pathologies to occur in different breeds. In addition, the occupation, or sport of the dog needs to be considered.

Kemp *et al.* (2005) tested an hypothesis of functional trade-off in limb bones by measuring the mechanical properties of limb bones in two breeds of domestic dog that have undergone intense artificial selection for; running (Greyhound) and fighting (Pit Bull) performance. They postulate that the physical demands of rapid and economical running would differ from the demands of fighting in ways that may prevent the simultaneous evolution of optimal performance in these two sports. The bones were loaded to fracture in three-point static bending. In Pit Bulls, the proximal limb bones differed from those of the Greyhounds in having relatively larger second moments of area of mid-diaphyseal cross-sections and in having more circular cross-sectional shape. The Pit Bulls exhibited lower stresses at yield, had lower elastic moduli, and failed at much higher levels of work. In the Greyhound, the stiffness of the tissue of the humerus, radius, femur and tibia was 1.5–2.4-fold greater than in the Pit Bulls. These differences between breeds were not observed in the long bones of the feet, metacarpals and metatarsals. These authors conclude that selection for high-speed running is associated with the evolution of relatively stiff limb bones, whereas selection for fighting performance leads to the evolution of limb bones with relatively high resistance to failure.

Speed of running is constrained by the speed at which the limbs can be swung forwards and backwards, and by the force they can withstand while in contact with the ground. Regarding sprinting Greyhounds, Usherwood & Wilson (2005) have shown that, on entering a tight bend, Greyhounds, unlike humans sprinting around banked bends, do not change their foot-contact timings. Greyhounds have to withstand a 65% increase in limb forces, whereas humans change the duration of foot contact to spread the time over which the load is applied, thereby keeping the force on their legs constant. These authors conclude there is no force limit on Greyhound sprint speed – they suggest that Greyhounds power their locomotion by torque about the hips, so that the muscles that provide the power are mechanically divorced from the structures that support weight.

4.9 Biomechanics of locomotion: the horse

This section includes an overview of the equine anatomical and biomechanical adaptations which allow this animal to be energy efficient and travel at relatively high speeds over moderate distance, even though it is a large mass. The horse, like the dog, locates approximately 57% of body weight on the forelimbs at rest, with this load increasing during locomotion (Schamhardt 1998). However, the forelimbs of the horse have adapted to a primary support role, providing little propulsive force, while the hindlimb supports less weight but provides more propulsion (Wilson *et al.* 2000). To achieve this, the forelimbs act as energy efficient springs, which store and release energy,

decreasing the cost of locomotion. The structures that are biomechanically unique to equine locomotion are described below.

As the size of an animal doubles, the weight of the animal is cubed, yet the cross-sectional area of the limb musculature is only squared (Wilson *et al.* 2000). This means to continue functioning, the animal must have grossly large muscle mass in the limbs to support the weight of the animal. To compensate for this, the horse has undergone many evolutionary adaptations to better meet the needs of an herbivorous quadruped while decreasing the cost of locomotion. These adaptations include an increase in the length of the limbs, restriction (via changed osteology) of the available range of movement in the limbs, and replacement of muscle tissue in the lower limbs with elastic tendons. This decreases the weight of the limb while increasing the capacity for energy storage, therefore decreasing energy cost of locomotion. Muscles are located at the proximal end of the limb to reduce inertia, as the muscles are closer to joint centres of rotation (e.g. spinning ice skater extending his/her arms). This adaptation has also occurred in the Greyhound, which is bred to sprint. The Greyhound is a long-legged dog, with large proximal muscle mass and light distal limbs.

There are mechanisms in the horse that increase the efficiency of locomotion as well as the efficiency of the horse's energy expenditure at rest. The passive stay apparatus of the forelimbs and hindlimbs will be described here, and the actions of these mechanisms during locomotion will be outlined further on in this section.

The passive stay apparatus in the horse

The forelimb passive stay apparatus allows the horse to rest on its feet, and cope with the stance phase of locomotion, with minimal muscular effort. In the forelimb, it involves the synsarcosis and all the joints distal to the pastern joint, the suspensory apparatus, and superficial and deep digital flexor tendons (SDFT and DDFT) (Budras *et al.* 2001). At the synsarcosis the serratus ventralis is the principal weight-bearing connection and contains a large amount of tendinous tissue. The biceps tendon position relative to the cranial surface of the glenohumeral joint – in the intertubercular groove – has a stabilising role. The joint is further stabilised by the biceps tendon anchoring the muscle to the proximal radius, and via the lacertus fibrosus and extensor carpi radialis (ECR), to the proximal third metatarsal. The weight of the trunk at the proximal scapula tenses the biceps–lacertus–ECR, causing relative extension at the elbow and the carpus.

The elbow is in turn further prevented from flexing by the carpal and digital flexors that arise from the epicondyles of the humerus. The carpus is stabilised by the ECR tendon. The attachment of flexor carpi ulnaris and ulnaris lateralis to the accessory carpal bone tends to keep the carpus extended. The fetlock is prevented from further extending

by the suspensory apparatus that is associated with the interosseus tendon and the superficial and deep digital flexor tendons (SDFT and DDFT respectively).

The suspensory apparatus

The interosseous ligament arises from the carpus and the proximal third metacarpal and attaches to the proximal sesamoid bones. As it descends it splits and sends extensor branches around the proximal phalanx to the common extensor tendon. Collateral ligaments attach the sesamoids to the metacarpal and proximal phalanx and a palmar ligament unites the sesamoids and forms a bearing surface for the flexor tendons. The tension in the interosseous ligament is carried distally by four sesamoidean ligaments.

The SDFT assists the suspensory apparatus via its accessory (check) ligament from the radius above the carpus to the proximal and middle phalanges. The DDFT and its check ligament provide additional support – this accessory ligament arises with the interosseous from the caudal aspect of the carpus and ends on the distal phalanx.

The suspensory apparatus acts to limit hyperextension at the metacarpophalangeal joint via the suspensory ligament, proximal sesamoidean ligaments, palmar ligaments, and superior and inferior check ligaments. The deep and superficial flexor tendons act as high-tension cables to support the passive ligamentous restraints via a powerful flexion moment.

Hindlimb

The ability for the horse to prevent collapse of the hindlimb with minimal muscular effort involves the stifle-locking mechanism, the reciprocal apparatus/mechanism of stifle, hock and fetlock and the suspensory mechanism, which is similar to the forelimb.

Locking of the stifle is related to the larger medial ridge on the femoral trochlea, and its proximal tubercle, the patella and divergence of the intermediate and medial patellar ligaments. The medial trochlea sits in between these two patellar ligaments. The trochlear surface has two parts – the larger, gliding surface faces cranially and the smaller resting surface forms a narrow shelf above the gliding surface. Even in hindlimb weight bearing the patella sits at the proximal end of the trochlea. When the horse rests a hindlimb, the patella on the supporting leg rotates medially about 15° and the medial patellar ligament slides caudally on the tubercle of the medial ridge, thus hooking the patella (via the parapatellar cartilage) on the tubercle, where it resists displacement. This converts the jointed column of the hindlimb to a weight-bearing strut. A conscious contraction of the quadriceps is required to unlock the patella from the tubercle by laterally rotating the patella (Budras *et al.* 2001; Dyce *et al.* 2002).

The *reciprocal mechanism* is provided by the tendinous peroneus tertius and the SDFT. These pass between the distal end of the femur and the hock – the peroneus tertius

arising from the lateral femoral condyle and passing cranially to the tibia to insert on tarsal bones and proximal metatarsal; the SDFT lying caudal to the tibia and connecting the caudal femur to the calcaneal tuber. This ensures that when flexion or extension of one joint occurs it necessitates the same movement at the other (Dyce *et al.* 2002; Budras *et al.* 2001).

The fetlock and pastern joint are supported in a manner similar to the forelimb suspensory apparatus, however there are two differences in the arrangement. The accessory (check) ligament of the DDFT is thinner, and the SDFT has no accessory ligament. The latter is compensated for by the tendon's strong attachment to the calcaneal tuber (Budras *et al.* 2001).

A legacy of these aforementioned biomechanical constraints is that the horse is predisposed to musculoskeletal injuries, especially in the distal forelimb. This is the most frequently injured site in horses of all types across all sports (Dyson 2000; Davies 2002; Brown *et al.* 2003). The extent and nature of compensation for injury reflects whether the injured structure is loaded more while absorbing energy and/or supporting the body or while actively moving the limb. Compensations become more difficult as the speed (racehorse) and vertical displacement (dressage horse) increase (Clayton 1996; Barrey *et al.* 2001; Barrey & Biau 2002) and the parameters that define superior performance in each case will show a measurable deficit.

Owing to lack of research into neuromotor control in the animal as compared with the human, biomechanics of the equine locomotor system, be it in anatomy texts or research, focus on the role of the major muscles of locomotion which cross multiple joints.

Gait

Horses use and are trained to utilise many variations of the main gaits.

- At walk, the inter-limb coordination changes with differing gaits as defined by Wilson *et al.* 2000.
- Trot is a symmetrical, two-beat gait with the diagonal limb pairs moving synchronously and a short suspension phase between ground contacts of alternate diagonals.
- Pacing is a two-beat gait where unilateral forelimbs and hindlimbs move synchronously and a suspense phase between placements of the alternating pairs.
- Gallop is a four-beat asymmetrical gait where the forelimbs and hindlimbs work in two skipping pairs, with overlap between each limb contacting the ground. Therefore on a left lead, the foot placement would be right hind, left hind, right fore, left fore and suspension.
- Canter is a three-beat gait, with the same sequence of footfalls as the gallop but with the second hind and first fore leaving the ground at the same time.

The equine gait is manipulated by changing firstly the inter-limb coordination, but also the timing of the phases

of the gait cycle and the angulation of the joints. Weyland *et al.* (2000) showed that as the speed increases, from trot to gallop, the protraction or swing phase of the gait cycle actually remains the same. Instead, the time spent in stance decreases, and the force applied to the ground is larger, thus the time spent during stance can be represented as a fraction of the time for the stride of the limb, expressed as the duty factor. As the fraction gets smaller, that is, as the time in stance decreases, the force experienced by the limb increases. The maximum speed of the horse is limited by the minimum duty factor that can be sustained, that is, the maximum force that the legs can withstand. Start to relate these principles directly to the horse sports and therefore the type of lesion you would expect as you go through each applied biomechanical principle.

Stress and strain in gait

Training variables (e.g. gait, frequency, duration, surface type, hoof balance) load the limb in different ways (Clayton 2002). Stress is a measure of load per unit area. Strain is the length change due to the applied stress. The loading rate is determined by the speed and frequency of impact; thus gait is of primary importance in strain characteristics, and helps determine which structures are at maximum risk. Mechanisms are in place to cope with these different stressors, and improve the efficiency of locomotion. Impact forces are absorbed by the hoof, the suspensory apparatus, the digital flexor (DF) muscles and the shoulder syncarcosis (Payne *et al.* 2005).

Sport-specific locomotion

The horse's ability to move at speed is not due to active muscle contraction alone (Brown *et al.* 2003; Wilson *et al.* 2003; Zarucco *et al.* 2004). The limbs rely on non-contractile structures to assist muscles by providing a passive role in joint stabilisation and elastic storage and release of energy. The biceps mechanism and suspensory apparatus of the distal forelimb, as described above, enables utilisation of stored elastic energy, reducing muscular energy expenditure and the weight of the distal limb (Dimery *et al.* 1986). The trade-off is a reduced capacity to make voluntary adjustments thus placing tendons and passive soft tissues at risk (Schamhardt 1998). The superior check ligament, inferior check ligament, and suspensory ligament combine to support more than 50% of the total moment developed about the metacarpophalangeal (MCP) joint in full extension in stance (Brown *et al.* 2003). These passive structures assist the superficial and deep digital flexor muscles to stabilise the MCP joint and provide assistance in propulsion into flexion of the joint at the completion of stance. The muscles themselves are relatively small and rely largely on a passive tendon and connective tissue contribution to support body weight. There is a linear relationship between speed and MCP joint angle (Brown *et al.* 2003), and passive structures are under greater load at higher speeds.

Wilson *et al.* (2003) described the biceps mechanism, 'as a catapult that accelerates the protraction of the forelimb'. This is achieved by exploiting elastic potential energy, which is stored during stance phase as the biceps is stretched. This catapult action produces a peak power output of 2200 watts, rivalling a muscle one hundred times the weight of the 0.4 kg biceps (Wilson *et al.* 2003). In a galloping horse, the biceps rapidly stretches up to 12 mm more than at the trot, releasing four times the energy.

Wilson *et al.* (2003) concludes that the biceps mechanism, through its substantial internal tendon elastic energy storage and release mechanism, is responsible for 80% of the shoulder extensor moment during limb protraction. The biceps internal tendon is stretched during limb retraction during stance thus storing the energy required for the swing phase. The extension moment is dependent on energy storage and thus on speed, so is less effective in the slower paces. Thus, dressage horses are unable to recruit biceps in the same way to reduce the muscle demands of protraction.

Most of the length change in the distal forelimb muscle-tendon complex occurs passively as a result of the in-series arrangement and tissue properties of the elastic components, and not by concentric contraction of the digital flexor (DF) muscles (Barrey *et al.* 2001). The DF muscles are 'tuned' by virtue of their short-fibred, deep heads to rapidly damp up to 88% of damaging vibrations (frequency 30–40 Hz) that transmit up the limb (Wilson *et al.* 2001a). Impact of this type is the most important factor in the development of degenerative joint disease, the most common cause of wastage in dressage horses (Clayton 1997).

The proximal spring, including the muscle-tendon units from the scapula to the elbow, has been shown (McGuigan & Wilson 2003) to shorten by 12 mm during stance phase at gallop. The distal limb spring, from the elbow to the foot, in contrast shortens by 127 mm in stance at the gallop. These authors have sound evidence that the role of the proximal spring is to achieve a small tuning effect on the distal spring and to drive the distal spring. The role of the two units is to achieve shock absorption and energy storage and return to drive locomotion and, once again, is mainly a passive one.

The advantages of these passive stabilisation and propulsive mechanisms are two-fold during locomotion: (i) there is a reduced requirement for muscle contraction and therefore a significant energy saving; (ii) adjustments to higher speeds and thus higher loads can be done quickly and automatically without central nervous system input. It should be noted, however, that the efficiency of the passive mechanisms described reduces with decreasing speed to a point where at slow speed, e.g. walk and slow trot, the majority of moment production is performed actively by the muscles. Hence, there is a relatively greater energy demand at these slow gaits and while the passive structures are partly relieved of strain, the active muscle units and their associated

structures are increasingly loaded. Consider the mechanisms involved in forelimb protraction as an example of the relationship between speed of gait and contributing mechanisms. Wilson (2003) found that the catapult action of the biceps and internal tendon (passive action) contributed approximately 80% of the shoulder extensor moment at a 3 m/s trot. The active concentric action of supraspinatus contributed the other 20%. The loss of the passive biceps catapult contribution would therefore have a huge impact on locomotion mechanics at slower speeds.

As previously stated, both Wilson *et al.* (2003) and Brown *et al.* (2003) report a linear relationship between speed of gait and MCP angle; as speed reduces, so does the angle of the MCP (preloading of passive structures), and the stance time increases. An increased stance time reduces peak ground reaction forces as the limb is loaded over a longer time period, thus reducing the stresses on the limb and reducing the likelihood of injury (Brown *et al.* 2003). A trot at 3 m/s produces a forelimb stance time of 227 ms (milliseconds) (Holmstrom *et al.* 1995). The Piaffe movement of Grand Prix dressage horses by comparison has a speed of 0.09 m/s and a forelimb stance time of 509 ms, more than twice the ground contact time at the trot (Holmstrom *et al.* 1995). Given the known linear relationship, it can be extrapolated that a significant quantity of passive elastic energy storage potential would be lost due to reduced elastic preloading and dampening of stored energy over time. Holmstrom *et al.* (1995) documents the speed of the Passage movement at 1.7 m/s with a forelimb stance time of 365 ms. This gait may also compromise passive mechanisms, and favour force production from active muscle contraction. Holmstrom *et al.* (1995) measured the hock extension angle during the Piaffe and concluded that due to reduced extension compared with other gaits, elastic strain energy may not be important in Piaffe.

At the quick end of the spectrum, a sprint horse may reach speeds of 20 m/s, requiring very short stance time and greatly increasing peak limb force. McGuigan and Wilson (2003) report, that it is stance time that drops with speed and that limb protraction time is relatively independent of the gait and speed of the horse. It is widely accepted (Batson *et al.* 2003; Brown *et al.* 2003; McGuigan & Wilson 2003) that it is due to high peak vertical forces producing hyperextension of the MCP joints that there is such a high rate of injury (50% of race horse injuries) to the superficial and deep flexor tendons, the superior and inferior check ligaments and the suspensory ligament, which are the predominant passive structures in the distal forelimb. Brown *et al.* (2003) calculated that the superior and inferior check ligaments and suspensory ligament combined supported more than 50% of the total isometric moment about the MCP joint at maximal extension. These are the soft-tissue injuries of the galloper which obviously accompany the sequelae of bony injuries resulting from rapid overloading and overuse of unaccustomed structures.

4.10 Considerations in sport-specific pathology

4.10.1 Flat racing

Owing to the rapid protraction occurring repeatedly in the forelimbs in flat racing, there are large peak forces and extreme forelimb flexor tendon and ligament strains (Barrey *et al.* 2001; Wilson *et al.* 2003).

Racehorses utilise the passive energy output of the biceps catapult, therefore, owing to the high duty cycle, they are predisposed to the development of bicipital tendinitis and rupture. Musculo-tendinous injuries may first manifest as behavioural problems, refusal to stride out, and slow training times, in addition to any of the classic signs of lameness such as unloading/head bobbing, altered gait parameters/asymmetry, and inability to protract the limb. Latissimus dorsi and triceps brachii are required to contract from a lengthened position (Payne *et al.* 2004, 2005a, b), producing powerful retraction to pull the body forward and thus are at risk of injury.

Ground reaction forces on a single forelimb at gallop can reach 2.5 times the body weight, and MCP joint hyperextension can reach angles that almost parallel the ground (Schamhardt 1998). Stabilisation of the MCP joint is provided by the flexor tendons and ligaments (suspensory ligaments and superior and inferior check ligaments). The ligaments increase their contribution to stability as joint extension increases, up to a maximum of half of the total support at MCP joint maximum extension (Brown *et al.* 2003). Failure has been shown to occur *in vitro* at strains between 12 and 19.7%. Hyperextension of the fetlock at faster paces produces flexor tendon strains of between 5 and 10% (Barrey *et al.* 2001) with the SDFT experiencing double the strain of the DDFT (Dimery *et al.* 1986; Brown *et al.* 2003). Other investigators have measured tendon strains of 3% at walk, 6–8% at trot and 12–16% at gallop. Combined with surfaces that are too hard, too soft or too irregular, racehorses risk catastrophic tendon damage, and exhibit a high incidence of tendon, check and suspensory ligament injuries (Brown *et al.* 2003).

Davies (2002) studied the bones of maturing Thoroughbreds (2–3 years of age) whose bones undergo remodelling at a high rate. Remodelling alters the composition and lowers the mineral density (Davies 2002). Bone is a pseudo-ductile material with a relatively large elastic zone. Under ideal conditions, deformation is contained within the elastic zone and results in normal desirable adaptive hypertrophy. Galloping combines high stress (concussive load concentrated on a small surface area) applied at high rate (frequency with which the limb impacts the ground) (Davies 2002).

Flat racing imposes stress and strain of sufficient magnitude and rate to change the quality of bone from pseudoductile to brittle, increasing the risk of fatigue and catastrophic fractures (Davies 2002). Fast tracks and hard training surfaces have a high resistance and do not absorb

impact forces well, increasing concussion and prolonging the attenuation time, and can lead to increased incidence of flexor tendinitis in racehorses.

Strain is defined as the change in length divided by the original length, and is measured at the dorsal mid-third of the forelimb cannon bone. Horses with third metacarpal pain will exhibit the signs of over training – loss of performance, appetite, behavioural problems, as well as palpable tenderness and swelling over the inflamed mid-third of the dorsal surface of the cannon bone.

Common racing injuries

- Concussive injuries – hoof/joint, flexor tendinitis, proximal suspensory desmitis
- Hyperextension of the fetlock – flexor tendon injuries/rupture, suspensory ligament rupture
- Fractures – particularly of the carpus and third metacarpal (cannon)
- Shin soreness and fatigue fractures of the cannon
- Biceps tendinitis and rupture

Compensations

- Shin sore – refusal to stride out, slow training times
- Biceps tendinitis – reduced cranial phase; inability to protract the limb – increased recruitment of brachiocephalicus, asymmetry, head bobbing and leaning on the bit
- Suspensory apparatus – reduced weight-bearing phase and lateral unloading – unwillingness/refusal to lead on a particular leg, refusing to stride out, head bobbing/crookedness

4.10.2 Dressage

Dressage training attempts to change the way horses carry themselves, and interact with the ground, altering braking and propulsion characteristics, ultimately aiming to shift weight caudally to lighten and enable elevation of the forehand (Clayton 1996). Barrey and Biau 2002 analysed the characteristics of dressage as:

- Exhibiting slow cadence-stride frequency and high regularity (similarity of acceleration patterns of each stride)
- Large dorsoventral displacement (vertical movement)
- Dorsoventral and longitudinal activity (power of the motion in vertical and longitudinal directions)

Thus dressage requires the horse to execute repetitive, controlled and powerful antigravity movement in all directions; forward, backward and lateral and degrees of collection. Transitions between and within gaits require great muscular power, control and coordination (Barrey & Biau 2002) (Figure 4.7). The characteristics of dressage movement are:

- Collection produces an increase in the upward acceleration and a decrease in the forward acceleration (Barrey

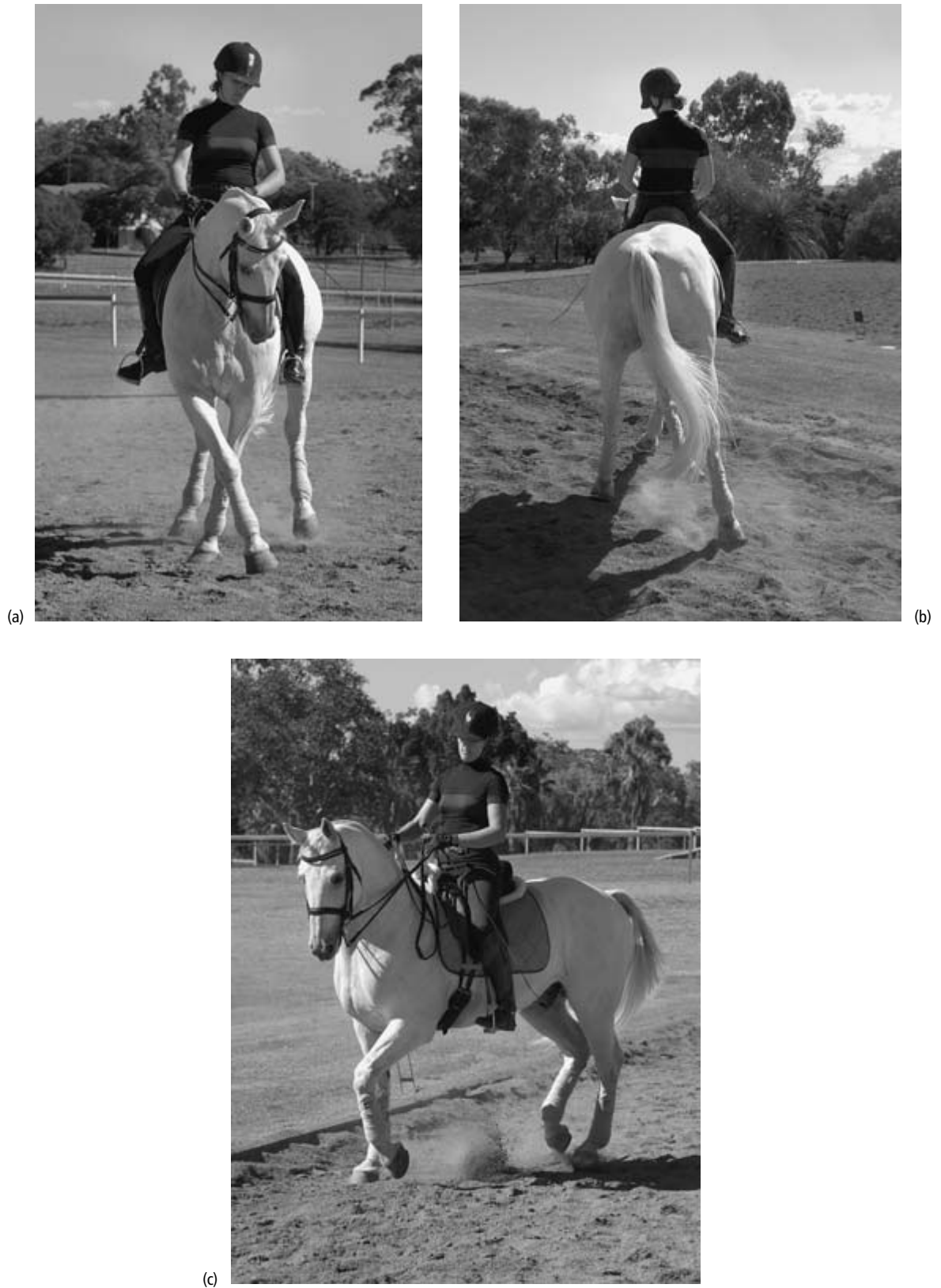


Figure 4.7 Dressage biomechanics.

& Biau 2002) – the hindlimbs apply a braking force to forward movement during Piaffe (Clayton 1996).

- There is a large range of motion at hock and at the elbow and carpus.
- Passage and Piaffe are characterised by a prolonged stride duration and slower stride frequency.
- Passage exhibits a higher longitudinal GRF in the hindlimb over the forelimb (Barrey & Biau 2002).
- There is repetitive concussive loading of limb joints.
- Front legs apply a braking force to forward movement during Piaffe.
- Circular and lateral movements are achieved by utilising the thoracic and pelvic muscular sling and adduction and abduction of the fore and hindlimb.

Vertical displacement at the trot is achieved through actively ‘springing’ off the ground via powerful concentric contractions of the propulsive muscles of the hindlimbs, flexors of the distal forelimb, and utilising return of elastic energy in the flexor tendons. The increased impact loads apply repetitive concussive stresses to the joints of the fetlock, carpus, hock, stifle and pelvis (Barrey & Biau 2002) and places the flexor tendons and suspensory apparatus under strain. During slower versions of the trot the reduced stretch on the flexor tendons (Robert *et al.* 2002) lessens their elastic energy storage and increases the antigravity workload placing greater concentric demands on the muscles that move the forelimb. The muscles of the syncarcosis (e.g. serratus ventralis, pectorals) are required to dissipate repetitive ground reaction forces and more muscle activity is required to stabilise the spine (Robert *et al.* 2002).

Horses are trained to alter the temporal characteristics of the trot to produce the more collected variations of trot – the Passage and the Piaffe. Clayton has studied the temporal characteristics of the trot, Passage and Piaffe: the tempo remains the same but forward movement is converted into vertical motion, resulting in a longer period of suspension, greater ground reaction forces, increasing joint concussion. During the Passage, the hindlimb is primarily responsible for propulsion and the forelimbs act to brake forward motion and elevate the front end. The exception is Piaffe which requires the hindlimbs to ‘brake’ the forward movement while the front limbs propel (Clayton 1996).

Apart from the kinematic differences from the standard trot and gallop, dressage movements often require full collection (extreme flexion of the spine, in particular the upper cervical and lumbosacral spine), and exaggerated forelimb and hindlimb protraction, and flexion of the shoulder, elbow, carpus and fetlock joints, well beyond the normal locomotive functional range of these joints. The soft-tissue structures commonly injured during galloping are mostly spared during this type of activity. Gibson *et al.* (1997) document the incidence of a galloper with a confirmed superficial digital flexor injury being unable to race but successfully competing injury-free in dressage. The muscular

physique of the top level dressage athlete is a testament to the extreme muscle power and control requirements of the discipline.

The types of injuries experienced by the dressage athletes are expected to be in the proximal limbs and trunk due to the increased loading of unaccustomed muscles to somewhat un-natural movements. At the extremes of movement, e.g. at the height of forelimb protraction, the prime movers are working to produce slow velocity movements with long, almost isometric holds at the end of range. Muscles prefer to work at a natural velocity within their most efficient length, which is usually close to their resting length. The brachiocephalicus becomes a prime mover in forelimb protraction in the Piaffe and Passage and it is forced to work in an extremely shortened position owing to collection of the neck and the extreme floating protraction of the forelimb. Working hard under these conditions is likely to cause muscle fatigue and strain. The isometric nature of the trunk postural muscles stabilising the spine while suspending the limbs in a slow temporal environment, may cause local ischaemia and hasten fatigue, and cause compensations.

The repetitive nature of dressage training predisposes to overuse injuries, with acute traumatic injuries rare (Ross & Dyson 2003). Impact loading can induce fatigue fractures of the metacarpal 3 and degenerative joint disease (Clayton 1996; Ross & Dyson 2003). There is an increased demand on range of motion at the elbow and carpal joints, and on antigravity and digital flexor muscle activity (dampening of vibration). Common soft-tissue injuries involve the suspensory apparatus (e.g. proximal suspensory desmitis), and superficial digital flexor tendinitis (Clayton 2002).

Compensations

- Injured dressage horses lose rhythm, elevation and regularity, altering the swing phase of protraction and the flight path of the distal limb.
- Injury to the biceps mechanism compromises efficient shoulder extension and forelimb protraction – substitutes with increased activation of cervical trapezius/rhomboids, brachiocephalicus, brachialis and supraspinatus.
- Painful joints and inflamed flexor tendons and/or suspensory ligaments may cause the horse to shift weight to one side, lift the head, become unwilling to lead on a particular leg, or carry the rider on the affected diagonal in an attempt to reduce the vertical GRF (Clayton 1996; Ross & Dyson 2003).
- The horse reduces impulsion, and the digital flexors work harder to damp the shock of impact.
- Loss of range of motion at elbow and carpus reduced flexion of forelimb joints, altering the height and flight and destroying the expression of the movement.
- Back pain and appendicular joint injuries are adversely affected by torsion – the horse avoids performing lateral

movements and moves asymmetrically (Ross & Dyson 2003).

- Back pain – the horse will hollow the back to avoid collecting and/or engaging the hindquarters.

Compensations to injury to the limbs are well documented. Wilson *et al.* (2001c) describe an early unloading of the heel onto the toe in early stance in horses with navicular pain. Weishaupt *et al.* (2004) measured a 15% reduction in vertical impulse in the lame limb by shifting load to the diagonal limb and by prolonging the stance time. Buchner *et al.* (2003) found a 34% reduction in vertical displacement and a 9 mm rearward shift of the body centre of mass in mid-stance in forelimb lameness. Buchner *et al.* (1996) found a significant reduction in fetlock extension in stance on the lame forelimb and increased shoulder flexion and retraction of the lame forelimb.

The common message from these studies is that the proximal joints act as load dampers to reduce peak forces in the lame forelimb, and compensations are made to avoid overloading of non-lame limbs. A well recognised compensation for forelimb lameness is a rising of the head during the stance phase on the lame limb. This phenomenon tends to shift the centre of gravity caudally and thus relieves the weight from the forelimbs (Back *et al.* 1993). Perhaps the most obvious compensation to injury and pain is the refusal to perform for the rider, or uncharacteristic behavioural signs such as bucking, propping and running off. While these signs are indicative of a problem, the problem area may be anywhere from the spine to the foot.

Identification of an injury would involve the same assessment whether in a galloper or a dressage horse, but the fundamentals of the functional and biomechanical requirements of the individual disciplines is important as an adjunct to understanding the mechanisms of injury and how to prevent them from occurring.

4.11 Biomechanics of the equine foot

It is beyond the scope of this chapter to describe in detail equine hoof biomechanics and locomotive related pathology in detail. But it is vital that physiotherapists have an in-depth knowledge of the equine foot and work closely with the treating veterinarian and farrier. Recognised as causing equine forelimb lameness for over a century, pathologies such as navicular disease and laminitis are complex acute–chronic conditions seen in riding and retired horses of all disciplines. On reviewing the vast amount of literature available it is evident the necessity for understanding foot biomechanics and the pathogenesis both from a musculoskeletal and medicine perspective. Gaining an accurate equine veterinarian differential diagnosis is essential as many other foot ailments have similar clinical signs, thus confusing the issue. Beneficial disease management includes farriery, once the possible aetiology or causations are understood.

Navicular disease is used here as an example. (This information is summarised from the following literature sources: Ostblom *et al.* 1982; Stashak 1987; Pool *et al.* 1989; Wright & Douglas 1993; McGuigan & Wilson 2001; Pardoe *et al.* 2001; Wilson *et al.* 2001b, 2001c; Eliasher *et al.* 2002.)

Anatomically, the navicular or sesamoid bone at the back of the foot beneath the frog, acts like a pulley, anchored/supported by the sesamoidean ligaments. Wrapping around it is the deep digital flexor tendon (DDFT) inserting into the pedal bone (distal phalanx), and is protected by the navicular bursa. The distal interphalangeal joint (coffin joint) also lies in close proximity. The navicular bone has two biomechanical functions and any alteration to these may predispose the animal to this multifactorial condition:

1. It provides a constant angle to maintain mechanical advantage for insertion of the DDFT like a fulcrum/pulley system.
2. It is an anticussion device aiding shock absorption with concussion being a definitive predisposing disease factor.

The navicular bone transmits a portion of the weight from proximal to the middle phalanx, with navicular pressure increasing further as it is forced against the DDFT/bursa as the body weight passes over the limb. Force alterations may be a vital factor in the disease process causing degenerative changes over time, along with bursitis, which in turn leads to hyperthermia and alteration of the flexor surface of the bone.

Research-based evidence now confirms that abnormal mechanical overload of the navicular bone results in gait changes and possibly the early or late disease stages; shifting leg lameness, shortened stride and the toe on the ground. The disease-associated gait is a vicious cycle resulting in a positive-feedback loop, as the horse attempts to unload the heels and avoid pain by contraction of the deep digital flexor (DDF) muscle. Unfortunately, this increases the peak stress/force on the navicular (double compared with normal). Regional analgesia of the palmar digital nerves (nerve block) confirms this by causing a lowered compressive force.

A narrow, small, boxy, upright foot may not be the initial cause but further exacerbates the condition and may be a result of the disease. A flat foot, long toe, low collapsed heels, and broken back foot/hoof pastern axis is a proven predisposing factor, stretching the DDFT hence increasing forces. The broken back foot pastern axis prolongs the breakover time, just before the heel leaves the ground (normal peak around 85% of stance) and increases toe first contact, increasing forces through the navicular. The distal interphalangeal (DIP) joint position is then in permanent extension causing greater tensile forces on the DDFT and ligaments and stress/concussive force on the navicular.

Logically then, poorly conformed and trimmed horses in work on hard surfaces are particularly liable to undergo mechanical changes.

As a result of the mechanical loading there is an irreversible high rate of bone remodelling (turnover) and erosion of navicular fibrocartilage surface in contact with the DDFT in the central portion, reflecting aging degeneration and adhesions of the DDFT. As a result blood vessels in the navicular bone increase. Radiographs and bone scans in the advanced stages often show; bony erosions, distal sesamoidean ligament ossification, arthritis of the coffin joint with the DDFT becoming progressively destroyed. This identifies a possible end point for a variety of heel-related conditions.

Biomechanical corrective farriery attempts to normalise navicular forces at breakover, and reduce surrounding forces for pain relief and functional improvement. A reduction in the angle of deviation of the DDFT around the navicular bone can occur by elevating the heels, hence the heels leaving the ground earlier. Foot balancing re-establishes the correct anatomical relationship of the foot/pastern axis (ideally a straight line parallel to the hoof wall) and enables the foot to strike the ground level in relation to individual skeletal conformation, accurately assessed on X-ray. Also important is the medial/lateral hoof balance with hoof walls the same height and an imaginary line bisecting the frog–sole, with heels equidistant from the coronary band. Foot balancing ensures that the centre of rotation of the DIP joint falls on the midpoint of the bearing surface (Figure 4.8).

Other temporary remedial farriery includes; raising the heel, shortening the toe or leaving the heels long. Increasing the thickness of the shoe from toe to heel by a tapered wedge with the bearing surface extending beyond the heels reduces



Figure 4.8 Example of good hoof/pastern axis.

DDFT tension and tensile navicular load, although it may delay the breakover and load the heels. But by lowering the heels allowing for the wedge (correct foot/pastern axis) encourages foot expansion reducing vertical impact forces. Although more controversial (because of increased overall navicular pressure), a measure for horses with collapsed heels is the Eggbar shoe. This is often combined with a rolled toe or tapered wedge. Improved weight distribution can occur by extending the ground-contact surface helping to re-establish a suitable heel for future support. Long-term use of heel wedges or Eggbar shoes is not advised as the vicious navicular cycle may recommence or it may be difficult to stop using them.

4.12 Conclusion

Knowledge of the concepts of functional biomechanics of animals from the orientation and movements available at individual joints, to the way in which movement differs between conformation breeds and between sports, will assist the animal physiotherapist in achieving the best outcome for performance in an animal.

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5

Comparative exercise physiology

Catherine McGowan and Brian Hampson

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5.1 Introduction

The aim of this chapter is to discuss principles of exercise physiology and follow up, with discussion in an applied manner to familiarise the physiotherapist to some of the sports their animal patients will be undertaking. The chapter presumes a basic understanding of the physiology of exercise in people, and aims to expand knowledge of animal exercise physiology so that physiotherapists can more appropriately develop rehabilitation and exercise programmes for their animal patients.

The physiology of exercise in animals is similar in physiological principles to humans and much of the research work in man has been and is still carried out on animals for the purposes of enhancing knowledge for human exercise physiologists. However, certain differences clearly occur that can have clinical significance and may affect exercise potential. Of course, animals are quadrupeds, but other factors can be equally important, such as the inability of horses to mouth breathe and dogs only sweat in localised regions of their body.

For species-specific exercise physiology in animals, the greatest amount of research and knowledge has centred on the athletic horse, although this is gradually changing. Despite the advancements in research and clinical application of exercise physiology principles in animals, it may still surprise the physiotherapist used to working in a human sports setting how limited the translation of this knowledge is at the sporting industry level.

5.2 Principles of exercise physiology

5.2.1 Energy production for exercise

It is clearly the aim of trainers of performance animals to maximise the animal's capacity for exercise. In simplest terms this means maximising the availability of energy for

muscle contraction, in the form of adenosine triphosphate (ATP) and the fuels required to produce it.

There are limited stores of ATP within muscles for muscle contraction (either as ATP, or high energy phosphates like phosphocreatine) so energy is produced during exercise either aerobically or anaerobically, depending on the availability of oxygen (and substrate).

5.2.2 Aerobic energy production

Aerobic production of ATP occurs via a series of reactions within the mitochondria called aerobic or oxidative phosphorylation because of its requirement for oxygen, and the ultimate step is the phosphorylation of adenosine diphosphate (ADP) to make ATP. Aerobic energy production can occur using stored muscle glycogen or glucose from blood as an energy substrate. This involves glycogen or glucose undergoing glycolysis to produce pyruvate in the cell cytoplasm. Pyruvate can then be transported into the mitochondria where it is converted to acetyl coenzyme A (CoA) in the mitochondria, which enters the tricarboxylic acid (TCA) or Krebs cycle. The net result of the TCA cycle is the production of ATP and the production of the coenzymes nicotinamide adenine dinucleotide (NADH) and flavin adenine dinucleotide (FADH₂), which enter the electron transport chain producing ATP.

Complete aerobic metabolism of one glucose unit (entering as glucose-1-phosphate) from glycogen yields 39 molecules of ATP – three from glycolysis, two from the TCA cycle and 34 from the electron transport chain. If glucose from the bloodstream is used it must first be converted to glucose-6-phosphate requiring one molecule of ATP, so the net energy yield is only 38 molecules of ATP.

Fatty acids can also be used as substrate for oxidative phosphorylation via a process called beta-oxidation producing acetyl CoA. Acetyl CoA then enters the TCA cycle

producing NADH and FADH₂, which enter the electron transport chain as for carbohydrate substrates. Because fatty acids are composed of many carbon atoms and only two are required to produce acetyl CoA, the yield of energy from a typical fatty acid is very high. For example, the complete aerobic breakdown of palmitic acid (16-carbon fatty acid) yields 129 molecules of ATP.

5.2.3 Anaerobic energy production

Anaerobic energy production is reliant on the metabolism of stored muscle glycogen or glucose via glycolysis with the resultant production of pyruvate, but pyruvate remains in the cytoplasm and is converted to lactate. The amount of ATP produced via anaerobic glycolysis is much less than aerobic glycolysis and oxidative phosphorylation. If glycogen is the original substrate, there are three ATP molecules per glucose unit produced, while only two ATP molecules are produced if blood glucose was the original substrate.

5.2.4 Energy sources during exercise

During exercise in the healthy animal, the usual sources of energy are carbohydrates and fat. Glycogen is predominantly stored in the muscles and liver, and glucose is available in the blood. Fats are stored in the body as triglycerides but can be broken down to free fatty acids, which can circulate in the blood and be taken up by exercising muscle. Muscle also has triglyceride stores, which can be broken down within the muscle to release free fatty acids. In the horse volatile fatty acids are produced as a result of their digestive process and these can be also be directly used via beta-oxidation.

At the onset of exercise, when oxygen supply may be limiting and during high intensity exercise, when the requirement for ATP exceeds the rate of ATP production aerobically, substrates are predominantly utilised anaerobically. Anaerobic energy production is rapid and does not require the delivery of oxygen to the muscle, but the ATP yield is low compared with aerobic pathways and the production of lactate will decrease muscle pH. During low to moderate intensity exercise aerobic metabolism predominates and will be a mixture of fatty acid and carbohydrate utilisation. Horses and dogs have both been shown to have the ability to utilise fatty acids for energy production, but this is probably limited to lower intensity exercise as it is in man (Hawley 2002). In most species, a greater ability to utilise aerobic energy sources during high exercise intensity is beneficial for maximum performance and delaying fatigue (see VO₂max below).

In dogs and horses the same principles apply. However, there are three important principles to consider that highlight the central role of energy production for exercise, especially aerobic energy production:

1. Energy utilisation pathways are not all or nothing. At any moment during exercise there are aerobic and anaerobic contributions to energy production.

2. Energy partitioning of different species may vary enormously and whether an animal is working 'aerobically' or 'anaerobically' should not be extrapolated directly from humans, but rather from research data pertinent to that species. This is explained in more detail in energy partitioning below.
3. The intensity of exercise is used to describe many aspects of exercise from substrate usage to specific training programmes. The intensity of exercise is often expressed as a proportion of maximal aerobic metabolic rate (VO₂max) (see both energy partitioning and VO₂max below) – yet in animals with very high VO₂max this needs to be put into perspective. For example, extrapolating from research in man, fat utilisation may only occur at intensities of <65% VO₂max (Hawley 2002). The same intensity in horses is a fast canter/working gallop and so fat usage may be important during a much greater proportion of exercise than in man.

5.2.5 Energy partitioning

The relative contributions of aerobic and anaerobic energy sources during exercise is an important indicator of the type of exercise performed by an animal in a particular athletic pursuit and will be central to dictating training programmes and adaptations to training. For athletic events that are primarily aerobic in nature, much effort in a training programme will be on maximising the aerobic pathways. Athletic events that are anaerobic in nature may focus on increasing muscle strength and acceleration.

As mentioned above, there are considerable species differences in proportions of aerobic and anaerobic energy supply during exercise. In man, short-term high intensity exercise such as a 60-second sprint to fatigue is a primarily anaerobic activity, yet the equivalent intensity sprint in a horse is primarily aerobic in nature (Table 5.1). This illustrates that the horse has a high reliance on aerobic energy supply, even in short-duration high-intensity exercise. A 60-second horse race is only approximately 1000 m, and commonly termed a 'sprint', but is not predominantly anaerobic exercise.

Horses have evolved to gallop fast, but to outrun predators they must be able to sustain that speed. Hence, the reliance on the more sustainable (aerobic) energy source during exercise. Horses do vary with breed, but even the Quarterhorse (bred to race over a quarter of a mile or 400 m or 2 furlongs) in a 400 m race would only have 60% of the energy supplied anaerobically. Of course, the Arabian has

Table 5.1 Energy partitioning for equivalent intensity and duration exercise in human athletes and Thoroughbred horses

Duration of <i>all out</i> exercise	Man (Hagerman 1992)	Horse (Eaton 1994)
60 s	30% aerobic	70% aerobic
2 min	60% aerobic	80% aerobic

an even greater reliance on aerobic energy sources during exercise and during endurance riding will work about 96% aerobically (Eaton 1994). Similarly, an eventing horse relies primarily on aerobic energy sources, with anaerobic energy during jumping efforts (Marlin *et al.* 1995).

Energy partitioning has not been well described for dogs. Despite the apparent variability in breeds that currently undergo sporting activities, wolves and dogs evolved to hunt in a pack over long distances and so have a greater reliance on aerobic energy sources during exercise. From the type of race and anecdotal information, it would seem logical that Greyhounds rely highly on anaerobic sources. However, this has not been tested and with evidence for a high VO_2 max of Greyhounds (143 ml/kg/min) contradicting this assumption (Staadon 1984), this area warrants further study.

5.3 The pathway of oxygen

The supply of oxygen to exercising muscle is vital to the performance ability of any animal, but particularly those that are relying aerobic metabolism for sustaining energy, such as both the horse and dog. The pathway of oxygen is fundamental to an understanding of exercise physiology as it is the key to aerobic energy production. *Limitations to oxygen supply anywhere along this pathway can seriously limit performance* (Figure 5.1).

5.3.1 Maximal oxygen uptake (VO_2 max)

The key measure of the aerobic contribution to energy production during exercise is the oxygen uptake. As mentioned above, this can be used to determine the relative intensity of exercise. It is measured by measuring oxygen and carbon dioxide concentration in expired respiratory gasses (Figure 5.2).

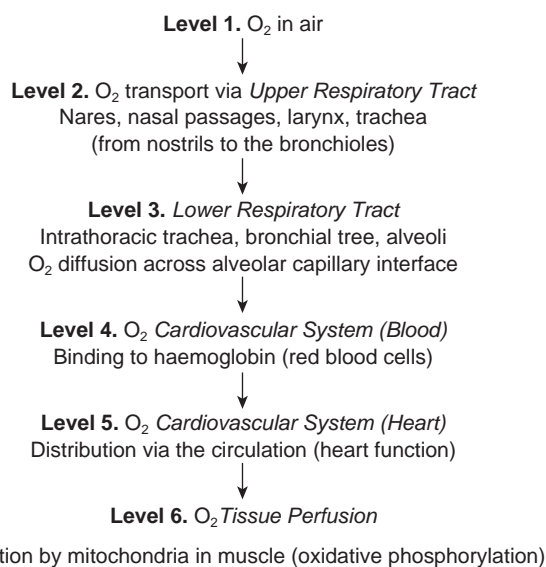


Figure 5.1 The oxygen transport chain.



Figure 5.2 VO_2 max being measured in an exercising horse on treadmill. Note the lightweight mask with holes to reduce resistance to breathing and large diameter tubing to remove expired gasses.

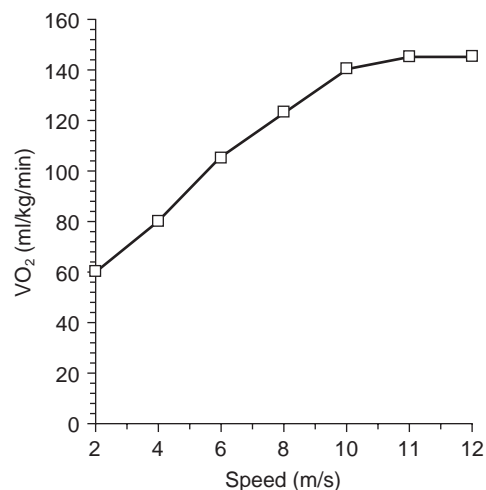


Figure 5.3 The relationship between VO_2 and speed in a horse during incrementally increasing speed.

Oxygen uptake or VO_2 is the overall use of oxygen by an animal. When measured during incrementally increasing exercise, VO_2 increases linearly, matched to demand during exercise (Figure 5.3), until it finally plateaus at a peak value called VO_2 max = maximal aerobic metabolic rate. It has been measured in many different species including horses (Evans & Rose 1987) and dogs (Wagner *et al.* 1977) and is closely related to (endurance) performance ability. VO_2 max is often used as an indicator of intensity of exercise with 100% VO_2 max usually indicating high intensity exercise. However, it is important to note that 100% VO_2 max is not 100% or maximal effort. One hundred per cent VO_2 max is only the limit of the aerobic energy pathways and the increased energy requirement at speeds/exercise intensities above VO_2 max are supplied by anaerobic energy sources.

In the horse, there is a relatively small anaerobic contribution and a horse can't reach speeds much greater than equivalent to about 120% VO_2max (Eaton *et al.* 1995) owing to its enormous ability to supply energy aerobically. Yet humans are capable of tests at 200% VO_2max .

5.3.2 Kinetics of oxygen uptake and effect of a warm-up

Animals with rapid oxygen kinetics have an advantage for performance because the faster the increase in aerobic energy production, the smaller the oxygen debt and lactate accumulation from anaerobic energy production at the beginning of intense exercise (Figure 5.4).

Horses have very rapid oxygen kinetics whereas humans have much slower oxygen kinetics. VO_2max can be reached in about 20 s in a fit horse, while a human athlete would take over 2 min to reach VO_2max under the same conditions. More specifically, the time to reach 63% VO_2max in horses has been measured as 10 s, in the dog it is approximately 20 s and in a fit human about 30 s (Poole *et al.* 2004).

In horses, oxygen kinetics have been shown to be improved by training (Bellenger *et al.* 1995). Oxygen kinetics is also generally improved by a warm-up before exercise (Tyler *et al.* 1996a; Geor *et al.* 2000). One study demonstrated a significant improvement in the aerobic contribution to energy demands during intense exercise shortly following 5 min of exercise at 50% VO_2max (Tyler *et al.* 1996a). Another study demonstrated similar effects irrespective of warm-up intensity (Geor *et al.* 2000). Physiological adaptations during the warm up include cardiovascular and respiratory responses that ensure an adequate supply of oxygen to the working muscles and the working muscles receive a greater proportion of the blood flow at the expense of other organs such as digestive system. The increased O_2 delivery to the muscles enhances their

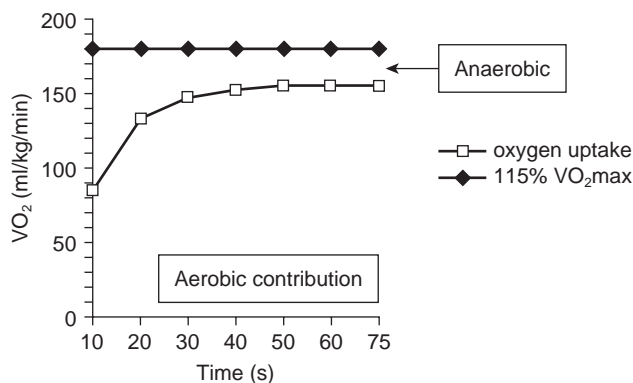


Figure 5.4 The relationship between VO_2 and time at a set speed (calculated to be 115% VO_2max) in a horse demonstrating energy partitioning during exercise as well as VO_2 kinetics. This figure shows the typical partitioning of energy in a horse performing supramaximal exercise (exercise >100% VO_2max). The area under the curve represents the aerobic contribution to exercise. Note: the large anaerobic contribution in the first seconds of exercise. However, this horse reaches close to VO_2max by 20 s into exercise.

ability to work aerobically and reduces lactate build up during exercise and delays the onset of fatigue due to lactate accumulation in high intensity sports (McCutcheon *et al.* 1999). Warm up in human athletes has been shown to increase VO_2max (Gray & Nimmo 2001). During the warm-up the temperature of the working muscles rises by 1°C which is beneficial as warm muscles contract more powerfully, and the fibres become more compliant, which reduces the risk of injury due to tearing of the fibres (Shellock & Prentice 1985). It is interesting to note that despite the evidence in horses for the physiological benefits of warm-up, there is limited warm-up performed before many equine competitions, especially Thoroughbred racing. Warm-up has also had a reported role in injury prevention, although the current evidence for this is limited.

5.4 Cardiorespiratory function during exercise

Clearly, animals have adapted to increase respiratory gas exchange and transport during exercise – indeed all components of the oxygen transport chain (Figure 5.1) – in order to maximise the delivery of oxygen for energy production during exercise.

During exercise there is:

- dilation of the nostrils and in some species mouth breathing to reduce upper respiratory tract resistance;
- increased ventilation of the lungs – (increases in minute ventilation from 80 l/min at rest to 1800 l/min in the horse);
- increased tidal volume – (ventilation per minute) owing to frequency of respiratory cycles and decreased physiological dead space;
- increased perfusion of alveoli – due to increased cardiac output and dilation of pulmonary blood vessels;
- decreased transit time of pulmonary capillary blood flow – faster blood flow past the alveoli;
- increased diffusion of O_2 and CO_2 from the alveoli into the capillaries perfusing the alveoli – due to increased gradient, increased blood flow;
- increased haemoglobin concentration (in the horse) due to splenic contraction – increased oxygen-carrying capacity (the splenic reserve) and can increase the measured packed cell volume from around 35–40 to over 60%, increase red cell volume by 1/3 (Poole 2004);
- increased heart rate (HR) and stroke volume (SV) = Increased cardiac output and overall transport of oxygen to the lungs and exercising muscle;
- increased peripheral perfusion – capillaries in the periphery (muscles) are better perfused;
- increased diffusion O_2 and CO_2 from capillaries to or from exercising muscle due to increased gradient and blood flow.

During exercise, heart rate increases linearly to heart rate max with increasing intensity of exercise, similar to VO_2 ,

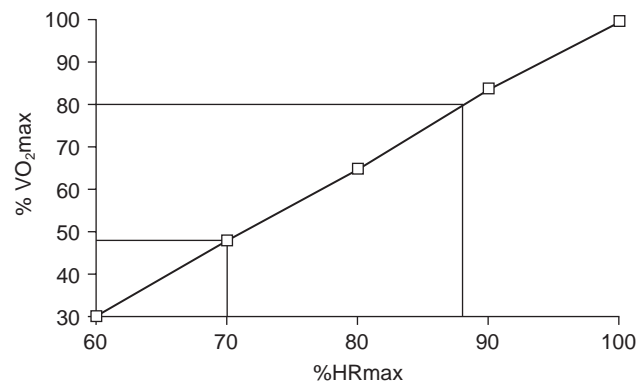


Figure 5.5 Schematic relationship between heart rate and VO_2 max in the horse showing that 50% VO_2 max is approximately 70% HRmax and 80% VO_2 max is approximately 88% HRmax (Evans & Rose 1987).

and heart rate is a good indicator of VO_2 while maximal heart rate is directly proportional to VO_2 max (Figure 5.5).

The higher the heart rate at a specific speed the more 'stress' the horse is under. (This stress can be anything from excitement, through lack of fitness to cardiac insufficiency).

5.5 The effect of training

Training results in improvements along the entire pathway of oxygen reflected as increased VO_2 max as a marker of overall aerobic metabolic rate. While this may be a major aim, training effects are not limited to cardiorespiratory system improvements, and improvements in components of anaerobic energy production including muscle buffering capacity, resting muscle glycogen concentration (McGowan *et al.* 2002) have been demonstrated in horses.

As well as improvements in energy production during exercise, improvements in muscle size (Tyler *et al.* 1998) indicating increased muscle strength and considerable adaptation of bone, cartilage, tendon, ligaments and other connective tissue occur (Marlin & Nankervis 2002). More difficult to measure in the animal world, but well evidenced in human studies, are training improvements in skill acquisition, refinement in motor pathways and motor unit recruitment pattern and speed, as well as development of sports-specific proprioceptive and tactile feedback mechanisms. These training responses combine to improve performance and reduce error and injury rates.

5.5.1 Cardiorespiratory responses to training

Increases of 30% in VO_2 max from the detrained state have been recorded after 28 weeks training in horses (Tyler *et al.* 1996b) and similar increases have been found in humans.

A few points to note regarding the effect of training in horses:

- VO_2 max continues to increase over 28 weeks of training, and then tends to plateau.

- Half of the increase and most rapid increase is in the first 7 weeks.
- Continual training stimulus will cause continual increases in VO_2 max – important as many trainers keep a very even workload following initial training (Tyler *et al.* 1996b).

In fact, there is a large increase in VO_2 max with very little initial training. The potential effects of this on perhaps more slowly adapting musculoskeletal structures should be considered and this time period may be speculated to be one of the danger periods for injury.

Cardiovascular responses to training have been shown to occur in horses (Poole 2004) in response to training:

- Heart rate decreases at the same speed – exercise at a lower proportion of heart rate max
- Faster recovery
- Stroke volume increases (heart size increases)
- Increased plasma volume (heart pre-load)
- Increased resting red cell volume (haemoglobin or Hb)
- Increased capillary density in trained muscle

These findings have been shown to occur to a greater or lesser extent in dogs, humans and horses. The horse appears to have a greater capacity to increase heart size and mass with training (Young 1999).

5.5.2 Skeletal muscle adaptations to training

There are significant adaptive responses in skeletal muscle in response to training in all species. In contrast to cardiorespiratory adaptations these changes are slow to occur, and slow to revert. Studies in horses have shown it can take 16 weeks to detect most of the skeletal muscle adaptations and these changes did not revert during the 12-week period of detraining (Tyler *et al.* 1998; Serrano *et al.* 2000).

Interestingly, the major adaptive response in equine muscle is to improve aerobic energy supply via improvements in the pathway of oxygen, even when very high-intensity training exercise is used. These include (Tyler *et al.* 1998):

- Increase in capillaries/mm²
- Decrease in diffusional index (area per capillary)
- No significant change in fibre types with training but significant increase in IIA to IIB ratio ($P < 0.05$)
- Increased mitochondrial volume
- Increased activity of enzymes associated with aerobic muscle metabolism (both the TCA cycle (citrate synthase) and β -oxidation of fatty acids (hydroxy acyl dehydrogenase), but no increase in the activity of the muscle enzymes associated with anaerobic glycolysis (lactate dehydrogenase)

Other adaptations occur with high-intensity training and include increased fibre area in all fibres (I, IIA, IIB) (Tyler *et al.* 1998) and increased muscle buffering capacity and

muscle glycogen concentration (McGowan *et al.* 2002). In humans, more exercise-specific adaptations to training have been achieved, but the genotype and phenotype variations between humans are far greater than in horses – compare the body type of a weight lifter with that of an endurance/marathon runner.

5.5.3 Muscle glycogen concentration

The horse has very high muscle glycogen stores compared with humans and while resting muscle glycogen concentration does increase with training, it is poorly responsive to 'glycogen loading' techniques used in humans. In short-duration high-intensity exercise, muscle glycogen does not become depleted, but in long-duration exercise muscle glycogen depletion may occur, e.g. endurance riding. Of greater importance in horses are the slow glycogen repletion rates – it takes approximately 48 h after glycogen depletion to replenish skeletal muscle glycogen stores, and this is not affected by feeding post exercise (but can be hastened by IV glucose infusions) (Lacombe *et al.* 2001). Both glycogen loading and attempts to hasten glycogen repletion in horses are very dangerous and can induce laminitis. This is a good example of where extrapolation from the human literature can be perilous.

5.6 Detraining

The rate of loss of fitness primarily depends on the duration and level of training stimulus. In studies in horses, prolonged training resulted in prolonged maintenance of cardiorespiratory fitness. VO_2max decreased slowly with no change for 6 weeks and by 12 weeks, it was still 15% above pre-training values (Tyler *et al.* 1996b). Indices of cardiac function also did not change for 4 weeks and did not return to normal until 12 weeks of detraining (Kriz *et al.* 2000).

Another factor affecting detraining responses is the amount of exercise during the detraining period. This relates well to a forced rest because of injury. A small amount of daily exercise is effective in maintaining cardiovascular and musculoskeletal fitness. Human studies involving strict bedrest during the detraining period have evidenced the dramatic loss of cardiovascular fitness, bone density and muscle and ligament integrity. It is important to consider the rate of loss of fitness before commencing training, e.g. if cardiorespiratory fitness is maintained following an injury and 6 weeks' rest to heal the injury, the animal could reinjure itself by exercising at a greater capacity than the recently healed injury can withstand.

5.7 Applied exercise physiology

5.7.1 Designing training programmes

As a physiotherapist, you will be designing training programmes predominantly to improve musculoskeletal and/or neurological function during treatment and rehabilitation.

In many cases you will be happy to restore function at a very low level of exercise intensity, yet in others you will be treating animals while they are actively competing. Therefore, when designing training programmes it is important to consider the principles of exercise physiology and question what you are trying to achieve.

A major aim will always be to improve components of the oxygen transport chain (Figure 5.1) through predominantly aerobic training. However, to be able to determine the intensity of training stimulus, exercise physiologists rely on use of heart rate and lactate measured during exercise. In horses, the major adaptive response to training is aerobic, irrespective of the intensity of training.

5.7.2 Use of heart rate in training programmes

Heart rate (HR) can be used to estimate VO_2 and is so used in training programmes to estimate the intensity of exercise performed. While heart rate during exercise in man has been studied on a population basis allowing calculations adjusted for age and gender, such information is not available in animals. The optimal method of determining the intensity of exercise using HR is by first finding out HRmax, as the intensity is most reflective of true VO_2 or exercise intensity, if expressed as a percentage of HRmax (Figure 5.5).

Maximal heart rate varies considerably between species and breeds of animals. For example the mongrel dog has an HRmax of approximately 300 beats per minute (Wagner *et al.* 1977) and the racing Greyhound 318 (Staadon 1984). The racing Thoroughbred or Standardbred horse can have an HRmax of 240 to 260 beats per minute (Tyler-McGowan *et al.* 1999). In horses, HRmax also decreases with age, with HRmax of horses over 20 <200 bpm compared with approximately 220 bpm in their younger and middle aged counterparts (Betros *et al.* 2002).

HRmax does not increase with training (Evans 1985) so ideally, a horse could be assessed early in its training and the same HRmax used to tailor the training over time. However, in the author's experience, early in training a horse may not be fit enough to reach its HRmax before fatigue. Therefore, it may be better to delay measurement of HRmax until 2 weeks or more into the training programme.

Using heart rate in training without having obtained HRmax could produce very different relative intensities of training. For example, in one group of Standardbred racehorses of the same age (mean 4 years), HRmax varied from 215 bpm to 260 bpm. The relative intensity of exercise at 160 bpm then varied in these individuals from 74% to 62% of HRmax. Therefore training intensity based on heart rate should be individualised for each animal.

In general, training heart rates are extrapolated from humans with early training heart rates in the 70% HRmax (50% VO_2max) range (154 bpm for a horse with HRmax of 220 bpm); medium or moderate intensity training 80% HRmax (70% VO_2max) range (176 bpm for a horse with

HRmax 220 bpm); high intensity training 90–100% HRmax (80–100% VO₂max) range (>198 bpm).

5.7.3 Lactate and its use in exercise and training

Lactate production is a normal response to energy production and while increasing lactate concentration has a negative effect on muscle contraction and energy production in the horse, it is unlikely to be a limiting factor for short-term high-intensity exercise (racing). Horses have an enormous ability to generate lactate and after a race or maximal exertion will frequently have plasma or blood lactate concentrations of over 25–30 mmol/l (resting 0.5 to 1.0 mmol/l) (Harris *et al.* 1987).

Lactate concentration increases when the speed increases above 7–9 m/s (Eaton *et al.* 1999). The speed at which it begins to increase depends on gait, breed, horse, diet and state of training (Figure 5.6). Lactate threshold or OBLA can be used in training programmes with a higher speed at OBLA in fitter horses and those with greater ability to transport and use oxygen (higher VO₂max).

In reality, determination of the lactate curves of horses are hard to achieve practically in a training situation, so shortcuts have been developed and validated, e.g. vLa4 (speed when lactate reaches 4 mmol/l or approximately OBLA), or La10 (the lactate concentration at a speed of 10 m/s). These derived values are comparable between horses and in the same horse over time. It should be noted that OBLA corresponds in the horse and human at least, to around 80% VO₂max. This is moderate intensity training and endurance athletes often use training at this intensity to maximise aerobic capacity or VO₂max. The racehorse at OBLA or about 80% VO₂max is travelling at 15 sec/furlong, or 13 m/s, or 800 m/min (that is a gallop) (Davie 2003). Where typically 'jogging' performed by Standardbred trainers and horsewalker exercise is quite low intensity. It is important to differentiate low-intensity exercise from moderate-intensity, and training programmes should incorporate moderate-intensity training as well as low- and high-intensity training.

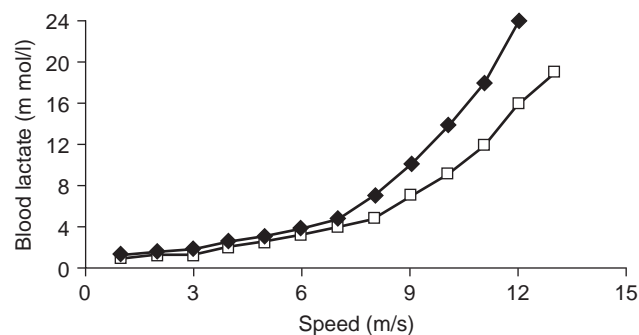


Figure 5.6 The typical relationship between blood lactate concentration and speed.

5.8 High altitude training

Current research for human athletes has demonstrated that living at high altitude and training at low altitude is superior to protocols involving high-altitude training, as the physiological benefits obtained by living and training at altitude are outweighed by the deficits produced in reduced VO₂ max and total work output (Hahn & Gore 2001). This protocol remains untested in horses. Certainly, physiological changes occur in the horse in response to hypobaric hypoxia, and these changes may be beneficial to athletic performance if they occurred at sea level. These include (Wickler & Greene 2004):

- Increased red cell number
- Increased blood volume
- Improved muscle capillarity
- Improved metabolic capacity of muscle

Unfortunately these changes have not yet been linked directly to performance improvements at sea level. The limitations of high-altitude exposure and training must also be considered. Apart from the risk of altitude sickness, VO₂ reduces by 15–30% (Navot-Mintzer *et al.* 2003) and the capacity to train at high intensity is reduced.

5.9 Maximal performance and factors limiting maximal performance in the horse

Performance in horses may be described as dependent primarily on energy supply (respiratory and cardiovascular systems and the transport of oxygen) and the ability to use that energy efficiently in the form of locomotion (musculoskeletal system). The equine athlete is efficient, coordinated and has been selected for athletic performance. Horses have a large aerobic capacity (>160 ml/kg/min), well over double that recorded on a per kilogram basis in elite human endurance athletes (70 ml/kg/min). This reliance on aerobic energy and oxygen supply during exercise in horses has helped the horse become a superior athlete, yet has implications in racehorse health because it leaves the racehorse highly susceptible to limitations in oxygen transport (Figure 5.1) and despite all these adaptations, the horse appears to have a respiratory limitation to maximal exercise. This is evidenced by the development of arterial hypoxaemia and hypercapnoea during intense exercise (galloping) (Art & Lekeux 1995). The horse has extremely high pulmonary vascular pressures to prevent rupture of the pulmonary capillaries under physiological conditions, so it has a relatively thick diffusion barrier, reducing the rate of diffusion, and this is the likely cause for much of the exercise-induced hypoxaemia (Christley *et al.* 1997).

5.9.1 Equine poor performance

Poor performance is an important area for the animal physiotherapist as many cases referred for physiotherapy have a

veterinary diagnosis of poor performance. Yet it is important to determine what could be occurring in the horse with poor performance. It is vital to assess nature of the performance problem: you should ascertain if the horse has never performed well, if there was a sudden decrease in performance, or if there was a decrease after improved class. When assessing horses for poor performance you should consider:

1. What might have contributed to the poor performance, and remember to look beyond your own sphere of expertise.
2. More than one problem can coexist.
3. In horses performing maximally, subtle, subclinical problems may be the reason for poor performance.

The performance issue may be different for horses of different disciplines, e.g. for dressage horses soundness, coordination and muscle control are essential or for endurance horses fluid and temperature regulation are essential. In the poor-performing racehorse poor performance resulting from disorders affecting oxygen transport or energy supply often presents similarly, despite a myriad of causes and is generally described as 'stopping' at the end of a race. Examples of such disorders include upper and lower respiratory tract disorders, cardiac disease and disorders of the red blood cell oxygen carrying capacity (anaemia) but can be any problem along the pathway of oxygen (Figure 5.1). Other problems may be present such as respiratory stridor (noise), myopathy (tying-up) or any musculoskeletal injury or disease (unsoundness). Muscle metabolic capacity is rarely limiting to performance, and anaerobic capacity is rarely routinely assessed (Martin *et al.* 2000).

A key point is that because racehorses exercise maximally, subtle or subclinical problems are enough to dramatically affect performance.

5.9.2 Upper respiratory tract disorders

Upper respiratory tract disorders affect oxygen transport by increasing the resistance of breathing and reducing airflow. This reduces the amount of oxygen available for the rest of the oxygen transport chain. The increased resistance to breathing is usually accompanied by a noise called respiratory stridor. The horses' unique anatomy and physiology only serve to compound any problems that exist. The horse is an obligate nasal breather so any anatomical narrowing of the nostrils or nasal passages will reduce performance. There is also a complicated relationship between the larynx and soft palate that make this area particularly susceptible to disorders. Owing to the importance of the diameter of the airway passages to airflow and the airway resistance that develops even with 'normal' breathing during maximal exercise, very small alterations in the diameter of structures can seriously affect performance.

The most common disorder is the paralysis of the left side of the larynx called idiopathic laryngeal hemiplegia

(ILH). The disease is caused by paralysis of the left recurrent laryngeal nerve that innervates the muscle of the larynx required for the opening and closing of the arytenoid cartilages. Other laryngeal problems include dorsal displacement of the soft palate and disorders of the epiglottis. Dorsal displacement of the soft palate is more common in Standardbred than Thoroughbred horses. It occurs when there is a loss of the seal between the soft palate and the larynx, resulting in a dorsal movement of the soft palate up in front of the larynx, obstructing airflow. Epiglottic and other disorders are less common.

5.9.3 Lower respiratory tract disorders

Lower respiratory tract disorders affect oxygen transport by reducing the diffusion of oxygen (and carbon dioxide) across the alveolar wall into the pulmonary capillaries, although some lower respiratory disorders also affect airflow through the smaller bronchioles (bronchoconstriction). It is postulated that the horse has evolved in such a way that its athletic capabilities are greater than the functional capacity of its respiratory system. Huge pulmonary vascular pressures are generated during maximal exercise and the diffusion membrane is as thick as it can be to still maintain diffusion. While this means that the horse can withstand (in many cases) the enormous vascular pressures that other species would not be able to, it leads to a diffusion limitation, evidenced by hypercapnoea (high CO₂) and hypoxaemia (low O₂) during maximal exercise. What this means is that the healthy equine lung is at the limit of its capabilities during maximal exercise and any alteration to its function will reduce performance significantly.

The three main lower respiratory tract diseases are:

- Inflammatory airway disease (IAD)
- Exercise-induced pulmonary haemorrhage
- Infectious disease

Inflammatory airway disease (IAD) is a syndrome of small airway inflammation and resultant obstruction without infection that results in poor performance. Some people believe that it is an early form of the more severe clinical disease called recurrent airway obstruction (RAO), or heaves, that has some similarities with human asthma. In many cases it is clinically inapparent, although at other times it may result in coughing or nasal discharge. It is frequently associated with poor housing management, may be seasonal and may be associated with exercise-induced pulmonary haemorrhage.

Exercise-induced pulmonary haemorrhage (EIPH) is another significant lower airway disorder of racehorses. It is due to bleeding from the dorsocaudal lung lobes and can result in clinical epistaxis (nose bleeding). The pathogenesis of the disease is not clear but is likely to relate to the abnormally high pulmonary vascular pressures during exercise, causing rupture of capillaries into the alveoli (despite the relatively thick diffusion barrier). EIPH actually occurs

to some degree in most (probably >90%) racehorses exercising maximally, detected by bronchoalveolar lavage. However, blood appearing at the nostrils (resulting in the horse being banned from racing in some countries, including Australia) occurs in considerably fewer than 1% of horses. It is not always associated with a reduction in performance, and performance is usually only reduced in those horses that have a severe episode.

5.9.4 Anaemia

Reduced circulating red blood cell volume, or anaemia, affects oxygen transport by reducing the binding of oxygen to haemoglobin because of reduced haemoglobin. Anaemia is, however, rare in the athletic horse. The horse has the unique ability to contract its spleen during exercise and as a result can release up to 12 l of extra blood into the circulation during exercise. Disease and possibly nutritional deficiencies could cause anaemia.

5.9.5 Cardiac disease

Cardiac disorders affect oxygen transport by reducing the transport of oxygen from the lungs to the muscle where it is required to produce energy. There is a high prevalence of cardiac murmurs in racehorses, many of which do not affect the horse at all, e.g. 16% of National Hunt racehorses have a right-sided murmur associated with tricuspid valve regurgitation (Patterson & Cripps 1993). Many murmurs simply represent turbulent blood flow and are called physiological murmurs. A number of murmurs however represent serious cardiac disease and limitations to performance through reduced cardiac output and cardiac failure. Horses may also suffer from arrhythmias reducing the cardiac output. If these are secondary to cardiac failure then they are always associated with a loss of athletic function. However many are associated with abnormal electrical activity and may be treated effectively. The most common rhythm disturbance is atrial fibrillation and it is more common (in the uncomplicated form) in young Standardbred horses.

5.9.6 Musculoskeletal disorders

Musculoskeletal disorders are the most common cause of poor performance either alone or in combination with other diseases. In the majority of cases the horse will be clinically lame or unsound and a veterinary examination and/or physiotherapist's assessment will often reveal the source of the problem. However, in some cases there is a reduction in performance without obvious clinical signs and more sophisticated techniques like gamma scintigraphy and gait analysis may need to be employed to find the cause (Chapter 6).

5.9.7 Other factors

A number of other factors are important for successful performance, including mental attitude or the 'will to win', and behavioural issues (particularly with overtraining or

staleness – see later). The racehorse, like all horses, is dependent upon appropriate levels of nutrition. Also, abdominal disease, particularly gastric ulcers (Chapter 3) can be an important problem in racehorse performance.

5.9.8 Overtraining syndrome in horses

Overtraining is simply an imbalance between training and recovery – either the training stimulus is too great or the recovery too short. Over time this can result in a syndrome of fatigue and poor performance, usually accompanied by one or more other signs, e.g. weight loss, psychological changes and susceptibility to infections. Overtraining is a chronic syndrome that takes many weeks to months for recovery. It must be differentiated from overreaching that is an acute form of overtraining that only takes a few days for recovery.

In humans, overtraining can result from a training programme using the principles of overload training, but poorly regulated. Overload training techniques are commonly used in elite human athletes and are a fine tuning of training and maximising the recovery response – in fact trainers aim to induce overreaching and time the 'super compensation' or overcompensation for a competition (Figure 5.7).

Horse trainers certainly don't commonly use overload training principles, however similar principles, e.g. 'tapering' or reducing exercise just before racing may cause similar responses. In humans, overload training is closely monitored by trainers and sports psychologists.

Signs of overtraining in horses include:

- Poor performance (by definition)
- Loss of body weight, loss of appetite
- Muscle pain or increased muscle enzymes
- Increased susceptibility to gastrointestinal or urinary infections
- Incoordination
- Increased rate of injury

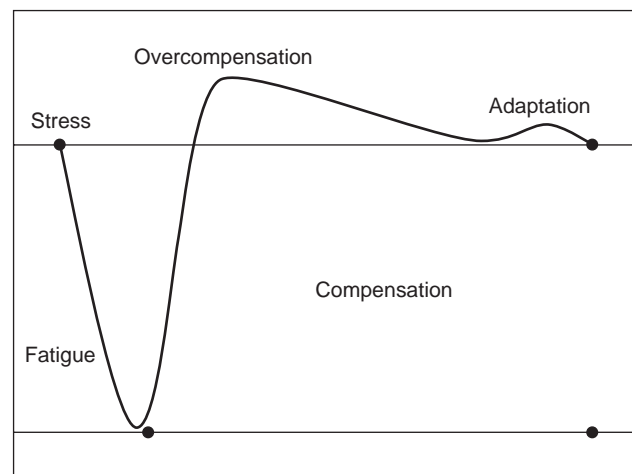


Figure 5.7 Overload training principles (Davie 2003).

- Altered heart rates/lactates/ VO_2
- Unwillingness to train
- Nervousness
- Behaviour changes

Diagnosis in human athletes relies heavily on mood profiling – behavioural changes in horses too can predominate. Diagnosis of overtraining in horses relies on identification of poor performance, exclusion of other major causes, identification of loss of body weight and behavioural changes. There may be subtle changes on blood tests, but these are frequently unrewarding. Human athletes tend to overtrain when social stresses are added to the stress of training and competition. Similar stresses in horses might be illness (e.g. viral respiratory disease), gastric ulcers, transport, boring routine, subtle lameness or musculoskeletal pain.

Ideal treatment of overtraining involves prolonged rest, decreased training load (especially high-intensity training) and addition of variety to the training and management routine. Examples might include hacking the horses if they are quiet, flat work, cavaletti work; turn the horse out into a paddock or a change of yard. Also good husbandry to minimise external stresses, e.g. transport, illness and gastric ulcers, is important. Differentiation from overreaching is important: e.g. horse prepared too quickly that will have a response to short-term rest – this response is likely to be great, i.e. super compensation, so it would be ideal to be able to use the response to the trainer's advantage.

5.10 Training the sled dog (Husky)

The following sections outline some of the principles of how exercise physiology may be utilised to design training programmes in the two extremes of dog sport – the Husky dog and the Racing Greyhound.

5.10.1 Profile of the Husky as an athlete

The Husky was bred in Siberia under very cold and harsh conditions. The main purpose of the breed is to pull a load (a sled) to transport people and goods across snow and ice for very long distances. The Husky is therefore a thick-coated dog, of medium build and designed for strength and endurance. The dog is obviously very robust and tough and readily trainable. In Australian conditions, overheating during training and racing would be expected to be a major issue.

VO_2max of 154 ml/min/kg was measured in a group of trained foxhounds (Musch *et al.* 1985). The aerobic capacity of the Husky, although not yet determined, could be even greater. This performance is impressive compared with humans (70 ml/min/kg) and elite Thoroughbreds (175 ml/min/kg). Van Citters & Franklin (1969) measured the working heart rate in Huskies at over 300 bpm for extended runs of over 1 hour duration. Resting heart rate was typically between 40 and 60 bpm. Stepien *et al.* (1998) trained a group of 77 Huskies pulling a sled for an average of

20 km/day for 5 months. There was no dropout during this 5-month period, attesting the durability of the Husky.

5.10.2 Profile of the sled dog race

The Iditarod Sled Dog Race in the USA is an example of top-level competition at the extreme end of the performance spectrum. The race covers 1692 km in 12 days, averaging 141 km/day. The race has an energy demand for the human sled driver alone of 10 740 kJ/day (Cox *et al.* 2003).

Australian conditions do not favour the sled dog racing industry as the window of opportunity for racing on snow is small, the terrain is limited, and conditions are generally too warm to allow longer races. Therefore the majority of sled dog racing in Australia is over ground not snow, and races are generally limited to 20–25 km. They are often raced in the early morning, as temperatures cannot be higher than 10–12°C (Canberra Sled Dog Club 2005).

A 25-km race in Australia is generally run in 1 hour. Therefore the speed at this middle distance race is 7 m/s. A 30-km race would be expected to take 75 min at a speed of approximately 7 m/s (Canberra Sled Dog Club). Stepien *et al.* (1998) recorded oxygen uptake of 40% VO_2max at 4.4 m/s. He notes that the majority of Husky training is done at this speed. Reynolds *et al.* (1995) recommends anaerobic conditions in Huskies at 3 min at 6.7 m/s on a 10° slope.

Ready & Morgan (1984) investigated physiological responses in Siberian Husky dogs during a 90-s sprint, a 7.5-km free run and a 6-km team sled run. They measured blood lactate levels of 1.74, 0.70 and 3.06 mmol respectively 3 min after the run, with heart rates of 190, 211 and 166 bpm immediately on completion. These figures suggest very efficient aerobic energy utilisation in the dog. The study does, however, highlight the significant anaerobic requirement of the sled run in comparison to the free run. This is probably due to the extra load requirements of pulling a sled and demands of challenging terrain.

Summary of race profile

- 30-km race over variable terrain
- 75 min duration
- Average speed of 7 m/s (25 km/h)
- 4.4 m/s is equivalent to 40% VO_2max
- Energy supply is predominantly aerobic but bouts of anaerobic metabolism are expected
- Typical VO_2max for the sled dog is assumed to be 145–155 ml/min/kg or higher
- Blood lactate levels under race conditions are unknown
- HR max exceeds 300 bpm and can be sustained over long periods

Components of a proposed sled training programme

- Fitness testing
- Play
- Cross-country course

- Dry treadmill – loaded and unloaded
- Wet treadmill
- Track work

5.10.3 Fitness testing

Fitness test data should form the basis of the exercise intensity prescription. Exercise prescription is best prepared for each individual dog. Evans (2000) notes that there are significant differences in the response of physiological parameters to exercise between individual horses, and within individuals at different levels of training. Fitness testing will include:

- Health and lameness assessment
- Determination of HR_{max} and VO₂max if possible
- Determination of blood lactate level vs. speed
- Determination of heart rate vs. speed

For health and lameness assessment see Chapter 6.

Determination of VO₂max

Maximum oxygen uptake defines the aerobic capacity of the horse (Evans 2000) and it has been used for many years as the measure of the aerobic capacity in humans. The ability of the sled dog to race over 30 km will be largely dependent on the VO₂max. Training dogs for 8–12 weeks of treadmill running at 80% max heart rate, 5 days per week, improved VO₂max by 31% (Musch 1985).

This measure should be used as one of the main indicators of the response of the dog to the training programme and will determine training speed for interval training. In a major review article on high intensity interval training in human endurance athletes, Laursen and Jenkins (2002) report the recent use of the velocity at which VO₂max is achieved as the interval intensity. Tyler-McGowan *et al.* (1999) also report value in this intensity for the aerobic capacity training of horses. Ideally, the velocity at VO₂max should be tested fortnightly during interval training periods so that the interval training speed can be adjusted with improvements in aerobic function. It is expected that the training velocity will require an increase of approximately 3% per week (Trilk *et al.* 2002).

Determination of blood lactate level vs. speed

Blood lactate levels indicate the contribution of anaerobic metabolism to the energy supply. This curve is well known in horses and humans and has been investigated in the dog. Blood lactate levels in the horse rapidly rise during exercise speeds greater than 65–85% VO₂max (Evans 2000), and regularly increase to 20–30 mmol. The only documented evidence of lactate levels in the trained Husky is at 3.06 mmol, 3 min after a 6-km sled run (Ready & Morgan 1984). Training in both humans and horses typically results in a shift of the curve to the right, indicative of an important training adaptation. Ready and Morgan (1984) observed no shift in the curve in response to a 12-week interval training

programme in Huskies, perhaps as a result of insufficient intensity of exercise. Musch *et al.* (1987) observed significantly lower blood lactate levels at a given level of sub-maximal exercise following an aerobic training period.

Although exercise speeds can be calculated according to VO₂max, blood lactate levels can be taken for future reference. The speed at which a blood lactate level of 4 mmol (vLa₄) is reached is significant as an interval training marker, as it is used widely in the horse industry, is repeatable, and reliably associated with racing success (Trilk *et al.* 2002). It is of interest that the same marker is used by elite human sprint and athletic trainers as a guide to training intensity (Steinacker 1993). Therefore, it is reasonable to assume that the same marker could be of value as a training tool for canine athletes.

Determination of heart rate vs. speed

The heart rate to running speed relationship in humans (Steinacker 1993) and horses (Trilk *et al.* 2002) is linear at moderate to high workloads and is reproducible. There is a well-documented shift to the right of the curve with training in horses (Evans 2000) and humans. Musch *et al.* (1985) reported a lower heart rate at any level of submaximal exercise following an aerobic training programme. Heart rate is easily measured in the field situation and is a simple way to measure workload. Some degree of caution should be noted however, as heart rate tends to rise in dogs with anticipation of a stressor (Vincent *et al.* 1993) without an actual increase in workload. Heart rate can be monitored and recorded during testing and training sessions by a portable monitor/computer. This information can be downloaded, along with speed and distance information from a portable GPS unit to give valuable feedback on training performance and be used for future reference. If heart rate data is consistently reliable, and in line with VO₂ and blood lactate levels and adaptations, heart rate responses alone may be used to modify exercise intensity.

5.10.4 Training

Play

Play is important to relieve training stress and for ongoing bonding between the trainer and dog. Play can also involve high-speed sprints (chasing a ball), jumps and swimming to add variety to the training programme. These sessions allow the trainer to include valuable suppling, balance and proprioception components to the programme design.

Cross-country course

The cross-country course is designed to give the dog long runs (10–15 km) in an interesting and stimulating environment, while challenging the dog with high work demands and variable terrain. The trainer will accompany the dog on the course on a wheeled sled or bicycle and monitor and control speed, workload and distance. An inventor has recently produced a lightweight jig, suitable for one or more

dogs to tow a driver. The driver is well balanced behind the wheel axle so that the load on the dog is minimal and can even be modified for preference to transfer some weight bearing off the dog and onto the jig axle.

Cross-country work forms a large part of basic training, to prepare the dog's musculoskeletal system for the training ahead, while providing a stimulus for aerobic work. Stepien *et al.* (1998) reported 77 Huskies were trained for 5 months at 4.4 m/s (40% VO_2max) for 20 km/day with no reported dropout. The proposed training goal of 120–150 km/week is therefore realistic, and given the robustness of the breed, is unlikely to lead to overtraining and injury under close monitoring.

Dry treadmill – loaded and unloaded

The treadmill provides a secure environment for speed and incline while allowing the close monitoring of several physiological parameters (fitness test). Treadmill training provides a level surface with reduced injury risk. The dog can be loaded as per sled condition by adding a harness and loaded at will, via a weight and pulley set-up. Overloading on the treadmill allows for accurate and measurable overload in a situation specific to the race conditions. Strength training is used extensively in human athletes and has been shown to improve the pattern of neural drive (Judge *et al.* 2003) and reduce muscle injury rate (Croisier *et al.* 2002) in track athletes. Gonyea and Sale (1982) describe the major features of muscle adaptation to weightlifting exercise as changed in contraction time, fibre size and fibre number. The same could be expected with weight-loaded dog training.

The training effect of sled towing has been measured in humans, as this is a popular form of resistance training in sprinters. Lockie *et al.* (2003) found that a sled load of 12.6% of body mass had a performance benefit above a load of 32.2% body mass. Kinematics were also observed during this study. It is important to note that the 32.2% body mass load had an effect of reducing stride length by 24% and stride frequency by a lesser extent. In addition, sled towing increased ground contact time, trunk lean and hip flexion. These are important issues for training the sled dog. It is well established that training needs to be as specific as possible so that the right systems and muscle functions can adapt optimally. Training in a predominantly unloaded situation will not prepare the dog to race optimally in a loaded situation.

Stepien *et al.* (1998) support the idea of sled pulling in training as it applies a significant isometric load as well as the isotonic training of endurance running. They found training adaptations in excess of those found in non-pulling training, particularly with respect to cardiac function.

Wet treadmill

The wet treadmill allows the dog to exercise against the resistance of the water at belly level while walking, trotting and running at variable speed. This exercise in particular

allows the loading of the limb protractors, which cannot be overloaded by pulling a sled. Human athletes benefit from weight training of these powerful forward flexors of the limbs, which are responsible for driving the limb forward to foot strike. The water environment also allows the Husky to be trained in a cool environment. Running in the water at the beach, dam or river is an obvious alternative.

Track work

Track work can form the basis of the interval training and high speed training programmes. Once again, work can be done under harness under the direction of the driver/trainer while monitoring speed and distance via GPS unit and heart rate monitor. The interval training programme should be split between the treadmill and track to relieve boredom and avoid staleness or overtraining.

5.11 Programme phases

An example is given of a 16-week programme, divided into four phases:

- *Basic training*: weeks 1–4
- *Sub-strenuous/resistance training*: weeks 5–8
- *Strenuous interval training*: weeks 9–14
- *Taper*: weeks 15–16

Basic training phase

Art *et al.* (1995) found significant reductions in performance-related variables in horses with 2–6 weeks of rest. Rest in human athletes has more profound effects. Four weeks of rest in endurance trained dogs is likely to have similar effects of reducing VO_2max by 15–20%. While allowing the musculoskeletal system recovery time, rest may leave the animal more vulnerable to injury under strenuous exercise conditions. The 4-week 'basic training' phase is designed to bring the dog gradually back into the training regime while allowing activities for skill development and 'fun'. During this period the trainer should form a close and confident relationship with the dog, which will assist in accomplishing the more strenuous activities later in the programme.

Sinha *et al.* (1991) reported no difference between VO_2max following basic training of 40% and 80% VO_2max . Musch *et al.* (1985), in a series of two studies, measured 31% gains in VO_2max , training at 80% maximum heart rate. It can be concluded that light exercise designed to prepare the musculoskeletal system is not conditional on intensity and that low to moderate intensity work is sufficient to stimulate some preparatory gain in VO_2max . It must be remembered, however, that the Husky is an endurance athlete, capable of pulling a load in excess of 140 km/day. A low intensity 10 km walk/jog for a previously fit dog is not a big ask. Second, the endurance Husky, unlike the racehorse, is a mature animal. There are no age

restrictions on races and no incentive to race at an early age. The incidence of injury due to immature musculoskeletal system is therefore likely to be lower than observed in horses.

During this period, the dog can be introduced to the various training venues and familiarised with the surroundings, regimes and equipment required in the training programme. It is best to familiarise the dog with a new procedure in a low-stress situation without time constraints. Attention should be given to address suppling, balance and proprioception tasks during this period to reduce incidence of training- and race-related musculoskeletal injuries.

Sub-strenuous/resistance training

The goals for this period are to:

- further improve aerobic performance in preparation for the next more intensive training phase;
- progressively overload the musculoskeletal system in a way that is specific to the event situation to promote performance-enhancing adaptation and injury prevention;
- further develop the training regimes and become expert with the training methods;
- continue to work on suppling, balance and proprioception tasks for injury prevention.

High-intensity exercise must be gradually introduced to allow adequate stimulation and time for musculoskeletal and metabolic changes to occur. Significant changes in endurance performance-related variables will occur in the dog during a short endurance-training programme (Musch *et al.* 1985), but evidence from human studies strongly suggests that further improvements will not occur unless the intensity is lifted. This period of training will gradually introduce the dog to the interval training protocol, which is used to obtain the required intensity of training. Training intensity should be based on velocity at VO_2 max or HRmax obtained at the pre-phase fitness test and increased each week.

Resistance training forms a part of all human athletic endurance and strength training programmes, but is underutilised in the training of animals. This is no doubt due to difficulties in training design. Animals cannot be easily taught to lift heavy weights. Saunders *et al.* (2004) concludes that 'strength training allows the muscles to utilise more elastic energy and reduce the amount of energy wasted in braking forces'. Apart from improvements in performance with resistance training (Saunders *et al.* 2004), there are the benefits of injury prevention, particularly following previous muscle injury (Proske *et al.* 2004).

Strenuous interval training

Laursen and Jenkins (2002), in a major review article on interval training in human endurance athletes, report that increases in volume of training in highly trained athletes do not further enhance either endurance performance or associated physiological variables. It seems that, for athletes who are already trained, improvements in endurance

performance can only be achieved through high intensity interval training.

The intensity of interval training in a previously fit athlete can be very high. Creer *et al.* (2004) found in human athletes, that a twice weekly, 4-week sprint interval programme, consisting of 4×30 -second sprints, in addition to an endurance training programme, was superior to an endurance training programme alone. They measured significant gains in motor unit activation, total work output and increased blood lactate levels.

Although some variety should be kept in the programme to relieve boredom and stress, this 6-week phase can focus on high intensity interval training on the track and on the wet and dry treadmill. More rest intervals and longer recovery times (Laursen & Jenkins 2002) are essential as intensity of training is increased. The velocity at which VO_2 max or HRmax is achieved may be selected as the goal interval speed. This intensity has been supported as the probable optimal training intensity for improving aerobic performance in both human studies (Laursen & Jenkins 2002) and horse studies (Tyler-McGowan *et al.* 1999). It should be noted that reassessment of aerobic capacity is required at regular intervals, as the intensity needs to increase with improvements in VO_2 max. Overtraining is a risk at this period of training and signs should be closely monitored.

Velocity at VO_2 max or HRmax can be determined on both wet and dry treadmills. Track training speed will be identical to the dry level treadmill speed. Ready and Morgan (1984) recorded high blood lactate levels with sled running (3.06 mmol) in Huskies compared with free running (0.70 mmol). It is expected also that the dog will exhibit high blood lactate levels and VO_2 at lower wet treadmill velocity than dry treadmill velocity.

Taper

The aim of the taper is to reduce the physiological and psychological stress of daily training and optimise performance. Good, evidence-based data on tapering methods in animals does not exist in the literature. However, tapering for human athletic events is a well-recognised practice and a well-researched topic. Tapering before a middle distance endurance event improves performance by 5–6%, due to positive changes in the cardiorespiratory, metabolic, haematological, hormonal, neuromuscular and psychological status of the athlete (Majika & Padilla 2003). It is agreed that tapering is best achieved by maintaining intensity of training while reducing training volume (60–90%) and slightly reducing training frequency (no more than 20%). Neary *et al.* (2003), report a 50% training volume reduction for 7 days before performance is superior to 30% and 80% reductions, and revealed a 5.4% performance improvement.

The dogs should be tapered progressively over a 2-week period before competition. Training volume can be reduced by 25% in week 1, and a further 25% in week 2. Training

intensity should remain the same. The number of training sessions will reduce over the 2-week taper period.

5.12 Aims of the programme design

Cardiorespiratory responses

- Training-induced bradycardia and lower HR at a given velocity
- Increased VO_2max by 30–40%
- Increased maximum cardiac output by 30–35 %
- Compensatory hypertrophy of the heart (up to 24% in Huskies)
- Reduced systemic vascular resistance by 25% during maximum exercise
- Increased velocity at which V_4 is reached, goal of at least 20% increase

Musculoskeletal responses

- Increased muscle fibre area for both slow-twitch and fast-twitch muscle fibres
- Better utilisation of elastic energy due to strength training
- Increased muscle strength in both the propulsive and protracting musculature
- Increased muscle contraction time
- Increased resting intramuscular glycogen (15–20%) content. Enhanced muscle oxidative capacity
- Increased motor unit activation/improved pattern of neural drive
- Increased tendon cross-sectional area
- Increased bone density

Injury prevention

- Low injury rate due to strength training and low-velocity long-distance training, in addition to improved flexibility, balance and proprioception through specifically targeted exercises
- Avoidance of overtraining by varying training activities and regular fitness testing/health and lameness checks

5.13 Training the racing Greyhound

5.13.1 Profile of the Greyhound as an athlete

The origins of the Greyhound are in recreational hunting. Dogs are bred to sprint and are very fast over a short distance and still have a strong hunting instinct. The dog typically weighs around 28–30 kg and has a very low percentage body fat (Schoning *et al.* 1995). The Greyhound is a long-legged dog, with large proximal muscle mass and light distal limbs.

Grandjean *et al.* (1983) in studies on the racing Greyhound, remarks on the similarities in the metabolisms of the Greyhound and human sprinters. There is surprisingly little research available on training methodology and adaptations

to exercise, so much of the theory is extrapolated from human sports medicine. The Greyhound is capable of running 500 m at 18 m/s. In comparison, the Quarterhorse can just beat him at 20 m/s and man is way behind at over 10 m/s over 400 m. The Greyhound has a higher percentage of fast-twitch fibres than other dog breeds (Guy & Snow 1981) and can produce blood lactate levels of 27 mmol (Rose & Bloomberg 1989). Lactate peaks 5 min after exercise and returns to resting levels by 30 min after a 400-m sprint race (Rose & Bloomberg 1989).

5.13.2 Profile of the Greyhound race

Greyhound races in Australia are typically over 297 m and are won at the elite level in about 17 s. The race is run in an anticlockwise direction from a standing start. There are several dogs in the race and dogs regularly interfere.

The Greyhound relies predominantly on anaerobic metabolism during the 17-s sprint race (Rose & Bloomberg 1989). VO_2max is not known, but a very high heart rate maximum suggests a higher VO_2max than expected for a sprint athlete with very little endurance training.

A dog is typically 'trained' by daily walks, some track work and the odd run on the bait to improve the 'desire'. It is generally thought that the dog has bred-in ability to run fast. However, research has shown that performance improvements can be achieved and that the dog has a similar metabolic reaction to training as the human sprinter.

Injuries on and off the racetrack are common in Greyhounds. The most common are right hindlimb gracilis muscle tears, fracture of the central metatarsal in the right hindlimb, fractured carpi in either forelimb (Schoning 1994) and shin splints on the left (railing) forelimb (Davis 1971). These bony injuries most likely result from training errors, either overtraining of young dogs leading to breakdown, or under-preparation of the musculoskeletal system to cope with the rigors of sprint training and racing. The high injury rate requires special attention in training design.

Cornering is a skill that requires learning at a slow speed and practising at increasing levels of difficulty until the dog is competent at race speed. We cannot expect the dog to have the motor control at pace in cornering to avoid injury if it is not well rehearsed in the task. In addition to motor control development, is the importance of musculoskeletal development to withstand the angular forces sustained during cornering. Tissue is organised along the lines of stress. Therefore there must be a significant amount of angular stress in the preparation and training programme to allow suitable bone, muscle, tendon and ligament adaptation. The issue of 'one sidedness' then appears. In reality, the racing greyhound is an athlete with a unilateral bias. Just like a right-handed tennis player, the dog should develop an acceptable asymmetry.

Summary of the race profile

- Approx. 300 m all-out sprint
- Run anticlockwise on circular track

- 17 s race time
- Predominantly anaerobic metabolism
- Soft-tissue and bony injuries are common

Programme components; evidence to support training methodology

- Fitness testing
- Skill development and basic training
- Track work under harness
- Wet treadmill
- Play/stretching/balance and proprioception training
- Sprint work
- Race track

5.13.3 Fitness testing

Regular health and lameness assessment will be important during the training programme. Particular care is required to assess for signs of bony soreness and muscle injury.

VO₂max can be measured in the Greyhound, as in the Husky, as an indicator of improved aerobic capacity. In reality HR is more commonly used as a training indicator, owing to the unavailability of VO₂ measurements in dogs.

As the athlete adapts to anaerobic-based training, the blood lactate level at a given velocity should reduce. This is an indication of efficiency (Creer *et al.* 2004). However, training adaptations should also allow the generation of higher blood lactate levels during maximal work. To measure these markers, the velocity must be controllable, thus the treadmill is ideal.

5.13.4 Skill development and basic training

The Greyhound has the same basic training requirements as the Husky in terms of musculoskeletal adaptations. Whereas the Husky is reliant on a large aerobic capacity to complete the 30-km race, the Greyhound requires aerobic fitness to endure the rigors of the pre-race training programme. Suppling, balance and proprioception training is important during this period.

Straight endurance work will be the main feature of this exercise but will be combined with skill development. The Greyhound must become very proficient at cornering left to cope with the racetrack situation. The trainer should regularly circle the dog left, first at a walk and later at a trot and run. The Greyhound will learn to balance itself during cornering and adapt neural mechanisms and tissues to suit the activity. This will start the grounding in injury prevention as well as performance enhancement.

Track work under harness

The dog can be taught to pull a harness. Weighted sled towing is a common resisted sprint training technique in human athletes (Lockie *et al.* 2003). While this technique has the disadvantage of changing sprint kinematics (Lockie *et al.* 2003), it allows the trainer to adjust the velocity and

load on the dog and regulate adequate rest breaks during interval training. It also allows further skill training and specific loading for cornering.

Wet treadmill

Lockie *et al.* (2003) and Judge *et al.* (2003) have good evidence that resistance training is beneficial for both sprint performance and injury prevention. Resistance training has the effect of improving the pattern of neural drive, increasing muscle fibre size and speed of contraction and increasing power output in the activity specifically trained. The wet treadmill at belly depth provides a resistance for the limb protractors during the swing phase of gait. This is combined with a harness weight to provide a load to the propulsive muscles of gait.

This activity closely matches fast running and fulfils some of the requirements for resistance training to be beneficial (Gonyea & Sale 1982). Saunders *et al.* (2004), in a major review on running training techniques, summarises the value of strength training as allowing muscles to utilise more elastic energy and reduce the amount of energy wasted in braking forces.

Play/stretching/balance and proprioception training

This session provides a good bonding opportunity between dog and trainer and can allow some controlled sprint work, e.g. ball chasing and retrieval. A muscle-stretching session, particularly of the hindlimb hamstrings and adductors should be incorporated. Balance and proprioception exercises can include a variety of terrain and obstacles to work over and around, to enrich the training environment.

Sprint work

The open field can be used. This session will form the basis of specific sprint speed development. A GPS unit secured to the Greyhound via a Lycra singlet can provide velocity feedback. Attention should be paid to behavioural management in the starting cage to avoid bad habits developing during racing.

Racetrack

Specific sprint work on the racetrack is required towards the end of the programme to prepare the dog psychologically for racing and to provide more specificity in incorporating the particular musculoskeletal stresses of the racetrack. Further work on behaviour in the starting cage should be done to assist in relieving race stress and bad behaviour which may affect performance.

5.14 Aims of the programme design

Cardiorespiratory responses

- Training-induced bradycardia and lower HR at a given velocity
- Increased VO₂max by 30%

- Increased maximum cardiac output by 30–35%
- Compensatory hypertrophy of the heart (up to 24% in Huskies)
- Reduced systemic vascular resistance by 25% during maximum exercise
- Increased velocity at which V4 is reached, goal of at least 20% increase
- Increase exercising blood lactate levels

Musculoskeletal responses

- Increased muscle fibre area for both slow-twitch and fast-twitch muscle fibres
- Better utilisation of elastic energy due to strength training
- Increased muscle strength in both the propulsive and protracting musculature (24%)
- Decreased muscle contraction time
- Increased resting intramuscular glycogen (15–20%) content. Enhanced muscle oxidative capacity
- Increased motor unit activation/improved pattern of neural drive
- Increased tendon cross-sectional area
- Increased bone density
- Selective musculoskeletal development to handle the stress of cornering left

Injury prevention

- Lower injury rate due to strength training and low velocity long distance training
- Avoidance of overtraining by varying training activities and regular fitness testing/health and lameness checks
- Selective musculoskeletal development to handle the stresses of high race speeds and cornering left
- Correction of muscle tightness due to stretching programme. Particularly in the hind limb adductors and hamstring muscles
- Improved balance, coordination and proprioception

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6

Equine and canine lameness

Nicholas Malikides, Thomas McGowan and Matthew Pead

6.1 Equine lameness
6.2 Canine lameness

References
Further reading

6.1 Equine lameness

Nicholas Malikides

6.1.1 Introduction

Orthopaedic disorders in horses occurring before and during their performance careers represents a significant cost to the equine industry and is the focus of considerable research. Surveys in racehorses indicate that orthopaedic disorders, primarily those that result in lameness, are the most frequent reason for loss of performance and interruption in training (referred to as wastage) (Bailey *et al.* 1997; Dyson 2000). This chapter will focus on lameness problems related to bones, joints, tendons and ligaments. For more information on muscle disease see Chapter 7.

Veterinarians and trainers have long recognised that many factors contribute to the development of orthopaedic problems, usually manifested as lameness, in horses. For example, lameness may be the result of trauma (related to ground surface factors and to exposures to injurious foreign bodies), congenital or acquired anomalies, infection, metabolic disturbances and circulatory disorders; or any combination of these. Furthermore, in all horses that are lame, consideration of inherent conformational faults, training, riding and shoeing factors or even character flaws in the horse is essential. In addition, because the nervous system is intimately connected to the musculoskeletal system, neurological disorders may secondarily result in gait deficits, unexplained lameness or muscle atrophy. Examples include stringhalt, shivers and equine herpes virus 1 myeloencephalopathy (Chapter 7).

Lameness as a result of orthopaedic or musculoskeletal problems is one of the most common reasons for veterinary attention and in turn, referral for physiotherapy, in all breeds and types of horses. Lameness is an abnormality in movement or locomotion, particularly evident in unloading patterns of gait associated with pain. However, despite the myriad causes of lameness, this unloading pattern may not be very different. It is therefore essential for physiotherapists

attending to lame horses to have an accurate diagnosis of the orthopaedic disorder made by a veterinary surgeon and to understand the pathoanatomical principles of this diagnosis. This will allow appropriate veterinary treatment, more specific physiotherapy treatment and an accurate prognosis to be made. For example, there is no advantage in treating secondary muscle pain in the shoulder, scapular and pectoral region of a horse with an undefined chronic distal interphalangeal joint osteoarthritis. While such treatment may result in short-term improvements, the absence of a definitive diagnosis will result in a 'revolving door' of unsuccessful treatment, and ultimately client dissatisfaction. Further, due to the changeable nature of musculoskeletal problems in athletic animals, it is important to always include in a physiotherapy assessment, a complete lameness examination and referral back to the veterinary surgeon for pathoanatomical diagnosis of new problems that may have arisen. See also Chapter 8.

In this section, we will attempt to outline broadly the important anatomical, pathophysiological, mechanistic and therapeutic principles of equine orthopaedics with particular emphasis on the lame horse.

Veterinarians and animal physiotherapists require a detailed knowledge of functional anatomy to assess orthopaedic disorders. For biomechanics see Chapter 4. Comprehensive anatomy texts are available (see Further reading). The benefits of becoming completely familiar with topographical anatomy cannot be overemphasised.

6.1.2 Conformational and clinical terms and definitions

There is a strong association between conformation and predisposition to lameness (Marks 2000). However, predicting the timing and exact nature of the lameness (particularly when examining weanlings or yearlings) may prove impossible. Not all conformational variations predispose to lameness in a racing career, e.g. in one study, carpal effusion and incidence of carpal fracture was decreased in Thoroughbreds with carpal valgus (Anderson *et al.* 2004).

It is important not to overemphasise conformation as a cause of unsoundness when examining horses. Common conformational faults are presented in Table 6.1.

Rule of thumb: Hoof conformation is more important than limb conformation. The conformation of the hoof, particularly the hoof pastern axis, is reflected in the conformation of the limb.

6.1.3 Approach to the lame horse

Lameness is by far the most common reason for performance and pleasure horses to be presented to a veterinarian. Remember, however, lameness may be the result of dysfunction in one or more structures and systems (Speirs 1994). While there are several definitions for the term lameness, it is important to realise that lameness is a *clinical sign*. Lameness is a manifestation of the signs of inflammation, degeneration, a mechanical defect, deformity or malformation, resulting in varying degrees of pain in one or more limbs, back or pelvis and subsequently in a gait abnormality that is evident while the horse is standing or in movement.

Poor performance will also be a common presenting complaint for many equine orthopaedic problems. However, when presented with a horse with poor performance, one of the difficult problems is to decide whether the problem results from a true lameness; is a training or riding problem, a shoeing problem, or a tack (saddle, biting, hobbles, boots) problem; results from the horse's character and physiology; is an inherent biomechanical limitation for this individual; or even exists at all (Marks 2003).

Veterinarians focus on making a pathoanatomical diagnosis of the cause of lameness using a database of historical, clinical and diagnostic test information. In doing so, there are a number of first principles that should be followed:

1. Define and verify the problem. Is the problem primarily a gait abnormality or a swelling, or a discharge? Determine exactly what the horse demonstrates and the duration and character of the problem, e.g. if it is transient or intermittent.
2. Localise the problem. An attempt is made to localise the problem to a particular part of the body (ruling out neurological or other body system problems) by categorising the lameness, using manipulative tests and diagnostic analgesic techniques (see below).
3. Consider pathophysiological and pathological causes.
4. Establish an order of priority of the potential cause(s). Have a list of diagnostic possibilities *and* a plan of attack to rule in or out these options using the most informative tests (see section 6.5 Diagnostic imaging).

Components of the lameness examination

A lameness examination should be performed in an orderly, logical and step-by-step manner. In some situations (e.g. financial and time constraints) the examination

can change or be shortened. However, except for circumstances such as suspected stress fracture or unsuitable environment or temperament of the horse, it is always recommended to perform a full lameness examination and appropriate diagnostic tests before advising on a lame horse.

Components of the lameness examination include:

1. A detailed clinical history and note the signalment of the horse.
2. A general physical examination (e.g. measure temperature, auscultate the thorax and abdomen).
3. Examination from a distance observing conformation, symmetry and posture.
4. Confirmation of lameness and that the leg indicated by the owner is truly the lame leg. The degree of lameness is graded.
5. General and brief palpation of each limb and use hoof testers on all hooves comparing degree of any pain between normal and affected feet.
6. Localisation of the lameness to a particular limb or limbs: On a flat dry surface (and sand arena if available), observe movement at straight walk and trot and lunge in a circle on a lead and/or in saddle.
7. Localisation of the source of the pain/lameness:
 - (a) Manipulate affected limb(s) using flexion tests.
 - (b) Detailed pressure/palpation of suspect limb.
 - (c) Confirm localisation using diagnostic local analgesia: does the horse become sound once pain is removed?
8. Development of a differential diagnostic list and consideration of pathogenesis.
9. Selection of the most appropriate imaging technique(s) to attempt to confirm the diagnosis and identify pathology.
10. Advising the client, making appropriate recommendations and beginning surgical and/or medical therapy.

A final component is taking detailed records of the examination, ideally performed using a standardised examination form. Such forms ensure that steps are not missed and the records can be reviewed later for follow-up or repeat visits, especially in horses with more subtle lameness. For further reading on lameness, there are excellent veterinary texts available (e.g. Ross & Dyson 2003; Stashak, T.S. (ed.) *Adam's Lameness in Horses*, 5th edn 2002, see Further reading).

Signalment and history

1. What are the age, sex, breed and use (signalment) of the horse?
 - (a) Many problems tend to develop at specific ages, e.g.
 - Neonates and foals: developmental orthopaedic disease (DOD), rupture of the common digital flexor tendon and physal fractures
 - Two-year olds (skeletal immature): bucked shins, bowed tendons and carpal bone fractures

Table 6.1 Common faults in conformation

Common term	Synonym	Definition and details	Predisposes to:
Forelimbs			
<i>Front perspective</i>			
Base-narrow		Standing with forelimbs inside 'plumb line'. Can be toe-in or toe-out. Found in large chest horses	Increased weight bearing and stress on outside of carpus, fetlock, phalangeal joints and hoof; articular windpuffs, lateral sidebone and lateral heel bruises
Base-wide		Standing with forelimbs outside 'plumb line'. Can be toe-in (rare) or toe-out. Found in narrow chest horses	Increased weight bearing and stress on inside of lower limb and hoof; misshapen feet; interference and medial splint bone problems
Toe-in	Pigeon-toed	Toes point toward one another; congenital and may involve limb from shoulder or fetlock down; common	Outward deviation of foot during flight (padding or winging-out) resulting in interference with hind limb
Toe-out	Splay-footed	Toes point away from each other; congenital, usually from shoulder down; uncommon	Inward arc when advancing; results in interference with opposite forelimb especially if combined with base-narrow stance
Knock knees	Carpus valgus, knee narrowed (angular limb deformity)	Medial angular deviation of carpus with lateral deviation below carpus; common in foals; usually corrects itself with maturity	Increased stress on medial carpal collateral ligaments, outward rotation of cannon bone, fetlock and foot May be protective for fetlock fracture or effusions in racing Thoroughbreds (McIlwraith <i>et al.</i> 2003)
Bowlegs	Carpus varus, bandy-legged (angular limb deformity)	Lateral or outward deviation of carpus with medial deviation below carpus	Increased tension on outside of carpus (lateral collateral ligament) and medial surface of carpal bones
Bench knees	Offset knees	Cannon bones are offset (or deviate) laterally and don't follow a straight line from radius; congenital; often combined with carpus valgus	Increased stress on medial splint bone (splints) and suspensory ligament Associated with increased risk of fetlock problems in racing Thoroughbreds (Anderson <i>et al.</i> 2004)
<i>Side perspective</i>			
Calf-knees	Hyperextended knees, sheep knees, back at the knee	Backward deviation of carpus; increased weight/stress on carpal ligaments and front aspect of carpal bones	Carpal chip fractures, injuries to check ligaments
Bucked knees	Knee sprung, goat knees, over in the knees	Forward deviation of carpus; knees knuckle forward so dangerous for rider; congenital form bilateral	Strain on sesamoid bones, suspensory ligament, and SDFT and extensor carpi radialis muscle
Open knees		Irregular carpal profile (side view) giving impression joint not apposed; 1–3 year old often with physisitis improves with age	Physisitis, carpal problems
Standing under front		Entire limb below elbow placed to far under body; can occur with disease as well as be a conformational fault	Overloading of forelimbs, shortened cranial phase of stride and low arc of foot flight = stumbling
Camped in front		Entire forelimb from body to ground is too far forward	Especially seen in navicular syndrome and laminitis
Short upright pastern		Often associated with base-narrow, toe-in conformation; in horses with short limbs and heavily muscled	Increased concussion on fetlock and phalangeal joints and navicular bone resulting in traumatic arthritis, ringbone and navicular syndrome
<i>Side perspective</i>			
Long sloping pastern		Pastern bone too long with pastern angle normal or subnormal ($\leq 45^\circ$)	Injury of flexor tendons, sesamoiditis, sesamoid fractures, suspensory desmitis
Long upright pastern		Pastern bone too long and angle steep	Same as short upright pastern but not as severe

Table 6.1 (Continued)

Common term	Synonym	Definition and details	Predisposes to:
Hindlimbs			
<i>Rear perspective</i>			
Base wide		Distance between hooves greater than distance between centre of thighs; commonly associated with cow-hocks; not as common as in forelimbs	Interference; strains to inner structures of limb
Base narrow		Distance between hooves less than centre of the thighs; heavily muscled horses; often accompanied by 'bowlegs' with hocks too far apart	Interference, strains to lateral structures of limb
Cow hocks	Tarsus valgus (medial deviation of hocks)	Hocks point toward each other (too close) and base-wide from hocks to feet; may be accompanied by 'sickle-hock' esp. Western performance horses; worst hindlimb fault	Excessive stress on hock leads to bone spavin
<i>Side perspective</i>			
Sickle hocks	Curby conformation, small hock angles	Excessive angulation of hock joints ($\leq 53^\circ$)	High stress on back of hock joint and the soft tissue support structures; curb
Straight behind		Excessively straight limbs (little angle between tibia, femur and hock)	Bog spavin and upward fixation of the patella
Camped behind		Entire limb too far back; often associated with upright pasterns	
Hooves			
Broken hoof/pastern axis	Broken back posture, run under heel	Low heel, long toe; hoof and pastern axis not in alignment; very common	Heel bruising, navicular syndrome, hoof cracks, interference Increased risk of carpal problems in racing Thoroughbreds (Anderson <i>et al.</i> 2004)
Coon footed	Broken forward posture	Too steep hoof angle, too low pastern angle; foot axis > pastern axis (short toe, high heel)	Extensor process of pedal bone injury, coffin joint degenerative joint disease, pedal osteitis
Hoof imbalance		One heel longer than other due to improper trimming; one heel lands before other	Fetlock osselets, pastern ringbone, navicular syndrome, hoof cracks, sheared heal
Flat feet		Lacks natural concavity of sole; more common in fore hooves; heritable; normal in some Draft breeds	Increased pressure on heels to avoid sole; sole bruising and lameness
Contracted heels/foot		Hoof narrower than normal especially back half; more common in front hooves; uni- or bi-lateral	Overly concave sole, recessed frog, chronic lameness; thrush
Bull-nosed foot		Hoof with a dubbed or curved toe wall	
Buttress foot	Pyramidal disease	Swelling on the front of the hoof wall at the coronary band due to new bone growth from low ringbone, fracture of extensor process of pedal bone	Degenerative joint disease of coffin joint, low grade lameness
Thin walls and sole		Hoof wall wears away too rapidly or doesn't grow fast enough to avoid effects of sole pressure; heritable	Low heel, which wears down; sole bruising and lameness after trimming
Club foot	Flexural deformity of coffin joint	Hoof axis $\geq 60^\circ$ secondary to injury, preventing proper use of hoof or to flexural deformity involving deep digital flexor tendon; usually 6 weeks–6 months	Boxy appearance to hoof, wear at toe growth at heels; lameness

Plumb line = line drawn from point of shoulder or hip bisecting the forelimb or hindlimb respectively to the foot, when viewed from the front or back of the horse.

- Adults: osteoarthritis, navicular syndrome and unexplained tendinitis
- (b) Breed usually determines the sporting activity or use, which has the greatest impact on lameness distribution.
2. How long has the horse been lame?
 - (a) Acute: may indicate fracture or infection.
 - (b) Chronic: one month or more: permanent structural changes may have taken place that usually prevent full recovery and prognosis is guarded.
 3. Was the onset gradual or sudden?
 - (a) Sudden, severe onset indicates trauma.
 - (b) Mild or insidious onset suggests infection or degeneration (osteoarthritis) respectively.
 4. Has the lameness worsened, stayed the same or improved?
 - (a) Rapidly worsening lameness suggests infection.
 - (b) No improvement even with rest and slowly worsening suggests degeneration (osteoarthritis).
 - (c) Slow improvement with rest suggests fracture or mild to moderate soft tissue injuries.
 - (d) Lamé – quickly sound with rest – work – lame cycle suggests stress-related bone injury.
 - (e) Marked improvement in lameness generally indicates a better prognosis than horses that remain static or have worsened.
 5. Does the horse 'warm into' (worsens with exercise) or 'out of' (improves with exercise) the lameness?
 - (a) Lameness associated with stress fractures, tendon or ligament injuries, splints, curb, and foot soreness worsens with exercise.
 - (b) In racehorses, a worsening lameness may appear as a progressive inability to maintain position on the track on corners, whereas riding horses may increasingly stumble, have problems taking leads or refuse to jump fences.
 - (c) Lameness associated with muscular or arthritic joint involvement (e.g. lower hock pain in racehorses) often improves with exercise. Some horses can race or perform successfully despite the pain of joint degeneration and lameness can be difficult to detect.
 6. Does the horse stumble?
 - (a) May be the result of heel pain (e.g. from navicular syndrome), causing a horse to land on its toes and stumble, or from interference with the synergistic action of flexion and extension (e.g. from carpal pain).
 - (b) Neurological disease, e.g. ataxia, proprioception deficits or weakness may also be considered.
 7. Is there a known cause such as trauma or foreign body removal?
 8. Was any treatment given and was it helpful?

No response to a recent and appropriate treatment regimen may indicate a poor prognosis; alternatively, recent

- administration of non-steroidal anti-inflammatory drugs (NSAIDs) might mask symptoms of lameness and give a false impression of severity (or lack thereof).
9. Have there been any management changes recently?
 - (a) *Shoeing*: hooves trimmed too short or trimmed aggressively, altered hoof balance, improper and irregular shoeing, or a nail driven into or near sensitive tissue may all predispose to lameness.
 - (b) *Training and performance intensity*: a recent increase in training intensity or return to the same level after a rest may be result in lameness related to stress-induced subchondral or cortical bone injury (e.g. bucked shins, stress fractures).
 - (c) *Surface*: harder surfaces are associated with hoof lameness and a sudden change in racing surface may lead to episodic lameness in racehorses; soft or uneven surfaces may exacerbate ligament or tendon injuries.
 - (d) *Diet and housing*: sudden changes in diet may result in excesses (e.g. grain) leading to laminitis or deficiencies (e.g. in calcium) leading to nutritional secondary hyperparathyroidism; shipping to and from sales; foaling indoors; being turned out onto pasture with other horses; and exposure to weather all may be associated with soft tissue injuries, puncture or kick wounds and other trauma.
 10. What health or lameness problems has the horse had in the past?

Not always possible to obtain an entire history but recurrence of lameness, results of previous diagnostic tests and response to previous medication should be noted.

Examination at rest

Symmetry, posture and conformation

Careful visual examination with the horse standing squarely in a flat surface at rest is performed from all angles, first at a distance and then up close. Always compare with the opposite side (assuming that it is normal) during every stage of examination.

From a distance, the following should be observed:

- General body condition and symmetry of skeletal and soft tissues
- Conformation of body, limbs and feet
- Alterations in posture such as weight shifting (normally horses will not shift weight in forelimbs but will in hindlimbs), foot pointing or refusing to bear weight (resting a forelimb usually suggests a problem in that limb)
- Presence of any overt tissue injury

At close observation, each limb and muscle group, particularly of the back and rump is scrutinised and compared with its opposing member for symmetry. Hooves are checked for abnormal wear, cracks, imbalance, size and heel bulb contraction, whereas all joints and tendons and their sheaths are inspected for swelling. For example, the

limb with the smaller hoof and higher heel is usually the (chronically) lame limb (see also Table 6.1: Common faults in conformation). Gluteal muscle wastage usually indicates the lame hind limb, while asymmetry of the position of the tubae coxae accompanies many types of pelvic fractures.

Basic abnormalities (in any segment of a limb or hoof) to observe include:

- Change in size, shape, height and width of any structure (especially hooves)
- Deformity (especially joints and hooves), skin wounds and muscle wasting
- Swelling (tissue oedema or joint effusions) and thickening, indicative of inflammation
- Draining (usually infected) sinus tracts from joints, bone or soft tissue
- Old lesions or scars

Palpation and hoof tester examination

Palpation and inspection of the hooves, limbs, back and neck should be performed methodically starting at the hoof, moving up the limb. Ideally, palpation should be performed briefly before watching the horse in motion and subsequently in greater detail after movement and the lame limb(s) has been identified. Comparison with the opposite 'normal' forelimb or hindlimb in the same horse, or in another sound horse if the problem is bilateral in the affected horse, is essential.

The basic palpable abnormalities (in any segment of a limb or hoof) are:

- Swelling (tissue oedema or joint effusions)
- Heat (often accompanied by increased blood flow or 'pulse' – especially of the hoof or 'digit')
- Pain (superficial or deep but usually results in adverse reaction to palpation)

All are indicative of *inflammation* as a result of some form of trauma and/or infection.

Examination of the hoof (Table 6.2)

- Coronary band: heat suggests laminitis; swelling \pm discharging sinus suggests subsolar abscess (gravel), or necrosis/infection of lateral cartilages (quittor); swelling with pain over extensor process of pedal bone suggests fracture of extensor process.
- Bulbs of heels and above quarters: swelling and pain \pm discharge suggests subsolar abscess.

Examination of the pastern (proximal interphalangeal joint)

- Structures that should be palpated for signs of inflammation (i.e. swelling, heat, pain) include the proximal and middle phalanges (fractures, ringbone); distal sesamoidian and collateral ligaments and superficial digital flexor (SDF) and deep digital flexor (DDF) tendons (sprains and tenosynovitis common).

Table 6.2 Examination of the hoof sole (with the hoof lifted)

1. Assess shoeing (common problem is excessive contact with the sole inside the white line) then remove shoe.
2. Clean and trim sole with hoof knife and take note of:
 - (a) Discoloration – from puncture wounds, nail pricks, bruise, or draining abscess
 - (b) Separation of white line (seedy toe) owing to laminitis
 - (c) Overgrown frog or frog atrophy (caused by not bearing normal weight on frog due to painful heels)
 - (d) Hoof imbalance (different heights to heels or sheared heels) as a result of improper and uneven trimming
 - (e) Abnormal wear of toe suggesting heel pain, or excessive lateral or medial wear suggesting compensation for a problem
 - (f) Contracted heels (abnormal narrowness to heels) caused by any painful condition resulting in the horse not putting weight on frog or hoof
 - (g) Flat hooves due to little or no sole concavity (sole should not be in contact with the ground) resulting in bruising and excessive heel contact to avoid sole
3. Use hoof testers (with the hoof lifted and on the ground). Use enough pressure to find pain but not enough to elicit pain where there is none; recheck a positive response repeatedly and always compare with the opposite hoof. The testers may also be used as a hammer to percuss the outer hoof wall.
 - (a) Diffuse sensitivity: laminitis, distal phalanx fracture, pedal osteitis
 - (b) Localised sensitivity: bruise, corns, abscess, nail prick
 - (c) Central frog sensitivity: caudal heel pain (navicular syndrome), sheared heels



Figure 6.1 Palpation for rotation and shear of distal interphalangeal (coffin, P2 P3) joint. It is important to stabilise the fetlock and pastern to isolate the movement.

- Rotating and flexing pastern (fetlock, pastern and coffin joints) may elicit crepitus or pain indicative of joint inflammation or osteoarthritis (Figure 6.1).

Examination of the fetlock (metacarpal–phalangeal joint)

- Structures that should be palpated for swelling, effusion or pain include:
 - (a) The dorsal pouch (suggestive of chip and articular fractures)

- (b) The palmar pouch (between the suspensory ligament and cannon bone) of the fetlock looking for bilateral synovitis or unilateral joint disorders
- (c) SDF tendon, DDF tendon and sheath suggestive of tendinitis or tenosynovitis
- (d) Suspensory ligament branches and sesamoid bones (desmitis, sesamoiditis, sesamoid fractures)
- Rotating and flexing fetlock to detect decreased range of motion (age or fetlock problem such as osteoarthritis, sprain or synovitis).

Examination of the metacarpus/metatarsus

- Major structures to palpate for swelling, effusion or pain are: the SDF tendon, DDF tendon, the suspensory ligament and its origin and the DDF (inferior) check ligament (high in the metacarpus behind suspensory), ensuring normal movement and separation.
- Pain, heat and swelling over the splint bones indicate 'splints' or splint fractures, whereas these signs when palpating the dorsal middle third of the cannon indicates 'bucked shins'.

Examination of the carpus

- Swelling or effusion on front and back of joint suggests carpalitis, chip/slab fractures and osteoarthritis; palpate individual carpal bones and flex and rotate carpus, noting reduced motion \pm pain (chips, synovitis, extensor tendinitis and collateral ligament strains).
- Examination for range of motion. Note the carpus has lateral and medial planes of motion as well as flexion-extension (Figure 6.2).

Examination of the forearm, elbow and arm

Major structures to palpate for swelling, crepitus or pain are the distal radius (fractures), elbow joint (*capped elbow*, olecranon fracture) and humerus (fracture).

Examination of the shoulder

- Palpate and visualise swelling or wastage of shoulder muscles (supraspinatus, infraspinatus muscles suggestive of nerve injury), and pain over greater trochanter, and bicipital tendon and bursa (bicipital bursitis, tendosynovitis).
- Flex, adduct and abduct shoulder to detect pain indicative of scapula fractures and joint osteoarthritis.

Examination of the hock (tarsus)

Major structures to palpate for swelling, effusion or pain include:

- Tarsocrural joint: 'moveable' effusion (*bog spavin*) between pouches of joint suggests idiopathic synovitis, intra-articular chip fractures or osteochondritis desiccans (OCD) of tibia or talus; 'non-moveable' distension suggests chronic inflammation of joint capsule associated with chronic fractures, capsule sprains and osteoarthritis.
- Distal intertarsal and tarsometatarsal joints: palpated on medial (inside) side, effusion suggests osteoarthritis (*bone spavin*).
- DDF tendon and tarsal sheath on inside back (medioplantar) of hock: non-moveable distension and swelling indicates synovitis or tendosynovitis (*thoroughpin*).
- Back of calcaneus bone: swelling indicates inflammation of long plantar ligament or *curb*.
- Point of hock: soft swelling/distension of subcutaneous bursa indicates acute bursitis (*capped hock*); swelling becomes firm and fibrous with time.

Examination of the stifle

Relevant structures to palpate and test include:

- Wastage and swelling of surrounding muscles.
- Patella ligaments: medial, middle and lateral ligaments (desmitis); patella (displacement, fracture).



(a)



(b)

Figure 6.2 Examination of the carpus. Note the carpus has lateral and medial planes of motion as well as flexion.

Table 6.3 Classification of lameness

Classification	Definition	Structures that may be affected
1. Support phase lameness	Characterised by abnormal body or head movement when limb is bearing weight	Usually bones, joints, ligaments and flexor tendons of lower limb, especially hoof
2. Swing phase lameness	Characterised by circumduction or reduced cranial or caudal swing phase in the affected leg	Usually joints, muscles, extensor tendons, tendon sheaths or bursae of the upper limb
3. Mixed lameness	Combination of supporting and swinging limb lameness	Any of above
4. Compensatory lameness	Occurs when pain in a limb results in uneven distribution of weight on another limb or limbs	Any of above

- Femoropatella joint pouch: distension on either side of middle patella ligament suggests a problem in femoropatella or femorotibial pouch as they communicate (capsulitis, osteoarthritis, OCD, intra-articular fracture).
- Positive patella displacement test (upward fixation of patella), positive cruciate rupture (cruciate ligament rupture), painful medial collateral ligament test (medial ligament rupture or sprain).

Examination of the upper limb and pelvis

- Abnormalities to inspect and palpate are asymmetry of the pelvis and thigh muscles (fractures), signs of inflammation in muscles of thigh (particularly semimembranosus and semitendinosus muscles indicating myopathy), and crepitus of hip and pelvis suggestive of pelvic and femoral neck fracture.
- Vaginal and rectal examination by a veterinarian is also useful to detect crepitus of a pelvic symphyseal fracture below vagina (while manipulating the limb at the same time), and ileum and acetabula fractures of the pelvis.

Examination of the back and neck

Muscles of the back, thorax and neck should be palpated for tension, sensitivity and flinching (see also Chapter 8) as well as muscle wastage, asymmetry, swelling and heat.

Important palpation tests (looking for above abnormalities) include:

- Light and firm palpation (generally using a blunt instrument) along back from withers to tail. Soft tissue or bone problems will cause horses to resist ventroflexion (extension of the thoracolumbar spine), resist dorsiflexion (flexion of the lumbosacral region) and resist lateral flexion (when blunt instrument is run over the lateral sides of the back on both sides).
- Palpate tips of spinous processes looking for malalignment, depressions or protrusions indicating fracture.
- Rectal examination by a veterinarian to confirm fractures of pelvis, sacrum and lumbar vertebrae as well as thrombosis of iliac artery.

Table 6.4 Lameness grade

Grades	Lameness is:
Grade 1	Difficult to observe, not consistently apparent when weight bearing or circling on inclines or hard surfaces
Grade 2	Difficult to observe at walk or trotting in straight line; consistently apparent when weight bearing or circling on inclines or hard surfaces
Grade 3	Consistently seen at trot under all circumstances
Grade 4	Obvious with marked head nodding or shortened stride
Grade 5	Obvious with minimal weight bearing in motion and/or at rest; inability to move

Adapted from: Definition and classification of lameness. In: *Guide for Veterinary Service and Judging of Equestrian Events*. American Association of Equine Practitioners Lexington, KY, 1991:19

Examination during movement

Confirmation and localisation of lameness

The primary purpose of examining a horse while in motion is to determine which limb(s) is (are) lame and whether there is any compensatory lameness as a result of the primary lameness. Classification of the lameness as support phase, swing phase or mixed and grading according to a standardised system (Tables 6.3 and 6.4) is also important.

Requirements for a thorough gait evaluation

- A hard, level, non-slip surface and freedom from distractions and dangers. Lameness in the distal limb is often exacerbated on hard ground whereas proximal limb lameness may be worsened on soft ground.
- A competent handler and consideration of safety, particularly with difficult horses.
- The horse must be led with a loose lead rope so it can move its head and neck freely and the horse must be moved at a consistent speed.
- The horse should be walked and slow trotted in a straight line, in a circle, and in both directions, and should be observed from the front, the side and behind.



(a)



(b)



(c)

Figure 6.3 Lunging on a straight level surface as well as a hard and soft circle.

- Space for lunging in circles (in both directions) to help to demonstrate more clearly subtle lameness (Figure 6.3).

Indicators of lameness (fore or hind)

- **Sound:** Listen for regularity of rhythm and sound of footfall. A lame horse usually lands harder on the sound limb, resulting in a louder hoof contact with the ground. Horses with disparity of hoof size may confound interpretation. In addition, horses that drag a hoof make a distinctive scraping noise.
- **Extension of fetlock:** Generally, the fetlock joint of the sound limb drops down further when this limb is weight bearing *at the walk* than does the fetlock joint of the lame limb (which is being spared). The opposite is true in horses with suspensory desmitis or superficial digital flexor (SDF) tendinitis when walked but not when trotted.
- **Drifting:** Horses with hindlimb lameness generally drift or list away from the lame limb, thus reducing weight bearing on that limb.
- **Abnormal limb movement** (best viewed from the side):
 - (a) **Reduced length of stride phase:** Decreased cranial phase of stride in one limb may indicate lameness in opposite limb. In normal horses, the length of stride of the paired forelimbs and hindlimb, measured from hoof print to hoof print, is nearly identical from side to side. Also, the degree of extension and flexion of joints are similar.
 - (b) **Altered limb and hoof flight:** For example, hoof travelling inwards (winging in) or outwards (winging out) or front hoof hitting the opposite forelimb or same side hindlimb; usually associated with conformational faults and may result in interference, self-trauma, pain and lameness.
 - (c) **Altered arc and path of hoof:** Lowering of the arc of the hoof during forward movement can occur to reduce impact when the hoof lands or to reduce limb flexion during protraction; if lameness is severe, dragging of the toes can result.
 - (d) **Reduced joint flexion:** Particularly in hindlimbs and most observable in hocks; may be associated with alterations in hoof flight and reduced stride phase. This may be more apparent when the horse is lunged or ridden.
 - (e) **Abnormal hoof placement:** If there is toe pain, weight will be placed on the heel and will land heel first. If pain is on the outside of the hoof sole then weight will be placed on the inside of the sole and the horse can be seen to land on the inside of the hoof first.

Lameness higher up the limb (above carpus or tarsus) may be exacerbated with the affected limb on the outside of a circle whereas lower limb lameness is usually worse with the affected limb on the inside of a circle. Lameness in horses with disorders located anywhere on the inside of the limb (e.g. stress fracture of the pedal bone, proximal suspensory desmitis, or fracture of third carpal bone, medial femoropatella joint disorder) is worse when the affected limb is on the outside of the circle and the lesion is being compressed.

Forelimb lameness

- The forelegs support 60% of a horse's weight and the weight of a rider increases the value to 70%.
- As a result of the increase in concussive forces ~75% of lameness is found in a forelimb, especially in Thoroughbred horses (TBs). However, compared with TBs, the prevalence of forelimb lameness is lower (~60%) in Standardbred (SB) racehorses pulling the added load of a cart, and hindlimb lameness is more common.
- The majority (~95%) of lameness problems in the forelimb occur at the level of or distal to the knee (carpus). The foot should always be suspected first.
- *Rules of thumb* for recognition of forelimb lameness:
 - (a) Best observed while the horse is trotted towards the examiner and when viewed from the side.
 - (b) Head nods down when bearing weight on the normal limb ('down on sound') and up when weight is borne on the lame limb (to shift weight onto back limbs). This is the most consistent indicator of forelimb lameness!
 - (c) Stilted action suggests both fore hooves involved.
 - (d) Stride length characteristics, height of hoof flight, sound and fetlock drop (described above) also are helpful.

Hindlimb lameness

- The hind legs serve as the horse's prime propelling force.
- Most lameness in the hindlimb is due to problems of the tarsus and below and the hock is by far the most common site.
- Hindlimb lameness is best observed while the horse is trotted away from the examiner and if the examiner observes from the side of the lame limb.
- Observation of asymmetrical movement of the pelvis on the side of the lame hindlimb is the most important and consistent abnormality.
- The tuber coxae (as well as the point of hip and the gluteal musculature) on the side of the lame limb rise higher (during weight bearing) and lower (when the sound limb bears weight – '*pelvic hike*') than the sound limb when straight trotting on a flat surface. Tape markers placed on the tuber coxae may help gauge this asymmetrical and excessive vertical movement.

- The horse's head is lowered when the lame leg contacts the ground. (Only in higher grade (>3) hindlimb lameness.) The horse's tail head rises when the lame hindlimb bears weight (not always visible).
- Choppy, short gaits lacking impulsion and no asymmetric pelvic movement may suggest bilateral hindlimb lameness.
- Stride length characteristics ('carrying' lame hindlimb when viewed from side), height of hoof flight, sound and fetlock drop (described above) are also helpful.

Multi-limb lameness

A prominent head nod can be seen in horses with simultaneous left fore and left hindlimb lameness (head moves upwards on lame forelimb, and is lowered on lame hindlimb), making diagnosis of multi-limb lameness difficult. However, the presence of a pelvic hike may increase suspicion of both limbs being affected. Diagnostic analgesia (see below) starting with the hindlimb is essential to investigate both limbs.

Simultaneous left forelimb and right hindlimb lameness (especially in SB trotters) manifests as a head nod that reflects the left forelimb lameness and drifting away from the right hindlimb.

Manipulative tests

After observing the horse at exercise and determining which limb (or limbs) is lame, the next step is to focus on the lame limb and perform detailed inspection and palpation. Manipulative or provocative tests should then be performed to exacerbate mild, subtle or 'hidden' (inapparent) lameness and to aid in localisation of the abnormality causing the lameness to a particular segment of the limb.

Joints should be examined for:

- Decreased range of movement
- Increased instability
- Crepitus
- Pain (superficial or deep but usually results in adverse reaction to manipulation)
- Willingness or resistance of horse to undergo procedure and lack of willingness to bear weight on lame limb when the opposite, sound limb is manipulated
- Exacerbation of lameness thus localising pain to the 'stressed' part of the limb

Provocative tests are not sensitive or specific and result in false-positive and false-negative results. Excessive force may 'create' lameness that may not have any clinical relevance to the lameness observed during movement examination. The results of provocative tests in the lame limb should be compared with the opposite, sound limb and interpreted in conjunction with previously collected information and with the more specific results of diagnostic analgesia (Busschers & Van Weeren 2001).



Figure 6.4 Flexion of the fetlock joint. Note this flexes all the phalangeal joints. It is important to ensure the carpus is not flexed at the same time.

Flexion tests

Flexion tests are always begun with the hoof, working up the leg. The joint under investigation is held in a firmly flexed position for 45–60 seconds after which the horse is immediately trotted off for at least 12–15 metres and any worsening of gait noted. Because some joints and associated structures are inherently linked together in flexion or extension (e.g. hock and stifle, phalangeal joints and fetlock), the exact differentiation of pain responses between these joints and structures is not possible (Figure 6.4).

It is important to use a consistent technique (i.e. force applied, duration of test) and a 'positive' result is defined as obvious lameness or a 1 to 2 grade increase in lameness observed persistently for more than 5–8 strides while the horse trots in a straight line after flexion. Sound horses warm out of the normally mild response seen in the first few strides.

Flexion results in compressive or tension forces being applied to both articular structures within a joint as well as surrounding soft tissue. Therefore a positive response to flexion of the lower limb (fetlock flexion test) can be observed with any disorder of the coffin, pastern and fetlock joints; navicular bone or bursa; other palmar heel structures; digital flexor tendon sheath; palmar pastern soft tissue; suspensory ligament branches or sesamoid bones.

Direct pressure tests

Response to localised pressure over any painful areas (limb or back), tendon swellings (suspensory branches or digital flexor tendons), splints, the front of the proximal phalanx and metacarpal 3, and specific areas of the hoof (using hoof testers) can be as useful as flexion tests for localising the source of pain. Compression is usually maintained for approximately 30 seconds and then the horse is trotted off and observed for exacerbation of lameness.

Wedge test

The wedge test is used specifically to stretch or compress the joints, subchondral bone, articular surfaces and associated soft tissues of the hoof, including the DDF tendon, SDF tendon, the suspensory ligament and collateral ligaments. A wedge with a 20° inclination is placed under the weight-bearing hoof to raise the toe (increased stress on DDF tendon, navicular bone and associated ligaments and bursa) or the heel (increased stress on suspensory ligament). The horse is made to stand for 30–60 seconds with the opposite limb elevated, after which the horse is trotted off in a straight line, observing for exacerbation of lameness. This test has relatively poor specificity and poor predictive value.

6.1.4 Diagnostic analgesia: nerves and joints

Although time consuming, invasive and sometimes hazardous to both horse and examiner, diagnostic analgesia ('nerve blocking') arguably remains the most valuable tool to localise lameness to a specific structure of a limb (Pasquini *et al.* 1995, Whitton *et al.* 2000, Bassage & Ross 2003). Common local anaesthetic drugs used in horses – 2% solutions of lidocaine, mepivacaine and bupivacaine – block or inhibit nociceptive nerve conduction by preventing the increase in membrane permeability to sodium ions. Mepivacaine has become the agent of choice because it causes minimal tissue reaction and has a slightly longer duration of action (2–3 hours).

Local analgesia may be used for:

- Perineural infiltration around specific nerves to desensitise the limb regions/structures supplied by that nerve distal to the site of the injection. (Therefore, if a horse becomes lame-free following the injection, one or more of these structures are the source of the pain and lameness.)
- Intrasynovial analgesia of joints, tendon sheaths or bursae.
- Direct local infiltration over suspect superficial lesions.
- Field analgesia, performed by circular injection around the suspected site of pathology, thereby blocking all nerve fibres entering the area.

Unless simply confirming a suspected lesion, use of local analgesia should start with the hoof and be continued sequentially up the limb anaesthetising specific limb segments. After waiting an adequate time (at least 5–10 minutes for lower limbs and up to an hour for upper limb blocks) the perineural block should be tested for its effect on removing superficial *and deep* pain using a blunt instrument or firm digital pressure. Improvement in degree of lameness >70% to 80% after perineural or intra-articular analgesia may be considered to be a positive response.

While local analgesia is considered an objective test, there can be problems with interpretation owing to the effects of diffusion of local anaesthetic solution up the nerve, into communicating joints, or from joint pouches into

surrounding tissues and nerves, which may result in unintended desensitisation of structures, so in general, horses should be re-evaluated no more than 5–10 minutes after administration. Examples of diffusion and structural communications resulting in desensitisation of the structures after local analgesia include diffusion between coffin joint, navicular bursa, navicular and pedal bones.

Response to local analgesia can be complete where the lameness (mostly) resolves and the examination can be stopped or the lameness switches to the opposite limb. Alternatively, the response can be incomplete owing to chronic or deep bone pain, which may remain resistant to analgesia. Individual variation in neuroanatomy and response to analgesia may also result in incomplete responses. Additionally, as a result of complex sensory innervation of joints, intra-articular analgesia inconsistently abolishes pain from many of the common articular problems, particularly subchondral bone pain (due to remodelling, cystic or erosive disorders, incomplete fractures and osteoarthritis). Because joint pain often arises from articular and periarticular tissues, perineural analgesia more consistently abolishes pain from all aspects of the joint and surrounding soft tissue structures.

6.1.5 Diagnostic imaging

The number of imaging modalities has increased over recent years and, in addition to radiography, ultrasound and nuclear scintigraphy, the newer modalities of thermography, computed tomography and magnetic resonance imaging provide important means for more accurate and detailed orthopaedic diagnoses (McIlwraith 2003). Although the latter three modalities tend to be restricted to referral or university establishments, equine veterinary practitioners commonly use the other three. Imaging should ideally be used in conjunction with findings from the history, physical and lameness examinations.

Radiology/radiography

Following identification of the site of pain during the lameness examination, the logical next step is to image the area beginning with radiography, the mainstay of diagnostic imaging in horses. Radiography gives information about bones and joints as well as soft tissues such as tendons, ligaments and joint capsule insertions. Most radiography of the lower limbs can be performed using a portable X-ray machine. However, larger radiographic machines found mostly at referral or university veterinary institutions allow better images of the proximal limbs and pelvis. A range of views has been developed for each individual segment of the forelimb and hindlimb (Park 2002).

It has become popular in Australia and many countries to radiograph yearlings during the yearling sale process. However, the association between radiographic findings and pain and dysfunction is not always clear cut and some lameness disorders are not correlated with radiographs at all (Kane *et al.* 2003).

Advanced imaging techniques such as computed tomography (CT) and magnetic resonance imaging (MRI) provide detailed images in any plane and are becoming more commonly used. However, for both techniques the machine size limits scans to the limbs below the level of the mid-radius and tibia, and to the upper and mid cervical spine and head in anaesthetised horses.

Thermography

In thermography, a visual image is produced from infrared radiation emitted from the skin surface and detected using a photon detector connected to a computer. The image is displayed in colours (isotherms) which correspond to different skin temperatures, and which accurately reflect changes in circulation, pain and temperature in deeper tissues. Although soft tissue and bony inflammation can be detected before clinical and radiographic changes by as much as 2 weeks, thermographic changes are non-specific and this technique should be complementary to other diagnostic modalities.

Ultrasonography

Ultrasonography has become an affordable and non-invasive diagnostic technique that enables repeated assessment of tissue lesion over time. Ultrasonography involves the use of high frequency sound waves (>1 MHz) emitted from a transducer to image tissues of the body. Sound is reflected (or echoed back to the transducer) from interfaces between tissue of different physical characteristics or density (called acoustic impedance). Large differences in acoustic impedance between two tissues (e.g. bone versus fluid) result in greater amplitude of the reflected echo. Most sound is reflected at the interface of soft tissue with bone or air, leaving insufficient sound to penetrate deeper structures. Sound is additionally reflected or scattered when it meets small reflectors, representing internal architecture, within tissues.

Reflected sound is received by the transducer and converted back into electrical impulses to produce a grey-scale image. A loud or highly reflected echo is seen as a white image (hyperechoic; e.g. bone), weaker echoes as varying levels of grey (hypoechoic; e.g. soft tissue, tendons), and no echo (anechoic; e.g. fluid, blood) as a black region.

Ultrasonography is indicated for:

- Diagnosis of soft tissue injuries especially tendons, tendon sheath and ligaments but also muscular, vascular, joint capsule and bursal defects
- Monitoring the effect of training on soft tissue structures, especially tendons and ligaments and prevention of potential injury in horses with subtle signs of tendon or ligament disease
- Comparison with the opposite limb
- Monitoring of healing of soft tissue injuries (correlates closely with histological changes)

- Assessment of fluid accumulation (in joints, bursae, other soft tissues and masses)
- Evaluation of bony surfaces (e.g. tuber coxae, metacarpus/metatarsus)
- Investigation of wounds

Nuclear medicine/scintigraphy

Nuclear scintigraphy involves the intravenous administration of technetium-99m, a radioactive isotope, which emits gamma-radiation and, conjugated with methylene diphosphate or hydroxymethane, is preferentially taken up into the mineral lattice of bone at a level dependent on the rate of bone remodelling. Sites with a high rate of turnover, as a result of physiological or pathological factors (i.e. disease), therefore accumulate more of the technetium than normal and this is reflected in increased gamma-radiation being emitted from a particular site ('hot spot'). The level of gamma emission is detected using an imaging system at a particular time after technetium administration.

The advent of scintigraphy over recent years, particularly in referral practice, has allowed *early* recognition of a range of subtle vascular (blood flow), soft tissue and especially bone lesions, such as inflammation (e.g. osteoarthritis, osteitis) and stress fractures, which in the past may have remained undiagnosed (Steyn 2002).

6.1.6 Selected orthopaedic diseases

Bone fracture

Complete fractures can occur in any bone depending on where an excessive load or traumatic event has been applied. In contrast, incomplete or stress fractures occur most commonly in the metacarpus/metatarsus, tibia, humerus and pelvis and primarily in racehorses. These latter fractures are the result of chronic microtrauma that weakens bone. While complete fractures usually are obvious, stress fractures frequently are difficult to diagnose and require specialised equipment such as scintigraphy.

Most acute, complete fractures cause significant lameness regardless of their size and location. In every horse presented for acute non-weight-bearing lameness, a fracture should be high on the differential diagnosis list (which should also include bone, joint or tendon sheath infection or foot abscess). However, some fractures such as an osteochondral chip fracture of a carpal bone may only produce low-grade lameness.

Classification and prognosis

Fractures are broadly classified as complete or incomplete (stress), stable or unstable (non-displaced or displaced), and open or closed. Fractures also are categorised according to their configuration (oblique, transverse, spiral, multiple and comminuted), and their location and character (articular (through a joint), non-articular, diaphyseal, epiphyseal, Salter–Harris physeal, chip and slab) (Nunamaker 2002).

Fractures that carry a poor to grave prognosis (and in which case euthanasia would be considered by the veterinarian) are:

- Unstable (no cortical continuity to prevent motion)
- Displaced (no alignment of bone fragments)
- Above the carpus (knee) or tarsus (hock)
- Open (with minimal to extensive skin laceration, contamination and vascular injury)
- Comminuted (multiple breaks that often communicate)
- Those in which much time has passed since injury and first aid measures were ineffective
- Those that occur in uncooperative patients and in horses >450 kg

Fracture healing

During healing, bone reunites either by:

- Primary remodelling without callus formation – this requires rigid fracture stabilisation and correct anatomical reduction of bone
- Secondary fibrocartilage formation between fragments, which is later replaced by periosteal and endosteal new bone (callus)

Key point

Depending on the above prognostic factors, it may take 12 months or more for complete fracture repair.

Fractures heal via a series of sequential but overlapping processes:

- *Inflammatory phase*: crucial for vascular and bone repair and protection from infection
- *Reparative phase*: depends on method of fracture fixation, stability and degree of displacement of fracture
- *Remodelling phase*: to replace avascular and necrotic regions and realign bone

It is important to note some of the undesirable consequences of fracture healing, which may include chronic lameness, athletic disability, and, rarely, laminitis (of the contralateral, weight-bearing limb). 'Fracture disease' (pain and lameness) may occur following bone repair after internal fixation with screws and plates or after a cast is removed.

- Unlike the situation in humans, joints become stiff from degenerative ankylosis rather than disuse, or develop increased laxity (especially young animals treated with casts).
- External immobilisation such as casts also results in tendon and muscle flaccidity and atrophy of surrounding muscles.
- Scar tissue formation involving tendons, ligaments and muscle may impair tendon function, stiffen adjacent joints and cause flexural deformity in growing horses.

In addition, fibrous tissue is far more pronounced if infection had previously complicated the fracture repair and the limb may become permanently thickened with severe loss of function.

- The non-fractured limb is at risk of developing angular limb deformities or laminitis, as a result of excessive weight bearing.

Key point

It is in these situations that physiotherapy would be of most use, particularly in the early stages after cast removal or several weeks after internal fixation.

Bone infection

It is worth summarising some important features of bone infection, which is commonly encountered in equine orthopaedics.

Osteitis and osteomyelitis describe inflammation of bone although the former term is applied to *inflammation* that begins in or involves the periosteum and outer bone cortex, whereas the latter term is used when *inflammation and infection* begins in or extends into the medullary cavity.

Osteitis

- Common in extremities (mostly cannon bones) owing to lack of soft tissue protection.
- Caused by trauma (kick with intact skin), break in skin or nearby infectious process.
- Damaged periosteum and outer cortex usually dies from lack of blood and results in:
 - (a) osteitis and bone sequestration if unexposed (i.e. skin still intact); or
 - (b) osteitis, sequestrum formation plus contamination and infection if bone exposed – the development of a draining sinus tract and non-healing wound subsequently occurs.
- Pathogenic organisms (commonly *Staphylococcus*) reside within avascular necrotic bone and avoid immune defences. (*Note: For this reason also, systemic antibiotics are of limited value unless there is associated cellulitis*).
- Lameness, swelling and a non-healing draining purulent wound are key signs (remains until sequestrum surgically removed).
- Radiographic evidence of a sequestrum is usually not visible until 2–3 weeks after the injury. Also, culture and sensitivity of the sequestrum is always warranted for accurate diagnosis of the offending organism.

Osteomyelitis

- More extensive bone inflammation than osteitis and begins within or extends into the medullary cavity.
- Key signs are severe lameness, swelling (cellulitis) and a draining wound if trauma involved.

- Culture and sensitivity of infected bone or joint is always warranted for accurate diagnosis of the offending organism(s).

Three types of osteomyelitis are: haematogenous, traumatic and iatrogenic.

Haematogenous

- Primarily in neonates (often with immune system suppression due to failure of passive transfer of maternal immunoglobulins).
- Sluggish metaphyseal blood flow allows bacteria, spread from a primary site (e.g. umbilicus, gastrointestinal tract (GIT) or lung), to localise in the synovial membrane of joints (S-type), and/or the physis (P-type), and/or the epiphysis (E-type).
- Most common organisms isolated include: *Escherichia coli*, *Salmonella* spp., and *Streptococcus* spp.
- Results in inflammation, blood vessel thrombosis and prostaglandin-induced bone necrosis and destruction.
- Frequently, foals that recover from an initial infection (umbilical or systemic) develop bone or joint infection several days later.

Traumatic

- As a result of a penetrating wound or open fracture, bacteria enter the medullary cavity through the open wound. Rarely, the skin is not broken but blood supply is compromised and necrotic tissue provides an ideal medium for bacteria that arrive from a haematogenous route.
- Many types of Gram-negative and Gram-positive organisms may be isolated.
- Open fractures in which bone fragments lose their blood supply and unstable, open fractures are particularly prone to osteomyelitis.

Iatrogenic

- Develops as a result of contamination during internal fixation (using metal plates) of open or closed fractures.
- Fracture haematoma, lack of blood supply at fracture site and the implantation of foreign pins, screws and plates provide favourable conditions for bacterial colonisation and growth.
- Highly resistant bacteria such as methicillin-resistant *Staphylococcus aureus* and Gram-negative bacteria usually are involved.

Injury and repair of tendons and ligaments

In a galloping Thoroughbred at maximal speed, the SDF tendon operates close to its physiological limits with a relatively narrow safety margin. Consequently, minor disruption of the tendon matrix composition and arrangement may increase the risk of excessive tendon strain resulting in tendinitis. Conversely, stronger and stiffer tendons are likely to be less prone to tendon injury.

Tendons suffer from either extrinsic (percutaneous) traumatic injury or, more commonly, intrinsic strain (leading to tendinitis). Strain may occur as a sudden event or be a cumulative subclinical process, with damage varying from minor disruption/rupture of individual fibrils to progressive involvement of groups of fibrils to complete tendon rupture.

Remember: The major *acute* pathological endpoints of strain are degeneration (initially subclinical) and inflammation of tendon components resulting in pain, oedema, heat, swelling and consequently lameness.

Broadly, injury to tendons may occur as a result of the following mechanisms:

1. Sudden excessive over-extension (e.g. of the fetlock joint) that mechanically disrupts tendon fibrils.
2. Direct low-grade mechanical forces experienced during strenuous exercise (with maximal loading). This results in cumulative fatigue micro-damage of the tendon matrix. The damage may be exacerbated by poor foot and limb conformation, landing from a jump, lack of fitness, hard dry ground surfaces, fatigue at the end of a performance event and uncoordinated muscle activity.
3. Age-related degenerative changes. In contrast to muscle and bone (in which increasing mechanical demand with age and exercise results in both tissues undergoing an increase in mass and architectural change), tendons appear to have little ability to adapt after skeletal maturity (≥ 2 years) and cumulative micro-damage weakens the tendon matrix.

Key point

These latter two points may explain the presence of pathological tendon damage in older horses without clinical signs of tendinitis and that tendinitis frequently is a bilateral condition with one limb more severely affected than the other.

4. Exercise-induced hyperthermia of the tendon. Galloping horses have hot tendons (up to 45°C), which damages matrix proteins.
5. Ischaemia and reperfusion injury due to variable blood flow during and after sub-maximal and maximal exercise.

The healing of tendon and ligament follows a sequence of haemorrhage, oedema, acute inflammation, fibroblastic proliferation, collagen production and chronic remodelling.

1. Unchecked inflammation in the early stages of tendinitis may result in release of proteolytic enzymes, which, although directed at removing necrotic collagen, also digest relatively intact tendon collagen causing progression of the lesion.
2. Tendon tissue is not regenerated. Rather, scar tissue (produced by paratendon and endotendon cells),

characterised by haphazardly arranged collagen (predominantly type III), is laid down. This scar tissue:

- Is *weaker* than normal tendon tissue.
- Is predisposed to *re-injury* at the injured site (especially if the horse is prematurely exercised in an uncontrolled manner).
- Results in *adhesions* reducing normal gliding function.
- Is slowly remodelled over many *months* (usually >6) into type I collagen, although the tissue never becomes normal tendon. Controlled exercise during this phase may encourage this remodelling, with improved alignment of collagen fibrils and mechanical properties of the scar tissue.
- Takes *15–18 months* to heal completely, although the tendon has poor elasticity, resulting in increased strain in adjacent regions of the same tendon, opposite tendon or other supporting ligamentous structure (e.g. suspensory ligament).

Strain of tendons and ligaments

Tendinitis is inflammation of tendon and tendon muscle attachments caused by excessive strain and commonly affects flexor tendons of the lower limbs (particularly the SDF tendon). In contrast, *desmitis* is inflammation of a ligament. Disorders range from minor tearing to complete rupture or avulsion from bony attachments.

- Flexor tendons of the forelimbs are more commonly affected than those in the hindlimbs, whereas the SDF tendon is more often affected than the DDF tendon in forelimbs.
- Lesions are generally localised to the core of the mid-metacarpal region of the SDF tendon in the forelimb (where cross-sectional area is smallest and blood supply poorest).
- In contrast, suspensory ligament (SL) desmitis is more often found in Standardbreds, particularly in the hindlimbs as a result of different gait and locomotor demands.

Tendons and ligaments suffer a higher frequency of injury:

- In racehorses on flat track versus steeplechase
- When raced on a hard track versus a muddy track
- When training has been inadequate especially first 3–4 starts
- When muscles are fatigued
- In horses with poor conformation such as excessive pastern slope and long toes

Key clinical signs are acute pain, swelling around the affected tendon or ligament resulting in varying degrees of lameness, or firm, diffuse swelling in chronic cases. Ultrasound is used to definitively define the extent of the lesion (core lesion or diffuse) and for determining progress during treatment and the prognosis for future performance.

*Principles of management of tendon injury***Key point**

Currently, there is no universal treatment method for tendinitis and, in most instances, clinical experience influences recommendations. In general, therapy should be aggressive, should include anti-inflammatory treatment, may combine strategies such as tendon stabbing and controlled exercise, and should be regularly monitored using ultrasonography.

Medical therapy

The key things veterinarians do when presented with an acute severe tendon injury is to:

- Stop training and complete stall rest, initially for 4–6 weeks, followed by controlled exercise for a minimum of 6–10 months (see below).
- Control inflammation (decrease oedema, swelling and pain) and progression of damage.
- Minimise excessive scar tissue and encourage normal repair.
- Recommend controlled exercise and rehabilitation, which involves an initial programme of hand-walking/swimming for 3 months followed by increasing strength exercise for a further 3–4 months. Progress is monitored via ultrasound.

Note: Although clinical signs of lameness are usually resolved after a short period of rest, *much longer is needed before substantial healing* of the tendon has occurred.

Key point

Factors that influence recovery and return to racing are severity of lesion and type of rehabilitation, with those undergoing a controlled exercise regimen more likely to return to racing.

Surgical therapy

Surgical treatment usually is indicated in severe and bilateral tendinitis (not desmitis). Some commonly used procedures include:

- Accessory ligament desmotomy. Transection of the accessory ligament of the SDF tendon to increase involvement of the SDF muscle and reduce load on the tendon itself when the horse returns to work. The success of the operation and results of return to racing are varied and conflicting.

- Percutaneous tendon splitting. For acute tendinitis where the intra-tendinous haematoma and oedema is relieved from a core lesion using scalpel stabs or needle sticks. The quality of repair and return to race form is variable.
- Suturing severed flexor tendons.

Key point

Synthetic tendon implants and counter-irritation (via application of topical 'blister ointments' or 'pin-firing') are now considered to be inappropriate therapies for tendinitis (although there is still support for them in some countries). As with many treatments for tendinitis in the horse, conclusive proof of the effectiveness of these controversial therapies is lacking and in many cases can be counterproductive.

The tendon sheath

Synovial effusion of a tendon sheath (tenosynovitis) is common in all types of horses and may be: idiopathic, acute, chronic or septic.

Idiopathic

- Occurs when synovial effusion results in sheath distension but without inflammation, pain or lameness.
- Most commonly affects the extensor tendon sheaths over the carpus (especially in foals), the tarsal sheath which encloses the DDF tendon over the hock ('thoroughpin'), and the digital flexor sheath enclosing the SDF tendon and DDF tendon above and below the fetlock ('windpuffs' or 'wingalls'; especially hindlimbs).
- Chronic low-grade trauma and poor conformation may be risk factors.
- Diagnosis is made by recognising typical clinical signs, obtaining normal synovial fluid findings and differentiating from effusion of the fetlock joint.

Acute

- Manifested as a rapidly developing effusion accompanied by distension, heat, pain and possibly lameness.
- Digital tendon sheath over fetlock is the most common site but also seen in the sheath of the extensor tendon over the carpus.
- Direct trauma, and DDF or SDF tendinitis or suspensory ligament desmitis are potential causes, the latter conditions visualised using ultrasonography.

Chronic

- Manifested as a persistent synovial effusion with non-inflammatory swelling and fibrous thickening of tendon sheath.
- Develops as a result of repetitive minor trauma and commonly follows acute unresolved tenosynovitis.

- Is usually accompanied by adhesion formation, stenosis within the sheath, diffuse or nodular sheath thickening and sometimes tendon damage.
- Results in reduced function with inability to flex the carpus (if extensor tendon sheath affected) or fetlock (if digital tendon sheath affected).
- Ultrasonography is essential to evaluate sheath thickening, adhesions and tendon injury.

Septic

- Marked infected synovial effusion with swelling, heat, pain and severe lameness – an emergency! Infection most commonly is introduced via a penetrating wound, which frequently goes undetected, particularly in the pastern region. Infection may also occur after contaminated intrasheath injection or rarely via haematogenous route. The pathogenesis, signs, diagnosis and treatment are similar to septic arthritis.
- Septic tenosynovitis is a critical condition because of the:
 - (a) Severity of lameness;
 - (b) Difficulty in eliminating infection and propensity to become chronic;
 - (c) High risk of long-term complications resulting in chronic lameness, e.g. adhesion formation, rupture of tendon (digested by inflammatory cell enzymes if progressed enough), extension of infection and laminitis in opposite limb.
- Major sites affected are the digital flexor sheath over fetlock (88%), digital extensor tendon sheath and tarsal sheath.

Diagnosis:

- Marked lameness unless sheath open and draining from wound or tract.
- Synovial fluid analysis (Ross & Dyson 2003, p. 582).
- Confirmation of penetrating tract.
- Culture of offending organisms (usually mixed bacteria, *Streptococcus*, Enterobacteriaceae, *Staphylococcus aureus* and *Klebsiella* most common).
- Ultrasonography to identify adhesions, complicating injuries of tendons and involvement of the annular ligament (in digital flexor tenosynovitis).

Key point

Treatment for septic tenosynovitis (see below) must be early (if possible) and involve aggressive intrasynovial and systemic broad-spectrum antibiotics, with copious lavage of the sheath. Usually these cases should be referred and hospitalised. However, adhesion formation may result in failure of the horse to return to athletic activity (Figure 6.5).



Figure 6.5 Stallion – 5 months after a septic tenosynovitis, demonstrating restrictive adhesion formation, physically restricting the hoof contacting the ground. Note also the clubfoot (Table 6.1) conformation as a result of prolonged non-weight bearing.

Principles of management of tenosynovitis

Treatment varies depending on the cause and different clinical manifestations. Some key principles:

- Stall rest the horse (not really necessary in idiopathic cases)
- Control inflammation (although in idiopathic cases no treatment is necessary)
- Reduce effusion and adhesion formation and return previous function
- Control infection in septic tenosynovitis
- Surgery

In chronic or septic tenosynovitis arthroscopy may be used to:

- Assess sheath and associated flexor or extensor tendons
- Drain excess fluid
- Break down adhesions
- Perform meticulous debridement, cleaning and flushing with isotonic fluid including antibiotics and dimethyl sulfoxide (DMSO)
- Place in-dwelling drain in infected cases to allow further irrigation and local therapy. Regional IV perfusion of antibiotics, slow-release antibiotic depot systems and antibiotic infusion pumps may be placed at this time
- Resect constriction of annular ligament

Arthritis

Although arthritis literally means joint inflammation, the term is used in a broader sense to cover a number of well-defined pathological entities (below) involving the major structures of the joint. Particular features of all of these entities (except immune-mediated disease) is that single or repetitive episodes of trauma are nearly always involved in the pathogenesis and that there is considerable overlap between them, or one disorder may progress into another:

- Traumatic arthritis
 - (a) Idiopathic synovitis, synovitis and capsulitis
 - (b) Chronic proliferative synovitis (see reference list for details)
 - (c) Sprain, luxations, meniscal tears and intra-articular fractures
- Osteoarthritis
- Septic arthritis
- Immune-mediated and autoimmune arthritis

*Osteoarthritis and septic arthritis***Osteoarthritis (degenerative joint disease)**

Osteoarthritis is defined as an essentially non-inflammatory disorder of moveable joints, characterised by degeneration and loss of articular cartilage (splitting and fragmentation) and the development of new bone on joint surfaces and margins.

Osteoarthritis may develop in four distinct ways:

1. *Acute*: associated with synovitis and capsulitis involving high motion joints (carpus and fetlock); especially in racehorses
2. *Insidious*: associated with high-load, low-motion joints (interphalangeal (ringbone), intertarsal (bone spavin) and tarsometatarsal); repetitive trauma main aetiological factor; young, mature and aged active horses
3. *Incidental or 'non-progressive' articular cartilage erosion*: associated with age and continued low-level use: questionable clinical significance
4. *Secondary to other problems*: including intra-articular fractures, luxations, sprain, wounds, septic arthritis and osteochondrosis

The main pathogenic mechanisms hypothesised are:

- Inherently defective cartilage matrix, which fails under normal loading
- Microfractures of the subchondral and epiphyseal bone secondary to excessive mechanical stresses, which result in failure to absorb repetitive physiological loads and subsequently in cartilage damage
- Repetitive impact trauma ('use trauma') resulting in cartilage microfracture and metabolic alterations in chondrocytes
- Damaged chondrocytes and synoviocytes results in:
 - (a) An imbalance towards matrix depletion over repair

- (b) Release of matrix-degrading enzymes (matrix metalloproteinases), cytokines (tumour necrosis factor-alpha, interleukin-1), nitric oxide and prostaglandins (e.g. PGE2)
- (c) Cartilage degeneration/loss, fissuring and separation, decreased viscoelasticity of tissues and secondary remodelling of bone

Septic (infective) arthritis

Septic arthritis, or bacterial infection of a joint, is the most severe joint problem in horses and is the most common cause of death in foals.

Bacterial colonisation of the synovial membrane results in:

- Mild to severe inflammation with necrosis of the synovial membrane and formation of fibrinopurulent exudation
- Release of a diverse range of inflammatory mediators, which potentially cause rapid loss of glycosaminoglycans, proteoglycans and collagen and eventually cartilage degradation

Bacteria enter joints via:

- Haematogenous spread from umbilical, respiratory or gastrointestinal infections in foals (especially *Actinobacillus* spp., *Escherichia coli*, *Streptococcus* spp., and *Salmonella* spp.)
- Local penetration or direct trauma in adults (especially *Streptococcus* spp., *E. coli* and anaerobes)
- Iatrogenic associated with intra-articular injection of steroids or joint surgery (especially *Staphylococcus* spp.)

Risk factors for development of septic arthritis include foal factors, such as failure of passive transfer of maternal immunoglobulins and septicemia (high sepsis score), and trauma in poorly managed environments. The tarsocrural (tibiotarsal) joint of the hock is the most commonly affected, followed by the fetlock, carpus and stifle.

*Principles of management of joint disease***Medical therapy**

- Prevent further physical damage to joint: principally by rest and immobilisation
- Control pain, inflammation and eliminate production of inflammatory mediators in osteoarthritis and maybe septic arthritis once infection resolved
- Remove preformed mediators from joint using joint lavage
- Antibiotic therapy for septic arthritis
- Allow sufficient time for cartilage healing and institute controlled exercise

Surgical therapy

- To treat primary causes of osteoarthritis such as osteochondrosis, intra-articular fracture and chips and meniscal tears. Arthroscopic keyhole surgery is the most common technique

- Arthrotomy/arthroscopy for open drainage in septic arthritis if fibrin accumulation and synovectomy needed, or foreign body suspected
- Surgical arthrodesis for joint luxation and end-stage osteoarthritis, particularly for low-motion joints (e.g. pastern and distal tarsal joints)
- Synovectomy to remove fibrotic non-productive synovial membrane and to lower concentrations of deleterious enzymes
- Surgical curettage of full- and partial-thickness defects in cartilage and bone

The vertebral column/back

The back of the horse is most commonly defined as the thoracolumbar spine and sacrococcygeal spine supported and maintained under tension by a complex arrangement of soft tissue structures (i.e. muscles and ligaments). The soft tissue structures of the back most commonly associated with injury, strain or inflammation, include the supraspinous ligament, the epaxial muscles (longissimus dorsi, multifidus muscles the middle gluteal muscle which extends cranially as far as L1), and the sublumbar or hypaxial muscles (e.g. iliopsoas and psoas minor muscles). The most common type of back injury is muscular or ligament damage or strain (33%), followed by over-riding spinous processes ('kissing spines'; 29%), sacroiliac strain (subluxation of the sacroiliac joint; 'jumper's bump'; 14%), 'undefined back problem' (12%) and temperament (8%). Fractures of the dorsal spinous processes, of the vertebral body and of the pelvis are rare.

The most common presenting sign of back pain is loss of athletic performance rather than 'back pain'. Definitive diagnosis is very difficult.

A wide range of conditions can be mistaken for back pain by owners including:

- Hind limb lameness (e.g. hock degenerative joint disease (DJD), upward fixation of the patella)
- Hypersensitivity of the back ('thin skinned')
- Initial stiffness and hypersensitivity to saddling and mounting ('cold backed')
- Ill-fitting saddle
- Poor schooling or riding
- Temperament problems
- Lack of ability of horse to perform to owner's expectations
- Cervical or thoracolumbar spinal cord compression presenting with weakness or stiffness behind

Predispositions to back injury include:

- Use (hunters or jumpers, dressage and event horses)
- Breed (Standardbred), conformation (inflexible short-backed horses more prone to vertebral injury, whereas flexible long-backed horses are often prone to muscle or ligament strains, especially behind withers and over loin)

Assessment of the equine back is covered in detail in Chapter 8.

6.2 Canine lameness

Thomas McGowan and Matthew Pead

6.2.1 Introduction

The aim of this section is to outline the canine veterinary orthopaedic examination, focusing on aspects pertinent to the animal physiotherapist.

A thorough examination will include:

- Evaluation of conformation
- Gait assessment
- Palpation and manipulation

The orthopaedic examination relies heavily on a thorough and accurate knowledge of the anatomy of the area of interest, including the underlying anatomy and topographical landmarks (see Further reading). This will allow the examiner to navigate their way around the animal and examine areas of concern.

As an animal physiotherapist, it is not your responsibility to make a pathoanatomical diagnosis and it will be illegal in some states and countries. Your role in accepting an orthopaedic case involves understanding the diagnosis, its implications and consequences. You should maintain communication with the referring veterinary surgeon. In assessing each case and developing a course of action for treatment and rehabilitation you should use your clinical reasoning process. The examiner should question the findings and relate them to the patient, and current presenting complaint, as well as questioning additional findings and inconsistencies.

6.2.2 Examination

Conformation

The dog should be checked for angular deformities (valgus and varus), internal and external rotations, and growth discrepancies between bones (e.g. radius and ulna). Every breed has a specific conformation requirement for that breed. Yet, desirable conformation in some dog breeds may predispose to orthopaedic conditions. Some types of conformation may be speculated to predispose to orthopaedic disease and the variation between breeds needs to be considered. For example the very upright stifles in a Staffordshire terrier cross (Figure 6.6.) may predispose to patellar or cruciate ligament problems, compared with the very low flexed stifle of a German shepherd dog that may predispose to lumbosacral disease. However, there is limited evidence to support such relationships.

Gait assessment

Providing the patient can move without excessive distress, the lame animal should always be examined in motion. Initially, this will be for diagnostic purposes, but the continual re-evaluation of the patient during the period of therapy is essential and therefore the physiotherapist must become comfortable with gait assessment. The basic assessment should take place on a solid, flat, non-slippery surface. The dog should be observed at a walk and a trot going away



Figure 6.6 A 2-year-old Staffordshire Terrier cross-breed dog with conformation change, but no lameness.

from and towards the examiner. It may also be wise to examine the gait as the dog passes the examiner so the stride length can be assessed. The examiner should initially concentrate on identifying the lame leg, before moving on to describing the gait abnormality in systematic, standard terms. If an animal that is reported being lame does not show the problem, circling and going up and down slopes will sometimes exacerbate a subtle lameness. A patient may also be examined before and after a heavier burst of exercise such as running or ball retrieval or after a period of enforced rest such as kennelling.

Palpation and manipulation

Everyone who examines an animal must have an understanding of animal behaviour and recognise the hazards of working with animals (Chapter 2). In the course of this section we will be referring to dogs but this will apply to most animals. Some exceptions may be noted but clearly this would be a monumental task and it is not a goal of this section to give in detail every scenario that could play out with numerous species. This section assumes that the examiner has been trained in canine handling and restraint techniques, such as placing muzzles, and how to restrain a dog so that the dog, handler and the examiner are safe from injury. When working with animals, you must remember that the animal can be fearful and at the same time protective of their owners (Chapter 2).

The examiner should develop a routine and should follow the same routine for all dogs. Palpation is the technique of using touch to identify anatomical features such as a tendon, joint or bony prominence and gross pathological features such as a joint effusion. The lightest pressure possible should be used to define the anatomy. Manipulation involves moving the joints and testing range, laxity, restriction and normality of that movement. Clearly, some elements of this examination such as manipulation to the end

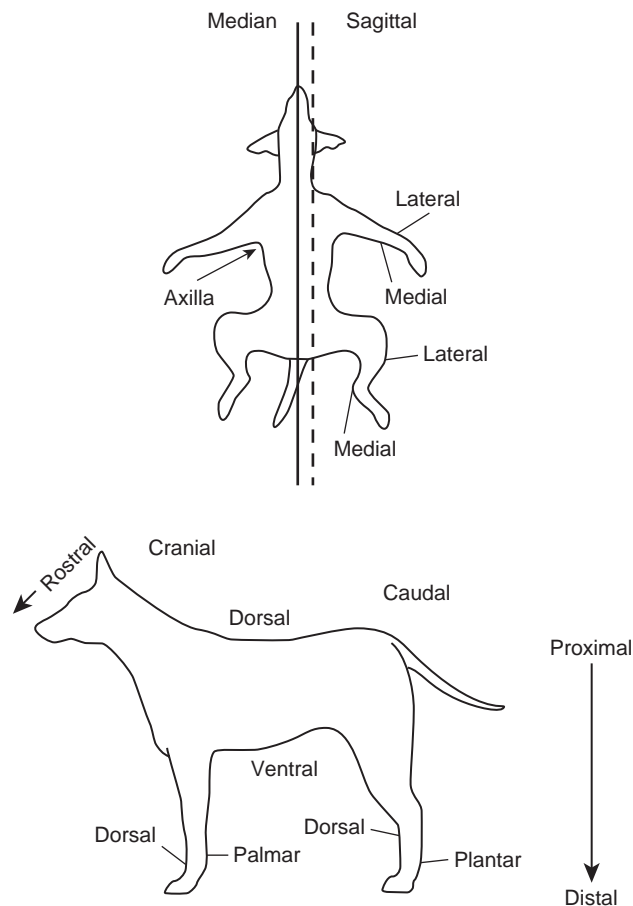


Figure 6.7 Anatomical nomenclature.

of the range of motion and deep palpation may elicit a pain response and should be left to the end of the examination in order to keep the cooperation of the patient for as long as possible. There will be times when certain examination techniques are not appropriate or are even contraindicated. These will be discussed throughout this section. The approach to examination given in this section is the '*distal to proximal*' system used by the authors, but there are numerous satisfactory systems and it is in no way intended to imply that this is the 'only' method. However the examination is undertaken, the examiner must try to catalogue any pain, heat, swelling (both acute fluid and chronic fibrous), crepitus, restriction of movement, increased range of movement and muscle wastage involving any part of the limb.

One thing to be aware of in quadrupeds is that the anatomical nomenclature for directions and locations in the dog are different from those in man (Figure 6.7).

Forelimb

Phalanges (paw)

The examination starts by examining the paw. There are numerous injuries and problems that dogs can have to this region including: nail disease or injury (broken nails,

infected nails, infected nail bed (paronychia, onychomycoses), skin disease or injury (cut pads, inter-digital dermatitis, foreign body penetration, neoplastic conditions, autoimmune dermatitis), as well damage to the underlying bone, tendon and ligamentous structures. Phalangeal fractures are relatively common.

Deviations of the nail should alert the examiner to pathology. Rupture of the SDF tendon may result in a slight elevation of the nail and a flaccid extension of the phalanges, while rupture of the deep digital flexor tendon should result in a much more dramatic elevation of the nail. A medial or lateral deviation of the nail could indicate rupture of the collateral ligaments between the second (P2) and third (P3) phalange.

Abnormalities to look for are pain on palpation (be careful of the dog's response to pain! See Chapter 2), swelling at the nail beds, deviated nails, swollen pads, heat, blood (wet or dried), purulent discharges (wet or dried), peeling of pads, redness, or discoloration of the hair (especially white dogs). Discoloured hair on the feet of light-coloured dogs can sometimes mean that the dog has been licking the area excessively. Saliva can turn the hair orange/brown.

When examining deeper structures it is important to visualise the anatomy of the region. During the examination you should observe for pain response, stability, normal range of movement, anatomical alignment and acute or chronic inflammation. An effort should be made to palpate for joint margins and stress each joint by end range of flexion, extension, rotation, and medial and lateral opening of the joint (for example collateral ligament rupture between the phalanges is common in racing Greyhounds). The dorsal and the palmar surface should be examined thoroughly making sure that the spaces between the pads are visualised. The pads should be felt to see if they are excessively hard (for example melanoma can occur in this site) or soft (for example abscesses within the soft tissue of the pad secondary to foreign body penetration).

It should be considered that the first digit (most medial) is the dewclaw, which may have been removed. The first nail that is on the ground medially is the second digit, with the fifth being most lateral. Some dogs may still have their dewclaws in the front and they can have extra dewclaws (polydactyly). Dogs with long hair on their feet can be difficult to examine and, to see adequately, the hair may need to be trimmed.

Metacarpus

The metacarpus is the next area of interest and the metacarpal bones should be examined individually and as a group. Each metacarpal bone has a pair of sesamoid bones on the palmar surface of metacarpophalangeal joints and a single dorsal sesamoid, which articulates with the dorsal head. The joint capsule envelops the metacarpophalangeal joint and two sesamoid bones. The sesamoid bones are labelled 1–8, medial to lateral from the second metacarpal bone.



Figure 6.8 A 4-year-old neutered male domestic shorthaired cat, fractured proximal metatarsals II–IV.

Fractures and subluxations represent a large proportion of the problems found in this region (Figure 6.8). The metacarpal bones should be examined for pain on response to palpation, fractures, subluxation from the carpus or non-specific swelling (acute or chronic inflammation). You should remember that there are only four metacarpal bones. The metacarpal–phalangeal joints should be mobilised individually as well as in a group. The sesamoid bones are incriminated for lameness and are over represented in Rottweilers and in dogs about 2 years of age. There is a predisposition for sesamoid 2 and 7. One study showed 22 out of 50 Rottweilers examined had radiographic lesions but only five dogs had clinical signs.

Carpus

The following bony landmarks should be identified and palpated:

- Radial styloid process
- Ulnar styloid process
- Each of the seven carpal bones, i.e. the accessory, radial and ulnar in the proximal and the 1st, 2nd, 3rd and 4th in the distal row.

It is important to recognise the complexity and interrelationships of the carpal bones, carpal ligaments and tendons of the foreleg (see Further reading for detailed anatomical

descriptions). It is ideal to examine this area in a weight-bearing position to assess for hyperextension. It can be difficult to assess mild joint effusions in the carpus. The joint should be assessed at the level of the carpal–metacarpal joint, the intercarpal joint and the radial–carpal joint. Most dogs will have a range of flexion that will bring the pads in very close proximity to the radius. There are some breed differences and it is important to recognise the normal range of motion. The joint should be checked for medial and lateral stability. The medial collateral has a long and a short portion. The long portion is under tension on extension and the short is under tension under flexion. The medial collateral ligament is a more common site of injury or pathology than the lateral collateral and may be associated with carpal valgus (Figures 6.9a and 6.9b). Attention should be paid to the accessory carpal bone on the palmar aspect of the joint.

Problems that occur in the carpus include fractures, particularly of the radial and accessory carpal bones. Hyperextension injuries usually occur as a result of a fall from height and present in a palmar grade stance. Collie breeds are prone to palmar ligament degeneration resulting in chronic hyperextension of the carpus with continued worsening. Eventually, the accessory carpal bone will make contact with the ground. Developmental carpal hyperextension presents itself at about 2–3 months of age and is common in German Shepherds (Figure 6.10).

Time should be taken to assess conformation defects that might affect the carpus, looking for angular deviations (carpal varus and carpal valgus). These are usually seen in the small chondrodysplastic breeds (i.e. Corgis, Dachshunds), but are also seen in giant breeds (Figure 6.11).

Tip

The accessory carpal bone is a good landmark to use to navigate around the carpus. It is important to be able to identify the bones of the carpus by palpation.

Radius and ulna

The long bones of the foreleg (radius and ulna) should be palpated along their entire length to assess for swelling and pain. (These bones can be the site of primary bone tumours that can cause a forelimb lameness.) You should recognise that dogs do not have much ability to pronate and supinate and that there is an interosseus ligament between the radius and ulna. The ulna is the long bone affected the most by panosteitis.

Young, fast-growing dogs are subject to hypertrophic osteodystrophy. This is a disease of destruction and regeneration of bone at the distal radius and ulna (distal tibia and fibula). The periosteum separates and there is severe inflammation and necrosis with microfractures. It is



(a)



(b)

Figure 6.9 (a) A 1-year-old female neutered Boxer, swollen medial aspect of the right carpus. (b) Radiographic image of the dog in Figure 6.9 (a).

accompanied by pyrexia, depression, weight loss and lameness. Retained cartilaginous cores in the ulna decrease ulnar growth and are implicated in cranial bowing of the radius, valgus deformities in the carpus and elbow subluxations.



Figure 6.10 Carpal hyperextension in a German Shepherd Dog.



Figure 6.11 A 2-year-old male neutered Dachshund cross.

Elbow

The following bony landmarks should be identified and palpated:

- Lateral epicondyle
- Medial epicondyle
- Olecranon

The elbow is a site of concern owing to the multifaceted problems that may affect the elbow. The elbow joint consists of three bones in the articular surface, the medial and lateral condyles of the humerus, the trochlear notch of the ulna and the radial head (Figure 6.12). Elbow dysplasia can be a combination of several defects at once, but the result is osteoarthritis at a very young age. Osteochondrosis dissecans (OCD), medial fragmented coronoid process (FCP), and ununited anconeal processes (UAP) are all implicated as a cause of elbow dysplasia. It has been suggested that a short radius can be the cause for FCP. Ununited anconeal process is a failure of fusion of two centres of ossification. This leaves the anconeal process as a separate portion and



Figure 6.12 Orientation of humerus, radius, and ulna.



Figure 6.13 Demonstration of elbow extension.

creating a fault in the joint surface. The elbow should be put through a full range of motion to assess pain on flexion, extension, internal and external rotation (Figure 6.13). The joint should be assessed for swelling, joint effusion, pain, and stability. It is important for elbow dysplasia to be diagnosed at as early an age as possible. This can make surgery a more rewarding procedure.

The extensor and flexor muscles of the antebrachium insert on the lateral and medial epicondyles, respectively.

Condylar fractures in mature animals are often a result of major trauma. Atraumatic condylar fractures are not uncommon and are a result of failure of the ossification centres in the medial and lateral condyles to fuse. One report showed Spaniel types to be over-represented at 78%. For skeletally immature dogs, more than 50% occurred in dogs less than 7 months old. The average age for skeletally mature dogs was 6 years. A history of no trauma and a fracture of this type should alert the examiner to a possible failure of ossification between the condyles and that the contralateral elbow should be radiographed to look for a failure of ossification in that elbow as well.

Tip

You should be able to work your way around the elbow joint by palpation of the major landmarks. Using the olecranon, medial and lateral epicondyles you should be able to identify most major structures.

Humerus

It is important to palpate the long bones in old dogs, as these are a major site for neoplastic disease. Osteosarcomas make up 75% of bone cancers. The median age is 7 years and it is most common in large breeds. There is a slight rise in incidence in the 2-year-old age group as well. The most common sites are distal femur and proximal humerus, but radius, ulna and tibia are not uncommon either. The mean survival time is 12 months post diagnosis. Amputation is a common surgical procedure for these dogs. They can present like the 'old dog with arthritis'. But at some point the dog will become non-weight-bearing and will probably have a pathological fracture through the tumour. Palpation of the long bones with a pain response should alert the examiner to be suspicious. The humerus (distal half) is also a site for panosteitis. Panosteitis is most commonly seen in large and giant breeds from 6 months to 18 months of age. This is inflammation of the long bones and is usually self-limiting and resolves with time. It often presents as 'shifting leg' lameness and often occurs bilaterally. Anti-inflammatories are used to make the dog more comfortable. In the 2-year-old large or giant breed dog it may be difficult to determine the cause of long-bone pain and it would be prudent to have a series of radiographs to determine the cause.

Dogs with long-bone pain can give a very aggressive pain response to palpation and care should be taken when palpating the humerus if it is suspected of being the source of pain.

Shoulder

The following bony landmarks should be identified and palpated:

- Spine of the scapula
- Scapula border
- Acromium process
- Greater tubercle

The shoulder has a large range of motion. It is important to be able to identify the muscles and bones in this area (Figure 6.14). The shoulder is an area of concern in racing greyhounds. The tendon of the biceps brachii muscle passes distally through the intertubercular groove, covered by the transverse intertubercular ligament. The groove is bordered by the greater and lesser tubercles. The greater tubercle is the point of insertion for the supraspinatus tendon, infraspinatus tendon and a portion of the deep pectoral muscle inserts on the greater tubercle and the subscapularis muscle inserts on the lesser tubercle. The shoulder should be placed through a full range of motion with extension, flexion, rotation, abduction and adduction. The biceps should be palpated via direct palpation over the intertubercular groove and to its insertion on the proximal radius and ulna. The whole biceps mechanism can be stressed by performing the biceps stretch test. This is accomplished by flexing the shoulder with the elbow at approximately 90° and then extending the elbow (Figure 6.15). The insertion should be palpated by using the hand opposite the leg to be examined, crossing underneath the dog's thorax and direct palpation from lateral to medial. Using the right



Figure 6.14 Orientation of the scapula and humerus.



Figure 6.15 Demonstration of the biceps stretch test.



Figure 6.16 Demonstration of palpation of the biceps tendon of insertion.

hand, facing the same direction as the dog, take your right hand and pass it under the dog's right side just behind the dog's right elbow, cross under to the dog's left elbow and use the index finger to find the tendon of insertion (Figure 6.16). The most common diseases in the shoulder joint are OCD and osteoarthritis. Though the disease is commonly bilateral, it usually presents unilaterally. Crepitus, periarticular fibrosis, effusion and decreased range of motion are not common clinical findings. The examiner

should be looking for mild atrophy of the scapular muscles. This is usually found in dogs aged 5–10 months. Bicipital tenosynovitis and bicipital tendon injury occur in middle-aged dogs. Shoulder luxations can be traumatic or congenital in nature. Another uncommon condition of the shoulder is infraspinatus contracture and results in a leg that is abducted and externally rotated.

Scapula

The scapula should be palpated thoroughly. The muscles should be identified with their corresponding tendons. The examiner should bear in mind the major difference from man in that the shoulder is a weight-bearing joint that is lateral on the thorax and not dorsal/posterior. Do not forget the ventral pectoral muscles. Fracture of the acromium process allows the deltoid muscles to displace the process, thus requiring surgical repair. In affected dogs, palpation directly over the process is very painful. The scapular cartilage can also be a site for neoplasia (osteosarcoma).

Tip

The glenoid process and the acromium process can be used in combination with the spine to locate and orientate the examiner to the insertions of the associated muscles of the shoulder and scapula.

Hindlimb

Examination of the hindlimb should start distally. The principles of examination of the phalanges and the metatarsals will be the same as for the forelimb.

Tarsus

The following bony landmarks should be identified and palpated:

- The medial and lateral malleolus
- Calcaneal process of the fibular tarsal bone
- Tibial and central tarsal bone
- Tarsal bones I, II, III, IV
- Metatarsal bones II, III, IV, V

The tarsus is a complex joint with numerous joints. It consists of the tarsometatarsal joint, the distal intertarsal joint, the proximal intertarsal joint and the tibiotarsal joint. The main joint of movement in the hock joint is the tibiotarsal joint. The calcaneus is a large tarsal bone on the caudal/palmar aspect of the tarsus, which has the insertion of the Achilles tendon and the superficial digital flexor tendon insertions.

It is important to check for instability of all the mentioned joints of the tarsus. It is not uncommon for dogs and cats to dislocate the tarsometatarsal joint. The joint should be put through a full range of motion with flexion,

extension, rotation and medial and lateral stress. Again, it is important to remember that when you are palpating you should be able to visualise and identify what you are palpating. Key landmarks around the joint are the calcaneus, lateral (distal end of fibula) and medial (distal end of the tibia) malleolus. The long digital extensor courses over the cranial/dorsal aspect of the hock joint. You can isolate this tendon and palpate the joint margins on the 'craniomedial' and the 'craniolateral' aspect of the hock. Joint effusions can be palpated here and on the 'caudomedial' and 'caudolateral' aspect. The superficial digital flexor and the gastrocnemius tendons insert on the calcaneus. It is important to be able to palpate these separately (this may be easier to do in a non-weight-bearing leg). It is not uncommon for the superficial digital flexor tendon to be dislocated medially and usually requires surgical replacement. Rupture of the Achilles tendon or the gastrocnemius muscle is not uncommon.

Tibia and fibula

Moving more proximally, you should palpate the entire length of the tibia and the fibula. The cranial tibialis muscle, long digital extensor, peroneus longus, and lateral head of the deep digital flexor all lie on the lateral aspect of the tibia. The semitendinosus tendon inserts on the proximal tibia medially, with the popliteus and lateral digital flexor. Caudally is the gastrocnemius and deep to that is the superficial digital flexor. The gastrocnemius and the superficial digital flexor originate from the caudal distal femur. It is important to be able to palpate the head of the fibula, as this is a landmark used for assessing cranial cruciate rupture. The distal tibia and fibula can also be a site for hypertrophic osteodystrophy.

Stifle

This brings us to the stifle (knee). It is very important to be able to palpate for joint effusions, instability, crepitus and cruciate laxity. The majority of hindleg lameness comes from the stifle joint. It is also important to be able to assess for patellar stability. It is very important for the examiner to be able to assess the cranial cruciate ligament (CCL). Rupture of the CCL is most commonly a degenerative process with a sudden worsening. Occasionally it is purely a traumatic incident. For a more complete understanding of the pathogenesis, the reader should be able to find an abundance of literature using common databases and texts. It is not a goal of this section to explain the disease process in cranial cruciate disease in dogs. The ability to properly assess the stifle is paramount. It cannot be performed without having a sound knowledge of the anatomy of the stifle joint (Figure 6.17). As with all joints, the stifle should be assessed in weight bearing as well as with the dog recumbent. With the dog in weight bearing, the examiner can position himself or herself behind the dog and use the left hand to palpate the left stifle and the right hand to palpate



Figure 6.17 Orientation of the femur and tibia.

the right stifle. Reaching from lateral across the cranial aspect, the examiner can use his/her fingers to assess the medial aspect of the stifles simultaneously. This is very important in detecting medial buttress (periarticular fibrosis on the medial aspect of the stifle joint). The joints can be assessed for joint effusion in this same position. By locating the patella and following it distally, the examiner can palpate the joint just medial and lateral to the patellar tendon. With the dog in either standing (but not weight bearing) or in lateral recumbency, the examiner should assess the integrity of the cranial cruciate ligament. The ligament has two portions. The 'craniomedial portion' is under tension in flexion and extension. The 'caudolateral portion' is under tension only in extension. For this reason it is important to assess the cranial cruciate ligament in extension and flexion. To examine the left stifle the examiner should place the left index finger on the tibial tuberosity, and the left thumb should be placed on the caudolateral aspect of the stifle over the head of the fibula. The right hand should be placed on the distal femur with the index finger on the patellar tendon just proximal to the patella and the right thumb on the lateral fabella (or on the lateral condyle on the femur) (Figure 6.18). The lateral fabella can be difficult to find in dogs with short thick, muscular legs (e.g. Staffordshire terriers). The examiner then attempts to move the tibia cranially with the left hand while stabilising the distal



Figure 6.18 Proper hand placement for assessment of cruciate stability.

femur with the right hand. In young, immature dogs there is more laxity and movement on assessing the cruciate but it will not be that free movement that comes to an abrupt stop. Another method of assessing cruciate integrity is the tibial compression test. It can be very difficult to assess the cruciate ligament in a dog that is stressed and tense or not cooperative. A negative result does not mean that it is not a cruciate injury. The presence of a 'click' when the dog is weight bearing or when the examiner is palpating or putting the stifle through flexion–extension and internal–external rotation would be suggestive of a meniscal injury. This would not mean that a negative exam result indicates no meniscal injury as this has poor sensitivity.

In one study of dogs with cranial cruciate disease, 100% of dogs had atrophy of the thigh muscles, 77% had joint effusion on physical examination but 100% had effusion on radiographs (Figure 6.19) and 70% had medial buttress (Figure 6.20).

Hip

The following bony landmarks should be identified and palpated (Figures 6.21, 6.22, 6.23):

- Wing of the ilium
- The ischial tuberosity/sciatic tuberosity
- The greater trochanter
- Body of the femur



Figure 6.19 Lateral stifle radiograph demonstrating joint effusion by displacement of patellar fat.



Figure 6.20 Caudocranial stifle radiograph demonstrating osteophyte production on the medial and lateral tibial plateau and an opaque periarticular thickening on the medial aspect of the stifle joint.



Figure 6.21 Orientation of the pelvis from a dorsal view.



Figure 6.22 Orientation of the pelvis and femur from a lateral view.

In medium to large breed dogs the coxofemoral joint is routinely affected by hip dysplasia and the accompanying secondary osteoarthritis. It may also be involved in trauma in all breeds. The joint itself is deep to the greater trochanter and its capsule cannot be palpated because of the overlying gluteal muscles, so examination largely depends on manipulation. This can be performed in lateral recumbency or in standing. Grasp and fix the stifle and then move the femur to manipulate the joint. Flex and extend the hip in the normal plane (cranial–caudal) of motion and then abduct it before returning it to a neutral position (femur almost perpendicular to the ground in the standing dog), then internally and externally rotating it. As hip dysplasia (HD) is so prevalent it is advisable to manipulate the joint gently at first and only test the limits of the range of motion, especially extension, at the end of the examination, as they may be painful. Placing the hip in extension tends to extend the lumbosacral joint, so the LS joint should be pressure tested with firm downward pressure in the standing animal as part of every hip examination.



Figure 6.23 Ventrodorsal radiograph of a 9-year-old male neutered German Shepherd's pelvis with severe hip dysplasia.

Tip

The greater trochanter of the hip is the most lateral point at the top of the thigh. Check its position by palpating with one hand while moving the stifle cranially and caudally with the other. Then locate the wing of the ilium cranially and the tuber ischii caudally to give you your landmarks for the hemipelvis.

Neck and back

Neck and back are not going to be covered in this section. This examination is to be covered in the neurology and physiotherapy assessment chapters (Chapters 7 and 8) as it is directly associated with trying to localise a spinal lesion. The assessment of back, neck and pelvis is also covered in the physiotherapy assessment chapter (Chapter 8). If a dog presents with back and/or neck pain they should be treated with extreme care and should never be manually manipulated until cleared of a vertebral instabilities.

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7

Neurological and muscular conditions

Philip A. Moses and Catherine McGowan

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7.1 Introduction

Animal neurology follows the same principles as human neurology, yet clearly there will be major differences in the neurological examination of animals and humans, particularly where verbalisation and comprehension are important in differentiating lesions in man. The aim of this chapter is to familiarise the reader with some differences in neuroanatomy between humans and animals, to describe the process of the neurological examination in small animals and horses, and to identify and explain some commonly encountered neurological and muscular conditions in animals.

7.1.1 Definitions

CNS – Central Nervous System (brain and spinal cord)

UMN – Upper Motor Neurone: brain and spinal neurones that initiate and control movement

LMN – Lower Motor Neurone: cell bodies are located in the ventral horn of the spinal cord; fibres are outside the spinal cord or brainstem – these neurones produce movement

Plegia and Paralysis – Complete loss of motor and sensory function

Paresis – Partial loss of sensation and partial to complete loss of motor function

Tetraparesis or Plegia – Paresis/plegia involving all four limbs

Paraparesis or Plegia – Paresis/plegia involving pelvic limbs

Hemiparesis or Plegia – Paresis/plegia involving thoracic and pelvic limbs on one side

Monoparesis or Plegia – Paresis/plegia involving one limb

7.2 Neuroanatomy

7.2.1 The spinal cord

The spinal cord is contained within the vertebral canal;

Table 7.1 Comparison of vertebrae between the dog, cat and the horse

Usual number of vertebrae	Dog and cat	Horse
Cervical (C)	7	7
Thoracic (T)	13	18
Lumbar (L)	7	6 (5 esp. Arabs)
Sacral (S)	3	5
Caudal (Ca)	Varies with tail length	Approximately 20

Table 7.2 The relationship between spinal cord segments and vertebral bodies in small animals

Vertebral segment	Spinal cord segments contained
L2	L2–3
L3	L3–4
L4	L4, 5, 6 (7)
L5	L7 S1, 2, 3

there is a larger epidural space in the cervical region. The spinal cord extends from the brainstem and terminates at L6 (dogs), L7 (cats) and S2 (horse) but there is both intra-breed and interbreed variation (Tables 7.1 and 7.2).

Spinal cord segments generally correspond to their respective vertebral segments from C1 to L1 or L2, caudal to this, spinal cord segments become shorter relative to the vertebral bodies. Similarly in the horse, the spinal cord segments generally correspond to the respective vertebral segments except caudally where the first three sacral segments occur within the last lumbar vertebra and the cord terminates within the cranial quarter of the sacrum (Dyce *et al.* 1987a, c).

In both small animals and horses the cord widens at the intumescences, cervical (C6–T2) and lumbar (L4–S3). The lower motor neurones (LMN) of the thoracic and pelvic

limbs arise from these segments respectively. LMNs are the efferent neurones for muscle contraction and are part of the simple reflex pathway. LMN reflex pathways are controlled for voluntary movement by higher motor centres – the upper motor neurones (UMN). UMN pathways tend to have a calming effect on reflexes, but their major role is in directing the various LMNs in voluntary movement (Mayhew 1989a). Major descending UMN pathways include the corticospinal, rubrospinal, reticulospinal and vestibulospinal tracts. As well as descending influences from the higher CNS centres (cerebral cortex, brainstem and cerebellum), ascending UMN pathways carry sensory information. These pathways include (from most superficial to deepest within the spinal cord) conscious proprioceptive, unconscious proprioceptive and nociceptive/pain pathways (Mayhew 1989a).

Descending UMN pathways can be divided into pyramidal and extrapyramidal systems. The pyramidal system is mostly involved in controlling finely adjusted movements, and the extrapyramidal, coarser movements, particularly in stereotypic locomotor patterns (Dyce *et al.* 1987b). The pyramidal system is of great importance in man, but less so in domestic animals. In dogs pyramidal fibres reach all levels of the cord, though fibre numbers decrease by 50% at the cervical cord. However, in horses the pyramidal system terminates at the level of origin of the brachial plexus and the extrapyramidal system is of much greater importance in controlling locomotion (Dyce *et al.* 1987b). This explains the decreased importance of corticospinal motor pathways in animals and why large lesions destroying the cerebrocortical motor centres do not cause permanent abnormality in gait, except for deficits in postural reaction testing in the contralateral limbs (De Lahunta 1983).

The blood supply to the spinal cord is from the paired spinal arteries. These give rise to the dorsal and ventral radicular arteries. The arterial supply in the dog and cat is more consistent with that present in man, with each spinal segment well supplied. There appears to be excellent capacity for collateral supply as well. For this reason vascular disturbances and infarcts appear to be less common and also less devastating than in man. Venous drainage is from small spinal veins, which drain into the large internal vertebral venous plexus that lies on the floor of the vertebral canal.

7.2.2 Vertebral anatomy of small animals

The cervical vertebrae numbered C1–7 contain spinal cord segments C1–8. The first vertebral body is the atlas, C1, a ring-shaped structure with prominent lateral wings. The axis, C2, has two smaller, caudally projecting transverse processes and a large dorsal spinous process. A strong dorsal atlantoaxial ligament joins C1–C2, ventrally the prominent dens on C2 articulates with C1 and has strong ligamentous attachments. There is no intervertebral disc between C1 and C2. Vertebrae C3–7 have similar morphology with a block-shaped vertebral body beneath an arched neural canal. There is a dorsal spinous process as well as transverse

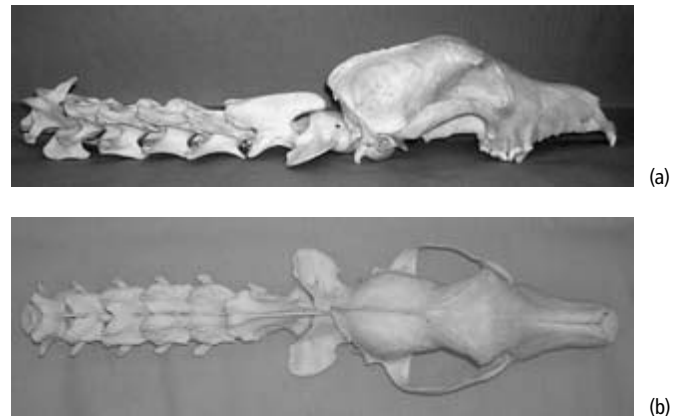


Figure 7.1 Normal anatomical specimens of the canine skull and cervical vertebrae, lateral (a) and dorsal (b) view; note the large transverse processes on C6.

processes extending ventrolaterally. C6 has very large transverse processes, thoughtfully placed there to assist surgeons in the identification and location of vertebral bodies (Figure 7.1).

The thoracic vertebrae are basically similar in structure to the caudal cervical vertebrae. The ribs arise from the costal fovea of the transverse processes laterally. There is a strong intercapital ligament between left and right rib heads from T1–T9, for this reason intervertebral disc prolapse is uncommon in this area. There are large dorsal spinous processes that tilt caudally on T1–10. T11 is termed the anticlinal vertebra, as the dorsal process is more vertical. T12 and 13 have dorsal processes that tilt cranially.

The lumbar vertebrae have larger, block-shaped vertebral bodies; the transverse processes are small and extend caudally. These transverse processes become larger more caudally and the dorsal processes become smaller more caudally.

The three sacral vertebrae are fused and articulate with the two ilial bodies via a C-shaped cartilaginous auricular surface. The dorsal spinous processes are small and the dorsal lamina is thin. There is an average of 20 caudal vertebrae although the number may vary from 6–23. Cats have less variation.

7.2.3 Vertebral anatomy of horses

The cervical vertebrae are similar to those in small animals, although long, with large transverse processes palpable to C5 or C6. The thoracic vertebrae have very long dorsal spinous processes (DSP) with the exception of T1, and T2 is usually deep to the scapulae, therefore T3 is usually the first palpable DSP and forms the start of the withers. The lumbar vertebrae have very long horizontal transverse processes, with synovial joints frequently developing between the L4–L5 or L5–L6 transverse processes or fusion may occur. There are variations in the lumbosacral pelvic anatomy in over 30% of horses and for more details the reader is referred to Chapter 4. The five sacral vertebrae are fused and S1 articulates with the ilium in the sacroiliac joint. The intervertebral discs in the horse are relatively thin, with a

less distinct boundary between the nucleus pulposus and annulus fibrosus than in other species.

7.2.4 The intervertebral discs and intervertebral disc disease (IVDD) in small animals

Intervertebral discs separate all vertebral bodies with the exception of C1–2. The discs provide flexibility between vertebrae and act to absorb shock along the spinal column. The discs have an external annulus fibrosus; composed of collagen fibres arranged in lamellae. Collagen, primarily of type I but with some type III, comprises 70–80% of the outer annulus. There are also elastic fibres arranged circularly, longitudinally and obliquely. The lamellae are separate to allow gliding movement during loading. The lamellae can form complete or incomplete rings. There are sensory (pain) fibres in the outer lamellae of the intervertebral disc. The annulus fibrosus may be weaker dorso-laterally as the lamellae are often incomplete in this region. This eccentric loading of the annulus fibrosus explains the propensity for the intervertebral disc to extrude dorsally.

The nucleus pulposus forms the inner portion of the intervertebral disc. The nucleus pulposus is an embryonic remnant of the notochord and is located slightly eccentrically. The nucleus pulposus is composed of an unorganised matrix of type II collagen and proteoglycans and is bound in 80–88% water. The nucleus pulposus is avascular and aneural. With age, the nucleus pulposus desiccates and undergoes chondroid metaplasia. This leads to cellular necrosis and a loss of the gelatinous nature as the nucleus pulposus is replaced with disorganised collagen and loses the capacity for movement and shock absorption. These changes occur very early in the chondrodystrophic breeds and may be complete by 2 years of age.

There are two distinct types of disc degeneration described by Hansen in his seminal work in 1952. They are known as ‘Hansen type I’ and ‘Hansen type II’. In Hansen type I disc disease, more commonly seen in the chondrodysplastic breeds, the intervertebral disc undergoes chondroid metaplasia. Type I disc disease is usually peracute or acute in presentation. It is characterised by increased collagen content, an alteration in the glycosaminoglycan concentration, a loss of water and an alteration of the proteoglycan content of the intervertebral disc. The disc becomes more cartilaginous and the nucleus more granular. There is a lack of gross distinction between the nucleus pulposus and the annulus fibrosus. The nucleus pulposus may undergo dystrophic calcification resulting in a loss of shock-absorbing qualities. Calcified intervertebral discs have been observed in Dachshund puppies as young as 5 months of age! Type I disc disease may occur in multiple discs within the vertebral canal.

In Hansen type II disc disease, more commonly seen in non-chondrodysplastic breeds, the disc undergoes fibroid metaplasia. Type II disc disease is slow and insidious. Water and proteoglycans are lost from the intervertebral disc

resulting in its narrowing. The nucleus pulposus is particularly affected and becomes indistinguishable from the inner lamellae of the annulus fibrosus. There is a reduction of glycosaminoglycans in the nucleus and annulus. The nucleus pulposus becomes fibroid in nature but rarely mineralises. Type II disc disease results from a bulging or protrusion of the annulus fibrosus and herniation of the nucleus pulposus through ruptured fibres in the annulus fibrosus. Ventral herniations are also reported and are thought to be associated with the formation of osteophytes and spondylosis deformans.

Note: There may be a blurring between Hansen type I and II disc disease and both processes may occur simultaneously.

7.3 Neurological examination

While the neurological diagnosis is clearly the realm of the veterinary surgeon, an animal physiotherapist should be able to perform a neurological examination, including localisation and grading of the findings as part of the assessment process. Recording and monitoring of these findings will allow an objective assessment of the patient’s response to treatment. Physiotherapists may be involved in treating and rehabilitating post-surgical neurological cases or may be the clinicians in charge of long-term follow up of many chronic neurological problems. *Many neurological cases require the team effort of both the veterinary surgeon and physiotherapist.*

When considering the equine patient it is also important to recognise the limitations in both diagnosis and treatment owing to the size of the patient. It is essential to have a respect and awareness of the potential danger involved. Even a quiet horse can become a serious threat in such situations where it is disorientated, ataxic or fearful. A key factor that may limit the rehabilitation of equine neurological patients is that if a horse is suffering from neurological deficits that make them ‘a danger to themselves or others’ euthanasia should be considered. This, of course, is the decision of the veterinary surgeon involved (usually involving communication with the insurance company) but one needs to be aware of potential dangers posed by a horse with major neurological disease.

In the large animal patient, simple recumbency presents its own challenges. Turning and taking care of a recumbent horse is difficult and a 24-hour-a-day concern. The rule of thumb is that serious myositis will occur following 6 hours of lateral recumbency in a large animal (Cox *et al.* 1982; Nout & Reed 2005). They are also susceptible to pressure sores, urine scalding, ocular damage and other problems of recumbency seen in other species (Nout & Reed 2005).

7.3.1 Preliminary examination and history

Initial collection of data, as for any other area, is important and should include:

- Patient signalment – age, sex, breed, use, value, history (of this and other problems)
- A veterinary physical examination
- Observation

A thorough veterinary physical examination will help differentiate neurological from metabolic, cardiovascular and musculoskeletal disorders. Also consider mental status, gait, posture, trauma, facial expression and breathing pattern.

Signalment is important to develop differential diagnoses and vital to characterise disorders as:

- Acute or chronic
- Progressive or static
- Persistent or intermittent

7.3.2 The examination procedure

The aim of the neurological examination is to establish the presence of neurological disease and hopefully, neuroanatomical location. A consistent, complete and methodical approach is essential. The most commonly used approach is the head to tail approach (Mayhew 1989b).

A neurological examination form (Figure 7.2) is vital to ensure the examination is carried out in a thorough and methodical manner.

Repeated/serial neurological examinations should be performed to assess any alteration in status. The neurological examination should be performed in an area free from distractions.

Examination of the head

The following abnormalities may indicate disease of the forebrain:

- Abnormal mentation (usually marked depression) – abnormal mentation can also indicate brainstem dysfunction (if affecting the reticular activating system or RAS)
- Seizures
- Dementia
- Progressive neurological dysfunction
- Behavioural changes
- Personality changes

The following abnormalities may indicate cranial nerve disease (or brainstem disease if associated with depression and gait abnormalities):

- Cranial nerve abnormalities
- Vestibular signs

The following abnormalities may indicate cerebellar disease:

- Tremors, and
- Ataxia

Head tremors may be a result of cerebellar disease. Cerebellar disease is rare in adult animals, but abiotrophy has been reported in Arabian foals, cats and other animals.

Cerebellar-associated locomotor dysfunction may be difficult to differentiate from ataxia induced by spinal cord lesions without advanced imaging (CT and MRI). Spinal lesions rarely cause changes in the above. The major exception is Horner's syndrome (first order) associated with cervical, spinal cord compression.

Initial examination of the head involves

1. Observation of behaviour and awareness or mentation, where abnormalities may indicate that the forebrain is involved. It may be important to observe the animal undisturbed to see these changes.
2. Observation of head posture, an abnormality that can be an indication of peripheral or central vestibular, central, cerebellar, musculoskeletal or even neuromuscular abnormality.

A *head tilt* is indicative of vestibular disease and occurs towards the side of the lesion. A head tilt is characterised by the poll deviated about the muzzle, versus a *head turn* which involves the whole head and often neck, and is associated with forebrain disease. In the latter case the animal may also turn and compulsively circle away from the site of the lesion.

7.3.3 Cranial nerve examination

Cranial nerve I – Olfactory

The olfactory nerve is sensory and involves integration in the forebrain. Olfaction can be tested with visual and non-visual smelling of food. A normal response is sniffing and licking. Irritating substances are best not used as they can also stimulate the trigeminal nerve and produce false results. It is almost impossible to demonstrate a unilateral olfactory nerve lesion owing to the ability of one olfactory nerve to sense the food adequately.

Cranial nerve II – optic nerve

The optic nerve is sensory. The facial nerve (VII) provides efferent motor innervation and information is processed in brainstem, forebrain and cerebellum.

The optic nerve is assessed by:

1. Pupillary size and symmetry (anisocoria is unequal pupil size) and pupillary light reflex. The pupillary light reflex examines both the optic nerve and oculomotor nerve supplying both eyes.
2. Vision. In small animals this may be assessed by seeing if the animal follows your hand, with a cotton wool ball or toy. In the horse, an obstacle course with the contralateral eye covered is often required to clearly demonstrate visual deficits.
3. The menace reflex. The menace reflex is a response to a threatening or menacing gesture. It is important not to touch the eye or create a wind effect, which will cause the eye to blink due to the trigeminal (V) nerve. Facial nerve damage can reduce the response owing to an inability to blink (lagophthalmus).

HISTORY PHYSICAL EXAMINATION OBSERVATION Mental status (e.g. alert, depressed, stupor, coma) Posture (e.g. normal paraparesis, hemiparesis, head tilt, tremor) Gait (e.g. ataxia, circling)		SPINAL REFLEXES Reflex (Nerve) (Spinal cord Segments)	
POSTURAL REACTIONS LEFT		LEFT	RIGHT
	Hopping		Triceps (Radial) (C7-T1)
	Front		Biceps (Musculocutaneous) C6-C8
	Rear		Withdrawal (thoracic limb) (Multiple) C6-T4
	Paw position		Patellar (Femoral) (L4-L6)
	Front		Gastrocnemius (Tibial, sciatic) (L6-S1)
	Rear		Withdrawal (pelvic limb) (Sciatic) (L6-S1)
	Reflex step		Perineal (S1-S2)
	Front		
	Rear		
	Tactile placing		
	Front		
	Rear		
	Visual placing		
	Front		
	Rear		
	Hemistanding		
	Hemiwalking		
	Wheelbarrowing		
	Extensor postural thrust		
CRANIAL NERVES		SPINAL HYPERAESTHESIA PANNICULUS REFLEX Level of cut-off (dermatome)	
LEFT	Test (Innervation)	LEFT	RIGHT
	Menace response (II & VII)		
	Vision (II)		
(SML)	Pupil size		
	Pupillary light reflex (II & III)		
	Stimulate left eye		
	Stimulate right eye		
	Strabismus (III, IV & VI)		
	Spontaneous nystagmus (III, IV, VI & VIII)		
	Positional nystagmus (III, IV, VI & VIII)		
	Oculovestibular response (III, IV, VI & VIII)		
	Facial sensation (V)		
	Jaw tone (V)		
	Temporal muscle mass (V)		
	Corneal reflex (V, VI & VII)		
	Facial symmetry (VIII)		
	Palpebral reflex (V & VIII)		
	Hearing (hand clap) (VIII)		
	Swallowing or gag reflex (IX & X)		
	Tongue (XII)		
URINARY FUNCTION Voluntary urination? Bladder distention? Overflow/ease of manual expression		DEEP PAIN PERCEPTION Thoracic limb Pelvic limb Tail	
		EYES Horner's syndrome (Sympathetic) Fundic examination	
		MUSCLE PALPATION Tone Atrophy	
		LESION LOCALISATION BRAIN Forebrain Brainstem Cerebellum Vestibular-peripheral Vestibular-central Multifocal	
		SPINAL CORD C1-C5 C6-T2 T3-L3 L4-S3	
		MULTIFOCAL CNS	
		PERIPHERAL NERVE Local Generalised	
		NEUROMUSCULAR	
		MUSCULAR	
		NORMAL	
SCORE: Absent = 0, Reduced = 1, Normal = 2 Increased = 3, Clonus = 4			

Figure 7.2 Example of a neurological examination form. This form is designed for small animals but could be adapted for horses.

Cranial nerves III, IV and VI – oculomotor, trochlear and abducens nerves

The oculomotor, trochlear and abducens nerves are motor nerves responsible for the innervation of eye movements. The abducens nerve controls globe retraction. The oculomotor nerve also mediates pupillary constriction in the pupillary light reflex. Therefore, mydriasis (pupil dilation) may indicate oculomotor nerve damage.

Abnormalities of the oculomotor, trochlear and abducens nerves are seen primarily as strabismus (abnormal eye position). This can be assessed by ensuring the animal is able to position its eyes appropriately when its head is raised and lowered and moved from side to side.

Cranial nerve V – trigeminal nerve

The trigeminal nerve provides sensory innervation to the face and motor innervation to muscles of mastication. The temporalis and masseter muscles are palpated for asymmetry and atrophy. Bilateral weakness of masticatory muscles may result in an inability to close the mouth.

There are three sensory branches of the trigeminal nerve: ophthalmic, maxillary and mandibular, that can be independently assessed:

1. Palpebral reflex elicits a blink reflex, which is mediated by the ophthalmic branch of trigeminal nerve.
2. Touching or pinching the upper lip lateral to the canine tooth in small animals results in wrinkling of face and blinking which is mediated by the maxillary branch of the trigeminal.
3. Touching or pinching the lower lip lateral to the canine tooth in small animals results in wrinkling of the face and blinking which is mediated by the mandibular branch of the trigeminal nerve.

To elicit the same response as (2) and (3) in horses, closed haemostats may be used to tap the face to produce a twitch response, often accompanied by a head nod. It is also important in cases where forebrain disease is suspected to determine that an appropriate behavioural response accompanies the local twitch response. To elicit more of a behavioural response the haemostats can be used to probe the sensitive nasal septum bilaterally.

Cranial nerve VII – facial nerve

The facial nerve provides motor innervation to the muscles of facial expression and also parasympathetic innervation of the salivary and lacrimal glands. The facial nerve is assessed by examining for facial asymmetry such as lagophthalmus, flaccid facial features and/or lips and/or abnormal ear carriage.

In the horse, motor control of the face is assessed at the nostrils where any asymmetry will be most evident and may be the only area affected in distal lesions. The eyelids or strength of eyelid closure may be palpated when trying to determine if the eyes are also involved in more proximally

located lesions. Lesions that occur close to the exit of the facial nerve from the cranium will involve the auricular branch and the affected side will show an ear droop.

Cranial nerve VIII – vestibulocochlear nerve

The vestibulocochlear nerve provides sensory innervation for hearing and vestibular function. Hearing can be tested by evaluating the response to loud noise; however, this does not differentiate between unilateral hearing loss and normal hearing. Vestibular dysfunction includes head tilt, abnormal nystagmus (abnormal rhythmical eye movements) and an ataxic, broad-based stance.

In horses, the vestibular part of this nerve may be assessed using either a blindfold (Figure 7.3a & b) or moving the horse from the light into a darkened area (e.g. stable), which will accentuate any head tilt (as well as nystagmus and ataxia).

Cranial nerves IX and X – glossopharyngeal and vagus nerves

The glossopharyngeal and vagus nerves provide sensory and motor innervation to the pharynx. The vagus nerve also controls laryngeal function. The gag reflex is elicited by touching left and right sides of the caudal pharyngeal wall in small animals, and observing elevation of soft palate and contraction of pharyngeal muscles. An asymmetric response is more significant than bilateral loss of gag reflex. Dysphagia, regurgitation, voice change and inspiratory stridor are other signs of nerve dysfunction.

Cranial nerve XI – accessory nerve

The accessory nerve provides motor innervation to the trapezius muscle. Muscle atrophy will be evident in accessory nerve lesions, although this may be difficult to assess.

Cranial nerve XII – hypoglossal nerve

The hypoglossal nerve provides motor innervation to the muscles of the tongue. The tongue is inspected for atrophy, asymmetry or deviation. Animals will usually lick their nose after finishing the gag reflex if the hypoglossal nerve is intact. Unilateral loss of innervation may result in the inability to lick one side of the nose or face.

In the horse, assessing the gag reflex is not possible without an endoscope. To assess pharyngeal function (as well as tongue tone and symmetry) a hand is placed into the horse's mouth at the diastema, the tongue palpated, then gently retracted out of the side of the mouth, then released. Normal horses should replace their tongue quickly and most swallow on replacing their tongue.

7.4 Posture, gait and reflexes in small animals

Gait and posture are important in the assessment of brainstem, spinal cord and peripheral nerve and muscle function. It should be noted which limbs are abnormal, and the deficits present.



(a)



(b)

Figure 7.3 (a) A horse with a mild head tilt and facial nerve paralysis including an ear droop on the left side; (b) with the head tilt worsened by applying a towel as a blindfold.



Figure 7.4 A 6-year-old Alaskan Malamute with a right hind conscious proprioceptive deficit.

Specific tests of neurological function include:

Posture

Posture should initially be assessed while the animal is free to move in the consulting room. Abnormalities that may be noted include head tilt, abnormal truncal posture, improper limb positioning (conscious proprioception), and decreased or increased muscle tone.

Gait

Gait should be assessed on a firm, non-slippery surface, observe the animal walking from the side, behind and moving away and toward the examiner.

Conscious proprioception

Indication of deficits include knuckling, foot misplacement and scuffing of toenails while walking as well as an inability to right the foot when placed over on the knuckles (Figure 7.4).

Ataxia

Ataxia while walking is characteristic of neurological, but not necessarily spinal, disorders. There may be lack of coordination with or without spastic, paretic or involuntary movements.

Palpation

Palpation is important to assess the musculoskeletal system and integument. It is important to compare symmetry between sides.

7.4.1 Postural and proprioceptive assessment

Proprioceptive positioning

If the dorsum of the paw is placed on the floor, the paw should be immediately returned to a normal position. Delayed (>1 second) or absent conscious proprioception indicates neurological disease.

Movement

Determine if the animal is ambulatory, weakly ambulatory or non-ambulatory. Assistance may be required in weak animals. The absence of voluntary movement indicates severe but not irreversible disease.

Wheel-barrowing

When wheel-barrowing the animal is supported under the abdomen with weight bearing on the forelimbs. A normal animal will walk forward with coordinated forelimb movement. Slow initiation may indicate a lesion in the cervical spinal cord, brainstem or cerebral cortex. Exaggerated movement may indicate a lesion in the cervical spinal cord, lower brainstem or cerebellum.

Hopping

The animal is supported as per wheel-barrowing but weight bearing on one leg. The animal is moved lateral and medial. Poor initiation of movement suggests a conscious proprioception deficit and poor movement suggests a motor deficit. Asymmetry may assist in lateralising the defect.

Extensor postural thrust

The animal is supported under the thorax and lowered to the floor. The pelvic limbs should move caudally with symmetric walking movements. Slow initiation may indicate a lesion in the spinal cord, brainstem or cerebral cortex. Exaggerated movement may indicate a lesion in the spinal cord, lower brainstem or cerebellum.

Hemistanding and hemiwalking

The front and rear limbs are elevated on one side and lateral walking movements assessed. Slow initiation may indicate a lesion in the spinal cord, brainstem or cerebral cortex. Exaggerated movements may indicate a lesion in the spinal cord, lower brainstem or cerebellum.

Placing reactions

It is important to assess tactile placing and then visual placing.

With tactile placing the animal is supported under the thorax and eyes covered as limbs touch a table edge. A normal response is to immediately place the feet on the table for positional support. Visual placing is the same except the animal is allowed to visualise the table edge. Visual placing requires visual pathways to the cerebral cortex, communication from the visual cortex to the motor cortex and motor pathways to the peripheral nerves of the forelimbs.

7.4.2 Spinal reflexes (or myotactic reflexes)

Myotactic reflexes test the integrity of sensory and motor components of the reflex arc and the influence of the descending motor pathways on this arc.

There are three possible responses:

- Absent or depressed reflex due to complete or partial loss of either sensory or motor component of the reflex arc, this is described as a LMN response
- Normal reflex
- Exaggerated reflex due to an abnormality in the descending pathway from brain to spinal cord, this is described as an UMN response

Forelimb reflexes*Triceps reflex*

The animal is placed in lateral recumbency with the uppermost leg supported under the elbow. The triceps tendon is struck with a reflex hammer just proximal to the olecranon. A normal response is slight extension of the elbow. The triceps reflex is innervated by the radial nerve originating from spinal cord segments C7–T1. An absent or depressed response should not be interpreted as abnormal. Exaggerated response indicates a lesion cranial to C7.

Biceps reflex

The animal is placed in lateral recumbency with the elbow slightly extended. A reflex hammer is struck on a finger placed over biceps tendon just proximal to the elbow. A normal response is slight flexion of the elbow. The biceps reflex is innervated by the musculocutaneous nerve and spinal cord segments C6–C8. Absent or depressed responses should not be interpreted as abnormal. An exaggerated response indicates a lesion cranial to C6.

Thoracic limb withdrawal reflex

The animal is placed in lateral recumbency and mild noxious stimuli inflicted on the foot. A normal response is flexion of the entire limb. Thoracic limb withdrawal reflex primarily involves the spinal cord segments C6–T1. Absent or depressed responses indicate a lesion of either spinal cord segments or peripheral nerves. An exaggerated response indicates a lesion cranial to C6.

Extensor carpi radialis reflex

The animal is placed in lateral recumbency with the elbow supported and flexed and carpus flexed. The proximal belly of the extensor carpi radialis muscle is tapped with the tendon hammer. A normal response is mild extension of the carpus. The extensor carpi radialis reflex innervated by the radial nerve and spinal cord segments C7–T1.

Hindlimb reflexes*Patella reflex*

The animal is placed in lateral recumbency with the affected leg uppermost and supported underneath the limb. The patella tendon is struck with a reflex hammer (Figure 7.5). A normal response is a single, quick extension of the stifle. A unilateral loss suggests a peripheral nerve lesion (femoral



Figure 7.5 Patella (femoral nerve) reflex being assessed on a dog.

nerve). Bilateral loss suggests a segmental spinal cord lesion L4–L6. An exaggerated response suggests a lesion cranial to L4.

Pelvic withdrawal reflex

The animal is placed in lateral recumbency and mild noxious stimuli inflicted on the foot. A normal response is flexion of the entire limb. Spinal cord segment L6–S1 and the sciatic nerve are in the withdrawal reflex arc. Unilateral loss suggests a peripheral nerve lesion (sciatic nerve). Bilateral loss suggests a segmental spinal cord lesion L6–S1. An exaggerated response suggests a lesion cranial to L6.

Sciatic reflex

The animal is placed in lateral recumbency with uppermost leg supported under the stifle and extended. A reflex hammer is tapped between greater trochanter and ischiatic notch. A normal response is a flexion of the stifle and hock.

Gastrocnemius reflex

The animal is placed in lateral recumbency and the tendon of insertion of the gastrocnemius is struck dorsal to the hock. A normal response is extension of the hock. The gastrocnemius reflex is innervated by the tibial branch of the sciatic nerve and L7–S1 spinal cord segments.

Panniculus or cutaneous trunci reflex

A pinprick stimulus is applied to the skin of the back beginning from L5 and progressing cranially. Both left and right sides are assessed. A normal response is unilateral twitching of the cutaneous trunci muscle at the point of stimulation and cranial. An absence of response suggests a lesion 1–2 segments cranially. This reflex is not always reliable.

Perineal or anal sphincter reflex

Gentle stimulation is applied to the perineal area with a needle or forceps. A normal response is contraction of the

anal sphincter muscle. The sensory and motor innervation is via the pudendal nerve and spinal cord segments S1–S3. An absent or depressed response indicates a lesion in the sacral spinal cord or pudendal nerve.

Crossed extensor reflex

This reflex is observed during withdrawal reflexes of either the pelvic or thoracic limbs. Toe pinching of one limb results in flexion of that limb and extension of the contralateral limb. A crossed extensor reflex is caused by a lesion affecting descending inhibitory pathways UMN and is suggestive of severity or chronicity.

The Schiff–Sherrington phenomenon

This phenomenon is usually an indication of severe spinal cord injury. The Schiff–Sherrington reflex is caused by loss of ascending inhibition from pelvic limbs resulting in forelimb and neck hypertonicity. The postural reactions and reflexes of the thoracic limb are otherwise normal.

7.4.3 Urinary bladder innervation

The bladder is innervated by both autonomic (hypogastric and pelvic) and somatic (pudendal) nerves. The pudendal nerve innervates the striated muscle of the urethra and maintains urinary continence. A lesion above S2–S3 will cause spasm of bladder outflow and difficulty in expression of the bladder UMN. A lesion below S2–S3 will cause lack of sphincter tone and an easily expressible bladder LMN. In general a LMN bladder carries a worse prognosis.

7.4.4 Pain perception

Pain perception provides important prognostic information and is the last test to be performed in a neurological examination. A painful stimulus is applied to each foot and the tail. Perception of pain is indicated by a significant behavioural response (i.e. turns and looks, vocalises or attempts to bite). Progressively stronger painful stimuli may be applied to assess presence of deep pain sensation. Deep pain perception may be modified by temperament, drugs, pain threshold and experience. The absence of deep pain is a poor prognostic sign.

Hyperpathic level

Pressure is applied to the spinous processes and paraspinal muscles of the thoracic and lumbar region and transverse processes and paraspinal muscles of the cervical region. Increased sensitivity may occur at the level of spinal cord disease.

7.4.5 Interpretation of gait posture and reflex abnormalities in small animals – spinal lesions (Table 7.3)

Spinal cord injury results in loss of function in the following order:

1. Conscious proprioception
2. Voluntary motor function

Table 7.3 Spinal cord lesions may be localised according to the neurological status of the limbs. Lower motor neurone (LMN) signs tend to predominate if both LMN and upper motor neurone (UMN) signs are present

Lesion	Forelimbs	Hindlimbs
C1–C5	UMN	UMN
C6–T2	LMN	UMN
T3–L3	Normo-reflexic	UMN
L4–S3	Normo-reflexic	LMN

3. Superficial pain
4. Deep pain

Return of function following spinal cord injury is in the reverse order and, roughly, in a similar time frame to loss of function. That is, a Dachshund with Type I IVDD that loses voluntary motor function in 6 hours will, with decompressive surgery often be ambulatory in 24 hours, whereas a German Shepherd with chronic Type II IVDD that has 6 months of deterioration before becoming non-ambulatory may not walk again.

Aetiology of spinal cord lesions may be assessed on the basis of history of pain and paresis.

1. Acute and static:
 - Vascular (e.g. fibrocartilaginous embolism (FCE) or infarction)
 - Trauma (e.g. fracture–luxation)
 - Degenerative (e.g. type I IVDD)
2. Acute and progressive:
 - Degenerative (e.g. type I IVDD)
 - Inflammatory (e.g. discospondylitis and vertebral osteomyelitis)
 - Trauma (e.g. fracture–luxation)
 - Anomalous (e.g. atlantoaxial subluxation) and tumour
3. Chronic and progressive:
 - Degenerative (e.g. type I and II IVDD, cauda equina syndrome, caudal cervical spondylomyelopathy or degenerative myelopathy)
 - Inflammatory (e.g. discospondylitis and vertebral osteomyelitis)
 - Neoplasia of the meninges or spinal cord

7.5 Posture, gait and reflexes in horses

Due to the simple problem of not being able to physically accomplish many of the tests outlined above for dogs, examination of the horse for brainstem, spinal cord and peripheral nerve and muscle function relies primarily on observation of normal gait, and posture during normal gait and during specific manoeuvres (Table 7.4). As with small animals it should be noted which limbs are abnormal, and the deficits present.

Table 7.4 Abnormalities vs. region – once the presence of ataxia, weakness and gait changes in all four limbs has been determined, this information can be used to localise the lesion

Region affected	Predominant signs
Brainstem/spinal cord white matter (UMN)	Ataxia, paresis, hypermetria (usually hindlimbs), spastic hypometria (usually forelimbs)
Vestibular system	Ataxia, hypometria
Cerebellum	Ataxia, hypermetria
Ventral grey matter or motor nerves (LMN)	Paresis, atrophy
Musculoskeletal system	Paresis, (hypometria)

(UMN) upper motor neurone; (LMN) lower motor neurone

The animal is examined for deficits including:

- Evidence of lameness or musculoskeletal abnormality (Chapter 6)
- Atrophy of muscle groups
- Paresis (weakness): (UMN or LMN?)
- Ataxia (general or unconscious proprioception deficits)
- Conscious proprioception deficits
- Spasticity (increased muscle tone)

As a general rule of thumb, lameness is a consistently abnormal gait pattern versus neurological disease, which is inconsistent.

7.5.1 Weakness (paresis)

It is important to always consider weakness when assessing gait of a horse. If it is an UMN disease, weakness as well as ataxia will mean a higher grade and more severe lesion. If there is marked weakness without much ataxia evident: a LMN or ‘neuromuscular disease’ may be suspected.

Specific tests for weakness (paresis):

- Look for hoof wear – dragging toes, low arc of flight of the hoof (more indicative of LMN weakness, low muscle tone)
- Tail pull – at rest (if unable to fix limb in extension more likely to be LMN); and during walking (UMN – able to reflexly resist while standing still, but due to combination of ataxia and paresis, weakness in response to pulling the tail is more pronounced at the walk) (Figure 7.6)
- Hopping (Figure 7.7), circling, slope – trembling, buckling of weak limb, knuckling over
- Often simply picking up one leg will be all that is required to elicit trembling, buckling of the weight-bearing limb in a very weak horse
- Note: horses with generalised weakness ‘walk better than they stand’ – i.e. they can’t fix themselves in standing



Figure 7.6 Pulling the tail of a horse while it is being walked as a test for weakness and ataxia. Note the handler is keeping a close eye on the person holding the tail.



Figure 7.7 Hop test to exacerbate weakness – the examiner holds one from limb up and first observes for stability on the weight-bearing contralateral limb. If the horse is not overtly weak, the examiner pushes the horse away from them eliciting a hop.

Interpretation

- If there is generalised weakness, with no ataxia and spasticity, neuromuscular disease should be suspected
- If there is localised weakness, LMN or peripheral nerve disease should be suspected
- If weakness and ataxia are both present, an UMN lesion affecting the descending motor pathway ipsilateral and caudal to the site of the lesion is suspected
- Note also there can be apparent ‘weakness’ associated with vestibular disease as horses tend to tend to fall or collapse towards the side of the lesion

7.5.2 Proprioception

Ataxia is the loss of control of general body and limb co-ordination often present as a swaying of the trunk and abnormal swing phase of the limbs and is indicative of



Figure 7.8 Walking down (and up) a slope will often exacerbate abnormalities in conscious proprioception (especially if the head is concurrently raised), weakness and gait abnormalities (hypermetria or hypometria).

general (unconscious) proprioceptive deficits. This may be due to UMN, vestibular or cerebellar lesions.

Specific tests for ataxia:

- Observe for poor coordination, swaying, limb moving excessively during swing phase – weaving, abduction, adduction, crossing of limbs and stepping on them.
- Exaggerated by tight circles – pivoting, circumduction, serpentine, sudden stopping, backing, walking up or down a slope (Figure 7.8), raising the head.
- Note: there appears to be a component of visual compensation for ataxia in horses and raising the head or blindfolding can often accentuate ataxia.

Loss of conscious proprioception indicates damage to ascending sensory pathways to the forebrain. While in small animals this is easily tested by seeing if the animal weight bears on the dorsum of the foot, this is not useful in the horse. The simplest test is observation of stance after the horse is suddenly stopped.

7.5.3 Gait abnormalities

- Hypermetria refers to increased range of limb flight, usually associated with increased muscle tone, hypereflexia and may be accentuated by ataxia.
- Hypometria refers to decreased range of limb flight. If a horse is hypometric, it may be due to either low muscle tone/weakness (LMN) or increased muscle tone and spasticity (UMN).
- Dysmetria refers to either hyper or hypo or combination, i.e. an abnormal range of limb flight.

7.5.4 Additional tests for cervical spinal cord lesions

For horses with forelimb signs and spinal cord lesion localised cranial to T2, a closer examination of the neck and forelimbs can be used for confirmation and more specific localisation of the lesion.

This might include:

- Observation and palpation of neck for muscle atrophy, asymmetry, sweating.
- Range of movement of the neck. This is rarely reduced in cervical vertebral malformation (CVM), but may be reduced in acute fractures.
- Sensory perception. Looking for local sensation and pain responses – two-step technique using haemostats at first lightly pinching the skin fold, then pressure is applied and response observed. Local cervical and cervicofacial reflexes can be applied assessing sensation over the neck with local or facial responses.
- Sway reaction can be used to assess strength and coordination in response to pushing the horse at the shoulder away from you (similar to hop test Figure 7.7).

7.5.5 Additional tests for horses with thoracolumbar or cauda equina lesions

For horses with lesions caudal to T2, careful examination of the trunk, hindlimbs, tail and anus is warranted to again localise the lesion and determine the extent, if any, of LMN involvement. It is useful to assess routinely perineal and tail reflexes in every neurological examination, but is essential in any horse with hindlimb signs and no forelimb signs.

Examination includes:

- Observation and palpation for muscle atrophy, asymmetry, sweating
- Sensory perception, including the panniculus reflex and pain response
- Tail – voluntary movement, tone
- Perineal reflex/tail clamp
- Male external genitalia
- Rectal examination by a veterinarian – assess lumbar, sacral or coccygeal vertebrae, bladder volume, tone

7.6 Diagnostic techniques

7.6.1 Survey radiographs

General anaesthesia is essential for accurate positioning of small animals. Although the spinal cord cannot be seen on plain films, they are essential to assess vertebral body shape for abnormalities. Assessment of vertebral alignment is important, particularly post trauma. Alignment should always be assessed in two planes. Spinal deviation may also occur with developmental anomalies such as hemivertebrae.

There are characteristic radiographic changes associated with IVDD. Collapse of the intervertebral disc space and narrowing of the facet space and foramen may be seen. Mineralised IVD, while not diagnostic alone, are characteristic of type I IVDD. Occasionally, extruded disc material may be seen in the intervertebral foramen.

Vertebral shape should be assessed for anomalies, fracture, or pathology, therefore, knowledge of normal anatomy and variations is important. Vertebral osteo-



Figure 7.9 Correct spinal needle placement for a lumbar myelogram.

myelitis or discospondylitis causes bone lysis and sclerosis of vertebral bodies and end plates respectively.

7.6.2 Myelography

Myelography is recommended for the determination of lesions within the neural canal and with the limited availability of advanced imaging such as CT and MRI, myelography is more widely practiced in veterinary medicine than in human medicine. Myelography can differentiate between extradural, intradural-extramedullary and intramedullary lesions. It is useful for IVDD, neoplasia, vertebral body abnormalities, vertebral instability and many other conditions (Figure 7.9).

7.6.3 Computed tomography and magnetic resonance imaging

These are diagnostic modalities that are becoming increasingly available within veterinary medicine. These modalities have been limited by expense to universities and large referral institutions but are more accessible today. CT has better contrast resolution than radiography and provides information in such areas as: cause of extradural compression, vertebral body neoplasia, presence of spinal cord swelling, and subtle abnormalities in vertebral shape and structure (Figure 7.10).

MRI uses the differential magnetic properties of atomic nuclei to distinguish between tissues densities. MRI is an excellent modality for the assessment of neural tissue parenchyma. T2 scans are particularly useful for assessment of lesions within the spinal cord. MRI has been used to a limited extent in horses simply due to the inability to fit the entire horse into the chamber; however, the head of small ponies and neck of larger horses can be successfully imaged.

7.6.4 Cerebrospinal fluid (CSF) analysis

CSF analysis is recommended for identifying some spinal and intracranial disease processes. In small animals it is rarely definitively diagnostic. An exception to this is the



Figure 7.10 A large, contrast-enhancing mass in the fore brain of a 10-year-old Boxer dog. The dog presented with multifocal neurological signs including ataxia, altered mentation and seizures.

meningeal inflammatory process – granulomatous meningoencephalitis (GME). GME is a non-infectious cellular inflammatory disorder that is not uncommonly seen in small animals, particularly small/toy breeds of dog.

In horses, CSF analysis is particularly useful for detecting equine protozoal myelitis (EPM), although this disease is exotic to Australia and the UK. CSF analysis may also be a useful indicator of inflammatory or haemorrhagic CNS conditions. Electromyography is not widely practised in veterinary medicine outside universities.

7.7 Neurological disease in small animals

7.7.1 Forebrain disease

Forebrain disease is a major red flag for physiotherapists and any case that develops or shows signs of forebrain disease that has not been diagnosed and treated by a veterinary surgeon should be immediately referred back. Examples of forebrain disease include brain tumours and meningitides.

A detailed discussion of brain lesions and treatment is not undertaken here.

Meningitis

Meningitis is not uncommon in small animals and meningitis should be considered for any animal with spinal pain, especially cervical pain. The more commonly encountered forms are listed below:

Suppurative meningitis

Bacterial meningitis is uncommon in cats and dogs. Many different organisms have been implicated. It is important to note that transmission of meningeal infection from animal

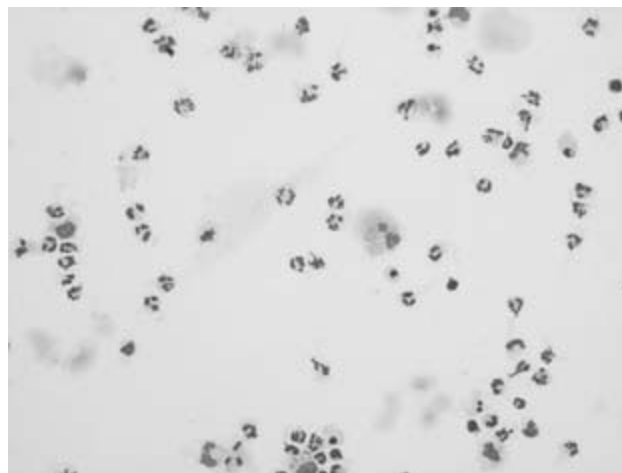


Figure 7.11 Haematoxylin and eosin (H&E) stained cytological preparation of the cerebrospinal fluid of a dog with granulomatous meningoencephalitis, note the large number of activated neutrophils. Total white cell count 394×10^9 . Original magnification $\times 1000$.

to man is very uncommon. Predisposing factors include paediatric animals, immunocompromised animals, bacteraemic animals and those housed in poor environmental conditions. Diagnosis is usually by neurological signs and CSF analysis. Prognosis is often poor due to diagnosis late in the course of disease.

Non-suppurative meningitis

Non-suppurative meningitis is relatively common in small animals. Among the more common manifestations are granulomatous meningoencephalitis (GME), steroid-responsive *meningial arteritis*, canine distemper and feline infectious peritonitis virus infections.

GME is most commonly encountered in small and toy breeds of dog (Figure 7.11). GME is of significant clinical importance as it may mimic many neurological conditions and small dogs with cervical pain should be examined very carefully for central neurological signs. GME is characterised by high CSF white-cell counts. Treatment with steroids or immunosuppressive drugs such as ciclosporin or cytosine may effect temporary relief, however, long-term prognosis is poor. GME is probably a group of non-suppurative meningitides that will be better understood in the fullness of time.

Steroid-responsive meningial arteritis is a breed-related condition and has been reported in Beagles and the Bernese Mountain Dog. As the name suggests the condition responds to treatment with steroids, however, long-term prognosis is poor.

Canine distemper virus infection was once common, however, with the advent of regular vaccination programmes it is uncommonly encountered today. Clinical signs are associated with multifocal CNS disease and vary from seizures to spinal pain. Treatment is limited to supportive care and prognosis is guarded.

Feline infectious peritonitis virus infection may cause neurological signs including, spinal cord signs; prognosis is poor.

7.7.2 Brainstem and cranial nerve disease

Vestibular disease

Vestibular disease is relatively common in the dog and some forms are responsive to medical management, residual neurological deficits are commonly seen. Vestibular disease is divided, anatomically, into peripheral, central and paradoxical.

Peripheral vestibular disease

Peripheral vestibular disease is a result of disturbance to either the peripheral vestibular sensing apparatus in the labyrinth of the inner ear or the vestibular nuclei in the medulla oblongata of the brainstem. There is usually no paresis or proprioceptive deficit as peripheral vestibular disease spares ascending and descending spinal tracts. The cranial nerves (other than the facial nerve) passing through the middle ear are also spared. Nystagmus is present and is horizontal or rotational.

Otitis media and interna

The middle and inner ear are contained within the petrous temporal bone and infection or inflammation of this region can lead to peripheral vestibular signs. The clinical signs include facial nerve paralysis or Horner's syndrome due to involvement of the facial nerve and ocular branch as they pass through the middle ear. Causes include infection and nasopharyngeal polyps (in the cat). The prognosis is good with definitive treatment, although head tilt usually remains.

Feline idiopathic peripheral vestibular disease

The aetiology of this condition is unknown although it is similar to Ménière's disease in humans. Resolution, with or without treatment usually occurs in 4–6 weeks although mild ataxia and head tilt may persist.

Squamous cell carcinoma

This tumour, usually arising from the pinna of the ear may extend into the middle ear resulting in vestibular signs. The tumour is aggressive in nature usually with significant local invasion and prognosis is poor.

Other tumours of the ear that may cause vestibular signs include:

- Fibrosarcoma
- Ceruminous gland
- Adenocarcinoma

Congenital peripheral vestibular disorders

This may be seen in certain breeds of both cat and dog (Siamese, Burmese, German Shepherd Dog, English Cocker Spaniel, Doberman Pinscher, Beagle and Shetland Sheepdog).

The cause is unknown and as it appears within a few weeks of birth, the condition is thought to be developmental. There is no treatment.

Canine geriatric vestibular disease

Geriatric vestibular disease is generally seen in older dogs, the aetiology and pathogenesis unknown. Onset is acute, however, the condition usually resolves within 1–2 weeks. Persistence of some neurological signs is common.

Central vestibular disease

Central vestibular disease may be caused by any brain disease involving the rostral medulla oblongata. The two most common causes are brain neoplasia and encephalitis. Clinical signs include hemiparesis or more commonly tetraparesis, depression and other cranial nerve deficits. Vertical nystagmus may be seen. Prognosis is usually poor.

Paradoxical vestibular disease

Paradoxical vestibular disease is uncommon but may be seen with any lesion in the caudal cerebellar peduncle. Clinical signs include contralateral head tilt, rolling and circling. UMN deficits are also seen. Prognosis is dependent on cause but is usually poor.

7.7.3 Spinal conditions affecting small animals

The most commonly encountered spinal conditions affecting small animals are:

- Vertebral body abnormalities
- IVDD types I & II
- Spinal fractures
- Atlantoaxial luxation/subluxation
- Caudal cervical spondylomyelopathy (wobbler syndrome)
- Lumbosacral conditions
- Fibrocartilaginous embolism
- Discospondylitis
- Chronic degenerative radiculomyelopathy

Vertebral body abnormalities

The most common vertebral body abnormalities seen in small animals are:

- Hemivertebrae
- Transitional vertebrae
- Block vertebrae
- Spina bifida

Hemivertebrae are most commonly seen in the screw tail breeds, i.e. Boston Terrier, Pug (Figure 7.12), English and French Bulldogs. This is the most clinically significant vertebral anomaly in the dog. There is a failure of ossification of part of the vertebral body, unilateral, dorsal or ventral. Clinical signs are usually secondary to cord compression and may be chronic or acute in onset. Decompressive

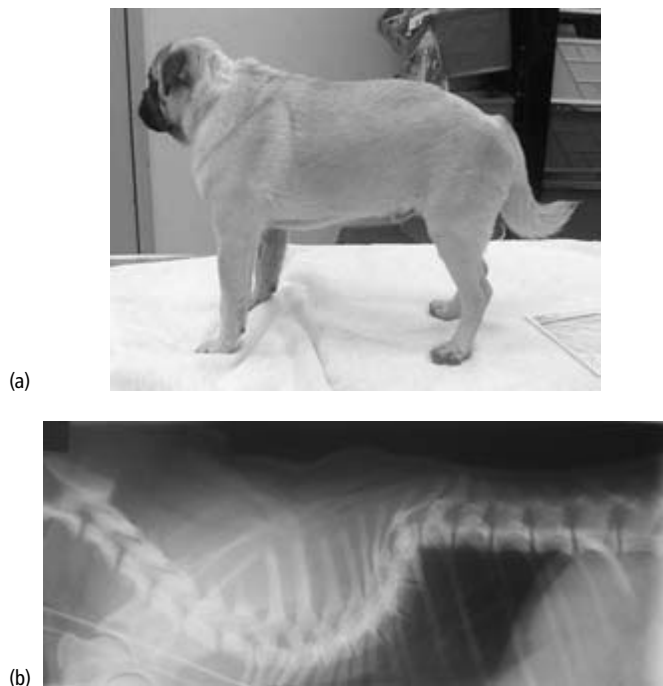


Figure 7.12 (a) A 6-month-old Pug with congenital hemivertebrae. (b) Lateral thoracolumbar radiographs of the same dog.

surgery may be indicated in some cases. Animals with vertebral body abnormalities should not be used for breeding.

Transitional vertebrae may be seen at any level of the spinal column. The main area of clinical significance is at the lumbosacral junction where transitional vertebrae may result in instability and spinal cord compression.

Block vertebrae result from improper separation of the vertebrae during development. Block vertebrae are inherently stable and are not usually clinically significant.

Spina bifida is characterised by an incomplete dorsal lamina and may not be of clinical significance in mild cases, however, in more severe cases with meningeal involvement (spina bifida cystica) neurological abnormalities are often evident and corrective surgery may be required. Spina bifida is more commonly seen in the screw tail breeds and in Manx cats.

IVDD types I & II

Due to the blurring between Type I and II IVDD these will be discussed together and within spinal cord regions.

Cervical disc disease

Clinical signs are related to location of lesion. Cervical disc extrusion causes severe pain occasionally with the absence of other clinical signs. Nerve root signs are common with more caudal lesions and Horner's syndrome may also be seen.

Diagnosis is based on signalment, clinical signs and imaging. Plain spinal films and myelography are essential prior to treatment.

Firstly, and very importantly the animal's collar should be removed and not replaced. Use of a harness for the remainder of the animal's life is advised.

Medical management of cervical disc disease may be attempted. Absolute rest and the judicious use of anti-inflammatories may be beneficial. However, in the author's experience, because of the pain associated with cervical disc disease, this condition is best treated surgically.

The most common site for cervical disc disease is C2–C3 and the frequency appears to diminish caudally.

Cervical IVDD in large breed dogs is usually Hansen type II and most commonly occurs between C5 and C7. This may be associated with caudal cervical spondylomyelopathy and is discussed later.

Surgical options

The common surgical treatments are:

- Ventral slot
- Dorsal laminectomy
- Dorsolateral hemilaminectomy
- Facetectomy
- Cervical disc fenestration

Ventral slot

A ventral slot is the most commonly performed technique for surgical management of cervical IVDD. The advantages include minimal muscle dissection and easy access to adjacent intervertebral discs for fenestration.

A ventral approach is made to the cervical spine, the relevant disc space identified and a ventral fenestration performed. A slot-shaped laminectomy is performed commencing ventrally and extending into the neural canal. Disc material is removed from the canal. Closure is then undertaken. Fusion between vertebral bodies occurs approximately 8–12 weeks postoperatively.

Disadvantages include, occasional severe intraoperative haemorrhage due to laceration of the venous sinus, incomplete removal of disc material, inability to perform the procedure on more than one vertebra owing to instability and post-operative vertebral body fracture or collapse.

Prognosis is dependent on the degree of sensory and motor loss as well as location of IVDD. The prognosis is improved if there are no thoracic limb sensory deficits in C2–C3 or C3–C4 lesions and if the animal is ambulatory within 96 hours of surgery. Complete recovery appears more likely with cranial cervical lesions than caudal cervical lesions. Long-standing tetraparetic animals have a significantly worse prognosis. Occasionally a C2–3 lesion will cause progression of neurological signs leading to brainstem swelling and respiratory and cardiac arrest. Following surgery the author expects 95–100% of dogs to be either normal or have mild cervical pain within 2 weeks of surgery. Recurrence of severe pain occasionally occurs within 1–2 weeks, usually because of incomplete disc removal or vertebral collapse



Figure 7.13 CT scan of the L2 of 4-year-old Dachshund, a large type I intervertebral disc extrusion is evident on the left side of the neural canal.

and should be investigated. Physiotherapy plays a vital role in recovery following spinal surgery and is increasingly becoming an invaluable component of therapy.

Thoracolumbar disc disease

This is the most common form of disc disease in small animals accounting for 66–86% of all cases.

Two forms are recognised:

- Type I disc disease – an acute, often explosive extrusion of disc material into the neural canal. Type I IVDD usually occurs in chondrodysplastic breeds of young age. T12–T13 is the most common site with incidence decreasing caudally in chondrodystrophic breeds (Figure 7.13).
- Type II disc disease – a chronic slow and insidious disc protrusion usually in older non-chondrodysplastic animals. L1–L2 is the most common site in non-chondrodystrophic.

Grading of these cases is important in deciding treatment options and prognosis.

The following grades are used by the author although other systems are available:

- Grade I – Single episode of mild, moderate or severe back pain with slight to no conscious proprioception deficits and no motor weakness. Prognosis is good with medical management and fenestration.
- Grade II – Recurrent back pain or persistent severe back pain with conscious proprioception deficits and ambulatory paraparesis. Prognosis is guarded to good with fenestration and conservative management but excellent with decompressive surgery.

- Grade III – Uncontrolled severe back pain with conscious proprioception deficits and ambulatory paraparesis. Prognosis is excellent with decompressive surgery.
- Grade IV – weakly or non-ambulatory paraparesis with back pain. Prognosis is good to excellent with decompressive surgery.
- Grade VA – Paraplegia less than 48 hours duration but with deep pain present. Prognosis is guarded to poor with decompressive surgery.
- Grade VB – Paraplegia greater than 48 hours duration but with deep pain absent. Prognosis is poor to grave, with decompressive surgery.

Treatment options – conservative or surgical

Many animals will respond to conservative management, however, recurrence at the same site is likely (up to 80%). Conservative management involves absolute cage rest and pain relief. Some authors advocate the use of corticosteroids in these animals but there is little scientific evidence that corticosteroids are of benefit and considerable evidence of deleterious side effects, care must also be exercised with the use of non-steroidal anti-inflammatory drugs (NSAIDs). A large number of human patients with spinal cord injury will develop gastrointestinal side effects, regardless of therapy; the situation is thought to be similar in animals.

Which animals are good candidates for medical management? Grade I and II animals have a similar prognosis with medical vs. surgical management, however, the recurrence rate is very high for medically managed animals. Some authors advise Grade VB animals have a similar recovery rate (albeit very low) for medical vs. surgical management; however, this author finds even severely affected animals have a better prognosis if treated surgically.

Surgical options

The commonly practised surgical options include:

- Disc fenestration
- Hemilaminectomy
- Pediculectomy
- Dorsal laminectomy

Dorsolateral hemilaminectomy

The author prefers to perform this procedure under magnification. With the animal positioned in sternal recumbency and rotated slightly away from the surgeon, a dorsal incision is made. The fascial attachments in the midline are incised and the epaxial musculature elevated from the dorsal spinous process. The muscular attachments onto the articular facets are sharply dissected. The articular facets are removed with rongeurs and the dorsolateral pedicles removed with a high-speed bur. Once the spinal cord is exposed the underlying disc material can be removed and the canal lavaged to remove remaining debris. The underlying disc and often, adjacent discs, may be fenestrated at this time. An

autogenous fat graft is usually placed over the spinal cord before closure to help prevent scar tissue causing narrowing of the neural canal. Closure is routine. Procedure for a dorsal approach is similar.

Advantages of hemilaminectomy are removal of the majority of the intervertebral disc, retrieval of disc material with minimal spinal cord trauma, ability to extend laminectomy cranially and caudally to improve retrieval of disc material and minimal effect of hemilaminectomy on torsional stability of the thoracolumbar spine.

Prognosis is very good, the success rates for non-ambulatory dogs, following hemilaminectomy is 79–95%. Physiotherapy plays a vital role in recovery following spinal surgery and is increasingly becoming an invaluable component of therapy.

Factors affecting neurological recovery

The time interval between the onset of neurological disease and surgical decompression is a major influencing factor. The recovery rate is far more rapid in dogs undergoing decompressive surgery within 48 hours of onset of clinical signs than those surgically treated after 48 hours. This delay has more profound effect on the prognosis of more severely affected animals.

The severity of neurological disease is also very important; the prognosis for recovery deteriorates with increasing neurological grade. Grade I recovery rate 95–99% – Grade V 45–65%.

The presence or absence of deep pain sensation is a major prognostic factor. The deep pain fibres are located in the dorsal aspect of the ventral commissure of the spinal cord. Severe spinal cord injury is required for animals to lose deep pain sensation. There are several studies (Duval *et al.* 1996; Scott & McKee 1999) with slightly differing outcomes but universal agreement that the absence of deep pain severely worsens prognosis. Addition of physiotherapy post surgery hastens neurological recovery.

Spinal fractures

Cervical fractures

Cervical fractures are uncommon. They can be associated with trauma or developmental weakness of a pathological nature. Overall, 80% of cervical fractures occur at C1–C2. Any animals with severe neck pain following trauma must be managed very carefully – especially if anaesthetised for radiography.

Non-surgical management

Conservative management may be attempted for minimally displaced fractures in the absence of severe neurological signs. Conservative management involves strict cage confinement, use of a neck brace and appropriate analgesia. Cage rest should be considered for 4–6 weeks. If there is any deterioration in neurological status the animal should immediately be reassessed.

Surgical management

Surgical management is indicated in unstable fracture–luxations, if the animal is weakly ambulatory or tetraparetic, or if conservative therapy is unsuccessful. Surgical management allows for decompression of the spinal cord as well as vertebral stabilisation. A variety of techniques have been described. The most common are the ventral and dorsal stabilisation techniques.

Ventral techniques involve identification of the affected site via a ventral approach. Stabilisation or fusion of the affected vertebrae and support until fusion is complete – usually 6–8 weeks. Dorsal techniques rely on restoring vertebral alignment and assisting with fixation of facet joints, fusion is unlikely, the dorsal spinous process does not allow plating to be undertaken.

Prognosis

Prognosis depends on neurological status before surgery as well as radiographic classification, method of repair and response to post-operative management. There is a poor correlation between degree of vertebral displacement and neurological condition. There appears to be a high peri-operative mortality rate (approaching 40%) with cervical fractures, however, if the animal survives the peri-operative period the recovery rate is excellent, with 97–100% of cases having a complete recovery. Poor prognostic factors include severe neurological status, deteriorating neurological status and an interval of more than 5 days before treatment.

In general, animals managed with conservative management tend to have milder neurological injury, a slower improvement but reduced hospital stay times, while those managed with surgery tend to have more severe neurological injury, more rapid improvement but increased hospital stay times.

Thoracolumbar fractures

The thoracolumbar spine is the most common site of spinal fracture–luxation in the dog and cat. Most are traumatic, usually with severe concurrent injuries both soft tissue and orthopaedic. It is important to remember that radiographic findings are not consistent with neurological findings. Up to 20% of animals have a second spinal fracture–luxation!

Functionally, the spine is made up of three connected units, the dorsal lamina and facet joints, the pedicles and vertebral body / intervertebral disc. This is the three-compartment theory. A disruption of the ventral compartment is most significant in destabilising the spinal column.

Treatment options are non-surgical management or surgical management.

Non-surgical management

Non-surgical management is best reserved for animals with stable fractures and minimal neurological deficits. Non-surgical management includes, strict cage rest, use of

a back splint and appropriate analgesia. If there is no improvement or there is neurological deterioration then the animal should be re-appraised.

A back brace aims to immobilise the vertebral segments cranial and caudal to fracture–luxation. The splint may be constructed from aluminium sheeting, thermosetting plastic or casting material. The splint may be best applied to an anaesthetised animal and then modified after recovery. In the author's experience, cats are intolerant of back splints.

Surgical management

Surgical management should be considered in animals with unstable fracture–luxation, weakly ambulatory or non-ambulatory tetraparesis, severe pain, or if conservative management is unsuccessful. The aim is to decompress the spinal cord and stabilise the fracture–luxation. Pathological fractures secondary to neoplasia are not usually treated surgically. In other cases of pathological fracture the underlying pathology must be diagnosed and treated.

A variety of surgical techniques have been described including: Steinmann or threaded pins and polymethyl methacrylate (PMMA) (Waldron *et al.* 1991), vertebral body plating, clamp rod internal fixator (CRIF) system, plastic dorsal spinous process plates, modified segmental spinal fixation (spinal stapling), external skeletal fixation (ESF) – both traditional ESF or circular ESF.

Post-operative management involves analgesia, cage rest and usually external support with cage rest for 4–6 weeks. Serial neurological examination is advised and any deterioration in neurological status should be thoroughly assessed.

Prognosis

Prognosis is dependent on neurological status, success of repair and response to repair. There is poor correlation between degree of vertebral displacement and neurological condition. In general, animals with presence of deep pain whose fractures are stabilised quickly have a good prognosis while those without deep pain have a poor prognosis.

Physiotherapy plays a vital role in recovery following spinal surgery and is increasingly becoming an invaluable component of therapy.

Lumbosacral and coccygeal fractures

Lumbosacral and coccygeal fractures are not uncommon in small animals. Animals subject to trauma from behind, usually in car accidents, often have fractures in this area. A major problem, along with loss of locomotion, is the loss of bladder control and anal tone. Bladder management in particular is vital both short and long term.

Non-surgical management

Non-surgical management can be considered for animals with stable fractures and minimal neurological deficits. Cage rest, application of a back brace and analgesia are

advised. Repeated neurological examinations are essential to determine response to therapy. Cage rest is advised for 6 weeks. Bladder management, if the animal is not urinating voluntarily, is vital.

Surgical management

Surgical management is advised for animals with unstable fracture–luxations, or that are poorly ambulatory or have non-ambulatory tetraparesis, or if conservative management is unsuccessful. The aim is to decompress the cauda equina and stabilise the vertebral bodies.

A dorsal approach is made and the fracture reduced. The most common method of maintaining reduction is the use of transilial pin fixation. A pin is placed between the ilial wings over the dorsal lamina of L7 to prevent dorsal luxation, the facet joints may be screwed or pinned as well. The use of pins or screws and PMMA bone cement has also been described. Most animals are too small for dorsal plating or stapling techniques. More recently, the use of transilial pins with an ESF has been described, as has use of the CRIF system.

Prognosis

In general, the prognosis depends on severity of cauda equina damage. The cauda equina nerve roots appear more resistant to compression than the spinal cord. The prognosis is good for animals with retained neurological function of the pelvic limbs, anus, urinary bladder, perineum and tail regardless of degree of compression; however, the prognosis is poor with loss of motor function and deep pain perception.

Atlantoaxial luxation

Atlantoaxial luxation may be either congenital or acquired.

Congenital luxations occur with little or no trauma and are secondary to failure of normal development of the dens (most common) or failure of normal development of the alar, apical and or transverse ligaments of the dens. This condition has been reported in many different breeds. Neurological signs tend to be less severe than with traumatic locations.

Acquired atlantoaxial luxation

This condition is most commonly seen in the toy breeds. Yorkshire Terrier, Lhasa Apso, Chihuahua, Pekinese, Toy Poodle and Pomeranian all appear predisposed. Clinical signs include abnormal low head carriage, progressive tetraparesis and ataxia associated with neck pain. There may be an acute presentation with very minor trauma and the animal may dislike its head being touched. Usually it occurs in young animals, less than 12 months of age.

Luxation may be due to axial fracture at synostosis between dens and body of the axis or luxation with an intact dens. The transverse ligaments ± apical ligaments must rupture for atlantoaxial luxation.

Diagnosis

Diagnosis is by clinical signs and usually plain radiography; although CT may be of benefit in planning surgical reconstruction.

Treatment may be conservative management or surgical.

Conservative management

Conservative management includes strict cage rest, a neck brace and appropriate analgesia. Cage rest should be for 6–8 weeks. Although conservative management may result in clinical improvement recurrence is common, as only limited soft tissue fibrosis appears to occur between the atlas and the axis.

Surgical management

Surgical management is more common. Indications include severe neck pain, severe neurological signs or if conservative management is not successful.

Either ventral stabilisation or dorsal stabilisation may be undertaken. Ventral stabilisation involves reduction of luxation and fusion of the articular joints with bone graft and screws, while dorsal stabilisation involves replacement or augmentation of the dorsal atlantoaxial ligament with wire, nylon or a fascial strip.

Prognosis

The prognosis depends on the severity of neurological signs and therapeutic management. Conservative management has a guarded prognosis due to recurrence. Surgical management has a good to excellent prognosis for ventral approach but a slightly reduced prognosis for the dorsal approach. Physiotherapy plays a vital role in recovery following spinal surgery and is increasingly becoming an invaluable component of therapy.

Caudal cervical spondylomyelopathy

It should be noted at this point that the author does not like the term ‘wobbler syndrome’ for this condition and instead uses the acronym CCSM.

CCSM is an abnormality of caudal cervical vertebrae or intervertebral discs causing spinal cord compression. Synonyms include:

- Canine caudal cervical spondylomyelopathy
- Cervical vertebral instability
- Spondylolisthesis
- Cervical spondylopathy
- Cervical malformation or malarticulation syndrome

The causes may be hereditary, nutritional, traumatic and acquired or a combination of these aetiologies.

There are five recognised different classifications:

1. Chronic degenerative disc disease
2. Congenital osseous malformation
3. Vertebral tipping

4. Hypertrophy of the ligamentum flavum

5. Vertebral arch abnormality and hourglass compression

C5–C6 is the most common site for non-IVDD-associated caudal cervical spondylomyelopathy, while C6–C7 is the most common site for IVDD-associated caudal cervical spondylomyelopathy. CCSM causes chronic spinal cord compression.

Clinical signs

Regardless of aetiology and classification type CCSM may be divided by functional appearance into static or dynamic compressive lesions. This is based on the myelographic appearance of the compression under traction and is important, as the treatment modality is dependent on this differentiation. Routine myelography of the caudal cervical spine is undertaken, mild traction is then applied and if the compression is unchanged the lesion is termed a static lesion, however, if the lesion resolves with traction it is termed a dynamic lesion.

About 80% of cases occur in the Great Dane and Doberman Pinscher, the author sees an increasing number of Labrador Retrievers as well as other breeds. Dogs may be seen at a young age (<2 years) with congenital compression of the spinal cord. Older animals presenting with progressive deterioration of neurological signs are most common.

The usual history is of progressive ataxia over a period of months to years. The thoracic and pelvic limbs are affected but signs begin earlier and are more pronounced in the pelvic limbs. Animal may also present acutely after minor trauma. There is cervical pain in only 40% of cases.

Neurological examination reveals stiff, short and choppy gait with straight legs, marked muscular atrophy and neck pain may be noted with manipulation in some cases. UMN reflexes are noted to the pelvic limbs and may include crossed extensor reflex if chronic.

Diagnosis

Diagnosis is based on clinical and neurological examination and plain and myelographic findings. Advanced imaging is not usually considered necessary in CCSM, as myelography is the best diagnostic modality for differentiating between static and dynamic lesions.

Treatment options may be divided into conservative or surgical.

Conservative management

Conservative management is not a good option. CCSM is a chronic progressive disease and conservative management is not effective for static lesions. Conservative management may provide short-term relief for dynamic lesions although underlying instability and malformation are not corrected and neurological deterioration will continue. Some authors state that use of a neck brace will lead to muscle atrophy resulting in a deterioration of neurological status.

Surgical management

The plethora of surgical techniques described highlights the disagreement among surgeons as to the best course of treatment. Like surgical management of cranial cruciate ligament disease, it would seem that each surgeon has his own technique!

The aims of surgery are to relieve spinal cord compression. The mechanism by which this is achieved will depend on the classification, clinical presentation, intervertebral space affected, number of lesions, location of lesions and the presence or absence of a dynamic lesion.

The techniques fall into four groups:

1. Ventral slot
2. Ventral stabilisation
3. Ventral distraction
4. Fusion and dorsal laminectomy

Fenestration alone is not recommended for animals with CCSM as it leads to collapse of the intervertebral space and may cause neurological deterioration. Many Dobermans have von Willebrand's disease and coagulation profile is highly recommended before surgery.

Prognosis

Prognosis for CCSM is generally good with surgery for ambulatory animals, however non-ambulatory dogs do not tend to do well (Jeffery & McKee 2001). Physiotherapy plays a vital role in recovery following CCSM surgery and is increasingly becoming an invaluable component of therapy (Figure 7.14 a, b and c).

Lumbosacral conditions / cauda equina syndrome

Cauda equina syndrome (CES) is a general term describing compression, inflammation or vascular compromise of the nerve roots of the cauda equina. CES is a relatively common condition and may be congenital or acquired. Like CCSM there are also static and dynamic forms of CES. In the normal anatomy the cauda equina is surrounded by the lamina, interarcuate ligament and articular facets dorsally,



Figure 7.14 (a) A 6-year-old Doberman with caudal cervical spondylomyelopathy (CCSM), standard myelogram notes large extradural compression of the spinal cord at C6–7.



Figure 7.14 (b) The same 6-year-old Doberman with caudal cervical spondylomyelopathy (CCSM), a traction myelogram note almost completes resolution of the extradural compression.

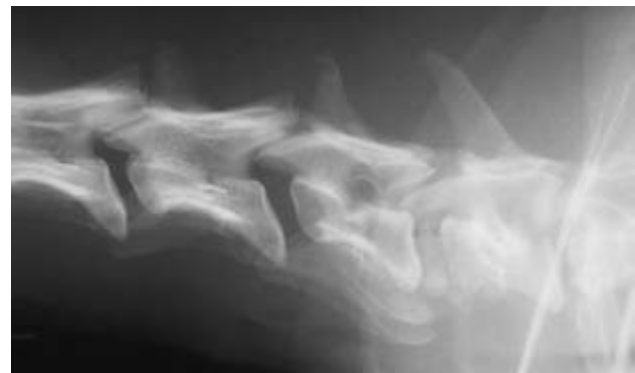


Figure 7.14 (c) The same 6-year-old Doberman with caudal cervical spondylomyelopathy (CCSM) following distraction-fusion surgery a polymethyl methacrylate (PMMA) plug has been used to maintain traction until fusion occurs.

the pedicles laterally, and the dorsal longitudinal ligament, and the vertebral venous sinus, dorsal annulus fibrosus and vertebral body ventrally. The vertebral bodies containing the cauda equina are L5–L7, S1–S3 and Cd1–Cd5.

Congenital cauda equina syndrome

Congenital CES involves congenital spinal canal stenosis, sacral osteochondrosis, transitional vertebrae affecting L7 and the sacrum. Congenital CES usually occurs in larger dogs and especially the German Shepherd Dog. Staffordshire Bull Terriers also appear to be over represented. There appears to be a male-dominated sex predilection.

Acquired cauda equina syndrome

Acquired CES conditions include vertebral fracture or luxation, discospondylitis, vertebral osteomyelitis and types I and II IVDD of the L7–S1 vertebrae, nerve roots or surrounding soft tissue. Degenerative lumbosacral stenosis is the most common cause of cauda equina syndrome. Acquired CES also occurs in large breeds of dog especially the German Shepherd Dog.

Clinical signs

Clinical signs are related to compression of the cauda equina and may be intermittent. Lower back pain, exercise-induced lameness, sphincter dysfunction, self-inflicted skin lesions of the tail, perineum, genitals or extremities may all be seen. The back is usually maintained in flexion and exercise-induced lameness, due to radiculopathy, may be evident. Clinical findings may also include, scuffing of pelvic limb toenails, reluctance to jump or sit, exercise intolerance or reluctance to exercise, urinary and faecal incontinence, abnormal tail carriage, pelvic limb muscle atrophy as well and hyperesthesia of the hind quarters. Intermittent claudication may be evident.

Diagnosis

Paraesthesia or dysaesthesia with excessive chewing on tail and lateral aspect of hind feet.

Diagnosis may be difficult, the condition is often dynamic at least in part, clinical signs may be intermittent and many older dogs will have spondylosis of the lumbosacral (LS) joint without clinical signs.

Plain radiographs with the LS joint in neutral, flexed and extended lateral views may show signs of increased mobility. Myelography is difficult, as the epidural sac of the spinal cord does not always cross the LS joint. Epidurography is very difficult to interpret. The author finds diagnosis best based on clinical examination and CT examination with the hind limbs forward under the body to accentuate compression.

Conservative management

Conservative management may be more successful than with other spinal conditions. Absolute rest for 6–8 weeks and the judicious use of NSAIDs are often of benefit.

Surgical management

Surgical management is advised if pain is severe or there is no improvement following conservative management.

The most common procedure is the dorsal laminectomy. A dorsal approach is made to the LS joint and dorsal lamina removed. The cauda equina/nerve roots are carefully retraced and disc material removed from the neural canal. If nerve root impingement is evident a foramenotomy may also be performed.

An alternate procedure is the distraction fusion procedure. This procedure involves distraction of LS space, which is maintained by screws across the facet joints and the addition of autogenous cancellous graft. A dorsal laminectomy may also be undertaken.

Prognosis

Strict confinement for 8–12 weeks after surgery is essential, excessive post-operative activity may result in a poor outcome. The prognosis is good to excellent in most cases with success rates of 85–100% being reported. Urinary and

faecal incontinence rarely resolve if present before surgery although many animals improve. Physiotherapy plays a vital role in recovery following spinal surgery and is increasingly becoming an invaluable component of therapy.

Fibrocartilagenous embolism (FCE)

FCE is the most common vascular spinal cord condition in the dog and second most common in the cat and contrasts greatly from the same condition in man. FCE occurs when a cartilaginous embolism lodges in a spinal vessel. The cartilaginous embolism is identical histopathologically to the nucleus pulposus of the intervertebral disc. FCE has been found in either arteries and veins as well as both arteries and veins! The origin of the FCE and the exact route of vascular penetration is the matter of some debate. The extrinsic spinal cord arteries are anastomotic and are not susceptible to vascular occlusion; however, the intrinsic arterial vasculature forms an end arterial flow system that is subsequently highly susceptible to embolisation.

FCE tends to occur in non-chondrodystrophic breeds. The Miniature Schnauzer is over represented. The condition is often lateralised. Clinical signs are acute onset; non-progressive (unless myelomalacia occurs), and non-painful neurological deficits. Often onset of the condition occurs during physical activity (61% of cases), a common history is that of an animal chasing a ball, suddenly crying out then having a neurological deficit in one limb. Neurological deficits usually stabilise at 24 hours. The specific neurological signs are dependent on the anatomical distribution of the FCE and the severity of the resultant ischaemia. FCE usually affects brachial or lumbosacral intumescences, 31% between C6 and T2 and 47% between L4 and S3. Lateralisation of neurological deficits is relatively common due to asymmetric branching of intrinsic spinal vasculature.

Diagnosis

Definitive diagnosis is difficult. Until recently an ante-mortem diagnosis could only be made by assessment of typical clinical features and exclusion of differential diagnoses. Mild cord swelling may be noted on myelography (Figure 7.15); however an MRI has been recently shown to clearly demonstrate spinal cord ischaemia.



Figure 7.15 Myelogram of a dog with a fibrocartilagenous embolism, note the slight swelling of the spinal cord at the level of L1.

Treatment

Treatment via conservative management, confinement to prevent self-trauma and extensive physiotherapy has been shown to produce excellent results (Lindsey Connell – Personal Communication).

Prognosis

Prognosis is generally good; however, a poorer prognosis has been reported for large breeds, older animals, animals with more severe neurological signs, extensive myelographic swelling, or changes in CSF and if there is deteriorating neurological status.

This contrasts greatly to the same condition in man where only 32 cases had been reported up to 1985 with only one surviving, the majority passed away in the short term.

Discospondylitis and vertebral phsitis

Discospondylitis involves infection in the disc space and is seen in middle-aged animals, while vertebral phsitis involves infection in the vertebral end physis and is seen in immature animals. The conditions will be discussed together. These infections are relatively common in dogs but not in cats. Discospondylitis tends to affect large breeds of dogs, especially the German Shepherd. Males outnumber females by 2:1 except for fungal discospondylitis, which is more common in females.

Clinical signs

Clinical signs are related to spinal cord compression as well as infection and are dependent on location of lesion(s). Animals tend to be pyrexia, lethargic and unwell, often with severe and progressive back pain and neurological deficits.

Diagnosis

Diagnosis is based on physical examination and radiological findings. Characteristic lytic lesions are found in the vertebral end plates or vertebral phsitis (Figure 7.16), more than one lesion may be present. Samples of urine and blood are taken for culture and sensitivity.

Treatment

Treatment is based on identification of causative agent and prolonged treatment with antibiotics is essential.



Figure 7.16 Vertebral phsitis of the caudal endplate of L4 evident on a lateral lumbar plain radiograph of a 5-month-old Mastiff breed dog. Note absence of normal physis and lytic changes in the vertebral body.

Surgery may be indicated if spinal instability is present or if the neurological deterioration occurs despite appropriate antimicrobial therapy. Fungal discospondylitis is not cured; however, it may be managed with medication for some time.

Prognosis

Prognosis is dependent on early diagnosis and treatment, the pathogen involved and sensitivity to antibiotic therapy and the degree and severity of neurological involvement. Physiotherapy plays a vital role in recovery following spinal injury and is increasingly becoming an invaluable component of therapy.

Chronic degenerative radiculo myelopathy (CDRM)

This debilitating neurological condition is usually seen in German Shepherd Dogs. These dogs may also present with type II IVDD and hip dysplasia. It is poorly understood and the deterioration is inevitable. Physiotherapy can play a major role in the treatment of these dogs.

The aetiology is unknown but the condition is similar to lesions seen in man due to subacute combined degeneration resulting from vitamin B₁₂ deficiency or associated with elevated methylmalonic acid. CDRM may be due to genetic, inherited or familial causes (breed predisposition) or immune-mediated disease. Vitamin E deficiency causes similar spinal cord degeneration in other species. However serum levels of Vitamin E are within normal reference range in dogs with CDRM.

Clinical signs

CDRM most commonly occurs in the German Shepherd Dog, however it may occur in any large breed. Older animals are more commonly affected with males more commonly affected than females.

Clinical signs are those of progressive loss of hindlimb motor function. Paresis is often asymmetrical in pelvic limbs. Urinary and faecal continence and pain sensation are preserved early in the condition but are lost later. Hindlimb reflexes are usually UMN although occasionally femoral nerve is LMN.

Diagnosis

Diagnosis is based on exclusion of other causes of neurological deterioration, as previously stated this can be difficult in animals with concurrent disease.

There is no specific treatment, although therapy with aminocaproic acid has been described. Other concurrent conditions are treated and extensive physiotherapy is advised to help maintain remaining motor function and muscle bulk. Deterioration of neurological status is inevitable.

Prognosis

Prognosis is poor, short term, and grave, long term.



Figure 7.17 A 6-year-old Australian Cattle Dog with a right brachial plexus injury secondary to trauma, note the left forelimb carriage and proprioceptive deficit.

7.7.4 Peripheral neuropathies

The peripheral nerves may be damaged anywhere along their pathway, from the cell body to the peripheral nerve ending on the effector organ. Abnormality of function will depend on the nerve(s) affected and the location of damage or disease (Figure 7.17). Peripheral neuropathy may be sensory or motor. Sensory neuropathy alone is uncommon. Motor neuropathy results in weakness, muscle atrophy and loss of reflexes.

Peripheral neuropathies may be acquired or congenital.

Acquired peripheral neuropathies

Acquired peripheral neuropathies include:

1. **Trauma.** This may be due to fractures, bite wounds or penetrating injuries (including injections). A common injury is brachial plexus injury or avulsion secondary to trauma. Complete loss of function owing to severance of the nerve is unlikely to improve although surgical repair may be possible with timely exploration.
2. **Endocrine.** Peripheral neuropathies may occur secondary to several endocrine conditions including: diabetes mellitus, hypothyroidism and hypoadrenocorticism.
3. **Paraneoplastic syndromes.** Several paraneoplastic syndromes can cause peripheral neuropathies.
4. **Infections.** Infections with *Toxocara* and *Neospora* have both been associated with peripheral neuropathies usually in young animals.
5. **Immune-mediated disease.** The most common manifestations are the equally perplexing polyradiculoneuritis (Coonhound Paralysis, USA) and distal denervating disease (UK).
6. **Toxins.** Toxicity from lead, organophosphates, mercury as well as chemotherapeutic drugs such as vincristine.

Congenital peripheral neuropathies

Hereditary conditions have been reported in a number of breeds including:

- Rottweiler, Brittany Spaniel – both may display an autosomal dominant neural degeneration
- Stockard's paralysis affecting Great Danes
- Abiotrophy reported in Swedish Lapland Dog and the Saluki
- Hereditary polyneuropathy of Alaskan Malamutes, Tibetan Mastiffs and Golden Retrievers
- German Shepherd Dogs have been reported with giant axonal neuropathy and spinal muscular atrophy

There are many other congenital conditions, these conditions are mostly found in young dogs and are generally progressive in nature. A full list of these conditions is reported by Summers *et al.* 1995 and Gough & Thomas 2004.

For further reading on peripheral neuropathies the reader is directed to Chrisman 1989 and LeCouter 1988.

7.7.5 Neuromuscular disease

A large number of conditions fall within this group. Neuromuscular diseases frequently present as non-ambulatory animals or weak animals. Diseases affecting the neuromuscular junction typically cause LMN-type reflexes with reduced reflexes.

Common causes include:

- Ixodes paralysis
- Snake envenomation, e.g. brown snake
- Myasthenia gravis
- Coonhound paralysis – polyradiculoneuritis
- Botulism
- Toadfish toxicity – tetrodotoxin
- Ciguartera toxicity

Alterations in ability to ambulate, altered mental status and paroxysmal events are common presenting signs in animals with neurological disease. However, these signs are not exclusive to neurological conditions. In most cases the information required to determine whether neurological disease is present can be obtained from a thorough physical examination and good clinical history. See Section 7.12 Neuromuscular disease.

7.8 Equine neurological diseases

7.8.1 Forebrain disease

As for small animals forebrain disease is a big red flag for physiotherapists and the hallmark signs of abnormal mentation or depression should be taken very seriously. Some animals might start off appearing quietly depressed and harmless and develop maniacal behaviour or seizures very suddenly, so be aware.

7.8.2 Brainstem/cranial nerve disease

Brainstem/cranial nerve disorders occurring in horses may well be amenable to physiotherapy in their treatment. This

section will focus on facial nerve paralysis and vestibular disease, which occur reasonably frequently.

Horner's syndrome will also be discussed in this section, as, although it is a deficit of the sympathetic nerve supply to the head, and not a specific cranial nerve or brainstem lesion, anatomically its signs manifest in the same region and it needs to be differentiated from eye abnormalities caused by specific cranial nerve deficits.

Major signs

- Vestibular disease – head tilt, nystagmus (horizontal, fast phase away from the lesion), ataxia. In addition, UMN gait quadraparesis and depression if central lesion
- Facial nerve paralysis
- Eye abnormalities: e.g. anisocoria, ptosis, and strabismus – it is important to differentiate cranial nerve deficits vs. Horner's syndrome
- Pharyngeal/laryngeal deficits: dysphagia. Dysphagia, apart from caused by botulism or facial nerve deficits is not very common in Australia. In the UK and USA it can be caused by fungal guttural pouch infections and is a serious red flag for referral back to a veterinarian
- Other cranial nerve deficits – see Section 7.3 Neurological examination

Facial nerve paralysis

Facial nerve injury is common in horses because the facial nerve runs superficially over the masseter muscles on the side of the face. General anaesthesia in the horse without removal of the head collar has caused instances of facial nerve paralysis, owing to the position of the buckle directly over branches of the nerve. Trauma and middle and inner ear infections can also cause facial nerve deficits.

Key to understanding the relationship of the facial nerve and vestibular nerve is their anatomical association – they arise from the brainstem close together and run together within the petrous temporal bone of the skull – so injuries to this area may cause injury and clinical signs involving both nerves.

Clinical signs

The signs exhibited by an affected horse give you the localisation of the lesion, which is important when working out both possible aetiology and prognosis. Deficits are only seen distal to the site of the lesion.

1. Facial nerve lesions over the side of the face – deviation of nares, muzzle only
2. Between stylo mastoid foramen (exit from skull) and mandible – cause signs of (1) above + ear & eyelid droop
3. Petrous temporal bone lesions (within skull) – cause signs of (2) above + loss of lacrimal secretion, \pm vestibular nerve signs
4. Brainstem lesions – causes signs of (3) above + other cranial nerve deficits, gait deficits, depression



Figure 7.18 Shows a horse with facial nerve paralysis that has resulted in the complication of corneal ulceration.

Horses with facial nerve paralysis with prolonged/permanent deficits can have serious complications including:

- Eye injury, dry eye (due to loss of lacrimation and lagophthalmus) (Figure 7.18)
- Dysphagia, feed pouching
- Poor performance – due to nostril collapse

Severe eye disease and nostril collapse may put an end to a performance career in some equestrian sports.

Prognosis

The prognosis for facial nerve paralysis as well as any peripheral nerve damage depends on the severity of damage. The terminology is the same as in other species (including man).

- *Neuropraxia* (loss of function only) – may resolve within 14 days
- *Axonotomesis* (severance of axons) – 6 months for recovery. Should be the worst scenario for closed lesions to the side of the face (i.e. iatrogenic)
- *Neurotmesis* (severance of entire nerve fibre) – prolonged to permanent loss of function. Scarring, fibrous tissue or callus formation may lead to (permanent) worsening of nerve injury

Vestibular disease

As for small animals vestibular disease is classified as peripheral if affecting the nerve after it leaves the brainstem, or central if involving the brainstem with additional signs.

Clinical signs and treatment

Trauma and infection are the major causes of peripheral vestibular disease in the horse (Blythe 1997a). Trauma usually involves fracture or injury to the petrous temporal bone. Infection is an insidious apparently haematogenous infection that can result in chronic osteomyelitis of the stylohyoid bone, temporohyoid joint and petrous temporal bone. Unlike in small animals it does not extend from external ear infections (otitis externa).

In either case, the lesion may be quite subtle to very severe. Subtle cases can be deceptive, as horses have the ability to compensate visually, particularly in chronic/longstanding cases. Hence blindfolding (or moving from light to dark) is an important part of the neurological examination. Vestibular disease should be considered in horses that are extremely one sided, and has also been associated with head shaking (Blythe 1997a). If vestibular disease is suspected, look for evidence of facial nerve paralysis as well, in longstanding cases an ear droop might be the best clue (Figure 7.3). To confirm the diagnosis endoscopy of the guttural pouches (to visualise the stylohyoid bone and temporohyoid joint), radiography and possibly nuclear scintigraphy are used.

Prognosis

Prognosis is guarded for full recovery, as chronic cases of either cause are very unlikely to improve long term owing to the likelihood of permanent bony changes affecting the nerve. However, acute cases, especially traumatic, can have a good outcome. Acute exacerbation of chronic otitis media/interna usually have very poor prognosis as fracture of the stylohyoid bone may have occurred.

Central vestibular disease may also be caused by trauma with a classic lesion involving a horse rearing over backwards and striking its poll, with forces transmitted through the basisphenoid bone creating a fracture over the region of the facial and vestibular nuclei in the brainstem. Other causes of central vestibular disease include equine herpes virus myelitis, cauda equina neuritis and equine protozoal myelitis.

Horner's syndrome

Signs

Horner's syndrome appears in the horse as in all species as an asymmetrical-appearing eye due to miosis (constricted pupil), enophthalmus (a sunken globe), and protruding nictitating membrane (3rd eyelid). However, unique to the horse, Horner's syndrome also causes sweating (and increased blood flow) of head and face due to interruption of sympathetic supply to blood vessels and sweat glands of the head.



Figure 7.19 Horner's syndrome in a horse; note the prominent sweating above the site of the lesion (a perivascular injection).

The extent and localisation of clinical signs enable the localisation of the lesion and hence information about aetiology and prognosis. The sympathetic nerves supplying the head originate at T1–T3 and travel via the cervicothoracic, middle and cranial cervical ganglions to the head where postganglionic fibres innervate the eyes and face.

A common cause is the perivascular injection of substances in the left jugular groove and the subsequent inflammatory reaction affecting the sympathetic trunk as it travels deep to the jugular vein (Figure 7.19). If Horner's syndrome is evident from a perivascular injection reaction, the larynx should be endoscoped as the recurrent laryngeal nerve runs with the vagosympathetic trunk and could result in development of laryngeal hemiplegia or roaring.

7.8.3 Spinal cord disease

Gait, posture and reflex abnormalities in horses with spinal lesions

With increasing compression more functional deficits occur:

- Loss of proprioception
- Motor weakness
- Loss of sensory perception, touch
- Loss of pain

Clinical signs

Clinical signs include ataxia, postural deficits, paresis, hypermetria, hypometria with recumbency (tetraparesis) in severe lesions.

Grade/classification of deficits

1. (+) Subtle – deficits just barely detected at normal gait, occur during backing, stopping, turning, swaying, neck extension etc

2. (++) Mild – detected at normal gait, exaggerated by above manoeuvres
3. (+++) Moderate – prominent at normal gait, tend to buckle and fall with above techniques
4. (+++++) Severe – tripping or falling spontaneously at normal gait to complete paralysis. (Some add a Grade 5, which is recumbent)

Note: The forelimbs are often a grade less than the hind limbs with focal cervical spinal cord and brainstem lesions.

Localisation – cervical, thoracic, lumbosacral, cauda equina region.

Thoracic limb signs with cervical lesions may need careful evaluation to detect, particularly with chronic lesions.

Spinal cord diseases

In Australia and the UK, the majority of equine spine lesions are the result of trauma or cervical vertebral malformation (CVM). In the USA, equine protozoal myelitis (EPM) is also very common. Other diseases include congenital malformations (e.g. atlanto-occipitalaxial malformation in Arabians) infection, degenerative myelo(encephalo)pathies and neoplasia (Mayhew 1999).

Spinal trauma

Spinal trauma usually presents with a history of sudden onset of ataxia or recumbency, and only sometimes is the incident observed. If there is not an unstable fracture, there may be no progression, frequently improvement, although later progression due to callus formation may occur. Trauma may play a role in the acute exacerbation of CVM; i.e. the horse is already neurologically abnormal, more likely to fall, and only after acute exacerbation do the owners notice the abnormalities.

Sites of predilection (Mayhew 1989b):

- Occipitoatlantoaxial region
- Caudal cervical
- Mid-back – but requires considerable force – usually unstable fracture, paraplegia and recumbency (dog sitting)

Signs of spinal cord disease occur, that vary from ataxia and paresis to recumbency. In horses there is no or short-lived spinal shock or Schiff–Sherrington phenomenon.

Management

Management includes anti-inflammatory therapy (steroidal), surgical therapy may be attempted, box rest if standing, and always involving intensive nursing care if recumbent (Figure 7.20).

Prognosis

Prognosis is guarded, but remarkable recoveries have been made. Poor prognosis with luxations, unstable fractures, poor initial response to treatment (24–48 hours) or continued recumbency.



Figure 7.20 A horse with a traumatic thoracic spinal cord lesion being managed using a sling. Splinting of the hindlimbs and use of bales of straw and shavings were also used in an attempt to support the mare.

Cervical vertebral malformation (CVM)

This syndrome has been termed wobbler syndrome in horses but this is a non-specific term as others use the term to describe any horse with spinal cord ataxia.

CVM occurs in large breed horses especially Thoroughbreds and Warmbloods. Aetiology is multifactorial but includes congenital, familial, dietary and managerial (including exercise) factors.

CVM affects young horses, large for their age and breed. CVM foals tended to be heavier and taller during some time intervals than the normal foals, but there were no significant differences between the two groups in any of the skeletal growth measurements. Body weight gain was faster in CVM foals from 31 to 60 days (Ruff *et al.* 1993). Nutrition is an important factor in young growing horses and the appearance of CVM is related to developmental orthopaedic disease (DOD) (Chapter 3). Signs of spinal cord disease occur with progressive ataxia of all four limbs, most notable in the pelvic limbs (or only in pelvic limbs in chronic cases). There may be acute exacerbation after an injury, but usually slow onset of increasing clumsiness. Note neck pain is rarely seen.

Diagnosis

Veterinary diagnostic work-up typically includes lesion localisation and plain radiography. Plain radiography may be all that is required in diagnosis. Lesions may include: stenosis of the vertebral canal (dynamic or static); abnormal articular processes; subluxation of vertebrae on flexion (C2–C6) or extension (C6–T1) of the neck; enlarged vertebral physal growth regions; overriding of the vertebral arch and next caudal vertebral body causing dynamic stenosis during flexion or extension; or proliferation of articular and periarticular soft tissues. Minimum sagittal diameters (MSD)

are used as a guide to diagnosis and represent the narrowest diameter from the dorsal aspect of the vertebral body to the ventral border of the dorsal laminae. Correcting for magnification errors MSD is divided by the maximal width of the cranial aspect of the vertebral body to give the sagittal ratio. There are published tables for Thoroughbreds. Myelography may be useful in demonstrating dynamic lesions and may be required to confirm the diagnosis.

Management

In the horse, surgery can be performed but is costly and may only improve the horse on average one grade. The fundamental problem is that a horse with any degree of ataxia should not be ridden.

Early detection in young foals (6 months) and dietary restrictions has resulted in resolution of ataxia and successful racing careers (Donawick *et al.* 1993). Once disease is advanced, prognosis for suitable riding animal is poor.

Cauda equina syndrome

Cauda equina syndrome (CES) describes lesions involving the sacrococcygeal spinal cord segments, cauda equina, sacral plexus and peripheral nerves to the bladder, rectum, anus, tail and perineum. CES may or may not involve lumbosacral nerve roots to the lumbosacral plexus producing gait abnormalities.

Signs

Signs are predominantly LMN of the tail, anus and perineal region presenting as various degrees of hypotonia, hyporeflexia and hypalgesia of the tail, anus and perineal region. There may be urinary bladder paresis, rectal dilation, penile prolapse.

There may also be LMN weakness and paresis of pelvic limbs tending to result in a 'dog sitting' posture or recumbency. In these cases it can be difficult to distinguish from UMN disease with urinary retention and secondary contusion of tail, anus from recumbency.

Causes

Causes of CES include:

- Trauma, e.g. sacrococcygeal fracture and luxation, avulsion of the cauda equina. There may be a history of rearing and falling on rump
- Infections, inflammatory, immune, e.g. polyneuritis equi, equine herpes virus 1 (EHV 1)
- Toxicity, e.g. *Sorghum* spp. ingestion
- Congenital anomalies
- Neoplasia

EHV 1 myeloencephalopathy is an uncommon manifestation of EHV1 infection. The neurological form may occur sporadically or as an outbreak. There may be a recent history of abortion or respiratory disease (other manifestations of EHV1 infection). Affected horses develop sudden onset signs of CES including ataxia, paresis and urinary

incontinence, with early stabilisation of signs. Some animals can be maintained with good nursing care including bladder catheterisation, faecal evacuation and physiotherapy during the recovery. Recovery is usually over days to weeks, although full recovery may take over 1 year. Prognosis is better if the animal is not recumbent owing to the complications of recumbency.

Equine protozoal myelitis

This disease is predominantly one of North America, and is exotic to Australia and the UK as these countries lack the definitive hosts (small carnivores) required to complete the life cycle of the parasite. It is caused by a protozoan parasite called *Sarcocystis neurona*, where the horse is an aberrant host and the disease can affect any part of CNS (UMN, LMN), including causing selective focal/multifocal CNS lesions. A hallmark of the parasitic disease is asymmetric lesions, or a combination of UMN and LMN signs. The disease is able to be confirmed by CSF analysis and usually responds to prolonged antibiotic therapy and is ideal for physiotherapy intervention once the protozoa have been inactivated.

7.8.4 Neuromuscular disease

There is a lot of overlap in the clinical presentation of muscle disease of primary muscular origin and of neurological (LMN) origin. Abnormalities in muscle contraction including tetany and weakness can also occur as part of an underlying metabolic problem. However, differentiation is possible with a thorough veterinary diagnostic work-up.

Signs

- Weakness – which may be localised or diffuse
 - (a) Diffuse weakness can be episodic
 - (b) Severe diffuse weakness may result in recumbency
- Increased tone, e.g. myotonia, tetany, spasm, synchronous diaphragmatic flutter
- Atrophy
- Abnormal gait due to:
 - (a) Decreased muscle contraction/range of movement
 - (b) Increased muscle contraction, e.g. stringhalt type gaits with stringhalt and equine motor neurone disease (EMND)
- Pain

Equine neuromuscular disorders may be grouped according to the major clinical presentations. These are localised weakness, abnormal hind limb gait, diffuse weakness and generalised tetany.

Localised weakness

Peripheral neuropathies are the most common cause of localised weakness. However, localised weakness and atrophy can also occur as a result of inflammatory myositis and have been described in the neck (Barrott *et al.* 2004), back (Sponseller *et al.* 2005) and masseter muscles (Lindsay *et al.*

1989). These are diagnosed by muscle biopsy. Localised weakness might also need differentiating from severe limb orthopaedic disease, e.g. septic arthritis or fracture.

Peripheral neuropathies

Peripheral neuropathies are usually traumatic in origin, although in horses they commonly occur after prolonged general anaesthesia with poor positioning and long surgical times (associated with a type of compartmental syndrome, Lindsay *et al.* 1989). Obturator nerve paralysis may occur after foaling. Obturator nerve paralysis is far more common in cattle where oversize calves cause dystocia and prolonged pressure while the calf is in the pelvic canal damages the obturator nerve. This is frequently termed ‘calving paralysis’. Sciatic nerve injury has occurred, especially in foals, due to accidental inappropriate injection of irritant drugs over the nerve.

In the hind limb, classically, femoral nerve injury results in lack of stifle extension. Obturator nerve injury results in loss of adductor function. Sciatic nerve injury results in knuckling of the fetlock, poor stifle flexion.

In the forelimb, damage to the brachial plexus will result in loss of extension of the entire limb (Figure 7.21). Branches of the brachial plexus include the suprascapular nerve, radial nerve, axillary nerve and musculocutaneous nerve. Injury over the point of the shoulder can specifically



Figure 7.21 Horse with brachial plexus injury of 4 weeks' duration. Note the marked atrophy. This horse fully recovered with extensive teamwork rehabilitation by a veterinarian and physiotherapist.

damage the suprascapular nerve resulting in atrophy and dysfunction of the supraspinatus and infraspinatus muscles or ‘Sweeney’. Radial nerve paralysis results in lack of elbow extension as well as the lower limb, and brachial plexus injury results in complete inability to extend the limb including the shoulder.

Diagnosis of peripheral nerve injuries relies on accurate localisation of clinical signs and is supported/monitored by mapping areas of reduced skin sensation or use of an electromyogram. However, skin dermatomes in the horse are difficult to detect as areas are smaller than in man and inconsistent in response (Blythe 1997b).

Treatment

Medical treatment initially includes anti-inflammatory medication. It is vital to ensure the contralateral limb is supported in horses that are non-weight bearing. Splinting may be indicated to assist in weight bearing. Surgery may be indicated especially in chronic, adhered suprascapular nerve paralysis. Physiotherapy is indicated (Chapter 14).

Abnormal hindlimb gait

Shivering

Affected horses have muscle fasciculation that give the affected horse the appearance of shivering, usually concentrated in the hindlimb area. The horse will lift the affected hindlimbs, holding each up or shifting, while fasciculation occurs, with the tail often held straight out, fasciculation may also occur along the trunk.

Shivering can be incited with backing, lifting one limb, and excitement. It most often occurs in draft breeds and their crosses, but any large horse may be affected. The condition may be progressive in draft breeds. However, mild cases can remain useful athletes (doesn't occur during normal forward movement) although specifically looked for in predisposed breeds at shows, e.g. Shires and Clydesdales. Diagnosis is based on clinical signs. The cause is unknown, although there has been a link to the metabolic muscle disorder equine polysaccharide storage myopathy (EPSM) so muscle biopsy is warranted.

Stringhalt

There are two forms of stringhalt – classic and Australian or pasture associated stringhalt.

Signs

Signs involve excessive flexion of one (classic) or both (Australian) hindlimbs and signs are usually worse when backing and turning. Classic stringhalt frequently occurs after an injury (probably a musculoskeletal disease) and may respond to surgical therapy (tenotomy and removal of about 10 cm of the lateral digital extensor tendon).

Australian stringhalt is a neurological disease due to a toxin present on pastures. It frequently occurs as an outbreak and has been reported in Australia, New Zealand and

the USA. The lesion is a peripheral neuropathy (axonopathy) affecting branches of the sciatic and also the recurrent laryngeal nerve. It has been associated with particular pasture species, especially *Hypochaeris radicata*. Although sweet pea intoxication (lathyrism) appears identical.

It is important to differentiate stringhalt from ossifying myopathy (more of a slapping gait see below) and aortic-iliac thrombosis (post-exercise stamping of feet and abnormal hindlimb gait due to thrombosis of the iliac arteries interrupting the blood supply to hind limbs – probably similar to ‘pins and needles’ in man). The residual spastic gait in horses with equine motor neurone disease may resemble mild stringhalt.

Prognosis

‘Australian stringhalt’ often results in marked generalised atrophy and prolonged recovery (up to 18 months) but eventual improvement usually occurs. Classic stringhalt may result in permanent gait abnormalities.

Diffuse weakness

There are many causes of diffuse weakness including:

- Neurological disease, e.g. equine motor neurone disease
- Metabolic disease, e.g. hyperkalaemic periodic paralysis, equine polysaccharide storage myopathy
- Neuromuscular toxins, e.g. botulism, ionophore toxicity

The role of the physiotherapist in these conditions is in management of recumbency and rehabilitation during recovery (Nout & Reed 2005). Some examples of diseases are presented below.

Equine motor neurone disease (EMND)

Acquired neurodegenerative disease is associated with depletion of antioxidants. It affects adult horses (mean 9 years) and there have been worldwide reports. The condition is probably under-diagnosed in Australia. Initial reports tended to occur in horses deprived of green forage, but it has also been reported to occur in pastured horses (McGorum *et al.* 2006). EMND is often seen in stabled heavily grain-fed show Quarterhorses. Horses may have a history of little or no access to pasture plus poor quality grass hay (usually also high concentrate diet).

Signs

Acute onset of trembling, excessive recumbency, shifting of weight, reluctance to stand still/confined and muscle atrophy (especially hindquarters), elevation of tail head (Figure 7.22). Appetite normal to ravenous. No ataxia. Can develop marked spasticity or stringhalt-like gait abnormalities.

Treatment

Treatment is usually attempted using high doses of Vitamin E – 5000–7000 IU/day as well as dietary addition of green pasture or alfalfa (lucerne) hay vs. grass hay.



Figure 7.22 Horse with equine motor neurone disease (EMND) demonstrating diffuse muscle atrophy and generalised weakness, demonstrated by his narrow based stance, elevated tail head and low head carriage.

Prognosis

The prognosis is poor. Cases may either continue to progress resulting in euthanasia, improve after 3–6 weeks (but are at risk of relapse) or stabilise but remain emaciated and develop further gait abnormalities, e.g. stringhalt, that eventually result in euthanasia. In this third group there have been no reports of physiotherapy intervention and this may be an area for involvement.

Botulism

Botulism is usually the result of ingestion of pre-formed toxin (usually A, B or C) of the bacterium *Clostridium botulinum* that interferes with transmission of nerve impulses at the neuromuscular junction of both striated and smooth muscles by stopping the release of acetylcholine at the neuromuscular junction and other cholinergic synapses (including the autonomic nervous system).

Usually associated with rotted vegetation, e.g. poor quality silage, or contamination of feed with a dead animal. A common source of the toxin for horses is usually contaminated feed (big bales) or grass/corn silage. Botulism can sometimes be associated with multiplication and elaboration of toxin in an animal, especially in foals (toxicoinfectious botulism) where it multiplies in the gut and sometimes in wounds if appropriate anaerobic conditions are present.

Clinical signs

Clinical signs include dysphagia, weakness, recumbency, faecal and urinary incontinence. It is typically an ascending paralysis with dysphagia a prominent early sign, but outbreaks have involved horses with paralysis and not dysphagia.

Signs include abrupt progressive onset flaccid paralysis of skeletal muscle (hindlimb paresis progressing to tetraplegia), symmetrical weakness (muscle tremors), tongue

weakness, protrusion, dysphagia (not always present), eventual recumbency and dyspnoea.

Treatment

Treatment involves supportive care and antitoxin where available.

Prognosis

Prognosis is poor if there is rapid onset tetraplegia. Management of mild cases may be rewarding, but if recumbent >24 hours, prognosis is poor. Manage dysphagia, incontinence, and secondary complications. Physiotherapy may well reduce recovery times.

Post-anaesthetic myoneuropathy

This occurs in fit performance horses that develop prolonged recumbency, tetra, para or monoparesis after general anaesthesia. There are compartmental pressure elevations, ischaemia of muscle and pressure neuropathy.

Treatment includes rest with or without slinging, medical support (fluids, anti-inflammatory medication, dantrolene). While recovery is good with more localised lesions, generalised myoneuropathy has a poor prognosis. There is a role for physiotherapy and undetermined strategies, but intervention would need to be aggressive.

Other syndromes appear similarly, including post-anaesthetic myaesthetic syndrome and post-anaesthetic haemorrhagic myelopathy.

Hyperkalaemic periodic paralysis

This occurs in young adult Quarterhorses of 'Impressive' bloodlines (Impressive was the name of the stallion the genetics were all traced back to) that have an inborn defect in potassium (K⁺) homeostasis. Affected horses have episodic trembling, sweating and weakness to recumbency, often associated with exercise or stress. The disease is an autosomal dominant inherited disease, with an identifiable genetic marker, so breeders can avoid the disease. Cases can be managed with long-term drug therapy.

Tetany

Tetanus

Horses are highly susceptible to generalised tetanus caused by the bacterium *Clostridium tetani*. Spores of this organism are found very commonly in soil and gastrointestinal bacteria. The usual route of infection is via penetrating wounds that allow bacterial spores to enter and multiply in an anaerobic environment. Multiplication results in release of the tetanus toxins, which are transported via motor neurone axons to the spinal cord.

Clinical signs

Clinical signs include an elevated tail head and stiff gait. The horse appears anxious with their ears back, eyelids wide open, prominent nictitating membranes, nostrils flared, head



Figure 7.23 Horse with tetanus following a small puncture wound above the right carpus.

extended. A classic sign is 'lock jaw' and horses may drool, and can't swallow. Severely affected horses will become recumbent with relatively increased extensor tone (Figure 7.23).

Differentiate from fasciculations of a weak animal, shivering, seizures or congenital myotonia. In horses, hypocalcaemia can present similarly to tetanus with stiffness, muscle fasciculation, sweating and anxiety.

Treatment

Treatment of tetanus includes support: quiet, dark, low stimulation room; hydration, good footing, deep bedding (secondary ulcers), elimination of the original infection by careful wound management, antibiotics, antitoxin and sedation. Mortality for tetanus in horses is high, but it is a preventable disease with vaccination highly effective and inexpensive.

7.9 Intrinsic muscle disease

Differentials for muscle disease include:

- Inflammation/infection (bacterial, viral, protozoal, traumatic, immune mediated)
- Metabolic, e.g. polysaccharide storage myopathy, hypocalcaemia, hypokalaemia and exhaustion
- Nutritional, e.g. vitamin E deficiency
- Genetic, e.g. myotonia and calcium channel disorders

7.9.1 Laboratory diagnosis of muscle disease

Muscle damage will result in the release of muscle enzymes and the muscle protein myoglobin into the blood (measured in the serum or plasma) and myoglobin is rapidly filtered by the kidneys and appears in the urine (making it appear dark red). The main measured muscle enzymes are creatine kinase (CK) and aspartate amino transferase (AST). CK and AST have very different kinetics, which need to be taken into consideration when diagnosing the extent and time frame of muscle damage. CK rises rapidly, peaking at around 12 hours and will be back to normal in 2 days,

while AST takes up to 24 hours to peak and several days to return to baseline. CK tends to be used for acute monitoring of muscle enzymes and AST for evaluation of changes over a longer period. While CK is quite specific for muscle, AST is also a liver enzyme in horses.

Muscle biopsy

Taking a biopsy is simple and only mildly invasive, but can yield vital information. The biopsy is examined for inflammatory cells, regeneration, degeneration and central nuclei. It is quite straightforward to make many diagnoses, e.g. neurogenic atrophy, myositis and polysaccharide storage myopathy, although biopsies from some horses with significant muscle disease may not reveal any information.

7.9.2 Delayed onset muscle soreness (DOMS) and muscle strain injury

DOMS is virtually unreported in the veterinary literature but is likely to occur in animals, especially animals used for athletic activities as it does in man (Cheung *et al.* 2003; Connolly *et al.* 2003). There have been reports of unexplained elevations of muscle enzymes of horses starting to be trained for the first time that were hypothesised to be due to DOMS (Kirby & McGowan 2004).

Muscle strain injury occurs as a result of overstretching of muscle leading to disruption of fibres, which can subsequently lead to inflammation and healing with fibrosis. While it certainly can be a cause of lameness in both horses and dogs, it can be difficult to diagnose or is sometimes even overlooked as a cause of lameness. Muscle strain injury is quite common in athletic horses and dogs. It occurs particularly in those muscles that cross two or more joints, especially near the musculotendinous junction but also at the origin and insertion of the muscle (Fitch *et al.* 1997, Steiss 2002).

Based on the system in humans there are four grades:

1. Tearing of a few fibres
2. Pain
3. Local spasm
4. Complete muscle rupture

Recovery is rapid with low-grade injuries, but fibrous tissue may predispose to reinjury or contracture.

Diagnosis

Muscle injury should be a differential as a cause of lameness. Palpation may reveal pain or even a defect. Ultrasonography is probably the most appropriate imaging technique for muscle injuries themselves, although if the injury occurs at the origin or insertion of a muscle, radiography is important to determine if there has been an avulsion fracture at the site. Nuclear scintigraphy, other imaging techniques may reveal the inflammation.

Common sites of muscle strain injuries in the dog are:

- Forelimb: rhomboideus, serratus ventralis, pectorals, triceps, biceps and flexor carpi ulnaris
- Hindlimb: iliopsoas, tensor fascia lata, sartorius, pectineus, gracilis and Achilles mechanism.

While in the horse, common sites include the gluteal muscles, especially with hill work (e.g. rapid acceleration on an inclined treadmill) and lumbar muscles. Injury to the serratus ventralis muscles is no longer common (carriage horse injury from slippery roads) but can occur. In both the dog and horse there are probably many more that are as yet poorly recognised.

Treatment of muscle strain injury

Low-grade injuries – conservative physiotherapy using the principles you would use in man and see also Chapter 13. High-grade injuries may be amenable to surgical treatment (dogs): including surgical debridement, repair or tenomyectomy.

7.9.3 Ossifying/fibrotic myopathies

Dogs

Semitendinosus fibrotic myopathy

Occurs in German Shepherds, uncommon, poorly responsive to surgery. They present with a characteristic gait pattern due to the tethering of the forward phase of limb flight, similar to the horse.

Myositis ossificans

Usually secondary to trauma (there have been some reported cases in Dobermans secondary to clotting disorders). Sites of predilection include the hip (Dobermans), shoulder, quadriceps and cervical regions; and it occurs in large, middle-aged, active dogs and presents as lameness from mechanical interference. Surgical debulking is the preferred treatment.

Horses

Fibrotic or ossifying myopathy

Semitendinosus, semimembranosus, biceps femoris or biceps brachii muscles may be involved. More common in Quarterhorses probably as a result of repeated muscle injury. Presents with a characteristic gait that involves a shortened cranial phase, where the limb is jerked back before being put to the ground. Treatment is by surgical correction – tenotomy of tibial insertion of semitendinosus.

7.9.4 Contractures

Dogs are the main species affected by contractures and they are not well recognised in horses.

Infraspinatous contracture

Occurs in large, active, middle-aged dogs, tethering of normal shoulder motion, circumduction of the limb. There is palpable atrophy of the muscle. Proposed to be secondary

to injury causing fibrosis and functional shortening of the muscle. Treatment is surgical – infrapinatus tenotomy.

Quadriceps contracture

Occurs in actively growing dogs <6 months old following fracture of distal femur with voluntary or enforced immobilisation. The result is prolonged hyperextension of stifle muscle contracture and adhesions. It is best prevented, but if occurs, early recognition is essential as prognosis once advanced is poor. Physiotherapy responses have been good (Chapter 13).

Gracilis contracture

Occurs in active, middle-aged, large dogs, secondary to injury like infrapinatus contracture. Affected dogs can maintain normal function but have a characteristic jerky gait and shortened stride. Affected dogs tend to have guarded long-term outlook owing to recurrence after surgery.

7.9.5 Equine rhabdomyolysis syndrome (ERS or tying-up)

Recurrent tying-up or ERS is a common disability in horses causing signs ranging from a stiff gait and muscle cramping to reluctance to move or recumbency. It has been referred to using a variety of synonyms including ‘Monday morning disease’, setfast, azoturia, tying-up and exertional rhabdomyolysis. In the UK it is most commonly referred to as equine rhabdomyolysis syndrome or ERS. It is common, affecting up to 6–7% of racing Thoroughbreds, polo horses and competition horses, i.e. about 1 in 15 horses (McGowan *et al.* 2002a, 2002b, Upjohn *et al.* 2005). ERS may occur sporadically or be recurrent. The recurrent form is the most problematic, and requires accurate diagnosis and management.

Two causes for ERS have been found:

1. EPSM or polysaccharide storage myopathy. This form occurs in Quarterhorses (Valberg *et al.* 1992), Warmbloods, draft breeds and draft breed crosses – including cobs and ponies (Valentine *et al.* 2000, 2001a).
2. A calcium channel disorder (similar to malignant hyperthermia in pigs and man). Affected horses have abnormal regulation of muscle contraction owing to a defect in intracellular calcium regulation. This form is found in Thoroughbreds as a distinct disorder from EPSM (Lentz *et al.* 2002).

Both these forms have been shown to have a familial basis, although the genetics of the disorders are still being researched. However, since the underlying causes of ERS are both likely to be inherited and prevalent in the population, the most crucial factor in managing the condition is the avoidance of risk factors. Many of the so-called ‘causes’ of episodes of tying-up are actually risk factors for precipitating an episode of tying-up. These include change in exercise pattern (e.g. several days off and then resumption of normal work), increased carbohydrate in diet, stress,

oestrus and insufficient electrolyte supplementation. These risk factors are not the same for all horses and so recommendations cannot be assumed to be beneficial for all.

For example, in polo horses, episodes tend to occur actually during a chukka (competition), are severe, and tend to be associated with excitable temperament horses exercising too hard for their level of fitness. There is no sex predisposition, no age predisposition and no relationship to dietary carbohydrates. In eventers, there is also no sex or age predisposition, nor is there a relationship to temperament or carbohydrates in the diet. The only risk factor was being confined for most or all of the day. In Thoroughbred racehorses (both flat racehorses and National Hunt racehorses) there is a sex predisposition (increased risk for fillies), but the association with temperament only occurs in the 2-year-olds – i.e. a 2-year-old filly with a nervous or excitable temperament is at great risk for tying-up, but above 2 years, there is no effect of temperament.

Diagnosis

Diagnosis of recurrent tying-up is quite straightforward and is primarily based on history, clinical signs and laboratory findings. A history of more than one episode of tying-up is a good indication that the condition is recurrent, but should be combined with a thorough management history and serum muscle enzyme results. It is important to realise that a single episode may be one of many more to come and that subclinical episodes can occur.

Some horses may not show overt tying-up and especially in young horses being backed or broken in with EPSM there may be behavioural problems such as unwillingness to go forward, bucking or rearing instead of classic tying-up signs of hard contracted muscle, sweating, pain and unwillingness to walk.

Differentials include lameness, aortoiliac femoral thrombosis, colic (many horses paw and sweat because of the pain and look as if they are having colic), laminitis, pleuritis (both of these cause the horse to be reluctant to walk, and laminitis in particular can cause such pain that all the muscles are hard and tense mimicking a horse that has tied up).

In subclinical or chronic/recurrent cases an exercise test to detect elevations in CK can confirm the problem, following collection of a pre-test blood sample for measurement of serum activities of CK and AST. The horse is lunged at a trot, or worked on the track at similar exercise intensity for 15–20 minutes, and 4 hours after exercise CK is collected. Many horses with recurrent ERS will show at least a doubling of CK, indicating ongoing recurrence.

A muscle biopsy is useful in differentiating EPSM as a specific cause for ERS. Knowing the specific cause for ERS can help in determining heritability and prognosis. Also, while both ERS and EPSM are managed principally by dietary therapy, ERS in light bred horses, not caused by EPSM, and may be amenable to medical therapy. ERS in these horses has been shown to be a calcium channel

disorder so drugs that affect muscle calcium regulation, e.g. phenytoin may be used. Muscle changes are often apparent in horses with recurrent disease, even without clinical signs. Interestingly, and important when considering physiotherapy treatment, despite the severity of signs in episodes of azoturia/tying-up, fibrosis is rarely seen (as opposed to neurogenic conditions).

Treatment of ERS

The acute case/episode should be examined by a veterinary surgeon because of the risk of renal disease or complications. Treatment might include analgesia (NSAIDs) and fluid and electrolyte therapy.

The mainstay of treatment of the chronic or recurrent case is threefold:

1. *Diet.* Level of carbohydrate should be relative to exercise and weight and minimised by use of other energy sources, e.g. oil or fat. A high fat diet is essential, even on 100% roughage-fed horses. Fat should constitute 20% caloric intake (Chapter 3).
2. *Exercise.* It is important to allow the horse to move and not be confined. Therefore maximise turnout (although be careful of excessively rich pasture as it may have the same effect as excessive carbohydrates). Resume exercise early – do not give prolonged periods of box rest following an episode – resume when the horse is able to walk freely without pain. Be careful of over-reliance on muscle enzymes, as prolonged elevations are likely for 4 months after dietary intervention (Valentine *et al.* 2001b). In some horses, restricted low intensity exercise (excitable horses held back on a tight rein) can be more likely to induce attacks so moderate intensity exercise on a loose rein, with minimal warm-up, can be beneficial. Ensure exercise is consistent throughout the week – ideally no days of rest.
3. *Drug therapy* can be employed in Thoroughbreds on a temporary basis to get horses back into regular training.

Prognosis

Overall prognosis is good and many horses may still continue an athletic career with appropriate management, although complete resolution of problem is unlikely and provided owners are well informed and can deal with that concern a good outcome can be achieved.

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8

Physiotherapy assessment for animals

Lesley Goff and Tracy Crook

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8.1 Introduction

The aim of this chapter is to provide the animal physiotherapist with guidelines for comprehensive physiotherapy assessment of the dog and horse. Emphasis lies with communication with the veterinarian, clinical reasoning, accurate history taking and physical assessment to guide the physiotherapist in selection of treatment. The chapter is designed to be used in conjunction with other chapters in this text such as orthopaedics, neurology, biomechanics and manual therapy.

In many countries, physiotherapists have been granted professional autonomy, and as such they are able to diagnose and treat human patients as primary contact practitioners. Yet most veterinary acts, for example the Veterinary Surgeons Act 1966 in the United Kingdom, currently preclude physiotherapists from being primary contact practitioners when providing physiotherapy for animals. The UK act clearly states that: ‘only veterinary surgeons registered with the Royal College of Veterinary Surgeons have the right to practice veterinary surgery’. Veterinary surgery is defined in the act as:

‘The art and science of veterinary surgery and medicine and, without prejudice to the generality of the foregoing, shall be taken to include:

- The diagnosis of diseases in, and injuries to animals, including tests performed upon them for diagnostic purposes.
- The giving of advice based on such a diagnosis.
- The medical and surgical treatment of animals.
- The performance of surgical operations on animals.’

It is essential that animal/veterinary physiotherapists obtain veterinary diagnosis of the animal’s condition

requiring treatment from the referring veterinarian, before carrying out physiotherapy assessment and devising a physiotherapy treatment plan. Good communication between physiotherapist and veterinarians is paramount, not only to comply with legislation but to maintain high standards of professional conduct in animal physiotherapy.

Physiotherapy assessment of the animal involves the integration of skills, which include:

- Knowledge of anatomy, functional anatomy and biomechanics
- The ability to observe movement and analyse movement patterns of the musculoskeletal system
- Animal husbandry skills
- Sound clinical reasoning based on the latest research evidence
- Ability to communicate with and educate the owner/carer
- Ability to communicate with veterinarians

Historically, veterinarians’ initial diagnosis of a condition, injury or disease in the animal has focused on both the anatomical location and the specific pathology involved, that is, pathoanatomical diagnosis. While a pathoanatomical diagnosis is ideal, it is not always possible to establish a pathoanatomical diagnosis prior to initialising patient management based on the identified problems. Physiotherapy treatment decisions can be then be based on the patient’s signs and presenting movement disorders.

Pathoanatomical diagnoses may be difficult in animals where the conditions affecting an animal’s performance are either non-specific in origin, or are difficult to diagnose with traditional veterinary diagnosis. For example, there is much written about the difficulty in diagnosing conditions such as chronic sacroiliac disease and temporomandibular disorders in the horse, due to the non-specific nature of

clinical signs and in the case of the former, difficulty diagnosing with traditional veterinary diagnostic modalities (Jeffcott *et al.* 1985, Moll & May 2002). An example of a condition of non-specific origin in a horse, for which physiotherapy may be of benefit, is a restriction of cervical spine mobility affecting the horse's ability to flex laterally on a small circle. A mechanical, musculoskeletal condition such as this in a horse may show no obvious lameness. A work-up involving lameness assessment, joint blocks, or imaging techniques readily available to veterinarians will be unlikely to reveal pathology. Imaging techniques available to most veterinarians, for example ultrasound or radiography, do not show the presence of movement dysfunction. The veterinarian may instead classify the cervical restriction as part of the 'problem list'. In the above case, a physiotherapy assessment involving movement analysis, physical examination and the employment of sound clinical reasoning, should be able to ascertain the cause of the mechanical dysfunction, and lead to development of a treatment plan for restoring mechanical function.

There are many instances where veterinarian- and physiotherapist-combined assessment, will deliver the best outcome for the animal patient. Veterinary expertise can be utilised to discover the source of many conditions and pathologies in animals, and physiotherapy expertise can be utilised to assess any secondary or associated mechanical dysfunctions. In some cases veterinary assessment and management will need to be instigated before physiotherapy assessment and treatment is warranted. In cases where there are contraindications or precautions to a physiotherapy assessment, or where a new development occurs within the presenting condition, the physiotherapist should discuss with the referring veterinarian.

8.2 Clinical reasoning

Clinical reasoning forms the basis for the assessment and the ongoing reassessment, which in turn is essential for, and concurrent with, delivery of the most effective treatment. Clinical reasoning is an essential part of each step of the animal physiotherapy assessment. It is influenced by the clinician's knowledge base, beliefs or values and skills associated with clinical practice (Refshauge & Latimer 1995). Unlike a human patient, an animal patient cannot describe to you their resting symptoms, or tell you assessment is causing an increase in their symptoms. An animal can only show signs of problems. Thus clinical reasoning is an essential skill for delivering animal physiotherapy safely.

Clinical reasoning begins with the clinician's interpretation of clues from the patient. These initial clues help the clinician formulate a preliminary working hypothesis. The working hypothesis should be considered throughout the rest of the subjective and physical examination, as well as during ongoing patient management (Jones 1994).

The following is a brief example of clinical reasoning process:

A horse presents with lameness in the right hindlimb, which is apparent when trotted on a right-hand circle in deep sand. Lameness is absent on the straight line or on hard surface. Lameness was unaffected by nerve blocking the lower limb or tibial and peroneal nerves by the referring veterinarian. The clinician forms a working hypothesis by making a note of the possible pain-producing structures (e.g. coxofemoral joint, sacroiliac joint, neuromeningeal structures, pelvic limb soft tissues). Further information is then sought through the subjective and physical examination with the working hypothesis in mind (Jones 1994). Information may include specific questioning of the owner (if it has not already been carried out) such as previous episodes of lameness, horse's working history and performance, veterinary investigations, medications and previous trauma. This subjective information may then guide the clinician 'where to start' with the physical examination.

The history directs the physiotherapist towards the starting point of the physical examination, which in the above case may include sacroiliac joint palpation and functional tests, including neural tension tests, to help narrow down the area or structure from where the signs are most likely to arise. It is important however, to realise that the physical examination is not a routine series of tests (Jones 1994). Whilst it is useful to have a systematic manner in which to carry out assessment, the physical tests should be an extension of the hypothesis testing performed through the subjective examination. If the history has provided the physiotherapist no working hypothesis, however, then it is essential to test and 'clear' all possible structures from contribution to the signs.

Hypotheses regarding precautions and contraindications (Chapter 9) to physical examination and treatment will guide the extent to which physical examination and initial treatment can be performed without risk of aggravation of signs (Jones 1994). The physical examination is used to test the hypothesis regarding potential sources of signs and contributing factors in such a way that each structure that could be implicated is specifically examined. In summary, the clinical reasoning process guides the examination and the treatment.

A clinical reasoning form, which can be used in clinical practice, is shown in Figure 8.1, along with further notes on clinical reasoning.

8.2.1 The assessment

Although the physical examination is not a routine 'recipe' of tests, it is useful to perform the physiotherapy examination in a systematic manner, to optimise efficiency of the process (Refshauge & Latimer 1995). Following a routine of examination also minimises the physiotherapist omitting crucial components of the examination, especially where there are

Immediately following the history (BEFORE physical examination)

1. List, in order of importance, three structures most likely to be contributing to presenting signs and symptoms. Even though there is unlikely to be ONE joint, muscle, or other tissue solely responsible, you need to be identifying structures down to a high level of accuracy, so be specific (e.g. upper cervical vertebrae; left brachial plexus; right cranial cruciate ligament of the stifle; right peroneus tertius).

	Structure	Justify your reasoning (e.g. distribution of symptoms; history of specific trauma)
1		
2		
3		

If you wish to include more contributing structures, please justify.

2. In consideration of the breed and/or conformation of the animal, are there any precautions or contraindications, prior to going on with physical examination?

No:

Yes: a) Are you confident it is safe to proceed with FULL physical examination?
Y/N

b) Are there any veterinary or physiotherapy tests, which can be carried out to indicate it safe to proceed with FULL physical examination?
Y/N

If yes, list.

c) Is it safe to proceed with a modified examination? Justify

Following the physical examination, BEFORE treatment.

3. Do you agree with your hypothesis of structures most likely to be contributing to the presenting condition?
Yes

No.

If no, please revise list

	Structure	Justify your reasoning (e.g. specific gait pattern, specific pain response or motion restriction)
1		
2		
3		

Figure 8.1 A clinical reasoning form.

4. At this point in the assessment, are there any precautions or contraindications to treatment?

Y/N

If Yes: a) Do you feel safe to proceed with treatment? Please justify.

b) Do you feel a neurological test or further veterinary investigation is warranted? Justify.

5. If your answer to above question was No, do you feel it is appropriate to begin initial treatment today? Justify.

6. What treatment will you implement as initial treatment and why? (Include dosage)

7. What response/s do you expect from treatment and the dosage?

a) Immediate response

b) Response after 24 hours

8. Do you envisage an exercise programme as being appropriate for this patient? If so, at what stage will it be introduced?

9. When would you like to implement the second treatment – justify.

10. If the response is as hypothesised, explain what is likely to be implemented as second treatment, including dosage.

11. Can you predict the outcome from the second treatment? If no, please justify.

To be completed after 2nd treatment

12: Is your outcome as expected? If no, please give reasons.

Notes regarding use of the Clinical Reasoning Form

This clinical reasoning form has been prepared to assist the animal physiotherapist in the process of clinical reasoning, and to help the clinician avoid examination or treatment when it is NOT warranted, or when it will be detrimental to the animal. Knowing when to cease examination of the animal, or when it is not appropriate to provide a full physical examination, is even more important than knowing which examination procedures to include in your assessment. There may be dire consequences in performing a physiotherapy examination or treatment when contraindicated.

When treating animals, clinical reasoning is possibly more important a skill to possess, than when treating humans (not to detract from the importance of clinical reasoning in human physiotherapy). A human patient can tell you they are feeling worse (e.g. pain, paraesthesia) during assessment, or can alert you to their resting symptoms of numbness or dizziness, or unwellness, but an animal cannot.

It may be useful to make a copy of the form and take it with your patient records, so it can be filled in as you proceed with your examination – this may be more time-consuming initially, but worth while to avoid exacerbating an animal's condition and to improve your animal patient outcomes.

Figure 8.1 (Continued)

complications such as multiple or difficult signs reported by the animal patient's owner, or distractions such as behavioural issues with the animal patient at time of examination.

An example of a systematic approach to the assessment process is set out below, however, individual physiotherapists may find their own order of systematic approach to be more suitable. The order of the components of the physical assessment are interchangeable, depending on the direction clinical reasoning takes the clinician.

- History/subjective information from the owner
- Observation and conformation (willingness to move; postural deformities, conformation)
- Analysis of provoking activity – sport- and occupation-specific
- Gait – sport- and occupation-specific
- Active physiological movements
- Passive physiological movements
- Passive accessory movements
- Soft tissues – palpation and testing
- Nervous system – testing relative spinal cord and peripheral nerve function plus status of neuromeningeal system

8.2.2 History

Taking an effective history is fundamental to optimal physiotherapy treatment; therefore it is arguably the most important part of the whole examination (Refshauge & Latimer 1995).

Following the diagnosis given by the veterinarian, it is important to take an extensive clinical history from a physiotherapist's point of view, to document the degree of functional disability perceived by the owner/handler/trainer, onset and progression of the disorder and the past history related to the disorder. The physiotherapist can employ a variety of questioning methods, but should avoid posing leading questions. Open-ended questions provide reliable and valid information. Taking a history requires good interpersonal skills as well as a good knowledge base. The time spent with the owner obtaining information allows the physiotherapist to observe the animal's general demeanour, and behaviour while observing its general condition, conformation, gait and posture.

The history of the animal's presenting condition should be taken from the owner and recorded in the physiotherapy records, supplemented by notes obtained from the referring veterinarian. A typical veterinary referral should include details of the breed, age and sex of the animal, veterinary diagnosis, previous medical history, history of the present complaint, current medication and veterinary management. If the veterinary referral does not include all this information, it is important for this to be followed up by the physiotherapist with the veterinarian.

The owner should be asked when they first became aware that their animal had a problem or dysfunction, and whether or not the problem is becoming worse or better. The ques-

tioning should cover whether there are observed changes in the animal's demeanour, and if the owner feels the animal is in pain, or is depressed or anxious. In the case of lameness, it is useful to ask the owner to identify the affected limb and to report any observed gait abnormalities. The owner may be questioned regarding activities or situations that exacerbate or ease their animal's condition. It is often these observations that will affect in the owner's eyes whether or not the physiotherapy intervention is of benefit to their animal.

In summary, for animal physiotherapy, the structure of a history includes:

- Recording of the area affected/effect of dysfunction
- Current history of condition/current veterinary diagnosis
- Past history (including past treatments)
- Questions to determine contraindications and precautions to treatment
- Questions related to the animal's occupation/activity (including equipment and tack used)
- Owner/handler expectations of future occupation/activity
- For ridden horses, questions related to rider biomechanics/existing injuries.

All information must be recorded in detail for legal purposes as well as liaison with the veterinarian and owner.

8.2.3 Observation

The patient should be observed statically and dynamically.

Static observation

The purpose of observing the animal is to glean information about any visible functional deficits and other abnormalities such as swellings, muscle atrophy, scarring, alignment of extremities and spine, and also the animal's conformation (see also Chapter 6) and condition score. The physiotherapist may observe the animal from a lateral, cranial, caudal, and sometimes dorsal aspect.

From the lateral aspect:

- Weight distribution between limbs
- Limb alignment (angles of long bones, hocks, stifles, carpi, placement of limbs caudocranially, hoof–pastern axis in the equine)
- Obvious swellings or scarring of limbs, body and head
- Angle of scapula, spinal curves and set of tail
- Head position
- Coverage of muscle/other soft tissue

From the cranial aspect:

- Mentation and head position (including head tilt)
- Limb alignment (angles at carpus, placement of limbs mediolaterally, foot/hoof alignment relative to metacarpals/proximal phalanges, hoof/claw growth)
- Trunk symmetry (including ribcage)
- Weight distribution between limbs
- Obvious swellings or scarring of limbs, body and head

From the caudal aspect:

- Symmetry and development of muscle/other soft tissue over pelvis and trunk
- Height of pelvic bony landmarks
- Set of tail
- Trunk symmetry (including ribcage)
- Limb alignment (hock height, angles at hock, stifles, fetlock (metacarpal/tarsal phalangeal) joints, placement of limbs mediolaterally)
- Weight distribution between limbs
- Obvious swellings or scarring of limbs, body and head

From the dorsal aspect (especially applicable to canines)

- Spinal curvatures
- Trunk symmetry (including ribcage)
- Alignment of pelvic bony landmarks
- Set of tail

Ideally, relevant abnormalities or asymmetries should be measured if the physiotherapist intends to alter them (Goodsell & Refshauge 1995). Useful measurement equipment includes tape measure, goniometer and digital camera.

Dynamic observation

Dynamic observation has been included in sections of this chapter specific to the dog and the horse.

Other gait tests (analysis of provoking activity)

It may be necessary to 'challenge' the animal if gait patterns are normal and the animal displays no functional disability during gait. These can be performed at this point of the examination but will be detailed in Section 8.3 The physical assessment.

In the case of dogs, obstacle courses are a useful means by which to challenge gait; for example, stairs or steps, poles to walk, trot or run over, or performing serpentine manoeuvres. Transitions from sit to walk and vice versa are also useful tests. Many of these gait tests fall under the category of 'functional tests' and may relate to the animal's occupation. Obstacle courses may also be used to emphasise functional disabilities in the horse, such as slopes (up and down), poles, serpentine manoeuvres. Simple tests such as small circles, walking backwards and displacing the horse's pelvis laterally during walk may also be used, and there is some overlap here with neurological tests as well as 'functional tests'. Testing the horse's gait on different surfaces, such as hard concrete, soft, deep sand or grass may be required to emphasise functional disabilities as well as lameness.

8.3 Physical assessment

The physical examination is aimed at confirming the hypotheses and ideas suggested by the history and observation and identifying the most appropriate treatment (Refshauge & Latimer 1995). It involves the physiotherapist

carrying out active movements, passive motion tests, palpation and functional tests. The latter often involve sport- or occupation-specific manoeuvres in animals. Functional tests may also look at integrity of groups of muscles and/or neuromeningeal tension. Neurological assessment, which involves testing of the conductivity of the neural system, may be grouped under functional tests (see Chapter 7).

For adequate physical assessment it is essential to have an understanding of the anatomy, functional anatomy and biomechanics of the animal, conditions and diseases occurring in animals as well as practical skills in animal handling, and experience in the practice of physiotherapy. This knowledge enables the physiotherapist to be accurate, identifying the structures underlying the surface during observation, palpation, active and passive movements; to ascertain which anatomical structures are contributing to the global or local movement dysfunction or painful condition; and to ascertain when it is not appropriate to continue with physiotherapy treatment for the animal. The veterinarian may have already diagnosed a disease or condition, but if at any point in the examination the physiotherapist is unsure of whether a disease or condition exists that has not been addressed, or if the condition has progressed, the veterinarian should be consulted or the animal returned for veterinary examination.

During the physiotherapy physical assessment process, continual reassessment is essential as signs are altered by the examination process itself.

8.3.1 Active movement tests

Understanding the normal pattern and amount of movement available at a joint or body segment allows detection of abnormalities of movement (Lee 1995). In addition to overall body motion and gait, active movements of regions of the animal patient are important to assess. The physiotherapist needs to know what movements the animal is willing to do, as, unlike humans, they cannot tell us which activities cause pain or difficulty. Active movement testing enables the physiotherapist to ascertain which movements are affected, in what way the movements are altered, the effect of pain on movement and the range of movement available (Refshauge & Latimer 1995). Importantly, baseline measures are made for reassessment so that it can be established whether the patient has improved as a result of treatment. The physiotherapist may need to set up tasks to view certain active movements as the animal patient cannot be asked to specifically move an affected body part (see other gait tests).

Facilitated active movements may be part of the manual examination and can be achieved using food treats or reflexes inherent in the animal. An example of an active movement readily facilitated by a treat is cervical lateral flexion range of motion. The vertebral level at which the lateral flexion can be approximately guided by the physiotherapist's positioning of the food treat (Figure 8.2). Reflexes, such as the rounding reflex in the horse, can facilitate the

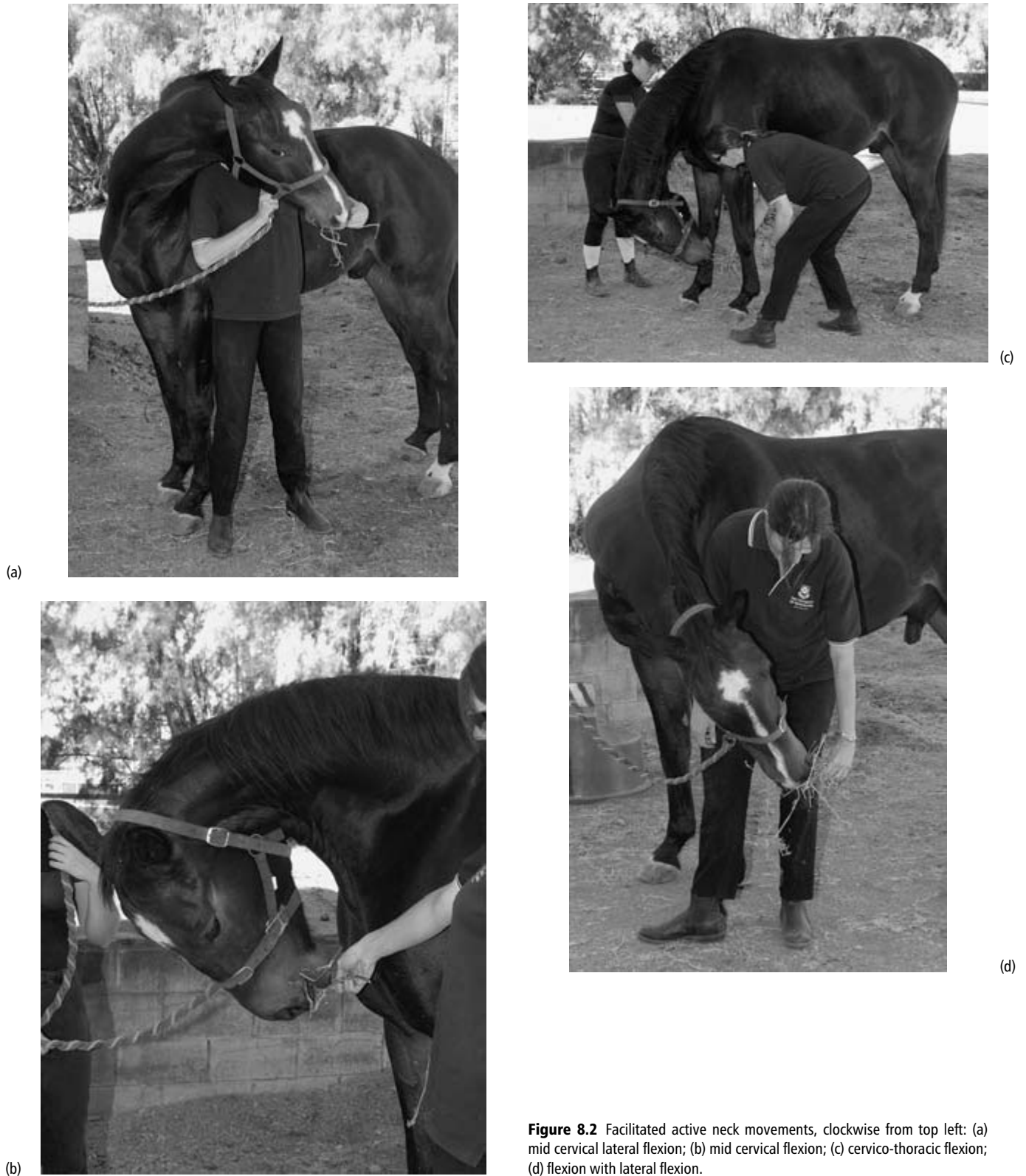


Figure 8.2 Facilitated active neck movements, clockwise from top left: (a) mid cervical lateral flexion; (b) mid cervical flexion; (c) cervico-thoracic flexion; (d) flexion with lateral flexion.

general movement of flexion of the vertebral column (Figure 8.3).

It may be desirable to reproduce the signs in an animal to enable a baseline to be established so treatment effect and

its dosage can be monitored. Applying further stress to the active movement may be required for the reproduction of signs. Examples of further stressing active movements are the hock, fetlock and carpal flexion tests, to further stress



Figure 8.3 Rounding reflex.

those joints for reproduction of signs. Further stressing a joint, or applying overpressure can also ‘rule out’ a joint or segment as being responsible for signs, if there is no pain response or no worsening of movement pattern following the overpressure.

8.3.2 Palpation

To carry out effective palpation, it is essential that the clinician has a sound knowledge of the animal’s anatomy, and has developed skills that enable differentiation between varying states of the soft tissues and articular structures, both within and between individual animals. For example, the musculature of a racing Greyhound will feel completely different from that of a more sedentary lap dog, and the joints of the thoracolumbar region of a Thoroughbred will have a different feel regarding resistance and range of motion from that of a Welsh Mountain Pony.

General soft tissue palpation

This may be performed safely on all animal patients and gives the physiotherapist information regarding temperature, soft tissue irritability muscle spasm/hypertonicity/hypotonicity, bony anomalies, soft tissue thickening/tightness/swelling and pain response. General palpation is also an important part of the physiotherapist’s communication with the animal patient, as a precedent to deeper palpation and further manual examination. This may be carried out before active movements if appropriate.

Specific soft tissue palpation

Muscles can be a source of pain and can be palpated for pain response as well as the parameters mentioned in the above paragraph. Muscle palpation is a subjective assessment technique based on the physiotherapist’s experience in feeling the quality of soft tissue. Algometry has been found to be a more objective measure of muscle pain pressure threshold (PPT) and has been used in both human and

animal research (Ohrbach & Gale 1989, Brown *et al.* 2000, Haussler & Erb 2006). In veterinary literature PPT has been referred to as mechanical nociceptive threshold (MNT) (Haussler *et al.* 2006). Algometry has been shown to be a repeatable modality for assessment (Varcoe-Cocks *et al.* 2006), and can be used in baseline assessment as well as reassessment.

Specific muscle palpation should be carried out when the working hypothesis has caused the physiotherapist to believe that groups or individual muscles are a primary source of signs of pain. Experience will help the physiotherapist to determine whether a pain response on deeper muscle palpation is due to reactivity of muscle groups, or symptomatic pain. Often, the quadruped is tender or ‘reactive’ along the epaxial muscles as a secondary aspect to another injury elsewhere. It is therefore necessary to compare palpation side to side, and develop a feel for localised spasm or alteration in muscle texture, as opposed to general reactivity. Alteration in muscle texture may manifest as strong, hypertonicity (spasm), hypotonicity, ‘boggy’ (oedematous), woody or fibrous. Occasionally the fascia covering the muscle group is not continuous. This may represent a recent or chronic fascial or muscle disruption.

Specific soft tissue palpation can also divulge the presence of scar tissue, effusions, oedema or general soft tissue thickening. These are signs that can readily be measured for reassessment during and after treatment.

Peripheral nerves may be palpated where they are found superficially, for example the distal limb of horses. Careful palpation of the peripheral nerves may provide information regarding pain response, quality of the tissue surrounding the nerve (including protective muscles spasm or muscle atrophy) and this should be carefully compared with the contralateral side.

8.3.3 Passive movement tests

The results of the passive movement examination in terms of the direction of motion loss or instability, the nature of the resistance perceived (tissue resistance of muscle spasm) and the nature and intensity of the pain provoked, direct the selection and application of manual therapy techniques. The physiotherapist is looking for reproduction of signs, or pain response in the animal, and local discrepancies in movement of the body part or segment such as increased or decreased stiffness. Comparison of movements or components of the movement to the contralateral side is essential while the physiotherapist gains experience in feeling for ‘normal’ motion, for any given part of the animal. Passive testing may be of body regions, or more localised to specific joints or soft tissues.

Passive joint tests

Passive testing of joint motion affects intra-articular and periarticular structures (joint surfaces, joint capsule, ligaments) and to an extent also affects extra-articular structures (musculature, fascia and neuromeningeal tissue)

(Coppieters *et al.* 2004). Passive joint movement tests can be divided into 'passive physiological' and 'passive accessory', or 'translatory joint movements' (see also Chapter 9).

Passive physiological joint testing

Passive physiological movements involve the movement patterns observed in active movements, but these are performed on the animal by the physiotherapist when the patient is passive. In the case of the horse, which for physiotherapy examination is usually conscious and standing, true passive physiological movement of certain joints is not possible. This is because of the tonicity of the postural muscles, particularly of the vertebral column. If required, the horse can be sedated by a veterinarian for physiotherapy examination, but the postural stabilising muscles remain recruited in standing. Depending on the state of relaxation of the dog patient, passive physiological joint movements can be carried out on most joints, especially if the dog is recumbent. If required, dogs can be sedated by the veterinarian for physiotherapy assessment. This enables true passive physiological assessment.

Passive physiological joint testing requires knowledge of the normal pattern and amount of movement available at a joint or body segment, to enable the physiotherapist to detect abnormalities of movement (Lee 1995). 'End-feel' of joint range of motion is an important guide as to the status of the joint. A joint may have abnormal range of motion, but in addition, qualifying the nature of the end-feel may help with assessment and reassessment and establish more clearly the structures involved in the dysfunction.

Passive accessory joint testing

Passive accessory motion tests are translatory movements applied by the physiotherapist to articular structures, and may be applied to the spine and the extremities. As for passive physiological joint movements, assessment using translatory joint movements involves the judgement of any pain response of the patient and the physiotherapist's perception of the quality and range of movement that result from the translatory forces applied to the joint. As these applied movements are passive, the same issue with postural muscles and passive physiological movement tests exists. However, translatory movements are small gliding motions, and with careful hand placement and positioning of direction of forces parallel to the 'plane' of the articular surface, information regarding the quality of motion at the joint, and pain response can be gathered. It is suggested that in assessment of vertebral levels in humans, a complete examination should include application of forces in a number of angulations of directions to the sagittal plane, to investigate the effect on stiffness, pain and muscle activity surrounding the segments (Caling & Lee 2001) (Figures 8.4 and 8.5).

Combined movements are another means by which to perform a more detailed passive movement assessment.



(a)



(b)

Figure 8.4 (a) Dorsoventral accessory translation of thoracic vertebral level.
(b) Rotational accessory translation of thoracic vertebral level.

Combined movements may allow a more accurate choice of manual treatment technique to be made, and may also help predict the outcome of a technique (Edwards 1994). The increase or decrease in signs that are produced when combining movements can help the physiotherapist to establish the direction of passive movement best suited as a treatment, as well as predicting response (Edwards 1994). For example, a combined movement in examination of extension and lateral flexion of the thoracolumbar spine may exacerbate a dysfunction at reassessment. A useful technique to relieve signs may then involve flexion and lateral flexion the opposite direction. If the same technique had improved the dysfunction (say, improved lateral flexion at the thoracolumbar spine) then placing that segment or segments into lateral flexion and extension to apply the chosen technique may be useful.

Combined movements may be performed in the extremities or the vertebral column. In the equine vertebral column, facilitated movement can be combined with overpressure to result in a combined movement (Figure 8.6). As the canine patient can be made recumbent, it is a little easier to obtain a 'true' passive combined movement in the spine (Figure 8.7). Peripheral joints in the horse may have combined movements applied in standing.

Combined movements may also give information about joint integrity in the periphery. An example is akin to the collateral ligament tests of the human knee, which indicate the possibility of damage of articular structures and will direct the type of management. Periarticular structures in peripheral joints in both the canine and equine can be similarly assessed using combined movements.

8.3.4 Functional tests

Muscles can be a source of pain but as previously addressed, can also be examined as a functional unit. Manual muscle testing is carried out in human patients to ascertain the ability of the individual muscles or muscle groups to contract through range and against resistance (Herbert 1995). Muscle testing can also help to localise pain, however it has been argued that strong muscle contraction, particularly isometric, can stress other pain-sensitive structures such as joint surfaces and ligaments. As manual muscle testing is a voluntary activity for the patient to perform at the request of the clinician, the value of such tests is difficult to determine in animals.

Human research has shown altered patterns of muscle recruitment with functional activities such as balancing on one leg (Hungerford *et al.* 2003) and straight leg raising (O'Sullivan *et al.* 2002). In the animal patient we can test patterns of functional muscle recruitment. An example is the ability of a horse or dog to unilaterally stabilise on one hind leg, by the clinician lifting the contralateral hind leg and displacing the horse towards the standing leg, can measure the integrity of the muscle groups of the hindlimb and pelvis on the weight-bearing limb (Figure 8.8).



Figure 8.5 Obliquely directed translation over costotransverse joint.



Figure 8.6 Combined movement assessment of flexion and left lateral flexion of equine thoracolumbar spine (using unilateral rounding reflex and overpressure with physiotherapist's left hand at desired level of vertebral column).



Figure 8.7 Combined movement assessment of flexion and left lateral flexion of canine thoracolumbar spine. Physiotherapist's left thumb is palpating approximation of spinous processes in a relatively flexed position.



Figure 8.8 Test for unilateral hind limb stability – horse is displaced toward the left by the physiotherapist. Note, this horse demonstrates hindlimb stability as there is no increase in lateral displacement of the pelvis over the weight-bearing hindlimb.

Neural provocation tests

Other manual tests to be grouped into functional tests include testing of the status of the neuromeningeal structures, specifically the ulnar, median and radial nerve of the forelimb, femoral and sciatic nerves in the hindlimb and the meninges of the spinal cord. As is the case in humans, neural provocation tests can be used in animals to assess the effects upon the neural tissue of the structures adjacent to the nervous system (the mechanical interface), and neurobiomechanics, that is sliding of nerve alongside the interface and elongation (Butler 2000). Neurodynamics are also affected by intraneural and extraneural oedema and circulation (Coppieters *et al.* 2004). Results from neural provocation testing may be sensitised by adding or removing a ‘central’ component such as cervicothoracic or thoracolumbar flexion (equivalent to slump test in human physiotherapy) (Coppieters & Butler 2001). A pain response may be reproduced during tension testing, and increased stiffness in neural tissue may limit movement (Boland 1995). Animal patients cannot report reproduction of symptoms, unlike humans, thus the physiotherapist must be sensitive to increase in perceived tension of the neuromeningeal structures, and compare side to side. Neural provocations tests are used not only to establish the contribution of the structure to symptoms, but for reassessment and treatment (Figure 8.9).



Figure 8.9 Neural provocation test of canine sciatic nerve (Babbage *et al.* 2006).

Neurological examination

Neurological examination is an important part of the functional assessment and is carried out when there are signs suggesting that neural conductivity is abnormal. Neurological tests may be used to monitor the progress of the patient (Chapter 7). When providing physiotherapy assessment or treatment, it is important to know where the neurological system may be compromised and to what degree. This will ensure the physiotherapy assessment does not further compromise the neural conductivity in the relevant part of the nervous system and the appropriate technique is eventually chosen for treatment. In all manual assessment, particularly when the vertebral column is involved, it is vital to perform a neurological examination focusing on the spinal cord to determine if there are neurological-based contraindications to manual therapy. Much of the neurological assessment will have been performed during the examination of the animal's gait (Chapter 6) and it is pertinent to always include specific neurological provocation tests in your gait assessment. Examples are, in the horse, small circles, walking up and down hills and backing (Chapter 7) and in the dog, the hindlimb-placing reflex. Scuffing of the toenails or areas of wear on the dorsum of the pad or hoof can alert the examiner to the likelihood of a reduced proprioception and this should be investigated further.

8.4 Special considerations in canine physiotherapy assessment

8.4.1 History

During the history taking, the physiotherapist may gain clues or red flags (contraindications or precautions for examination) as to breed-typical diseases, just from observing the breed and conformation of the dog. For example, some toy breeds are predisposed to patella subluxation; young, large-breed dogs are predisposed to ununited anconeal process,

osteochondritis dissecans, and panosteitis; and old, large-breed dogs are predisposed to chronic hip dysplasia. It is important, however, not to make assumptions regarding the dog's condition based only on the breed or conformation.

Recording the age of the patient is essential. Immature animals may have skeletal developmental growth disorders, and aged dogs may have degenerative conditions that require modification of examination techniques. Information should be gathered from the owner concerning the animal's home environment, diet, exercise levels and occupation (companion animal or working dog).

The following are suggested questions that may be useful:

- Does the dog live inside or out?

If the dog lives inside, ask the following questions:

- Has there been a change in the home which coincides with the onset of their symptoms, for example have wood floors/tiled floors or vinyl floors recently been fitted?
- Do they live with other animals, cats, dogs, etc.?
- Is there a boisterous puppy or child in the house that they regularly play with?
- Where is their bed, and what type of bedding is used? For example, is it a hard or soft surface? Could it possibly be in a draft? Are they confined to a cage?
- Do they regularly climb the stairs or are there steps in or out of the house?
- Do they jump in or out of the car, on and off the owner's bed/settee, etc.?

If the dog lives outside, ask the following questions:

- Are they chained?
- What type of shelter do they have?
- Has there been a change to the garden layout or surfaces?
- Plus any of the questions already listed for indoor dogs.

It is useful to ask the owner to describe in detail the animal's daily routine from the time it wakes in the morning to the time it goes to sleep. This will gather information of the amount and type of exercise that the dog has.

The following questions may be of benefit when discussing the patient's exercise levels:

- Is it a working dog or companion animal?

If it is a working dog, specific information related to the discipline undertaken should be gathered.

If it is a companion animal ask the following general questions:

- What is the frequency of exercise, once, twice daily, weekly etc.?
- How long is the typical exercise session in terms of minutes?
- Is the dog leash-walked or allowed free exercise, or a combination of both?
- Does the dog play with other dogs when out?

- What type of lead/harness does it wear?
- Does it play with toys, e.g. chase balls/frisbees/squirrels/rabbits?

In addition, the owner should be asked if exercise seems to exacerbate or lessen signs. For example, the animal may initially appear stiff on an affected limb and may subsequently warm up and move more freely after exercise. Conversely, some animals may appear more uncomfortable after exercise. Discomfort/lameness may become more apparent several hours later or the next morning.

8.4.2 Canine static observation

It is useful to consider individual breed differences when conducting a visual examination. Each breed will have its own stance and posture, for example chondrodystrophic breeds such as Bull Dogs and Bassett Hounds will stand completely differently from breeds such as the German Shepherd and Labrador Retriever. Any deviation from the breed standard should be recorded, together with information relating to the patient's general condition in terms of weight and muscling. Obesity is known to exacerbate chronic lameness in dogs and condition scoring can be a useful objective measure during treatment (Chapter 3).

Observation of the position of the head and neck and trunk, will often give clues as to the amount of weight that an animal is putting through its limbs. Observation has been covered in Section 8.2.3 of this chapter.

8.4.3 Canine dynamic observation and gait assessment

The dog should be observed both statically and when moving, to determine information about its static and dynamic conformation and posture. Its willingness to move in general, coupled with the fluidity of motion when changing postures should be noted. For example, is the animal able to get from lying to sitting, sitting to standing with ease and vice versa? Is it symmetrical in its movement patterns, and equally weight bearing on each limb? Does it appear to be in pain? Does it verbalise? When changing position does it do so in a consistent pattern?

If possible, it is preferable to observe the dog ambulating on a number of different surfaces at different gait speeds. This should be done in a controlled environment, without evident distractions for the patient. Ideally, the dog should be observed on and off the lead. If it is only possible to see the animal moving on the lead, the owner should be asked to walk on either side of the animal with a slack lead to allow the dog to move freely without interference. The dog should be observed from different angles.

8.4.4 Canine palpation

Before beginning palpation of a dog, it is important to have carried out a risk assessment. Part of this will have been done by observing the dog during the subjective assessment, and

observation of static and dynamic postures. The physiotherapist needs to be aware that one of the dog's defence mechanisms is biting, especially if it is in pain (Chapter 2). A restraint such as a muzzle is a useful piece of equipment to carry when providing physiotherapy for dogs. The owner may need to acclimatise the animal to wearing a muzzle in advance of the physiotherapy sessions.

Many dogs become tense when standing on an examination table, making it difficult to assess muscle tone accurately. A 'vet bed' can provide a useful alternative.

Pain response as a result of palpation in an individual is often dependent on the pain tolerance of the animal concerned. Some breeds are known to be more stoic than others.

Every clinician will develop his or her own examination sequence when palpating a dog. Some prefer to start with the head and work caudally; others will start at the toes and work dorsally. All parts of the dog should be palpated sequentially, even if the presenting problem is an obvious injury such as a 'knocked up/sprung toe' in a competitive animal. All animals learn compensatory movement patterns, very often resulting in secondary musculoskeletal problems.

8.5 Assessment and palpation of canine extremities

8.5.1 General palpation of the limbs

The dog's limbs may be palpated with the patient in the standing or the recumbent position, or both. The advantage of having the dog in lateral recumbency is that they can be more readily restrained by the physiotherapist or handler allowing a more thorough examination of the limb in question. Palpation of animals in standing allows a comparison of left and right limbs, by palpating both sides simultaneously. Both limbs should be similarly positioned.

It is preferable to start proximally and work distally following the lie of the coat, initially applying gentle pressure with the flat of the hand to allow the patient to become acclimatised to contact by the physiotherapist. Differences in muscle definition, atrophy/hypertrophy, soft tissue swelling, bony anomalies, and temperature and pain responses between the left and right side should be noted. Differences noted may be examined in more detail when the animal is later placed in lateral recumbency.

8.5.2 Palpation of the canine limbs

The joints of the thoracic and pelvic limbs should be assessed, including palpation of:

- Bony landmarks
- Ranges of motion
- Joint stability

A detailed assessment of each muscle is beyond the scope of this chapter. However, it is expected that the examining physiotherapist should have an excellent grasp of the regional muscle anatomy, so that they are able to evaluate

systematically each muscle in turn, noting clinical signs of atrophy, hypertrophy, spasm, haematomas, tears, adhesions and discomfort. Specific muscle and tendon pathologies related to individual joints will be highlighted. When assessing joint range of motion, the affected and the contralateral joint should be simultaneously tested, comparing the quality and resistance of the joint movement, crepitus and end-feel. Ranges of motion vary between breeds and have been published for the Labrador Retriever (Jaegger *et al.* 2002; Crook *et al.* 2007) and Greyhound (Nicholson *et al.* 2006). If joint instability is suspected following physiotherapy examination, the referring veterinarian should be consulted so that any underlying pathologies can be further evaluated. Orthopaedic assessment of the thoracic and pelvic limbs is detailed in Chapter 6.

8.5.3 Palpation of the canine vertebral column

Assessment of the head, vertebral column and pelvis often require detailed neurological evaluation. For further details refer to the neurology chapter (Chapter 7).

Head and cervical spine

The position of the head on the neck should be observed from above, the front and either side. Any obvious neurological signs such as head tilt, nystagmus and paralysis/paresis of the facial muscles should be investigated fully before commencing further examination. The general demeanour of the animal should be noted, together with its responsiveness to verbal stimulation.

Palpable bony landmarks of the head

- Occipital protuberance
- Nuchal line
- Mastoid process
- Temporomandibular joint

Palpable bony landmarks of the cervical spine

- Wings of the atlas
- Vertebral bodies of C2–C6
- Vertebral body of C7? (This depends on the size of the dog but generally difficult to palpate.)

Active range of motion

The dog should be encouraged to go through the full active range of neck motion. This can be done in the form of a baited stretch where the dog reaches for a treat. For the caudal cervical spine, the dog is asked to flex fully to the middle of the chest, extend, side-flex and rotate throughout the whole cervical spine to either side. The upper cervical spine motion can be differentiated from the caudal cervical spine by gently stabilising dorsally over C2 region and guiding the nose in a forward and upward motion to simulate upper cervical extension. The nose can be guided closer toward the cranial chest to gain the upper cervical 'nodding'

motion. Upper cervical rotation can be guided with a food treat, in a motion simulating the nose rotating around the longitudinal axis of the dens of C2.

Passive range of motion

With the animal in the sitting or standing position, one hand is used to stabilise the shoulder girdle, while the other hand gently guides the head through available range, noting any resistance from the animal, the quality of the movement and end-feel. Care should be taken not to exceed the range of motion achieved by the dog actively, or past the dog's comfortable range. Again, with careful hand placement, upper cervical motion can be differentiated from the caudal cervical motion. (Refer to contraindications to cervical manual techniques for dogs in Table 9.2).

Ranges of motion in the upper cervical spine (O–C1–C2)

- Flexion (O–C1): Nose to upper chest
- Extension (O–C1): Nose moved in a forward direction first then up toward ceiling
- Rotation (C1–2): Nose 'pivots' around the estimated longitudinal axis of the dens of C2

Ranges of motion in the caudal cervical spine (C2–C7)

- Flexion: Nose to lower chest
- Extension: Nose to ceiling (90°) to remainder of vertebral column
- Sideflexion/rotation: Nose to lateral chest wall

The greatest amount of flexion and extension (nodding movement) occurs at the atlanto-occipital joint. Rotation is greatest at the atlantoaxial joint, with smaller amounts of flexion, extension, side flexion and rotation occurring throughout the rest of the cervical spine. It should be noted that coupled motion occurs throughout the spine owing to the orientation of the facet joints; hence, lateral flexion is accompanied by rotation (see Chapter 4).

Assessing for pain/restricted motion

If the dog is unwilling to go through full active/passive range of motion, this may be because of pain or restriction of soft tissues or articular structures. Palpation of the muscles, soft tissues and joints may give further clues as to the source of the limitation. Pain response or altered motion arising from vertebral segments can be determined by palpating ventrolaterally, dorsolaterally and via a lateral translatory glide (Figure 8.10). The plane of the articular facets should guide the direction of passive movement assessment (see Chapter 4). The accuracy of palpation of vertebral levels is dependent on the size of the dog.

8.5.4 Thoracic spine

The dog should be observed for degree of thoracic lordosis or kyphosis from the front, above and either side, plus symmetry of the rib cage noted.



Figure 8.10 Lateral accessory translation of C3–4.

Palpable bony landmarks of the thoracic spine

- Spinous processes T1–T13 inclusive
- Anticlinal vertebrae T10/11
- Ribs 1–13
- Sternum

Ranges of motion of the thoracic spine

Specific ranges of intervertebral motion have not been documented for the thoracic spine in the dog. It is thought that orientation of the articular facets guide the direction and amount of available motion (see Chapter 4).

Active assessment of thoracic spine

The dog may be observed in various positions and activities which move the thoracic spine, such as scratching or stretching. Lateral flexion-baited stretches can be utilised, similar to those for the cervical spine, but encouraging the dog's nose to approximate the flank. Lateral flexion can be combined with extension and flexion of the thoracic spine by directing the treat appropriately.

Passive assessment of motion of the entire thoracic spine

With the animal in the standing position:

- Dorsal flexion: The flat of one hand is placed under the dog's chest on the sternum, and applies pressure in a dorsal direction encouraging the dog to round its back.
- Ventral flexion: The flat of one hand is placed on the thorax of the dog at about the level of T6–10 and applies pressure in a ventral direction encouraging the animal to arch its back.
- Side flexion/rotation: The flat of one hand applies dorso-lateral pressure to the side of the chest wall, while the other hand stabilises at the shoulder/hip region.

Assessing individual joint mobility in the thoracic spine and rib cage

The spinous processes in the dog are readily palpable, thus it is possible to determine joint mobility at each segmental level. As for the cervical spine, accuracy of individual vertebral level palpation is dependent upon the size of the dog.

Thoracic dorsoventral mobility

This can be assessed by applying digital dorsoventral pressure either side of the spinous process, and repeating at each level.

Thoracic rotation

This can be tested by applying unilateral dorsoventral pressure either side of the spinous process. In both cases the quantity and quality of the movement and the end-feel should be noted, together with any indication of discomfort from the animal.

Mobility of the rib cage

With the dog in the standing position it is possible to determine mobility of the rib cage, by laying the flat of each hand laterally on either side of the chest wall at approximately the same level, comparing left and right sides. Rib excursion may be felt during respiration.

Mobility of the costovertebral and costosternal joints

This can be achieved by applying digital pressure in a ventral direction over the costovertebral joints and by applying a dorsal pressure over the costosternal joints. Range and quality of motion is noted, and compared side to side.

See Chapter 9, for passive physiological and accessory glide testing of thoracic vertebral motion segments.

8.5.5 Lumbar spine

Palpable bony landmarks

- Spinous processes L1–7
- Lumbosacral junction
- Wings of the ilia

Ranges of motion of the lumbar spine

The facet joints of the canine lumbar spine display mostly sagittal alignment, with interlocking of the caudal and cranial articular processes; therefore flexion–extension tends to be the main movement available at this part of the vertebral column. Preliminary data from *in vitro* study suggest that flexion–extension is variable throughout the lumbar spine, increasing from 5–10° at L4–5, to 40° at L7–S1. The greatest amount of lateral bend occurs at L4–5, and very little axial rotation was observed at all lumbar segments. Flexion–extension is coupled with slight axial rotation, which increases cranial to caudal. During lateral flexion and axial rotation the coupling of motion is greatest in the lumbosacral segment, followed by L4–5 (Benninger *et al.* 2004).

At the lumbosacral junction the caudal articular facets face mediadorsally and cranial facets face lateroventrally – they are more angled to the transverse plane than the more cranial lumbar joints. Flexion–extension is significant at this articulation (Benninger *et al.* 2006).

Passive assessment of movement of the entire lumbar spine

With the animal in the standing position:

- Dorsal flexion: The flat of one hand is placed under the dog's belly and applies pressure in a dorsal direction encouraging the animal to round its back.
- Ventral flexion: The flat of one hand is placed on the dorsal lumbar spine of the animal at the levels L1–6 and applies pressure in a ventral direction encouraging the animal to arch its back.
- Side flexion/rotation: The flat of one hand applies dorso-lateral pressure to the side of the lumbar spine, while the other hand stabilises at the thoracic/hip region.

Assessing individual joint mobility in the lumbar spine

The spinous processes in the dog are readily palpable thus it is possible to determine joint mobility at each segmental level.

Lumbar dorsoventral mobility

This can be assessed for extension of a given vertebral motion segment by applying dorsoventral pressure either side of the spinous process, and repeating at each level for comparison of range of motion, end-feel and pain response. The relative 'opening' or fanning out between spinous processes, as an indication of flexion at a given motion segment, can be localised between vertebral segments by careful palpation with one hand and application of ventral 'lift' via the abdomen with the other hand.

Assessment of dorsoventral mobility can also be carried out via passive physiological movement tests in lateral recumbency. This is described in detail for thoracic segments in Chapter 9, and can be applied to any vertebral level.

Lumbar lateroflexion/rotation

This can be tested by applying unilateral dorsoventral pressure either side of the spinous process, in a direction obliquely angled toward the midline. This is mostly applicable for more caudal levels. Comparison between levels and from side to side should be performed. See Chapter 9, for passive physiological movement test in lateral recumbency as described for thoracic vertebral levels.

8.5.6 Pelvis and sacroiliac joints

Palpable bony landmarks

- Iliac crest
- Tuber sacrale
- Sacrum

- Cranial dorsal iliac spine
- Cranial ventral iliac spine
- Greater ischiatic notch
- Lesser ischiatic notch
- Greater trochanter
- Caudal vertebrae

Note any differences in the relative positions of the bony landmarks on the left and right sides.

Ranges of motion of the pelvis and sacroiliac joints

The main movements available at the sacroiliac joint are flexion–extension with a total of range of 7° thought to be available (Gregory *et al.* 1986).

Assessing passive mobility of the sacroiliac joints

Palpation of the ilial wing and the sacral body simultaneously (with flat of hand or two fingers) allows relative motion between the ilium and the sacrum to be assessed during protraction/retraction of the hindlimb. This may be done in lateral recumbency or in standing (Figure 8.11). A



(a)



(b)

Figure 8.11 (a) Palpation of cranial rotation of ilium relative to sacrum via hindlimb retraction. (b) Palpation of caudal rotation of ilium relative to sacrum via hindlimb protraction. Fingers palpate the sacral spine and ilium.

dorsal glide of the ilium on the sacrum can be applied via the hindlimb and any relative dorsoventral motion between ilium and sacrum assessed. Gapping and approximation of the sacroiliac joint can be assessed via the described palpation of ilium and sacrum, with adduction and abduction of the hindlimb.

8.6 Special considerations in equine physiotherapy assessment

As for dogs, there are certain breeds of horse which tend to carry certain conditions. Examples are atlantoaxial occipital malformation in Arabians, cervical vertebral malformation (CVM or ‘wobbler’) in Thoroughbreds and Warmbloods, and developmental (congenital) peripheral joint disease in Warmbloods and also Miniature breeds. It is important, however, to take a full, unbiased history without ‘labelling’ the horse into a category.

Special questions pertaining to the equine patient include:

- Horse’s occupation or use – current and past, including current level of training
- Tack – type, fit, recent changes
- Nutrition
- Shoeing/trimming
- Rider musculoskeletal dysfunction existing concurrently

8.6.1 Equine static observation

General static observation of the quadruped has been covered in Section 8.2.3. Specifically for horses, it is important to observe the following:

Trunk

- Areas of white hair under girth or saddle region (may indicate poor tack fit)
- The curves of the cervical, thoracolumbar and lumbosacral spine in relation to the horse’s age, breed and conformation

Extremities

- Specific detail to hoof wear, hoof axes and shoes
- Observe closely for scars, swelling and asymmetries of limb alignment

Head

- Asymmetries of facial features
- Mentation, ear position

8.6.2 Equine dynamic observation and gait assessment

Observation of gait will ascertain the degree of lameness of the animal, if present. Lameness scoring will not be dealt with in detail here, rather, the observation of gait from a physiotherapy movement analysis point of view.

At the walk

In the absence of obvious lameness, the walk may bring out subtle movement dysfunction that faster gaits may mask. This is because in the walk cycle there are stages where a limb must be able to stabilise unilaterally at a given point. Kinematic data reveals greater ranges of motion for the thoracolumbar spinal segments at the walk compared with the trot (Roethlisberger *et al.* 2006), giving another reason for the subtle dysfunction of gait being more apparent to the observer at the walk. The horse may be led in hand or observed on the lunge. For the slower gaits of walk and trot it may be useful to assess both in a straight line and on the circle.

From the lateral aspect (straight line or circle)

- Timing of limb contact with the ground (audible as well as visual)
- Placement of hindlimb with respect to forelimb (degree of 'overreach')
- Placement of hoof (flat/toes first/toes up)
- Head carriage/head 'bob'
- Maintenance of spinal curves
- Tail carriage
- Recruitment of abdominal musculature
- General mobility of trunk, pelvic and neck fascia

From the caudal aspect (straight line)

- Timing of limb contact with the ground (audible as well as visual)
- Tracking of hindlimbs compared with forelimbs
- Specifically for hindlimb – plaiting/winging/mediolateral placement
- Mediolateral placement of hind hoof with ground
- Tail carriage
- Symmetry of rise of gluteal musculature
- Symmetry of lateral swing of pelvis
- Symmetry of lateral swing of ribcage/abdomen

From the cranial aspect (straight line)

- Head carriage/head bob
- Tracking of forelimbs compared with hindlimbs
- Specifically for forelimb – winging/mediolateral placement
- Mediolateral placement of fore hoof with ground
- Symmetry of shoulder motion
- Symmetry of swing of ribcage/abdomen

At the trot

Concussive limb lameness may be more apparent at the trot than the walk, owing to increased loading forces. In the absence of lameness the following are some useful points regarding gait analysis at the trot:

From the lateral aspect

- Head carriage/bob
- Symmetry of the diagonal limbs striking the ground

- Tail carriage
- Maintenance of spinal curves
- Recruitment of abdominal musculature
- General mobility of trunk, pelvic and neck fascia
- (On the circle) ability of inside hind leg to 'drive' on the circle – often it will adduct if difficulty stabilising
- Degree of overreach

From the caudal aspect

- Timing of limb contact with the ground (audio as well as visual)
- Tracking of hindlimbs compared with forelimbs
- Specifically for hindlimb – plaiting/winging
- Tail carriage
- Symmetry of rise of gluteal musculature
- Symmetry of lateral swing of pelvis
- Symmetry of lateral swing of ribcage/abdomen

From the cranial aspect

- Head carriage/head bob
- Tracking of forelimbs compared with hindlimb
- Specifically for forelimb – winging/mediolateral placement
- Symmetry of shoulder motion
- Symmetry of swing of ribcage/abdomen

At the canter

Important in assessment of gait is the transition from trot to canter and back to trot again. The physiotherapist looks for ease of transition, coordination of limbs and ability of the horse to engage the hind (pelvic) limbs, which involves some lumbosacral flexion as well as recruitment of pelvic and abdominal stabilising musculature (Faber *et al.* 2001). It is important to assess the recruitment of hindlimb musculature with each direction of the canter on the lunge. Canter assessment is usually performed on the lunge owing to the speed of gait.

8.7 Equine palpation**8.7.1 Head, neck and temporomandibular joint (TMJ)**

The physiotherapy history should follow on from the veterinary history and examination, in which a *full dental examination* should have been undertaken, along with any other diagnostic tests described above.

Special questions regarding temporomandibular joint (TMJ) dysfunction include asking the owner about the following clinical signs (Moll & May 2002):

- Head shaking
- Quidding
- Apparent masticatory problems

As well as:

- Biting problems

The above list is not exhaustive, and there may be more subtle clinical signs, which are associated with TMJ dysfunction, such as minor deviations of the poll during work or inability of the horse to accept the bit on one rein.

Observation

- Symmetry of the visible muscles of mastication, primarily the masseter and temporalis muscles at rest
- Symmetry of facial bones, especially mandible
- Position of upper cervical spine regarding extension and flexion, plus symmetry of occipital and upper cervical muscles
- Mastication during eating
- Mentation

Palpation

Careful palpation of the horse's TMJ and comparison with the other side can detect joint effusion. The TMJ is located by following a horizontal projection from the lateral aspect of the eye directly caudal (Moll & May 2002). The distance between the condyle of the mandible and the mastoid process can be palpated and compared side to side as can the distance between the mastoid process and the atlas. Gentle movement of the mandible in a lateral direction can be used to confirm the location of the mandibular condyle, as the joint line is palpated.

Muscles of mastication should be palpated bilaterally for symmetry of bulk, quality of the muscle belly and pain response. The masseter, temporalis, medial pterygoid (jaw closure) and anterior belly of digastric (jaw opening) are the most easily palpable muscles. The anterior belly of digastric, and medial pterygoid muscle are palpated on the medial aspect of the mandible. The strap-like digastric belly can be distinguished medially and rostrally from the medial pterygoid. The occipitomandibular part of caudal belly of digastric, which assists in raising the tongue and hyoid bone, can be palpated just caudal to the mandible. Other muscles assisting jaw opening are geniohyoid, inferior genioglossus, sternohyoid and omohyoid (Baker 2002). The latter two muscles can be palpated just caudal to the hyoid bone and run caudally to the scapula, crossing the larynx (Sisson 1975).

Algometry has been used in assessment of pain in the muscles of mastication in human TMJ dysfunction (Farella *et al.* 2000, Michelotti *et al.* 2004), and has been shown to be useful in assessment of pain response in horses (Varcoe-Cocks *et al.* 2006). Algometry therefore may be an objective measure of TMJ dysfunction where there is muscular adaptation or spasm.

Motion tests

Lateral mandibular glide

Symmetry of lateral glide of the mandible may give an indication of altered mediolateral excursion. The mandible can be moved laterally in relation to the stabilised maxilla, where the examiner should see an initial lateral displacement of



Figure 8.12 Lateral excursion test to examine dental/temporomandibular joint movement.

the mandible, followed by an oblique glide of the mandible relative to the maxilla which 'gaps' the upper and lower incisor rows (Figure 8.12).

Flexion test

This tests the rostral movement of the mandible relative to the maxilla. The front incisors are palpated by the examiner's index finger while the head is flexed on the upper cervical spine. There should be a relative rostral movement of the lower incisors compared with the upper incisors, indicating a slight rostral movement of the mandible. If the head is moved into a relative upper cervical extension, then the lower incisors should be felt to glide caudally on the upper incisors (Figure 8.13).

8.7.2 Equine cervical spine

Palpation

The muscles of the occiput, temporal and hyoid region should be palpated for symmetry, tone, thickening, tenderness and spasm. This can be done bilaterally, taking care of your position if in front of the horse when palpating occipital region, as the horse can throw his head up if there is a pain response. The larger muscles of the cervical spine should also be palpated with the same parameters in mind.

Each vertebral body can be palpated from C1–C6. When palpating horses with a lot of soft tissue coverage, it is often useful to count the spaces between the vertebral bodies to enable identification of each vertebral level.

Active movements

Observation of the horse during gait, on small circles and ridden, during grazing/feeding will give an overall impression of cervical range of active motion.

Baited active movements for upper cervical spine

- Extension: Guide horse's muzzle forward and up, (with a treat) so effecting upper cervical extension.



Figure 8.13 Flexion test to examine jaw movement relative to head position.

- Flexion: Guide horse's muzzle towards upper chest to effect a nodding movement (at the poll).

For caudal cervical spine

- Flexion: For lower cervical/upper thoracic flexion, guide horse's muzzle down between fetlocks (or observe horse grazing – check for even weight distribution between forelimbs); also guide horse's muzzle towards sternum to check mid-cervical flexion.
- Lateral flexion: Guide muzzle around along horse's lateral trunk towards the flank and compare range side to side.
- Lateral flexion/flexion: Guide muzzle around towards the carpal region and compare range side to side (Figure 8.2).

Passive physiological movements

Many of these manoeuvres are passive-assisted movements (not true passive physiological movement tests) as the horse is not truly relaxed in the standing position.

Upper cervical spine

- Extension: Guide muzzle as described above (with or without treat); stabilise with one hand gently over C1 and apply gentle overpressure from underneath the muzzle. Assess end-feel and any asymmetrical deviation (laterally or rotatory) of the occiput on C1.



Figure 8.14 Passive physiological assessment of equine atlantoaxial (C1–2) joint.

- Flexion: Guide muzzle towards the upper chest; stabilise with one hand gently over C1 and apply gentle overpressure to the front of the muzzle. Assess end-feel and any deviation of occiput on C1.
- Rotation: Stabilise with one hand over C2 and guide horse's muzzle toward you on an axis that is approximately through the longitudinal axis of the dens. Apply gentle overpressure via the muzzle. Compare range of motion and end-feel side to side (Figure 8.14).

Caudal cervical spine

- Flexion: Passive neck flexion is difficult to assess as it requires the horse to initiate the action.
- Extension: As above.
- Lateral flexion: Motion at each cervical level between C3–C6 can be assessed by palpating the 'opening' of the cervical vertebra when an assistant laterally flexes the horse's neck away from the assessor.
Or: Stabilise with one hand over the vertebral body to effectively 'block' motion from the chosen level caudal, and gently guide the horse's muzzle toward you, in a lateral flexion direction. Apply gentle overpressure; assess range of motion and end-feel and compare side to side.

Passive accessory palpation

Lateral glide technique to caudal cervical spine

This technique is described in detail in Chapter 9, and involves assessing the relative lateral glide of the more cranial level on the more caudal level. Range of motion and end-feel is compared side to side.

Oblique dorsoventral translation

This technique assesses the combined ventral and lateral glide of a vertebral level relative to the segment above and below. Translation is applied in an oblique dorsoventral direction on the body of the vertebra, while stabilising the level cranial to that from the contralateral side (Figure 8.15).



Figure 8.15 Oblique dorsoventral translation at C3–4. Near hand is applying translation to C4 vertebral body while far hand is stabilising C3 contralaterally.

Oblique ventrodorsal translation

Translation is applied in an oblique ventrodorsal direction on the body of the vertebra, while stabilising the level caudal to that from the contralateral side.

8.7.3 Thoracic and thoracolumbar spine

Palpation

The musculature of the trunk, including the epaxial muscles, abdominal muscles, and wither region should be palpated for symmetry, tone, thickening, swelling, tenderness and spasm. The spinous processes of T4–T18 should be identified and the corresponding rib angles. Often it is helpful to identify the last thoracic vertebrae via the 18th rib and palpate cranially. The sternum and manubrium and costal cartilages should be palpated ventrally.

The lumbar spinous process should be identified from L1–L6 – often it is easier to count back cranially from the lumbosacral junction. The transverse processes can be palpated, depending on the depth of soft tissue coverage. Note, some horses may only have five lumbar vertebrae and/or transitional thoracolumbar and sacral vertebrae (Chapter 4).

Active movements

Active movements are assisted with either baited stretching or using reflexes inherent in the horse.

- **Lateral flexion:** Observation of the relative contribution of the cranial thoracic spine to a baited lateral flexion test of the cervical spine, or making the horse perform a tight circle, can be compared side to side.

The lateral flexion reflex can be initiated by stimulating the contralateral gluteal region of the horse with a firm object such as fingernail, pen cap or blunt hoof pick. The horse will tend to shift the pelvis away from the irritant, and as there is minimal lateral flexion in the lumbar vertebral column, much of the movement occurs at the thoracic spine (this is often combined with some flexion of the thoracolumbar region).

- **Flexion:** A ventrodorsal ‘lift’ reflex can be performed via the manubrium/sternum or more caudally at the level of the girth. A firm pressure with fingernails, pen cap or blunt hoof pick applied to the midline of the above-mentioned regions can cause the horse to ‘lift’ the cranial thoracic region. Observation of the motion at the thoracic spinous processes is carried out.
- **Extension:** Dorsoventral extension or ‘hollowing’ of the thoracic spine can be induced by stimulating as mentioned above along the epaxial muscles. This can be performed bilaterally for extension or unilaterally for a combined extension/lateral flexion movement. Differences side to side can be noted.

Passive physiological movements

As for the cervical spine, these manoeuvres are passive-assisted movements (not true passive physiological movement tests) as the horse is not truly relaxed in the standing position.

- **For lateral flexion:** Use of the lateral flexion reflex can be localised to a given level of the thoracic spine by stabilising with one hand over the lateral aspect of the spinous process, and causing the horse to flex laterally. The amount of intervertebral lateral flexion can be compared between levels and then side to side. Depending on the size of horse and examiner, the horse’s pelvis may also be pulled towards the examiner to create a lateral flexion movement as an alternative to using the reflex. (Figure 8.16). Motion can also be assessed between ribs and rib angles with this technique.
- **For flexion:** Using reflexes described above, the horse can be encouraged to perform a ventrodorsal ‘lift’. The physiotherapist can palpate the relative ‘opening’ movements between the spinous processes and compare between levels.

Passive accessory palpation

- **For extension:** A dorsoventral translation can be applied in a direction perpendicular to the spinous process, to effect an extension movement between vertebral segments (or groups of vertebral levels). Care should be taken to be aware of the angles of the individual spinous processes, as cranial thoracic processes tend to override the body of the next caudal vertebra. T1–T15 spinous processes are angled dorsocaudally, change at the anticlinal vertebra T16 to an upright position, and are angled dorsocranially from T16–T17.
- **For lateral flexion/rotation:** A relative latero-rotatory translation of one vertebra relative to the next can be applied in variety of ways, and it is to be noted that lateral flexion is coupled with rotation in the thoracic spine (see Chapter 4 for definition).

Digital pressure may be used to ‘pull’ a spinous process towards you from the contralateral side, effecting a lateral flexion at that given level combined with an



Figure 8.16 Use of reflex to induce left lateral flexion at thoracolumbar spine. Physiotherapist localises the lateral flexion at desired level with hand.

ipsilateral rotation. Comparison between levels and side to side, regarding range and quality of motion should be performed.

Translation of the vertebral body via the spinous process from the ipsilateral side may achieve a relative movement between adjacent vertebrae – it is useful to provide a ‘counter’ stabilisation at the spinous process cranial or caudal.

- For rotation/lateral flexion: An obliquely, medially and ventrally directed glide applied over the costotransverse joint (between transverse process and tubercle on rib) may effect a relative rotation and lateral translation between vertebral levels in the thoracic spine. The quality of movement and end-feel should be compared between levels and side to side.

8.7.4 Lumbo-pelvic and sacroiliac/hip region

Palpation

The gluteal musculature in particular (note middle gluteal; extends as far cranial as the lumbar spine) should be palpated for symmetry, tone, thickening, swelling, tenderness and spasm, along with biceps femoris, hamstring group, tail head muscles, adductor and medial and lateral thigh muscles. The lumbosacral junction, tuber coxae, tuber sacrale, ischial tuberosity, sacral spinous processes, caudal verte-

brae, pelvic symphysis and greater trochanter should be identified – in particular, symmetry of the tubera coxae and sacrale, sacral spines and pelvic symphysis. The dorsal sacroiliac ligament is palpable running from tuber sacrale to abaxial surface of sacral spine. The sacrotuberous ligament is palpable medial and cranially to the ischial tuberosity.

Active movements

Active movements of the lumbopelvic and sacroiliac region can be observed during straight line gait (look for symmetry at walk and trot), tight circles, canter transitions, rein-back, walking up and down hills, and unilateral hind limb stance. Rounding reflex via the gluteal musculature can indicate ability to rotate the pelvis caudally and extension reflex can indicate ability to rotate the pelvis cranially.

Passive movements

The tail can be extended and moved laterally to ascertain tail head muscle tone, as well as comparison of motion side to side, as far cranial as the sacrum (one hand moves the tail, while the other palpates over the relevant vertebral level).

The hindlimb can be moved into protraction to assess relative caudal rotation of the pelvis on that side, and retraction to assess relative cranial rotation (Figure 8.17). It is difficult to differentiate between coxofemoral joint and pelvic motion when using the hindlimb to cause physiological movement, so these joints could be considered a functional unit. The limb can be adducted and abducted to assess relative motion of the hip in these ranges. Compare range and quality of motion side to side.

Passive physiological and passive accessory

- Ilium on sacrum: Cranial, caudal, and oblique rotations can be applied to the ilium around the sacrum as well as dorsoventral translations via the tuber coxae and tuber sacrale. The latter may be angulated laterally to follow the plane of the ilial wing. Compare range and quality of motion, and end-feel side to side (Figure 8.18).
- Sacrum on ilium – The sacrum can be translated laterally by stabilising the ipsilateral tuber sacrale and gripping the sacral spines and gliding towards the operator. Compare range and quality of motion, and end-feel side to side (Figure 8.19).

A dorsoventral translation can be applied to the sacrum, centrally, and a relative longitudinal distraction translation can be applied by ‘cupping’ the most anterior sacral spinous process and gliding the sacrum caudally.

8.7.5 Scapulothoracic articulation

Palpation

The borders and surfaces of the scapula including the supraglenoid tubercle, the dorsal scapular cartilage as well as the superficial muscles such as deltoid, supraspinatus, infraspinatus, triceps group, serratus ventralis, subclavius



(a)



(b)



(c)

Figure 8.17 Palpating relative iliosacral motion at tuber sacrale and sacrum via movement of hindlimb. (a) Neutral; (b) in protraction; (c) in retraction.



(a)



(b)

Figure 8.18 Assessing movement of ilium on sacrum (a) cranial rotation; (b) oblique rotation.

and trapezius, should be palpated and compared side to side. The muscles on the ventral thorax, that is the pectoral groups (ascending, transverse and descending), sternocephalicus and brachiocephalicus should also be palpated.

Active movements

The movement of the scapula on the thorax can be observed during varieties of gait, including changes of direction that involve adduction and abduction of the forelimb.



Figure 8.19 Lateral translation of sacrum relative to the ilia – physiotherapist's right hand stabilises against right tuber sacrale and left hand glides sacrum laterally towards the right.

Passive movements

Gliding or translatory movements of the scapula can be performed in craniocaudal, dorsoventral and abduction directions. The forelimb is elevated and flexed at the carpus, elbow and shoulder and the physiotherapist faces the scapula, holding the flexed forelimb and supporting underneath the elbow. The scapula is then translated as described above, in its plane against the thorax. Range and quality of movement is noted and compared side to side.

Relative flexion/extension excursion of the scapula can also be observed and/or palpated with full retraction and protraction of the forelimb. Relative abduction and adduction of the forelimb can provide an observable assessment for abduction/adduction excursion of the scapula.

8.7.6 Glenohumeral joint

Palpation

The greater and lesser tubercles of the humerus are readily palpated, and the joint line can be identified with the shoulder in flexion, applying a slight cranial force along the axis of the humerus. The biceps tendon, from supraglenoid tubercle via the intertubercular groove can be palpated.

Active movements

Active movements of flexion and extension can be observed during various gaits.

Passive movements

Passive physiological

The humerus lies at right angles to the scapula and may be further flexed, with the rest of the forelimb also flexed for ease of handling. The physiotherapist faces the trunk of the horse and applies flexion to the shoulder via the distal humerus. The integrity of the muscular support of the joint can be assessed by applying medial and lateral rotations to the humerus, via its distal end, and comparing end-feel and range of motion side to side. Extension of the glenohumeral joint is most easily assessed when combined with a caudal translation of the scapulothoracic articulation (see above).

Passive accessory

The forelimb is flexed for ease of handling with physiotherapist facing slightly cranially, palpating the glenohumeral joint line. Cranial and caudal translation of the humerus can be performed to assess joint range of motion (Figure 8.20). Medial and lateral rotations can be applied via the humerus (as above) and assessed via palpation of the joint line.

8.7.7 Elbow joint

Palpation

Medial and lateral epicondyles of the humerus, olecranon, lacertus fibrosus (crossing the flexor aspect of the elbow),



Figure 8.20 Assessing cranial translation of the humerus at glenohumeral joint – left hand is palpating the joint line, while cranial translation of humerus is applied along the humeral longitudinal axis via the physiotherapist's forearm.

brachialis and triceps may be palpated and compared side to side. Musculature distal to the elbow (muscles of the forearm) includes the extensor and flexor groups.

Active movements

Active movements of the elbow may be observed during various gaits.

Passive movements

Passive physiological

The elbow may be flexed passively and extended via the radius/ulna (with the carpus in flexion for ease of handling), using counter-pressure on the distal humerus. Medial and lateral movements may be applied to the joint at various angles of flexion and extension to test joint integrity. For all passive physiological movements, joint end-feel, quality and range of motion are compared side to side.

Passive accessory

With the elbow in a neutral flexed/extended position, a longitudinal force can be applied along the radius/ulna, and the relative translation palpated via the heel of the hand at the olecranon (humerus is stabilised distally). Medial and lateral translation can be applied, and joint end-feel, quality and range of motion are compared side to side.

8.7.8 Carpal joint

Palpation (including metacarpal region)

In extension (standing), the styloid processes of distal radius, extensor tendons and accessory carpal bone are readily palpable. In flexion, the radiocarpal joint space (which has greatest mobility) and mid-carpal joint space are able to be palpated on the extensor aspect, for relative range of motion and the presence of any joint distension. The joint line of the carpometacarpal joint may also be palpated on the extensor aspect. The palmar carpal ligament can be palpated on the flexor aspect, and just distally, the accessory (check) ligament of the deep digital flexor tendon. Distal to these structures, the superficial and deep digital flexor tendons should be distinguishable from each other. The 3rd metacarpal should be palpated for irregularities, as should the 2nd and 4th metacarpals (medial and lateral splint bones).

Active movements

Active movements of the carpal joint may be observed during various gaits.

Passive movements

Passive physiological

The carpus as a whole may be fully flexed, and mediolateral movements and medial and lateral rotations applied to the fully flexed position, via the metacarpal, to test joint integrity and range of motion. The carpus is in extension in standing, but overpressure may be applied with the



Figure 8.21 Assessment of carpal extension via overpressure.

forelimb in protraction, and via application of force over the flexor surface of the carpus (the physiotherapist may differentiate the articular rows) (Figure 8.21). Joint end-feel, quality and range of motion are compared side to side.

Passive accessory

In flexion, cranial translation of the proximal carpal row on the radius may be performed. The physiotherapist faces cranially and supports the metacarpal and applied translation with thumb or web of hand. The translation may be biased towards the radial carpal bone or ulna/intermediate carpals, and the amount of flexion of the carpal joint required to access the proximal carpals varies between horses. Joint end-feel, quality and range of motion are compared side to side.

The amount of appreciable motion at the middle carpal joint and carpometacarpal joint make accessory translation a difficult task. As extension is the close-packed position of the carpal joint, translation of the carpals in a caudal direction is also a difficult task.

8.7.9 Metacarpophalangeal joint (fetlock)

Palpation

The distal 3rd metacarpal, proximal sesamoids, and proximal phalanx (PI) should be palpated for irregularities and compared side to side. Proximally and palmolaterally the interosseus ligament may be identified. The palmar ligament, collateral and straight sesamoidean ligament may be palpated at the palmar aspect. Distension of the joint capsule ('wind puffs') may be palpated between the interosseous ligament and the sesamoid bones.

Active movements

Active movements of the metacarpophalangeal joint may be observed during various gaits.

Passive movements

Passive physiological

The joint is extended in standing, thus may be further flexed in non-weight bearing, and overpressure applied to assess the end-feel and quality and range of motion, and compared side to side. Even though the shape of the joint restricts the motion to flexion–extension, medial and lateral rotation may be applied to the joint in various flexed positions to assess the integrity of the joint. Joint end-feel, quality and range of motion is compared side to side.

Passive accessory

A relative cranial translation of the proximal phalanx may be performed upon the metacarpal. The physiotherapist may face cranially or caudally, and stabilising the distal metacarpal, apply the cranially directed translation close the joint line of the proximal phalanx. Joint end-feel, quality and range of motion are compared side to side.

8.7.10 Proximal interphalangeal joint (PIP) – PI/PII – pastern joint

Palpation

Palpation of the bony surface of PI and PII, and along the coronet band for irregularities can be performed and compared side to side. Axial and abaxial ligaments may be palpated along the palmar aspect of PI.

Active movements

Active movements at the PIP joint may be more difficult to observe than more proximal joints when observing gait, but should be compared from side to side.

Passive movements

Passive physiological

Passive flexion and extension can be assessed by stabilising PI and flexing or extending PII. Overpressure may be applied in the flexion–extension direction, and also medial and lateral rotation, and abduction/adduction applied at various angles of flexion–extension. Joint end-feel, quality and range of motion are compared side to side.

Passive accessory

Cranial translation of PII relative to PI is achieved by stabilising PI and gliding PII in a cranial direction holding close to the joint line (Figure 8.22). Accessory rotations may be delivered using the same stabilisation and handholds. Joint end-feel, quality and range of motion are compared side to side.

8.7.11 Distal interphalangeal joint (DIP) – PII/PIII – coffin joint

Palpation

The distal phalanx (PIII) is contained within and conforms to the interior of the hoof, so it is not readily palpable. The



Figure 8.22 Accessory translation of distal phalanx (P3) on middle phalanx (P2). Left hand stabilising proximal phalanx (with joint in neutral) and right hand-applied translation.

hoof should be observed and palpated for irregularities and compared side to side (Chapter 6).

Active movements

Active movements are difficult to ascertain by observation but are thought to mirror those of the PIP joint.

Passive movements

Careful stabilisation of the middle phalanx (P2) can allow a relative translation of the distal phalanx via the hoof. A relative cranial glide of P3 on P2 may be achieved, and medial and lateral rotation accessory glides can be assessed and compared, as for other joints, side to side.

8.7.12 Coxofemoral joint (hip)

Palpation

The greater (cranial and caudal parts) trochanter and the third trochanter, plus the musculature covering the region (gluteal muscles, tensor fascia latae, biceps femoris, semitendinosus, semimembranosus, adductors, pectineus, gracilis, sartorius, quadriceps femoris) should be palpated and compared side to side.

Active movements

The active movements of the hip joint as part of the lumbo-pelvic-hip complex should be observed during gaits (including movements which cause abduction/adduction of the hindlimb). (See also Section 8.7.4 on the lumbo-pelvic and sacroiliac/hip region.)

Passive movements

Passive physiological

The hindlimb can be moved into protraction to assess relative caudal rotation of the hip–pelvis on that side, and retraction to assess relative cranial rotation. It is difficult to differentiate between coxofemoral joint and pelvic motion

when using the hindlimb to cause physiological movement, so these joints could be considered as a functional unit. The limb can be adducted and abducted to assess relative motion of the hip in these ranges. Palpation of the greater trochanter or third trochanter during these hindlimb movements may give an indication of relative coxofemoral movement as opposed to that of the ilium/hemipelvis. Compare range and quality of motion side to side.

Passive accessory

Due to the depth of the coxofemoral joint, passive accessory glides are difficult to deliver. (See: passive accessories delivered via the ilium; Section 8.7.4)

8.7.13 Stifle (tibiofemoral and patellofemoral joints)

Palpation

The patella and the three patellar ligaments should be palpated – often these ligaments are more accessible to palpation when the stifle is in flexion. Medial and lateral femoral condyles may be palpated as well as insertions of musculature of the anterior, medial and lateral thigh.

Active movements

The active movements of the stifle as a whole should be observed during gaits, with particular attention to the presence of any patellar locking.

Passive movements

Passive physiological

The tibiofemoral joint may be flexed, as well as extended (both these movements are coupled with the hock due to the reciprocal apparatus – see Section 4.9), and medial and lateral rotation applied to the joint via the tibia to assess the integrity of the joint. Quality of movement, end-feel, and range of motion should be compared side to side.

The patellofemoral joint may be palpated as the tibiofemoral joint is flexed and extended to ascertain its pattern of passive glide in the femoral trochlea. It also may be palpated as the horse walks, to ascertain the quality of the movement. Mild locking can sometimes be identified in this way.

The proximal tibiofibular joint has little movement (there is no distal tibiofibular joint in the horse).

Passive accessory

The patella may be translated in the trochlea of the femur in a craniocaudal direction and a mediolateral direction.

8.7.14 Tarsal joint (hock)

Palpation

The muscles covering the tibia (craniolateral group and caudal group) should be palpated, as well as the medial and lateral malleoli, the calcaneal tuber and calcaneal tendon (gastrocnemius). Proximal metatarsal, fourth (lateral) and third (medial) tarsal bone may also be palpated. Superficial

and deep digital flexor tendons may be palpated on the flexor aspect from an area proximal to the calcaneal tuber. On the medial (extensor) aspect of the hock the cunean tendon (medial branch of tibialis cranialis) is palpable.

Active movements

Hock action is readily observed during a variety of gaits.

Passive movements

Passive physiological

The hock as a whole may be fully flexed (see Section 6.3.5 Manipulative tests), and compared side to side. Most of the motion occurs at the tarsocrural joint. Extension of the hock is coupled with stifle movements (see reciprocal apparatus in Section 4.9) and if full extension is independently available at the hock, then partial or full rupture of the peroneus tertius should be considered.

Passive accessory

It is difficult to apply translatory joint movements to the hock because the hock movement is coupled with stifle movement, and the 'stack' of tarsal bones and the tarsometatarsal joint allow little appreciable movement. Small amounts of mediolateral translation may be applied to the calcaneal tuber, which may reflect accessory lateral movement of the calcaneus.

8.7.15 Metatarsophalangeal joint and interphalangeal joints

See similar in forelimb: Sections 8.7.9, 8.7.10 and 8.7.11

8.8 Conclusion

This chapter has provided a summary of the key components of physiotherapy assessment for animals. The importance of an accurate history and the use of clinical reasoning, careful and logical motion analysis, and palpation skills have been emphasised in guiding selection of treatment. Physical examination of the main joints and soft tissues of the dog and horse vertebral column and extremities has also been outlined. Please refer to orthopaedic (Chapter 6), neurological (Chapter 7), manual therapy (Chapter 9) – and other chapters in this book to provide information necessary for a comprehensive assessment of the animal patient.

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9

Manual therapy

Lesley Goff and Gwendolen Jull

9.1 Introduction
9.2 Technical aspects of manual therapy
9.3 Manual therapy in practice
9.4 Dogs

9.5 Horses
9.6 Conclusion
References

9.1 Introduction

Manual therapy (or manipulative therapy) refers to the practice, within musculoskeletal physiotherapy, of therapist-applied passive or assisted active movement techniques for the management of pain and impairments in the articular, neural and muscle systems. Its use is based on a detailed assessment of the presenting pathophysiological features in the neuro-musculoskeletal system underlying the pain and loss of normal function, for the purposes of diagnosis and management. Manual therapy is a vital component of the multimodal management approach to musculoskeletal disorders within the field of musculoskeletal physiotherapy. Manual therapy complements other management strategies inclusive of therapeutic exercise, electrophysical agents, functional retraining, education, and self-management procedures. The passive movement techniques of manual therapy are used both as assessment and management techniques.

The theory and practice of manual therapy is well established in the management of human musculoskeletal disorders. This chapter will review the practice of manual therapy and how it may be applied to animals. While there can be some direct transfer of principles and practices from the human to the animal, there are obviously issues unique to the animal patient. For example, there are anatomical, kinesiological and functional differences, animals cannot be instructed to relax, unlike human patients. As a consequence of these factors, the animal physiotherapist is required to modify techniques. This chapter offers some suggestions on the manner in which techniques may be modified as well as providing some insight into the rationale and application of manual therapy.

9.2 Technical aspects of manual therapy

Passive mobilisation is often discussed in terms of effects on intra-articular and periarticular structures (joint surfaces, joint capsule, ligaments). The movements also affect extra-

articular structures (muscles, fascia and neuromeningeal tissue). In the application of manual therapy or passive movement, treatment techniques can be applied at different amplitudes, velocities and positions within a joint's range of movement (Maitland *et al.* 2006).

Manual therapy encompasses two predominant technique types:

- Joint manipulation
- Passive joint mobilisation

Joint manipulation is differentiated from joint mobilisation by the speed of application of a single movement (high velocity thrust). Passive joint mobilisation includes any manual therapy directed at joint or soft tissue structure that does not involve a high velocity thrust (Hurwitz *et al.* 1996). Passive joint mobilisation involves the rhythmical application of movement, which is performed at a comfortable speed (approximately two to three oscillations per second). Passive mobilisation as its name infers is traditionally performed as a passive technique, but evolution of the practice has seen the introduction of components of active mobilisation in combination with the therapist-applied passive technique (Mulligan 1995).

The movements performed passively include the joint's physiological movements as well as its accessory (or translatory) movements. Passive physiological movements refer to the rotations that occur around the joint's three axes and are performed by the physiotherapist, usually without the patient's active assistance. Passive accessory movements are the translations, which automatically accompany the rotations but, in contrast to the physiological movements, cannot be performed voluntarily by the patient. In a typical joint, translation of a joint surface upon another tends to involve a combination of rolling and sliding (MacConnail 1964). This is partly due to the concavo-convex nature of most joint surfaces (Lee 1995). The direction of translation is related to the plane of the joint and the direction of motion that the physiotherapist wishes to effect. Passive

physiological and passive accessory movements may be applied to both the spine and extremity joints in both humans and animals.

Either of the movement types can be performed in assessment or treatment. The physiological or accessory movements can be performed singularly or in combination (Edwards 1994), or the passive mobilisation may be combined with the patient performing directed active movement, termed, *mobilisation with movement* (Mulligan 1995). In mobilisation with movement (MWM), the translatory movement of a painful or restricted joint is performed passively by the therapist and the painful or restricted physiological movement is performed simultaneously. The physiological movement may be accomplished actively by the patient or in an assisted active or passive manner (Mulligan 1995). In the case of the animal, the physiological movement is accomplished by instigating an active assisted, passive movement, or reflex movement (Figure 9.1). MWM can be applied to both spinal and extremity joints. When applied to spinal joints, this manual technique is termed the *sustained natural apophyseal glide* (SNAG). Mulligan (1995) points out that the technique

applied to the joints in the spine is generally in the direction of the physiological movement that is restricted, and in the extremity joints, the sustained translation is often applied in a related but often different direction from the physiological movement.

9.2.1 Proposed physiological effects of manual therapy

There is a growing bank of research regarding beneficial effects of manual therapy with studies carried out using animal and human models. Randomised control trials in humans have provided evidence of the effectiveness of manual therapy for relief of pain and for an increase in function (Boline *et al.* 1995; Nilsson *et al.* 1997; Jull *et al.* 2002; Childs *et al.* 2004). Research in more recent times has also attempted to explain these beneficial effects on a physiological basis.

There have been many theories proposed to explain the mechanism of manual or manipulative therapy. Theories on the effect have ranged from biomechanical, for example, movement of the nucleus pulposus in a discal disorder (Haldeman 1978) to effects on the muscle system such as a reduction in paraspinal muscle hypertonicity (Herzog *et al.* 1999). The biomechanical effects were thought to correspond with an alteration in electromyographical activity and/or a temporary increase in degree of joint displacement due to a hysteretic effect (Herzog *et al.* 1999). There are biomechanical effects of the movement-based manual therapy; however, the evidence is not secure for a predominant biomechanical basis to explain the effect of manipulative therapy when used as a single modality. For example, Hearn & Rivett (2002) investigated the reported biomechanical effects of a cervical mobilisation technique – the sustained natural apophyseal glide. The technique was proposed to cause a repositioning of the articular facet as well as affecting the whole functional spinal unit (Mulligan 1995). Hearn & Rivett (2002) found no empirical evidence to support such a biomechanical effect. Likewise in a single case study with a similar technique applied to a subluxed carpometacarpal joint of the thumb Hsieh and colleagues (2002), using MRI imaging, were not able to measure any improvement in the positional fault, despite the patient achieving pain relief. Immediate gains in range of movement from a manipulation in an acute neck pain model were lost within 48 hours of treatment (Nansel *et al.* 1990), which highlights the need and supports the evidence (Gross *et al.* 1996) for a multimodal approach inclusive of exercise to assist in maintaining range of movement gains. In a randomised controlled trial, immediate reduction in quadriceps stretch reflex following passive and dynamic oscillations applied to the human knee lasted less than one minute (Newham & Lederman 1997). Thus, although biomechanical features are inherent in a movement-based treatment technique, they do not seem to explain in large part, the beneficial effects of manual and manipulative therapy techniques at this stage of research.

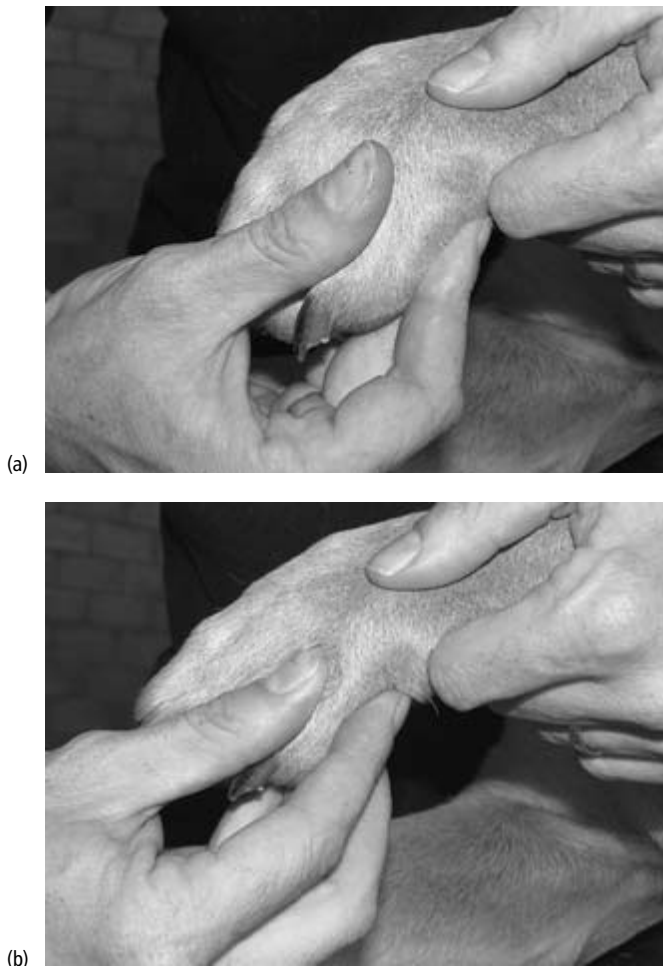


Figure 9.1 (a) Flexion of canine metacarpophalangeal joint; (b) MWM (flexion/external rotation) of canine metacarpophalangeal joint.

In more recent times there has been a shift toward investigations of neurophysiological mechanisms to explain the pain-relieving effects of manipulative therapy. Neurophysiological studies are investigating models of pain relief resulting from the afferent input and stimulation provided by the locally induced movement. It is thought that such afferent input may stimulate neural inhibitory systems at various levels in the spinal cord (Allen *et al.* 1984; Wyke 1985; Christian *et al.* 1988), as well as in higher centres in the central nervous system (Wright 1995). A growing body of evidence is showing that one mechanism could be through activation of descending inhibitory pathways via the lateral periaqueductal grey area of the midbrain suggesting that manipulative therapy produces a non-opioid mediated hypoalgesia (Vicenzino *et al.* 1994; Wright *et al.* 1994; Wright 1995; Vicenzino *et al.* 1998; Vicenzino *et al.* 2001). Recent evidence indicates that spinal manual therapy produces a treatment-specific initial hypoalgesic and sympathoexcitatory effect beyond that of a placebo or control condition (Vicenzino *et al.* 1998; Sterling *et al.* 2001). A concurrent motor response, a decrease in superficial neck flexor activity, was also noted in the study by Sterling *et al.* (2001) of manual therapy to the C5–6 segment, which would further support the proposal of activation of the periaqueductal grey region. Furthermore, in a series of studies of a manual therapy technique applied to the elbow for lateral epicondylalgia, Paungmali and colleagues (Paungmali *et al.* 2003a; Paungmali *et al.* 2003b; Paungmali *et al.* 2004), again confirmed the concurrent hypoalgesic and sympathoexcitation effects of manual therapy, and that the initial effect was not antagonised by naloxone, in concord with a non-opioid mechanism of action. Manual therapy has also been shown to produce mechanical not thermal hypoalgesia (Vicenzino *et al.* 1998). This implicates an endogenous noradrenergic, over a serotonergic mechanism – that is a non-opioid form of analgesia.

Manipulative therapy is likely to exert its effects via multiple mechanisms. For example, Heikkila and colleagues (2000) showed some improvement in cervical proprioception (measured as joint position error) following manipulative therapy to the cervical spine. Further research is required to fully understand all mechanisms of effect of manipulative therapy techniques on the sensory, muscular, articular and postural control systems.

9.2.2 The broader scope of manual therapy

The use of manual therapy is not restricted to articular structures. The muscle and neural systems can also contribute to restriction of movement, pain and functional loss. The prevention and treatment of adaptations of muscle and connective tissue that impede the performance of motor tasks is a core part of manual therapy (Herbert 1995). Manual therapy can be utilised to effect muscle length changes via stretching, various massage, or soft tissue techniques. Stretching may be either sustained,

static, or delivered via hold–relax or ‘proprioceptive neuromuscular facilitation’ (PNF) techniques. PNF techniques involve lengthening of target muscles and contraction of opposing muscle groups (Chalmers 2004). This involves a voluntary contraction at command of the therapist, thus sustained or static stretching of muscles and soft tissue, as a technique, may be more applicable to animals than PNF techniques. A recent review of the literature suggests that the effects of mobilisation of muscle via sustained or static stretch, that is, a lengthening of the muscle, is achievable because of the viscoelastic properties of muscle (Chalmers 2004). This may partly explain the efficacy of other soft tissue manual techniques such as trigger point therapy and myofascial release, both of which involve applying sustained pressure to the muscle and connective tissue.

The other soft tissue system that is viscoelastic and must slide and extend in response to the body’s movement is the neural system. The neural system is innervated and of itself can cause pain if its movement is impeded or the neural structures are irritated by movement (Elvey 1994; Elvey 1997; Butler 2000). The involvement of neural structures is elicited clinically in humans through the use of neural tissue provocation tests (Elvey 1994; Elvey 1997; Butler 2000), which involve a combination of movements of the limb that stress neuromeningeal structures to a greater extent than non-neural structures (Boland 1995). Although little evidence exists as to the physiological effects of neural mobilisation, Butler (2000) has hypothesised that these include dispersion of intraneural oedema, lengthening of neural tissue, improved intraneural blood supply and improved axonal transport. In human physiotherapy, neural tissue provocation tests are performed with standardised procedures, which are based on the pathways that the peripheral nerves take through the body, from emergence of the nerves from the intervertebral canals into the formation of the plexuses. Standardisation of neural tissue provocation tests in animal physiotherapy has been explored in the canine sciatic nerve (Babbage *et al.* 2006). The sequence proposed for the straight leg raising (SLR) test in dogs is hip flexion, stifle extension, hock flexion and extension of the digits. The cumulative increase in strain in the sciatic nerve recorded in this study is comparable to measurements in humans (Figure 9.2). Until further research on neural tissue provocation tests in animals is carried out, knowledge of anatomy of the neural plexuses of the thoracic and pelvic limbs in the quadruped will guide the physiotherapist in the application of neural tissue provocation tests in animals.

Mobilisation of neuromeningeal structures invariably imparts movement to the physiological mechanical interface, which is the tissue of the nervous system that can move independently from the system (Butler 2000). The interface may include muscle and fascia, ligaments and joints. Pathological interfaces may include scar tissue, osteophytes, ligamentous swelling or oedema (Butler 2000).



Figure 9.2 Neural provocation tests of canine sciatic nerve – hip flexion, stifle extension, hock flexion and digit extension (Babbage *et al.* 2006).

9.3 Manual therapy in practice

9.3.1 Assessment

Assessment of the movement impairment in the articular, muscle and neural systems as well as the reactions to the movement test, in terms of pain response, tissue resistance and muscle guarding, is fundamental to guiding the application of manual therapy techniques. Specific tests are used to examine each system.

With respect to the articular system, both active movements and functional performance are assessed in the first instance. This is followed by a passive examination of the joint's movements. Both passive physiological and accessory movements of the joint are examined in all directions. Physiotherapists using passive physiological movements in the vertebral column of animals need to be aware that different spinal levels demonstrate varying ranges of motion. Range of movement varies between species and is related to biomechanics of the spinal column (Chapter 4). For example, there is minimal intervertebral lateral flexion available in the equine lumbar spine, owing to the size of the transverse processes and the presence of extra articulations between the transverse processes (Denoix 1999). In the limbs, there is variation in range of motion and resting positions of the peripheral joints, which is breed or species specific. For example, the resting weight-bearing position of wrist (carpus) is more extended in dogs such as Collies compared with dogs with more upright carpi such as Boxers.

In the tests of physiological and accessory movements, the physiotherapist assesses:

- The range, quality and end-feel of movement that result from the gentle manual forces applied to the joint during the test
- The pain response of the patient to the test

A thorough knowledge of the normal patterns and ranges of movement available at a joint or body segment provides the basis for decisions of abnormalities of movement (Lee 1995). The results of the passive movement examination in terms of the direction of motion loss or instability, the nature of the resistance perceived (tissue resistance of muscle spasm) and the nature and intensity of the pain provoked, direct the selection and application of manual therapy techniques. Passive movements can also be examined with simultaneous performance of an active movement. Assessment for the potential efficacy of an MWM as a treatment involves the reduction of pain or improvement in the range of a joint motion, when the passive translation is applied as the physiological movement is undertaken.

9.3.2 Reliability

It is important that assessment techniques are reliable. Inter-rater reliability refers to the ability of raters to assign scores in a consistent fashion and is determined when two or more raters judge a given event at the same point in time (Domholdt 2000). Studies of inter-therapist reliability of spinal joints have indicated that judgements of joint stiffness or motion by experienced manual therapists have poor to moderate inter-rater reliability (Maher & Adams 1994; Smedmark *et al.* 2000; Hoving *et al.* 2005). Judgements that include a pain response (pain provocation tests) however, have shown good to excellent reliability (Potter & Rothstein 1985; Maher & Adams 1994; Jull *et al.* 1988; Jull *et al.* 1997; Hoving *et al.* 2005). The studies regarding inter-rater reliability and pain response involve human subjects verbalising the presence of pain during a manual assessment technique. Animals give various signals as pain responses, which include vocalisation, but this is not always the case. This poses a challenge for physiotherapists in animal physiotherapy. It is suggested that animal physiotherapists need to become very sensitive with their manual palpatory skills, as they cannot rely on the pain response alone in assessment and treatment. There is some evidence that it is the complexity of biological tissue that is responsible for the poor reliability in joint stiffness and movement judgement. Physiotherapists have been shown to be accurate and reliable testing stiffness and very small ranges of motion on non-biological models (Latimer *et al.* 1998; Goff *et al.* 2004).

9.3.3 Selection of manual therapy technique

The selection of technique evolves via clinical reasoning processes that require careful consideration of hypotheses such as sources of symptoms, precautions, prognosis and further contributing factors (Butler *et al.* 1994). Information to form the hypotheses is derived from questioning, handling skills and previous clinical reasoning experiences that must include due attention to what is known about the presenting pathoanatomy, pathophysiology and psychological traits of the animal. Thus, it goes without saying

that a sound knowledge of the anatomy, physiology, conditions and diseases which can affect the animal, as well as animal behaviour, is crucial in such reasoning processes. Close liaison with veterinary professionals is important for knowledge of disease processes and when further examination by a veterinary surgeon is indicated.

There are no recipe approaches for the selection and application of manual therapy interventions in animal or human treatment. Neither is there one correct treatment approach for a given patient's presentation or condition. There are several manual therapy approaches within physiotherapy and recent randomised clinical trials in humans have shown that they have similar efficacy (Rosenfeld *et al.* 2000; Hoving *et al.* 2002; Jull *et al.* 2002). The skill of the physiotherapist is to select the most appropriate manual therapy intervention for the particular patient, based on the clinical examination of that patient's pain and movement impairments.

Selection of a specific passive mobilisation technique is based on several factors (Maitland *et al.* 2006). These are inclusive of the specific direction of movement impairment, the limiting features to that movement (pain, tissue resistance or muscle guarding) and the response to the manual tests that have been applied. The technique selected usually relates to the physiological direction (or its associated translatory movement) with the most restriction. In addition, the pain response to that movement will guide how gently or firmly the technique can be applied. The selection and application of the initial passive mobilisation technique is further guided by assessment of the responsiveness to the technique in the examination (Maitland *et al.* 2006). During the reassessment phase of the examination, if an animal's condition is improved following a passive mobilisation technique, then it is likely that implementing such a technique in treatment will be beneficial. Similarly if the animal's condition is improved following deep muscle palpation or testing of extensibility in a certain

muscle or group of muscles, then it is possible that techniques designed to lengthen or relax those tissues may be of benefit. Neural tissue provocation tests, as discussed, are an important component of the physical examination. If the animal's active movements, pain responses, or other signs such as joint signs are improved after such tests, then selection of gentle neural tissue mobilisation techniques may be warranted at some stage in the management.

9.3.4 Safety

Safety in initial selection and application of manual therapy techniques is a prime consideration for the welfare of the animal. Contraindications to the use of manual therapy techniques are presented in Table 9.1 and precautions are presented in Table 9.2. Precautions and contraindications to manual therapy assessment and treatment are related to certain syndromes or diseases in animals, which can be breed or species specific. This list is not exhaustive. During the assessment, these should be considered as possible breed- and signalment-related red flags. It is the physiotherapist's responsibility to assess each case and to request more information or refer back to a veterinarian for further tests if there are concerns.

As well as the safety of the animal, the safety of the physiotherapist is also paramount. Physiotherapists must be able to apply techniques without the risk of being kicked, bitten or crushed. Injuries from large animals such as horses can be life threatening. Handling animals, large and small has occupational risks for physiotherapists and other animal handlers. Physiotherapists must always be aware of optimal positioning for themselves. Manual therapy techniques can be physically demanding and require postures and positions that are not potentially causative of injuries.

9.3.5 Treatment dosage

Ongoing reassessment as the treatment is progressing is of fundamental importance. Reassessment is the key to delivering

Table 9.1 Contraindications to manual therapy

Contraindication	Species	Breed/signalment	Action
Severe splinting spasm often seen at a spinal segment	Canine Equine	Spinal fracture, any breed. Chondrodysplastic dogs breeds for thoracolumbar lesions	Diagnostic imaging or neurological testing by veterinarian. Avoid manual testing of affected segment
Ataxia ± upper motor neurone signs – usually indicates spinal cord disease	Canine Equine	Cervical vertebral malformation in Doberman, Weimeraner, Great Dane (canine) Thoroughbred, Warmblood (equine). Atlantoaxial/occipital deformity in Arabian horses	No treatment should be initiated without accurate diagnosis – further veterinary and diagnostic imaging investigation
Severe progressive pain or non-weight bearing in any limb that does not seem to follow a mechanical pattern on manual testing	Canine	Osteosarcoma of the long bone, particularly in canine breeds such as Rottweiler. Sites of predilection include proximal humerus and distal femur, but can be anywhere	Further veterinary and diagnostic imaging investigation
Non-weight bearing in horses – infection or fracture are main two causes	Equine	Any	Precaution – diagnostic imaging. If hot, swollen peripheral joint, may be infective arthropathy – a veterinary emergency

Table 9.2 Precautions to manual therapy techniques

Precaution	Species	Breed/signalment	Action
Brachycephalic dogs – may have underlying cervical vertebral malformation	Canine	Basset Hounds, Pugs, Pekinese	Active movements only for cervical spine followed by very careful palpation
Lethargy, vomiting, unexpected deterioration of hind limb weakness	Canine	Any	Any medical problem, which may arise, needs to be referred back to veterinarians – these could indicate metabolic, toxic or organ disease unrelated to the initial complaint

the optimal dosage of treatment. It provides guidance as to the effectiveness of the particular technique chosen and, importantly, provides the direction for the progression of technique as the animal improves. If any improvement within or between treatments does not continue, this indicates that the selection of the particular technique needs to be reappraised and another technique chosen. Continual assessment for responsiveness is integral to the practice of manual therapy. A lack of responsiveness to a technique, or an aggravation of the condition can also be a useful guide as to what may help to ease symptoms. Past experience and expected responsiveness of an intervention can also guide the physiotherapist in judging whether the animal has a condition that will or will not respond to manual therapy.

Ideally, the animal should receive as many treatments as are required to effect the change desired by the owner, within the limits of the presenting condition, the animal's age and activity/skill requirements, and the length of time the condition has been present. Improvement in function should be measured and recorded between sessions. If functional improvement does not occur, then the physiotherapist should reappraise the treatment approach. Other factors such as possible reinjury, compliance with home programmes by the owner, activity levels and other interventions may need to be considered.

It must be stressed that manual therapy is one component of the multimodal management programme provided to the animal. Within the multimodal programme, specific exercises to facilitate the muscle system and functional muscle lengthening exercises, as required, are conducted with the animal. Such procedures capitalise on the improved mechanical function of the animal following a manual therapy intervention and facilitate movement patterns and muscle groups, which may have been inhibited by pain or altered movement (see Chapter 14). A home programme is designed to be carried out by the owner and this is an essential component of overall management.

9.3.6 Considerations in manual physiotherapy for animals

There are both similarities and differences between providing physiotherapy for animals and physiotherapy for humans. From a manual physiotherapy perspective, the concepts of manual assessment and treatment are similar

when dealing with human and animal patients, but there are some practical differences. For example, when noting a pain response, the physiotherapist should be very aware of the body language of the animal. Animals, particularly dogs, may vocalise when experiencing pain from palpation or movement, but often the response is to move away from the application, and sometimes to bite, kick or cause deliberate or unintentional harm to the physiotherapist or handler (Chapter 2). This points to the need for the palpation or movement to be applied with care. With such care, subtle changes can be detected in the segment that the physiotherapist is examining. Such changes may include tissues retracting or moving away from palpation or pressure, a mild spasm reaction or change in surrounding musculature or soft tissue. It is essential for a manual physiotherapist treating animals to develop their palpatory and tissue-perception skills to be able to detect subtle changes and response of tissues, especially as a pain response is not always indicative of movement dysfunction.

9.4 Dogs

Dogs, like humans can be examined in functional positions as well as recumbent positions. The advantage of recumbency is that joints and soft tissue may be palpated or moved through a range of motion in a relaxed state. Being able to perform passive accessory movements as part of manual assessment gives the physiotherapist information regarding movement occurring at the joint's surface, in relation to the movements that take place during physiological movements. The degree of relaxation available when undertaking manual assessment for a dog depends on the arousal or distress levels of the dog and its willingness to be in a recumbent position. Unlike in a human patient, the therapist cannot request a dog to relax in a prone position on a treatment couch, although practice and calm, skilful handling may induce relative relaxation in a dog. Owner cooperation here is paramount. The canine physiotherapist may instruct the owner in appropriate mannerisms and handling to maximise the cooperation of the canine patient. Assistance from a veterinarian regarding sedation or anaesthesia may occasionally be required to complete a full manual examination on a dog that is very anxious about the examination.

It is good practice to conduct all relevant tests in one position to maximise comfort of the dog before moving on to the next position. For example, an efficient approach might be to first observe gait and active movements, then carry out manual testing procedures which may be done in standing, such as preliminary neurological tests and certain passive physiological tests. The assessment can then be conducted in recumbency for the tests, such as reflex testing, passive accessory spinal or peripheral movements and neural tissue testing. Manual testing must be tailored to that to which the dog will comply. Most manual tests can be modified for position or an alternative test can be devised to minimise distress to the dog. Use of a checklist or consistent examination routine will minimise the chance of omitting parts of the examination.

Breed variation is another consideration when performing a manual musculoskeletal examination of a dog. There are vast differences in size and conformation within the canine species, and the physiotherapist's examination may be required to be tailored accordingly. An example is palpation of thoracic spine segments. Palpation of individual spinous processes will not be as specific in a small dog such as a Miniature Fox Terrier compared with a middle- or large-sized dog. A skilled manual physiotherapist should be able to modify the examination to account for this, without missing vital information. The canine physiotherapist should be familiar with the conformational differences that exist between dog breeds, as well as related difference in gait.

It is not possible in this section to describe all manual therapy techniques that may be used in the management of dogs, but the following examples illustrate a range of techniques and importantly, illustrate modifications for animals.

9.4.1 Extremity joints

As for human extremity joints, the joints of dog forelimbs and hindlimbs can be moved through their passive physiological and accessory ranges of motion for purposes of both assessment and treatment. Principles of manual handling for both passive physiological and accessory techniques, ordain that the proximal segment is usually stabilised and the distal segment moved. The physiotherapist's hands support the limb and joint as close to the joint line as possible, without blocking movement at the joint. In relation to the accessory (translatory) movements, the physiotherapist needs to be aware of the plane of the articular surfaces and the direction of glide of the moving segment. The planes of the articular surfaces are listed in Table 4.1.

Practical descriptions of manual therapy techniques for assessment and treatment of the stifle joint are presented as an example of the process of adaptation for dogs. These concepts can be applied to any peripheral joint in the dog, where the functional anatomy and biomechanics of the joint are understood. These techniques may be applied to the dog in any position, but a position of lateral

recumbency is ideal to allow manual handling of joints and relative relaxation of the dog.

Techniques for the canine stifle joint

Passive physiological movements of tibiofemoral and patellofemoral joint complex

The physiotherapist stabilises the femur with one hand while the other hand grasps the proximal tibia. The stifle may be flexed or extended to the end of the available physiological range, or the movement is ceased at a certain point in range in response to pain. In assessment, the end-feel and pain response is noted and compared with the other side. The joint may be further stressed if more information is required by flexing or extending the joint in differing ranges of hindlimb protraction or retraction. This may be done with dog recumbent or standing.

Ligamentous integrity of tibiofemoral joint

Ligament tests should be performed in lateral recumbency to ensure the dog is as relaxed as possible. The therapist tests ligamentous integrity by applying medial and lateral stresses (testing joint capsule and collateral ligaments) with the stifle in neutral and then various positions of flexion and extension. The cranial cruciate ligament test involves a cranially directed draw of the tibia upon the femur, with stifle flexed approximately 120°. The physiotherapist faces cranially, grasping the distal femur, with the thumb stabilising over the lateral femoral condyle and index finger palpating close to the joint line cranially. The other hand grasps the proximal tibia, with the thumb stabilising via the lateral condyle of the tibia and index finger palpating close to the joint line at tibial tuberosity. The hand stabilising the tibia performs a cranial translation of the tibia upon the femur. Index fingers palpate the distance travelled cranially by the tibial tuberosity relative to the distal femur (joint line), and the physiotherapist notes the quality of end-feel (Figure 9.3). The test should be compared with the other



Figure 9.3 Cranial drawer test of canine stifle.

stifle. Sedation may be required to attain a true passive cranial drawer test.

Passive accessory (translation) movements

1. Patellofemoral joint: The patella is grasped between the index finger and thumb, and may be translated medially and laterally or dorsoventrally in the trochlea of the distal femur. A comparison of the relative amount of lateral and medial excursion should be made, and the directions of glide compared overall with the contralateral joint. Patella translations or glides may be performed in various tibiofemoral joint angles.
2. Tibiofemoral joint: The therapist grips the distal femur and the proximal tibia with both hands close to the joint line. The primary accessory movement glide or translation of the tibia on the femur in relation to physiological flexion–extension will be in a cranial and caudal direction. This can be performed in various angles of flexion–extension. Other accessory movements or glides that are assessed at the tibiofemoral joint, include mediolateral glides, internal and external rotation of the tibia on the femur and distraction–compression of tibia on the femur. Again, a judgement is made regarding quality of the translatory movements and/or pain response and responses compared with the contralateral side. Crepitus or meniscal clicks may be felt if there is degenerative change or meniscal pathology. To further assess the medial and lateral compartment of the tibiofemoral joint and the associated menisci, a test similar to McMurray's test in the human can be applied.

Treatment decisions regarding the direction of treatment movement and dosage are based on the assessment findings of range, tissue resistance and pain response. For example, if there has been primarily a pain response to passive testing, this directs that treatment by either passive physiological or accessory movements should be delivered within a pain-free range. In the tibiofemoral joint, this may be gentle passive flexion or extension mobilisation, a mid-range translatory glide in a neutral position of the stifle or the stifle position is found that allows a more pain-free delivery of either the passive physiological or accessory movement. This could involve positioning into directions other than the main physiological movement, for example; flexion of the joint in slight internal rotation of the tibia or reducing neuromeningeal tension by slightly ipsilaterally, laterally flexing the thoracolumbar spine.

If passive testing has revealed that stiffness or restriction is the main problem, then the technique chosen may be to end of range. Techniques to improve flexion or extension include: end-range passive physiological movements into flexion or extension, applied in a rhythmical oscillation at a comfortable speed; or using a passive accessory mobilisation, stabilising the femur and translating the tibia upon it

in a craniocaudal direction towards end of range, with the stifle in a physiologically flexed or extended position.

An MWM may involve holding the tibia or patella in a position that allows greater flexion or extension to be achieved (rotation, deviation, compression or distraction for tibia and mediolateral or dorsoventral glide for patella) and then applying the passive physiological or accessory movement.

The treatment can be progressed in several ways: the stifle can be held in greater ranges of the movement direction to be achieved, the mobilisation can be performed more towards end range or at end range; neuromeningeal tension may be superimposed on the joint; or number and/or vigour of mobilisation repetitions increased.

9.4.2 Canine vertebral joints

Passive physiological and translatory movements of the vertebral joints are likewise used in both assessment and treatment. The orientation of the facet (zygapophyseal) joints directs the movements that can occur at each level of the vertebral column, thus it is important for the physiotherapist to be aware of their anatomical orientation. Intervertebral discs will also affect motion available at a given vertebral level (see Chapter 4). The example presented here will be passive movement assessment of the canine mid-thoracic spine. With an understanding of anatomy, these principles can be applied throughout the vertebral column. Passive movements to any area of the vertebral column can be applied to the dog in sitting, standing or recumbency (lateral recumbency or supine).

Assessment of joints of the mid-thoracic spine (T7–8)

The facet joints of T1–9 tend to be orientated in a transverse plane, with the cranial articular processes facing dorsally and the caudal processes facing ventrally. The main movement available from T1–9 is lateral flexion, which tends to be coupled with contralateral rotation (see Chapter 4). The ribs attach to the vertebral bodies of two thoracic vertebrae and the intervening disc to form the costovertebral joints (e.g. the 2nd rib attaches to T1 cranially and T2 caudally). These joints have a small role in limiting motion in the thoracic spine. The costotransverse joints attach to the transverse process and the rib level corresponds to the vertebral level. Thus passive physiological and accessory movement examination of, for example, the T7–8 motion segment, involves the facet joints, intervertebral joints and costotransverse joint of T7 and T8 plus the costovertebral joints of ribs 6, 7, 8 and 9. During palpation, it should also be noted that the spinous processes of this section of thoracic spine overlap the body of the next most caudal vertebrae.

Examination of passive physiological movements

1. T7–8 (R) lateral flexion with dog standing: The physiotherapist is positioned standing over the dog and faces the dog's head. Depending on the size of the dog, the T8

level is stabilised with the right hand over the side of the vertebral body or with the thumb on the spinous process. The dog's trunk from T7 cranially is guided into right lateral flexion with the left hand down to the T8 segment. The right hand palpates for closing of the vertebral body and ribs on the right side and the therapist notes range of motion, quality of motion, and end-feel of the movement. Alternatively the physiotherapist may face the tail of the dog and, stabilising T7 with the left hand, move the trunk caudal to this level into right lateral flexion.

2. The examination of T7–8 (R) lateral flexion in left lateral recumbency: The physiotherapist faces the dog's ventral trunk. The T8 spinous process is stabilised with the left hand and the dog's upper trunk is lifted into right lateral flexion. The stabilising hand palpates either opening or closing movement between T7 and T8 just lateral to the spinous process. Alternatively, the spinous process of T7 is stabilised with the right hand and the dog's lower trunk is lifted into right lateral flexion. Palpate with the right hand as described above (Figure 9.4 a and b).



(a)



(b)

Figure 9.4 (a) Passive physiological right lateral flexion of T7–8, stabilising T7 and moving T8; (b) Stabilising T8 and moving T7.

Examination of T7–8 segmental flexion and extension

The relative flexion–extension mobility of the T7–8 motion segment can be assessed by palpating the interspinous space of T7–8 with the dog in lateral recumbency or standing. In standing, the spine and rib cage are moved into relatively flexed and extended positions via the sternum and the ribcage. The relative opening or fanning out of the spinous processes, or rib angles (flexion) or the relative closing of the spinous processes and ribs is palpated. In lateral recumbency, the spinous process of T7 or T8 is palpated while either the caudal or cranial trunk of the dog is moved into flexion and extension. Again the relative opening and closing is palpated between T7 and T8 as for standing position. Passive physiological movements are compared with levels cranial and caudal to T7/8.

Accessory movement for right lateral flexion of T7–8 in standing or left lateral recumbency

The physiotherapist stabilises the spinous process/laminar region of T7 with the left thumb and use the right thumb to apply a lateral glide on the right side of T8 towards the left (Figure 9.5 a). The amount and quality of motion and end-feel are assessed. The glide may also be performed at the costotransverse joint and the accuracy of application depends on the size of the dog's vertebra. The lateral translations are compared with cranial and caudal levels and compared with motion in left lateral flexion.

9.5 Horses

As a general rule equine manual physiotherapy techniques will be carried out with the horse in standing. Horses are rarely assessed in recumbency, as this mostly requires the horse to be anaesthetised. Horses may be sedated by veterinarians for manual examination, but even when sedated the horse can remain in the standing position. As the postural muscles of the horse are active when standing, true passive accessory movement testing is not possible for some joints, such as those of the thoracolumbar region. Palpation of some soft tissues in a relaxed state may not be possible. Modified passive physiological joint movements can be carried out in standing.

Horses requiring physiotherapy are generally accustomed to being handled by owners, farriers and veterinarians, so are quite receptive to manual physiotherapy examination. Great care must always be taken with regard to safety of the physiotherapist at all times. Even under sedation horses may deliver accurate kicks. Owner cooperation regarding handling of the equine patient is paramount to the safety of the physiotherapist.

9.5.1 Extremity joints

Passive physiological movements may easily be performed on equine limb joints. The approach to accessory/translatory joint movements of the equine limb joints however differs slightly from that of the canine, as some of the joints,



(a)



(b)

Figure 9.5 (a) Right lateral flexion passive accessory translation of T7–8 (left thumb is applying translation to T8 spinous process relative to T7); (b) Hand position for passive accessory translation in caudal thoracic vertebral column.

especially the carpus and hock, are stable and the small flat bones, which comprise part of those joints are difficult to palpate. The coxofemoral joint is also difficult to approach in a translatory manner owing to its coverage by large musculature. The example of the equine fetlock (metacarpophalangeal joint) will be used, but principles can be applied to all peripheral joints. As equine limbs can be heavy and dangerous if the horse strikes or kicks, the physiotherapist must be careful with positioning at all times.

Techniques for the metacarpophalangeal (fetlock) joint

Passive physiological movements of equine fetlock joint – flexion and extension

For the technique to be performed in a safe manner, the physiotherapist faces the caudal end of the horse and picks up the hoof. For the assessment of flexion, the toe or front of the hoof is grasped and the fetlock is fully flexed. Any pain response, range, quality and end-feel of the movement is assessed. When flexed, the fetlock has accessory lateral and rotatory movements (see Chapter 4). To further stress the joint, medial and lateral stresses are added to the flexed



Figure 9.6 Extension with overpressure for equine metacarpophalangeal (fetlock) joint.

fetlock. The stress can be applied at various angles of flexion and the amount of medial versus lateral motion is noted and compared. The same process is conducted for medial and lateral rotatory accessory movement. All movements should be compared with the contralateral fetlock.

The fetlock is positioned in an extended position when the horse is weight bearing, however, extension can be further stressed in the following way. The forelimb is picked up and brought into a protracted position. The fetlock is extended further via the toe of the hoof. Overpressure may be applied to the dorsal surface of the fetlock, and the therapist assesses the very slight accessory motion in extension (Figure 9.6). The movement should be compared to the contralateral fetlock.

Assessment of craniocaudal translation of the metacarpophalangeal (fetlock) joint

The physiotherapist picks up the hoof in a safe manner, holding fetlock in a semi-flexed position (non-close-packed position). The metacarpal is stabilised and the proximal phalanx is grasped close to the joint line. A dorsal glide of the phalanx is directed upon the metacarpal (Figure 9.7). The range of motion, quality of motion and the end-feel of the joint is assessed. The glide can be repeated in various degrees of flexion and also in relative extension. Medial and lateral rotations may be applied to the fetlock joint via the proximal phalanx. All translations should be compared with the contralateral side.

9.5.2 Equine vertebral joints

As for dogs, joints of the equine vertebral column may be taken through passive physiological and translatory movements in both assessment and treatment. Again, the orientation of the facet (zygapophyseal) joints directs the movements that can occur at each level of the vertebral column, the intervertebral discs will also affect motion available at a given vertebral level. The illustrative example



Figure 9.7 Cranio-caudal accessory translation of the equine metacarpophalangeal (fetlock) joint – left hand stabilising distal metacarpal; right hand directing glide of proximal phalanx.

will be the passive movement assessment of the mid-cervical spine. Passive movements are applied to the horse in standing.

Techniques for mid-cervical spine (C4–5)

Passive physiological movements, lateral flexion C4–5

Note this is a modified passive physiological movement, as the horse's cervical spine is not relaxed in standing. To examine the movement of lateral flexion at the C4–5 segment, the C5 level is stabilised with one hand. The horse's head and neck is guided by the therapist into lateral flexion by directly pulling the head around, or using an active stimulus such as a carrot. The range of lateral flexion and the end-feel are assessed via the stabilising hand and the motion is compared with the lateral flexion in the opposite direction. Lateral flexion can be tested in various positions of cervicothoracic flexion and extension.

Accessory lateral glide of C3–4

The physiotherapist stands facing the horse's rump and places one hand on either side of the vertebral body. The

forearms are positioned perpendicular to the horse's neck to enable a lateral glide to be delivered through the body of the vertebra. To test for left lateral flexion of C3–4, stabilise either side of C3 and glide the vertebral body towards the right (Figure 9.8). The same can be repeated for level C4.



(a)



(b)

Figure 9.8 Accessory lateral flexion glide of equine cervical vertebral. (a) Right hand directs glide to C4 towards the right; (b) Forearms are positioned perpendicular to vertebral body level.

The quantity and quality of the movement is assessed and the perceived motion is compared between levels.

9.6 Conclusion

Manual therapy is an essential part of the multimodal approach to assessment and treatment of musculoskeletal disorders in animal physiotherapy. The basic requirements for the application of effective manual therapy are, knowledge of the functional anatomy of the musculoskeletal and neuromeningeal systems, and how these systems are affected by pathology and movement dysfunction. Good palpation skills enhance the effectiveness of the manual therapist in both assessment of disorders and delivery of treatment techniques. Manual therapy may be the sole form of treatment chosen for a given condition, but is usually delivered in conjunction with other therapeutic modalities such as electrotherapy and functional exercise programmes.

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10

Principles of electrotherapy in veterinary physiotherapy

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10.1 Overview	10.6 Ultrasound therapy
10.2 Electrical stimulation of tissue	10.7 Evidence-based practice
10.3 Electrical stimulation for pain relief	10.8 Summary and conclusions
10.4 Electrostimulation of muscles	References
10.5 Laser therapy	

10.1 Overview

Electrotherapy has been an essential core of physiotherapy since the inception of the profession. Electrotherapy is an umbrella term to identify a range of treatments ranging from electrical stimulation for muscle strengthening, to the use of sound waves (ultrasound) and light (laser) to treat physical injuries.

There are four broad types of electrotherapy in routine clinical use, including:

- Electrostimulation (for pain relief and for stimulation of nerve and muscle, e.g. transcutaneous electrical nerve stimulation (TENS); neuromuscular electrostimulation (NMES); interferential therapy (IF))
- Ultrasound therapy
- Laser therapy
- Electromagnetic energy (e.g. pulsed electromagnetic energy)

Of these, the first three are most commonly used in animal therapy, and will form the basis of the current chapter.

Electrotherapy finds many applications in physiotherapy, however the three primary applications in animal therapy are:

- The treatment of pain
- Stimulation of muscle
- Promotion of tissue repair

However it is important to stress that electrotherapy is rarely used in isolation by physiotherapists, but rather is usually combined with other treatment approaches such as exercise, and manual techniques.

Detailed descriptions of the biophysical principles, methods of application, indications and evidence for electrotherapy are beyond the scope of the current chapter.

Rather, the following is designed to present the key principles of electrotherapy treatment as a background to the material on animal treatment in subsequent chapters. For a more detailed account of these modalities, the reader is directed to dedicated electrotherapy texts (e.g. Baxter 1994; Walsh 1997; Nelson *et al.* 1999; Kitchen 2002; McDonough & Kitchen 2002; Robertson *et al.* 2006).

10.2 Electrical stimulation of tissue

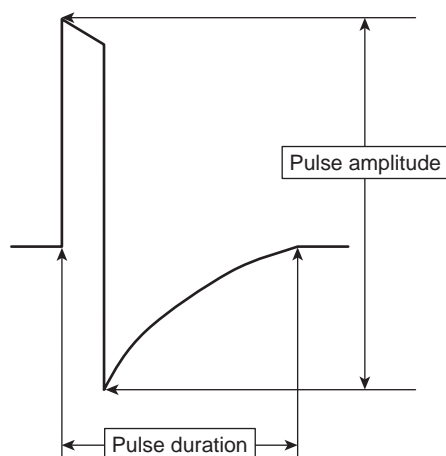
10.2.1 Basic principles

Effective application of low- or medium-frequency electrical currents in order to stimulate sensory and motor nerves to produce or to facilitate muscle contraction, or alternatively to produce pain relief, requires an understanding of the basic biophysical principles underpinning such application. The following presents an overview of this area.

10.2.2 Activation of peripheral nerves

In order to activate sensory or motor nerves, electrical stimulation should be applied in the 'low frequency range' (<250 Hz), using appropriate pulse durations and intensities powerful enough to activate the nerve. Manipulation of machine settings allows selection of the most appropriate parameters to produce the desired effects. While there are a variety of electrostimulation devices available for the animal therapy market, ranging from NMES for muscle stimulation/strengthening, to TENS for pain relief (and IF devices which are promoted for both of these applications), it is important to realise that the effects of therapy are essentially determined by the parameters of stimulation, principally: intensity, pulsing frequency and pulse duration (Figure 10.1).

It is also important to remember that most peripheral nerves are mixed and include (see Figure 10.2):



Notes:

- (i) The figure represents a schematic of a typical electrostimulation waveform: while it is asymmetric, the positive and negative components are equivalent and thus overall there is no net direct current effect.
- (ii) *Pulse Amplitude* represents the *intensity* of the stimulation; when the intensity of the stimulation is increased by the therapist, the amplitude is increased.
- (iii) *Pulse Duration* (coupled with the number of pulses per second) is a key parameter in determining the effects of stimulation (e.g. muscle stimulation or pain relief).

Figure 10.1 Pulse waveform with key parameters.

Type	Characteristics		
A α	Motor fibres	Diameter 13–20 μm Speed 80–120 ms^{-1}	
A β	Sensory fibres Mechanoreceptors Low-threshold	Diameter 6–12 μm Speed 35–75 ms^{-1}	
A δ	Sensory fibres Mechanothermal High-threshold	Diameter 1–5 μm Speed 5–35 ms^{-1}	
C	Nociceptors	Diameter <1.5 μm Speed <2 ms^{-1}	

Figure 10.2 Peripheral nerve: key characteristics.

- Motor nerve fibres (myelinated A α fibres)
- Large diameter (myelinated) sensory fibres, including Type II, A β ‘touch’ pressure receptors
- Thinly myelinated fibres, Type III (A δ)-mechanothermal receptors
- Unmyelinated nociceptive fibres subserving pain, Type IV-unmyelinated (diffuse pain)

Based upon these fibre types, a variety of effects are possible with electrical stimulation of a single nerve:

1. *Intensity of stimulation.* Large diameter, myelinated fibres have a lower threshold for activation, and therefore are most easily stimulated with lower stimulation intensities. In Figure 10.2 it can be seen that the largest diameter fibres are motor fibres, followed by the large diameter sensory fibres. However, in practice when surface electrodes are placed on the skin, normally these large diameter sensory fibres are activated first as they are closer to the stimulating electrodes than the motor nerves. Intensities are usually set using arbitrary units on most commercially available stimulators – typically with a ‘wheel’ control or similar – but intensity essentially refers to the magnitude of the current flow and is specified in milliamperes (mA).
2. *Frequency of stimulation.* This is specified in pulses per second – or Hertz (Hz) – and is limited physiologically by the absolute refractory period of the nerve, that is, how long it takes to recover from the production of an action potential, and be ‘ready’ for the next stimulus. It is longer for slowly conducting nerve fibres, which have a smaller diameter, and consequently these fibres respond preferentially to low frequencies.
3. *Pulse duration.* This is specified in milliseconds or microseconds; the shortest (microsecond) pulse durations preferentially stimulate sensory fibres, while the longer pulse durations preferentially stimulate motor nerve fibres. The reason why the large diameter sensory fibres are stimulated first can be explained by the relative proximity to the surface electrodes.

There is a variable threshold for stimulation of different types of nerve fibre, which depends upon the intensity (strength), pulse duration of the applied stimulus and the distance from the stimulating surface electrodes: this is illustrated schematically in Figure 10.3. Apart from the different slopes of the curve for different types of nerve, denoting sensory modalities versus motor activation, it is also important to note that for longer pulse durations, lower stimulus intensities are required to stimulate a nerve.

10.2.3 Application of electrical stimulation

For stimulation of muscle or nerve, two electrodes are applied. While, by convention, the black electrode is the cathode, and the red electrode is the anode, in the application of biphasic currents, polarity is not critically important. Selection of sites for stimulation or electrode placement will depend upon the desired effect of stimulation and include: over the bulk of the muscle, motor points, over peripheral nerves, acupuncture points, over spinal nerve roots, or directly to the painful area.

Selection of electrode type will depend upon a variety of considerations; the two principal types of electrode available are carbon electrodes, which require application of gel

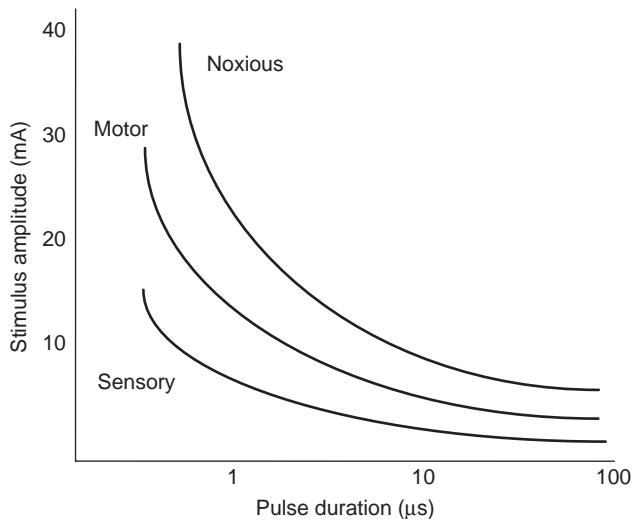


Figure 10.3 Nerve stimulation: relevance of pulse characteristics.

to transmit electrical current, as well as tapes for attachment onto the skin, and reusable, pre-gelled, self-adhesive electrodes. While the former are less costly, the latter tend to be more commonly used clinically, are less time-consuming to apply and are more convenient for application by the owner or carer.

10.3 Electrical stimulation for pain relief

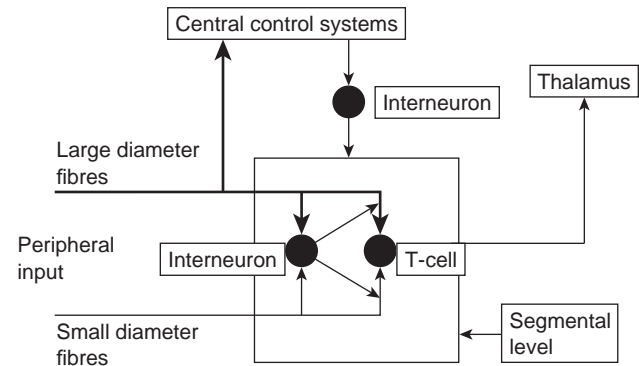
10.3.1 Overview

Electrical stimulation has been used for pain relief since ancient times; however, until the publication of the original paper outlining the pain-gate theory by Melzak and Wall (1965), electrostimulation for pain relief (or electroanalgesia) was not widely accepted outside the profession. Based upon initial work by Wall and Sweet (1967) (and others) so called transcutaneous electrical nerve stimulation (TENS) for pain relief has become a popular alternative to pharmacological methods of pain relief, particularly after the introduction of the first portable and affordable TENS units in the 1970s (see Walsh 1997).

While the term TENS could arguably be applied to any form of electrostimulation, its use has become limited to the description of electrostimulation for pain relief using small, compact portable units. Equally, while there are various types of electrostimulation device available, based upon different waveforms or methods of application, ranging from interferential therapy to H-wave therapy, the underlying principles are identical.

10.3.2 Mechanisms of action

While other physiological effects are possible with electrical stimulation (such as increases in blood flow and localised blocking of peripheral nerve fibres), the two main mechanisms by which electrostimulation produces pain relief are:



Notes:

- (i) The figure illustrates as a schematic the main elements of the pain control mechanisms underpinning pain relief using electrotherapeutic agents.
- (ii) Sensory input from the periphery reaches the relevant segmental level of the spinal cord, where this is transmitted to rostral levels of the nervous system via T-Cells or wide dynamic range cells.
- (iii) The relative degree of activity in large and small diameter fibres determines the activation of inhibitory mechanisms via interneurons in the substantia gelatinosa (also called SG-cells); these can block the passage of nociceptive information through the spinal cord. (Called segmental inhibition or pain-gating mechanisms).
- (iv) Parallel activation of central mechanisms stimulate descending inhibitory control mechanisms through the activation of opiate interneurons, which modulate the processing of nociceptive information through the spinal cord.

Figure 10.4 Pain control mechanisms.

segmental inhibition through pain-gating mechanisms, and via descending inhibitory mechanisms (see Baxter & Barlas 2002). The first of these relies on the selective stimulation of larger diameter fibres in peripheral nerves, which in turn helps to 'block' nociceptive activity in smaller afferents at segmental level (see Figure 10.4). For this, so-called conventional TENS is used, based upon higher frequency stimulation, coupled with longer pulse durations.

Descending inhibitory effects are also possible, based upon the release of endogenous opiate-like substances, in response to more intense levels of stimulation at lower pulse frequencies. Because of the similarity to (needle) acupuncture, this type of TENS is frequently termed acupuncture-like TENS. While the effects of opiate release are widespread, one of the primary sites of action is at the relevant segmental level, where opiate interneurons mediate the pain-relieving effects.

10.3.3 Indications: clinical use of electroanalgesia

Electrostimulation can be used for the relief of pains of various aetiologies, including:

- Acute pain associated with surgery (post-operative pain), ligament sprains, fractures and with labour pain

- Chronic musculoskeletal pain, including spinal pain and neuralgia

While electrostimulation can be used to treat most types of pain, it tends to produce best results in cases of more localised pain, of more moderate intensity, and where the site of pain is more superficial. More variable results are reported with more severe, widespread and deeply seated pain.

10.3.4 Principles of application

Before initial application, the patient should be adequately assessed and a diagnosis obtained. The patient should be checked for contraindications, and where possible the site or area of application should be tested and cleansed to improve conductivity. For initial treatments, time of application should be kept relatively short e.g. 15–20 min, to reduce any anxiety and to familiarise the animal with the sensations associated with stimulation. Treatment should generally be initiated with ‘conventional TENS’ as this is more comfortable and is therefore easily tolerated. At subsequent sessions, treatment time can be increased up to one hour at a time, and where appropriate, ‘acupuncture-like TENS’ can then be introduced or trialled.

Electrode placement (Figure 10.5)

TENS can be applied at a variety of sites, and some degree of experimentation may be necessary to determine the optimal site for electrode placement for effective pain relief. The most common site of electrode application is directly over the area of pain, with the electrodes placed to ensure that the majority of current, and therefore nerve stimulation, will pass through the site of pain. Alternatively, the TENS electrodes can be placed (proximally) directly over the relevant peripheral nerve that supplies the affected area, or over the relevant spinal nerve roots. In the latter case, the electrodes are placed lateral to the spine, so they are over the relevant spinal nerve roots. The effectiveness of both of these types of application is dependent upon an adequate knowledge of anatomy, especially relating to nerve supply of relevant structures (e.g. relevant dermatomes or myotomes for the animal patient).

Electrodes can also be placed over acupuncture, motor and trigger points. For therapists wishing to gain the benefits of acupuncture, use of acupuncture points for electrical stimulation can represent an effective and practical alternative to needles, which may be difficult in some animal patients, or limited by regulation or scope of practice. Alternatively, there is a high degree of correspondence between acupuncture and ‘trigger points’, which can also be used as an electrode placement site for electrostimulation, where these are identified upon palpation. Finally, motor points represent the best site for the treatment of muscle pain; when using acupuncture-like TENS, electrodes should be placed over the relevant muscle/appropriate myotome, and intensity increased until a muscle contraction is obtained.



Figure 10.5 Electrode placement for transcutaneous electrical nerve stimulation (TENS) in a dog 3 days after hemilaminectomy surgery. Four electrodes are used close to the surgical site. Note similar placement of electrodes could also be used for neuromuscular electrostimulation (NMES); however care should be taken to avoid the surgical site when using NMES settings.

10.4 Electrostimulation of muscles

The preferred term for electrical stimulation applied to effect contractions of muscle is neuromuscular electrical stimulation or NMES (McDonough & Kitchen 2002). The terms functional electrical stimulation (FES) or functional neuromuscular stimulation (FNS) are used to describe therapy using units that aim to ‘mimic’ the stimulation patterns of intact nerves, in cases where the nerve supply is damaged, e.g. peripheral nerve damage. Other terms, therapeutic electrical stimulation (TES) and electrical stimulation are sometimes used, but typically refer to devices that are designed to elicit sensory effects only.

It is important to note that the waveforms used in TENS and NMES are essentially the same; furthermore the current capacities (i.e. the electrical intensities produced by available units) are identical. The differing effects of each type of therapy are based upon the selection of appropriate stimulation parameters, and particularly, pulse duration, or width, and pulsing frequency; this is reflected in the design of casing and dials of units to allow manipulation of waveform characteristics (Figure 10.1; Table 10.1).

Table 10.1 Comparison of TENS and NMES

Electrostimulation	Effects	Pulse duration	Pulse amplitude	Pulse frequency
Neuromuscular electrostimulation	Muscle stimulation	<1 ms (200–600 μ s)	Sufficient intensity to provide muscle contraction	>50 Hz
Transcutaneous electrical stimulation	Pain relief	200 μ s 50 μ s	Sufficient intensity to provide muscle contraction 'Strong but comfortable'	<20 Hz >100 Hz

10.4.1 Mechanisms of action

Skeletal muscle fibres can be contracted by conscious (voluntary) or automatic control from the central nervous system; electrical currents can also 'artificially activate' muscle fibres by activation of the peripheral nerve to the muscle. There are different types of skeletal muscle fibres, which produce different types of muscle work. Type I are red in colour, slow twitch and relatively stable; they are activated at relatively low levels of muscle activity, over relatively long durations. Because of their aerobic metabolism, they fatigue slowly. Type IIB muscle fibres are white in colour, fast twitch, and produce muscle power. While they provide high levels of muscle activity, because of their anaerobic metabolism, they fatigue quickly and thus are only active for relatively short periods. Depending upon the pattern of the electrical stimulation, either or both types of fibre can be activated.

10.4.2 Indications

Electrical stimulation has been used for strengthening of normal muscle, however the evidence for electrical stimulation over voluntary exercise is limited. In atrophied muscles, the evidence from human studies is variable: in quadriceps and abdominal muscles, the evidence is conflicting, while there is some evidence of benefit in the muscles of the lumbar spine. Electrical stimulation has also been used for muscle stimulation in neurological damage, promoting motor recovery and strength, in the treatment of subluxations (in the early stages of recovery) and for the reduction of muscle spasticity. In animals, the primary use is the treatment of muscle atrophy, to underpin re-education of muscle function, and muscle strengthening (Figures 10.6 and 10.7). As in humans, muscle strengthening in muscle atrophy resulting from neurological lesions has also been successfully achieved with electrical stimulation. Suggested uses have also included strengthening of muscles and promotion of muscle recovery post-operatively, where resolution of oedema has also been reported; in such cases application near the incision site should be avoided (although TENS can be applied close to the wound for pain relief, as in Figure 10.5).

10.4.3 Principles of application

Before electrode application, the animal's hair should be clipped to lower resistance to passage of current, and



Figure 10.6 Electrode placement for neuromuscular electrostimulation (NMES) in a dog 3 days after hemilaminectomy surgery, electrodes are moved until appropriate muscle contraction is obtained, then taped into place for the treatment.

if possible, the area of application should be washed with soap and water (alcohol should be avoided). There are two techniques used to locate electrodes. The first is placement of a primary electrode over the motor point which can be located using a probe electrode, with the current pulsed at 1 Hz. Once the optimal site for muscle stimulation has been identified, this should be marked where possible with an indelible marker. A second dispersive or indifferent electrode must be placed elsewhere on the body part, at a convenient location near the muscle being treated. This electrode should be larger, so that the current density across it is lower and it is therefore unlikely to elicit either motor or sensory responses. Alternatively, electrodes of a similar size can be placed at either end of a muscle belly so that the bulk of the muscle is covered. In this method, location of the motor point is not important, however, in order to identify where best to place the electrodes it is important to have a good knowledge of the anatomy of the muscle. If using this technique, electrodes should be large enough to cover the bulk of the muscle (without touching one another); the standard-sized electrodes which come with most battery-powered and line-powered units are likely to be too small for larger animals.



Figure 10.7 Electrode placement for neuromuscular electrostimulation (NMES) in a horse with disuse atrophy secondary to non-weight bearing lameness for 2 months. The upper electrode is over the triceps muscle, the lower electrode is at the top of the splint (partially tucked in) over the head of the extensor carpi radialis.

Electrostimulation parameters should be set to obtain the best effect from each treatment session; as part of this, it is worth taking time to identify the optimum placement sites for electrodes. As far as is possible, regular treatment (e.g. every other day) is preferable, with the length of each treatment session set to allow adequate periods of stimulation interspersed with adequate 'recovery periods'. Treatment should be initiated with stimulation at 60–100 Hz, with an on:off ratio of at least 1:3 (i.e. 5:15 s) set to induce fatigue within the stimulated muscle. The intensity should be adjusted to provide the strongest contraction possible within the animal's tolerances. However, it has been suggested that to strengthen muscle, it may be necessary to use muscle contractions of the order of 25% and 50% of maximum voluntary isometric contractions (MVIC) to obtain clinically meaningful outcomes (Alon 2005). Treatment should be started with stimulation of 8–15 contractions per session, with sessions provided 3–5 times per week. Subject to adequate response, the treatment should be continued over 3–5 weeks of training.

10.4.4 Safety, contraindications and precautions

There are limited reports of side effects or adverse reactions with electrical stimulation in humans. However, the common contraindication of treatment over the pregnant

uterus should be observed with electrical stimulation, as well as no application over indwelling stimulators or similar devices. Electrical stimulation should not be applied over the eyes or gonads. Other areas in which caution should be exercised include infected or broken skin (although some forms of electrical stimulation can be used as a means of stimulating wound repair), as direct application may exacerbate existing conditions.

10.5 Laser therapy

10.5.1 Overview

Laser is an acronym for 'light amplification by stimulated emission of radiation'; it is a form of electromagnetic radiation in the visible and near visible part of the spectrum. Lasers have found a variety of applications in veterinary medicine: their uses range from surgical to diagnostic devices. Low intensity laser therapy (also known as low level laser therapy, laser biostimulation, or more simply laser therapy) may be defined as the use of low power – typically 500 mW or less – laser sources and superluminescent diodes (similar in many respects to laser diodes, but lacking the property of coherence) for the treatment of medical conditions; it is based upon athermic tissue reactions.

10.5.2 Mechanisms of action

The basic interaction underlying laser therapy is the absorption of light in irradiated tissue by specific biomolecules. These are known as chromophores, typically found within the cell mitochondria. Based upon this absorption, light energy is transformed into biochemical energy. Following this initial absorption, there are a variety of secondary reactions, which result in modulation of cellular functions, and typically the stimulation of tissue-repair mechanisms. In addition, laser irradiation may also help reduce pain when used at appropriate treatment parameters. Absorption of light is the key to the basis of the effectiveness of laser therapy: thus while tissue penetration is often quoted in terms of millimetres, it should be realised that tissue absorption is more important.

10.5.3 Specific effects of therapy

A variety of effects of laser irradiation have been reported under controlled laboratory conditions including altered nerve conduction, changes in blood flow circulation, and stimulation of angiogenesis. Neurochemically, laser irradiation has been shown to increase the metabolism of endogenous opiates, acetylcholine and serotonin. At cellular level, laser irradiation has been shown to enhance the production of ATP within cells, and thus mediate or modulate a variety of other events including release of growth factors, cytokine reactions and cell replication; the ultimate effect of these events is acceleration of delayed tissue healing. This is the primary, or cardinal effect, of laser therapy, and thus the term laser photobiostimulation (or

simply 'biostimulation') is frequency used to describe the therapy, especially in the USA.

10.5.4 Indications: conditions treated

Laser therapy finds wide application in the treatment of a variety of conditions. These principally include:

- Wounds and ulcers: Pressure sores, chronic wounds/delayed wound healing, diabetic ulceration, burns, skin abrasions
- Acute injuries/trauma: Tendon and muscle tears/haematoma, ligament sprains, fractures, subluxations, and various types of sporting and soft tissue injuries
- Musculoskeletal conditions: repetitive strain injuries, rotator cuff tears, carpal tunnel syndrome, complex regional pain syndrome/reflex sympathetic dystrophy, fibromyalgia and temporomandibular joint pathologies
- Inflammatory conditions (acute and chronic): Tendinitis, bursitis, myositis, fascitis, synovitis
- Arthritis and related conditions: Rheumatoid arthritis (and other autoimmune diseases), osteoarthritis, chondromalacia patella, calcifications (e.g. bone spurs)

10.5.5 Treatment principles: devices and specifying parameters

Contemporary laser treatment devices used in veterinary practice are based upon either single (pen style) or multiple source diode units, the latter being available as fixed (commonly called clusters) or flexible units (typically based upon rubberised units). The output of the unit – radiant power output – should be specified in milliWatts (mW), and is fixed by the selection of the treatment 'head'. Control units allow setting of treatment parameters, which might include time of irradiation/treatment, and setting of pulsing frequency (Table 10.2). The treatment time is important in determining the dosage of treatment: energy in Joules (J) is calculated as:

$$\text{Energy (J)} = \frac{\text{Radiant power (mW)} \times \text{Time (s)}}{1000}$$

Thus a 33-s treatment with a 30 mW unit will provide a treatment dosage at the irradiated point of approximately one Joule (0.99 J). This means of specifying dosage is useful

for routine clinical treatment of points on intact skin. For the treatment of open wounds, it is more appropriate to use *energy density* (also called radiant exposure, based upon the area irradiated), which is also typically used in research reports on wound healing. For this, the area of the wound is calculated (in cm²), as well as the total energy in joules delivered over the surface of the wound.

Energy density is then calculated as:

$$\text{Energy density (J/cm}^2\text{)} = \frac{\text{Energy (J)}}{\text{Wound area (cm}^2\text{)}}$$

In such cases, treatment should be initiated at dosages of 4 J/cm² and progressed depending upon response to treatment.

Pulsing frequencies may also be selected on some machines: in such cases, lower pulsing frequencies (e.g. <20 Hz) are typically used in more acute conditions, while higher pulsing frequencies (hundreds to thousands of Hz) are typically used in the treatment of chronic conditions and open wounds.

Effectiveness of routine clinical treatment is dependent upon systematic and comprehensive treatment of sites: this starts with direct application to lesion/wounds or site of pain (using contact technique where possible, i.e. intact skin), relevant nerve roots and trunks, as well as trigger or tender points for pain management, and acupuncture points if appropriate. In treatment, especially of wounds and burns, lymphatic and blood vessels can also be usefully targeted for treatment.

10.5.6 Safety, contraindications and precautions

The safety of laser therapy is well established: there are limited reports of side effects or adverse reactions; in humans, there are extremely rare reports of nausea following irradiation over nerve roots, or sensations (tingling) during treatment.

Contraindications to treatment principally include active or suspected carcinoma, or areas of haemorrhage; care should also be taken in performing treatment near the eyes, even though the risk of eye damage is limited. Treatment over the pregnant uterus is also contraindicated.

Table 10.2 Laser therapy: parameters

Wavelength	Power	Pulsing	Dosage
600–1000 nm	milliWatts (mW)	May be Continuous Wave (CW)	Joules (J)
Red (visible) in 600 nm range	Typically: (for single sources) 30–500 mW for animal therapy	If pulsed, settings can range from 1 Hz to 20 000 Hz (20 kHz)	Typically: Up to 30 J per point in animal therapy
Infrared (invisible) >700 nm	May be higher for multisource arrays		Joules per cm ² (J/cm ²) Typically: 4 J/cm ² over open wounds

10.5.7 Treatment of wounds: key principles

In treating wounds, dosages over the wound surface should be kept relatively low (4 J cm^2). In addition, systematic treatment is essential to success. Treatment approaches can vary, depending upon the treatment unit available (i.e. single diode probe or treatment head versus a multisource array), and upon the size of wound. Treatment of larger wounds in large animals might only be feasible with multisource arrays, while in smaller animals, single diodes might be necessary.

Wound treatment is a two-stage process, including the treatment of the wound bed (using non-contact technique, distance of approximately 1 cm), followed by contact treatment of the intact skin around the wound margin, approximately 1 cm from the edges of the wound. As already indicated, lymphatic vessels can also be usefully treated to enhance the effectiveness of therapy.

Laser treatment of wounds should be harmonised with concomitant treatment procedures; e.g. any necessary debridement of the wound should be performed before applying laser treatment. For treatment of the wound bed of a larger wound, if only a single diode probe is available, care should be taken to ensure that as far as possible, a standardised dosage is applied across the whole of the wound. For this, some therapists use a grid or 'chess board' approach, based upon the same principles used for assessment with the probe applied to squares of $1 \text{ cm} \times 1 \text{ cm}$. Alternatively, 'manual scanning' can be used, similar to ultrasound, however, in such cases it is difficult to standardise the treatment. However, for larger wounds, treatment with a single diode can be prohibitively time-consuming and thus multisource arrays, or in rare cases, scanner devices, are preferred as they provide the ability to cover an extensive area with one shot (typically $12\text{--}20 \text{ cm}^2$ coverage). Treatment of the wound margin represents an essential second phase of treatment, applying laser through integral/patent skin using 'in-contact' principles with appropriate dosages (at least $1\text{--}2 \text{ J}$ per point).

Infected wounds are typically regarded as a 'relative' contraindication to laser treatment, based upon fears of photobiostimulation of the infection. However, stimulation of the host response is frequently overlooked as the primary effect of treatment; based upon this, the presence of infection can represent an indication for laser treatment. However in such cases, aseptic technique is essential, as part of which a pragmatic approach such as the use of 'Clingfilm' wrap either over the treatment head, or wrapped around the wound can provide an effective aseptic barrier. As already indicated, treatment should be initiated at a dosage of 4 J/cm^2 and progressed, depending upon response. While overdosing is theoretically possible, therapist's time is a more practical consideration and thus treatment dosages above 12 J/cm^2 are commonly used, thus decreasing treatment time, particularly with larger wounds.

In parallel with improved or accelerated wound healing, commonly observed benefits of treatment include reduced oedema, pain relief and improved patterns of sleep.

10.5.8 Applications in rehabilitation: practical considerations

Stimulation or restoration of wound-healing processes represents a common effect in all tissue types, and coupled with the pain-relieving effects of treatment, laser therapy thus finds wide application in animal therapy. In comparison with other electrophysical modalities, such as ultrasound or electrostimulation, this combination of increased healing plus pain relief, coupled with the athermic nature of its effects (with less risk of burns or exacerbation in acute stages), means that laser therapy is often the modality of choice. However, as in humans, laser therapy represents only one element of comprehensive physiotherapy treatment and rehabilitation for the animal patient. While laser treatment can be safely and effectively combined with most other modalities and integrated easily with other treatment approaches, it is important to recognise the relevance of the sequence of application with other modalities. In particular, thermal modalities can have a significant influence on the effectiveness of therapy, given their effects upon blood flow, and thus the 'saturation' of blood chromophores in the tissue. Where tissue perfusion is reduced, as in cryotherapy, the distribution of absorption of laser energy will be affected, so that chromophores in deeper structures will be targeted by treatment. This might well be a useful treatment strategy in larger animals in which the lesion or target tissue is deeply sited. In contrast, in cases of application of laser following tissue heating, the effects of treatment will be much more systematic than local, given the absorption by chromophores within the blood.

Laser devices (single diodes) can also be used to stimulate acupuncture points as an alternative to needles. Advantages associated with such 'laser acupuncture' include the fact that it is non-invasive and therefore easier and safer to apply; avoids problems with needle phobia; and although it requires reduced training, it is claimed by many therapists to be just as effective as needle treatment in some cases.

10.6 Ultrasound therapy

Therapeutic ultrasound (US) is one of the most commonly used electrotherapeutic modalities. It is based upon the application of longitudinal sound waves to the body for a therapeutic effect, i.e. molecules of the tissues treated oscillate in the same direction as the sound wave (see Table 10.3 for parameters).

10.6.1 Mechanism of action

US has been shown to have a direct effect upon cells, and, in the laboratory, to stimulate healing. The effect can be either

Table 10.3 Ultrasound therapy: parameters

Frequency	Power	Pulsing	Dosage
Megahertz (MHz)	Watts per cm ² (W/cm ²)	May be continuous wave (CW)	Typically specified in minutes for each area of treatment
1–3 MHz	Power settings typically used range: <1–3 W/cm ²	Usually pulsed, settings typically range 1:1–1:9 (Duty cycle 50–10%)	Times may be several minutes to 20 min

thermal or non-thermal, depending upon the ultrasound parameters (e.g. intensity) or the pattern of absorption in the tissue. Penetration of ultrasound in insonated tissues is determined by attenuation of US beam as it travels through the tissue. Scattering of the beam can occur, as can reflection and refraction of the beam. These events are determined by the frequency of the ultrasound: e.g. 3 MHz US is absorbed more readily than 1 MHz US, and hence doesn't penetrate as deeply. The penetration of US is also dependent upon the nature of the insonated tissue: for example, muscle proteins act as major absorbers of ultrasound energy.

10.6.2 Biophysical principles

US application can produce a variety of effects in tissue, depending upon the parameters of application. Acoustic streaming and micromassage are considered as the most relevant effects of ultrasound energy upon tissues in terms of its claimed clinical benefits. Acoustic streaming is caused by the differential pressures acting along the axis of the US beam, and has been linked to effects such as increased secretion and growth factor release from insonated cells. Micromassage refers to the rapidly alternating pressures exerted upon insonated cells, and the resultant mechanical stresses are considered important to the therapeutic benefits associated with US therapy, particularly in reducing tissue oedema, although the precise mechanisms of action remain unclear.

Cavitation (the production of gas bubbles in insonated tissue) occurs as a transitory event, at high intensities, and where continuous US is used. Whilst it is considered unlikely that cavitation is produced during routine US treatments, it is theoretically possible that this effect could potentially be damaging. This is one of the main reasons why pulsed ultrasound is typically employed, and intensities kept relatively low (generally athermal). The formation of standing (or stationary) waves is also theoretically possible, and are likewise potentially damaging to tissue. However, like cavitation, the creation of standing waves during routine ultrasound treatment is unlikely owing to tissue geometry and the movement of the treatment head during therapy.

Beyond this, it is also thought that pain-gating mechanisms underpin some of the pain-relieving effects of ultrasound, as a result of the contact stimulation of the

treatment head, possibly coupled with the effects of acoustic streaming and micromassage.

10.6.3 Indications for use

Tissue healing

Promotion of tissue healing is the cardinal application for US therapy: this includes the treatment of open wounds, or the treatment of muscle tears, ligament sprains, etc. In such cases, US can be safely used at any stage of healing, although in open wounds it is more commonly used where healing is delayed. In the more acute stages following injury, ultrasound should only be used at relatively low intensities to limit the potential of exacerbating oedema; used appropriately, ultrasound can be effective in helping to reduce oedema.

Fracture healing

US, and particularly so-called low-intensity ultrasound, has been promoted as an effective therapy for the treatment of delayed or complicated fracture healing. While it is usually recommended that conventional units are only applied in the first two weeks post fracture, units specially designed for fracture treatment (e.g. sonic accelerated fracture healing system (Warden *et al.* 2000) can be applied throughout the process of fracture healing (which is similar in many ways to healing in other tissues).

Pain relief

Although not a primary indication for ultrasound treatment, the relief of pain is typically regarded as a parallel effect of US therapy. Despite this, the underlying mechanisms of action are unclear (e.g. claimed pain-gating mechanisms, increased blood flow reducing local levels of pain-relieving substances, etc.). Furthermore, most electrotherapy textbooks make scant reference to such effects.

10.6.4 Safety, contraindications and precautions

Ultrasound is generally regarded as a safe modality for routine use in physiotherapy. Typically, side effects are associated with higher intensities (>3 W/cm²) and associated thermal reactions, e.g. exacerbation of inflammatory response in acute conditions. Contraindications to treatment include: active or suspected carcinoma, or areas of haemorrhage; application to eyes and gonads; application over

pregnant uterus. Other contraindications include application to ischaemic tissue, suspected deep venous thrombosis. Caution should also be exercised with treatment over metal or plastic implants, or in areas with vascular problems.

10.7 Evidence-based practice

In common with other areas of physiotherapy practice, the available evidence to support the clinical use of electrophysical agents can be limited or variable. Common problems with published research include a lack of high quality studies (i.e. internal validity is often limited, with lack of controls or blinding). However, there are also widespread problems owing to the use of inappropriate, or inadequately specified treatment parameters in research studies.

10.8 Summary and conclusions

Electrophysical agents can play a significant role in the success of animal therapy. However these modalities are not panaceas. In particular, biophysical principles and the rules of good clinical practice remain; for example, the need for thorough assessment. Beyond this, the effectiveness of the therapy is dependent upon the quality of the machine and regular testing and servicing, especially in situations where the machine can be subjected to high levels of wear and tear.

Given the potential risks associated with some treatments, care should be taken with anxious or temperamental animals, where application of electrophysical agents may not be appropriate. General contraindications to treatment with electrophysical agents include application over the eyes, the pregnant uterus or the gonads. In addition caution should be exercised in all such applications, as animals are

unable to provide the therapist with the requisite feedback, such as feelings of discomfort or heating.

There are several key issues that underpin the effectiveness of all electrophysical modalities. These include the importance of the selection of appropriate treatment parameters (including treatment intensity, time and site of application) for the desired effects or clinical benefits. Frequently, therapists are unclear about the relevance of treatment parameters, and thus fail to achieve the desired or anticipated benefits of electrotherapy treatment.

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11

Hydrotherapy

Michelle Monk

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11.1 Introduction

Hydrotherapy has long been recognised for its benefits in the rehabilitation of humans (Davis & Harrison 1988; Langridge & Phillips 1988; Hall *et al.* 1990; Konilan 1999; Kelly *et al.* 2000) and is now growing in popularity in the rehabilitation of the small (Taylor 1992; Gentry 1993; Harasen 2001) and large animal patient. Although, in horses, research has to date been limited to swimming as a form of training in normal horses (Hobo *et al.* 1998; Misumi *et al.* 1994; Misumi *et al.* 1995). Hydrotherapy allows for earlier intervention, with patients being able to begin moving within days of injury or surgery with little or no risk of reinjury (Konilan 1999). For the animal patient, exercising in water allows unloading of painful joints, as well as earlier weight bearing on otherwise very weak or painful limbs (Steiss 2003).

Recently, hydrotherapy or aquatic therapy has become an important part of the rehabilitation of many small and large animal conditions, as the recognition of the importance of rehabilitation after injury grows. No longer are these patients discharged back to owners or handlers for rest and gradual return to normal exercise, but specific rehabilitation protocols and programmes are in place in many animal facilities.

There is sufficient evidence now available for the effectiveness of hydrotherapy on humans. There is however, a paucity of information in the veterinary or physiotherapy literature with respect to protocols, guidelines and implementation of these programmes for small or large animals. There is thus a need for animal physiotherapists to perform and publish research in this area, to provide scientific evidence for hydrotherapy and its applications and effectiveness in the rehabilitation of animals.

In the interim, clinical application of many of our human hydrotherapy principles and techniques, to the management of our animal patients, taking into consideration, differences in size, body type, injuries and communication differences, may be utilised.

11.2 Physical properties of water

Movement and exercise performed in water is very different from that performed on land. We are unable to simply transfer land exercises over to similar practice in water. To do so would be ignoring the opportunities available to us through exercising in water and the physiological effects of immersion on the body. There are several properties of water we must understand, in order to develop efficient hydrotherapy programmes for animals. These include:

- Density
- Specific gravity
- Buoyancy
- Hydrostatic pressure
- Viscosity
- Surface tension
- Refraction

11.2.1 Density

The density of a substance is the relationship between its mass and volume, measured in kg/m^3 . Density = mass / volume, kg/m^3 . Water is most dense at 4°C, expanding at both higher and lower temperatures. Density increases with dissolved substances; hence seawater is denser than pure water. The *relative density* of a substance compares the density of a substance using water as a standard.

Densities of various substances are defined by a number value called *specific gravity*. The specific gravity of pure water is 1.0. Relative density and specific gravity of an object will depend on the composition of the object and will determine whether an object will float or sink. If the ratio of an object's specific gravity to that of water is greater than 1.0; the object will tend to sink; if the ratio is less than 1.0, the body will float. The upper and lower limits of body density in the human community are typically 0.939 g/ml in the very obese and 1.10 g/ml in the leanest individuals (Edlich *et al.* 1987). The relative density of the substance also



Figure 11.1 (a and b) Comparison of dogs swimming with different in buoyancies.

determines how much of it will sit outside of the water. If the specific gravity of a cork is 0.2, only 20% of it will sit under the water. A human with air in the lungs, with a specific gravity of 0.95–0.97, will have 95–97% of their body under the water.

Application to animal hydrotherapy (Figure 11.1a and b):

- Lean animals and heavily muscled animals have a tendency to sink, and may need to use a buoyancy vest to reduce effort.
- Animals with a greater amount of body fat will float more easily.
- Animals with osteoporosis will have reduced specific gravity and will float.
- Adding flippers or weights will increase specific gravity and cause the animal to sink, so increased effort will be required to keep the head above water.

11.2.2 Buoyancy

When a body is immersed in water, it is subject to the forces of gravity and buoyancy. The principle of buoyancy was

discovered by Archimedes. Archimedes' principle states, 'that when a body is wholly or partially immersed in a fluid at rest, the body experiences an upthrust, equal to the weight of the water it has displaced' (Edlich *et al.* 1987). Buoyancy is the force experienced as an upthrust, which acts in the opposite direction to the force of gravity. The body immersed in the water appears to lose weight, and the weight loss is equal to the weight of water displaced.

So the body at rest in water is subject to two opposing forces:

- Gravity – acting through the centre of gravity (COG)
- Buoyancy – acting through the centre of buoyancy, which is the centre of gravity of the volume of the displaced liquid (Edlich *et al.* 1987)

In women, the percentage weight bearing when immersed to the level of C7 is 6–9%, when immersed to the xiphoid process is 25–31% and when immersed to the anterior superior iliac spine, 40–51% (Harrison & Bulstrode 1987).

In dogs, a similar study comparing weight bearing in standing as a percentage of body weight borne on land, demonstrated that when dogs were immersed to the level of the greater trochanter weight bearing was 38%, at the lateral femoral condyle weight bearing was 85% and at the lateral malleolus 91% (Tragauer & Levine, 2002). Further studies need to be performed in various breeds and sizes of dog to confirm these values.

If the centres of gravity and buoyancy are in the same vertical line, the body is kept in *equilibrium*. If the centres of gravity and buoyancy are not in a vertical line, the resultant effect of the two forces will cause a turning force called a *moment* about a pivot point. The body will continue rotating until it finds a point of equilibrium. The *moment of force* = the force of buoyancy \times distance between the centre line of buoyancy and a vertical line through the axis of rotation/pivot point. Buoyancy will have a greater effect on a long lever than on a short lever. We can also use the force of buoyancy to assist movement in weak or injured limbs, and provide support and reduced loading to painful or healing structures.

Implications for animal patients:

- Left hind limb amputee swimming: COG moves towards right, will rotate up on right side to reach equilibrium.
- A right hind limb held up in flexion: moves COG to left will rotate down on the affected side to reach equilibrium – a dog may struggle to overcome this.
- Spinal injured patients or those with asymmetrical tone may not be able to control trunk rotation that occurs during swimming – may use buoyancy vest in early stages, but then progress to the dog having to strengthen trunk against moment of force.
- Care with position of flotation devices as they will alter buoyancy and, if asymmetrical, can cause the dog to tip over.

11.2.3 Hydrostatic pressure

Pascal's law states that fluid pressure is exerted on all surfaces of an immersed body, while at rest, and at any given depth. Hydrostatic pressure is the sum pressure exerted on all surfaces of a body immersed in water, for any given depth. Pressure is directly proportional to the depth of the part immersed and the density of the water (Edlich *et al.* 1987). Pressure increases with increased depth, and increased density of the fluid.

Implications for animal patients:

- The principles of hydrostatic pressure tell us that peripheral pooling of blood can be reduced in extremities deep in the water, assisting reduction of oedema.
- Hydrostatic pressure will affect lung volumes hence care needs to be taken with patients with respiratory distress or compromise.

11.2.4 Viscosity

The resistance to movement through water is caused by the friction between water molecules. Water acts as a resistance to movement, as the molecules tend to adhere to the surface of the body moving through it. Viscosity *decreases* as water temperature *increases*. This means weaker and smaller muscles move more easily in warmer water.

Reynolds theorem: Reynolds demonstrates that there are three types of flow:

- *Laminar flow:* Streamlines of molecules in even and regular patterns. The rate of movement at any fixed point is constant.
- *Transitional flow:* As velocity increases, molecules make small movements sideways.
- *Turbulent flow:* Velocity increases further; fluid moves irregularly, gives rises to 'eddies'.

Turbulence is the term describing the 'eddies' that follow in the wake of an object moving through fluid. The degree of turbulence will depend on the speed of movement and the shape of the object. Increased speed produces increased turbulence. The more unstreamlined, the more turbulence will be produced. When there is turbulence, there is *drag*. Drag is also increased when the velocity of the limb movement is increased.

Skin friction drag: water molecules are attracted to a submerged body. This accounts for 56% of the drag experienced by a moving object. Movement in the water is 799 times slower than in air. This is due to the weight of the water, plus the skin friction drag.

Implications for animal patients:

- Animals with poor swimming technique, limbs thrashing out of the water, or those that are poorly balanced, cause increased turbulence, and increased *drag*, or resistance to movement – increased difficulty and effort.
- We also use turbulence to provide resistance for strengthening such as the reciprocal movement of limbs in the

water, or swimming against a swim-jet, or adding floats or aqua-fins.

- Drag is reduced when a limb moves in the same direction as the turbulent flow, and can be used to assist movement, e.g. hip extension may be aided by swimming against swim-jets, or walking on the underwater treadmill.
- Other smaller animals in the water may be affected by turbulence created by larger animals.
- Animals with poor balance on land may be able to stand in water, with less chance of falling as they can correct themselves before falling over. We often see patients with spinal injury able to walk in the underwater treadmill before they can on land.

11.2.5 Surface tension

Surface tension is the force exerted between the surface molecules of the fluid. Water molecules have a greater tendency to adhere together at the surface. Resistance to movement is slightly greater at the surface as there is more cohesion of the molecules here. That's why an insect can land on the surface if the water, or dog hair can float on the top.

Implications for animal patients:

- Weak animals may be able to move a limb under the surface of the water, but have difficulty lifting the limb out of the water.
- More effort is required for swimmers thrashing limbs out of the water.

11.2.6 Refraction

Bending of light occurs as it passes from a denser medium to a less dense medium, or vice versa, e.g. air to water. This means that objects can appear shallower than they are, or steps can appear deeper. It also makes viewing limb movement through the top of the water more difficult.

Implications for animal patients:

- Limb movements look different from below the water rather than through the top – (have a look at the underwater treadmill).
- Animals may mis-judge steps.

11.3 Physiological responses to exercising in water

Exercising in the water causes different physiological responses from exercising on land. A number of studies have compared energy expenditure of exercising in water with the same activity on land (Evans 1978; Gleim & Nicholas 1989).

11.3.1 Energy expenditure

Energy expenditure in the water may be greater, the same or less, compared with the same exercise performed on land,

depending on the activity, the water depth, the temperature of the water and the speed at which the activity is carried out. Energy expenditure in the water is different for the same movement pattern performed on land owing to the buoyancy of the water, which reduces the body weight, so less force is required to lift the body against gravity (Cureton 1997). Also the viscosity of water increases the energy required to overcome the resistance to movement through water (Cureton 1997). The resistance encountered is directly related to the body weight, size, shape, position and speed of movement (Cureton 1997). In addition, exercising in cool water may require more energy to be expended in an attempt to maintain core body temperature, because of the greater conductivity of heat to water (Cureton 1997).

Walking in water

Walking in the water has become a common form of hydrotherapy for rehabilitation for humans and animals, and is thought to be particularly beneficial for injuries of the limbs (Cureton 1997). Evans (1978) found that only half to one third of the speed was needed to walk or jog across a pool in waist deep water at 31°C, to achieve the same energy expenditure as walking or jogging on a dry treadmill (1.6–3.5 miles/hour in water; 3.4–8.3 miles/hour on treadmill). They found that the resistance to movement through the water had a greater effect on energy expenditure than the reduction in body weight caused by the force of buoyancy.

Gleim & Nicholas (1989) investigated the effect of water depth on energy expenditure while walking at different speeds on a land treadmill and an underwater treadmill. These authors found that there were complex opposing effects of buoyancy and water resistance on energy expenditure in water. They found that the rate of oxygen uptake (VO_2) was increased during water walking in ankle- (25–55%), knee- (26–67%), thigh- (34–72%), and waist-deep (14–67%) water at speeds of 2–4.5 miles/hour. At these speeds, VO_2 increased curvilinearly with speed for both dry and water treadmill walking. At speeds above 5 miles/hour, VO_2 during jogging in ankle-, knee-, and thigh-deep water was higher than jogging on the dry treadmill, but by a lesser amount than at lower speeds. In waist-deep water at speeds above 5 miles/hour, there was no difference in the VO_2 compared with jogging on the dry treadmill. It appears that at this depth, the effect of buoyancy offset the effect of water resistance so that energy expended in the water and on the dry treadmill was the same.

Swimming

Swimming tends to be the other common form of hydrotherapy used in the rehabilitation animals, and there have been several studies looking at energy expenditure during swimming in humans. There are dramatic differences in energy expenditure, depending on the swimming stroke

used and the skill of the swimmer, making it difficult to predict the energy cost of swimming (Cureton 1997). In general, the energy cost of swimming a given distance, is about four times the cost of running the same distance.

Water temperature

Energy expenditure during exercise in water may be increased when exercising in cold water owing to the additional effect of shivering (Cureton 1997). The size of the effect is a function of the fatness of the participant, the exercise intensity and duration and the water temperature. At rest in the water, shivering tends to occur in humans when the water temperature is below 28–34°C (Craig & Dvorak 1970) depending on the degree of body fitness and duration of immersion. Even with light to moderately heavy submaximal exercise, energy expenditure is increased when water temperature is below 26°C (Craig & Dvorak 1970).

11.3.2 Maximal oxygen uptake

Maximal oxygen uptake is generally lower in water compared with similar exercise on land (Cureton 1997). Maximal heart rate and blood lactate are also lower during maximal water running, suggesting the work rate achieved at exhaustion is less in the water than on the land (Cureton 1997).

These findings are important as they suggest that we cannot base exercise prescriptions in water on maximal heart rates or oxygen uptakes measured on land. Reduced maximal heart rate accounts for much of the reduced maximal oxygen uptake observed (Cureton 1997).

11.3.3 Circulation

During upright, head-out-of-water immersion in humans, there is a shift of blood volume from the lower limbs and the abdomen to the thorax. This shift of blood increases central venous pressure, left ventricular end-diastolic volume, stroke volume and cardiac output, and decreases systemic vascular resistance at rest and during submaximal exercise.

While the body is at rest, or exercising at lower intensities, the heart rate remains mostly unchanged during immersion. At higher intensities of submaximal and at maximal exercise intensities, heart rate is decreased compared with exercise on land.

It is commonly, but not always, observed that heart rate is lower during exercise in the water compared with exercise at the same maximal oxygen uptake on the land. This is dependent on exercise intensity and water temperature. During moderately light head-out-of-water exercise in water 31–33°C, heart rate does not differ from when exercise is performed on land at the same VO_2 . During strenuous exercise, the heart rate is usually, but not always, lower by approximately 10 beats/minute (Craig & Dvorak 1970; Evans 1978).

These studies demonstrate that the relationship of VO_2 to heart rate during exercise in water compared with that on land is variable and depends on several factors including

exercise intensity, exercise mode and water temperature. Therefore, we must be very careful in using heart rate to prescribe or monitor exercise intensity in the water, or using a maximal heart rate derived from a land-based test, to determine expected exercise intensity in the water.

11.3.4 Thermoregulation

In humans, the regulation of body temperature in water is very different than it is in air because the primary means of dissipation of heat during exercise on land, evaporation of sweat, is not possible. The loss or gain of heat is also much greater in water through convection and conduction (Cureton 1997). Heat conductance is about 25 times greater in water than air, so when water temperature is above or below skin temperature, heat gain or loss is greater in water respectively, than in air (Cureton 1997). In the water, there is a much narrower range of water temperatures through which core temperature is not affected by exercise compared with the land.

During exercise in the air, environmental temperatures of between 5°C and 35°C do not affect the rise in central body temperature caused by increased exercise intensity (Cureton 1997). During exercise in water, the temperature needed to prevent a rise in core temperature during prolonged exercise varies from 34°C to 17°C, depending on the intensity of the exercise performed and the degree of body fat of the person (Craig & Dvorak 1970).

Exercising in warm water over 33°C can cause a feeling of fatigue and exhaustion, owing to elevated core temperature. Exercising in cold water below 25°C causes thermal stress and brings about metabolic and cardiovascular adjustments geared towards maintaining core body temperature, because the heat flow from the body is substantial. Exercising in water at 18°C can cause the muscles to fatigue, and an inability to contract the muscles fully. For most humans, the optimal water temperature for exercising is around 28–30°C, where little heat would be stored, and performance would not be impaired (Edlich *et al.* 1987). Most pools for human hydrotherapy are kept at ‘thermo-neutral’ temperature: 32–34°C as we are not ‘swimming’.

The optimal water temperature for small animal hydrotherapy is unknown, but will vary somewhat, depending on the size of the animal, how fast it can swim or walk, how fit or old it is, how fat it is, the duration of the exercise session, the depth of the water and any pre-existing respiratory or cardiac conditions.

It is expected that lower temperatures will be required for dogs that are swimming or generating considerable body heat, or if the dog will spend extended periods in the water. Pools for swimming should be kept at sub-thermal temperatures: 25–27°C. However, a small puppy will quickly chill if left standing in cooler water and may need temperatures up to 32°C.

When using the underwater treadmill to exercise patients after neurological or orthopaedic surgery, exercise intensity

will mostly be low. Hence, water temperature should be in the therapeutic range of 27–32°C, depending on the needs of the individual patient, speed of exercise and depth of immersion.

Rectal temperatures may need to be taken if there is concern for hyperthermia.

Implications for animal patients:

- Exercising in colder water may cause an increased work of swimming and increase oxygen uptake due to shivering, as well as difficulty contracting muscles.
- Thinner animals will feel the effects more in colder water.
- If the water is cold, sessions should be shorter (e.g. in the sea in winter).
- Similarly, water should not be too warm (suggest no more than 30°C), due to the thermal load on the body, the risk of fatigue and exhaustion.
- Monitoring of animal patients is imperative – and strict home exercise instructions should be given regarding water temperature, depth of immersion and duration of exercise (difficult to control for intensity).

There are several important differences between the physiological responses to exercise on land and in water, but as long as the therapist is aware of these differences, the benefits of exercising in an environment with reduced weight-bearing stresses on the limbs and the increased resistance offered by the water make it an ideal environment for rehabilitating many injuries.

11.4 Evidence for effectiveness of hydrotherapy

In humans, the benefits of exercising in water have long been recognised and more recently, evidence has been provided to confirm that hydrotherapy is beneficial for pain, function, self-efficacy, joint mobility, strength and balance (Davis & Harrison 1988; Langridge & Phillips 1988; Hall *et al.* 1990; Konilan 1999; Kelly *et al.* 2000).

Hydrotherapy is used widely in humans by therapists and patients for the management of osteoarthritis and rheumatoid arthritis. Hydrotherapy has been shown to reduce pain and improve function in patients with hip osteoarthritis, and increase strength and range of motion in joints affected by osteoarthritis. Reduction in pain, improved range of joint motion, along with reduced difficulty in the performance of functional tasks for those affected by rheumatoid arthritis, have also been demonstrated as a result of hydrotherapy programmes. Patients with rheumatoid arthritis also experienced an improved emotional and psychological state, along with improved joint range of motion and reduced pain, following a hydrotherapy programme (Hall *et al.* 1990). Hydrotherapy has also been shown to improve function in subjects with back pain.

Hydrotherapy is also commonly used by physiotherapists for post-operative therapy. Following shoulder surgery,

movement of the shoulder in the water requires much less muscle activation than on land, meaning that rehabilitation can begin earlier, with less stress on the injured structures (Kelly *et al.* 2000). An investigation of the effects of exercise in water and on land, following intra-articular anterior cruciate ligament reconstruction, showed that exercising in the water was more effective in reducing joint effusion, and hastened return to function, and that hydrotherapy was as effective for increasing knee range of motion and quadriceps muscle strength.

While hydrotherapy appears to be widely used for the rehabilitation of animals, currently there are few studies investigating the benefits of this form of exercise, or the parameters to use when prescribing exercise. Unlike human hydrotherapy, much of the hydrotherapy performed on animals consists of swimming or walking through the water. Fewer one-to-one therapist-patient treatments are performed, largely due to the danger of performing such activities, particularly in horses, or the inability of the patient to relax in the water. More one-to-one treatments may be performed in dogs, and with acclimatisation, the animals can become quite relaxed in the water and allow limbs or body parts to be moved for them.

Swimming is often used as part of the rehabilitation programme for the canine patient following surgery for cranial cruciate ligament deficiency. A programme of physiotherapy exercise and swimming, commenced at 3 weeks after surgery, was shown to improve limb function over cage confinement, with no difference evident between the affected and unaffected limbs measured with a force plate at 6 months after surgery (Marsolais 2002).

Range of motion of the stifle was compared in a group of dogs swimming versus those exercising on a land treadmill following cranial cruciate ligament stabilisation surgery. More stifle flexion was achieved while swimming than while exercising on the treadmill, so swimming was recommended if increasing stifle flexion is the goal (Marsolais 2003).

There have been several investigations of the physiological responses of horses to swimming (Hobo *et al.* 1998), but as with humans, it is very difficult to standardise the intensity and velocity of the movement of the horse in the pool, therefore measurements are often inaccurate.

Underwater treadmill therapy has recently become very popular for the rehabilitation of both canine and equine injuries. Joint kinematics of dogs studied while walking on an underwater treadmill, showed that joint flexion is greatest when the water is filled higher than the joint of interest, but that full joint extension is also achieved with underwater treadmill walking, which is not the case in swimming (Jackson *et al.* 2002).

Recently, the effects of an early intensive underwater treadmill, physiotherapy exercise and home-walking programme was compared with a typical home-walking programme, for their effects on limb function following tibial plateau-levelling osteotomy surgery in dogs with cranial

cruciate ligament deficiency (Monk *et al.* 2006). Dogs in the underwater treadmill physiotherapy group increased thigh circumference, and stifle flexion and extension range of motion, so no difference was evident between the affected and non-affected limbs at 6 weeks after surgery, while the home-walking group continued to lose muscle mass and their stifle joints became stiffer.

11.5 Benefits of hydrotherapy for animals

Based on the evidence we have for human hydrotherapy, the suggested benefits of hydrotherapy or exercising in water for animals include:

- Reduces loading on painful or healing structures – exercise can be commenced earlier with less muscle activation required and less load on limbs
- Provides additional support to limbs, reducing likelihood of injury to muscles, tendons and ligaments
- Allows exercise to continue while land-based exercise is restricted or contraindicated
- Eases performance of difficult activities and movements through the force of buoyancy
- Water provides more resistance/drag than air: great for strengthening
- Prevents atrophy
- Increases in muscle mass and strength
- Increases cardiovascular fitness and endurance
- Increases joint range of motion, reduces stiffness
- Increases soft-tissue extensibility
- Reduces muscle spasm and hypertonicity
- Increases tone in hypotonic body parts
- Allows gradual progression and return towards more normal function
- Assists in management of oedema through hydrostatic pressure
- Provides relaxation
- Reduces pain in joints with degenerative joint disease
- Increases circulation and assists in promotion of healing

11.6 Assessment of the small animal patient for hydrotherapy

Although this subject will not be covered in detail, it is expected that all patients attending for hydrotherapy will be assessed and cleared by their veterinarian before commencing hydrotherapy.

As with all human physiotherapy patients, a thorough assessment, including documentation of findings, needs to be performed. The physiotherapist needs to consider which patients will be suitable for hydrotherapy. Apart from their current medical or physical conditions, factors to consider include temperament, previous history of swimming, including good and bad experiences. On the whole, most patients are suitable for hydrotherapy in some form.

All horses, dogs and cats can swim. How well they do it and with how much anxiety will depend on prior experience, and experience of the handler now attempting to swim them.

Most animals will become acclimatised in a few visits, and it is worthwhile trying to persevere with even the most anxious patients. Use of buoyancy vests and equipment to lead the animal, extra staff members to assist and a short session help to reduce anxiety and enable acclimatisation.

11.6.1 Subjective questioning

In addition to your standard subjective questioning of the owner or handler, further questions (specific to hydrotherapy), which will help you determine the type of hydrotherapy that will be suitable and where to start, include:

- Date of last vaccination and worming?
- Previous level of exercise/fitness?
- Has the animal swum previously and where? In a pool/beach/dam – beware those that swim well at the beach may not swim well in your pool. Also, those that are fearful of water often are more relaxed in an underwater treadmill where feet can still touch the bottom.
- Did they actually initiate the swimming or did you place them in the water?
- Do they like the bath? The hose?
- Any incontinence?
- Any ear problems?
- When did they last eat?
- Did the vet advise you how much hydrotherapy to do?

11.6.2 Objective assessment

Along with your standard objective assessment, if you are planning to take a patient into the water, things to check before, include:

- Skin condition – any dryness, redness, flaking (not contraindicated, just be aware)
- Ear problems – check inside
- Make sure the dog has toileted
- Any open wounds / torn nails, etc.
- Heart rate and respiratory rate assessment at rest

11.6.3 Contraindications to hydrotherapy for animals

Hydrotherapy is not recommended for animals with any of the following conditions:

- Open, infected or draining wounds
- Unhealed surgical incisions without a waterproof cover
- Active gastrointestinal disease (vomiting and/or diarrhoea)
- Elevated body temperature / infection
- Systemic compromise such as severe cardiac, liver, kidney disease, hypotension or hypertension
- Respiratory compromise or distress

- Advanced debility
- Uncontrolled epilepsy
- High temperature / infection
- Kennel cough in dogs (contagious)

11.6.4 Precautions

Animals with any of the following conditions may still be suitable for hydrotherapy depending on the equipment and facilities available. No animal should *ever* be left unattended while participating in hydrotherapy in any form.

- Faecal incontinence – use nappies or evacuate bowel beforehand in dogs
- Urinary incontinence if urinary tract infection
- Wounds that have been given the ‘OK’ by the veterinarian – get written permission
- Older age patients
- Mild systemic compromise – monitor
- Extremely obese patients
- External fixators
- Skin problems
- Ear problems
- Epilepsy

11.6.5 Treatment plan

After assessment, a treatment programme and plan should be written and discussed with the owner, including expected time frames for improvement. Goals of treatment need to be devised and discussed with the owner. The owner may simply want the dog to return to getting up the back steps independently or walking to the local shops for the paper, or they may wish to return them to high-level agility or hunting and retrieving.

Objective measures need to be used to determine the effectiveness of treatment. These may include:

- Lameness score / gait analysis with video
- Range of motion of affected limb
- Muscle mass / limb circumference
- Functional measures: able to sit squarely / use stairs / get into the car

11.7 Types of hydrotherapy for animals

Following your assessment, you will have to determine the most suitable form of hydrotherapy, and the expected dosage for that day (or for home exercise).

This will be determined not only by your assessment findings, but by the size of the animal, the equipment you or the owners have available, any equipment you have to help you, and the goals of treatment.

Small animals

- Sink
- Bathtub
- Whirlpool



Figure 11.2 Dog pool.



Figure 11.3 Underwater treadmill.

- Swim-spa
- Children's wading pool
- Beach
- Dam
- Lake
- River
- Above-ground or in-ground human swimming pools – swimming and hydrotherapy
- Purpose-built dog pools – swimming and hydrotherapy (Figure 11.2)
- Underwater treadmills (Figure 11.3)

Large animals

- Swimming in purpose-built pools (Figure 11.4)
- Beach



Figure 11.4 Horse pool.

- Dam
- River
- Water walker / underwater treadmill (Figure 11.5 a and b)

11.7.1 Equipment

There are several different pieces of equipment that may be used to reduce or increase effort of the animal in the water.

- *Children's arm floaties*: may be used to assist joint flexion when placed below the joint of interest, or increase the work required to overcome the buoyancy. They may help to increase weight bearing in an affected limb if placed on the contralateral limb while walking in the underwater treadmill.
- *Theraband*: can be used to provide assistance or resistance to movement: looping around both hindlimbs will increase the work of walking while in the treadmill. Be sure it can be easily released if the animal becomes distressed.
- *Weighted straps around limbs*: can increase the work of swimming or walking to increase strength.
- *Buoyancy vests*: good for initial swimmers, those with poor trunk control and anxious dogs. Can reduce the work of swimming on the heart and lungs, meaning dogs can exercise their limbs for longer before tiring (Figure 11.6).
- *Harnesses*: to assist front, rear or the entire dog: sometimes a buoyancy vest gives too much support and we want the animal to work a little harder. Harnesses can be helpful to provide support and assistance to particular body areas, e.g. holding the rear end higher in the water for a dog with spinal injuries, but still making it use its trunk muscles to control body rotation.
- *Hoists*: useful for assisting large animals and those quite disabled in and out of the pool or treadmill.
- *Aquatoys*: great for motivation to encourage swimming.
- *Steps*: can be used to provide alternating terrain to negotiate while walking through the water.



(a)



(b)

Figure 11.5 (a and b) Horse aquawalker.

- *Ramps / inclines / declines:* to assist entry/exit as well as increasing work of hindlimbs if walking uphill, or forelimbs if walking down hill

11.7.2 Hydrotherapy for specific conditions – small animals

There are many conditions that will benefit from hydrotherapy in some form, including joint surgeries, fracture repairs, non-use of a limb, spinal injuries or surgeries and many degenerative conditions such as arthritis. Hydrotherapy, where possible, should not be the only form of therapy the animal receives, and each patient should be assessed by a physiotherapist before commencing a hydrotherapy programme. The following information dis-



Figure 11.6 Buoyancy vest.

regards the land-based therapy and is intended as a general guideline only. It is by no means a recipe for treatment, and each patient case must be assessed individually and an individual programme devised which considers the patients condition, other health conditions, fitness, previous experiences, fear of water, etc. Programmes should be revised regularly and progressed as needed.

Cruciate stabilisation surgery

Hydrotherapy has an important place in the rehabilitation of all dogs recovering from the various forms of cruciate surgery. Hydrotherapy would generally be commenced 10 days to 2 weeks after surgery, when the wound has healed. Swimming and underwater treadmill are great for increasing joint range of motion, thigh muscle mass, encouraging limb use, and maintaining or increasing general fitness while other land-based exercise is contraindicated. Muscle mass increases with underwater treadmill therapy once or twice a week more rapidly than with swimming or land-based exercise, and is the method of hydrotherapy chosen wherever possible. There is more control over intensity of exercise, with speed, length of treatment, water temperature and depth all able to be controlled easily and adjusted as required. Equipment can be used later on down the track to increase strength. It is recommended that dogs with severe arthritis of the stifle continue hydrotherapy in some form once or twice a week indefinitely.

Fracture repair

Hydrotherapy is often extremely beneficial after fracture repair to allow exercising of the injured part, thus increasing joint range of motion and preventing atrophy, contractures and non-use of limbs; and then building muscle strength, but in a controlled fashion, without compromising repair. The physiotherapist should check with the referring veterinarian as to how much weight they are happy for the animal to take on the limb and the stability of the repair.

In general, hydrotherapy would start when the wound has healed and any casts or bandages have been removed. Sometimes hydrotherapy will be performed when a dog attends for a bandage change, then the limb is dried and the bandage reapplied. This is very useful for preventing excessive joint stiffness and muscle wastage. Depending on the area fractured and type of fixation used, some animals may be more suitable for underwater treadmill initially as it is more controlled than swimming. If water quality is kept at human standards, and the referring veterinarian is happy, dogs with external fixators may be suitable to commence hydrotherapy also, as long as the limb is dried and kept clean afterwards.

Once radiographs demonstrate bone healing, weights and floats may be added to increase resistance.

Non-use of a limb

Non-use of a limb can occur after any limb injury or surgery, but is typically worse in dogs with complex injuries, multiple surgeries, those that have been immobilised for lengthy periods and anxious dogs. Complications of prolonged non-use of a limb include joint contractures, muscle shortening, muscle wasting, altered mechanics of other body parts, anxiety, muscle pain and hypersensitivity. It is important for the referring veterinarian to assess and treat the dog for any condition that may continue to promote non-use, such as pain. Hydrotherapy can be very beneficial to encourage gentle limb use, stretching and strengthening of the limb, often with swift carry-over to the land. The warmth of the water can relax the muscles holding up the limb and allow the limb to begin to be used. The buoyancy of the water reduces the load on the limb and often makes walking in the underwater treadmill, or swimming possible. Even if the dog is still reluctant to use the limb outside the pool or treadmill, the water provides an avenue to start to stretch and strengthen in the interim. Assisting the dog's limb to move either in the pool or treadmill can also be very beneficial to start limb movement in those somewhat more reluctant.

Spinal injury or surgery

Dogs with paralysis and paresis and one or multiple limbs affected, benefit greatly from hydrotherapy. Hydrotherapy can commence once the spine is stable. Patients are exercised in the underwater treadmill from day two after surgery, where they are assisted to move their limbs gently, with buoyancy supporting them, keeping the wound out of the water. Limbs that are very weak can be assisted to swim and walk, often resulting in a faster return to walking on the land. Dogs that are too weak to walk on the land will often swim or walk on the underwater treadmill first, then using the hydrotherapy to gain strength and control, can transfer over to independent walking on land. The use of a buoyancy vest is beneficial to assist in trunk control, and can be removed as the dog strengthens, to help trunk control

develop. Even if limb movement is not initially seen in the water, perseverance is worth while, as the weightless environment means small muscle contractions are often possible here initially, compared with attempting to move against gravity out of the water. Where possible, daily hydrotherapy is recommended for these patients until they can walk independently on land. This group of patients often benefits from ongoing weekly hydrotherapy where available, particularly if they have any deficits remaining after rehabilitation is complete.

Arthritis

Hydrotherapy is very beneficial in this group of patients for allowing strengthening without loading, increasing and maintaining joint range of motion, maintaining cardiovascular fitness and assisting with weight loss, when exercise on land may be difficult. This group of patients benefits from ongoing hydrotherapy, where available, once or twice a week. A variety of devices can be used to make the work of swimming or walking in the water more or less difficult depending on how the dog is affected and in which joints. Dogs may need assistance in and out of the water, but often cope well once the buoyancy of the water supports them, and can exercise in the water with minimal assistance.

11.7.3 Exercise prescription and monitoring

Warm-up, exercise component and cool down

As with all exercise programmes, each hydrotherapy session should have a warm-up, exercise component and cool down. This may simply be that exercise commences slowly, increases in intensity and then slows down before ceasing. Alternatively, the warm-up and cool down may be done outside the pool. In the case of exercising for particular conditions, massage, stretching or other land-based exercises may also make up part of the warm-up and cool down, or be done during the rests breaks between bouts of exercise.

Intensity of treatment

Intensity is also a consideration for each animal's hydrotherapy programme. Intensity of exercise in the underwater treadmill is easily controlled through depth of water, and speed of walking. Swimming intensity cannot be so easily controlled, but control over the number of laps swum before a rest, rest time, and use of a buoyancy vest are all ways to control exercise intensity while swimming. In general, as animals improve, the intensity of exercise is increased so they can adapt and strengthen.

Duration of treatment

Treatment time, will be affected by several factors including the dog's prior experience of being in water, anxiety, age, weight, pre-existing conditions, e.g. heart problems, recent limb surgery, previous fitness level.

In general, the more debilitated the animal is, the shorter the treatment time will need to be. The initial session may

consist of swimming three or four half-laps in the pool, with several minutes rest in between, or walking for three sets of 1 minute at a time on the underwater treadmill, with rests in between.

Monitoring

Monitoring of heart rate can be useful to help decide the length of exercise and rest sessions. Alternatively, watching for breathing rate and signs of distress can be helpful to avoid exhaustion.

Normal resting heart rates for dogs range between 50 bpm and 160 bpm, depending on size and fitness. Some puppies and toy breeds may have heart rates up to 180 bpm at rest.

At this stage, there are no documented 'target heart rates' for dogs. It has been suggested that the desirable difference between pre- and post-exercise heart rates is 20–50 bpm (Lamoreaux 2002). Until further studies are performed to determine appropriate heart rates for exercising dogs in water, it is better to do a small amount of hydrotherapy initially and increase slowly each session as the animal allows.

Normal respiratory rate is 16–20 breaths/min, with larger, fitter dogs having a rate as low as 10 breaths/min, and overweight or unfit dogs may be at 30 breaths/min. There are no values for exercising respiratory rates at present.

Further studies are required to determine the best way to monitor and when to monitor.

Frequency of treatment

As in human physiotherapy, we generally attain faster return to function with more intensive rehabilitation. The same occurs with rehabilitation and hydrotherapy in small animals.

For *orthopaedic* cases, the recommendation is two or three times per week for the first 4–6 weeks. After this time, depending on how the patient is progressing, they may reduce down to one or two per week until the 12-week mark. This will not be possible for many owners and once a week or fortnight has to suffice. Hydrotherapy will still benefit these patients but the rehabilitation process will take longer.

For *neurological* cases, daily hydrotherapy is recommended, especially in the first 2–3 weeks, to make use of neural plasticity.

Progression of treatment

Treatment needs to be progressed in order to overload the body slightly, and cause it to adapt and strengthen. Following each session, ask the owner to monitor their animal at home over the next 24 h and record anything that indicates we may have done too much.

Questions to ask on the next visit:

- How was the animal after the session? (Lameness same/worse/better; may have been tired, developed muscle soreness, been a little stiff)
- If the animal was more lame, how long did it last? (Few hours or until the next morning is OK, 24 h+ and we have done too much)
- How has the animal been since then? (May have been same, continued to improve, got worse)
- Did you increase any other exercises? (May have increased walk or other home exercises – decide if this contributed to lack or progression or deterioration if occurred)
- Did you give the dog any NSAIDs or ice/heat/massage, etc. if worse?
- How is the dog today and what exercise have you done before coming here today?

The answers to these questions will help you decide how to progress the patient's exercises for that day's session, as well as advising on what home exercise to do afterwards.

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12

Acupuncture and trigger points

Tina Souvlis

12.1 Introduction
12.2 Traditional acupuncture
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12.1 Introduction

Trigger points or areas of increased sensitivity within muscle are frequently diagnosed and managed in musculoskeletal pain conditions in both humans and animals. The aetiology of these trigger points is currently not fully understood, however, an understanding of the complex pathophysiology underlying the development of these points is emerging. Use of needling by physiotherapists for the treatment of musculoskeletal conditions, including trigger points, is becoming more widespread in people.

There are a number of philosophies that underpin the use of dry needling. For example, acupuncture may be considered as one of the principle elements used in Traditional Chinese Medicine (TCM) for the treatment of many conditions including those treated predominantly by physiotherapists such as musculoskeletal pain and disability. In a limited description, acupuncture can be considered as the application of fine needles to specific points throughout the body for the management of many conditions such as musculoskeletal pain, respiratory, endocrine and immune conditions. Although it is primarily thought of in TCM to be a method of balancing energy flow throughout the body, research has demonstrated that it can elicit a strong multi-system physiological response. This treatment approach has a long history over many thousands of years and indeed acupuncture has long been used as part of veterinary practice, with good results being reported for disorders of the musculoskeletal, cardiovascular, respiratory and gastrointestinal systems.

Modern practice often uses a modified or Western approach to acupuncture, which is based on modern physiology and anatomy instead of the ancient 'point' system. However, in order to integrate this treatment approach into a broader medical frame – knowledge of the traditional practice, the evidence base and the clinical implications of the treatment should be understood.

This chapter will outline:

- The use of acupuncture
- Potential mechanisms for its effect
- Evidence of the clinical efficacy of acupuncture in humans and animals in the management of musculoskeletal pain conditions

The nature of trigger points in musculoskeletal pain and the role of physiotherapy, including dry needling, will be discussed.

12.2 Traditional acupuncture

Underpinning TCM and the use of acupuncture is the concept of Qi (pronounced Chi). All things in the world are thought to be composed of Qi. It sustains movement and change and it is thought to be what animates living beings. It can protect against harmful influences and flows with blood in the blood vessels and in special channels or meridians. Both too much and too little Qi is thought to be harmful and can produce disease in the body.

In addition to the concept of Qi, that of *yin* and *yang* is fundamental to the practice of TCM and acupuncture. These constructs apply to the universe – not just to the body. In the biological sense, yin is considered female and yang is male. They are opposites, but not mutually exclusive; meaning that yin always contains some yang and vice versa. They cover a wide array of options, for example – yin may refer to cold, rest, passive, inward movement whereas yang refers to heat, movement, vitality and outward movement. Yin and yang interact with each other and can transform from one to the other. Hence a concept of constant flow and movement is represented. The health state depends on a balance between yin and yang, with disease states occurring when either one or the other is dominant. The meridians or channels in which the energy of yin and yang flows are thought to carry Qi to the organs. The cosmic influence of 'wind' is also thought to disrupt the balance of yin and yang.

The meridians are thought to run within the body and come to the surface only at specific points. These are considered to be the acupuncture points. However, there are a few points that do not lie on channels (so called extra-meridian points). There are 12 main channels that carry acupuncture points. The paired channels are related to and named for the organs through which they run, such as liver, gallbladder lung, etc. Although they have the same names, the Western and TCM concept of organs are only loosely related. There are thought to be six primarily yin channels linked to solid organs such as the liver and six yang channels linked to hollow organs such as the stomach. Acupuncture points are a mechanism for perturbing the flow of energy and restoring balance (Vickers & Zollman 1999). In addition, acupuncture points are also thought to be the entry points in the skin for entry and exit of 'wind'.

Whilst there are many different methods using acupuncture points for treatment – it is generally accepted that successful activation of the acupuncture point is accompanied by a sensation known as 'de Qi'. This evokes in the patient a sense of heaviness, dullness or deep ache within the tissue. It is thought that inserting the needle in the area surrounding the acupuncture point will not produce this sensation and the accompanying (desired) effect.

12.3 Acupuncture analgesia

Although acupuncture can be used in the management of many conditions including nausea, asthma, drug addiction and dental pain (Birch *et al.* 2004), the focus of this chapter is on the use and possible mechanisms of acupuncture in musculoskeletal pain conditions. The analgesic effect of acupuncture is thought to be related to activation of descending pain-inhibitory pathways. These pathways can be activated by a number of stimuli including stress, pain and activity.

12.3.1 Descending pain-inhibitory system (DPIS)

The perception of pain and analgesia involves a number of levels within the nervous system from periphery to cortex and broadly speaking, two systems produce this cognition (Kanjhan 1995). The *ascending system* relays information regarding peripheral nociception via the spinal cord to higher centres (Cross 1994). The *descending inhibitory system* also involves activation of the central nervous system (CNS) at many levels, including cortex, thalamus and brainstem, and determines the response to this peripheral input by modulating the functional state of the nociceptive system at spinal cord level and at higher centres within the neuraxis (Cross 1994; Kanjhan 1995). Activation of peripheral nociceptors does not necessarily result in the perception of pain, and as such, this perception reflects the balance of ascending input and descending controls. Although the CNS has also been shown to facilitate processes to increase pain, the focus of this chapter will be only

the analgesic responses induced by activation of inhibitory functions with the CNS.

It has been demonstrated that descending inhibition of pain is produced by a number of mechanisms that can be categorised as either opioid or non-opioid (Watkins *et al.* 1992). The definite nature of this terminology may not reflect the true character of the complex interactions that exist to modulate pain. The terms '*opioid*' and '*non-opioid*' may describe two ends of a continuum rather than two separate systems (Watkins *et al.* 1992). However, for the purposes of this review, the terms opioid and non-opioid will be used to summarise the characteristics of the different modulatory responses.

12.3.2 Opioid analgesia

It has been recognised for thousands of years that opiates such as morphine are highly effective pain relievers. However, it was not until recently, in the middle to late twentieth century that the mechanism was uncovered by which opioids produced their effects. Opioid receptors to which these substances bind to produce the analgesic effects were located in the nervous system. It was reasoned that there must be peptides produced within the body that can also bind to these receptors. Subsequently, endogenous opioid peptides, such as β -endorphins, which also produced analgesic effects were identified and characterised (Kieffer *et al.* 1992).

These receptors are differentially located throughout the CNS in areas where pain modulation occurs; however their roles in the modulation of pain transmission have yet to be fully revealed (Fields & Basbaum 1994; Mansour *et al.* 1995; Gaveriaux-Ruff & Kieffer 1999). It appears that opioids can exert analgesic effects both peripherally and centrally (Stein *et al.* 1990; Spike *et al.* 2002) and by pre-synaptic or post-synaptic mechanisms. The most important supraspinal sites for pain modulation by opioids are the periaqueductal grey region (PAG) and the rostral ventromedial medulla (RVM) (Mayer & Price 1976; Yaksh *et al.* 1988; Heinricher & Morgan 1999). Opioids injected into or released at these sites produce an inhibitory effect on nociceptive processes at spinal cord level, as well as attenuating supraspinal responses to nociceptive stimuli (Heinricher & Morgan 1999). Opioids can also exert a powerful effect when administered directly at the level of the dorsal horn of the spinal cord by controlling the release of neurotransmitters such as substance P (SP) and calcitonin gene-related peptide (CGRP) by nociceptive afferents (Cesselin *et al.* 1999). Yaksh *et al.* (1980) were able to demonstrate the pre-synaptic inhibition of release of substance P following noxious stimulation. In addition, Trafton *et al.* (1999) have suggested that internalisation of the μ receptor in neurones may point to a post-synaptic mechanism.

As mentioned above, opioid peptides both endogenous and exogenous, act by binding to opioid receptors. Opioid peptides are released in response to a number of stimuli

including pain, injury, exercise and stress (Meyer *et al.* 2000; Vaccarino & Kastin 2001). The endogenous opioid families include endorphins, enkephalins and dynorphins and the recently cloned endomorphins (Zadina *et al.* 1997). The cells that are genetically programmed to produce opioids are extensively distributed throughout both the peripheral and central nervous systems (Roques *et al.* 1999; Przewlocki & Przewlocka 2001).

Opioid analgesia has specific characteristics. Note that, the pain relieving (and other effects) of opioids can be reversed by the administration of an opioid antagonist, for example naloxone. Antagonists act by occupying the receptor sites and preventing the ligand-receptor binding of the opioids. Another characteristic of opioid-induced analgesia is the development of tolerance that can be described as a reduction in the analgesic effect following repeated administration of the opioid.

Analgesia that does not exhibit naloxone sensitivity or tolerance is described as non-opioid (Fields & Basbaum 1994). The mechanisms behind non-opioid analgesia have attracted recent investigation. Jacquet (1988) demonstrated involvement of the N-methyl-D-aspartate (NMDA) receptor in this form of analgesia. This receptor is widely located throughout the CNS and has previously been implicated in memory and learning. NMDA receptors are a specific subgroup of glutamate (an excitatory amino acid) receptors, which reportedly act in the mediation of non-opioid analgesia and studies have demonstrated reversal of non-opioid analgesia by administration of the non-competitive NMDA receptor antagonist MK-801 (Marek *et al.* 1991; Vaccarino *et al.* 1992). This receptor may also play an important role in the development of tolerance (Trujillo & Akil 1991). Noradrenergic and serotonergic mechanisms are also thought to be involved in mediating non-opioid analgesia.

How does acupuncture produce its pain-relieving effects? Acupuncture analgesia can occur following needling with and without associated electrical stimulation. It is thought that acupuncture stimulates the A δ fibres which are thinly myelinated fibres activated by a range of noxious mechanical and thermal stimuli. Although acupuncture needles may not cause any pain or sensation on insertion, the patient will often describe a sense of soreness or heaviness ('de Qi'), which the practitioner may use as evidence that the acupuncture point has been located. Afferent input from these fibres can inhibit pain through activation of the DPIS (Vickers & Zollman 1999).

Both opioid and non-opioid analgesia can be demonstrated following stimulation by acupuncture. Responses appear to be dependent on the parameters of the acupuncture stimulus when acupuncture is augmented by electrical stimulation. Low frequency and high intensity stimulation are related to an opioid mechanism of pain relief whereas high frequency and low intensity stimulation may produce an analgesic effect that is dependent on a γ -aminobutyric

acid (GABA) mediated system (Jeong *et al.* 1995). Chen and Han (1992) have suggested that different frequencies of stimulation result in activation of different opioid receptors. Low frequency stimulation (2 Hz) activates μ -receptors and δ -receptors, high frequency (100 Hz) stimulation are mediated by κ -receptors, whereas 2–15 Hz stimulation activates all three receptors. These findings show some similarities in results to investigations by Sluka *et al.* (1999) who have demonstrated that analgesia following high frequency (100 Hz) TENS stimulation was δ -receptor mediated whereas analgesia mediated by low frequency stimulation (4 Hz) was associated with μ -opioid receptors. A recent study in healthy human volunteers demonstrated that low frequency (4 Hz) high intensity stimulation that was tolerable but not noxious produced a significantly greater hypoalgesia than placebo stimulation (Barlas *et al.* 2006). Low intensity rated by the participants as strong but comfortable did not show any statistical difference to the placebo condition.

Sympathetic nervous system responses, which accompany analgesia, have also been demonstrated. Takeshige *et al.* (1992) have demonstrated that an initial excitatory response is followed by subsequent sympathoinhibition within a time frame of 20–45 minutes. Acupuncture needles are commonly left *in situ* for between 10 and 40 minutes (Vickers & Zollman 1999) as it may take this length of time for sympathetic nervous system inhibitory effects to take place. The significance of these findings may be related to the fact that the PAG is not a homogenous structure. It is made up of columns of cells that vary in length and thickness from rostral to caudal. The nature of the response may therefore depend on which part of the PAG is activated by the peripheral stimulus. The ventrolateral columns (vlPAG) and the lateral columns (lPAG) have a modulatory role over the sympathetic nuclei in the brain stem and these can be differentially activated depending on the originating site within the PAG for the neuronal drive (Lovick 1993; Lovick 1997). Deep somatic and visceral nociceptive afferents have been found to project to a discrete region of the vlPAG (Keay *et al.* 1994). This region is also responsible for a functionally significant response to pain and stress which includes quiescent behaviour and opioid-mediated analgesia (Keay *et al.* 1994).

12.4 Clinical effectiveness of acupuncture

Acupuncture is a relatively safe procedure with limited reporting of serious events (Birch *et al.* 2004) and although widely used clinically, there is limited support for the effectiveness of acupuncture for the management of chronic pain (Ezzo *et al.* 2000).

In research, issues with blinding and difficulty with the development of an adequate placebo technique have led to many of the studies investigating the efficacy of acupuncture being poorly controlled (Sjolund 2005). However, the

advent of the placebo needle (Streitberger & Kleinhenz 1998) has begun to impact positively on the quality of research in the area and recent studies have started to show the efficacy of acupuncture and acupressure in a number of conditions.

The effect of acupuncture and acupressure on low back pain (LBP) has been investigated. A large study investigating the long-term benefits (over 24 months) of acupuncture compared with standard medical (GP) care for chronic LBP found that 81% of patients stated that acupuncture had helped their back pain compared with 52% who had standard GP care (Thomas *et al.* 2005). Hsieh *et al.* (2006) demonstrated that acupressure had a statistically superior effect to physical therapy, both after the course of treatment and at the 6-month follow-up.

In a systematic review of the efficacy of acupuncture, Birch *et al.* (2004) suggest that the evidence is limited for the use of acupuncture in the management of conditions such as fibromyalgia, osteoarthritis of the knee, neck pain and tennis elbow. In general, the results are considered inconclusive and further research is required.

12.5 Use of acupuncture in animals

Acupuncture is becoming a more widely recognised treatment option in animals over the last few decades and The International Veterinary Acupuncture Society was founded in 1974 (IVAS 2006; Chan *et al.* 2001). The more recent rapid development of veterinary acupuncture has been driven by owners following media focus on its efficacy (Chan *et al.* 2001; Scott 2001). Acupuncture has been used for a variety of conditions including bovine reproductive disorders, canine paralysis and lameness and equine back pain (Chan *et al.* 2001). Conditions treated by physiotherapy that may also be amenable to acupuncture can include vertebral disorders, degenerative disc disease, joint disease and pain. The particular acupuncture points used are transposed from human points as TCM texts only provide points for large animals and birds (Yu & Lin 2000; Chan *et al.* 2001).

As for humans, there is limited evidence of the effectiveness of acupuncture in animals. A study investigating the efficacy of acupuncture on induced arthritis in the stifle joint of dogs has demonstrated a superior effect of acupuncture in over non-treatment. Skin temperature was measured using infrared thermography and following treatment for 3 weeks (one session/week), there was a return to normal of skin temperature only in the acupuncture group (Um *et al.* 2005).

In conclusion, further research is required to provide support to anecdotal reports of the efficacy of acupuncture. Although early studies were predominantly of low methodological quality, more recent research has begun to elucidate the mechanisms and the efficacy of acupuncture compared with both placebo treatment and more traditional

therapies. The mechanisms underpinning hypoalgesia following acupuncture are complex and most likely include activation of multiple central nervous system pathways. Veterinary acupuncture is becoming established to treat a variety of conditions including musculoskeletal pain and dysfunction and further well-controlled studies are required to provide an evidence base for its effectiveness.

12.6 Trigger points

Areas of increased sensitivity within a muscle are commonly referred to as 'trigger points'. It must be noted that although Travell and Simons (1993) have described trigger points to be pathognomic of myofascial pain syndrome (MPS), local muscle pathology may only be one of the mechanisms by which this hypersensitivity occurs. Tenderness or mechanosensitivity in muscle may be related to changes in muscle activity and sensitivity as a result of joint pathology, visceral pathology and nervous system pathology (Giamberardino *et al.* 1999; Johansen *et al.* 1999). It may occur due to both peripheral mechanisms of sensitisation (Graven-Nielsen & Mense 2001) and represent areas of secondary hyperalgesia related to central sensitisation mechanisms (Graven-Nielsen & Arendt-Nielsen 2002). In this section, the mechanisms and management underpinning so-called 'trigger point' development and their management will be discussed.

Trigger points can be described as local tender spots located in a band of skeletal muscle that produce pain on compression which is felt as pain locally, but can also evoke pain in a referred pattern (Travell & Simons 1993). The aetiology and pathophysiology of trigger points remains speculative; however, clinically, trigger points are thought to produce pain, restrict movement, cause muscle weakness and alter the muscle activation patterns in both local and distal muscle groups (Alvarez & Rockwell 2002). Autonomic signs such as hyperaemia, vasomotor and temperature changes and symptoms including dizziness and paraesthesia may be evident in patients with trigger points (Fricton *et al.* 1985; Jay 1995).

12.6.1 Diagnosis of trigger points

In addition to local and referred pain, which reproduces symptoms, a local twitch response and presence of a taut pain on palpation are considered to be characteristic of a trigger point (Borg-Stein & Stein 1996; Borg-Stein & Simons 2002). Although trigger points have long been recognised as a pathological entity clinically, these diagnostic criteria are not universally accepted (Borg-Stein & Simons 2002) and there is equivocal support in the literature that these symptoms are reproducible between examiners. Some authors contend there is limited agreement (Nice *et al.* 1992; Wolfe *et al.* 1992; Lew *et al.* 1997; Hsieh *et al.* 2000) whereas others have shown statistically significant reliability for location and identification of trigger points

(Gerwin *et al.* 1997; Sciotti *et al.* 2001). Use of specific diagnostic criteria by specifically trained and experienced investigators for research studies could assist in reducing this discrepancy.

12.6.2 Possible mechanisms

Pathophysiology of either the motor endplate or the muscle spindle has been proposed as potential causes of the trigger point. Measuring electromyogram activity with a needle electrode, Hubbard & Berghoff (1993), hypothesised that this activity originated in the muscle spindle. They suggested that the tenderness at the trigger point site was due to increased pressure within the spindle, however no studies have been able to measure intrafusal muscle fibre activity (Hong & Simons 1998) and therefore allow a comparison with the activity described by Hubbard and Berghoff (1993).

It has been suggested that there are physiological characteristics that are shared by the twitch response and the stretch reflex. Rivner (2001) demonstrated bursts of activity from insertion of a needle electrode into rabbit trigger points, which showed similar profiles for amount and duration of activity and behaviours to those occurring in the stretch response resulting from muscle spindle activity.

In contrast, a conflicting view by some researchers postulates that the activity observed by Hubbard & Berghoff (1993) is more likely to be recording of endplate noise (Simons *et al.* 2002). In their review on myofascial pain, Borg-Stein and Simons (2002) have suggested that endplate noise is related to a 'pathologic increase in release of acetylcholine (ACh) by the nerve terminal of an abnormal motor endplate under resting conditions'. This assertion is supported by evidence demonstrating endplate noise significantly more frequently in trigger points than in areas outside trigger points (Coupe *et al.* 2001; Simons *et al.* 2002).

As mentioned earlier, tenderness in muscle is thought to include elements of peripheral and central sensitisation of the nervous system. The pathophysiology of the trigger point has been described as a complex interplay between peripheral and central mechanisms (Borg-Stein & Simons 2002) whereby affected endplates are thought to release excessive ACh causing sustained depolarisation of the muscle fibre, thereby producing a sustained shortening of sarcomeres. As a consequence, an increase in local energy consumption and decrease in local circulation occurs, resulting in localised ischaemia and hypoxia. The release of inflammatory mediators, such as prostaglandin, bradykinin, serotonin and histamine, stimulated by the local ischaemia, is thought to sensitise afferent nerve fibres accounting for the hyperalgesia or tenderness in the muscle (Borg-Stein & Simons 2002). A recent study has demonstrated that change in the muscle microcirculation is thought to play a role in the pain and development of

tender points associated with trapezius overactivation (Larsson *et al.* 2004). Sustained sarcomere shortening may produce contraction within the muscle and explain the presence of the palpable taut band (Borg-Stein & Simons 2002).

In order to effect the most appropriate treatment, more research is required to clarify the pathophysiology of MPS trigger points. It may be that there is not a single pathophysiology, but instead that both muscle spindles and endplates are implicated in the pathogenesis of trigger points. Peripheral and central nervous system mechanisms play a role in the development, maintenance and spread of trigger points and therefore these mechanisms should be considered when considering the effects of treatment.

12.6.3 Treatment

To guide patient management, the physical examination should consider posture, biomechanics and joint function to establish the presence of underlying mechanisms for the development of local or referred pain of MPS trigger points (Borg-Stein & Simons 2002). The presence of focal tenderness and reproduction of the patient's complaints as well as taut bands within the muscle should be elicited. Treatment should then include specific interventions to modify or prevent factors that may have predisposed to the development of trigger points.

There is limited evidence for the use of pharmacological treatments in the management of MPS trigger points. Medical management of patients with pain may include the use of non-steroidal anti-inflammatory drugs, antidepressants and anticonvulsants although there is no evidence for their use in MPS; however their prescription may be based on research for these medications in fibromyalgia (Borg-Stein & Simons 2002). There is some evidence however for the use of *Botulinum* toxin as it has the potential to disrupt the abnormal endplate dysfunction by blockade of the release of ACh (Borg-Stein & Simons 2002). It may also have a central mechanism at the spinal cord and brainstem (Borg-Stein & Simons 2002; Gobel *et al.* 2001).

Common physiotherapy interventions for trigger points include massage, stretching and exercise. Electrophysical agents such as TENS, ultrasound and laser have also been used. Although there are anecdotal reports of the effectiveness of these treatments, there is limited evidence of their usefulness. Well designed placebo-controlled trials are necessary to provide therapists with support for these interventions.

Wet and dry needling have proved useful in the management of MPS trigger points to augment non-invasive stretching and exercise. Injection of steroids, local anaesthetics and, as discussed above, *Botulinum* toxin have been used to inactivate the points. The lack of inflammatory mechanisms does not support the use of steroids, however use of anaesthetic may reduce the amount of post-needle soreness (Borg-Stein & Simons 2002).

Dry needling refers to the use of acupuncture needles to stimulate muscle or inactivate trigger points without the injection of substances such as local anaesthetics or corticosteroids. There is no evidence to support the superior effects of wet needling, over and above the effects of dry needling (Cummings & White 2001). Dry needling has shown benefits in treating musculoskeletal pain (Edwards & Knowles 2003) and in previous, uncontrolled, clinical studies needling of the trigger point has been reported to be effective in reducing pain (Gunn *et al.* 1980; Hong 1994).

The mechanisms supporting the use of dry needling include those that were previously mentioned for acupuncture. For example, the use of superficial dry needling as advocated by Baldry *et al.* (2001) may activate the DPIS through stimulation of the A δ fibres. However, additional specific local effects may be produced by deeper stimulation (e.g. intramuscular stimulation) (Gunn 2003), which produces a twitch response. Alterations in peripheral circulation including an increase in muscle blood flow for tibialis anterior and in the overlying skin was demonstrated in asymptomatic and fibromyalgia patients (Sandberg *et al.* 2005). These authors showed that deep needling was superior to subcutaneous needling in healthy and trapezius myalgia subjects for increasing muscle blood flow. However, superficial needles produced more increase in blood flow than deep needles in fibromyalgia patients (Sandberg *et al.* 2005). Acupuncture can also be applied to trigger points and the location of trigger points is often coincident with traditional acupuncture points (Borg-Stein & Simons 2002). Although the needles used in the two interventions are the same, the application method differs with acupuncture needles often left *in situ* for 20 minutes or longer whereas techniques for muscle stimulation will be introduced and removed from the muscle quickly. Electrical stimulation can be used and added to both interventions. Therefore the type of needling used may be relevant to the patient and the mechanism of their complaint.

12.6.4 Trigger points in animals

In animals, trigger points may be associated with pain and lameness. They may also be associated with autonomic disturbance (Schoen 2001). Factors such as arthritis, trauma, stress, postoperative status and infection may predispose to the development of trigger points in animals (Schoen 2001). They are diagnosed by palpation, which produces vocalisation in the animal and may highlight the presence of the taut band. They can also cause a reduction in range of motion (Schoen 2001) and as in humans – they may be co-localised with acupuncture points (Janssens 1992). Trigger points have been described in the triceps, infraspinatus, quadriceps, pectineus, iliocostalis lumborum, peroneus longus, and gluteus medius muscles (Janssens 1991) in dogs.

Treatment approaches are similar to those used in humans and include stretching, massage, TENS and laser

(Schoen 2001), as well as wet and dry needling. These approaches to management have very limited research support. In a study in which lame dogs were treated with needling or injection with local anaesthetic into trigger points, 60% of the animals ($n = 48$) showed complete recovery (Janssens 1991). These animals had previously been unsuccessfully treated with medication and acupuncture. Further research in this area would clearly be useful to guide management.

In conclusion, acupuncture has its basis in TCM and has been used for centuries for the management of many conditions including those regularly managed by physiotherapists. The evidence for the mechanism of pain relief, suggests that activation of the DPIS is important in mediating the effects of acupuncture. Although the location of trigger points and acupuncture points may coincide, the potential mechanisms for the development of trigger points suggests changes at either the muscle spindle or the motor endplate including abnormal release of ACh at the motor point. Further investigation is necessary to determine conclusively the underlying pathophysiology of the trigger point, and the relationship between the acupuncture and the trigger point should be established so that management can be optimal. In addition, the direct effect of dry needling as an isolated treatment should be established to guide physiotherapists in the most appropriate ways to manage trigger points and musculoskeletal pain.

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13

Canine treatment and rehabilitation

Laurie Edge-Hughes and Helen Nicholson

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13.1 Introduction

It is clearly impossible in this textbook to cover all potential small animal physiotherapy applications. However, this book is intended to support physiotherapists in translating their human physiotherapy skills to animals, not to provide generic recipe-based treatment and rehabilitation. Therefore, principles and clinical reasoning will be the focus of this chapter with clinical case studies to illustrate this approach. The chapter will cover five main areas: orthopaedic and sporting, by Laurie Edge-Hughes; and respiratory, cardiac and neurological, by Helen Nicholson.

13.2 Canine orthopaedic rehabilitation

Laurie Edge-Hughes

Physiotherapy for orthopaedic conditions has a strong scientific background in human medicine. The canine physiotherapist should be aware of and rely on what is known to be effective in human practice and apply many of the same therapeutic goals, strategies and techniques to the animal patient when at all possible. Crucial to success of application to small animals is knowledge of the differences in canine anatomy, biomechanics, disease processes and surgical interventions. Many of the techniques are simple to apply, but it is the clinical reasoning as to when each should be applied that takes professional judgment and skill.

13.2.1 Soft tissue lesions: muscle, tendon and ligament

Forces great enough to cause musculotendinous injuries often occur during acceleration–deceleration phases of activities. Very high forces within the musculotendinous complex occur during eccentric muscle contractions, especially at high velocity and in an oblique direction (Fitch *et al.* 1997; Steiss 2002; Sharma & Maffulli 2005). A repetitive

overload beyond the physiological threshold may result in fibre degeneration, disordered arrangement of collagen fibres and an increase in vascularity (Khan *et al.* 1999). Various types of tendon degeneration occur, when some calcium deposits accompany the lesions in both humans and animals. Pain in these cases can be attributable to both mechanical and biochemical factors but not necessarily due to inflammation alone (Sharma & Maffulli 2005). Inflammatory lesions are infrequent and most commonly associated with partial ruptures (Khan *et al.* 1999). Degenerative tendinopathy is the most common histological finding in spontaneous tendon ruptures (Sharma & Maffulli 2005). Tendinopathy can refer to *tendinosis* (an intratendinous degeneration), *tendinitis/partial rupture* (symptomatic degeneration of the tendon with vascular disruption and inflammatory repair response) and *paratenonitis* (inflammation of the outer layer of the tendon alone (Khan *et al.* 1999). Ligamentous injuries are usually caused by traumatic overloads in atypical joint movements (i.e. lateral shearing or rotation of the stifle) or an overextension of a normal physiological motion (i.e. hyperextension of the carpus or stifle). However overuse degenerative lesions may also occur in ligamentous structures such as the cranial cruciate ligament in dogs (Jerram & Walker 2003).

13.2.2 Grading of soft tissue injuries

Utilising a grading system of sprains and strains and healing stages greatly enhances the practitioner's ability to successfully rehabilitate muscles, ligament or tendon lesions. When ligaments or tendons are sprained or strained, micro- or macro-tearing (partial rupture) has occurred at the level of the fibres with a predictable damage pattern (Magee 1986; Kujala *et al.* 1997; Steiss 2002).

- *Grade one:* Micro-tearing of the inner fibres within the structure, with the surrounding sheath intact.

- *Grade two*: Both micro- and macro-tearing of the inner fibres and the sheath occurs. Grade two lesions can vary in degrees of fibre damage.
- *Grade three*: Complete rupture of the fibres. Non-surgical healing of the structure is impossible, because no fibres are left intact. Nerve fibres within the structure are also ruptured, therefore animals may exhibit less pain than those with a partial lesion. However, the associated joint(s) may be unstable or functionally compromised as a result of a complete lesion (Kujala *et al.* 1997); an example being a rupture calcaneal tendon in the dog.

13.2.3 Assessment of soft tissue injuries

Assessment of the injury requires an in-depth knowledge of anatomy and exceptional palpation skills, because animals cannot verbalise where and when pain occurs, or understand verbal instructions, such as voluntary muscle contractions or resisting motion. Assessment relies on palpation of the specific muscle structure, including its entire length, while targeting the musculotendinous and teno-osseous junctions where structural failure most commonly occurs (Maganaris *et al.* 2004). A cross-fibre, deep palpation can be utilised to determine tenderness or tissue irritability. Irritability on palpation can be correlated with specific muscle stretching of that structure. A strained muscle or tendon will be painful to stretch (Edge-Hughes 2004, 2005). Ligament injuries are assessed similarly; utilising direct palpation of the entire length of the ligament and ligamentous stress/provocation test (i.e. gap testing). As in human orthopaedic medicine practices, the intracapsular ligaments, although non-palpable, can be examined using both functional assessment and provocation tests, such as the cranial drawer and tibial thrust tests for the cranial cruciate ligament in the dog (see Chapter 6).

13.2.4 Healing stages and treatment of acute soft tissue injuries (partial ruptures)

The treatment goals are to first relieve the signs and facilitate healing then if possible identify and correct the causative factor(s) (Wang *et al.* 2006).

Stages of connective tissue repair are sequential and predictable, the following phases occur in the healing process.

Early Stage 1

This is the *haemorrhagic phase* (24–48 hours). Cellular changes and swelling occur as erythrocytes and inflammatory cells (i.e. neutrophils) enter the area (Sharma & Maffulli 2005). The treatment goals are to minimise the initial bleeding, control swelling, prevent stress and tension, control inflammation and reduce pain; therefore following the RICE principles; *Rest, Ice, Compression and Elevation*. Depending on the severity of the lesion, generally rest is beneficial only in the first 24–48 hours to allow the active bleeding to stop. After 48 hours it is counterproductive to healing, hence controlled, pain-free movements should

be initiated while considering the lesion's vulnerability (Kerkhoffs *et al.* 2002; Geffen 2003). Ice assists in the reduction of swelling and pain by promoting vasoconstriction, decreasing inflammation and reducing nerve conduction velocity (Rees *et al.* 2006). Evidence in the literature varies depending on the site of the lesion, insulating effect of the hair, adipose tissue and the method of application. As a general rule ice should only be applied for 10–15 minutes per application. Longer applications may result in cyanosis of the skin and also be counterproductive due to the 'hunting response'; in humans, vasodilation occurs with prolonged icing thus more bleeding and swelling occurs in the area (Ho *et al.* 1995). Cyclical icing using a procedure of 10–15 minutes on and 10–15 minutes off is recommended in human practice. A compression or elastic bandage should be used around the affected joint, combined with icing – especially in lesions where the animal is predominantly recumbent. Care should be taken to avoid impairing the circulation distal to the compression bandage by wrapping a limb too tight, thus the animal must be supervised and circulation monitored. Elevation, while good in theory, is not always practical for the canine or feline patient. Environmental considerations are vital, such as providing the animal with non-slip surfaces, adequate confinement and sleeping surfaces. Use of inexpensive rugs to create a pathway over hardwood or linoleum flooring can aid in avoiding slip-related reinjury and gaining animal confidence in ambulation with early use of the affected limb (Edge-Hughes 2005).

Late Stage 1

This is the *substrate phase* (days 3–5). Dead cells and damaged collagen are removed or broken down. Monocytes and macrophages predominate and phagocytosis of necrotic material occurs. Vascular permeability is increased. Granulation tissue begins to form, which needs oxygen and nutrients, so capillaries divide and grow in the area (Sharma & Maffulli 2005). The goal here is to assist in removal of traumatic inflammatory exudates so that healing may begin. At this point you can still choose ice and compression, but also consider physiotherapy modalities such as pulsed (non-thermal) ultrasound, low dose laser or pulsed electromagnetic field therapy (PEMF) at the lowest setting (Michlovitz 1990; Lee *et al.* 1997; Saini *et al.* 2002; Green *et al.* 2003). Grade one Maitland's mobilisations have been utilised in resolving joint pain and inflammation, and may be useful in addressing adjacent or intra-articular ligament sprains (Maitland 1966; Gross-Saunders *et al.* 2005).

Stage 2

This is the *regeneration phase*, which occurs during days 5–21, but can last for up to 15 weeks, depending upon the severity of the injury or the body's ability or opportunity to heal. Formation of new collagen fibres commences by *fibroblast, tenoblasts* or *myofibroblasts* (depending upon the structure). These cells lay down new, small, weak fibres in a

disorganised fashion. The tensile strength of the muscle, tendon or ligament has the chance to increase during this time as long as appropriate stimulus is applied (Sharma & Maffulli 2005). In this stage of healing, treatment should be directed toward:

1. The aim of this phase is to *help the new fibres to align* in the strongest format possible and to reduce adhesion formation: organised and parallel to each other in the best direction to withstand force. Within the first 2 weeks following injury (severity dependent), it is best to adhere to relative rest incorporating pain-free activity and range of motion (ROM) (Wilson & Best 2005). In a canine model, passive stretching of repaired flexor digitorum profundus tendons was shown to be an effective therapy to lessen adhesions and improve tensile strength (Zhao *et al.* 2002). This can be done with progressive walking, balancing or weight-shifting exercises, passive or active motion of the joint/limb in pain-free ranges. At 2 weeks post injury, collagen synthesis is at its greatest (Sharma & Maffulli 2005). This is the most crucial time to start adding a small degree of stress to the injured tendon or ligament in order to promote collagen alignment. This may create a stronger wound and ultimately a faster return to function. Treatments in this stage can incorporate regaining lost ROM or flexibility by stretching into a small amount of discomfort (but not pain) and beginning strengthening (Sharma & Maffulli 2005; Wilson & Best 2005). Early strengthening for the dog could include three-leg or two-leg static standing balance and easy concentric muscle contractions, which might be achieved by flat land walking, pawing action or stepping over obstacles.
2. *Strengthening*: Once pain has subsided, advanced strengthening should be considered (Wilson & Best 2005). Eccentric strength training can be particularly effective in treating tendinopathies and may help promote the formation of new collagen or reverse degenerative changes if present (Khan *et al.* 1999; La Stayo *et al.* 2003; Wilson & Best 2005; Rees *et al.* 2006). Mechanical loading accelerates tenocyte metabolism and may speed repair (Khan *et al.* 1999). Eccentric strengthening in the dog may include walking backwards or sideways, use of hills or stairs, trotting or even small jumps depending upon the target muscle group or ligament. Exercising with neuromuscular electrical stimulation (NMES) may also aid in healing (Khan *et al.* 1999). For dogs, a functional application of NMES is very effective while encouraging weight shifting or static balancing on two legs (i.e. cross leg standing with NMES on the gluteal muscles or quadriceps complex).
3. *Enhancing circulation to the affected area*. Circulation allows for the delivery of oxygen and nutrients to the healing area and takes away the products of inflammation. Promotion of circulation can be accomplished several ways, including many therapeutic modalities as

described in Chapter 10; heat, cyclical hot/cold, laser, ultrasound, PEMF and NMES (Nelson & Currier 1987; Michlovitz 1990). Other circulatory enhancing methods include: prescription exercises, deeper transverse frictions and stimulating active muscle contractions (Cyriax 1982; Nelson & Currier 1987; Michlovitz 1990).

4. *Encourage tissue regeneration and metabolism*. Physiotherapy modalities such as laser, ultrasound and PEMF encourage collagen synthesis and metabolism of tenocytes and myocytes (Lee *et al.* 1997; Khan *et al.* 1999; Green *et al.* 2003; Sharma & Maffulli 2005). Use of ultrasound has been shown to speed up the production of more normal fibre matrix, and parallel bundle formation within the fibres, thereby accelerating the time to normal tendon tissue status in severed and repaired Achilles tendons in dogs (Saini *et al.* 2002).
5. *Restoring co-ordination and body awareness*. A widely researched phenomenon in humans following an injury or surgery is a diminished sense of proprioception (Lephart *et al.* 1997), leading to altered neuromotor control owing to a compromised feed-back mechanism. Therefore sensory facilitation techniques should be used to retrain proprioception and neuromotor control to avoid reinjury, secondary dysfunction and hasten return of an athletic animal to full performance. Retraining proprioception allows for a much more responsive musculoskeletal system which is needed for any high-end athletic venture (Lephart *et al.* 1997). This may include running off-leash in a park through to agility sports. Some techniques may include joint compressions, postural facilitation balancing activities, and coordination training that is not so challenging as to reinjure the area (i.e. walking over poles, walking on uneven surfaces, agility training, etc.).

Stage 3

This is the *remodelling phase*, which commences at approximately 6 weeks and can take up to a year. It is further divided into a *consolidation stage* and a *maturation stage* (Magee 1986). The *consolidation stage* may take from 6 to 10 weeks and is marked by the change of the repair tissue from cellular to fibrous. Fibroblast (tenoblast or myofibroblast) activity remains high and the fibres become aligned in the direction of stress. At 10 weeks the *maturation stage* occurs and is characterised by a change of the fibrous tissue to scar-like tissue. Vascularity is decreased and metabolism of repair tissue gradually ceases (Sharma & Maffulli 2005). Treatment targets building up muscular strength, maintaining ligament or musculotendinous extensibility, enhancing joint mobility and improvement of the proprioceptive system and neuromotor control. Treatments can include prescribed exercises that allow for a gradual return to function. This may include leash walks followed by short allowances of off-leash running with a gradual increase of the off-leash time. Destination jumping (onto something like a bed or over a small jump), trotting up or down hills

and zigzag trotting in the other direction, and gaiting exercises (specific techniques to address any gait adaptations) are useful at this stage. Gradual return to sport could include modified activity with more rest periods, specific turning directions and lower jump heights. Joint ROM may be gained simply by continuing to range the joint, therapeutic joint mobilisations (Grades 3 or 4; see Chapter 9) (Maitland 1966; Gross-Saunders *et al.* 2005) and exercises that target full joint ROM. Stretching should continue during this stage of rehabilitation to avoid scar tissue contracture and to promote collagen orientation. Proprioceptive exercises can progress to include more difficult balancing or displacement exercises (i.e. use of a wobble board or mini-trampoline), walking on different surfaces or over sporadically spaced objects of varying heights, walking on a narrow plank of wood, backing up or walking sideways over poles and destination jumping.

13.2.5 Healing and treatment of chronic soft tissue injuries

Chronic ligamentous (or musculotendinous) injuries will have random collagen orientation, wound contracture, restrictive adhesions/scars and may be degenerative (Khan *et al.* 1999; Maganaris *et al.* 2004; Edge-Hughes 2005; Sharma & Maffulli 2005). Chronic lesions may either be a result of a lesion not being properly addressed in the acute stage, resulting in incomplete healing, or repetitive overuse or micro-traumas, resulting in a degenerative lesion. The injury site may have healed in a weak, disorganised fashion, therefore continuing to receive micro-strains whenever it is overly stressed. Since scar tissue tends to contract or retract, tissue mobility can be reduced, hence the importance of beginning a stretching regime and slowly working on strengthening with these patients. Clinically successful treatment outcomes have been achieved with chronic soft tissue injuries (suspected degenerative tendinopathy). This can be achieved by aiming to start or increase an inflammatory process using high doses of modalities (continuous ultrasound or laser), aggressive stretching regimes and controlled exercise. This, however, must not promote a marked increase in any lameness and should progressively increase in intensity within the dog's tolerance. Contraindications and precautions must be closely monitored, for example continuous ultrasound at high intensities is absorbed by the hair raising the superficial skin temperatures high enough to causes burns; however the effectiveness of pulsed ultrasound when dealing with an intact hair coat has not been studied (Steiss & Levine 2005). Clinically, it is advisable to utilise pulsed ultrasound if unable to clip, shave or successfully separate the animal's hair coat. The type of protocol above has been shown to have effectiveness in chronic cases (Edge-Hughes 2004; Mikail & Campos 2004; Canapp *et al.* 2005 (unpublished)). Prescription anti-inflammatories or cortisone injections are of little benefit since a chronic injury is a non-inflammatory condition (Khan *et al.* 1999; Paavola *et al.* 2002).

13.2.6 Prevention of soft tissue injuries

Numerous cases of lameness may be undiagnosed in veterinary medicine and many are probably associated with muscle injury (Fitch *et al.* 1997).

Causes of soft tissue sprains and strains may be one or many of the following:

- Poor flexibility
- Inadequate warm up
- Fatigue
- Sudden forceful contraction
- Hyperextension or over stretching
- Strength imbalances
- Intense interval training and insufficient rests/breaks (Steiss 2003)

Stretching has been shown to be effective in increasing joint and muscle flexibility (Magnusson *et al.* 1998; Knudson 1999; Power *et al.* 2004; Thacker *et al.* 2004; Davis *et al.* 2005; Decoster *et al.* 2005). Regular stretching has been shown to improve eccentric and concentric force production, velocity of contractions, maximal volitional contractions, counter-movement jump height, 50-metre dash and athletic performance (Hunter & Marshall 2002; Shrier 2004). One study found that regular stretching can induce hypertrophy in immobilised muscles and another speculated that this effect might improve performance in the long term (Coutinho *et al.* 2004; Shrier 2004).

However, studies have shown that implementing stretching 'pre-event' or immediately before exercise or testing decreases:

- Isokinetic performance
- Velocity of contraction
- Muscle force produced with contractions
- Musculotendinous unit compliance
- Ability to store elastic energy in the eccentric phase (Behm *et al.* 2004; Fletcher & Jones 2004; Power *et al.* 2004; Shrier 2004; Thacker *et al.* 2004)

These negative effects have been reported to last for up to 1 hour after stretching (Thacker *et al.* 2004). An effective recommendation for regular stretching can be made for daily or every second day after a training session, but not before competition or a training bout. Stretches are effective for muscle elongation when utilised this often, holding 15–30 seconds and at only one repetition (Bandy & Irion 1994; Thacker *et al.* 2004; Davis *et al.* 2005; Decoster *et al.* 2005).

Warming-up the animal before racing or exercise is of great importance to achieve superior performance and prevent injuries (Strickler *et al.* 1990). A warm-up of 5 to 10 minutes is more beneficial for improving oxygen kinetics than a shorter warm-up period (de Vries 1986; Tyler *et al.* 1996). There are conflicting findings however, as to whether warming-up has any effect on performance in speed activities (de Vries 1986). Some literature also cites that endurance athletes perform better with 5 minutes of

vigorous high intensity warm-ups that include some sprinting (de Vries 1986). Essentially, heating of muscle tissues by 1–2°C can improve musculotendinous extensibility and may thereby reduce its susceptibility to strain injury (Steiss 2002). The cool down should be at least 15–20 minutes of walking after an especially strenuous session (de Vries 1986; Skinner 1987).

Attention to skill training, training on different surfaces or in adverse conditions can aid in avoiding injuries on ‘race day’ (Evans 2000). Human studies have cited the need to enhance neuromuscular control and proprioception in athletes as prevention against soft tissue injuries or as part of an advanced rehabilitation programme (de Vries 1986; Lephart *et al.* 1997). Consistent and regular repetitive training utilising balance training and joint repositioning should be implemented in a progressive manner in respect to difficulty (Lephart *et al.* 1997).

The training targets:

- Joint mechanoreceptors
- Ruffini endings
- Pacinian corpuscles
- Unmyelinated free nerve endings
- Golgi tendon organs
- Muscle spindles in order to target the central nervous system (CNS)

The CNS receives input from these sensory receptors and responds accordingly via spinal cord reflexes, the brainstem and cognitive programming to promote dynamic joint stability and functional stability (Lephart *et al.* 1997). Human endurance athletes have utilised variable intensity training to enhance performance, and sprint training should incorporate mandatory rest periods to avoid muscular fatigue (Paavola *et al.* 2002; Esteve-Lanao *et al.* 2005). Recommendations for conditioning in canine athletics have been made but have yet to be studied (Blythe *et al.* 1994; Zink & Daniels 1996; Zink 1997).

13.2.7 Rehabilitation example for grade one cranial cruciate ligament injuries

Characteristics of a Grade One cranial cruciate ligament (CCL) injury:

- Mild swelling detectable at the parapatellar tendon
- Mild discomfort on stress testing (unanaesthetised)
- Partial weight-bearing use of limb

The following treatment protocol can be used in dogs with partial cruciate tears or when surgery is not an option or has been declined.

- Keep the dog on a leash for 2–3 months without exception.
- Modalities that may encourage circulation to the cruciate ligament (i.e. PEMF or laser) on a regular basis (see also Chapter 10).

- Joint proprioception techniques such as joint compressions and cross leg standing.
- Strengthening of the adjacent musculature: up-hill walking (steep going up and gradual coming down).
- Balancing/coordination: walking on different terrain (i.e. to cause high stepping or somewhere with uneven footing), rocker boards, mini-trampolines, obstacle course and cushions off the couch, etc.
- At 2–3 months (individually based): add some ‘destination jumping’ (i.e. onto a bed or couch or over a small jump) and/or tug-of-war exercises if the dog is safe doing so.
- Supplementation: Glucosamine HCl and methylsulphonyl methane (MSM) have been found to be useful for joint protection and soft tissue healing respectively (Holt 1998). A short-term (3 week) triple dose of glucosamine HCl/MSM may be helpful, then a double dose for the long term or indefinitely, under the veterinarian’s guidance.
- Owners must be educated to *never* throw ball or play ‘Frisbee’ again with their dog (unless doing baby tosses), they need to be educated on the mechanics of injury – how the cruciate was injured in the first place and what to avoid (i.e. hyperextension or ‘pivot-shift’).

13.3 Additional concepts regarding soft tissue injury

Helen Nicholson

13.3.1 Potential indications

Animals may present with similar conditions to those in humans including but not limited to (see also Chapter 7):

- Delayed onset muscle soreness
- Muscle strain injury
- Myofascial trigger points
- Ossifying or fibrotic myopathies, e.g. semitendinosus fibrotic myopathy, myositis ossificans
- Contractures, e.g. infrapatellar contracture, quadriceps contracture

Grade of soft tissue injury, stages of healing and management of soft tissue injuries have been covered earlier in this chapter. Special note will be made here regarding contractures, with a relevant case study included.

13.3.2 Ossifying or fibrotic myopathies and contractures

Conditions such as myositis ossificans and fibrotic myopathy of the semitendinosus and other muscles (such as gracilis, biceps femoris or semimembranosus) are uncommon in dogs (Steiss 2002; see also Chapter 7).

Muscle contractures may occur acutely after injury, more chronically after periods of recumbency or post operatively due to tethering to a fracture site (Chapter 7).

Case study: Cat quadriceps contracture

(Courtesy of: Lindsey Connell, MAnimSt (Animal Physiotherapy))

The cat had sustained an injury to her right hindlimb (RHL) causing a grade 4 medially luxating patella and subluxation of the calcaneoquartal joint (between the calcaneus and the 4th tarsal bone) resulting in non-weight bearing (NWB) lameness. Veterinary management of the injury included casting the limb for 3–4 weeks. Initially the cat was weight bearing (WB) on the cast limb. However, on its removal she was again NWB lame on her RHL. At 5 weeks post-injury the cat was referred to a specialist veterinary hospital. Surgical stabilisation of the stifle and hock joints was performed and included right medial patella luxation repair and calcaneoquartal arthrodesis using cancellous bone graft taken from the right proximal humerus and stabilised with a pin and tension wire. The limb was then placed in a Robert Jones bandage and splint post operatively. Post-operative instructions were to keep the cat confined in a small room for 4–6 weeks. Antibiotic and anti-inflammatory medications were initiated and follow-up radiographs planned for 4 weeks after surgery.

The Robert Jones bandage was removed at 2 weeks and at 3 weeks after surgery, the cat was still not using the leg at all. Although the arthrodesis was stable, it was evident that there were marked hip and stifle contractures. The case was referred to the physiotherapist and a plan made to assess and treat the cat under sedation or general anaesthetic (GA), as the veterinarian felt that the stability and position of patella and tarsus was sufficient for physiotherapy to commence.

Physiotherapy consultation under GA: The RHL passive extension ROM at the hip was 90° pre-treatment; stifle flexion ROM 75° and extension 145° (Figure 13.1 a and b). Manual physiotherapy techniques performed on the RHL hip and stifle included: passive accessory and physiological joint mobilisations including mobilisation with passive movement; soft tissue myofascial release techniques; passive joint and whole limb ROM exercises into flexion–extension; and hip rotation, flexion and extension sustained stretches (Figure 13.2 a, b and c). The hip ROM post-treatment was 110° with the stifle remaining unchanged. The owner was instructed on how to perform the passive stretches and ROM exercises (4 × a day). Weight-bearing exercises were given to the owner as able, i.e. three-point standing by lifting a forelimb to encourage right hind toe touch (4 × a day). These were demonstrated to the owner and the nursing staff by the physiotherapist, before and after the GA.

After 2 weeks of in-hospital physiotherapy treatments under GA three times per week, a review of the cat showed much improved hip ROM, however the stifle was still very flexed and extremely restricted especially involving semimembranosus, semitendinosus and biceps femoris. However, the cat was beginning to WB on the limb. The cat was discharged to continue with home exercise and physiotherapy.

After 3 weeks of physiotherapy, it was evident that the cat did not require GA for treatment, which continued with hip and stifle mobilisations and soft tissue manipulation including trigger-point therapy and myofascial release techniques to the aforementioned musculature.

Six weeks after surgery, radiography revealed a good tarsal fusion and the pin was removed. There were still moderate hip and tarsal contractures, although the cat was now WB with the musculature noticeably beginning to return. Physiotherapy was to continue along with the owner's home treatment programme.

Two months after surgery the cat had regained full hip and stifle extension with minimal pain, and she was increasing in strength and muscle bulk. The owner was instructed to continue with her 'home exercise programme' and allow the cat freedom to exercise outside the room, to progress to outdoor exercise after three more weeks.

Three months after surgery the cat was doing extremely well and returned to her normal functional activities both in and outdoors. There was no visible RHL lameness with full ROM at hip and stifle, but still some visible and palpable reduction of muscle mass, which with her normal activity was expected to return.



Figure 13.1 (a and b) A cat with contracture after right hindlimb immobilisation and surgery showing reduced hip and stifle range of motion under general anaesthetic.



Figure 13.2 (a, b and c) Same cat as Figure 13.1 showing: (a) myofascial release; (b) extension and (c) flexion whole-limb ROM/soft tissue release exercises performed under general anaesthesia.

13.3.3 Ice stretching

Although no evidence exists to date in the literature, ‘ice stretching’ can be effective clinically as a method of treating muscular hypertonicity as the cause of contracture. Ice stretching is a process whereby ice is rubbed briskly over the skin above the affected muscle to stimulate the cold receptors in the skin. The cold receptors activate an inhibitory interneurone in the spinal cord that prevents the stretch reflex from being activated, thus allowing a better quality of relaxed stretch of the affected muscle. Ice stretching provides an easy method of stretching deep muscles, as, provided the skin over the muscle is stimulated, even deep muscles will have their stretch reflex inhibited. It is vital, however, that the ice is continuously and briskly applied to the skin to maintain the activation of the cold receptors. For example, in a case where a dog had bilateral iliopsoas shortening, improvement was noted on only one side until the owner demonstrated exactly how she was performing the stretch at home – she was slightly less coordinated with the ice on the unimproved side and therefore stopped the

brisk movements of the ice as she initiated the stretch – this was apparently all the opportunity the dog needed to resist relaxation. Teaching the owner to perform the stretch with the ice in the other hand saw the unimproved side quickly catch up to the improved leg. Clinically, it is the author’s experience that most dogs tolerate 4×30 -second stretches very well, and that this yields satisfactory results. The use of vapocoolant sprays is typically very poorly tolerated by dogs owing to the noise of the aerosol. Additionally, plastic ice packs tend to drag on the skin during the brisk rubbing, causing discomfort. A preferred technique of administration is via direct skin contact of a large ice block (typically encased in a foam cup so the physiotherapist’s hands do not freeze), with use of towels to ensure water does not run down the dog causing discomfort (Figure 13.3).

13.4 Osteoarthritis Laurie Edge-Hughes

Osteoarthritis (OA) is characterised by progressive loss of articular cartilage and by reactive changes at the margins



Figure 13.3 Ice stretch on caudal hindlimb muscles.

of the joints and bones (Schumacher 1988). Clinical manifestations include aching discomfort that worsens with activity and is relieved by rest, a restriction of activity level, a limitation in the ability to perform, poorer proprioception, pain and discomfort, joint stiffness, effusion and enlargement, and loss of strength and flexibility (Schumacher 1988; Millis & Levine 1997; Buckwalter 2003; Shrier 2004; Snibbe & Gambardella 2005).

The causes of OA are age, genetics and conformation, as well as obesity, repetitive stress and joint trauma (such as subchondral bone damage, intra-articular derangement or surgical interventions) (Schumacher 1988; Johnson & Johnson 1993; Olmstead 1995; Lahm *et al.* 2004; Millis 2004). Physiotherapy can prove useful in both the treatment of OA as well as in attempting to prevent or delay the onset and progression of OA.

13.4.1 Assessment of osteoarthritis

Early detection and subsequent treatment of OA will yield the most favourable outcomes. Physiotherapists have long used manual testing techniques and clinical reasoning to diagnose early-onset joint OA (Magee 1986; Cibulka & Threlkeld 2004). If there is a joint capsule lesion or a total joint reaction present (OA lesions), a characteristic pattern of restriction in the passive ROM will occur (Magee 1986; Cibulka & Threlkeld 2004). This is called a capsular pattern, and in humans, the common capsular patterns for all joints are documented (Magee 1986; Gross-Saunders *et al.* 2005). The same is not true for animals; however speculation can be made from clinical practice as to common patterns of restriction, i.e. hip extension is commonly restricted in OA at the canine hip joint (Gross-Saunders *et al.* 2005). Pain and a loss of end-range extension is a frequent finding in early OA of many canine joints (Olmstead 1995). Additionally, physiotherapists utilise 'joint end-feel' to assist in determining the pathology at the joint (Magee 1986). This

practice can also be transferred to the canine patient by a practitioner skilled in manual therapy (see Chapter 9).

13.4.2 Treatment of osteoarthritis

The goal for the treatment of OA is to improve the joint and overall function and quality of life of the animal; relieve pain and associated muscle spasm, maintain and regain joint ROM, improve joint health, strengthen supporting muscles, address proprioceptive deficits and advise on lifestyle modifications.

Relief of pain may be accomplished by use of modalities (ultrasound, laser, PEMF, and NMES, see Chapter 10) (Nelson & Currier 1987; Michlovitz 1990; Millis & Levine 1997; Gur *et al.* 2003; Sutbeyaz *et al.* 2005). Massage has been shown to reduce pain, increase pain tolerance and stimulate a release of endorphins, so long as regular massage sessions are administered (Tappan 1988; Corbin 2005; Plews-Ogan *et al.* 2005). For this reason, owners could be instructed in how to perform massage techniques properly as a regular home-based intervention. Thermal agents such as heat or cold are both reported to have pain-relieving effects and application of each should be taught to owners and/or utilised as part of a therapy session (Michlovitz 1990; Millis & Levine 1997; Steiss & Levine 2005). Additionally, isokinetic muscle-strengthening exercises are also capable of significantly reducing pain (Huang *et al.* 2005). Many other forms of manual therapy techniques can also be utilised to reduce pain as described in Chapter 9.

The goals of improving joint ROM, joint health, muscular strength, functioning and proprioception can be accomplished by case-specific strengthening and exercise (Millis & Levine 1997; Brosseau *et al.* 2003; Buckwalter 2003; Roddy 2005; Roos & Dahlberg 2005; Stitik *et al.* 2005). Strengthening the supporting muscles aids in shock absorption, with the increase in strength minimising fatigue-related injuries in general conditions (Millis & Levine 1997; Buckwalter 2003). Both aerobic walking and quadriceps-strengthening exercises have been shown to reduce pain, increase joint ROM and improve function in human knee OA patients, thus reducing disability (Roddy *et al.* 2005; Roos & Dahlberg 2005; Snibbe & Gambardella 2005). Moderate exercise has also been shown to improve knee cartilage glycosaminoglycan content in humans at risk of developing osteoarthritis (Roos & Dahlberg 2005). Articular cartilage can be stressed and damaged with repetitive impact loads, blows to the joint or torsional loads (Buckwalter 2003). Thus, aquatic exercises have been praised for OA patients because of the buoyancy effect of the water which allows exercise without significant joint impact (Cochrane *et al.* 2005; Stitik *et al.* 2005). Cross training has been proposed as a method to reduce the repetition of the same patterns of joint loading and motion (Buckwalter 2003). This information can be translated to the care of dogs with OA. Regular exercise (walks or trotting)

on a softer surface (grassy areas) should be encouraged. The use of an underwater treadmill or swim therapy can be very beneficial and utilised as a cross-training tool (Millis & Levine 1997; Hamilton 2002; Huang *et al.* 2005). Walking on hilly terrain could also alter joint loading and build different muscle groups (Edge-Hughes 2002). Competitive athletics should be discouraged, however, as canine sports such as agility, fly ball and racing, etc. could impart excessive joint loading or torsional forces.

Manual physiotherapy techniques such as joint mobilisations, stretching and joint traction/distraction have been found to be effective in improving function, walking tolerance and quality of life in humans and ROM in dogs (Crook 2004; Hoeksma *et al.* 2004). Manual therapy as an adjunct to exercise therapy has been shown to have a greater effect than exercise therapy alone on OA of the human hip (Hoeksma *et al.* 2004; Deyle *et al.* 2005). A physiotherapist trained in manual therapy is the most qualified practitioner to apply mobilisation treatments, as a keen appreciation of arthrokinematics, end-feel and an ability to grade the mobilisation is required (see Chapter 9). Owners can be instructed in muscle stretching and joint traction if deemed safe to perform these manoeuvres.

Joint health, cartilage regeneration and slowing of the progression of OA lesions can be targeted with use of modalities and nutritional supplementation. PEMF has been shown to increase chondrocyte matrix synthesis and proliferation *in vitro* and *in vivo* and has been shown to preserve the morphology of articular cartilage and retard the development of OA lesions in the knee of aged osteoarthritic guinea pigs (Fioravanti *et al.* 2002; Fini *et al.* 2005; Sutbeyaz *et al.* 2005). Laser therapy (Chapter 10) is able to enhance the biosynthesis of arthritic cartilage, and results in the improvement of arthritic histopathological changes (Cho *et al.* 2004; Lin *et al.* 2005). Additionally, human studies have found a correlation with glucosamine use and a reduction in joint-space narrowing and erosive effects of OA over a period of 3 years (Verbruggen *et al.* 2002; Bruyere *et al.* 2003). Canine studies have found that the use of a glucosamine/chondroitin sulphate mixture can enhance synthesis and turnover of the matrix of proteoglycans and collagen and hence can have a protective effect against synovitis and associated bone remodelling (Canapp *et al.* 1999; Johnson *et al.* 2001). Cetylated fatty acids have been shown in both human and animal studies to modulate the immune response and inflammatory process of osteoarthritis and in turn improve ROM and overall function (Richardson *et al.* 1997; Curtis *et al.* 2002; Hunter & Marshall 2002; Kraemer *et al.* 2005). Advice regarding nutritional supplementation should be considered just as important as physical management of the disease.

Overall improvement in functioning and quality of life is the ultimate treatment goal for OA. Excessive body weight can impact the stresses on articular cartilage. In a human

study each pound of body weight lost resulted in a four-fold reduction in the load exerted on the knee per step during daily activities (Messier *et al.* 2005). A canine study found that dogs with hip OA that were fed 60% of their current calorie intake lost 11–18% of their body weight and experienced a significant decrease in hindlimb lameness (Impellizeri *et al.* 2000). Weight management should be deemed an integral part of rehabilitation of the osteoarthritic dog.

13.4.3 Prevention of osteoarthritis

Preventing OA should be an important goal for all ageing animals or animals that have suffered a joint trauma (including surgical interventions). One canine study found that lifelong exercise did not cause cartilage erosion, osteophytes or meniscal injuries (Newton *et al.* 1997). In fact, a separate study found that: dogs exercised 4 km/day \times 5 days/week \times 40 weeks had increased cartilage thickness, proteoglycan content and indentation stiffness; dogs exercised 20 km/day \times 5 days/week \times 15 weeks had a decrease in cartilage thickness and proteoglycan content, but no degeneration; and dogs that were exercised 40 km/day \times 1 year had a decrease in proteoglycan concentration and indentation stiffness and stimulated remodelling of subchondral bone but no degeneration (Buckwalter 2003). The authors commented that increasing exercise causes changes in cartilage composition and mechanical properties but does not accelerate joint degeneration. So, given that muscle strengthening aids in shock absorption capabilities for joints (Buckwalter 2003), exercising to strengthen muscles may help to prevent the development of OA (Berend *et al.* 2004). Lifelong weight management is also an excellent prevention aid. The results of a 14-years-long study suggest that a 25% restriction in food intake can increase median life span and delay the onset of signs of chronic disease in dogs (Kealy *et al.* 2002).

In summary, to prevent or delay the onset of OA, dogs should be kept lean, exercised regularly on surfaces which reduce joint impact and torsions, and owners advised on nutritional supplementation as the animal ages or should joint injuries/surgical procedures arise.

13.5 Post-operative rehabilitation

Laurie Edge-Hughes

Stringent protocols for post-operative rehabilitation may be unnecessary when the physiotherapist utilises clinical reasoning skills, creates problem lists, sets rehabilitation goals and is cognisant of healing times. Anterior cruciate ligament (ACL) surgeries are common in both humans and dogs. Post-operative rehabilitation has been studied more thoroughly for this condition than any other orthopaedic surgery and can be used as a model for post-operative rehabilitative care in general.

First, it is important to understand what happens to soft tissues and articular structures after joint irritation, surgical intervention and/or a period of immobilisation or disuse. The disuse effects to muscles include: a loss of lean tissue mass; atrophy of type I slow-twitch muscle fibres (generally the extensor muscle groups) and associated tendons; biochemical conversion of slow-twitch muscle fibres to fast twitch and changes in the sarcoplasmic reticulum within muscles which decreases strength (Uthhoff *et al.* 1985; Francis *et al.* 2002; Taylor & Adamson 2002). In human ACL-deficient knees, the sartorius, gracilis and vastus medialis muscles exhibit a small decrease in power, while there is a significant decrease in power of the semitendinosus, rectus femoris, tensor fascia lata, vastus lateralis and the lateral head of gastrocnemius (Williams *et al.* 2003). Muscle firing patterns were also altered and displayed a greater magnitude of co-contractions than in normal legs (Williams *et al.* 2003). Ligaments were affected by immobilisation in that it can lead to ligament atrophy, a decrease in maximum load to failure and decreased biochemical, structural and mechanical properties (Uthhoff *et al.* 1985; Taylor & Adamson 2002).

Cartilage can also be affected by immobilisation: degenerative changes occur via a gradual reduction in proteoglycan content and production, a thinning or loss of articular cartilage, a decrease in cartilage matrix and cellular components, and a decrease in synovial fluid thus reduced cartilage nutrition (Uthhoff *et al.* 1985; Taylor & Adamson 2002). Loss of subchondral bone, osteoporosis, osteopenia, osteophytes and periarticular fibrosis are problems with immobilisation which affect bone (Uthhoff *et al.* 1985; Francis *et al.* 2002; Taylor & Adamson 2002). Proprioception is compromised; the ability to receive input from muscles, tendons and joints, and process information in a meaningful way in the central nervous system and assist in the knowledge of where the limb is in space, thus ultimately reducing the regulation of reflexes and neuromotor control (Hewett *et al.* 2002). Human ACL reconstruction results in proprioception deficits in both the involved and non-involved limbs at 3, 6, 9 and 12 months after surgery (Hewett *et al.* 2002).

13.5.1 Treatment of post-operative joints

Rehabilitation can allow the salvaging and acceleration of recovery time of some patients and it can make either the best or worst surgical procedure better (Taylor & Adamson 2002). The goals for any post-operative case are to: reduce pain; promote healing; maintain muscle mass and promote muscular development and joint stability; maintain joint flexibility; retrain proprioception, balance and coordination; facilitate early return to function; prevent degenerative joint disease and rebuild cardiovascular endurance (Hewett *et al.* 2002; Francis *et al.* 2002; Taylor & Adamson 2002; Nwadike & Hesbach 2004). Physiotherapy is also important

for addressing complications that can ensue with any post-operative case, such as identification of infections or surgical failure, treatment of secondary tendinitis/tendinopathies, and/or soft tissue adhesions (Nwadike & Hesbach 2004).

The general goals for all post-operative rehabilitation will be very similar and can be categorised into stages of healing. A two-part division of physiotherapy goals has been documented for dogs (Clark & McLaughlin 2001).

1. *Phase one:* Immediately following surgery and in the initial inflammatory stage of tissue healing through to the end of the reparative stage, which could last approximately 3–4 weeks. Goals in this stage are to:
 - Resolve pain and inflammation
 - Stimulate early tissue healing
 - Preserve muscle mass
 - Joint ROM and articular homeostasis
 - Prevent mechanically dysfunctional compensatory postures and movement strategies by the patient
2. *Phase two:* Targets challenging the healing tissues during remodelling and maturation stages of healing to improve strength and mobility, mobilise scar tissue and enhance functional return (Clark & McLaughlin 2001; Taylor & Adamson 2002; Nwadike & Hesbach 2004).

From clinical observations it may be more appropriate to divide the post-operative rehabilitation into four stages:

- Acute phase
- Sub-acute phase
- Mid-stage
- End stage

Human post-operative protocols for athletes recovering from ACL reconstruction recognise six stages/phases to a post-operative strengthening programme (Hewett *et al.* 2002). A combination of goals, theories and suggestions is presented below (Delitto *et al.* 1988; Bocobo *et al.* 1991; Johnson & Johnson 1993; Johnson *et al.* 1997; Lephart *et al.* 1997; Zink 1997; Fitzgerald *et al.* 2000; Clark & McLaughlin 2001; Ross *et al.* 2001; Hewett *et al.* 2002; Marsolais *et al.* 2002; Fitzgerald *et al.* 2003; Berend *et al.* 2004; Kvist 2004; Davidson *et al.* 2005; Perry *et al.* 2005; Shaw *et al.* 2005; Edge-Hughes 2006).

Acute stage (weeks 1–3)

Lifestyle management: To reduce the risk of reinjury, it is imperative to educate the owners about: avoiding rough play; toileting on-leash only; short 5-minute leash walks building up to 10 minutes by the end of week three; no excessive stair negotiations; no jumping; and promotion of non-slip flooring (i.e. rubber-backed slip-rugs). In very early stages after surgery slings may be necessary to help the dog ambulate to perform toileting tasks.

Manage inflammation: Low doses of modalities (ultrasound, laser or PEMF) can be useful to reduce inflammation

after the first 24–48 hours after surgery. Cryotherapy can also be used on a regular basis for home management. Grade 1 Maitland's mobilisations for the joint/s (joint glides, joint distraction or joint compression) may also assist in settling the joint. NMES to the surrounding musculature may also have a positive effect on joint inflammation and adjacent soft tissue swelling.

Enhance or maintain ROM: Passive ROM, avoiding pain at end ranges, can help to maintain normal joint ROM, and may also improve circulation and, via stimulation of proprioceptive mechanoreceptors, may aid in sensory awareness. Active ROM may be accomplished through easy weight-shifting activities to regain some extension in the elbow, carpus, stifle or hock. Easy, active hip extension may be accomplished by 'tummy rubs' at this stage. Gentle active flexion could be accomplished by 'shake-a-paw' activity in the forelimbs or stimulating a 'scratching' reflex for the hind end or by gently pinching the toes in either the front or hind legs to stimulate a reflexive pulling away response. Additionally, active hindlimb flexion can be accomplished by proper 'sit' training; having the owners give a verbal command to sit, correct any improper postures such as side sitting (which avoids flexing the hindlimb) by tapping on the offending limb or reverbalingising the command until a proper 'sit' is given before giving a treat reward. Forelimb elbow and shoulder flexion may be accomplished similarly by asking for a 'down' (sternal recumbency position) and correcting any offending limb compensations before rewarding with a treat.

Proprioception: At this stage, ROM and grade 1 Maitland's mobilisations may stimulate Ruffini ending or Pacinian corpuscles within the joint capsule that can reawaken the mind's awareness of the body, which is necessary after any surgical intervention. Massage to the affected limb may stimulate Golgi tendon organs and muscle spindles. Weight-shifting activities and the allowance of partial weight bearing will not only aid in return to normal function, but is also a proprioceptive stimulus. The use of NMES with the weight-shifting activities may target both early strengthening as well as proprioception.

Address axial skeleton issues: Clinically, physiotherapists have reported finding spinal or sacroiliac joint dysfunctions subsequent to prolonged lameness issues (Kerfoot 1997; Edge-Hughes 2001). A study of a population of horses with orthopaedic problems found that 74% of horses with back problems had lameness and that back problems were diagnosed in 32% of the lame horses; these findings were significantly higher than those recorded in a control population (Landman *et al.* 2004). This would mean that the post-operative animal could be at risk of developing back pain secondary to postural alterations following surgery. Therefore, clinically it is recommend that the entire animal is evaluated, especially the spine and pelvis, on a regular basis throughout the rehabilitation process.

Subacute phase (4–6 weeks)

Strengthening: In this stage, the animal must start to build its muscular support surrounding the affected joint. Both closed-chain and open-chain exercises have been recommended and found to be of benefit when combined in a post-operative exercise programme. The therapist may prescribe slowly, beginning to challenge the weight-bearing status of the limb with activities such as static balancing by lifting one or two of the unaffected limbs off the ground (with or without the additional use of NMES), walking up or down gradually sloping hills, or stepping over poles or objects (closed-chain). Muscular endurance should also be targeted and increasing walking times/distances should begin, gradually increasing to 20 minutes by the end of this stage. Use of a hydrotreadmill or pool swimming may be effective for strengthening at this stage as well (open-chain).

Soft tissue stretching and ROM: Care should be taken to not only address ROM deficits of the affected joint, but to also manage ROM of uninvolved joints, as compensatory gait alteration may have affected them. Muscle stretching of two-joint muscles should also be undertaken, as they will not be stretched with simple ROM (e.g. with any elbow joint surgery, the long head of triceps should be stretched, which is accomplished by combined simultaneous shoulder extension and elbow flexion).

Proprioception: Proprioception can be challenged to a greater extent in this phase. Use of balance boards, slow walking on uneven or unstable surfaces, or walking over obstacles may target this goal. Near the end of this stage, the static balancing exercises described above can be progressed to include small manual balance perturbations while holding positions.

Mid Stage (7–9 weeks)

Strengthening and proprioception: Increasing time, distances, speed or terrain traversed during leashed walks will aid in strengthening. Steeper hill and/or stairs at walk may aid in advanced strengthening. Trotting exercises may be appropriate. Static balancing on two legs, such as making the animal balance on the surgical leg and its opposite thoracic limb by holding the other diagonal pair off the ground should be introduced. This may include manual displacement or while on a novel surface (i.e. foam or mini trampoline) to challenge both strength and proprioception. Stepping-over exercises in all planes of motion, including lateral, rotational, or backwards, assists in increasing the complexity of the neuromotor task. Walking on a plank of wood elevated a few inches above the ground will address coordination issues, and may be combined with volitional balance disturbances (e.g. such as making the dog turn to take treats from side to side).

Gait retraining: Often dogs will develop compensatory postural or movement strategies following a surgery. Many of the exercise and proprioceptive techniques described

above can address these issues, but other therapeutic interventions might include taping or bandaging techniques or the use of mild, noxious mechanoreceptive stimuli to promote proper limb usage.

End stage (10 weeks +)

Strengthening and proprioception: If healing has occurred appropriately and according to schedule, then advanced strengthening techniques can be encouraged, such as destination jumping (i.e. on to a bed or over a jump). Longer trotting sessions may be necessary to build up muscular and cardiovascular endurance. A gradual return to off-leash activities may begin after 12 weeks, depending on safety issues, e.g. time of the year, condition of the ground for non-slip footing, only when not near other dogs that might initiate rough play, etc. Owners need to be instructed to allow for a long warm-up (10 minutes or more) before allowing a short off-leash run (consisting of just 5 minutes off leash to start) or that the initiation of the off-leash time should occur in the middle of the dog's walk. Straight line running can be attempted (i.e. asking the animal to run back and forth between two people).

Return to sports (14 weeks +)

Advanced strengthening and proprioception: As mentioned above, if healing has not been compromised during the post-operative period and the owners are determined to return the animal to sporting activities, then advanced training and intensive rehabilitation must be accomplished. Plyometrics, jumping, agility training, sport-specific training, sprints, training using greater balance perturbations, quick directional changes/pivoting/figure-of-eight exercises at submaximal effort at first, then progressing to greater speeds, cardiovascular endurance and a continuation of strength training should be targeted.

Important to note for post-operative rehabilitation

Not all surgical interventions will progress at the same rate, although they will go through the same healing stages. It is important to make the post-operative rehabilitation specific to the extent of the surgery and the animal's rate of healing. The physiotherapist should regularly assess when it is appropriate to progress through the various stages/phases of rehabilitation or to determine if a referral back to the surgeon is required.

13.6 Fracture healing

Laurie Edge-Hughes

Successful healing of a fracture is not only determined by complete bony union on radiographs, but also by the functional use of the limb thereafter. Physiotherapy following veterinary management of a fracture can be beneficial to fracture healing and is just as important as the correc-

tive procedure itself in determining long-term outcome (Olmstead 1995; Nwadike & Hesback 2004). Comprehension of healing stages and times is required for the implementation of an appropriate rehabilitation plan.

13.6.1 Stages of fracture healing

- *Stage 1:* Haematoma formation
- *Stage 2:* Traumatic inflammation (vasodilation, local swelling and loosening of attachment of periosteum to bone)
- *Stage 3:* Demolition (macrophages remove red blood cells, debris and fibrin. Detached bone fragments necrose and are attacked by macrophages and osteoclasts)
- *Stage 4:* Granulation (tissue formation, capillary loops form)
- *Stage 5:* Formation of woven bone and cartilage (woven bone is made of collagen ground substance called osteoid a process which is known as callus formation). Cartilage eventually dies and calcifies
- *Stage 6:* Lamellar bone formation: woven bone or cartilage is invaded by capillaries, and osteoblasts create osteoid
- *Stage 7:* Remodelling: osteoclastic removal and osteoblastic formation occur simultaneously until bone is close to its pre-fracture state. Callus is slowly removed and becomes lamellar bone. The internal callus hollows and forms the marrow cavity (Magee 1986)

13.6.2 Expected bone healing times

Time to clinical union for a fracture may be dependent upon age along with fracture type, severity and site (Olmstead 1995; Nwadike & Hesback 2004). In canine patients younger than 3 months, bone healing takes roughly 2–4 weeks, for animals 3–6 months of age, healing is 4 weeks to 3 months. When animals are between 6 and 12 months, healing can take 5 weeks to 5 months and if the dog is more than 1 year of age, then one might expect 7 weeks or up to 1 year for healing. There are known factors in human medicine that can contribute to delayed healing times such as insufficient frame stability, smoking (in the case of the canine patient, perhaps owner smoking could retard healing), poor weight bearing (perhaps due to pain), poor vascularisation, diabetes and poor nutrition (Gebauer 2002; Gebauer & Correll 2005; Steim & Lerner 2005).

13.6.3 Physiotherapy management of fractures

Physiotherapy management of fractures is very similar to post-operative joint surgery rehabilitation. Treatments can begin when the area is accessible (i.e. when a bandage or cast is off) or immediately if an internal fixation has been used. See Section 13.5.1 'Treatment of post-operative joints' to determine goals for different stages of healing.

Additional physiotherapeutic interventions and modalities (see Chapter 10) have been recommended

specifically for fracture management and may warrant consideration.

Ultrasound (US)

Low intensity, pulsed US (0.03–0.05 W/cm² with either a 1.0 MHz or 1.5 MHz or 3.3 MHz sound head used for 10–20 min per session daily, beginning day one post-operative fracture repair) has been shown to stimulate endochondral ossification due to stimulation of bone-cell differentiation and calcified matrix production by intracellular calcium signalling and incorporation in chondrocytes (Gebauer 2002; Parvizi *et al.* 2002; Korstjens *et al.* 2004). US has been shown to be useful in increasing the stiffness of bone and speeding healing in diabetic rats (Gebauer 2002), and may be effective by stimulation of angiogenesis (Childs 2003; Steim & Lerner 2005). Additionally, US has been shown to be effective on delayed unions and non-unions in children and did not have any effect on the growth plates (Gebauer & Correll 2005).

Laser

The use of laser has been reported to increase bone stiffness by forming a smaller, stronger callus, which was more osseous in nature compared with controls in rats (Luger *et al.* 1998). This study used a 632.8 nm, 35 mW laser for 30 min daily, delivering 892 J/cm² superficially for 14 consecutive days.

Pulsed electromagnetic field

Pulsed electromagnetic field (PEMF) has been reported to stimulate osteoblasts and chondroblasts (Childs 2003), however in the literature there are contradictory findings over the effectiveness of PEMF in bone healing. For this reason, particular attention should be paid to the parameters utilised in the studies that found this technique to be efficacious. Utilising a dog model, one study found that by using a PEMF setting of 1.5 Hz for 1 h/day at 4 weeks post-operatively and lasting for 8 weeks, significantly increased stiffness and promoted greater new bone formation (Inoue *et al.* 2002). This study also found an increase in mineral apposition rate and reduced porosity in the cortex adjacent to an osteotomy site. Delayed unions and non-unions have been shown to improve with electrical stimulation and PEMF versus placebo and were comparable to bone grafting (Aaron *et al.* 2004). Preservation of bone mass was demonstrated in animals with osteotomy gaps and a significantly reduced size of gap was noted versus controls using 15 Hz for 3 h a day (Ibiwoye *et al.* 2004). The prevention of osteoporosis following ovariectomy was demonstrated with PEMF at 7.5 Hz, for 8 h/day, for 30 days versus controls. This same setting was utilised in an earlier study, which demonstrated its ability to heal fracture non-union (Yang *et al.* 1994). However, one study found that PEMF at 100 Hz for 4–8 h/day for 2–4 weeks was effective in pro-

moting bone formation around a rough-surfaced implant (Matsumoto *et al.* 2000). A study looking at the combined use of PEMF, ice and exercise following fractures found that the combination group had better pain reduction and joint ROM than an ice and exercise group or a PEMF and exercise group (Cheing *et al.* 2005). This study utilised 50 Hz for 30 min/day. Other studies have found no effect with PEMF on healing of fractures, using similar settings. Clearly, more research needs to be conducted on different types and settings of PEMF machines to improve their role in clinical decision making for practitioners.

Electrical stimulation

Some studies utilise implantable electrodes (Clark 1987), but as this is not generally considered standard physiotherapy practice, this section will address use of surface neuromuscular electrical stimulation (NMES). An NMES protocol used in rabbits (surface electrode placed 3 cm proximal to the fracture site and the other proximal to the first electrode, using 25 mA of current, at a pulse width of 50 μ s, at 4 Hz with an on cycle of 20 s, and an off cycle of 15 s and a ramp of 5 s, 1 h daily, beginning on day 4 after surgery for 25 days) demonstrated an increase in mineralised callus by 2 weeks, and a significant increase in callus mineral content and mineralised callus compared with controls by 8 weeks (Park & Silva 2004). Bone had greater torsional parameters, for stiffness, maximum torque withstood, and required a greater amount of energy to create failure (Park & Silva 2004). It is reported that the cathode should be placed closest to the site of the fracture and the anode near the first electrode for proper application (Childs 2003).

Weight bearing and early mobilisation

A study of the effects of weight bearing on healing of cortical defects in the canine tibia (Meadows *et al.* 1990) found significantly less woven bone formed in the defects in the non-weight-bearing tibia than in the weight-bearing tibia. This was determined to be due to the disuse response in the underloaded tibia, in which less bone formed, rather than to the formation of more bone in the weight-bearing tibia. In one review paper, it was found consistently that early mobilisation was preferable over rest in human patients and the authors recommended that mobilisation may be able to be employed more often and perhaps more vigorously (Nash *et al.* 2004). They did, however, caution that it would also be naïve to assume that mobilisation is better than immobilisation in all circumstances. Other studies on humans and animals have had similar findings and have reported that early mobilisation has the potential to result in earlier recovery of mobility and strength, facilitation of an earlier return to activities and did not affect fracture alignment (Meadows *et al.* 1990; Kamel *et al.* 2003; Feehan & Bassett 2004; Nash *et al.* 2004; Cheing *et al.* 2005; Davidson *et al.* 2005).

Case study: Post-operative rehabilitation after internal femoral fracture stabilisation

Courtesy of Lindsey Connell, MAnimSt (Animal Physiotherapy)

A 6-month-old female Belgian Shepherd was diagnosed with a fractured femur by her veterinarian, who referred her to a specialist veterinary hospital for surgery. The surgeon found a Salter–Harris Type II fracture of her medial distal femoral condyle (Figure 13.4). The fracture was stabilised via internal fixation by reconstructing the medial ridge of the femoral condyle with lag screws via a medial approach to the distal femur. Following this a 90/90 bandage was applied and the dog medicated with nonsteroidal anti-inflammatory medication. The veterinarian's instructions for post-operative care were to keep the dog confined in a cage, with toileting privileges only, and for physiotherapy review and removal of the 90/90 bandage 7 days post operatively. Follow-up radiographs were scheduled for 4–5 weeks post operatively.



Figure 13.4 A 6-month-old Belgian Shepherd, Salter–Harris Type II fracture of her medial distal femoral condyle.



Figure 13.5 A 6-month-old Belgian Shepherd post surgery; surgically, the fracture was internally fixed by reconstructing the medial ridge of the femoral condyle with three lag screws via a medial approach to the distal femur.

Initial physiotherapy consultation (7 days post operatively)

After removal of the 90/90 bandage, the dog was non-weight bearing (NWB) on right hindlimb (RHL) (Figure 13.5). Examination findings: right stifle ROM was measured using goniometry and found to be reduced (flexion 80%, extension 50% normal), as was the right hip (flexion 90%, extension 50% normal) and right tarsus (flexion 70%, extension 80% normal).

Treatment: soft tissue manipulative techniques were implemented to treat the loss of soft tissue extensibility and joint ROM treatment, for the hip flexors (iliopsoas complex and cranial sartorius) and quadriceps complex, along with stifle flexors and the medial thigh musculature. The stifle and hip were gently mobilised through the available ROM into extension passively.

The owner was shown passive ROM exercises to all joints of the RHL and given advice about confining the dog as well as a slow leash-walking programme for toileting 3–4 times a day to encourage toe-touch weight bearing. Anti-inflammatory medication prescribed by the veterinary surgeon was continued.

Physiotherapy consultation (2 weeks post operatively)

The owner was concerned regarding the dog's lack of progress and that she was having difficulty doing the home physiotherapy programme. Dog was NWB on her RHL with it held up, into flexion. Right stifle, hip and tarsal ROM were unchanged. Dog was very 'tense' and with marked tissue irritability throughout iliopsoas, quadriceps, sartorius and hamstring musculature.

After discussing the case with the consulting veterinarian and owner, it was decided to treat the dog three times per week as a minimum owing to the fear of a flexion contracture developing.

Treatment sessions included soft tissue manual therapy of the hip including myofascial release techniques and trigger-point therapy combined with gentle, slow, sustained passive stretches through the entire available length of the associated tissues, i.e. iliopsoas complex, sartorius and quadriceps into hip and stifle extension and stifle flexion. The coxofemoral joint was mobilised via passive accessory joint manual therapy techniques, i.e. cranial glides to the femoral head and passive physiological motions to encourage extension through the entire available ROM, combined with passive stretches. The tibiofemoral joint was treated by soft tissue techniques as described above to semimembranosus, semitendinosus, biceps femoris and gracilis, followed by gentle tibiofemoral passive physiological joint extension mobilisations via caudal glide to proximal tibia on femur. The tarsal joints were also mobilised into extension via passive physiological extension mobilisations and soft tissue structures

passively stretched into extension. Repeated functional ROM exercises into flexion and extension and sustained passive stretches for the whole limb were performed gently.

Hydrotherapy/physiotherapy consultation (2¹/₂ weeks post operatively)

The dog was still NWB RHL. Assessment and treatment consisted of swimming with buoyancy vest with assistance by the physiotherapist, as she required facilitation of right hindlimb to obtain any active motion of this limb while swimming (Chapter 11). The dog completed 24 m swimming (two lengths), with rest periods to minimise post-treatment soreness.

Physiotherapy consultation (3 weeks post operatively)

Weight bearing (WB) starting with toe touching evident in RHL. ROM improving: hip and stifle extension now 70% with treatment continuing. Veterinary and physiotherapy plan: the dog is to continue to be caged and only walk on lead 3–4 times a day for 5 minutes and to continue physiotherapy three times a week and hydrotherapy twice a week. Treatment progression guided by the clinical findings at each session.

Physiotherapy consultation (4 weeks post operatively)

Consistent partial WB on RHL, although limb was still held in excessive flexion and lameness graded 3.5/5. ROM of hip extension now 90% with a tight 'restrictive capsular end-feel' however full flexion, abduction and rotation were now possible. The stifle could be fully flexed, however extension remained at 70% with some crepitus noted during and into end of ROM. The tarsus was now full ROM. Thigh circumference reduced (29 cm affected vs. 34 cm unaffected limb).

Due to the nature of the fracture and surgery to the tibiofemoral joint the veterinarian needed to re-examine the dog before progressing with the intensity of the treatment of the stifle to regain extension of the joint.

Follow-up radiographs (5 weeks post operatively)

Follow-up radiographs demonstrated good healing of fracture. Assessment and treatment by the physiotherapist was then performed under anaesthetic to gauge the joint integrity without muscle tone altering available joint ROM. Techniques used: passive accessory physiological joint manual therapy techniques to the hip, stifle and tarsus; sustained stretching into end-of-range hip and stifle extension. During treatment, full hip ROM was attained and 70% stifle extension, N.B. the stifle was not 'over-pressed' due to the fracture. Plan to continue with current physiotherapy and hydrotherapy treatment.

Physiotherapy consultation (8 weeks post operatively)

Reduced lameness (2/5 at walk). Marked improvement in RHL thigh circumference 32 cm (left 34 cm) and ROM improved with full hip extension and 85% stifle extension. As the dog was much more functionally active, the treatment frequency was reduced to twice a week, while continuing to attend hydrotherapy twice a week; consistently using RHL in the water.

Physiotherapy consultation (3 months post operatively)

A structured walking programme was commenced and graduated to including exercise from a bike at trot. Physiotherapy treatment was reduced to one session per week, and hydrotherapy kept at two sessions per week.

Physiotherapy consultation (4 months post operatively)

Dog was no longer lame at walk and minimally intermittently lame at trot 0.5–1/5. Thigh circumference was now 32.5 cm and stifle extension 90% with no pain on end-of-range joint overpressure, with minimal irritability and 'tightness' in the stifle flexors and gracilis. Exercise programme included leash walking and trotting for 30 minutes twice a day and hydrotherapy (swimming twice weekly). Dog was now allowed to be off lead and live with other dogs normally again and has returned to a successful showing career.

13.7 Hip dysplasia

Laurie Edge-Hughes

Even though affected puppies are born with normal hips, by 2 weeks changes have already occurred that predispose to excessive joint laxity and alterations in the shape of the

femoral and pelvic components of the joint (Read 2000). The joint incongruity leads to osteoarthritis and varying degrees of dysfunction and pain (Read 2000).

Environmental factors, including diet, have been shown to have significant effects on the incidence and severity of degenerative joint disease (DJD) in dogs with canine hip

dysplasia (CHD) (Smith *et al.* 2001). There is a natural tendency for many immature dysplastic dogs to overcome acute hip pain as they mature (Black 2000). This may be due to fibrosis of the joint capsule and acetabular remodelling which increases stability and healing of microfractures (Black 2000).

There are several surgical options to treat the condition (total hip replacement, triple pelvic osteotomy, femoral head and neck ostectomy, or capsular denervation to name a few) (Olmstead 1995; Moses 2000; Read 2000). Post-operative physiotherapy goals and treatments can be addressed according to stages of healing (see section 13.5 Post-operative rehabilitation).

Conservative measures may provide a specific role for physiotherapists. A long-term study followed 68 dogs diagnosed with CHD which were managed conservatively for 10 years (Barr *et al.* 1987), with 76% of the animals evaluated at the end of this time. Of these 63% had no discomfort with forced hip extension, 79% had normal ROM, and 72% had normal exercise tolerance. Another paper discussed rationale for conservative management that included controlled exercise, prevention of obesity and use of heat (Johnston 1992). Physiotherapy management of the non-surgical CHD patient aims to create the best possible musculoskeletal environment for pain-free hip functioning and to slow down the process of DJD.

Treatments should include controlled gross motor strengthening (general exercise and conditioning) and fine neuromotor control (balance and proprioception activities). ROM and stretching should focus on hip extension and rotation. Joint nutrition may be addressed by: exercise, joint compressions, ROM and nutritional advisement (Chapter 3). When pain is symptomatic, pain management can be assisted by manual therapy techniques and electrotherapy (Chapters 9, 10). Additional factors to consider are, secondary and compensatory movement patterns and neuromuscular dysfunctions that may ensue, such as spinal management to address facet joint dysfunctions or DJD elsewhere.

13.8 Conditioning canine athletes

Laurie Edge-Hughes

Research on training and conditioning for the canine athlete is not readily available, however there is much to be gained and extrapolated from horse and human research and literature. Fitness for sport is of utmost importance. Fitness should encompass cardiorespiratory function, muscle strength, endurance, flexibility and coordination (de Vries 1986; Zink & Daniels 1996; Zink 1997; Marcellin-Little *et al.* 2005).

A full conditioning programme should not be implemented until the dog is skeletally mature and physeal closure has occurred (Zink & Daniels 1996; Zink 1997; Marcellin-Little *et al.* 2005). Generally, the epiphyseal plates close at 10 months in large dogs, a few months earlier in small dogs

and a few months later in giant breed dogs (Marcellin-Little *et al.* 2005). Before engaging in a training regime, animals should be examined for medical and musculoskeletal health by a veterinarian. A physiotherapist trained in animal physiotherapy can assess musculoskeletal function.

The physical requirements and physiological adaptations required to perform a sprint race (Greyhound), endurance race (Sled Dog) or sporting event (agility dog) are very different. The Greyhound is required to sprint at speeds up to 45 mph (72 km/h) (Geffen 2003). The distance can vary from 5/16, 3/8, 7/16, or 9/16 of a mile (500 metres, 600 metres, 700 metres, or 900 metres) (The Greyhound Racing Association of America website: <http://www.gra-amerifca.org/>). In the Iditarod, the sled dog must race for distances up to 1150 miles (1840 km) (Ultimate Iditarod website: <http://www.ultimateiditarod.com/>), and often will run continuously for 10 to 12 hours at speeds of 10 to 12 mph (16–19.2 km/h) between rests (Kathy Topham 2005, personal communication). Shorter races usually see mushers running their dogs for only 6 or 7 hours continuously (Kathy Topham 2005, personal communication). Agility competitions require competitors to sprint in one or several trials daily, going over, under, through or around all kinds of equipment (Zink & Daniels 1996). The agility athlete is like a football, basketball or rugby player; needing to possess coordination, strength, speed and the ability to recuperate quickly between runs.

Training programmes aim principally to improve cardiorespiratory and musculoskeletal responses, including strengthening (see also Chapter 5). Bone density and the thickness of the calcified cartilage layer of articular cartilage can both be increased to strengthen and improve overall load-bearing capabilities (Marlin 2003). Proprioception and exceptional body awareness is important for all animals, but is of critical importance for agility dogs.

A training programme that can promote dynamic joint and functional stability is desirable and should incorporate the following three levels of motor control:

- Spinal reflexes
- Cognitive programming
- Brainstem activity (Lephart *et al.* 1997)

When a joint is placed under mechanical loading, reflex muscular stabilisation is stimulated through the spinal reflexes (Lephart *et al.* 1997). Voluntary movements that are repeated and stored as central commands are considered cognitive programming (utilising the highest level of CNS functioning) (Lephart *et al.* 1997). Brainstem reflexes include righting reactions and displacement reactions (Umphred & McCormack 1990).

13.8.1 Injury prevention

Stretching

In sprinting, muscle and fascial flexibility should be adequate to allow full ROM required for the activity

(de Vries 1986); but not be overly flexible, which could impede the immediate transference of musculotendinous forces to the bones and potentially reduce the speed of movement (Witvrouw *et al.* 2004). Stretching related to performance and injury prevention is very sports specific and a contentious issue in the literature. Some studies have shown there to be a negative impact on immediate athletic performance following stretching, thus stretching needs to be appropriately applied in a sports-specific manner during or after competition or training sessions (Behm *et al.* 2004; Power *et al.* 2004; Shrier 2004; Thacker *et al.* 2004) (see Section 13.2.6 Prevention of soft tissue injuries).

The racing Greyhound requires flexibility in the multiple-joint muscles that act in propulsion of both the fore and hind legs, such as gracilis, biceps femoris, semitendinosus, and semimembranosus, gastrocnemius muscle and the calcaneal tendon, as well as the long head of triceps, and latissimus dorsi. The goal for implementing a stretching programme for the racing Greyhound is to ensure adequate muscle length in order to achieve the exaggerated ROMs and eccentric muscle contractions in those ranges. A stretching programme should be implemented only after training sessions for these athletes as a warm-up period is essential, and should include all the muscles/muscle groups (bilaterally) mentioned above, consisting of one 30-second stretch every second day excluding race days.

Stretching to gain flexibility may not be essential for injury prevention in the Sled Dog as the gait and speed at which an endurance race is run only utilises the mid-ranges of the extremity joints (Witvrouw *et al.* 2004). The Sled Dog may be required to traverse (at a trot) over land or snow for several hours. End-of-range flexibility is not a requirement or an asset for this athlete and there is little need in this sport to utilise the energy-absorbing capacity of the muscle-tendon unit. No studies found reported any performance enhancement in endurance athletes with stretching. The current literature does not support a static end-of-range stretching regime in endurance animals as part of a regular or pre- or post-event activity.

The agility dog utilises its body in a very ballistic manner with rapid 'bursts' of power: jumping, changing directions and performing agile feats. This dog relies on kinetic/potential energy from elastic recoil of its musculotendinous apparatus as well as muscle power in jumping performance. A regular stretching programme may assist with this athlete, targeting the biceps brachii, long head of triceps, and the carpal and digit flexors of the forelimbs (bilaterally) as well as the gluteals, hamstrings, quadriceps, and the calcaneal tendon of the hindlimbs (bilaterally). One 30-second stretch every second day to all the muscles/muscle groups mentioned should be utilised for these athletes.

Avoiding overtraining

Overtraining and overreaching should be avoided (Chapter 5) (Feehan & Bassett 2004). During any training pro-

gramme, an animal should be allowed adequate rest and recovery periods between exercise bouts to avoid overtraining and overreaching. Monitoring appetite, body weight, recovery of body weight after racing, behavioural changes, reluctance to exercise, muscle soreness, reduction in performance and slow recovery following exercise may be advised (Evans 2000).

Warming-up and cool down

It has been stated that warming-up before competition is important for warming muscular tissues, enhancing muscle extensibility and improving oxygen kinetics (de Vries 1986; Tyler *et al.* 1996; Steiss 2002). The cool down should be at least 20 minutes of walking after an especially strenuous session (Evans 2000) (see Section 13.2.6 Prevention of soft tissue injuries).

Skill training

Attention to skill training, training on different surfaces or in adverse conditions can aid in avoiding injuries on 'race day' or 'trial weekend' (Evans 2000). Appropriate racing paces for Sled Dogs should be trialed to resist fatigue in both normal conditions and adverse weather (i.e. excessive heat) (Evans 2000; Ultimate Iditarod website: www.ultijmateiditarod.com). Racing Greyhounds should practise on different tracks, course styles or course lengths. Sled Dogs should train with varying sled weights, and over variable terrain in different kinds of weather. Agility dogs should train on different surfaces, with different pieces of equipment arranged in different orders.

Health checks

All dogs should be regularly evaluated for signs of overtraining during a conditioning or training programme. The owner/trainer should be instructed to monitor for signs of overtraining, and advised that monthly or bimonthly veterinary and physiotherapy evaluation should be conducted to monitor the medical and musculoskeletal well-being of the dog in training, so that treatment can be provided as needed.

13.8.2 Treatment of athletic injuries

The main difference in treating injuries in athletic dogs is the owner's expectations and desire to return to sport. Trauma to tissues or joints may result in a partial deafferentation and can lead to proprioceptive deficits (Lephart *et al.* 1997). Regaining neuromuscular control after injury or surgery is a necessary prerequisite for athletes wishing to return to competition (Lephart *et al.* 1997). Additionally, because these animals are more highly conditioned before injury, they may be able to tolerate a faster progression through the rehabilitation phases and most certainly will require advanced proprioception training and eccentric muscle strengthening exercises to make a safe return to sport (Lephart *et al.* 1997; La Stayo *et al.* 2003).

End-stage athletic rehabilitation

An end-stage or advanced level of rehabilitation may include the following (Zink & Daniels 1996; Zink 1997; Edge-Hughes 2002; Steiss 2003; Edge-Hughes 2004):

1. Exercises up and down hills
 - Varying hill steepness
 - May incorporate retrieving
 - Using different gaits and speeds
2. Trotting exercises
 - In straight lines
 - In serpentine, circular or figure-of-eight directions
 - On different surfaces
3. Acceleration/deceleration activities
 - Retrieving
 - Short burst sprints (such as practising racing starts or recall exercises with the owner a distance away from the dog)
 - Jumping (gradually increasing the jump height or breadth)
4. Cutting or rapid turning exercises
 - Playing games of chase or keep away
 - Running a mock agility course (starting with easy turns and low jumps and progressing to more difficult turns and manoeuvres)
5. Jumping exercises
 - In different directions (circles, serpentine, figure-of-eight)
 - Over varying heights
 - Over jumps raised at odd angles
6. Concentric strengthening
 - Pulling exercises (attaching a weight to a pulling harness)
 - Mushing or skijoring
 - Swimming
 - Treadmills (land or water)
7. Endurance
 - Gradual increases in duration of exercise
 - Gradual increases in distances travelled
 - Gradual increases in intensity of exercise
8. Static balancing
 - Using wobble boards or mini-trampolines
 - Standing on narrow planks of wood or small stools
 - Standing on a physio ball
9. Dynamic balancing
 - Walking on different surfaces (on foam, in sand, in tall grass, on uneven ground, in shallow water or in the woods)
 - Walking across narrow planks of wood
 - Walking across a seesaw
 - Weave poles
 - Jumping and recall exercises
 - A-frame training
 - Walking through ladders
 - Walking through an obstacle course with poles spaced unevenly apart (forwards, backwards and sideways)

13.8.3 Summary

All rehabilitation, regardless of the species of patient, should be undertaken from a critical analysis perspective. A detailed physical diagnosis or at minimum a working diagnosis, presumption or problem list is required before proceeding with treatment. Communication between veterinarians and physiotherapists is essential to accomplish this goal. Treatment selection should be approached with a clear understanding of the mechanism of injury or surgical technique, healing times and physiological effects of each treatment modality or therapy chosen. Physiotherapy clearly has an expanding role in the health management of the dog.

13.9 Respiratory physiotherapy

Helen Nicholson

13.9.1 Introduction

Despite respiratory physiotherapy being routine for a number of human conditions, it is not yet well established in veterinary medicine. Where respiratory 'physiotherapy' has been used in veterinary medicine, it tends to be limited to 'coupage' (percussion) despite the spectrum of techniques that physiotherapy encompasses. This limitation is interesting as many of the studies upon which the human evidence-base is built were performed on animals, including a number of them on dogs (Gee & Williams 1979; Zidulka *et al.* 1989; Tomkiewicz *et al.* 1995).

13.9.2 Potential indications

It is vital to remember that pathological conditions of the cardiorespiratory system in dogs are often quite different from those in humans. When accepting veterinary referrals, it is important that the physiotherapist understands the underlying pathophysiological basis of the condition, rather than just the clinical signs or problems. For example, when a dog is referred for coughing due to pulmonary oedema as a result of 'heart disease', the physiotherapist should consider that the disease may be a result of left atrioventricular valve dysfunction rather than myocardial disease (Ettinger & Feldman 2002).

Common indications for respiratory physiotherapy in small animals include:

- *Pulmonary oedema*, e.g. from cardiac disease (left-sided heart failure) or tick (*Ixodes holocyclus*) poisoning
- *Upper respiratory tract obstruction*, e.g. during anaesthetic recovery in brachycephalic dogs
- *Aspiration pneumonia*, e.g. tick poisoning, megaesophagus, myasthenia gravis, after snake bite or other poisoning, laryngeal paralysis
- *Pneumonia*, e.g. bacterial, viral (interstitial), fungal

Pulmonary oedema

The treatment of pulmonary oedema has both an acute and post-acute phase in humans. The acute treatment focuses

on the provision of non-invasive positive-pressure ventilation (Pare *et al.* 1983; Rasanen *et al.* 1985a, b; Bersten *et al.* 1991; Mondejar *et al.* 1996; Mehta *et al.* 1997; Nava *et al.* 2003; Crane *et al.* 2004; Lin *et al.* 2005) and as recovery progresses, treatment focus changes to post-acute cardiac rehabilitation programmes (Isles *et al.* 2002).

Non-invasive positive-pressure ventilation

Non-invasive positive-pressure ventilation is mechanical ventilation that does not involve intubating the trachea and is a common treatment of humans with pulmonary oedema, hypoxia and/or hypercapnia. Several varieties of non-invasive positive-pressure ventilation exist including: positive end expiratory pressure (PEEP); non-invasive intermittent positive-pressure ventilation (NIPPV); positive expiratory pressure masks (PEP masks); nasal mask ventilation; continuous positive airway pressure (CPAP); bi-level positive airway pressure (BiPAP) and nasal inspiratory pressure support ventilation (NIPS).

Canine and other animal models of pulmonary oedema demonstrate that rather than forcing fluid back into the circulation, PEEP improves lymphatic drainage (Deshpande *et al.* 1996) and increases lung volume (Gee & Williams 1979; Lumb 2000). Reduction in hypoxia may be seen from PEEP as previously unventilated alveoli are re-expanded (Deshpande *et al.* 1996; Lumb 2000). However, porcine models have suggested that PEEP is more effective at reducing extravascular lung water when applied immediately pulmonary oedema occurs (Ruiz-Bailen *et al.* 1999).

A number of well-designed human studies have demonstrated the ability of NIPPV, nasal mask ventilation (Benhamou *et al.* 1992), CPAP (Bersten *et al.* 1991) and PEP masks (Bellone *et al.* 2002) to reduce the intubation rates, length of time to weaning from mechanical ventilation, complication frequencies and in-hospital mortality rates of humans with hypercapnic respiratory failure. Further, PaO₂ has been shown to improve within the first hour of non-invasive ventilation via nasal mask ventilation (Benhamou *et al.* 1992) or CPAP (Bersten *et al.* 1991), where oxygenation alone is used as a control; and non-invasive ventilation has also been shown to reduce respiratory muscle fatigue (Fauroux *et al.* 1999). CPAP has been shown to be more effective than PEEP in reinflating alveoli through collateral ventilation (Andersen *et al.* 1979). Although BiPAP is commonly used in humans, it has been associated with more myocardial infarcts than CPAP (Mehta *et al.* 1997).

CPAP has been used in dogs with respiratory signs including pulmonary oedema, hypoxia and hypercapnia. In practice, the biggest challenge when using non-invasive ventilation in dogs is to achieve an airtight seal without causing the animal undue stress. Although commonly available anaesthetic masks achieve a sufficient seal, they blow the positive pressure directly into the dog's face and constrict the nasal region, which may not always be well-tolerated. Transparent hoods have been trialled, which blow the positive pressure over the top of the dog's head from

behind. These appear to be well tolerated, however it is harder to achieve an airtight seal even with short coats, as the pressure tends to escape down the chest and over the thoracic girdle. The effective delivery of CPAP to non-intubated dogs is therefore still in the trial phase in Australia.

Neurophysiological facilitation

Pulmonary oedema patients are often hypoxic with an elevated respiratory rate. Neurophysiological facilitation, or 'breathing assist techniques', is an area of respiratory physiotherapy developed to improve oxygen saturation by reducing respiratory rate and encouraging more effective inspiration and lung inflation. Despite its widespread use in human intensive care units, few studies have objectively tested the effect of neurophysiological facilitation techniques. Two techniques, *perioral stimulation* and *intercostal stretching*, were found to result in significant improvements in oxygen saturation (Chang *et al.* 2002) (Figures 13.6 & 13.7).

Clinically, intercostal stretching in particular has an effect on slowing the respiratory rate of dyspnoeic dogs (although difficult to perform on small dogs with respiratory rates in the vicinity of 60 breaths per minute). Perioral stimulation can be very difficult to perform on anxious, distractible dogs, as the head needs to be held reasonably still to maintain the moderate pressure required to perform the technique. The research protocol described by Chang *et al.* (2002) used perioral stimulation for 10 seconds followed by bilateral intercostal stretch for 20 seconds, repeated in a cycle for three minutes. The intercostal stretch was performed on the anterior (equivocal to ventral in the dog) aspects of ribs 2 and 3 in the human patients studied, however due to the positioning and size of canine patients, it is often more practical to perform intercostal stretching on the dorsolateral aspects of the middle to lower ribs (Figure 13.7).



Figure 13.6 Perioral stimulation being demonstrated in a dog; the head needs to be held reasonably still to maintain the moderate pressure required to perform the technique.



Figure 13.7 Intercostal stretching. The pressure directed from, for example, rib 7 towards rib 8, as opposed to directing pressure internally. The stretch should be applied during expiration; this may need to be done every second breath with dogs with particularly high respiratory rates. In theory, this technique works by inducing a stretch reflex in the intercostal muscles, causing them to contract more strongly during the subsequent inspiration, improving the 'bucket-handle' action of the lower ribs, therefore increasing the basal expansion of the lungs and the depth of inspiration, thus lowering respiratory rate and improving oxygenation.

From Chang *et al.* (2002) it can be anticipated that the application of the therapist's other hand on the back of the dog's head during perioral stimulation, to provide stability and counterpressure, will have no effect on the neurophysiological facilitation. Chang *et al.* (2002) included analysis of sensory stimulation (defined as stroking the upper and lower limbs for 90 s at 1 Hz) and found no negative effects on respiratory function.

Pneumonia and aspiration pneumonia

Pneumonias are considered conditions for expectoration, however, physiotherapy is delayed until the consolidative phase of viral or bacterial pneumonia begins to resolve, but is aggressively applied from the outset in aspiration pneumonia (Hough 2001).

Treatments for expectoration

The 'active cycle of breathing technique' is commonly used in human physiotherapy for expectoration but is too cognitive in nature to facilitate its ease of use with dogs. However, the literature does describe other less-cognitive aspects of respiratory physiotherapy that have a suitable evidence-base as being better than no or control intervention in humans. Such treatments include percussion, vibration and postural drainage.

Postural drainage positions for dogs have been well described by Manning *et al.* (1997) (Table 13.1). Clinically, the importance of postural drainage – and therefore the need for the dog's cooperation regarding being positioned –

Table 13.1 Description of postural drainage positions for the canine lungs (from Manning *et al.* 1997)

Area of lungs to be drained	Postural drainage position
Lateral segment of the left caudal lung lobe	The patient is placed in left lateral recumbency with the hind end elevated 40°
Left and right caudal dorsal lung fields	The patient is in sternal recumbency with hind end elevated 40°
Left and right caudal ventral lung fields	The patient is in dorsal recumbency with hind end elevated 40°
Left and right cranial ventral lung fields	The patient is in dorsal recumbency with front end elevated 40°
Left and right cranial dorsal lung fields	The patient is in sternal recumbency, with the front end elevated 40°
Right middle lung lobe	The patient is in dorsal recumbency. A pillow is placed under the right side of the thorax so that the right side is higher than the left side. The hind end is elevated 40°, and the front end is rotated one quarter turn to the right
Lateral segment of the right caudal lung lobe	The patient is in left-lateral recumbency with the hind end elevated 40°

is generally much greater the sicker the dog. Sometimes, the position may need to be modified to ensure patient compliance; while on other occasions, positions have had to be modified because of concomitant pathologies. In all cases, as when treating humans, oxygen saturations and heart rate should be closely monitored throughout postural drainage and the same precautions and contraindications followed as per human treatment (Irwin & Tecklin 1990).

13.9.3 Summary

Although studies are yet to be published documenting the efficacy of chest physiotherapy for dogs with pneumonia, these anecdotal findings support those of findings in humans chest physiotherapy (Sutton *et al.* 1982). In common with human intensive-care physiotherapists, animal physiotherapists do not restrict their treatment to the cardiorespiratory system; the animal physiotherapist should also consider the effects of decreased mobility or immobility on the entire body and implement treatment strategies to address these. Positioning to help reduce the work of breathing, maintain normal passive/active joint function and nutrition and reduce pressure area development may prove valuable. Owners should be encouraged to be involved in in-hospital physiotherapy where possible. This may be of benefit in several ways: some dogs are soothed by their owner's presence; it takes some of the burden off busy staff; and it may even increase compliance with outpatient physiotherapy upon discharge if owners have had this extra opportunity to become confident with the physiotherapy programme.

Case study: Respiratory physiotherapy

An 11-year-old desexed male West Highland white terrier presented to a veterinary practice with presumed aspiration pneumonia. Pneumonia predominantly affecting the right middle lung lobe was found on radiographs. The dog was hospitalised for stabilisation and intensive care and was subsequently referred for in-hospital physiotherapy on day three.

Initial physiotherapy assessment revealed a respiratory rate of 28 breaths per minute, with moist crackles on auscultation. Initial in-patient physiotherapy for acute pneumonia consisted of percussion, vibration and cough stimulation in the correct postural drainage position for the right middle lobe, i.e. 45° from right-side-up lateral recumbency with a 40° head-down tilt, owing to the lobar bronchus coming from the ventral floor of the right principal bronchus and travelling in a ventrolateral and sometimes caudal direction. This position was maintained while percussion was performed for 30 seconds, followed by expiratory vibrations on the subsequent three breaths, and this pattern of percussion–vibrations was repeated three times before using tracheal stimulation at the level of the third tracheal cartilage to ensure the dog coughed (Pryor *et al.* 1990; Pryor 1999). The physiotherapy was performed in this sequence in an effort to mimic the 'active cycle of breathing technique'. The programme was performed three times a day and resulted in observed increases in expectoration.

A final diagnosis of *pulmonary fibrosing alveolitis* was made and the dog discharged from hospital. Before discharge, his owners were taught to position the dog correctly and perform the percussion, expiratory vibration and tracheal stimulation techniques three times a day. At follow-up 4 days later, the owners reported that the dog had consistently spontaneously coughed after the percussion and expiratory vibration treatment, negating the need for tracheal stimulation. Although the owners had not visualised any sputum as a result of the dog coughing, evidence of the treatment working included much improved breath sounds on auscultation on interim veterinary examination, as well as a reported increase in exercise tolerance. A further veterinary examination 3 days later revealed resolution of pneumonic changes. The owners were given advice about weaning from physiotherapy by staging return to normal activity over the next 10 days.

13.10 Cardiac rehabilitation

Helen Nicholson

Current cardiac rehabilitation of humans consists of a combination of exercise, psychological and educational interventions. A systematic review and meta-analysis of randomised controlled trials of exercise-based rehabilitation of patients with cardiac disease demonstrated that, compared with usual care, patients participating in cardiac rehabilitation programmes had lower rates of all-cause mortality and lower systolic blood pressure (Taylor *et al.* 2004).

Exercise rehabilitation is recommended in the post-acute phase of pulmonary oedema and other cardiac conditions because it is understood that physical deconditioning occurs as a result of the immobility associated with such severe illness. Clinical guidelines outline safety issues to be considered, reporting a frequency of only one major complication per 67 126 hours of patient exercise in human programmes where safety considerations are met (Isles *et al.* 2002). Pre-exercise assessment is essential in human cardiac rehabilitation and programmes should be designed to include warm-up, aerobic conditioning and cool-down phases. The intensity of exercise recommended for humans differs according to the patient's typical level of daily exertion, i.e. patients with physically demanding lifestyles need to achieve a higher intensity level of rehabilitation than

those with more sedentary lifestyles, although the progression should be carefully graded throughout the rehabilitation programme (Isles *et al.* 2002). The duration of a human cardiac rehabilitation programme is recommended to be a minimum of 8 weeks, with at least two exercise sessions per week. The evidence shows no difference between programmes with a majority of sessions at home compared with a majority of sessions at hospital (Boddy *et al.* 2004). There is limited literature available on the effects and benefits of exercise in canine patients suffering from cardiac disease.

An exercise programme may prove to be of benefit to dogs demonstrating early signs of cardiac disease. Most aspects of human cardiac rehabilitation, including pre-exercise assessment tests (e.g. 6-minute walk test) are easily adapted to the treatment of dogs and are something that most owners can learn to use to monitor progress at home between physiotherapy appointments. It can, however, be harder to teach owners to reliably monitor their dog's response to the intensity of the exercise; hence techniques such as respiratory and heart-rate monitoring must be practised with the owner until you are both confident the dog will be safely monitored at home. Ideally, the programme should be started early in the pathogenesis of the disease and strictly monitored by both the veterinarian and physiotherapist.

Case study: Cardiac rehabilitation

Courtesy of: Sue Potelli Demajo, M. Anim. St. (*Animal Physiotherapy*)

An 11-year-old Labrador-cross dog was diagnosed with a moderate-grade left-sided heart murmur over the mitral valve by a veterinarian. The dog was still asymptomatic so a cardiac rehabilitation programme was recommended by the veterinarian in consultation with an animal physiotherapist. Cardiac function was monitored via a portable Polar heart rate (HR) monitor strapped to the dog's chest (www.polarusa.com). A functional 6-minute walk test (Guyatt *et al.* 1985) was performed on the treadmill at a controlled speed of 3.1 mph and at a 0% incline before and after the completion of the exercise programme. Since it is not possible to use the subjective Borg scale (Guyatt *et al.* 1985) of perception of fatigue in dogs, during the testing procedures fatigue was assessed in the dog by more objective signs including tension in the lead; behavioural indications, e.g. the dog started pulling at the owner's trousers or shoelaces to bring his/her attention to the fact that she was tired; and recovery rate – both immediately and the day following the exercise programme.

The progressive programme was performed for eight sessions on a treadmill over 3 weeks with increasing incline of 1% and increasing velocity of 0.5% from a minimum speed of 1 mph to a maximum of 6 mph every minute and a recording of the HR was done every minute for 20 minutes. The owner of the dog was asked to walk with the dog on the treadmill, to reduce the dog's stress and improve accommodation to treadmill walking (Figure 13.8a, b and c). Three ECG leads were attached to the dog's chest, one on each side close to the axilla area, and one at the centre of the chest, just under the xiphisternum. The exercise programme was initiated at a conservative exercise heart rate of 130 beats per minute (bpm) (approximately 45% HR max (Wagner *et al.* 1977)).

The diet of the dog was also changed from a wet-food diet with a caloric distribution of 24% protein 17% fat and 59% carbohydrate, to the dry food Eukanuba (<http://www.eukanuba.com>) restricted calorie diet, of calorific content of 22% protein, 6.6% fat and a 54.7%



Figure 13.8 Cardiac rehabilitation case study: 11-year-old Labrador cross diagnosed with a moderate grade left-sided heart murmur over the mitral valve. Exercise programme performed (a) the initial phase; (b) getting closer to the exercise heart rate; (c) the cool-down period.

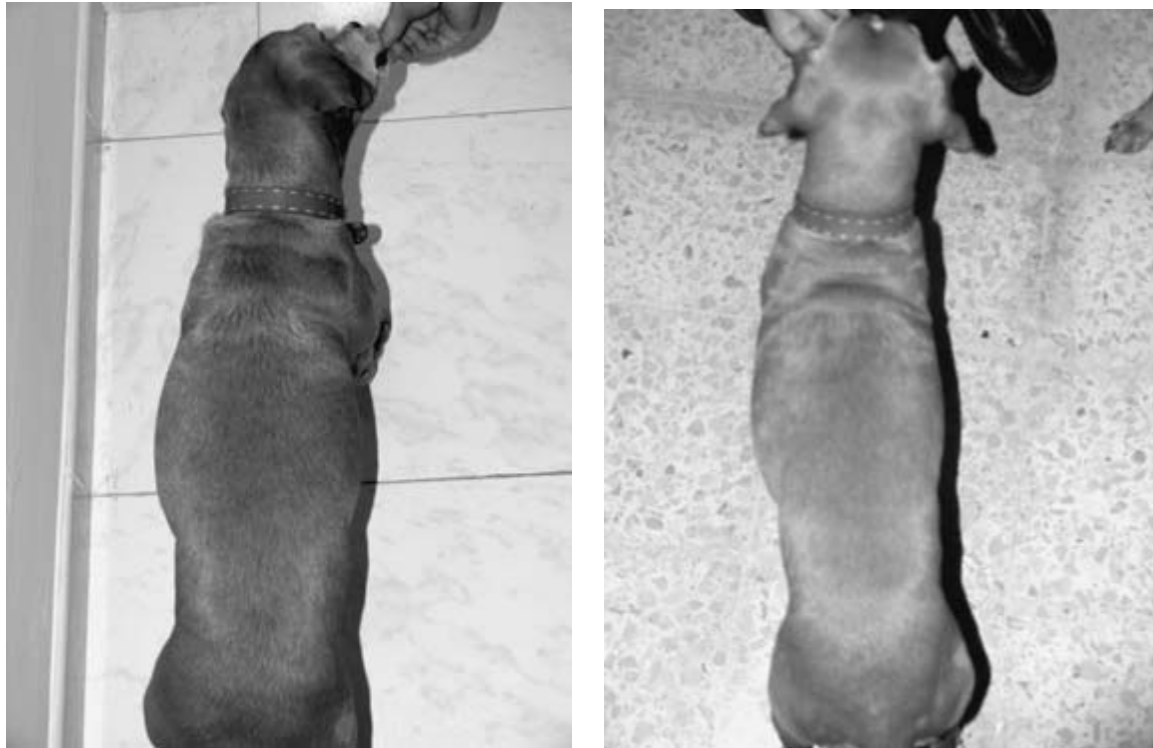


Figure 13.9 Cardiac rehabilitation case study: (a) conformation before the diet; (b) conformation after the diet.

carbohydrate. During the programme the dog did not lose weight but her conformation changed according to the Purina conformation analysis (<http://www.purina.com/>) in that she lost fat around her ribs, neck and waist area (Figure 13.9).

The dog showed increase in fitness with the programme. Resting HR decreased by 21 bpm and the time at which the exercise HR was reached during the incremental training session increased by a total of 7 minutes following the eight sessions of exercise. The 6-minute walk test showed a decrease in heart rate by 41 bpm and by 43 bpm before and after the cool down respectively.

However, during the fifth session the dog was showing signs of fatigue with the exercise HR reaching exercise levels very early on in the exercise. When the exercise session was stopped and the HR returned to resting levels again, upon reintroduction of exercise the HR shot up to maximum immediately. At this point the exercise programme was stopped and the dog rested for a few days, highlighting the need for monitoring techniques.

This single case suggests that a cardiac rehabilitation programme may be applied to dogs as it is to humans, especially if it is started at an early stage in the disease as soon as it is diagnosed by a veterinarian and before clinical signs become severe. Research in this area is warranted.

13.11 Neurological physiotherapy

Helen Nicholson

13.11.1 Introduction

Like so many other aspects of physiotherapy in veterinary medicine, neurological rehabilitation is not yet routinely or always appropriately applied. The evidence base for human neurological physiotherapy is constantly expanding and with many studies performed on animal species, e.g. cats, the use of physiotherapy in neurological rehabilitation of animals is likely to continue to increase. The use of physiotherapy to rehabilitate animals with severe neurological lesions may reduce the incidence of euthanasia, depending on owner commitment and ability to perform the home

exercise programme competently and frequently enough. The ability of the physiotherapist to exploit the healing process and encourage neural plasticity plays a vital role in the success of rehabilitation.

13.11.2 Potential indications

Potential indications for neurological physiotherapy include:

- *Cranial nerve disease*, e.g. trigeminal neuralgia; facial nerve dysfunction, which may be localised after tick poisoning; traumatic vestibular disease or post surgical, e.g. after ear-canal ablation
- *Spinal cord disease*, cervical, thoracolumbar and cauda equina, e.g. caudal cervical vertebral malformation

(CCVM); Hansen's type I and type II intervertebral disc disease; fibrocartilaginous embolism (FCE) and chronic degenerative radicular myelopathy (CDRM)

- *Peripheral nerve disease*, e.g. traumatic peripheral nerve paralysis
- *Generalised neuromuscular disease*, e.g. polymyositis; tick paralysis; polyradiculoneuritis (also known as 'coonhound paralysis') (see also Chapter 7).

It is important when treating any of these conditions that a thorough initial neurological examination is performed and repeated neurological examinations are performed and recorded to assess changes in grade and status. For example, with intervertebral disc lesions, it is possible to have more than one site involved or for another to become involved later on. As visits to the veterinarian tend to become less frequent some time after the original insult, but regular physiotherapy rehabilitation can persist for months, it can be the physiotherapist who has the first opportunity to notice red flags and promptly refer back to the veterinarian. *Please ensure that if the patient no longer fits the pattern of the condition they were referred for, urgent veterinary review is sought.*

Bladder function signs require particular attention and review as these may change rapidly, especially when associated with spinal cord injury (Chapter 7). It is not uncommon for animals to be discharged from hospital before developing full bladder control and owners may not be able to competently manage their bladder care at home. Often owners may be fooled into thinking that a wet bed means the dog has voluntarily urinated and/or completely emptied their bladder. 'Red flags' to watch for include:

- Distended bladder on palpation or (worse) observation
- Increase in intensity of the smell of the urine
- Haematuria
- Darkening of the colour of the urine
- Loss of appetite or interest in daily activities
- Fever

A urinary tract infection in a neurological patient is a medical emergency and veterinary treatment should not be delayed under any circumstances.

13.11.3 Neurological physiotherapy for animals

After neurological insult, spontaneous recovery of function may occur as a result of: reduction in swelling or reabsorption of haemorrhage; due to remyelination in the case of demyelinating disorders; or due to collateral circulation in the case of infarcts (Stephenson 1993). Alternatively, neuroplasticity (defined as the reorganisation or adaptation of neural tissue after insult) and Wallerian degeneration, via: regeneration of axons or dendrites; the collateral sprouting of remaining nerve fibres; or the unmasking of latent synapses; may be responsible for recovery (De Lahunta 1983). Physiotherapy therefore aims to: take advantage of spontaneous recovery; manipulate neuroplasticity to assist

functional return; prevent or minimise complications; and to implement compensatory strategies when poor prognosis results in the threat of euthanasia.

13.11.4 Principles of neurological rehabilitation

Many of the principles of neurological rehabilitation in humans have focused on rehabilitation of the stroke, head or spinal-cord injured patient and/or development or redevelopment of finely adjusted movements and skills characteristic of the pyramidal system, which is the predominant descending upper motor neurone system in the human spinal cord. However, it should be remembered that the pyramidal system in dogs is far less important and it reduces by half after the cervical spine (Chapter 7). This means the focus of neurological rehabilitation should be on functional gait retraining and development of the stereotypic patterns of gait and coarse movements compared with fine movements in humans.

The following information on motor learning has been derived from work in humans (Marteniuk 1979; Carr & Shepherd 1987; Magill 1989).

Phases of motor learning

- *Cognitive (elementary) phase*: A large number of errors occur with variable performance and slow reaction time. The patient's full attention is required (which can be difficult in animals, see Chapter 2).
- *Associative (intermediate) phase*: Fewer errors occur as components of the task become more integrated and performance becomes more consistent and efficient.
- *Autonomous (advanced) phase*: Movement becomes habitual with fast reaction times. Attention is released for other tasks.

Motor learning is enhanced by practice schedules that include:

- *Motivation*: The use of positive reinforcement and owner involvement keeps motivation levels high in most dogs, although different forms of positive reinforcement work with different patients, such as: food rewards; vocal encouragement; familiar toys or fetch items; environmental stimulus or familiarity (outside, grass surfaces, own home); other household animals being present; owner's touch and sensory stimulation (scratching favourite spot or tickling tummy); smells and sounds. When working with food-motivated but overweight dogs, subtract rewards from daily rations and encourage patients to work for meals to reduce exacerbating the weight problem.
- *Specificity*: Goals must be attainable and appropriate in order for rehabilitation to be effective – discuss treatment goals with the owners from the outset. Break complex tasks into parts, but always include practice of the whole task (with assistance as required to ensure successful performance). Owners must be made aware of

the smaller goals or components of the task that make up the larger goal or functional activity. For example, if the dog is able to stand for 1 minute with one person's light assistance of the pelvis following a thoracolumbar upper motor neurone disc lesion, this does not mean it is able to coordinate or has the strength to get into standing or walk under its own volition.

- *Meaningful context:* Relevance is important – many dogs find food lures make motor-learning exercises meaningful. In people, mental practice alone has proved to help improve motor performance – it is therefore possible that using familiar verbal commands may help dogs mentally 'rehearse' the commands that they are as yet unable to perform, however this has not been studied objectively.
- *Contiguity:* The patient must be able to perceive the relationship of events that occur together in time – instead of practising exercises in isolation, practise them in sequence. For example, once a tetraplegic dog has lifted the head, assist it into sternal recumbency, sitting and then standing so that the total task gives the dog feedback from the outcome of the initial head turning task, even if the dog has to be fully supported by a sling in standing.
- *Reinforcement:* To maintain and enhance motivation, the use of positive reinforcement is vital – excited tones of verbal praise and prompt delivery of food or other rewards both help to reinforce that the correct movement has just been performed and encourage the animal to remain motivated.
- *Feedback:* Feedback is essential if skill acquisition is to be maximally enhanced and must be positive and motivating, specific and accurate. Extrinsic feedback – food rewards, verbal praise, or luring the dog into position – can provide feedback about the desired position to be attained. Intrinsic feedback is derived from sensory receptors, e.g. tactile input can be used to help provide proprioceptive feedback. Feedback must be instantaneous after each attempt for the animal to understand best what it is that the physiotherapist is trying to teach. For further advice on giving feedback and motivating dogs, please research modern methods of teaching dogs obedience, as these techniques translate well to neurological physiotherapy.
- *Activation:* Try to relate learning to past function, e.g. if a dog habitually trotted pre-injury, teach stepping in the same sequence. Reduce distractions to enable selective attention to the task.
- *Repetition:* Repetition of tasks is vital, however the physiotherapist should be aware of rapid development of fatigue in many neurological cases. Fatigue interferes with the performance of a task – work 'to' not 'through' fatigue. Prolonged or too-frequent practice sessions do not significantly improve learning. Rest is important and

should be emphasised to overenthusiastic owners. For example, when practising sit to stand with a dog recovering from paresis of its pelvic limbs, have it start by sitting on a block to raise its pelvic position from the ground, thus making the task easier and more repetitions possible. Include rehabilitation exercises designed to improve cardiovascular fitness and discuss the pathophysiology of the diagnosis with the veterinarian to reduce the risk of catabolism occurring as a result of overexertion. The use of a treadmill, either underwater or land based, may facilitate neurological rehabilitation when used in conjunction with sound clinical reasoning skills.

Stages of motor control

- *Mobility:* Requires adequate range of motion and motor unit activity to make use of that range of motion.
- *Stability:* Requires ability to co-contract agonist and antagonist muscles to allow maintenance of anti-gravity weight-bearing postures.
- *Controlled mobility:* Requires at least one of the weight-bearing limbs to be lifted while the animal continues to control posture.
- *Skill:* This is the highest level of motor control and requires proximal stability so that distal body segments can undertake free skilled movement.

A dog may present at any stage on this continuum of stages of motor control to be attained during neurological physiotherapy. Thus, thorough assessment skills are required to ascertain at what stage a patient presents; and likewise a strong repertoire of therapeutic skills must be acquired to allow the physiotherapist to assist dogs of a variety of temperaments to progress from one stage of motor control to the next.

13.11.5 Therapeutic approaches to neurological rehabilitation

Many different therapeutic approaches are advocated in different regions throughout the world. As for the above information on skill acquisition and the stages of motor control, information on the various therapeutic approaches is included to help compensate for the different training that physiotherapists receive throughout the world. No one therapeutic approach is described in complete detail – the reader is encouraged to refer to the cited literature to learn more about specific approaches with respect to central and peripheral neural plasticity along with sensory and motor integration and retraining. The use of a variety of principles from the following approaches is necessary when working with canine patients, to compensate for their inability to speak and perform various key components of movements on command. Where verbal communication is lacking, some of the sensory stimulation techniques can often help to bridge the gap between desired and actual function, as is evidenced by the case studies.

The Bobath approach

The Bobath approach (Bobath 1990) is based on the concept that abnormal posture and movement 'shunts' sensation so that the abnormal patterns begin to feel normal to the patient. Treatment, therefore, aims to facilitate normal movement and posture so the patient experiences the sensation of normal movement patterns. Selective, isolated control is developed by the reduction of hypertonus, dissociation of mass patterns, avoidance of associated reactions and prevention of effort. Functional skills are built upon selective isolated control.

Bobath techniques include:

- Handling of 'key points' (regions of the body where movement is centred or initiated from)
- Awareness and active correction of differences between positions and postures
- Reflex inhibition of patterns and control of associated reactions
- Tapping and joint compression or weight bearing
- Accurate feedback
- Repetition of normal movement as often as possible throughout the day
- Sensorimotor re-education

Although none of these techniques has been defined in small animals it can be seen clinically that some of these techniques may lend themselves better than others to small animal neurological physiotherapy, with great variability expected between individual dog and cat acceptance of these techniques.

The Rood approach

The Rood approach (Rood 1956) is the concept of using very specific sensory stimuli to facilitate specific responses.

Rood advocated the following sensory stimuli:

- Cutaneous – fast brushing, light touch, stroking
- Thermal – ice, warmth
- Proprioception – stretch, tapping, resistance, joint compression
- Labyrinthine – rocking, rolling
- Visual
- Auditory
- Olfactory
- Taste

In the Rood approach, the initial reflex muscular response is used in developmental sequences before being incorporated into purposeful skilled movement. Rood's developmental sequence components can be modified for dogs to include:

- Prone (drop)
- Sitting
- Standing
- Walking

Rood considered feedback and repetition crucial for learning to occur.

Proprioceptive neuromuscular facilitation

Proprioceptive neuromuscular facilitation (PNF) (Voss *et al.* 1985) aims to use specific proprioceptive stimulation to re-educate the neuromuscular system. The spiral and diagonal movement patterns used in PNF are considered to be characteristic of mature movement.

Specific PNF techniques include:

- Hold relax – an isometric contraction of the muscle limiting motion
- Contract relax – a concentric contraction of the muscle limiting motion
- Slow reversal – alternating slow rhythmical concentric isotonic contraction of first the agonist then the antagonist muscle
- Rhythmic stabilisation – uses simultaneous isometric contraction of agonist and antagonist to encourage co-contraction, stability or even relaxation. Clinically, this tends to be the easiest technique used with dogs, working with the obedience command 'stay'; the technique can be progressed from four-point stance to three and then diagonal two-point stance.

The Brunnstrom approach

Brunnstrom advocated that movement recovery occurs through six successive stages (Sawner 1992):

1. Flaccidity
2. Synergies developing with increasing spasticity
3. Synergies performed voluntarily with spasticity at its peak
4. Some movement out of synergy as spasticity decreases
5. Independence from basic synergies develops as spasticity further decreases
6. Isolated co-ordinated movements performed at will

In early treatment with the Brunnstrom approach, synergies are facilitated through the use of postural reactions and proprioceptive and other stimuli as precursors to normal movement. A clinical example of this application is for a dog with a central thoracolumbar disc herniation (post-surgical bilateral pelvic limb flaccid paresis): with the dog supported in standing, the head can be lured to one side with a food treat, stimulating the asymmetrical tonic neck reflex, which increases the extensor tone of the ipsilateral limbs and the flexor tone of the contralateral limbs. By alternating food treats to the left and right, the physiotherapist stimulates the precursors of normal extension tone for stance phase and flexion tone of swing phase of gait.

The motor relearning programme

Carr and Shepherd have turned the focus of the inductive neurofacilitation models developed by Bobath, Brunnstrom

and colleagues towards the deductive motor relearning model (Carr & Shepherd 1992; Carr & Shepherd 2000). In *A motor relearning programme for stroke* (Carr & Shepherd 1992), the (human) patient's performance is compared with normal movement, based upon knowledge of the essential components of that task. The physiotherapist then assists the patient to activate the required muscles and practise so that selective control over those muscles is improved. The physiotherapist teaches the patient how to perform that motor task while concentrating on those task components that are both critical to the successful performance of the task and difficult to perform. The patient is therefore encouraged to become an active learner and problem solver and as such one might expect difficulties in applying this cognitive strategy to dogs. In practice, however, most dogs are easily motivated and easily taught a motor skill in much the same way that positive reinforcement, shaping and operant conditioning are used to teach puppies the basic motor tasks of 'sit', 'drop', 'come' and 'stay'. Clinically,

it has, however, often been the case that dogs that have previously been taught motor tasks, such as the basic obedience commands, adapt more easily to the motor relearning approach.

13.11.6 Neurological rehabilitation in animals

Cranial nerve dysfunctions may present for physiotherapy as primary neurological complaints, however they are often associated with a multitude of other neurological dysfunctions or secondary lesions or complications. A dog presented with a fractured hock and during assessment it was noted that she had a mild right-sided facial asymmetry following the previous attachment of a paralysis tick near the facial nerve. The dog responded to sensory stimulation techniques including brisk rubbing with an ice cube and gentle brushing with a vibrating electric toothbrush to try to improve sensory and motor tone. Please note that not all cases will tolerate all sensory stimuli, owing to neuralgia or hypersensitivity that may occur.

Case study: CCVM post-operative rehabilitation (ventral slot stabilisation)

A 9-year-old male neutered Doberman presented to the veterinarian with tetraplegia (neurological gait grade 5/5), diagnosed via myelogram with C5–6 CCVM and underwent ventral slot and stabilisation surgery. The surgery was successful, however, the dog did not recover spontaneously and was referred to physiotherapy 6 weeks later. The dog had previously undergone right cranial cruciate surgery which had not received physiotherapy. Physical findings on presentation included: tetraplegia; recumbency requiring assistance to hold his head up to drink; variable soft tissue contractures at all major peripheral joints; atrophy of the epaxial musculature, thoracic limb and pelvic limb muscles bilaterally; and thickening of the right stifle.

Treatment: It was necessary to restore normal passive range of motion before active control of movement was attempted (Sullivan *et al.* 1982). After 2 weeks of an intensive home programme of 2-hourly turns, passive range of motion exercises, heat and soft tissue mobilisation techniques, the functional pain-free passive range of motion available at his peripheral joints had improved to near normal. A motor relearning programme was initiated to teach the dog to move from the lateral recumbency position to half sternal lying. Progress was slow, learning the positions half sternal lying, full sternal lying, sitting and then standing, with his willingness to participate in exercises depending on his proclivity for the various treats offered. Ten weeks after physiotherapy commenced, the dog took his first unaided steps, taking approximately 15 minutes to walk 47 metres. His programme was upgraded over the subsequent 2 months to include negotiating stairs and bushwalks. The dog continued to function well and follow-up 2 years later showed that he had maintained his neurological gait grade of 2/5, with only mild proprioceptive deficits during bushwalks, remaining independent with the family home which had five stairs. His owners carried out a maintenance home programme weekly, consisting of various neurofacilitatory exercises used throughout his rehabilitation programme.

Case study: General neuromuscular disease

An 8-year-old obese female entire Cocker Spaniel presented to a veterinary neurologist with a 48-hour history of a hoarse bark and rapid deterioration resulting in flaccid tetraplegia. The veterinary neurologist diagnosed polyradicular neuritis, or 'coonhound paralysis', and the dog was admitted to hospital for in-patient management and intensive physiotherapy. On initial presentation to physiotherapy, the dog was obese (grade 9/9 on the Purina scale (<http://www.purina.com>), with her only voluntary movements being of the head and cervical spine. Cranial nerves and mentation were normal.

The dog's obesity was a significant impediment to successful manual handling and gait re-education. The dog was therefore concurrently placed on a restricted calorie diet, monitored in hospital to ensure compliance and safety.

Physiotherapy treatment began with sensory stimulation to all four limbs, the lumbar and thoracic spines and abdomen. The aim was to stimulate the sensorimotor system via mechanoreceptive and proprioceptive facilitation techniques to assist in increasing muscle activity, particularly of the antigravity muscles, via local spinal reflexes, such as activating stretch receptors with rapid vibration or tapping, or mechanoreceptors via icing. Passive range of motion exercises for the peripheral joints and soft tissue mobilisation or massage of the

entire dog was also an integral part of treatment. The dog's weight was supported via a sling in partial weight bearing, owing to the benefits of weight bearing on bone density, muscle and joint health, bowel function and behaviour. As the dog found this type of exercise quite tiring, it was hoped that it would also contribute towards weight loss and help to maintain whatever cardiovascular fitness she had before the onset of her illness.

Treatment focused strongly on the implementation of motor relearning theory, commencing with teaching the dog to move from lateral recumbency to half sternal recumbency, then to full sternal recumbency, then to sitting, followed by standing and finally stepping. At all stages, the dog was also taught relevant balance strategies via both internal and external perturbations in each position and how to descend safely to the floor again to minimise the risk of injury due to a fall. The dog was not motivated by food or toys, but could be motivated by having her lead and collar on and a walk with her companion dog simulated. The dog was repeatedly analysed to compare her performance of a task to that of normal dogs and was assisted to perform the missing or difficult components until she could perform them independently and thus move to the next level of task.

Fourteen weeks after presentation, the dog took her first steps. This type of time frame is not rare for such debilitated dogs, which owners often find difficult to appreciate, failing to understand that a human with a similar intensity of injury would probably take far longer to rehabilitate.

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14

Equine treatment and rehabilitation

Lesley Goff and Narelle Stubbs

14.1 Introduction

14.2 Exercise-based rehabilitation

14.3 Stretching for injury prevention and rehabilitation

14.4 Assessment of the horse and rider unit

References

14.1 Introduction

In the majority of forms of equitation, including the pleasure horse and specifically the competition athlete, the horse performs to its optimum, and is often performing to the limit of, or close to its physiological limits. In many cases the horse is loading the body systems in a repetitive or cyclical way. This may involve and ultimately affect the neuromuscular, orthopaedic, cardiovascular and metabolic systems, hence all must be considered during the rehabilitation process. As in the canine athlete, the lack of direct verbalisation with the animal necessitates quality veterinary and physiotherapy assessment, diagnosis and management in all levels of equine sport, from the child's aged pony to the elite athletes, with respect to injury prevention, treatment, rehabilitation, quality of life and performance enhancement.

Assessment–treatment–rehabilitation in the horse should follow the current human and veterinary evidence-based practice as described throughout this text. This not only encompasses both orthopaedic and neuromuscular principles (Chapters 8, 9, 13) but also exercise physiology (Chapter 5), biomechanics (Chapter 4) and performance enhancement. The use of a 'neuromotor or neuromechanical control' model for rehabilitation is encouraged. Ironically, these neurophysiological terms describe the equine locomotive system – balance of stability and mobility (Chapter 4).

Physiotherapy for equine orthopaedic and neurological conditions, similar to canine physiotherapy, is based on a strong scientific background in human medicine. There are various professional and lay texts available to the reader in relation to equine treatment and rehabilitation, however, while there have been considerable advances in diagnostic methods and veterinary management, the current equine therapeutic literature often lacks a solid scientific evidence base. Hence it is a necessity that physiotherapists develop expert clinical reasoning skills and are able to apply the

available evidence-based knowledge to treatment, rehabilitation and management of the equine athlete, continually keeping abreast of new evidence-based advances. Denoix and Pailloux assimilate physiotherapy and sports medicine concepts with veterinary management, describing physiotherapy as being 'based on the understanding of, and respect for, biomechanical structures. It is however more than just a therapeutic technique through touch and gesture; it is also a means of sensory communication, of bridging the silence between horse and rider' (Denoix & Pailloux 2001).

The pathophysiology of injury and healing of the animal's body systems are presented elsewhere in this text, including that presented in the canine treatment and rehabilitation chapter (Chapter 13). These principles can be adapted to the horse's comparative anatomical and biomechanical differences. Unlike the dog and human, in the majority of circumstances, the horse is treated in standing; thus much of the musculoskeletal system is treated in the weight-bearing (WB) or, partially weight-bearing (PWB) postures. This is because the horse can only be made recumbent via analgesia; i.e. you can't make them lie down! However, clinically, the majority of physiotherapeutic techniques, including manual therapy, soft tissue and skeletal mobilisation and manipulation (Chapters 9 and 12), proprioceptive facilitation techniques (Chapter 13), electrotherapy (Chapter 10) and exercise-based rehabilitation can be adapted and performed successfully. These are all part of the multimodal approach to physiotherapy management of athletic, traumatic, and degenerative injuries seen in the performance and pleasure horse.

A vital aspect of equine physiotherapy that will be demonstrated in this chapter is the pivotal role played by the rider in all aspects of the horse's neuromuscular system. The rider could therefore be seen as an extension of the horse, thus the concept of 'cause and effect' may ensue in many circumstances of chronic neuromuscular and performance-related problems. Hence a global approach must

be taken to both the horse and rider, and cover all aspects of the prevention–rehabilitation–performance–enhancement management time line.

14.2 Exercise-based rehabilitation

The introduction of exercise-based rehabilitation to the physiotherapy management of orthopaedic and neurological conditions in horses is based largely on human research, but there is also a growing bank of research in equine exercise physiology and injury rehabilitation. Understanding the responses to exercise of injured and non-injured soft tissue, cartilage and bone can guide us in exercise prescription for rehabilitation, and possibly injury prevention.

Exercise-based rehabilitation should be introduced at a stage of physiotherapy management that is appropriate for the injury. It is a broad concept, encompassing strengthening, specific training regimes, facilitation of neuromotor control systems, and stretching. Acute conditions may require initial interventions to reduce inflammatory response, so as not to compromise tissue-level healing, with mobilisation implemented in later treatments (Warden 2005). Chronic conditions, such as chronic sacroiliac joint injury, or sacroiliac degenerative joint disease which often result in poor performance (Jeffcott *et al.* 1985) may benefit from specific exercises aimed at facilitating normal neuromotor control at initial treatment.

In the horse, many musculoskeletal injuries are directly related to locomotion. Locomotion involves cyclical loading of both soft tissue and bone, in the vertebral column and extremities. There are some mechanisms in the horse, such as the passive stay apparatus, that predispose the horse to developing particular pathologies; these mechanisms have been discussed in Chapter 4. Prevention or management of musculoskeletal injuries thus relies on an understanding of the mechanical properties of bone, tendon, ligament, muscle and cartilage and how they respond to repetitive loading and unique equine locomotion.

14.2.1 Tendon

A common cause of lameness in athletic horses is injury to the tendons of the forelimb, most commonly the superficial digital flexor tendon (SDFT); but also the interosseous tendon and accessory ligament of the deep digital flexor tendon (Meershoek *et al.* 2001; Batson *et al.* 2003). These tendons contribute to locomotor efficiency by storing energy, that is, they act like springs, returning 93–95% of stored energy, as a result of elastic deformation (Wilson *et al.* 2000).

Tendons that store energy during locomotion have a low safety margin – this refers to the tendon operating close to the level of gross tensile failure (Batson *et al.* 2003). Such tendons are described as having conflicting requirements for strength and elasticity. The matrix composition of SDFT has been found to differ from that of the common

digital extensor tendon (CDET) regarding water and total sulphated glycosaminoglycan content, to allow the SDFT to remain more elastic as a material (Batson *et al.* 2003). Tendons such as the opposing CDET and the deep digital flexor tendon (DDFT) which function to position the limb, have fewer instances of atraumatic injury in the equine athlete than the SDFT (Batson *et al.* 2003).

A frequent clinical finding is partial SDFT rupture with a central core lesion (Wilmink *et al.* 1992). The central fibres tend to rupture at strains lower than that required to rupture the peripheral fibres, owing to differences in crimp characteristics of the collagen fibres of the two areas of the tendon (Wilmink *et al.* 1992). This is particularly so in older horses. This common SDFT injury is a degenerative type of injury, related to cyclical loading, and can be likened to tendinopathy in human athletes. The most common tendinopathies in humans are degenerative tendinoses of the Achilles, quadriceps and rotator cuff tendons. Tendinosis is distinct from acute tendinitis, in that the latter is characterised by heat, swelling, and pain due to inflammation (Gillis *et al.* 1993; Peers & Lysens 2005). In tendinitis, early tendon inflammation is characterised by decreased echogenicity on sonography, but maintenance of fibre alignment (Reef 2001). Tendinosis is characterised by local painful thickening and structural changes on imaging (decreased echogenicity or anechoic areas and fibre disruption) and hypertrophy of tendon (Reef 2001; Malliaras *et al.* 2006). Morphological characteristics of tendinosis include increased glycosaminoglycans, irregular fibre structure and arrangement, and no inflammatory cell infiltrates (Movin *et al.* 1997). The pain in chronic tendinopathy is not inflammatory in nature but its exact origin remains unclear. It is thought that some of the pain may be due to vascularisation into the tendon (Ohberg & Alfredson 2002). In equine literature it seems tendinosis is often misnamed tendinitis – it is important to understand the differences in pathology between the two conditions, as they are managed differently, and rehabilitation has a different role in each condition.

Appreciation of the mechanisms involved in response of normal tendon to training has implications for developing rehabilitation strategies in the management of tendon injuries, particularly the degenerative type injuries caused by repetitive microtrauma (Firth 2006).

Response of equine tendon to exercise

Results of responses of tendon to training have been conflicting in equine literature, highlighting the fact that the level and type of exercise may be critical in rehabilitation.

During the first 4 months of race training, it was found that there was an increase in mean cross-sectional area, and decrease in echogenicity in SDFT of six young Thoroughbreds, measured ultrasonographically (Gillis *et al.* 1993). Increase in cross-sectional area may lead to an

increase in stiffness, thereby reducing the elasticity and energy-storing capacity of the tendon (Marlin & Nankervis 2003). The race training programme involved a gradual increase in speed through trotting to gallop, and at 10 weeks, work at race speed every fifth training day. After the first work at race speed, two horses developed clinical signs involving grade 1 lameness, mild heat and swelling in mid-metacarpal region of SDFT. Only one of these horses demonstrated histological changes in the tendon, involving increased vascularity and lymphatics.

Another study involved horses being given controlled treadmill exercise for up to 18 months. Changes in cross-sectional area of the extensor tendons were not observed (Birch *et al.* 1999).

Tendon rehabilitation in humans

Much of the rehabilitation of tendinopathy in humans has revolved around eccentric loading of muscle and tendon. Eccentric loading of tendon has had good clinical results over a 12-week training period, but the mechanism behind the results is unknown (Alfredson *et al.* 1998). Eccentric calf muscle training in patients with mid-portion Achilles tendinosis resulted in decreased tendon thickness and normalised structure on imaging (Ohberg *et al.* 2004).

Sustained (3 minutes, three times daily) and intermittent stretching (five sets of 20 seconds, twice daily) increased Achilles tendon flexibility, with a corresponding decrease in pain. There was no significant difference between sustained and intermittent groups over 4 months (Porter *et al.* 2002).

In volleyball players with chronic patellar tendinopathy, eccentric quadriceps training was found to have no effect on knee function after a 12-week programme in which players continued to train (Visnes *et al.* 2005). Tendons of volleyball players undergoing eccentric loading responded variably to the increased load associated with a competitive volleyball season. It was concluded that change in pain and tendon appearance on imaging does not appear to be entirely dependent on load (Malliaras *et al.* 2006).

Implications for rehabilitation of chronic tendon injury in horses

Tendons (especially the SDFT) are already under eccentric load in the normal weight-bearing situation in the horse. As speed of gait increases, there is increased eccentric load on the tendons. Loads will also vary depending on the occupation of the horse, so rehabilitation should be tailored for the load requirements of tendon at return to full performance.

Research suggests that adaptations in equine tendons are already occurring before onset of clinical signs (Gillis *et al.* 1993). In the case of a chronic tendinopathy (tendinosis), a programme of mobilisation is preferable to a programme of immobilisation (Kannus *et al.* 2003). It is beyond the scope of this chapter to prescribe exercise rehabilitation for

every situation and occupation in equine athletics, so listed below are some general guidelines for rehabilitation of chronic tendon injury.

From onset of clinical signs, exercise should be restricted to walking. The validity of total box rest may be questioned in these cases – it is up to the discretion of the veterinarian and physiotherapist but prolonged immobilisation may contribute to further detrimental changes in chronic tendon pathology. Evenly distributed, low- to moderate-intensity exercise in young Warmbloods was more effective in developing strong, flexible tendons than single episodes of high intensity exercise superimposed on box rest (Cherdchutham *et al.* 2001). Keeping the horse in a small paddock or yard, where they can mobilise in a limited way is paramount. Progression of rehabilitation will depend on astute observation of clinical signs, and diagnostic ultrasonography at regular intervals throughout the rehabilitation (initially and then every 8 weeks – Reef 2001). Eccentric loading can be gradually increased in the following way:

1. Early
 - Standing/walking on varying surfaces, such as springy grass and sand will increase the loading.
 - Progressing walking into trotting.
 - Walk/trot on a lunge or circle will load different parts of tendon.
2. Mid
 - Progression of in-hand exercise to variable terrain, gentle gradients.
 - Ridden work at trot; straight line then circle – weight of rider will increase tendon load.
 - Graduate into canter; straight line then circle.
3. Late (36 weeks +)
 - Use of slopes and speed work will increase loading and should be brought in towards the end of the programme. Use of either or both will depend on horse's occupation.
 - Jumping horses – slopes, poles and cavallettis should be introduced. Consider that the highest forces are expected in the trailing limb during landing from jumps – fence height affects forces in the SDFT, minimally in the interosseous tendon and did not affect on the accessory ligament, therefore reduction in fence height may limit risks for SDFT injury but not effect accessory ligament or interosseous tendon (Meershoek *et al.* 2001).
 - Consider also the age of the horse – older horses have decreased crimp in the core region (Wilmink *et al.* 1992), so the rate of change of load that can be applied to the tendon may differ from that of younger horses.

It is recommended horses with severe tendon injury do not gallop until 6 months post injury, but preferably 9–12 months (Reef 2001).

Implications for rehabilitation of acute tendon injury in horses

As in all acute soft tissue injury, tendinitis involves the acute inflammatory phase, proliferative phase and maturation and remodelling phase (Kannus *et al.* 2003). (See Chapter 13 Canine rehabilitation, for the description of the response to injury at tissue level.) In acute injuries there should be a short period of immobilisation followed by controlled and progressive mobilisation (Kannus *et al.* 2003). Gibson *et al.* (1997) found in cases of tendinitis of distal insertion of SDFT, swelling, peritendinous fluid accumulation, disruption of normal fibre alignment and some loss of echogenicity ultrasonographically. As healing progressed there was return of echogenicity but normal fibre alignment did not return and there was some adhesion formation between branch of the SDFT and adjacent structures. Even though there is a distinct inflammatory phase in tendinitis, the latter may implicate the importance of mobilisation and exercise from the remodelling phase of the injury.

14.2.2 Bone

Bone has a relatively large elastic zone, and thus is described as a pseudoductile material (Marlin & Nankervis 2003). When rate of loading of bone is increased, the behaviour of bone can change from pseudoductile to brittle. A common condition in performance horses, especially young racehorses, is sore, or 'bucked' shins (third metacarpal bone) (Marlin & Nankervis 2003). This often interferes with the training programme. It has been found that increasing exercise distances at a canter and high speed in short periods (up to 1 month) are associated with an increased risk of sore shins, as a result of remodelling of bone and associated microdamage (Verheyen *et al.* 2005). Increasing cumulative exercise distances upon entering training were associated with a decreased risk of disease. Early signs of sore shins should result in the exercise programme being reduced but not completely removed (Marlin & Nankervis 2003). This is so that bone can be given time to adapt and remodel.

Implications for rehabilitation for bony injuries

For young Thoroughbred racehorses with shin soreness

This has implications for the training programmes of young Thoroughbreds in that gradual introduction of small amounts of high-speed exercise may be beneficial when designing rehabilitation programmes for re-entry into training. Canter should be kept to a minimum on high-speed work days and large amounts of both canter and high speed work should be avoided in the early stages of training and rehabilitation (Verheyen *et al.* 2005).

For shin soreness in other sports and other fracture healing

Exercise should be carried out at gaits that simulate the sport for which the horse is intended (compare racing with dressage). In encouraging bone remodelling, the horse

should be exposed to the surfaces and terrain appropriate for the sport. For instance, the surfaces horses compete on in dressage differ greatly from endurance riding and eventing. It is recommended that the more demanding the sport, the smaller the increment in training load increase. Training load should be increased no more than once per 2 weeks. Short periods of high-impact loading are as beneficial as prolonged periods of impact loading, therefore it is unnecessary to spend long periods performing high-impact activity in rehabilitation to gain bone remodelling (Marlin & Nankervis 2003).

Studies in rats have found that age does not affect loading and bone-strength gains, and the gains were from changes to bone geometry (Bennell *et al.* 2002). We may extrapolate that bone-strength gains can be achieved in all ages of horse. This is particularly applicable for dressage and eventing horses, who tend to be competitive into their teen years.

14.2.3 Cartilage

Articular cartilage is adapted to resist compressive forces, which are taken through the joint surfaces during weight bearing. Some joints, such as the sacroiliac joint, and possibly some joints of the vertebral column are thought to be exposed to shear forces rather than compressive forces (Dalin & Jeffcott 1986).

Degenerative joint disease is a very common cause of wastage in dressage horses (Dyson 2000). Degenerative changes are often seen in the tarsal joints of performance horses, the vertebral column facet joints and sacroiliac joint surfaces. In one study of 36 racehorses, all had degenerative or adaptive changes in the articular facet joints of the vertebral column (Haussler *et al.* 1999). In a population of horses studied for sacroiliac joint degeneration (SID), dressage and showjumpers were considered to be most at risk of SID (Dyson & Murray 2003). Carpal osteoarthritis is frequently seen in 2–3-year-old racehorses and other horses undergoing high-intensity training (Murray *et al.* 1999). Carpal articular cartilage in strenuously trained horses showed more fibrillation and chondrocyte clusters than cartilage from animals that underwent gentle exercise. Thus, strenuous training may lead to deterioration of cartilage at sites with a high clinical incidence of lesions such as dorsal radial carpal cartilage (Murray *et al.* 1999).

Implications for rehabilitation of articular cartilage injuries

In support of the case for controlled exercise in rehabilitation of cartilage injury or degeneration, animal studies have shown that unloading of a joint rather than overloading of the joint, combined with poor muscular control and weakness may be a risk factor in joint degeneration (Herzog *et al.* 2004; Laurent *et al.* 2006). In humans with anterior cruciate ligament injury, resulting in instability and often decreased loading at the knee, rapid development of degenerative

changes characteristic of osteoarthritis occurs, including damage to type II collagen and an increase in proteoglycan content (Nelson *et al.* 2006). Strengthening and exercise have a role in increasing joint range of motion and improving joint health – improvement in morphology of articular cartilage – by increasing muscular strength and joint proprioception (Buckwalter 2003). Deficits in neuromuscular reflex pathways as a result of decreased proprioception have been shown to have a detrimental effect on joints. Proprioceptive rehabilitation to facilitate dynamic joint stabilisation is thought to improve the neuromuscular control mechanism (Lephart *et al.* 1997).

Regarding improvement in morphology of articular cartilage, moderate exercise in human subjects has been shown to improve knee cartilage glycosaminoglycan content in individuals at risk of developing osteoarthritis (Roos *et al.* 2005). Chondrocytes have been shown to respond to both shear and compressive loading, regarding metabolic regulation and biosynthesis of chondrocytes (Eckstein *et al.* 1999; Frank *et al.* 2001).

Thus for weight-bearing joints, such as the carpus, in which the cartilage resists compressive forces, we can extrapolate from equine and human research that moderate exercise can have a role in maintaining or improving the status of joints with articular cartilage degeneration. Research suggests this is also the case for joints subjected to shear forces, such as the sacroiliac joint (Eckstein *et al.* 1999; Frank *et al.* 2001). It is thought that the equine musculoligamentous ‘sling’ and fascial system surrounding the vertebral column and sacroiliac joint (SIJ) may be under proprioceptive control of neural elements within the tissue, as is the case in humans and pigs (Indahl *et al.* 1999; Brolinson *et al.* 2003). Any cause of altered proprioception or altered load transfer through the SIJ or vertebral column may be a reason for development of joint surface changes in horses. Thus exercise designed to improve the neuromotor control around this musculoligamentous sling may be required for articular cartilage health in this area.

The cartilage of young horses seems likely to be better able to adapt to mechanical loads than those of older horses (Marlin & Nankervis 2003). Thus the age of the horse should be taken into consideration when designing an exercise rehabilitation programme when articular cartilage is damaged. The older horse may require more gentle exercise over an increased time, before it is prepared to return to sport, as the articular cartilage adapts more slowly.

In summary, specific exercise should be part of the multi-modal approach both within physiotherapy modalities and alongside the nutritional (Johnson *et al.* 2001; Bruyere *et al.* 2003) and veterinary modalities that are used to enhance articular cartilage, reduce pain and inflammation.

14.2.4 Muscle

Muscle injuries in the athletic horse range from fibre disruption to delayed-onset muscle soreness and

problems with neuromuscular control following injury or surgery.

Even though the jury is out on benefits of stretching muscle for injury prevention (Taylor *et al.* 1990; Cornwell *et al.* 2002; Herzog *et al.* 2003), combinations of stretching and strengthening may be of great importance in injury management. In the treatment of grade 2 hamstring injuries, the total time to return to full training was decreased in a group of human athletes exposed to an intensive stretching programme (Malliaropoulos *et al.* 2004). However, progressive general agility and trunk stabilisation exercises were found to be more effective than isolated hamstring stretching and strengthening in promoting both return to sport and reducing injury recurrence in athletes with acute hamstring strain (Sherry & Best 2004). Chronic groin strains in athletes responded more favourably to an 8–12-week strengthening programme, including resistive exercises for agonists and antagonists, proprioceptive training and trunk strengthening, than a programme of stretching and other passive modalities (Holmich *et al.* 1999). Active and passive stretching have been shown to be equally effective for increasing hip flexor range of motion, but the authors were unsure if the results were due to increase in range of motion of the agonist, or improved function of the antagonist muscle group (Winter *et al.* 2004). A systematic review of literature has found that stretching in humans who do not have a functionally significant contracture of musculature, has a convincing effect, but the lasting effect of intensive stretching programmes is still yet to be investigated (Harvey *et al.* 2002).

In animal studies, stretching and passive mobilisation to lacerated rat gastrocnemius muscle has been shown to have a strong antifibrotic, and muscle regeneration effect, especially when implemented 14 days after laceration (Hwang *et al.* 2006). Another study using rats showed that stretching did not prevent muscle shortening in immobilised soleus, but reduced muscle atrophy. Stretching also induced hypertrophic effects in control muscles (Coutinho *et al.* 2004).

It has been shown that increases in strength and an accompanying decrease in pain and disability occurred in women with chronic neck pain who performed intensive training for neck and shoulder musculature. The control group, who performed stretching and aerobic exercise to achieve only minor changes in subjective and functional measures, embarked on a progressive, high-intensity strength training during a second year of rehabilitation. This led to significant improvements in neck strength, and decrease in pain and disability, suggesting that a combination of strength training and stretching may be effective for management of chronic neck pain (Ylinen *et al.* 2006).

In addition to retraining the muscular system for strength and power changes in rehabilitation (Crewther *et al.* 2005), changes in the neuromotor or neuromechanical control are an important contribution to the overall outcome following injury, as well as to enhancing performance.

14.2.5 Neuromechanical control

Motor control of equine locomotion and performance may be likened to that in the human, as it also requires an integrated system that has sensory pathways (mechanoreceptors, proprioceptors, stretch receptors, thermoreceptors and nociceptors) to detect the status of the body; a control system to interpret the requirements of stability and motion and plan appropriate responses (central nervous system (CNS)); and the muscles to execute the highly tuned responses required (Hodges 2003). Consideration of all these elements may be the key to successful rehabilitation and performance enhancement. Although not yet reported in the horse, as it has been shown in the human spine that even after resolution of acute, first-episode low back pain, the multifidus muscle recovery is not automatic (Hides *et al.* 1996) (Chapter 4).

Motor control theories or systems

Two motor control theories or systems exist: the open loop system and the closed loop system.

The *open loop system* implies that movement occurs from a pre-planned pathway from the CNS without modification via sensory feedback. These movements are often ballistic and repetitious. Data from humans and animal studies with de-afferented limbs has shown that movement can occur which is almost indistinguishable from the limb that has normal sensory input; except for fine motor control tasks which appear clumsy, such as finger motion (Taub & Berman 1968; Hodges 2003). This system occurs when the motion and environmental constraints are predictable; 'an internal system of body dynamics' (Gurfinkel 1994). Owing to a lifetime of experience, the CNS plans in advance the muscle activity required to overcome the predicted movement effects on the body due to the known interaction between the internal and external forces (Massion 1992; Cresswell & Thorstensson 1994; Hodges & Richardson 1997a, b). Thus the CNS tightly couples the feed-forward responses with the predictable demands of control of the system (Hodges & Moseley 2003).

The *closed loop system* implies that when a movement command is generated the intended motion is compared with feedback regarding the body's status and the relationship with its environment. The motion is continually modulated, if the feedback differs from the command, the performance is corrected, hence sensory feedback is used to mould performance (Schmidt & Lee 1999). This modulator system of the body's segments, posture and overall functional performance encompasses multiple sensory systems; skin, muscle, joint and ligament receptors as well as visual and vestibular systems.

This closed-loop feedback or modulator system can be utilised by the animal physiotherapist very effectively during rehabilitation and performance enhancement in many forms, by tapping into the equine sensory modulation, which includes the mechanoreceptive and proprioceptive

systems. Clinically, this can be achieved by utilising sensory facilitation aids applied directly to the horse's skin or body, for example utilising taping techniques, proprioceptive aids such as light chains, stretch bandaging, and training aides, such as the Pessoa lunging system or long-reining techniques (Figure 14.1 a, b, c). These modalities are highly applicable to the horse as the skin is densely innervated with sensory nerves and the cutaneous trunci muscle system exists. For example horses are able to feel an insect walking across their back, and twitch their skin in a given area voluntarily! Clinically, and from a neuromotor control perspective, it is an extremely valuable clinical tool to utilise the closed loop to facilitate appropriate or change an existing trunk or limb motion in the equine rehabilitation and performance enhancement.



(a)



(b)

Figure 14.1 Proprioceptive facilitation techniques: (a) taping technique; (b) wrapping technique.



(c)

Figure 14.1 Proprioceptive facilitation techniques: (c) Theraband technique.

14.3 Stretching for injury prevention and rehabilitation

Stretching is often used for prevention of injury in pre- and post-exercise situations, and in the rehabilitation of injuries and conditions that affect soft tissue. There is conflicting evidence, however, regarding effects of stretching and lengthening of the musculotendinous unit in relation to improved performance and injury prevention (Marek *et al.* 2005).

14.3.1 Effects of stretching

The assumption that stretching lengthens muscles and extracapsular joint structures leading to increased musculoskeletal flexibility has been tested in a number of studies. Fukunaga & Kurokawa (1996) summarise the response of muscle tissue to stretch stimulus as elongation of the muscle fibre by addition of new sarcomeres serially to the ends of existing myofibrils. Woo *et al.* (1982) provide evidence for the lengthening response of tendons and ligaments to stretching. The connective tissue component of a muscle-tendon unit changes in response to exercise stimulus and can thus alter the elasticity of the unit. Stretching may be implicated if the goal of stretching before or after exercise is simply to lengthen the muscle-tendon unit and improve joint range of motion (Hampson *et al.* 2005).

It is agreed that there is increased compliance in the muscle-tendon unit *immediately* following either active or

passive stretching (Taylor *et al.* 1990; Cornwell *et al.* 2002; Herzog *et al.* 2003), but it is unclear how long the increased compliance of the tissue is maintained after stretching. It is suggested that four repetitions of a 30-second hold, passive stretch, is the optimal combination for most elongation in the muscle-tendon unit (Taylor *et al.* 1990).

A common belief is that post-exercise stretching may reduce or prevent delayed onset muscle soreness, however a systematic review of literature has found no evidence to support this belief, or that of a more rapid return to previous levels of performance (Herbert & Gabriel 2002; Cheung *et al.* 2003). Clinicians should be aware that the combination of heat and/or static stretching has been shown not to reduce soreness, swelling, or muscle damage (Jayaraman *et al.* 2004).

Pope *et al.* (2000) provide good evidence that pre-exercise stretching does not affect injury rate in the type of activities carried out by army recruits. Hennig & Podzielnny (1994), Nelson & Kokkonen (2001), Cornwell *et al.* (2002) and Herbert & Gabriel (2002) have all found insufficient evidence to support the use of stretching in relation to injury prevention, delayed-onset muscle soreness and performance. Another study using military recruits has shown that stretching may reduce the instances of muscle/tendon injury, but have no effect on prevention of bone and joint injury (Amako *et al.* 2003). However, care must be taken in extrapolating these conclusions to the equine elite athletic population, flat race horse or eventer, whose performance more closely reaches the limits of physiological capacity, as some of the studies used formerly sedentary people who rose to moderate fitness level over the course of the study.

Implications for stretching in the horse

It has been demonstrated that the type of activity performed by an athlete should be taken into consideration, as stretching before a jumping or lifting type activity, where maximal force and power output is critical, has been shown to be deleterious to performance (Hennig & Polzielnny 1994; Cornwell *et al.* 2002; Avela *et al.* 2004). Two mechanisms have been identified as contributing to this diminished performance; depression of muscle activation via neural mechanisms and a decrease in musculotendinous stiffness (Kokkonen *et al.* 1998; Cornwell *et al.* 2002). This has led to the better understanding of why conflict exists in the literature (Hampson *et al.* 2005). Cornwell *et al.* (2002) and Wilson *et al.* (1994), offer an equine athlete biomechanical research model, by applying what is known from human exercise physiology and sports science. Using these models, sporting activities can be broadly separated into two types of muscle demand; high intensity of stretch-shortening cycles (HSSC) and low intensity limited stretch-shortening cycles (LSSC) (Hampson *et al.* 2005).

An example of HSSC activity is the jumping activity of the show jumper, or eventer. In this activity, the horse

relies on a muscle–tendon unit that is compliant enough to store and release a high amount of elastic energy to enhance performance in the jumping and sprinting phases. This is equally important in landing from height as an insufficiently compliant muscle–tendon unit may not cope with the energy absorption demands placed on the structures. In the jumping effort, there is an eccentric preloading phase of the prime mover muscles coupled with elastic preloading of passive structures (potential energy) contributes to an increased power output (kinetic energy) in the concentric lifting phase of the jump. Obviously, a stiff non-compliant musculotendinous unit would be deleterious under these conditions and not only limit performance but contribute to a high injury risk. Consequently, the rationale for performance enhancement and injury prevention in this type of sport is to increase the compliance of the musculotendinous unit through controlled stretching (Hampson *et al.* 2005).

The LSSC activity is well represented by the endurance horse, where the locomotion is at a relatively slow, steady pace for an extended period – up to 25+ km. However, in the endurance horse the moderate to slow pace still utilises the extensor apparatus and biceps/lacertus mechanism in a somewhat preloaded manner, as in HSSC exercise. Biomechanically, during LSSC locomotive activity, most of the power generation is by active concentric muscle work, with force generated transferred to the moment arm most efficiently by a non-compliant muscle-tendon unit (Chapter 4). A more compliant muscle–tendon unit in this case would effectively reduce the amount of muscle work converted into external work. Clearly, a stretching programme to lengthen and increase the compliance of the musculotendinous unit would be deleterious to performance in these types of activities (Hampson *et al.* 2005).

Much of the research regarding the biomechanics of stretching has been carried out on laboratory animals; thus we may extrapolate findings to the equine athlete owing to the similarities in muscle and connective tissue physiology across quadruped species. The value of stretching in HSSC and LSSC situations is likely to be even more relevant than in the human, due to the cursorial locomotor anatomy of the horse which utilises passive structures to aid a more efficient gait (Stashak 2002). For example, in the galloping horse about 80% of the shoulder extensor moment has been attributed (Wilson 2003) to the biceps/lacertus fibrosis passive elastic storage mechanism (Chapter 4). Hence a very compliant and high energy-storing musculotendinous unit would be very beneficial to performance, in reducing energy consumption of gait, and would also assist in injury prevention (prevent overloading the tendon) in any HSSC activity (Hampson *et al.* 2005).

Injury risk of the activity

HSSC activities (jumping, sprinting) have a higher soft tissue injury rate than LSSC (steady jogging) activities (Witvrouw *et al.* 2004) due to the less predictable nature,

higher impulse loading, and increased soft tissue preloading in the lengthened position, combined with eccentric muscle activity, for the former. However an accurate assessment for potential injury rate for a particular activity can be determined only by well-designed epidemiology studies; for example in the Thoroughbred racing industry, specific injury patterns such as the high incidence of suspensory ligament injuries in the race horses (Brown *et al.* 2003). Such studies will equip the physiotherapist with information that may be used to assess the requirements for a stretching programme.

Types of stretching

Stretching techniques can be divided into four basic categories: ballistic, static, passive and proprioceptive neuromuscular facilitation (PNF). Ballistic stretching is characterised by repeated bouncing movements at the end of range and has been shown to be less beneficial and has been implicated as a causative factor in injury to the muscle–tendon unit (Stanish 1982). Static stretching refers to the subject stretching a muscle to just short of the point of pain and holding this position for 30–60 seconds. The technique suggests that the subject is in control of the stretch and it is therefore not realistic in the animal population (Hampson *et al.* 2005). Passive static stretching is performed by a partner, and is stretch applied by an external force. In the horse, a mild degree of muscle activity is often present because it is standing during treatment. Hence passive stretch may be seen as variation of the human hold–relax and contract–relax stretching technique. Clinically this form of stretching enables the physiotherapist to utilise contractile elements or enables eccentric loading of the musculotendinous unit very effectively. If the stretch is performed repeatedly through a full functional range of constant motion it would be termed a PNF technique, which uses reflex activation and inhibition (Stanish 1982). This may also be termed a neuromechanical mobilisation technique as the therapist is repeatedly also stretching the myofascial and neurological systems which can more closely mirror the sport-specific nature of the animal. Hence these stretching methods may prepare the animal's neuromuscular system for performance. Although true PNF is often the technique of choice for human athletes, it requires training and a very compliant horse.

14.3.2 Examples of stretches and sport-specific stretching

Show jumpers

Horses performing at high speeds and/or jumping are classic examples of HSSC activities. There are limited statistics in the literature concerning injury rates in horses performing in jumping events but Pinchbeck *et al.* (2004) report a high injury rate of 8.9% in a large study of hurdle and steeplechase events. Studies (Fukunaga & Kurokawa 1996; Cornwell *et al.* 2002) on human jumping efforts reveal an

interesting pattern: pre-jump stretching has a deleterious effect on a jump from a standing start but no effect on a lunge jump. The difference is that a lunge jump allows an eccentric pre-loading of the musculotendinous unit. As discussed earlier, the biomechanics of equine locomotion favours this type of energy production over direct muscle energy transmission. Although no biomechanical studies exist on the effects of pre-stretching on the equine jumping effort, it can be extrapolated by combining what we know of human jumping and equine locomotion biomechanics that pre-stretching would certainly not reduce the performance (Hampson *et al.* 2005). Given the extremely high incidence of tendon and ligament overloading injuries below the tarsus and carpus in equine athletes, there is a strong incentive for a very compliant musculotendinous and ligament system. The equine athlete not only has to generate sufficient energy to jump, but requires a compliant musculotendinous system to land safely. Hence, the use of stretching as a training and pre-event warm-up technique is highly recommended. The recommendation is strengthened in the presence of previous and existing muscle, tendon and ligament injury.

Dressage

Dressage requires very slow and powerful limb movements with the majority of the joint moments being produced by active muscle contraction (Chapter 4). In this situation a low compliant musculotendinous unit is considered desirable. Pre-dressage stretching with the goal of improving performance flexibility would therefore be counterproductive. If, however, the horse were carrying an injury to muscle, tendon or ligamentous tissue which was at risk of further injury, the small potential performance loss would be outweighed by the potential benefit in injury prevention (Hampson *et al.* 2005). In fact, dressage-based 'supplying exercises' are used in many forms of equitation to enhance and optimise performance as well as to prepare the musculoskeletal system for the sport.

Eventing horse

The jumping section of the one- or three-day event can be considered as being applicable to the show jumper above. The cross-country course involves jumping and is not a race, although it is necessary to complete the course within a given time limit. The straights between the jumps could certainly be considered to contain LSSC demands, given the moderate speed and the repetitive nature of the gait. However, given the unique locomotor biomechanics of the equine gait and the large role of the passive-energy storing mechanisms, there is a sound argument to exclude the cantering equine from the definition of a LSSC activity. Therefore, the stretching protocol for both sections is covered by that of the show jumper. The first section of the event, the dressage phase, certainly qualifies as an LSSC activity as mentioned above. However, to ensure that the horse was best prepared to complete the three phases intact,

most riders would risk a small potential performance loss in the dressage phase, if stretching were to be technically counterproductive. Stretching then, would probably be part of the everyday training programme, as for the show jumper (Hampson *et al.* 2005).

Race horses

It is possible that the muscles of these sprinting athletes may suffer repeated strain, which often goes unnoticed, undiagnosed or untreated because the animal cannot verbalise – with the only sign being a poorer performance and mild gait changes or subtle stiffness. These muscles work under HSSC conditions and undergo a high eccentric load, particularly the semitendinosus, semimembranosus middle gluteal and biceps femoris at the end of range. In this case, flexibility is certainly an advantage, particularly when the muscle–tendon unit is probably in a shortened state from previous injury. In terms of injury prevention during training and competition, it is likely that the race horse would benefit from an activity-based stretching programme during warm-up. It should also be noted that any incidence of muscle injury may also be a result of training error, or more specifically lack of training, and lack of appropriate warm-up.

Examples of stretches

Many riders and practitioners attempt to stretch the 'top line' or epaxial musculature of the horse. This is an active stretch combining both eccentric and concentric activity or a functional ballistic stretch. It utilises the horse's own biomechanical myofascial and ligamentous 'pulley' system via lengthening the nuchal ligament, thoracolumbar fascia and the complex myofascial attachments of longissimus. To be effective however, this form of functional stretching requires coordinated neuromotor control, as this would be an ineffective stretch if the hypaxial muscles were inactive. These primarily include the iliopsoas complex, rectus abdominus, subclavius and serratus ventralis to flex actively and dorsally the lumbosacral, thoracolumbar and cervicothoracic regions. It must be noted that in the horse, most of the epaxial, hypaxial and pelvic muscles are myofascially interconnected directly. Hence, stretching in the horse often requires a combination of limb and spinal positions to ensure the desired effect occurs, while monitoring for on-going cheating strategies the animal may adopt to avoid the posture. For example, Figures 14.2, 14.3 and 14.4 demonstrate variations that can be used to stretch the pelvic limb and thoracolumbar and lumbosacral complexes. Note that when using the 'slump' to stretch the thoracolumbar and pelvic complex, it is also very important to consider that this position also places the central and peripheral nervous system under tension.

Posterior pelvic limb muscles

To stretch the semimembranosus effectively the hock must be kept in flexion to wind up the calcaneal tendon or



Figure 14.2 Hindlimb stretch.

insertion, the limb needs then to be abducted to a greater degree than is shown in Figure 14.2. Biceps femoris would be stretched by adducting the limb from this position. It is important to note the cheating strategies this horse has implemented to prevent a true stretch of middle gluteal; the muscle is unloaded by pelvic rotation, hitching and thoracolumbar extension and raising of the head and neck. Because of the lumbar attachment of middle gluteal onto longissimus and the thoracolumbar fascia as far cranially as the first lumbar vertebrae, the thoracolumbar and cervical (due to longissimus cervicus) spine must be dorsally flexed and well as the limb (Figure 14.2).

Thoracolumbar vertebral dorsal flexion stretch

This utilises the rounding response via a noxious stimulus. This stretch, if performed repeatedly on a daily basis, may assist in preventing loss of spinal motion, notably in the facet joints, in all horses but especially those with an increased lordotic posture or conformation (Figure 14.3).



Figure 14.3 Rounding reflex.



Figure 14.4 Slump stretch.

Equine baited slump stretch (neuromechanical)

This affects many structures including nuchal and supra-spinous ligament, the entire epaxial myofascial system, thoracolumbar vertebral column and the neurological system (spinal cord, and components of the brachial and lumbosacral plexus and associated fascia) (Figure 14.4). To place neuromeningeal and myofascial structures on more stretch, unilateral hindlimb protraction may be added.

14.3.3 Summary of implications for rehabilitation of muscle injury in horses

Strengthening has a role in assisting muscle rehabilitation, as has been outlined in cases of both acute and chronic injury (Holmich *et al.* 1999; Sherry & Best 2004; Hwang *et al.* 2006). A great deal of literature has investigated the effects of various resistance training programmes on strength and power change in humans, yet the stimuli that effect muscle adaptation are still not fully understood (Crewther *et al.* 2005). This type of training, where combinations of sets and repetitions of isolated muscle groups is utilised, is not applicable to horses, as we cannot instruct the horse to perform a number of repetitions on an isolated group of muscles as we can do in humans. The evidence from Sherry & Best (2004) shows that global trunk strengthening and agility were more useful for return to performance than specific muscle group strengthening and stretching – this may be the most effective path in returning horses to activity following a muscle injury. There is some further evidence in human research that specific stabilisation exercise is effective in reducing pain and disability in chronic back pain, neck pain, cervicogenic headache and pelvic pain as well as reducing recurrence after acute back pain (Ferreira *et al.* 2006). It may also be the case for muscle-based injury, as regaining neuromuscular control after injury or surgery is necessary for the athlete wishing to return to competition (Lephart *et al.* 1997).

Should the desired effect of stretching be to improve muscle–tendon compliance in the horse during a bout of activity, then the stretching should be performed immediately before performance, as the lasting effect of stretching is unknown from the human studies. The type of activity also needs to be taken into account before implementing a stretching programme, if it is for injury prevention or enhanced performance (Hampson *et al.* 2005). Factors affecting length of time to hold a passive stretch in a horse are dictated by (a) extrapolation from human studies; (b) the type of stretch that is desired (active versus passive) – there is evidence that both types of stretch are useful in rehabilitation; (c) the length of time a stretch can be physically maintained on a horse by the operator. In the latter case, functional stretches may be of more benefit than isolating a muscle for stretch, for example, standing the horse on an uphill slope with the affected leg in retraction, if the aim is to stretch hip flexors.

In cases where there is moderate contracture of muscle, or potential adhesion formation in a muscle as a result of a laceration, then there is evidence that stretching has a role in enhancing muscle hypertrophy and reducing fibrosis (Coutinho *et al.* 2004; Hwang *et al.* 2006).

Despite some conflicting evidence regarding the role of stretching in rehabilitation, particularly in prevention of injury, the physiotherapist should realise that many techniques employed clinically affect the length of muscle, as well as other soft tissue. In our physiotherapy management of the horse, we may use stretching, either alone or in combination with other techniques, to change the range of motion of a segment or limb, with the goal of reducing pain or restriction of motion and/or enhancing performance. Such techniques may include joint mobilisation, neuromeningeal mobilisation, hold–relax technique, and even trigger points – this is not an exhaustive list. In the horse, we can also use the closed-loop feedback or modulator system, which will cause stretching of some structures whilst facilitating others.

To obtain any benefit when implementing a myofascial stretching programme, the individual horse and its sport-specific functional requirements must always be assessed. This should entail a detailed assessment of the biomechanical requirements of the activity, including the contribution of anatomical and neuromotor control components of the animal to the activity. Only then can it be determined if a stretching regime will in fact be beneficial to the performance of the activity, and if so, an accurate regime based on scientific principles can be formulated.

14.4 Assessment of the horse and rider unit

Total rehabilitation of the horse involves assessment and treatment of the horse–rider unit. Horse riding is unique in the world of sports because one of the members of the team is not human and the team must be in absolute harmony to execute even the simplest manoeuvres (Sorli 2000).

Gait in performance horses has been shown to be affected by type of rider (novice versus experienced) (Licka *et al.* 2004). The horse's performance is also affected by the way the rider contacts the horse and the ability of the rider to perform a part of the athletic team. A rider carrying a lower back problem may have difficulty sitting to a trot due to pain or discomfort. A rider with an ankle injury may have difficulty positioning the foot in the stirrup. More subtle movement dysfunction in the rider can cause the rider not only to look unbalanced, but can affect the way a horse moves. An example of the latter is a rider with a mechanical dysfunction of the pelvis causing increased contact through the right ischial tuberosity compared with the left. This will affect delivery of aids; i.e., what the horse is feeling through the epaxial muscles. A rider with poor scapular control, owing to shoulder girdle or cervical spine injury, may have difficulty maintaining position of the upper limb.

14.4.1 Role of equine physiotherapists in rider management

Existing injuries

The physiotherapist should manage existing injuries which result from the equestrian sport as well as concurrent injuries such as lower back pain, or other sporting injuries. Common injuries in equestrian sport are as follows:

- Head/face
- Upper limb
- Lower limb
- Trunk
- Other

(Williams & Ashby 1995; Watt & Finch 1996; Sorli 2000; Petridou *et al.* 2004).

The physiotherapist may need to apply appropriate physiotherapy modalities to maximise function and minimise pain so that the rider can return to their sport and ride to the best of their ability. If the equine physiotherapist does not possess the skills or experience required (such as manual therapy, experience in sports physiotherapy, management of conditions of the vertebral column, or neurological problems) then it is their role to refer to a practitioner who can help the rider.

Rider mechanical function

Rider mechanical function needs to be optimised to ensure the best horse–rider integration for optimal equestrian performance. The physiotherapist should assess:

1. *The mounted rider.* Observe the rider from the back side and front, when seated on the horse. Look at position of legs, pelvis, trunk, shoulder, neck, arms and head and note asymmetries of posture or muscle bulk (Figure 14.5).
Question the rider regarding their awareness of contact with the saddle and horse, and symmetry of their



Figure 14.5 Assessment of rider.

own posture. Compare the posture of the horse that you have observed before the rider mounted, to after the rider mounted.

Repeat the same observations when the rider is in motion with the horse moving through various gaits and transitions (Figure 14.6).



Figure 14.6 Assessment of rider in motion.

2. *The rider's 'on ground' biomechanics.* Assess the rider standing on level ground from behind, front and the side and note postural asymmetries and anomalies (including muscle bulk). Compare these findings to what you have seen when the rider is mounted (statically and riding). If the same conditions you see 'on ground' are present when mounted, these may need to be addressed before/concurrently with physiotherapy management of the horse. If the postural anomalies or asymmetries of the rider are more marked when the rider is mounted (particularly in motion) then it may be more pertinent to manage existing biomechanical or musculoskeletal problems in the horse. Then, reassess the horse–rider unit to see if the rider problems remain.

The total assessment and treatment of horse and rider may not be able to be completed in one session – it is at the discretion of the physiotherapist as to how long is spent in one session. Clinical reasoning may also dictate that only one 'problem' (either horse OR rider) is addressed at once.

14.4.2 Contact areas

Contact areas to consider when assessing rider are:

- Caudal bony landmarks of the pelvis
- Pelvic floor
- Medial surfaces of lower limbs
- Feet
- Hands via the bit
- Voice
- Mood/temperament
- Other artificial aids

Rider aids are given via subtle change in weight shift or pressure from the rider's seat, positioning and change in pressure via the rider's lower limbs and hands through the bit.

The function and position of the lumbopelvic region of the rider is particularly important to assess when the contact areas with the horse are considered. It is known that the pelvic girdle is important for transfer of forces between legs and trunk (Lee & Vleeming 2000). The lumbopelvic local system stabilises the joints of the spine and pelvis in preparation or response to external loads (Hodges & Richardson 1996; Hungerford *et al.* 2003). Control of the upper limb and trunk is affected by motor control issues which originate in the lumbopelvic region. It is important that this system functions well in the rider for trunk rotation and stability, head and neck position and motion, and subtle positioning of hands and arms.

Physiotherapists can affect the bony alignment and soft tissue contact of rider contact areas (Huysmans *et al.* 2005), and optimise motor control via coordination of activation of muscles and fascia, neural patterning and awareness (Lee & Vleeming 2000). Physiotherapists also have a role in minimising rider pain and discomfort.

14.4.3 Conclusion

When providing physiotherapy assessment and treatment of the horse–rider unit, it is important to be able to utilise your physiotherapy skills to restore anatomical position and function of musculoskeletal structures in the rider in addition to treating the horse. If the animal physiotherapist does not have the skills or experience to manage the rider musculoskeletal/neurological conditions, then he/she needs to refer the rider to a practitioner who has the skills/equipment to do so.

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