Soft tissue lesions: muscle, tendon and ligament

Soft tissue injuries are an often underdiagnosed source of canine lameness. Sporting and working dogs may be particularly at risk of suffering acute traumatic muscle strains, ligamentous sprains or chronic overuse degenerative tendinosis lesions resulting from poor healing of repetitive strain injuries. Less conditioned animalsmay alsobe at riskwhenperforming infrequent burst activities or endurance tasks, much like the phenomenon known as weekend warrior syndrome in humans. Physical therapy skills and knowledge lend the ability to systematically assess, diagnose and conservatively treat soft tissue injuries in the canine patient.

Muscle strains may be caused by poor flexibility, inadequate warm-up, fatigue, sudden forceful contraction or forced extension/flexion, strength imbalances, intense interval training, insufficient breaks and overtraining (Steiss 2002). The potential for certain muscles to be strained or torn is greater for somemuscles than others.Multi-jointmuscles are those that cross two ormore joints and are at greatest risk for strain because they can be stretched by the movement at more than one joint. A strain may also occur when high forces are put through tendons and muscles, as occurs during eccentric muscle contractions (where amuscle is contracted during a stretch), when forces are applied quickly and obliquely, or during an explosive burst of movement. Muscle strains most often affect the muscle origin or insertion, typically at the musculotendinous and teno-osseous junctions, but can occur within the muscle belly as well.

Tendon injuries may be secondary to acute trauma or repetitive loading. The designation of tendon pain as ‘tendonitis’ is often a misnomer as it implies an inflammatory process. Tendinopathy is a better generic descriptor that can be used to include all pathologies that arise in and around tendons (i.e. tendonitis, tendinosis or paratenonitis). There is a lack of good-quality histological data from symptomatic tendon disorders of short duration to unequivocally state that tendon lesions are actually inflammatory in nature. Marr et al. (1993) described an inflammatory reaction in superficial digital flexor tendon injuries in horses, but only within the first 2 weeks. Other animal models suggest that an inflammatory reaction is present in acute situations but that a degenerative process soon supersedes this (Rees et al. 2006). Classic inflammatory changes are not frequently seen in chronic athletic tendon conditions, and it has been suggested that the time at which the tendon becomes symptomatic for pain does not coincidewith onset of pathology. On a practical note, in clinical practice most tendinopathies are chronic (tendinosis lesions) by the time the patient (or animal owner) seeks medical attention. So perhaps, the clinician should place only minimal, if any, focus on inflammation for tendon pain conditions. Paratenonitis occurs when a tendon rubs over a bony protuberance and is alternatively known as peritendinitis, tenosynovitis and tenovaginitis (Khan et al. 1999). It is clinically characterised by acute oedema and hyperaemia of the paratenon with infiltration of inflammatory cells and within hours or days, fibrinous exudate fills the tendon sheath. Despite these results, pathologists and scientists in this field argue that inflammation of the paratenon is a rare occurrence.

Tendinosis describes intratendinous degeneration without clinical or histological signs of an inflammatory response. This form of tendinopathy is typically considered an overuse injury that involves excessive loading of the tendons, frequent cumulative microtrauma and subsequent mechanical breakdown of the loaded tendon. In order to mediate the repair process, local tenocytes must maintain a fine balance between extracellular matrix network production and degradation, and unless fatigue damage is actively repaired, tendons will weaken and eventually rupture. In humans, tendinosis is a common problem that is characterised by persistent, localised, activityrelated pain and swelling associated with common Achilles, patellar and supraspinatus tendons. The histological appearance of tendinosis is that of collagen disorientation, disorganisation and fibre separation with an increase in mucoid ground substance, increased prominence of cells and vascular spaces with or without neovascularisation and focal necrosis or calcification. Additionally, affected tendons are characterised by fibrocartilaginous metaplasia of tenocytes and hypercellularity. On visual inspection of affected portions of a tendon, they are lacking their normal glistening-white appearance and have been reported to have a grey-brown or pink-yellow appearance. Pain in these cases can be attributable to both mechanical and biochemical factors but not necessarily due to inflammation alone. Inflammatory lesions are infrequent andmost commonly associated with partial ruptures (Khan et al. 1999). It has been hypothesised that the neovascularisation in the regain of nerve endings accounts for the pain and swelling in the tendon. Barring a direct trauma or muscle strain, it is more likely that a soft tissue injury is a tendinosis lesion, and the practitioner should be aware of the pathology. 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 physiologicalmotion (i.e. hyperextension of the carpus or stifle). However, overuse degenerative lesionsmay also occur in ligamentous structures such as the cranial cruciate ligament in dogs.