A look at chronic pain in dogs

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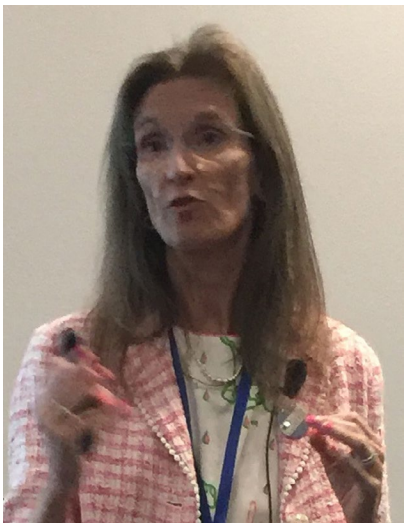

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ABSTRACT: Chronic pain can be found in all mammals that have a nociceptive pathway. It is defined as pain that extends beyond the normal time of healing. This article deals with chronic pain that affects canine patients. Chronic pain and depression have both been shown to occur in mammals. Chronic pain can be divided into inflammatory pain, non-osteoarthritis–non-malignant pain and cancer pain. The pet owner is key in the recognition of chronic pain in dogs. There are validated chronic pain scales available for dogs. The article will discuss various examples of chronic pain that affects dogs and ways to alleviate the effects of this pain in selected disease processes.

Introduction

Chronic pain presents a challenge to any nurse, whether veterinary or human. Problems arise not only from the diagnosis itself, but from the number of species involved and the situations in which they live and function (pets, working animals, research animals and zoo animals). Veterinary nurses are presented with the difficulty of recognising and assessing chronic pain states in their patients on a daily basis. This article will deal specifically with canine chronic pain and conditions that involve chronic pain. It is beyond the scope of this paper to talk about the neurophysiology of pain.

What is chronic pain?

Chronic pain is generally described in human medicine as pain that persists beyond the normal time of healing, or as persistent pain caused by conditions where healing has not occurred or which remit and then recur (Mathews et al., 2014). Chronic pain may be considered as a disease state. The 2015 AAHA/AAFP Pain Management Guidelines for Dogs and Cats states, "chronic pain is defined as that which exists beyond the expected duration associated with acute pain" (Epstein et al., 2015), but suggests that therapy should be addressed to the underlying cause of pain rather than on arbitrary labels based on duration (Woolf, 2010) (see **Textbox 1**). Neuropathic pain is defined as pain caused or initiated by a primary lesion, injury or dysfunction in the peripheral nervous system or central nervous system (Mathews et al., 2014).

What are the consequences of chronic pain?

Chronic pain and clinical depression are quite clearly comorbid conditions. This should come as no surprise when considering that the same pathways and neurotransmitters are involved. A search on PubMed found studies have been done about clinical depression behaviour in animals. Most studies have been done in rats and mice (Heinzmann et al., 2014). In animals (again rats), pain-related depression of behaviour can similarly be assessed by measures of locomotor activity, feeding or social interactions with conspecifics (National Research Council, 2009, 2011; Negus, 2013). Dogs (Tontodonati, Fasdelli, Moscardo, Giarola, & Dorigatti, 2007; Rutherford et al., 2012; Chalkley & Bouljihad, 2014), cats (Lee et al., 2011) and rhesus macaques (Hennessy, McCowan, Jiang, & Capitanio, 2014; Qin et al., 2015) have been studied with similar outcomes. We should accept

that our patients are little different from their human counterparts in this regard, and that it is possible that chronic pain contributes negatively to their overall well-being far beyond the sensation and discomfort itself. The ultimate consequence of under-recognised and/or under-managed chronic pain and its impact on mobility, abilities and overall quality of life is the highest price: death or, termed appropriately, humane euthanasia. The data support this conclusion. In a life-long study of labradors, fully 2/3 of the study population was euthanised due to orthopaedic conditions (the others were cancer and other medical issues) (Lawler et al., 2005). This is consistent with a study documenting that one of the two most common reasons for euthanasia in military working dogs was degenerative joint disease (Moore, Burkman, Carter, & Peterson, 2001) and the other was cancer.

Textbox 1. How does pain become persistent?

a. Ongoing inflammation – anywhere
b. Nerve injury, whether a single original insult, or chronic
c. Maladaptive, neuropathic changes in the nervous system, secondary to (a) or (b), or can be spontaneous over time or post-operative

Chronic pain groups

1. Chronic inflammatory (especially but not limited to: osteoarthritis (OA), but also chronic otitis, chronic pancreatitis, and all the other chronic medical "itis" conditions)
2. Non-OA, non-malignant pain
3. Cancer pain

Cancer patients tend to have more serious health restrictions and are prescribed more opioids and other medications than patients with chronic non-malignant pain. Cancer pain can originate from invasion of the tumour into tissues densely innervated by primary afferent neurons (e.g. pleura, peritoneum) or directly into a peripheral nerve plexus. In the latter, neuropathic symptoms may be predominant.

Chronic pain recognition in dogs

The behavioural changes associated with chronic pain may develop gradually and may be subtle, so that they can only be detected by someone very familiar with the animal (usually the owner).

The pet owner may see:

- Increasingly diminished function and mobility that indicate progressive disability
- Diminished exercise tolerance and general activity (see **Figure 1**)
- Difficulty standing, walking, taking stairs, jumping or getting up (see **Figure 2**)
- Decreased grooming
- Changes in urination or defaecation habits (see **Figure 3**)

At present, a few tools have been described to evaluate chronic pain in dogs and these have provided information about the range of alterations in the demeanour, mood and behaviour of dogs because of chronic pain (see **Textbox 2**). Broadly, these can be categorised as follows (Mathews et al., 2014):

- Vitality and mobility – how energetic, happy, active/lethargic, contented, playful is the dog; ease of lying, sitting, jumping up, tolerance to exercise
- Mood and demeanour including states of alertness, anxiety – for example, whether it is withdrawn, sad, dull, confident – its playfulness and sociability
- Levels of distress – e.g. vocalization (moaning, groaning), demeanour (e.g. depressed) and response to other dogs and humans
- Indicators of pain – e.g. comfort levels, stiffness, lameness

Textbox 2. Signs of chronic pain in dogs (Hielm-Björkman, 2014)

Positive behaviours reduced with chronic pain

- Decreased socialisation/play with human family
- Decreased socialisation/play with other dogs
- Decreased movement (quality and quantity)
- Decreased interest in hygiene/grooming
- Decreased tail wagging
- Hypo- or anorexic
- Decreased curiosity

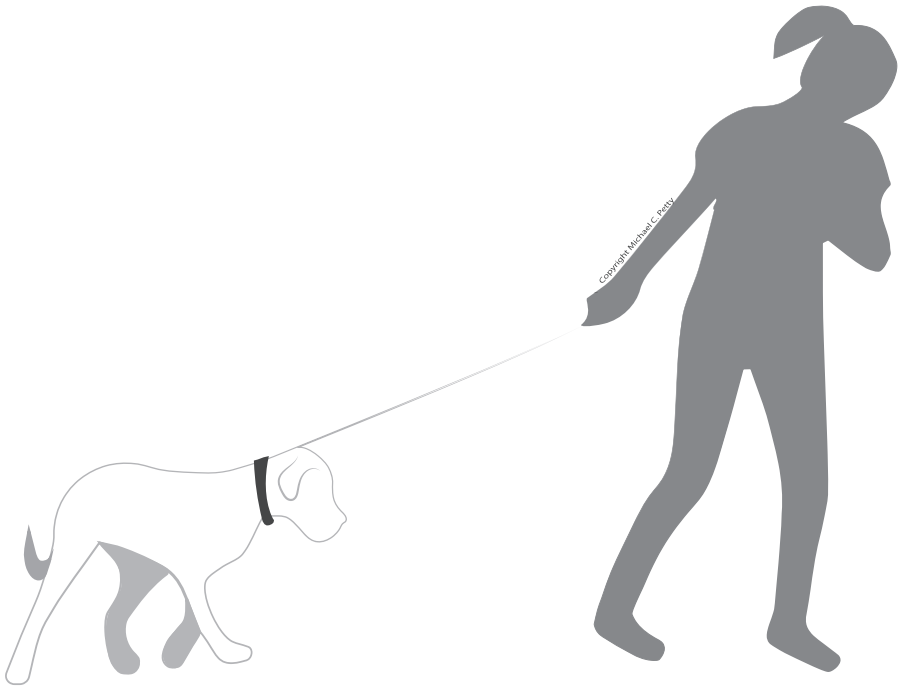

Figure 1: A dog that refuses to walk because of pain (with permission from Dr Petty)

Negative behaviours more frequent with chronic pain

- Aggression towards humans and/or other dogs
- More dependent on owner, jealous, "clingy"
- Sleeping more
- Does not come up to greet owner
- Fearful
- Guarding behaviour, guards body parts
- Biting painful areas
- Licking painful areas or dorsal aspects of front limbs
- Sudden, excessive scratching
- Sudden, excessive negative reaction (compulsive behaviour)
- Under- or overactive

Abnormal posture or movement seen with chronic pain

- Reluctance to move (walk, trot, gallop, jump)
- Inability to turn in one or both directions
- Hind legs tucked under abdomen
- Tail between hind legs
- Ears back
- Restlessness, wandering, circling
- Rigid posture and gait
- Sitting or lying down in the middle of walks
- Head hanging; will not lift or turn head (neck pain)
- Praying position (abdominal pain)
- Decreased weight bearing (limb pain)
- Sitting abnormally (e.g. knee out in stifle pain)
- Trembling or shaking

Mental and physiological behaviour

- Depressed, sad, and/or anxious demeanour
- Visible white sclera around the iris (not always pain, some breeds show this all the time)
- Panting or tachypnea or tachycardia without exercise

Other

- No change in behaviour
- Decreased vocalisation and/or quiet whining or whimpering
- Increased vocalisation including screaming or howling with breakthrough pain or manipulation of painful area
- Allodynia
- Hyperesthesia or hyperalgesia

What are hindrances to chronic pain management for animals?

A lack of appreciation that many chronic disease processes and cancers are associated with significant pain. An inability to assess chronic pain in dogs and cats. A lack of knowledge of drugs, drug therapy and other pain-relieving techniques. A lack of communication with clients and lack of involvement of clients in the assessment and treatment phases. And finally, an under-use of nursing staff for assessment and re-evaluation of pain in hospitalised patients (Lascelles & Gaynor, 2011).

What are some examples of chronic pain scales that can be used for the veterinary team and for clients?

They are quite different than the various pain scales and tools one would use to assess acute, post-surgical pain. The validated chronic pain clinical measurement instruments (CMIs) in veterinary medicine include:

Helsinki Chronic Pain Index (Hielm-Björkman, Rita, & Tulamo, 2009)

Canine Brief Pain Inventory (Brown, Boston, Coyne, & Farrar, 2008)

Liverpool Osteoarthritis in Dogs (Walton, Cowderoy, Lascelles, & Innes, 2013)

Cincinnati Orthopedic Disability Index (Gingerich & Strobel, 2003)

Health-Related Quality of Life (Reid, Wiseman-Orr, Scott, & Nolan, 2013)

At one time, the most objective pain assessment method currently available for dogs with chronic limb pain would be force place, i.e. ground reaction forces, using force platform analyses. A normal healthy dog puts ~60% of its weight on the forelimbs and ~40% on the hind limbs. Force platform analysis assumes that a dog will put less weight on a limb if it is painful. Force platform analysis has long been considered the gold standard for evaluation of canine lameness. This method can give rise to 50% variability in outcomes of control groups, never mind breed and conformation variabilities on

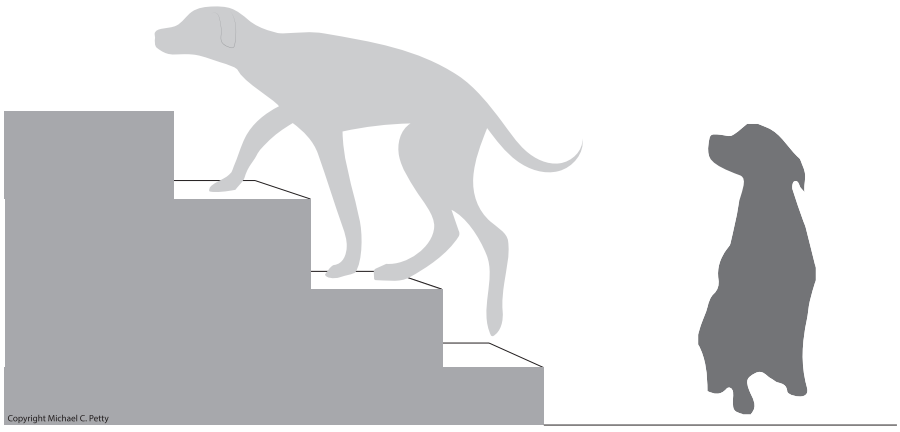

Figure 2. A dog that is reluctant to go up the stairs because of painful mobility issues (with permission from Dr Petty)

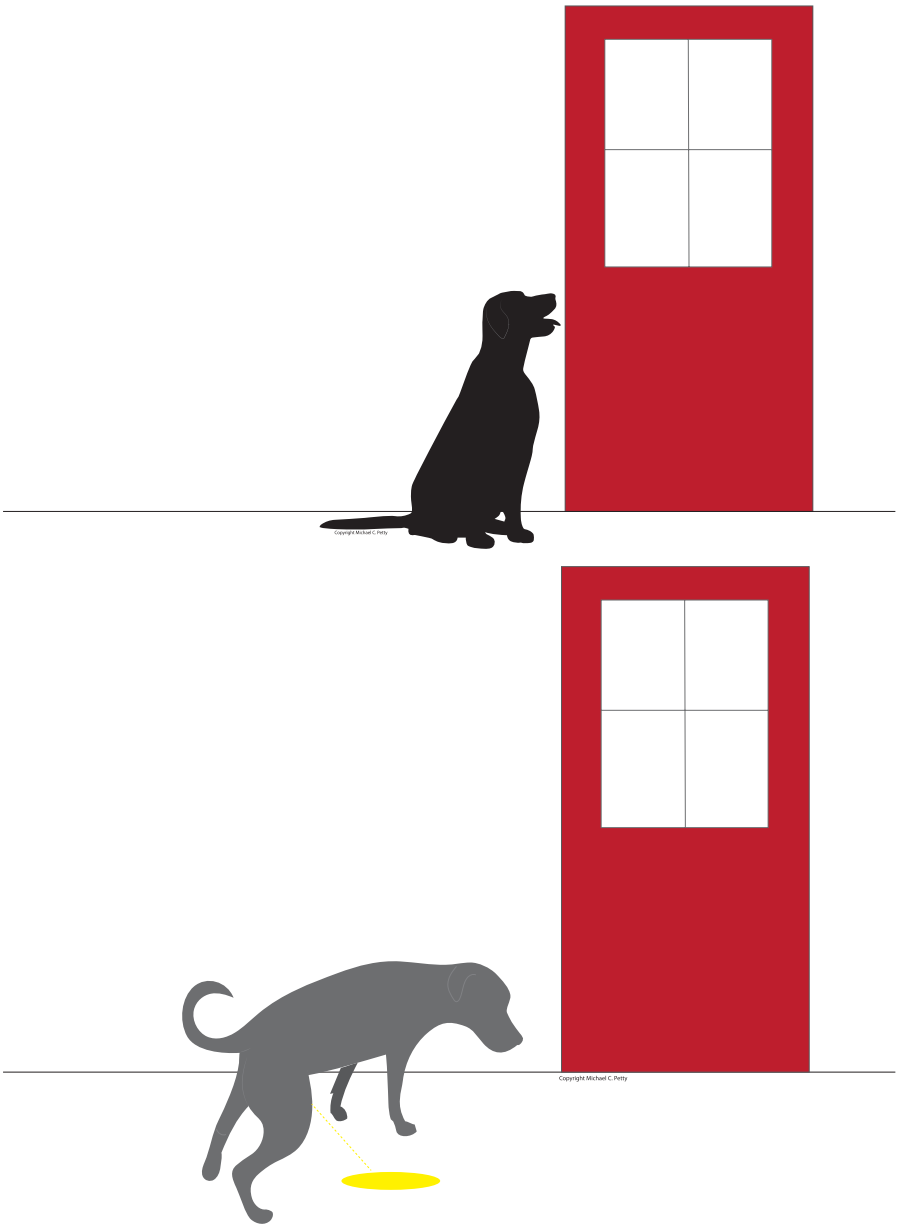

Figure 3. A dog that is unable to control urination and soils the house (with permission from Dr Petty)

top of that, and are only reliable when a single joint in one leg is painful (Moreau, Lussier, Ballaz, & Troncy, 2014). The limitations are significant enough that the US the Food and Drug Administration no longer accepts force platform analysis as an outcome measure for pain medication efficacy or pivotal studies in the USA. Behaviour-based CMIs are generally considered among the most reliable tools. What about in the future? Wearable activity monitors and accelerometers might provide the next instrument to provide objective data, and indeed there is work in dogs (Brown, Boston, & Farrar, 2010) suggesting responsiveness validity (picking up increased activities in degenerative joint disease (DJD) patients after initiating non-steroidal anti-inflammatory drugs (NSAIDs)).

Canine chronic pain conditions

Degenerative joint disease/ osteoarthritis

First a quick definition: although the terms degenerative joint disease (DJD) and osteoarthritis (OA) are commonly used interchangeably in veterinary medicine, a distinction has been made between the two (Clarke et al., 2005). DJD is a general term used to describe any degenerative change in a synovial, cartilaginous, or fibrous articulation in the skeleton. OA, however, is a pathological change of a diarthrodial synovial articulation and includes deterioration of articular cartilage, osteophyte formation, bone remodelling, soft tissue changes and low-grade, non-purulent inflammation.

The clinical signs of OA are similar regardless of whether the disorder is primary or secondary. The onset is often insidious but progressive. Early during the disease, the animal may sporadically be reluctant to perform previous tasks or activities, i.e. jumping into the car. In the next stage, a lameness or stiffness occurs following periods of excess activity or over-exertion. These signs often disappear after several days of rest. As the degeneration progresses, the stiffness and lameness may be most pronounced following periods of rest. The pet typically "warms out" of the signs with activity. Any cold or damp weather will increase the severity and duration of the symptoms. Continuous stiffness, lameness and chronic pain typify the final stage producing an irritable, reclusive and restless pet (see **Textbox 3**).

Textbox 3. Signs of canine OA

- Stiffness after exercise
- Wasting away of muscle – atrophy
- Limited movement
- Pain
- Joint swelling
- Trouble getting up, laying down, walking, climbing stairs or jumping
- A grating sound in a joint
- Altered behaviour

The objectives of treatment for OA are: reduce pain and discomfort, decrease clinical signs, slow the progression of the disease, promote the repair of damaged tissue and improve the quality of life (Fox & Millis, 2010). OA is the most common orthopaedic problem in dogs (see **Textbox 4**), encompassing about 20% of the population across all age ranges.

Textbox 4. Identify patients at risk of OA

- All large breed dogs over 6 years
- All giant breed dogs over 4 years
- All dogs with congenital issues – hip dysplasia, elbow dysplasia, etc.
- All dogs with history of past orthopaedic injury, especially those involving a joint
- All athletes

The incidence increases with age. Multimodal therapy encompasses not only drugs which act at various levels of the nociceptive pathways, but in the case of OA, multiple non-drug therapies (see **Textbox 5**).

Textbox 5. Aids for arthritic dogs

- Slip-free flooring
- Soft bedding
- Ramps instead of steps
- A warm, dry environment
- Help with grooming
- Help 'Em Up™ harness or assistive devices
- Orthotics and prosthetics

Management of OA

The top modalities, from an evidence-based perspective, for the management of canine OA (focus on medical rather than surgical management) are:

1. Weight optimisation – near or at the top of the list, and can certainly be said to be the number 1 preventative measure for the progression of OA in the dog (Lawler et al., 2005; Marshall, Hazewinkel, & Mullen, 2010; Kirkby & Lewis, 2012). The reality that 35–40% of adult pets and 50% of pets over age 7 are overweight or obese, making it the most common medical disorder of companion animals and by extension a major welfare concern for them (Burns, 2013; Raffan, 2013).
2. Symptom-modifying drugs: NSAIDS – if weight optimisation can be said to be the number 1 preventative measure for the progression of OA in dogs, and a crucial element to the treatment of it, the modality that is said to offer the most immediately, predictably and observably effective improvement in canine OA is the NSAID class of drugs. Through multiple systematic reviews in recent years we have a very good grasp on the highest, wisest, safest use of NSAID in dogs (Innes, Clayton, & Lascelles, 2010; KuKanich, Bidgood, & Knesl, 2012; Monteiro-Steagall, Steagall, & Lascelles, 2013).

   Amantadine – an NMDA-receptor antagonist (among other effects). This is the only medication for which we have evidence as an adjunct to NSAID for canine OA given at 3 mg/kg BID for 3 weeks (Lascelles et al., 2008).

   Gabapentin – effective against the sensitisation component of chronic joint inflammation. Start at 5–10 mg/kg PO q12–24 h and in some dogs working up to a target dose of 20+ mg/kg. Initial higher doses can be given at bedtime for 7–14 days.

   Tramadol – in dogs, it has a very short half-life (1.7 h), low plasma levels and negligible amounts of the opioid M1 metabolite are produced. Convincing evidence for a pain-modifying effect of oral tramadol, however, remains elusive, and already low plasma levels quickly diminish with sequential administration (Malek et al., 2012). Despite pharmacokinetic data demonstrating limited production of the M1 metabolite in dogs following parenteral administration of tramadol (McMillan et al., 2008), there appearsto be some evidence that injectable tramadol does provide reasonable analgesia in dogs immediately after surgery, which may indicate that the M1 metabolite is of less importance in providing analgesia in dogs than it is in people (Murrell & Flaherty, 2014). Current data suggest that it is not appropriate to rely on oral tramadol as a sole means of providing analgesia in dogs and that, when it is used, frequent dosing, probably at doses of around 4 mg/kg every 8 hours, is necessary to achieve therapeutic concentrations (Murrell & Flaherty, 2014).
3. Structure-modifying drugs – the main example includes pentosan polysulfate (Cartrophen; Biopharm Australia), which is a licensed product made from beech and similar in structure to heparin (Pettitt & German, 2015). It is produced by Biopharm Australia. The dose is 3 mg/kg PPN (pentosan polysulfate sodium) given subcutaneously every 5–7 days for four treatments. Subsequent single booster shots are usually given every 3–6 months.

   Injectable polysulfated glycosaminogylcan; in the USA, Adequan®, an FDA-approved chondroprotective, disease-modifying osteoarthritis drug; 2–5 mg/kg IM every 3–5 days for 3 weeks, twice weekly for 4 weeks. Following initial induction of 2 injections per week for 4 weeks, booster dosing every 2–4 weeks is based on patient need. Clients are instructed on administration of subcutaneous injections at home and Adequan® is dispensed.

   Omega 3 fatty acid diets – (Hill's j/d®) Science supports that the eicosapentaenoic acid (EPA) fraction of omega 3 fatty acids have both anti-inflammatory and anti-cartilage degradation effects. Diets are the only way to assure the delivery of adequate levels (EPA: 50–100 mg/kg).
4. Non-pharmacologic treatments – physical rehabilitation, myofascial trigger point therapy, acupuncture, shockwave therapy, thermal

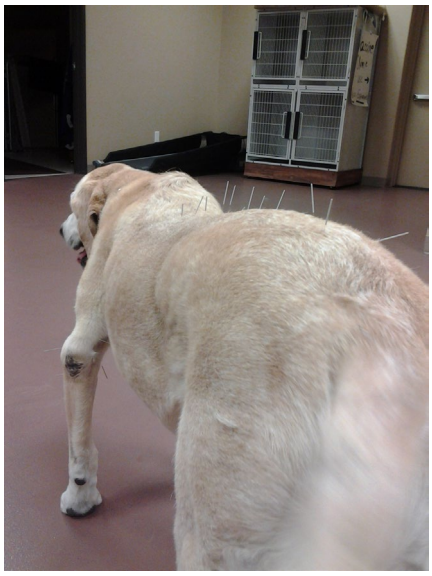

Figure 4. Coda is an 11-year-old labrador that has severe arthritis of the left elbow. Dr Douglas Stramel has done two rounds of stem cell therapy and is performing monthly maintenance of acupuncture. They also acupuncture the lower spine

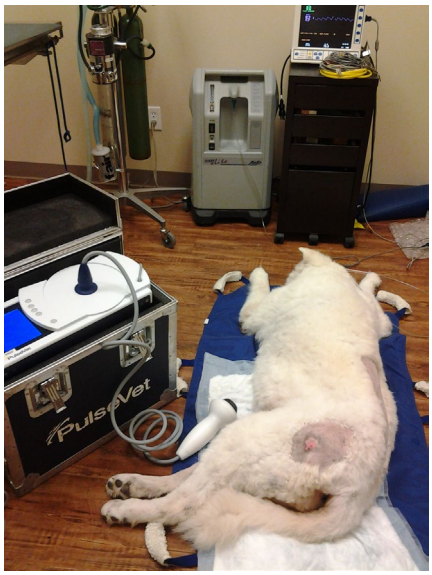

Figure 5. A 13-year-old Kuvasz that was getting ready to be euthanised because it could not walk. Dr Douglas Stramel has been doing hyaluronic acid joint injections into both stifles, and shockwave therapy for both hips and lower spine. Additionally, he has performed weekly acupuncture and laser sessions. Kudda can now get up and walk around the back yard

modifications and environmental modifications are all tools that can be added to the treatment of chronic pain (see **Figures 4** and **5**).

Non-OA, non-malignant pain

Neuropathic pain requires several classes of medications and procedures as it cannot be adequately managed with a single pharmacological or non-pharmacological therapy (Mathews, 2008).

Intervertebral disc disease

Intervertebral disc disease can occur in any area of the spinal cord. Dachshunds appear predisposed. Young to middle-aged dogs are most commonly affected. Dogs less than 1 year of age rarely have intervertebral disc disease (Fingeroth & Thomas, 2015). Geriatric dogs can also be affected. It is observed most frequently in the chondrodystrophoid breeds, especially the dachshund, but also: Pekingese, French bulldog, beagle, American cocker, shih tzu and lhasa apso. Non-chondrodystrophoid dogs (labrador retrievers, German shepherd dogs, etc.) usually present between 5 and 12 years of age with chronic, Hansen Type 2-disc herniation (Fingeroth & Thomas, 2015).

Post-amputation

Chronic residual limb pain and phantom limb pain is a known syndrome in human patients, but may also occur in veterinary patients. The cause may be attributable to peripheral sensitisation as a result of spontaneous activity from sprouting regenerating nerve endings or neuroma formation that gives rise to secondary changes in otherwise silenced small dorsal root ganglia cells, central sensitisation, or cortical reorganisation (Mathews, 2008).

Ocular conditions

Ocular pain can be quite marked. Keratitis, iritis and glaucoma can produce a deep, dull pain with inflammation. Corneal lesions and foreign bodies can produce sharp, acute pain (Fox, 2010). The cornea has the highest concentration of free nerve endings in the body and can therefore be particularly painful (Kurita, Ulrich, Jensen, Werner, & Sjøgren, 2012; Marfurt, Murphy, & Florczak, 2001).

Otic conditions

A very common ailment of dogs seen in a veterinary practice is ear disease. From mild erythema to severe otitis media, approximately 15–20% of all canine patients suffer with it. In humid climates, the incidence of otitis in dogs approaches 50%. Chronic inflammation may lead from hyperplasia to dysplasia (Fox, 2010). Infections, yeasts, improper treatments and over-treatment exacerbate matters.

Neuropathic pain associated with trauma: accidental and surgical

When repairing fractures, or handling neural tissues that may be inadvertently incorporated in the surgical procedure, it is prudent to identify neural tissue and ensure that this is not incorporated in ligatures at any surgical site to prevent the potential for the development of neuropathic pain that may be difficult to identify and treat later. Should transection or excessive manipulation or traction of neural tissue be necessary, application of a lidocaine and bupivacaine mixture to neural tissue at least 5 min before handling is recommended (Mathews, 2008).

Pelvic fractures occur with some frequency in veterinary medicine. Pelvic fractures and repair may result in nerve injury to the femoral nerve and cauda equina. The cauda equina lies within the lumbosacral canal and is composed of the seventh lumbar (L7), sacral and coccygeal nerve roots. Injury of these roots causes deficits of sciatic, pudendal, pelvic, perineal and caudal rectal nerve function. Motor dysfunction is readily recognised in these injuries in veterinary patients (Mathews, 2008).

If it is trauma and/or gross nerve injury of any kind, whether external such as Hit by Car, or internal such as IVDD or surgical, the veterinary team's number 1 job is prevention through a well-planned, aggressive, peri-operative, multimodal protocol which makes use not only of the standard modalities (NSAIDs, opioids) but also locoregional anaesthetics and medications designed specifically to address and mitigate central sensitisation, such as subanaesthetic ketamine CRI (Constant Rate Infusion) and gabapentin.

Cancer pain

Pain in cancer is produced by pressure on, or chemical stimulation of, nociceptors (nociceptive pain), or it may be caused by damage or illness affecting the nerve fibres themselves (neuropathic pain). Between 40% and 80% of patients with cancer pain experience neuropathic pain (Kurita et al., 2012). Cancer pain has varying degrees of severity that is dependent on duration, location and type of cancer. Inflammation due to tumour necrosis or direct pressure causes pain. Pain may originate from nerve root compression, from muscle spasms around the lesions or directly from lesions, or from tissue that has been infiltrated. Most patients with cancer suffer pain to some degree. Some cancers such as lymphomas and leukaemia have a lower incidence of pain suffering in humans. However, even in these, the pain can be excruciating (Mathews et al., 2014). An example would include painful osteosarcoma, or any bone metastasis, simultaneously or not with OA in a dog.

Other examples include: large mast cell tumour with waxing and waning degranulation in a dog; late-stage nasal adenocarcinoma causing local destruction, invading the orbit and penetrating the cribriform plate and the cranial vault in a dog; pancreatic carcinoma with peritoneal carcinomatosis in a dog; very large hepatic tumour causing capsular distension in a dog; inflammatory mammary carcinoma with lymphatic invasion in a dog (Lucroy, 2013) (see **Textbox 6**). Cancers that induce osteolysis are most obviously painful processes. The animal diagnosed with appendicular osteosarcoma can present for excruciating pain resistant to even the most potent analgesics. Intense cancer pain may be attributed to stretching the capsule of certain visceral organs, or by causing flow obstruction (biliary, urinary or gastrointestinal tracts) (Mathews, 2008). Additionally, the host's immune response to the cancer itself may generate pain as an epiphenomenon through the liberation of inflammatory cytokines.

Textbox 6. Tumours associated with pain (Lucroy, 2013)

1. Primary bone tumours such as osteosarcoma, fibrosarcoma, chondrosarcoma or hemangiosarcoma.
2. Tumours metastatic to bone, such as prostate carcinoma or mammary carcinoma.
3. Multiple myeloma.
4. CNS tumours, particularly spinal tumours.
5. Inflammatory mammary carcinoma, although large mammary tumours may also be painful.
6. Lower urinary tract tumours and those involving the prostate may be painful.
7. Oral tumours, especially if invasive to bone.
8. Invasive cutaneous tumours.
9. Intrathoracic tumours, particularly if disseminated and involving the pleura.
10. Intra-abdominal tumours, particularly if placing traction on the root of the mesentery, causing complete or partial obstruction of the intestinal tract, or resulting in distension of the capsule of solid organs.

The goal in managing dogs with cancer is to control pain and improve a patient's overall quality of life using traditional anti-cancer therapeutic modalities, various analgesic therapies and supportive care. Some patients will benefit from palliative surgeries to remove painful tumours, although the surgery itself may not positively affect the overall prognosis for survival (de Lorimier & Fan, 2005). A classic example is a painful appendicular osteosarcoma in which metastatic disease ultimately determines the survival time. In this example, amputating the limb provides immediate relief to the patient, and adjuvant chemotherapy is administered to prolong survival and delay the growth of micrometastatic disease (de Lorimier & Fan, 2005).

Conclusion

The treatment of chronic pain in dogs remains an ongoing learning process. Veterinary Nurses play a huge role in the care and assessment provided. With focus, continued learning and leadership, this area of veterinary medicine is a means for personal and professional growth and, ultimately, compassionate care of companion animal populations and for the Veterinary Nurse.

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