pISSN 1598-298X / eISSN 2384-0749
J Vet Clin 34(5) : 347-352 (2017)
http://dx.doi.org/10.17555/jvc.2017.10.34.5.347

Journal of Veterinary Clinics
www.ksvc.or.kr

Pain in Animals: Anatomy, Physiology, and Behaviors

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(Received: May 23, 2017 / Accepted: August 11, 2017)

Abstract : Pain, an adaptive but unpleasant sensation, is the most common symptom of numerous diseases in humans and animals. Although animal patients express this symptom frequently, a lack of communication abilities hinders its recognition by veterinary physicians, thereby leading to unsatisfactory management of the symptom. On the other hand, pain itself has its own neurological mechanisms, regardless of the disease that causes it. Thus, a physician may need to know the mechanisms underlying pain development in order to properly manage the symptom in a particular disease. In this review, we attempt to provide a brief introduction to the anatomical, physiological, and neurological basis of pain transmission and sensation. Although most knowledge about these mechanisms comes from studies in humans and laboratory animals, it is generally applicable to pet, farm, or zoo animals. In addition, we summarize pain behavior in several pet, farm, and laboratory animals for its proper identification. This information will help to identify and manage pain, and thus improve welfare, in animals.

Key words **:** Pain, Spinal cord dorsal horn, Pain pathway, Pain behaviors, Animal pain.

Introduction

The definition of pain is “an unpleasant sensory and emotional experience with actual or potential tissue damage, or described in terms of such damage”, as described in the reference for humans (15). In a manner similar to humans, animals perceive and react to the pain sensation (2) to protect their bodies from existing or potential tissue damage. Pain is therefore called an adaptive and early warning sign. Regarding the perception of pain, there are abundant similarities in anatomical pathways and biochemical and physiological mechanisms as well as in behavioral responses to pain stimuli, which have been collectively used to justify the validity of animal research for the development of pain management tools in humans. However, unlike in humans, pain assessment in animal patients is complicated by their lack of communication abilities. Therefore, the recognition and control of pain are far more difficult challenges in animals than they are in humans, thereby leading to insufficient healthcare in veterinary clinics. It is thus necessary for veterinary physicians to not only be familiar with pain behaviors in animals, but also understand the anatomical and physiological basis of pain mechanisms.

In this review, we will first summarize the anatomical basis of pain, particularly in the dorsal horn (DH) of the spinal cord. The spinal DH receives various peripheral sensory inputs, including pain, through the primary afferent fibers, whose cell bodies are located in dorsal root ganglia (DRG). It is thus the initial central nervous system (CNS) area that processes and integrates sensory and pain information, and provides the source of signals transmitted to the higher brain. On the other hand, nerve fibers, descending from the brainstem and other higher brain structures, and intrinsic neurons participate in the central processing of peripheral information in the DH, while projection neurons are in charge of the transmission of integrated information from the DH to various higher brain regions, particularly to the thalamus. In addition, we will describe the pathway of pain transmission to higher brain regions, and also the physiological aspects of pain, most of which are derived from studies in laboratory animals, that is, rats and mice. Finally, we will briefly discuss pain behaviors in animals; it should however be noted that pain treatments and their principles are not included here. The basic but essential knowledge of pain mechanisms provided in this short review would help veterinary clinicians properly manage pain in animals and increase awareness of animal welfare.

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The spinal cord dorsal horn: the first central area for pain transmission

The spinal cord is a continuum of the brain, consisting of an outer layer of white matter and an inner core of gray matter, and is typically described as a series of segmental components determined by the emergence of spinal nerves. In the case of the dog, there are eight cervical, thirteen thoracic,

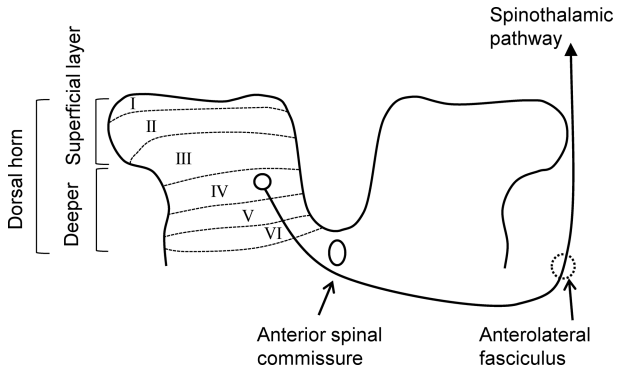

Fig 1. A diagram showing the spinal cord dorsal horn and the spinal thalamic pathway. The dorsal horn includes laminae I to VI; its superficial layer consists of laminae I and II, and its deeper layer of laminae III to VI. The axons of projection neurons decussate in the anterior spinal commissure, and are added to the spinothalamic pathway through the anterolateral fasciculus.

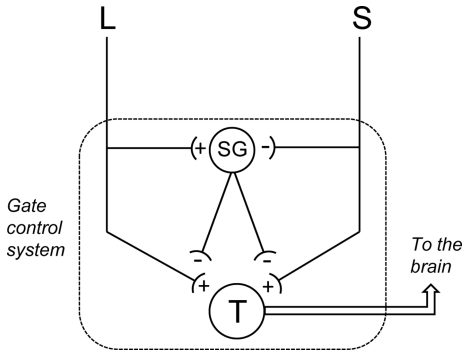

Fig 2. A simplified diagram of the gate control theory of pain mechanisms. Large diameter afferent fibers (L) excite (+) both substantia gelatinosa (SG) neurons in lamina II and transmission (T) neurons in the deeper layer, while small diameter fibers (S) inhibit (–) SG neurons but excite T neurons. The SG neurons further inhibit both the L and S fibers presynaptically. Therefore, the activation of L fibers blocks the transmission of pain signals through the S fibers (i.e., closing the pain gate), whereas the activation of S fibers allows transmission (i.e., opening the gate).

seven lumbar, three sacral, and about five caudal segments (28). Longitudinally oriented myelinated axons, including some non-myelinated axons, are responsible for white color of the white matter. The gray matter occupies the central portion of the spinal cord, and contains neuronal cell bodies, dendrites, axons, and glial cells. On the basis of the size, shape, cytological features, and density of neurons, the gray matter is divided into 10 laminae (or layers) (as seen in transverse sections), an organization that was first proposed in the cat (20), at all levels of the spinal cord except for lamina VI, which is present only in the cervical and lumbar segments. The first six distinct laminae (I-VI) in the dorsal-to-ventral direction are collectively called "the DH" of the spinal cord (Fig 1). The first and second laminae form the superficial layer of the spinal DH (6), and laminae III-VI form the deeper layer.

Primary afferent fibers, arranged on the basis of fiber size, are distinctly distributed to the laminae of spinal DH (25), which affects, physically, the velocity of nerve conduction and, physiologically, the modality of sensory information. Most fine myelinated (Aδ fiber; conduction velocity, 1.5 to 10 m/sec) or unmyelinated (C fiber; conduction velocity, < 1.5 m/sec) primary afferent fibers, which transmit mechanical and thermal pain sensation, end predominantly in the superficial layer of the spinal DH (9,10). On the other hand, most large-myelinated cutaneous primary afferents (Aβ fiber; conduction velocity, > 10 m/sec), which function as low-threshold mechanoreceptors for light touch sensation, have a characteristic pattern of termination in the deeper layer of the spinal DH (7). Further, the unmyelinated C fibers can be divided into two major groups: peptidergic and non-peptidergic (25). Peptidergic C fibers express tropomyosin receptor kinase A (TrkA) or transient receptor potential cation channel subfamily V member 1 (TRPV1) as cellular markers and contain neuropeptides such as calcitonin gene-related peptide (CGRP) and/or substance P (17). These fibers are considered as nociceptors because of their responsiveness to noxious stimuli (8). The peptidergic CGRP/substance P-containing C fibers terminate mainly in lamina I and the outer layer of lamina II. Non-peptidergic C fibers can be identified by staining with Bandeiraea simplicifolia isolectin B4 (IB4) (24), and are distributed in the inner layer of lamina II; this population is also nociceptive (3).

In the spinal DH, lamina I, known as the marginal nucleus, contains projection neurons whose axons contribute to the spinal thalamic tract for sensory and pain transmission to higher brain areas. Due to a concentration of small neurons and a relatively small number of myelinated axons, lamina II is observable as a translucent band under the naked eye or light microscope, thus being called "substantia gelatinosa (SG)" (11,16). All SG neurons are heterogeneous interneurons, and 30% of them are γ-aminobutyric acid (GABA)ergic (26). Although interneurons also exist in the deeper layer (i.e., laminae III-VI) of the spinal DH, it is characteristic that this area contains many projection neurons of which axons contribute to the spinocervicothalamic and spinothalamic tracts for sensory and pain transmission (28). Many of the projection neurons are neurokinin (NK) 1-positive neurons. These neurons project to pain-relating areas in the brain, and receive inputs from GABAergic interneurons that contain neuropeptide Y (NPY) (18). This contrasts with the fact that a subpopulation of projection neurons, called giant marginal cells, in lamina I lack NK1 and express the glycine receptor-associated protein gephyrin (19).

The synaptic circuitry in the spinal DH, as an integration and transmission system for pain, is not clearly demonstrated yet. However, Melzack and Wall (14) proposed a theory that describes how peripheral pain signals are transmitted from the DH to higher brain areas (called the gate control theory; Fig 2). In this theory, SG neurons in lamina II of the DH play a significant role in the gate for pain transmission by presynaptically controlling both large and small diameter fibers that innervate transmission neurons. Because the main function of SG neurons is an inhibitory one to the terminals of both small and large fibers, the activation of SG neurons by the large diameter fibers (Aβ fibers) closes the gate for passing on the pain signals conveyed through both fibers. However, the inhibition of SG neurons by the small diameter

fibers (C fibers) opens the gate and transfers the pain signals to the transmission system. The transmission system, conceivably consisting of projection neurons, transmits the signals to the higher brain areas that are involved in the processing of pain signals. Although much recent information needs to be incorporated, the gate control theory helps our neurological understanding of pain symptoms.

Pain pathways to the brain

Pain sensation is not simple; rather, it has a distinct quality with different psychological dimensions in humans, as well as, probably, in pets and animals. Several anatomical pain pathways, which convey the integrated signal in the spinal DH to the higher brain regions, play roles in the quality of pain, that is generally described as a combination of sensory-discriminative (temporal, spatial, thermal/mechanical), affective (subjective and emotional; e.g., fear, tension, and autonomic responses), and evaluative (cognitive, describing the magnitude of the quality; e.g., stabbing/pounding or mild/severe) components (12).

The first pathway is the spinothalamic pathway (27) that involves the projection neurons in lamina I and the deeper layer of the spinal DH (Fig 1). This pathway is fast-conducting and located in the anterolateral fasciculus of the spinal cord (Figs 1 & 3), and it decussates in the anterior spinal commissure in one to three segments above the level of the root entry. While ascending through the anterolateral fasciculus, the bundle of axons from above-levels of the spinal segment are added to the inner side of the tract one by one, terminating in the several brainstem and the thalamic structures. This pathway helps animals to identify the location (spatial) and time (temporal) of pain stimuli, therefore contributing to the sensory-discriminative component of pain. The main arrival region of the spinothalamic pathway in the thalamus is the ventrobasal and posterior nuclei for its lateral division and, for its medial contingent, the intralaminar complex of nuclei and in the nucleus submedius (21).

Another fast-conducting pathway is the postsynaptic dorsal column pathway (Fig 3), which follows dorsal column nuclei (gracile nucleus and cuneate nucleus in the brainstem), exerting a modulating effect on pain transmission. This pathway reaches the ventrobasal and ventroposterior nuclei of the thalamus (21).

The rest pain pathways, rather slowly-conducting, include the spinoreticulothalamic and spinohypothalamic pathways (Fig 3) (21). The spinoreticulothalamic pathway directly projects to the reticular core of the medulla and the midbrain, forming synapses in the nucleus gigantocellularis and, more rostrally, in the nuclei of the parabrachial region, the midbrain reticular formation, the periaqueductal gray matter, and the hypothalamus. The spinohypothalamic pathway targets both sides of the hypothalamus, the thalamus, the superior colliculus, and reticular formations in the midbrain, the pons, and the medulla. The pain signals passing through these slowly-conducting pathways then terminate in the medial and intralaminar nuclei of the thalamus, and contribute to the affective component of pain, including various reflexes, endocrine adjustments, and emotional changes caused by pain. The evaluative component of pain involves broad areas of the brain, including the primary and secondary somatosensory cortex, and may also be applicable to animals.

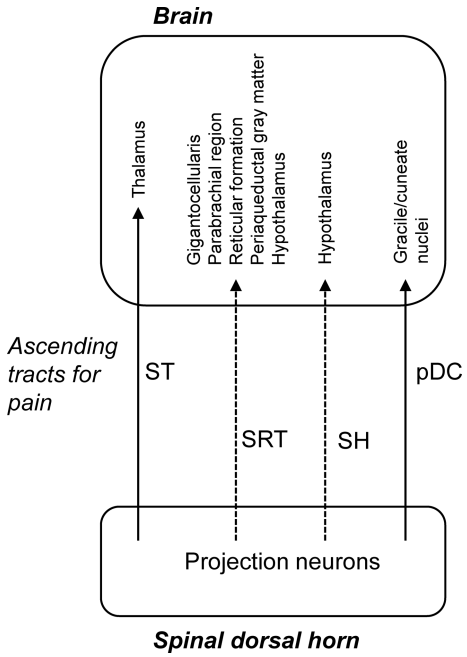

Fig 3. A summary of ascending pain pathways. Projection neurons in the spinal dorsal horn, particularly, the deeper layer, send axons to the various regions of the brain through ascending tracts. The lateral fast-conducting pathways (*solid lines*) includes spinothalamic (ST) and postsynaptic dorsal column (pDC) pathways; the medial slowly-conducting pathways (*dotted lines*) include spinoreticulothalamic (SRT) and spinohypothalamic (SH) pathways.

Physiological aspects of pain

Pain is typically evoked by the sequential activation of peripheral nociceptors, pain pathways, and the central pain centers of the brain. The particular stimuli that activate specific nociceptive receptors are diverse. For example, nociceptive receptors in the skin can be activated by pricking, cutting, crushing, burning, and freezing. However, those stimuli do not activate the joints, which are sensitive to hypertonic saline or inflammation in the synovial membrane. In addition, pain in skeletal muscles, as well as in cardiac muscles, is caused by ischemia, necrosis, hemorrhage, injection of irritating solutions, prolonged contraction, or injuries of connective tissue sheaths. Therefore, to identify the exact sources that cause pain in animals, veterinary physicians need to delineate the stimuli sensitive to a specific tissue in the pain area.

Tissue damage, activating the nociceptors and/or pain pathways and causing "primary pain", induces the release of proteolytic enzymes. These enzymes locally act on tissue proteins to liberate substances, for instance, histamine, prostaglandins, serotonin, and potassium ions. The nociceptors in the periphery respond to the released pain substances, thereby increasing vascular permeability. The activation of nociceptors, especially C fibers, further releases substance P, which

Table 1. Symptoms of pain in some of pets, farm and laboratory animals, and domestic poultry (*modified from* Short, 1994)

Species	Posture	Temperament	Vocalization	Locomotion	Other
Dog	Tail between legs, arched or hunched back, twisted body to protect pain site, drooped head, prolonged sitting position, tucked abdomen, lying in flat, extended position	Aggressive (Typical aggressive dogs sometimes show the opposite behavior), biting, clawing, attacking, escaping	Barking, howling, moaning, whimpering	Reluctance to move, carrying one leg, lameness, unusual gait, unable to walk, refused to climb stairs	Continuous localized licking, altered breathing, unable to perform normal tasks, attacks other animals or people if painful area is touched, chewing painful areas (self-trauma), tearing, changes in sleeping, drinking & eating patterns
Cat	Tucked limbs, arched or hunched head and neck or back, tucked abdomen, lying flat, slumping of the body, drooping of the head	Aggressive, biting, scratching, chewing, attacking, escaping	Crying, hissing, spitting, moaning, screaming	Reluctant to move, carrying one leg, lameness, unusual gait, unable to walk	Attack if painful areas are touched, failure to groom, dilated pupils
Horse	Standing with head down, standing on three legs, recumbency in severe pain, tucked abdomen, arched back, abnormal position of feet and legs, dropped ears	Aggressive, kicking, striking, biting, uncooperative, fighting, defeated, docile, escaping	Quiet, grunting, moaning	Lameness, reduce speed, abnormal gait, non-weight-bearing, reluctance to move, walks on toes or hocks	Self-trauma, significant reduction in performance, dull eyes
Cattle	Prolonged sternal recumbency, head and neck extended or flexed into flank area	Aggressive, kicking, head butting, grinding teeth, docile	Bawling or quiet	Reluctance to move, abnormal gait, lameness, dragging leg, incoordinated hopping or lunging	Reduced mile production
Pig	Prolonged recumbency, arched back, drooping head	Aggressive, escaping, biting, slashing	Squealing or quiet	Reluctance to move, walking with arched back	Poor growth or loss of weight
Rat	Persistent recumbent posture, hiding	Aggressive or docile	Squealing	Accelerated or depressed movement	Abnormal writhing, eats bedding
Domestic poultry	Prolonged setting on feet, drooping of wings, head & neck, head turned and under wing, hiding	Docile, appears asleep	Excessive noise or quiet	Reluctance to move, no effort to use wings	Reduced egg production

induces vasodilation, erythema, and edema ("neurogenic inflammation"), causing "secondary pain" in a much broader area than just the area that is injured.

Regarding the perception of pain, it should be mentioned that the pain threshold depends on the state of an animal. If an animal is in a state of distraction, strong emotion (e.g., fear or rage), or depression, the threshold to evoke pain would be lowered, i.e., the animal is more sensitive to pain stimuli. In addition, pain is heterogeneous, regarding etiological factors, mechanisms and temporal characteristics (22). A single mechanism may potentially produce different symptoms, such as spontaneous pain or shock-like pain, while the same symptom (a pain response to light touch) may involve several different mechanisms. In addition, the mechanisms contributing to the early state of pain will change in later (chronic) states, and therefore require different pain management strategies.

Pain itself may cause various physiological responses that can sometimes be used for objective measurements of pain. Those physiological parameters are measured before and during pain, or after the administration of pain medication. As neurophysiological signs, outward symptoms of the peripheral and/or central nervous system, such as twitching, tremors, convulsions, paralysis, dilated pupils, etc., can be measured; alternatively, using complex techniques, such as evoked potential analyses, standard electroencephalograms or brain wave analyses, may be considered. As cardiovascular signs, changes in heart rate, blood pressure, blood flow, cardiac output, etc., can be measured. Changes in respiratory rate, minute volume, and blood gases and pH levels can be measured as respiratory signs. In addition, digestive and urinary signs can be measured.

Pain behaviors in animals

The recognition of pain in animals is problematic, and relies on the interpretation of an animal's behavior by an observer, because there is no effective means of communication between them. Therefore, animals experiencing pain can be evaluated by parameters that relate to their behaviors (Table 1) (23). Visible pain behaviors could be a limping gait or a behavior not to apply weight on the affected limb. In addition, animals' nocifensive signs, to escape from nociceptive stimuli, could be measured, which are paw licking, excessive grooming, excessive exploratory behavior, and guarding of the affected area. In some of serious pain conditions, particularly in the case of peripheral nerve injury, an affected animal attacks and mutilates the denervated areas ("autotomy").

In veterinary hospitals and laboratories, pain behaviors can be tested by applying mechanical or thermal stimuli to the affected area, which can be determined as allodynia or hyperalgesia. Allodynia is the case of withdrawal responses to non-painful mechanical or thermal stimuli, for example, light touch and brushing, while hyperalgesia describes stronger or early withdrawal responses to painful mechanical (e.g., Von Frey filaments) or thermal (e.g., hot or cold water) stimuli. It should be noted that the degree of pain behavior depends on the species, the pain type (*not described in this review*), and the affected areas.

Regardless of the advances that have been made in understanding pain behaviors in animals, finding convincing scores for pain identification is still challenging. Visual analogue scores (VAS) (1), numerical rating scales (29), or any other pain score indices usually involve translating subjective assessment values into numbers. It is not rare to find significant differences in pain scores among multiple observers or veterinary clinicians. In addition, these methods are sometimes unreliable in the assessment of acute pain in dogs (5). In this regard, the Glasgow Composite Measure Pain Scale (GCMPS) would be a reliable way to assess pain in animals (4). The GCMPS was developed by using methods similar to McGill Pain Questionnaire in humans (13), and took the form of a structured questionnaire completed by an observer, while following a standard protocol which includes the assessment of spontaneous and evoked behaviors by evaluating the interaction between the observer and the animal.

Concluding remarks

The understanding, treatment and management of pain is of great importance in the veterinary clinic. Although most knowledge about pain mechanisms comes from studies in humans and laboratory animals, the understanding of pain mechanisms may also help to manage pain in veterinary clinics. Furthermore, extensive efforts need to be made to define pain behaviors in pet, farm, zoo, or laboratory animals, and to understand the neurological etiology of pain. In addition, careful observation of specific pain behaviors in different species by veterinary clinicians would help identifying the adequate treatment for individual animal patients. Such an effort would highly improve animal welfare.

Acknowledgements

This manuscript was designed by DY and written and read by DY, TWK and HC. All authors approved the final manuscript. The authors have no conflict of interests regarding this work.

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