ABSTRACT

ANATOMICAL CHARACTERIZATION OF THE CANINE BRACHIAL PLEXUS AND EVALUATION OF THREE BLIND BRACHIAL PLEXUS BLOCK TECHNIQUES

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This thesis describes the ventral spinal rami that contribute to the formation of the brachial plexus and its peripheral nerves in cadaver dogs and evaluated three blind techniques for performing the brachial plexus block. In phase I of this study the region of the brachial plexus of cadaver dogs was injected using one of three blind approaches for performing the brachial plexus block. The brachial plexus was then dissected to evaluate the resultant nerve staining followed by dissection and identification of the nerves that formed the brachial plexus. In phase II of this study the degree of anesthesia and analgesia that resulted from each of the three approaches for performing the brachial plexus block were evaluated in clinical cases of dogs undergoing surgical procedures of the thoracic limb.

Phase I of this study identified that the canine brachial plexus in this group of dogs was formed from the ventral spinal nerve rami of C6, C7, C8 and T1. Additionally, three anatomical landmarks were identified as fundamental for successful dye injection at the level of the brachial plexus. These landmarks were the transverse process of C6, the point of the shoulder, and the first rib. No differences in dye dispersion and nerve staining were identified between the three blind brachial plexus block techniques.
The clinical phase, phase II, of this study did not identify any differences between the three approaches to block the brachial plexus and the resultant postoperative pain scores in dogs undergoing surgical procedures of the thoracic limb. Additionally, blockade of the brachial plexus provides better analgesia in dogs undergoing surgical procedures of the distal thoracic limb than dogs undergoing thoracic limb amputation.
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DECLARATION OF WORK PERFORMED

I declare that the work reported in this thesis was performed by myself and the contributing authors as stated.

The authors’ contribution is as follows:

**Alicia Skelding:** Acquisition and preparation of cadaver dogs, brachial plexus injection and dissection of cadaver dogs, anesthetic monitoring and pain assessment for clinical study, data collection, manuscript preparation. **Alexander Valverde:** Study design and funding application, cadaver dog staining score assignment, brachial plexus blocks and assistance with pain assessments for clinical study, data collection, statistical analyses, manuscript review and revision. **Rodrigo Aguilera:** Assistance with anesthetic monitoring and of pain assessment for clinical study. **Noel Moens:** Thesis and manuscript revision. **Jeffrey Thomason:** Assistance with preparation of cadaver dogs and thesis revision. **Roman Poterski & David Robinson:** Assistance with preparation of cadaver dogs. **Melissa Sinclair:** Thesis revision.
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<tbody>
<tr>
<td>AA</td>
<td>Axillary approach</td>
</tr>
<tr>
<td>ASA</td>
<td>American Society of Anesthesiologists</td>
</tr>
<tr>
<td>BP</td>
<td>Brachial plexus</td>
</tr>
<tr>
<td>BPB</td>
<td>Brachial plexus block</td>
</tr>
<tr>
<td>C6</td>
<td>Cervical spinal nerve six</td>
</tr>
<tr>
<td>C7</td>
<td>Cervical spinal nerve seven</td>
</tr>
<tr>
<td>C8</td>
<td>Cervical spinal nerve eight</td>
</tr>
<tr>
<td>CMPS-SF</td>
<td>Glasgow composite pain scale-short form</td>
</tr>
<tr>
<td>CNS</td>
<td>Central nervous system</td>
</tr>
<tr>
<td>ETCO₂</td>
<td>End tidal carbon dioxide</td>
</tr>
<tr>
<td>ETIₔO</td>
<td>End tidal isoflurane</td>
</tr>
<tr>
<td>Hz</td>
<td>Hertz</td>
</tr>
<tr>
<td>IM</td>
<td>Intramuscular</td>
</tr>
<tr>
<td>IV</td>
<td>Intravascular</td>
</tr>
<tr>
<td>LA</td>
<td>Local anesthetic</td>
</tr>
<tr>
<td>L1</td>
<td>Landmark 1</td>
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<td>L2</td>
<td>Landmark 2</td>
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<td>L3</td>
<td>Landmark 3</td>
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<tr>
<td>L₄</td>
<td>Lumbar spinal nerve four</td>
</tr>
<tr>
<td>L₅</td>
<td>Lumbar spinal nerve five</td>
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<tr>
<td>MAC</td>
<td>Minimum alveolar concentration</td>
</tr>
<tr>
<td>Symbol</td>
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</tr>
<tr>
<td>mA</td>
<td>Milliampere</td>
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<td>MHz</td>
<td>Megahertz</td>
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<td>mL</td>
<td>Milliliters</td>
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<tr>
<td>ms</td>
<td>Milliseconds</td>
</tr>
<tr>
<td>PA</td>
<td>Perpendicular approach</td>
</tr>
<tr>
<td>PAG</td>
<td>Periaqueductal gray matter</td>
</tr>
<tr>
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<td>Thoracic spinal nerve one</td>
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<td>T₂</td>
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<tr>
<td>TA</td>
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</tr>
<tr>
<td>SpO₂</td>
<td>Peripheral capillary oxygen saturation</td>
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CHAPTER 1

GENERAL LITERATURE REVIEW
1.1 INTRODUCTION

The application of local anesthetic techniques plays a fundamental role in providing anesthesia and analgesia in veterinary species. Local nerve blocks can be very useful as part of multimodal analgesia in anesthetized patients through the anesthetic/analgesic effects of local anesthetic drugs in the intra- and postoperative period. Local anesthetic techniques can block the transmission of nociceptive information from peripheral sensory nerves to the central nervous system and therefore prevent pain perception. In this way, local nerve blocks also play an important role in preventing central and peripheral sensitization in response to peripheral nerve injury. Local anesthetic techniques target specific sites of the pain pathway depending on the technique that is implemented. When administered peripherally, local anesthetics can block nociceptive fiber endings (nociceptors) or whole nerves and the contained fibers versus epidural or spinal administration, which targets the nerve roots and/or spinal cord. The end goal of any local anesthetic technique is blocking all sensory afferent nociceptive fibers, namely Aδ and C fibers. However, local nerve blocks also affect afferent and efferent fibers involved in motor activity (Aα), proprioception (Aγ), touch (Aβ), and sympathetic (B) activity; therefore, these functions can also be affected at the onset of the block. Occasionally local anesthetic drugs are administered in combination with opioids, α2-agonists or epinephrine to enhance and prolong the effects of the nerve block.

A number of different nerve blocks have been described in veterinary species. Regardless of the nerve block to be performed, comprehensive knowledge of the relevant anatomy is important for successful and safe performance of the peripheral nerve blockade, as most of these techniques require great reliance on anatomical landmarks. Considerable anatomical variation exists across veterinary species. Species-specific knowledge of...
anatomical landmarks is necessary because the soft tissue target areas for peripheral nerve blocks will vary in relative location to these anatomical landmarks among species.
1.2 NERVE ANATOMY, HISTOLOGY & PHYSIOLOGY

The peripheral nervous system is composed of the somatic and autonomic nervous systems, in addition to peripheral ganglia and connective tissue elements (Goldstein 2001). The somatic peripheral nervous system consists of spinal nerve roots, spinal nerves, dorsal and ventral rami and peripheral nerves with associated branches that supply every tissue for sensory (afferent) and motor function (efferent) (Stewart 2003). The autonomic nervous system is structurally divided into sympathetic and parasympathetic divisions. The primary role of the autonomic nervous system is the maintenance of homeostasis and the regulation of visceral functions (Shields 1993).

Both somatic and autonomic anatomical components of the peripheral nervous system are contained in the peripheral nerve as an assorted mix of individual nerve fibers (axons) that can be myelinated or unmyelinated (Stewart 2003). This bundle of nerve axons within the peripheral nerve is known as a fascicle or fasciculus. Fascicles can vary considerably in number and size from one peripheral nerve to the next but may also vary in size within and among nerves (Stewart 2003). The fascicles can be arranged in two distinct combinations: the cable structure in which fascicles remain discrete throughout the entire length of the nerve and the plexiform structure in which there is branching and rejoining of fascicles at various locations along the course of the nerve (Stewart 2003). The distal parts of nerves often contain fascicles adopting the cable structure, whereas the plexiform arrangement is usually observed more proximally in the nerve (Stewart 2003). Sensory nerve fibers that originate from the skin and motor nerve fibers that travel to specific muscle groups remain grouped together within fascicles as they course through the peripheral nerve (Stewart 2003).
The cellular components of peripheral nerve fibers are bound together by three layers of connective tissue: the epineurium, perineurium and endoneurium (Goldstein 2001; Stewart 2003), Figure 1.1.

**Figure 1.1.** Schematic cross section of a peripheral nerve (Lin & Liu 2013).
The endoneurium is a loose connective tissue layer that wraps each individual nerve fiber (Goldstein 2001). The loose connective tissue structure of the endoneurium differs from the structure of the perineurium and epineurium. It surrounds the neural and glial components of the peripheral nerves (Goldstein 2001). The resultant microenvironment is continuously in flux with various ions and macromolecules being added to and removed from it, by the surrounding arterial and venous systems (Goldstein 2001). The fibers within these fasciculi are ensheathed by a dense, highly organized connective tissue layer called the perineurium (Goldstein 2001). The cells of the perineurium are metabolically active and the structure itself is relatively rigid, resisting any expansion or challenge associated with fluid build up (Goldstein 2001). The role of the perineurium is to maintain homeostasis of the endoneurial fluid (Stewart 2003). The epineurium surrounds the entire nerve, made up of numerous fasciculi, and is composed of strong, primarily collagenous, connective tissue (Goldstein 2001, Stewart 2003).

Peripheral nerves receive blood supply via two difference sources (Neal 2003; Sinnott et al. 2003), Figure 1.2. The first source is an extrinsic source that does not supply nutrients to the nerve itself and exists within the epineurium and perineurium (Neal 2003). The extrinsic blood vessels are responsive to adrenergic stimulation, and eventually anastomose with the intrinsic blood vessels (Sinnott et al. 2003). The second source is an intrinsic source that exists within the endoneurium and provides nutrients to the peripheral nerve (Neal 2003).

Fibers within each peripheral nerve are usually classified into three major categories based on fiber diameter and rate of conduction: A, B and C fibers (Goldstein 2001). Both class A and B fibers are myelinated, whereas class C fibers are unmyelinated. In general, myelinated fibers have faster conduction velocities than unmyelinated fibers, and the presence of myelin also makes these nerve fibers larger (Waxman 1980).
Class A fibers consist of somatic afferent (sensory) and efferent (motor) fibers and are the largest peripheral fibers with conduction velocities from 12 to 100 m/sec (Goldstein 2001). The somatic afferent class A fibers are further divided into groups I, II, and III. Group I (Aα) fibers are heavily myelinated, and have a diameter of 12-20 μM. These fibers bring proprioceptive sensory information from muscle spindle cells (subgroup Ia or Aα) and tendon organs (subgroup Ib or Aα), with a conduction velocity of 80 to 100 m/sec (Goldstein 2001). Group II (Aβ) fibers are moderately myelinated, with a diameter of 5-15 μM. These fibers bring sensory information from cutaneous receptors and muscle spindle cells, with conduction velocities of 20 to 90 m/sec (Goldstein 2001). Group III fibers (Aδ) are lightly myelinated, with a diameter of 2-5 μM. These fibers are responsible for bringing sensory information through the neospinothalamic tract from peripheral nociceptors as well as sensory information from the walls of blood vessels. They have the slowest conduction velocity for this type of fiber, which ranges from 12 to 30 m/sec (Goldstein 2001). Somatic efferent class A fibers can also be further divided into Aα and Aγ motor fibers; somatic efferent Aα fibers are similar in myelination and conduction speed to the afferent Aα fibers described above, but have effector actions on extrafusal muscle fibers. They have a higher conduction velocity (80 to 100 m/sec)
than the $A_\gamma$ fibers, which are moderately myelinated. $A_\gamma$ fibers have a diameter of 3-6 $\mu$M and conduction velocities ranging from 4 to 24 m/s for effector actions on intrafusal muscle fibers (Goldstein 2001).

Class B fibers are preganglionic fibers for the sympathetic nervous system with conduction velocities from 3 to 15 m/sec, being lightly myelinated, and having a diameter of 1-3 $\mu$M (Goldstein 2001).

Finally, class C fibers are unmyelinated fibers. They are also somatic afferent fibers and bring information through the paleospinothalamic and archispinothalamic tracts from peripheral nociceptors, have a diameter of 0.4-1.2 $\mu$M and very slow conduction velocities from 0.3 to 1.5 m/sec. These fibers also have postganglionic sympathetic actions (Goldstein 2001).

In addition to the nerve fibers, the nervous system contains numerous ganglia. A ganglion is a structure that contains nerve cell bodies linked by synapses and forms a swelling on the nerve fiber. Both sensory and autonomic ganglia exist within the nervous system. The sensory ganglia consist of the dorsal root ganglia and those of the cranial nerves – trigeminal ganglion, geniculate ganglion, petrous ganglion and nodose ganglion (Goldstein 2001; Mizeres 1955). These ganglia are surrounded by periganglionic tissue, a type of connective tissue that is very similar in structure to the perineurium and epineurium (Goldstein 2001). For autonomic ganglia, the anatomical arrangement consists of a first (preganglionic) neuron located in the central nervous system that relays input to a second (postganglionic) neuron, via the ganglion in the peripheral nervous system, and from here the output is relayed to target cells (Evans & de Lahunta 2013; Goldstein 2001). Blood–ganglionic barriers do not exist; the blood vessels within the ganglia are fenestrated and allow passage of large molecules into the extracellular space (Goldstein 2001).
In mammals the spinal cord is organized in segments and, from each segment, paired dorsal and ventral spinal nerve roots emerge (Dyce et al. 2010; Goldstein 2001). The dorsal root brings afferent information into the spinal cord and the ventral roots carry efferent information from the spinal cord to target cells. Two types of sensory neurons enter the dorsal root of the spinal cord, a somatic afferent neuron and a visceral afferent neuron (Amann & Constantinescu 1990; Dyce et al. 2010; Goldstein 2001). Somatic afferent neurons enter the spinal cord via the dorsal root and bring information from body wall structures (Dyce et al. 2010; Goldstein 2001). Visceral afferent neurons also enter the spinal cord via the dorsal root but bring sensory information primarily from viscera (Goldstein 2001). Two types of efferent neurons exit the ventral root of the spinal cord: a somatic efferent neuron and a visceral efferent neuron (Amann & Constantinescu 1990; Dyce et al. 2010; Goldstein 2001). Somatic efferent neurons have their cell bodies in the ventral horn of the spinal grey matter and innervate skeletal muscle (Dyce et al. 2010; Goldstein 2001), while visceral efferent neurons have their cell bodies in the intermediolateral portion of the spinal grey matter and exit the spinal cord through the ventral root of the spinal nerve to become preganglionic, myelinated fibers of the autonomic nervous system (Dyce et al. 2010; Goldstein 2001).

1.2.1 Visceral Afferent & Efferent Pathways

The peripheral autonomic nervous system can be divided into the sympathetic and the parasympathetic systems (Amann & Constantinescu 1990; Goldstein 2001; Evans & de Lahunta 2013). The autonomic nervous system is an arrangement of neurons: a sensory neuron providing information to the central nervous system, an interneuron within the central nervous system and two motor neurons in sequence providing the response from the central nervous system (Amann & Constantinescu 1990; Goldstein 2001; Shields 1993; Figure 1.3).
The sensory receptors that provide information from visceral sensation are located in the body walls of the viscera (Amann & Constantinescu 1990). The sensory portion of the autonomic nervous system is very similar to its somatic counterpart, with receptors that respond to temperature, pressure and movement of the viscera (Amann & Constantinescu 1990).
The two motor neuron sequence of the visceral efferent system has the cell body of the preganglionic neuron located in the central nervous system and the cell body of the postganglionic neuron located in a peripheral ganglion (Goldstein 2001). The arrangement of the sympathetic and parasympathetic systems differ in the location of their peripheral ganglia, but in general a preganglionic neuron from the central nervous system synapses on the postganglionic neuron within the peripheral ganglion (Amann & Constantinescu 1990). This postganglionic neuron extends outside of the central nervous system from the peripheral ganglion to a target organ (Amann & Constantinescu 1990; Goldstein 2001).

The preganglionic axons of the sympathetic nervous system are short and exit from the ventral nerve roots of C₈/T₁ through L₄/L₅ to synapse on postganglionic neurons located in the sympathetic trunk (Evans & de Lahunta 2013). The sympathetic trunk is a paired strand of ganglia located ventrolateral to the thoracic and lumbar vertebrae. The preganglionic axons of the parasympathetic nervous system leave as part of cranial nerves III, VI, IX and X and as part of the ventral roots of the sacral spinal nerves (Evans & de Lahunta 2013). These long axons synapse on short postganglionic neurons near their target organs, Figure 1.3.

1.2.2 Somatic Afferent Pathways

Somatic afferent (sensory) pathways arise from the skin, fascia and muscles in the body (Goldstein 2001). The receptors of these pathways have developed to respond to a definite stimulus for that type of receptor (Goldstein 2001). Exteroceptive impulses arise from the integument in response to receptors stimulated by temperature, touch, pressure and pain (Goldstein 2001). A second type of impulse is described as proprioception. Proprioceptive impulses arise from deeper receptors located within ligaments, muscles and joints (Goldstein 2001), which function to provide a sense of body position. Cutaneous receptors are subdivided into unencapsulated (free) or encapsulated nerve endings. Perhaps more clinically important,
is the functional division of cutaneous receptors: mechanoreceptors, thermoreceptors and nociceptors (Goldstein 2001). Mechanoreceptors respond to touch, pressure and vibration by converting these stimuli into an electrical signal (Goldstein 2001). Stimulation of these receptors opens voltage-gated sodium channels and an action potential is developed. Thermoreceptors respond to changes in temperature, which results in depolarization of these specific fibers (Goldstein 2001). Nociceptors are free nerve endings that respond to tissue injury or potential injury. There are two principal fiber types that transmit nociceptive information: the Aδ fibers transmit information in response to initial, rapid pain and C fibers transmit information in response to chronic, slow pain (Goldstein 2001).

1.2.3 Somatic Efferent Pathways

Somatic efferent neurons arise from motor neuron cells in the ventral horn of the spinal cord. They exit the ventral spinal nerve root to carry motor impulses to the skeletal system (Evans & de Lahunta). Two types of motor neurons are described in the nervous system: the large Aα motoneurons innervate skeletal muscle and the smaller Aγ motoneurons innervate muscle spindles (Goldstein 2001). The Aα motoneurons are highly active cells and have the single function of causing muscle cell contraction (Goldstein 2001). Each motor nerve innervates a group of muscle fibers and together they comprise a motor unit (Goldstein 2001). The neurotransmitter at the terminal end of these motor nerves that results in muscle cell contraction is acetylcholine, released in response to nerve discharge (Goldstein 2001).

1.3 NOCICEPTION AND PAIN

Pain management is an important consideration in veterinary practice that improves the animal’s well-being and quality of life. The advancements in pain recognition and assessment
have led to improvements in analgesic protocols and techniques for animal husbandry procedures, peri- and post-operative pain, as well as management of chronic and cancer pain.

Pain was defined by the International Association for the Study of Pain in 1979 as “an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage” (Landa 2012; Egger et al. 2014). The definition most commonly cited to describe animal pain was presented by Molony and Kent in 1997: “Animal pain is an aversive sensory and emotional experience representing awareness by the animal of damage or threat to the integrity of its tissues; it changes the animal’s physiology and behaviour to reduce or avoid damage, to reduce the likelihood of recurrence and to promote recovery; non-functional pain occurs when the intensity or duration of the experience is not appropriate for the damage sustained and when physiological and behavioural responses are unsuccessful in alleviating it”.

Physiologically, pain can be divided according to the duration of time it is experienced or according to the origin of tissue injury. For example, acute pain is of short duration, disappearing soon after the injury has healed (Landa 2012); whereas, chronic pain persists beyond healing of the original injury (Landa 2012). In regards to origin, pain can be divided into somatic, visceral and neuropathic pain (Vinuela-Fernandez et al. 2007; Landa 2012; Okafor et al. 2014). Somatic pain is the sensation generated as a result of skin or muscle injury and is usually well localized (Landa 2012). Visceral pain is the result of inflammatory damage or distension of the hollow organs and, because of the physiology of visceral sensory innervation, is often difficult to localize (Landa 2012). Primary visceral afferent neurons enter the dorsal horn of the spinal cord and synapse nonspecifically over several spinal segments, often crossing to the contralateral dorsal horn (Robinson & Gebhart 2008; Fu et al. 2011). Clinically, animals experiencing visceral pain often demonstrate referred pain in other parts of the body as a result of visceral afferent neurons receiving concurrent input from somatic...
structures (Robinson & Gebhart 2008; Landa 2012). Neuropathic pain is a peripheral or central pathology of the nervous system (Okafor et al. 2014) and often has a component of disproportionate clinical response to stimuli such as hyperalgesia or allodynia (Landa 2012). The differences in origin and type of pain experienced are related to the type of nociceptive fibers involved in the transmission of the impulse from the tissue to the brain and the arrangement of these fibers along that pathway.

Nociception is defined as the physiologic process that results from stimulation of specialized fibers (nociceptors) and nerve signaling to the central nervous system for processing (Gaynor & Muir 2015). These neural processes lead to the conscious interpretation of pain if the hypothalamus and cortex are subsequently stimulated; if the signal does not reach the cortex, they only result in nociceptive stimulation and autonomic responses without the perception of pain. This implies that there is nociception without pain but there is no pain without nociception. Nociceptors are the free endings of peripheral afferent (sensory) neurons that respond to noxious or partially noxious stimuli (Egger et al. 2014). Information from peripheral nociceptors is conveyed to the central nervous system via primary afferent nerve fibers (Gaynor & Muir 2015; McKune et al. 2015); of which there are three defined types of nociceptive fibers: Aβ fibers, Aδ fibers and C fibers.

Adequate knowledge and understanding of the physiologic pathways and mechanism of pain is necessary for its effective management and provision of sufficient analgesia. The course of a painful stimulus from a peripheral nociceptor to conscious perception in specific regions of the brain is multifaceted, and knowledge of the numerous modifications that a painful stimulus undergoes on the route to perception is described below. Its understanding will improve the ability to alleviate or control pain through the use of various analgesic drugs.

1.3.1 Pain Transmission
Pain transmission from stimulus to conscious perception is divided into five stages. The noxious stimulus that initiates the pain pathway can be mechanical, chemical, or thermal in origin (Gaynor & Muir 2015). The stimulus is (1) transduced into an action potential that is (2) transmitted via sensory pathways to the dorsal horn of the spinal cord. At the level of the spinal cord, these action potentials are (3) modulated before being (4) projected to the brain for final processing and (5) conscious perception (Gaynor & Muir 2015; Meintjes 2012).

Considered in a simplified form, the pain pathway can be regarded as a chain of three orders of neurons (Lamont et al. 2000), Figure 1.4: a first–order neuron transmits information from peripheral nociceptors to a second–order neuron within the dorsal horn of the spinal cord. From there, the second–order neuron projects to the hypothalamus to a final, third–order neuron which projects within the brain from the hypothalamus to the cortex (Lamont et al. 2000).
1.3.1.1 First Order Neurons: Transduction & Transmission

The process of transduction and transmission of a noxious stimulus involves the first order neurons. These conduct information from the peripheral nociceptors (Aβ fibers, Aδ fibers and C fibers) to the macula densa in the dorsal horn of the spinal cord (Fu et al. 2011). Peripheral nociceptors respond to noxious stimuli that have the potential to cause tissue damage (Lamont et al. 2000; Meintjes 2012) transducing a noxious stimulus into an electrical signal. These various stimuli include heat, pressure, inflammatory mediators, neurotransmitters and alterations in electrolyte concentrations (Meintjes 2012).

The action potentials created by stimulation of peripheral nociceptors are transmitted to the dorsal horn of the spinal cord via their accompanying afferent (sensory) axons: Aβ fibers, Aδ fibers or C fibers. C fibers are small, unmyelinated fibers that exhibit slow conduction...
velocity (< 1.5 m/s) (Dubin & Patapoutian 2010). C fibers respond to a variety of thermal, mechanical and chemical stimuli and are thus termed polymodal nociceptors (Gaynor & Muir 2015; McKune et al. 2015). Their activation results in the sensation of slow, dull, burning, aching pain. Aδ fibers are lightly myelinated with a conduction velocity of 2 to 10 m/s and are predominantly stimulated by high intensity thermal and/or mechanical stimuli, which results in the sensation of fast pricking or sharp pain (Egger et al. 2014). Aβ fibers are large diameter, heavily myelinated afferent axons. They exhibit a very rapid conduction velocity (> 10 m/s), characteristically in response to non–noxious, mechanical stimuli.

Typically, stimulation of Aδ fiber signaling results in primary hyperalgesia or “first pain”, arising from stimulation of mechanical and thermal nociceptors (Lamont et al. 2000; Meintjes 2012). If a stimulus is of sufficient magnitude, C fibers are also activated resulting in the sensation of secondary hyperalgesia or “second pain”. Each type of afferent (sensory) fiber terminates in a specific location of the dorsal horn grey matter and stimulates a specific second order neuron on its ascent to the hypothalamus.

1.3.1.2 Second Order Neurons: Modulation & Projection

Second order neurons relay information from the dorsal horn of the spinal cord to the thalamus. The process of modulation occurs locally in the spinal cord when afferent axons enter the dorsal horn of the spinal cord carrying sensory information where they synapse within the grey matter of the spinal cord with various interneurons of the pain pathway. Following this synapsing in the dorsal horn, nociceptive modulation occurs.

There are three populations of neurons within the dorsal horn that primary afferent (sensory) axons can interact with (Lamont et al. 2000; Meintjes 2012). One type is the interneurons, which can result in inhibitory or excitatory responses and play a role in local processing of nociceptive input (Lamont et al. 2000). There are also propriospinal neurons that
extend over a number of spinal segments for modulation of reflex activity (Lamont et al. 2000). And finally, projection neurons, which have axons that travel long distances before terminating (Evans & de Lahunta 2013). For the most part, these neurons project cranially and terminate in supraspinal centers within the brain (Lamont et al. 2000; Meintjes 2012). However, the projection pathway can begin in the brain and travel caudally to activate final motor pathways in the brainstem and spinal cord (Evans & de Lahunta 2013). In humans, these pathways are referred to as ascending and descending, respectively (Evans & de Lahunta 2013).

Projection neurons are further subclassified based on the lamina of the grey matter that they are concentrated in and the type of noxious stimuli that they respond to. Nociceptive specific neurons respond to noxious thermal and mechanical input from Aδ and C afferent (sensory) fibers. They are found primarily in superficial lamina I and II (Lamont et al. 2000). Wide dynamic range neurons respond in a graded manner to low–threshold receptors as well as nociceptive input and are primarily located in lamina V (Lamont et al. 2000). Finally, complex neurons respond to somatic and visceral afferent (sensory) information and are located in lamina VII (Lamont et al. 2000). These projection neurons carry nociceptive information to supraspinal centers via one of several ascending pathways, also known as spinal tracts (Lamont et al. 2000).

1.3.1.2.1 Ascending Spinal Tracts

The ascending spinal tracts are projection neurons that travel cranially to terminate in the brain. Each ascending tract is named based on the tract’s point of origin and location of termination (Evans & de Lahunta 2013). There are three types of ascending spinal tracts that project nociceptive information from the dorsal horn of the spinal cord to the thalamus: neospinothalamic tract, paleospinothalamic tract and archispinothalamic tract (Fu et al. 2011). The grey matter of the spinal cord dorsal horn has been divided into 10 layers known as Rexed’s
laminae I–X (Meintjes 2012), Figure 1.5. The Aδ afferent fibers primarily terminate superficially in lamina I, with a few fibers that travel to terminate in lamina V (Lamont et al. 2000). The C afferent fibers terminate superficially in lamina II (Lamont et al. 2000), Figure 1.6.

![Diagram of the spinal cord](image)

**Figure 1.5.** Representation of the 10 layers (Rexed’s laminae) in the grey matter of the spinal cord (Fu et al. 2011).
The spinothalamic tract is the major ascending pathway for projection of nociceptive information to supraspinal centers. This tract is orientated vertically along the ventrolateral portion of the spinal cord (Bourne et al. 2014). Second order neurons that comprise this tract originate mainly from contralateral laminae I and IV–VI (Bourne et al. 2014). As previously mentioned, there are three pathways within the spinothalamic tract that relay nociceptive
information to the thalamus: the neospinothalamic, paleospinothalamic and archispinothalamic tracts (Fu et al. 2011).

The neospinothalamic tract is a monosynaptic pathway that projects directly to the lateral thalamus carrying information transmitted from peripheral Aδ fibers (Almeida et al. 2004); therefore it is responsible for the sensation of fast pricking or sharp pain. The paleospinothalamic tract is a multisynaptic pathway transmitting information primarily from peripheral C fibers to various regions of the thalamus (Almeida et al. 2004); and is responsible for the sensation of slow, dull, burning, aching pain. The archispinothalamic tract is a multisynaptic pathway that mediates visceral, emotional and autonomic reactions to pain transmitted via C fibers (Fenton et al. 2015).

1.3.1.3 Third Order Neurons: Perception

Third order neurons transmit information from the thalamus to the primary sensory cortex where the nociceptive signal is consciously perceived. Pain results from activation of a number of structures within the brain, rather than a single region (Okafor et al. 2014). Nociceptive neurons exist within the cerebral cortex, thalamus, hypothalamus and brainstem (Lamont et al. 2000) and allow for central processing of nociceptive information. The medulla, pons and midbrain are three structures of the brainstem that contribute to pain processing via contribution to the reticular formation and the periaqueductal gray matter (Lamont et al. 2000). The periaqueductal gray matter (PAG) is a portion of the midbrain that not only sends information to the thalamus and hypothalamus but also plays an important role in descending modulation of nociceptive information (Lamont et al. 2000) so that the nociceptive information does not escalate in intensity and repetition.
1.4 ANATOMY OF THE CANINE BRACHIAL PLEXUS

The brachial plexus has been studied in dogs and is described to originate from the ventral rami of cervical spinal nerves six (C₆), seven (C₇) and eight (C₈) and the first thoracic spinal nerve (T₁) (Allam et al. 1952; Guilherme & Benigni 2008; Mahler & Adogwa 2008; Dyce et al. 2010). There is also contribution from cervical spinal nerve five (C₅) and thoracic spinal nerve two (T₂) according to some authors (Allam et al. 1952; Guilherme & Benigni 2008; Mahler & Adogwa 2008). Similarly, in humans, spinal nerve contribution to the brachial plexus is quite variable; the brachial plexus typically originates from C₅ to C₈ and T₁ (Williams et al. 1989), but up to 60% of human patients have been documented to have contributions from cervical spinal nerves C₃ and C₄, and the thoracic spinal nerve T₂ (Williams et al. 1989). In these situations, the contribution of T₁ and C₅ is reduced, respectively.

In one of the first documented studies investigating canine brachial plexus anatomy, 58 plexuses were dissected. In 58.6% of dogs the brachial plexus had contributions from the ventral rami of spinal nerves C₆ through T₁; 20.7% of dogs had contributions from C₅ through T₁; 17.2% of dogs had contributions from C₆ through T₂; and only 3.4% of dogs had contribution from C₅ through T₂ (Allam et al. 1952).

1.4.1 Nerves of the Brachial Plexus

There are twelve nerves of the canine brachial plexus (Dyce et al. 2010; Mahler & Adogwa 2008). These nerves exit at the ventral border of the scalenus muscle before heading towards the axilla to form the brachial plexus (Allam et al. 1952; Mahler & Adogwa 2008; Campoy et al. 2010). Some nerves innervate their corresponding structures from above the plexus (long thoracic, lateral thoracic, cranial pectoral, caudal pectoral, suprascapular,
subscapular, and thoracodorsal nerves) and the rest from below the plexus (axillary, musculocutaneous, radial, median, and ulnar nerves).

Two of the nerves above the plexus provide local innervation to the surrounding musculature, namely the 1) long thoracic nerve to the serratus ventralis muscle and the 2) lateral thoracic nerve to the cutaneous trunci muscle (Dyce et al. 2010). The major contributing nerves are described below in cranial to caudal order as they emerge from the ventral aspect of the scalenus muscle. The 3) cranial pectoral nerve provides innervation to the superficial pectoral muscles (Dyce et al. 2010; Mahler & Adogwa 2008). The 4) caudal pectoral nerve provides innervation to the deep pectoral muscles (Dyce et al. 2010; Evans & de Lahunta 2013). The 5) suprascapular nerve exits from the cranial aspect of the plexus and is formed from C₆ and minor contributions of C₇ with or without a branch from C₅ (Allam et al. 1952; Guilherme & Benigni 2008; Dyce et al. 2010) and travels from medial to lateral around the cranial border of the scapular neck to innervate the supraspinatus and infraspinatus muscles (Allam et al. 1952; Mahler & Adogwa 2008; Dyce et al. 2010). The 6) subscapular nerve provides innervation to the subscapularis muscle and its origin is from contributions of C₆ and C₇. The 7) axillary nerve originates from C₇ (Allam et al. 1952; Guilherme & Benigni 2008) and flexes the shoulder through stimulation of the subscapularis, teres major, teres minor, and deltoideus muscles. At the level of the subscapularis muscle and the teres major muscle the axillary nerve splits into three branches, one of which will provide innervation to both of these muscles themselves (Allam et al. 1952). The remaining branches provide innervation to the deltoideus muscle and the teres minor muscle (Allam et al. 1952; Mahler & Adogwa 2008). The axillary nerve terminates as the lateral cutaneous brachial nerve (Allam et al. 1952). The 8) musculocutaneous nerve is composed of two nerve fascicles of C₇ (Allam et al. 1952; Guilherme & Benigni 2008) and descends to the medial surface of the thoracic limb and divides to stimulate contraction in the biceps brachii and brachialis muscles (Allam et al. 1952;
Guilherme & Benigni 2008; Mahler & Adogwa 2008), which flexes the elbow joint (Dyce et al. 2010; Lemke & Creighton 2008). In addition, the musculocutaneous nerve sends a small branch to innervate the superficial pectoral muscle (Allam et al. 1952; Guilherme & Benigni 2008) and detaches a branch at the level of the distal humerus that joins the median nerve (Allam et al. 1952). Additionally, the musculocutaneous nerve provides sensory innervation to the medial aspect of the antebrachium. The 9) thoracodorsal nerve typically originates from C7 and C8, however it has been recognized that on occasion the nerve is produced from contributions of C7 or C8 only, and very rarely from united contributions of C6, C7 and C8 (Allam et al. 1952). The thoracodorsal nerve provides innervation to the latissimus dorsi muscle (Allam et al. 1952; Dyce et al. 2010; Mahler & Adogwa 2008) and concurrent sensory innervation to the ventral thoracic and abdominal walls. The 10) radial nerve is formed predominantly from C8 and small contributions from C7 and T1 (Allam et al. 1952; Guilherme & Benigni 2008). In dogs that receive contribution of T2 to the formation of their brachial plexus, the contributing branch of T2 will add to a component to the radial nerve (Allam et al. 1952; Guilherme & Benigni 2008). The radial nerve divides into four branches and passes between the heads of the triceps to provide innervation to the extensor muscles of the upper limb, including the coracobrachialis and triceps brachii muscles, and subsequently, damage to this nerve results in a characteristic stance of the affected patient, in which the elbow and carpus cannot be extended and these joints remain flexed, such that the top surface of the paw drags on the ground. Then it continues to provide innervation to the forelimb extensors: ulnaris lateralis, lateral digital extensor, common digital extensor, extensor carpi radialis, and extensor carpi obliquus muscles (Allam et al. 1952; Guilherme & Benigni 2008; Dyce et al. 2010). The radial nerve terminates on and provides sensory innervation to the craniolateral aspect of the forelimb, carpus and digits (Mahler & Adogwa 2008). The 11) median nerve and 12) ulnar nerve reach the brachial plexus within a common sheath (Allam et al. 1952). They are formed
from C₈ and T₁ with occasional contribution from T₂ (Allam et al. 1952; Guilherme & Benigni 2008) and divide at the level of the proximal forelimb. Both nerves provide innervation to the digital flexors. Specifically, the median nerve supplies the flexor carpi radialis, superficial digital flexor, and deep digital flexor muscles; whereas the ulnar nerve innervates the flexor carpi ulnaris and deep digital flexor muscles. The ulnar nerve provides sensory innervation to the skin on the caudal aspect of the forearm, carpus and digits (Mahler & Adogwa 2008) and the median nerve provides sensory innervation to the palmar manus (Evans & de Lahunta 2013). Table 1.1 provides a summary of the twelve nerves of the canine brachial plexus along with their origin, structures innervated and major motor and sensory functions.

Table 1.1. Summary of the twelve nerves of the canine brachial plexus.

<table>
<thead>
<tr>
<th>Nerve</th>
<th>Origin</th>
<th>Innervation</th>
<th>Motor Function</th>
<th>Sensory Function</th>
</tr>
</thead>
<tbody>
<tr>
<td>Long thoracic</td>
<td>C₇</td>
<td>Serratus ventralis muscle</td>
<td>Raise thorax</td>
<td></td>
</tr>
<tr>
<td>Lateral thoracic</td>
<td>C₈ – T₁</td>
<td>Cutaneous trunci muscle</td>
<td>Twitch skin</td>
<td></td>
</tr>
<tr>
<td>Cranial pectoral</td>
<td>C₈ – C₈</td>
<td>Superficial pectoral muscles</td>
<td>Extend/adduct shoulder</td>
<td></td>
</tr>
<tr>
<td>Caudal pectoral</td>
<td>C₈ – T₁</td>
<td>Deep pectoral muscles</td>
<td>Extend/adduct shoulder</td>
<td></td>
</tr>
<tr>
<td>Suprascapular</td>
<td>C₆ – C₇</td>
<td>Supraspinatus, infraspinatus muscles</td>
<td>Extend/flex shoulder</td>
<td></td>
</tr>
<tr>
<td>Subscapular</td>
<td>C₆ – C₇</td>
<td>Subscapular muscle</td>
<td>Adduct shoulder</td>
<td></td>
</tr>
<tr>
<td>Thoracodorsal</td>
<td>C₈</td>
<td>Latissimus dorsi muscle</td>
<td>Flex shoulder, advance body</td>
<td>Ventral thoracic and abdominal wall</td>
</tr>
<tr>
<td>Axillary</td>
<td>C₇ – C₈</td>
<td>Teres major, teres minor, deltoid muscles</td>
<td>Flex shoulder</td>
<td>Cranialateral antebrachium</td>
</tr>
<tr>
<td>Musculocutaneous</td>
<td>C₆ – C₇</td>
<td>Biceps brachii, brachialis, coracobrachialis muscles</td>
<td>Flex elbow</td>
<td>Medial antebrachium</td>
</tr>
<tr>
<td>Radial</td>
<td>C₇ – T₁</td>
<td>Triceps, tensor fasciae antebrachii, anconeus, extensor carpi radialis, extensor carpi obliquus, common digital extensor, lateral digital extensor, ulnaris lateralis muscles</td>
<td>Extend elbow, carpus and digits</td>
<td>Cranial antebrachium</td>
</tr>
<tr>
<td>Median</td>
<td>C₈ – T₁</td>
<td>Flexor carpi radialis, superficial digital flexor, deep digital flexor muscles</td>
<td>Flex carpus and digits</td>
<td>Palmar manus</td>
</tr>
<tr>
<td>Ulnar</td>
<td>C₈ – T₁</td>
<td>Flexor carpi ulnaris, deep digital flexor muscles</td>
<td>Flex carpus and digits</td>
<td>Caudal antebrachium, palmar manus</td>
</tr>
</tbody>
</table>
1.5 LOCAL BLOCKADE OF THE BRACHIAL PLEXUS

Blockade of the brachial plexus with local anesthetic can be performed to provide analgesia/anesthesia and muscle relaxation for patients undergoing surgical or diagnostic procedures of the thoracic limb. The block can be used as an adjunct to general anesthesia to provide multimodal analgesia, allowing for a reduction in the overall dose of general anesthetics required and to prolong the local analgesic effect into the postoperative period to reduce the amount of systemic analgesics required to provide patient comfort (Rioja et al. 2012).

Blockade of the brachial plexus can be performed using two anatomical approaches, directly injecting the local anesthetic at the level of the brachial plexus or by injecting the ventral rami that form the brachial plexus as they emerge from the spinal cord. Direct injection of the brachial plexus is referred to in this thesis as the traditional approach (Wenger 2004; Campoy & Read 2013; Gaynor & Muir 2015; McKune et al. 2015). Injection of the ventral rami is referred to as the paravertebral approach (Lemke & Dawson 2000; Lemke & Creighton 2008; Campoy & Read 2013). Depending on the site chosen for injection of local anesthetic, the analgesia/anesthesia can include the thoracic limb from the proximal shoulder and humerus to the digits (paravertebral approach) or from the elbow to the digits (traditional approach) (Campoy & Read 2013).

1.5.1 Indications for Use

Blockade of the brachial plexus can be beneficial to the patient for any surgical or potentially painful diagnostic procedure of the thoracic limb where provision of anesthesia and analgesia is warranted. The area of the limb that is to be involved in the procedure will dictate the choice of block technique. For procedures involving the shoulder and humerus, a
paravertebral approach will be necessary to provide anesthesia to the proximal aspect of the limb (Campoy & Read 2013). For procedures involving the elbow, antebrachium and distal forelimb, blockade of the brachial plexus at the level of the axilla will provide sufficient anesthesia (Campoy & Read 2013). Alternatively, specific nerves that originate from the brachial plexus (radial, ulnar, median, and musculocutaneous) can be blocked individually on their anatomical paths for procedures that only involved circumscribed areas of the limb (Trumpatori et al. 2010).

1.5.2 Technique and Equipment Required

There are three modalities used to administer local anesthetic agents to the brachial plexus or the ventral rami: blind technique using anatomic landmarks, location of the nerves using electrical stimulation (electrostimulation or electrolocation), and location of the nerves using ultrasonography (Campoy et al. 2010; Rioja et al. 2012).

1.5.2.1 Traditional Approach with Blind Technique

The traditional axillary approach for blockade of the brachial plexus is performed using anatomic landmarks, primarily the scapulohumeral joint, as a guide for needle placement and provides anesthesia to the elbow and distal structures of the thoracic limb (Campoy & Read 2013; McKune et al. 2015).

1.5.2.1.1 Technique

The patient is positioned in lateral recumbency with the leg to be blocked up and at a standing angle (Campoy & Read 2013). The key anatomic landmarks are identified: scapulohumeral joint, acromion, greater tubercle of the humerus, jugular groove, and first rib (Campoy & Read 2013), Figure 1.7. An imaginary line is drawn to connect the acromion and
the cranial border of the greater tubercle. A second line drawn perpendicular to this one demonstrates the direction of needle advancement. A third line is drawn to indicate the position of the first rib, palpated beneath the scapula. A fourth line is drawn to trace the course of the jugular vein as it would disappear beneath the scapula. The intersection of the third and fourth lines indicates the caudal aspect of the brachial plexus and the location of the axillary artery and vein (Mahler & Adogwa 2008; Campoy & Read 2013). This is the maximum point of needle advancement (Campoy & Read 2013). The entry point of the needle is cranial to the acromion and it is advanced caudally on the medial aspect of the scapula (Campoy & Read 2013).

![Figure 1.7](image.jpg)

**Figure 1.7.** Landmarks for brachial plexus block in a dog using the traditional approach. The long red line indicates the course of the jugular groove; its most caudal point is at the first rib, which indicates the most caudal aspect of the brachial plexus. The perpendicular red line connects the acromion to the cranial border of the greater tubercle. The intersection of these two lines is the insertion point of the needle (Campoy & Read 2013).
1.5.2.1.2 Equipment and Skill Required

The traditional axillary approach is considered an intermediate level technique (Campoy & Read 2013; McKune et al. 2015). The technique can be performed using an appropriate size spinal needle (19-22 gauge) depending on the size of the animal, 0.25-0.3 mL/kg of local anesthetic (Campoy et al. 2008) and sterile gloves.

1.5.2.1.3 Advantages

This technique is advantageous, as it does not require any specialized equipment and these landmarks are readily identifiable in most canine patients.

1.5.2.1.4 Disadvantages

Although the technique is relatively easy to perform it typically requires deposition of large amounts of local anesthetic because there is no confirmation of appropriate needle placement over the plexus, which may result in an incomplete block or block failure. In addition, this approach has been documented to not reliably anesthetize structures proximal to the elbow (Lemke & Dawson 2000).

1.5.2.2 Paravertebral Approach with Blind Technique

The use of the paravertebral technique to provide anesthesia to the brachial plexus was first described by Lemke & Dawson (2000), in a review paper describing local anesthetic techniques in dogs and cats. Following investigation, poor success was achieved with the technique (Hofmeister et al. 2007), and the description of the technique was subsequently modified (Lemke & Creighton 2008). The technique claims to provide anesthesia for procedures involving the shoulder, upper thoracic limb and associated soft tissue (Lemke & Creighton 2008; Campoy & Read 2013). The key anatomical landmarks are the transverse
process of C₆ and the head of the first rib (Lemke & Dawson 2000; Lemke & Creighton 2008; Campoy & Read 2013).

### 1.5.2.2.1 Technique

For the modified paravertebral approach, the patient is placed in lateral recumbency with the limb to be blocked up and at a standing angle. The transverse process of C₆ is identified. The needle is inserted dorsal to the transverse process in a dorsoventral direction until the needle contacts the transverse process of C₆, Figure 1.8. The needle is walked off the cranial and caudal margins of the transverse process to block the ventral branches of C₆ cranially, and the ventral branches of C₇ caudally. For the other portion of the block, the head of the first rib is palpated as well as its costochondral junction. Local anesthetic is instilled at the cranial border of the first rib dorsal to the level of the costochondral junction to block the converging ventral branches of C₈ and T₁ (Lemke & Creighton 2008; Rioja et al. 2012; Campoy & Read 2013).

### 1.5.2.2.2 Equipment and Skill Required

The paravertebral approach is considered an advanced level technique (Campoy & Read 2013; McKune et al. 2015), as correct identification of the landmarks is necessary for a successful block. The technique can be performed using an appropriate size spinal needle (19-22 gauge) depending on the size of the animal, local anesthetic (1-3 mL per site) and sterile gloves (Lemke & Creighton 2008).
Figure 1.8. Landmarks for brachial plexus block in a dog using the paravertebral approach. The transverse process of C₆ and the head of the first rib are palpated to identify needle insertion points. Local anesthetic is instilled cranial and caudal to C₆ and cranial to the first rib (Hofmeister et al. 2007).

1.5.2.2.3 Advantages

The technique is theoretically advantageous over the traditional approach as it allows for more precise deposition and lower volume of local anesthetic agent (Lemke & Creighton 2008; Campoy & Read 2013), as well as providing anesthesia to the entire thoracic limb (Lemke & Dawson 2000).

1.5.2.2.4 Disadvantages

The technique is difficult to perform; even more so in overweight and obese dogs as identification of the landmarks are critical for its execution (Lemke & Creighton 2008; Campoy & Read 2013). In addition to the potential complications of brachial plexus blockade, this
technique has its own unique risks that must be considered. Inadvertent puncture of the thoracic cavity and resultant pneumothorax, intravascular injection of the vertebral arteries, epidural migration and diaphragmatic paresis are further potential complications. The phrenic nerve, which provides innervation to the diaphragm, is composed of contributions from cervical spinal nerves five, six and seven (Ogawa 1959). Therefore blockade of the ventral roots of C₆ and C₇ at this level increases the risk of hemidiaphragmatic paresis (Lemke & Creighton 2008; Campoy & Read 2013), because of the risk of paralysis of the phrenic nerve some authors recommend that this block not be performed bilaterally (Lemke & Dawson 2000). In a cadaver study that evaluated the use of ultrasonography for performing the paravertebral brachial plexus block in dogs, 20% of dogs were found to have dye staining of the phrenic nerve following performance of the block (Bagshaw et al. 2009).

The success rate of this block has been lower than anticipated. In a study using canine cadavers evaluating the success rate of the originally described technique for paravertebral forelimb block (Lemke & Dawson 2000), only 33% of the cadavers had successful staining of the ventral nerve roots of C₆, C₇, C₈ and T₁ while 66% of the cadavers had only successful staining of three of those four nerves (Hofmeister et al. 2007). A recent report investigated the success rate of this block in cadavers and compared the use of the originally described blind technique to ultrasound or electrostimulation for nerve location (Rioja et al. 2012). The study found poor success rates with all three methods and high complication rates. In particular, 29-39% of dogs developing resultant staining of new methylene blue in the spinal cord (Rioja et al. 2012), which clinically could result in life–threatening depression of the respiratory and/or cardiovascular system.

1.5.2.3 Traditional Approach with Ultrasound Technique
The use of an ultrasound–guided technique for brachial plexus blockade has been recently described (Campoy et al. 2010) and the ultrasonographic anatomy of the canine brachial plexus has been previously defined (Guilhereme & Benigni 2008). The method allows direct visualization of the nerve and needle position (Campoy & Read 2013), decreases the risk of intravascular injection, and reduces the required volume of local anesthetic (Campoy et al. 2010).

1.5.2.3.1 Technique

To perform this technique, dogs are placed in dorsal recumbency with the thoracic limbs in a naturally flexed position (Campoy et al. 2010; Campoy & Read 2013; McKune et al. 2015). The palpable anatomic landmarks are the superficial pectoral muscle, manubrium, brachiocephalicus muscle and sternocephalicus muscle, Figure 1.9. The transducer is placed in a parasagittal plane in the fossa between the manubrium and scapula. The axillary artery and vein are identified as key anatomic landmarks as the nerves of the brachial plexus lie dorsal to them. The needle is advanced craniocaudally just dorsal to the identified axillary artery and vein, and can be visualized as it comes into proximity of the nerves (Campoy et al. 2010; Campoy & Read 2013). Local anesthetic can also be visualized as it is infused around the nerves.
1.5.2.3.2 Equipment and Skill Required

This technique requires access to an ultrasound with a high frequency transducer as well as an appropriate size spinal needle (19-22 gauge) depending on the size of the animal, local anesthetic (0.15-0.2 mL/kg) and sterile gloves (Campoy et al. 2010).

1.5.2.3.3 Advantages
The use of ultrasound allows direct visualization of the individual nerves, which increases the accuracy of deposition of local anesthetic, thereby decreasing total volume required. Visualization also decreases the likelihood of intravascular or intraneural injection.

1.5.2.3.4 Disadvantages

This technique requires access to an ultrasound and the skill level to be able to identify each of the nerve roots ultrasonographically, which increases the difficulty of the block. The technique can be challenging to perform without being skilled in ultrasonography resulting in low success rates. A recent study demonstrated poor success rates in sensory nerve blockade with the use of ultrasonography to perform the traditional brachial plexus block in dogs, with only 16.7-50% of dogs developing antinociception in the skin innervated by the radial, musculocutaneous, or median and ulnar nerves (Akasaka & Shimizu 2017). Optimal position and restraint of the animal is necessary to perform this block successfully (Guilhereme & Benigni 2008). Additionally, positioning of the ultrasound transducer can be challenging in smaller patients (Rioja et al. 2012).

1.5.2.4 Paravertebral Approach with Ultrasound Technique

The use of an ultrasound–guided technique for a paravertebral approach for performing the brachial plexus block in dogs has been described in two studies (Bagshaw et al. 2009; Rioja et al. 2012). The initial study used cadaver dogs and evaluated staining of only C6, C7 and C8 (Bagshaw et al. 2009); whereas more recently, the technique was evaluated in anesthetized dogs that were euthanized following dye injection to evaluate staining of C6, C7, C8 and T1 (Rioja et al. 2012).

1.5.2.4.1 Technique
With the dog in lateral recumbency, the transverse process of C₆ is identified by scanning in a cranial to caudal direction centered on the ventrolateral aspect of the vertebrae, with the probe parallel to the dorsal plane of the dog. The intervertebral foramen between C₅ and C₆ is identified in a longitudinal plane, cranial to the transverse process of C₆ and the intervertebral foramen between C₆ and C₇ is identified caudal to the transverse process of C₆. At each foramen, an artery and respective C₆ and C₇ nerve can be seen as hypoechoic, rounded structures. The needle is inserted and local anesthetic deposited at the level of each foramen in order to block nerves C₆ and C₇ (Rioja et al. 2012). For blockade of C₈ and T₁, the probe is aligned at the cranial aspect of the thoracic inlet in a slightly oblique direction. The axillary artery and vein can be identified and followed until a bundle of hypoechoic, rounded structures with echoic septations consistent with the nerves are identified (Guilhereme & Benigni 2008; Rioja et al. 2012) deposition of local anesthetic at this location can then be visualized.

1.5.2.4.2 Equipment and Skill Required

This technique requires access to an ultrasound with a high frequency transducer (12.5 MHz) as well as an appropriate size spinal needle (19-22 gauge) depending on the size of the animal, local anesthetic (0.3 mL/kg) and sterile gloves (Rioja et al. 2012).

1.5.2.4.3 Advantages

The use of ultrasound allows direct visualization of the individual nerves, which increases the accuracy of deposition of local anesthetic, thereby decreasing total volume required. Visualization also decreases the likelihood of intravascular or intraneural injection.

1.5.2.4.4 Disadvantages
This technique requires access to an ultrasound and the skill level to be able to identify each of the nerve roots ultrasonographically, which increases the difficulty of the block. In one study, this technique took significantly more time (6.3 ± 2.7 min) to perform than a blind technique (3.6 ± 1.8 min), but was faster than electrostimulation (12.2 ± 5 min). Despite the skill of an experienced operator, the success of staining of all four nerves was only 9%, due to difficult visualization of the nerves and the appropriate landmarks (Rioja et al. 2012).

1.5.2.5 Electrostimulation with Traditional Approach or Paravertebral Approach

The use of electrolocation using a peripheral nerve stimulator can be applied to any of the above-mentioned techniques for performing the brachial plexus block in dogs in an attempt to improve precision (Mahler & Reece 2007; Campoy et al. 2010; Rioja et al. 2012; Campoy & Read 2013; McKune et al. 2015) and success rate (Mahler & Adogwa 2008; Ricco et al. 2013; Akasaka & Shimizu 2017) of the block.

1.5.2.5.1 Technique with Traditional Approach

The positive electrode is connected to the patient and electrostimulation applies an electric current to the peripheral nerve that will result in a corresponding muscle contraction if the proximity is appropriate (Futema et al. 2002; Mahler & Adogwa 2008; Campoy & Read 2013; McKune et al. 2015). The initial current on the peripheral nerve stimulator is set at 1.0 to 1.5 mA (McKune et al. 2015) and gradually decreased to a threshold current of 0.4 mA that elicits a muscle response to verify appropriate needle position (Campoy & Read 2013; McKune et al. 2015). The current is typically delivered at a frequency of 2 Hz for 0.1-0.2 milliseconds (Lenke & Creighton 2008), Figure 1.10.
1.5.2.5.2 Technique with Paravertebral Approach

Using the same landmarks as the blind technique, an electrical stimulus of 1 mA current, 0.1 ms duration, at a frequency of 1 Hz is delivered. When muscle contractions associated with each of the nerves occurs, the current is decreased in decrements of 0.2 mA until the muscle contractions disappear. Once muscle contractions disappear, the current is increase by 0.2 mA to obtain muscle contractions again and the local anesthetic is injected (Rioja et al. 2012).

At the proximal level of the brachial plexus, electrostimulation of the ventral nerve root of C₆ results in rotation of the shoulder. Electrostimulation of C₇ results in contraction of the biceps, triceps and extension of the carpus. Electrostimulation of C₈ causes extension of the elbow, carpus and digits. Electrostimulation of T₁ results in flexion of the carpus and digits (Campoy & Read 2013).
1.5.2.5.3 Equipment and Skill Required

Blockade of the brachial plexus by electrostimulation requires use of a peripheral nerve stimulator and an insulated needle, local anesthetic (0.25-0.3 mL/kg) and sterile gloves. For brachial plexus block in most dogs a 22-gauge, 1.5-inch pinpoint tip insulated needle is appropriate (Lemke & Creighton 2008; Rioja et al. 2012).

1.5.2.5.4 Advantages

This technique is advantageous because it provides electrophysiologic confirmation of nerve identification and needle placement. It may also shorten the time to onset of the block and prolong the duration of action as a result of increased accuracy (Lemke & Creighton 2008).

1.5.2.5.5 Disadvantages

The technique of electrostimulation places the needle tip in very close proximity to the nerve, potentially increasing the risk of intraneural injection. High pressure or resistance at the onset of injection of local anesthetic may indicate intraneural injection and the needle should be repositioned to prevent potential nerve damage (Mahler & Adogwa 2008). For the traditional approach, the addition of electrolocation was found to result in low success rate for sensory blockade with only 16.7-50% of dogs developing antinociception present in the skin innervated by the radial, musculocutaneous, or median and ulnar nerves (Akasaka & Shimizu 2017). For the paravertebral approach, this technique required a longer time to complete than ultrasound–guided or blind injection of the brachial plexus, and it was only effective in staining one of the four nerves upon examination of dogs in necropsy after brachial plexus injection while under anesthesia (Rioja et al. 2012).

1.5.3 Potential Complications of Brachial Plexus Block
Potential complications of performing the brachial plexus block include blockade failure, inadvertent intravascular puncture/injection, intraneural injection or damage to surrounding structures, epidural or spinal anesthesia, phrenic nerve block and pneumothorax (Lemke & Creighton 2008; Mahler & Adogwa 2008; Campoy & Read 2013; Rioja et al. 2012). Horner’s syndrome secondary to brachial plexus blockade, although rare, has also been reported in a dog (Viscasillas et al. 2013).

1.5.3.1 Blockade Failure

Partial or complete failure of local block is a possibility with any of the described techniques. Failure can result from inadequate or incomplete contact of all or any of the nerves that compose the brachial plexus with the local anesthetic. The initial evaluation of brachial plexus blockade in dogs using the traditional approach reported good to excellent results in 86% of cases (Nutt, 1962). Moens & Caulkett (2000) reported a tendency of nerves in the cranial aspect of the brachial plexus to demonstrate a more rapid onset of block and longer duration of action when compared to nerves in the caudal aspect of the brachial plexus as a result of channelization of local anesthetic agent toward the cranial aspect of the axilla.

Literature evaluating the success of the paravertebral approach for brachial plexus block in dogs is limited. In a cadaveric study evaluating the initially described technique of the paravertebral block it was reported that only one third (33%) of the dogs had the ventral nerve rami of C6, C7, C8 and T1 were successfully stained while the remaining two thirds (66%) of the dogs had only three of the four nerve rami forming the brachial plexus successfully stained (Hofmeister et al. 2007). Even poorer results were establish in a more recent evaluation of the technique, with the ventral nerve rami of C6, C7, C8 and T1 demonstrating staining in only 17% of dogs using the revised technique for paravertebral block of the brachial plexus.
(Rioja et al. 2012). In addition, no improvement in success rate was demonstrated using peripheral nerve stimulation or ultrasound guidance (Rioja et al. 2012).

Electrostimulation has been described to perform a number of peripheral nerve block techniques in dogs. In papers describing performance of the canine brachial plexus block utilizing electrolocation, the success rate varies between 91.6-100% (Futema et al. 2002; Ricco et al. 2013), with improved success of nerve staining when higher volumes of injectate were used (Campoy et al. 2008). The paper that describes the use of ultrasonography for blockade of the canine brachial plexus reports that C7, C8 and T1 were successfully stained in all dogs, whereas C6 was not sufficiently stained in any of the dogs (Campoy et al. 2010).

1.5.3.2 Intravascular Injection, Hemorrhage

Inadvertent intravascular injection is a possible complication as the arteries and veins that provide the blood supply to the thoracic limb run in close proximity to the nerve plexus (Dyce et al. 2010). Intravascular injection can be avoided by aspirating back prior to injection of local anesthetic. Hemorrhage and subsequent hematoma formation from inadvertent laceration of a peripheral vessel is difficult to avoid when using a blind technique but is not a frequently reported complication. Studies documenting the risk of hemorrhage and hematoma development are limited. Hematoma formation has been documented to range from 7.1-21.4% when performing the brachial plexus block in dogs via a blind approach or with electrolocation (Ricco et al. 2013). In a feline study evaluating brachial plexus block techniques using ultrasonography, jugular hematoma was reported in 33.3% of the cats (Anson et al. 2015).

1.5.3.3 Epidural or Spinal Anesthesia

Rioja et al. (2012) evaluated complications associated with the paravertebral approach to block the brachial plexus in canine cadavers, and found staining of the spinal cord to be a
common complication with an incidence of 29-39%. Although no clinical reports of this complication exist, cervical spinal anesthesia could result in unilateral or bilateral phrenic nerve paralysis and impairment of diaphragmatic function with life–threatening cardiopulmonary complications.

1.5.3.4 Phrenic Nerve Block

The phrenic nerve is formed by the ventral braches of cervical spinal nerves five, six and seven (Ogawa 1959; Dyce et al. 2010). The risk of paralysis of the phrenic nerve is increased when performing the paravertebral approach to brachial plexus block, with a reported incidence of 20% when using high volumes of local anesthetic (Bagshaw et al. 2009). Although in healthy dogs, unilateral or bilateral blockade of the phrenic nerve does not appear to have negative implications for pulmonary function, it is not recommended that these blocks be performed in dogs with cardiopulmonary compromise (Lemke & Creighton 2008). In feline patients the risk of phrenic nerve block may be higher than that in dogs, one cadaver study documented straining of the phrenic nerve in 50% of cats (Anson et al. 2015).

1.5.3.5 Pneumothorax

Puncture of the thoracic cavity at the level of the thoracic inlet is a potential complication of the brachial plexus block. The incidence of staining of the visceral pleura in cadaver dogs has been reported to be 4-13%, indicating entry into the thoracic cavity (Rioja et al. 2012). Pneumothorax following brachial plexus blockade is a rare but potentially severe complication. A single case report of unilateral pneumothorax in a dog following brachial plexus block via the traditional approach using a peripheral nerve stimulator has been documented (Bhalla & Leece 2015). The pneumothorax became evident 16 hours post block.
and treatment involved thoracentesis and placement of a chest tube. The pneumothorax resolved by 48 hours following the brachial plexus block.

1.5.3.6 Horner’s Syndrome

Horner’s syndrome is characterized by myosis, ptosis, enophthalmos and prolapse of the nictitating membrane, secondary to a disorder of the sympathetic nerves that supply the eye. A case report of the development of Horner’s Syndrome in a dog following brachial plexus block via the paravertebral approach has been documented (Viscasillas et al. 2012). The dog was noted to have developed Horner’s syndrome following recovery from anesthesia, which disappeared when the patient’s motor and sensory function returned to the forelimb. It was hypothesized that deposition of local anesthetic close to the first rib resulted in paralysis at the level of the cervicothoracic ganglia and subsequently, transient Horner’s syndrome (Viscasillas et al. 2012). A recent clinical study evaluating the paravertebral block in dogs with lidocaine or lidocaine combined with epinephrine documented the development of Horner’s syndrome in 25% of dogs that persisted for 130 minutes following the dogs’ recovery from anesthesia (Choquette et al. 2017).

1.5.4 Species Comparison of Techniques

Blockade of the brachial plexus for provision of anesthesia and analgesia of the thoracic limb is commonly utilized in human patients and has been well described in the dog (Campoy & Read 2013). Blockade of the brachial plexus has been investigated and described in a number of other veterinary species to provide anesthesia and analgesia for surgical and diagnostic procedures. These include: feline, avian, bovine, and ovine species (Anson et al. 2013; Brenner et al. 2010; da Cunha et al. 2013; Figueiredo et al. 2008; Iwamoto et al. 2012; Mencalha et al. 2014; Mosing et al. 2010).
1.5.4.1 Brachial Plexus Blockade in the Feline Patient

The anatomy of the brachial plexus in cats varies considerably from dogs, being reported to originate from C₆, C₇, C₈, T₁ and T₂ (Mencalha et al. 2014) and with the nerves that are located within the connective tissue of the axillary space being limited to the suprascapular, subscapular, axillary, musculocutaneous, radial, median and ulnar nerves in cats (Anson et al. 2013; Mencalha et al. 2014; Anson et al. 2015). Identification and discussion of the pectoral nerves, lateral thoracic nerve, long thoracic nerve and thoracodorsal nerve with respect to the feline brachial plexus is lacking in these papers. One study reports the existence of a “double” subscapular nerve, labeling these nerves as subscapular nerve I and subscapular nerve II (Mencalha et al. 2014). Limited literature exists describing techniques for brachial plexus blockade specifically in cats, and it is usually performed using anatomic landmarks via the traditional approach as described for dogs. Electrostimulation has been used in conjunction with the traditional approach to brachial plexus blockade to improve the success rate of the block, and clinically the technique was found to be inhalant sparing and improved postoperative analgesia in cats undergoing orthopedic surgery of the thoracic limb (Mosing et al. 2010). With the recent characterization of the ultrasonographic appearance of the feline brachial plexus (Anson et al. 2013), an ultrasonographic technique for brachial plexus blockade in cats has been described in feline cadavers (Anson et al. 2015). For this technique to be successful the cat is placed in dorsal recumbency with the limb to be blocked abducted 90° to the body wall and the ultrasound transducer positioned parallel to the spine to identify the nerve roots of the brachial plexus (Anson et al. 2015), which is similar to the description of the technique for canine patients. The application of the ultrasonographic technique for performing the brachial plexus block in feline patients has not yet been evaluated in a clinical setting.
1.5.4.2 Brachial Plexus Blockade in the Avian Patient

Although surgical procedures of the wing are very common in avian patients, literature on performing the brachial plexus blockade and the subsequent anesthesia and analgesia is limited. The anatomic location and nerve composition of the avian brachial plexus is highly variable between species (da Cunha et al. 2013). Typically, the brachial plexus is comprised of four spinal nerves. The brachial plexus of ducks (*Anas platyrhynchos*) and geese (*Anser anser*) is derived from the last two cervical spinal nerves and the first two thoracic spinal nerves (Brenner et al. 2010). The existing literature suggests that the technique is difficult to perform successfully in avian species. A study evaluating two novel techniques for brachial plexus blockade in mallard ducks (*Anas platyrhynchos*) has been reported. The study evaluated an axillary approach and a dorsal approach to block the brachial plexus in mallard ducks (*Anas platyrhynchos*) (Brenner et al. 2010). For the axillary approach, the ducks were placed in lateral recumbency with the test wing up and suspended. In birds, the brachial plexus lies craniodorsally in a depression in the axillary space formed by the medial edge of the pectoralis muscle, the cranial edge of the biceps brachii muscle and the dorsal aspect of the serratus ventralis muscle (Brenner et al. 2010). These muscle margins were used as the landmark to perform this block. For the dorsal approach, the ducks were positioned the same way and the brachial plexus was approached from the dorsal aspect of the bird. From this approach, the brachial plexus is located beneath the scapula cranioventral to the depression formed between the last cervical vertebra and the first thoracic vertebra, which was used as the landmark to perform this block. Regardless of the technique evaluated to perform the brachial plexus block, the authors found inconsistent motor and sensory block (Brenner et al. 2010). An earlier study that evaluated an axillary approach to brachial plexus blockade with the use of a nerve stimulator in chickens found that the block was easy to perform but was also associated with a
high rate of failure (Figueiredo et al. 2008). The brachial plexus in chickens is near the ventral border of the scapular caudal to the first rib and coracoid bone. This study used these orthopedic landmarks and advanced the needle through the pectoralis muscle to a region around the first rib. Flexion of the wing during electrostimulation was considered to indicate appropriate proximity to the brachial plexus and the local anesthetic was injected (Figueiredo et al. 2008). Recently, an ultrasound–guided technique for brachial plexus blockade was evaluated in Hispaniolan Amazon parrots (Amazônia ventralis) and compared to a blind brachial plexus block performed based on anatomical landmarks (da Cunha et al. 2013). For the blind approach, the birds were placed in dorsal recumbency with the limb to be blocked extended. The brachial plexus was approached from the caudal axilla and the space formed between the pectoral muscle, triceps muscle and supracoracoideus aticimus muscle was the insertion point for the needed. The ultrasound–guided technique was not found to be efficacious in providing sensory or motor block (da Cunha et al. 2013). The current literature describing the brachial plexus block in avian patients lacks consistency with respect to the technique itself, likely as a result of species variation.

1.5.4.3 Brachial Plexus Blockade in the Bovine Patient

Local anesthetic techniques have become quite useful in bovine medicine, particularly during field procedures. Similar to dogs, the brachial plexus in cattle is derived from cervical spinal nerve six, seven, eight and the first thoracic spinal nerve (Iwamoto et al. 2012). The technique for brachial plexus blockade in cattle is typically performed blind with the use of anatomical landmarks and presents similar complications to those listed for canine patients (Thurmon & Ko, 1997). Recently, the ultrasonographic appearance of the brachial plexus in calves has been reported and subsequently an ultrasound–guided technique for brachial plexus blockade was described (Iwamoto et al. 2012). Sensory and motor blockade of the axillary,
radial, musculocutaneous, median and ulnar nerves was evaluated. The technique only resulted in consistent sensory blockade in the region innervated by the musculocutaneous nerve. Motor blockade of short duration was successful in 87.5% of blocks (Iwamoto et al. 2012).

1.5.4.4 Brachial Plexus Blockade in the Ovine Patient

Similar to bovine patients, the brachial plexus in sheep is derived from the ventral nerve roots of cervical spinal nerve six, seven, eight and the first thoracic spinal nerve (Ghadirian et al. 2016). Brachial plexus blockade has been achieved in sheep using a peripheral nerve stimulator to identify the nerves of the brachial plexus via a technique similar to the traditional approach described in dogs, which was reported to be easily performed, (Ghadirian et al. 2016) as well as via a paravertebral approach (Rodrigo-Mocholi & Schauvliege 2016). The paravertebral approach was reported to successfully provide analgesia by permitting a stable plan of anesthesia during thoracic limb amputation (Rodrigo-Mocholi & Schauvliege 2016).
1.6 DRUGS FOR BLOCKADE OF THE BRACHIAL PLEXUS

1.6.1 Local Anesthetics

All local anesthetics have a common chemical arrangement, which is utilized for their classification. Local anesthetics are weak bases are composed of a lipophilic aromatic ring, hydrophilic tertiary amide which are linked together by an intermediate hydrocarbon chain (Becker & Reed 2006). The classification of local anesthetics is based on the makeup of this intermediate chain, comprising either an amide or ester linkage (Becker & Reed 2006), Figure 1.11.

![Chemical structure of an ester local anesthetic (left) and an amide local anesthetic (right) (Campoy & Read 2013).](image)

**Figure 1.11.** Chemical structure of an ester local anesthetic (left) and an amide local anesthetic (right) (Campoy & Read 2013).

Local anesthetics administered as a depot in close proximity to peripheral nerves prevent the transmission of somatic, motor, and autonomic nerve conduction by reversible inhibition of sodium channels on the nerve cell membrane (Becker & Reed 2006). In addition, systemic administration of lidocaine has been found to have a number of other benefits including analgesic, anti-inflammatory, and antiarrhythmic effects, through different mechanisms of action. In human patients, lidocaine and procainamide demonstrate ventricular antiarrhythmic properties by elevating the electrical stimulation threshold (Harrison et al. 1963). Local anesthetics are thought to exhibit an anti-inflammatory effect via reducing the formation of leukotriene B4, which is a potent stimulator of polymorphonuclear granulocytes.
and cause inhibition of free radical release from macrophages (Suzuki et al. 2013). Without activation of these cells, the inflammatory response is markedly suppressed. These anti-inflammatory properties are thought to be the mechanism by which lidocaine decreases the length of time that horses hospitalized for colic experienced postoperative ileus and nasogastric reflux (Cook & Blikslager 2008).

1.6.1.1 Physiology of the Sodium Channel

The voltage-gated sodium channel is a four domain transmembrane protein with a central ion pore (Payandeh et al. 2011). The sodium channel has three forms: activated (open), inactivated and resting (closed). At resting membrane potential the voltage-gated sodium channel is in its resting (closed) state and the ion pore is closed (Suzuki et al. 2013; Grimm et al. 2015). The voltage-gated sodium channel has two gates: an activation gate on the extracellular surface of the channel and an inactivation gate on the intracellular surface of the channel. Cell depolarization causes the voltage-gated sodium channel to activate (open) to allow for an influx of sodium ions to pass through the channel and enter the cell (Suzuki et al. 2013; Grimm et al. 2015). Within milliseconds the intracellular inactivation gate of the sodium channel closes and the channel becomes inactivated while the cell membrane undergoes repolarization. Repolarization of the cell membrane leads to closure of the activation gate and opening of the inactivation gate and the voltage-gated sodium channel returns to the resting (closed) state (Suzuki et al. 2013; Grimm et al. 2015).

1.6.1.2 Mechanism of Action for Nerve Block

To exhibit their mechanism of action on an individual nerve cell, local anesthetics must diffuse through the plasma cell membrane because the binding site on the voltage-gated sodium channel is on the intracellular portion of the ion channel (Becker & Reed 2006). The
more acidic pH within the cell favours the protonated form of the local anesthetic, which antagonizes the sodium channel (Suzuki et al. 2013). Local anesthetic drugs can reach the interior binding site via the hydrophilic pathway through the pore interior from the cytoplasmic side of the cell or the hydrophobic pathway through the lipid cell membrane (Nau & Wang 2004; Suzuki et al. 2013). Local anesthetics preferentially bind voltage-gated sodium channels that are in the activated and inactivated state (Suzuki et al. 2013), Figure 1.12.

The higher affinity that local anesthetics demonstrate for sodium channels in the activated and inactive states is because the binding site of local anesthetics is more accessible with the channel in this state than in the resting state (Suzuki et al. 2013), as well as a postulated increase in affinity secondary to the conformation change that the sodium channel undergoes during activation (Nau & Wang 2004). Once bound, local anesthetics inhibit further activation of the sodium channel by maintaining it in the inactivated state (Suzuki et al. 2013). Local anesthetics inhibit sodium channel conduction by generating reversible reduction of the sodium current and preventing passage of the electrolyte through their selective ion channel therefore preventing transmission of nerve impulses, which has been termed a conduction blockade (Lin & Liu 2013; Maheshwari & Naguib 2015; Nau & Wang 2004). Failure of passage of sodium ions through the sodium channel results in the inability of the threshold potential to be reached and therefore an action potential cannot be propagated (Lin & Liu 2013; Maheshwari & Naguib 2015).
Figure 1.12. Different states of voltage–gated sodium channel. At resting membrane potential the channel is closed. Depolarization results in channel opening and passage of sodium into the cell. During repolarization, the channel is inactivated following closure of the inactivation gate. Local anesthetics (LA) preferentially bind activated and inactivated state (Suzuki et al. 2013).

Local anesthetics exhibit use–dependent block, meaning that they bind to the voltage–gated sodium channels when they are depolarized (activated) (Balser et al. 1996) and the block becomes cumulative, with more local anesthetic becoming bound to the sodium channels the
more frequently the channel is opened (Wann 1993). The drug is released from the sodium channel during states of hyperpolarization and rest as the affinity for the channel declines (Balser et al. 1996). When associated with local anesthetic drug this repriming (return from the inactivated state back to the resting state) of the sodium channel occurs more slowly (Balser et al. 1996). Local anesthetics also block voltage-gated potassium and calcium channels, however the clinical significance of this remains unclear (Suzuki et al. 2013).

1.6.1.3 Specific Local Anesthetic Pharmacologic Comparisons

1.6.1.3.1 Lidocaine

Lidocaine is the most commonly used local anesthetic in veterinary medicine. Lidocaine is an aminoamide local anesthetic and has a pKa of 7.9 (Campoy & Read 2013; Egger et al. 2014). Its onset of action is rapid, taking effect within 5-10 minutes of local infiltration and the duration of effect is relatively short, lasting 1-2 hours (Egger et al. 2014) making it an ideal local anesthetic for rapid procedures it which persistent sensory and motor blockade is unnecessary. Lidocaine is approved for use in small and large animal species in North America and is available in many formulations including a 10% topical spray and 2% solution (Egger et al. 2014).

1.6.1.3.2 Bupivacaine

Bupivacaine is an aminoamide local anesthetic and has a pKa of 8.1 (Campoy & Read 2013; Egger et al. 2014). Bupivacaine is four times as potent as lidocaine and its onset of action is relatively slow in comparison to lidocaine, taking effect within 10-20 minutes of local infiltration and the duration of effect is long, lasting 3-6 hours (Egger et al. 2014), making it advantageous to patients enduring procedures that require prolonged anesthetic and analgesic
effects. Bupivacaine has not been approved for use in veterinary species in North America but is gaining popularity in small animal medicine. Bupivacaine is available in many formulations including 0.25%, 0.5% and 0.75% solutions (Egger et al. 2014).

1.6.1.3.3 Mepivacaine

Mepivacaine is an aminoamide local anesthetic with a pKa of 7.7 and a very similar profile to lidocaine being commonly used in both small and large animal species, being approved for use in dogs and horses (Campoy & Read 2013; Egger et al. 2014). Its onset of action is very similar to lidocaine, taking effect within 5-10 minutes however the duration of action is slightly longer (Egger et al. 2014). It is available in a 2% solution (Egger et al. 2014).

1.6.1.3.4 Ropivacaine

Ropivacaine is an aminoamide local anesthetic with a pKa of 8.1 that does not have approval for use in veterinary species (Campoy & Read 2013; Egger et al. 2014). Its onset of action is slow, taking effect within 10-20 minutes of local infiltration and the duration of effect is long, lasting 2-4 hours (Egger et al. 2014). It is available as a 0.75% solution and other formulations (Egger et al. 2014). Ropivacaine is a newer local anesthetic and has gained popularity due to an improved safety profile with respect to toxicity and a decreased efficacy for causing motor blockade (Sakonju et al. 2009). In a study comparing the brachial plexus block in dogs using bupivacaine versus ropivacaine, no difference was noted with respect to onset of action; however ropivacaine was noted to be of shorter duration of action (Sakonju et al. 2009).

1.6.1.4 Toxicity of Local Anesthetics
With the administration of a local anesthetic agent for the provision of anesthesia and analgesia, there also exists the potential risk of toxicity to the patient. Local anesthetic drugs exhibit a toxic effect through the same mechanism by which they act as analgesic and anesthetic agents, via blockade of the sodium channel. Toxicity from local anesthetic drugs may result from overdose as a result of accidental intravascular injection, incorrect dose calculation when being administered systemically, or disease conditions that result in decreased drug clearance and sustained high plasma concentrations.

The manifestations of systemic toxicity to local anesthetic drugs are dose–dependent and related to the achieved plasma concentrations. Therefore, they are also affected by the speed of absorption from the injection site and the clearance of the drug from the body, the latter being species dependent. Peak plasma concentrations are achieved faster from different injection sites; in general peak plasma concentrations following intercostal injection are, > epidural, > brachial plexus, > infiltration > subcutaneous administration (Tucker & Mather 1979); and cats are more sensitive to the toxic effects of local anesthetics than dogs.

The central nervous system and the cardiovascular system are affected in that order in a dose–dependent fashion. The lipid solubility of local anesthetics allows them to cross the blood–brain barrier to produce central nervous system toxicity (Groban 2003). Within the central nervous system, local anesthetics depress cortical inhibitory pathways allowing for persistent activity of excitatory pathways, which manifests as nystagmus, muscular twitching and seizure activity (Groban 2003). With the exception of bupivacaine, most local anesthetics require larger systemic doses to produce clinical signs of cardiovascular toxicity than central nervous system toxicity (Groban 2003). The cardiotoxic effect of local anesthetics is variable between agents, but in general they result in depression of myocardial contractility and impulse conduction velocity (Groban 2003).
Although toxic doses of local anesthetic to the central nervous system are well tolerated in anesthetized patients due to the protective effects of central nervous system depression associated with unconsciousness (Copeland et al. 2008), cardiovascular signs will equally occur in conscious and anesthetized patients after exposure to similar doses (Copeland et al. 2008).

1.6.1.4.1 Toxic Doses of Lidocaine

In conscious dogs, signs of CNS toxicity manifest after a cumulative intravenous dose of 22 mg/kg (Liu et al. 1983). The dose reported to produce irreversible cardiovascular toxicity in conscious dogs was 3.5-6.7 times that which produced clinical signs of CNS toxicity (Liu et al. 1983). Signs of CNS excitement; including tremors, sedation, salivation, rigidity, and convulsions, can be observed following a single intravenous dose of < 10 mg/kg (Liu et al. 1983). The single intravenous dose resulting in cardiovascular collapse in anesthetized dogs is reported to be 28 mg/kg, with fatality resulting from progressive hypotension, bradycardia and asystole (Liu et al. 1982); however, intravenous doses between 3-10 mg/kg can begin to decrease stroke volume and cardiac output (Liu et al. 1982).

1.6.1.4.2 Toxic Doses of Bupivacaine

The doses of bupivacaine that result in signs of toxicity are much less than those reported for lidocaine since it is a more potent local anesthetic. In conscious dogs, signs of CNS toxicity manifest after a cumulative intravenous dose of 5 mg/kg (Liu et al. 1983). Signs of CNS excitement, including tremors, salivation, rigidity and convulsions, can be observed following a single intravenous dose of 3 mg/kg (Liu et al. 1983). Similar to lidocaine, the dose reported to produce irreversible cardiovascular toxicity in conscious dogs was 3.5-6.7 times that which produced clinical signs of CNS toxicity (Liu et al. 1983). The single intravenous
dose resulting in cardiovascular collapse in anesthetized dogs is reported to be 11 mg/kg, with fatality resulting from progressive hypotension, bradycardia and asystole (Liu et al. 1982). Marked decreases in stroke volume and cardiac output can begin to occur at intravenous doses of 3 mg/kg (Liu et al. 1982) and therefore, the intravenous route of administration is not recommended for bupivacaine.

1.6.2 Other Adjuvants

With respect to peripheral nerve blocks, adjuvants are agents that are used in combination with the local anesthetic drug to improve the effectiveness and duration of effect of both epidural and peripheral nerve block.

1.6.2.1 Vasoconstrictors

Epinephrine has been frequently combined with local anesthetic drugs prior to administration (Moens & Caulkett 2000; Futema et al. 2002; Brenner et al. 2010) and has a number of beneficial effects (Campoy & Read 2013). The addition of epinephrine will increase the intensity and duration of blockade, reduce systemic absorption of the drug and decrease the intensity of surgical bleeding as a result of local vasoconstriction (Campoy & Read 2013).

The dual blood supply to peripheral nerves through an extrinsic and intrinsic source contributes to the actions of vasoconstrictors in prolonging the duration of blockade. The extrinsic artery is responsive to adrenergic stimulation (Neal 2003), which retards the rapid removal of local anesthetic from the epineurial compartment and allows more local anesthetic to enter the deeper perineurial compartment early in the block (Sinnott et al. 2003). At the endoneurial compartment, the prolonged duration of block by vasoconstrictors appears to correspond to an enlarged local anesthetic content in nerve at later times, from a very slowly emptying from this compartment (Sinnott et al. 2003).
In a recent study, the efficacy and duration of the modified paravertebral brachial plexus block in dogs using lidocaine or lidocaine in combination with epinephrine was evaluated. It was found that the addition of epinephrine did not result in an increased duration of sensory blockade, however it did result in significantly longer motor blockade as evident by gait abnormalities in the dogs for up to 180 minutes post block administration (Choquette et al. 2017).

1.6.2.2 Alpha 2 (α2) Agonists

The combination of α2–agonists and local anesthetics for peripheral nerve blockade has been found to prolong the duration of action of the local anesthetic via actions of the α2–agonist on the peripheral α2A receptor (Campoy & Read 2013). It has been demonstrated that in combination with local anesthetic agents, dexmedetomidine provides a dose–dependent increase in the duration of local anesthetic sensory blockade in rats (Brummett et al. 2009) and humans (Esmaoglu et al. 2010; Yoshitomi et al. 2008). In these species, dexmedetomidine has been evaluated in combination with lidocaine (Yoshitomi et al. 2008), levobupivacaine (Esmaoglu et al. 2010) and ropivacaine (Brummett et al. 2009) and increased the interval of sensory blockade when combined with either local anesthetic agent. Clonidine was also demonstrated to increase the duration and degree of local anesthetic blockade in humans (Yoshitomi et al. 2008).

In dogs, the addition of α2–agonists to local anesthetic agents for peripheral nerve blockade has demonstrated inconsistent results. The addition of medetomidine to mepivacaine for peripheral radial nerve block prolonged sensory and motor blockade when compared to mepivacaine alone (Lamont & Lemke 2008). However, no significant differences in onset or duration of sensory or motor blockade of the femoral and sciatic nerves were found when
blocks were performed in dogs using ropivacaine alone or ropivacaine in combination with dexmedetomidine (Trein et al. 2017).

A number of possible mechanisms for this prolonged duration of action of the local anesthetic when combined with an α2–agonist have been proposed, the first being delayed absorption of the local anesthetic drug as a result of vasoconstriction (Yoshitomi et al. 2008), very similar to the mechanism described for epinephrine. The second is direct inhibition of peripheral nerve impulse conduction (Yoshitomi et al. 2008). More recently, it has been suggested that α2–agonists prolong peripheral nerve blocks by inhibiting the hyperpolarization–activated cation current (Ih), which is important to reestablish the resting membrane potential (Trein et al. 2017). This inhibition maintains the peripheral nerve in a hyperpolarized state and inhibits conduction of new action potentials.

1.6.2.3 Opioids

The combinations of opioids and local anesthetics are primarily used for epidural anesthesia and analgesia, however they are sometimes combined for administration of peripheral nerve blockade to enhance the quality of the nerve block (Campoy & Read 2013). In the human literature the use of various opioids to enhance peripheral nerve blocks has provided conflicting results. However, it has been consistently reported that buprenorphine enhances and prolongs the effect of peripheral nerve blocks when combined with local anesthetics (Candido et al. 2010; Leffler et al. 2012). Unlike other opioids, it has been demonstrated that buprenorphine behaves like a local anesthetic in that it results in use dependent block of voltage gated sodium channels on the nerve cell membrane (Leffler et al. 2012). Although evidence for the use of buprenorphine as an adjuvant are encouraging, reports of the use of opioids for this purpose in veterinary species are very limited. A recent study in lambs demonstrated that the addition of morphine or tramadol to the local anesthetic for
blockade of the brachial plexus did not affect the onset or duration of peripheral sensory blockade (Ghadirian et al. 2016).
1.7 RATIONALE, HYPOTHESIS, AND OBJECTIVES

Blockade of the brachial plexus with local anesthetics provides anesthesia and analgesia, and improves patient comfort postoperatively when performed in canine patients undergoing surgical procedures of the thoracic limb. This block is most commonly performed using a blind approach that uses specific anatomical landmarks for appropriate needle placement. However, the description does not establish the exact end point for needle position, which may lead to block failure. The inclusion of ultrasonography or electrolocation for performance of the canine brachial plexus block have both been described to guide the injection and better locate the delivery of the local anesthetic; however, utilization of these modalities requires a certain level of skill of the operator, requires specialized equipment, and is not exempt of failure. A recent study has demonstrated poor success rates with both imaging modalities (Akasaka & Shimizu 2017).

There were three objectives of this study. The first objective was to dissect and describe the ventral spinal nerve rami that contributed to the formation of the canine brachial plexus and each of its nerves. The second objective was to compare dye distribution and nerve staining, between the currently described traditional blind technique for brachial plexus blockade and two novel blind approaches for brachial plexus blockade in dogs: axillary and perpendicular approaches. The final objective was to compare the degree of anesthesia and analgesia resulting from these three techniques when used on clinical cases of dogs undergoing a surgical procedure of the thoracic limb. The hypotheses were that the novel approaches would be alternatives to the traditional approach for performing the brachial plexus block in dogs, and that the degree of anesthesia and analgesia would not differ between the techniques.
1.8 REFERENCES


CHAPTER 2

ANATOMICAL CHARACTERIZATION OF THE BRACHIAL PLEXUS IN DOG CADAVERS AND COMPARISON OF THREE BLIND TECHNIQUES FOR BRACHIAL PLEXUS BLOCKADE
2.1 SUMMARY

**Objective:** To describe the ventral spinal nerve rami contribution to the formation of the brachial plexus (BP) and to compare ease of performing and nerve staining between three blind techniques for BP blockade in dogs.

**Study design:** Prospective, randomized, blind study.

**Animals:** Eighteen dog cadavers weighing 28.2 ± 9.7 kg (mean ± standard deviation).

**Methods:** Dogs were randomly assigned to one of three BP treatments: TA- traditional approach, PA- perpendicular; or AA- axillary. Dye (0.2 mL kg\(^{-1}\)) was injected in the left BP using a spinal needle; another BP treatment was used in the right BP. Landmarks (L) included: L1, midpoint between point of the shoulder and sixth cervical (C\(_6\)) transverse process; L2, scapulohumeral joint; and L3, first rib. For TA, the needle was introduced craniocaudally through L1, medial to the limb and cranial to L3. For PA, the needle was directed perpendicular and caudal to L2, aligned with L1, until cranial to L3. For AA, the needle was directed ventrodorsally, parallel and cranial to L3 until at L1. All BP were scored for dyeing quality (0-poor to 5-excellent). The left BP was dissected for nerve origins. Kruskal-Wallis test was used to compare scores (\(p < 0.05\)).

**Results:** In all dogs, the musculocutaneous nerve originated from C\(_7\), C\(_8\); the radial nerve from C\(_8\), the first thoracic vertebra (T\(_1\)) (16/18 dogs) and C\(_7\) (2/18); and the median and ulnar nerves from C\(_8\), T\(_1\) (17/18) and C\(_7\) (1/18). Scores (median [95% confidence intervals]) were TA 2.5
(0-5); PA 4 (3-5); AA 5 (0.5-5) and not significantly different ($p = 0.54$; ranks TA 15.8; PA 19.4; AA 20.3).

**Conclusion and clinical relevance:** The musculocutaneous, median, ulnar, and radial nerves originate from C7, C8, and T1. Regardless of technique, knowledge of anatomy and precise landmarks are relevant for correct dye dispersion.

*Keywords* anatomy, brachial plexus block, dog.
2.2 INTRODUCTION

The brachial plexus is a network of nerves that provides innervation to all structures of the thoracic limb. The canine brachial plexus has been documented to receive contributions from the ventral spinal nerve rami of the fifth cervical (C₅) to the second thoracic (T₂) vertebrae (Allam et al. 1952; Mahler & Adogwa 2008; Dyce et al. 2010). However, contributions from C₅ and T₂ are usually minor, if present at all (Allam et al. 1952).

Blockade of the brachial plexus is commonly performed in canine patients undergoing surgical procedures of the thoracic limb to provide local anesthesia, analgesia and improve patient comfort. In people and dogs undergoing general inhalation anesthesia, successful block of the brachial plexus provides an inhalant sparing effect allowing a reduction in the requirements of anesthetic drugs needed to maintain a surgical plane of anesthesia (Mosing et al. 2010; Yuan et al. 2014).

The innervation to the thoracic limb can be blocked at its origin from the spinal cord (cervical paravertebral block), at the brachial plexus, or once the specific nerves travel from the plexus to the limb. The cervical paravertebral block provides regional anesthesia of the distal thoracic limb as well as the proximal humerus and shoulder (Lemke & Creighton 2008). Alternatively, individual blockade of the radial, ulnar, median and musculocutaneous nerves can be performed below their origin from the brachial plexus to provide specific local anesthesia and analgesia of the distal thoracic limb (Trumpatori et al. 2010).

A number of techniques have been described in dogs for performing the block at the level of the brachial plexus. A blind technique has been thoroughly described that uses specific anatomic landmarks (Campoy & Read 2013; Campoy et al. 2015); however, block failure is still relatively common despite following the descriptions of this block (Nutt 1962; Futema et al. 2002; Campoy et al. 2008; Ricco et al. 2013). Alternatively the location of the plexus to
perform the brachial plexus block can be more accurately determined using electrical stimulation or ultrasonography (Futema et al. 2002; Mahler & Reece 2007; Mahler & Adogwa 2008; Campoy et al. 2010; Campoy & Read 2013; Campoy et al. 2015). The use of sophisticated equipment to identify anatomical structures and precise brachial plexus location may be cost or skill limited by most veterinary practitioners and it does not substantially improve success rates (Akasaka & Shimizu 2017). Therefore, a review and better description of the current blind technique could provide more concise instructions for improved performance and success rate of the block.

This study had two objectives. The first objective was to dissect and describe the ventral spinal nerve rami that contribute to the formation of the canine brachial plexus and each of its nerves. The second objective was to compare in dogs the dye distribution and nerve staining between the currently described traditional blind technique for brachial plexus blockade and two alternative blind approaches that used different accesses to the plexus, axillary and perpendicular. It was hypothesized that the alternative approaches would provide easier approaches and better dye distribution than the traditional approach for brachial plexus blockade in dogs.
2.3 MATERIALS AND METHODS

2.3.1 Animals

A total of 18 cadavers (six mixed breed dogs, four Labrador Retrievers, two Siberian Huskies, one Bassett Hound, one Border Collie, one German Shepherd, one Golden Retriever, one Rottweiler, one Alaskan Malamute), mean ± standard deviation (SD) weight of 28.2 ± 9.7 kg (range, 14–44 kg) were obtained from a private source and used for evaluation of three techniques for blockade of the brachial plexus and dissection of the brachial plexus. A proposal was submitted to the Institutional Animal Care Committee but no approval was necessary for the use of cadavers.

2.3.2 Techniques for Blockade of the Brachial Plexus

Dogs were assigned to one of three techniques, traditional, perpendicular or axillary approach by a randomization scheme of random block sizes (http://www.randomization.com) with six dogs for each approach. Each dog was first injected in the region of the left brachial plexus with yellow tissue marking dye (0.2 mL kg⁻¹; Cancer Diagnostics Inc., NC, USA). Then the plexus was dissected to identify the location of the dye and each nerve associated with the brachial plexus, including their origin from the spinal cord. Following this phase, the dog was injected in the right brachial plexus with the same volume of dye, using one of the alternative techniques, also randomly assigned with block sizes. Consequently, each dog was treated with two of the three approaches and the study design resulted in 12 injections for each approach, six injections on the left and six on the right.
2.3.2.1 Traditional Approach (TA)

The dog was placed in lateral recumbency with the upper thoracic limb positioned in a relaxed position and extended away from the body perpendicular to the long axis of the torso (standing angle). The point of the shoulder, the transverse process of C₆, the caudal aspect of the scapulohumeral joint and the first rib were palpated. A 20 gauge, 3.5-inch (8.9-cm) spinal needle (Campoy & Read 2013) was introduced at the midpoint between the transverse process of C₆ and the point of the shoulder (landmark L1), medial to the limb and between the scapula and caudal section of the jugular groove, and advanced in a cranial to caudal direction, beyond the scapulohumeral joint (L2) to just cranial to the first rib (L3) (Figure 2.1). The entire volume of dye was injected at this location.

Figure 2.1. Landmarks for performing the traditional approach.
2.3.2.2 Perpendicular Approach (PA)

The PA was also performed with the dog in lateral recumbency with the thoracic limb position as for TA. A 20 gauge, 3.5-inch (8.9-cm) spinal needle was introduced perpendicular to the scapula, caudal to scapulohumeral joint (L2) and aimed cranial to the first rib (L3), at a level that corresponded to the midpoint between the transverse process of C₆ and the point of the shoulder (L1), (Figure 2.2). The entire volume of dye was injected at this location.

Figure 2.2. Landmarks for performing the perpendicular approach.
### 2.3.2.3 Axillary Approach (AA)

The AA approach was performed with the dog in dorsal recumbency with the limb to be blocked at a standing angle. The point of the shoulder, transverse process of C₆, caudal aspect of the scapulohumeral joint and first rib were palpated. A 20 gauge, 3.5-inch (8.9-cm) spinal needle was introduced at the level of the thoracic inlet in a ventral to dorsal direction, lateral to the manubrium and medial to the limb, parallel and cranial to the first rib (L2), and advanced to the level of the midpoint between the transverse process of C₆ and the point of the shoulder (L3) (Figure 2.3). The entire volume of dye was injected at this location.

![Figure 2.3. Landmarks for performing the axillary approach.](image)

Figure 2.3. Landmarks for performing the axillary approach.
2.3.3 Dissection of the Brachial Plexus

With the dog in right lateral recumbency, the plexus was dissected as described elsewhere, figure 2.4 (Evans & de LaHunta, 2010). Briefly, a circumferential skin incision was made 5 cm distal to the left elbow. A midventral incision was made through the skin from the cranial neck to the umbilicus. From the mid–cranial neck and umbilicus a transverse incision was extended to the dorsal midline on the left side. A transverse incision was extended from the midventral incision to the elbow on the medial aspect of the left antebrachium to join the previously made circumferential incision.

The skin and the cutaneous trunci muscle of the neck and thorax were reflected to the dorsal midline. The extrinsic muscles of the thoracic limb (brachiocephalicus, omotransversarius, trapezius, rhomboideus, serratus ventralis and pectoral muscles) were cut at their attachments to the scapula. The scapula was abducted from the thorax to expose the axillary space. The nerve rami of C₅, C₆, C₇, C₈, and T₁, and T₂ were identified and dissected from the ventral border of the scalenus muscle. All nerves connected to the brachial plexus were dissected from the overlying connective tissue and fat, and included the pectoral nerve, the suprascapular nerve, the subscapular nerve, the musculocutaneous nerve, thoracodorsal nerve, the axillary nerve, the radial nerve, the ulnar nerve and the median nerve. The individual nerve rami to the pectoral, suprascapular, subscapular, musculocutaneous, thoracodorsal, axillary, radial, ulnar, and median nerves were identified and photographed. Subsequently, the dog was placed in left lateral recumbency and the dissection repeated for dye distribution on the right brachial plexus.

Based on the contribution of each nerve ramus to the nerves in the plexus, a scoring system was developed and both brachial plexus were scored for dyeing quality using the following score system: 0 = No staining of any nerve; 1 = Staining of C₆; 2 = Staining of C₇, or C₆ and C₇; 3 = Staining of C₈ or T₁; 4 = Staining of C₇ and C₈, or C₈ and T₁, or ulnar and
median nerve, or radial nerve; and 5 = Staining of C7 and C8 and T1, or ulnar and median and radial nerves; where 0 was considered a poor block and 5 an excellent block (Table 2.1). The same investigator, unaware of the group allocation, assigned all scores at a later date using the images generated from each plexus.

2.3.4 Statistical Analysis

The sample size was calculated to detect a 2–point difference in the staining score between groups with a type 1 error of 0.05 and a power of 90%, which required 7 of each approach.

Descriptive statistics were done for weight and nerve origins (MedCalc, version 17.8, Ostend, Belgium). Scores for dye distribution were compared with the Kruskal-Wallis test (http://vassarstats.net accessed May 2016) (p < 0.05).
2.4 RESULTS

Demographic descriptions of the dogs and the brachial plexus block technique used for each plexus are provided in Table 2.2.

Dog 1: The left brachial plexus was stained using the TA and resulted in no staining of any nerves of the brachial plexus, Figure 2.5. The dye was located at the caudal aspect of the nerve rami in the ventral scalenus muscle and latissimus dorsi muscle. The right brachial plexus was stained using the PA and resulted in complete staining of the musculocutaneous, axillary, thoracodorsal, radial, ulnar, and median nerves at the level of the brachial plexus.

Dog 2: The left brachial plexus was stained using the PA and resulted in incomplete staining but included the medial aspects of the radial, ulnar, and median nerves at the level of the brachial plexus, Figure 2.6. The right brachial plexus was stained using the AA and resulted in complete staining of the ventral nerve rami of C6, C7, C8, and T1.

Dog 3: The left brachial plexus was stained using the TA and resulted in complete staining of the musculocutaneous, axillary, thoracodorsal, radial, ulnar, and median nerves at the level of the brachial plexus, Figure 2.7. The right brachial plexus was stained using the AA and resulted in complete staining of the ventral nerve rami of C7, C8, and T1.

Dog 4: The left brachial plexus was stained using the PA and resulted in complete staining of the cranial pectoral, suprascapular, subscapular, musculocutaneous, axillary, thoracodorsal, radial, ulnar, and median nerves at the level of the brachial plexus, Figure 2.8. The right brachial plexus was stained using the TA and resulted in complete staining of the ventral nerve rami of C6, C7, C8, and T1.

Dog 5: The left brachial plexus was stained using the AA and resulted in no staining of any nerves of the brachial plexus, Figure 2.9. The dye was located caudal to the plexus in the caudal pectoral muscles. The right brachial plexus was stained using the TA and demonstrated
no staining of any nerves of the brachial plexus. The dye was located medial to the brachial plexus within the pectoral muscles.

*Dog 6:* The left brachial plexus was stained using the TA and resulted in incomplete staining, but included the medial aspect of the thoracodorsal and radial nerves at the level of the brachial plexus, Figure 2.10. The right brachial plexus was stained using the AA and resulted in complete staining of the cranial pectoral, suprascapular, subscapular, musculocutaneous, axillary, thoracodorsal, radial, ulnar, and median nerves at the level of the brachial plexus as well as complete staining of the ventral nerve rami of C7, C8, and T1.

*Dog 7:* The left brachial plexus was stained using the AA and resulted in incomplete staining, but included the medial aspect of the cranial pectoral, suprascapular, subscapular, musculocutaneous, axillary, and thoracodorsal nerves at the level of the brachial plexus, Figure 2.11. The right brachial plexus was stained using the PA and resulted in incomplete staining, but included the lateral aspect of the radial, ulnar, and median nerves below the level of the brachial plexus.

*Dog 8:* The left brachial plexus was stained using the AA and resulted in complete staining of the ventral nerve rami of C7, C8, and T1, Figure 2.12. The right brachial plexus was stained using the TA and resulted in no staining of any nerves of the brachial plexus. The dye was located deep to the triceps muscle and staining the biceps brachii muscle.

*Dog 9:* The left brachial plexus was stained using the PA and resulted in no staining of any nerves of the brachial plexus, Figure 2.13. The dye was located on the lateral aspect of the scapula within the triceps muscle. The right brachial plexus was stained using the AA and resulted in complete staining of the ventral nerve rami of C6, C7, C8, and T1.

*Dog 10:* The left brachial plexus was stained using the TA and resulted in only staining of C6 and its contribution to the pectoral, suprascapular, and subscapular nerves, Figure 2.14. The dye was mostly located on the medial aspect of the scapula within the subscapularis muscle.
The right brachial plexus was stained using the PA and resulted in no staining of any nerves of the brachial plexus. The dye was located caudal to the exit point of the nerve rami within the scalenus muscle.

Dog 11: The left brachial plexus was stained using the AA and resulted in complete staining of the ventral nerve rami of C₆, C₇, C₈, and T₁, Figure 2.15. The right brachial plexus was stained using the PA and resulted in complete staining of the lateral aspect of the radial, ulnar, and median nerves at the level of the brachial plexus.

Dog 12: The left brachial plexus was stained using the PA and resulted in no staining of the nerves of the brachial plexus, Figure 2.16. The dye was located within the fascia medial to the brachial plexus. The right brachial plexus was stained using the TA and resulted in no staining of any nerves of the brachial plexus. The dye was located dorsal to the exit of the ventral nerve roots within the scalenus muscle.

Dog 13: The left brachial plexus was stained using the PA and resulted in complete staining of the cranial pectoral, suprascapular, subscapular, musculocutaneous, axillary, thoracodorsal, radial, ulnar, and median nerves at the level of the brachial plexus as well as complete staining of the ventral nerve rami of C₈ and T₁, Figure 2.17. The right brachial plexus was stained using the AA and resulted in no staining of any nerves of the brachial plexus. The dye was located above the exit point of the ventral nerve rami within the scalenus muscle.

Dog 14: The left brachial plexus was stained using the TA and resulted in incomplete staining, but included the musculocutaneous, thoracodorsal, and radial nerves at the level of the brachial plexus, Figure 2.18. The right brachial plexus was stained using the AA and resulted in incomplete staining, but included the medial aspect of the ventral nerve ramus of T₁ with the remaining dye within the fascia on the medial aspect of the nerve roots.

Dog 15: The left brachial plexus was stained using the TA and resulted in no staining of any nerves of the brachial plexus, Figure 2.19. The dye was located caudal to the brachial plexus.
within the fascia and external abdominal oblique muscle. The right brachial plexus was stained using the PA and resulted in complete staining of the musculocutaneous, thoracodorsal, radial, ulnar, and median nerves at the level of the brachial plexus.

**Dog 16:** The left brachial plexus was stained using the AA and resulted in no staining of any nerves of the brachial plexus, Figure 2.20. The dye was located medial to the ventral nerve roots surrounding the trachea. The right brachial plexus was stained using the PA and resulted in incomplete staining, but included the ventral nerve ramus of T1.

**Dog 17:** The left brachial plexus was stained using the AA and resulted in complete staining of the cranial pectoral, suprascapular, subscapular, musculocutaneous, axillary, thoracodorsal, radial, ulnar, and median nerves at the level of the brachial plexus as well as complete staining of the ventral nerve rami of C7, C8, and T1, Figure 2.21. The right brachial plexus was stained using the TA and resulted in complete staining of the musculocutaneous, thoracodorsal, radial, ulnar, and median nerves at the level of the brachial plexus.

**Dog 18:** The left brachial plexus was stained using the PA and resulted in complete staining of the musculocutaneous, axillary, thoracodorsal, radial, ulnar, and median nerves at the level of the brachial plexus, Figure 2.22. The right brachial plexus was stained using the TA and resulted in complete staining of the radial, ulnar, and median nerves distal to the brachial plexus.

**2.4.1 Brachial Plexus and Nerve Anatomy:**

All brachial plexuses were formed from the cervical ventral nerve rami C6, C7, C8, and thoracic ventral nerve ramus T1 (Tables 2.3 and 2.4). No contribution to the brachial plexus from C5 or T2 was identified in any dog.

In 100% of the dogs, the pectoral nerve was formed from a branch of C6 (Table 2.2). In 94% of the dogs, the suprascapular nerve was formed from C6 and C7. In dog 10 the
suprascapular nerve was formed from a single branch of C6. In 94% of dogs, the subscapular nerve was from C6 and C7. In dog 2 the subscapular nerve was formed from a single branch of C7. In 100% of dogs, the musculocutaneous nerve was formed from C7 and C8. In 72% of dogs, the axillary nerve was formed from C7 and C8. In dog 7, 9, 10, 12, and 13 the axillary nerve was formed from a single branch of C7. In 83% of dogs, the thoracodorsal nerve was formed from a single branch of C8. In dog 9, 13, and 17 the thoracodorsal nerve was formed from C8 and had contribution from T1. In 89% of dogs, the radial nerve was formed from C8 and T1. In dog 4 and 11 the radial nerve was formed from C7, C8, and T1. In 94% of dogs the trunk of the median and ulnar nerve was formed from C8 and T1. In dog 2 the trunk of the median and ulnar nerve also received contribution from C7.

2.4.2 Brachial Plexus Nerve Staining:

Scores for dyeing quality of each brachial plexus are presented in Table 2.2. The median (95% confidence interval) score was 2.5 (0.5-5) for TA, 4.0 (3-5) for PA, and 5.0 (0.5-5) for AA. There was no difference between scores based on ranks from the Kruskal-Wallis test ($p = 0.54$; ranks TA 15.8, PA 19.4, AA 20.3).

Incomplete staining (scores of 0, 1, and 2) (Figure 2.9 and 2.14 A) with no contribution to the main nerves or their ventral rami, to the distal limb were present in 6/12 plexus (50%) in TA, 2/12 plexus (17%) in PA, and in 3/12 (25%) in AA. In these scores, the dye stained C6 and/or C7, or no nerves or ventral rami. No score of 2 was recorded. For scores of 0, the dye was not in contact with any nerve or rami, and instead was located in the surrounding area of the plexus. These, included locations such as the ventral scalenus muscle and latissimus dorsi muscle, or in the area of the pectoral muscles, or in the area of the triceps and biceps brachii muscles, or in the area of the subscapularis muscle, or in the fascia of the external abdominal oblique muscle, or ventral to the trachea at the thoracic inlet.
In score 3, staining was incomplete and included staining of at least one of the ventral nerve rami (C₈ or T₁) to the main nerves to the distal limb, including the musculocutaneous, ulnar and median or radial nerve (Figure 2.6 A). Score of 4 was also incomplete staining because the dye stained at least one of those main nerves (Figure 2.10 A). Partial staining was achieved in 2/12 plexus (17%) in TA, in 4/12 plexus (33%) in PA, and in 2/12 plexus (17%) in AA.

Complete staining of the main nerves, musculocutaneous, ulnar, median and radial, or of the ventral nerve rami that these nerves originate from, namely C₇, C₈ and T₁, was assigned a score of 5 (Figure 2.7). This score was achieved in 4/12 plexus (33%) in TA, in 6/12 plexus (50%) in PA, and in 7/12 plexus (58%) in AA.
2.5 DISCUSSION

The contributions of spinal nerves to the brachial plexus of dogs have been documented in two previous anatomical studies. The first study documented minor contribution to the canine brachial plexus from the ventral nerve rami of spinal nerves C5 and T2 (Miller, 1934). A second study identified that in 58 brachial plexus, 58.6% of dogs had contributions from the ventral rami of spinal nerves C6 through T1; 20.7% had contributions from C5 through T1; 17.2% had contributions from C6 through T2; and only 3.4% had contribution from C5 through T2 (Allam et al. 1952). The current study describes that the nerve contribution to 18 canine brachial plexus does not include the ventral rami of spinal nerves C5 or T2. Recent reviews on local regional anesthesia also mention that the spinal nerve ramus contribution to the brachial plexus originates from C6, C7, C8, and T1 (Lemke & Dawson 2000; Campoy & Read 2013), although specific dissection of the spinal nerve rami was not performed to document these statements.

The most important rami to be included in the block of the brachial plexus to desensitize the distal thoracic limb are the caudal nerves (C8 and T1), since they are the major contributors to the musculocutaneous, radial, ulnar and median nerves. The musculocutaneous nerve originates from C7 (Allam et al. 1952) and innervates the biceps brachii and brachialis muscles to cause flexion of the elbow and provide sensory innervation to the medial aspect of the antebrachium (Allam et al. 1952; Mahler & Adogwa 2008; Dyce et al. 2010). In the current study 100% of the dogs had contribution from the ventral rami of spinal nerves C7 and C8 to the musculocutaneous nerve. The radial nerve innervates the extensor muscles of the thoracic limb and provides sensory innervation to the forearm (Mahler & Adogwa 2008; Dyce et al. 2010). It is consistently documented that the ventral ramus of spinal nerve C8 is a major contributor to the radial nerve (Miller, 1934; Allam et al. 1952). In the current study 100% of
the dogs had contribution from spinal nerve C_8 and T_1 to the radial nerve. The ulnar and median nerves are the two other nerves that provide major sensory innervation to the thoracic limb in addition to innervating the flexor muscles (Dyce et al. 2010). Consistent with what has been previously documented (Miller, 1934; Allam et al. 1952; Dyce et al. 2010), all dogs in the current study received major contribution from the ventral rami of spinal nerves C_8 and T_1 to the formation of the ulnar and median nerves.

The scoring system used in this study differs from other scores previously used in similar studies. The landmarks described in the traditional approach (Campoy & Read 2013; Campoy et al. 2015), and those of the alternate approaches presented in this study, resulted in deposition of the dye in different areas of the plexus and surrounding tissue. Scoring systems used in other studies have counted the sensory distal nerves (musculocutaneous, ulnar, median, and radial) stained after injection of the plexus (Campoy et al. 2008; Ricco et al. 2013) or the ventral rami stained at the plexus itself (Campoy et al. 2010), but not both. We considered that a scoring system should include both ventral rami at the plexus and/or the distal nerves formed from the plexus, due to their contribution to an adequate brachial plexus block. Therefore, in the current scoring system, the best scores were those that include all distal thoracic nerves or the rami that contribute to those nerves (C_7, C_8, and T_1).

Regional techniques for local blockade rely on accurate identification of anatomical landmarks for appropriate deposition of the local anesthetic drug in the proximity of the nerves to facilitate its anesthetic actions, as well as to decrease the likelihood of complications from damaging surrounding structures, such as blood vessels. Additional modalities, including electrolocation and ultrasonography have been utilized to describe more precise injection of local anesthetics to several specific nerves, including provision of brachial plexus blockade in dogs (Mahler & Reece 2007; Mahler & Adogwa 2008; Campoy et al. 2010; Ricco et al. 2013; Campoy et al. 2015), with the goal of improving success rate of the block and avoiding
surrounding structures. However, these techniques have not necessarily improved the success rate, require costly equipment, and a level of expertise to perform. In a study comparing blind cervical paravertebral block with electrolocation or ultrasound guidance, in dogs before humane euthanasia, there was no improvement in the success rate of blockade of the ventral nerve rami of C₆ to T₁ (Rioja et al. 2012). Better success rates (91.6-100%) have been described for electrolocation and ultrasound guided blockade of the brachial plexus when using the traditional approach (Futema et al. 2002; Campoy et al. 2008; Ricco et al. 2013), although C₆ was not successfully stained in any dog, despite ultrasonography in one study (Campoy et al. 2010). This is in contrast to a clinical study where a tendency for a more rapid onset and longer duration of action of block was observed for nerves in the cranial aspect of the brachial plexus, compared to nerves in the caudal aspect, as a result of channelization of local anesthetic agent toward the cranial aspect of the shoulder (Moens & Caulkett 2000).

In this study, two alternative approaches (PA, AA) using anatomical landmarks for performing the canine brachial plexus block were described and compared with the TA that is currently described in the literature. Partial or complete failure of local blockade is most often due to inadequate or incomplete contact of the local anesthetic with all or any of the nerves in the brachial plexus as a result of inadequate needle location (Campoy et al. 2008; Ricco et al. 2013). Good to excellent results were reported in 86% of dogs that received a brachial plexus block using the TA (Nutt, 1962). In our study, a score of 5 was considered a complete block, based on complete staining of all relevant ventral rami or distal nerves to the limb, and was achieved in 33% of cadavers using the TA, in 50% of cadavers with the PA, and in 58% of cadavers using the AA. Including partial blocks (scores of 3 and 4) with the complete block, the scores improve to 50% for the TA, 88% for the PA, and 75% for the AA.

Evaluation of the dye scores in the current study demonstrated that scores of incomplete staining (0-2) did occur throughout the study period regardless of repetition of the different
approaches. Blocks were always performed by the same person, following the described landmarks set for each technique. The description of the TA for blockade of the canine brachial plexus uses the scapulohumeral joint as the primary anatomic landmark to guide needle placement for deposition of local anesthetic drugs (Campoy & Read 2013). Our hypothesis was that the novel approaches (PA, AA) would be better alternatives than the TA for brachial plexus blockade in dogs; however, in retrospect, regardless of the technique, three key anatomical landmarks were identified after dissection of the plexus and assessment of the dyeing scores, based on the description of each of the three techniques used in the methods. These landmarks were the transverse process of C₆, the point of the shoulder, and the first rib; it was appreciated that if the needle point was positioned at the level midway between the transverse process of C₆ and the point of the shoulder and advanced to the cranial aspect of the first rib, the success of dye deposition within the brachial plexus was increased, regardless of the technique used. The authors have realized that with any of the three techniques, keeping a finger on the first rib during needle advancement would help ensure appropriate needle placement prior to dye injection, by making contact with the rib and directing the tip of the needle cranial to it, where the brachial plexus is located.

The volume of dye used to evaluate techniques for performing the brachial plexus block varies among studies. A volume of 0.3 mL kg⁻¹ of a lidocaine/methylene blue dye combination injected into the brachial plexus using electrolocation was successful in staining all nerves of the brachial plexus in the majority of dogs, whereas volumes of 0.075 and 0.15 mL kg⁻¹ were unsuccessful in the majority of dogs (Campoy et al. 2008). However, in a study using ultrasound-guided brachial plexus block in dogs, 0.15 mL kg⁻¹ of a local anesthetic/methylene blue dye combination was able to achieve good staining of three of the four ventral nerve rami of the brachial plexus (Campoy et al. 2010). Proper identification of anatomic landmarks and needle position with respect to the plexus are important for successful blockade of the desired
nerves and has allowed for reduced volumes (0.15-0.4 mL kg\textsuperscript{-1}) in several studies using electrolocation, ultrasound guidance, or blind techniques (Wenger 2004; Wenger et al. 2005; Mahler & Reece 2007; Campoy et al. 2008, Campoy et al. 2010, Ricco et al. 2013). In the current study, 0.2 mL kg\textsuperscript{-1} of dye were used for injection of each brachial plexus. When the dye was injected directly on the plexus, this volume of dye resulted in adequate staining of all nerves of the brachial plexus; however, clinical studies would be required to determine if this volume results in successful local anesthesia.

This study had some limitations. The utilization of cadaver dogs may have affected dye dispersion differently than what would occur in a live animal. Additionally, because cadaver dogs were used, any increased risk of vascular complications such as hemorrhage or hematoma formation with a particular technique could not be evaluated. Finally, as only the left brachial plexus nerve formation was dissected in its entirety any anatomical differences to nerve formation of the right brachial plexus were not identified.

In clinical practice, the utilization of anatomical landmarks for provision of brachial plexus blockade may be more cost effective than techniques that require special equipment, and more straightforward to perform, because they require less time (Rioja et al. 2012). In conclusion, all of the three techniques described here are feasible methods to perform a brachial plexus block in canine patients. Proper identification of the anatomical landmarks described in this study are key to provision of a successful block. Further work is needed to determine if any clinical differences exist between these techniques.
2.6 REFERENCES


Miller RA (1934) Comparative studies upon the morphology and distribution of the brachial plexus. Am J Anat 54, 143-175.


**Table 2.1.** Score system for dye distribution on rami or nerves associated with the brachial plexus after administration of 0.2 mL kg\(^{-1}\) of dye in cadaver dogs.

<table>
<thead>
<tr>
<th>Score</th>
<th>Ventral ramus(i) and/or peripheral nerve(s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>No staining of any nerve</td>
</tr>
<tr>
<td>1</td>
<td>Staining of C(_6)</td>
</tr>
<tr>
<td>2</td>
<td>Staining of C(_6), or</td>
</tr>
<tr>
<td></td>
<td>Staining of C(_6) + C(_7)</td>
</tr>
<tr>
<td>3</td>
<td>Staining of C(_8), or</td>
</tr>
<tr>
<td></td>
<td>Staining of T(_1)</td>
</tr>
<tr>
<td></td>
<td>Staining of C(_7) + C(_8), or</td>
</tr>
<tr>
<td></td>
<td>Staining of C(_8) + T(_1), or</td>
</tr>
<tr>
<td>4</td>
<td>Staining of the musculocutaneous nerve, or</td>
</tr>
<tr>
<td></td>
<td>Staining of the ulnar + median nerves, or</td>
</tr>
<tr>
<td></td>
<td>Staining of the radial nerve</td>
</tr>
<tr>
<td>5</td>
<td>Staining of C(_7) + C(_8) + T(_1), or</td>
</tr>
<tr>
<td></td>
<td>Staining of the musculocutaneous + ulnar + median + radial nerves</td>
</tr>
</tbody>
</table>
Table 2.2. Description of dog cadavers assigned to injection of yellow dye simulating brachial plexus block by two of three techniques and overall nerve staining score (score 0 no staining to score 5 all nerves stained) determined after dissections.

<table>
<thead>
<tr>
<th>Dog number</th>
<th>Breed</th>
<th>Weight (kg)</th>
<th>Dye volume (mL)</th>
<th>Approach technique</th>
<th>Left side</th>
<th>Score</th>
<th>Right side</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Basset Hound</td>
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<td>4.6</td>
<td>Traditional</td>
<td>0</td>
<td></td>
<td>Perpendicular</td>
<td>5</td>
</tr>
<tr>
<td>2</td>
<td>Mixed breed</td>
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<td>3.2</td>
<td>Perpendicular</td>
<td>3</td>
<td></td>
<td>Axillary</td>
<td>5</td>
</tr>
<tr>
<td>3</td>
<td>Mixed breed</td>
<td>32</td>
<td>6.4</td>
<td>Traditional</td>
<td>5</td>
<td></td>
<td>Axillary</td>
<td>5</td>
</tr>
<tr>
<td>4</td>
<td>Mixed breed</td>
<td>16</td>
<td>3.2</td>
<td>Perpendicular</td>
<td>5</td>
<td></td>
<td>Traditional</td>
<td>5</td>
</tr>
<tr>
<td>5</td>
<td>Labrador Retriever</td>
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<td>8.8</td>
<td>Axillary</td>
<td>0</td>
<td></td>
<td>Traditional</td>
<td>0</td>
</tr>
<tr>
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<td>Mixed breed</td>
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<td>7</td>
<td>Traditional</td>
<td>4</td>
<td></td>
<td>Axillary</td>
<td>5</td>
</tr>
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<td>Border Collie</td>
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<td>7</td>
<td>Axillary</td>
<td>3</td>
<td></td>
<td>Perpendicular</td>
<td>4</td>
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<td>Siberian Husky</td>
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<td>7</td>
<td>Axillary</td>
<td>5</td>
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<td>Traditional</td>
<td>0</td>
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<td>German Shepherd</td>
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<td>6.4</td>
<td>Perpendicular</td>
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<td>Axillary</td>
<td>5</td>
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<td>Golden Retriever</td>
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<td>6.8</td>
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<td></td>
<td>Perpendicular</td>
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</tr>
<tr>
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<td>Labrador Retriever</td>
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<td>7.6</td>
<td>Axillary</td>
<td>5</td>
<td></td>
<td>Perpendicular</td>
<td>5</td>
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<tr>
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<td>Alaskan Malamute</td>
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<td>8</td>
<td>Perpendicular</td>
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<td></td>
<td>Traditional</td>
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<tr>
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<td>Rottweiler</td>
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<td>Perpendicular</td>
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<td>Axillary</td>
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<td>Traditional</td>
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<td>3</td>
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<tr>
<td>15</td>
<td>Labrador Retriever</td>
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<td>16</td>
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<td>17</td>
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<td>Axillary</td>
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<td>Traditional</td>
<td>5</td>
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<tr>
<td>18</td>
<td>Mixed breed</td>
<td>14</td>
<td>2.8</td>
<td>Perpendicular</td>
<td>5</td>
<td></td>
<td>Traditional</td>
<td>5</td>
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</table>
Table 2.3. Number (percentage) of dogs in which each nerve ramus contributed to the nerves of the left brachial plexus ($n = 18$). C, cervical; T, thoracic.

<table>
<thead>
<tr>
<th>Nerve</th>
<th>Nerve ramus</th>
<th>C6</th>
<th>C7</th>
<th>C8</th>
<th>T1</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pectoral</td>
<td></td>
<td>18 (100)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Suprascapular</td>
<td></td>
<td>18 (100)</td>
<td>17 (94)</td>
<td></td>
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<tr>
<td>Subscapular</td>
<td></td>
<td>17 (94)</td>
<td>18 (100)</td>
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</tr>
<tr>
<td>Musculocutaneous</td>
<td></td>
<td>18 (100)</td>
<td>18 (100)</td>
<td></td>
<td></td>
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<tr>
<td>Thoracodorsal</td>
<td></td>
<td></td>
<td></td>
<td>18 (100)</td>
<td>3 (17)</td>
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<tr>
<td>Axillary</td>
<td></td>
<td>18 (100)</td>
<td>13 (72)</td>
<td></td>
<td></td>
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<tr>
<td>Radial</td>
<td></td>
<td>2 (11)</td>
<td>18 (100)</td>
<td>18 (100)</td>
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<tr>
<td>Ulnar</td>
<td></td>
<td>1 (6)</td>
<td>18 (100)</td>
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<tr>
<td>Median</td>
<td></td>
<td>1 (6)</td>
<td>18 (100)</td>
<td>18 (100)</td>
<td></td>
</tr>
</tbody>
</table>
Table 2.4. Number of dogs in which the nerve ramus/rami contributed to the nerves of the left brachial plexus (n = 18). C: cervical; T: thoracic.

<table>
<thead>
<tr>
<th>Nerve</th>
<th>C_6</th>
<th>C_6-C_7</th>
<th>C_7</th>
<th>C_7-C_8</th>
<th>C_8</th>
<th>C_7-C_8-T_1</th>
<th>C_8-T_1</th>
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<td>Pectoral</td>
<td>18</td>
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Figure 2.4. Dissection position and first skin incisions (Evans & de LaHunta 2010).
Figure 2.5. Brachial plexus block of dog #1, showing dye dispersion for both blocks and nerve origin for the left plexus. A- Traditional approach on left limb with score of 0; B- Perpendicular approach on right limb with score of 5.

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Figure 2.6. Brachial plexus block of dog #2, showing dye dispersion for both blocks and nerve origin for the left plexus.  A- Perpendicular approach on left limb with score of 3; B- Axillary approach on right limb with score of 5.

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Figure 2.7. Brachial plexus block of dog #3, showing dye dispersion for both blocks and nerve origin for the left plexus. A- Traditional approach on left limb with score of 5; B- Axillary approach on right limb with score of 5.

A.

B.
Figure 2.8. Brachial plexus block of dog #4, showing dye dispersion for both blocks and nerve origin for the left plexus.  A- Perpendicular approach on left limb with score of 5; B- Traditional approach on right limb with score of 5.

A.

B.
Figure 2.9. Brachial plexus block of dog #5, showing dye dispersion for both blocks and nerve origin for the left plexus. A- Axillary approach on left limb with score of 0; B- Traditional approach on right limb with score of 0.

A.

B.
**Figure 2.10.** Brachial plexus block of dog #6, showing dye dispersion for both blocks and nerve origin for the left plexus. A- Traditional approach on left limb with score of 4; B- Axillary approach on right limb with score of 5.

A.

B.
Figure 2.11. Brachial plexus block of dog #7, showing dye dispersion for both blocks and nerve origin for the left plexus. A- Axillary approach on left limb with score of 3; B- Perpendicular approach on right limb with score of 4.

A.

B.
**Figure 2.12.** Brachial plexus block of dog #8, showing dye dispersion for both blocks and nerve origin for the left plexus. A- Axillary approach on left limb with score of 5; B- Traditional approach on right limb with score of 0.

A.

B.
Figure 2.13. Brachial plexus block of dog #9, showing dye dispersion for both blocks and nerve origin for the left plexus.  A- Perpendicular approach on left limb with score of 0; B- Axillary approach on right limb with score of 5.

A.

B.
Figure 2.14. Brachial plexus block of dog #10, showing dye dispersion for both blocks and nerve origin for the left plexus. A- Traditional approach on left limb with score of 1; B- Perpendicular approach on right limb with score of 0.

A.

B.
Figure 2.15. Brachial plexus block of dog #11, showing dye dispersion for both blocks and nerve origin for the left plexus. A- Axillary approach on left limb with score of 5; B- Perpendicular approach on right limb with score of 4.

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Figure 2.16. Brachial plexus block of dog #12, showing dye dispersion for both blocks and nerve origin for the left plexus. A- Perpendicular approach on left limb with score of 0; B- Traditional approach on right limb with score of 0.

A.

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Figure 2.17. Brachial plexus block of dog #13, showing dye dispersion for both blocks and nerve origin for the left plexus. A- Perpendicular approach on left limb with score of 5; B- Axillary approach on right limb with score of 0.

A.

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Figure 2.18. Brachial plexus block of dog #14, showing dye dispersion for both blocks and nerve origin for the left plexus. A- Traditional approach on left limb with score of 5; B-Axillary approach on right limb with score of 3.

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**Figure 2.19.** Brachial plexus block of dog #15, showing dye dispersion for both blocks and nerve origin for the left plexus. A- Traditional approach on left limb with score of 0; B-Perpendicular approach on right limb with score of 5.

A.

B.
Figure 2.20. Brachial plexus block of dog #16, showing dye dispersion for both blocks and nerve origin for the left plexus. A- Axillary approach on left limb with score of 0; B- Perpendicular approach on right limb with score of 3.

A.

B.
**Figure 2.21.** Brachial plexus block of dog #17, showing dye dispersion for both blocks and nerve origin for the left plexus. A- Axillary approach on left limb with score of 5; B- Traditional approach on right limb with score of 5.

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**Figure 2.22.** Brachial plexus block of dog #18, showing dye dispersion for both blocks and nerve origin for the left plexus. A- Perpendicular approach on left limb with score of 5; B- Traditional approach on right limb with score of 4.

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B.
CHAPTER 3

ASSESSMENT OF THE DEGREE OF ANALGESIA OF THREE BLIND BRACHIAL PLEXUS BLOCK TECHNIQUES IN DOGS UNDERGOING SURGICAL PROCEDURES OF THE THORACIC LIMB
3.1 SUMMARY

Objective: To evaluate the degree of anesthesia and postoperative analgesia resulting from three different brachial plexus block (BPB) local anesthetic techniques in canine patients undergoing surgery of the thoracic limb.

Study design: Prospective, randomized, blinded, clinical trial.

Animals: Twenty-four client-owned dogs 3.5 (95% CI, 0.94 to 6.88) years of age, weighing 26.8 (95% CI, 18.0 to 31.1) kg.

Methods: Dogs premedicated with IM hydromorphone (0.05 mg kg\(^{-1}\)) and acepromazine (0.02-0.05 mg kg\(^{-1}\)), induced with IV propofol to effect and maintained with isoflurane (1.5% end-tidal) using mechanical ventilation. Dogs were randomized in a blinded study and received one of 3 BPB techniques: TA- traditional, PA- perpendicular, or AA- axillary, with 0.2 mL kg\(^{-1}\) of bupivacaine, after induction. Heart rate, systolic arterial pressure, end-tidal isoflurane and CO\(_2\), temperature, and anesthetic maintenance score (0–best to 4–responsive) were recorded throughout anesthesia. Dogs were scored for anesthetic recovery quality (0–not responsive to 3–responsive) and up to 24-hours postoperatively for pain (Glasgow pain scale). A Kruskal-Wallis test was used to compare groups and a Wilcoxon rank sum test to compare dogs undergoing amputation versus no amputation with a Monte Carlo estimate ($p < 0.05$).

Results: No differences in the measured variables or scores were noted between groups. Nine of 24 dogs had thoracic limb amputations (4 in TA, 3 in PA, 2 in AA). Dogs undergoing
amputations vs. no amputations had higher pain scores (2.4 [2.4-3.0, interquartiles] vs. 1.6 [1.4-2.2]) in the first 3–hours post–extubation, recovery scores (1 [0-2] vs. 0 [0-0]), anesthesia score (0 [0-1] vs. 0 [0-0]), anesthesia time (203 [186-235] min vs. 160 [140-230]), surgery time (115 [100-138] min vs. 50 [41-90]), age (8 [6.5-10] years vs. 1 [0.5-3]), and body weight (30.4 [27.6-36.2] kg vs. 20.2 [5.2-28.3]). All dogs recovered uneventfully from anesthesia without BPB complications.

**Conclusions and clinical relevance:** The three anatomical approaches to block the brachial plexus provided a similar degree of comfort. Dogs undergoing thoracic limb amputation exhibited less comfort than dogs not undergoing thoracic limb amputations after BPB.
3.2 INTRODUCTION

Locoregional anesthesia is commonly performed in veterinary medicine as an adjunct to general anesthesia to decrease the requirement of anesthetic drugs and improve patient comfort postoperatively. In canine patients, surgical procedures of the thoracic limb are commonplace and can include, but are not limited to: fracture repair, arthroscopy and limb amputation. Major procedures such as these require general anesthesia to be performed, however general anesthesia poses an inherent risk and as such, balanced anesthetic techniques that include local anesthetic blocks are becoming increasingly popular across species in veterinary medicine.

The innervation to the thoracic limb is provided entirely by the brachial plexus, which is a network of nerves that is formed from the ventral spinal nerve rami of C6 through T2 (Allam et al. 1952; Dyce et al. 2010). Blockade of the brachial plexus using local anesthetic is commonly performed in canine patients undergoing surgical procedures of the thoracic limb, and when performed correctly, administration of 0.5% bupivacaine can result in anesthesia and analgesia of the thoracic limb for more than six hours (Sakonju et al. 2009).

The most commonly used technique for performing a brachial plexus block (BPB) in canine patients is a blind approach to the plexus that employs specific anatomical landmarks for needle positioning (Campoy & Read 2013; Grimm et al. 2015). Although techniques for performing the BPB in dogs using ultrasonography (Campoy et al. 2010) or electrolocation (Mahler & Adogwa 2008) have been described, the blind approach to the brachial plexus is still commonly performed because it does not require additional equipment or an advanced skill level to be performed well. What can be difficult to ascertain from the description of the current blind technique is the final location of the needle tip that will result in an adequate
contact of local anesthetic with the nerves of the brachial plexus and subsequently and appropriate degree of anesthesia and analgesia.

In Chapter II, three blind techniques for BPB in dogs were compared to evaluate ease of performance and dye distribution in a population of cadaver dogs. Although no differences in nerve staining between these three techniques were identified, three key anatomical landmarks were used to improve the distribution of the dye on the brachial plexus. These landmarks were the transverse process of C6, the point of the shoulder, and the first rib; it was appreciated that if the needle point was positioned at the level midway between the transverse process of C6 and the point of the shoulder and advanced to the cranial aspect of the first rib the success of dye deposition within the brachial plexus was increased, regardless of the technique used. As discussed in Chapter II, the investigators realized that keeping an index finger on the first rib during needle advancement would help ensure appropriate needle placement prior to dye injection.

Blind techniques for BPB have not been compared in dogs for degree of analgesia in clinical cases undergoing surgical procedures of the thoracic limb. In cats undergoing distal thoracic limb surgery, brachial plexus block has been demonstrated to have an inhalant–sparing effect and improve post-operative comfort (Mosing et al. 2010). The objective of the current study was to evaluate the degree of anesthesia and post-operative analgesia resulting from three different blind BPB techniques in canine patients undergoing surgical procedures of the thoracic limb. It was hypothesized that there would be no difference between the three techniques in the degree of post-operative analgesia.
3.3 MATERIALS AND METHODS

3.3.1 Animals

Twenty-four client-owned dogs 3.5 (0.94 to 6.88) [median and 95% CI] years of age, weighing 26.8 (18.0 to 31.1) kg of various breeds presented to the Ontario Veterinary College Health Sciences Centre for surgical procedures of the thoracic limb, and for whom owner consent was obtained, were used for evaluation of three blind techniques for blockade of the brachial plexus. Surgical procedures of the thoracic limb included: amputation (n=9) and soft-tissue or orthopedic repair (n=15). Dogs were classified as an American Society of Anesthesiologists (ASA) status I or II.

The study was carried out in accordance with the guidelines of the Canadian Council on Animal Care and was approved by the Institutional Animal Care Committee at the University of Guelph.

3.3.2 Anesthesia

All anesthetic procedures were carried out by one of two anesthesia residents, except for the brachial plexus block. Dogs were premedicated with intramuscular (IM) hydromorphone (0.05 mg kg\(^{-1}\)) (Hydromorphone HCl, Sandoz Canada, Boucherville, Quebec, CA) and acepromazine (0.02-0.05 mg kg\(^{-1}\)) (Atravet, Boehringer Ingelheim, Burlington, Ontario, CA). In one case, an intravenous (IV) catheter was present and the preanesthetic medication was delivered IV. A peripheral catheter (BD Insyte-W, BD Infusion Therapy Systems Inc. Sandy, Utah, USA) was placed in the right or left saphenous vein depending on the surgical procedure. Twenty to twenty five minutes following premedication, the dogs were scored for degree of sedation at the time of catheterization using a scale from 0 to 3, with 0– indicating no obvious sedation and 3– indicating profound sedation (Appendix 1). Anesthesia
was induced with propofol (2-3 mg kg\(^{-1}\), IV) (Propofol 1%, Pharmascience Inc. Montreal, Quebec, CA) titrated to effect to allow orotracheal intubation and maintained with isoflurane (Aerrane, Baxter Corporation, Mississauga, Ontario, CA) vaporized in 100% oxygen. Dogs were immediately ventilated using a volume–cycled ventilator (EMC 2002IE; Hallowell, Pittsfield, Massachusetts, USA) to maintain an end–tidal CO\(_2\) (ETCO\(_2\)) of 35-45 mmHg and an end–tidal isoflurane (ETIso) of 1.5%. All dogs were administered isotonic fluids (Plasma–Lyte A, Baxter Corporation, Mississauga, Ontario, CA) IV at a rate of 5-10 mL kg\(^{-1}\) h\(^{-1}\). Instrumentation for monitoring was completed within 5 minutes following induction with a Doppler (Benson Medical Industries Inc. Markham, Ontario, CA) for indirect systolic blood pressure and with a multiparameter monitor (Cardiocap 5; Datex–Ohmeda, Madison, Wisconsin, USA) for heart rate and rhythm, respiratory rate, Sp\(_O_2\), ETT\(_O_2\), ETCO\(_2\), and esophageal temperature. Systolic arterial pressure was maintained above 90 mmHg by adjusting the intravenous fluid rate and by administering dopamine infusion (3-7 \(\mu\)g kg\(^{-1}\) min\(^{-1}\)) (Dopamine HCl, Baxter Corporation, Mississauga, Ontario, CA) as required. Adjustments in anesthetic depth were made in accordance with the following protocol: following initiation of surgery if the patient demonstrated no response to surgical stimulation the vaporizer setting was adjusted such that the ETT\(_O_2\) was decreased to 1.3%. Dogs were administered hydromorphone (0.03 mg kg\(^{-1}\), IV) every 2 hours during surgery, and one dose of meloxicam (0.1 mg kg\(^{-1}\), IV) (Metacam, Boehringer Ingelheim, Burlington, Ontario, CA) was administered just prior to extubation. The quality of anesthetic maintenance was scored at the end of the procedure using a scale from 0–4, with 0– indicating the best plane of anesthesia and 4– indicating the less desirable plane of anesthesia (Appendix 2). If dogs demonstrated response to surgical stimulation (anesthesia scores of 1 or higher), the vaporizer setting was adjusted to deliver an ETT\(_O_2\) of 1.5% and not readjusted thereafter; in addition, administration of IV lidocaine (Lidocaine HCl, Alveda Pharmaceuticals, Toronto, Ontario, CA) (2 mg kg\(^{-1}\)) was
included for anesthesia scores of 2 or higher, and IV hydromorphone (0.03 mg kg\(^{-1}\)) for anesthesia scores of 3 or higher. Body temperature was supported using a circulating warm water blanket and forced warm air. At the completion of surgery, dogs were transferred to recovery for extubation and pain assessments. Dogs were extubated when they were spontaneously ventilating, developed a brisk palpebral reflex and the eye demonstrated return of consciousness. Immediately following extubation the dogs were scored for quality of recovery using a scale from 0 to 3, with 0– indicating profound sedation and 3– indicating no sedation to an excitable recovery (Appendix 3). Pain assessments were completed at 0, 15, 30 minutes, 1, 2, 3, 6, 9, 12 and 24 hours following extubation using the shortened Glasgow Composite Pain Scale (CMPS-SF) (Appendix 4). Postoperative analgesia with hydromorphone (0.03-0.05 mg kg\(^{-1}\), IV or IM) was scheduled routinely at 3-6 hours post–extubation and repeated Q 6-8 h, as deemed appropriate. If the dog received a score of 5/20 or 6/24, earlier than the post-operative scheduled analgesia, rescue analgesia with hydromorphone was administered. For dogs that underwent thoracic limb amputation and had pain diffusion catheters placed during surgery, bupivacaine (Marcaine 0.5%, Hospira Healthcare, Montreal, Quebec, CA) infusion was also provided at 4 hours following completion of surgery or as part of the rescue analgesia if required by the dog.

3.3.3 Brachial Plexus Block

Dogs were assigned to one of three techniques equally by a randomization scheme (http://www.randomization.com): traditional, perpendicular, or axillary approach. Following instrumentation, the brachial plexus of the affected limb was blocked by a single board certified anesthetist (AV) and the anesthetist (AS, RA) carrying on the anesthesia of the dog remained unaware of the type of block performed.
For the traditional approach, the dog was placed in lateral recumbency with the limb to be blocked up and at a standing angle. A 20 gauge, 2.5- to 3.5-inch spinal needle (BD Medical, Franklin Lakes, New Jersey, USA) was introduced medial to the limb, between the scapula and caudal section of the jugular groove, in a craniocaudal direction, from the midpoint between the transverse process of C₆ and the point of the shoulder, and advanced beyond the scapulohumeral joint and just cranial to the first rib. For the axillary approach, the dog was placed in dorsal recumbency with the limb to be blocked at a standing angle; the spinal needle was introduced at the level of the thoracic inlet in a ventral to dorsal direction, lateral to the manubrium and medial to the limb, parallel and cranial to the first rib, and advanced to the level of the midpoint between the transverse process of C₆ and the point of the shoulder. For the perpendicular approach, the dog was placed in lateral recumbency with the limb to be blocked up and at a standing angle; the spinal needle was introduced perpendicular to the scapula, caudal to scapulohumeral joint and cranial to the first rib, at a level that corresponded to the midpoint between the transverse process of C₆ and the point of the shoulder, until it reached a depth that exceeded only slightly the width of the lateromedial aspect of the limb at the scapulohumeral area. Each dog was administered 0.2 mL kg⁻¹ of bupivacaine 0.5% (Marcaine 0.5%, Hospira Healthcare, Montreal, Quebec, CA) for the block.

3.3.4 Statistical Analysis

Comparisons between the three groups were completed using a Kruskal–Wallis test. For comparison of dogs undergoing amputation versus no amputation, that included dogs from the three groups, a Wilcoxon rank sum test was used. A Monte Carlo estimate was used for significance \((p < 0.05)\). Statistical analysis was performed using the SAS program (SAS Institute, version 9.4, Cary, North Carolina, USA).

3.4 RESULTS
Results are presented as median [interquartile range]. Dogs underwent a variety of surgical procedures of the thoracic limb, which included: fracture repair (10), amputation (9), arthroscopy (3), mass removal (1), and digit amputation (1).

There were no differences in acepromazine dose used for preanesthetic sedation, propofol dose required for orotracheal intubation, or total anesthesia time between groups (Table 3.1). There were no differences in sedation, anesthetic or recovery scores or cardiorespiratory variables and body temperature between groups (Table 3.2). Five dogs in the traditional approach group, three in the perpendicular approach group, and four in the axillary approach group required dopamine support for hypotension. Six of these dogs were treated with glycopyrrolate for initial bradycardia and hypotension (4 in traditional approach group, 1 in the perpendicular approach group, and 1 in the axillary approach group). Three dogs only required a single dose of glycopyrrolate and no further cardiovascular support (2 in the perpendicular approach group, 1 in the axillary approach group).

Nine of 24 dogs had thoracic limb amputations: four dogs in the traditional group, 3 dogs in the perpendicular group and 2 dogs in the axillary group. Dogs undergoing thoracic limb amputations versus those not undergoing thoracic limb amputations had higher pain scores (2.4 [2.4-3.0] vs. 1.6 [1.4-2.2]; median [lower and upper quartile]) in the first 3 hours post extubation, higher recovery scores (1 [0-2] vs. 0 [0-0]), higher anesthesia scores (0 [0-1] vs. 0 [0-0]), longer anesthetic times (203 [186-235] vs. 160 [140-230] minutes), longer surgery times (115 [100-138] vs. 50 [41-90] minutes), were of older age (8 [6.5-10] vs. 1 [0.5-3] years), and higher body weight (30.4 [27.6-36.2] vs. 20.2 [5.2-28.3] kg), Table 3.3

There were no differences in anesthetic scores or recovery scores between groups, indicating that maintenance of anesthesia and analgesia postoperative scores were similar regardless of the approach used to block the brachial plexus. Only one dog from the
The perpendicular approach group received two doses of lidocaine for rescue analgesia in the intraoperative period. Based on the duration of surgery and the planned frequency of hydromorphone administration, in the traditional approach group, 5 dogs received a single dose and 3 dogs received two doses; in the perpendicular approach group, 5 dogs received a single dose, 2 dogs received two doses, and one dog did not receive intraoperative analgesia; and in the axillary approach group, all dogs received a single dose.

The post-operative pain scores using the CMPS–SF indicate the need for rescue analgesia if scores ≥ 5/20 (non ambulatory) or 6/24 (ambulatory) were obtained. For the traditional approach, there were 4 dogs that had scores ≥ 5 for a total of 8 assessments; 3 dogs had amputations with 5 scores ≥ 5, including one score before anesthesia, and one young dog (3 months of age) had a fractured radius/ulna repair with 3 scores ≥ 5. For the perpendicular approach, 6 dogs had scores ≥ 5 for a total of 12 assessments; 2 dogs had amputations with 5 scores ≥ 5, whereas the other 4 dogs did not have amputations and had 7 scores ≥ 5, including 2 scores before anesthesia. For the axillary approach, 2 dogs had 2 scores ≥ 5; one dog had an amputation and the other did not have an amputation and showed this score before anesthesia (Table 3.4). The median (95% confidence interval) number of doses of hydromorphone received in the 24-hour postoperative period for each group was 3 (1.6-4.0) for the traditional approach group, 3 (2.0-3.2) for the perpendicular approach group, and 2 (0.8-3.0) for the axillary approach group.
3.5 DISCUSSION

In this study the anesthetic maintenance score, recovery score, and postoperative analgesia resulting from three blind brachial plexus block techniques in canine patients undergoing surgical procedures of the thoracic limb were compared. The results of this study supported the hypothesis that the degree of analgesia provided was not different between the three techniques. From Chapter II, a previous cadaver study, the transverse process of C₆, the point of the shoulder and the first rib were identified as fundamental landmarks when performing the brachial plexus block to ensure appropriate needle location and these three landmarks were utilized during the performance of each of the three techniques in this clinical study. Blind techniques are inexpensive and if anatomical landmarks are accurately identified, they can be clinically effective. A dog cadaver study using blind techniques for staining of the saphenous, obturator, and lateral cutaneous femoral nerves also showed consistent and optimal nerve staining when anatomical landmarks were followed closely (Echeverry-Bonilla et al. 2017) and this should result in effective clinical blocks with local anesthetics.

Differences in degree of analgesia were identified when evaluating dogs that underwent thoracic limb amputation versus those that did not. Amputation of the thoracic limb can be performed as a salvage procedure following traumatic injury or, as for the dogs in this study, for the removal of the primary lesion of painful and metastatic cancer (Smeak & Lisano 2012). The surgical technique for amputation of the thoracic limb in dogs includes a skin incision that begins at the most dorsal aspect of the scapular spine and dissection of the muscles participating in synsarcosis to reflect the scapula from the body wall (Smeak & Lisano 2012). The innervation to the skin of the caudal cervical region is provided by the dorsal rami of the C₅, C₆, T₂ and T₃ spinal nerves and innervation to the trapezius muscle, omotransversarius muscle and part of the brachiocephalicus muscle is provided by cranial nerve eleven (spinal accessory
nerve) (Dyce et al. 2010; Grimm et al. 2015); none of which would be anesthetized by the brachial plexus block. This innervation of the dorsal shoulder and surrounding skin would explain why dogs undergoing amputation of the thoracic limb did not experience the same degree of postoperative comfort as dogs undergoing surgical procedures of the antebrachium and distal forelimb, as demonstrated by higher postoperative pain scores.

Pain evaluation in veterinary species is challenging, particularly when comparing the degree of pain across individuals. A number of pain scales have been developed for use in veterinary species in an attempt to standardize pain evaluation, for provision of sufficient analgesia and for purposes of clinical comparison. Pain assessments were performed using the CMPS–SF scale (Reid et al. 2007), which aims at providing a clinical and objective method for evaluating the degree of post-operative pain in dogs using a descriptive statement for six behavioral categories that best describe the conduct of the dog with regard to comfort level (Reid et al. 2007). The scale has a maximum score indicating the need for provision of rescue analgesia. The scores indicating the need for rescue analgesia were ≥ 5/20 (non ambulatory) or 6/24 (ambulatory).

Since the dogs in this study were undergoing major surgical procedures, the anesthetic protocol for the dogs in this study utilized a balanced anesthetic technique. Such balanced anesthetic techniques have become popular across species in veterinary medicine and refers to the use of a combination of analgesic and anesthetic drugs to exploit the benefits of each drug while minimizing the adverse effects that would occur from large doses of a single anesthetic agent, typically inhalant anesthetics (Ilkiw, 1999). Inhalant anesthetics are most commonly used in veterinary medicine for maintenance of general anesthesia; however, large doses of inhalant anesthetics can result in severe cardiovascular and respiratory depression (Ilkiw, 1999; Grimm et al. 2015). In dogs, opioids are known to decrease inhalant requirements to maintain a surgical plane of anesthesia. Hydromorphone is a μ opioid receptor agonist that was chosen
as part of the balanced anesthetic protocol in this study because it has been demonstrated to both reduce isoflurane requirements (Machado et al. 2006) and to provide effective postoperative analgesia (Hoelzler et al. 2005; Bateman et al. 2008) in dogs.

The minimum alveolar concentration (MAC) is a measure of potency of volatile anesthetic agents and is defined as the concentration of inhalant anesthetic required to prevent purposeful movement in response to a noxious stimulus in 50% of a population (Eger et al. 1965; Ilkiw, 1999). In order to prevent purposeful movement in 95% of a population in response to a noxious stimulus (a surgical plane of anesthesia), typically values of 1.2 to 1.4 times MAC are required (Ilkiw, 1999). The MAC of isoflurane in dogs varies depending on the study but typically is reported to be between 1.2-1.4% (Valverde et al. 2003; Machado et al. 2006). In this study, the initial ETIso was set at 1.5% considering the reported range of MAC of isoflurane for dogs and that dogs had received anesthetic drugs and techniques that would decrease their inhalant requirements including acepromazine (Boyd et al. 1991), hydromorphone (Machado et al. 2006) and the brachial plexus block. Cardiorespiratory parameters were similar between the three groups and values considered optimal for anesthetized dogs. The number of dogs requiring blood pressure support with hypotension was also similar between groups. The hypotension that occurred during anesthesia may be the result of the dose-dependent vasodilatory effects of isoflurane (Mutoh et al. 1997), since the initial ETIso of 1.5% exceeded the 1.0 MAC value reported for isoflurane in dogs. In addition, the use of hydromorphone and acepromazine in the premedication may have exacerbated the cardiovascular depression of isoflurane from synergistic effects.

This study had several limitations. The dogs included in this study were administered a variety of analgesics for pain management including an opioid, a non-steroidal anti-inflammatory and a local block technique with a local anesthetic. The anesthetic protocol was the same across all dogs included in the study and thus the authors believe the differences
detected, or lack thereof, have to do with block technique rather than inclusion of other analgesics. Furthermore, it would be unethical for dogs undergoing such extensive surgical procedures to only receive a local block and no other analgesic agent. A placebo group was not included in this study for two reasons. First, the objective of the study was to compare the degree of analgesia that resulted from the three brachial plexus block techniques. Second, it was anticipated that owner enrolment in the study would be difficult if one possibility was that the dog would not receive any sort of local block. Dogs were also administered postoperative analgesia according to a pre–scheduled format, following the standards of our hospital. Analgesia was restricted in the first three hours post-extubation to allow for a more critical assessment of the degree of analgesia that resulted from the brachial plexus block. Thereafter, routine analgesia was included as deemed necessary, sometimes regardless of the pain score, to prevent discomfort, to facilitate the handling of the patient, and to comply with the requirements for the surgical intervention. This type of analgesic management could have influenced the pain scores; however, analgesia after brachial plexus block can last 11 hours with bupivacaine and epinephrine administered at a dose of 4 mg kg\(^{-1}\) (volume of 1.07 mL kg\(^{-1}\)) (Futema et al. 2002). The dose used in our study was 1 mg kg\(^{-1}\) (volume of 0.2 mL kg\(^{-1}\)) and may have lasted for a shorter time because it was a lower volume.

In conclusion, the three anatomical approaches evaluated here to block the brachial plexus in canine patients undergoing surgical procedures of the thoracic limb provided similar degree of comfort in the post-operative period. Dogs undergoing thoracic limb amputations exhibited less comfort postoperatively, regardless of provision of brachial plexus block, than dogs not undergoing thoracic limb amputations.
3.6 REFERENCES


Table 3.1. Description of dogs anesthetized with isoflurane undergoing thoracic limb surgery (no amputation [n=15] versus amputation n=9]) that received 0.2 mL kg\(^{-1}\) (0.09 mL lb\(^{-1}\)) of bupivacaine at the brachial plexus before surgery.

<table>
<thead>
<tr>
<th>Brachial Plexus Block</th>
<th>Age (years)</th>
<th>Body weight (kg)</th>
<th>Acepromazine (mg/kg)</th>
<th>Propofol (mg/kg)</th>
<th>Intra-operative hydromorphone doses</th>
<th>Anesthesia time (min)</th>
<th>Surgery time (min)</th>
<th>No amputation / amputation</th>
<th>Surgical procedure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Traditional</td>
<td>5.5</td>
<td>26.8</td>
<td>0.03</td>
<td>2.3</td>
<td>2</td>
<td>235</td>
<td>123</td>
<td>4 / 4</td>
<td>4 amputations, 1 radius/ulnar fracture, 1 shoulder arthroscopy, 1 mass removal 3 radius/ulna fracture, 2 amputations, 1 humeral condylar fracture, 1 shoulder/elbow arthroscopy, 1 elbow arthroscopy</td>
</tr>
<tr>
<td>Perpendicular</td>
<td>2.6</td>
<td>20.5</td>
<td>0.025</td>
<td>3.1</td>
<td>1</td>
<td>185</td>
<td>85</td>
<td>5 / 3</td>
<td>3 radius/ulnar fractures, 1 humeral fracture, 1 digit amputation</td>
</tr>
<tr>
<td>Axillary</td>
<td>2.9</td>
<td>25.8</td>
<td>0.03</td>
<td>2.8</td>
<td>1</td>
<td>165</td>
<td>52</td>
<td>6 / 2</td>
<td>3 amputations, 3 radius/ulnar fractures, 1 humeral fracture, 1 digit amputation</td>
</tr>
</tbody>
</table>

Data expressed as median (lower-upper quartile). * No significant differences between groups.
Table 3.2. Anesthetic parameters in dogs anesthetized with isoflurane undergoing thoracic limb surgery that received 0.2 mL kg\(^{-1}\) (0.09 mL lb\(^{-1}\)) of bupivacaine at the brachial plexus before surgery using one of three anatomical approaches (n=8 per group).

<table>
<thead>
<tr>
<th>Brachial Plexus Block</th>
<th>Pain score</th>
<th>Sedation score</th>
<th>Anesthesia score</th>
<th>Recovery score</th>
<th>End-tidal isoflurane (%)</th>
<th>End-tidal CO(_2) (mmHg)</th>
<th>Systolic arterial pressure (mmHg)</th>
<th>Heart rate (beats per min)</th>
<th>Temperature (°C)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Traditional</td>
<td>2.1 (1.6-2.8)</td>
<td>2 (0.5-3)</td>
<td>0 (0-0.5)</td>
<td>0.5 (0-1)</td>
<td>1.41 (1.37-1.45)</td>
<td>40 (39-40)</td>
<td>95 (92-103)</td>
<td>83 (78-94)</td>
<td>36.5 (36.4-36.9)</td>
</tr>
<tr>
<td>Perpendicular</td>
<td>1.7 (1.5-3.3)</td>
<td>2 (1.5-2)</td>
<td>0 (0-0.5)</td>
<td>0 (0-2)</td>
<td>1.40 (1.35-1.44)</td>
<td>40 (37-43)</td>
<td>103 (99-122)</td>
<td>100 (91-104)</td>
<td>36.1 (35.3-37.0)</td>
</tr>
<tr>
<td>Axillary</td>
<td>1.6 (1.2-2.0)</td>
<td>2 (1.5-2)</td>
<td>0 (0-0)</td>
<td>0 (0-0)</td>
<td>1.38 (1.36-1.39)</td>
<td>41 (38-44)</td>
<td>93 (87-105)</td>
<td>87 (74-97)</td>
<td>36.2 (35.8-36.9)</td>
</tr>
</tbody>
</table>

Data expressed as median (lower-upper quartile). No significant differences between groups.
Table 3.3. Anesthetic parameters in dogs anesthetized with isoflurane undergoing forelimb surgery (no amputation [n=15] versus amputation [n=9]) that received 0.2 mL kg\(^{-1}\) (0.09 mL lb\(^{-1}\)) of bupivacaine at the brachial plexus before surgery.

<table>
<thead>
<tr>
<th>Surgery</th>
<th>Pain score 0-3 h post-extubation</th>
<th>Pain score 0-24 h post-extubation</th>
<th>Recovery score</th>
<th>Post-operative hydromorphone (doses)</th>
<th>Anesthesia score</th>
<th>Anesthesia time (min)</th>
<th>Surgery time (min)</th>
<th>Age (years)</th>
<th>Body weight (kg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>No amputation</td>
<td>1.6* (1.4-2.2)</td>
<td>1.5 (1.2-2.2)</td>
<td>0* (0-0)</td>
<td>3* (3-3)</td>
<td>0* (0-0)</td>
<td>160* (140-230)</td>
<td>50* (41-90)</td>
<td>1.0* (0.5-3.0)</td>
<td>20.2* (5.2-28.3)</td>
</tr>
<tr>
<td>Amputation</td>
<td>2.4 (2.4-3.0)</td>
<td>2.0 (1.8-2.8)</td>
<td>1 (0-2.0)</td>
<td>4 (4-5)</td>
<td>0 (0-1)</td>
<td>203 (186-235)</td>
<td>115 (100-138)</td>
<td>8.0</td>
<td>30.4 (27.6-36.2)</td>
</tr>
<tr>
<td>* p value</td>
<td>0.011</td>
<td>0.081</td>
<td>0.005</td>
<td>&gt; 0.0001</td>
<td>0.048</td>
<td>0.071</td>
<td>0.006</td>
<td>0.0001</td>
<td>0.01</td>
</tr>
</tbody>
</table>

Data expressed as median (lower-upper quartile). * Significantly lower than for amputation.
Table 3.4. Number of dogs with Glasgow composite pain scores equal to or higher than 5/20 (non-ambulatory) or 6/24 (ambulatory) that indicated need for rescue analgesia after thoracic limb surgery (no amputation [n=15] versus amputation n=9]) that received 0.2 mL kg⁻¹ (0.09 mL lb⁻¹) of bupivacaine at the brachial plexus before surgery. Value in parenthesis indicates the specific score. Values in brackets indicate the group [TA- traditional; PA- perpendicular; AA- axillary].

<table>
<thead>
<tr>
<th>Surgery</th>
<th>Number of dogs before anesthesia</th>
<th>0.25</th>
<th>0.5</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>6</th>
<th>9</th>
<th>12</th>
<th>16</th>
<th>24</th>
</tr>
</thead>
<tbody>
<tr>
<td>No amputation</td>
<td>3 (5,6,7) [PA, AA, PA]</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>2</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Amputation</td>
<td>1 (6) [TA]</td>
<td>3</td>
<td>2</td>
<td>1</td>
<td>2</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>(11)</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

Note: Values in brackets indicate the group [TA- traditional; PA- perpendicular; AA- axillary].
APPENDIX 1

<table>
<thead>
<tr>
<th>Sedation score at the time of catheterization or induction</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 Bright and alert, no apparent sedation and/or excitable-dysphoric (excited, anxious, difficult to restrain in recumbency, very interactive and responsive, vocalizing, reactive to voice and touch)</td>
</tr>
<tr>
<td>1 Calm however minimal sedation (quiet but still alert and aware of surroundings, mild resistance to restraint for lateral recumbency, moderate response to voices and touch; mild resistance to catheterization)</td>
</tr>
<tr>
<td>2 Very calm, with moderate sedation (quiet, relaxed, minimal restraint required for lateral recumbency, mild response to voices or touch; no resistance to catheterization)</td>
</tr>
<tr>
<td>3 Profound sedation (quiet, very relaxed, no restraint necessary for lateral recumbency, does not respond to voice or touch)</td>
</tr>
</tbody>
</table>
**APPENDIX 2**

<table>
<thead>
<tr>
<th>Anesthetic Maintenance Quality Score</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 0</td>
<td>No adjustments required in the depth of anesthesia during maintenance. No autonomic responses (less than 20% increase in BP from previous value and/or signs of bucking the ventilator)</td>
</tr>
<tr>
<td>1 1</td>
<td>Dog shows autonomic responses (BP increases suddenly &gt; 20% from previous value and/or signs of bucking the ventilator), which requires of increasing the end-tidal isoflurane to 1.5% for return of a stable anesthetic depth</td>
</tr>
<tr>
<td>2 2</td>
<td>Dog shows autonomic responses (BP increases suddenly &gt; 20% increase from previous value and/or signs of bucking the ventilator), which requires of increasing the end-tidal isoflurane to 1.5% and administration of 1-2 boluses of lidocaine (2 mg kg(^{-1}), IV) for return of a stable anesthetic depth</td>
</tr>
<tr>
<td>3 3</td>
<td>Dog shows autonomic responses (BP increases suddenly &gt; 20% increase from previous value and/or signs of bucking the ventilator), which requires of increasing the end-tidal isoflurane to 1.5%, administration of &gt; 2 boluses of lidocaine (2 mg kg(^{-1}), IV), and repeat of hydromorphone (0.03 mg kg(^{-1})) &lt; 2 hours from the previous dose, for return of a stable anesthetic depth</td>
</tr>
<tr>
<td>4 4</td>
<td>As 3, but there is no return of a stable anesthetic depth despite all actions</td>
</tr>
</tbody>
</table>
# APPENDIX 3

**Recovery score from the time of extubation to the first 15 minutes**

<table>
<thead>
<tr>
<th>Score</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Profound sedation (quiet, very relaxed, no restraint necessary for lateral recumbency, does not respond to voice or touch)</td>
</tr>
<tr>
<td>1</td>
<td>Very calm, with moderate sedation (quiet, relaxed, minimal restraint required for lateral recumbency, mild response to voices or touch)</td>
</tr>
<tr>
<td>2</td>
<td>Calm however minimal sedation (quiet but still alert and aware of surroundings, mild resistance to restraint for lateral recumbency, moderate response to voices and touch)</td>
</tr>
<tr>
<td>3</td>
<td>Minimal or no apparent sedation and/or excitable-dysphoric (excited, anxious, difficult to restrain in recumbency, very interactive and responsive, vocalizing, reactive to voice and touch)</td>
</tr>
</tbody>
</table>
APPENDIX 4

A. Look at dog in Kennel

Is the dog

(i)

<table>
<thead>
<tr>
<th>Behavior</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Quiet</td>
<td>0</td>
</tr>
<tr>
<td>Crying or whimpering</td>
<td>1</td>
</tr>
<tr>
<td>Groaning</td>
<td>2</td>
</tr>
<tr>
<td>Screaming</td>
<td>3</td>
</tr>
</tbody>
</table>

(ii)

<table>
<thead>
<tr>
<th>Behavior</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ignoring any wound or painful area</td>
<td>0</td>
</tr>
<tr>
<td>Looking at wound or painful area</td>
<td>1</td>
</tr>
<tr>
<td>Licking wound or painful area</td>
<td>2</td>
</tr>
<tr>
<td>Rubbing wound or painful area</td>
<td>3</td>
</tr>
<tr>
<td>Chewing wound or painful area</td>
<td>4</td>
</tr>
</tbody>
</table>

In the case of spinal, pelvic or multiple limb fractures, or where assistance is required to aid locomotion do not carry out section B and proceed to C

Please tick if this is the case [ ] then proceed to C

B. Put lead on dog and lead out of the kennel

When the dog rises/walks is it?

(iii)

<table>
<thead>
<tr>
<th>Behavior</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>0</td>
</tr>
<tr>
<td>Lame</td>
<td>1</td>
</tr>
<tr>
<td>Slow or reluctant</td>
<td>2</td>
</tr>
<tr>
<td>Stiff</td>
<td>3</td>
</tr>
<tr>
<td>It refuses to move</td>
<td>4</td>
</tr>
</tbody>
</table>

C. If it has a wound or painful area including abdomen, apply gentle pressure 2 inches round the site

Does it?

(iv)

<table>
<thead>
<tr>
<th>Behavior</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Do nothing</td>
<td>0</td>
</tr>
<tr>
<td>Look round</td>
<td>1</td>
</tr>
<tr>
<td>Flinch</td>
<td>2</td>
</tr>
<tr>
<td>Growl or guard area</td>
<td>3</td>
</tr>
<tr>
<td>Snap</td>
<td>4</td>
</tr>
<tr>
<td>Cry</td>
<td>5</td>
</tr>
</tbody>
</table>

D. Overall

Is the dog?

(v)

<table>
<thead>
<tr>
<th>Behavior</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Happy and content or happy and bouncy</td>
<td>0</td>
</tr>
<tr>
<td>Quiet</td>
<td>1</td>
</tr>
<tr>
<td>Indifferent or non-responsive to surroundings</td>
<td>2</td>
</tr>
<tr>
<td>Nervous or anxious or fearful</td>
<td>3</td>
</tr>
<tr>
<td>Depressed or non-responsive to stimulation</td>
<td>4</td>
</tr>
</tbody>
</table>

Is the dog?

(vi)

<table>
<thead>
<tr>
<th>Behavior</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Comfortable</td>
<td>0</td>
</tr>
<tr>
<td>Unsettled</td>
<td>1</td>
</tr>
<tr>
<td>Restless</td>
<td>2</td>
</tr>
<tr>
<td>Hunched or tense</td>
<td>3</td>
</tr>
<tr>
<td>Rigid</td>
<td>4</td>
</tr>
</tbody>
</table>

Total Score \((i+ii+iii+iv+v+vi) = \) _______
CHAPTER 4

GENERAL DISCUSSION AND FINAL CONCLUSIONS
4.1 GENERAL DISCUSSION

This investigation had a main objective of improving our knowledge of the canine brachial plexus anatomy and to determine the best technique to perform an effective brachial plexus block by way of a blind approach using only anatomical landmarks without the use of devices (ultrasound, nerve locator), during anesthetic management of cases undergoing surgical procedures of the thoracic limb.

Pain management has become an important interest in veterinary medicine over the last few decades. Veterinarian attitudes to animal pain have resulted in more consistent and widespread use of analgesic drugs and techniques that relieve animal discomfort and stress. Often, pain management starts before its onset, but in most instances, this is only feasible in elective procedures. This preemptive approach can prevent pathophysiological mechanisms associated with nociception, from the transduction of the nociceptive stimulus to cortical perception and the associated pain response. In other instances, pain management is in response to an ongoing and actively painful condition that may require more aggressive management that incorporates multimodal analgesia and multiple techniques for pain management. Therefore, a preemptive approach has advantages and is preferred where achievable.

Local anesthetic techniques are widely used to provide anesthesia and analgesia in veterinary species. It was Albert Nieman who, in 1860, first isolated the active alkaloid in its crystalline form from the cocoa plant and named it “cocaine” (Deschner et al. 2007). The substance was reported to result in numbness of the tongue upon contact and thus began the basis for the use of this substance for blocking reflexes and treating pain in
animals (Keys 1942; Deschner et al. 2007). Reports of regional anesthesia in veterinary species began to emerge in the early nineteen hundreds with subarachnoid and epidural anesthesia being reported in horses, cattle and dogs (Campoy & Read 2013). Currently, locoregional anesthesia has become commonplace across species in the practice of veterinary medicine to provide anesthesia and analgesia, facilitate diagnostic or minor surgical procedures and/or to decrease the requirement of other anesthetic agents. Locoregional anesthesia encompasses a variety of techniques that include: topical anesthesia, local anesthesia, regional anesthesia, neuraxial anesthesia and intravenous regional anesthesia (Campoy & Read 2013). These techniques can be used independently or as part of multimodal analgesia in anesthetized patients. Local anesthetic techniques that target specific nerves and the contained nociceptive fibres (Aδ and C) can block all afferent (sensory) input from the corresponding innervated area and therefore decrease the requirements for other systemic analgesic and anesthetic drugs during anesthesia. For the veterinary practitioner, the safe and successful delivery of locoregional anesthesia requires species-specific knowledge of anatomical landmarks due to the anatomical variation that exists across species.

Surgical procedures of the thoracic limb are common in canine patients and include: fracture repair or other orthopedic procedures, soft tissue procedures or entire limb amputation. As part of a balanced anesthetic and analgesic protocol, blockade of the brachial plexus is commonly performed in these patients. In the literature, it is reported most commonly that the canine brachial plexus is formed from the ventral spinal nerve rami of C6, C7, C8 and T1 (Campoy & Read 2013; Grimm et al. 2015). Older reports document minor contributions in a small percentage of dogs from C5 and/or T2 (Miller
In the cadaver study performed here, no contributions to the brachial plexus from the ventral spinal nerve rami of C5 or T2 were identified in any of the study dogs. While clinically, the consequence of these findings are undoubtedly minimal our cadaver work confirmed the reports in most recent literature that the canine brachial plexus is formed from combinations of these four ventral spinal nerve rami. The most relevant nerves that provide sensory innervation to the thoracic limb include the musculocutaneous, radial, ulnar and median nerves. This study determined that all of these nerves are formed from the ventral spinal rami of C7, C8 and T1. The musculocutaneous nerve was always formed from C7 and C8 (100% of dogs). The radial nerve was predominantly formed from C8 and T1 (89% of dogs), but also received contribution from C7 in the remaining dogs. Similarly, the median and ulnar nerves were predominantly formed from C8 and T1 (94% of dogs) but also received contribution from C7 in the remaining dog. Therefore, the most important ventral spinal rami to block in the brachial plexus are C8 and T1, and some dogs may also require C7.

The current description in the literature for performing the blind brachial plexus block in dogs is very thorough and defines a number of anatomical landmarks that must be identified: scapulohumeral joint, acromion, greater tubercle of the humerus, jugular groove, and first rib (Campoy & Read 2013; Grimm et al. 2015). Despite the very comprehensive description, it can be difficult to ascertain the end point for needle positioning that will result in successful and complete anesthesia and analgesia of the brachial plexus. This difficulty was encountered during the cadaver study, regardless of the technique used to approach the brachial plexus. The cadaver dye study was a randomized, blinded study using the traditional approach and two novel approaches.
(axillary and perpendicular) to the brachial plexus with the objective of determining which technique was more effective at placing the dye at the level of the brachial plexus. Results of the cadaver study demonstrated that, regardless of technique, needle placement was often inaccurate and that unsuccessful dye injection was as high as 50%; whereas incomplete staining was not higher than 33% of the cadavers; and complete staining was never higher than in 58% of the cadavers.

As a result of these dye scores throughout the study and careful anatomical dissection, it was possible to identify a simpler approach to the brachial plexus block in dogs that includes three key anatomical landmarks: the transverse process of C₆, the point of the shoulder, and the first rib. If the needle was advanced at a level that was midway between the transverse process of C₆ and the point of the shoulder and advanced to an end point that was just cranial to the first rib, there was consistent success in staining of the brachial plexus, regardless of the technique used. Keeping a finger on the first rib during needle advancement would help ensure appropriate needle placement prior to dye injection. Although techniques for performing the brachial plexus block in dogs using advanced equipment (electrolocation or ultrasonography) have both been described (Futema et al. 2002; Mahler & Reece 2007; Mahler & Adogwa 2008; Campoy et al. 2010; Campoy & Read 2013; Grimm et al. 2015), these modalities are not readily available to veterinary practitioners that work outside of large specialty hospitals and it has not been demonstrated that they improve the success rate of the block (Akasaka & Shimizu 2017). One of our goals was to describe a simplified, blind approach to performing the brachial plexus block in dogs that can be executed without the use of advanced pieces of equipment.
The second part of this study investigated the clinical application of the three approaches (traditional, perpendicular, and axillary) to the brachial plexus, with a better understanding of the anatomy and proper use of the landmarks identified from the cadaver study. The objective was to evaluate the degree of anesthesia and postoperative analgesia resulting from the three approaches in canine patients undergoing surgical procedures of the thoracic limb. Using a prospective, randomized, blinded study, the three techniques were compared in 24 dogs presenting for surgical procedures of the thoracic limb (n = 8 dogs/technique), which included limb amputation, and soft tissue or orthopedic procedures of the thoracic limb.

Results from this study demonstrated that there were no differences in anesthetic scores or recovery scores between groups, indicating that maintenance of anesthesia and postoperative analgesia scores were similar regardless of the technique used to perform the brachial plexus block. These pain scores were derived using the Glasgow Composite Pain Scale; which indicates the need for rescue analgesia if dogs receive scores ≥ 5/20 (non ambulatory) or 6/24 (ambulatory). With the design of this investigation and the inclusion of amputation cases, we were able to also compare dogs undergoing thoracic limb amputation with those cases undergoing surgical procedures that involved the more distal aspect of the thoracic limb. Dogs undergoing thoracic limb amputations (n = 9) versus those not undergoing thoracic limb amputations (n = 15) had higher pain scores in the first 3 hours post-extubation, as well as higher recovery scores and higher anesthesia scores, which provided confirmation of incomplete analgesia due to the more extensive and dorsal surgical approach to the limb. The innervation to the most proximal aspect of the shoulder muscles and skin comes from nerves that do not originate within the brachial plexus (Dyce
et al. 2010; Grimm et al. 2015). Despite the logic for this realization in clinical practice, this is the first study that demonstrates that the brachial plexus block provides less effective analgesia for thoracic limb amputations. However, due to the balanced anesthetic technique used in our study, the postoperative pain scores obtained are still acceptable and the anesthetic effects of the block for the distal aspect of the thoracic limb are most likely beneficial to the patient during the surgical procedure.

This project had several limitations. With respect to the cadaver phase of the investigation, the utilization of cadaver dogs may have resulted in different dye dispersion in the area of the brachial plexus than would occur in the live animal and no comments could be made on any increased risk of hemorrhage or hematoma formation between techniques. Furthermore, only the left brachial plexus was dissected and there could have been differences in the right brachial plexus anatomy that were not identified in our study. For the clinical phase of the investigation, the power of the study could have affected the interpretation of the results. We considered three alternatives to determine the sample size. If the Glasgow Pain Score was used as the end-point to compare the groups, assuming a 2 point difference in the score with a standard deviation of 1, a type I error (α value) of 0.05 and a desired power of 80%, a sample size of 5 dogs per group was required; for a power of 90%, a sample size of 7 dogs per group; and for a power of 99%, a sample size of 11 dogs per group was required. If using the end-tidal concentration of isoflurane required to maintain a stable plane during anesthesia was used as the end-point, and assuming a 0.5% difference and a standard deviation of 0.25%, the same results as for the pain score were obtained. Finally, if differences in systolic blood pressure were used as the end-point, assuming a difference of 30 mm Hg and a standard deviation of 20 mm Hg, the sample size
was 8 dogs per group for a power of 80%, 11 per group for a power of 90%, and 18 per group for a power of 99%. From these calculations we selected 8-10 dogs per group, according to the availability of cases and time. A placebo group was not included in this study for two reasons. First, the objective of the study was to compare the degree of analgesia that resulted from the three brachial plexus block techniques. Second, it was anticipated that owner enrolment in the study would be difficult if one possibility was that the dog would not receive any sort of local block.
4.2 FINAL CONCLUSIONS

Overall, the investigations performed and discussed here demonstrated the following results:

1. The canine brachial plexus in this population of dogs was formed from the ventral spinal nerve rami of C₆, C₇, C₈ and T₁.

2. The transverse process of C₆, the point of the shoulder, and the first rib are key anatomical landmarks for provision of an accurate and successful brachial plexus block in dogs.

3. Any one of the three blind approaches described here to perform the brachial plexus block in canine patients provides postoperative analgesia in patients undergoing surgical procedures of the distal thoracic limb.

4. Blockade of the brachial plexus alone does not provide sufficient analgesia and comfort in canine patients undergoing amputation of the thoracic limb.

5. Veterinarians can perform the blind brachial plexus block in dogs successfully following the anatomical landmarks described.
4.3 REFERENCES


Miller RA (1934) Comparative studies upon the morphology and distribution of the brachial plexus. Am J Anat 54, 143-175.