Canine Ovariectomy

Subjects: Veterinary Sciences

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Canine ovariectomy is an elective surgery with a moderate level of pain.

local anaesthetic in canine ovariectomy
analgesia in canine neutering
regional techniques in canine ovariectomy

1. Introduction

In recent decades, in veterinary medicine, the focus on the prevention, assessment, and treatment of surgical pain has substantially increased. As is known from the physiology of domestic animals, untreated pain results in many untoward responses involving all body systems: tachycardia, hypercoagulability, hypoventilation, hypoxemia, sepsis, stress, anxiety, reduced food intake, release of stress hormones, weight loss, immunosuppression, and increased blood pressure [1][2]. One of the objectives of veterinary medicine is to provide adequate analgesia to help the patient not feel pain and to move, eat, and sleep without discomfort, particularly in the first hours after the surgery [3]. Multimodal analgesia involves the use of multiple analgesic agents and provides the administration of both systemic and regional drugs. This method includes local and regional blocks that are safe and efficacious in dogs, when performed correctly. Local anaesthetic drugs can be injected directly into tissues to provide analgesia for manipulation or wounds or can be injected perineurally to provide analgesia for a wide variety of painful conditions. Owing to the potential to provide profound analgesia, these classes of drugs are recommended as part of the analgesic protocol in the majority of patients undergoing surgical procedures, such as canine ovariectomy. There are numerous local and regional blocks described for canine use, with the aim of reducing the dosage of the systemic drugs used and their possible side effects, while still maximising the desired effect [4]. Local anaesthetics have been used in analgesic protocols in the form of peripheral nerve blocks, epidural injections, intracavitary instillation, and regional blocks [5].

2. Local Anaesthetics

The mechanism of action of local anaesthetics is based on the blockade of sodium channels and the reversible blockage of the generation and propagation of electrical nerve impulses, which causes the blockage of the action of sensory and motor nerves. In veterinary medicine, local anaesthetics are extensively used in local and regional anaesthetic techniques. These techniques provoke desensitization of a localized area of the body, primarily causing ion channel blockers to act mainly on voltage-gated Na^+ channels. The use of local anaesthesia results in the blockade of dependent K^+ and Ca^{2+} channels, albeit with lower affinity [6][7][8][9][10], showing differential sensory

and motor blocking behaviour; when this blockage is carried out at the level of the central neuraxis or peripheral nerves, it becomes clinically observable [11][12]. Loss of sensation regarding temperature, acute pain, light touch, and motor activity is among the actions of local anaesthetics [13]. Differential blockade is the name of this mechanism, and many factors influence it, such as the length of the nerve into which the anaesthetic will be inoculated, as well as the concentration and type of drug. After the administration of the anaesthetic drug, its availability is regulated by several concurring factors such as mass flow, diffusion, binding to neural and non-neural structures, and vascular absorption. The rate and amount of systemic absorption of these kinds of drugs should be taken into account due to the possibility of reaching toxic plasma concentrations. For this reason, local anaesthetics with low systemic absorption are more manageable. This is influenced by many factors such as the site of inoculation, the vasoactivity and lipid solubility of the drug itself, the dose used and additives (such as vasoconstrictors), vasodilation, i.e., the influence of local nerve block, and the physiological condition of the animal [14]. Lidocaine is used for infiltration anaesthesia, peripheral nerve block, epidural and intrathecal block, and regional intravenous anaesthesia. Its duration of action is around 1–2 h, but can be increased to 3 h by adding adrenaline (epinephrine) [15]. Mepivacaine is a similar drug, but has a somewhat longer duration of action due to its less vasodilatory properties. Due to its lower neurotoxicity than other local anaesthetics, it is widely used in equine medicine for peripheral diagnostic blocks [16]. Bupivacaine and levobupivacaine are strongly lipophilic molecules and are about four-times stronger than lidocaine; they have an onset of about 20–30 min and a longer effect duration of 3 to 10 h [17]. Bupivacaine has cardiotoxic properties in dogs (if injected IV at a dosage of 4–20 mg/kg); in fact, it is not recommended for intravenous regional anaesthesia, nor for topical anaesthesia. Its clinical use concerns the procedures of infiltrative, peripheral nerve, epidural, and intrathecal blocks. Ropivacaine has a structure and onset similar to bupivacaine and mepivacaine; they also have intrinsic properties of differential blocking, mainly at low concentrations; for this, they are very suitable when looking for a sensory block associated with a minimum motor dysfunction. This drug sees the same clinical use of bupivacaine and at equivalent doses is slightly less potent in motor blockade and determines a shorter marginal sensory blockade (6 h) [17]. Its biphasic effect means that at concentrations lower than 0.5% causes vasoconstriction, while at concentrations higher than 1%, it causes vasodilation [18]. Several substances can be combined with local anaesthetics to prolong the duration and increase the potency of nerve blocks. These include vasoconstrictors, corticosteroids, buffering substances, and alpha-2-agonists. The most common association is with adrenaline, which with its vasoconstriction effect decreases the absorption of the anaesthetic by the tissue vessels, thus prolonging the effect of the molecule and the duration of the blockade [19][20]. Sodium bicarbonate can increase the speed of onset and the potency of the anaesthetic used and is able to decrease the pain accompanying the injection [20][21][22]. In human medicine, it has been shown that administering local anaesthetics in combination with dexamethasone perineurally enhances and prolongs peripheral nerve block [23]. The mechanism of action is not yet known, but dexamethasone succeeds in decreasing the activity of C-type fibres [24]. Molecules belonging to the alpha-2-adrenergic class can decrease the clearance of local anaesthetics and have an inhibitory effect on C-fibres and A-delta fibres [25][26]. Their role is widely discussed in prolonging and intensifying regional anaesthetic blockade [27][28]. In addition, the pKa of different local anaesthetics affects their absorption differently. The alkalization of the local anaesthetic solution before it is injected, the speed of onset of the nerve block, and the alteration of tissue pH (e.g., inflammation, infections) can modify the correct absorption of the drug [21][22][29]. In general, LA can cause toxic effects in the

nervous and cardiovascular systems. When a toxic dose is reached in plasma, inhibitory response mechanisms in the brain are blocked, leaving excitatory mechanisms free to act. This can lead to the onset of clinical signs such as muscular contractions and seizures. At the cardiac level, sodium channel blockade interferes with phase 0 of cardiac depolarization, the phase associated with the opening of the fast sodium channels. This mechanism is seen on the electrocardiogram (ECG) as an increase in PR and QRS intervals. It is known that the agents having high lipid solubility are more associated with toxicity, and R-enantiomers are more toxic than their laevorotatory counterparts. The system for treating signs of toxicity varies according to the severity. Convulsive activity can be kept under control with the use of benzodiazepines, and it is advisable to start supportive activity immediately with oxygen supplementation and, if necessary, mechanical ventilation. In the event of clinical signs suggestive of cardiovascular depression, action can be taken first with the use of fluids and cardiac inotropes. If arrhythmias occur, the use of other molecules acting as sodium channel blockers should be avoided. Local anaesthetics can also cause direct damage to tissues (neurotoxicity, chondrotoxicity), allergic reactions, and methemoglobinemia [30]. The toxic doses of the most commonly used LA reported in the literature for the canine species are lidocaine 20 mg/kg, bupivacaine 4.3 mg/kg, and mepivacaine 80 mg/kg [30].

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