Veterinary
Anaesthesia and Analgesia
Formerly the Journal of Veterinary Anaesthesia

Veterinary Anaesthesia and Analgesia, 2016, **43**, 371–378 doi:10.1111/vaa.12303

RESEARCH PAPER

Effects of intravenous and topical laryngeal lidocaine on heart rate, mean arterial pressure and cough response to endotracheal intubation in dogs

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Abstract

Objective To compare the effects of intravenous (IV) and topical laryngeal lidocaine on heart rate (HR), mean arterial pressure (MAP) and cough response to endotracheal intubation (ETI) in dogs.

Study design Prospective, randomized, blinded clinical study.

Animals Forty-two client-owned dogs (American Society of Anesthesiologists class I and II status) undergoing elective orthopaedic surgery.

Methods Dogs were randomized to three groups. Dogs in group SALIV received 0.1 mL kg^{-1} IV saline. Dogs in group LIDIV received 2 mg kg^{-1} IV 2% lidocaine. Dogs in group LIDTA received 0.4 mg kg^{-1} topically sprayed laryngeal 2% lidocaine. All dogs were premedicated with methadone (0.2 mg kg^{-1} IV). After 30 minutes, IV propofol was administered to abolish the lateral palpebral reflex and produce jaw relaxation. The allocated treatment was then administered and, after 30 seconds, further propofol was administered to abolish the medial palpebral reflex and facilitate ETI. HR and MAP were measured at four time-points using cardiac auscultation and automated oscillometry, respectively. The cough response at ETI was recorded. One-way ANOVA and *post hoc* Tukey adjustment were used to analyse parametric data. The Kruskal–Wallis test was used to analyse non-parametric data. Odds ratios were calculated for the cough response. A p-value of ≤ 0.05 was considered to indicate statistical significance.

Results In response to ETI, changes in MAP differed significantly between groups. In SALIV, MAP increased (4 ± 6 mmHg), whereas it decreased in LIDIV (6 ± 13 mmHg) ($p = 0.013$) and LIDTA (7 ± 11 mmHg) ($p = 0.003$). Dogs in SALIV were almost 10 times more likely to cough than dogs in LIDIV (odds ratio 9.75, 95% confidence interval 0.98–96.60; $p = 0.05$).

Conclusions and clinical relevance In propofol-anaesthetized dogs, IV and topical laryngeal lidocaine attenuated the pressor response to ETI, whereas IV lidocaine reduced the cough response.

Keywords cough, intravenous lidocaine, intubation, laryngotracheal lidocaine, pressor response.

Introduction

The sympathetic response to larygoscopy and endotracheal intubation (ETI) is well documented in humans (King et al. 1951; Forbes & Dally 1970). Reported responses include tachycardia (Shribman et al. 1987), elevation in arterial blood pressure (McCoy et al. 1995; Singh et al. 1995), increased intracranial pressure (Shapiro et al. 1972) and elicitation of the cough reflex (Yukioka et al. 1993). In cats, mechanical stimulation of the upper

respiratory tract was found to evoke a reflex increase in systemic blood pressure (Tomori & Widdicombe 1969). In dogs, an elevation in systolic blood pressure (Holzchuh et al. 1991; Jolliffe et al. 2007) and elicitation of the cough reflex (Jolliffe et al. 2007) have been reported at ETI. These haemodynamic disturbances may be variable in magnitude and duration and, although they may be of little or no consequence in healthy individuals, may be detrimental in patients with pre-existing hypertension, myocardial insufficiency or cerebrovascular disease (Fox et al. 1977). In vulnerable patients, failure to attenuate the sympathetic response to intubation may risk myocardial ischaemia (Edwards et al. 1994) or cardiac failure (Fox et al. 1977). Although the precise mechanism is not known, ETI in patients with head injury may also result in raised intracranial pressure and intracranial haemorrhage mediated via the pressor response or possibly a neuronal pathway linked to the cough reflex (Robinson & Clancy 2001).

Lidocaine is an amide local anaesthetic and has the greatest diversity of routes of application of all local anaesthetics (Webb & Pablo 2009). Mechanisms of action of intravenous (IV) lidocaine include the suppression of autonomic reflexes, a neuronal membrane stabilizing effect and inhibition of the release of excitatory neuroamines, particularly glutamate (Terada et al. 1999). In humans, IV lidocaine administration prior to intubation has been shown to reduce the pressor response (Wilson et al. 1991), prevent intracranial hypertension (Hamill et al. 1981) and reduce the cough reflex (Steinhaus & Gaskin 1963; Jakobsen et al. 1991). In anaesthetized infants in whom laryngospasm was induced, IV lidocaine reduced the incidence of laryngospasm (Erb et al. 2013). By contrast, other studies failed to find a clinical benefit of IV lidocaine in attenuating haemodynamic responses to ETI in humans (Miller & Warren 1990; Pathak et al. 1990; Singh et al. 1995). In dogs, the evidence for lidocaine use prior to ETI is limited. A study by Jolliffe et al. (2007) found that 1 mg kg^{-1} IV lidocaine failed to attenuate the pressor response or reduce the cough response in propofol-anaesthetized dogs.

The use of topical laryngotracheal lidocaine prior to ETI to prevent laryngospasm in infants is supported by results from a recent meta-analysis (Mihara et al. 2014). In adults, topically applied lidocaine is reported to attenuate the pressor response to intubation (Sklar et al. 1992; Mostafa et al. 1999) and reduce the cough reflex (Bülow et al. 1996). However, topical lidocaine application failed to prevent intracranial hypertension at intubation (Hamill et al. 1981). Hamilton et al. (2012) reported that topical lidocaine failed to suppress the cough response at ETI in children and was in fact associated with a higher incidence of desaturation than when it was not administered.

To the present authors' knowledge, the use of laryngotracheal lidocaine to attenuate the sympathetic response to ETI has not yet been investigated in dogs. A higher dose of IV lidocaine (2 mg kg^{-1}) was selected for this study because Jolliffe et al. (2007) reported no beneficial effect at ETI when using 1 mg kg^{-1} lidocaine IV. In the human literature, higher IV lidocaine doses (1.5 mg kg^{-1}) are reported to be associated with beneficial effects at ETI (Jakobsen et al. 1991; Wilson et al. 1991).

This study aimed to investigate the effects of IV and topical laryngeal lidocaine on heart rate (HR), mean arterial pressure (MAP) and cough response to ETI in dogs. Its secondary objective was to investigate whether IV or laryngotracheal lidocaine administration may have a propofol-sparing effect in ETI in dogs.

Materials and methods

Ethical approval was granted by the University of Liverpool Ethics Committee (VREC107a). Informed owner consent was obtained for all animals prior to their participation in the study.

Animals

Forty-two client-owned dogs [American Society of Anesthesiologists (ASA) class I and II status] with a median age of 3 years, 6 months (range: 7 months to 10 years, 2 months) and weighing a mean ± standard deviation of 30.4 ± 16.3 kg were included in the study. All dogs were scheduled for elective orthopaedic surgery at the Small Animal Teaching Hospital, University of Liverpool. Dogs with any form of cardiovascular disease were excluded from the study. All dogs were fasted from food for 8 hours and water was removed 2 hours prior to the administration of general anaesthesia.

Anaesthetic protocol

Each of the 42 dogs was randomized to one of three treatment groups using an envelope selected by an