Int.J.Curr.Microbiol.App.Sci (2019) *8*(4): 2382-2388

International Journal of Current Microbiology and Applied Sciences
***ISSN: 2319-7706* Volume 8 Number 04 (2019)**
Journal homepage: http://www.ijcmas.com

Original Research Article https://doi.org/10.20546/ijcmas.2019.804.277

Effect on Clinico-Physiological Parameters after Administration of Buprenorphine-Propofol Anaesthesia in Atropinized Goats

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Keywords

Atropine sulphate, Buprenorphine, Clinico-physiological, Goats, Propofol

Article Info

Accepted:
17 March 2019
Available Online:
10 April 2019

A B S T R A C T

The experiment was conducted on six healthy non-descript goats of either sex weighing between 20-25 kg by administrating atropine sulphate @ 0.04 mg/kg I/M followed by buprenorphine @ 10 mg/kg I/M and 10 min. later followed by induction of anaesthesia with propofol @ 5mg/kg I/V. After buprenorphine administration, lowering of head was observed in all the animals within 8.20±0.58 min. After propofol injection, there was rapid and smooth onset of anaesthesia (0.50±0.55 min). Swallowing reflex, corneal and palpebral reflexes abolished within 3 min after onset of anaesthesia which remained throughout the period of duration of anaesthesia. The anal pinch and pedal reflexes were fully abolished along with complete muscle relaxation of jaw, tail, anus sphincter and limbs which was good but for short duration. The mean duration of anaesthesia was 31.33±1.20 min. and lasted by raising of head. The mean returned to sternal recumbency was 43.45± 2.50 minutes. All the animals tried to stand with ataxia at 51.20 ±1.50 and complete recovery i.e. animals stand without ataxia took 60.00 ± 2.58 minutes after propofol administration. Rectal temperature did not show any significant variation whereas heart and respiration rate showed significant ($P<0.05$) decrease after buprenorphine-propofol administration and returned to near base value by 180 min. It can be concluded that buprenorphine-propofol combination may be safely used for short duration anaesthesia in atropinized goats.

Introduction

Goat is one of the earliest domesticated animals in livestock farming due to its higher prolificacy and short generation interval. Its rearing is a great economic support to landless, small and marginal farmers in our country as it is a multifunctional animal, providing meat, milk and wool. Goats undergo many surgical procedures, such as hernia, dystocia, traumatic injuries and they need a safe anaesthesia. General anaesthesia can be induced in goats using the same drugs commonly used in other species. The choice of anaesthetic drugs and timing of surgical interventions is important in normal and diseased animals. Goats are not amongst the commonly anaesthetized animals, which is partly why information on goat anaesthesia is scarce. There is no available anaesthetic drug

which can provide proper anaesthesia alone now a days. The anaesthetic combination should congregate different characteristics, having adequate sedation and a deep unconsciousness state, enough muscle relaxation without greatly changing the patient's physiologic parameters (Alma *et al.*, 2002). Therefore, combinations of sedatives and other anaesthetics have been widely used in animal practice. Propofol is an ultrashort acting, non-barbiturate, non-dissociative intravenous anaesthetic agent which the reduced post anaesthetic complications, because of its rapid metabolism. It is popular both in animals and human beings as an intravenous anaesthetic agent primarily because of quality of induction and quick recovery from anaesthesia (Hall and Peshin, 1996). Propofol should be combined with analgesic drugs in anaesthesia protocols for noxious procedures because it is devoid of any substantial analgesic effect (Beths, 2008). It is recommended to premedicate goats so that they are calm before administering these induction agents (Galatos, 2011). Atropine sulphate is used as preanaesthetic to prevent salivary, bronchial, tracheal and gastric secretions and to inhibit the bradycardiac effects of vagal stimulation.

Buprenorphine is a partial μ opioid agonist and a strong kappa antagonist. It has high affinity for μ receptors, slow dissociation resulting in a long duration of action. At higher doses, it begins to behave more like an antagonist, limiting the maximal analgesic effect. Thus, it has very wide margin of safety (Sporer, 2004). It is about 25 times more potent than morphine and has a low level of physical dependence. Most opioids require redosing at every 4-6 hours. Buprenorphine provides analgesia for upto 6 to 8 hours after administration. Therefore, it has an advantage over other short acting opioids. Because of its longer analgesic action compared to other opiates, buprenorphine can be used before and during surgery to provide post analgesia. This drug is metabolized in the liver and excretes through bile. Reports regarding use of buprenorphine with propofol combination in goats are limited; therefore, the present study was undertaken to assess the effects on clinico-physiological parameters after administration of buprenorphine-propofol anaesthesia in atropinized goats

Materials and Methods

The present study was conducted in six healthy non-descript goats of either sex weighing between 20-25 kg using atropine sulphate @ 0.04 mg/kg I/M followed by buprenorphine @ 10 mg/kg I/M and 10 min. later followed by induction of anaesthesia with propofol (5mg/kg I/V). The following clinical parameters were studied viz., onset of sedation / anaesthesia, spontaneous activity, lowering of head, salivation, onset of sternal or lateral recumbency and duration of anaesthesia. Depth of anaesthesia was judged by monitoring the loss of swallowing reflex, corneal, conjunctival, palpebral reflexes, relaxation of anal sphincter anal pinch, pedal reflexes and extent of muscle relaxation. Recovery from anaesthesia was monitored as raising of head, trying to stand with ataxia and complete recovery i.e. standing without ataxia. The physiological parameters includes rectal temperature, heart rate and respiratory rate which were recorded before and 10 minutes after premedication and 10, 20, 40, 60, 90, 120 and 180 minutes after propofol anaesthesia. The data obtained were statistically analyzed by analysis of variance (ANOVA) as per the standard procedure outlined by Snedecor and Cochran (1994).

Results and Discussion

The effects on clinical parameters after administration of buprenorphine-propofol anaesthesia in atropinized goats are shown in

Table 1. There was little decrease in the activity in all the animals after buprenorphine administration. The onset of analgesia of sedation i.e. lowering of head was observed within 8.20±0.58 min after buprenorphine administration. The onset of sedation is affected by lipid solubility of the drug. A slow onset of opioids was also reported by Hansraj *et al.,* (2000). These findings are supported by Pathak *et al.,* (2012) for spinal analgesia in buffalo calves. The eye and pedal reflexes were present and maintained actively. The anal pinch reflexes were present. There was mild cutaneous analgesia of abdomen, tail and base of horn. This might be due to buprenorphine is not considered appropriate for relief of severe pain because it is a partial agonist and does not have the full analgesic or intrinsic effects of a pure agonist (Pathak *et al.,* 2012). After administration of propofol, the animals showed marked sedation, drooping of head and latereal recumbency. There was rapid and smooth onset of anaesthesia (0.50±0.55 min.). In the present study, propofol provides smooth induction of general anaesthesia. Similar results were also achieved in sedated goats by Dzikiti *et al.,* (2009). Rapid onset of action is caused by rapid uptake of propofol into the CNS (Zoran *et al.,* 1993) and induction of depression occurs by enhancing the effect of the inhibitory neurotransmitter GABA and decreasing the metabolic activity of the brains (Concas *et al.,* 1991). Administration of premedicants had no any effect on quality of induction and almost all the animals recorded smooth, quick, and excitement free induction of anesthesia. These features of induction might be due to propofol's inherent quality of induction (Robinson and Borer-Weir, 2013). Swallowing reflex, corneal and palpebral reflexes abolished within 3 min after onset of anaesthesia which remained througout the period of duration of anaesthesia. The anal pinch and pedal reflexes were fully abolished but for short duration. There was complete muscle relaxation of jaw, tail, anus sphincter and limbs and was good. Complete analgesia at fetlock, base of tail, abdomen, ribs, periosteum and base of horn was observed after propofol administration. The mean (± S. E.) duration of anaesthesia was 31.33±1.20 min. Various pre-anaesthetic combination have been used with propofol to prolong the duration of anaesthesia along with shorter recovery time, thereby improving quality of anaesthesia (Potliya *et al.,* 2015). Increase in the duration of anaesthesia was correlated with the additive effect of preanaesthetics with propofol in depressing the activity of the cerebral cortex. In the present study, buprenorphine was combined with propofol to prolong the duration of anaesthesia and produce profound analgesia with good muscle relaxant. The mean recovery was manifested by raising of head at 31.33 ±1.20 minutes. The mean returned to sternal recumbency was 43.45± 2.50 minutes. All the animals tried to stand with ataxia at 51.20 ±1.50 and complete recovery i.e. animals stand without ataxia took 60.00 ± 2.58 minutes after propofol administration. The recovery was smooth, free from excitement, without any struggling and uncomplicated in all the animals. Singh *et al.,* (2014) reported complete recovery time of 182.33 min. in buffalo calves after propofol administration using tripflupromazine as preanaesthetic agent. Propofol is extensively redistributed from blood to tissue. Therefore, the propofol has no cumulative effect and it leads to smooth recovery (Carroll *et al.,* 1998).

Physiological parameters

The effects on physiological parameters after administration of buprenorphine-propofol anaesthesia in atropinized goats at various time intervals are shown in Table 2. Rectal temperature showed a non significant decrease upto 60 min (from 103.1±0.13 to 102.5±0.15°F) after administration of

propofol which progressively increased and returned to base value by 180 min. in all the animals. Reduced rectal temperature had been reported during propofol anaesthesia in goats (Carroll *et al.*, 1998; Amarpal *et al.*, 2002). The fall in rectal temperature may be a sequel to thermoregulatory depression and decreased metabolic rate due to propofol which was further potentiated by buprenorphine. As it also exerts depressive effect on thermoregulatory centres in hypothalamus, the reduction in rectal temperature is considered secondary to CNS depression and reduction in the muscular activity (Kammar *et al.*, 2014).

Table.1 Mean ± SE values of clinical parameters after administration of buprenorphine-propofol in atropinized goats

Clinical Parameters	Mean±S.E. (Time in minutes)
Onset of sedation after buprenorphine (Lowering of head)	8.20±0.58 min.
Onset of anaesthesia	0.50±0.55 min.
Duration of anaesthesia	31.33±1.20 min.
Time for raising of head up	31.33 ±1.20 min.
Time for sternal recumbency	43.45± 2.50 min.
Time for stand with ataxia	51.20 ±1.50 min.
Time for complete recovery (Standing without ataxia)	60.00 ± 2.58 min.

Table.2 Mean ± S E values of physiological parameters after administration of Buprenorphine-Popofol in goats at various time intervals

Parameters	Period of observation (min)								
	0	10 min after premedication	10 min after G.A.	20	30	60	90	120	180
Rectal Temperature (°F)	**103.1 ±0.13**	**103.0±0.13**	**103.0 ±0.13**	**102.9 ±0.14**	**102.8± 0.14**	**102.5 ± 0.15**	**102.65 ±0.14**	**102.8 ±0.09**	**103.1 ±0.13**
Heart Rate (Beats/ Minute)	**78.83 ±0.65**	**75.66±0.88**	**73.15* ±1.40**	**71.33** ±0.95**	**70.00** ±0.93**	**70.40** ±0.93**	**72.50* ±1.72**	**73.33* ±0.76**	**77.00 ±1.18**
Respiration Rate (breaths/ minute)	**22.16 ±0.61**	**20.33±0.95**	**16.00* ±0.91**	**14.33** ±0.61**	**14.16** ±0.70**	**15.50* ±0.34**	**18.16 ±0.74**	**21.33 ±0.55**	**22.06 ±0.54**

* P < 0.05 = Significant at 5% level when compared to base value

** P < 0.01 = Significant at 1% level when compared to base value

A significant (P<0.05) decrease in heart rate was observed with a peak decrease at 30 minutes (70.00±0.93 beats/min.) after administration of buprenorphine-propofol and thereafter a progressive increase in heart rate tending to returned near the base value by 180

min. In contrast to other intravenous anaesthetic agents, propofol does not depress barroreflex sensitivity directly but may produce an increased vagal tone and decreased in sympathetic tone by central mechanism (Cullen *et al.,* 1987). The cardiac depressant effect of buprenorphine is due to withdrawal of sympathetic activity, resulting in depressed ventricular function and heart rate. The decrease in heart rate might also be due to propofol induced vasodilatation leading to a fall in systemic vascular resistance as well as dose related depression of myocardial contractibility (Duke, 1995). Non-significant change in heart rate has been reported in dog treated with buprenorphine and acepromazine (Stepien *et al.,* 1995). Similarly, Anandmay *et al.,* (2012) reported significant decrease in heart rate after propofol and buprenorphine anaesthesia in atropinized dog.

Respiration rate decreased significantly ($P<0.05$) at 30 min (14.16±0.70 breath/min.) as compared to base value (22.16±0.61 breaths/min.) later on, the values increased and returned to near base value by 180 min. Opioids in combination with propofol increase the probability of respiratory depression during anesthesia (Short and Bufalari, 1999). Dahan *et al.,* (2006) reported that buprenorphine has also the property of respiratory depression and is due to its partial µ agonistic activity in human. Buprenorphine being a partial µ opioid agonist may have a wider safety profile compared to full µ agonist, especially with regards to respiratory depression. In the present study, transient apnoea was observed immediately after propofol administration which lasted for 30 to 40 seconds in all the animals. Propofol is a potent respiratory depressant and apnoea is common upon induction (Benson, 1997). Bufalari *et al.,* (1998) also opined that respiratory depression might cause transient apnoea. Transient apnoea has been reported in goats (Prassinos *et al.,* 2005) after propofol administration. Propofol can induce significant depression of respiratory function characterized by a reduction in the rate of respiration. Anandmay *et al.,* (2012) reported significant decrease in respiration rate after propofol and buprenorphine anaesthesia in atropinized dog.

In the present study, after buprenorphine-propofol administration there was significant ($p<0.05$) decrease in the respiration rate and heart rate, thereafter it returned to near normalcy level by 120 min of the study period. Whereas, rectal temperature did not show any significant variation. However, the transient changes in physiological parameters were compensated within 180 min. and remained within physiological limits. Therefore, on the basis of above study, it was concluded that buprenorphine-propofol combination may be safely used for short duration anaesthesia in atropinized goats as it produced good analgesia alongwith quick onset of anaesthesia showed good muscle relaxation with smooth and uneventful recovery.

References

Alma, A.G., Hector, S., and Enrique, N. (2002). Bases Farmacologicas de la Anestesia General Endovenosa De Corta Duracion Ee El Equino Veterinaria Mexico, *Universidad Nacional Autónoma de México Distrito Federal, México*, 33 (3):309-333.

Amarpal, Kinjavdekar, P., Aithal, H. P., Pathak, R., Pratap, K. and Singh, V. 2002. Effect of xylazine and medetomidine premedication of propofol anaesthesia in goats. *Indian J. Anim. Sci.* 72: 565-566.

Anandmay, A.K., Dass, L.L. and Sharma, A.K. 2012. Clinico-anaesthetic

changes following administration of propofol alone and in combination of buprenorphine in dogs. *Indian Vet. J.*, 89(10): 77-79.

Benson, G.J., 1997. Anaesthesia and analgesia: physiologic effects o pharmacologic agent. In Anaesthesia and analgesia in laboratory animals (Kohn, D. F., S. K. Wixson, W. JJ. White, G. J. Benson, Eds) American College of Laboratory Animal Medicine Series, Academic Press, New York. P.10

Beths, T., 2008. Total intravenous anaesthesia in dogs: Development o a target-controlled infusion (TCI) scheme for propofol. Ph.D. thesis, Department of Companion Animal Sciences, University of Glasgow.

Bufalari, A., S.M., Millar, C., Giannoni and Short, C. E. 1998. The use of propofol as an induction agent for halothane and isoflurane anaesthesia in dogs. *J.Am.Anim.Hosp.Assoc.*, 34:84-91.

Carroll, G.L., Hooper, R. N., Slater, M. R., Hartsfield, S. M. and Matthews, N. S. 1998. Detomidine-Butorphanol-Propofol for carotid artery translocation and castration or ovariectomy in goats. *Veterinary Surgery*, 27: 75–82.

Concas, A., Santoro, G. and Serra, M. 1991. Neurochemical action of the general anaesthetic propofol on the chloride ion channel coupled with GABA receptors. *Brain Research*, 542: 225-232

Cullen, P.M., Turtie, M., Pry, S., Roberts, C., Way, W. L. and Dye. 1987. Effect of propofol anaesthesia on baroreflex activity in humans. *Anaesthesia & Anal*., 66: 1115.

Dahan, A., Yassen A, Romberg A, Sarton E, Teppema L, Olofsen E, Danhof M. Buprenorphine induces ceiling in respiratory depression but not in analgesia. *Br. J. Anaesth*. 96(5):627–663.

Duke, T., 1995. A new intravenous anaesthetic agent propofol. *Can. Vet. J*., 36 (1):181-182.

Dzikiti, T.B., Stegmann, G. F., Hellebrekers, L. J., Auer. R. E. J. and Dzikiti, L. N. 2009. Sedative and cardiopulmonary eects of acepromazine, midazolam, acepromazine-butorphanol and midazolam-butorphanol on propofol anesthesia in goats. *J S Afr Vet Assoc* 80:10-16

Galatos, A., D. 2011. Anaesthesia and analgesia in sheep and goats. *Veterinary Clinics of North America: Food Animals*, 27:47-59

Hall, L.W., and Peshin, P.K. (1996). Propofol-halothane-nitrous oxide/oxygen anaesthesia for mega-voltage radiotherapy in dogs. *Vet. Anaesth. Analg*. 23: 20-22.

Hansraj, Amarpal, Singh G. R. and Aithal, H. P.2000. Postoperative pain management in dogs. Efficacy of premptive analgesia with lignocaine and ketamine. *Indian J. Anim.Sci*.70:362- 365.

Kammar, M.H., EL., Gad, S. B. and Korittum, A. S. 2014. Evaluation of the sedative, analgesic, physiological and haematological effects of intravenous detomidine, detomidine-butorphanol, romifidine and romifidine-butorphanol in Baladi goats. *Global Veterinaria* 12(1): 36-44.

Pathak, R., Pratap, K., Kinjavdekar, P. and Aithal, H. P. 2012. Comparison of bupivacaine, xylazine and buprenorphine with ketamine combination for spinal analgesia in buffalo calves. *Vet World*. 5 (12):754-761.

Potliya, S., Kumar, S. Singh, S. and Kumar, S. 2015. Evaluation of efficacy and safety of glycopyrrolate - xylazine –

propofol anesthesia in buffalo calves. *Vet World*, 8(3):251-256.

Prassions, N.P., Galatos, A.D., and Raptopoulos, D. 2005. A comparison of propophol, thiopental or ketamine as induction agents in goat. *Veterinary Anaesthesia and Analgesia* 32: 289-296.

Robinson, R,. and Borer-Weir, K. 2013. A dose titration study into the effects of diazepam or midazoalm on the propofol dose requirements for induction of general anaesthesia in client owned dogs, premedicated with methadone and acepromazine. *Vet. Anaesth. Analg.* 40 (5):455–463.

Short C. E and Bufalari A. 1999. Propofol anaesthesia. *Vet. Clin. North Am. Small Anim. Pract*. 29 (3): 747–778.

Singh, K., Kumar, A., Kumar, S., Potliya, S. and Singh, S. 2014. Evaluation of triflupromazine-propofol as a anaesthetic combination in buffalo calves. *Haryana Vet*. 53 (2):78-83.

Snedecor, G.W., and Cochran, W. G. 1994. *Statistical Methods*, 8th Edition, Oxfords and IBH Publishing Co., New York, p.59.

Sporer, K.A., 2004. Buprenorphine: a primer for emergency physicians. *Ann. Emerg. Med.*, 435: 580-584.

Stepien, R.C., Bonagura, J. D. and Bednarbki, R. M. 1995. Cardiorespiratory effects of acepromazine maliate and buprenorphine hydrochloride in clinically normal dogs. *Am. J. Vet. Res*., 56: 78-84.

Zoran, D.L., Reidesel, D. H. and Dyer. D. C. 1993. Pharmacokinetics of propofol in mixed breed dogs and grey hounds. *Am. J. Vet. Res*., 54: 755-760.

How to cite this article:

Rukmani Dewangan, M.S. Maravi, S.K. Tiwari, M.O. Kalim and Sharda, R. 2019. Effect on Clinico-Physiological Parameters after Administration of Buprenorphine-Propofol Anaesthesia in Atropinized Goats. *Int.J.Curr.Microbiol.App.Sci.* 8(04): 2382-2388.
doi: https://doi.org/10.20546/ijcmas.2019.804.277