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Clinico-Physiological Response to Detomidine-Propofol Anaesthesia in Atropinized Goats

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A B S T R A C T

The study was conducted on six healthy non-descript goats of either sex weighing 20-25 kg by giving atropine sulphate (0.04 mg/kg I/M) followed by detomidine (15 µg/kg I/M) and 10 min. later followed by induction of anaesthesia with propofol (5mg/kg I/V). There was decrease in spontaneous activity with marked sedation as well as lowering of head after detomidine administration in all the animals. After administration of propofol, there was rapid and smooth induction of anaesthesia (0.46±0.51 min). Transient apnoea was observed immediately after induction with propofol which lasted for 40-50 seconds. This was followed by loss of swallowing reflex, corneal, palpebral and conjunctival reflexes were abolished within 3 min and remained so throughout the anaesthetic period. The anal pinch and pedal reflexes were also fully abolished. Muscle relaxation was excellent. Complete analgesia at fetlock, base of tail, abdomen, ribs and base of horn was observed. The duration of anaesthesia was 52.50 ± 8.44 min and complete recovery which was smooth with no excitement occured within 91.66±14.24min. There was non-significant decrease in the rectal temperature. Heart and respiration rate significantly decreased upto 60 min. after detomidine-propofol administration. It can be concluded that detomidine-propofol anaesthesia may be safely used for inducing longer duration of anaesthesia in goats.

Introduction

General anaesthesia produces unconsciousness, analgesia and muscle relaxation but might also suppress autonomic reflex activity and consequently leads to inadequate function of vital physiological systems such as the cardiovascular and respiratory system (Antognini and Carstens, 2002). The choice of anaesthetic drugs and timing of surgical intervention is important in both normal and diseased animal. However, rapid and smooth induction of anaesthesia and lesser duration of recumbency are desirable. No single anaesthetic is known to posses all the components of the ideal general anaesthetic agent without depressing some vital function. Therefore, multiple drug approach is exploited to diminish sensory, motor, sympathetic and parasympathetics reflex activities and to attenuate individual components of the anaesthetic state. Propofol is a non-barbiturate, intravenous anaesthetic agent commonly used in veterinary and human

anaesthetic practice (Bodh *et al.*, 2013), which rapidly induces smooth anaesthesia, and permit fast and uneventful recovery. The rapid induction and short duration of action, with rapid recoveries make propofol potentially useful in calves (Branson and Gross, 1994). However, when used alone, propofol is unsatisfactory; consequently, it has been combined with various sedatives and analgesic drugs to produce adequate surgical conditions (Cullen and Reynoldson, 1993). 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. Detomidine HCl is a new synthetic alpha-2 adrenorecptor agonist and is imidazole derivative with sedative and analgesic properties (Anonymous, 1996) which is primarily used as sedative in horses. Reports regarding use of propofol anaesthesia in combination with detomidine as premedicant in goats are limited; therefore, the present paper deals with the clinico-physiological response to detomidine-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 detomidine (15 µg/kg I/M) and 10 min. later followed by induction of anaesthesia with propofol (5mg/kg I/V). The following clinical parameters were studied are 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 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 mean and standard error of recorded values were calculated. One way analysis of variance (ANONA) and Duncan's multiple range test were used to compare the means at different intervals with base values as per the standard procedure outlined by Snedecor and Cochran (1994).

Results and Discussion

There was decrease in spontaneous activity in all the animals after detomidine administration with marked sedation as well as lowering of head. The sedative action of α_2-adrenergic agonists is due to inhibition of firing of locus coeruleus (LC) neurons in the pons of the lower brain stem and its ascending activating projection to the forebrain after systemic injection. Urination and defaecation was observed in two animals after detomidine administration. Moderate cutaneous analgesia was observed in animals on pin-prick at abdomen, tail and base of horn. Pedal and palpebral reflexes were present in all animals. All the animals fell down to lateral recumbency after administration of detomidine. Singh *et al.*, (1991) reported salivation after administration of detomidine in goats. Head drooping with detomidine had also been reported in horses (Skarda and Muir., 1996) and in sheep (Malhi *et al.*, 2015). After administration of propofol, there was rapid and smooth induction of anaesthesia. The onset of anaesthesia was 0.46±0.51 minutes. Rapid onset of action is caused by rapid uptake of propofol into the CNS 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). Adetunji *et al.*, (2002) also found that propofol provided rapid induction of anaesthesia after its administration due to its high lipid solubility resulting in rapid blood / brain equilibrium. The duration of anaesthesia was 52.50±8.44 minutes. There was loss of swallowing reflex, corneal, palpebral conjunctival and panniculus reflexes and abolished within 3 min which remained throughout the period of anaesthesia. The anal pinch and pedal reflexes were fully abolished. The muscle relaxation was excellent. Complete analgesia at fetlock, base of tail, abdomen, ribs and base of horn was observed. Sluggish to absent corneal reflex was observed after induction of propofol anaesthesia (Singh *et al.*, 2014 and Potliya *et al.*, 2015 a, b) in buffalo calves. Increase in the duration of anaesthesia was correlated with the additive effect of preanaesthetics with propofol in depressing the activity of the cerebral cortex.

Kilic (2008) concluded that the combination of detomidine, midazolam and ketamine resulted in anaesthesia lasting about 45 min in calves. Various pre-anaesthetic combination have been used with propofol to prolong the duration of anaesthesia alongwith shorter recovery time, thereby improving quality of anaesthesia (Potliya *et al.*, 2015 b). In the present study, detomidine was combined with propofol to prolong the duration of anaesthesia and produce profound analgesia with excellent muscle relaxation. After detomidine-propofol administration, there was excellent muscle relaxation which might be due to prior administration of detomidine which activates alpha-2adrenoceptors present in the spinal cord (Branson *et al.*, 1993). Recovery was manifested by raising of head at 52.50 ±8.44 minutes and was smooth, free from excitement, without any struggling. All the animals returned to sternal recumbency at 60 ± 4.50 minutes. All the animals tried to stand with ataxia at 75.5 ±3.50 and complete recovery occurred 91.66 ± 14.24 minutes after propofol administration. Potliya *et al.*, (2015 a, b) observed that complete recovery took 198.0 ±9.75 min. and 132.0 ±8.63 min. to occur in buffalo calves after administration of atrophine-xylazine-propofol and glycopyrrolate-xylazine-propofol combinations, respectively. The effects on physiological parameters after detomidine-propofol anaesthesia in atropinized goats at various time intervals are shown in Table 1. A non-significant decrease in the rectal temperature was observed after administration of propofol in combination with detomidine which persisted upto 90 min. (from 103.5±0.20 to 102.±0.17°F). The decrease in rectal temperature was also probably the result of a reduced basal metabolic rate (BMR) and muscle activity, and depression of thermoregulatory centre (Kilic, 2008).

Rectal temperature slightly decreased after administration of detomidine in Pateri goat and this decrease was non-significant as compared to the control values (Tunio *et al.*, 2016). Reduced rectal temperature had been reported during propofol anaesthesia in goats (Carroll *et al.*, 1998; Amarpal *et al.*, 2002). The reduction in rectal temperature is considered secondary to CNS depression and reduction in the muscular activity (Kammar *et al.*, 2014). Highly significant ($p<0.01$) decrease in heart rate was observed from base value upto 120 min (from 77.66 ± 0.76 to 66.16±0.40 beats/min.) after the administration of atropine-detomidine-propofol with a peak decrease at 60 min. (50.16±1.35 beats/min). However, the values increased and returned to preadministration level by 180 min. The decrease in the HR might be attributed to vasoconstriction due to alpha-2 agonist administration leading to reflex bradycardia (Lemke, 2004).

Table.1 Effects on physiological parameters after detomidine-propofol anaesthesia in atropinized goats at various time intervals (Mean±S.E.)

Groups	Time Intervals(min)								
	0	10 min after pre-medication	10 min after G.A.	20	30	60	90	120	180
Rectal Temperature (°F)	103.5 ±0.20	103.0 ±0.20	102.8 ±0.20	102.6 ±0.19	102.4 ±.0.17	102.2 ±0.18	102.00 ±0.17	103.0 ±0.19	103.3 ±0.19
Heart Rate (Beats/ Minute)	77.66 ±0.76	72.00 ±1.26	61.66** ±1.66	58.83** ±2.30	53.50** ±2.12	50.16** ±1.35	56.16** ±0.40	66.33* ±1.89	74.83 ±0.90
Respiration Rate (per minute)	22.00 ±0.51	18.00 ±0.25	15.00* ±0.81	12.16** ±0.30	10.50** ±0.34	9.66** ±0.49	16.00* ±1.39	17.05* ±1.28	21.36 ±0.60

* P < 0.05 = Significant at 5% level when compared to base value
** P < 0.01 = Significant at 1% level when compared to base value

Inhibition of sympathetic tone from the central nervous system, inhibition of norepinephrine release from sympathetic nerve terminals, vagal activity in response to detomidine induced vasoconstriction and direct increase in the release of acetylcholine from the parasympathetic nerves in the heart have been described as possible mechanisms by which detomidine induces bradycardia (MacDonald and Virtanen, 1992). Tunio *et al.,* (2016) in goats reported decreased HR after detomidine administration. 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).This might be due to direct action of α_2-agonist on the post synaptic receptors of the vascular smooth muscles leading to vasoconstriction and an initial transient hypertension followed by pronounced hypotension. Xylazine-propofol and medetomidine-propofol combinations produce bradycardia in goats which was pronounced after premedication with medetomidine (Amarpal *et al.,* 2002). The animals showed a significant ($p< 0.05$) decrease in the respiration rate up to 120 min. (from 22.00±0.51 to 17.05±1.28 per min.) which became highly significant ($p<0.01$) at 60 min. (from 22.00±0.51 to 9.66±0.49 per min.) with peak decrease after anaesthesia. Apnoea is common following bolus administration of propofol or thiopentone and increasing the dose or speed of injection will increase the incidence and duration of apnoea (Bodh *et al.,* 2013). In the present study, transient apnoea was observed immediately after propofol administration which lasted for 40 to 50 seconds in all the animals. Bufalari *et al.,* (1998) also opined that respiratory depression might cause transient apnoea. A transient apnoea of about 2 minutes has been reported immediately after administration of thiopentone in buffalo calves premedicated with atropine and diazepam (Singh *et al.,* 2006). Similar findings were reported with propofol by Carroll *et al.,* (1998) in goat. Decrease in respiration rate, might be due direct depressant action of α_2-agonist (detomidine) on central nervous system in general and respiratory centre in particular (Kim *et al.,* 1999). In the present study, after propofol injection there was significant ($p<0.05$) decrease in the respiration rate, thereafter it returned to near normal values by 120 min of the study period. However, in the present study, the transient changes in

physiological parameters were compensated within 180 min. and remained within physiological limits. Therefore, it can be concluded that the detomidine-propofol anaesthesia may be safely used for inducing longer duration of anaesthesia in atropinized goats as it produced profound analgesia with quicker onset of induction of anaesthesia with excellent muscle relaxation along with smooth and uneventful recovery.

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