



Physiological properties of different doses of Detomidine in cattle

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Abstract

The present study was undertaken to find out an ideal sedative dose of detomidine for minor surgical and diagnostic procedures in cattle. The study was conducted on a total of 18 cattle, randomly divided into 3 groups, each group consisting of 6 cattle. The cattle were administered with detomidine at the dose rates of 10, 20 and 30 $\mu\text{g}/\text{kg}$ body weight I.V. in group I, II and III respectively.

The parameters like time for ataxia, time to attain peak sedation, duration and quality of sedation were studied. In addition, physiological parameters like body temperature, heart rate and respiratory rate were recorded before administration of detomidine, during sedation, after recovery and after taking feed and water. The study revealed quicker onset of ataxia and peak sedation when detomidine was administered at the rate of 30 $\mu\text{g}/\text{kg}$ body weight compared to 20 and 10 $\mu\text{g}/\text{kg}$ body weight. It showed that ataxia and onset of sedation were directly proportional to the dose of detomidine. The quality and duration of sedation also significantly increased in 30 $\mu\text{g}/\text{kg}$ body weight followed by 20 and 10 $\mu\text{g}/\text{kg}$ body weight revealing dose dependent depth and duration of sedation. In all cattle, dose dependent decrease in heart rate, dose dependent increase in respiratory rate and a non significant decrease in rectal temperature noticed. The values stayed below normal after recovery and comparable with normal values only after taking feed and water.

Key words: Ataxia, Cattle, Detomidine, Sedation.

Introduction

Although a lot of minor surgical procedures in cattle can be done under local or regional analgesia, sedation in cattle is often required for certain diagnostic procedures like endoscopy, laparoscopy, radiography etc., minor surgical problems like opening an abscess in a vicious bullock and even to the extent of performing a major surgical procedure like caesarean section under xylazine sedation due to its oxytocic effect.

Among the a_2 - adrenergic agonists, xylazine is widely used in cattle as a sedative and a premedicant to general anaesthesia (Clarke and Hall, 1969). Xylazine as a sedative and a premedicant at the dose rate of 0.1 mg - 0.5 mg/kg body weight was reported by several authors to induce AV block (transient hypertension) and regurgitation due to the relaxation of gastro-oesophageal sphincter, ruminal tympany (Ruckebusch and Allal, 1987) due to its a_2 - adrenergic agonistic action and abortion due to the oxytocic effect (LeBlanc *et al.*, 1984).

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Detomidine, an α_2 - adrenergic agonist is directly proportional to dose related sedation and has a wide margin of safety (5 to 300 $\mu\text{g}/\text{kg}$ body weight) as reported by Vainio (1985). It does not cause abortion and regurgitation even in unfasted animals (Alitalo, 1986; Jedruch and Gajewski, 1986 and Schatzmann *et al.*, 1994).

Since the study was undertaken to find out an ideal sedative dose rate of detomidine in cattle.

Materials and methods:

The study was conducted on a total of 18 cattle, randomly divided into 3 groups, each group consisting of 6 cattle. The cattle were administered with detomidine at the dose rates of 10, 20 and 30 $\mu\text{g}/\text{kg}$ body weight I.V. in group I, II and III respectively.

The parameters studied were time for ataxia (in min) was assessed by the degree of incoordination, time for peak sedation (in min) was assessed by the attainment of ataxia, dropping of upper eyelid and lowering of head, duration of sedation (in min) recorded as the time interval between peak sedation and recovery in terms of unassisted standing and quality of sedation was assessed by the score card system proposed by Hamm *et al.* (1995) (Table 1).

Table 1: Score card to assess the quality of sedation

Details	Grading	
	Present	Absent
Absence of noise response	2	0
Absence of visual arousal	2	0
Ataxia	2	0
Instability and/or recumbency	2	0
Analgesia (clinical evaluation)	2	0
Total	10	0

In addition, heart rate per min was recorded by direct auscultation, respiratory rate per min was recorded by observing abdominal movements and the body temperature ($^{\circ}\text{C}$) was recorded using digital thermometer before detomidine administration, at peak sedation, after recovery and after taking feed and water.



Results and Discussion:

Time for ataxia, time for peak sedation, duration and quality of sedation are shown in Table 2.

Table 2: Mean \pm SE values of Time for ataxia, Time for peak sedation, Duration and Quality of sedation

Parameters	Group I (10 $\mu\text{g}/\text{kg}$ b. wt)	Group II (20 $\mu\text{g}/\text{kg}$ b. wt)	Group III (30 $\mu\text{g}/\text{kg}$ b. wt)
Time for ataxia	1.85 ^c \pm 0.01	1.60 ^b \pm 0.02	1.37 ^a \pm 0.04
Time for peak sedation	5.27 ^c \pm 0.02	5.05 ^b \pm 0.02	4.59 ^a \pm 0.04
Duration of sedation	14.80 ^a \pm 0.73	24.26 ^b \pm 0.40	38.17 ^c \pm 0.41
Quality of sedation	2.59 ^a \pm 0.05	8.53 ^b \pm 0.04	9.46 ^c \pm 0.93

Means bearing different superscripts in a row differ significantly ($P < 0.01$)

The present study showed that quicker onset of ataxia and peak sedation at a shorter duration when detomidine was administered at the rate of 30 $\mu\text{g}/\text{kg}$ body weight, when compared with 20 and 10 $\mu\text{g}/\text{kg}$ body weight revealing that ataxia and onset of sedation were directly proportional to the dose of detomidine. The findings concurred with Gnanasekar *et al.* (2013), Yamashita *et al.* (1999), England *et al.* (1992), Peshin *et al.* (1993), Singh *et al.* (1994) and Hamm *et al.* (1995).

In the central nervous system α_2 -adrenoceptors regulate the neuronal release of noradrenaline and several other transmitter substances which are involved in the modulation of sympathetic outflow, cardiovascular and endocrine functions, vigilance and nociception. The α_2 -adrenoceptor located in the presynapsis regulate the release of sympathetic transmitter substance norepinephrine from the nerve endings. Hence detomidine, the α_2 -adrenergic agonists produced major sedation with associated analgesia (Alitalo, 1986 and Jochle and Hamm, 1986).

Detomidine induced sedation in a dose dependent manner (Virtanen, 1986). McCleary and Leander (1981) and Paalzow and Paalzow (1982) attributed the analgesic effect of detomidine to the inhibition of pain modulating noradrenergic neurons in the central nervous system.

The quality and duration of sedation also significantly increased in 30 $\mu\text{g}/\text{kg}$ body weight followed by 20 and 10 $\mu\text{g}/\text{kg}$ body weight revealing dose dependent depth and duration of sedation.

During sedation with 10 $\mu\text{g}/\text{kg}$ body weight, detomidine induced calming and the animals were able to stand revealing lesser score. Virtanen and Macdonald (1985) and Hamm *et al.* (1992) also reported that detomidine at lower doses did not affect the locomotor activity. Detomidine at 10, 20 and 30 $\mu\text{g}/\text{kg}$ body weight during sedation produced sternal recumbency in all the animals although drooping of head noticed, which could be attributed to the protection of righting reflex (Virtanen, 1986). The score for the quality of sedation was assessed on the absence of noise response



and visual arousal, presence of ataxia, instability and recumbency and analgesia. This score was maximal at 30 µg/kg body weight revealing that the intensity of sedation increased as the drug dose increased. The findings concurred with the observation of Oijala and Katila (1988) and Singh *et al.* (1994). The dose dependent longer duration of action could be attributed to the lipophilic nature of drug (Salonen, 1986) and slow metabolism and these findings concurred with those of Singh *et al.* (1994) and Gnanasekar *et al.* (2013).

Table 3. Mean ± SE values of Heart rate, Respiratory rate and Body temperature

Parameters	Group	Base value	At peak sedation	After total recovery	After taking feed and water
Heart rate / min	I	64.31 ^e ± 1.14	56.82 ^c ± 1.50	60.52 ^d ± 1.69	62.33 ^{de} ± 1.86
	II	68.91 ^f ± 1.99	56.23 ^c ± 1.43	66.73 ^{ef} ± 1.92	68.42 ^f ± 1.91
	III	56.19 ^c ± 2.04	42.35 ^a ± 2.23	52.76 ^b ± 1.75	54.96 ^{bc} ± 1.84
Respiratory rate / min	I	18.24 ^{ab} ± 0.37	20.69 ^c ± 0.49	18.72 ^b ± 0.49	17.94 ^{ab} ± 0.36
	II	21.43 ^{cd} ± 0.80	24.09 ^e ± 0.46	22.11 ^d ± 0.52	21.37 ^{cd} ± 0.54
	III	16.98 ^a ± 1.01	19.87 ^c ± 0.69	18.23 ^{ab} ± 0.89	17.02 ^a ± 0.68
Body temperature (°C)	I	38.8 ± 0.13	38.7 ± 0.16	38.8 ± 0.12	38.8 ± 0.14
	II	39.0 ± 0.16	38.8 ± 0.10	38.9 ± 0.16	39.0 ± 0.13
	III	38.5 ± 0.12	38.2 ± 0.18	38.4 ± 0.13	38.5 ± 0.12

Means bearing different superscripts in a parameter differ significantly (P<0.01)

The mean heart rate, respiratory rate and body temperature of the three groups were shown in table 3. At peak sedation, dose dependent decrease in heart rate could be noticed. The decrease in heart rate due to the administration of α_2 - adrenergic agonists was attributed to the effect of drug on carotid sinus baroreceptor reflex (Garner *et al.*, 1971), withdrawal of sympathetic tone, increase in



parasympathetic tone, direct depressive action on cardiac pace maker and conduction tissue and reduction in myocardial inotropic effect (Klide *et al.*, 1975 and Campbell *et al.*, 1979). Wagner *et al* (1991) reported more persistent duration of bradycardia with detomidine particularly at high doses. Sarazan *et al.* (1989) and Flavahan and McGrath (1982) attributed the dose dependent bradycardia of detomidine to the direct negative chronotropic effect on the heart. After taking feed and water, the heart rate still remained less than the base value and the mean values after taking feed and water were comparable with base values. The findings concurred with Clarke and Taylor (1986), Jones (1993) and Oijala and Katila (1998).

Following sedation with detomidine the mean respiratory rate increased in all the groups. These findings concurred with Vainio (1985) and Singh *et al.* (1994). Short (1992) reported elevated respiratory rate during high doses of detomidine which could be attributed to the stimulation of chemoreceptors due to elevated carbondioxide tension.

The mean rectal temperature revealed non significant decrease following sedation in all the groups when detomidine was administered as sedative. This concurred with Virtanen and macdonald (1985). Xylazine induced interference in the thermoregulatory system (Ponder and Clarke, 1980) leading to hypothermia or hyperthermia in animals. Detomidine maintained the mean rectal temperature due to the maintenance of cardiac output (Wagner *et al.*, 1991 and Short, 1992) and better tissue perfusion (Gasthuys *et al*, 1990).

The present study revealed detomidine is a very useful sedative in cattle. Both 20 µg/kg and 30 µg/kg body weight detomidine provided adequate sedation, analgesia and muscle relaxation. 10 µg/kg body weight detomidine can be used very well for standing restraint.



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