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SEDATIVE AND ANALGESIC EFFECTS OF DETOMIDINE IN CAMELS (*Camelus dromedarius*)

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ABSTRACT

Detomidine hydrochloride was administered intravenously to three groups of camels, using three different doses (25, 50 or 75 µg/kg). The levels of sedation and analgesia were graded and recorded. Sedation and analgesia were dose dependent. Detomidine at a dose rate of 75 µg/kg produced profound sedation and analgesia. Significant hyperglycemia and bradycardia were recorded after administration of detomidine and till recovery. No significant changes in hemoglobin concentration (Hb%), PCV, WBCs and RBCs counts and serum concentration of creatinine and blood urea nitrogen levels were recorded at any dose level.

Key words : Analgesia, camel, detomidine, sedation

Drugs for sedation and tranquilisation are very useful in camel husbandry, medicine and surgery. Deep sedation as well as analgesia is a mandatory for dealing with camels either for some routine examinations or many surgical interventions. Several anaesthetics, tranquilisers and analgesics have been used in camels (Fouad and Morcos, 1965; Khamis *et al*, 1973; Peshin *et al*, 1980; Sharma *et al*, 1983; El-Amrousi *et al*, 1986; White *et al*, 1986 and Fahmy *et al*, 1995). Chlorpromazine hydrochloride, propyonil promazine and acepromazine have also been evaluated as sedatives in camels (Said, 1972; Khamis *et al*, 1973; Ali *et al*, 1989). Despite advances in the field of tranquilisers and their uses in domestic animals, experience with their application on the camel have not been exhaustive (Fouad, 2000).

Alpha-2 adrenoceptor agonists (Xylazine, detomidine, medetomidine and romifidine) have been extensively used in the field of veterinary anaesthesiology for their sedative properties (Hall and Clark, 1991). These drugs have been used as sole agents for restraint or calming of camels or to reduce stress (Ali, 1988). If these agents are inadequate to complete involved surgical procedures, supplementation with local analgesics or general anaesthesia has been used.

Xylazine was the initial alpha-2 adrenogenic agent which had been introduced for sedation in camels (Denning, 1972; Sharma *et al*, 1982). Xylazine (0.25mg/kg, i.m.) is adequate for many clinical uses in camels and seems to be superior to chlorpromazine and propionyl promazine (Khamis *et al*, 1973).

Detomidine, a relatively new alpha-2 adrenoceptor agonist, is a sedative, muscle relaxant and analgesic that has been shown to be effective in a wide range of animal species (Hall and Clark, 1991; Raekallio *et al*, 1991 and El-Maghraby and Atta, 1997). Generally, detomidine induces stronger and longer lasting sedation and analgesia in comparison with other members of the same group such as xylazine (Jochle *et al*, 1989). Preliminary trials indicated that intramuscular injection of detomidine (50µg/kg) in camels revealed marked sedation and analgesia (Hall and Clark, 1991). Intravenous administration of detomidine in dromedary camels has not been evaluated in the available literature. The purpose of the controlled study reported here is to evaluate objectively the efficacy of various doses of detomidine in dromedary camels with special reference to its sedative, analgesic, haematological and biochemical effects.

Materials and Methods

Fifteen adults apparently healthy camels (9 males and 6 females), aged 6 to 15 years and weighing 300 to 450 kg were used. Resting rectal temperature, pulse, and respiratory rates were measured and a complete blood count was made

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before each treatment.

Camels were randomly divided into three groups (5 camels in each group). One per cent detomidine hydrochloride (Domosedan: Orion Corporation, Animal Health Division) was injected intravenously at the dose levels of 25, 50 and 75 µg/kg, respectively in the three groups. Dropping of the head, external concheae of the ear, lower lip and/or upper eyelid, prolapse of the penis and frequency of urination were recorded. Sedation was graded as mild, moderate or deep. Analgesia was assessed by recording the response of the animal to needle pricks and electrical stimulation. Needle pricks were applied at the shoulder, flank area and perineum. Electrical stimulation was applied through two electrodes fixed around a closely clipped fetlock joint of both fore limbs and connected to a variable output stimulator (BioScience stimulator, 10550). The amplitude of the electrical current output was increased until the response of the animal by moving or raising one of the examined limbs. The amplitude of the current to which response occurred was recorded and accordingly analgesia was graded from 0 to 3 as described in horses by Jochle and Hamm (1986). The time of onset, degree, duration of sedation and analgesia and the recovery time were recorded and monitoring continued upto 3 hours after drug administration.

Heart and respiratory rates were recorded at 0 (to serve as a control), 15, 30, 45 and 60 minutes and at apparent recovery time. Blood samples were collected from the jugular vein at 0, 15, 30 and 60 minutes and at recovery time for determination of haemoglobin (Hb%), packed cell volume (PCV %) and RBCs and WBCs counts. Blood serum was also analysed for urea nitrogen and creatinine concentrations.

Statistical analysis of the data was done by one-way ANOVA followed by pairwise comparison of probabilities (Bonferroni correction). Values of $p < 0.05$ were considered to be statistically significant.

Results

Intravenous injection of detomidine induced apparent sedative effect within 2-3 minutes. No difference in latency was detected between the three doses of detomidine. All animals remained calm and appeared to be unaware of their surroundings. Drooping of the lower lip, head, upper eyelid and external conchea of the ear were recorded (Figs 1 and 2). Mild salivation and lacrimation were also detected. Ataxia varied from mild to moderate, the

Fig 1. A camel after sedation with intravenous detomidine hydrochloride Notice the drooped head and abduction of the limbs.

Fig 2. The sedative effect of detomidine in a camel. Notice drooping of the lower lip, lower eyelid and external conchea of the ear.

Table 1. The effect of various doses of detomidine on the duration and grade (mean ± SD) of sedation and analgesia.

Dose of Detomidine	Sedation		Analgesia		Recovery (min)
	Duration	Grade	Duration	Grade	
25 µg/kg	26 ± 4.43	Mild	20 ± 6.17	0-1	55 ± 10.2
50 µg/kg	40 ± 2.17	Mild-Moderate	28 ± 4.13	2	90 ± 5.0
75 µg/kg	55 ± 3.11	Deep	37 ± 5.19	3	95 ± 9.78

Table 2. Heart rate, respiratory rates and temperature (means ± SD) of camels injected with different doses of detomidine.

Dose	Time (min)	Respiratory rate	Temperature	Heart Rate
Detomidine 25 µg/kg	t_0	13.66 ± 1.5	37.3 ± 0.64	44.6 ± 2.06
	t_{15}	11.33 ± 1.15	37.7 ±.7	29.66 ± 4.93 *
	t_{30}	12.33 ± 3.21	37.36 ± 0.05	30 ± 3.60 *
	t_{60}	12 ± 1.57	37.53 ± 0.05	31.33 ± 4.16*
	Recovery	16.66 ± 3.05	37.7 ± 0.1	34.66 ± 2.30
Detomidine 50 µg/kg	t_0	14.33 ± 5.0	37.5 ± 0.66	46.33 ± 12.4
	t_{15}	13.33 ± 2.08	37.9 ± 0.65	30.66 ± 5.03 *
	t_{30}	12.66 ± 3.05	38 ± 0.6	31.33 ± 6.02 *
	t_{60}	12.33 ± 0.57	38.06 ± 0.55	36.55 ± 3.46
	Recovery	14.33 ± 0.57	37.8 ± 0.6	33.33 ± 4.16
Detomidine 75 µg/kg	t_0	13.66 ± 1.69	37.8 ± 0.18	37.66 ± 4.18
	t_{15}	13.0 ± 2.64	38.1 ± 0.2	22.33 ± 5.13 *
	t_{30}	12.0 ± 2.0	38.1 ± 0.15	23.66 ± 7.23 *
	t_{60}	11.33± 1.15	38.2 ± 0.1	24.66 ± 6.65 *
	Recovery	13± 1.73	38.1 ± 0.0	31.33 ± 11.37

* Statistically different (P<0.05) by pairwise analysis.

degree of ataxia increased by increasing the dose of detomidine. Although all camels remained in a standing position after administration of detomidine at dose rate of 25 or 50 µg/kg, camels which received 75 µg/kg attained sternal recumbency within 10 minutes. Frequent urination commencing about 40 - 60 minutes after administration of detomidine was observed. Protrusion of the penis was not observed in any animal. The sedative effect persisted for 26 ± 4.43, 40 ± 2.17 and 55 ± 3.11 minutes after intravenous injection of detomidine at 25, 50 and 75 µg/kg, respectively. The degree of sedation was more or less dose dependent and rated from mild to deep. The depth of sedation induced by dose of 75 µg/kg was greater than that induced by either by 25 or 50 µg/kg (Table 1).

The period of analgesia was shorter than the period of sedation (table 1). The analgesic effect persisted for 20 ± 6.17, 28± 4.13 and 37± 5.19 minutes after intravenous administration of detomidine at doses of 25, 50 and 75 µg/kg, respectively. Intravenous administration of detomidine at a dose of 25 µg /kg induced a poor analgesic effect which ranged from 0 (no obvious analgesia) to grade 1 analgesia. The analgesic effect of 75 µg/kg b.wt. was excellent (grade 3) as indicated by lack of response to painful and electrical stimulations.

Significant bradycardia was recorded in all camels after intravenous injection of all three doses (Table 2). Twenty beats/minute was the lowest rate recorded. Auscultation also showed irregular rhythm and dropped beats. Respiratory rate and rectal temperature were not affected. There were no significant changes in the blood biochemical parameters except glucose. A significant ($P< 0.05$) hyperglycemia was observed 15 minutes after detomidine administration which persisted till recovery.

Discussion

Alpha-2 agonists are used to sedate animals for a variety of diagnostic and surgical procedures. These include procedures such as dental working, radiology, endoscopy and minor surgeries with local analgesia (if necessary). While many veterinarians still prefer the intramuscular route of administration, intravenous administration of alpha-2 agonists gives the most reliable sedation and rapid onset of action (Hall and Clark, 1991 and Short, 1992). This might be due to the variability in the response which may be influenced in part by unpredictable drug absorption from the IM administration site.

The onset of sedation started soon (2-3 minutes) after intravenous injection of detomidine. No difference in latency period was detected between the different doses of detomidine. The analgesic effect of detomidine in camels was nearly dose dependent. While the low dose (25 g/kg) showed mild analgesic effect, the higher doses (50 g /kg and 75 g/kg) produced moderate to deep analgesic effect. It should be pointed that the high dose was associated with high degree of ataxia and even recumbency. Usually, increasing the dose of alpha-2 agonist increases ataxia without preventing the response of the animal to painful stimulation (Short, 1992).

Salivation was minimal in all the three tested dosage in this study. Increased salivation after detomidine injection has been reported in cattle (Short, 1992). A significant bradycardia has been observed after intravenous administration of detomidine. Similar findings had been reported in other species after sedation with detomidine (Short

Table 3. Some haematological and biochemical values (means ± SD) after I/V administration of Detomidine 25, 50 and 75 µg/kg body weight.

Dose	Time (minute)	Glucose mmol/L	BUN mmol/L	Creatinine mmol/L	Total protein gm/dl	RBC X 10^6	Hb gm/dl	PCV %	WBC X 10^3
Detomidine 25 µg/kg	t_0	4.9 ± 0.88	8.3 ± 2.1	157 ± 3.07	6.6 ± 0.71	6.9 ± 1.01	10.96 ± 0.77	24 ± 1.5	15 ± 5.8
	t_{15}	5.96 ± 0.35	8.7 ± 1.7	139 ± 33.7	6.14 ± 0.34	7.3 ± 0.34	9.5 ± 1.9	21.7 ± 2.1	11.5 ± 3.6
	t_{30}	5.78 ± 0.87	11.1 ± 4.2	146 ± 12.2	6.17 ± 0.67	7.4 ± 0.8	8.8 ± 1.01	22 ± 2.0	16.3 ± 5.9
	t_{60}	7.76 ± 0.74 *	12.0 ± 6.7	132 ± 29.8	7.04 ± 0.08	7.35 ± 0.35	8.8 ± 0.77	22.5 ± 0.7	15 ± 2.7
	Recovery	6.95 ± 1.94 *	8.55 ± 1.5	144 ± 11.0	7.4 ± 1.6	7.48 ± 0.73	8.6 ± 0.81	21 ± 2.3	14.8 ± 6.5
Detomidine 50 µg/kg	t_0	5.44 ± 1.83	9.7 ± 2.76	159 ± 9.18	6.31 ± 0.32	7.48 ± 0.54	10.5 ± 0.76	25 ± 1.15	10.37 ± 1.0
	t_{15}	7.29 ± 2.50 *	9.01 ± 0.6	166 ± 6.69	6.6 ± 1.9	7.16 ± 0.77	10.1 ± 0.93	23 ± 0.8	8.7 ± 0.49
	t_{30}	7.17 ± 1.08 *	9.0 ± 2.7	145 ± 20	5.7 ± 0.37	7.33 ± 0.90	9.8 ± 0.59	24 ± 1.0	9.9 ± 3.2
	t_{60}	11.35 ± 1.4 *	10.4 ± 5.1	153 ± 9.17	6.3 ± 0.19	7.3 ± 1.15	9.2 ± 0.9	22 ± 1.5	10.4 ± 6.5
	Recovery	11.49 ± 0.8 *	8.7 ± 2.3	160 ± 17.0	6.7 ± 0.50	7.6 ± 1.23	10.0 ± 0.75	22.6 ± 1.15	8.7 ± 1.65
Detomidine 75 µg/kg	t_0	6.05 ± 0.71	8.1 ± 0.74	139 ± 16.2	7.2 ± 0.45	6.82 ± 0.38	8.2 ± 1.1	21 ± 1.15	11.4 ± 5.09
	t_{15}	7.01 ± 0.57 *	9.2 ± 2.30	132 ± 24	6.2 ± 0.67	8.57 ± 1.8	8.56 ± 1.96	23 ± 1.0	11.8 ± 1.37
	t_{30}	7.47 ± 0.83 *	9.14 ± 2.4	160 ± 29	6.17 ± 1	7.02 ± 0.75	8.66 ± 1.28	21.6 ± 2.0	9.8 ± 2.85
	t_{60}	9.6 ± 1.5 *	9.74 ± 3.5	160 ± 17	6.01 ± 0.34	7.4 ± 0.66	9.5 ± 1.32	24 ± 0.0	11.1 ± 4.40
	Recovery	9.8 ± 2.0 *	8.9 ± 3.66	155 ± 42	6.11 ± 1.04	6.8 ± 0.49	9.06 ± 0.51	21 ± 1.0	9.6 ± 2.38

* Statistically different ($P<0.05$) by pairwise analysis.

et al, 1986 and El-Maghraby and Atta, 1997). Bradycardia has been also documented in dromedary after premedication with xylazine (Khamis *et al*, 1973; Bolbol *et al*, 1980 and White *et al*, 1987). These significant changes in heart rate after the use of detomidine in camel are contrary to the findings of some other reports for xylazine (Peshin *et al*, 1980). Bradycardia following administration of alpha-2 adrenoceptor agonist may be due to central stimulation that mediated through the vagus nerve (Hall and Clarke, 1991).

The reported respiratory depression associated with detomidine is a common adverse effect of alpha-2 agonists. This result might be in agreement with the findings of other studies in horses (Short *et al*, 1986). However, the decrease of respiratory rate was not significant. This result might be in agreement with that reported after the use of xylazine (Peshin *et al*, 1980). Although alpha-2 agonists have a relaxing effect on the gastrointestinal tract and are associated with decreased motility (Hall and Clark, 1991), no marked tympany was noticed on camels of this study.

The significant hyperglycaemia seen following detomidine administration concurs with the results reported after camel sedation with xylazine in some studies (Peshin *et al*, 1986 and Ali *et al*, 1989). It may be attributed to increased adrenergic activity, decrease in the secretion or effects of insulin or increase in the secretion or effect of glucagons (Custer *et al*, 1977 and Ali *et al*, 1989).

The frequent urination after administration of alpha-2 agonists was thought to be through inhibition of antidiuretic hormone release and hyperglycemia (Hall and Clark, 1991). The absence of penis protrusion even in deeply sedated camels is consistent with the result observed after sedation of camels with xylazine (Khamis *et al*, 1973). The later authors attributed this observation to some anatomical features; where the preputial orifice of the dromedary is relatively narrow, surrounded by muscular tissues of the prepuce, which are directed backwards enabling the protrusion of the penis only in its erected state.

In conclusion, detomidine seems to be safe and effective sedative and analgesic agent for camels. The intravenous administration of detomidine in a dose rate of 75 g/kg produced profound sedation and analgesia. Detomidine could

be used for variety of diagnostic and minor surgical procedures in camels.

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References

Ali BH (1988). A survey of some drugs commonly used in the camel. Veterinary Research Communications 12: 67 - 75.

Ali BH, El-Sanhouri AA and Musa BE (1989). Some clinical, haematological and biochemical effects of four tranquilisers in camels (*Camelus dromedarius*). Revue Elev. Med. Vet. Pays Trop. 42 (1): 13-17.

Bolbol AE, Hassanein A and Ibrahim H (1980). Some studies in the camel after sedation with Rompun. Vet. Med. Rev. 1: 55 - 60.

Custer R, Kramer L and Kennedy S (1977). Haematologic effects of Xylazine when used for restraint of Bactrian camels. Journal of American Veterinary Medical Association 171: 899 - 901.

Denning HK (1972). The use of Rompun in the dromedary in diagnostic splenectomy (Infection with *Trypanosoma evansi / surra*). Vet Med. Rev 3/4: 239-242.

El-Amrousi S, Gohar HM, Hafez AM and Ramadan RO (1986). Saffan Anesthesia in camels. Assiut Veterinary Medical Journal 15 (30): 191-195.

El-Maghraby HM and Atta A (1997). Sedative and analgesic effects of detomidine with or without butorphanol in donkeys. Assiut Veterinary Medical Journal 37(73): 201-211.

Fahmy Lotfia S, Farag KA, Mostafa MB and Hegazy AA (1995). Propofol anesthesia with xylazine and diazepam premedication in camels. Journal of Camel Practice and Research 2(2): 111-113.

Fouad KA (2000). Camel surgery and anesthesia. Egyptian Journal of Veterinary Science 34: 1 - 10.

Fouad KA and Morcos MB (1965). Comblen (Bayer) as a premedication before epidural anaesthesia in Camel. Berline und Munchen Tierarztiche. Wochen 78: 44 - 45.

Hall LW and Clark KW (1991). Veterinary Anesthesia. 9th Ed, Bailliere Tindal. pp 51 - 71.

Jochle W and Hamm D (1986). Sedation and analgesia with Domosedan in horses: Dose response studies on efficacy and duration. Acta. Vet. Scand. 82:69-84.

Jochle W, Moore JJM and Brown J (1989). Comparison of detomidine, butorphanol, flunixine meglumine and xylazine in cases of equine colic. Equine Veterinary Journal (Suppl) 7: 111-114.

Khamis Y, Fouad K and Sayed A (1973). Comparative studies on tranquilisation and sedation in *Camelus dromedarius*. Veterinary Medical Review 4: 336 - 345.

Peshin PK, Nigam JM, Singh SC and Robinson BA (1980). Evaluation of xylazine in camels. Journal of American Veterinary Medical Association 177 (9): 875 - 878.

Peshin PK, Singh AP, Sing J, Chawla SK and Lakharu JC (1986). Acid-base and blood gass changes following xylazine administration in buffalo and camel. Indian Journal of Animal Science 56(2): 198 - 202.

Raekallio M, Kivalo M, Jalanka H and Vainio O (1991). Meditomidine/ketamine sedation in calves and its reversal with atipamezole. Journal of Veterinary Anaesthesiology 18:45-47.

Said AH (1972). Clinical comparative studies on different tranquilisers in camel. MVSc thesis, Cairo University, Egypt.

Sharma DK, Behl SM, Khanna BM and Datt SC (1982). Use of xylazine as anesthetic in caesarean section in a camel. Haryana Veterinarian 21(1): 50-51.

Sharma SK, Singh J, Peshin PK and Singh AP (1983). Evaluation of chloral hydrate anesthesia in camels. Zentralbl Veterinarmed A 30 (9) : 674-681.

Short CE (1992). Alpha-2 agents in animals: sedation, analgesia and anesthesia. Ist Ed, Veterinary Practice Publishing Company, California. pp 23-81.

Short CE, Matthews N, Harvey R and Tyner CL (1986). Cardiovascular and pulmonary function studies of a new sedative/analgesic (Detomidine) for use alone or in horses as a preanaesthetic. Acta Vet Scand 82: 139-155.

White RJ, Bark H and Bali S (1986). Halothane anaesthesia in the dromedary camel. Veterinary Record 119: 615-617.

White RJ, Bali S and Bark H (1987). Xylazine and Ketamine anaesthesia in the dromedary camel under field conditions. Veterinary Record 120 (5): 110 - 113.