SEDATIVE AND ANALGESIC EFFECTS OF DETOMIDINE IN CAMELS (CAMELUS DROMEDARIES)

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ABSTRACT

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

INTRODUCTION

Drugs for sedation and tranquilization 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 anesthetics, tranquilizers 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 aeepromazine have been early evaluated as sedatives in camels **(Said 1972, Khamis et al, 1973, Ali et al, 1989)**. Despite the advances in the field of tranquilizers and their uses in domestic animals, experienee with their appliеation on the camel have been still lacking until recent years **(Fouad, 2000)**.

Alpha-$_2$ adrenoceptor agonists (Xylazine, detomidine, medetomidine, and romifidine) have been extensively used in the field of veterinary anesthesiology for their sedative properties **(Hall and Clark, 1991)**. These drugs have been used as sole agents for restrain or calming of camels or to reduce stress **(Ali, 1988)**. If these agents are inadequate to complete involved surgical procedures, supplementation with local analgesic or induction with general anesthesia 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 trails 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, hematological and biochemical effects.

MATERIALS AND METHODS

Fifteen mature apparently healthy one humped camels, (9 males and 6 females), aged six to fifteen years and ranged from 300 to 450 body weight were used along this study. Resting rectal temperature, pulse, and respiratory rates were measured and a complete blood count was made before each treatment to asses' animals' health.

Camels were divided randomly into three equal groups (5 camels in each group). 1% detomidine hydrochloride (Domosedan: Orion Corporation, Animal Health Division) was injected intravenously at the dose levels of 25, 50 & 75µg/kg body weight 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 assed and graded to mild, moderate and deep. Analgesia was detected and 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 for 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 appearing recovery time for determination of hemoglobin (Hb%), packed cell volume (PCV%) and RBCs and WBCs counts. Blood serum was also analyzed for blood urea nitrogen and creatinine concentrations.

Statistical analysis of the data was performed by using 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 & 2). Mild salivation and lacrimation were also detected. Ataxia varied from mild to deep, the degree of ataxia increased by increasing the dose of detomidine. Although all camels remained in a standing position after administration of detomidine in a dose rate of 25 or 50µg/kg b.wt., camels which received 75µg/kg b.wt. revealed sternal recumbency within 10 minutes. Frequent urination commencing about 40- 60 minutes after administration of detomidine was observed along this study. 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 & 75µg/kg b.w. respectively. The degree of sedation was more or less dose dependant and rated from mild to deep. The depth of sedation induced by 75µg/kg was greater than that induced by either 25 or 50µg/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 ± 4.17 and 37± 5.19 minutes after intravenous injection of detomidine at 25, 50 & 75µg/kg b.w. respectively. Intravenous administration of detomidine in a dose rate of 25µg/kg induced a poor analgesic effect which ranged from 0 (no obvious analgesia) to grade 1 analgesia. The analgesic effect of 75µg/kg b.w. was excellent (grade 3) as indicated by lack of response to painful and electrical stimulations.

Significant bradycardia was recorded in all camels which received detomidine (Table 2). Heart

rates were significantly reduced after intravenous injection of the three doses of detomidine. Twenty beats/minute was the lowest rate recorded. Auscultation showed also irregular rhythm and dropped beats. Minimal depression in the respiratory rate was also recorded (Table 3). However, the respiratory depression was not significant from induction of sedation till the recovery of camels in the three tested doses. The changes in rectal temperature were also not statistically significant.

Intravenous administration of the three different doses of detomidine did not reveal significant changes in some of the hematological and biochemical values (Hb%, PCV%, WBCs and RBCs counts and blood urea nitrogen, blood urea nitrogen levels). A significant (P< 0.05) hyperglycemia was observed 5 min. after detomidine administration (Table 3). The increased blood glucose was recorded in the range of recovery.

DISCUSSION

Alpha-2 agonists are now frequently used in camels for a variety of diagnostic and surgical procedures. These include, restraint, teeth rasping, 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 intramuscular depot.

The onset of sedation (2-3 minutes) after intravenous injection of detomidine. No difference was observed between the different doses of detomidine. The analgesia was dose dependent, while the low dose (25µg/kg) was not sufficient, the higher doses (50 µg/kg and 75µg/kg) revealed moderate analgesic effect. It should be pointed that the high dose was associated with high degree of ataxia. As a result, increasing the dose of alpha-2 agonist increases ataxia without improving the response of the animal to painful stimulation (**Short, 1992**).

Salivation was minimal in the three tested dosage in this study. This might contradict with that reported in cattle that show obvious increased salivation after detomidine injection (**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 et al, 1990 and El-Maghraby and Hiba, 1997**). Bradycardia has been also documented in domestic animals premedicated with xylazine (**Thurmon 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 **(Penshin 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 el 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 **(Penshin 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 hyperglycemia seen following detomidine administration concurs with the results reported after camel sedation with xylazine in some studies **(Penshin 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 thought to be through inhibition of antidiurtic 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.

Although both of detomidine and xylazine belongs to the same group, one of the marked differences between them appears to be in their effect on the uterus, whereas xylazine has eebolic effects, detomidine slows electrical uterine activities in pregnant ruminants **(Hall and Clark, 1991)**. No antagonist has been tried to reverse the effects of detomidine in dromedary camels. However, atipamezole might reverse the action of detomidine in camels, it has been demonstrated to be effective in reversal of both sedative/analgesic and physiologic changes in ruminant receiving alpha-$_2$ adrenoceptor agonists **(Rackallio et al, 1991)**. Although, yohimbine didn't reverse the hematological effects of xylazine in camels (Al-Busadan and Osman, 2001). In the llama, a related species, a combination of intravenous yohimbine and 4-amino-pyridine gave rapid reversal of xylazine induced sedation but doxpram was ineffective **(Reibold et al, 1989)**.

In conclusion, detomidine seems to be safe and effective sedative and analgesic agent for cam-

els. The intravenous administration of detomidine in a dose rate of 75µg /kg b.wt. revealed profound sedation and analgesia. Detomidine could be used for varity of diagnostic and minor surgical procedures in camels.

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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 and 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
	t15	11.33± 1.15	37.7±.7	29.66± 4.93 *
	t30	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	37.5± 0.66	46.33± 12.4
	t15	13.33± 2.08	37.9± 0.65	30.66± 5.03 *
	t30	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
	t15	13.0± 2.64	38 1± 0.2	22.33± 5.13 *
	t30	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.

Table 3: Some hematological 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. ± 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	22.5± 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 ± 8.0	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.

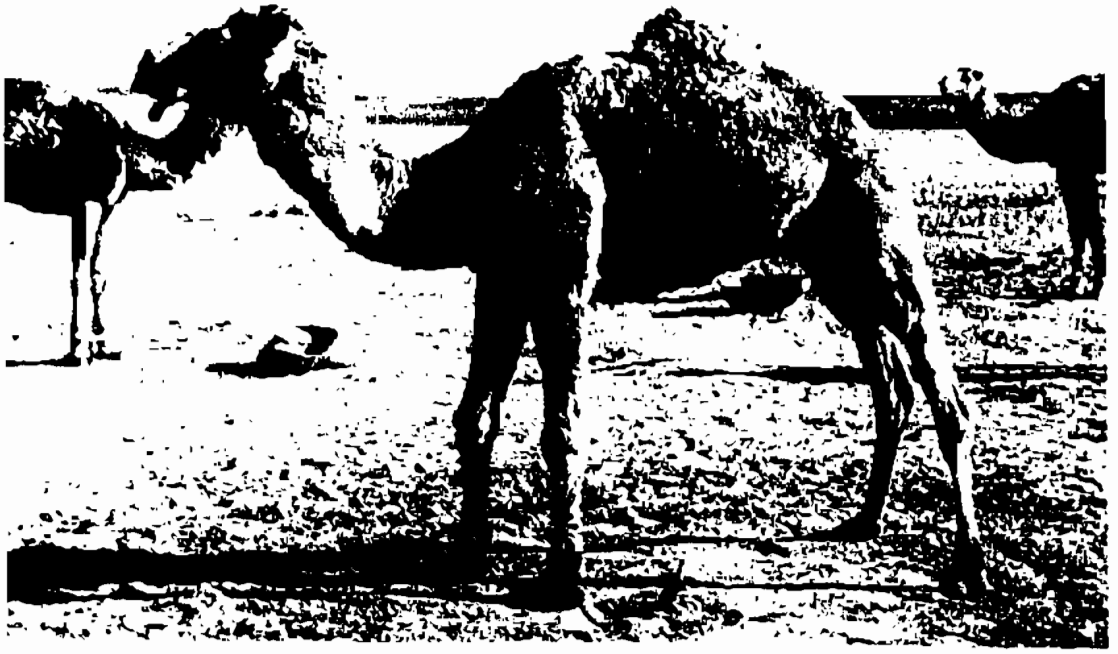

Fig 1: A camel after sedation with sedation with intravenous detomidine hydrochloride Notice the drooped head and abduction of the limbs.

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

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الملخص العربى

التأثير المهـدئ والمسكن لعقـار الديتومـدين فى الجمـال

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حديثاً تم دراسة إستخدام عقار الديتومدين فى مجال التخدير فى العديد من الحيوانات ولكن لم يتم التقييم الموضوعى له فى الجمال. وقد تم في هذه الدراسة تقييم الحقن الوريدى لعقار الديتومدين فى الجمال حيث تم حقن ثلاث جرعات مختلفة من ذلك العقار (٢٥ ، ٥٠ ، ٧٥ ميكروجرام لكل كيلو جرام من وزن الحيوان) في ثلاث مجموعات مختلفة من الجمال تتراوح أعمارهم من ستة إلى خمسة عشر عاماً وتكونت كل مجموعة من خمسة جمال، وتمت دراسة التأثير المهدىء والمسكن وكذلك التغيرات فى صورة الدم بصورة منتظمة بتلك المجموعات.س

وقد لوحظ تأثر الجمال بعد فترة تراوحت من دقيقتين إلى ثلاث دقائق بعد الحقن الوريدى للعقار وظهر ذلك بوضوح من خلال الهدوء الواضح للحيوان بالإضافة إلى بعض العلامات الأخرى مثل إرتخاء وتدلى الشفة السفلى والجفن الأسفل للعين والصوان الخارجى للأذن وترنح الحيوان بصور متفاوتة أدت إلى رقود الجمال وعدم القدرة على الوقوف فى بعض الأحيان (خصوصاً بالنسبة للجرعة الأعلى) وقد امتدت فترة التهدئة إلى حوالى ٢٦، ٤٠، ٥٠ دقيقة بالنسبة للجرعات الثلاثة على التوالى وتزامن التأثير المهدىء مع تأثير تسكينى واضح للعقار وإن كان قد إمتد التسكين لفترة أقل إمتدت إلى حوالى ٢٠، ٢٨، ٣٧ دقيقة بالنسبة للجرعات الثلاثة على التوالى، ولقد إختلفت درجة التسكين باختلاف جرعة الديتومدين وتناسبت معها تناسباً طردياً، ووجد أن للديتومدين تأثير واضح على ضربات القلب التى إنخفضت بصورة معنوية، وباستثناء إرتفاع نسبة السكر بالدم فإنه لاتوجد فروق ذات دلالة معنوية فى تحليل صورة الدم على مستوى الجرعات الثلاثة. ولقد تبين أن الجرعة الأعلى فى هذه الدراسة (٧٥ ميكروجرام لكل كيلوجرام من وزن الحيوان) ذو فاعلية عالية السيطرة على الجمال وفحصها بدون مضاعفات جانبية وتؤدى إلى مستوى عالى من التسكين يسمح بإجراء بعض التدخلات الجراحية البسيطة تحت تأثيرها.