Journal of Animal and Veterinary Advances 11 (23): 4460-4464, 2012
ISSN: 1680-5593

Efficacy of Intravenous Administration of Ephedrine for Prevention of Xylazine-Induced Hypotension in Camels (*Camelus dromedarius*)

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Abstract: The effects of ephedrine on Arterial Blood Pressure (ABP), heart rate, respiratory rate and some haematology and biochemistry parameters were carried out on six camels in two periods. In the first period, all camels received Intravenous (IV) 0.2 mg kg^{-1} xylazine while in the second period, same camels received IV 0.2 mg kg^{-1} ephedrine followed 10 min later by IV 0.2 mg kg^{-1} ephedrine and 0.2 mg kg^{-1} xylazine administered at same time. Cardiopulmonary measurements were taken at baseline and were further recorded at 10, 20, 30, 40, 50 and 60 min after treatment in both groups. Mean heart rate, respiratory rate and ABP measurements decreased significantly after xylazine in the first period. Mean heart rate in the second period decreased significantly at all times compared to baseline and to first period while mean ABP measurements were significantly higher at 10, 20, 30 and 40 min compared to first period. There was a significant decrease of Platelet count (PLT), Haemoglobin (HB), Urea Nitrogen (BUN), Albumen (ALB), Alanine aminotransferase (ALT) and Glucose (GLU) in the first period compared to the second period and there was significant increase of Calcium (Ca) and potassium (K^+) after treatments in the second period. Ephedrine was safe and effective in improving ABP in xylazine induced hypotension in camels.

Key words: Camel, ephedrine, xylazine, hypotension, haematology, biochemistry

INTRODUCTION

Ephedrine is a synthetic, noncatecholamine sympathomimetic that stimulates alpha-and beta-adrenergic receptors directly and indirectly by causing endogenous norepinephrine release (Stoelting, 1987; Hemmings and Hopkins, 2005). Ephedrine is used to counteract hypotension (Lee *et al*., 2002) and it is most commonly administered after the onset of hypotension in human (Hemmings and Hopkins, 2005) and in small animals (Chen *et al*., 2007). Ephedrine administration in anaesthetized dogs caused significant increases in mean ABP, cardiac output and stroke volume and significant decrease in heart rate (Wagner *et al*., 1993). Similar results were found in anaesthetized horses (Grandy *et al*., 1989; Hellyer *et al*., 1998; Lee *et al*., 2002) where ephedrine caused significant increase in ABP. Xylazine is an alpha2-adrenoceptor agonist used commonly as sedative in veterinary anaesthesia (England and Clarke, 1996). It produces respiratory depression, bradycardia, initial hypertension followed by a prolonged hypotension and decreased cardiac output due to sympathetic blockade and vagal stimulation (Clarke and Hall, 1969; Maze and Tranquilli, 1991; Wagner *et al*., 1991). There is no information regarding the use of ephedrine in camels therefore the objectives of this study were to determine the effects of ephedrine on heart rate, respiratory rate, ABP and some haematology and biochemistry parameters in healthy camels premedicated with xylazine.

MATERIALS AND METHODS

Six healthy dromedary camels of two breeds, 4 Shoael and 2 Majaheem, 4 males and 2 females with mean age±SD 4.0±1.8 years and weight 393.7±136.8 kg were used for this two periods study. All camels were considered to be healthy, based on the results of physical examination. Food but not water was withheld for 24 h before trials. Camels were restrained manually in sternal recumbency at least 3 h before start of trials. In the first period, all camel received xylazine (X-group) (Rompun, Bayer, Turkey) as a single IV dose of 0.2 mg kg^{-1} into the jugular vein. Trials on the second period was conducted one day later where all camels received ephedrine (Ephedrine Hydrochloride, Martindale Pharmaceuticals, Essex, UK) as a bolus IV dose of 0.2 mg kg^{-1} followed 10 min by next IV dose of 0.2 mg kg^{-1} ephedrine and 0.2 mg kg^{-1} xylazine (E-X-group) administered at same time. Baseline heart rate (manually by a stethoscope), respiratory rate (counting thoracic movements) and rectal temperature (electronic thermometer) were assessed. Baseline indirect blood pressure values were assessed by oscillography

using a cuff placed around the base of the tail and connected to a patient monitor (Infinity Delta XL, Drager Medical, Germany). These parameter values were further recorded at 10, 20, 30, 40, 50 and 60 min after xylazine administration in both groups and at 5 min (5AE) and 10 min (10AE) after ephedrine administration in the E-X-group. Blood samples (14 mL) were taken at baseline, 10 and 60 min after administration of xylazine or ephedrine-xylazine in both groups. They were collected from the jugular vein via disposable syringes and divided into EDTA tubes for haematologic evaluation and to plain tubes without anticoagulant for the biochemical analysis. For haematological evaluation, each tube was inverted 2-3 times to ensure thorough mixing and analyzed within 2 h using an automated haematology analyzer (VetScan HM2, Abaxis Veterinary Diagnostics, USA) for total erythrocyte count (RBC), Haemoglobin (HB), Haematocrit (HCT), White Blood Cell count (WBC), Lymphocytes (LY), Monocytes (MO), Neutrophil (NE), Mean Cell Volume (MCV) and Platelet count (PLT). For biochemical analysis, serum was harvested by centrifugation and stored at -80°C until analyzed by automatic analyzer (VetScan VS2, Abaxis Veterinary Diagnostics, USA) for Albumen (ALB), Alkaline Phosphatase (ALP), Alanine aminotransferase (ALT), Amylase (AMY), Urea Nitrogen (BUN), Calcium (Ca), Phosphorus (PHOS), Creatinine (CRE), Glucose (GLU), sodium (Na^+), potassium (K^+), Total Protein (TP) and Globulin (GLOB).

Statistical analysis was performed using the General Linear Model (GLM) procedure (SAS Institute Inc., Cary NC USA, 2002). Data presented as mean±SD unless otherwise stated. Data was calculated and tested for the significance using the t-test. Moreover, arc sine transformation was done to percentage data. The $p<0.05$ was considered significant.

RESULTS AND DISCUSSION

Table 1 shows that mean heart rate, respiratory rate and ABP measurements decreased significantly in X-group after treatment. Following the initial dose of ephedrine in the E-X-group, mean heart rate was signficantly lower than baseline at AE5 and AE10. Mean respiratory rate did not differ significantly between AE5, AE10 and the baseline. Mean ABP measurements at AE5 were lower than baseline but were then higher at AE10 when compared to baseline. After administration of treatments in the E-X-group, mean heart rate decreased significantly at all times when compared to baseline and to X-group. Mean ABP measurements in E-X-group were significantly higher at 10, 20, 30 and 40 min compared to X-group. There was a significant decrease of Haemoglobin (HB), Platelet count (PLT) (Table 2), Urea Nitrogen (BUN), Albumen (ALB), Alanine aminotransferase (ALT) and Glucose (GLU) (Table 3) in the X-group compared to the E-X-group and significant increase of Calcium (Ca) and potassium (K^+) (Table 3) after treatments in the E-X-group.

The recommended IV dose, route, timing and frequency of ephedrine for use in human and animals is controversial (Di Roio *et al*., 1997; Egger *et al*., 2009). The recommended IV dose of ephedrine ranges from 0.02-0.25 mg kg^{-1} for dogs and cats (Egger *et al*., 2009; Carroll and Martin, 2007), 0.06-0.2 mg kg^{-1} in horses (Grandy *et al*., 1989; Lee *et al*., 2002) and 0.08-0.9 mg kg^{-1} in human (Hemmings and Hopkins, 2005). There is no information regarding the dose rate of ephedrine in camels therefore based on earlier studies, a dose rate of 0.2 mg kg^{-1} was chosen in this study to examine its efficacy and safety. Ephedrine has been administered prior to premedication (Cleary-Goldman *et al*., 2005), after premedication (Egger *et al*., 2009) and during general

Table 1: Mean values±SD of Heart Rate (HR) beats/minute, respiratory rate (f_R) breaths/min, Mean Arterial blood Pressure (MAP), Systolic Arterialblood Pressure (SAP) and Diastolic Arterial blood Pressure (DAP) at the Baseline (BL) at 5 min after ephedrine Administration (5AE) at 10 min after ephedrine Administration (10AE) and 10-60 min after xylazine and ephedrine-xylazine administration

	Time (min)								
Variables	BL	5AE	10AE	10	20	30	40	50	60
HR									
Xylazine	47.5±4.90^{ax}	-	-	33.7±5.40^{b}	36.3±4.50^{bx}	34.2±8.70^{b}	31.8±5.90^{b}	31.3±5.40^{b}	33.2±9.20^{b}
Ephedrine	41.0±5.20^{ay}	34.3±3.70^{b}	34.5±3.80^{b}	29.0±2.80^{b}	29.3±4.80^{by}	29.3±4.20^{b}	29.3±6.40^{b}	29.3±4.70^{b}	28.8±3.70^{b}
f_R									
Xylazine	20.5±7.80^{ax}	-	-	14.3±3.80^{b}	13.3±3.90^{b}	15.7±4.10^{b}	16.7±3.90^{b}	15.0±2.10^{b}	15.8±1.80^{b}
Ephedrine	14.2±5.80^{ay}	14.3±3.70^{a}	14.7±3.50^{a}	18.0±6.30^{a}	17.3±3.60^{a}	15.5±2.40^{a}	17.3±2.20^{a}	17.2±2.60^{a}	14.5±1.90^{a}
MAP mmHg									
Xylazine	129.3±19.8^{a}	-	-	119.5±10.8^{acx}	109.3±11.1^{acx}	108.3±10.1^{acx}	105.3±11.7^{bcx}	111.3±18.3^{ac}	109.8±21.8^{ac}
Ephedrine	142.2±23.2^{ac}	133.7±27.6^{ac}	146.5±24.0^{a}	156.7±36.1^{abcy}	146.8±17.0^{acy}	146.5±11.6^{acy}	138.3±10.6^{acey}	130.7±16.7^{ac}	123.8±12.4^{bc}
SAP mmHg									
Xylazine	161.5±18.5^{a}	-	-	143.8±10.7^{acx}	139.0±11.9^{ax}	131.3±16.1^{bcx}	129.7±13.7^{bcx}	131.2±20.9^{bcx}	140.3±31.3^{a}
Ephedrine	171.3±24.3^{a}	150.7±33.9^{ab}	179.8±37.3^{ac}	190.5±33.6^{acy}	176.8±17.9^{dy}	174.0±18.4^{ay}	165.5±13.5^{ay}	162.8±19.9^{abdy}	155.2±18.4^{abd}
DAP mmHg									
Xylazine	96.0±22.2^{a}	-	-	96.7±15.5^{ax}	91.0±9.50^{ax}	92.0±7.40^{ax}	87.5±8.70^{ax}	92.7±14.7^{a}	88.3±20.9^{a}
Ephedrine	112.8±23.3^{a}	109.7±31.8^{ac}	123.5±19.3^{a}	128.8±27.6^{ay}	122.8±16.7^{ay}	120.3±11.1^{ay}	114.3±11.9^{ay}	112.8±15.5^{a}	90.5±35.4^{bc}

$^{a-d}$Means within a row with different superscripts differ significantly ($p<0.05$); $^{x, y}$Means within a column with different superscripts differ significantly ($p<0.05$)

Table 2: Haematology (mean±SD) at baseline, 10 and 60 min after premedication with xylazine in the X-group and after ephedrine-xylazine administration in the E-X-group

	Time (min)		
Variables	Baseline	10	60
RBC (cells µL^{-1})			
Xylazine	11.2±1.20	10.7±1.600	9.8±1.60
Ephedrine	11.1±2.90	11.4±1.600	9.9±1.10
HB (g dL^{-1})			
Xylazine	13.9±1.90^{ac}	13.1±1.200^{ax}	11.7±1.10^{ab}
Ephedrine	14.4±1.90^{a}	15.4±1.400^{ay}	12.4±0.90^{b}
HCT (%)			
Xylazine	32.5±3.30	30.8±4.800	28.0±4.10
Ephedrine	31.9±7.90	33.0±3.800	28.2±2.90
WBC (g L^{-1})			
Xylazine	18.4±3.60	17.0±3.100	16.1±2.60
Ephedrine	16.1±2.50	17.6±1.700	14.6±1.80
LY (%)			
Xylazine	36.9±8.70	35.9±9.300	34.2±8.70
Ephedrine	36.7±8.20	35.4±7.800	34.3±8.70
MO (%)			
Xylazine	6.7±1.20	5.9±0.600	6.1±0.90
Ephedrine	4.2±0.50	4.0±1.000	3.4±0.90
NE (%)			
Xylazine	56.5±8.30	58.2±9.400	59.7±8.90
Ephedrine	59.4±58.9	59.3±8.200	62.3±8.30
MCV (fl)			
Xylazine	28.8±0.80	28.7±0.800	28.7±0.80
Ephedrine	28.5±0.60	28.7±0.800	28.3±0.50
PLT (g dL^{-1})			
Xylazine	173.0±60.80	124.7±61.70^{x}	144.0±49.8
Ephedrine	202.3±118.4	250.0±115.4^{y}	159.8±40.7

$^{a-d}$Means within a row with different superscripts differ significantly ($p<0.05$); $^{x, y}$Means within a column with different superscripts differ significantly ($p<0.05$)

Table 3: Serum chemistry (mean±SD) at baseline, 10 and 60 min after premedication with X in first group and after XE administration in the second group

	Time (min)		
Variables	Baseline	10	60
ALB (g dL^{-1})			
Xylazine	4.9±0.500^{a}	4.4±0.400^{ax}	4.8±0.400^{ax}
Ephedrine	4.9±0.200^{ac}	5.3±0.300^{ay}	5.6±0.500^{aby}
ALP (U L^{-1})			
Xylazine	91.2±39.80	72.1±32.60	81.2±31.30
Ephedrine	61.8±10.10	63.2±10.70	64.5±13.70
ALT (U L^{-1})			
Xylazine	7.2±1.700^{x}	6.3±1.900^{x}	6.8±1.600^{x}
Ephedrine	18.8±0.500^{ay}	21.3±0.400^{by}	24.2±1.500^{cy}
AMY (U L^{-1})			
Xylazine	629.5±237.5	621.8±241.5	616.7±238.7
Ephedrine	561.1±22.70	572.2±26.00	583.7±24.60
BUN (mg dL^{-1})			
Xylazine	15.5±1.900	14.2±1.600^{x}	15.8±0.900
Ephedrine	17.1±0.500	17.4±0.400^{y}	17.2±0.700
Ca (mg dL^{-1})			
Xylazine	4.1±0.800^{x}	3.4±1.700	4.5±0.700
Ephedrine	2.5±0.300^{acy}	2.9±0.800^{a}	3.8±1.200^{ab}
PHOS (mg dL^{-1})			
Xylazine	14.0±2.100^{x}	11.9±1.800	13.4±1.700^{x}
Ephedrine	11.0±1.900^{y}	10.3±1.200	11.1±2.300^{y}
CRE (mg dL^{-1})			
Xylazine	1.5±0.300^{x}	1.3±0.300	1.4±0.300
Ephedrine	1.7±0.600^{acy}	1.5±0.300^{a}	1.7±0.600^{ab}
GLU (mg dL^{-1})			
Xylazine	7.5±1.900	6.5±0.600^{x}	7.3±1.000^{x}
Ephedrine	10.1±1.700	9.9±3.500^{y}	11.0±4.100^{y}
Na^{+} (mmol L^{-1})			
Xylazine	152.5±8.200^{bcx}	142.3±9.800^{a}	146.3±6.900^{ac}
Ephedrine	143.8±1.400^{y}	144.0±1.600	147.3±1.300
K^{+} (mmol L^{-1})			
Xylazine	6.3±2.500	7.1±2.200	7.4±1.600
Ephedrine	6.5±0.300^{a}	6.0±0.400^{a}	7.9±0.200^{b}
TP (g dL^{-1})			
Xylazine	7.8±1.000^{x}	8.2±1.000	7.8±1.300
Ephedrine	9.0±0.300^{acy}	8.2±0.100^{a}	7.7±0.200^{ab}
GLOB (g dL^{-1})			
Xylazine	2.1±0.500^{x}	2.2±0.500	2.2±0.500
Ephedrine	2.7±0.000^{y}	2.7±0.100	2.6±0.200

$^{a-d}$Means within a row with different superscripts differ significantly ($p<0.05$); $^{x, y}$Means within a column with different superscripts differ significantly ($p<0.05$)

anaesthesia (Hellyer *et al*., 1998; Lee *et al*., 2002). In this study, ephedrine was initially administered prior to premedication to observe its cardiopulmonary effects during first 10 min in healthy camel then a second dose of ephedrine was administered in combination with xylazine which was included in the design of this study as the most common premedication used in camels (Al-Mubarak *et al*., 2008) and due to its cardiopulmonary depression characteristics (England and Clarke, 1996) that mimic clinical situation. Mean heart rate, respiratory rate and ABP measurements in the X-group decreased significantly after premedication with xylazine, similarly as with the previous studies (Doherty *et al*., 1987; Mama *et al*., 1996). Mean heart rates in the E-X-group were significantly lower after ephedrine administration at all time points compared with baseline and at 20 min when compared to the X-group which is in similar to previous studies in horses (Grandy *et al*., 1989; Hellyer *et al*., 1998) and in dogs (Wagner *et al*., 1993; Egger *et al*., 2009). In contrast, heart rate was increased in other studies in horses (Lee *et al*., 2002) and dogs (Chen *et al*., 2007). Lee *et al*. (2002) explained that by the additive effect of extra dose of E and use of halothane which increases the sensitivity of the heart to epinephrine-induced arrhythmias while Chen *et al*. (2007) explained that by including glycopyrrolate in their protocol and by the underlying mechanism of action of ephedrine that stimulates cardiac β1-receptors (Stoelting, 1998). Chen *et al*. (2007) demonstrated in anaesthetized dogs that mean ABP increased immediately following 0.2 mg kg^{-1} ephedrine administration with significant differences from baseline at 2.5 min then decreased rapidly, returning to near or below baseline within 5 min while Egger *et al*. (2009) found in premedicated dogs that systolic ABP was significantly lower than baseline at 5 and 40 min after 0.1 mg kg^{-1} ephedrine administration. In anaesthetized horse, mean ABP measurement was significantly increased at 5 and 15 min (Hellyer *et al*., 1998) and at 5, 10, 15 and 30 min (Lee *et al*., 2002). In this

study, mean ABP measurements at AE5 were slightly lower than baseline and this could be attributed to the considerable differences in response between individual animals as the mean ABP measurements were higher than baseline at 5 min in 4 camels involved in this study. However, mean ABP measurements were then significantly higher at 10, 20, 30 and 40 min compared to the X-group which is in agreement to earlier studies (Lee *et al*., 2002; Hellyer *et al*., 1998). It is possible that immediate or transient increase in ABP measurements after ephedrine administration in this study were not observe because measurement were made after 5 min and then at every 10 min intervals and the use of indirect ABP monitoring have not allowed continuous monitoring of ABP. However, direct ABP measurement was not carried out in this study as arterial catheterization is problematical in this species due to their thick skin and muscle layers. Nevertheless, the method of indirect oscillometry for blood pressure measurement used in this study provides useful information in most horses but may produce erroneous values in a small number (Hall *et al*., 2001).

In this study, there were a significant decrease of HB and PLT in the X-group compared with E-X-group. Similar effects of xylazine on HB and PLT in camels have been reported elsewhere (Custer *et al*., 1977; Ahmed *et al*., 1996). Such effects may be due to haemodilution or increased spleen storage function (Ahmed *et al*., 1996). GLU and ALT during this study were significantly lower in the X-group compared with E-X-group. This decrease in GLU and ALT after xylazine administration was earlier reported (Custer *et al*., 1977; Al-Busadah, 2002) and attributed to various factors such as changes in body temperature, haemodilution or more leakage of aspartate aminotransferase into plasma during xylazine sedation. However, in this study, most haematological and biochemical parameters obtained were within the normal range of camels (Mohri *et al*., 2008; Hussein *et al*., 2012).

CONCLUSION

In summary, this current study demonstrates that ephedrine at the doses given was safe and effective in improving ABP compared to the X-group although there was a minimum decrease in heart rate after ephedrine administration. However, further research needs to be done to determine the optimal dose, pharmacokinetic parameters (half-life, clearance and volume of distribution), blood gas analysis and continuous capnography recording and direct ABP measurement in order to establish its use and safety in camels.

ACKNOWLEDGEMENTS

The researcher gratefully acknowledges Dr. M. Alkhalifah and Dr. K. Abursais for their assistance in data collection. Prof. M. Mandoor for the statistical analysis. Deanship of Scientific Research in King Faisal University for the financial support.

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