INTERNATIONAL JOURNAL OF AGRICULTURE & BIOLOGY
1560–8530/2003/05–3–316–321
http://www.ijab.org

Effects of Detomidine with Chloral Hydrate Anaesthesia in Horses

MUHAMMAD ARIF KHAN, MUHAMMAD ASHRAF, KHALID PERVEZ, H.A. HASHMI AND ASIM K. MAHMOOD
Department of Clinical Medicine and Surgery, University of Veterinary and Animal Sciences, Lahore–Pakistan

ABSTRACT

Effect of detomidine with chloral hydrate was studied in 18 local bred horses. Three treatments were given to separate groups of horses, i.e. A= chloral hydrate alone @ 100 mg kg^{-1} body weight intravenously, B= detomidine @ 50 µg kg^{-1} body weight intravenously 10 min before chloral hydrate (100 mg kg^{-1} body weight) administration, C= chloral hydrate @ 60 mg kg^{-1} body weight with the same dose rate of detomidine used as in group B. The results of group B were quite encouraging in terms of ideal anaesthesia which is normally required for major surgical intervention in Horses. The treatment used in group C also proved better but the duration of surgical anaesthesia was shorter.

Key Words: Detomidine; Chloride hydrate; Anaesthesia; Horse

INTRODUCTION

The use of horse in race, polo, riding, tent pegging and draft purpose has made it an important part of livestock in Pakistan. This particular species often suffers from certain problems, which require surgical manipulation as a sole remedy. To serve the purpose of anaesthesia to perform various surgical exercises in horses, chloral hydrate has been a drug of choice due to the unavailability of proper anaesthetics in field practice. The drug is also being used now a days by an average practitioner engaged in equine practice. Chloral hydrate was first introduced in 1869 by Liebrick as a somificient. It was the first drug used as intravenous anaesthetic (Oehme & Prier, 1974). Malone (1917) first time reported its use as an intravenous general anaesthetic after a number of trials in horses. The drug is found in colorless crystals with penetrating odour and bitter taste. The hypnotic effect of chloral hydrate is due to the formation of trichloroethanol after metabolism in liver. The sensory routes are mildly affected which results into loss of muscle control leading to frightening and struggling. The drug also depresses respiration and blood pressure and has prolonged recovery period, incordination and less safety margin (Booth & McDonald, 1988).

Various drugs have been tried in the past as pre-anaesthetics in equine to overcome the side effects of the general anaesthetics. Detomidine HCl (Domosedan) is a potent sedative and analgesic for use in veterinary practice (Hall & Clark, 1991). The drug has been tried as pre-anaesthetic with chloral hydrate anaesthesia in this project. The sedative and analgesic actions of detomidine are related to CNS depressions mediated by stimulation of alpha two receptors (Anonymous, 1996).

This study was conducted to evaluate the problems associated with chloral hydrate anaesthesia, to evaluate the effect of detomidine premedication and to observe reduction in the dose rate of chloral hydrate.

MATERIALS AND METHODS

Source of animals. The study was conducted on 18 local bred horses between the age of 5-6 years. The horses were kept at indoor hospital, Department of Clinical Medicine and Surgery, University of Veterinary and Animal Sciences, Lahore. The health status was judged to find out any abnormality. A week prior to study, the animals were treated for ecto and endo parasites. The horses were provided normal feed and water as per routine.

Pre-anaesthetic considerations. General health status of the animal was assessed before the administration of anaesthetic by recording body temperature, pulse rate, respiration rate and auscultation of heart and lungs. The induction and recovery were managed in quiet and isolated place, well padded to prevent the chances of injuries and bruises to the animals.

Fasting. All the animals were kept fast at least 12-18 h before the administration of anaesthetic agents. This was infact done to minimize the hazards of casting i.e. to reduce vigourness, to decrease the mechanical compression of lungs by full gastro-intestinal tract.

Medicine used

Detomidine. Detomidine [4-(5)-(2,3 dimethyl benzyle) imidazole HCl] is a newly developed potent sedative and analgesic for use in veterinary practice. It is found in the form of colourless clear solution, which can be administered intravenously and intramuscularly in various animals. The drug is available with a trade name "Domosedan" containing 10 mg/mL detomidine HCl. It is the product of Orion Pharmaceutical Company, Turku, Finland. Detomidine was used at the dose rate of 50 µg kg^{-1} body weight intravenously in this project.

Chloral hydrate. Chloral hydrate (Trichloroacetyl aldehyde: CCl_3. CHO) is a non-selective CNS depressant. It is readily soluble in water but solutions are irritant. Chloral hydrate was used in the form of intravenous injection

manufactured by Syman's Pharmaceuticals, Lahore, Pakistan in the powder from and 10% solution was prepared and administered at a dose rate of 100 mg kg^{-1} body weight intravenously.

Experimental protocol. The horses were divided into three groups i.e. group A, B and C comprising six horses each. Animals of group A were administered 10% solution of chloral hydrate alone at the dose rate of 100 mg kg^{-1} body weight intravenously in the jugular vein. The animals of group B, were administered detomidine as pre-anaesthetic at the dose rate of 50 µg kg^{-1} body weight and 10 min later chloral hydrate was administered (100 mg kg^{-1} body weight) intravenously, while in group C detomidine was administered at the same dose rate as in group B, but the dose of chloral hydrate was reduced to 60 mg kg^{-1} body weight.

Parameters. Following parameters were recorded during this experiment.

Nature of induction. The animals were vigilantly monitored between the administration of anaesthesia to the disappearance of reflexes to evaluate the nature of induction, which was graded as smooth, shivering, and struggling movements. The induction period was also monitored.

Recumbency period. The total period between disappearance and reappearance of reflexes was recorded

Nature of recovery. It was recorded as smooth, with staggering, difficulty in standing, regurgitation, excessive salivation, lacrimation or excitement showing circling and kicking.

Evaluation of sedation. Sedation was evaluated on the basis of body reflexes in all animals at different intervals.

Evaluation of analgesia. The analgesia was evaluated at different intervals by giving painful stimulus so the animals.

Evaluation of clinical parameters. Temperature, pulse and respiration were recorded before, during and after anaesthesia.

Body reflexes. Ear twitching, protrusion of tongue, palpebral reflex, corneal reflex, pupillary reflex, front pedal reflex, hind pedal reflex, anal reflex, tail response and relaxation of penis in males were observed.

Liver function test (LFTs). The effects of chloral hydrate alone and in combination with detomidine on the liver were also monitored by performing liver function tests such as: Serum Glutamic Pyruvate Transaminase (SGPT) or Alanine Amine Transferase (ALT) or Serum Alkaline Phosphatase (SAP or ALP)

For the measurement of these enzymes, kits prepared by Randox Laboratory, U.K were purchased from local market and used under standard conditions.

RESULTS

Induction. Nature of induction was not satisfactory in group A (Table I). All animals of this group showed vigorous staggering and extra man power was needed to control the animal during this stage, induction time was in the range of 16–18 min with a mean of 16.66 ± 0.81 min (Table II). In group B, induction was better and smooth. The horses first bent their knees and hocks and then gently laid on the ground. The horses did not show any sign of incoordination, discomfort or distress during the induction phase. None of the horses showed straining and movements of legs after attaining recumbency during this phase (Table I). Induction time was in the range of 5-9 min with a mean of 6.83 ± 1.47 min (Table II). The induction was also ideal and smooth in the members of group C. None of the animals of this group showed struggling and shivering during induction (Table I). Induction time was in the range of 6-10 min with a mean of 7.33 ± 1.63 min. Statistical analysis showed a significant difference ($P < 0.05$) (Table II).

Table I. Effect of detomidine on nature of induction and recovery with chloral hydrate induced anaesthesia in equines

Animal No.	Nature of Induction			Nature of Recovery		
	A	B	C	A	B	C
1	Staggering	Smooth	Smooth	Staggering	Smooth	Smooth
2	Staggering	Smooth	Smooth	Staggering	Smooth	Smooth
3	Staggering	Smooth	Smooth	Staggering	Smooth	Smooth
4	Staggering	Smooth	Smooth	Staggering	Smooth	Smooth
5	Staggering	Smooth	Smooth	Staggering	Smooth	Smooth
6	Staggering	Smooth	Smooth	Staggering	Smooth	Smooth
Mean	Staggering	Smooth	Smooth	Staggering	Smooth	Smooth

A = Chloral hydrate 100 mg kg^{-1} body weight; B = Detomidine (50 µg kg^{-1}) with Chloral hydrate 100 mg kg^{-1} body weight; C = Detomidine (50 µg kg^{-1}) with Chloral hydrate 60 mg kg^{-1} body weight

Table II. Effect of detomidine on time of induction, recumbency period and time of recovery with chloral hydrate induced anaesthesia in equine

Animal No.	*Time of induction (minutes)			*Recumbency Period (minutes)			*Time of Recovery (minutes)		
	A	B	C	A	B	C	A	B	C
1	16	8	10	19	65	53	76	62	60
2	18	6	8	22	61	51	73	64	57
3	17	6	6	24	63	55	70	60	54
4	16	9	8	21	60	56	78	65	51
5	16	7	6	22	58	54	71	63	62
6	17	5	6	24	62	50	77	67	59
Mean	16.66	6.83	7.33	22	61.50	53.16	74.16	63.50	57.16
+SD	±0.81	±1.47	±1.63	±1.89	±2.42	±2.31	±3.31	±2.42	±4.07

* indicates significant difference ($P < 0.05$); A = Chloral hydrate 100 mg kg^{-1} body weight; B = Detomidine (50 µg kg^{-1}) with Chloral hydrate 100 mg kg^{-1} body weight; C = Detomidine (50 µg kg^{-1}) with Chloral hydrate 60 mg kg^{-1} body weight

Table III. Comparative effect of chloral hydrate (alone) and detomidine premedication on temperature, pulse and respiration at 30 and 75 minutes of anaesthesia

Animal No.	Group A Chloral hydrate (alone)						Group B Detomidine premedication with chloral hydrate induced anaesthesia						Group C Detomidine premedication with chloral hydrate at reduced dose rate induced anaesthesia					
	30 min			75 min			30 min			75 min			30 min			75 min		
	*T	*P	R	T	*P	R	*T	*P	R	T	*P	R	*T	*P	R	T	*P	R
1	99.6	39	10	99.8	40	10	99.2	36	10	99.8	38	12	99.0	34	8	100	34	9
2	98.8	39	9	98.8	42	10	98.2	34	8	98.6	38	9	98.2	32	8	98.4	34	10
3	99.4	40	10	100	39	9	98.4	38	8	100	41	10	98.6	34	10	100.2	36	12
4	99.6	39	9	99.8	40	11	98	36	9	98.6	39	9	98.2	34	9	98.4	38	12
5	98	38	8	98.6	38	10	98	34	8	98.4	39	10	98	36	8	98.2	38	10
6	99.2	39	10	99.6	41	10	98.2	36	8	98.8	40	10	98.2	32	8	98.8	36	12
Mean	99.1	39	9.33	99.4	40	10.0	98.3	35.6	8.50	99.0	39.1	10±	98.3	33.6	8.50	99.0±	36	10.8
+SD	±0.6	±0.6	±0.8	±.58	±1.4	±.63	±.45	±1.5	±.83	±.68	±1.1	1.09	±.36	±1.5	±.83	.81	±1.7	±1.3

* indicates significant difference ($P < 0.05$)

Recumbency period. Recumbency period in group A was short with a range of 19-24 min with a mean of 22 ± 1.89 min (Table II). There was incomplete disappearance of reflexes during recumbency period. Palpebral reflex disappeared only in two horses when evaluated at 30 min of the administration of chloral hydrate. Front and hind pedal reflexes were also not affected and all the horses responded with similar behaviour, when painful stimulus was applied on pasterns. Jaw tone disappeared at 30 min after the injection of chloral hydrate, which reappeared right after 15 min. Similarly, tongue protrusion was also observed at 30 min in all horses. Tail reflexes and anal tone disappeared at 30 min in all horses except one in which the anal tone was not affected.

In group B, the recumbency period was long with a range of 58-65 min with a mean of 61.50 ± 2.42 min (Table II). None of the horses showed any signs of pain when painful stimulus was applied. The reflexes disappeared altogether at 30 min and the animal remained in complete surgical anaesthesia up to 60 min after the injection of detomidine. However, the front and hind pedal reflexes were observed at 60 min in four animals. Tongue protrusion and penile relaxation were noticed 15 min after the injection of detomidine and remained in same state up to 60 min with all animals.

In group C the recumbency period was longer than group A. It was with a range of 40-46 min with a mean of 43.16 ± 2.31 min (Table II). Surgical anaesthesia was better than group A. There was no evidence of pain tone in all the animals when tested at 15 min post detomidine injection. The tongue protrusion was also seen in all animals, but the other reflexes were still present at this time. At 30 min all the reflexes disappeared with the exception of palpebral reflex, which was observed in three animals. The anal tone was also present in one horse. Statistical analysis showed significant difference ($P<0.05$) in the recumbency period of three groups (Table II).

Recovery. In this, emphasis was given on nature of recovery and time of recovery of individual animals. In group A, recovery was also not smooth and the animals exhibited unwanted responses. There was staggering in animal No. 1, 3 and 6, shivering in animal No. 2, circling of head in animal No. 4 and kicking in animal No. 5 (Table I). Recovery period was in the range of 70-78 min with a mean of 74.16 ± 3.31 min (Table II). In group B, the recovery was smooth in all cases with exception of animal No. 1, which showed shivering, recovery period was in range of 60-67 min with mean of 63.50 ± 2.42 min (Table II). The animals of group C, also behaved similarly and showed ideal and smooth recovery. None of the animals showed any unwanted response while standing from ground. All were found standing at their first attempt. The recovery period was in range of 51-62 min with a mean of 57.16 ± 4.07 min (Table II). Statistical analysis showed a significant difference ($P < 0.05$) in recovery period between the groups.

Clinical parameters

Temperature. In group A, all the animals showed decrease in body temperature. At 30 min, the mean temperature was 99.10 ± 0.61°F. Later the temperature started increasing in the next 45 min (99.43 ± 0.58°F). It indicated that chloral hydrate alone caused slight hypothermia which improved later on. The animals of group B, in which detomidine pre-medication was used, the temperature decreased in the first 30 min. The mean temperature was 98.33 ± 0.45°F. Later the temperature increased to 99.03 ± 0.68°F. In the next 45 min the animals of group C, also behaved similarly. The temperature was 98.36 ± 0.36°F at 30 min and in the next 45 min increased to 99.0 ± 0.87°F. The hypothermia was noticed in all three groups in the first 30 min which was recovered up to 75 min. The statistical analysis revealed significant differences between the groups at 30 min ($P<0.05$). The results are presented in Table III.

Table IV. Comparative effect of chloral hydrate (alone) and detomidine premedication on liver enzymes

Animal No.	Group A Chloral hydrate (alone)				Group B Detomidine premedication with chloral hydrate induced anaesthesia				Group C Detomidine premedication with chloral hydrate at reduced dose rate induced anaesthesia			
	SAP before IU/lit.	SAP after IU/lit.	SGPT before IU/lit.	SGPT after IU/lit.	SAP before IU/lit.	SAP after IU/lit.	SGPT before IU/lit.	SGPT after IU/lit.	SAP before IU/lit.	SAP after IU/lit.	SGPT before IU/lit.	SGPT after IU/lit.
1	146.3	147	7.4	7.8	148	150	6.2	6.4	146	146.8	7.2	7.8
2	172	175	9.8	10.2	178	178.6	9.8	10.2	174	176.2	8.4	9.2
3	204.2	208.3	22.4	22.6	180.2	182	22.2	22.8	192.2	194.4	14.6	14.8
4	215.4	217	15.7	17.2	210.2	214.6	14	15.2	180.6	182.2	10.4	11.2
5	220.4	222.4	11.3	14.2	215.4	218	19.4	20.2	190	194.2	16.8	17.2
6	189	190.8	19.2	20.2	192	194	11.3	12.2	204.2	207.4	11.2	11.4
Mean	191.22	293.42	14.30	15.37	187.3	189.53	13.82	14.50	181.17	183.53	11.43	11.93
+SD	±28.27	±28.70	±5.8	±5.73	±24.57	±25.29	±6.03	±6.18	±20.09	±21.0	±3.66	±3.50

Normal Range: SAP = 143-395 I.U/lit; SGPT = 2-23 I.U/lit; SD = Standard Deviation

Pulse. In case of chloral hydrate alone, pulse/min of animals decreased in the first 30 min (39±0.63) which increased to 40±1.41 in the next 45 min. In group B, the pulse rate decreased in the first 30 min (35.66±1.50), which later increased in the next 45 min (39.16±1.16). Similarly, in animals of group C, first showed decrease in pulse rate at 30 min (33.66±1.50), which later increased in the next 45 min (36±1.78). There was significant difference between the groups ($P<0.05$) (Table III).

Respiration. In group A, respiration per minute of the animals decreased in the first 30 min (9.37 ± 0.81) then it increased to 10.00 ± 0.63. In group B, in which detomidine was used as pre-medication, respiration per minute decreased initially and it was 8.50 ± 0.83 at 30 min. Which later increased to 10 ± 1.09 in next 45 min. The animals of this group exhibited similar change in respiration as in other groups. At 30 min respiration per minute was 8.50 ± 0.83 which increased later to 10.83 ± 1.32 in the next 45 min. Statistical analysis showed no significant differences between the groups ($P>0.05$) (Table III).

Liver enzymes (LFTs)

Serum alkaline phosphatase (SAP). In group A, the concentration of alkaline phosphatase before and after the experiment was 191.22 ± 28.27 and 193.42 ± 28.70, respectively (Table IV). In group B, the concentration before and after experiment was 187.30 ± 24.57, 189.53 ± 25.29 I.U/lit. While in group C, the concentration before experiment was 181.17 ± 20.09 I.U/lit. which increased to 183.53 ± 21.01 when evaluated after the experiment. Statistically no significant difference was noticed in the change of enzyme activity before and after the experiment in any group indicating that this enzyme remained unaffected ($P>0.05$).

Serum glutamic pyruvic transaminase (SGPT). The enzyme concentration in case of chloral hydrate alone, before experiment was 14.30 ± 5.80 I.U/L and after experiment was 15.37 ± 5.73 I.U/L, while in case of detomidine premedication, the concentration before experiment was 13.82 ± 6.03 and after experiment was 14.50 ± 6.18 I.U/L. In group C, detomidine premedication with reduced chloral hydrate, the concentration before experiment was 11.43 ± 3.66 and after experiment was 11.93 ± 3.50. The observation revealed that there was no significant difference in the enzyme activity before and after the experiment in any group indicating that this enzyme also remained unaffected ($P>0.05$) (Table IV).

Table V. Mean evaluation of sedation under the effect of chloral hydrate (alone) and detomidine premedication

Parameters	Groups	Time (min)				
		0	15	30	45	60
Sedation	A	0	0	2	0	0
	B	0	1	3	3	2
	C	0	1	3	3	0
Analgesia	A	0	0	1	0	0
	B	0	0	3	3	2
	C	0	0	3	2	0

Each value is the mean of 6 animals; 0 = No Sedation / Analgesia; A = Chloral hydrate alone; 1 = Mild Sedation / Analgesia; B = Detomidine premedication with chloral Hydrate; 2 = Moderate Sedation / Analgesia; C = Detomidine premedication with chloral Hydrate at reduced dose rate; 3 = Deep Sedation / Analgesia

Evaluation of sedation. Sedation was evaluated in all groups at an interval of 15 min, just after the administration of drug to 60 min post injection. The sedation was moderate in group A and severe in group B and C when tested at 30 min post injection. Later the sedation decreased gradually and at 60 min it was moderate in group B, and no sedation in group A and C (Table V).

Evaluation of analgesia. The painful stimulus used to evaluate the degree of analgesia in the animals showed slight analgesia in group A, deep analgesia in group B and C at 30 min post injection. In the animals of group B, deep analgesia was seen at 45 min, which decreased gradually onward. At 45 min post injection, moderate analgesia was observed in animals of group C, which was completely abolished afterwards (Table V).

DISCUSSION

Various drugs are used as pre-anaesthetic medication to overcome the problems associated with the use of anaesthetics. Pre-anaesthetic agents are so named because they are usually given to prepare the patient for administration of anaesthetic agent. They are used to reduce the amount of general anaesthetics needed and to increase the margin of safety. Keeping in view these things, this project was designed to solve the problems associated with chloral hydrate to evaluate detomidine pre-medication to overcome the problems associated with chloral hydrate induced anaesthesia and to evaluate the reduction in the dose rate of chloral hydrate in equine.

Different studies in the past have proved the detomidine HCl to be an effective pre-anaesthetic with ideal compatibility with both injectable and inhalant anaesthetics (Short *et al.,* 1984). Detomidine is an alpha-2 agonist drug which is used mainly as a sedative in the horses (Alitalo & Vainio, 1982). The drug is metabolised in the liver and excreted through urine and feces (Salonen & Suolinna, 1988; Stanley *et al.,* 1992; Salonen *et al.,* 1992). Detomidine is used as a sedative and analgesic agent alone and also with other drugs to achieve accumulative effect like ketamine, butorphanol, morphine, thiopentone and carfentanil citrate (Muir *et al.,* 1977). Detomidine can also provide analgesia and sedation for minor work of short duration (Jochle *et al.,* 1991; Klein, 1975). However, recently opiates, which are partial agonists, have gained popularity as they are less liable to abuse (Paton & Clarke, 1986). Of such drugs, buprenophine and butorphanol have been used in combination with detomidine in horses (Robertson & Muir, 1983).

In the present work, chloral hydrate was used in combination with detomidine. It is metabolised to trichloroethanol, which accounts for most of the pharmacological activity. In group A, where chloral hydrate was used alone the induction was not smooth and the animals showed staggering and incoordination with exception of one animal which showed smooth induction. This may be due to selective action of chloral hydrate on brain and spinal cord. The sensory routes may be mildly affected which may result in frightening and struggling (Booth & McDonald, 1988). Extra manpower was also needed to control the animal on the ground. The horses showed severe straining and stretching of limb muscles. Induction time in these animals was 16.66 ± 0.81 min. Recumbency was short with a mean of 22 ± 1.89 min and the reflexes did not disappear completely during recumbency period. Recovery was prolonged with mean time of 74.16 ± 3.31 min and recovery was not very encouraging because all animals exhibited different types of unwanted attempts at this moment. On recovery staggering and shivering were observed. The shivering was due to hypotensive action of the drug. These findings are quite in agreement with the results of Sobti and Mattu (1990), but they used chloral hydrate with thiopental. Moreover, they used this combination for anaesthesia in crossbred calves. One animal showed vigorous kicking and was looking hyper-excited which might be due to increased release of adrenaline in the blood or due to the aggressive temperament of the animal. This finding is in agreement with the results of Shaheen *et al.* (1984) but he used chloral hydrate with magnesium sulphate in buffaloes. A slight decrease in pulse rate, respiration rate and temperature was also observed. This picture was similar to that reported by Karram and Yousaf (1991) who carried out trials of chloral hydrate on donkeys.

Premedication with detomidine HCl in chloral hydrate induced anaesthesia exhibited very smooth induction and non of the animals showed staggering and incoordination. The time of induction was with a mean of 6.83 ± 1.47 min. This has also been reported in the past by Varshney *et al.* (1996). But he used xylazine HCl in his trials. Recumbency period was good with a mean of 63.50 ± 2.42 min and there was complete disappearance of reflexes during recumbency period. As detomidine has a sedative and analgesic effect mediated by stimulation of alpha–2 receptors (Ruksoaho *et al.,* 1983). However in two of the animals front and hind pedal reflexes were seen at 60 min. After the injection of chloral hydrate the animal attained recumbency gradually and smoothly without requiring any assistance. The limbs were almost relaxed. The respiration was slow and in normal rhythm. Recovery was rapid and smooth in all cases except one animal showed shivering. Recovery time was short with a mean of 63.50 ± 2.42 min. On recovery the animals first attained sitting and then standing posture without manual assistance. After getting up from the ground they balanced their bodies and then started walking. A decrease in temperature, pulse and respiration rate was also noticed in this groups. The animals of groups C showed similar results as seen in group B, but the time of induction was slightly longer with a mean of 7.33 ± 1.63 and recumbency period was shorter with a mean of 53.16 ± 2.31 min. The nature of recovery was also smooth with mean recovery time 57.16 ± 4.07 min. A significant difference in temperature and pulse rate was noted at 30 min post injection between the groups. However, at 75 min post injection evaluation revealed significant difference only in pulse rate between the groups.

The liver function test of all groups revealed an increase in the concentration of serum alkaline phosphatase and serum glutamic pyruvic transaminase in the sample collected after the experiment. But the increase did not exceed the normal range, which indicated that the liver remained unaffected. The results clearly indicated that chloral hydrate alone should not be used in horses as it possess several disadvantages. Moreover, the prolong induction time offered practical difficulties in physical restraint before the animals became recumbent. The chloral hydrate like barbiturate lacks analgesic activity at sub-

anaesthetic dose rate (Brander *et al.*, 1991). The study also provided evidence that chloral hydrate at reduced dose rate with detomidine premedication can be used for minor surgical intervention or surgical exercise of short duration.

REFERENCES

Alitalo, I. and O. Vainio, 1982. Detomidine as a sedative and premedicine in large animals. A clinical study in horses and cattle. *In: Proc. 14th Congr. Eur. Vet. Surg.*, Istanbul, pp: 69–75

Anonymous, 1996. Dermosedan (Detomidine hydrochloride). Sedative and analgesic for use in horses only. Orion-Farmos, Espoo, Finland.

Booth, N.H. and L.E. McDonald, 1988. *Veterinary Pharmacology and Therapeutics.* 6th Ed., p. 243. Iowa State University Press, America

Brander, G.C., D.M. Pugh., R.J. Bywater and W.L. Jenkins, 1991. *Veterinary Applied Pharmacology and Therapeutics*, 5th Ed. pp: 469–70. ELBS, Bailliere Tindall

Hall, L.W. and P.M. Clarke, 1991. *Veterinary Anaesthesia.* 8th Ed. Bailliere Tindall. London

Jochle, W., G.L. Woods, T.V. Little, R.B. Hillman and B.A. Ball, 1991. Detomidine hydrochloride versus xylazine plus morphine as sedative and analgesic agents for flank laparotomies and ovary and oviduct removal in standing mares. *Equine Vet. Sci.*, 11: 225–8

Karam, M.H. and M. Yousef, 1991. Clinical observation of chloral hydrate narcosis in donkeys. *Assiut. Vet. Med. J.*, 23: 168–72

Klein, L.V., 1975. Abstracts of 20th World Vet. Congress. 739.

Malone, P.J., 1917. Chloral hydrate as intravenous anaesthesia in horses. *Vet. Rec.*, 29: 531

Muir, W.W., R.T. Skarda and D.W. Miln, 1977. Evaluation of xylazine and ketamine hydrochloride for anaesthesia in horse. *American J. Vet. Res.*, 38: 195–201

Oehme, H.V. and F.L. Prier, 1974. Chloral hydrate as intravenous anaesthetic. Animal anaesthesia colchester: Spottiswoode Ballantyne.

Paton, B.S. and K.W. Clarke, 1986. A preliminary trial of Detomidine / Opiate combination in the Horse. *J. Ass. Vet. Anaesth*., 7: 14–43

Robertson, J.T. and W.W. Muir, 1983. A new analgesic drug combination in horse. *American J. Vet. Res.*, 44: 1667–9

Ruskoaho, H., J.M. Savola, S. Kaipiainen, J. Puurunen and N.T. Karki, 1983. The cardiovascular effects of MPV-295 [4-(5)-2-(2,6_dimethylphenyl) ethylimidaole], a new antihypertensive agent with α2-adrenoceptor agonistic properties. *Naunyn-Schmiedeberg's Arch. Pharmacol*., 322: 279–85

Salonen, J.S. and E.M. Suolinna, 1988. Metabolism of detomidine in the rat. I. Comparison of 3H-labelled metabolites formed *in vitro* and *in vivo*. *European J. Drug Metab. Pharmacokinetics*, 13: 53–8

Salonen, J.S., L. Vuorilehto, M. Giolbert and G.A. Maylin, 1992. Identification of detomidine carboxylic acid as the major urinary metabolite of detomidine in the horse. *European J. Drug. Metab. Pharmacokinet*, 17: 13–20

Shaheen, H.M., I.N. Chaudhry and M. Sharif, 1984. Chloral hydrate and its combination with magnesium sulphate as central anaesthetic in buffaloes. *Pakistan Vet. J.*, 4: 4

Short, C.E., N. Matthews, C.L. Tyner and R. Harvey, 1984. Cardiovascular and Pulmonary function of a new sedative / analgesic (detomidine) for use in horses. pp: 243–50. *Proc. 13th Ann. Conv. Am. Eq. Pract.*, Dallas, Texas, USA.

Sobti, V.K. and S.J. Mattu, 1990. Haemodynamics, acid base status and blood gas tension during chloral hydrate induced thiopental maintained anaesthesia in calves. *Indian Vet. J.*, 67: 1048–52

Stanley, S.D., Y.M. Yang, T.W. Wood, J.S. Salonen, Di Watt, H.H. Tai and T. Tobin, 1992. Detomidine clearance in the horse as determined by ELISA. pp: 193–201. *Proc. 9th Int. Conf. Rac. Ana. Vet.* New Orleaus.

Szeligowski. E., A.M. Janicki and M. Krzeski, 1986. Detomidine (Domosedan®) in sedation, premedication and general anaesthesia of Horses. *Acta. Vet. Scand*., 82: 181–5

Varshney, J.P., P.K. Uppal and M.P. Yadav, 1996. Evaluation of xylazine hydrochloride as sedative in donkeys. *Indian Vet. J*., 73: 24–7

(Received 23 February 2003; Accepted 30 May 2003)