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Effects of Xylazine and Dexmedetomidine on clinico-physiological and haemato-biochemical parameters during total intravenous anaesthesia in horses

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ABSTRACT

The present study evaluated the clinico-physiological and haemato-biochemical parameters in 12 native breed horses, irrespective of age and sex, using two different total intravenous anaesthesia (TIVA) triple drip combinations, viz. xylazine, ketamine and guaifenesin (XKG group); and dexmedetomidine, ketamine, and guaifenesin (DKG group). Sedation in both groups was achieved with acepromazine and butorphanol, with xylazine used in the XKG group and dexmedetomidine in the DKG group, followed by ketamine induction and maintenance with the respective TIVA protocols. During different time intervals in the pre, peri and post-operative periods, both groups were assessed for clinico-physiological, haematological and biochemical parameters. The results showed that horses in the DKG group showed balanced and better cardiopulmonary and haemato-biochemical changes when compared to XKG group.

Keywords: Cardiopulmonary, Dexmedetomidine, Guaifenesin, Haemato-biochemical, Ketamine, Xylazine

Equine anaesthesia involves administering a combination of drugs to achieve the desired effects of sedation, analgesia, muscle relaxation and loss of consciousness in horses (Muir and Hubbell 2009). Horses face higher anaesthetic risk compared to other domestic species, with volatile agents commonly used for longer procedures causing dose-related cardiopulmonary depression (Steffey and Howland 1978). Cardiovascular problems significantly contribute to the high mortality rate in horses (Johnston *et al*. 2002). Balanced anaesthesia ensures better intraoperative cardiopulmonary function, reduces surgical pain and facilitates smoother recoveries (Bettschart-wolfensberger and Larenza 2007). Total intravenous anaesthesia (TIVA) has commonly been used in equine anaesthesia for short term procedures, though under general anaesthesia various clinico-physiological and haemato-biochemical changes are induced. Anaesthesia leads to cardio-pulmonary depression, including decreased heart rate, blood pressure and oxygen haemoglobin saturation in blood, challenging the maintenance of vital parameters. General anaesthesia has an impact on both haematological and biochemical parameters.

The use of dexmedetomidine, a newer alpha-2 agonist, in equine anaesthesia has limited literature available regarding its efficacy and safety compared to xylazine-based TIVA combination. This study aimed to provide valuable insights into the efficacy and pitfalls of using dexmedetomidine in equine anaesthesia, focussing on clinic-physiological and haemato-biochemical parameters. The findings of this study will enhance the understanding of dexmedetomidine role in equine anaesthesia and potentially expand its usage in the field.

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MATERIALS AND METHODS

The study was conducted from 2022 to 2023 at the Department of Veterinary Surgery and Radiology, College of Veterinary Science and Animal Husbandry, Kamdhenu University, Junagadh, Gujarat, India. The study included 12 horses randomly divided into two groups: group I and II with six animals in each group. All the animals underwent preoperative evaluation of clinico-physiological and haematology parameters based on American Society of Anaesthesiologists (ASA) classification (Kalchofner *et al*. 2009) followed to categorize the health status of the horses.

In group I (XKG), acepromazine (0.02 mg/kg), xylazine (0.5 mg/kg) and butorphanol (0.02 mg/kg) were administered intravenously as a premedication, followed by ketamine (2.2 mg/kg) as induction and maintenance of anaesthesia by using TIVA with xylazine (0.5 mg/mL), ketamine (2 mg/mL) and guaifenesin (50 mg/mL) as a CRI at a dose of 1-3 mL/kg intravenously. In group II (DKG), acepromazine (0.02 mg/kg), dexmedetomidine (3.5 µg/kg) and butorphanol (0.02 mg/kg) were given intravenously as a premedication, followed by ketamine (2.2 mg/kg) as induction and maintenance of anaesthesia by using TIVA with dexmedetomidine (2.5 μg/mL), ketamine (2 mg/mL) and guaifenesin (50 mg/mL) as a CRI at a dose of 1-3 mL/kg intravenously. The study assessed various clinico-physiological and haemato-biochemical parameters during anaesthesia. Rectal temperature (RT), heart rate (HR) and respiration rate (RR) were recorded prior to anaesthesia (Base value), after induction (0 min) and at 15 min intervals during anaesthesia. Electrocardiogram (ECG) readings were assessed at specific time intervals. Oxygen haemoglobin saturation in blood (SPO_2) and blood pressure was recorded using a non-invasive vital monitor (Veterinary Monitor, NT3B-V). Blood samples were collected at different intervals, including prior to anaesthesia (base value), after induction (0 min) at 15 min intervals during anaesthesia and post-anaesthesia. Haematological parameters such as haemoglobin, total erythrocyte count, packed cell volume, total leukocyte count, differential leukocyte count and platelet count were assessed using an auto-haemoanalyzer. Biochemical parameters including total protein, albumin, blood urea nitrogen, creatinine, aspartate aminotransferase, alanine transferase, alkaline phosphatase, total bilirubin, and direct bilirubin were analyzed using fully automated analyzer. The blood glucose level was determined in fresh samples with glucometer (Morpen Laboratories Limited, Delhi). The methods involved the use of specific instruments and techniques to measure and monitor these parameters in order to evaluate the effects of anaesthesia on the horses.

The difference between two data sets between groups were statistically analysed by using student 't' test (parameteric data). The difference between more than two data sets within group were statistically analysed by one way ANOVA followed by using Duncan's Multiple Range Test (DMRT) (Snedecor and Cochran 1980).

RESULTS AND DISCUSSION

Physiological observations: In both the groups, there was a significant ($p<0.05$) decrease in heart rate after induction and throughout the surgery, returning to baseline after recovery (Table 1). The observed reduction in heart rate after induction can be attributed to the potent cardiovascular depression caused by the alpha-2 agonists used in both groups. Dexmedetomidine does not have direct effect on cardiac muscles Day and Muir 1993). Dexmedetomidine reduces heart rate more than xylazine in a dose-dependent manner due to physiological reflex bradycardia, without deleterious effects on myocardial oxygen demand or causing hypoxia. Activation of α_1 and α_{2b} receptors produced systemic and peripheral vasoconstriction without altering heart rate much in dexmedetomidine compared to xylazine due to their specificity (Kable *et al*. 2000, Giovannitti *et al*. 2015). On the other hand, ketamine administration produces cardiac stimulation, increasing cardiac output and heart rate (Zielmann *et al*. 1997).

In both the groups, there was a significant ($p<0.05$) decrease in respiration rate after induction, which remained stable with minor changes throughout the surgery (Table 1). The decrease in respiration rate observed between the groups was attributed to the suppressive effects on baroreceptors and adrenergic receptors caused by the administration of xylazine, dexmedetomidine and total intravenous anaesthesia (TIVA) (Yamashita *et al*. 2000, Hopster *et al*. 2014). Sleiman *et al*. (2016) observed a decrease in respiratory rate (RR) during total intravenous anaesthesia (TIVA) in horses undergoing surgery, with the most significant reduction occurring within the first 30 min.

In both the groups rectal temperature decreased non-significantly and returned to base value after recovery (Table 1). The decline in rectal temperature was attributed to the comparatively lower rate of oxygen utilization and basal metabolic rate observed under anaesthesia compared to conscious horses. The reduction in basal metabolism was influenced by the effects of alpha-2 agonists and adjunctive medications, which could interfere with thermoregulatory centres and muscular activity (Lemke *et al.* 1993, Thakur *et al*. 2011, Duke-Novakovski *et al*. 2015).

In both the groups SpO_2 significantly ($p<0.05$) decreased after induction, reaching as low as 85%, but quickly returned to normal range 90-100% within few minutes and maintained within this range throughout the surgery (Table 1). DKG group showed lower values than xylazine due to least vascular resistance, decrease in the saturated partial pressure of oxygen, which could be attributed to the respiratory depression caused by the alpha-2 agonist and acepromazine. This decrease in oxygen saturation resulted in a ventilation-perfusion mismatch (Thakur *et al*. 2011). Furthermore, the administration of ketamine further contributed to the reduction in saturated partial pressure of oxygen due to its direct relaxant effect on bronchial smooth muscle causes hypoventilation (Muir *et al*. 2000).

In both the groups, non-significant changes were observed in systolic arterial blood pressure (mmHg), diastolic arterial blood pressure (mmHg) and mean blood pressure (mmHg) which remained relatively stable throughout different time intervals, showing only minor fluctuation (Table 1). These results suggest that both the groups maintained hemodynamic stability throughout the anaesthetic period. The administration of dexmedetomidine in group DKG resulted in stable systolic and mean blood pressure levels during surgical anaesthesia, without significant variations. Compared to xylazine in group XKG, dexmedetomidine exhibited slightly better control over blood pressure, reducing myocardial oxygen demand and maintaining vital blood flow without increasing the preload on the heart (Lawrence *et al*. 1996, Giovannitti *et al*. 2015). Similar to these findings Hopster *et al*. (2014) reported no significant difference between xylazine and dexmedetomidine groups while using TIVA in horses.

In the ECG, there were no significant changes noticed on between groups. DKG group showed increased PR interval, QT interval in some cases due to potent cardiovascular depression effect of dexmedetomidine and acepromazine causing reduced sympathetic outflow and vasodilation. In XKG group both bradycardia and tachycardia ECG

Table 1. Mean±SE values of physiological parameters in group XKG and DKG

Variable	Group	Base value	0 min	15 min	30 min	45 min	60 min	After recovery
HR (Beats/min)	XKG	51.00 ± 2.35^{bA}	37.17 ± 2.07^{aA}	36.33 ± 2.48^{aA}	37.83 ± 3.97^{aA}	36.80 ± 4.21^{aA}	37.00 ± 2.51^{aA}	48.00 ± 2.58^{bA}
	DKG	49.66 ± 1.81^{bA}	29.83 ± 2.82^{aB}	29.17 ± 2.56^{aA}	32.16 ± 2.28^{aA}	34.00 ± 5.03^{aA}	32.50 ± 6.50^{aA}	45.67 ± 0.95^{bA}
RR (Breaths/min)	XKG	20.16 ± 1.60^{bA}	8.00 ± 0.73^{aA}	7.66 ± 1.05^{aA}	8.33 ± 1.33^{aA}	7.40 ± 0.92^{aA}	8.00 ± 1.15^{bA}	17.16 ± 1.04^{bA}
	DKG	20.50 ± 2.39^{bA}	8.83 ± 0.54^{aA}	6.83 ± 0.54^{aA}	6.33 ± 0.55^{aA}	6.66 ± 0.33^{aA}	7.50 ± 1.50^{aA}	16.66 ± 0.80^{bA}
RT (°F)	XKG	100.7 ± 0.17^{aA}	100.48 ± 0.20^{aA}	100.10 ± 0.42^{aA}	100.38 ± 0.18^{aA}	100.30 ± 0.13^{aA}	99.87 ± 0.31^{aA}	100.03 ± 0.17^{aA}
	DKG	99.96 ± 0.20^{bB}	99.48 ± 0.31^{abB}	98.55 ± 0.40^{abB}	98.03 ± 0.49^{aB}	98.33 ± 1.18^{abA}	97.45 ± 1.45^{abA}	99.46 ± 0.09^{abA}
SpO_2 (%)	XKG	$97.16. \pm 0.40^{bcA}$	93.83 ± 1.77^{abA}	93.33 ± 1.25^{aA}	93.33 ± 1.08^{aA}	94.00 ± 1.04^{abA}	94.66 ± 0.33^{abcA}	98.00 ± 0.25^{bcA}
	DKG	98.33 ± 0.40^{dA}	94.33 ± 0.55^{bcA}	93.00 ± 1.03^{abA}	91.83 ± 0.65^{aA}	93.80 ± 0.73^{abcA}	95.33 ± 0.33^{cA}	98.00 ± 0.36^{dA}
SAP (mmHg)	XKG	133.67 ± 5.17^{aA}	133.66 ± 9.15^{aA}	128.33 ± 9.28^{aA}	$125. \pm 11.57^{aA}$	124.8 ± 17.95^{aA}	151.66 ± 33.73^{aA}	142 ± 1.91^{aA}
	DKG	118.33 ± 3.08^{aA}	126 ± 3.33^{abA}	120.83 ± 5.67^{aA}	119.83 ± 8.53^{aA}	130.67 ± 6.98^{abA}	137.50 ± 2.50^{abA}	144 ± 3.33^{bA}
DAP (mmHg)	XKG	86 ± 3.73^{aA}	87.33 ± 12.74^{aA}	88.66 ± 10.95^{aA}	85.66 ± 11.96^{aA}	80.4 ± 15.03^{aA}	99.33 ± 30.22^{aA}	91 ± 1.12^{aA}
	DKG	80.00 ± 1.73^{abA}	75.83 ± 2.66^{aA}	76.33 ± 2.62^{aA}	74.66 ± 5.39^{aA}	77.33 ± 4.5^{aA}	81.00 ± 3.00^{abA}	91.66 ± 2.50^{bA}
MAP (mmHg)	XKG	98.5 ± 3.09^{aA}	100.33 ± 12.07^{aA}	101.67 ± 10.16^{aA}	97.83 ± 11.83^{aA}	95.6 ± 15.89^{aA}	118.66 ± 30.68^{aA}	101.67 ± 1.86^{aA}
	DKG	93.00 ± 1.98^{aA}	93.67 ± 2.85^{aA}	91.17 ± 3.77^{aA}	92.33 ± 5.18^{aa}	94 ± 6.55^{aA}	100.00 ± 3.00^{aA}	103.00 ± 2.32^{aA}

Means bearing different small alphabet (a, b, c, d) superscript indicates significant difference ($p<0.05$) value within group at different time intervals. Means bearing different capital alphabet (A, B) superscript indicates significant difference ($p<0.05$) value between group at corresponding time intervals.

changes were observed, reduced PR interval and QRS duration during maintenance due to prolonged hypotension caused by xylazine. No major abnormalities were thus noticed in ECG findings.

Haematological parameters: The mean±SE values of various haematological parameters are listed in Table 2. Haemoglobin, total erythrocyte count and packed cell volume showed decreased pattern changes, there was no statistical significance between and within groups. Hb, TEC, PCV returned to baseline or near to baseline values after recovery. Kullman *et al*. (2014) observed decrease in haemoglobin count when xylazine was given along with guaifenesin and ketamine for maintenance in horses. The administration of drugs like acepromazine and alpha-2 agonists can lead to changes in total erythrocyte count (TEC) and packed cell volume (PCV) due to the dilation of splenic vessels leading into sequestration of blood cells in the spleen (Ballard *et al*. 1982, Kullaman *et al*. 2014 and Nanda *et al*. 2014). This phenomenon may result in alterations in blood cell counts and volume, potentially impacting overall haematology and circulation.

In group XKG, during surgical anaesthesia, there was a non-significant decrease in total leukocyte count after induction, followed by a gradual decreasing pattern throughout the maintenance of anaesthesia. Neutrophil, monocyte, and basophil counts showed non-significant decrease after induction, with no definite pattern during the surgical period. Lymphocyte counts exhibited a non-significant increase after induction, but no specific trend was observed at different intervals. Overall, no significant variations were observed within or between groups, indicating a stable immune response during the surgical procedure. In Group DKG, there were no significant variations in total leukocyte count, neutrophil count, lymphocyte count, monocyte count or basophil count within the group. Similar to Group XKG, the immune response remained relatively stable during the surgical period. Significant differences were seen in the eosinophil count when compared to Group XKG, but these differences remained within the normal range, therefore they had no clinical significance. Both groups exhibited no major changes in immune response during surgical anaesthesia. Similar findings have been reported by Kaur (2018) and Deepesh (2019).

In both XKG and DKG groups, there was non-significant decrease of platelet count during surgical period. However, it showed thrombocytopenia in animals even after recovery, with the platelet count remaining low compared to the preoperative levels. This decrease in platelet count could be attributed to the sequestration of blood in the spleen due to the administration of acepromazine and the effect of alpha-2 agonists on platelet reduction in horses (Meagher and Tasker 1972, Gathyus *et al*. 1990). Similar results were reported by Agivale *et al*. (2019) who also observed platelet reduction in horses.

Biochemical parameters: The mean±SE values of various biochemical parameters are listed in Table 3. In

Table 2. Mean±SE values of haematological parameters in group XKG and DKG

Variable	Group	Base value	0 min	15 min	30 min	45 min	60 min	After recovery
TEC ($\times 10^6/\mu L$)	XKG	7.66 ± 0.87^{aA}	6.66 ± 0.62^{aA}	6.55 ± 0.51^{aA}	6.11 ± 0.49^{aA}	6.35 ± 0.45^{aA}	7.14 ± 0.65^{aA}	6.55 ± 0.87^{aA}
	DKG	8.08 ± 0.29^{aA}	7.44 ± 0.21^{aA}	6.99 ± 0.20^{aA}	6.90 ± 0.19^{aA}	6.68 ± 0.28^{aA}	7.00 ± 0.07^{aA}	8.07 ± 0.67^{aA}
Hb (g/dL)	XKG	12.44 ± 1.45^{bA}	10.13 ± 0.75^{abA}	9.78 ± 0.54^{abA}	8.82 ± 0.55^{aA}	9.36 ± 0.49^{abA}	10.33 ± 0.57^{abA}	9.80 ± 1.28^{abA}
	DKG	10.66 ± 0.37^{cA}	9.80 ± 0.34^{bA}	9.15 ± 0.29^{abA}	8.86 ± 0.42^{abA}	8.57 ± 0.70^{abA}	8.04 ± 0.96^{aA}	9.22 ± 0.30^{abcA}
PCV (%)	XKG	39.82 ± 3.55^{aA}	35.07 ± 2.57^{aA}	34.44 ± 1.97^{aA}	32.21 ± 1.99^{aA}	33.01 ± 1.72^{aA}	36.39 ± 1.83^{aA}	34.45 ± 4.62^{aA}
	DKG	37.22 ± 1.71^{abA}	34.22 ± 1.39^{abA}	31.39 ± 1.34^{aA}	31.22 ± 1.34^{aA}	31.22 ± 1.17^{aA}	29.69 ± 1.52^{aA}	33.68 ± 1.37^{abA}
TLC ($\times 10^3/\mu L$)	XKG	11.35 ± 1.00^{aA}	10.37 ± 1.18^{aA}	9.56 ± 1.22^{aA}	9.30 ± 1.19^{aA}	7.92 ± 0.29^{aA}	7.57 ± 0.72^{aA}	9.96 ± 1.77^{aA}
	DKG	8.93 ± 0.90^{aA}	8.12 ± 0.63^{aA}	7.67 ± 0.63^{aA}	7.48 ± 0.63^{aA}	7.72 ± 1.12^{aA}	8.29 ± 1.36^{aA}	8.75 ± 0.86^{aA}
Neutrophils (%)	XKG	69.30 ± 5.49^{aA}	62.92 ± 4.68^{aA}	66.68 ± 4.58^{aA}	68.42 ± 5.08^{aA}	64.12 ± 5.40^{aA}	67.70 ± 10.65^{aA}	71.63 ± 4.79^{aA}
	DKG	61.87 ± 5.52^{aA}	59.68 ± 5.39^{aA}	61.30 ± 2.52^{aA}	59.15 ± 3.59^{aA}	56.50 ± 3.03^{aA}	59.20 ± 7.80^{aA}	58.27 ± 6.95^{aA}
Lymphocytes (%)	XKG	23.70 ± 4.87^{aA}	27.47 ± 4.73^{aA}	23.65 ± 4.25^{aA}	23.05 ± 4.97^{aA}	27.88 ± 5.37^{aA}	24.10 ± 9.15^{aA}	21.22 ± 5.24^{aA}
	DKG	25.97 ± 6.56^{aA}	27.61 ± 6.68^{aA}	27.58 ± 5.72^{aA}	28.76 ± 6.31^{aA}	27.04 ± 12.57^{aA}	37.85 ± 13.55^{aA}	27.23 ± 7.20^{aA}
Monocytes (%)	XKG	6.33 ± 1.31^{aA}	9.02 ± 0.29^{aA}	9.03 ± 0.50^{aA}	8.32 ± 0.85^{aA}	7.32 ± 0.91^{aA}	8.00 ± 1.57^{aA}	6.33 ± 1.45^{aA}
	DKG	6.57 ± 0.92^{aA}	7.00 ± 0.98^{aA}	6.10 ± 1.44^{aA}	6.62 ± 1.17^{aA}	5.47 ± 3.04^{aA}	5.70 ± 2.60^{aA}	6.00 ± 1.18^{aA}
Basophils (%)	XKG	0.18 ± 0.08^{aA}	0.10 ± 0.05^{aA}	0.12 ± 0.04^{aA}	0.15 ± 0.06^{aA}	0.06 ± 0.04^{aA}	0.13 ± 0.09^{aA}	0.17 ± 0.07^{aA}
	DKG	0.73 ± 0.33^{aA}	0.37 ± 0.09^{aA}	0.30 ± 0.08^{aA}	0.30 ± 0.10^{aA}	0.23 ± 0.12^{aA}	0.20 ± 0.10^{aA}	0.25 ± 0.08^{aA}
Eosinophils (%)	XKG	0.48 ± 0.19^{aA}	0.47 ± 0.15^{aA}	0.53 ± 0.20^{aA}	0.58 ± 0.24^{aA}	0.42 ± 0.15^{aA}	0.30 ± 0.10^{aA}	0.45 ± 0.18^{aA}
	DKG	1.78 ± 0.41^{aA}	1.72 ± 0.31^{aA}	1.53 ± 0.30^{aA}	1.53 ± 0.30^{aA}	1.17 ± 0.48^{aA}	1.00 ± 0.70^{aA}	1.55 ± 0.31^{aA}
Platelets ($\times 10^3$ μL)	XKG	206.67 ± 16.00^{bA}	179.00 ± 21.42^{abA}	162.00 ± 18.17^{abA}	154.17 ± 19.00^{abA}	153.60 ± 20.83^{abA}	137.33 ± 27.67^{aA}	174.67 ± 8.34^{abA}
	DKG	208.50 ± 27.52^{bA}	183.17 ± 15.91^{aA}	186.83 ± 22.01^{aA}	179.33 ± 23.18^{aA}	158.00 ± 15.01^{aA}	141.00 ± 11.00^{aA}	172.83 ± 14.06^{aA}

Means bearing different small alphabet (a, b, c) superscript indicates significant difference ($p<0.05$) value within group at different time intervals. Means bearing different capital alphabet (A, B) superscript indicates significant difference ($p<0.05$) value between group at corresponding time intervals.

both the groups XKG and DKG, significant ($p<0.05$) variations in blood glucose levels were observed during the surgical anaesthesia period. Blood glucose levels significantly increased upon induction and were elevated over the duration of anaesthesia maintenance. After recovery, there was a significant decrease in blood glucose levels from elevated values in both groups. This decrease was attributed to temporary insulin resistance induced by the administration of the alpha-2 agonists xylazine and dexmedetomidine. However, as the effects of alpha-2 agonist wore off during the post-recovery period, blood glucose levels returned to near base values. This transient hyperglycemia led to increased urine output without the presence of glucosuria or any adverse effects. The present study found that xylazine induced higher levels of hyperglycemia compared to the dexmedetomidine group, which is consistent with previous studies (England and Clarke 1996, Daunt and Steffey 2002, Ambrosio *et al*. 2012).

In both the groups, XKG and DKG, non-significant decrease of total protein and albumin up to 60 min after increase pattern was observed and it returned to base values without major alterations. The decrease pattern can be attributed to the haemodilution caused by the administration of fluids during the surgery. Additionally, as reported by Wagner *et al*. (1991), the shift of fluid from the extravascular compartment to the intravascular compartment to compensate the hypotension for increasing the cardiac output may contribute to a decrease in total serum proteins and albumin. Similar findings were reported by Kullaman *et al*. (2014) and Deepesh (2019).

In both the groups XKG and DKG, significant ($p<0.05$) variations in BUN levels were observed between the groups, with Group II exhibiting higher values compared to Group I. However, these changes were not clinically significant and could be attributed to individual variations and age differences. Within both groups, non-significant decreases in BUN levels were observed. In contrast, no significant variations in serum creatinine levels were found between or within the groups, indicating stable renal function throughout the surgical period. Kinjavdekar *et al*. (2000) and Fani *et al*. (2008) reported that the anaesthesia temporarily inhibited renal

Table 3. Mean±SE values of biochemical parameters in group XKG and DKG

Parmeter	Group	Base value	0 min	15 min	30 min	45 min	60 min	After recovery
Blood glucose (mg/dL)	XKG	128 ± 7.47^{aA}	150 .83 ± 13.17^{abA}	204 ± 19.84^{bcA}	245.83 ± 22.38^{cA}	269.40 ± 25.37^{dA}	253.75 ± 27.10^{cA}	224.50 ± 19.61^{cA}
	DKG	108 ± 4.07^{aB}	122 ± 7.32^{abA}	145.33 ± 8.09^{bcB}	164.50 ± 10.52^{cdB}	191.67 ± 12.03^{dA}	198 ± 27^{dA}	154.17 ± 13.75^{bcB}
Total protein (g/dL)	XKG	6.08 ± 0.56^{aA}	5.94 ± 0.84^{aA}	5.50 ± 0.78^{aA}	5.74 ± 0.78^{aA}	5.31 ± 0.78^{aA}	6.66 ± 0.23^{aA}	5.89 ± 0.24^{aA}
	DKG	7.17 ± 0.50^{aA}	6.61 ± 0.52^{aA}	6.50 ± 0.28^{aA}	5.81 ± 0.40^{aA}	5.44 ± 0.89^{aA}	5.33 ± 1.65^{aA}	5.91 ± 0.42^{aA}
Albumin (g/dL)	XKG	3.51 ± 0.14^{aA}	3.32 ± 0.31^{aA}	3.22 ± 0.30^{aA}	3.27 ± 0.23^{aA}	3.12 ± 0.28^{aA}	3.47 ± 0.10^{aA}	3.06 ± 0.16^{aA}
	DKG	3.92 ± 0.29^{bA}	3.56 ± 0.22^{abA}	3.54 ± 0.10^{abA}	3.19 ± 0.21^{abA}	2.99 ± 0.35^{aA}	2.84 ± 0.77^{aA}	3.20 ± 0.18^{abA}
AST (IU/L)	XKG	130.74 ± 4.50^{aA}	142.85 ± 11.76^{aA}	147.50 ± 14.04^{aA}	144.46 ± 6.80^{aA}	161 ±6.36^{aA}	140.67 ± 10.37^{aA}	129.69 ± 17.02^{aA}
	DKG	120.51 ± 7.72^{aA}	114.63 ± 12.93^{aA}	111.17 ± 8.83^{aA}	115.26 ± 10.29^{aA}	135.81 ± 29.95^{aA}	120.62 ± 17.02^{aA}	113.80 ± 18.33^{aA}
ALT (IU/L)	XKG	9.13 ± 1.14^{aA}	9.55 ± 1.28^{aA}	8.84 ± 1.60^{aA}	7.47 ± 0.81^{aA}	7.69 ± 0.82^{aA}	10.76 ± 2.81^{aA}	8.00 ± 1.32^{aA}
	DKG	10.83 ± 0.67^{aA}	10.47 ± 0.98^{aA}	9.88 ± 1.33^{aA}	9.88 ± 1.19^{aA}	10.35 ± 3.03^{aA}	7.43 ± 0.33^{aA}	8.65 ± 1.24^{aA}
ALP (IU/L)	XKG	146.90 ± 11.49^{aA}	128.33 ± 14.40^{aA}	127.65 ± 11.85^{aA}	128.52 ± 11.03^{aA}	114.46 ± 10.73^{aA}	129.73 ± 7.08^{aA}	119.17 ± 6.02^{aA}
	DKG	136.57 ± 26.96^{aA}	117.25 ± 12.64^{aA}	117.83 ± 15.57^{aA}	112.05 ± 21.02^{aA}	100.87 ± 38.31^{aA}	107.45 ± 67.75^{aA}	115.08 ± 23.15^{aA}
Total Bilirubin (mg/dL)	XKG	3.10 ± 0.26^{aA}	2.83 ± 0.29^{aA}	2.90 ± 0.22^{aA}	2.80 ± 0.23^{aA}	3.02 ± 0.19^{aA}	3.07 ± 0.36^{aA}	3.17 ± 0.21^{aA}
	DKG	2.39 ± 0.50^{aA}	2.24 ± 0.43^{aA}	2.15 ± 0.35^{aA}	1.57 ± 0.36^{aB}	2.16 ± 0.59^{aA}	2.44 ± 0.63^{aA}	1.99 ± 0.41^{aB}
Direct Bilirubin (mg/dL)	XKG	0.41 ± 0.05^{aA}	0.28 ± 0.07^{aA}	0.36 ± 0.15^{aA}	0.27 ± 0.12^{aA}	0.29 ± 0.12^{aA}	0.30 ± 0.15^{aA}	0.29 ± 0.04^{aA}
	DKG	0.24 ± 0.03^{aB}	0.22 ± 0.04^{aA}	0.19 ± 0.05^{aA}	0.29 ± 0.12^{aA}	0.29 ± 0.09^{aA}	0.17 ± 0.07^{aA}	0.13 ± 0.04^{aB}
BUN (mg/dL)	XKG	30.42 ± 6.16^{aA}	30.91 ± 5.59^{aA}	30.08 ± 4.50^{aA}	31.50 ± 6.06^{aA}	26.36 ± 4.66^{aA}	36.98 ± 2.63^{aA}	29.15 ± 5.13^{aA}
	DKG	50.51 ± 3.77^{aB}	48.32 ± 4.02^{aB}	48.86 ± 3.65^{aB}	44.65 ± 3.93^{aA}	37. 15 ± 4.61^{aA}	37.64 ± 1.98^{aA}	43.92 ± 3.13^{aB}
Creatinine (mg/dL)	XKG	1.43 ± 0.15^{aA}	1.49 ± 0.23^{aA}	1.39 ± 0.15^{aA}	1.57 ± 0.18^{aA}	1.51 ± 0.26^{aA}	1.66 ± 0.38^{aA}	1.56 ± 0.13^{aA}
	DKG	1.77 ± 0.27^{aA}	1.69 ± 0.12^{aA}	1.65 ± 0.14^{aA}	1.63 ± 0.23^{aA}	1.52 ± 0.24^{aA}	1.45 ± 0.54^{aA}	1.71 ± 0.20^{aA}

Means bearing different small alphabet (a, b, c, d) superscript indicates significant difference ($p<0.05$) value within group at different time intervals. Means bearing different capital alphabet (A, B) superscript indicates significant difference ($p<0.05$) value between group at corresponding time interval

blood flow which raised the plasma urea nitrogen level but adequate surgical fluid administration improved renal function by increasing cardiac output.

Both the groups demonstrated that liver enzymes AST (aspartate aminotransferase), ALT (alanine aminotransferase) and ALP (alkaline phosphatase) remained within the normal range during anaesthetic period, showing no significant changes, with only minor fluctuations within the normal limits. Similar findings were reported by Thakur *et al*. (2011), Agivale *et al*. (2019) and Deepesh (2019).

Both the groups showed higher levels of total bilirubin during the preoperative period, which remained stable with minor fluctuations throughout the surgical period. The increase in total bilirubin levels above the normal range, was attributed to the longer fasting time than recommended fasting time, which led to a decrease in the elimination of unconjugated bilirubin from the blood, resulting in hyperbilirubinemia leads to hyperlipidemia and hepatic lipidosis (Gronwall and Mia 1972). In both groups XKG and DKG, no significant variations were observed in direct bilirubin levels during the anaesthesia, only minor fluctuations within the normal range. Similar findings were reported by Chaturdevi (2005).

In both the groups, the maintenance of heart rate and blood pressure within the desired anaesthetic range ensured cardiac and pulmonary stability during total intravenous anaesthesia (TIVA) in horses, without any adverse effects. Both haematological and biochemical changes remained within acceptable limits, indicating that no significant abnormalities were observed. Notably, the DKG group demonstrated slightly better results compared to the XKG group, suggesting that the newer dexmedetomidine drug may offer advantages in multiple aspects. However, further studies are necessary to fully explore the potential applications and benefits of dexmedetomidine in horses.

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REFERENCES

Agivale S M, Khandekar G S, Tripathi S D and Sawandkar P G. 2019. Clinical evaluation of xylazine, ketamine and guaifenesin as total intravenous anaesthesia in equines. *Advances in Animal and Veterinary Sciences* **7**(4): 265–71.

Ambrosio A M, Casaes A G, Ida K K, Souto M T M R, Zechetto L D S, Furtado P V and Fantoni D T. 2012. Differences in blood glucose increase between horses receiving xylazine and detomidine for surgical and non-surgical clinical procedures. *Brazilian Journal of Veterinary Research and Animal Science* **49**(6): 493–99.

Ballard S, Shults T, Kownacki A A, Blake J W and Tobin T. 1982. The pharmacokinetics, pharmacological responses and behavioral effects of acepromazine in the horse. *Journal of Veterinary Pharmacology and Therapeutics* **5**(1): 21–31.

Bettschart-Wolfensberger R and Larenza M P. 2007. Balanced anesthesia in the equine. *Clinical Techniques in Equine Practice* **6**(2): 104–10.

Chaturvedi V. 2005. 'Comparative study on evaluation of diazepam and midazolam prior to xylazine – ketamine anaesthesia for gelding in equine.' M. V. Sc Thesis, Maharashtra animal and fishery science University, Nagpur, India.

Daunt D A and Steffey E P. 2002. Alpha-2 adrenergic agonists as analgesics in horses. *Veterinary Clinics: Equine Practice* **18**(1): 39–46.

Day T K and Muir W W. 1993. Alpha 2-adrenergic receptor agonist effects on supraventicular and venticular automacity in dogs with complete atrioventricular block. *American Journal of Veterinary Research* **54**(1): 136–41.

Deepesh K. 2019. 'Comparative evaluation of total intravenous anaesthesia vis a vis inhalation anaesthesia in equine.' M.V.Sc Thesis, Guru Angad Dev Veterinary and Animal Sciences University, Ludhiana, India.

Duke-Novakovski T, Palacios-Jimenez C, Wetzel T, Rymes L and Sanchez-Teran A F. 2015. Cardiopulmonary effects of dexmedetomidine and ketamine infusions with either propofol infusion or isoflurane for anesthesia in horses. *Veterinary Anaesthesia and Analgesia* **42**(1): 39–49.

England G C and Clarke K W. 1996. Alpha 2 adrenoceptor agonists in the horse–a review. *Bristish Veterinary Journal* **152**(6): 641–57.

Fani F A, Mehesare S P, Pawshe D B, Khan K M, and Jadhav D. 2008. Haematological and biochemical changes during epidural xylazine hydrochloride anaesthesia in dogs. *Vet World* **1**(6): 175-7.

Gasthuys F, De Moor A and Parmentier D. 1990. Haemodynamic changes during sedation in ponies. *Veterinary Research Communications* **14**: 309–27.

Giovannitti J A, Jr Thoms S M and Crawford J J. 2015. Alpha-2 adrenergic receptor agonists: a review of current clinical applications. *Anesthesia Progress* **62**(1): 31–39.

Gronwall R and Mia A S. 1972. Fasting hyperbilirubinemia in horses. *The American Journal of Digestive Diseases* **17**(5): 473–76.

Hopster K, Muller C, Hopster-Iversen C, Stahl J, Rohn K and Kastner S. 2014. Effects of dexmedetomidine and xylazine on cardiovascular function during total intravenous anaesthesia with midazolam and ketamine and recovery quality and duration in horses. *Veterinary Anaesthesia and Analgesia* **41**(1): 25–35.

Johnston G M, Eastment J K, Wood J L N and Taylor P M. 2002. The confidential enquiry into perioperative equine fatalities (CEPEF): Mortality results of Phases 1 and 2. *Veterinary Anaesthesia and Analgesia* **29**(4): 159–70.

Kable J W, Murrin L C and Bylund D B. 2000. *In vivo* gene modification elucidates subtype-specific functions of α2-adrenergic receptors. *Journal of Pharmacology and Experimental Therapeutics* **293**(1): 1–7.

Kalchofner K S, Picek S, Ringer S K, Jackson M, Hassig M and Bettschart-Wolfensberger R. 2009. A study of cardiovascular function under controlled and spontaneous ventilation in isoflurane–medetomidine anaesthetized horses. *Veterinary Anaesthesia and Analgesia* **36**(5): 426–35.

Kaur A. 2018. 'Evaluation of midazolam and guaifenesin as preanaesthetic agents for general anaesthesia in equine.' M. V. Sc Thesis, Guru Angad Dev Veterinary and Animal Sciences University, Ludhiana, India.

Kinjavdekar P, Amarpal G S, Aithal H and Pawde A M. 2000. Physiologic and biochemical effects of subarachnoidally administered xylazine and medetomidine in goats. *Small Ruminant Research* **38**(3): 217–28.

Kullmann A, Sanz M, Fosgate G T, Saulez M N, Page P C and Rioja E. 2014. Effects of xylazine, romifidine, or detomidine on hematology, biochemistry, and splenic thickness in healthy horses. *The Canadian Veterinary Journal* **55**(4): 334.

Lawrence C J, Prinzen F W and De Lange S. 1996. The effect of dexmedetomidine on the balance of myocardial energy requirement and oxygen supply and demand. *Anesthesia & Analgesia* **82**(3): 544–50.

Lemke K A, Tranquilli W J, Thurmon J C, Benson G J and Olson W A. 1993. Hemodynamic Effects of atropine and glycopyrrolate in isoflurane-xylazine-anesthetlzed dogs. *Veterinary Surgery* **22**(3): 163–69.

Meagher D M and Tasker J B. 1972. Effects of excitement and tranquilization on the equine hemogram. *Modern Veterinary Practice* **53**(3): 41–43.

Muir W W and Hubbell J A E. 2009. Intravenous anesthetic and analgesic adjuncts to inhalation anesthesia. *Equine Anesthesia Monitoring and Emergency Therapy*. 2nd edition. Pp 260–276. Saunders, Elsevier.

Muir W W, Lerche P, Robertson J T, Hubbell J A, Beard W, Miller T and Bothwell V. 2000. Comparison of four drug combinations for total intravenous anesthesia of horses undergoing surgical removal of an abdominal testis. *Journal of the American Veterinary Medical Association* **217**(6): 869–73.

Nanda V S, Kumar A, Kanwar M S, Sharma A, Sharma S K and Khurana A. 2014. Continuous maintenance anaesthesia using guaifenesin or diazepam combined with xylazine and ketamine in horses. *Indian Journal of Veterinary Surgery* **35**(2): 89–92.

Sleiman M, Diner D and Simeonora G. 2016. The effects of dexmedetomidine continuous rate infusion (cri) on isoflurane anesthesia in healthy horses. *International Journal of Clinical Medicine* **7**(6): 371–80.

Snedecor G W and Cochran W G. 1980. *Statistical methods*. 9th edn. Iowa state university Press.

Steffey E P and Howland D E. 1978. Cardiovascular effects of halothane in the horse. *American Journal of Veterinary Research* **39**(4): 611–15.

Thakur B P S, Sharma S K, Sharma A and Kumar A. 2011. Clinical evaluation of xylazine-butorphanol-guaifenesin-ketamine as short-term TIVA in 18 equines. *Veterinary Medicine International* **6**.

Wagner A E, Muir W W 3rd and Hinchcliff K. 1991. Cardiovascular effects of xylazine and detomidine in horses. *American Journal of Veterinary Research* **52**(5): 651–57.

Yamshika K, Satoh M, Umikawa A, Tsuda A, Yajima Y, Tsubakishita S, Seno T, Katoh S, Izumisawa Y and Kotani T. 2000. Combination of continuous intravenous infusion using a mixture of guaifenesin-ketamine-medetomidine and sevoflurane anesthesia in horses. *The Journal of Veterinary Medical Science* **62**(3): 229–35.

Zielmann S, Kazmaier S, Schnull S and Weyland A. 1997. S-(+)-Ketamine and circulation. *Der Anaesthesist* **46**: 43–6.