

## Research Article

### Dexmedetomidine with Butorphanol and Propofol for Total Intravenous Anaesthesia in Uraemic Buffalo Calves

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#### ABSTRACT

Sixteen clinical cases of urolithiasis in male buffalo calves of 4–6 months of age and 40–55 kg of weight were used in groups A and B. Group A animals were premedicated with dexmedetomidine (2.5 µg/kg body wt, IV), while in group B dexmedetomidine (2.5 µg/kg body wt, IV) and butorphanol (0.05 mg/kg body wt, IV) were used for premedication. Anaesthesia was induced by 1 % propofol in group A (2.50 ± 0.37 mg/kg) and B (1.99±0.29 mg/kg), and maintained by 1 % propofol with continuous intravenous infusion. A lower dose of propofol for induction and maintenance was required in group B than in group A, whereas recovery time, sternal recumbency time, standing time and duration of anaesthesia were similar in both groups. In both groups, HR and RR decreased significantly (P<0.01) up to 75 min. There was a gradual increase in RR after induction of anaesthesia in both groups, and a significantly higher RR was recorded from 45 to 75 min in group A. Rectal temperature decreased significantly in both groups up to 90 min. Haemoglobin, PCV and TLC decreased in both groups. Significant neutrophilia was recorded in group B and lymphocytopenia in both groups. Urea nitrogen, creatinine and glucose increased in both groups. MAP decreased significantly (P<0.05) after premedication up to the end of observation period in both groups. Decrease in SpO<sub>2</sub> after premedication was recorded in both groups. All the animal of both groups recovered without any complications. It was concluded that dexmedetomidine–propofol combination can be used safely for total intravenous anaesthesia in uraemic buffalo calves, however, addition of butorphanol has a dose sparing effect on propofol induction and maintenance anaesthesia in uraemic buffalo calves.

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#### INTRODUCTION

Urolithiasis is a common surgical condition of almost all domestic animals but higher incidence has been recorded in bovine and caprine (Radostits *et al.*, 1994). Several anaesthetic techniques using different drugs have been reported for the management of urolithiasis. Epidural lignocaine hydrochloride (Kinjavdekar *et al.*, 2005), bupivacaine (Singh *et al.*, 2007a), ropivacaine (Singh *et al.*, 2005) and xylazine and ketamine (Singh *et al.*, 2007b) have been used for the anaesthetic management of goats suffering from urolithiasis. General anaesthesia may be preferred over local anaesthesia for any surgical intervention as it provides complete unconsciousness, better insensitivity to pain, good muscle relaxation, and freedom from reflex responses and loss of motor ability (Thurmon *et al.*, 1996). Dexmedetomidine, a newer  $\alpha_2$ -agonist, reduces the dose requirements of opioids and anaesthetic agents, and attenuates the haemodynamic responses to tracheal intubation and surgical stimuli. Effects of dexmedetomidine have been studied in buffaloes (Singh, 2011), however, there are only a few reports on the use of dexmedetomidine in large

ruminants, especially buffaloes. Butorphanol, an opioid agonist–antagonist with sedative and analgesic properties, is known to induce mild sedation accompanied by small decreases in arterial blood pressure, heart rate and arterial oxygen tension in dogs (Trim, 1983). Synergistic interactions have been reported between  $\alpha_2$ -agonists and opioids or benzodiazepines in earlier studies (Amarpal *et al.*, 1998; Kojma *et al.*, 2002). Synergistic action of butorphanol and dexmedetomidine showed excellent muscle relaxation after anaesthetic induction with propofol in sheep (Monsang, 2011). Propofol, a short-acting hypnotic agent, is usually injected as a single bolus for anaesthetic induction to allow intubation and initiation of inhalant anaesthesia (Short and Bufalari, 1999). Propofol has been investigated as intravenous anaesthetic in sheep (Lin *et al.*, 1997), goats (Amarpal *et al.*, 2002) and buffaloes (Malik *et al.*, 2011). There are no reports available in literature on the use of dexmedetomidine, butorphanol, and propofol for the anaesthetic management of uraemic buffalo calves. Therefore, the present study was designed to compare the suitability of

propofol anaesthesia with two different preanaesthetic protocols.

#### MATERIALS AND METHODS

Sixteen male buffalo calves of 4–6 months of age and 40–55 kg of weight suffering from urolithiasis, brought for treatment to the Referral Veterinary Polyclinic, Indian Veterinary Research Institute, were used. The animals were divided randomly into A and B groups (n=8 each). The animals were restrained in right lateral recumbency and premedicated with intravenous dexmedetomidine (2.5 µg/kg body wt) alone in group A, and dexmedetomidine (2.5 µg/kg body wt) with butorphanol (0.05 mg/kg body wt) in group B. After 10 minutes of premedication, anaesthesia was induced by IV administration of 1 % propofol till effect. Anaesthesia was maintained with continuous intravenous infusion (CII) of propofol. The different treatments were evaluated on the basis of following parameters:

##### *Clinical Observations*

###### *Jaw Relaxation*

*Jaw relaxation* was taken as a measure of muscle relaxation and was evaluated at 0, 5, 10, 15, 20, 30, 45, 60, 75, and 90 min intervals by observing the resistance to opening of the jaw on applying pressure on lower and upper jaws, and graded on a 0 to 3 score scale as 0: not allowing to open the jaws, 1: resistant to opening the jaws and closed quickly, 2: less resistance to opening of jaws and closed slowly, and 3: no resistance and jaws remained open. At each interval mean value for jaw relaxation score was calculated and the muscle relaxation was graded as nil on a score of 0, very mild when the score was > 0 but < 1, mild when the score was ≥ 1 but < 2, moderate when the score was ≥ 2 but < 3 and excellent when the score was 3.

###### *Palpebral Reflex*

*Palpebral reflex* was recorded as a measure of depth of sedation by observing the blink of eye lids on touching the medial canthus with index finger and graded on a 0 to 3 score scale as 0: intact and strong reflex (quick blink), 1: intact but weak reflex (slow response), 2: very weak reflex (very slow and occasional), and 3: abolished reflex. Palpebral reflex was recorded at 0, 5, 10, 15, 20, 30, 45, 60, 75, and 90 min intervals. At each interval mean value for palpebral reflex score was calculated and the sedation was graded as absent on a score of 0, mild when the score was > 0 but < 1, moderate when the score was ≥ 1 but < 2, deep when the score was ≥ 2 but < 3 and very deep when the score was 3.

###### *Pedal Reflex*

*Pedal reflex* was recorded as a measure of depth of analgesia by recording the response to pin prick at coronet region on the digits of hind limbs and graded on a 0 to 3 score scale as 0: intact and strong reflex (strong withdrawal), 1: intact but weak reflex (animal responding slowly), 2: intact but very light reflex (slow and occasional response), and 3: reflex abolished completely. Pedal reflex was recorded at 0, 5, 10, 15, 20, 30, 45, 60, 75 and 90 min intervals. At each interval mean value for pedal reflex score was calculated and the analgesia was graded as no analgesia on a score of 0, very mild analgesia when the score was > 0 but < 1, mild analgesia when the score was ≥ 1 but < 2, moderate analgesia when the score was ≥ 2 but < 3, and complete analgesia when the score was 3.

###### *Salivation*

*Salivation* was recorded at different intervals and graded on a 0 to 3 score scale as 0: No salivation, 1: mild salivation, 2: moderate salivation, and 3: excessive salivation.

###### *Induction*

*Induction dose* of propofol was calculated in mg/kg for each group.

###### *Maintenance*

*Maintenance dose* of propofol was calculated in mg/kg/min for each group.

###### *Recovery Time (RET)*

*Recovery time (RET)* was recorded as the time elapsed from discontinuation of injection of drugs to the reappearance of pedal reflex.

###### *Sternal Recumbency Time (SRT)*

*Sternal recumbency time (SRT)* was recorded as the time elapsed from discontinuation of injection of drugs until the spontaneous regaining of sternal recumbency.

###### *Standing Time (ST)*

*Standing time (ST)* was recorded as the time elapsed from the time of discontinuation of injection of drugs until the spontaneous regaining of standing position and able to walk.

###### *Duration of Anaesthesia*

*Duration of anaesthesia* was recorded as the time elapsed from the time of abolition of pedal reflex to the time of reappearance of pedal reflex.

###### *Urination*

*Urination and defecation* were also recorded.

###### *Physiological Observations*

Heart rate (beats/min) by non-invasive blood pressure (NIBP) monitor, respiratory rate (breaths/min) by counting the movement of the thorax, and rectal temperature (°C) by digital thermometer were recorded before administration of drug(s) at 0 minute and at 5, 10, 15, 20, 30, 45, 60, 75 and 90 min after administration of drugs.

###### *Haematological Observations*

One ml blood was collected in heparinized (1:1000) disposable syringes, at 0 min (base line), 15, 30, 60 and 90 min after administration of drugs for the estimation of haemoglobin (Hb in g/L) using Sahli's haemoglobinometer, packed cell volume (PCV in L/L) by microhaematocrit method, total leukocyte count (TLC in  $\times 10^9/L$ ) by using Neubauer counting chamber and differential leukocyte count (DLC in %) by a thin, stained blood smear.

###### *Biochemical Observations*

Five ml venous blood was collected in heparinized (1:1000) disposable syringes (1 ml in sodium fluoride for glucose estimation) at 0 min (base line) and at 15, 30, 60 and 90 min after injection of drug(s) for separation of plasma for the estimation of urea nitrogen (mmol/L) by diacetyl monoxide (DAM) method, glucose (mmol/L) by GOD/POD method and creatinine (µmol/L) by alkaline picrate method.

###### *Haemodynamic Observations*

Systolic blood pressure (SBP), diastolic blood pressure (DBP) and mean arterial pressure (MAP) were recorded at 0 min (base line) and at 5, 10, 15, 20, 30, 45, 60, 75 and 90 min after administration of the drugs by non invasive blood pressure (NIBP) monitor. Oxygen saturation of haemoglobin (SpO<sub>2</sub>) by pulse oxymetry was also recorded.

###### *Statistical Analysis*

Analysis of Variance (ANOVA) and Duncan's multiple range test (DMRT) were used to compare the means at different time intervals between the groups. Paired 't' test was used to compare the means at different intervals with respective base values in each group (Snedecor and Cochran, 1994).

#### RESULTS

##### *Jaw Relaxation*

In animals of both groups, the jaws were mildly relaxed after premedication at 5 and 10 min followed by excellent relaxation after induction of anaesthesia up to the end of anaesthetic period (Figure 1). After discontinuation of anaesthesia a normal jaw tone was attained.

**Palpebral Reflex**

In animals of group A, the reflex was moderately depressed after premedication at 5 and 10 min, followed by complete abolition of reflex up to the end of anaesthetic period (Figure.2). In group B, the palpebral reflex was completely abolished from 10 min after premedication up to the end of anaesthesia. Comparison between groups A and B revealed that the palpebral reflex was more depressed in group B.

**Pedal Reflex**

In group A, pedal reflex was mildly depressed after premedication at 5 and 10 min, followed by excellent depression

of reflex after induction of anaesthesia up to the end of the anaesthetic period (Figure 3). In animals of group B, pedal reflex was mild to moderately depressed after premedication at 5 and 10 min, followed by excellent depression of reflex up to the end of anaesthetic period.

**Salivation**

In animals of group A, a very mild salivation was recorded at premedication followed by a moderate salivation up to the end of anaesthetic period. In group B, a mild salivation was recorded after premedication, which persisted up to the end of anaesthetic period.

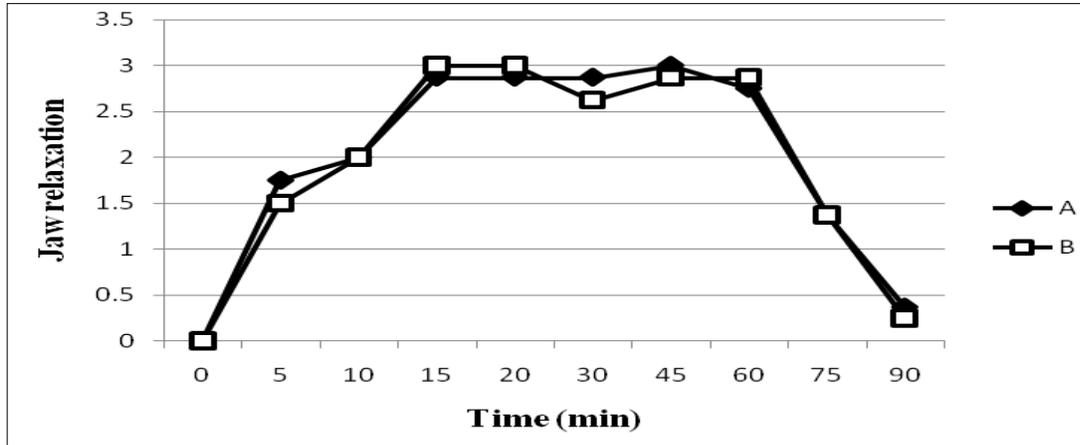


Figure 1: Mean±SE values of score for jaw relaxation in animals of groups A and B at different time intervals

Figure 2: Mean±SE values of score for palpebral reflex in animals of groups A and B at different time intervals

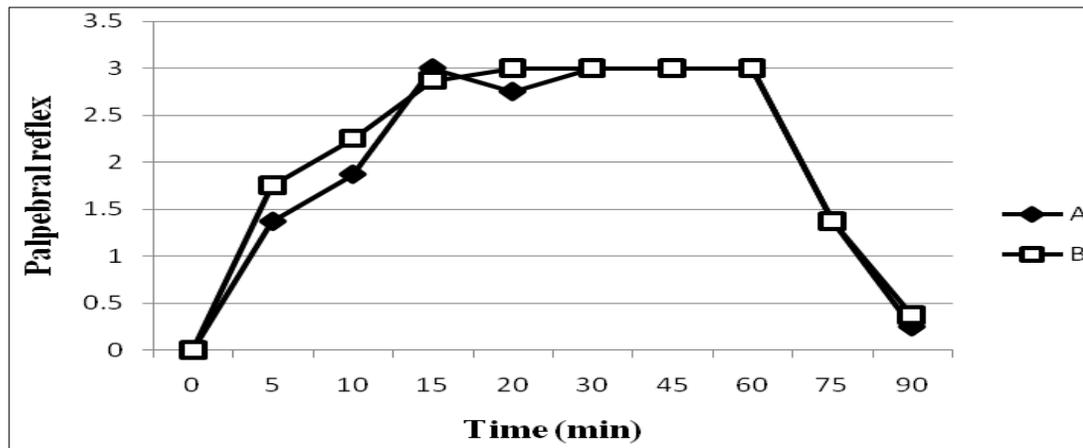


Figure 3: Mean±SE values of score for pedal reflex in animals of groups A and B at different time intervals

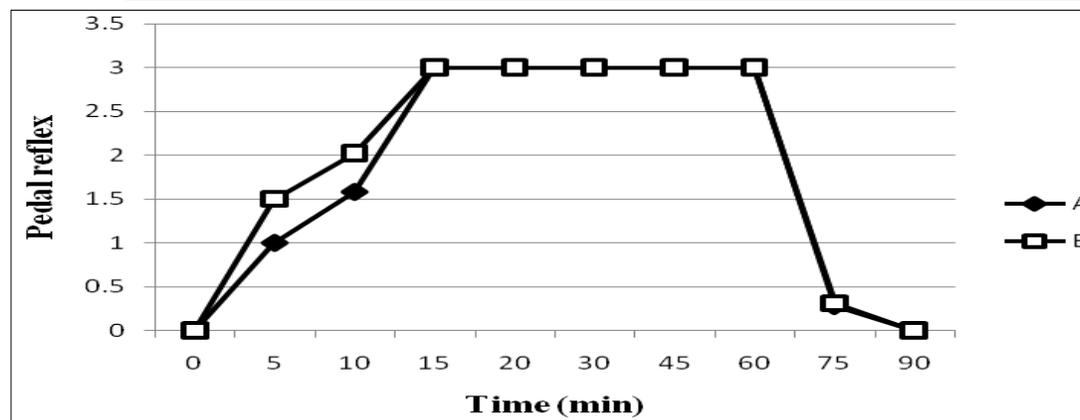


Figure 3: Mean±SE values of score for pedal reflex in animals of groups A and B at different time intervals

Figure 4: Median  $\pm$  SE values of recovery time, sternal recovery time, standing time and duration of anaesthesia in animals of groups A and B

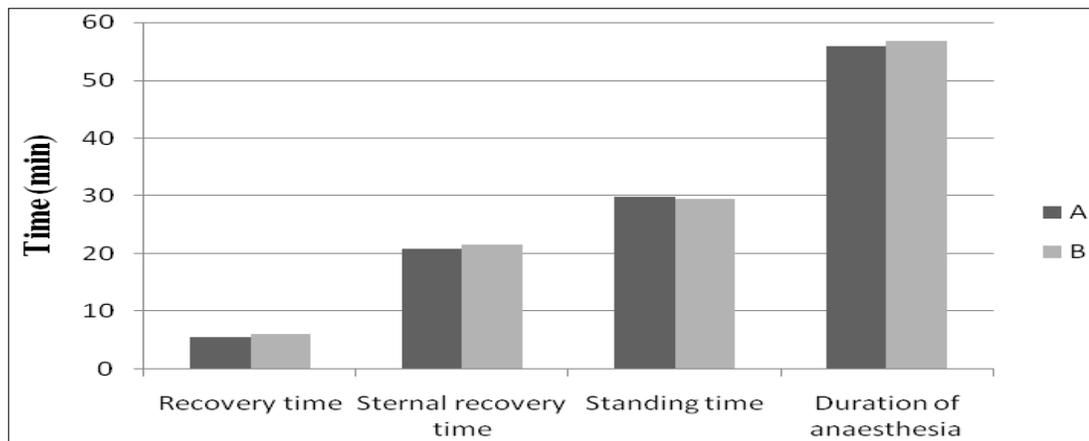


Figure 5: Mean  $\pm$  SE values of heart rate in animals of groups A and B at different time intervals

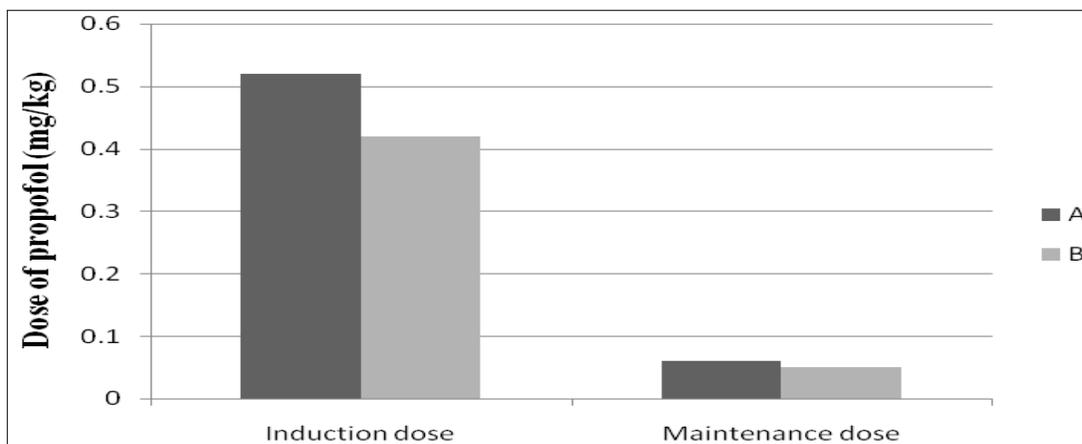
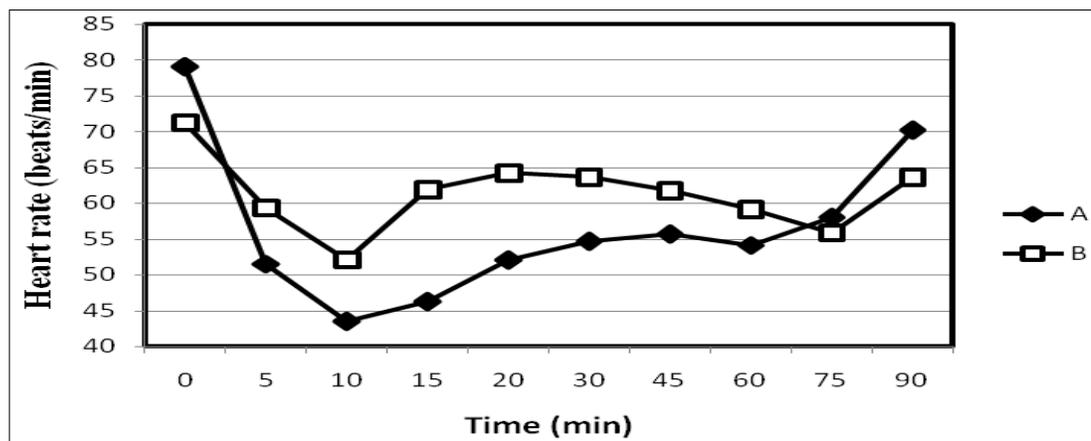


Figure 5: Mean  $\pm$  SE values of heart rate in animals of groups A and B at different time intervals

Figure 6: Mean  $\pm$  SE values of heart rate in animals of groups A and B at different time intervals



**Recovery Time**

The median  $\pm$  SD values of recovery time in groups A and B were 5.62 $\pm$ 0.62 min and 6.12 $\pm$ .54 min, respectively (Figure 4).

**Sternal Recumbency Time**

The median  $\pm$  SD values of sternal recumbency time in groups A and B were 20.87 $\pm$ 2.91 min and 21.50 $\pm$ 3.02 min, respectively (Figure 4).

**Standing Time**

The median  $\pm$  SD values of standing time recorded in groups A and B were 29.87 $\pm$ 3.26 min and 29.50 $\pm$ 3.13 min, respectively (Figure 4).

**Duration of Anaesthesia**

The median  $\pm$  SD values of duration of anaesthesia recorded in groups A and B were 55.87 $\pm$ 0.95 min and 56.72 $\pm$ 1.12 min, respectively (Figure 4).

**Induction Dose**

The mean values of induction dose of propofol required in groups A and B were 0.52 $\pm$ 0.08 mg/kg and 0.42 $\pm$ 0.06 mg/kg, respectively (Figure 5).

**Maintenance Dose**

The mean values of maintenance dose of propofol recorded in groups A and B were 0.06+ 0.00 mg/kg/min and 0.05+0.00 mg/kg/min, respectively (Figure 5).

**Physiological Observations**

**Heart rate (HR):** HR decreased ( $P<0.01$ ) significantly in the animals of group A after premedication. Thereafter, HR improved and remained significantly ( $P<0.05$ ) below the base line from 20 min to 75 min interval (Figure 6). In the animals of group B, HR decreased significantly ( $P<0.01$ ) following premedication. A slight increase in HR was recorded after induction, however, the values remained significantly ( $P<0.05$ ) decreased from the base line till the end of observation period.

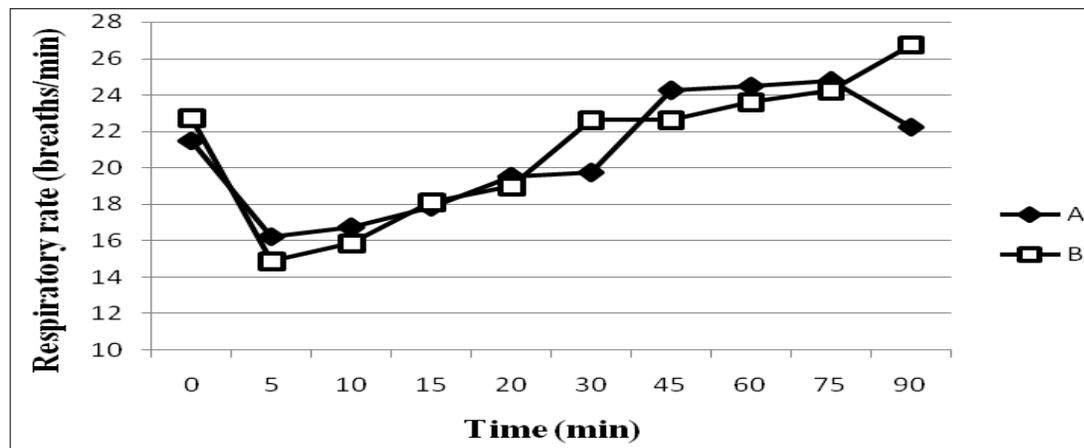


Figure 7: Mean±SE values of respiratory rate in animals of groups A and B at different time intervals

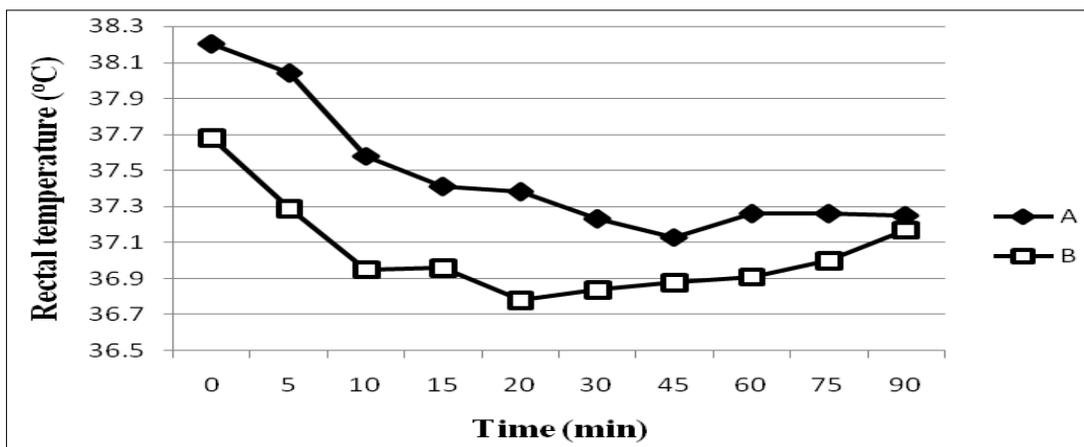
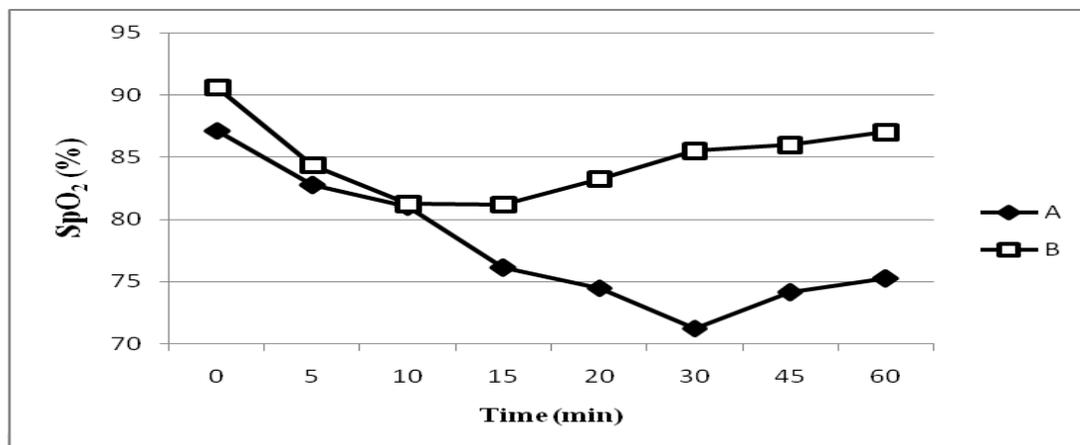


Figure 8: Mean±SE values of rectal temperature in animals of groups A and B at different time intervals

Figure 9: Mean±SE values of mean arterial pressure in animals of groups A and B at different time intervals



### Respiratory Rate (RR)

RR decreased ( $P<0.05$ ) significantly after premedication in both groups but after induction it increased significantly ( $P<0.01$ ) in group A from 45 to 75 min (Figure 7). In group B, there was a gradual increase in RR after induction of anaesthesia and it remained above the base line up to the end of observation period.

### Rectal Temperature (RT)

In group A, RT decreased significantly ( $P<0.01$ ) in both groups during almost the entire observation period (Figure 8).

### Haematological Observations

#### Haemoglobin (Hb), Packed Cell Volume (PCV) and Total leukocyte count (TLC)

In both groups, there was a significant ( $P<0.01$ ) decrease in Hb, PCV and TLC from 15 to 90 min (Table).

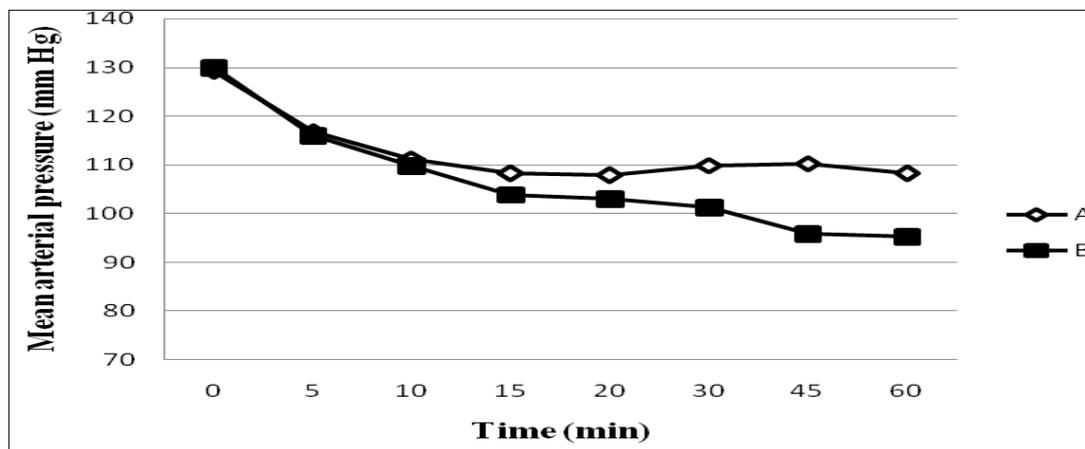
#### Neutrophils and Lymphocytes

In both groups there was a significant ( $P<0.01$ ) increase in neutrophils and corresponding decrease in lymphocytes from 15 to 90 min of observation period (Table).

Table: Mean  $\pm$ SE of haematobiochemical parameter in animals of groups A and B at different intervals

Parameters	Groups	Time Intervals (min)				
		0	15	30	60	90
Haemoglobin (g/L)	A	117.50 $\pm$ 2.50	108.00 <sup>**</sup> $\pm$ 2.09	106.56 <sup>**</sup> $\pm$ 2.16	108.69 <sup>*</sup> $\pm$ 3.11	106.87 <sup>**</sup> $\pm$ 2.98
	B	113.75 $\pm$ 5.32	101.37 <sup>**</sup> $\pm$ 5.16	101.25 <sup>**</sup> $\pm$ 4.30	99.50 <sup>ab</sup> <sup>**</sup> $\pm$ 2.78	99.87 <sup>**</sup> $\pm$ 2.31
Packed cell volume (L/L)	A	0.38 $\pm$ 0.00	0.34 <sup>**</sup> $\pm$ 0.01	0.33 <sup>**</sup> $\pm$ 0.01	0.34 <sup>b</sup> <sup>**</sup> $\pm$ 0.00	0.34 <sup>*</sup> $\pm$ 0.00
	B	0.38 $\pm$ 0.01	0.33 <sup>**</sup> $\pm$ 0.00	0.34 <sup>**</sup> $\pm$ 0.01	0.31 <sup>**</sup> $\pm$ 0.00	0.33 <sup>**</sup> $\pm$ 0.00
Total leukocyte count (x10 <sup>9</sup> /L)	A	10.61 $\pm$ 0.47	9.17 <sup>**</sup> $\pm$ 0.68	8.93 <sup>**</sup> $\pm$ 0.79	9.10 <sup>**</sup> $\pm$ 0.52	8.91 <sup>**</sup> $\pm$ 0.58
	B	9.97 $\pm$ 0.47	8.95 <sup>**</sup> $\pm$ 0.62	8.76 <sup>**</sup> $\pm$ 0.60	8.44 <sup>**</sup> $\pm$ 0.67	8.01 <sup>**</sup> $\pm$ 0.63
Neutrophil count (%)	A	30.12 $\pm$ 1.99	34.25 <sup>**</sup> $\pm$ 2.16	34.50 <sup>**</sup> $\pm$ 2.10	34.50 <sup>**</sup> $\pm$ 1.50	32.75 <sup>**</sup> $\pm$ 1.76
	B	27.87 $\pm$ 2.15	32.00 <sup>**</sup> $\pm$ 2.57	34.25 <sup>**</sup> $\pm$ 2.32	33.87 <sup>**</sup> $\pm$ 1.61	31.00 <sup>**</sup> $\pm$ 2.40
Lymphocyte count (%)	A	64.87 $\pm$ 1.99	60.75 <sup>**</sup> $\pm$ 2.16	58.50 <sup>**</sup> $\pm$ 2.10	58.50 <sup>**</sup> $\pm$ 1.50	60.25 <sup>**</sup> $\pm$ 1.76
	B	67.12 <sup>**</sup> $\pm$ 2.15	63.00 <sup>**</sup> $\pm$ 2.57	58.75 <sup>**</sup> $\pm$ 2.32	59.12 <sup>**</sup> $\pm$ 1.61	62.00 <sup>**</sup> $\pm$ 2.40
Urea nitrogen (mmol/L)	A	11.43 $\pm$ 1.37	12.91 <sup>**</sup> $\pm$ 1.54	13.39 <sup>**</sup> $\pm$ 1.48	13.98 <sup>**</sup> $\pm$ 1.50	11.98 $\pm$ 0.74
	B	11.32 $\pm$ 1.09	12.23 $\pm$ 1.26	14.79 <sup>**</sup> $\pm$ 1.14	12.91 <sup>**</sup> $\pm$ 1.10	12.20 $\pm$ 0.77
Creatinine ( $\mu$ mol/L)	A	533.17 $\pm$ 62.14	455.17 $\pm$ 72.75	437.50 <sup>ab</sup> <sup>**</sup> $\pm$ 49.31	436.05 <sup>*</sup> $\pm$ 66.20	437.87 <sup>ab</sup> $\pm$ 53.18
	B	488.80 $\pm$ 66.97	478.40 $\pm$ 57.64	477.10 $\pm$ 49.62	448.50 $\pm$ 62.51	477.10 $\pm$ 55.95
Glucose (mmol/L)	A	6.91 $\pm$ 0.69	8.54 <sup>**</sup> $\pm$ 2.00	9.09 <sup>**</sup> $\pm$ 0.61	9.15 <sup>**</sup> $\pm$ 0.68	8.60 <sup>**</sup> $\pm$ 0.82
	B	7.41 $\pm$ 1.55	8.58 $\pm$ 1.04	8.73 $\pm$ 0.41	9.22 $\pm$ 0.43	8.91 $\pm$ 0.44

Figure 10: Mean $\pm$ SE values of oxygen saturation of haemoglobin (SpO<sub>2</sub>) in animals of groups A and B at different time intervals



### Biochemical Observations

**Urea nitrogen:** In group A the urea nitrogen increased significantly ( $P<0.01$ ) from 15 to 60 min of observation period (Table). In group B, urea nitrogen increased significantly ( $P<0.01$ ) at 30 min and 60 min ( $P<0.05$ ).

### Glucose

In group A, glucose increased significantly ( $P<0.01$ ) from 15 min to 90 min of observation period (Table). In group B, glucose increased significantly ( $P<0.05$ ) at 15 min. From 30 min of observation period, the plasma glucose started decreasing to return to its base level by 90 min.

### Creatinine

In group A, creatinine decreased significantly ( $P<0.05$ ) from 15 min up to the end of the observation period (Table). In group B, creatinine decreased ( $P<0.05$ ) significantly at 15 min.

### Haemodynamic Observations

**Systolic blood pressure (SBP) and Diastolic blood pressure (DBP):** SBP and DBP decreased significantly ( $P<0.01$ ) after premedication up to 90 min of observation in both groups.

### Mean Arterial Blood Pressure (MAP)

In group A, a more significant ( $P<0.01$ ) decrease in MAP from premedication up to 75 min of observation was recorded except at 90 min where the decrease was less significant

( $P < 0.05$ ) (Figure 9). In group B, a significant ( $P < 0.01$ ) decrease in MAP was recorded throughout the period of observation. MAP was significantly ( $P < 0.05$ ) lower in group B as compared to that in group A at 75 min.

#### **Haemoglobin Oxygen Saturation (SpO<sub>2</sub>)**

In group A, a significant ( $P < 0.05$ ) decrease in SpO<sub>2</sub> at 15 min and 20 min and a highly significant ( $P < 0.01$ ) decrease from 30 to 60 min was recorded (Figure 10). In group B, a significant ( $P < 0.01$ ) decrease in SpO<sub>2</sub> was recorded from premedication up to 15 min of observation period.

#### **DISCUSSION**

The jaw relaxation was excellent at most of the intervals in both groups. Alpha 2-adrenergic agonists have been reported to produce profound muscle relaxation when used alone or in combination with opioid agonist-antagonists (Ko *et al.*, 1996; Pratap *et al.*, 2000; Ahmad *et al.*, 2011; Singh, 2011). The muscle relaxation effect that accompanies sedation is due to inhibition of alpha 2-adrenoceptors at the interneuron level of the spinal cord (Cullen, 1996). Excellent muscle relaxation recorded after induction with propofol in the present study might be due to combined effects of alpha 2-agonist, opioid and propofol.

Dexmedetomidine induces a dose dependent sedation but increasing the dose beyond a certain level does not cause a further increase in sedation (Kuusela *et al.*, 2000). Synergistic sedative and analgesic activity between alpha 2-agonist and opioid agonist-antagonist has been reported in ruminants (Malik *et al.*, 2011; Singh, 2011). In the present study, mild to moderate analgesia recorded in both groups during preanaesthetic period might be due to administration of dexmedetomidine and butorphanol. However, excellent analgesia observed after administration of propofol might be due to the action of anaesthetic propofol.

The reduction in the induction dose of propofol in group B than in group A might be due to synergistic action of dexmedetomidine and butorphanol. A marked synergism between alpha 2-agonists and propofol has been reported previously (Hammond and England, 1994). Synergism between alpha 2-agonists, butorphanol and propofol might have played an important role in reducing the induction and maintenance dose of propofol in group B in the present study.

Rapid recovery after propofol and halothane anaesthesia has been reported in goats (Carroll *et al.*, 1998). Similar findings were also observed in the present study. There is lack of any cumulative effect of propofol after its administration either by repeat bolus injection or by continuous infusion (Adetunji *et al.*, 2002).

Bradycardia has been reported following dexmedetomidine administration in goats (Kastner *et al.*, 2005) and sheep (Kastner *et al.*, 2006; Monsang, 2011). Butorphanol has been reported to produce mild lowering of HR and minimum cardiovascular effects (Greene *et al.*, 1990). It has also been reported that butorphanol facilitates the increase in parasympathetic tone and thereby contributes to bradycardia (Ko *et al.*, 2000). Bradycardia recorded in the present study could be attributed to the action of dexmedetomidine and butorphanol.

The initial reduction in RR recorded in the present study might be due to dexmedetomidine and butorphanol. More reduction in RR recorded in group B was in accordance with the earlier studies where a greater respiratory depression was recorded when medetomidine was combined with butorphanol in buffaloes (Malik *et al.*, 2011). Similarly, decreased RR was reported following administration of dexmedetomidine along with butorphanol in dogs (Gupta, 2010; Surbhi *et al.*, 2010),

sheep (Monsang, 2011) and buffaloes (Ahmad, 2009). Propofol caused a further decrease in mean RR in groups A and B, plausibly by depressing central inspiratory drive and ventilatory response to arterial carbon-dioxide response (Goodman *et al.*, 1987).

A decrease in RT recorded in both groups might be attributed to a decrease in the skeletal muscle tone, reduced metabolic rate and muscle relaxation along with depression of thermoregulatory centers. Alpha-2 adrenergic agonists have been reported to induce prolonged depression of thermoregulation (Ponder and Clarke, 1980). These agents have also been found to depress hypothalamic noradrenergic alpha 2 receptors to cause hypothermia (MacDonald *et al.*, 1988). Decrease in RT has been reported after dexmedetomidine in dogs (Ahmad *et al.*, 2011; Santhosh, 2011) and sheep (Monsang, 2011), and dexmedetomidine and butorphanol in dogs (Gupta, 2010) and sheep (Monsang, 2011). Hypothermia has also been reported during propofol anaesthesia in goats (Carroll *et al.*, 1998; Amarpal *et al.*, 2002) and buffaloes (Malik *et al.*, 2011).

Haemoglobin, PCV and TLC decreased significantly in both groups during post-anaesthetic period. Pooling of circulatory blood cells in the spleen or other reservoirs secondary to decreased sympathetic activity may explain the decrease in Hb, PCV and TLC (Wagner *et al.*, 1991). The decrease in PCV and Hb during the period of anaesthesia or sedation may also be due to shifting of fluid from extravascular compartment to intravascular compartment in order to maintain normal cardiac output in the animals (Wagner *et al.*, 1991). The decreased Hb and PCV have also been reported after administration of dexmedetomidine in dogs (Gupta, 2010) and sheep (Monsang, 2011), butorphanol alone (Ahmad, 2009) and along with midazolam in buffaloes (Malik *et al.*, 2011). Neutrophilia and lymphocytopenia recorded in the present study might be due to the stress caused by the preanaesthetic and anaesthetic drugs and subsequent stimulation of adrenal glands. Similar findings have been reported after administration of dexmedetomidine in dogs (Ahmad *et al.*, 2011) and sheep (Monsang, 2011), or combinations of dexmedetomidine-midazolam-fentanyl in dogs (Ahmad *et al.*, 2011), midazolam-butorphanol in sheep (Monsang, 2011), butorphanol-medetomidine or butorphanol-halothane in buffaloes (Malik *et al.*, 2011).

The increase in urea nitrogen might be attributed to the temporary inhibitory effects of anaesthetic drugs on the renal blood flow, which in turn might have caused a rise in urea nitrogen level as suggested by Kinjavdekar *et al.* (2000). The increased hepatic urea production from amino acid degradation could also account for the observed increase in blood urea values during the maximum depth of anaesthesia (Eichner *et al.*, 1979). Increased BUN has been recorded during propofol anaesthesia in dogs premedicated with xylazine (Surbhi *et al.*, 2010).

The increase in plasma glucose observed in the present study might be attributed to an alpha 2-adrenergic inhibition of insulin release from beta cells of pancreas and increased glucose production in the liver (Gasthuys *et al.*, 1987). Increased glucose has also been reported following administration of medetomidine/dexmedetomidine-butorphanol followed by propofol anaesthesia in canine orthopaedic patients (Gupta, 2010), buffaloes (Malik *et al.*, 2011) and sheep (Monsang, 2011), or propofol in dogs (Surbhi *et al.*, 2010; Ahmad *et al.*, 2011).

Dexmedetomidine might have been responsible for adequate renal blood flow and enough glomerular filtration rate to maintain creatinine near the base value in both groups.

However, creatinine was reported to increase following administration of butorphanol–xylazine (Surbhi *et al.*, 2010) or medetomidine in propofol anaesthetized canine orthopaedic patients (Gupta, 2010; Surbhi *et al.*, 2010) and healthy buffaloes (Ahmad, 2009; Malik *et al.*, 2011).

Decreased blood pressure after premedication might be attributed to the effects of dexmedetomidine as IV administration of alpha-2 agonists have been reported to cause a prolonged hypotension (Ruffolo *et al.*, 1993). Hypotension is also attributed to bradycardia and vasodilation, stimulation of central alpha-2 adrenoceptors, peripheral sympatholytic action and enhanced parasympathetic outflow (Tibirica *et al.*, 1991). Decreased BP has been reported following administration of medetomidine–butorphanol during halothane anaesthesia in buffaloes (Ahmad, 2009; Malik *et al.*, 2011). A decrease in blood pressure after propofol administration has been associated with arterial and venous vasodilatation and decreased contractility of the heart (Ilkiw *et al.*, 1992).

Decrease in SpO<sub>2</sub> was possibly due to a certain degree of respiratory depression in both groups. Detomidine, medetomidine and romifidine produce severe hypoxaemia when administered IV at equipotent sedative doses in conscious sheep (Celly *et al.*, 1997a, b). Similarly, decreased SpO<sub>2</sub> has been reported following administration of butorphanol–medetomidine or dexmedetomidine in propofol anaesthetized dogs (Gupta, 2010; Surbhi *et al.*, 2010) and sheep (Monsang, 2011). Propofol infusion has been reported to cause significant respiratory depression with decrease in all measures of ventilation in animals (Kuusela *et al.*, 2003).

Based on the present study, it is concluded that dexmedetomidine (2.5µg/kg) induces good sedation, analgesia and muscle relaxation in uraemic buffalo calves but causes transient cardiopulmonary depression. Butorphanol can augment sedation and analgesia produced by dexmedetomidine, and can spare the dose of propofol needed for induction and maintenance of anaesthesia.

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