Haryana Vet. **50** (December, 2011), pp 1-4

Research Article

EVALUATION OF ATROPINE AS PREANAESTHETIC TO DIAZEPAM-THIOPENTONE ANAESTHESIA IN BUFFALOES

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ABSTRACT

The study was done to evaluate atropine as preanaesthetic to diazepam-thiopentone anaesthesia in 12 buffalo calves by administering atropine sulphate (0.04 mg/kg, IM), diazepam (0.2 mg/kg, IV) and thiopentone sodium (5% 'to effect', IV). Diazepam was administered 10 minutes after atropine and thiopentone 5 minutes after diazepam administration. The calves became calm, ataxic and recumbent within one minute of diazepam administration, but responsive to pin prick stimulation. Swallowing and palpebral reflexes were abolished within one to three minutes of thiopentone administration. The panniculus and periosteal scratching reflexes remained intact. Relaxation of tail, limbs and ballooning of anus were observed only in two calves within two minutes after thiopentone administration. The calves stood up with ataxia at 86±3 minutes but complete recovery occurred at 214±4 minutes. There was a gradual increase in heart rate after atropinization. The pulse pressure remained low throughout the experiment. Mean arterial pressure (MAP) increased gradually after atropine and diazepam administration. However, at two minutes of thiopentone administration, the MAP dropped below the base value. The change in central venous pressure was non-significant. There were no significant changes in the ECG time and voltage components except biphasic T-wave and inversion of T-wave.

Key words: Atropine, diazepam, thiopentone and buffalo calves

General anaesthesia, for surgical manipulations, may require the intravenous administration of certain drugs with different actions to ensure sedation, analgesia, muscle relaxation and control of visceral reflex responses. Diazepam (0.5 mg/kg, IV) and thiopentone produced severe hypotension without compensatory tachycardia in non-atropinised buffalo calves (Singh *et al*., 2007). Diazepam appears to have a parasympathomimetic effect in buffaloes and partially desensitizes baroreceptor reflexes thus inhibiting compensatory tachycardia (Singh *et al*., 2007). Since atropine blocks the cardiac vagus, it greatly reduces or abolishes cardiac inhibitory effects of drugs acting through a vagal mechanism. Thus the study was done to evaluate the benefits of atropine as preanaesthetic to diazepam-thiopentone anaesthesia in buffaloes.

MATERIALS AND METHODS

The present study was done on 12 clinically healthy male buffalo calves of six months to one year of age and weighing between 70 to 150 kg. The animals were randomly divided into two groups (groups I and II) of six animals each. Atropine sulphate (0.04 mg/kg, IM), diazepam (0.2 mg/kg, IV) and thiopentone sodium (5% 'to effect', IV) were administered. Diazepam was administered 10 minutes after atropine and thiopentone 5 minutes after the diazepam administration. In group I, behavioral changes (weak time, down time, relaxation of limb muscles, qualitative analgesic effects of drugs, recovery from analgesia, return to sternal recumbency, head rightening reflex, standing time with ataxia, browsing/munching time and complete clinical recovery without ataxia), physiological (rectal temperature and respiratory rate), haematological [haemoglobin (Hb), packed cell volume (PCV) and erythrocytic sedimentation rate (ESR)] and blood biochemical (blood glucose, blood urea nitrogen (BUN), plasma bilirubin, creatinine and total proteins) parameters were studied before and at 10 minutes of atropine administration, at 5 and 10 minutes of diazepam and thiopentone administration,

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respectively; then at recovery and, thereafter, at 24 hours after recovery. Whereas, in group II, systolic (SP) and diastolic pressure (DP; through carotid artery cannulation under local anaesthesia), central venous pressure (CVP; through jugular vein cannulation under local anaesthesia); mean arterial pressure (MAP), ECG, heart rate, pHa, partial pressure of oxygen (paO_2), partial pressure of carbon dioxidepa (pCO_2), base excess-extra cellular fluid (BE-ECF) and bicarbonate (HCO_3) were evaluated before administration of atropine, at 10 minutes of atropine, at 5 minutes of diazepam administration and at 2, 15, 30 and 45 minutes after the administration of thiopentone. The statistical analysis of data was done by one-way-analysis of variance and Duncan's multiple range tests.

RESULTS AND DISCUSSION

Different behavioral signs, hematological and biochemical parameters are presented in Tables 1 and 2. In group I, administration of diazepam made the calves calm and quiet. The calves became ataxic and recumbent within one minute of diazepam administration, but they were responsive to pin prick stimulation. Sarma and Kumar (1998) observed similar findings after diazepam administration in the atropinized cow calves. This might be due to state of more cerebral depression (Dodman *et al.*, 1989). The abolition of swallowing reflex was noticed within one minute after induction of anaesthesia with thiopentone. The palpebral reflex was abolished within two to three minutes of thiopentone induction in three calves, whereas, corneal reflex remained intact in all the calves. The panniculus as well as periosteal reflexes also remained intact in all the calves. Relaxation of tail, limbs and ballooning of anus were observed only in two out of six calves within two minutes after thiopentone administration. The return to sternal recumbency along with head rightening reflex was noticed at 62±4 minutes. The calves stood up with ataxia at 86±3 minutes but complete recovery took 214±4 minutes. The mean effective dose of thiopentone was found to be 6.54 mg/kg. Amarpal and Kumar (1994) observed head rightening reflex at 126.75±15.07 minutes and complete recovery at 305.00±21.89 minutes in atropine premedicated calves during the diazepam-thiopentone anaesthesia. However, goats premedicated with atropine have been reported to recover completely from the effect of diazepam-thiopentone anaesthesia at 85±10 minutes (Singh and Kumar, 1988).

Table 1
Behavioral changes after atropine-diazepam-thiopentone anaesthesia in buffalo calves (n=6)

Parameters	Atropine-Diazepam-Thiopentone (Mean time±S.E. in minutes)
Weak time	<1
Down time	<1
Abolition of swallowing reflex	<1
Head rightening reflex	62±4
Standing time with ataxia	86±3
Complete clinical recovery without ataxia	214±4

Mirakhur *et al.* (1989) observed that dose requirements of thiopentone sodium for providing surgical anaesthesia in cow calves did not decrease when diazepam (1.0 mg/kg) was used as a premedication agent. In buffalo calves, thiopentone sodium @ 10 mg/kg has been reported to produce surgical anaesthesia (Singh *et al.,* 1980). However, results of present study are in contrast to the observations of Mirakhur *et al.* (1989) in cow calves. Our results indicated that dose of thiopentone required for general anaesthesia was considerably reduced i.e. 6.54 mg/kg when buffalo calves were premedicated with diazepam (0.2 mg/kg).

There were no significant changes in rectal temperature and respiration rate during the entire period of observation. Mirakhur *et al.* (1989) and Singh *et al.* (2007) did not observe any appreciable changes in the rectal temperature after diazepam-thiopentone anaesthesia in cow and buffalo calves, respectively. However, Amarpal and Kumar (1995) observed significant decrease in rectal temperature in cow calves during atropine-diazepam-thiopentone anaesthesia. After diazepam administration, the respiration rate was elevated (34±6 minutes) as compared to base value (26±4 minutes). Diazepam has been shown to cause hypoxaemia and decrease in tidal volume in cow calves (Mirakhur *et al.,* 1988a). The increase in respiration, therefore, appears to be a compensatory mechanism in an attempt to offset the hypoxaemia and decrease in tidal volume.

There were no significant variations in any of the

Table 2

Effects of atropine-diazepam-thiopentone anaesthesia on certain clinico-pathological parameters in buffalo calves (n=6)

Parameters (Units)	Base value	10 minutes after atropine administration	5 minutes after diazepam administration	10 minutes after thiopentone administration	At recovery	24 hours after recovery
Ambient temperature (^{0}C)	32.5^{bc}±0.5	33.2^{abc}±0.3	33.5^{ab}±0.4	34.0^{a}±0.4	31.8^{c}±0.7	32.0^{c}±0.3
Rectal temperature (^{0}C)	38.3^{a}±0.2	38.4^{a}±0.2	38.2^{a}±0.2	37.9^{a}±0.4	38.4^{a}±0.6	38.2^{a}±0.2
Respiration rate (per minute)	26^{ab}±4	26^{ab}±3	34^{a}±6	27^{ab}±5	29^{ab}±1	22^{b}±2
Haemoglobin (g%)	9.9^{a}±0.2	9.7^{a}±0.1	9.9^{a}±0.3	9.7^{a}±0.4	9.9^{a}±0.4	10.0^{a}±0.3
Packed cell volume (%)	28^{a}±1	28^{a}±1	29^{a}±1	27^{a}±1	29^{a}±2	29^{a}±1
ESR (mm first hour)	52^{a}±8	55^{a}±7	54^{a}±7	53^{a}±8	55^{a}±8	56^{a}±7
Blood glucose (mg/dl)	60.9^{a}±8.5	72.2^{a}±11.1	67.1^{a}±8.4	82.2^{a}±9.5	80.8^{a}±11.6	56.6^{a}±12.1
Total plasma proteins (g/dl)	6.4^{a}±0.5	5.9^{a}±0.9	5.5^{a}±0.9	5.2^{a}±0.5	5.9^{a}±0.5	5.6^{a}±0.4
Blood urea nitrogen (g/dl)	24.9^{a}±2.8	26.7^{a}±1.4	26.7^{a}±1.7	26.9^{a}±0.7	25.8^{a}±1.6	21.4^{a}±2.3
Plasma creatinine (mg/dl)	0.63^{a}±0.04	0.62^{a}±0.04	0.65^{a}±0.04	0.68^{a}±0.04	0.64^{a}±0.04	0.56^{a}±0.01
Plasma bilirubin (mg/dl)	0.47^{a}±0.11	0.50^{a}±0.12	0.72^{a}±0.18	0.74^{a}±0.11	0.77^{a}±0.09	0.49^{a}±0.11

haematological and blood biochemical parameters (Table 2). However, 10 minutes after thiopentone administration, the blood glucose increased to 82.2±9.5 mg/dl as compared to base value of 60.9±8.5 mg/dl. The total plasma proteins at this stage decreased as compared to base value. The plasma bilirubin increased to 0.77±0.08 mg/dl at recovery time as compared to the base value of 0.47±0.11 mg/dl. Singh *et al.* (2007) observed similar changes in haematological and blood biochemical parameters in buffalo calves during diazepam-thiopentone anaesthesia. Amarpal and Kumar (1995) also observed non-significant changes in total plasma proteins, serum urea nitrogen and creatinine and non-significant fall in Hb and PCV in calves but a significant increase in blood glucose level. In the present study, hyperglycaemia may be due to decreased glucose utilization or due to increased glycogenolysis.

There was a gradual increase in heart rate after atropinization which further increased even up to 15 minutes of diazepam administration. The increase in heart rate was about 140% after diazepam administration as compared to the base value. Immediately after thiopentone administration, the calves developed respiratory arrest. At that time, the heart rate was 86 beats per minute as compared to base value of 27 beats per minute. The endotracheal intubation was done and calves were resuscitated. The pulse pressure remained low throughout the experiment. The MAP increased gradually after atropine administration. At 5 minutes after diazepam administration, the MAP increased further than the base value (142.6 mmHg). However, at two minutes of thiopentone administration, the MAP (107.2 mmHg) dropped below the base value. The change in CVP was non-significant throughout the experiment.

There were no significant changes in the ECG time and voltage components except for some primary T-wave changes which included biphasic T-wave and inversion of T-wave. Such changes can occur after variations in myocardial oxygenation which were expected under diazepam-thiopentone anaesthesia.

There was considerable reduction in arterial pH along with marked increase in arterial pCO2 values once the calves were disconnected from the ventilator at 30 minutes after the thiopentone. There was also acute arterial hypoxaemia. These changes may be due to alveolar hypoventilation (Singh *et al.*, 1980; Mirakhur *et al.*, 1988b; Amarpal and Kumar, 1995) and increased alveolar-arterial oxygen gradient due to venous admixture (Mirakhur, 1991). There were no significant changes in arterial HCO_3 and BE-ECF. There were also no appreciable variations in the blood concentrations of sodium, potassium, calcium, magnesium and chloride. As the effects of the anaesthesia on cardiopulmonary systems were marked and inconsistent, the anaesthetic combination may not be safe in diseased buffaloes and may prove fatal in absence of positive pressure ventilation.

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