Full Length Article

HAEMATO-BIOCHEMICAL CHANGES IN PROPOFOL WITH 2% LIGNOCAINE CONTINUOUS RATE INFUSION (CRI) IN DOGS

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ABSTRACT

The present research work was conducted to study the effect of propofol on haemato-biochemical and physiological parameters. A total of twelve adult dogs divided into six (6) animals in each group viz Group-I (Maintenance by propofol) and Group-II (maintenance by propofol and 2% lignocaine CRI) were presented for removal of infected uterus. Haematological parameters and biochemical parameters were within their normal physiological range in all groups. Based on the clinical, biochemical, hematological parameters propofol-lignocaine combination was more suitable.

Keywords: Atropine sulphate, Dog, Lignocaine, Propofol, Xylazine HCl

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INTRODUCTION

Perfect anaesthesia is characterised by adequate muscular relaxation, analgesia, sedation and reversible loss of consciousness for surgical intervention. The key to successful surgery is the implementation of proper anaesthesia and peri-operative monitoring. Balanced anaesthesia provides proper analgesia, sedation and rapid and smooth recovery (Mc Keni 2008). So an appropriate selection of anaesthetic agents and techniques is required for surgical intervention. Parenteral drugs sedation selection can be accomplished by the premedication with sedative and analgesic agents and continuous rate infusion during the maintenance of anaesthesia by an anaesthetic agent. Impediment of inward breath sedation is that it requires the utilization an awkward and expensive sedation machines. In field condition, intramuscular or intravenous sedation/anaesthesia is typically the technique for decision.

MATERIALS AND METHODS

The study was carried out on twelve adult clinical cases of dogs that were brought

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to the Teaching Veterinary Clinical Complex and Department of Veterinary Surgery and Radiology, Bihar Veterinary College, Patna for removal of infected uterus.

Dogs were kept off fed for 12 hours before surgery. The animals were randomly divided into groups consisting of six animals in each group viz. Group-I and II. All the animals were injected Atropine Sulphate @ 0.04 mg/kg body weight I.M. and after 10 minutes, Xylazine Hydrochloride @ 1 mg/kg body weight I.M. with minimum forcible restraint as pre-anaesthetic.

Once the signs of sedation become evident, I.V. was maintained with slow infusion of Normal Saline Solution @ 15-20 drop/ min in two groups (I and II) and as constant rate infusion @30 mcg/kg/min of normal saline solution mixed with appropriate 2% lignocaine. After 15 minute of pre-anaesthetic administration, Propofol was administered intravenously @ 4 mg/kg body weight for induction of anaesthesia in both the groups. Then anaesthesia was maintained in group I animals with Propofol by intermittent bolus injection technique with a syringe at the dose rate of 2 mg/kg body weight each time as required when the movement was detected on the application of noxious stimuli as per Adetunji *et al.,* 2002 and Morgan and Legge 1989. In group II animals anaesthesia was maintained by Propofol at the dose rate of 2 mg/kg body weight along with 2% Lignocaine as CRI @ 30 mcg/kg/min. Dogs were maintained surgical anaesthesia plane by assessing pedal reflex, palpebral reflex, eyeball position, and jaw tone. By using a microinfusion set in CRI we had a fixed volume of fluid (60 drops = 1ml) delivered in 60 seconds (1 Min) that was 1 ml/min.

RESULTS AD DISCUSSION

The mean ±SE values of induction time after intravenous administration of propofol (Group-I), Propofol with lignocaine CRI (Group-II) in atropine – xylazine premedicated dogs were as 22.34±0.20 and 22.42 ± 0.59 seconds, respectively. The induction time was rapid in all animals. The induction time in group -I and Group-II did not differ significantly. The mean ± SE values of duration of anaesthesia in Group-I and Group-II were observed to be 16.16 ± 0.82 and 17.75 ± 0.91 minutes, respectively. The duration of analgesia was statistically significant ($p<0.05$). The present findings of group-II were insignificantly different from the group-I that mean lignocaine as CRI has little role in the variation of the duration of surgical anaesthesia. The shorter duration of recumbency in group I might be due to the induction and maintenance agent propofol which gets rapidly redistributed from the brain to other tissues and also efficiently eliminated from plasma, this may explain its short action and the rapid recovery as compared to Ketamine (Zoran *et al.,* 1993). The mean ± SE values for the recovery time in Group-I and Group-II were recorded 20.37 ± 0.21 and 22.10 ± 0.80, respectively. Non-significant recovery time was seen. The present findings of recovery time in group-I corroborated with the findings of Bayan *et al.* (2002) observed recovery time 19.92 ± 0.40 minutes during propofol anaesthesia in dogs without premedication. Nursory (2011) observed recovery time 23.83 ± 0.48 minutes during propofol anaesthesia in dogs premedicated with xylazine and atropine. Group-II were insignificantly different from the group -I that means lignocaine CRI did not bring much variation in recovery time. Rectal temperature decreased insignificantly (P>0.05) up to 15 minutes during maintenance in the groups but remained within normal physiological limit. A decreased rectal temperature during continuous infusion of propofol in dogs has also been reported by Jena *et al*. (2014). However, a minimum or non - significant decrease in rectal temperature might indicate that propofol and propofol – lignocaine CRI had minimal effect on thermoregulatory mechanism of the subject. Heart rate increased significantly (p≤0.05) up to 10 minutes after premedication and it decreased gradually till the end of observation but remained within the normal range. Increase in heart rate was observed after administration of xylazine in all groups. This is in accordance with the earlier studies in which anticholinergic atropine and glycopyrrolate were found capable of reversing alpha-2-agonist-induced bradycardia in dogs and caused tachycardia (Alibhai *et al*. 1996). Therefore, increase in heart rate after the administration of xylazine might be due to the vagolytic effect of atropine. Lignocaine did not cause much variation in group II.

Respiratory rate at 0 minute (28.05 ± 0.52^{aA}) and at 30 minute (17.84 ± 0.33^{abE}) decreased significantly (P<0.05) up to 30 minutes during maintenance and then increased. However minimum value recorded was within the physiological limits. Respiratory depression in dogs following propofol administration was also observed by Jena *et al.* (2014). It might be due to propofol causing a decrease in mean respiratory rate by the depressing central respiratory response to arterial carbon dioxide tension (Bayan *et al.* (2002). In group II respiratory rate at 0 minute (27.30 ± 0.86^{abA}) and at 30 minute 16.59 ± 0.52^{bD}) decreased significantly (P<0.05) up to 30 minutes during maintenance and then increased.

In group-I, haemoglobin level showed a significant (P<0.05) decrease with maximum fall at 60th minute (Table-1). Thereafter, it returned gradually to the pre-induction level. A significant decrease in haemoglobin has also been reported during continuous infusion propofol in dogs by Jena *et al*. (2014). The decrease in haemoglobin level in the present experiment might be due to the splenic pooling of erythrocytes that occurred with most of the other anaesthetics. It might also be due to the shifting of fluids from the extra vascular compartment to the intravascular compartment to maintain the cardiac output in animals (Church *et al.*, 1994). In group II, haemoglobin level showed insignificantly (P>0.05) decreasing trend till 30 minutes and then increased at 60 min and then decrease. The decrease in haemoglobin level might be due to the splenic pooling of erythrocytes that occur with most of the anaesthetics. In both the groups significant decrease in PCV has been recorded. The decrease in PCV in these groups might be due to pooling of circulating erythrocytes secondary to decreased sympathetic stimulation (Das, 2013) or due to inter compartmental fluid shift to normal cardiac output (Das, 2013).

In both Group I and II total erythrocyte count showed an insignificant decrease. Similar findings were reported by Nursory (2011) and Sankar *et al.* (2011) in dogs following administration. The value of TLC did not register any significant change.

In both the groups blood glucose was increased significantly at 15 minutes then insignificant increase was observed (Table-2). A similar observation was also reported by Ratnesh *et al.* (2014) during propofol or ketamine anaesthesia in xylazine premedicated dogs. An increase in serum glucose level might be due to decreased membrane transport of glucose, decreased glucose utilization, inhibition of insulin release mediated by alpha-2 receptors in pancreatic beta cells, and increased blood concentration of adrenocortical hormone (Das, 2013).

Table 1. Haematological observation

Group	0 min	15 min	30 min	60 min	120 min
			Hemoglobin (mg/dl)		
I	12.41±0.64	12.19±0.63	11.90±0.61	10.99±0.56^{c}	11.36±0.58
II	13.68±0.43	13.08±.41	12.89±0.41	13.17±0.42^{ab}	12.56±0.40
			TEC (10^6 / cu.mm)		
I	5.78±0.30	5.68±0.29	5.54±0.28	5.12±0.26^{c}	5.29±0.27
II	6.37±0.20	6.09±0.19	6.00±0.19	6.14±.19^{ab}	5.85±0.18
			PCV (%)		
I	33.13±1.70	32.55±1.67	31.76±1.63	29.33±1.50^{c}	30.32±1.55
II	36.52±1.15	34.92±1.10	34.41±1.08	35.17±1.11^{ab}	33.54±1.06
			TLC (103/cu.mm)		
I	9.96±0.72^{b}	8.79±0.63^{b}	8.68±0.62^{b}	8.98±0.65^{b}	9.93±0.67^{b}
II	11.47±0.59^{ab}	10.18±0.53^{ab}	10.05±0.52^{ab}	10.40±0.54^{ab}	10.85±0.56^{ab}

Table 2. Biochemical observation

Group	0 min	15 min	30 min	60 min	120 min
			Glucose		
I	75.38±0.65 ^{cD}	81.64±0.71 ^{bC}	82.69±0.72 ^{cBC}	84.76±0.73 ^{dAB}	86.82±0.75 ^{dA}
II	87.38±0.61 ^{aD}	94.68±0.66 ^{aC}	95.94±0.67 ^{bBC}	98.53±0.68 ^{bAB}	101.06±0.70 ^{bA}
			BUN		
I	11.47±0.42^{abC}	13.38±0.49^{abBC}	16.31±0.60^{bA}	14.36±0.53^{bAB}	12.50±0.46^{abBC}
II	13.59±0.63^{aC}	15.83±0.73^{aBC}	19.26±0.89^{aA}	17.00±0.78^{aAB}	14.87±0.69^{aBC}
			Creatinine		
I	1.12±0.05^{abC}	1.47±0.07^{aAB}	1.70±0.08^{aA}	1.51±0.07^{abAB}	1.37±0.07^{abBC}
II	1.34±0.09^{aB}	1.75±0.11^{aAB}	2.03±0.14^{aA}	1.81±0.12^{aAB}	1.64±0.11^{aAB}
			SGPT (ALT)		
I	37.92±0.38^{bC}	39.30±0.39^{bBC}	42.23±0.42^{bA}	42.52±0.42^{bA}	39.30±0.39^{bB}
II	33.18±0.73^{cB}	34.46±0.76^{cAB}	37.07±0.82^{cA}	37.35±0.82^{cA}	34.85±0.77^{cAB}

A, B, C - Values in the same row with different superscripts differ significantly (P<0.05)

a, b, c - Values in the same column with different superscripts differ significantly (P<0.05)

In group-I, significant elevation of blood urea nitrogen (BUN) was recorded at 30 minutes during total intravenous anaesthesia (TIVA) and thereafter decreased towards preanesthetic. Similar finding was also reported by Tiwari *et al.* (2006). In group-II significant elevation of blood urea nitrogen was also recorded.

In group I and Group II serum creatinine was significantly increased. The significant increase in creatinine value might be due to the temporary inhibitory effects of anaesthetic drugs on the renal blood flow leading to a decrease in glomerular filtration rate as suggested by Kinjavdekar *et al*. (2005) or due to hypotension induced by propofol anaesthesia might leading to reduced renal flow (Singh *et al.,* 2014).

No significant differences in ALT values within the groups were recorded. In the groups it was seen that more or less same pattern. The transient variation in ALT values was within physiological limits during the anaesthetic period might be indicative of the non-toxic effect of all the anaesthetic drugs on heart, liver, kidney, pancreas, brain tissue, or skeletal muscle.

CONCLUSION

In group-I, induction with propofol was rapid, recovery was smooth and rapid, maintenance of anesthesia was smooth and stable with good muscle relaxation but analgesia was poor and a brief period of apnoea was observed at the time of induction. In group-II, induction with propofol was rapid, recovery was smooth and rapid, maintenance of anesthesia was smooth and stable with good muscle relaxation but analgesia was better than group-I and a brief period of apnoea was observed at the time of induction.

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