CLINICO-PHYSIOLOGICAL ALTERATIONS AFTER EPIDURAL ADMINISTRATION OF DEXMEDETOMIDINE ALONE AND IN COMBINATION WITH BUPIVACAINE IN BUFFALO CALVES

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ABSTRACT

The present study was conducted to evaluate clinico-physiological alterations after epidural administration of dexmedetomidine alone and in combination with bupivacaine in buffalo calves. The animals of dexmedetomidine-bupivacaine group showed quick onset and longer duration and good depth as compared to animals of dexmedetomidine alone group. The recovery was smooth in both the treatments. There was significant decrease in rectal temperature in both the groups while heart rate and respiration rate was decreased non significantly.

KEYWORDS : Clinico-physiological changes, Epidural Dexmedetomidine, Epidural Bupivacaine.

INTRODUCTION

In veterinary practice most of the surgical procedures can be performed safely and humanely in ruminants by using a combination of physical restraint, mild sedation and regional anaesthesia (Edwards, 2001). Dexmedetomidine is the latest and most potent alpha-2 adrenergic receptor agonist and is the active stereoisomer of the racemic medetomidine preparations. It produces sedation, anxiolysis and sympatholysis and possess some analgesic properties without any respiratory depression (Cormack *et al.,* 2005). Bupivacaine hydrochloride is most widely used local analgesic for epidural use because of its tendency to block sensory fibres preferentially to relative sparing of motor fibers (Alvarez *et al*., 1983). In a study Kamble et al. (2016) reported that combination of dexmetedomidine and ropivacaine hydrochloride is a better option for epidural analgesia instead of alone dexmedetomidine. Rayees and Shukla (2013) reported significant haemato-biochemical changes after Bupivacaine administration in buffalo calves. The present study was conducted to evaluate the clinico-physiological changes following dexmedetomidine alone and in combination with bupivacaine as an epidural analgesia.

MATERIALS AND METHODS

The study was conducted on 12 healthy male buffalo calves, weighing between 50 to 70 kg aged 4-6 months and divided into two groups of six animals each. The animals of group 1 received dexmedetomidine (@ 5 µg/kg body wt.), while animals of group 2 received bupivacaine (@ 0.15 mg/kg. body wt.) and dexmedetomidine (@ 5 µg/kg body wt.) simultaneously through sacrococcygeal space. The clinico-physiological changes were recorded at different time intervals in both treatments.Time from injection to loss of reflexes at inguinal region was considered as time of onset of analgesia. Depth of analgesia was recorded at flank, inguinal region, hind limbs, perineum and tail by observing response to pain at a particular region and was graded by using orbital score card (Nil: no analgesia, +: mild analgesia, ++: moderate analgesia, +++: strong/complete analgesia). Time taken from loss of sensation from any region to return of sensation at all the sites was considered as duration of analgesia. Animals were observed for incoordination in gaits after epidural administration of drugs. Nil, +, ++, +++ score was given on observation of without staggering, with staggering, unable to stand and recumbency respectively. All the animals were observed for their standing recovery that is time taken for standing alert and walking without support.The rectal

temperature (ºF), heart rate (per min) and respiration rate (per min) were recorded at 0 min, 30 min, 60 min, 90 min, 3 hrs and 8 hrs intervals.

RESULTS AND DISCUSSION

The onset of analgesia was quicker (4.5 ± 0.52 minutes) in treatment 2 than (17.83 ± 1.16 minutes) in treatment 1. Kalim *et al.* (2014) reported that combination of bupivacaine and medetomidine was proved to be better as it produced analgesia with quicker onset and of longer duration. The epidural administration of dexmedetomidine alone and its combination with bupivacaine induced duration of analgesia of 30 ± 1.06 and 120.33 ± 2.99 minutes in treatment 1 and treatment 2 respectively. El Shamaa and Ibrahim (2014) suggested that use of dexmedetomidine, as an additive to the local anaesthetic bupivacaine in caudal epidural analgesia prolongs the duration of post-operative analgesia following lower abdominal as well as perineal surgery compared with caudal morphine with no side-effects on the vital signs. Dexmedetomidine alone induced mild to moderate analgesia while dexmedetomidine in combination with bupivacaine induced moderate to strong/complete analgesia. Singh *et al.* (2005) also concluded that medetomidine alone or in combination with ketamine or bupivacaine produced complete analgesia of the tail, perineum, inguinal region and upper parts of hind limbs. Animals of both treatments became recumbent but the higher score for motor incoordination was observed in treatment 2. Singh *et al.* (2005) suggested that motor incoordination was mild to moderate in animals of medetomidine-bupivacaine group. In the present study complete recumbency in both groups might be due to more potent effect of dexmedetomidine than medetomidine and xylazine.

Table 1:- Mean level ± SE of rectal temperature (ºF), respiration rate (per minute) and heart rate (per minute) at different time intervals following epiduralanalgesia in buffalo calves in two treatment groups.

Groups	Time (minutes)	Temperature (ºF)	Respiration rate (per minute)	Heart rate (per minute)
Group I	0	100.74 ± 0.19	21.16 ± 0.60	58.66 ± 0.88
	30	97.14 ± 0.16*	20.00 ± 0.57	54.33 ± 2.56
	60	97.97 ± 0.24*	18.83 ± 0.94	53.83 ± 1.97
	90	98.34 ± 0.23*	18.33 ± 1.20	52.50 ± 2.45
	180	99.23 ± 0.19*	18.66 ± 1.25	52.83 ± 2.31
	480	100.23 ± 0.16	19.83 ± 0.70	54.16 ±1.85
Group II	0	100.61 ± 0.20	21.66 ± 0.49	54.66 ±3.91
	30	97.39 ± 0.17*	18.66 ± 0.49*	49.33 ±4.43
	60	96.99 ± 0.16*	17.33 ± 0.42*	49.16 ± 2.93
	90	98.32 ± 0.15*	17.00 ± 0.36*	49.66 ±4.14
	180	98.88 ± 0.14*	17.66 ± 0.49*	51.33 ±4.58
	480	100.49 ± 0.17	21.33 ± 0.49	53.16 ± 4.06

shows significance at 5 % level as compared to 0 hour value ($P<0.05$)

The order of complete recovery was 71.16 ± 2.21 and 174.66 ± 3.05 minutes in treatment 1 and 2 respectively. The recovery was smooth in both treatments. Dexmedetomidine induced sedation seems to be partially induced by endogenous sleep pathway, which explains why medetomidine or dexmedetomidine sedated veterinary and human patients can easily be aroused from normal sleep (Nelson *et al*., 2003). From table 1 it is revealed that rectal temperature decreased significantly ($P<0.05$) in both treatment groups between 30 to 180 minutes.The heart rate decreased non significantly in both treatments. Bradycardia following administration of bupivacaine alone might be due to a generalized decrease in sympathetic activity (Lumb and Jones, 1984). The respiration rates showed non significant decreasing pattern in treatment 1. In treatment 2, respiration rate decreased significantly ($P<0.05$) from 18.66 ±0.49 to 19.66 ± 0.49 between 30 to 180 minutes and minimum value 17.00 ± 0.36 was observed at 90 minutes. Administration of medetomidine or dexmedetomidine has been found to depress the medullary centre resulting decreased respiratory rate with minimal effect on blood gases in dogs (Amarpal *et al.,*1996; Kuusela *et al.,* 2000). There was mild salivation in treatment 1 and treatment 2 animals. Salivation was maintained upto 20 to 25 minutes.

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