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Caudal epidural analgesia in calves using different local analgesics

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Abstract

Analgesic effects were conducted with 2% lignocaine hydrochloride (LH), 2% lignocaine hydrochloride with adrenaline (LHA) and 0.5% bupivacaine hydrochloride (BH) in calves of both sexes. Analgesic drugs were injected into caudal epidural space in 30 apparently healthy calves of below one year of age. A total of 30 analgesic trials were done, where heart rate, respiration rate and rectal temperature were monitored. Heart rate and respiratory rate significantly ($P<0.05$) decreased from preanalgesic values, during caudal epidural analgesia period with 0.5% bupivacaine hydrochloride. Heart rate increased significantly ($P<0.001$) from preanalgesic values when 2% lignocaine plus adrenaline was used. Only lignocaine hydrochloride showed a short onset, rapid spreading without having any side effect. Duration of analgesia was prolonged with 0.5% bupivacaine hydrochloride compared to other analgesic agent. Drowsiness, tympany and shivering were observed when 0.5% bupivacaine hydrochloride was used. Bupivacaine seemed to be better for longer duration of analgesia compared to lignocaine. But lignocaine hydrochloride may be used for rapid onset of analgesia in calves.

Keywords: Analgesia, Onset, Caudal epidural, Calves

Introduction

The majority of the surgical procedures in ruminants are done using local analgesics where epidural analgesia is commonly used. There are very few reports of epidural analgesia in calves in Bangladesh. Therefore, the objectives of the study were: a) to study the action of various local analgesics in caudal epidural analgesia calves b) to investigate the effect of various analgesics on the clinical parameters in calves and c) to compare the effect of 2% lignocaine hydrochloride, 2% lignocaine hydrochloride with adrenaline and 0.5% bupivacaine hydrochloride in caudal epidural analgesia.

Materials and Methods

A total of 30 clinically healthy calves (20 males and 10 females) of below one year of age were used. Two percent lignocaine hydrochloride (Jasocaine®, Jayson Pharmaceuticals Ltd. Bangladesh), 2% lignocaine hydrochloride with 0.0005% adrenaline (Jasocaine-A®, Jayson Pharmaceuticals Ltd. Bangladesh), 0.5% bupivacaine hydrochloride (Ultracaine®, Jayson Pharmaceuticals Ltd. Bangladesh), at the dose rate 3 ml were administered in group A, B and C, respectively

Caudal epidural analgesia was produced in calves ($n= 10$) by injecting local analgesic into the sacrococcygeal space using a 6-cm long, 18 -gauge needle at an angle of about 45° to the skin surface, directed anteriorly and ventrally to a depth of about 2 cm and the analgesic agents was pushed. Respiration rate, heart rate and rectal temperature were monitored before administration and at 5 min. and 10 min. after recovery from analgesia.

Analgesic agents were injected in calves in the standing position. The state of analgesia was observed in every 5 min. with the help of a needle by pricking in the region. Analgesia was assessed as 1-excellent (no response), 2-adequate (slight movement or reflex response), 3-poor (avoidance response) to needle pricking. Onset and duration of analgesia were recorded. The desensitized area was measured by a scale. The measurement was carried out up to recovery. Side effects were closely observed and recorded during the analgesic period. Care was taken not to excite the animal before and during monitoring.

Student's t-test was performed to compare the obtained data before and after analgesia. Analysis of variance (ANOVA) in completely randomised design was carried out for significant variation of analgesic effects in among different groups and significant difference was done for mean differences.

Results and Discussion

Effects of various analgesic agents on clinical parameters

Before analgesia with 2% lignocaine hydrochloride, the mean values of heart rate, respiration rate and rectal temperature were 76.0 ± 4.2 /min, 25.8 ± 3.4 /min, 102.9 ± 0.6 $^{\circ}$ C, respectively. The heart rates significantly ($p < 0.01$) decreased but there was no variation in case of respiration rate and rectal temperature during 5 min after analgesia as compared to pre analgesic values and at that time, the mean values of heart rate, respiration rate and rectal temperature were 68.2 ± 6.3 /min, 27.8 ± 9.1 /min, 103 ± 1.1 $^{\circ}$ C, respectively (Table 1). In 0.5% bupivacaine hydrochloride anaesthesia, the mean values of heart rate, respiration rate and rectal temperature before anaesthesia were 77.6 ± 2.6 /min, 27.6 ± 3.6 /min and 102.4 ± 0.9 $^{\circ}$ F, respectively. The same parameters were monitored during 5 min after analgesia and during 10 min after analgesia (Table 2). The heart rate was significantly ($p < 0.05$) decreased and respiration rate was insignificantly decreased.

Table 1. Effects of various local analgesics on heart rate, respiration rate and rectal temperature during caudal epidural analgesia

Calves N=10/ groups	Before analgesia			5 min after giving local analgesic			10 min after giving local analgesic		
	Heart rate	Res. rate	Rectal temperature	Heart rate	Res. rate	Rectal temperature	Heart rate	Res. Rate	Rectal temperature
A(LH)	76.0 ± 4.24	25.8 ± 3.49	102.9 ± 0.58	68.2 ± 6.34**	27.8 ± 9.12	103.3 ± 1.06	73.2 ± 5.72	30.2 ± 7.29	102.9 ± 1.17
B (LHA)	74.0 ± 8.34	32.6 ± 11.61	103.2 ± 0.65	80.8 ± 7.29**	36.2 ± 12.85*	102.5 ± 0.60	82.8 ± 7.16**	34.0 ± 8.80	102.3 ± 1.30
C(BH)	77.6 ± 2.61	27.6 ± 3.58	102.4 ± 0.88	71.8 ± 4.71*	22.8 ± 3.44*	100.5 ± 0.72	68.4 ± 5.55*	24.6 ± 1.95	102.4 ± 0.83

LH-lignocaine hydrochloride, LHA-lignocaine hydrochloride with adrenaline, BH-bupivacaine hydrochloride ** Significant (<0.01)
* Significant (<0.05)

In caudal epidural analgesia, 0.5% bupivacaine hydrochloride and lignocaine hydrochloride (2%) significantly decreased heart rates. This finding is in agreement with the earlier studies (Kinjavdekar and Pratap, 2002). When 2% lignocaine hydrochloride was used, respiration rates decreased insignificantly. This observation corresponds with the study of the earlier investigators (Hossain and Kumar, 1988). In this study, rectal temperature in calves of all groups decreased. There is no report on the effect of local analgesic agents on body temperature. Since, the spinal nerve always carries sympathetic fibre, peripheral nerve block always produces vasodilatation (Hall and Clarke, 1989). There is usually fall in body temperature due to peripheral vasodilatation in the area of blockade.

In 2% lignocaine with adrenaline analgesia, the mean values of heart rate, respiration rate and rectal temperature before anaesthesia were 74.0 ± 8.3 /min, 32.6 ± 11.6 /min, 103.2 ± 0.7 $^{\circ}$ C respectively. Heart rate and respiration rates were significantly ($p < 0.01$) increased during 5 min after analgesia as compared to preanalgesic values but temperature decreased insignificantly (Table 1). This observation corresponds with the study of Luna *et al.*, (2004). Generally, adrenaline in local analgesic solution causes peripheral vasoconstriction. Several compensating physiological mechanisms are activated when a partial sympathetic blockade occurs. There is an increase in heart rate as a compensating mechanism to maintain cardiac output and blood pressure (Soma, 1971). Adrenaline increases heart and respiration rates by activating α and β receptors (Brander *et al.*, 1991).

Onset, extent and duration of analgesia

In Group A, the mean values of onset, area of desensitization and duration of analgesia were 3.2 ± 0.8 min, 18.9 ± 2.9 cm and 31.2 ± 10.8 min, respectively. With Group B, the mean values of onset, area of desensitization and duration of analgesia were 7.4 ± 1.8 min, 7.2 ± 0.7 cm and 20.8 ± 5.5 min, respectively and the mean values of onset, area of desensitization and duration of analgesia in Group C were 6.8 ± 1.5 min, 17.1 ± 2.4 cm and 31.8 ± 5.7 min, respectively (Table 2). The onset was short in Group A, but it had more area of desensitization and Group C had long duration.

Table 2. Effects of various local analgesics during caudal epidural analgesia in calves

Calves N=10/ groups	Amount of agents (ml)	Onset of analgesia (min) (MN±SD)	Desensitized area (cm) (MN±SD)	Duration (min) (MN±SD)
A(LH)	3	3.2±0.84 ^b	18.9±2.92 ^a	31.2±10.76 ^a
B (LHA)	3	7.4±1.82 ^a	7.2±0.70 ^b	20.8±5.45 ^b
C(BH)	3	6.8±1.48 ^a	17.1±2.41 ^a	31.8±5.67 ^a

LH-lignocaine hydrochloride, LHA- lignocaine hydrochloride with adrenaline, BH- bupivacaine hydrochloride ** Significant (P<0.01), a,b,c-Significant difference (LSD 1%)

Different row showing superscript dissimilar letter's differed significantly

Significant (P<0.01) differences were found in the onset of analgesia, extent of analgesia and duration of analgesia after caudal epidural injection of three different analgesic agents with the same dose in different calves. These findings were supported by Caulkett *et al.* (1993) and Lewis *et al.* (1999). The major reason for variation might be due to the protein binding characteristic of analgesic agents that preliminary influences the duration of action of anaesthetic agents (Gissen *et al.*, 1980). During epidural analgesia, the onset was rapid in case of 2% lignocaine hydrochloride, whereas the onset was slower in case of 0.5% bupivacaine hydrochloride and lignocaine hydrochloride with adrenaline. Howel *et al.* (1990) observed that the onset of analgesia was slow with 0.5% bupivacaine hydrochloride and the onset was more rapid with 2% lignocaine hydrochloride, while Trim (1989) stated that lignocaine hydrochloride with adrenaline had a more rapid onset than bupivacaine hydrochloride. The onset of epidural analgesia is generally governed by the rate of disappearance of anaesthetic solution from the site where absorption of the analgesic agents through blood and lymphatic channel, duramater, epidural fat were involved. The protein binding characteristics of local analgesic agents influence the duration of action (Hall and Clarke, 1989). The duration of analgesia with 0.5% bupivacaine hydrochloride was significantly longer than 2% lignocaine hydrochloride. This observation supports the previous findings (Grubb *et al.*, 1992).

Tympany, shivering and drowsiness were found in Group C after using 0.5% bupivacaine hydrochloride. Muscle tremor was present after using 2% lignocaine hydrochloride with adrenaline. These side effects were not observed in all animals. The variability among patients in their responses to local anaesthetic toxicity accounts for these side effects (Laishley *et al.*, 1988).

It is suggested that 2% lignocaine hydrochloride is suitable for minor surgery but 0.5% bupivacaine hydrochloride may be used if a longer time of analgesia is required for surgery. However, lignocaine plus adrenaline should be avoided in case of cardiac insufficiency.

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