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## Comparative Effects of Different Local Analgesics and Ketamine on Clinical Parameters for Cranial Epidural Analgesia in Black Bengal goats

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**Abstract**

The research was directed to exposing the effect of analgesics on clinical parameter for cranial epidural analgesia in black Bengal goats. A series of thirty-two (32) analgesic trials were conducted in goats aged between 8 to 12 months and with an average body weight of 8.1 kg. Goats were split into four groups (n=4) and a replication of eight trails was implemented in every group at most one week gap. Lignocaine hydrochloride (2%, 6 mg/kg), lignocaine hydrochloride with adrenaline (2%, 6 mg/kg), bupivacaine hydrochloride (0.5%, 1.5 mg/kg) and ketamine hydrochloride (5 mg/kg) were used to perform cranial epidural analgesia. Heart rate, respiration rate, and rectal temperature were examined. Lidocaine hydrochloride significantly (P<0.01) declined the heart rate and significantly (P<0.05) raised respiration rate during cranial epidural analgesia, whereas lidocaine hydrochloride with adrenaline significantly (P<0.01) raised the heart and respiration rate. Bupivacaine hydrochloride non-significantly (P>0.05) raised heart rate and significantly (P<0.01) down turned the respiration rate while ketamine hydrochloride significantly (P<0.01) increased the heart and respiration rate. All anaesthetic agents significantly (P<0.01) declined the rectal temperature during cranial epidural analgesia. The effects of these analgesics on the onset time of anaesthesia, peak time of anaesthesia, area of desensitization and duration of anaesthesia, biochemical and hematological parameter analysis, rumen motility measurement and other tests for enteric response were not performed especially during cranial epidural analgesia in this experiment, this would be of interest in future studies.



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## Introduction

Bangladesh is an agriculture based country. Livestock has a direct contribution of 2.95% of agricultural Gross Domestic Product (GDP) in the country's economy. Among ruminants, goats comprise 17.46 million, which occupies the second largest position in livestock [1]. The leather and leather goods of black Bengal goats have a supreme position in the international leather market, which contributing about 6 to 7 percent of total export earnings. Sometimes, epidural anesthesia is useful to implement main surgeries for the treatments of goat diseases. Anaesthesia is one of the miracles of medicine, without which modern surgical techniques would have been impossible. It was first developed to alleviate pain and provide relaxation for surgery. It is employed in animals for a wide variety of operative interventions. The choice of different types of anaesthesia, use of anaesthetic and analgesic agents and route of administration of anaesthetic agents all depend on the animals as well as patients.

For the treatment of goat diseases, general anaesthesia often leads to tympanitis or regurgitation, which is threatened for life [2]. In ruminants, the occurrence of regurgitation or gastro-esophageal reflux of rumen material and its subsequent inhalation during general anaesthesia has been widely reported [3]. Since ruminants are amenable to physical restraint, most of the regular surgical procedures can be performed using local anesthetics [4]. These procedures are uncomplicated, secure, and cost-effective and need not particular machinery. Among many types of local and regional anesthesia, epidural anesthesia has been practiced in veterinary medicine for the correction of surgical interventions, e.g., tail removal, amputation of the udder, rectal examination, abscess opening and others.

Injection of anaesthetic and analgesics within the spinal canal, but outside the dura mater is termed as epidural anaesthesia. Epidural anaesthesia was first reported in dogs by Cathelin in 1901, but due to the larger anaesthetic requirement and the toxicity of cocaine, it was not used until 1921 [5].

The drug so injected, temporarily paralyses the spinal nerve roots, its contacts, resulting in analgesia of tissues innervated by the nerves [6]. It is classified according to the site at which the injection is made, i.e., cranial (high) and caudal (low). The use of epidural analgesia in veterinary medicine is most common in large animal practice. The method is also frequently used in small animals [7].

Troncy et al. [8] stated that in spite of the availability of a number of local anesthetics, lidocaine hydrochloride, and bupivacaine hydrochloride are being predominately administered for epidural anesthesia. The local analgesic agents vary in their potency, duration of action and cost. Lignocaine is an amide-type local analgesic and has a comparably fast induction and a moderate span ranged from one to two hours [9, 10]. Bupivacaine produces an expanded period of analgesia, four times more robust than lidocaine and is practiced mostly for regional and epidural analgesia [11]. The effect of a local analgesic is increased by the addition of vasoconstrictor agents. Adrenaline is commonly used for elongation of local analgesia [5]. Adrenaline indiscriminately blocks the sensory, sympathetic and motor fibers [12]. Ketamine hydrochloride, a dissociative drug has been practiced epidurally to mitigate pain before and after the operation. Spontaneous, unprovoked movements may occasionally be occurred in deeply anaesthetized patients [13]. In this research, the effect of analgesics on the clinical parameter of cranial epidural analgesia in black Bengal goats was evaluated.

## Materials and Methods

### Management of experimental animals

The proposed trial was carried out in four groups; each group contained eight goats (*Capra hircus*) of both sexes. These animals were apparently healthy, especially during the experiment. Their weight was 7.5 kg to 8.6 kg and their ages were 8 months to 1 year. A total of 32 experimental trials were performed in these animals to study the effect of certain local anaesthetics in the cranial (Lumbo-sacral) epidural space. The work was executed in the

operation theater of the veterinary clinics, Sylhet Agricultural University, Sylhet-3100. The period of the experiment was about six months starting from March 2011 to August 2011. The ethical guidelines of animal handling and experimentation advised by the animal care committee of the Sylhet Agricultural University, Bangladesh were strictly followed.

The animals were kept under good hygienic condition. They were grazed in the grassland for 6 hours every day and had a free access to water *ad libitum*. Standard concentrate feed was supplied. Deworming and vaccination were performed. The animals were frequently examined to detect any pathological condition.

### Preparation of the animals

The goats were taken to the operation theater 20 to 30 minutes prior to administration of analgesic or anaesthetic agents. Age, sex and body weight were recorded before starting the experiment. The body weight of each of the animals was taken by using the weighing machine. The animals were then examined clinically to find any pathological changes. The animals were laid in lateral recumbency and were controlled physically by an assistant and also casting by ropes. Clipping, shaving, and painting with tincture of iodine were chronologically performed before any injection. The anaesthetic injection was given without premedication. The analgesia was always performed in the morning throughout the course of the investigation.

### Anaesthetic agents

#### *Lignocaine hydrochloride (2%)*

Lignocaine HCl (Jasocaine<sup>®</sup>, Jayson Pharmaceuticals Ltd.) is widely used a local analgesic agent which is available in Bangladesh in 50 ml vial. Each milliliter contains 20 mg Lidocaine HCL (an HDD.).

#### *Lidocaine Hcl including 0.0005% adrenaline (2%)*

Lidocaine HCl (2%) including 0.0005% adrenaline (Jasocaine A<sup>®</sup>, Jayson Pharmaceuticals Ltd.) is available in 50 ml vial. Each milliliter contains 20 mg Lidocaine and 5 µg adrenaline.

#### *Bupivacaine HCl (0.5%)*

Bupivacaine HCl (Ultracaine<sup>®</sup>, Jayson Pharmaceuticals Ltd.) is a local anesthetic of an amide group available in Bangladesh in 30 ml vial.

Each milliliter contains 5 mg Bupivacaine hydrochloride (anhydrous).

#### *Ketamine hydrochloride (a dissociative anaesthetic agent)*

Ketamine hydrochloride (G-KETAMINE<sup>®</sup>, Gonoshasthaya Pharmaceuticals Ltd., Dhaka) is available in Bangladesh in 10 ml vial. Each milliliter contains 50 mg ketamine.

### Methods of cranial epidural analgesia

Epidural analgesia is a useful technique in veterinary anaesthesia. Epidural analgesia is produced by injecting the anaesthetic solution into the epidural space. In this experiment, a completely randomized design was used. The animals were divided into four different groups. In each group, eight experimental trials were accomplished. To mitigate the possible development of resistance to anaesthetic agents, a replication of the trails was made at least one-week interval. Studies in these goats were carried out in the absence of surgical stress. Laterally, restraining of the goats on the operating table, the back was flexed and the hind limbs were held forward. The site of injection was immediately posterior to the last lumbar spine. Clipping, cleaning, and disinfection were performed on the injection site. A 3 inch 18 gauge needle was pricked at the lumbosacral space and the analgesic solutions including distilled water for control were injected. The drugs were allocated for epidural analgesia as described in Table 1.

**Table 1** Doses and concentration of drugs used for cranial epidural analgesia.

Groups	Concentration and doses of drugs
A	3 ml 2% Lignocaine hydrochloride (Jasocaine <sup>®</sup> )
B	3 ml 2% Lidocaine hydrochloride including 0.0005% adrenaline (Jasocaine A <sup>®</sup> )
C	3 ml 0.5% Bupivacaine hydrochloride (Ultracaine <sup>®</sup> )
D	1 ml Ketamine hydrochloride (G-KETAMINE <sup>®</sup> )

### Duration of anesthesia

The state of analgesia was observed in every 5 minutes with the help of a needle, by pricking the region. Analgesia was assessed as “+++” excellent (no response), “++” adequate (slight movement or reflex response), “+” poor (avoidance response) to needle pricking. Meantime duration from onset of analgesia to recovery from analgesia was 75.86, 70.63, 99.63 and 28.29 min after treating with 2%

Lignocaine hydrochloride, 2% Lidocaine HCl including 0.0005% adrenaline, 0.5% Bupivacaine hydrochloride, and ketamine hydrochloride, respectively.

### **Monitoring of clinical parameters**

#### *Heartbeat*

Heart rate was counted 10 minutes before administration of the analgesic agent, during anaesthesia and 10 minutes after recovery of anaesthesia from indirect auscultation of heart after placing the stethoscope between the 4<sup>th</sup> and 5<sup>th</sup> intercostal space.

#### *Respiration rate*

Respiration rate was recorded 10 minutes before administration of the analgesic agent, during anaesthesia and 10 minutes after recovery of anaesthesia from indirect auscultation of respiration after placing the stethoscope between the 4<sup>th</sup> and 5<sup>th</sup> intercostal space and counting the abdominal movements simultaneously.

#### *Rectal temperature*

Rectal temperature was recorded 10 minutes before administration of analgesic agents, during anaesthesia and 10 minutes after recovery of anaesthesia by a digital clinical thermometer (°F) into the anus and was kept for 1 minute.

### **Statistical analysis**

Student's paired t-test was performed to compare the obtained data before and after anaesthesia. Analysis of variance (ANOVA) was carried out according to Steel and Torrie [14] to test significance variation among the effects in the different time interval. The results were analyzed by the least significant difference test in MSTAT software.

## **Results**

The results of analgesics on heartbeat, the rate of respiration and body temperature in different groups of goats are presented in Table 2-4, respectively. Before anaesthesia, the mean values of heartbeat, rate of respiration and body temperature were 79.88 per min, 26.75 per min and 102.19°F, respectively in group A. The heartbeat and body temperature were significantly decreased during anaesthesia as compared to pre anaesthetic values and that time the

mean values of heartbeat and body temperature were 74.5 per min and 101.81°F, respectively. After recovery, the mean values of heartbeat and body temperature were 80.75 per min and 102.0°F, respectively. The heartbeat was increased non-significantly compared to pre anaesthetic value. The body temperature was decreased significantly compared to pre anaesthetic value. The mean values of rate of respiration during and after anaesthesia were 28.25 per min and 27.13 per min, respectively. The rate of respiration during anaesthesia was increased significantly and after anaesthesia was increased non-significantly compared to pre anaesthetic values.

In group B, the mean values of the heartbeat, the rate of respiration and body temperature before anaesthesia were 77.13 per min, 26.25 per min and 102.66°F, respectively. The heartbeat and rate of respiration were significantly ( $P < 0.01$ ) increased during anaesthesia compared to pre anaesthetic values and that time the mean values of the heartbeat and rate of respirations were 83.0 per min and 28.5 per min, respectively. After recovery, the mean values of the heartbeat and rate of respiration were 79.38 per min and 28.63 per min, respectively. The heartbeat and the rate of respiration both showed a significant increase compared to pre-anaesthetic values. The mean values of body temperature during and after anaesthesia were 102.49°F and 102.55°F, respectively. The body temperature was decreased significantly during anaesthesia and decreased insignificantly after anaesthesia compared to pre-anaesthetic values.

In group C, the mean values of the heartbeat, the rate of respiration and body temperature were 77.5 per min, 32.25 per min and 102.95°F, respectively before anaesthesia. The rate of respiration and body temperature were significantly decreased during anaesthesia compared to pre-anaesthetic values and that time the mean values of rate of respiration and body temperature were 29.13 per min and 102.58°F, respectively. After recovery, the mean values of rate of respiration and body temperature were 30 per min and 102.78°F, respectively, which showed a significant decrease compared to pre-anaesthetic values. The mean value of heartbeat during and after

**Table 2** Effects of anaesthetic and analgesic agents on per minute heartbeat of goats.

Group	Before anaesthesia	During anaesthesia	After recovery
A	79.88±5.47	74.5±4.67**	80.75±3.06
B	77.13±3.45	83.0±4.37**	79.38±3.62*
C	77.5±4.33	77.63±3.04	76.63±3.57 <sup>ns</sup>
D	73.75±5.47	80.88±4.81**	78.88±5.07**

**Table 3** Effects of anaesthetic and analgesic agents on the rate of per minute respiration of goats.

Group	Before anaesthesia	During anaesthesia	After recovery
A	26.75±3.03	28.25±2.74*	27.13±2.71
B	26.25±3.45	28.5±2.49**	28.63±1.93**
C	32.25±2.81	29.13±1.88**	30.0±1.97*
D	31.63±2.16	35.0±2.96**	33.0±2.67

**Table 4** Effects of anaesthetic and analgesic agents on the body temperature (°F) of goats.

Group	Before anaesthesia	During anaesthesia	After recovery
A	102.19±0.36	101.81±0.41**	102.0±0.40*
B	102.66±0.23	102.49±0.22*	102.55±0.22
C	102.95±0.29	102.58±0.23**	102.78±0.24*
D	102.78±0.25	102.4±0.36**	102.39±0.27*

\* = significant (P<0.05) ; \*\* = highly significant (P<0.01)

anaesthesia was 77.63 per min and 76.63 per min, respectively, and this difference was statistically non-significant.

In group D, the mean values of the heartbeat, the rate of respiration and body temperature were 73.75 per min, 31.63 per min and 102.78°F, respectively, before anaesthesia. The mean values of the heartbeat and rate of respiration during anaesthesia were 80.88 per min and 35.0 per min, respectively, which showed a significant increase (P<0.01) compared to pre-anaesthetic values. After recovery, the mean values of the heartbeat and rate of respiration were 78.88 per min and 33.0 per min, respectively. The heartbeat was increased significantly whereas the rate of respiration was increased non-significantly compared to pre-anaesthetic values. The mean values of body temperature during and after anaesthesia were 102.4°F and 102.39°F, respectively. During and after anaesthesia, the body temperature was decreased significantly compared to pre-anaesthetic values.

## Discussion

The results showed that both heartbeat and body temperature were dropped in cranial (lumbosacral) epidural analgesia. The rate of respiration was increased in cranial epidural analgesia. Lidocaine HCl including adrenaline and ketamine hydrochloride

increased heartbeat and rate of respiration in experimental animals. Bupivacaine hydrochloride raised the heartbeat and lessened the rate of respiration. Though the impact of vascular intake has been overlooked, it is well settled that the effect of local anesthetic on clinical parameters during epidural analgesia result from both local and systemic absorption [5]. The veins composing venous plexus are comparatively large and afford a large vascular surface for rapid vascular intake [15]. In cranial epidural analgesia with lignocaine hydrochloride, heartbeat and temperature were lowered whereas De Rossi et al. [16] observed that the heartbeat, respiratory rate, and blood pressure were stable after lidocaine produced analgesia. It increased the rate of respiration whereas Hossain and Kumar [17] found decreased respiration and heartbeat in epidural analgesia. It can be overlooked as lidocaine has a short time action [18]. Xylazine combined with lidocaine hydrochloride-induced a decrease in the heartbeat and respiratory rates, but not in blood pressure [16]. In another study, the lidocaine produced analgesia showed average heartbeats and respiratory rates while the body temperature was low [19].

The use of lignocaine hydrochloride with adrenaline increased the heartbeat and rate of respiration. There is no such information in the

literature. Adrenaline is used as a vasoconstrictor agent. It acts on both  $\alpha$  and  $\beta$  receptors on sympathetic fibers [13]. Usually, adrenaline in local analgesic solution creates constriction of the peripheral vessel. Many balanced physiological pathways are triggered when a partial sympathetic blockade ensues. There is a hike in the heartbeat as a balanced mechanism to maintain cardiac output and blood pressure [20]. Adrenaline raises heartbeat and rate of respirations by stimulating  $\beta$  receptors [13].

In the present study, bupivacaine hydrochloride boosted the heartbeat and dropped the rate of respiration. Conversely, a decrease in heartbeat was recorded after epidural administration of medetomidine or bupivacaine [21], though Gill et al. [22] found an unchanged heartbeat, the rate of respiration and body temperature using 0.5% bupivacaine HCl in epidural analgesia. In the cranial epidural, analgesia with 0.5% bupivacaine HCl decreased the rate of respirations and in the caudal epidural; analgesia also decreased the rate of respiration. The bradycardial effect recorded in this study might be due to blockade of sympathetic nerves. Since spinal nerve always carries sympathetic fibers, blockade of the sympathetic nerves occurs first [20]. Decreased rate of respiration might consequence from their weaken action on respiratory center in central nervous system [23].

When ketamine hydrochloride was used, heartbeat and rate of respirations were increased. The Same result was observed by Kinjavdekar et al. [24]. In earlier studies [25], spinal application of ketamine was always linked with rising in the rate of respiration in dogs and goats. But controversy remains as Aithal et al. [26] and Kinjavdekar and Singh [27] observed no significant change in the rate of respiration, heartbeat and body temperature during epidural administration of ketamine hydrochloride. Dadafarid and Najafpour [28] reported that the heartbeat increased significantly with ketamine hydrochloride and the respiratory rate showed a decline with ketamine trail. Pathak [29] reported a significant decrease in the rate of respiration after epidural injection of xylazine and ketamine combination. The initial bradycardia followed by gaining in heartbeat might be due to the effect of

ketamine on the cardiovascular system [30]. The exact cause is unknown. Increased heartbeat and rate of respiration might result from their stimulant action on centers controlling the outflow in sympathetic nerves. This accounts for tachycardia and bronchodilator action [13].

In this research, the body temperature in goats of all groups diminished. As the spinal nerves transmit sympathetic fiber peripheral nerve desensitization induces vasodilatation [23]. A decline in temperature is due to dilatation of peripheral vessels in the desensitization region. The immobility and inability to shiver also contributed to the heat loss during epidural analgesia [20]. Díaz and Becker [31] reported a decline in temperature due to dilatation of peripheral vessels in the desensitization region and from weakening of the hypothalamic center for heat regulation. Kumar and Singh [30] suggested that these changes are because of suppressing the impact of anesthetics on metabolism.

## Conclusions

During cranial epidural analgesia, 2% lidocaine HCl including adrenaline and ketamine HCl significantly increased the heartbeat and respiration rate. Lignocaine HCL (2%) with adrenaline produced straining and muscle tremor. Shivering and drowsiness were recorded after using 0.5% bupivacaine HCL. Tympany, excitement, and muscle tremor were observed after using ketamine HCL. No side effect was found with 2% lignocaine HCL. So 2% Lignocaine HCL is safer than other local analgesics for cranial epidural analgesia.

## Conflict of interest

The authors declared that they have no conflict of interest.

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