CAUDAL ANAESTHESIA FOR PAEDIATRIC PATIENTS: 
COMPARISON BETWEEN LOCAL ANAESTHETIC MIXTURE ALONE AND LOCAL ANAESTHETIC MIXTURE WITH CLONIDINE 
(1 µg/Kg)

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ABSTRACT

Background & Objectives: To evaluate the effects of clonidine as an adjuvant to local anesthetic mixture, for caudal anesthesia, in pediatric patients undergoing sub umbilical surgeries. 

Material & Methods: Fifty ASA –I and II, children from age group of 1 year to 6 years, from either sex, were selected for study. Patients were randomly divided into two groups of 25 each: Clonidine group received: Local anaesthetic mixture + Inj. Adrenaline + Inj. Clonidine (1µg/kg). While in Control group received: Local anaesthetic mixture + Inj. Adrenaline through caudal epidural route. Onset of blockade, hemodynamic and respiratory parameters, sedation and postoperative analgesia were observed.

Results: Onset of sensory and motor blockade was significantly earlier in Clonidine group (sensory blockade: 1.32±0.39 minutes, motor blockade: 1.92 ± 0.31 minutes), than control group (sensory blockade: 3.02 ± 0.36 minutes, motor blockade: 3.70 ± 0.45 minutes) (‘p’<0.05). Peak effect was achieved significantly earlier in Clonidine group(sensory: 4.76±0.50 minutes, motor: 8.02 ±0.6 minutes ), than control group(sensory: 8.46 ± 0.91 minutes, motor: 10.96 ±0.78 minutes). (‘p’<<0.05). Duration of post operative analgesia was significantly longer in Clonidine group (444 ± 54.77 minutes), as compared to Control group (177 ± 36.66 minutes). 

Conclusion: we conclude, clonidine as an adjuvant to local anesthetic solution, for caudal block significantly shortens time for onset and peak effect for sensory and motor blockade, prolongs duration of analgesia.
KEY WORDS: Caudal anaesthesia, clodine, Lignocaine, Bupivacaine.

INTRODUCTION
Currently, paediatric pain relief is recognized as a very important issue all over the world. There have been various methods for providing post operative analgesia in paediatric patients, each with its own advantages and disadvantages. Out of these, caudal epidural anaesthesia is recognized as a very effective and safe technique for sub umbilical surgeries. But with Single shot caudal epidural, duration is not long enough to provide postoperative analgesia for long duration. This mandate use of either catheter technique or addition of adjuvants to prolong the duration of post operative analgesia.

Traditionally, opioids have been used as adjuvants to local anaesthetics by neuraxial route. The use of clonidine as an alternative to epidural opioids offers several potential benefits. Clonidine does not have respiratory depressant effects and the incidence of vomiting and pruritus is less frequent compared with that seen after administration of epidural morphine.[1]

Clonidine, a mixed $\alpha_2/\alpha_1$ adrenoreceptor and imidazoline receptor agonist, is a lipophilic drug. Safety and efficacy of clonidine by caudal route in paediatric age has been proved by previous studies. They found that clonidine prolongs duration of post operative analgesia without significant adverse effects.[2,3,4] With this background, we undertook this study to evaluate the effects of addition of clonidine (1µg/kg) to local anaesthetic mixture for caudal epidural in paediatric age group for sub umbilical surgeries.

MATERIAL AND METHODS
In our prospective, randomized control study, 50 patients of either sex; in age group of 1 year to 6 years; of ASA physical status I and II, weighing less than 20 kg, posted for sub umbilical surgeries were selected. After approval from institutional ethical committee, this study was carried out at S.S.G. Hospital, Vadodara during the year 2009-2010.

[A] Pre-operative assessment
All the patients selected were subjected to a thorough pre-anaesthetic check-up. Routine investigations like hemogram, and urine examination and weight of patient were asked for. Parents were explained about the technique of anaesthesia in their language. Informed written consent of them was obtained.
Inclusion Criteria
1. Age group of 1 to 6 years 
2. Sub umbilical surgeries. 
3. ASAPS I and II. 
4. No coagulation abnormalities. 
5. No history of cardiac, respiratory, or neurological disease. 
6. No known hypersensitivity to local anaesthetics or clonidine.

Exclusion Criteria
1. Patients with h/o allergy to local anaesthetics. 
2. Patients with coagulation abnormalities. 
3. Patients with infection at local site of caudal epidural puncture. 
4. Patients with grossly abnormal sacral anatomy. 
5. Patients with spinal or neurological disease 
6. patients with h/o convulsions 
7. Patients of ASAPS III and above. 
8. Patients with cardiovascular disease or hemodynamic instability 
9. Patients with respiratory disease.

[B] Anaesthetic Management
All Patients were kept nil by mouth for 6 hours for solid foods and 4 hours for clear fluids.
On the morning of surgery; premedication in form of oral glycopyrrolate(0.2 mg/ml) 20 µg / Kg and oral midazolam (5mg/ml) 0.5 mg / Kg were administered 45 minutes before induction of Anaesthesia. 25 % Dextrose was added (1-2 cc) to mixture of midazolam and glycopyrrolate to mask unpalatable test. Before taking the patient to OT, sedation score was assessed. A four point sedation score was assigned as follows.\[8\]
1 = Asleep, not arousable by verbal command 
2 = Asleep, arousable by verbal command 
3 = Drowsy/not sleeping 
4 = Alert/aware

Monitors like NIBP, SpO₂ probe, ECG were attached to patient. Baseline preoperative vital parameters like pulse rate, BP, respiratory rate and arterial oxygen saturation were noted. Patients were pre-oxygenated with 100% oxygen through Jackson-Ree’s modification of Ayre’s t-piece, for 3 minutes. Anaesthesia was induced with O₂ and Sevoflurane through
mask till the loss of eyelash reflex. Intra venous line was secured under aseptic precautions with 22G-24G intra venous line and Injection RL was started. Patients were turned to lateral position for caudal epidural block. Anaesthesia was maintained with \( O_2 \) and Sevoflurane (2-2.5\%). Caudal epidural block was given under all aseptic and antiseptic precautions with 24 Gauge 1.5 inch hypodermic sterile needle.

**Patients were divided into two groups randomly**

**Group – I :- Clonidine Group** received
- Injection Lignocaine (2\%) (7mg/Kg )
- Injection Bupivacaine (0.5\%) (2mg/Kg )
- Injection Adrenaline (1:2,00,000) (5µg/ml )
- Injection clonidine (1µg/Kg )
- Injection sterile water to make 1ml/Kg volume.

**Group–II:-Control Group** received
- Injection lignocaine (2 \%) (7mg/ Kg)
- Injection Bupivacaine (0.5\%) (2 mg/Kg )
- Injection Adrenaline (1:2,00,000) (5µg/ml )
- Injection sterile water to make total 1ml/Kg volume.

Caudal epidural space was located and confirmed by absence of CSF on aspiration, loss of resistance and Whoosh test. When caudal Injection was completed, patients were turned to supine position. Sevoflurane was discontinued, to ensure early recovery from Sevoflurane and for assessment of sensory and motor blockade. Onset of anaesthesia was assessed from loss of scrotal reflex in male and loss of patellar reflex in female and pin prick test every 30 seconds. Time to achieve maximum sensory level and motor blockade and maximum level achieved were noted.

Vital parameters like pulse rate, BP, respiratory rate, \( SpO_2 \) were monitored at the interval of 5 minutes initially and then 15 minutes later on throughout intra-operative period. Sedation score was also monitored during intra-operative period. No additional analgesic medications were given during perioperative period. Caudal block was considered failed if adequate level was not achieved after 15 to 20 minutes of block. These patients were given general anaesthesia and were excluded from further study. In patients, who have achieved adequate level, if child moved upper limbs, and if required, supplemental sedation was given in form
of oxygen + Sevoflurane just to induce sleep and discontinued after that. Total duration of surgery was noted.

After completion of surgery, pulse rate, BP, respiratory rate and SpO₂ were noted; then all patients were shifted to recovery room and observed for following parameters
(i) Pulse rate, BP, Respiratory rate, oxygen saturation.
(ii) Motor block (complete / partial)
(iii) Hanallah’s Modified Objective Pain Score (OPS) for postoperative analgesia at 1 hour intervals.
(iv) First spontaneous voiding of urine.
(v) complications like respiratory depression (RR<10 minute), excessive sedation, dryness of mouth, urinary retention, hypotension, bradycardia, nausea / vomiting, fever and any neurological deficit.

[C] Assessment of post operative analgesia
Post operative analgesia was assessed using modified Hanallah’s Objective Pain Score devised by Hanallah R.S. containing 6 parameters. Each parameter containing 0, 1, 2 points. Maximum score is 12. Total score of 7 or more was considered as an indication for supplemental analgesic requirement. Supplemental analgesic were administered in the form of syrup. Paracetamol 5 mg/Kg orally, or Inj. Paracetamol 5 mg/kg intravenously and study was terminated.

All results in our study were subjected to statistical analysis using student’s unpaired ‘t’ test for quantitative data and chi-square test and standard error difference between two proportions for qualitative data. p<0.05 was taken as significant and p<0.001 as highly significant.

RESULT
Our results are as follows
Demographic data were comparable in both groups. Duration of surgeries was 77.2±35.3 minutes in clonidine group and 73.2±33.93 minutes in control group. The difference between two groups was not statistically significant.(‘p’>0.05). Most of the surgeries in both groups were herniotomy, circumcision, hypospadiasis repair and urethral fistula repair. Number of different surgeries in both groups were comparable and there was no statistically significant difference (‘p’>0.05).
Pre op sedation score was II in 88% of cases in clonidine group and 84% cases in control group. In clonidine group, 12% patients had sedation score of III, while in control group, 12% and 4% patients had sedation score of I and III respectively. There was no statistically significant difference in two groups for pre operative sedation score.

Onset of sensory blockade was 1.32± 0.39 minutes in clonidine group and 3.02 ± 0.36 minutes in control group. Onset of motor blockade was 1.92 ± 0.31 minutes in clonidine group and 3.70 ± 0.45 minutes in control group. Time for peak sensory level was 4.76±0.50 minutes in clonidine group and 8.46 ± 0.91 minutes in control group. Thus onset and peak of sensory and motor blockade was significantly earlier in clonidine group. \( 'p' <0.05 \).

In clonidine group \( T_{10} \) level was achieved in 80% of cases and \( T_8 \) level in 20 % of cases, while in control group, \( T_{10} \) level was achieved in 84% of cases and \( T_8 \) level was achieved in 16 % of cases. The difference was statistically not significant. \( 'p'>0.05 \).

**Table – Assessment of blockade**

<table>
<thead>
<tr>
<th>Group</th>
<th>Clonidine group</th>
<th>Control group</th>
<th>‘p’ value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Onset (min)</td>
<td>Sensory</td>
<td>1.32±0.39</td>
<td>3.02 ± 0.36</td>
</tr>
<tr>
<td></td>
<td>Motor</td>
<td>1.92 ± 0.31</td>
<td>3.70 ± 0.45</td>
</tr>
<tr>
<td>Peak (min)</td>
<td>Sensory</td>
<td>4.76±0.50</td>
<td>8.46 ± 0.91</td>
</tr>
<tr>
<td></td>
<td>Motor</td>
<td>8.02 ± 0.66</td>
<td>10.96 ± 0.78</td>
</tr>
</tbody>
</table>

**Table: Maximum level achieved**

<table>
<thead>
<tr>
<th>Level achieved</th>
<th>Clonidine group</th>
<th>Control group</th>
<th>‘p’ value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No. of cases</td>
<td>Percentage</td>
<td>No. of cases</td>
</tr>
<tr>
<td>( T_{12} )</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>( T_{10} )</td>
<td>20</td>
<td>80</td>
<td>21</td>
</tr>
<tr>
<td>( T_8 )</td>
<td>5</td>
<td>20</td>
<td>4</td>
</tr>
<tr>
<td>( T_6 )</td>
<td>00</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

As shown in figure, from inter group ‘p’ values, there was no statistically significant difference between two groups for pulse rate in, pre op or intra op period at any time. As seen from intra group ‘p’ values (‘p’ >0.05), there was no statistically significant change in pulse rate during intra operative period as compared to preoperative values in either of two groups. As evident from intergroup ‘p’ values, there was no significant difference between systolic blood pressure of two groups in pre op and intra op phase.(‘p’>0.05). As evident from
intragroup ‘p’ values, (‘p’ >0.05), systolic blood pressure remained stable in both groups during intra operative phase.

Diastolic blood pressure also showed similar trends. There was no statistically significant difference between two groups, for pre op and intra operative values (inter group P values>0.05). During entire intra operative phase diastolic blood pressure remained stable in both groups (intra group ‘p’ values >0.05). There was no difference in respiratory rate during perioperative period between two groups which was statistically insignificant (inter and intra group ‘p’ values >0.05).

Arterial oxygen saturation during perioperative phase was comparable in both groups and there was no statistically significant difference between two groups. (inter group ‘p’ values>0.05). There was no episode of desaturation (SpO₂<93 %) or any significant change in arterial oxygen saturation in either of two groups. (intra group ‘p’ value <0.05) Intra operatively sedation score was III in 48 % of cases, II in 32% of cases and IV in rest of cases in clonidine group, while in control group, sedation score was II in 60 % of cases, III in 12% of cases, and I in 28 % of cases. The difference was statistically significant. (P<0.05). Thus sedation score were significantly higher in clonidine group during intra operative phase. Supplemental sedation was required in 8 % of cases in clonidine group and 28 % of cases in control group. Thus significantly less number of patients in clonidine group required supplemental sedation (‘p’<0.05).Post operatively pulse rate, systolic BP, diastolic BP and respiratory rate were comparable in both groups and with corresponding preoperative values, thus suggesting that postoperatively there was no hemodynamic or respiratory compromise. There was no statistically significant difference between two groups (P>0.05).
Duration of post operative analgesia was defined from completion of surgery to the time of rescue analgesic supplement. It was 444 ± 54.77 minutes in clonidine group and 177 ± 36.66 minutes in control group. It was significantly longer in clonidine group than control group.(‘p’<0.001) Total duration of analgesia was defined as time from giving caudal block to rescue analgesic supplement. It was 518.8±31.46 minutes in clonidine group and 250.80 ± 24.22 minutes in control group. The difference was statistically highly significant (P< 0.05). In clonidine group, no patient required rescue analgesia till 5th hour post operatively, and by 6th and 7th hour 28 % and 40% patients required rescue analgesia. While in control group, 100 % of patients required rescue analgesia by 4th hour. Duration of motor blockade was slightly longer 197.2±35.35 minutes in clonidine group than control group (170.4 ± 25.07 minutes), but the difference was not statistically significant. (P>0.05) First spontaneous urinary voiding time in after surgery was 193.33 ± 48.5 minutes in clonidine group, and 171 ± 41.4 minutes in control group, the difference was statistically and clinically not significant and no intervention was required in any case. Only side effect seen in the study was dryness of mouth in 6 patient in clonidine group, it was not seen in control group. No other side effect or complication were seen.

**DISCUSSION**

Caudal epidural block is one of the most common regional anaesthetic techniques used in pediatric patients. It is generally considered a simple and safe procedure and its main disadvantage is its relatively short duration of action, even with the use of long-acting local anaesthetic agents such as bupivacaine. In order to improve the duration of action and quality of analgesia of a caudal block with bupivacaine, various drugs have been used, e.g. opioids, epinephrine, midazolam, neostigmine, ketamine and clonidine.\(^3\)

Clonidine has been shown to produce analgesia without causing significant respiratory depression after systemic, epidural or spinal administration.\(^3\) Clonidine action, similar to local anaesthetic action, and its interaction with local anaesthetics have been explained by
three possible mechanisms. First, clonidine blocks Aδ and C fibres as a consequence of an increase in potassium conductance in isolated neurons, thus intensifying local anaesthetic conduction block. Secondly, clonidine may cause local vasoconstriction, thus decreasing local anaesthetic spread and removal around neural structures. Thirdly, clonidine combined with spinal local anaesthetics or used in peripheral blocks intensifies and prolongs analgesia. Spinal α2 adrenergic agonists may also induce analgesia by activating spinal cholinergic neurons resulting in acetylcholine release.[7]

In our study mean age was 4.42 ± 1.79 years in clonidine group and 4.29 ± 1.67 years in control group, and there was no statistically significant difference between two groups. (P >0.05). We used oral Glycopyrrolate in dose of 20 µg/kg which helped in decreasing vagal response and secretions during inhalation induction and caudal anaesthesia. We used oral midazolam which helped in sedating the child, in reducing separation anxiety from parents, improving mask acceptance, and also in reducing Sevoflurane requirement for induction.[3]

Patients were induced with O2 with Sevoflurane through mask with modified Jackson-Rees circuit, till loss of eyelash reflex and then Sevoflurane concentration was rapidly reduced to 2—2.5% for maintenance till caudal space was located, when Sevoflurane was discontinued to ensure rapid awakening for assessment of sensory and motor blockade and reflexes. Ivani et al used same method of induction with Sevoflurane via face mask for study of ropivacaine-clonidine mixture. While in our study Sevoflurane was used as and when required to induce sleep in child and discontinued after that.

We have used caudal block as a sole anaesthetic technique for paediatric patients as this is our institutional practice and Fellmann C et al also described that awake regional anaesthesia for inguinal hernia repair was suggested to avoid life-threatening respiratory complications known to occur after general anaesthesia in these infants.[15]

We used local anaesthetic solution because whole surgery was to be carried out in caudal anaesthesia only with O2/Sevoflurane by mask only when needed to induce sleep. Bupivacaine (0.25 %) alone may result in incomplete motor blockade To improve motor blockade Lignocaine was used along with bupivacaine. Clonidine was added in 1 ug/kg body weight dose with help of tuberculin syringe. Upadhyay et al also used Clonidine 1 ug/kg in their study.[8] Van elstraete et al used Lignocaine- bupivacaine mixture in their study. While all other authors used bupivacaine alone with clonidine or Ropivacaine alone with clonidine
in their study.[6] In both groups there was predominance of males but it was comparable. It was probably because of higher incidence of congenital hernia in males and some surgeries done exclusively in males like orchidopexy, circumcision, urethroplasty etc. There was only one female in clonidine group and no female patient in control group.

Duration of surgery was 77.2 ± 35.3 min in clonidine group and 73.2 ± 33.93 min in control group. There was no statistically significant different between two groups (P>0.05). Most of the surgeries lasted from 30 minutes to 120 minutes. Constant et al showed caudal anaesthesia can used for surgeries lasting for more than 90 minutes and addition of clonidine increased duration of surgical analgesia to more than 130 minutes.[8] Our study was done for sub umbilical surgeries. Type of surgeries were comparable in both the groups and there was no statistically significant difference between two groups.

Pre operative pulse rate was 103.76 ± 8.27 per minutes in clonidine group and 102.54 ±7.55 per minute in control group. There was no statistically significant difference between two groups (P>0.05). Pulse rate remained stable in both the groups during perioperative period (p >0.05). Pre operative systolic blood pressure was 104.69 ± 4.88 mm Hg in clonidine group and 104.16 ± 4.75 mm Hg in control group. Preoperative diastolic blood pressure was 66.88 ± 3.46 mm Hg in clonidine group and 66.16 ± 3.46 mm Hg in control group. There was no statistically significant difference between two groups in systolic or diastolic blood pressure. (P>0.05). And there was no change in systolic and diastolic blood pressure during perioperative period in both groups. (‘p’ >0.05). No bradycardia or hypotension was found in any case.

Our study correlates with results obtained by all authors who have done study of clonidine in paediatric caudal anaesthesia.[3,8,9] Motsch et al used clonidine in 5 µg/kg BW dose in paediatric caudal anaesthesia and found no difference in intra operative hemodynamic parameters but during emergence and post op period pulse and BP were significantly lower in clonidine group.[10] Jamali et al. only studied baseline mean arterial pressure (MAP) and MAP 3 h after the procedure. No differences in MAP were seen with the administration of 1 µg/kg of caudal clonidine. Furthermore, it is possible that caudally administered clonidine results in less haemodynamic change than systemically administered clonidine.[14]

In paediatric patients, immaturity of sympathetic system, less blood volume in lower limb capacitance vessels and splanchnic circulation, diminished autonomic adaptability of heart, is
presumably responsible for minimal or no fall in blood pressure or bradycardia after caudal epidural clonidine. Perioperatively there was no statistically significant change in arterial oxygen saturation and respiratory rate in either group. There was no episode of apnoea or desaturation in either of the groups during perioperative period. Our findings match with all the authors who studied clonidine in paediatric caudal anaesthesia.

Onset time of sensory block was $1.32 \pm 0.39$ minutes in clonidine group and $3.02 \pm 0.36$ minutes in control group. Onset time of motor block was $1.92 \pm 0.31$ minutes in clonidine group and $3.7 \pm 0.45$ minutes in control group. Onset of both sensory and motor block was earlier in clonidine group and the difference was statistically highly significant. Peak sensory level was achieved in $4.76 \pm 0.50$ minutes in clonidine group and $8.46 \pm 0.91$ minutes in control group. Peak motor level was achieved in $8.02 \pm 0.66$ minutes in clonidine group and in $10.96 \pm 0.78$ minutes in control group. Peak effect was achieved earlier in clonidine group and the difference was statistically significant. ($P<0.001$). Peak level achieved were comparable in both groups. In clonidine group, $T_8$ level was achieved in 20 % of cases, $T_{10}$ level was achieved in 80 % of cases. While in control group $T_8$ level was achieved in 16 % of cases and $T_{10}$ level was achieved in 84 % of cases. There was no statistically significant difference between two groups for level achieved ($P>0.05$). Clonidine enhances sensory and motor blockade by local anaesthetic mixture and thereby potentiates their action which may be responsible for earlier onset and peak effect observed in clonidine group. Ivani et al found onset time of 9 minutes in clonidine group and 10 minutes in control group, however they used Ropivacaine 0.1% in clonidine group and 0.2% in control group. In case adequate sensory or motor block was not achieved by 15 minutes then it was considered failed caudal epidural, general anaesthesia was given and case was excluded from study.

Pre operative sedation score was 2 in 88 % cases and 3 in 12 % cases in clonidine group and 2 in 84 % cases and 1 in 12 % cases in control group. There was no statistically significant difference between two groups. Intra operatively sedation score was higher in clonidine group than control group. The difference was statistically significant. ($p<0.05$). Supplemental sedation was required in 8 % cases in clonidine group and 28 % cases in control group. The difference was statistically significant. ($p<0.05$). No other author could evaluate intra operative sedation because they have given general anaesthesia along with caudal epidural.
Duration of surgical analgesia was considered to be throughout duration of surgery if during surgery there was no significant change in pulse rate (>15%), blood pressure, or no response to surgical incision or other painful stimuli during surgery. In our study, there was no significant change in hemodynamic parameters in intraoperative phase. In case of inadequate analgesia conventional GA with intubation was given to patient. Those cases were excluded from study. Constant et al, assessed surgical analgesia, and found it to be present in 93% patients throughout duration of surgery, in clonidine group and in 57% patients in control group.[8] Duration of post operative analgesia was defined as, time from completion of surgery to rescue analgesic administration, and was assessed in our study with the help of modified Hanallah’s objective pain score. When patient’s score was 7 or more analgesia was considered inadequate and rescue analgesia was administered in form of paracetamol syrup or i.v. Injection. Duration of post operative analgesia was 444 ± 54.77 minutes in clonidine group and 177.6 ± 36.66 min in control group. The difference was statistically highly significant.(P<0.001).

We choose modified Hanallah’s objective pain score because it is based on objective parameters to assess pain, it is simple to use, it requires minimum or no patient cooperation and it covers many indirect sign of pain like facial expression, limb movement etc.[16]

Total duration of analgesia was defined as time from caudal Injection to rescue analgesic supplement. Total duration of analgesia was 518.8 ± 31.46 minutes in clonidine group and 250.80 ± 24.22 minutes in control group. The difference was statistically highly significant.(p<0.001). Motsch et al observed a marked effect of the combined caudal injection of clonidine and bupivacaine in children, resulting in prolongation and enhancement of caudal analgesia postoperatively.[2] Our results matched with all authors who found prolonged postoperative analgesia with caudal clonidine in their studies.[2,4,6,8,10,11]

Duration of motor block was considered till patient were fully able to move both limbs without any weakness. It was 197.2 ± 35.35 minutes in clonidine group and 170.4 ± 25.07 minutes in control group. The difference was not statistically significant.(P>0.05). Our result matched with Van elstraete et al, who found time to first supported standing 148 min and 141 min in clonidine and control group respectively.[6] Cook et al also found no significant difference in motor block between two groups.[11]
Time to first spontaneous voiding was 193.33 ± 48.5 minutes in clonidine group, and 171 ± 41.4 minutes in control group, the difference was statistically and clinically not significant and no intervention was required in any case. Previous study also found no difference in time to first spontaneous voiding between two groups. Van Elstreate et al in their study also did not find any difference between two groups. Sympathetic outflow to the urinary tract promotes an increase in urethral resistance and depresses detrusor contraction, favouring urinary retention. Therefore, clonidine facilitates micturition. They hypothesize that the small dose of clonidine given caudally was unlikely to have a peripheral effect on the bladder. We did not find any complication during perioperative period in both the groups.

Therefore, we conclude clonidine added as an adjuvant to local anaesthetic solution for caudal epidural block in paediatric patients shortens time for onset and peak effect for sensory as well as motor blockade, provides intra operative sedation and stable hemodynamics and prolongs duration of postoperative analgesia without any side effects.

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BIBLIOGRAPHY


