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Addition of dexmedetomidine to caudal bupivacaine in paediatric patients: Hemodynamic changes

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Abstract

Neurotransmitters in the spinal cord attenuate or amplify the pain signals from the periphery. Substance P, calcitonin and gene related peptides amplify while endogenous opioids, norepinephrine, serotonin, GABA and glycine attenuate the pain signal. Nociceptive impulse reaches the thalamus by second order neurons in the spinothalamic, spinoreticular and spinomesencephalic tracts. This study included 60 children, of both genders, coming for various elective infra-umbilical surgical procedures such as herniotomy, circumcision, orchidopexy, urethroplasty etc. After obtaining clearance from the hospital ethical committee a written informed consent was obtained from parents before commencing the study. The baseline oxygen saturation (SpO₂) was 98.67 ± 1.24% in group B. In group D, the baseline oxygen saturation (SpO₂) was 98.17 ± 1.51%. There was no significant difference between the groups. At 5, 15 and 30 minutes SpO₂ values were 99 ± 0.74%, 98.1% ± 0.89 and 97.9% ± 1.24 respectively in group B and 98.23% ± 1.4, 98.67% ± 1.18 and 98.6% ± 1.04 24 respectively in group D. The differences were statistically significant but clinically insignificant.

Keywords: Dexmedetomidine, bupivacaine, hemodynamic changes

Introduction

Acute pain is a highly complex, dynamic and subjective experience that is useful to growing children, serving to warn them of the danger and limiting exposure to additional injury. However, untreated acute, recurrent or chronic pain related to disease or medical care may have significant physiological and psychological consequences in children^[1].

Historically, it was thought that children neither suffer nor feel pain or respond to or remember the painful experiences as adults did. This assumption was due to immaturity of nervous system in children. As a result, it was common to ignore the need for analgesia during painful procedures. However, various studies off late have shown that pain pathways, as well as cortical and sub-cortical centres necessary for pain perception, are well developed late in gestation. Newborn infants, even preterm, can appreciate pain and react to it with tachycardia, hypertension, increased neuro-endocrine response and intracranial pressure^[2].

The basic mechanism of pain perception in infants and children are similar to those of adults. Noxious mechanical, thermal and chemical stimuli excite primary afferent fibres that transmit information from the periphery to dorsal horns of the spinal cord. Aδ (large, myelinated and fast conducting) and C (small, unmyelinated and slow conducting) nerve fibres are primarily responsible for pain impulse transmission. These signals can be amplified by the inflammatory mediators caused by tissue injury like bradykinin, prostaglandins, cytokines, substance P, catecholamines and potassium^[3].

Neurotransmitters in the spinal cord attenuate or amplify the pain signals from the periphery. Substance P, calcitonin and gene related peptides amplify while endogenous opioids, norepinephrine, serotonin, GABA and glycine attenuate the pain signal. Nociceptive impulse reaches the thalamus by second order neurons in the spinothalamic, spinoreticular and spinomesencephalic tracts.

Descending modulation occurs when efferent projections from supra spinal areas, such as peri-aqueductal gray, raphe nucleus and locus coeruleus release inhibitory neurotransmitters. The major neurotransmitters mediating descending inhibition are nor-epinephrine, serotonin, endogenous opioids, GABA and acetylcholine^[4].

The cardiovascular effects of dexmedetomidine are mediated via adrenoreceptors in both the central and peripheral nervous systems result in sympatholysis. It has a biphasic blood pressure response, a short hypertensive and subsequent hypotensive response. The initial reaction can be explained by the peripheral α₂B-adrenoceptor stimulation of vascular smooth

muscle and can be attenuated by a slow infusion over 10 or more minutes. The initial response lasts for 5 to 10 minutes and is followed by a decrease in blood pressure of approximately 10 to 20% below baseline and a stabilization of the heart rate, also below baseline values; both of these effects are caused by the inhibition of the central sympathetic outflow overriding the direct stimulating effects. Bradycardia and sinus arrest can occur which will respond to anticholinergics [5].

Dexmedetomidine reduces cerebral blood flow and cerebral metabolic rate of oxygen. Activation of the receptors in the brain and spinal cord inhibits neuronal firing causing analgesia, sedation and anxiolysis. Sedation mediated through the locus coeruleus closely mimics endogenous sleep. It produces good degree of sedation, still patients are easily arousable. Dexmedetomidine does not affect intracranial or lumbar cerebrospinal fluid pressure or cerebral perfusion pressure [6].

When administered via the neuraxial route, it confers some analgesic and antinociceptive actions. Being highly lipophilic, dexmedetomidine is rapidly absorbed into CSF and binds to α_2A adrenoceptors of dorsal horn of spinal cord. It prolongs the duration of both sensory and motor blockade caused by local anaesthetics.

Methodology

This study included 60 children, of both genders, coming for various elective infra-umbilical surgical procedures such as herniotomy, circumcision, orchidopexy, urethroplasty etc. After obtaining clearance from the hospital ethical

committee a written informed consent was obtained from parents before commencing the study.

Inclusion criteria

- Patients in the age group of 1-10 years, of both the genders, undergoing infraumbilical surgeries.
- Patients belonging to ASA physical status 1 and 2.
- Parental consent.

Exclusion criteria

- Patients with known allergy to the study drugs.
- Suspected coagulopathy.
- Infection at the site of caudal block.
- History of developmental delay and neurological diseases.
- Skeletal deformities in the caudal region.
- Parental refusal.

Group B ($n = 30$) received Bupivacaine 0.25%, 1ml/kg + 0.5ml normal saline

Group D ($n = 30$) received Bupivacaine 0.25%, 1ml/kg + Dexmedetomidine 1 μ g/kg making the volume 0.5 ml.

Results

The mean age in group B was 3.87 ± 2.49 years and in group D was 4.7 ± 2.54 years. The two groups did not differ significantly with respect to their age, which is depicted in table 1.

Table 1: Age Distribution

Group	No of Individuals	Mean age (months)	Standard Deviation	p value	Statistical Significance
B	30	46.43	± 29.86	0.21	NS
D	30	56.4	± 30.46		

In group B there were 29 (96.66%) males and 01 (3.33%) female. Group D had 30 (100%) males and no (0%)

females. The groups were comparable with respect to sex as shown in the table 2.

Table 2: Sex Distribution

Gender	Group B n (%)	Group D n (%)	P value	Statistical Significance
Male	29 (96.66)	30 (100)	0.31	NS
Female	01 (3.33)	00 (00)		
Total	30	30		

The mean weight of the children in group B was 12.46 ± 3.83 kg. In group D the mean weight of the children was 13.67 ± 3.92 kg. The two groups did not differ significantly with respect to weight. The weight distribution is depicted in table 3.

Table 3: Weight Distribution

Group	Mean Wt kg	Standard Deviation	p value	Statistical significance
B	12.46	± 3.83	0.23	NS
D	13.67	± 3.92		

Baseline heart rate was 105.47 ± 15.77 per minute in group B and 108.13 ± 17.12 per minute in group D; the difference

is statistically not significant.

After induction, at 5 minute, heart rate was 106.17 ± 14.96 per minute in group B and 107.1 ± 14.06 per minute in group D. The difference between the changes is statistically insignificant. After 15 minute of induction heart rate was 108.7 ± 17.21 per minute in group B and 106.67 ± 14.24 per minute in group D the difference is statistically not significant.

At 90 minutes there was reduction in heart rate in both the groups 99 ± 18.39 per minute in group B and 93 ± 7.64 per minute in group D. The difference between the groups is statistically not significant. There was no significant difference in the heart rate between the two groups at any time interval as shown in table 4

Table 4: Changes in Heart Rate per min

Time Interval (min)	Group B Mean \pm SD	Group D Mean \pm SD	p value	Statistical Significance
Base line	105.47 \pm 15.77	108.13 \pm 17.12	0.53	NS
0	105.57 \pm 15.75	108.57 \pm 17.36	0.49	NS
5	106.17 \pm 14.96	107.1 \pm 14.06	0.80	NS
10	108.07 \pm 16.52	105.43 \pm 14.03	0.51	NS
15	108.7 \pm 17.21	106.67 \pm 14.24	0.62	NS
30	104.73 \pm 14.96	103.57 \pm 12.33	0.74	NS
45	106.25 \pm 14.62	99.39 \pm 14.63	0.16	NS
60	92.25 \pm 20.27	94.7 \pm 15.73	0.81	NS
90	99 \pm 18.39	93 \pm 7.64	0.48	NS

The baseline mean arterial pressure in group B was 61.1 \pm 8.31 mm Hg where as in group D, it was 64.63 \pm 7.42 mm Hg, which is statistically not significant. MAP gradually decreased to 56.47 \pm 7.44 mm hg at 45 minutes in group B, where as in group D, it gradually decreased to 55 \pm 3.6 mm

hg at 90 minutes.

At all the time intervals except at 10 min, the p value was > 0.05 and hence the differences in the mean arterial pressure were not significant. Changes in mean arterial pressure are shown in table 5.

Table 5: Changes in Mean Arterial Pressure mm Hg

Time Interval (min)	Group B Mean \pm SD	Group D Mean \pm SD	p value	Statistical Significance
Base line	61.1 \pm 8.31	64.63 \pm 7.42	0.09	NS
0	61.33 \pm 8.38	64.57 \pm 7.43	0.12	NS
5	59.77 \pm 7.44	63.53 \pm 8.39	0.07	NS
10	57.1 \pm 6.4	61.57 \pm 7.45	0.02	S
15	59.1 \pm 7.46	61.8 \pm 7.41	0.17	NS
30	57.17 \pm 8.23	59.45 \pm 6.56	0.24	NS
45	56.47 \pm 7.44	59.09 \pm 6.14	0.23	NS
60	57.5 \pm 9.03	56.3 \pm 4.57	0.74	NS
90	59.5 \pm 0.7	55 \pm 3.6	0.14	NS

The baseline oxygen saturation (SpO₂) was 98.67 \pm 1.24% in group B. In group D, the baseline oxygen saturation (SpO₂) was 98.17 \pm 1.51%. There was no significant difference between the groups. At 5, 15 and 30 minutes SpO₂ values were 99 \pm 0.74%, 98.1% \pm 0.89 and 97.9% \pm

1.24 respectively in group B and 98.23% \pm 1.4, 98.67% \pm 1.18 and 98.6% \pm 1.04 24 respectively in group D. The differences were statistically significant but clinically insignificant. There were no desaturation events at any time interval. Changes in SpO₂ are shown in table 6.

Table 6: Changes in SpO₂

Time interval (min)	Group B Mean \pm SD	Group D Mean \pm SD	p value	Statistical significance
Base line	98.67 \pm 1.24	98.17 \pm 1.51	0.17	NS
0	98.67 \pm 1.24	98.23 \pm 1.55	0.24	NS
5	99 \pm 0.74	98.23 \pm 1.4	0.01	S
10	98.77 \pm 0.9	98.2 \pm 1.45	0.07	NS
15	98.1 \pm 0.89	98.67 \pm 1.18	0.04	S
30	97.9 \pm 1.24	98.6 \pm 1.04	0.02	S
45	98.24 \pm 1.09	98.61 \pm 0.99	0.27	NS
60	98 \pm 0.81	98.55 \pm 1.44	0.49	NS
90	98.5 \pm 0.71	98.57 \pm 2.15	0.96	NS

Discussion

This study, using caudal epidural block with bupivacaine alone and bupivacaine with dexmedetomidine combination was conducted in 60 children in the age group of 1 to 10 years, of ASA status I and II coming for various elective infra-umbilical surgeries. Epidural dexmedetomidine has been used in the range of 1.5–2 μ g/kg without any incidence of neurological deficits [7, 8]. El-Hennawy A M *et al.* used dexmedetomidine 2 μ g/kg with bupivacaine 0.25%, 1 ml/kg caudally [9]. In our study a cautious study design was adopted by using a low dose of dexmedetomidine 1 μ g/kg to avoid the side effects like excessive sedation and bradycardia.

In the present study, both the groups were similar with respect to demographic data like age, weight and sex of the children. The mean age in bupivacaine group was 3.87 \pm 2.5

years and in dexmedetomidine group it was 4.7 \pm 2.5 years. The mean weight was 12.46 \pm 3.8 kg in group B and 13.67 \pm 3.9 kg in group D. There was only one female in the study. This could be due to the fact that common paediatric infraumbilical surgeries are circumcision, herniotomy and orchidopexy which are male-specific surgeries or more common in males. Cook *et al.* [7] studied the effect of caudal analgesia in paediatric patients in the age group of 1-10 years, undergoing only orchidopexy, hence all the cases were male (100%).

In the present study, heart rate and blood pressure of all the patients were recorded at 5 minute intervals.

There was no significant difference in the heart rate within the groups over time or between the two groups at any time interval. The mean baseline heart rate was similar in both groups. Baseline heart rate was 105.47 \pm 15.77 per minute

in Bupivacaine group and 108.13 ± 17.12 per minute in dexmedetomidine group; the difference is statistically insignificant.

After induction of anaesthesia and the caudal block was performed the heart rate was stable intra-operatively in both the groups, the differences between the changes is statistically insignificant.

At 90 minutes there was reduction in heart rate in both the groups; 99 ± 18.39 per minute in group B and 93 ± 7.64 per minute in group D. The difference between the groups is statistically not significant.

The baseline mean arterial pressure was 61.1 ± 8.31 mm Hg in Bupivacaine group and 64.63 ± 7.42 mm Hg in Dexmedetomidine group, which was statistically insignificant. At 10 minutes post-induction the mean arterial pressure decreased to 57.1 ± 6.4 mmHg in group B and 61.57 ± 7.45 mmHg in group D, the difference was statistically significant but clinically not significant. Mean arterial pressure was stable throughout the intra-operative period. It gradually decreased to 56.47 ± 7.44 mm Hg at 45 minutes in group B, where as in group D, it gradually decreased to 55 ± 3.6 mm Hg at 90 minutes. At all time intervals, the p value were > 0.05 and hence the differences in the mean arterial pressure were not significant. Similar haemodynamic stability was seen in previous studies^[10].

The baseline oxygen saturation (SpO_2) was $98.67 \pm 1.24\%$ in bupivacaine group and $98.17 \pm 1.51\%$ in dexmedetomidine group. At 5, 15 and 30 minutes the SpO_2 values were $99 \pm 0.74\%$, $98.1\% \pm 0.89$ and $97.9\% \pm 1.24$ respectively in group B and $98.23\% \pm 1.4$, $98.67\% \pm 1.18$ and $98.6\% \pm 1.04$ respectively in group D. The differences were statistically significant but clinically insignificant. There were no desaturation events at any time interval.

Conclusion

At 90 minutes there was reduction in heart rate in both the groups 99 ± 18.39 per minute in group B and 93 ± 7.64 per minute in group D. The difference between the groups is statistically not significant. There was no significant difference in the heart rate between the two groups at any time interval.

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