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The Effects of Intravenous Dexmedetomidine on Spinal Anaesthesia: Comparison of Different Dose of Dexmedetomidine

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Abstract: ***Background: The Effects Of Intravenous DEXMEDETOMIDINE On Spinal Anaesthesia: Comparasion Of Different Dose Of DEXMEDETOMIDINE Material and Methods: This is a prospective, randomized, interventional Study. Total sample size is 75. Patients who were undergoing surgeries under Spinal Anaesthesia were divided into three groups, 25 patients in each group. DEX I Group: 0.5 microgram/kg dexmedetomidine iv DEX II Group: 0.3 microgram/kg dexmedetomidine iv DEX III Group: 0.2 microgram/kg dexmedetomidine iv with subarachnoid block with 3ml of 0.5%hyperbaric bupivacaine in all groups. Result: The duration of regression of sensory and motor block was prolonged with higher doses of Intravenous Dexmedetomidine in case of spinal anaesthesia with there is no significant effect on time of onset of spinal anaesthesia. Conclusion: We conclude in our study that with higher dose of intravenous dexmedetomidine as an adjuvant to 0.5% hyperbaric bupivacaine there was significantly prolonged both sensory and motor block along with higher Ramsay sedation score.***

Keywords: Intravenous Dexmedetomidine, Spinal anaesthesia

1. Introduction

The effective use of sedative - hypnotic agents and analgesics is an integral partof comfort and safety of patients. **Dexmedetomidine** is a potent and highlyselective α - 2 adrenoceptor agonist with sympatholytic, sedative, amnestic, andanalgesic properties. It provides a unique " conscious sedation" (patientsappear to be asleep, but are readily roused), analgesia, without respiratorydepression [1]. It decreases central nervous system (CNS) sympathetic outflowin a dose - dependent manner and has analgesic effects best described as opioidsparing.

Spinal anaesthesia: Bradycardia, hypotension, hypothermia and shivering are common complications after spinal anaesthesia (SA). SA impairs thermoregulation, inhibits tonic vasoconstriction, and causes the redistribution of core heat from the trunk to the peripheral tissue. Dexmedetomidine by inhibition of central thermoregulation and attenuation of hyperadrenergic response to perioperative stress are known to prevent postoperative shivering [2]. Activation of presynaptic α 2 - A receptors at locus ceruleus causes sedative and hypnotic effects, whereas its effect on descending medullo spinal noradrenergic pathway results in analgesia. At substantia gelatinosa of the spinal cord, it decreases firing in nociceptive neurons and release of substance P, thus producing analgesia. Prolongation of spinal anaesthesia after IV dexmedetomidine is by its supra - spinal action at locus ceruleus and dorsal raphe nucleus. Activation of post - synaptic α 2 - A receptors in CNS results in hypotension and bradycardia by decreasing the sympathetic activity. Activation of post - synaptic α 2 - C receptors in CNS results in anxiolysis, whereas activation of post - synaptic α 2 - B receptors in peripheral vasculature results in transient hypertension [3].

2. Material and Method

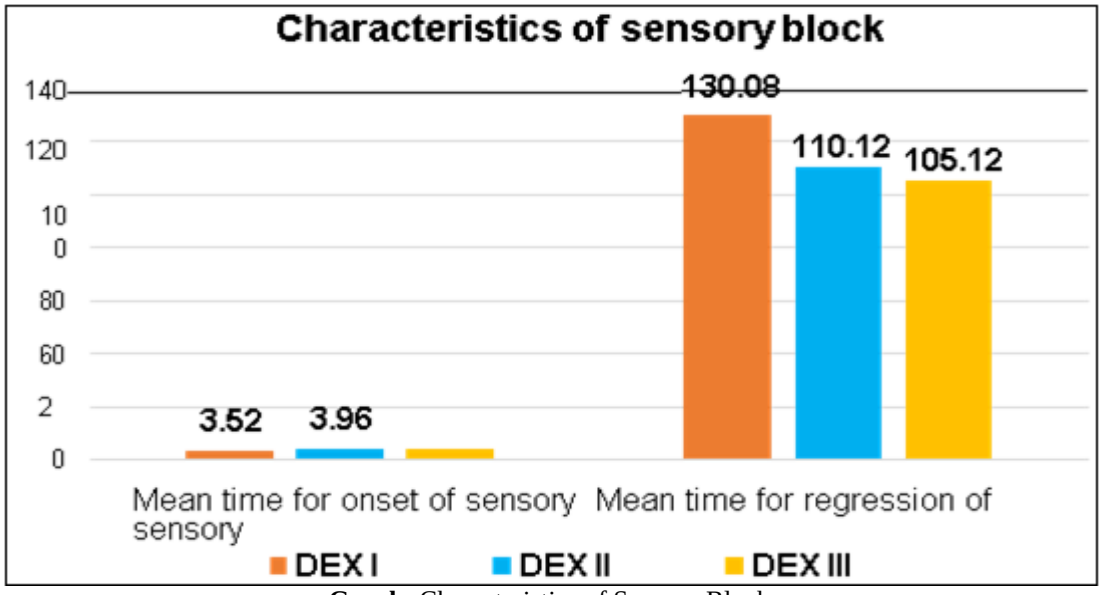
Written and informed consent was obtained who would be scheduled for surgeries. pre operative evaluation was carried out a day before the Surgery. A thorough history was taken and examination was carried out in all patients. Patients were advised for routine and relevant investigations like CBC, renal And liver function tests, Random blood sugar, Chest X - Ray, 12 lead ECG and Reports were reviewed. Patients were kept nil per oral (NPO) for 6 hours before Surgery.

Preparation in OT:
The procedure was explained to the patient and taken inside the operation Theatre. All the minimum mandatory monitors, ECG, NIBP (noninvasive Blood pressure), pulse oximetry were applied, baseline hemodynamics were recorded (ECG, NIBP, SpO2). Securing an intravenous line with 18 - or 20 - gauge Intravenous cannula and an infusion of lactated ringers solution was started at a Rate of 10 -

20ml/kg before spinal anaesthesia. Patient had received oxygen 3litres/min by facemask throughout the procedure. **DEX I group** - subarachnoid block with 3 ml of 0.5% hyperbaric bupivacaine+ 0.5 microgram/kg dexmedetomidine IV **DEX II group** - subarachnoid block with 3 ml of 0.5% hyperbaric bupivacaine+ 0.3 microgram/kg dexmedetomidine IV and **DEX III group** - subarachnoid block with 3 ml of 0.5% hyperbaric bupivacaine + 0.2 microgram/kg dexmedetomidine iv

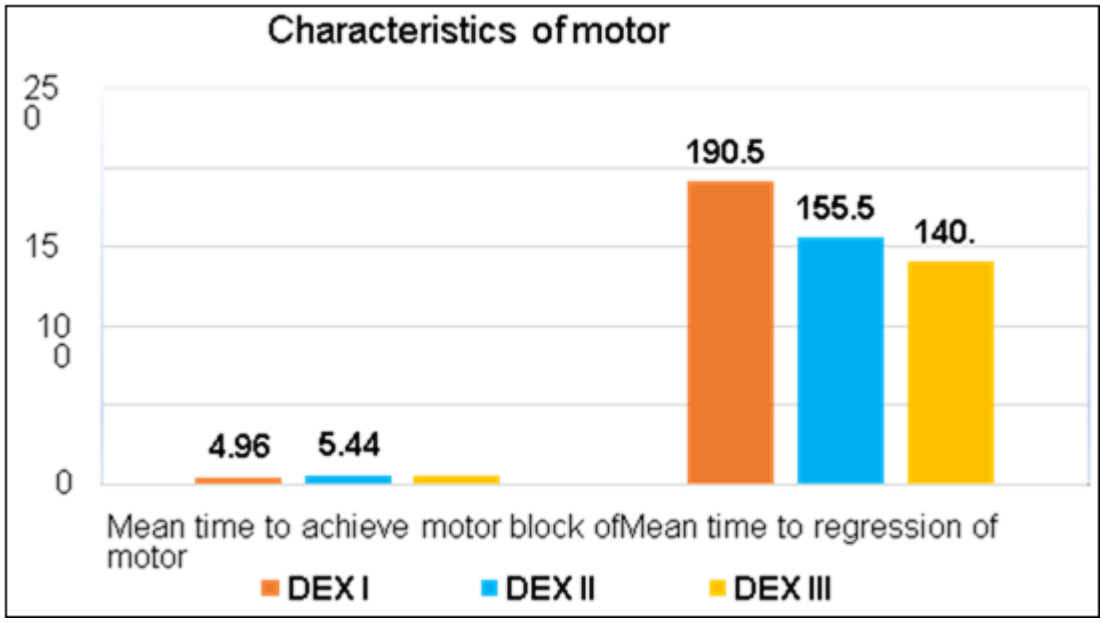
Spinal Anaesthesia:
Under all aseptic and antiseptic precautions, subarachnoid block was given with inj. Bupivacaine 0.5% (heavy) 3 ml in the sitting position using a 23 - gauge Quincke spinal needle positioned at the L3 - L4 interspace. Patients were immediately turned to the supine position. Onset and level of sensory and motor block was recorded. Sensory level was checked with pin prick method and achieved between T8 to T10segment and motor level by Bromage scale. Intra - operative Blood pressure, Heart rate, Spo2 and ECG will be monitored till the completion of surgery.

Post procedure:
Following details will be recorded post - operatively 1st, 2nd, 4th, 6th, 12th & 24th hour: 1) VAS [Visual Assessment score]2) Time for 1st rescue analgesia 3) RSS (Ramsay Sedation Scale) for sedation 4) Bedside Shivering Assessment Scale 5) Modified Bromage scale 6) Incidence of complications and side effect



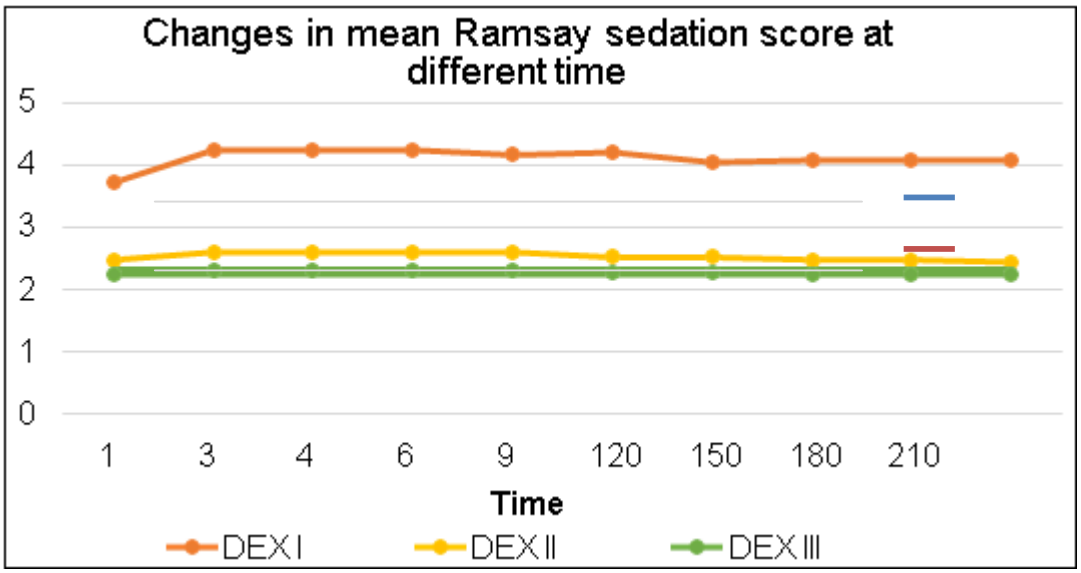
Graph: Characteristics of Sensory Block

Graph shows that, there is no significant changes in onset of sensory effect. The duration of regression to S2 dermatome was 130.08, 110.12 and105.12 min in DEX I, DEX II and DEX III group respectively. This difference was statistically significant ($p < 0.001$). Mean time for regression of sensory block to S2 dermatome was prolonged in DEX I group followed by DEX II and DEX III group.



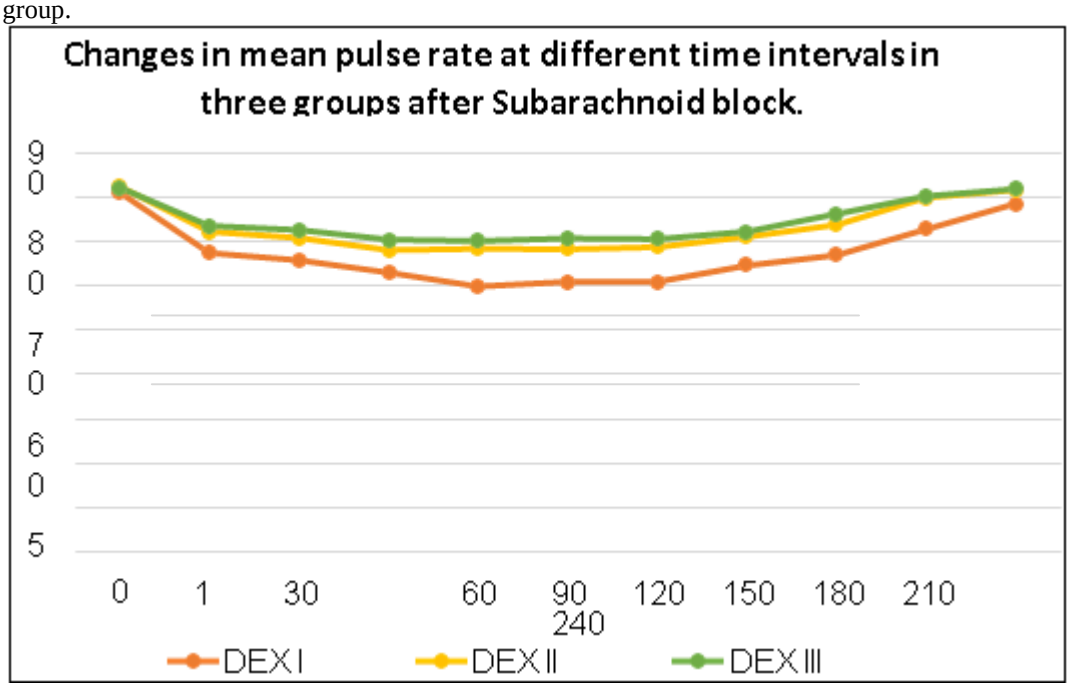
Graph: Characteristic of Motor Effect

Graph shows that the mean time to achieve motor block of grade 3 was 4.96, 5.44 and 5.72 min in DEX I, DEX II and DEX III group respectively which was statistically not significant (p=0.066). Time taken to regression of motor block from grade 3 to 0 was 190.52, 155.52 and 140.20 min in DEX I, DEX II and DEX III group respectively. This difference was statistically significant (p value < 0.001). Mean time to regression of motor block from grade 3 to 0 was prolonged in DEX I group followed by DEX II and DEX III group



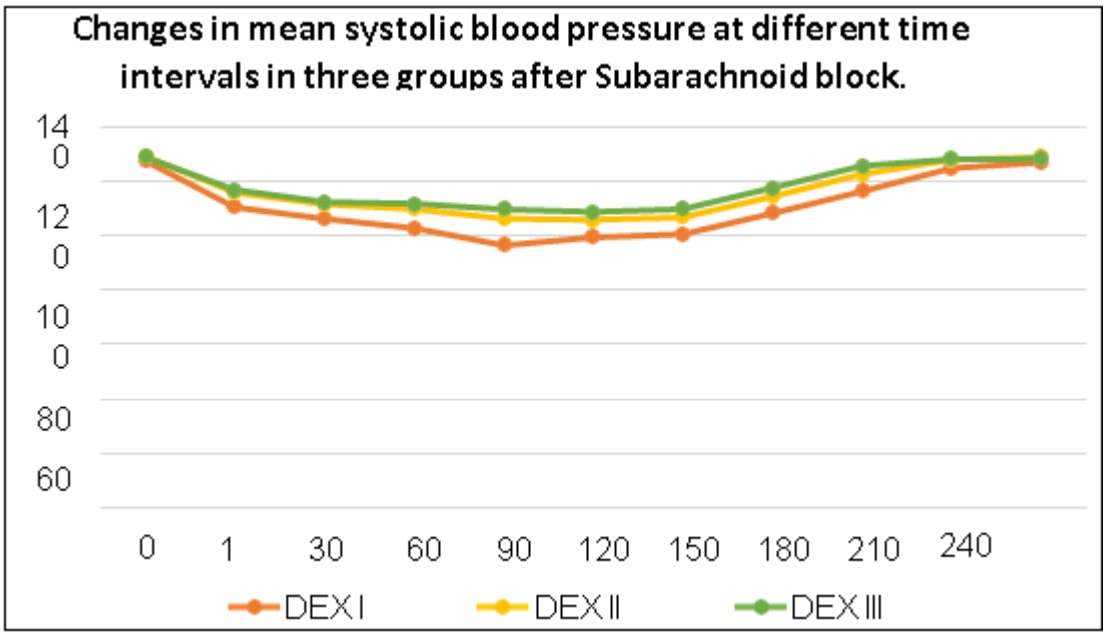
Graph: Changes in mean ramsay sedation score at different time

Graph shows that the mean Ramsay sedation score was significantly higher in DEX I group which was followed by DEX II and DEX III group.



Graph: Changes in mean pulse rate at different time intervals in three groups after subarachnoid block

Graph compares mean pulse rate among the groups after Subarachnoid block. Baseline (0 min) mean pulse rate was comparable among the three groups. After baseline (0 min) there was significant difference in mean pulse rate among three groups. Mean pulse rate fall significantly in DEX I group followed by DEX II and DEX III group.



Graph: Changes in mean systolic blood pressure at different time intervals in three groups after subarachnoid block

Graph compares mean systolic blood pressure among the groups after Subarachnoid block. Baseline (0 min) mean SBP was comparable among the three groups. After baseline (0 min) there was significant difference in mean SBP among three groups. Mean SBP fall significantly in DEX I group followed by DEX II and DEX III group.

3. Discussion

Recent studies have shown the efficacy of both intrathecal and IV dexmedetomidine in prolonging spinal anaesthesia. Prolongation of spinal anaesthesia after IV dexmedetomidine is by its supra - spinal action at locus ceruleus and dorsal raphe nucleus. Dexmedetomidine is a more selective α2 - A receptor agonist than clonidine, with more sedative and analgesic effects. Activation of presynaptic α2 - A receptors at locus ceruleus decreases norepinephrine release and causes sedative and hypnotic effects, whereas its effect on descending medullo spinal noradrenergic path way results in analgesia by terminating pain signal propagation. At substantia gelatinosa of the spinal cord, it decreases firing in nociceptive neurons and release of substance P, thus producing analgesia. So, dexmedetomidine has a role in modulating pain and inhibiting the transmission and perception of pain. Activation of post - synaptic α2 - A receptors in CNS results in hypotension and bradycardia by decreasing the sympathetic activity. Activation of postsynaptic α2 - C receptors in CNS results in anxiolysis, whereas activation of post - synaptic α2 - B receptors in peripheral vasculature results in transient hypertension.

4. Conclusion

We conclude in our study that with higher dose of intravenous dexmedetomidine as an adjuvant to 0.5% hyperbaric bupivacaine there was significantly prolonged both sensory and motor block along with higher Ramsay sedation score. Time for the requirement of 1st rescueanalgesic was also prolonged with higher dexmedetomidine dose. However, higher dexmedetomidine dose was reported with high incidence ofhypotension and bradycardia

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