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COMPARATIVE STUDY OF 0.5% BUPIVACAINE AND 0.5% BUPIVACAINE WITH DEXMEDETOMIDINE FOR SPINAL ANAESTHESIA

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AUTHORS' CONTRIBUTIONS

This work was carried out in collaboration among all authors. Authors NVK and NKN designed the study, performed the statistical analysis, wrote the protocol, and wrote the first draft of the manuscript. Authors AG, SAP and AJB managed the analyses of the study. Author SG managed the literature searches. All authors read and approved the final manuscript.

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ABSTRACT

Spinal anaesthesia remains one of the basic methods in the arsenal of modern aesthetics, which has diminished its prevalence over the last 100 years since its introduction into clinical practise. Stop physiological and metabolic changes that alleviate the discomfort of general anaesthesia during surgery. As the optimum condition for surgery is given. The purpose of this study was to evaluate the effects of dexmedetomidine added to hyperbaric bupivacaine for spinal anesthesia. The design of our study consisted of 75 patients, aged 18 to 50, undergoing ASA physical condition I, II spinal anesthesia undergoing elective lower limb orthopedic surgery, who were randomly assigned three after taking informed consent was divided into groups. The addition of dexmedetomidine to 0.5 per cent of hyperbaric bupivacaine in spinal anaesthesia greatly decreases onset time, prolonging the duration of both sensory and motor blockade.

Keywords: Bupivacaine; dexmedetomidine; spinal anaesthesia; lower limb; orthopaedic surgery.

1. INTRODUCTION

The best blessing that God has given to humankind isn't happiness, however help of agony. There have been many attempts since time immemorial to relieve pain during and after surgery, especially pain. Spinal anesthesia was brought into clinical practice in 1898 by Carl August Bier [1]. Over a century has passed and even today, it is one of the most popular methods for both elective and crisis surgeries especially Cesarean segments, lower abdominal medical procedures, orthopedic and urological surgery to

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name a few [2]. This study was intended to assess the efficacy of the addition of dexmedetomidine to bupivacaine in spinal anaesthesia and to compare its use with bupivacaine.

2. AIM & OBJECTIVES

The purpose of the study is to compare the following variables in three groups, i.e. Group A: 0.5 per cent Hyperbaric Bupivacaine 15 mg + Regular Salt 0.5 ml. Community B: 0.5 percent Bupivacaine 15 mg Hyperbaric + Dexmedetomidine 5 µg (0.5 ml) Community C: 0.5 per cent of Hyperbaric Bupivacaine 15 mg + Dexmedetomidine 7.5 µg (0.5 ml).

3. REVIEW OF LITERATURE

Goodison, R.R. 1982: states hyperbaric bupivacaine produces predictable and adjustable level of blocks which contrasts to isobaric mixture. Hyperbaric bupivacaine has rapid onset and longer duration of action [3]. Sundens KO, et al., 1982: The onset of sensory and motor blockade was faster in all three groups in their double blind study compared to 3 ml of 0.5% bupiviken, 1.5, 2.0 and 3 ml in 30 patients undergoing spinal anesthesia. The duration of motor blockade and analgesia increased with increasing volume [4]. Rachana Joshi, et al. 2013: This study included 50 patients of ASA grade I/II posted for lower abdominal. Patients were divided in to 2 groups: group B received (n=25) received 0.5% heavy bupivacaine 3ml(15mg) with 0.9% normal saline 0.2ml intrathecally. group BD received (n=25) received 0.5% heavy bupivacaine 3 ml (15 mg) with dexmedetomidine 0.05 ml (5 µg) and 0.9% normal saline 0.15 ml intrathecally [5]. Sherief A Abdelhamid et al., 2013: They concluded dexmedetomidine 5 µg intrathecally with bupivacaine provides earlier sensory and motor onset, with less postoperative analgesic requirements, with no sedation or neurological complications [6].

3.1 The Pathophysiology of Pain [7-12]

Pain is characterised as a sensory response evoked by stimuli that damage or threaten to kill tissue, and is more precisely identified as a feeling of an aversive stimulus that originates in a particular region of the body. Psychological pain occurs when a noxious stimulus activates high threshold sensory receptors (nociceptors). This informs the body of potential or actual damage and correlates with withdrawal reflexes. Pathological pain occurs in response to non-noxious stimulus or even in the absence of a definable stimulus. This promotes healing by avoidance of all stimuli but is truly pathological in its chronic form. Theories of pain: Although the exact mechanism of pain relief is not clear, various theories have been put forward of all the theories, the Gate control theory of pain is the most widely accepted. Gate control theory of pain [13]: Proposed by Melzack and Wall in 1965 and then later modified by them in 1982. They initially took into consideration the evidence of physiological specialization, central summation, patterning modulation of input and the influence of psychological factors.

3.2 Pharmacology of Local Anaesthetic [14-19]

Local anaesthetics are drugs that reversibly block nerve conduction, when locally to nerve tissue in the appropriate concentrations. The structure of anesthetic drug consists of a lipophilic aromatic ring and a hydrophilic tertiary amine. The intermediate link is neither by an ester or an amide. Local anesthetics have to cross the axonal membrane to reach the binding site. A swift change in the valency of amino nitrogen moiety lakes place for penetration. High concentration of base is required for penetration and cation moiety is required for action on target organ.

$R{=}N + H^+ \qquad R = NH^+$

(Unchanged base water insoluble) (Changed base water soluble)

Local anesthetics exist in an aqueous solution in a chemical equilibrium between base and cation. This depends on pH of solution and pKa of drug. pH can change the equilibrium but pKa is constant. When pH= pKa, Cation base. At physiological pH (7.4), concentration of cation is more than that of the base. Increase in the pH causes increase in base and hence increases penetration.

Local anesthetics prevent generation and conduction of nerve impulses in all excitable tissues. It affects the permeability of the nerve to Na^+ and K^+. Local anesthetics probably inhibit Na^+ flux by specific interaction with voltage gated Na^+ channels. It is hypothesized to act on the outer and inner surface of the axonal membrane. Uncharged local anesthetics enter the axoplasm and become positively charged to become an active cation. It acts as a receptor, blocking the Na^+ channel. Another theory is the membrane expansion theory. Drugs, which do not form cations at physiological pH, act by penetration the axonal membrane. The membrane swells and blocks Na^+ channel. At the resting process, the interior of the peripheral nerve fibre has a potential difference of about-70mV from the outside. When the nerve is stimulated there is a rapid increase of the

membrane potential by about + 20mV, followed by immediate restoration of the resting level. This depolarization/ repolarization sequence lasts for 1-2 ms and produces the action potential associated with the passage of a nerve impulse. Depolarization is the result of sudden increase in membrane permeability to Na+, which enters the cell through Na+ channels that are closed during resting phase. This increases the membrane potential to approximately +20 mV.when the electrochemical and concentration gradients of Na+ balance each other and the channels close. This gradient favours the movement of K+ outside the cell till resting potential is reached. The impulse is transmitted along the axons because a local current flows between depolarized (positive charge) and non-depolarized (negative charge) segment of the nerve. The voltage change due to this current induces a structure change in the Na+ channel in the next section, such that the potential for action is distributed along the nerve.

4. MATERIAL & METHODS

This clinical on 75 adult patients of ASA physical condition 1 and 2 from 18 years to 50 years under orthopedic surgery of voluntary body parts under spinal anesthesia after consenting over a period of 12 months at Krishna Hospital, Karad.

Group A: (Bupivacaine group): patients received intrathecal 0.5% hyperbaric bupivacaine 15mg (3 mL) + normal saline 0.5 ml and total volume 3.5 mL
Group B: (Dexmedetomidine group): patients received intrathecal 0.5% hyperbaric bupivacaine 15mg (3 mL) +dexmedetomidine 5 µg and total volume 3.5 mL
Group C: (Dexmedetomidine group): patients received intrathecal 0.5% hyperbaric bupivacaine15mg (3mL) +dexmedetomidine 7.5 µg and total volume 3.5 mL. Those patients scheduled to undergo elective lower limb orthopaedic surgeries under subarachnoid block.

5. OBSERVATIONS & RESULTS

As shown in Table 1, the mean age of patient in group A was 36.88 ± 9.08, in group B was37.52±8.93 and in group C was 38.68 ±8.79 years. In group A there were 17 males and 8 were females, Group B had 18 males and 7 females and Group C had 13 males and 12 females.

The mean height of patient in Group A was 5.45 ± 1.67, in Group B 5.50 ± 1.46 and in Group C 5.34 ± 1.51 (feet). The mean weight of patient in group A was 57.72 ± 11.26, in Group B was 56.68 ± 10.68 and in Group C was 54.28 ± 11.01 kg (Table 2).

According to Table 3, in Group A 8 per cent of patients had hypotension, 12 per cent had bradycardia, 12 per cent had nausea and vomiting, and 16 per cent had shivering. In Group B, 16 per cent of patients had hypotension, 16 per cent had bradycardia, 8 per cent had nausea / vomiting and shivering, and 20 per cent had hypotension in Group C, 20 per cent had bradycardia, 8 per cent had nausea / vomiting and shivering.

6. DISCUSSION

In spinal anesthesia there is a temporary interruption of nerve transmission within the subarachnoid space produced by the injection of a local anesthetic solution into the cerebrospinal fluid. In this study addition of 7.5 µg (Group C) of dexmeditomedine to bupivacaine accelerated the onset of sensory by 112 seconds and motor blockade by 125 seconds whereas addition of 5 µg (Group B) of dexmeditomedine to bupivacaine accelerates onset of sensory and motor blockade by 72 seconds and 105 seconds respectively when compared to control group (Group A).

Table 1. Age and sex wise distribution in group A, B and C

Age in years	Group A		Group B		Group C	
	Male	Female	Male	Female	Male	Female
<20	1	1	1	0	1	0
20-30	5	1	8	2	2	3
30-40	6	2	1	2	4	7
40-50	4	4	4	3	6	2
>50	1	0	4	0	0	0
Total	**17**	**8**	**18**	**7**	**13**	**12**
Mean ± SD	**36.88 yrs. ± 9.08yrs.**		**37.52yrs. ± 8.93yrs.**		**38.68yrs. ±8.79**	

Table 2. Distribution of mean and SD of height and weight in group A, B and C

	Group A	Group B	Group C
	Mean ± SD	Mean ± SD	Mean ± SD
Height in feet	5.45 ± 1.67	5.50 ± 1.46	5.34 ± 1.51
Weight in Kgs.	57.72 ± 11.26	56.68 ± 10.68	54.28 ± 11.01

Table 3. Side effects

Adverse effects	Group A n (%)	Group B n (%)	Group C n (%)
Nausea/Vomiting	3(12)	2(8)	2(8)
Drowsiness	0	0	0
Bradycardia	3(12)	4(16)	5(20)
Hypotension	2(8)	4(16)	5(20)
Respiratory depression	0	0	0
Shivering	4(16)	2(8)	2(8)

In our study, we observed that adding 7.5 µg dexmedetomidine (Group C) to bupivacaine prolonged sensory and motor blockade by 141 minutes and 132 minutes respectively while adding 5 µg dexmedetomidine (Group B) to bupivacaine prolonged sensory and motor blockade by 116 minutes and 108 minutes respectively compared to control group (Group A).

In our study, there is no significant difference between mean values of heart rate (min) from 0 min to after 120 min in Group A, while there is statistically significant change in mean heart rate in group B and group C but it is clinically insignificant.

7. CONCLUSION

The study concluded that the addition of dexmedetomidine to 0.5 per cent of hyperbaric Bupivacaine in spinal anaesthesia greatly improves onset time and prolongs the length of both sensory and motor blockades. It also prolongs the duration and increases the efficiency of postoperative analgesia with improved hemodynamic stability compared to bupivacaine alone. It is an attractive adjuvant for extended spinal anaesthesia. Dexmedetomidine can extend the spectrum and boost the reliability and effectiveness of regional anaesthesia. Thus, the study concluded that "Addition of dexmedetomidine potentiates bupivacaine spinal anesthesia."

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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