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Comparision of post operative analgesia for intravenous dexmedetomidine and clonidine as an adjuvant in subarachnoid block in lower abdominal surgeries

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Abstract

Introduction: Spinal anesthesia is the most commonly used technique for anesthesia and post operative analgesia in a variety of infraumbilical surgeries. An adjuvant may enhance the effect of the local anaesthetic and prolong the duration of sensory, motor blockade, and postoperative analgesia obtained with spinal anaesthesia.

Objectives: To compare the duration of post operative analgesia for dexmedetomidine and clonidine.

Methods: This is a comparative study of sixty patients undergoing lower abdominal surgeries. Patients were divided into 2 groups of 30 each. In this study drugs were given intravenously over 10 minutes prior to subarachnoid block. Patients in Group D(n=30) received dexmedetomidine 1 µgkg-1 and patients in Group C (n = 30) received clonidine 1 µgkg-1. It was followed by infusion dexmedetomidine 0.5 µg/kg/hr (Group D) and infusion of Clonidine 1 µg kg/hr (Group C) till the end of surgery. Postoperative pain was assessed by using the VAS (0 – No pain; 10 – Worst possible pain) every 30 minutes for first 4 hours, every 1 hour for next 4 hours, every 2 hours till 12 hours every 4 hours till 24 hours. Collected data will be analysed by SPSS software.

Results: There was statistically significant prolongation of postoperative analgesia in dexmedetomidine group (251.8 ± 11.94 min) compared to clonidine group (179.4 ± 6.926 min).

Conclusion: Statistically significant difference between post operative analgesia between dexmedetomidine and clonidine group. Dexmedetomidine had a delayed first analgesic request than clonidine.

Keywords: Clonidine, Dexmedetomidine, Subarachnoid Block, Lower abdominal surgeries

Introduction

The primary goal of anaesthesia is to relieve pain during and after surgery. Lower abdominal procedures can be performed under spinal, epidural, or general anaesthetic, as well as nerve blocks. Due to its ease of use, efficiency, and safety, regional anesthesia has become a crucial technique.

In particular for infraumbilical area and lower extremity surgeries, spinal anaesthesia is a well-established modality of anaesthesia because of its simplicity, convenience of administration, and lack of general anesthesia's side effects.1However, numerous limitations, such as needle phobia, Recall of the procedure, and a shorter duration of postoperative analgesia, have turned into issues for anesthesiologists. These elements emphasize the requirement for postoperative analgesia and intraoperative sedation for surgeries carried out under subarchnoid block. In the course of regional anaesthesia, a variety of methods and medication regimens have been tested to reduce anxiety and lengthen postoperative analgesia.

Due to their sedative and analgesic effects, alpha2 - adrenergic agonists can be employed as an adjuvant to regional anaesthesia. Alpha 2 adrenergic agonists include clonidine and dexmedetomidine. Compared to Clonidine, Dexmedetomidine is a highly selective agonist at the 2 receptor. When used with local anaesthetics, these medications may enhance their effects and extend the time that sensory, motor, and postoperative analgesia achieved with spinal anaesthesia exist.[2] For the comfort of both patients and surgeons during subarachnoid block surgeries, appropriate sedation is necessary. Additionally, it will help patients accept regional anaesthesia treatments.Comparing Dexmedetomidine to Clonidine, the latter offers better intraoperative sedation and a shorter arousal time.In 1898, August Bier used the local anaesthetic cocaine to execute the first human case of spinal anaesthesia at the Royal Surgical Hospital of the University of Kiel in Germany. This incident marked a turning point in the development of anaesthetic. The first significant regional method to be used in clinical settings was spinal anaesthesia. The successful use of procaine by Braun in 1905, tetracaine by Sise in 1935, lidocaine by Gordh in 1949, chloroprocaine by Foldes and McNall in 1952, bupivacaine by Emblem in 1966, and ropivacaine and levobupivacaine in the 1980s led to the development of spinal anaesthesia.[4]

Pagés originally described lumbar epidural anaesthesia in humans in 1921. Hingson offered continuous caudal for obstetrics in 1941, and Curbelo introduced lumbar epidural catheterization for surgery in 1947. Factors that affect Intrathecal spread of drug[5] includes Baricity ,Dose, volume, concentration,Temperature of injection, Viscosity, Additives and Patient factors include CSF volume, Advanced age ,Pregnancy,Height and weight Spinal anatomy Intraabdominal pressure.

Physiological responses of central neuraxial blockade[6] are as follows

Cardiovascular physiology: Neuraxial blockade can cause decrease in blood pressure (33% in non-obstetric populations) decrease in heart rate (13% in non-obstetric populations) and decrease in cardiac contractility. These cardiovascular changes are due to the blockage of sympathetic efferent fibers and are mostly related to block height.Veins and arteries enlarge as a result of sympatheticectomy. The arterial system only holds 25% of the blood's volume, whereas the venous system holds around 75% of it. Decrease in blood pressure are mostly caused by venous system dilatations. Bradycardia is

brought on by sympathetic blockage of the cardioaccelerator fibres from T1 to T5, which results in parasympathetic control of heart rate through the vagus nerve. Heart rate may also decline as a result of a decrease in SVR, decreased right atrial filling, and decreases in the intrinsic chronotropic stretch receptor response (Bezold – Jerisch Reflex).[6]

Respiratory physiology : In patients without any underlying lung illnesses, pulmonary function and respiration are typically retained with spinal blockage to midthoracic level gas exchange. In healthy people, there is no change in tidal volume, respiratory rate, minute ventilation, or lung volume. The intercostal and abdominal muscles, which are important for active expiratory function as well as the ability to cough and remove secretions, may function less effectively as a result of neuraxial blockage.

Gastrointestinal, Hepatic, and Genitourinary Physiology: Due to "parasympathetic dominance," the sympathectomy of spinal anaesthesia results in sphincter relaxation, intestinal constriction, and an increase in secretions. The occurrence of nausea during spinal anaesthesia is explained by this autonomic nerve system imbalance. Although there is no discernible difference, hepatic blood flow declines when mean arterial pressure falls. Similar to this, when perfusion pressure is sufficient, renal blood flow and renal function are maintained under spinal anaesthesia. At the lumbar and sacral levels, a subarachnoid block effectively inhibits sympathetic and parasympathetic regulation of the bladder. Loss of autonomic bladder control results in urinary retention. In the recovery unit, postoperative urine retention affects roughly 16% of patients.

Earlier adjuvant medications in neuraxial anaesthesia were mainly centered in increasing the duration and intensity of blockade. After the number of ambulatory surgeries are increasing interest is shifting to identifying adjuvants that will augment block depth and reliability without prolonging the recovery especially motor recovery. Various intrathecal, intravenous and inhalational agents have been used as adjuvants.Classification of intravenous drugs used to augment regional anaesthesia[7] includes opiods like Fentanyl, Nalbuphine ,Diamorphine and Nonopioids like Alpha 2 agonist – Clonidine, Dexmedetomidine, Ketamine ,Magnesium,Calcium channel blocker – Nimodipine and NSAIDS - Tinoxicam

The sympathomimetic substances known as alpha-adrenergic agonists stimulate alpha adrenergic receptors. The two subtypes of the alpha-adrenergic receptor are alpha1 and alpha 2. Target tissues are stimulated by postjunctional a1 receptors whereas they are inhibited and negatively fed back by prejunctional a-2 receptors. The G-protein-coupled a2 receptors come in three subtypes: a-2A, a-2B, and a-2C.[8]A-2 agonists include guanabenz, guanfacine, clonidine, tizanidine, medetomidine, and dexmedetomidine..Mechanism of action is as follows.The central nervous system houses the majority of the a-2A and a-2C receptors. These receptors may be stimulated in order to provide sedation, analgesia, and sympatholytic effects.The vascular smooth muscle has a-2B receptors that mediate the effects of vasopressors. [9]

All three a-2 receptor subtypes block adenylyl cyclase, which lowers levels of cyclic adenosine monophosphate and causes noradrenergic neurons in the locus ceruleus of the medial dorsal pons to become hyperpolarizedAs a result of which potassium efflux through activated calcium channels. It suppresses neuronal activation by preventing calcium ions from entering the nerve

terminal. Hypnosis and sedation are the results of this suppression, which prevents norepinephrine release and lowers activity of ascending noradrenergic pathways.

This negative feedback loop's activation will also lower blood pressure and heart rate. Nociceptive neurons are inhibited and the production of substance P is reduced when a-2 receptors in the dorsal horn of the spinal column are stimulated. Although there is some evidence of supraspinal and peripheral sites of action, the majority of the analgesic activity of a-2 agonist drugs is produced by this spinal mechanism.

Dexmedetomidine is a powerful and highly selective agonist of the -2 adrenoceptor that has sedative, anxiolytic, analgesic, and sympatholytic effects without depressing breathing.[10] Dexmedetomidine was licenced by the Food and Drug Administration (FDA) in December 1999 and made available in clinical settings as a short-term sedative (less than 24 hours).[11]It has an approximately 1600:1 a-2 to a-1 adrenoceptor specificity ratio, which is 7–8 times larger than clonidine's.[12.]Dexmedetomidine is a dextrorotatory S-enantiomer of medetomidine, is an 2-adrenergic receptor agonist with the chemical structure being (S)-4- [1-(2, 3-dimethylphenyl) ethyl]-3H-Imidazole.[13]

Dexmedetomidine is rapidly distributed after intravenous injection, with a distribution half-life of about 6 minutes, a terminal elimination half-life of about 2 hours, and a steady distribution volume of around 118 litres.The estimated clearance speed is 39 L/h[14]. Through direct glucuronidation and cytochrome P450 metabolism, dexmedetomidine goes through a full biotransformation. Metabolites of biotransformation about 95% is excreted in urine and 4 % excreted in faeces. [15] Even in senior patients, the pharmacokinetic profile does not differ according on gender or age, and patients with renal failure do not see any variations in the pharmacokinetics of the active dexmedetomidine molecule.

Sedative and analgesic effects are as follows .Dexmedetomidine induces an “arousable sedation” or “cooperative sedation”, which shows an easy transition from sleep to wakefulness. [16] Dexmedetomidine produces dose-dependent sedative effects. It can provide adequate sedation in critically ill patients to “complete” anaesthesia when used in high concentrations. High concentrations of dexmedetomidine is limited due to its potential for systemic and pulmonary hypertension, direct peripheral vascular effects and its potential to compromise myocardial function.[17]Dexmedetomidine has helped to reduce opioid requirements in the intraoperative period and post anesthesia care unit (PACU). Intra operative use of dexmedetomidine has helped to reduce doses of anesthetics, resulting in more rapid recovery from anesthesia and reduced need for pain medication in the PACU, thereby reducing the length of stay. Dexmedetomidine is about 3.5 times more lipophilic than clonidine and 8–10 times more selective α2 adrenergic receptors agonist. So it prolongs the duration of sensory and motor blockade induced by local anaesthetics, irrespective of the route of administration.[18]

Haemodynamic and Respiratory Effects are as follows. Dexmedetomidine's primary pharmacological impact is to lessen the sympathetic nervous system's excitability, which in turn dampens the stress response and stabilises hemodynamics. Dexmedetomidine's loading dose causes a brief rise in blood pressure and a reflex decrease in heart rate due to vasoconstriction brought on by the stimulation of peripheral -2B receptors in vascular smooth muscle, followed by hypotension when the

central -2A receptors' vasodilatory effects predominate.. Dexmedetomidine has a cardioprotective effect by inhibiting norepinephrine activity in the locus coeruleus, which lowers blood catecholamine levels, cardiac strain, and myocardial oxygen consumption.[19] As a result, it lessens the likelihood of arrhythmias in high-risk patients and prevents myocardial ischemia during the perioperative period.Even at greater doses, dexmedetomidine causes very slight respiratory depression. In some circumstances, such as awake intubation and awake craniotomy, this may offer excellent protection against unfavourable respiratory events.

A centrally acting 2-adrenergic agonist is clonidine. In 1962, Germany performed the first synthetic synthesis of it. It was first sold as an antihypertensive drug, but later studies focused on its effects on analgesia, antiemesis, bleeding control, hemodynamic stability, hormonal stability, reduced oxygen consumption, renal protection, anaesthetics-sparing effect, anxiolysis, sedation, anti-shivering, and myocardial protection..[20]Clonidine hydrochloride is an imidazoline derivative which is a centrally acting alpha-2 adrenergic agonist. The chemical name for clonidine is 2-((2,6-dichlorophenyl) amino)-2-imidazoline hydrochloride.[21]

An intravenous infusion of clonidine hydrochloride has an initial period of rapid dispersion lasting 11–9 minutes, followed by a phase of slower elimination lasting 9–2 hours over the course of 24 hours. Clonidine has an overall clearance rate of 219–92 mL/min. The distribution half-life of bolus dosages of clonidine is 1.2 hours, and the terminal half-life is 14.6 hours.22 Clonidine is metabolised to alkaline, neutral, and acidic conjugates in around 50% of the supplied dose.[23]These metabolites have minor role in human and are excreted mainly by kidney. Elimination half life is prolonged in patients with renal impairment. Hemodynamic and cardiac effects of clonidine includes it reduces blood pressure and cardiac output in patients. The brainstem's pre-synaptic alpha-2 adrenoreceptors are stimulated, which reduce norepinephrine release and boost parasympathetic activity. Reduced heart rate and stroke volume without a corresponding change in peripheral vascular resistance cause a reduction in cardiac output. Due to enhanced cardiac vagal activity and decreased peripheral sympathetic activity, clonidine lowers heart rate. Myocardium is not directly affected by it. The contractility of the myocardium was unaffected by clonidine. Clonidine induced reduction in blood pressure and heart rate will reduce cardiac workload.[24] So this drug is found to be beneficial in patients with coronary artery disease and it has a coronary vasodilatory effect.

Clonidine's sedative and analgesic effects are likely brought on by how it affects the locus coeruleus. Clonidine may be useful as an adjuvant analgesic to opioids in the perioperative context, reducing the overall narcotic requirements. Clonidine's sedative and anti-nociceptive actions are caused by its stimulation of central post-synaptic alpha-2 adrenoreceptors in the brain stem and spinal cord. However, nucleinidine did not lessen cardiac contractility.[25] In patients undergoing lower abdominal procedures under subarachnoid block, alpha 2 agonist is utilised as an adjuvant to extend the duration of postoperative analgesia. Dexmedetomidine and clonidine are used as an adjuvant to local anaesthetics in sub arachnoid blocks in the majority of trials. In this study, the duration of post-operative analgesia will be assessed in relation to the use of intravenous dexmedetomidine and clonidine as adjuvants to local anaesthetics in subarachnoid

blocks.We hypothesised that there would be no difference between the post-operative analgesia provided by dexmedetomidine and clonidine in lower abdominal procedures that involved subarachnoid blocks.

Materials And Methods

This prospective observational study was conducted among 60 Patients undergoing lower abdominal surgery in MES medical college from January 2021 to December 2021.

To estimate an appropriate sample size, the formula used will be

n= (Z(1-α/2) +Z(1-β)) 2 (Ϭ1 2 +Ϭ2 2 /r)/ (μ1 -μ2) 2

α =0.05, β=0.2

Mean in group 1 (μ1) = 243.85

Standard deviation in group 1 (Ϭ1) = 56.82

Mean in group 2 (μ2) = 190.93

Standard deviation in group 2 (Ϭ2) = 42.38

r= ratio = 1

Sample size in each group(n) = 30

A total of 60 patients were selected through convenient sampling.

Those patients of age between 20 and 60 years havin BMI of range 18.5-24.9 Kg/m2 and belongs to ASA I and ASA II patients of either sex were included in the study after taking consent and those with contraindications to tramadol, Diclofenac and local annaesthetics and spinal anaesthesis were excluded from the study along with pregnants and those with spinal deformities .

The institutional ethics committee approval was obtained for the conduct of the study. Patients who got intravenous clonidine on the day of surgery were placed in Group 2, while those who received intravenous dexmedetomidine were placed in Group 1. Electrocardiography (ECG), non-invasive blood pressure (NIBP), and pulse oximetry (SpO2) were attached when the patient was moved to operating room standard monitors, and the baseline data were recorded. According to standard routine protocol patients in Group 1(n=30) received dexmedetomidine 1 μgkg-1 and patients in Group 2 (n = 30) received clonidine 1 μgkg-1, each premixed with saline to a total volume of 10 ml and administered intravenously over a period of 10 min as a single dose through a syringe pump at the rate of 60 ml/hr. A lumbar puncture was conducted at the L3-L4 spinal interspace using a typical midline technique and a 25-G Quincke spinal needle five minutes after the infusion had ended. As per departmental policy, 3 ml of 0.5% Bupivacaine were administered intrathecally during hyperbaric treatment. Dexmedetomidine 0.5 g/kg/hr (Group 1) and Clonidine 1 g/kg/hr (Group 2) were then infused until the surgery was complete.

The VAS (0 - No pain; 10 - Worst possible pain) was used to measure postoperative pain for the first four hours, then every hour for the following four, every two hours for the following 12 hours, and every four hours for the following 24 hours. Rescue analgesic iv. TRAMADOL 50 mg in 100 ml Normal Saline was administered over 10 minutes to patients with a VAS score of 3 or above. The length of postoperative analgesia was determined by recording the time at which postoperative analgesia was first requested following surgery.. Following which post-operative pain was managed with multimodal analgesics as per institutional protocol i.e. injection DICLOFENAC SODIUM 75 mg in 100 ml normal saline as slow iv over 15 minutes after test dose and injection PARACETAMOL 1 gm over 10 minutes every 8th hourly. Break through pain was managed with injection TRAMADOL 50 mg in 100 ml normal saline over 10 minutes. The total number of

times break through which analgesic was given after the first rescue dose was recorded for each group.

Data collected was entered in Microsoft Excel. Quantitative data was analyzed in terms of means and standard deviations. All data collected was analyzed by SPSS version 15 (Statistical Package for Social Sciences). Comparison of normally distributed continuous variables between the 2 groups were done using Z test. A p value <0.05 will be considered as statistically significant.

Results

Out of 60 participants, male and female cases are almost same in Group D (33.3%) and Group C (30.0%). So, sex is comparable between groups. The age is almost same in Group D (40.87 ± 9.684) and Group C (41.17 ± 9.660). So, age is comparable with a p value >0.05. ASA I cases are almost same in Group D (66.7%) and Group C (53.3%). So, ASA grade is comparable (Figure 1).

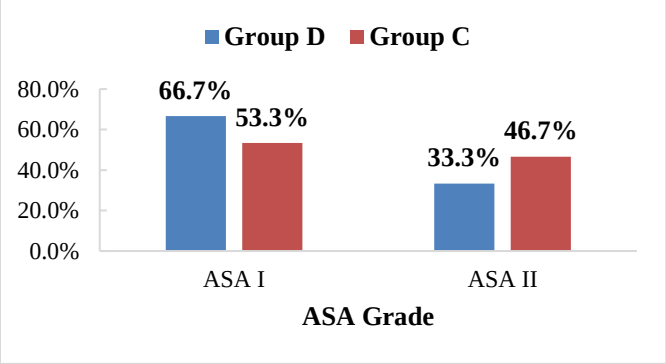

Figure 1: Comparison of ASA grade between groups

Table 1 shows the difference in visual analog scale between the groups. Table & figure 2 shows that the time of first postoperative analgesic request is significantly higher in dexmedetomidine group (251.8 ± 11.94) compared to clonidine group (179.4 ± 6.926).

Table 1: Difference in Visual Analog Scale between Groups

Visual Analog Scale	Dexmedetomidine	Clonidine	p - value
0 Minutes	0.00 ± 0.00	0.00 ± 0.00	1.000
30 Minutes	0.00 ± 0.00	0.00 ± 0.00	1.000
1 Hour	0.00 ± 0.00	0.00 ± 0.00	1.000
1.5 Hours	0.00 ± 0.00	0.40 ± 1.04	0.040
2 Hours	0.00 ± 0.00	1.90 ± 1.47	0.000
2.5 Hours	0.30 ± 0.92	0.80 ± 1.35	0.098
3 Hours	1.70 ± 1.51	0.00 ± 0.00	0.000
3.5 Hours	0.90 ± 1.40	0.00 ± 0.00	0.001
4 Hours	0.10 ± 0.55	0.00 ± 0.00	0.317
5 Hours	0.00 ± 0.00	0.00 ± 0.00	1.000
6 Hours	0.00 ± 0.00	0.00 ± 0.00	1.000
7 Hours	0.00 ± 0.00	0.20 ± 0.76	0.154
8 Hours	0.10 ± 0.55	0.30 ± 0.92	0.305
10 Hours	0.30 ± 0.92	0.00 ± 0.00	0.078
12 Hours	0.00 ± 0.00	0.00 ± 0.00	1.000
16 Hours	0.20 ± 0.76	0.10 ± 0.55	0.557
20 Hours	0.00 ± 0.00	0.00 ± 0.00	1.000
24 Hours	0.00 ± 0.00	0.00 ± 0.00	1.000

Test Applied: Mann-Whitney test

P value <0.05 significant.

Table 2: Difference in Time of First Postoperative Analgesic Request between Groups

Group	Mean	SD	P - value
Dexmedetomidine	251.8	11.94	0.000[S]
Clonidine	179.4	6.926	

Test applied: Independent sample t-test

P value <0.05 significant

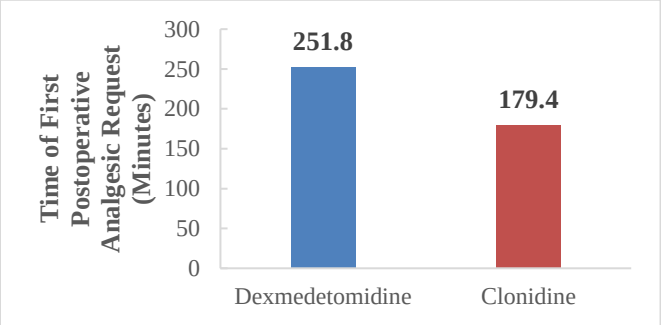

Figure 2: Difference in Time of First Postoperative Analgesic Request between Groups

Discussion

For peripheral nerve blocks as well as intrathecal, epidural, caudal, and intravenous local anaesthetic adjuvants, clonidine and dexmedetomidine have been utilised. Compared to clonidine, dexmedetomidine has an eight-fold higher affinity for alpha 2 adrenergic receptors and much fewer effects on alpha 1 receptor. When it comes to alpha 2A receptor selectivity, which is what gives these medications their hypnotic and analgesic effects, dexmedetomidine has a major edge over clonidine..[26]

By acting on spinal, supra-spinal, directly analgesic, and/or vasoconstricting activities on blood vessels, alpha 2-agonists cause analgesia. The locus ceruleus and the dorsal raphe nucleus are the major neuronal regions where these medicines operate to cause drowsiness and analgesia. This supraspinal effect may be the reason why dexmedetomidine and clonidine administered intravenously extend spinal anaesthesia.[2]

In our study the time when patient was given first post-operative analgesic was 251.8 minutes in Group D and 179.4 minutes in Group C. This was highly statistically significant between two groups showing significant increase in post-operative analgesia in dexmedetomidine group.

In study conducted by Reddy et al, dexmedetomidine had an increased time to first request for postoperative analgesia (243.35 ± 56.82 min) compared with clonidine (190.93 ± 42.38 min) and placebo (140.75 ± 28.52 min) which is similar to our study.

A study by Hasoor et al. compared 12.5 mg of hyperbaric bupivacaine with 0.5 mcg/kg of IV dexmedetomidine and a comparable volume of intravenous normal saline prior to subarachnoid block, followed by a maintenance infusion of dexmedetomidine at a rate of 0.5 mcg/kg/h and a similar rate of infusion of normal saline. They noticed that the dexmedetomidine group's analgesia duration was much longer than the normal saline group's (222.81±23.4 min) was (138.36±21.62 min).This is comparable to our study.[27]

Agarwal et al compared 2.5 ml of 0.5% bupivacaine mixed with 10 µg fentanyl for subarachnoid block with intravenous dexmedetomidine 1 µg/kg/h for 15 min followed by infusion of 0.3 µg/kg/h, IV Clonidine 2 µg/kg/h for 15 min followed by infusion of 0.5 µg kg/h or 15 ml of normal saline for 15 min followed by infusion at 50 ml/h. They observed first requirement for postoperative analgesic was after 353.13 ± 39.60 min, 314.38 ± 30.64 min and 193.25 ± 17.74 min in Groups I, II and III respectively. Though statistically significant duration of analgesia in dexmedetomidine group and clonidine group was more than our study. It could be due to addition of 25 mcg fentanyl with bupivacaine for subarachnoid block for lower limb surgeries. [1]

Conclusion

In our present study we have studied 60 patients divided into two groups with 30 patients in each group. Patients in Group 1 (n=30) received dexmedetomidine 1 µgkg-1 and patients in Group 2 (n = 30) received clonidine 1 µgkg-1 5 minutes prior to subarachnoid block. It was

followed by infusion dexmedetomidine 0.5 µg/kg/hr (Group 1) and infusion of Clonidine 1 µg kg/hr (Group 2) till the end of surgery.From this study we have concluded that dexmedetomidine has a prolonged post operative analgesia than clonidine.

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