Intravenous Dexmedetomidine or Propofol Adjuvant to Spinal Anesthesia in Total Knee Replacement Surgery

*Abdelkarim S. AlOweidi, [*1] Mahmoud M. Al-Mustafa, [1] Jihad M. Al Ajlouni, [2] Diana F. Mas'ad, [3] Mohammad Q. Hamdan, [2] Subhi M. Alghanem, [1] Ibraheem Y. Qudaisat, [1] Sami A. Abu Halaweh, [1] Islam M. Massad [1]*

Abstract

Background: The purpose of this study was to compare the effect of intravenous dexmedetomidine with the intravenous propofol adjuvant to spinal intrathecal anesthesia on the duration of spinal anesthesia and hemodynamic parameters during total knee replacement surgery.

Methods: Seventy five patients were enrolled into this randomized study from the 1st of April 2008 till the 30th of August 2009 for total knee replacement surgery under spinal anesthesia. They were randomly assigned into three groups, group D, group P and group C. Following intrathecal injection with bupivacaine 12.5 mg in all patients in the three groups, patients in group D received intravenous loading dose of 1μg/kg/hr dexmedetomidine over 10 minutes and a maintenance dose of 0.2 to 0.5 μg/kg/hr. Patients in group P received intravenous propofol 4 mg/Kg/hr over 10 min and a maintenance dose of 0.5-2 mg/Kg/hr. Patients in group C (control group) received nothing extra to the regular IV fluid. The regression times to reach S1 sensory level and Bromage 0 motor scale, the hemodynamic parameters, the Bispectral Index level of Sedation (BIS), and arterial CO_2 level were recorded.

Results: The regression time to reach S1 dermatome was 149.4±14.6 min in group C, 152.8±16.6 min in group P and 209.6±25.9 min in group D. The regression to Bromage 0 was184.6±22.8 min in group C, 190.0±21.0 min in group P, and 255.8±36.7 min in group D. Statistical analysis of regression of sensory and motor block was significant among groups (C vs. D, P vs. D, $P < 0.05$). The heart rate was significantly decreased in group D in comparison to groups C and P. Sedation levels were within accepted ranges in groups D and P and not affected in the control group. Minimal respiratory depression occurred in group P and D, clinically it was not significant.

Conclusion: Supplementation of spinal anesthesia with intravenous dexmedetomidine or propofol produces good sedation levels without significant clinical hemodynamic changes. Adding dexmedetomidine produces significantly longer sensory and motor block than propofol.

Keywords: Dexmedetomidine, Propofol, Spinal Anesthesia, Total Knee Replacement.

1. **Department of Anesthesia and Intensive Care, Jordan University Hospital, The University of Jordan, Amman, Jordan.**
2. **Department of Special Surgery, Orthopedic Division, Jordan University Hospital, The University of Jordan, Amman, Jordan.**
3. **Department of Family and Community Medicine, Jordan University Hospital, University of Jordan, Amman, Jordan.**
* *Correspondence should be addressed to:*
Abdelkarim S. AlOweidi
P. O. Box: 13046, Amman, Jordan 11942
E-mail: akaloweidi@hotmail.com

Introduction

Total Knee Replacement (TKR) is common orthopedic surgery that can be performed using Spinal Anesthesia (SA) or General Anesthesia (GA). Spinal anesthesia reduces the operating time, the need for blood transfusion and the incidence of thromboembolic disease (deep-vein thrombosis and pulmonary embolism). [1] Spinal anesthesia therefore seems to improve the outcome of patients undergoing total hip or knee replacement.

During the operations performed under spinal anaesthesia, sedation of the patients is often required. Many agents (Midazolam, Ketamine, Remifentanil, Propafol and Dexmedetomidine) have been used for this purpose. Continuous infusion of propofol is a useful sedation method because of the easy management by titration and rapid emergence. [2] Dexmedetomidine is a α_2 - agonist that has been used for pre-medication and as an adjunct to general anesthesia. [3-5] Intravenous Dexmedetomidine decreases the inhalational anesthesia and opioid requirement during general anesthesia. [6] Intravenous dexmedetomidine, proved to prolong the duration of spinal anesthesia, provided sufficient sedation and had few side effects. [7, 8]

Dexmeditomidine and propofol were not compared as sedative agents adjuvant drug to spinal anesthesia before. The aim of this study was to compare the intravenous dexmedetomidine with the intravenous propofol adjuvant to spinal anesthesia in patients who underwent total knee replacement surgery on the duration of spinal anesthesia, hemodynamic and sedation effect.

Methods

After obtaining the approval of the ethics committee of the Jordan University Hospital and an informed consent was given by each patient, seventy five patients ASA I-III scheduled for TKR surgery were enrolled in this randomized study from the 1st of April 2008 till the 30th of August 2009 at the University of Jordan, Jordan

University Hospital. All the surgeries were done by the same surgeon. Patients using α_2 – adrenergic receptors antagonists, calcium channel blockers, angiotensin converting enzyme inhibitors, having dysrhythmia in the electrocardiogram, a body weight more than 120 Kg, or height less than 150 Cm were excluded from the study. All the patients were hydrated with 300 ml of Ringer's Lactate solution via an 18-gauge IV cannula in the dorsum of the hand before spinal anesthesia. All patients had an invasive arterial blood pressure monitoring through insertion of an arterial canula in the right or left radial artery to measure arterial CO_2 level. Standard monitoring was used, including Invasive Arterial Blood Pressure (IABP), ECG, Heart Rate (HR), Bispectral Index (BIS) and pulse oximetry. Using a computer-generated random list, the patients were divided into three groups of 25 individuals, group D, P and C.

With the patient in the sitting position, a spinal analgesia was performed at the level of L3-L4 through a midline approach using a 25 –gauge Quincke spinal needle (B/Braun Medical, Messenger, Germany) with the hole pointing upwards. If the spinal block failed at the level of L3-L4, we changed the level to L2-L3. In case of failure at both levels, the procedure was planned to be abandoned, general anesthesia alone will be given and the patients will be excluded from the study protocol. All patients in the three groups received standard intrathecal local anesthesia (isobaric 0.5 % bupivacaine, 12.5 mg, 2.5 ml). The spinal injection rate was 1ml / 3-4 seconds. Immediately after spinal anesthesia, patients in group D were put to supine position, then started to receive intravenously dexmedetomidine. (Precedex 100 µg /ml; Hospira, Inc.) which was diluted with normal saline in a concentration of 4µg/ml with loading dose of 1µg/kg/hr dexmedetomidine over 10 minutes and a maintenance dose of 0.2 to 0.5 µg/kg/hr to keep the BIS monitor reading above 70 and below 85. The patients who were allocated to group P received intravenously a loading dose of 4 mg/kg/hr propofol over 10 min and a maintenance dose of 0.5 to 2 /kg/hr to keep the BIS monitor reading above 70 and below 85.

Patients in group C (control group) had no adjuvant sedative agent to spinal anesthesia. Patient motor power and sensation to cold using alcohol solution up to T10 dermatome were examined in both sides and lower extremities. All the patients received 4 L/ min of O2 by simple face mask.

The anesthesiologist performing the block recorded the baseline value of vital signs (Mean blood pressure, heart rate and peripheral oxygen saturation) and after performing the spinal analgesia, the vital signs were recorded every 15 minutes in the operation room and in the Post Anesthesia Care Unit (PACU) until the patients were discharged from PACU. Arterial blood gas sample was sent for analyses before spinal anesthesia and at 15, 60 and 120 min after spinal anesthesia to measure CO_2 levels in all patients. BIS monitor attached to the patient before performing spinal anesthesia and numerical values recorded before spinal anesthesia and every 15 min after spinal anesthesia till 120 min. Our aim is to keep the BIS value above 70 and below 85. In the PACU, the sensory level and Bromage scale were recorded every 15 minutes until the patient was discharged from the PACU. The times of regression to the S1 dermatome and Bromage scale 0 in PACU were recorded. The sensory level was assessed by cold sensation using alcohol swab along the mid-clavicular line bilaterally. The motor level was assessed according to the modified Bromage scale: Bromage 0, the patient is able to move the hip, knee and ankle; Bromage 1, the patient is unable to move the hip, but is able to move the knee and ankle; Bromage 2, the patient is unable to move the hip and knee, but is able to move the ankle; Bromage 3, the patient is unable to move the hip, knee and ankle. All durations were calculated considering the time of spinal injection at time zero. When sensory levels of anesthesia were not equal bilaterally, the higher level was used for the statistical analysis. Patients were discharged from the PACU after sensory regression to the S1 segment and Bromage scale 0.

In our study, hypotension was defined as a systolic blood pressure of less than 90 mmHg and was treated with a bolus administration of 300 ml of lactated Ringer's solution over 10 min and 6 mg of intravenous ephedrine. Bradycardia was defined as HR<50 beats/min, and if maintained was treated with 0.5 mg of intravenous atropine.

All patients were examined in the outpatient clinic two weeks following discharge. The doctor aimed to assess any neurological impairment related to spinal analgesia such as back, buttock or leg pain, headache or any neurological deficit.

Statistical Methods

Statistical analysis was done using Microsoft Excel 2003. Data were expressed as either mean and standard deviation or numbers and percentages. The demographic data of patients were studied for each of the three groups. The means for the continuous variables Age, Body Mass Index (BMI), the American Society of Anesthesiologists (ASA) classification, duration of surgery, total intravenous infusion and motor or sensory block regression time were compared between the three groups using analysis of variance ANOVA. The p value of ≤ 0.05 was considered significant.

Results

All the seventy five patients who were enrolled in the study completed the study protocol and were included in the data analysis. No spinal analgesia failure was anticipated. Thus, each group consisted of 25 patients. Demographic data, the duration of surgery, the total amount of fluids administered following spinal analgesia did not differ between the three study groups (Table 1). The time to regression to S1 dermatome and Bromage scale 0 was significantly prolonged in group D in comparison with groups P and C. The regression time to S1 was 149.4±14.6 min in group C, 152.8±16.6 min in group P and 209.6±25.9 min in group D, (P<0.0001). The regression time to reach the Bromage 0 scale was 184.6±22.8 min in group C, 190.0±21.0 min in group P, and 255.8±36.7 min in group D, (P <0.0001) (Table 2). The need to give ephedrine

or atropine, breadycardia, hypotension, the need to additive analgesia, blood transfusion and nausea or vomiting in the intraoperative or PACU time were comparable in the three groups (Table 3).

The mean values of mean arterial pressure in the first 90 min after performing the spinal analgesia and the first 90 min the PACU (Recovery room) were comparable between the three groups (Figure 1). The mean value of heart rate was significantly decreased in group D in comparison with group P and C, and significantly decreased in group P in comparison with group C in the first 90 min in the operation room and first 90 min in the PACU (Figure 2).

The BIS monitor readings were within our target range between 70 and 85 in group D and P after 15 min from the spinal anesthesia and not affected in group C (control group) (Figure 3). The oxygen saturation was higher than 95% in all patients.

The arterial CO_2 levels were comparable before spinal anesthesia and at 15 min after spinal anesthesia in the three groups and after that increased significantly in groups P and D in comparison to group C. This increase in arterial CO2 had no clinical significant since the reading is still within the normal range (Figure 4).

Two weeks following discharge, in the outpatient clinic the follow up did not show any neurological impairment related to spinal analgesia such as back, buttock or leg pain, headache or any new neurological deficit.

Table (1): Demographic data of 75 patients who underwent TKR surgery under spinal anesthesia, Values are the means ± standard deviations or numbers.

	Group C (n=25)	Group D (n=25)	Group p (n=25)
*Age (years)**	63.8±5.21	64.64±6.75	66.56±7.66
*Sex (Male/ Female)**	6/19	7/18	7/18
BMI	31.845±3.41	30.76±5.54	30.53±7.74
*ASA I/ II/ III**	4/17/ 4	4/ 18/ 3	3/ 19/ 3
*Duration of Surgery (min)**	96.2±8.9	98.8±8.3	97.2±7.1
*Total IV infusion (ml)**	1502±161	1508±227	1522±226

* P value > 0.05, statistically not significant.
BMI: Body Mass Index
ASA: American Society of Anesthesiologists

Table (2): Spinal block regression times in minutes of 75 patients who underwent TKR surgery. Values are the means ± standard deviations.

Spinal Characteristics	Group C (n=25)	Group D (n=25)	Group P (n=25)	P value
Motor block regression to Bromage 0	184.6±22.8	255.8±36.7	190.0±21.0	<0.0001
Sensory regression to S1 segment	149.4±14.6	209.6±25.9	152.8±16.6	<0.0001

Table (3): Adverse events and treatment. Values are the means ± standard deviations or numbers.

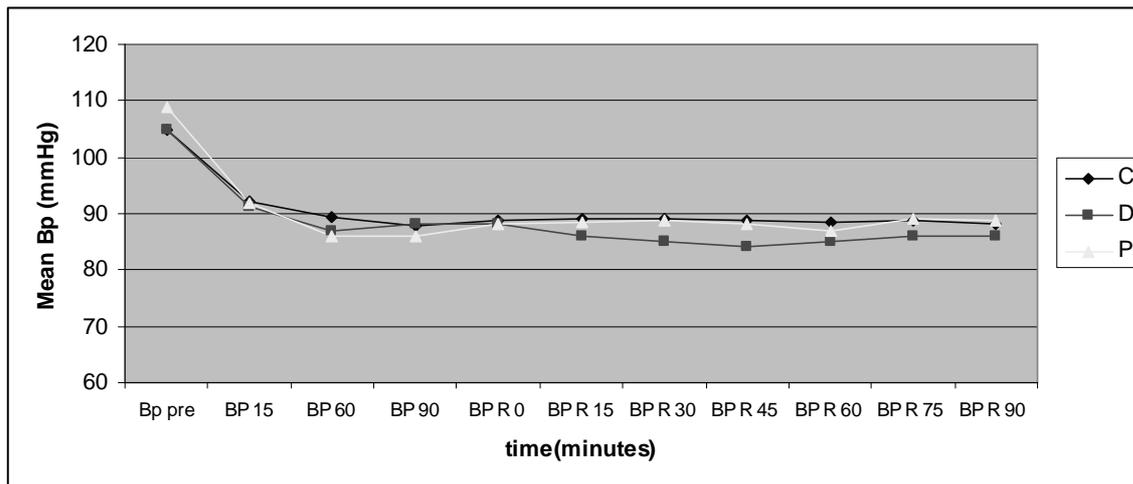| Adverse events and treatment	Group C No. of patients	Group D No. of patients	Group P No. of patients
Blood transfusion	1	1	1
Additive analgesia	0	0	0
Nausea / Vomiting	0	0	0
Bradycardia	0	1	0
Hypotension	1	1	2
Atropine	0	1	0
Ephedrine	1	1	2

Figure (1): Comparison of mean Blood Pressure (BP) levels among patients in C, D and P groups in the first 90 min after spinal anesthesia and the first 90 min in Recovery Room (R). Values are expressed as means.
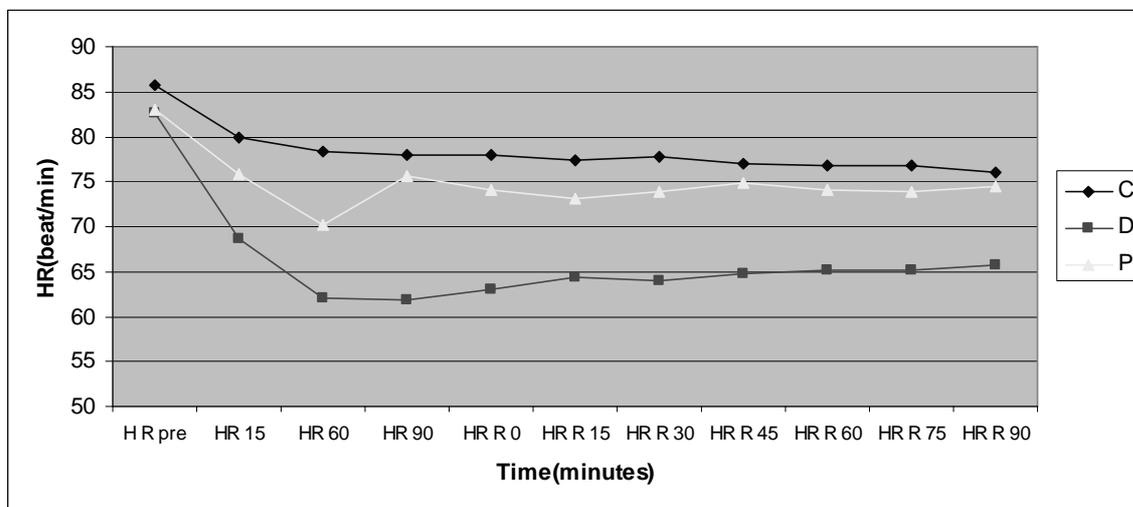

Figure (2): Comparison of Heart Rate (HR) levels among patients in C, D and P groups in the first 90 min after spinal anesthesia and in the first 90 min in Recovery Room(R). Values are expressed as means.
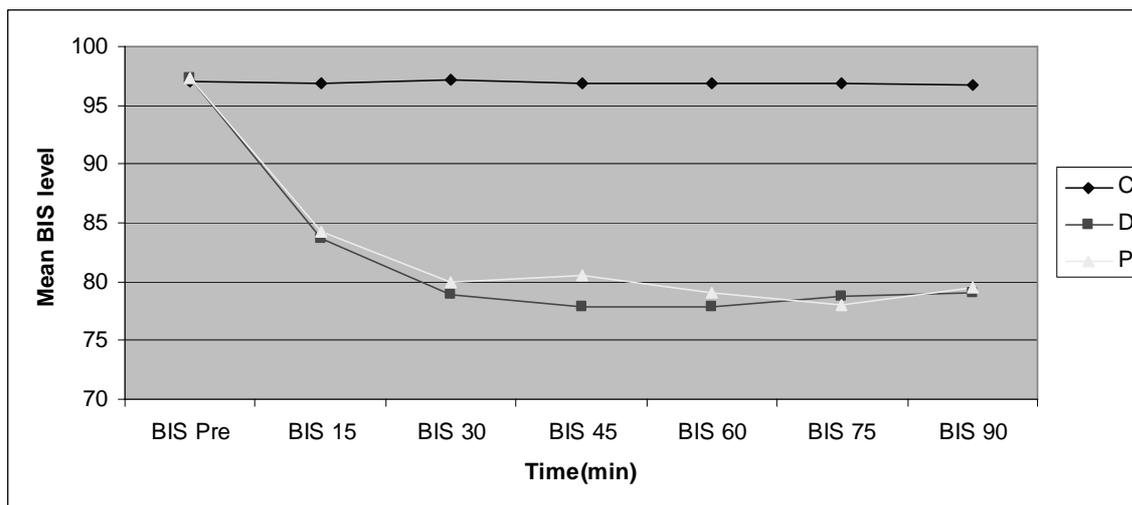
Figure (3): Comparison of Bispectral Index (BIS) levels of sedation among patients in C, D and P groups in the first 90 min after spinal anesthesia. Values are expressed as means.
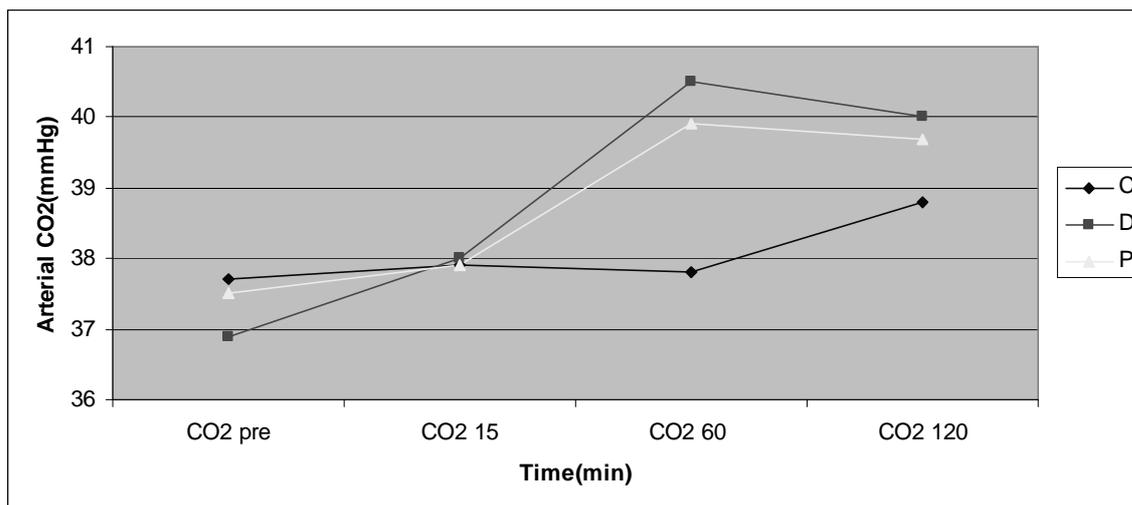

Figure (4): Comparison of Carbon dioxide levels (CO_2) values among patients in C, D and P groups before spinal anesthesia and at 15, 60 and 120 min after spinal anesthesia. Values are expressed as means.

Discussion

Different intravenous drugs have been used as adjuvant to spinal anesthesia as sedative agents such as propafol, dexmedetomidine, remifentanil, ketamine and medazolam. In our study, we compared the effect of using intravenous dexmeditomidine and propofol adjuvant to spinal anesthesia on the duration of spinal anesthesia. No previous studies were compared to these two agents when used as sedatives with spinal anesthesia.

Propofol is a short-acting, intravenously administered hypnotic agent. Its uses include the induction and maintenance of general anesthesia, sedation for mechanically ventilated adults, and procedural sedation and adjuvant to spinal anesthesia as sedative agent. [9] It has several mechanisms of action, [10] both through potentiation of $GABA_A$ receptor activity, thereby slowing the channel-closing time, and also acting as a sodium channel blocker.

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Recent research has also suggested that the endocannabinoid system may contribute significantly to propofol's anesthetic action and to its unique properties. [11] The characteristics of rapid onset and recovery along with its amnestic effects have led to its widespread use for sedation and anesthesia. Intravenous dexmedetomidine was used as adjuvant sedative agent with spinal anesthesia and it had the property of prolongation of the motor and sensory regression of spinal anesthesia. [8] Dexmedetomidine is α_2 agonist and produces sedation and anxiolysis by binding to α_2 receptors in the locus ceruleus, which diminishes the release of norepinephrine and inhibits sympathetic activity, thus decreasing heart rate and blood pressure. It produces analgesia by binding to adrenoreceptors in the spinal cord. Jorm et al. [12] found in their study that dexmedetomidine has an inhibitory effect on the locus ceruleus (A6 group) which is located at the brain stem. This supraspinal action could explain the prolongation of spinal analgesia after intravenous administration of dexmedetomidine.

The noradrenergic innervation of the spinal cord arises from the noradrenergic nuclei in the brain stem including the locus ceruleus, the A5, and the A7 noradrenergic nuclei. Neurons in the locus ceruleus are connected to the noradrenergic nuclei in the brain stem. Axon terminals of the noradrenergic nuclei reach lamina VII and VIII of the ventral horns of the spinal cord. The activity of the noradrenergic neurons is decreased by agonists acting at α_2-adrenergic receptors on the locus ceruleus cell bodies. Therefore, inhibition of the locus ceruleus results in the disinhibition of the noradrenergic nuclei and exerted descending inhibitory effect on nociception in the spinal cord. [13]

Dexmedetomidine has been used as adjuvant to local anesthesia in the intrathecal route; it has a significant effect on the onset and duration of spinal analgesia. [14] Konakci et al. [15] in their study found dexmedetomidine may have a harmful effect on the myelin sheath when administered via the epidural route. This result alarm anesthesiologist about the risk of usage of dexmedetomidine in the epidural or intrathecal route. Side effects of dexmedetomidine as hypotension and breadycardia are dose dependent, infusion of loading dose over 10 min and then infusing the maintenance dose decrease the incidence of this side effect.

In our study, the time to regression to S1 dermatome and Bromage scale 0 was significantly prolonged in group D in comparison with groups P and C. The regression time to reach S1 dermatome was 149.4±14.6 min in group C, 152.8±16.6 min in group P and 209.6±25.9 min in group D. The regression to Bromage 0 was184.6±22.8 min in group C, 190.0±21.0 min in group P, and 255.8±36.7 min in group D ($P < 0.0001$) (Table 2).

Propofol and dexmedetomidine are known to have sedation effect [9, 16] sedation level gives a better condition for the surgeon and the patient, provided that haemodynamic stability is preserved. In our patients, in group D a loading dose of 1μg/kg/hr dexmedetomidine over 10 minutes and a maintenance dose of 0.2 to 0.5 μg/kg/hr and in group P a loading dose of 4 mg/Kg/hr propofol over 10 min and a maintenance dose of 0.5-2 mg/Kg/hr were used. The numerical values of BIS monitor were within our target range and after 15 min started to decrease and remain between 70 and 85 in all patients in group D and P as shown in Figure (3).

The heart rate decreased significantly in group P and D after the start of intravenous infusion loading dose and extends in the PACU compared to group C (Figure 2) and this decrease in the heart rate was more clear and significant in group D in comparison with group P. The lower HR observed in group D could be explained by the decreased sympathetic outflow and circulating levels of catecholamines that are caused by dexmedetomidine. [17, 18] Only one patient in group P developed bradycardia (HR< 50 beat/min) treated with atropine, and no patients in group D or C had significant bradycardia which had no clinical significance. Hypotension after conduction of spinal anesthesia are common and may lead to intraoperative cardiac problems. [19] In our patients, the mean arterial pressure was also

decreased in the three groups in a comparable manner (Figure 1). Four patients developed hypotension (decrease the systolic blood pressure less than 90 mmHg), two in group D, one in group P and one in group C; all patients were treated successfully with IV fluid and ephedrine. Clinically, this was not significant.

In spontaneously breathing patients, the respiratory depressant effect of dexmedetomidine was less remarkable compared with that observed with propofol. [20] Both propofol and dexmedetomidine were known to have minimal respiratory depression when used as sedative agents and this was clear in our results where the arterial CO_2 level increased to acceptable values and clinically had no harm to the patients.

Conclusion

The supplementation of spinal analgesia with intravenous dexmedetomidine or propofol produce good sedation levels in all patients that enable patient's cooperation and potentially better operating conditions for the surgeons without significant respiratory depression. Adding intravenous dexmedetomidine produce significantly longer duration of sensory and motor block after spinal anesthesia, while adding intravenous propofol had no effect on the duration of spinal anesthesia. We endorse the addition of intravenouse dexmedetomidine to spinal anesthesia when prolongation of spinal anesthesia is desired, for example, anesthesia for surgeries such as the revision of hip or knee surgeries.

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Dexmedetomidine أو Propofol وريديا مضافاً إلى التخدير النخاعي في عمليات استبدال مفصل الركبة الكلي

عبد الكريم العويدي، [1] محمود المصطفى، [1] جهاد العجلوني، [1] ديانا مسعد، [2] محمد حمدان، [3] صبحي الغانم، [1] إبراهيم قديسات، [1] سامي أبو حلاوة، [1] إسلام مساد [1]

1– فسم التخدير والعناية الحثيثة، مستشفى الجامعة الأردنية، عمان، الأردن؛ 2– قسم الجراحة الخاصة، شعبة العظام، مستشفى الجامعة الأردنية، عمان، الأردن؛ 3– قسم طب الأسرة والمجتمع، الجامعة الأردنية، عمان، الاردن

الملخص

الخلفية: الغرض من هذه الدراسة مقارنة تأثير (Dexmedetomidine) وريديا مع (Propofol) وريديا كدواء مضاف للتخدير النخاعي على دوام التخدير النخاعي ومؤشرات الديناميكا الدموية خلال عمليات استبدال مفصل الركبة الكلي.

الأساليب: اجريت الدراسة بالطريقة العشوائية على 75 مريضاً من الأول من نيسان 2008 حتى الثلاثين من آب 2009 لعمليات استبدال مفصل الركبة الكلي. قسموا بشكل عشوائي الى ثلاث مجموعات D, P, C . بعد الحقن داخل القراب لـ bupivacaine 12.5 مغ في المجموعات الثلاث مرضى المجموعة D اعطوا وريديا جرعة تحميل 1مكغ/كغ/الساعة من Dexmedetomidine لعشر دقائق ثم جرعة مداومة 0.2–0.5 مكغ/كغ/الساعة. مرضى المجموعة P اعطوا Propofol 4 ملغ/كغ/الساعة لعشر دقائق ثم جرعة مداومة 0.5–2 ملغ/كغ/الساعة. مرضى المجموعة C (مجموعة المقارنة) لم يعطوا الا المحاليل الوريدية. المدة لعودة المستوى الحسي S1 والمقياس الحركي Bromage 0، مؤشرات الديناميكا الدموية، bispectral index لمستوى التهدئة، قيمة ثاني اكسيد الكربون الشرياني قد سجلت.

النتائج: المدة لعودة المستوى الحسي S1 كانت 149.4±14.6 دقيقة في المجموعة C، 152.8±16.6 دقيقة للمجموعة P، 209.6±25.9 للمجموعة D وللمقياس الحركي 0 كان دقيقة 184.6±22.8 للمجموعة C، 190.0±21.0دقيقة للمجموعةP، 255.8±36.7 دقيقة للمجموعة D. التحليل الاحصائي لانحدار الاحصار الحسي والحركي كان هاما ضمن المجموات الثلاث (C vs. D, P vs. D, P < 0.05). سرعة القلب انخفضت بشكل واضح في المجموعة D مقارنة في المجموعتين C وP. مستوى التهدئة كان ضمن المقبول في المجموعتين P,D ولم يتأثر في مجموعة المقارنة . حدث تثبيط تنفسي قليل في المجموعتين P,D لم يكن هاماً من الناحية السريرية.

الاستنتاج: اضافة Dexmedetomidine او Propofol وريديا للتخدير النخاعي يعطي تهدئة جيدة دون تاثير واضح في الديناميكا الدموية سريريا. اضافةDexmedetomidine يسبب احصار حسي وحركي اطول من Propofol بشكل ملحوظ.

الكلمات الدالة: Dexmedetomidine ، Propofol ، تخدير نخاعي، استبدال مفصل الركبة الكلي.