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Research Article

Dexmedetomidine versus nimodipine for controlled hypotension during spine surgery

Hatem Saber Mohamed *, Salah Mostafa Asida, Ossama Hamdi Salman

Anesthesia & Intensive Care, Egypt

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KEYWORDS

Dexmedetomidine;
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Controlled hypotension

Abstract *Background:* Controlled hypotension is a technique that is used to limit intraoperative blood loss, improve operative field, decrease duration of surgery, and thus decrease the amount of blood transfused.

Objective: The aim of this double-blind, randomized, controlled study is to compare the effects of sevoflurane combined with dexmedetomidine (DEX) or nimodipine (NIMO) on blood loss in the surgical field, recovery time, and patient tolerability in spine surgery.

Methods: Forty-eight (48) patients, 20–50 years of age, ASA I-II, randomly divided into DEX group ($n = 24$) and NIMO group ($n = 24$). In the DEX group, a loading dose of DEX infused over 10 min at a rate of 1 μg/kg/h, followed by a maintenance rate of 0.4–0.8 μg/kg/h. In the NIMO group, the dose infused at 15 μg/kg/h for 2 h (approximately 1 mg/h). The infusion rates were titrated to maintain mean arterial pressure (MAP) of 60–65 mm. Hg. We recorded MAP, intraoperative blood loss, total recovery time, total fentanyl consumption, incidence of arrhythmia or ischemia, and postoperative nausea and vomiting.

Results: No significant difference in the amount of blood loss between the two groups was observed. Total fentanyl consumption was significantly higher in the NIMO group (350 ± 8.9 μg) versus (200 ± 5.5 μg) in the DEX group ($p = 0.002$). Recovery time was shorter in the NIMO group (6.8 min) versus (8.9 min) in the DEX group ($p = 0.001$).

Conclusions: Dexmedetomidine and Nimodipine provided effective method of controlled hypotension limiting the blood loss, and NIMO was associated with significantly shorter extubation and recovery times compared with DEX.

* Corresponding author. Address: Department of Anesthesia, Qena University Hospital, Qena, Egypt. Tel.: +20 1005257062.
E-mail address: dr.hatem_saber@hotmail.com (H.S. Mohamed).

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1. Introduction

Significant blood loss during post-traumatic spinal stabilization surgery is a major anesthetic challenge that might require transfusion of whole blood or blood products [1].

Decreasing bleeding is important to maintain patient's hemodynamic stability and improve the surgical field. In spine surgery, improving the surgical field is especially important

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due to the presence of major and easily traumatized neurological structures. Improving the surgical field helps the surgeon to decrease the operative time, which further decreases bleeding [2].

Various agents have been used to provide controlled hypotension including direct acting vasodilators (sodium nitroprusside-nitroglycerin), ganglion-blocking agents, beta adrenergic blockers (esmolol), calcium channel blockers (nicardipine), α2-agonists (clonidine–dexmedetomidine), volatile agents, and magnesium sulfate [3–5].

Unfortunately, the available hypotensive agents are still far from ideal. Some have myocardial depression like esmolol and magnesium sulfate, and others have long postanesthetic recovery when used in high concentration like inhalation anesthetics, while tachyphylaxis and cyanide toxicity are potential side effects of nitroprusside. Moreover, potent hypotensive agents have their own concentration dependent side effects, e.g., inhalation anesthetics when used alone require such a high concentration to achieve a significant reduction in bleeding that hepatic or renal injury might occur.

Ideally, hypotensive agent should be easy to administer, has a short time to onset, has effects that disappear quickly when administration is discontinued with rapid elimination without toxic metabolites, negligible effects on vital organs, and has predictable and dose-dependent effects [3].

Dexmedetomidine is a highly selective α 2-adrenoceptor agonist recently introduced to anesthesia practice. Mediated by its central and peripheral sympathetic action, it has dose-dependent sedative, anxiolytic, and analgesic action without respiratory depression [6]. Its easy administration profile, predictability with anesthetic agents and lack of toxic side effects while maintaining adequate perfusion of the vital organs, makes it a near-ideal hypotensive agent. Spinal fusion surgery for idiopathic scoliosis has been safely undertaken with dexmedetomidine-controlled hypotension [7]. Moreover, dexmedetomidine associated peripheral vasoconstriction resulted in minimal bleeding either with skin incision or intraoperative [8].

Nimodipine, a 1-4 dihydropyridine derivative, is a calcium channel blocking agent. It is a highly lipid-soluble nifedipine analog with ready access to the CNS. It has been used as a prophylactic and therapeutic agent for intracranial hemorrhage associated with cerebral vasospasm [9]. Unlike dexmedetomidine, its desirable hypotensive effect is rather due to peripheral vasodilatation.

The aim of this study is to compare the effects of dexmedetomidine or nimodipine used for controlled hypotension on the amount of blood loss in the surgical field, recovery time, and tolerability in adult patients undergoing posterior lumbar spinal fusion due to traumatic fracture.

2. Patients and methods

2.1. Patient selection

This study was done in Qena University Hospital in the time period from December 2011 to December 2012, the study protocol was approved by the ethics committee of Qena Faculty of Medicine, and Written informed consent was taken from every patient sharing the study or from any legally responsible person. We studied 48 patients aged from 20 to 50 years classified as American Society of Anesthesiologists (ASAs) physical status I-II who were candidate for posterior fusion of the lumbar spine due to traumatic fracture.

We excluded patients with cardiac disease (hypertension-arrhythmia-ischemic heart disease – valve lesions), preexisting coagulation defects, hepatic or renal dysfunction, diabetes mellitus, neuromuscular disorder, and seizure disorder; patients receiving beta blockers, calcium channel blockers, digoxin, anticoagulants, tricyclic antidepressants, and known allergy to any of the drugs used in the study.

2.2. Anesthesia

In this prospective, randomized, double-blind study, we randomly allocated 48 patients into 2 groups according to a computer-generated allocation numbers table. All patients were hospitalized at the department of orthopedic surgery, Qena University Hospital, on the day before surgery and had fasted for at least 6 h before surgery.

No premedication was given and on arrival to the operating room, and two cannulae were inserted at different sites on the same arm (under complete aseptic technique): one (20-G) for the infusion of dexmedetomidine or nimodipine and the other (18-G) for the administration of fluids, blood, and other drugs.

A 20-G cannula was inserted into a radial artery near the wrist joint in the non-dominant hand under complete aseptic technique for direct measurement of arterial blood pressure which was recorded continuously.

Patients in both groups received 7 ml/kg of ringer's solution before induction of anesthesia and were monitored by the same system for heart rate (HR) and mean arterial pressure (MAP) [invasive and noninvasive], 12 lead electrocardiography (ECG), pulse oximetry (SPO2), urine output, and esophageal temperature were also monitored (Nihon Khoden monitor). Normothermia was maintained throughout the procedure with heating mattress and infusion of warm fluids.

Anesthesia was induced with fentanyl 1 μg/kg and propofol 2 mg/kg IV, while 100% oxygen was administered by mask for the first 3 min of induction, IV atracurium 0.5 mg/kg was used to facilitate tracheal intubation.

All patients were mechanically ventilated (Ohmeda Aespire 7100 anesthesia machine) with a tidal volume of 6–10 ml/kg, inspiratory/expiratory ratio of 1:2, and a respiratory rate adjusted to maintain end-tidal carbon dioxide ($ETCO_2$) concentration of 30–35 mmHg (10–16 breaths/min).

All patients were placed prone on a Relton Frame with a chest and pelvic rolls, leaving the abdomen hanging free. All patients were positioned and operated on by the same surgery team.

General anesthesia was maintained with sevofurane (end-tidal concentration of sevoflurane, 1.8–2.5%) in 100% oxygen and adequate muscle relaxation maintained with incremental bolus doses of 10 mg atracurium when indicated by the peripheral nerve stimulation (TOF guard).

Infusions were initiated with dexmedetomidine or nimodipine to achieve MAP between 60 and 65 mmHg, prior to skin incision. This was done by an anesthesiologist other than that who records the results of the study. In the dexmedetomidine (PRECEDEX, Hospira) group, a loading dose was infused intravenously over 10 min at 1 μg/kg diluted in 10 ml. 0.9% normal saline followed by a maintenance infusion rate of 0.4–0.8 μg/kg/h. In the nimodipine (NIMOTOP, BAYER)

group, 15 μg/kg/h (equivalent to 5 ml nimodipine infusion solution per hour) was infused for 2 h and if this dose is well tolerated and there is no severe drop of blood pressure, the dose can be increased after the second hour up to 30 μg/kg/h (equivalent to 10 ml nimodipine infusion solution per hour). Nimodipine infusion solution was administered using an infusion pump and not mixed with other drugs or added to infusion bottles (1 bottle of 50 ml infusion solution contains 10 mg nimodipine).

Intraoperative fluids administered for all patients included ringer lactate as a maintenance fluid and normal saline for deficits and losses, including packed RBCs transfusion for blood loss to a transfusion threshold of hemoglobin = 10 gm/dl and hematocrit of 25–30%.

The intraoperative estimated blood loss (EBL) for each procedure was calculated by weighing the surgical gauze pads and measuring the contents of the suction bottle (with adjustment made for the amount of saline irrigation used) by the same anesthesiologist for all patients who was unaware of the study details. The hemoglobin (HB) level and the hematocrit value (packed RBCs) were obtained on the preoperative day and first postoperative day.

In both groups, signs of inadequate anesthesia (e.g., increases in MAP greater than the target level, movement, tearing, or sweating) were treated with additional I.V. boluses of fentanyl in a dose of 1 μg/kg and recorded. Nitroglycerine was infused if these target levels could not be achieved with the uppermost dose.

Our primary end point was achieving MAP of 60–65 mmHg before skin incision, while secondary end points included; occurrence of tachycardia, the need to use rescue hypotensive agent, and recovery time.

Reflex tachycardia was defined as a heart rate (HR), and more than 15% of baseline (before induction of anesthesia) for 10 min with urine output > 1 ml/kg/h (to eliminate the possibility of tachycardia secondary to hypovolemia) was recorded and treated by increasing depth of anesthesia.

Infusion of the study drugs and fentanyl was stopped 5 min before the anticipated end of surgery, and sevoflurane was stopped after skin closure. At the end of surgery, any residual neuromuscular blockade was reversed with neostigmine and atropine to achieve a train of four ratio of at least 0.9 (T of guard). Extubation time and time to total recovery from anesthesia were recorded (Modified Aldrete's score $\geqslant 9$ on a scale of 0–10). After complete recovery from anesthesia, patients were transferred to the recovery room.

The two groups were compared with reference to patient's characteristics, intraoperative clinical data (operative time, urine output, crystalloid infusion, estimated blood loss, spinal levels fused, surgeon satisfaction, occurrence of reflex tachycardia, time to restoration of MAP, intraoperative fentanyl consumption), requirements for additional hypotensive agent (Nitroglycerine), and postoperative nausea and vomiting were recorded.

Blood samples were drawn for measuring HB%, Hct value in the preoperative day and first postoperative day, and blood samples were taken also for measuring blood urea nitrogen (BUN), serum creatinine, SGOT, and SGPT. Arterial blood gas analysis was used to determine changes in pH, partial pressure of CO_2 ($PaCO_2$), and bicarbonate (HCO_3^-).

2.3. Statistical analysis

Data were analyzed using SPSS statistical package version 15. Results were presented as mean ± (SD), median (Range), number, and percentage.

Forty-four patients (22 in each group of the study) were calculated based on a preliminary analysis and a desired power of 80% to detect a difference between groups of 20% regarding the amount of blood loss in the surgical field with a significant level of 5%. Thus, we studied 24 patients in each group.

t-tests were used to compare the two groups with respect to age, weight, intraoperative clinical data (operative time, crystalloid administration, urine output, levels fused, time to restoration of MAP, and occurrence of reflex tachycardia), HB% and Hct values, arterial blood gas analysis, and kidney and liver functions. Mann–Whitney test was used to compare the two groups with respect to estimated blood loss, time to modified aldrete score >9(min) and intraoperative fentanyl consumption. MAP and HR were compared between the two groups using unpaired *t*-test and using paired *t*-test for comparison in each group. Sex, presence of hypotensive agent, hypotension, bradycardia, nausea, and vomiting were compared in the two groups using Fisher exact test. *P* value of less than 0.05 was considered significant.

3. Results

WE enrolled 50 patients undergoing posterior lumbar spine fusion. Two patients had been excluded because of hypertension. Forty-eight patients were divided equally to either the dexmedetomidine group ($n = 24$) or the nimodipine group ($n = 24$).

There was no statistically significant difference between the two groups with regard to age, sex, weight, or ASA physical status (Table 1).

There was no statistically significant difference between the two groups with regard to operative time, crystalloid administration, urine output, estimated blood loss, and occurrence of reflex tachycardia, while there was a statistically significant difference in the time to restoration of MAP between the two groups (Table 2).

There was no statistically significant difference between the two groups in MAP (Fig. 1) or HR (Fig. 2) at baseline, after induction, during the hypotensive period or at the end of surgery, but within each group, MAP and HR during the hypotensive period were significantly lower than at baseline after induction and at the end of the surgery in each group ($P < 0.005$).

Mean intraoperative fentanyl consumption was highly significant in the nimodipine group compared with the dexmedetomidine group ($P = 0.002$), while the mean time to extubation was significantly shorter in the nimodipine group than in the dexmedetomidine group ($P = 0.001$). Similarly, the time to total recovery from anesthesia (modified aldrete score $\geqslant 9$) was significantly shorter in the nimodipine group compared with the dexmedetomidine group ($P = 0.002$) (Table 3).

During this study, there was no need to use nitroglycerine as a rescue hypotensive agent.

There was no statistically significant difference between the two groups regarding HB%, Hct value, pH, $PaCO_2$, (HCO_3^-),

Table 1 Demographic characteristics in adults undergoing spine fusion surgery.

	Dexmedetomidine group (n = 24)	Nimodipine group (n = 24)	P value
Age (year): mean ± SD	37.8 (± 10.7)	35.9 (± 14.8)	0.117
Sex (number, %)			
Female	8 (33.3%)	7 (29.2%)	
Male	16 (66.7%)	17 (70.8%)	
Weight (kg): mean ± SD	67.1 ± 14.5	68.9 ± 18.1	0.899
ASA physical status:			
ASA I	22 (91.7%)	21 (87.5%)	
ASA II	2 (8.3%)	3 (12.5%)	

Table 2 Intraoperative clinical data: values are expressed as mean ± SD, $P \leqslant 0.05$.

	Dexmedetomidine group (n = 24)	Nimodipine group (n = 24)	P value
Operative time (h)	3.9 ± 0.8	4.1 ± 0.9	0.189
Urine output: (cc/kg/h)	2.1 ± 0.3	2.4 ± 0.5	0.275
Crystalloid (cc/kg/h)	10.7 ± 3.9	9.9 ± 2.7	0.398
Estimated blood loss (cc)	385 ± 65	415 ± 78	0.869
Levels fused	2.8 ± 0.4	2.6 ± 0.3	0.754
Time to restoration of MAP (min.)	11.3 ± 3.2	26.8 ± 4.1	0.05
Occurrence of reflex tachycardia	2/24	3/24	

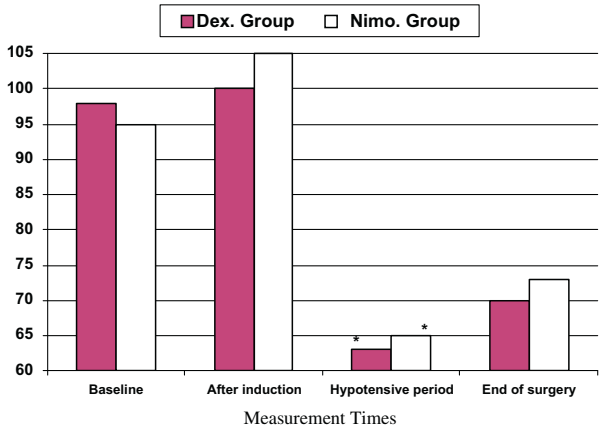

Figure 1 Perioperative mean arterial pressure (MAP) in both data are mean ± SD. $^{*}P < 0.05$ relative to the baseline value.

serum creatinine, blood urea nitrogen (BUN), SGOT, and SGPT at preoperative and postoperative periods (Table 4).

4. Discussion

Extensive blood loss during spine surgery leads to greater transfusion needs and serious consequences of the patient's hemodynamic equilibrium and can cause severe neurological damage because of the vicinity of major and highly fragile neurological structures. In terms of the surgeon, it is important to

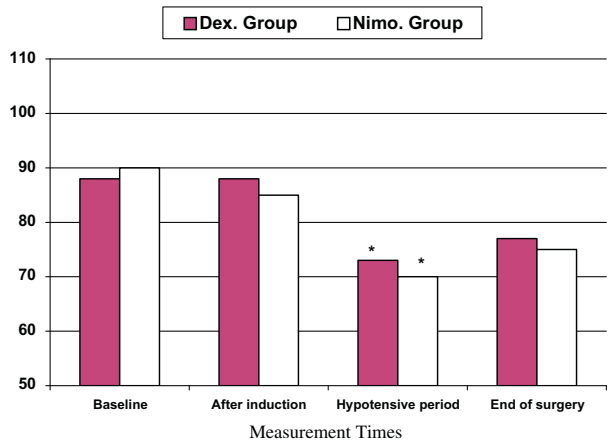

Figure 2 Perioperative heart rate (HR) in both groups: data are mean ± SD. $^{*}P < 0.05$ relative to the baseline value.

Table 3 Hemoglobin and hematocrit values in both groups; preoperative day and 1st postoperative day: values are mean ± SD.

	Normal value	Dexmedetomidine group (n = 24)	Nimodipine group (n = 24)	P value
Hemoglobin (gm%)				
Preoperative day		13.4 ± 1.6	13.1 ± 1.2	0.563
1st Postoperative day		13.1 ± 1.4	12.8 ± 1.9	0.657
Hematocrite (%)				
Preoperative day		0.39 ± 0.04	0.40 ± 0.04	0.756
1st Postoperative day		0.35 ± 0.05	0.35 ± 0.05	0.683

reduce bleeding to allow a better view of the surgical field, thereby increasing the surgeon's control and shortening surgical time which further decreases bleeding (see Table 5).

Controlled hypotension is the most commonly used technique for reducing blood loss in various types of surgeries, including spine surgery [10], but it may be associated with increased risk of neurological deficit due to reduced spinal cord perfusion [11].

The key equation in the provision of deliberate hypotension is MAP = C.O.P.X S.V.R. So, MAP can be reduced by either decreasing S.V.R. or C.O.P. or both. Inducing hypotension only by a reduction in C.O.P. is not feasible because the maintenance of tissue blood flow is mandatory. S.V.R. can be reduced by peripheral V.D. of the resistance vessels, while C.O.P. can be reduced by decreasing V.R., H.R., myocardial contractility or a combination of these.

In our study, dexmedetomidine or nimodipine, when used in combination with sevoflurane, provided a comparable optimal surgical field. Both drugs were effective in reaching MAP of 60–65 mmHg and ensure good surgical field. This is the first study to compare the effects of dexmedetomidine or nimodipine combined with sevoflurane in providing controlled hypotension during spine fusion surgery.

Both drugs demonstrated similar hemodynamic effects during spine fusion surgery. A stable MAP was easily achieved in both groups using the protocol described. However, the time

Table 4 Fentanyl requirements and recovery characteristics values are expressed as mean ± SD.

	Dexmedetomidine group (n = 24)	Nimodipine group (n = 24)	P value
Intraoperative fentanyl consumption (μg)	200.0 ± 55	350 ± 78*	0.002
Time to extubation (min)	8.9 ± 1.8	6.8 ± 1.3*	0.001
Time to modified aldrete score ⩾9 (min)	7.6 ± 2.1	5.7 ± 1.9*	0.001

Table 5 Arterial blood gas analysis kidney and liver function tests in both groups; preoperative and postoperative: values are mean ± SD.

	Normal value	Dexmedetomidine group (n = 24)	Nimodipine group (n = 24)	P value
pH:	7.35–7.45			
Pre		7.4 (0.1)	7.4 (0.1)	0.747
Post		7.4 (0.1)	7.4 (0.1)	0.747
$PaCO_2$ (mmHg)	35.0–45.0			
Pre		38.7 (5.9)	34.4 (5.9)	0.136
Post		30.3 (5.7)	31.2 (5.8)	0.96
HCO_3 (mmol/L)	22–26			
Pre		22.6 (3.8)	22.6 (3.8)	0.673
Post		22.6 (2.8)	22.6 (2.8)	0.673
S. creatinine (mg/dl)	0.8–1.2			
Pre		0.8 (0.2)	0.8 (0.2)	0.253
Post		0.8 (0.2)	0.8 (0.3)	0.567
BUN (mg/dl)	8–20			
Pre		12.4 (4.1)	10.4 (3.4)	0.256
Post		12.6 (4.8)	11.0 (3.1)	0.383
SGOT	Max 45 IU/L			
Pre		19.6 (5.8)	21.7 (9.3)	0.665
Post		19.2 (5.3)	20.9 (7.4)	0.363
SGPT	Max 45 IU/L			
Pre		20.8 (12.8)	20.3 (8.9)	0.817
Post		19.7 (13.2)	19.8 (10.8)	0.992

to restoration of baseline blood pressure was significantly different between the two groups, with the nimodipine group requiring approximately 15 min longer to return to the baseline blood pressure than the dexmedetomidine group. The difference in the pharmacokinetics of the two drugs may explain the difference in time courses of the two drugs. Dexmedetomidine is a highly selective alpha-2 (α 2) agonist with a biphasic half-life time ($t1/2$) with a distribution ($t1/2$) of 6 min and terminal elimination ($t1/2$) of 2 h [12]. On the other hand, Nimodipine is a calcium channel blocker which interferes with the calcium dependent regulatory mechanism of vascular smooth muscle tone. Removal of nimodipine would not result in restoration of baseline blood pressure until the drug diffused off the receptor site. The terminal elimination half-life time ($t1/2$) of nimodipine is 3 h [13]. However, gradual return of blood pressure may be helpful by preventing excess blood loss in the immediate postoperative period.

In accordance to our research, Hynynen et al. [13], and Zhou et al. [14], reported that intraoperative infusion of nimodipine and dexmedetomidine, respectively, maintained hemodynamic stability and was well tolerated.

Regarding the intraoperative clinical data of our study, the results obtained suggest that the intentional moderate hypotensive general anesthesia is safe and enables good control of blood loss in young healthy adult patients undergoing elective spine surgery. All patients should be carefully assessed before surgery for any contra-indications to hypotensive anesthesia, and the technique has the advantage to lesser major blood loss, produce a drier surgical field, and decrease operating time.

The precise mechanism by which controlled hypotension decreases blood loss is still obscure. Some studies have postulated that hypotensive anesthesia (leads to an ischemic field) (i.e., ↓local blood flow) which then causes less blood loss. But, other studies have measured local blood flow through scientific measures such as flowmetry [15].

Lee et al. [16] measured blood flow in paraspinal muscles during spine surgery with two hypotensive drugs, reaching a similar degree of hypotension. They found widely differing values for local blood flow, although blood loss did not differ. This result indicates that blood loss is influenced by many factors other than local blood flow such as systemic blood pressure [17] and blood flow in the epidural plexus [18].

In the field of spinal fusion surgery, some studies report that bleeding is essentially venous [1], so blood loss will be influenced by factors other than a decrease in art pressure. Factors other than blood pressure postulated to affect intraoperative blood loss include intra-abdominal pressure (related to prone positioning on the Relton-Hall frame which allow the abdominal viscera to hang freely, reducing the inferior vena caval pressure), the number of spinal segments operated on, body weight, pathological nature of the disease necessitating surgery, and surgeon's experiences [1].

In our study, all patients were operated due to traumatic fractures of the spine on 2 to spinal segments and by the same surgical team. There was no significant difference in weight between the two groups, and all patients were positioned in the same way and by the same term. So, the effects of the above mentioned factors have been obtunded.

The estimated blood loss (EBL) in both groups was much less than 500 ml. So, these values together with the postoperative HB and Hct values ensured minimal blood loss which did not necessitate intraoperatively packed RBCs transfusion.

It is not desirable to replace all the blood lost during the surgery because an adult patient with normal cardiopulmonary function can compensate for up to 20% loss of circulating blood volume.

The optimal hematocrit value is determined by the oxygen carrying capacity of the blood and its fluidity in the absence of hypoxemia. According to Messmer [19], normovolemic patients who have normal compensatory mechanisms should be able to tolerate values as low as 0.27–0.3%. This study showed that the overall hematocrit value dropped to 0.35% which is higher than the established tolerable range.

Many studies show a significant reduction in the need for homologous transfusion when moderate hypotensive anesthesia is used [20,21].

In our study, fentanyl consumption was significantly lower in the dexmedetomidine group compared with the nimodipine group ($P = 0.002$). Several studies have demonstrated that perioperative use of dexmedetomidine was associated with a significant reduction in the consumption of fentanyl in a dose-dependent manner [22,23]. This could be explained by the sedative and analgesic sparing effects of dexmedetomidine through central actions in the locus coeruleus and in the dorsal horn of spinal cord [24,25]. However, dexmedetomidine was associated with significantly longer times to extubation ($p = 0.001$) and to total recovery from anesthesia ($p = 0.001$). Similar to our study, Iclal et al. reported that extubation time was significantly longer in patients receiving dexmedetomidine compared with those receiving esmolol for controlled hypotension during tympanoplasty [26]. In contradiction to our study, Zhou et al. reported that the use of dexmedetomidine in aged patients with total hip replacement surgery under general anesthesia resulted in reduction in the extubation time and waking time after surgery.

Our study showed normal blood gas analysis, liver, and kidney functions, probably because MAP was maintained at approximately 60 mm. Hg in all patients. Also, the regimen prescribed in both groups for inducing hypotension (whether dexmedetomidine ornimodipine combined with sevoflurane and fentanyl on request) avoided the use of a single hypotensive agent with its subsequent dose-related side effects. Degoute [3] reported that adjuvant treatment for deliberate hypotension is an ideal technique which can reduce adverse effects of the individual hypotensive agents by reducing their total doses.

5. Limitations

Our study though has number of limitations. To begin with, there was no untreated control group, as all patients have to receive hypotensive agent. Second, we investigated single dose of hypotensive agent. Third, the difficulty of investigating subtle clinical factors, for instance, the proficiency of anesthesiologists or surgeons might mask the nature of a procedure that would be associated with more bleeding in less skilled hands. Finally, larger prospective controlled studies are necessary to ensure definite conclusions about the comparable effects of both drugs.

6. Conclusion

Both dexmedetomidine and nimodipine, combined with sevoflurane, provided an effective method for achieving a bloodless surgical field with controlled hypotension in these patients undergoing posterior lumbar spine fusion surgery. Nimodipine was associated with a comparable intraoperative clinical and recovery times compared with dexmedetomidine.

References

[1] Raw DA, Beattie JK, Hunter. Anesthesia for spinal surgery in adults. Br J Anaesth 2003;91(6):886–904.
[2] Palski SZ, Gunzburg R, Sztern B. An overview of blood-sparing techniques used in spine surgery during the perioperative period. Eur Spine J 2004;13(Suppl. 1:S):18–27.
[3] Degoute CS. Controlled hypotension: a guide to drug choice. Drugs 2007;67:1053–76.
[4] Tobias JD. Sevoflurane for controlled hypotension during spinal surgery: preliminary experience in five adolescents. Paediatr Anaesth 1998;8:167–70.
[5] Elsharnouby NM, Elsharnouby MM. Magnesium sulphate as a technique of hypotensive anesthesia. Br J Anaesth 2006;96:727–31.
[6] Maze M, Scarfini C, Cavaliere F. New agents for sedation in the intensive care unit. Crit Care Clin 2001;17:881–97.
[7] El-Gohary MM, Arafa AS. Dexmedetomidine as a hypotensive agent. Efficacy and hemodynamic response during spinal surgery for idiopathic scoliosis in adolescents. Egypt J Anaesth 2010;26:305–11.
[8] Lawrence CJ, Prinzen FW, de Lange S. Effect of dexmedetomidine on nutrient organ blood flow. Anesth Analg 1996;83:1160–5.
[9] Blardi P, Uros R, Volpi L, et al. Nimodipine: drug pharmacokinetics and plasma adenosine levels in patients affected by cerebral ischaemia. Clin Pharmacol Ther 2002;72:556–61.
[10] Park SH, Do SH, Kim CS, et al. Controlling deliberate hypotension in hypertensive patients undergoing spinal surgery: a comparison between remifentanil and sodium nitroprusside. Anesth Pain Med 2010;5(1):38–44.
[11] Krengel 3rd WF, Robinson LR, Schneider VA. Combined effects of compression and hypotension on nerve root function. A clinical case. Spine 1993;18:306–9.
[12] Dexmedetomidine (Precedex) Package Insert. Hospira, Inc. 2010.
[13] Hynynen M, Siltanen T, Sahlamn A, et al. Continuous infusion of nimodipine during coronary artery surgery haemodynamic and pharmacokinetic study. Br J Anaesth 1995;74:526–33.
[14] Zhou J, Wang J, Keng F. The use of dexmedetomidine in aged patients with total hip replacement surgery under general anesthesia: changes in hemodynamics, cerebral state index and wakening quality. African Journal of Pharmacy and Pharmacology 2012;6(25):1833–6.
[15] Szpalski M, Gunzberg R, Sztern B. An overview of blood-sparing techniques used in spine surgery during the perioperative period. Eur Spine J 2004;13(Suppl. 1:S):18–27.
[16] Lee TC, Buerkle H, Wang CJ, et al. Effect of isoflurane versus nicardipine on blood flow of lumbar paraspinal muscles during controlled hypotension for spinal surgery. Spine (Phila Pa 1976) 2001;26(1):105–9 [discussion 9].
[17] Sivarajan M, Amory DW, Everett GB, Buffington C. Blood pressure, not cardiac output, determines blood loss during induced hypotension. Anesth Analg 1980;59(3):203–6.
[18] Ullrich Jr PF, Keene JS, Hogan KJ, Roecker EB. Results of hypotensive anesthesia in operative treatment of thoracolumbar fractures. J Spinal Disorder 1990;3(4):329–33.
[19] Messmer KF. Acceptable haematocrit levels in surgical patients. World J Surg 1987;11:41–6.
[20] Fordyce AM, Telfer MR, Stassen LF. Cross-matched blood for major head and neck surgery: an analysis of requirements. Br J Oral Maxillofac Surg 1998;36:103–6.
[21] Anvari Zahra Taghipour, Afshar Fereydouniyan Nader, Imani Farnad, et al. Effect of clonidine premedication on blood lol.ss in spine surgery. Anesthesiol Pain Med 2012;1(4):252–6.
[22] Getter R, Brown HC, Mitchell DH, Silvius EN. Dexmedetomidine: a novel sedative-analgesic agent. BAYLOR Univ Med Center Proc 2001;14(1):13–21.
[23] Ayoqlu H, Yapakci O, Uqur MB, et al. Effectiveness of dexmedetomidine in reducing bleeding during septoplasty and tympanoplasty operations. 2008;20(6):437–41.
[24] Arpino PA, Kalafatas K, Thompson BT. Feasibility of dexmedetomidine in facilitating extubation in the intensive care unit. J Clin Pharm Ther 2008;33(1):25–30.

[25] Hoy SM, Keating GM. Dexmedetomidine: a review of its use for sedation in mechanically ventilated patients in an intensive care setting and for procedural sedation. Drugs 2011;71(11): 1481–501.

[26] Kol Iclal Ozdemir, Kaygusuz Kenan, Yildirim Altan, et al. Controlled hypotension with desflurane combined with esmolol or dexmedetomidine during tympanoplasty in adults. Curr Therapeutic Res 2009;70:197–207.