Egyptian Journal of Anaesthesia 33 (2017) 183–188

HOSTED BY

Contents lists available at ScienceDirect

Egyptian Journal of Anaesthesia

journal homepage: www.sciencedirect.com

Research article

Evaluation of dexmedetomidine as a sole agent in sedation of cancer patients undergoing radiological interventional procedures

Ayat Abdelfattah Hassan [a,*], Wafaa Taha Salem [a], Maie Kamaleldein Helaly [a], Ghada M. Bashandy [a], Reda Tabashy [b], May Abdelfattah [c]

[a] Department of Anesthesia and Pain Relief, National Cancer Institute, Cairo University, Egypt
[b] Department of Radiology National Cancer Institute, Cairo University, Egypt
[c] Department of Clinical Pathology, Faculty of Medicine, Cairo University, Egypt

ARTICLE INFO

Article history:
Received 27 June 2016
Revised 9 December 2016
Accepted 20 December 2016
Available online 13 January 2017

Keywords:
Dexmedetomidine
Sedation
Intervention
Radiological
Sole

ABSTRACT

Background: Previous studies have shown that dexmedetomidine has proven effectiveness as sedative in many outpatient settings and several reports are now available documenting its success for sedation of both non-invasive and invasive procedures.
Objective/purpose: This investigation aimed at evaluation of dexmedetomidine efficacy when used alone for sedation of patients undergoing radiological interventional procedures and measuring its different outcome variables.
Methods: A total of sixty patients who underwent interventional procedures requiring sedation in radiology department were enrolled. Only ages from 18 to 65 years and ASA physical status of I–II were allowed into the study. A loading infusion of one μg/kg over 10 min was started to be followed by a maintenance infusion of 0.2–1 (0.6) μg/kg/h. HR, blood pressure and Spo2 were continuously monitored while pain and sedation were assessed every 10 min by using visual analogue scale (VAS) and Ramsay sedation score (RSS) respectively. cortisol and blood glucose levels were measured pre and post interventionally in addition to the recording of the previously mentioned hemodynamics.
Results: Compared to the pre-sedation values, we observed an acceptable reduction; 11% for blood pressure and 10% for heart rate. Fentanyl was required as a rescue analgesia in 61% of patients enrolled in the study the levels of cortisol and blood glucose in the post intervention period showed statistically significant increase in the post-intervention samples as compared with pre-intervention ones ($P < 0.001$)
Conclusion: Dexmedetomidine can be used alone for sedation of interventional procedures when minimal to mild pain is in prospect thus provides an alternative for anesthesiologists for high risk patients but cannot be used alone when intense pain is anticipated.

1. Introduction

Conscious sedation is the gold standard for a wide range of outpatient interventions like endoscopy, dental procedures and interventional radiological procedures. The combination of an opiate and a benzodiazepine is known to provide excellent analgesia and sedation during such procedures [1]. However, they carry the risk of respiratory depression among other adverse effects. Local anesthesia alone has been advocated for many interventional radiological procedures in order to avoid risks as well as cost and of conscious sedation. But this approach is likely to be less acceptable to patients and reduce their willingness to undergo repeated procedures e.g. chemoembolization. On the other hand moderate sedation is a logical way to avoid hypoxia in the more susceptible patients.

The research for an ideal sedative is being carried out constantly [2]. Dexmedetomidine is a potent and highly selective α-2 adrenoceptor agonist with sympatholytic, sedative, amnestic and analgesic properties. It has continuously expanding uses, as the FDA approved its use as a sedative in non-intubated patients in late 2008, it received a special consideration to be used in many outpatient settings [3]. It also has the privilege of having a minimal depressant effect on the respiratory system.

Peer review under responsibility of Egyptian Society of Anesthesiologists.
* Corresponding author.
E-mail addresses: dr_ayat_fattah@yahoo.com (A.A. Hassan), Taha1212@hotmail.com (W.T. Salem), Maiehelaly@aol.com (M.K. Helaly), Ghada_pashandy@yahoo.com (G.M. Bashandy), reda.tabashy@Yahoo.com (R. Tabashy), dr_maya_fattah@cu.edu.eg (M. Abdelfattah).

http://dx.doi.org/10.1016/j.egja.2016.12.001
1110-1849/

The objective of this study was evaluating dexmedetomidine when used as a sole IV drug for sedation during interventional radiological procedures for cancer patients, and eventually gaining insight about the feasibility and efficacy of dexmedetomidine use in interventional radiology settings. Primary outcome was measuring dexmedetomidine different outcome variables in terms of Ramsy sedation score, VAS score, blood pressure and pulse. Secondary outcome was the determination of dexmedetomidine effect on serum cortisol as a stress hormone and subsequently blood glucose.

2. Patients and methods

The current study enrolled sixty patients scheduled for interventional procedures requiring sedation in radiology department of National Cancer Institute. This clinical trial is registered in clinical trials website as (NCT02180737). Interventions included nephrostomy, radiofrequency ablation, arterial and venous chemoembolization under radiographic imaging. Only ages from 18 to 65 years and ASA physical status of I–II were allowed into the study. Patients suffering from bradycardia or heart block were not included in the study. Patient who received an alpha2-agonist or antagonist within 14 days, IV opioid within 1 h, or an oral or IM opioid within 4 h of the start of study drug administration were excluded. Patients who are allergic to dexmedetomidine were also excluded.

In the pre-procedural holding area; patients were instructed in how to report their pain using VAS pain score, where 0, "no pain" and 100, "Worst pain imaginable". We asked the patients to describe their pain with standardized adjectives that corresponded to numerical scores as follows: 0, none; 10–20, mild; 30–40, discomforting; 50–60, distressing; 70–80, horrible; and 90–10, excruciating Fentanyl was given incrementally by a dose of (0.5–2 μg/kg) as a rescue analgesia when VAS score exceeded 40 mm.

After recording a baseline measurements of heart rate (HR), mean arterial blood pressure (MAP) and Spo2, a peripheral IV access was obtained. We inserted a 18- or 20-gauge intravenous catheter to facilitate fluid and drug administration. Dexmedetomidine diluted in 0.9% saline (4 μg/ml) was prepared in a syringe infusion pump. A loading infusion of one μg/kg over 10 min was started to be followed by a maintenance infusion of 0.2–1 (0.6) μg/kg/h to be given through a separate intravenous line. The rates were adjusted to reach Ramsay Sedation Score (RSS) [4] of 3–4. The RSS 1–6 were recorded at 10 min intervals for conscious sedation [4]. We have chosen the RSS score of 3 as our target level of sedation as it meets the conditions of conscious sedation, that is a minimally decreased level of consciousness, preserving the patients' ability to maintain their airway and to respond appropriately to verbal command.

Venous blood samples were collected before starting dexmedetomidine and immediately after the procedure for serum cortisol and blood glucose measurements and was conducted as follows:

2.1. Sample collection

Blood samples were collected from all patients on clot activator vacutainer tubes for serum cortisol assessment and sodium fluoride containing vacutainer tubes for plasma glucose assessment, before and after procedure. Specimens were centrifuged immediately for serum and plasma separation.

2.2. Methods for assessment

The quantitative measurement of cortisol was done using the IMMULITE® 2000 system analyzer which is a solid-phase competitive chemiluminescent enzyme immunoassay, and enzymatic UV test (hexokinase method) for the quantitative determination of plasma glucose level was done using the Beckman Coulter AU 680 analyzers.

Patients were transferred to the procedure room fully monitored. Patients received oxygen through nasal cannula 4 L/min, followed by a local skin anesthetic of 5–15 mL of 2% lidocaine with a 22 Gauge needle to decrease the initial patient discomfort and to eliminate pain associated with needle placement by the interventionist.

HR, MAP, SaO_2, drugs administered, RSS and time to achieve desired level of Sedation, VAS pain scores were recorded in a flow chart against time. Also adverse events including air way obstruction, apnea, bradycardia, failed sedation and total time of sedation were treated and documented.

Hypotension was defined as systolic blood pressure below 90 mmHg which was managed by IV fluid administration of 10 ml/kg initially and/or ephedrine (0.25–1 mg/kg). Bradycardia was defined as pulse rate below 55 bpm where atropine was given in a dose of 0.01 mg/kg. Desaturation was defined as SpO_2 below 90%. If the oxygen saturation decreased to between 90% and 95%, the patient was asked to take deep breaths if responding to commands while chin left and jaw thrust were applied in case of deep sedation. If the saturation decreased to 90% or less, supplemental oxygen was administered at a rate of 6 L/min via oxygen mask instead of supplying it through nasal cannula. Dexmedetomidine infusion was stopped once the intervention was terminated. Patients were discharged in two stages: first to the recovery room were again HR, MAP and Spo2 recorded and finally home discharge, the discharge criteria required that the patients be awake and alert with stable vital signs, able to ambulate without assistance, pain scores as pre-procedure level, and free of side effects.

3. Statistical methods

Data management and statistical analysis were performed using the Statistical Package for Social Sciences (SPSS) vs. 21. Numerical data were summarized using means and standard deviations or medians and ranges. Categorical data were summarized as numbers and percentages.

Comparisons pre and post procedure were done by paired *t* test while comparisons overtime intraoperative were done by repeated measure analysis of variance.

All p-values are two-sided. P-values < 0.05 were considered significant.

4. Results

4.1. Demographic data

48 males (80%) and 12 females (20%) with age range of 24–68 years old were included in this study. Patients underwent 63 procedures Table 1, least duration of the procedure was 20 min and maximal was 60 min.

4.2. Clinical parameters

Compared to the pre-sedation values, both mean systolic as well as diastolic blood pressures showed a clear and statistically significant post-procedural decline by 11% and 13% respectively ($P < 0.001$), Table 2. The 95% confidence interval for the difference in mean systolic blood pressure was −23.8 to −14 and that for mean diastolic blood pressure was −13.5 to −7.5. Blood pressure decreased steadily on repeated measurements over time during the intraoperative period ($P < 0.001$), Table 3. Intra procedural

Table 1
Patient characteristics and types of procedures.

Age (yrs.) (Mean ± SD)		53.4 ± 10.4	
Gender (No., %)	Female	12	20%
	Male	48	80%
Procedure (No., %)	Cementoplasty	2	3.3%
	Chemoembolization	6	10%
	Gastrostomy	2	3.3%
	Nephrostomy	4	6.7%
	PTD	3	5%
	Radio frequency	33	55%
	Splenic artery embolization	1	1.7%
	Ureteric stenting	1	1.7%
	Ureteric dilatation + double J	2	3.3%
	Ureteric stent + nephrostomy	1	1.7%
	Uterine fibroid embolization	3	5%
	Radiofrequency of bone	2	3.3%

mean systolic blood pressure was 125 ± 19 mmHg while mean diastolic was 74 ± 11 mmHg.

Pre-procedure mean heart rate was 80 ± 17 bpm while the post-procedure was 71 ± 13 bpm ($P < 0.001$), Table 2. The mean intra procedural pulse was decreased significantly from baseline ($P < 0.001$). Although there was a gradual and steady decrease in the mean pulse on 10, 20, 30, 40, 50 and 60 min Table 4, this decline was not statistically significant (p = 0.9). Average decrease in the mean pulse rate was 10% from the pre-sedation values.

Mean intraoperative SpO_2 was 94% ± 12.7. There was no statistically significant difference in the mean SpO_2 as measured throughout the procedure, ($P = 0.814$). Clinically significant respiratory depression was not observed in any of our patients only 3 patients required jaw thrust (5%).

At the 6 study points after starting dexmedetomidine infusion, RSS was observed to be less than 3 (where patients needed increasing rate of infusion) in 83.3%, 34%, 17.5%, 31.6%, 25%, 20% of patients at 10, 20, 30, 40, 50 and 60 min. respectively. On the other hand, 16.7%, 66%, 82.5%, 68.4%, 75%, 80% patient at the same time points didn't need any increase in dexmedetomidine infusion as their RSSs were 3 or 4, Fig. 1.

Based on the above mentioned observations; the group of patients whose RSS (3–4) were considered adequately sedated patients, and included the following procedures (chemoembolization, percutaneous nephrostomy, radiofrequency of liver metastases for centrally located lesions, ureteric stenting, splenic artery embolization, uterine artery embolization, PTD and gastrostomy) while the patients with a score of less than three were inadequately sedated and included (RFA of bone, Cementoplasty and radiofrequency of hepatic metastatic lesions abutting the diaphragm.

Table 4
Intra procedural pulse assessment (beats/min).

	Mean	SD
Pulse10Intra	75	16
Pulse20Intra	71	14
Pulse30Intra	70	12
Pulse40Intra	70	12
Pulse50Intra	67	7
Pulse60Intra	66	3
P value	0.971	

Values are mean ± standard deviations (SD). Pulse 10: after 10 min of starting dexmedetomidine infusion.

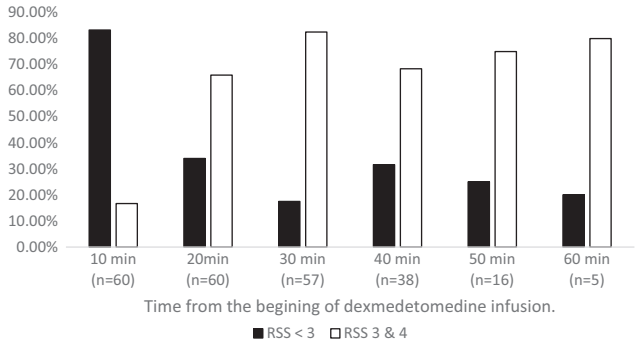

Fig. 1. RSS during dexmedetomedine infusion.

At 10, 20 min after starting dexmedetomidine infusion. VAS score more than four was obtained in 20% of patients (a total of 60 patients). While the remaining 80% had a score of less than four. At 30 min a VAS score more than 4 was obtained in (17.5%) of patients while a score of less than 4 was encountered by 82.5% (Total number of patients was 57).

At 40 min a VAS score more than 4 was obtained in (26%) of patients while a score of less than 4 was in obtained in (74%) (total number 38). At 50 min a score above 4 was obtained in (12.5%) while a score less than 4 was obtained in (87.5%) of patients (total number 16). At 60 min no patient experienced a VAS score more than 4 (total number was 5).

Table 2
Comparison between pre-sedation and post-sedation hemodynamic values.

	Pre-procedure		Post-procedure		Mean	95% CI of the difference		
	Mean	SD	Mean	SD	Difference	Lower	Upper	*P* value
Systolic (mmHg)	141	22	122	19	−19.0	−23.8	−14.3	**<0.001**
Diastolic (mmHg)	83	11	73	13	−10.4	−13.2	−7.5	**<0.001**
Pulse rate (beats/min)	79.4	17	71	11	−8.5	−12.9	−4.0	**<0.001**

SD: standard deviation, P < 0.05 is significant.

Table 3
Intraoperative blood pressure assessment.

	Intra 10		Intra20		Intra30		Intra 40		
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	
Systolic BP (mmHg)	133.6	23.3	123.8	21.4	118.4,	20.7	119.5	22.7	<0.001
Diastolic BP (mmHg)	77.2	13.1	73.1	12.6	73.2	11.4	69	13.3	0.016
Mean BP (mmHg)	94	16	87	15	85	15	53	17	<0.001

SD: standard deviation, P < 0.05 is significant, all groups are significant from each other's (Intra 10: after 10 min of starting dexmedetomidine infusion).

Table 5
Comparison between pre-sedation and post-sedation chemical values.

	Pre-procedure		Post-procedure		Mean	95% CI of the difference		
	Mean	SD	Mean	SD	Difference	Lower	Upper	*P* value
Cortisol (μg/dl)	14	12	26.5	18.4	12.7	8.3	17.0	<**0.001**
Glucose (mg/dl)	104	31	136	42	31.9	22.0	41.8	<**0.001**

Supplemental fentanyl was required in 61% of all patients to achieve a satisfactory level of analgesia with a mean dose of 1.2 μg/kg ranging from 0.4 μg/kg to 2.2 μg/kg.

No patients experienced major hemodynamic instability in the intra or post procedure period. No evidence of rebound hypertension, tachycardia or acute reversal of sedative and analgesic effects after drug discontinuation. There was no need for prolonged post-procedure monitoring, unplanned admission or subsequent medical attention.

4.3. Laboratory findings

As regard the laboratory data; mean serum glucose levels showed statistically significant increase in the post-intervention samples as compared with pre-intervention ones ($P < 0.001$). Also the mean serum cortisol levels showed statistically significant increase in the post-operative values ($P < 0.001$). The average increase in the post intervention blood cortisol was 86% from pre sedation values while average increase in blood glucose was 25% as shown in (Table 5).

Out of the 60 patients enrolled in our study 6 patients underwent chemoembolization; aged 59 ± 7 years old, mean cortisol preoperatively was 28 μg/dl showing a clear and significant rise postoperatively 62 μg/dl, same occurred with blood glucose; mean preoperative blood glucose was 79.5 mg/dl while postoperatively was 163.5 mg/dl and median VAS was 0.

5. Discussion

The primary aim of this study was to evaluate the hypothesis that dexmedetomidine can be used alone in the sedation for interventional radiological procedures. In this setting heterogeneous outcomes in terms of sedation and analgesia were obtained; while it was satisfactory in certain procedures it was not as competent in others. Compared to the pre-sedation values, a slight reduction of blood pressure and heart rate was observed, after the loading dose of dexmedetomidine that continued throughout the intervention time. Our data affirm that a dosage adequate to create sedation was not adequate to deliver adequate analgesia. The current observational study represents the first prospective evaluation of dexmedetomidine for sedation in this setting and adds to the limited data regarding its use in invasive procedures.

The interventional procedures that were studied in our trial were notably diverse, and it is possible that dexmedetomidine may prove more suitable for some procedures than others, thus providing an alternative choice for anesthesiologists which has no deleterious clinical effects on respiration, especially for procedures which was routinely performed under general anesthesia (e.g. hepatic RFA for centrally located lesions). Dexmedetomidine gave proof for its efficacy in both sedation and analgesia, similar success was reported by Bavullu et al. [5] who concluded that dexmedetomidine provided better sedation than midazolam in sedation for percutaneous drainage of hepatic hydatid cysts under local anesthesia but in fact their study differed from ours as propofol was given to all their patients in sub hypnotic doses (0.5 mg/kg).

Only few other studies used dexmedetomedine for conscious sedation. Kaygusuz et al. evaluated the utility of dexmedetomidine when compared with propofol during extracorporeal shockwave lithotripsy (ESWL) procedures in spontaneously breathing patients. The combination of dexmedetomidine with small dose of fentanyl was used safely and effectively for sedation and analgesia during ESWL [6].

Dexmedetomidine use was reported to cause hypotension in 30–40% and bradycardia in 9% of cases in previous studies evaluating its effects on hemodynamic parameters [7,8]. Unlike our results Jalowiecki et al. [9] reported average 26% decrease in blood pressure and mean HR was 17% lower than pre sedation values though he used a lower dose of dexmedetomidine (0.2 μg/kg) for maintenance infusion. Interestingly, Ickeringill et al. [10], mentioned that a dexmedetomidine infusion has a predictable cardiovascular effect such that within an hour of commencing an infusion of 1.0 μg/kg/h, a 10% drop in systolic blood pressure and a 10–15% drop in heart rate are expected, he also mentioned that these effects are exaggerated in patients who receive a loading dose, or who are hypovolemic.

Jalowiecki et al. [9] evaluated the ability of dexmedetomidine to provide analgesia and sedation for outpatient colonoscopy, they suggested that the use of dexmedetomidine to provide analgesia/sedation for colonoscopy is limited by its distressing hemodynamic instability. In contrast, our study differed in that pronounced, hemodynamic instability rarely occurred. However, the association of dexmedetomidine usage with such invasive painful procedures may explain the rarity of occurrence of hemodynamic instability and also the patients were not deeply sedated to maintain the arousability which may left a blood level of catecholamines that avoided excessive sympathetic blockage.

In our study the post procedure time passed unremarkably; no patient experienced rebound hypertension or tachycardia after drug discontinuation in concordance with the results by Wu et al. [11] who compared dexmedetomidine versus midazolam for sedation in upper gastrointestinal endoscopy.

Although the patients appeared to have been moderately sedated, they suddenly indicated pain; most of the pain encountered by patients occurred mainly during track dilatation or when the interventionist is exceeding the area covered by local anesthesia or even upon facing technical difficulty this finding was supported by Ohata et al. [12] and Ramsay and Luterman [13], who reported the success of the use of dexmedetomidine in airway surgeries and procedures but stressed on the importance of usage of adequate topical anesthesia.

Our observations in terms of VAS score, percentage of patients who required fentanyl and the amount required suggests the limited analgesic properties offered by dexmedetomidine when used in invasive procedures, similar finding was confirmed by Hammer et al. [14] who concluded that the lack of a significant effect of dexmedetomidine infusion of (1 μg/kg/h) on the EC50 of propofol in children undergoing esophagogastroduodenoscopy and Mason and Lerman [15] considered dexmedetomidine neither a complete anesthetic nor a complete analgesic.

Other studies which investigated the "opioid sparing effect" confirmed the decrease in morphine consumption as Arain et al. [16] who concluded in his study that administration of

dexmedetomidine significantly reduced, by 66%, the early postoperative need for morphine.

Our study did not directly evaluate the amount of fentanyl that is minimized upon using dexmedetomidine as the fentanyl was given as we anticipated pain or subsequent to the painful episodes until adequate analgesia was obtained, beside the lack of control group for comparison; as this was not our primary objective.

As an imidazole compound, dexmedetomidine has the potential to have inhibitory effects on cortisol synthesis similar to etomidate. It has been shown in dogs that the cortisol response to adrenocorticotrophic hormone is blunted three hours after a dexmedetomidine bolus of 80 μg/kg Maze et al. [17]. The levels of cortisol and blood glucose increased post procedural (25% and 84% respectively) but were significantly higher in patients who underwent chemoembolization; the results presented in our study were supported by Venn et al. [18] who concluded that dexmedetomidine infusion dose not inhibit adrenal steroidogenesis for short term sedation after surgery.

In contrast with the present study, Yacout et al. and Uyar et al. found that plasma concentration of cortisol and glucose had increased significantly in the placebo group, than in the dexmedetomidine group, but actually Yacout also measured the serum cortisol level on the first postoperative day not just in the immediate postoperative samples [19,20].

In agreement with the present study, Aantaa et al. and Al-Medani et al. [21,22] who concluded that, there was no statistically significant difference in the mean cortisol and glucose values immediately postoperatively, Al-Medani et al. added in research that at 6 and 24 h postoperatively, the mean values of cortisol and glucose increased significantly in the group of patients receiving morphine relative to the group of patients receiving dexmedetiomidine. We might say that dexmedetomidine may have modified cortisol release but not completely inhibited steroidogenesis as part of an inevitable physiological response, putting into consideration the time of starting dexmedetomidine administration, we also did not measure the level of serum catecholamine and IL6 as a part of the whole process to fully judge the entire scene.

Venn et al. added in his research that dexmedetomidine inhibited cortisol synthesis at supra therapeutic concentrations and decreased the inflammatory response to surgical trauma [18].

Despite the favorable results obtained with the six patients whom underwent chemoembolization in terms of their hemodynamics, clinical parameters, sedation and pain control, there was an unexpected rise of post interventional blood cortisol level, this finding may be attributed to the nature of the chemotherapeutic agent itself or its effect on endocrine response as mentioned in previous literature which documented a 10% average increase in weekly-treated patients reaching 64% increase in daily-treated patients in the blood cortisol levels [23].

Target level of sedation was reached in our study in 66% of the patients in 20 min and in 82.5% after 30 min, inadequate sedation was mainly attributed to inadequate pain relief. Similarly, Song et al. [24] observed a significant rise of RSS at 20 min in all his study groups utilizing (0.25 μg, 0.5 μg, 0.75 μg) of dexmedetomidine. Koroglu et al. [25] have chosen RSS of 6 when they used dexmedetomidine for pediatric sedation in MRI unit, and concluded that this level of sedation was reached after 30 min in 80% of the patients in their study upon using 0.5–0.7 μg/kg/h for maintenance.

A significant superiority of dexmedetomidine over other commonly used sedative drugs is that it has a broad therapeutic index. Dexmedetomidine does not cause clinically significant respiratory depression even at doses 15-fold higher than the recommended doses [26]. In healthy subjects, increasing dexmedetomidine doses lead to linearly decreasing pain sensation [27]. It might be possible that level of analgesia increases upon increasing the dose beyond the chosen one but the privilege of "arousability" and providing sedation without delaying recovery may not be guaranteed as mentioned by Kim [28] in his study. He used high doses as 7 μg/kg and criticized the delayed emergence from the dexmedetomidine, lasting from 3 to 8 h, considering this is a major fault of the sedative resulting in an extended fasting period for the patient, similar to general anesthesia with a muscle relaxant. Interestingly, even with this high dose Kim [28] used ketamine to fasten the onset of dexmedetomedine, ketorolac and fentanyl to treat the intractable pain caused by surgical incision in minimally invasive spine surgery.

6. Limitation of the study

This study has several limitations. First, was that we didn't make comparisons with the control groups. In our study, cortisol was measured immediately before starting the intervention and only once in the immediate post intervention period it could be possible that effects of dexmedetomidine on endocrinal stress response are not fully apparent at the immediate post intervention or postoperative period and becomes clearer in the subsequent times. Another limitation was the small number of certain cases e.g. RFA of bone and Cementoplasty which happened mainly due to the unfavorable results occurred in both rendering it unethical to enroll more patients from such entity, in addition to the diversity of the interventions included in our study. Our study should be considered a hypothesis-generating evaluation that provides pilot data for future large-scale investigations.

7. Conclusion

The efficacy of dexmedetomidine to provide sedation for patients undergoing interventional procedures is extremely variable; site and size of the lesion, type of procedure and technical skills of the interventionist are influencing factors, efficacy is much dependent on adequately applied local anesthesia.

Dexmedetomidine use in interventional procedure led to an acceptable reduction in both heart rate and blood pressure and did not abolish the adrenal steroidogenesis. Results presented in the current study suggests the limited analgesic properties offered by dexmedetomidine when used in invasive procedures.

Dexmedetomidine could be used in interventional radiological procedures as a sole sedating agent in selected cases, thus providing an alternative choice for anesthesiologists which has no deleterious clinical effects on respiration, especially for procedures which was routinely performed under general anesthesia (e.g. hepatic RFA for centrally located lesions), this choice will much be dependent on the balance between cost and benefit.

References

[1] Venn RM, Bradshaw CJ, Spencer R, Brealey D, Caudwell E, Naughton C, et al. Preliminary UK experience of dexmedetomidine, a novel agent for postoperative sedation in the intensive care unit. Anaesthesia 1999;54:1136–42.
[2] Carollo DS, Nossaman BD, Ramadhyani U. Dexmedetomidine: a review of clinical applications. Curr Opin Anaesthesiol 2008;21:457–61.
[3] Shukry M, Miller JA. Update on dexmedetomidine: use in nonintubated patients requiring sedation for surgical procedures. Ther Clin Risk Manage 2010;6:111–21.
[4] Ramsay MA, Savege TM, Simpson BR, Goodwin R. Controlled sedation with alphaxalone-alphadolone. Br Med J 1974;2:656–9.
[5] Abdullayev R, Bavullu EN, Aksoy E, Göğüş N, Dede D. Comparison of dexmedetomidine and midazolam in sedation for percutaneous drainage of hepatic hydatid cysts. Turk J Anaesth Reanim 2013;41:195–9.
[6] Kaygusuz K, Gokce G, Gursoy S, Ayan S, Mimaroglu C, Gultekin Y. A comparison of sedation with dexmedetomidine or propofol during shockwave lithotripsy:

a randomized controlled trial. Anesth Analg 2008;106:114–9. table of contents.
[7] Bhana N, Goa KL, McClellan KJ. Dexmedetomidine. Drugs 2000;59:263–8. discussion 269–70.
[8] Aho M, Erkola O, Kallio A, Scheinin H, Korttila K. Comparison of dexmedetomidine and midazolam sedation and antagonism of dexmedetomidine with atipamezole. J Clin Anesth 1993;5:194–203.
[9] Jalowiecki P, Rudner R, Gonciarz M, Kawecki P, Petelenz M, Dziurdzik P. Sole use of dexmedetomidine has limited utility for conscious sedation during outpatient colonoscopy. Anesthesiology 2005;103:269–73.
[10] Ickeringill M, Shehabi Y, Adamson H, Ruettimann U. Dexmedetomidine infusion without loading dose in surgical patients requiring mechanical ventilation: haemodynamic effects and efficacy. Anaesth Intensive Care 2004;32:741–5.
[11] Wu W, Chen Q, Zhang L-C, Chen W-H. Dexmedetomidine versus midazolam for sedation in upper gastrointestinal endoscopy. J Int Med Res 2014;42:516–22.
[12] Ohata H, Tanemura E, Dohi S. Use of high-dose dexmedetomidine infusion for anesthesia and sedation in a patient for microlaryngeal surgery maintained with spontaneous breathing. Masui 2008;57:428–32.
[13] Ramsay MAE, Luterman DL. Dexmedetomidine as a total intravenous anesthetic agent. Anesthesiology 2004;101:787–90.
[14] Hammer GB, Sam WJ, Chen MI, Golianu B, Drover DR. Determination of the pharmacodynamic interaction of propofol and dexmedetomidine during esophagogastroduodenoscopy in children. Paediatr Anaesth 2009;19:138–44.
[15] Mason KP, Lerman J. Review article: dexmedetomidine in children: current knowledge and future applications. Anesth Analg 2011;113:1129–42.
[16] Arain SR, Ruehlow RM, Uhrich TD, Ebert TJ. The efficacy of dexmedetomidine versus morphine for postoperative analgesia after major inpatient surgery. Anesth Analg 2004;98:153–8. table of contents.
[17] Maze M, Virtanen R, Daunt D, Banks SJ, Stover EP, Feldman D. Effects of dexmedetomidine, a novel imidazole sedative-anesthetic agent, on adrenal steroidogenesis: in vivo and in vitro studies. Anesth Analg 1991;73:204–8.
[18] Venn RM, Bryant A, Hall GM, Grounds RM. Effects of dexmedetomidine on adrenocortical function, and the cardiovascular, endocrine and inflammatory responses in post-operative patients needing sedation in the intensive care unit. Br J Anaesth 2001;86:650–6.
[19] Yacout AG, Osman HA, Abdel-Daem MH, Hammouda SA, Elsawy MM. Effect of intravenous dexmedetomidine infusion on some proinflammatory cytokines, stress hormones and recovery profile in major abdominal surgery. Alexandria J Med 2012;48:3–8.
[20] Uyar AS, Yagmurdur H, Fidan Y, Topkaya C, Basar H. Dexmedetomidine attenuates the hemodynamic and neuroendocrinal responses to skull-pin head-holder application during craniotomy. J Neurosurg Anesthesiol 2008;20:174–9.
[21] Aantaa R, Kanto J, Scheinin M, Kallio A, Scheinin H. Dexmedetomidine, an alpha 2-adrenoceptor agonist, reduces anesthetic requirements for patients undergoing minor gynecologic surgery. Anesthesiology 1990;73:230–5.
[22] Osman M, Al-Medani S, Beltagi R, El-Sawy M, Neemat-Allah F. Effects of dexmedetomidine versus morphine on surgical stress response and analgesia in postoperative open cardiac surgery. Res Opin Anesth Intensive Care 2015;1:16.
[23] Limberaki E, Eleftheriou P, Gasparis G, Karalekos E, Kostoglou V, Petrou C. Cortisol levels and serum antioxidant status following chemotherapy. Health 2011;3:512–7.
[24] Song J, Kim W-M, Lee S-H, Yoon MH. Dexmedetomidine for sedation of patients undergoing elective surgery under regional anesthesia. Korean J Anesthesiol 2013;65:203–8.
[25] Koroglu A, Teksan H, Sagir O, Yucel A, Toprak HI, Ersoy OM. A comparison of the sedative, hemodynamic, and respiratory effects of dexmedetomidine and propofol in children undergoing magnetic resonance imaging. Anesth Analg 2006;103:63–7 [table of contents].
[26] Jorden VSB, Pousman RM, Sanford MM, Thorborg PAJ, Hutchens MP. Dexmedetomidine overdose in the perioperative setting. Ann Pharmacother 2004;38:803–7.
[27] Ebert TJ, Hall JE, Barney JA, Uhrich TD, Colinco MD. The effects of increasing plasma concentrations of dexmedetomidine in humans. Anesthesiology 2000;93:382–94.
[28] Kim KH. Safe sedation and hypnosis using dexmedetomidine for minimally invasive spine surgery in a prone position. Korean J Pain 2014;27:313–20.