Dexmedetomidine versus Propofol: Is One Better Than the Other for MRI Sedation in Children?

Sheikh S. Ahmed¹ Tamara L. Unland² James E. Slaven³ Mara E. Nitu¹

¹ Section of Pediatric Critical Care, Riley Hospital for Children at Indiana University Health, Indianapolis, Indiana, United States

² Pediatric Procedural Sedation, IU Health North Hospital, Carmel, Indiana, United States

³ Department of Biostatistics Indiana University, School of Medicine Indiana University, Indianapolis, Indiana, United States

Address for correspondence Sheikh S. Ahmed, MD, Section of Pediatric Critical Care, Riley Hospital for Children at Indiana University Health, 705 Riley Hospital Dr., Riley Phase 2, 4900, Indianapolis, IN 46202, United States
(e-mail: ssahmed@iu.edu; sheikh921@yahoo.com).

J Pediatr Intensive Care 2017;6:117–122.

Abstract

Objective The aim of this article is to determine whether dexmedetomidine or propofol is better for MRI sedation in children.

Design This study is a retrospective review of patients sedated with dexmedetomidine or propofol for MRI between July 2007 and July 2015. Dexmedetomidine group (group D) was administered a bolus of 2 µg/kg over 10 minutes followed by a 1 µg/kg/hour infusion. Propofol group (group P) received a bolus of 2 mg/kg over 2 minutes followed by 83 µg/kg/minute infusion.

Results Of the 996 cases completed, 452 were in group P and 544 were in group D. Patients in group P were heavier and older than those in group D. All the patients except one in group D completed the procedures. Hypotension occurred in 59% in group P versus 4% in group D (89 ± 11.4 SBP vs. 103.80 ± 19.4 ; $p < 0.05$). Bradycardia was observed in 2.9% in group P versus 0.6% in group D. Apnea occurred in two patients in group D. Although procedure time was longer in patients receiving propofol versus dexmedetomidine (58.87 ± 28.17 vs. 45 ± 23.6 ; $p < .05$), the discharge time was significantly shorter ($37. \pm 12.30$ vs. 92.61 ± 28.19 ; $p < 0.05$).

Conclusion Dexmedetomidine appears to provide a useful alternative to propofol for MRI sedation with a longer recovery time, stable hemodynamics, and less reliable respiratory profile, while the propofol had the advantage of quicker onset and rapid recovery.

Keywords

- sedation
- dexmedetomidine
- propofol

Introduction

Magnetic resonance imaging (MRI) is a noninvasive, radiation-free diagnostic procedure. MRI testing is required for diagnostic purposes of various underlying conditions (neurological or oncological diseases, orthopedic abnormalities, vascular malformations, epilepsy, and developmental delay, among others).^{1,2} An MRI scan may last from 30 to 90 minutes depending upon the particular patient and the diagnostic needs. It is very important for patients to stay still for optimal image quality for precise

diagnosis. Younger patients are usually unable to cooperate and sedation is required. Ideally, the agent used for sedation in these procedures should have a rapid onset of action, short duration, and safety profile with low risk of complications (e.g., hemodynamic instability and respiratory depression).

Dexmedetomidine is the pharmacologically active d-isomer of medetomidine. It is a potent, highly selective α_2 -adrenoceptor agonist with a distributive half-life of approximately 8 minutes and a terminal half-life of 3.5 hours.^{3,4} At therapeutic doses, dexmedetomidine is a potent sedative without significant

received

December 1, 2015

accepted after revision

April 27, 2016

published online

June 24, 2016

Copyright © 2017 by Georg Thieme
Verlag KG, Stuttgart · New York

DOI <http://dx.doi.org/10.1055/s-0036-1584683>.
ISSN 2146-4618.

cardiovascular and respiratory instability.^{5,6} On the other hand, there is a significant interest in the use of propofol for MRI sedation in children because of its rapid induction and recovery characteristics.⁷ This is a retrospective study comparing sedative, hemodynamic, and respiratory effects as well as procedure time and discharge time of dexmedetomidine and propofol in children undergoing MRI.

Methods

This study is an institutional review board–approved retrospective chart review of children who underwent procedural sedation with dexmedetomidine or propofol for MRI scan between July, 2007, and July, 2015, at Riley Hospital for Children at Indiana University Health North campus. Patients were exclusively sedated for MRI using dexmedetomidine until December of 2012, and then a transition was made to use propofol for MRI starting January, 2013. Of the 996 cases completed, 544 were in the dexmedetomidine group and 452 were in the propofol group. Data included patients' demographics, underlying and acute diagnosis, drug dosages, occurrence of adverse events, physiologic variables, and procedures, sedation, and recovery times.

Our facility has an intensivist-based sedation program that adheres to policies and guidelines based on the recommendations by the Joint Commission on Accreditation of Health Care Organization (JCAHO) and the American Academy of Pediatrics (AAP).^{8,9} Patients are prescreened via phone interview by a sedation nurse with a parent/guardian, and a review of the primary care physician's chart is performed. Study inclusion and exclusion criteria are shown in ►Table 1. The sedation nurses and physicians also assess patients at the time of sedation.

A standardized periprocedure protocol occurs for all the children undergoing MRI sedation, including a short phone discussion between the sedation nurse and a caretaker providing education prior to the procedure, a quiet room setting near the MRI suite with minimal separation from the attachment figure (e.g., mother), use of distraction techniques such as iPad games and music for intravenous (IV) placement, and monitoring leads placement and an option of oral medication for the very anxious children if deemed necessary after discussion with the family.

Our sedation team utilized a standard approach to procedural sedation for MRI. For sedation with dexmedetomidine (group D), an IV bolus of 2 µg/kg over 10 minutes was administered, followed by a maintenance infusion of 1 µg/kg/h. A second bolus

of 2 µg/kg of dexmedetomidine was administered immediately to patients who were not adequately sedated prior to the initiation of maintenance infusion. Additional bolus medication, such as midazolam or fentanyl, was also used in an individualized way when necessary, per physician discretion. When propofol was used for sedation (group P), patients received a bolus of propofol of approximately 2 mg/kg over 1 to 2 minutes followed by continuous infusion of 83 µg/kg/minute using a syringe pump. If, at any time, unwanted movement occurred, the procedure was stopped and an additional bolus of propofol was given.

Throughout the procedure, patients were monitored continuously by a dedicated sedation nurse via continuous pulse oximetry, heart rate, and noninvasive blood pressure monitoring and nasal capnography (with concomitant oxygen delivery via nasal cannula). The sedating physician was readily available to assist the nurse and the patient. Patients were monitored until they were awake and drinking fluids and had a minimal Aldrete score of 9 points.¹⁰

Clinically significant hypotension, respiratory depression, and bradycardia were defined as a decrease of >20% from the baseline values of any of these vital signs. Other studies reported a decrease in values of >20% from the baseline that was considered to be clinically insignificant.⁶ To have a true comparison with previously published data, we use the same definition to define adverse events in our study.

Peak onset of sedation has been defined as the time from the start of the loading dose to achievement of a Ramsay score of 4. Procedure time has been defined as the time from achieving the acquired Ramsay score to the end of the procedure (end of the MRI testing and of the drug administration). Recovery/discharge time was defined as the time from the end of the procedure to the actual time the patient was discharged from the recovery room to home.

The sedation groups were compared with respect to demographic, clinical, and time variables. Data were analyzed using dedicated statistical software, SAS v.9.3 (SAS Institute, Cary, North Carolina, United States). Dexmedetomidine- and propofol-induced vital sign changes from baseline in each group were compared using Student *t*-test and the Mann-Whitney rank-sum test, depending on the distribution of data, for continuous data and with Fisher exact test for categorical data. Data were presented as means ± standard deviation and frequency (percent), unless otherwise specified. A *p*-value < 0.05 was considered statistically significant. Before analysis, patients were matched on gender and within 2 months on age to create equal sample sizes and to control for age and gender. Analyses were also performed excluding those participants who had additional medicine. This enabled a comparison using only dexmedetomidine and propofol.

Table 1 Inclusion and exclusion criteria

Inclusion criteria	Exclusion criteria
ASA classification	All inpatients
Outpatients only	Intubated
MRI only	Patient taking digoxin + beta-blocker

Abbreviations: ASA, American Society of Anesthesiologists; MRI, magnetic resonance imaging.

Results

A total of 996 patients were sedated for MRI, 452 were in group P with 100% completion and 544 in group D with one failure, resulting in 99.8% satisfactory completion. The most common indications included seizure disorder, developmental delay and behavioral disorder, autism, neoplasia, and

Table 2 Patient characteristics

	Propofol (n = 452)	Dexmedetomidine (n = 544)	p-Value
Gender			
Female	208 (46.0)	229 (42.1)	0.2143
Male	244 (54.0)	315 (57.9)	
Age (in mo)	63.39 (41.65)	54.07 (39.29)	0.0003
Weight (kg)	21.23 (12.27)	19.19 (10.94)	0.006

Note: Values are means (standard deviations) for continuous variables and frequencies (percent) for categorical variables. *p*-Values are from Student *t*-tests and chi-square tests.

orthopedic abnormalities. No significant group difference was found for gender ($p = 0.2$), but the difference was significant for age ($p = 0.0003$) and weight ($p = 0.006$) among the group (► **Table 2**). The American Society of Anesthesiologists (ASA) physical status classification system is a system used to indicate one's overall physical health or "sickness" preoperatively. ASA data are shown in ► **Table 3**. In ► **Table 3**, 21 and 13 patients in group D have been placed as ASA I/II and ASA II/III, respectively. It is logical to expect a missing class between ASA II and ASA III for systemic disease which is neither mild nor severe but is of a moderate nature. It is also not clear what will be the ASA classification of a case who is suffering simultaneously from one, two, three, or more systemic disease (which might be of different severity). As a result, in some places patients were documented as being in one ASA class and in other places in another.

A total of 324 (60%) patients in group D received one-time bolus, while two boluses were given to 103 patients (19%). In the 544 patients of group D, additional medications (fentanyl or midazolam) were required in 87 (16%) for spontaneous movements to avoid motion artifacts that can compromise MRI quality (22 patients with one-time bolus and 65 patients with two boluses). Thirty (5.5%) patients received oral midazolam for anxiety and IV placement before the start of the procedure. In group P, all patients completed MRI with propofol only without any need for additional medicine.

Table 3 ASA outcomes

ASA	Propofol	Dexmedetomidine	
I	83 (18.4)	235 (43.3)	<0.0001 ^a
I/II	0 (0)	21 (3.9)	
II	326 (72.3)	237 (43.7)	
II/III	0 (0)	13 (2.4)	
III	42 (9.3)	36 (6.6)	
IV	0 (0)	1 (0.2)	

Abbreviation: ASA, American Society of Anesthesiologists.

Note: Values are frequencies (percent). *p*-Value is from chi-square test.

^aSignificant statistical association at $p \leq 0.05$.

Several studies reported a decrease in heart rate of <20% from baseline, which is considered to be clinically insignificant.⁶ Using the same definition of bradycardia, we observed a higher incidence of this side effect in group P at 33% (147 children) versus 13% in group D (70 children). Bradycardia (heart rate < 60/minute), as defined according to Pediatric Advance Life Support (PALS) guidelines, was observed in 13 children (2.9%) in group P versus 3 children (0.6%) in group D. Oxygen saturation and respiratory rates were comparable between the groups (► **Table 4**). Two of the patients (0.1%) had apneic episodes in dexmedetomidine group, but no one required bag and mask ventilation or mechanical ventilation. Most of the patients received prophylactic supplemental oxygen to maintain oxygen saturation >95% as per protocol, which makes it impossible to determine the actual incidence of desaturation at room air. Hypotension (systolic blood pressure >20% below the normal limits) was observed in 248 (59%) and 22 (4.2%) patients in group P and group D, respectively. Transient hypertension (systolic blood pressure >20% above age-specific high limits) was observed in 137 (25%) of the patients of group D only.

Procedure times were longer in patients receiving propofol versus dexmedetomidine (group P, 58.8 ± 28.17 minutes, vs. group D, 45.0 ± 23.6 minutes; $p < 0.0001$; ► **Fig. 1**), but patients receiving propofol had significantly shorter discharge time (group P, 37.0 ± 12.3 minutes, vs. group D, 92.6 ± 28.19 minutes; $p < 0.05$; ► **Fig. 2**). Onset of action to achieve Ramsay score of >4 was significantly longer in group D versus group P (13.6 ± 4.6 vs. 2.0 ± 0 minutes; $p < 0.05$). None of the patients in either group experienced any type of allergic reaction. For the subset of participants with no additional medicines, all outcomes had results similar to the full sample, in terms of both mean values and statistical significance/nonsignificance.

Discussion

This is a large retrospective study (996 patients) comparing dexmedetomidine to propofol as a sedative agent for diagnostic MRI procedures in pediatric patients. While both sedatives can be used effectively and safely for outpatient MRI, propofol has rapid induction and recovery compared with dexmedetomidine at a lesser cost (\$19.50 for propofol vs. \$52.40 for dexmedetomidine). This financial benefit becomes even more impactful if we factor in the effect of rapid recovery with subsequent higher efficiency in the MRI suite.¹¹

One of the main goals of the procedural sedation for MRI is to provide the necessary level of sedation to avoid potential movement that would affect image quality while maintaining a wide margin of safety along with hemodynamic and respiratory stability.¹² Inadequate sedation during MRI has been reported in previous studies in 5 to 15% of cases, causing failure to complete the study in 3.7%. This occurred more frequently in hyperactive, uncooperative older children.^{13,14}

Respiratory events make up a large proportion (5.5%) of sedation complications in children.¹⁵ In our study, none of the patients in either group had low respiratory rates or decreased respiratory efforts, and incidence of apnea was reported only in

Table 4 Clinical outcomes at beginning and end of procedure

	Propofol	Dexmedetomidine	p-Value
HR (initial)	103.50 (19.56)	100.80 (19.77)	0.0335 ^a
HR (complete)	87.78 (15.78)	91.74 (16.03)	0.0001 ^a
Resp (initial)	20.19 (3.10)	20.59 (3.71)	0.0729
Resp (complete)	19.99 (4.91)	20.31 (5.04)	0.3211
SBP (initial)	115.40 (13.52)	112.50 (13.53)	0.0011 ^a
SBP (complete)	89.40 (11.44)	103.80 (9.44)	<0.0001 ^a
DBP (initial)	67.0 (13.03)	66.37 (11.89)	0.4436
DBP (complete)	42.91 (10.75)	60.50 (8.48)	<0.0001 ^a
SpO ₂	99.08 (1.30)	99.04 (1.20)	0.1830

Abbreviations: DBP, diastolic blood pressure; HR, heart rate; Resp, respiratory rate; SBP, systolic blood pressure; SpO₂, oxygen saturation as measured by pulse oximetry.

Note: Values are means (standard deviations). p-Values are from Student t-tests.

^aSignificant statistical association at $p \leq 0.05$.

two patients (0.1%) in dexmedetomidine group. All the patients in both groups received prophylactic supplemental oxygen to maintain oxygen saturations > 95 as per protocol, which makes it impossible to determine the actual incidence of desaturation at room air. Despite that, monitoring of respiratory function during administration of sedation either with propofol or dexmedetomidine (may depress respiratory function) is warranted.

Propofol's powerful inhibitory effect on sympathetic outflow has been demonstrated in a laboratory study.¹⁶ Dexmedetomidine also causes a decrease in heart rate and blood pressure similar to propofol due to its inhibiting effect of the sympathetic outflow and circulating catecholamine's level.^{17,18} As expected, the heart rate decreased significantly from baseline in our patient cohort. During sedation, this is an expected effect of anxiolysis and reflects higher baseline levels from anxiety. Other studies reported a decrease in heart rate of <20% from baseline, considered to be clinically insignificant.⁶ Using the same definition of bradycardia, we observed a higher incidence of this side effect in group P at 33% (147 children) versus 13% in group D (70 children).

In our study, the systolic blood pressure was decreased from baseline in both groups, which could be the result of a sympathetic inhibitory effect of both sedatives. However, direct effect of dexmedetomidine at postsynaptic vascular smooth muscles causes vasoconstriction that opposes sympathoinhibitory effects. This vasoconstrictive effect appears earlier than the central sympatholytic effects^{19,20} and may be the reason for lower incidence of hypotension seen in patients sedated with dexmedetomidine versus propofol (4.2 vs. 59.1%). None of the hypotensive episodes were considered clinically significant, finding consistent with observations reported in other studies.^{21–23}

In this study, onset of sedation with propofol was rapid compared with dexmedetomidine alone (2.0 ± 0 vs. 13.6 ± 4.58 minutes). The longer induction time with dexmedetomidine was due to the slow infusion over 10 minutes to avoid the undesirable hemodynamic changes that occur with faster infusion. In a study by Arain and Ebert, sedation onset with propofol was 10 minutes in comparison to 25 minutes with dexmedetomidine.²⁴ Although we demonstrated rapid onset of sedation with propofol compared with dexmedetomidine, the

Fig. 1 Comparison of procedure times between dexmedetomidine versus propofol group. **Fig. 2** Comparison of recovery + discharge times between dexmedetomidine and propofol group.

onset of targeted sedation in both groups was accomplished faster in our study than those reported by Arain and Ebert. This is likely explained by the difference in the sedation protocols and the sedation assessment methodology used in the two studies. The loading dose in Arain and Ebert study was 0.075 mg/kg for propofol and 1 µg/kg for dexmedetomidine targeting a bispectral index score of 70 to 80. Furthermore, the patient population is different, as our study is conducted in a pediatric population averaging 5 years of age versus a geriatric population with an average age of 62 years.

The discharge time observed in our patient cohort was 92 minutes with dexmedetomidine and 37 minutes with propofol, which is comparable to the 90 and 39 minutes discharge time of Heard et al and Koroglu et al, respectively.^{25,26} Lubisch et al observed a recovery time of 47 minutes, and Mason et al reported recovery times ranging from 24.8 to 35.2 minutes depending on the dose of dexmedetomidine used, while Pershad et al reported recovery times of 17 minutes for propofol.^{21,27,28} In some studies, recovery time is defined as the time lapsed until the patient meets discharge criteria, while our study, like a few others, defined discharge time as the actual time of leaving the recovery room.²⁵

Several small clinical trials have compared dexmedetomidine to propofol. In a recent trial, 95 children, ages 1 to 7 years, were randomly assigned to be sedated either with propofol or dexmedetomidine for MRI. They found shorter induction, recovery, and discharge time in propofol group with higher parental satisfaction.²⁶ In a trial of 60 children undergoing MRI, patients were randomized to receive either dexmedetomidine or propofol. Adequate sedation was achieved in 83 and 90% of the patients, respectively. Onset, recovery, and discharge time were all significantly shorter in the propofol group. Adverse effects, such as lower blood pressure, heart rate, and respiratory rate, were also found more in the propofol group.²⁹

In another trial, 40 children, ages 1 to 10 years, were randomized to receive either a combination of midazolam + dexmedetomidine or propofol only for MRI sedation. Recovery and discharge times were 15 minutes longer in the dexmedetomidine group. No adverse events were recorded in either group.¹¹ Koroglu et al compared dexmedetomidine versus propofol in children for sedation and found dexmedetomidine preserved heart rate and mean arterial pressure better than propofol. Also, the incidence of oxygen desaturation occurred more frequently with propofol, but onset of sedation, recovery, and discharge time were significantly shorter with propofol.²⁹

Our study strengthens the above-mentioned results, particularly because of a much larger patient cohort with good matching of demographic variables. Compiling all these data, it is safe to state that both sedation techniques are effective. Propofol alone can be used successfully to provide procedural sedation for MRI studies in 100% of cases, while high-dose dexmedetomidine was effective alone in 78.5% of the cases; the remaining 21.5% of the patients required additional medications. The effects of addition of fentanyl or midazolam to a patient receiving dexmedetomidine could increase the risk of respiratory depression and hypotension. Considering these possible effects on clinical data of dexmedetomidine sedation, we have performed the same analyses excluding

these 21.5% with additional medications. The results were similar with no changes in significant outcomes. The slightly safer hemodynamic and respiratory profile of dexmedetomidine could be offset by a significant increase in induction and recovery time and therefore a decreased turnover and a decreased efficiency of a busy MRI suite.

The limitations of our study include its retrospective nature and single-center experience. The current study presents a 99.8% (one failure) success with dexmedetomidine and 100% success with propofol for MRI. It could be asserted that the reported efficacy is due to the use of intensivist-based specialized sedation team rather than due to dexmedetomidine or propofol itself. This is reasonably true to some extent as specialization and experience should increase both success and efficiency. In spite of that, this can be stated with confidence: much of the reported success is specifically a function of dexmedetomidine or propofol. This is a descriptive study and few, if any, conclusion can be drawn about safety, is also underpowered to comment on safety, because the occurrence of serious sedation-related side effects are, fortunately, rare.³⁰ Additional prospective studies of the sedation for MRI in children using a greater number of patients are warranted to provide a true idea of safety.

In conclusion, dexmedetomidine appears to provide a useful alternative to propofol for MRI sedation with a longer recovery time, stable hemodynamics, and less reliable respiratory profile, while propofol had the advantage of quicker onset and rapid recovery. Dexmedetomidine has also been used successfully to sedate mechanically ventilated patients, including its recently expanded indication for orthopedic, vascular, and ophthalmic procedures in operation room in nonventilated patients. This could also be a good alternative to propofol for patients with metabolic disorders and soy and egg allergies.

References

- 1 Funk W, Hörauf K, Held P, Taeger K. Anesthesia for magnetic resonance tomography in neonates, infants and young children [in German]. *Radiologe* 1997;37(2):159–164
- 2 Kannikeswaran N, Mahajan PV, Sethuraman U, Groebe A, Chen X. Sedation medication received and adverse events related to sedation for brain MRI in children with and without developmental disabilities. *Paediatr Anaesth* 2009;19(3):250–256
- 3 Bhana N, Goa KL, McClellan KJ. Dexmedetomidine. *Drugs* 2000; 59(2):263–268, discussion 269–270
- 4 Shelly MP. Dexmedetomidine: a real innovation or more of the same? *Br J Anaesth* 2001;87(5):677–678
- 5 Nelson LE, Lu J, Guo T, Saper CB, Franks NP, Maze M. The alpha2-adrenoceptor agonist dexmedetomidine converges on an endogenous sleep-promoting pathway to exert its sedative effects. *Anesthesiology* 2003;98(2):428–436
- 6 Koroglu A, Demirbilek S, Teksan H, Sagir O, But AK, Ersoy MO. Sedative, haemodynamic and respiratory effects of dexmedetomidine in children undergoing magnetic resonance imaging examination: preliminary results. *Br J Anaesth* 2005;94(6):821–824
- 7 Deegan RJ. Propofol: a review of the pharmacology and applications of an intravenous anesthetic agent. *Am J Med Sci* 1992;304(1):45–49
- 8 American Society of Anesthesiologists Task Force on Sedation and Analgesia by Non-Anesthesiologists. Practice guidelines for sedation and analgesia by non-anesthesiologists. *Anesthesiology* 2002;96(4):1004–1017

- 9 Coté CJ, Wilson S; American Academy of Pediatrics; American Academy of Pediatric Dentistry; Work Group on Sedation. Guidelines for monitoring and management of pediatric patients during and after sedation for diagnostic and therapeutic procedures: an update. *Pediatrics* 2006;118(6):2587–2602
- 10 Aldrete JA, Kroulik D. A postanesthetic recovery score. *Anesth Analg* 1970;49(6):924–934
- 11 Heard C, Burrows F, Johnson K, Joshi P, Houck J, Lerman J. A comparison of dexmedetomidine-midazolam with propofol for maintenance of anesthesia in children undergoing magnetic resonance imaging. *Anesth Analg* 2008;107(6):1832–1839
- 12 Schulte-Tamburen AM, Scheier J, Briegel J, Schwender D, Peter K. Comparison of five sedation scoring systems by means of auditory evoked potentials. *Intensive Care Med* 1999;25(4):377–382
- 13 Malviya S, Voepel-Lewis T, Eldevik OP, Rockwell DT, Wong JH, Tait AR. Sedation and general anaesthesia in children undergoing MRI and CT: adverse events and outcomes. *Br J Anaesth* 2000;84(6):743–748
- 14 Voepel-Lewis T, Malviya S, Prochaska G, Tait AR. Sedation failures in children undergoing MRI and CT: is temperament a factor? *Paediatr Anaesth* 2000;10(3):319–323
- 15 Hasan RA, Shayevitz JR, Patel V. Deep sedation with propofol for children undergoing ambulatory magnetic resonance imaging of the brain: experience from a pediatric intensive care unit. *Pediatr Crit Care Med* 2003;4(4):454–458
- 16 Ebert TJ, Muzi M, Berens R, Goff D, Kampine JP. Sympathetic responses to induction of anesthesia in humans with propofol or etomidate. *Anesthesiology* 1992;76(5):725–733
- 17 Ebert TJ, Hall JE, Barney JA, Uhrich TD, Colino MD. The effects of increasing plasma concentrations of dexmedetomidine in humans. *Anesthesiology* 2000;93(2):382–394
- 18 Talke P, Chen R, Thomas B, et al. The hemodynamic and adrenergic effects of perioperative dexmedetomidine infusion after vascular surgery. *Anesth Analg* 2000;90(4):834–839
- 19 Aantaa R. Assessment of the sedative effects of dexmedetomidine, an alpha 2-adrenoceptor agonist, with analysis of saccadic eye movements. *Pharmacol Toxicol* 1991;68(5):394–398
- 20 Bloor BC, Ward DS, Belleville JP, Maze M. Effects of intravenous dexmedetomidine in humans. II. Hemodynamic changes. *Anesthesiology* 1992;77(6):1134–1142
- 21 Mason KP, Zurakowski D, Zgleszewski SE, et al. High dose dexmedetomidine as the sole sedative for pediatric MRI. *Paediatr Anaesth* 2008;18(5):403–411
- 22 Berkenbosch JW, Wankum PC, Tobias JD. Prospective evaluation of dexmedetomidine for noninvasive procedural sedation in children. *Pediatr Crit Care Med* 2005;6(4):435–439, quiz 440
- 23 Tecanivate A, Verawattagnon T, Saiyuenyong C, Areeruk P. A comparison of dexmedetomidine versus propofol on hypotension during colonoscopy under sedation. *J Anesth Clin Res* 2012;3:257
- 24 Arain SR, Ebert TJ. The efficacy, side effects, and recovery characteristics of dexmedetomidine versus propofol when used for intraoperative sedation. *Anesth Analg* 2002;95(2):461–466
- 25 Heard CM, Joshi P, Johnson K. Dexmedetomidine for pediatric MRI sedation: a review of a series of cases. *Paediatr Anaesth* 2007;17(9):888–892
- 26 Wu J, Mahmoud M, Schmitt M, Hossain M, Kurth D. Comparison of propofol and dexmedetomidine techniques in children undergoing magnetic resonance imaging. *Paediatr Anaesth* 2014;24(8):813–818
- 27 Lubisch N, Roskos R, Berkenbosch JW. Dexmedetomidine for procedural sedation in children with autism and other behavior disorders. *Pediatr Neurol* 2009;41(2):88–94
- 28 Pershad J, Wan J, Anghelescu DL. Comparison of propofol with pentobarbital/midazolam/fentanyl sedation for magnetic resonance imaging of the brain in children. *Pediatrics* 2007;120(3):e629–e636
- 29 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(1):63–67
- 30 Cravero JP, Blike GT, Beach M, et al; Pediatric Sedation Research Consortium. Incidence and nature of adverse events during pediatric sedation/anesthesia for procedures outside the operating room: report from the Pediatric Sedation Research Consortium. *Pediatrics* 2006;118(3):1087–1096