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High dose dexmedetomidine as the sole sedative for pediatric MRI

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Summary

Objective: This large-scale retrospective review evaluates the sedation profile of dexmedetomidine.

Aim: To determine the hemodynamic responses, efficacy and adverse events associated with the use of high dose dexmedetomidine as the sole sedative for magnetic resonance imaging (MRI) studies.

Background: Dexmedetomidine has been used at our institution since 2005 to provide sedation for pediatric radiological imaging studies. Over time, an effective protocol utilizing high dose dexmedetomidine as the sole sedative agent has evolved.

Methods/Materials: As part of the ongoing Quality Assurance process, data on all sedations are reviewed monthly and protocols modified as needed. Data were analyzed from all 747 consecutive patients who received dexmedetomidine for MRI sedation from April 2005 to April 2007.

Results: Since 2005, the 10-min loading dose of our dexmedetomidine protocol increased from 2 to 3 $\mu g \cdot kg^{-1}$, and the infusion rate increased from 1 to 1.5 to 2 $\mu g \cdot kg^{-1} \cdot h^{-1}$. The current sedation protocol progressively increased the rate of successful sedation (able to complete the imaging study) when using dexmedetomidine alone from 91.8% to 97.6% ($P = 0.009$), reducing the requirement for adjuvant pentobarbital in the event of sedation failure with dexmedetomidine alone and decreased the mean recovery time by 10 min ($P < 0.001$). Although dexmedetomidine sedation was associated with a 16% incidence of bradycardia, all concomitant mean arterial blood pressures were within 20% of age-adjusted normal range and oxygen saturations were 95% or higher.

Conclusion: Dexmedetomidine in high doses provides adequate sedation for pediatric MRI studies. While use of high dose dexmedetomidine is associated with decreases in heart rate and blood pressure outside the established 'awake' norms, this deviation is generally within 20% of norms, and is not associated with adverse sequelae. Dexmedetomidine is useful as the sole sedative for pediatric MRI.

Keywords: pharmacology:dexmedetomidine; safety drug; sedation

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Introduction

Infants, children and some developmentally compromised adolescents frequently require sedation to ensure motionless conditions during magnetic resonance imaging (MRI) studies. Historically, chloral hydrate, fentanyl, midazolam and pentobarbital have been the drugs of choice in radiology departments (1–5). Pentobarbital (Nembutal; Abbott, North Chicago, IL, USA) and chloral hydrate (Major Pharmaceuticals, Rosemont, IL, USA), the drugs most commonly administered have half-lives which approach 24 h and have been associated with prolonged recovery times and sedation-related morbidity (6–8). Dexmedetomidine is a highly selective $\alpha 2$ adrenoceptor agonist that has sedative and analgesic effects (9). In 2005, following approval by the Radiology and Hospital Sedation Committee, dexmedetomidine (Precedex; Hospira, Lake Forest, IL, USA) replaced pentobarbital as the standard for computerized tomography (CT) imaging in our institution (10). Thereafter, it was introduced to the MRI environment using the same dosing protocol that had already been established in CT. When used for MRI sedation, it became immediately apparent that there were challenges not previously encountered in the CT environment. Specifically, the noise generated by the scanner (despite insertion of earplugs), the vibration of the MRI table during image acquisition, and the lengthy duration of studies often resulted in patient movement sufficient to render the subsequent images nondiagnostic. At our institution, patients undergoing CT scanning are sedated on the CT table of the CT scanner. In contrast, patients undergoing MRI are sedated outside the scanner in a designated sedation room and are subsequently transferred to the MRI scanner. This transport of the patient can be stimulating, especially as the MRI table bumps over the door threshold and is 'locked' in place in the magnet bore.

After review of monthly Quality Assurance (QA) data over the course of several months, it soon became apparent that the protocol in current use for CT imaging needed to be modified for the MRI environment. Motionless conditions were not as reliably being attained for the duration of the MRI scans as compared to CT studies. Over the course of 2 years, the dexmedetomidine protocol for MRI sedation evolved into the current protocol. To our knowledge, this is the first large-scale evaluation of the hemodynamic effects, efficacy and adverse events associated with high dose dexmedetomidine as the sole sedative agent for pediatric MRI.

Methods

Sedation database

In December 1993, our institution established the Radiology Sedation Committee to create sedation guidelines for the Department of Radiology and to monitor quality assurance (QA) data. At our institution, all QA data is reviewed monthly at Radiology Sedation Committee meetings. Routine data collection includes patient demographics, information on the imaging conditions, the occurrence of adverse events, physiologic variables, drug dosages, the time necessary to sedate the patient, the time necessary to obtain the imaging study, and the recovery time (time spent in recovery room prior to meeting discharge criteria). Data are entered into a computerized database (FILEMAKERPRO, version 2.1; Claris, Cupertino, CA, USA) by a single designated staff member.

Sedation protocol

At our institution, Sedation Policies and Guidelines are based on those recommended by the Joint Commission on Accreditation of Healthcare Organization (JCAHO) and the American Academy of Pediatrics (AAP) (11,12). Prior to arrival, all patients are screened to confirm that they are appropriate sedation candidates. The screening process includes review of all pertinent past medical, surgical, sedation and anesthetic histories. All parents are directly contacted by telephone to clarify any medical issues. Our contraindications to dexmedetomidine sedation are listed in Table 1. Physiologic monitoring during MRI sedation is consistent with recommendations by the AAP (12). Continuous pulse oximetry, heart rate, noninvasive blood pressure monitoring and nasal capnography (with concomitant oxygen delivery via the cannula) are documented every 5 min throughout sedation.

Preprinted templated dexmedetomidine order sheets approved by the Pharmacy and Therapeutics Committee are used. All patients have an intra-

Table 1
Medical conditions which contraindicate dexmedetomidine and nurse-administered sedation

Active, uncontrolled gastroesophageal reflux – an aspiration risk
Active, uncontrolled vomiting – an aspiration risk
Current (or within past 3 months) history of apnea requiring an apnea monitor
Active, current respiratory issues that are different from the baseline status (pneumonia, exacerbation of asthma, bronchitis, respiratory syncytial virus)
Unstable cardiac status (life threatening arrhythmias, abnormal cardiac anatomy, significant cardiac dysfunction)
Craniofacial anomaly which could make it difficult to effectively establish a mask airway for positive pressure ventilation if needed
Current use of digoxin
Moya Moya disease
New onset stroke

venous catheter placed upon arrival to the MRI area. The dexmedetomidine order sheet specifies the following: a bolus of dexmedetomidine (Bolus 1) is administered at a specified dose in $\mu g \cdot kg^{-1}$ over 10 min. A minimum Ramsay Sedation Score (RSS) of 4 is targeted (13). RSS 4 or 5 is a clinically derived scoring system that is generally accepted as the depth of sedation which is adequate to facilitate diagnostic imaging studies (10,14). After bolus 1, the sedation score is assessed, and the bolus is repeated at the same dosage over 10 min (bolus 2) if a RSS 4 has not been achieved. As soon as the RSS 4 is achieved (following completion of either the first or second bolus), an infusion (Iradimed 3850 mRidium MR IV pump, Iradimed Corp, Winter Park, FL, USA) at a specified dosage in $\mu g \cdot kg^{-1} \cdot h^{-1}$ (Infusion) is immediately started. All sedation is performed outside the MRI suite in specially designated sedation rooms. Once the patient has achieved adequate sedation, he is transferred into the MRI suite. Should the patient awaken during transport or once in the MRI scanner, a repeat bolus can be administered. A patient can receive no more than three boluses during the entire sedation period. If adequate sedation is not achieved after these boluses, 2 $mg \cdot kg^{-1}$ pentobarbital is administered as a 'rescue' in an effort to induce sedation adequate to complete the scan. A failed sedation is defined as: i) the inability to successfully complete the imaging study and/or ii) the inability to obtain images of diagnostic quality as determined by the attending radiologist. Furthermore, a successful sedation always implies an imaging study that was acceptable to the radiologists for appropriate interpretation. Should a study be deemed uninterpretable related to patient movement, then the sedation would be classified as a 'failed sedation'. Should the pentobarbital not produce adequate sedation, the child is rescheduled for a general anesthetic. Following completion of the MR scan, the dexmedetomidine is discontinued, the patient is transferred to the radiology recovery room and a recovery nurse continues to monitor the vital signs every 5 min until discharge criteria are met. As per our institutional guidelines, discharge criteria requires a minimum Aldrete Score of 9 points (15).

Our current dexmedetomidine protocol has evolved over a 2-year period. Specifically, the bolus and infusion dosages were modified in response to review of sedation efficacy and adverse event data. All dosage changes were approved by the Radiology and Hospital Sedation Committee, as well as the Pharmacy and Therapeutics Committee prior to implementation. In this manner, the protocol evolved from a 10-min loading dose of 2–3 $\mu g \cdot kg^{-1} \cdot h^{-1}$. The infusion rates went through three successive changes from 1 to 1.5 $\mu g \cdot kg^{-1} \cdot h^{-1}$ and finally to 2 $\mu g \cdot kg^{-1} \cdot h^{-1}$.

Record review

After obtaining Institutional Review Board (IRB) approval for retrospective record review, the QA data were reviewed to compare outcome variables for all children who underwent MRI with dexmedetomidine. The IRB waived the need for informed consent for this retrospective review of QA data. A total of 747 pediatric patients received dexmedetomidine for MRI sedation between April 2005 and April 2007.

Statistical analysis

Dexmedetomidine doses were compared with respect to age, weight, time to sedation, duration of sedation, and time to recovery using the *F*-test in ANOVA with Bonferroni adjustment for multiple group comparisons. ASA level and gender were assessed between groups using chi-square testing. The entire pediatric study cohort was assessed to identify patients whose lowest heart rates were below the age-based normal range and thus showed

an occurrence of bradycardia (16). Among the subgroup of patients below the normal range for heart rate, their mean arterial blood pressures (MAPs) were evaluated based on established age-specific 'awake' normal ranges (16). Percentages of patients below the normal range for heart rate were compared among the three doses using chi-square, and multivariate logistic regression was used to determine if the dexmedetomidine dose was associated with a heart rate below the normal range after adjusting for age, sex, weight, and ASA level (17). The percentages of each of the three dexmedetomidine dosing groups requiring intravenous pentobarbital and rebolusing either because of failure of initial sedation or because the patient awoke in the scanner were analyzed using the Pearson's chi-square test. Fisher's exact test was used to test whether adjuvant use of pentobarbital increased the incidence of bradycardia. Multiple logistic regression was used to evaluate whether differences in adjuvant pentobarbital requirements and requirement for repeat bolus of dexmedetomidine were explained by initial dexmedetomidine dose independent of gender, age, weight, and ASA level. Adjusted odds ratios and 95% confidence intervals (CI) were calculated to establish risk reduction because of protocol dose. Statistical analysis was performed using SPSS version 16.0 (SPSS Inc, Chicago, IL, USA). Power analysis (NQUERY ADVISOR, version 6.0, Statistical Solutions, Boston, MA, USA) indicated that sample sizes of at least 100 patients in each of the dexmedetomidine protocol groups would provide 90% power ($\alpha = 0.05$, $\beta = 0.10$) to detect significant differences of 10% in the percentage of patients below the normal range in heart rate based on chi-square test of proportions (18).

Results

Demographic characteristics and time to sedation, duration, and recovery are presented in Table 2 for all three dexmedetomidine dosing groups: dose 1, bolus dose 2 $\mu g \cdot kg^{-1}$, infusion rate 1.0 $\mu g \cdot kg^{-1} \cdot h^{-1}$; dose 2, bolus dose 3 $\mu g \cdot kg^{-1}$, infusion rate 1.5 $\mu g \cdot kg^{-1} \cdot h^{-1}$; dose 3, bolus dose 3 $\mu g \cdot kg^{-1}$, infusion rate 2.0 $\mu g \cdot kg^{-1} \cdot h^{-1}$. All patients were sedated to RSS 4 or 5. No patients reached RSS 6. No significant group differences were found for gender ($P = 0.31$), age ($P = 0.47$), or weight ($P = 0.70$), however, ASA levels tended to be higher in patients receiving the highest dose of dexmedetomidine ($P < 0.001$). Time to sedation was longer for patients in dose group 1 than doses 2 and 3 ($P < 0.001$). Duration of sedation was longer and recovery time was shorter for patients in dose 3 group compared with groups 1 and 2 ($P < 0.001$). There were no adverse events reported during the recovery phase.

As shown in Table 3, among 55 patients in the study who required adjuvant pentobarbital for sedation because of scans that could not be

Table 2
Demographic characteristics and sedation times according to dexmedetomidine (Dex) dosing protocols (April 2005–February 2007)

Characteristic	*Dex dose 1* (n = *416*)	*Dex dose 2* (n = *164*)	*Dex dose 3* (n = *167*)	*P-value*
Sex, no. (%)				0.31
Male	243 (58.4)	93 (56.7)	86 (51.5)	
Female	173 (41.6)	71 (43.3)	81 (48.5)	
Age, years	4.8 ± 3.4	4.4 ± 3.5	4.5 ± 3.4	0.47
Range, years	0.1–19.9	0.2–18.7	0.2–17.9	0.70
Weight, kg	20.5 ± 12.0	19.6 ± 12.5	20.0 ± 12.7	
ASA, (%)				<0.001[a]
1	37 (8.9)	21 (12.8)	35 (21.0)	
2	311 (74.8)	129 (78.7)	95 (56.9)	
3	68 (16.3)	14 (8.5)	37 (22.2)	
Time to sedation, min	13.4 ± 6.1	11.8 ± 4.1	11.8 ± 4.4	<0.001[b]
Duration, min	49.8 ± 16.9	47.6 ± 16.4	58.6 ± 22.8	<0.001[c]
Time to recovery, min	35.2 ± 29.4	32.1 ± 20.0	24.8 ± 19.5	<0.001[d]

Plus and minus values are mean ± SD.
Dose 1, bolus dose 2 $\mu g \cdot kg^{-1}$, infusion rate 1.0 $\mu g \cdot kg^{-1} \cdot h^{-1}$; dose 2, bolus dose 3 $\mu g \cdot kg^{-1}$, infusion rate 1.5 $\mu g \cdot kg^{-1} \cdot h^{-1}$; dose 3, bolus dose 3 $\mu g \cdot kg^{-1}$, infusion rate 2.0 $\mu g \cdot kg^{-1} \cdot h^{-1}$.
[a]All doses significantly different from each other regarding ASA distribution. [b]Dose 1 significantly longer than dose 2 and 3. [c]Dose 3 significantly longer than doses 1 and 2. [d]Dose 3 significantly shorter than doses 1 and 2.

Table 3
Comparison of adjuvant pentobarbital and rebolusing requirements

Event of interest	*Dexmedetomidine dose 1* (n = *416*)	*Dexmedetomidine dose 2* (n = *164*)	*Dexmedetomidine dose 3* (n = *167*)
Received pentobarbital	34 (8.2%)	17 (10.4%)	4 (2.4%)[a]
Rebolus – initial sedation	112 (26.9%)	25 (15.2%)[b]	25 (15.0%)[b]
Rebolus – awakes in scanner	65 (15.6%)	24 (14.6%)	32 (19.2%)

Dose 1, bolus dose 2 $\mu g \cdot kg^{-1}$, infusion rate 1.0 $\mu g \cdot kg^{-1} \cdot h^{-1}$; dose 2, bolus dose 3 $\mu g \cdot kg^{-1}$, infusion rate 1.5 $\mu g \cdot kg^{-1} h^{-1}$; dose 3, bolus dose 3 $\mu g \cdot kg^{-1}$, infusion rate 2.0 $\mu g \cdot kg^{-1} h^{-1}$.
[a]$P < 0.001$ compared to dexmedetomidine dose 1 and 2. [b]$P = 0.007$ compared to dexmedetomidine dose 1.

completed (7.4% overall), there was a significantly lower percentage in dexmedetomidine dose 3 (2.4%) compared with dose 1 (8.2%, $P = 0.009$) and dose 2 (10.4%, $P = 0.003$). Multivariate logistic regression analysis indicated that dexmedetomidine dose 3 (highest dose) was associated with significantly lower rate of incomplete MRI scans than doses 1 and 2 (both $P < 0.01$), even after adjusting for the potentially confounding effects of gender, age, weight, and ASA level. The estimated reduction in the odds of an incomplete scan using dexmedetomidine dose 3 was 72% (95% confidence interval of 21–90%).

Interestingly, bradycardia occurred in 7 of the 55 patients requiring adjuvant pentobarbital for sedation (13%) and in 113 of the 692 patients (16%) who did not require pentobarbital ($P = 0.57$, Fisher's exact test), indicating that use of pentobarbital did not increase the risk of bradycardia in this pediatric population. In general, the duration of the bradycardia in individual patients was highly variable, ranging from 5 min to the duration of the dexmedetomidine sedation. Rebolusing during the initial sedation was significantly lower for dose 2 (15.2%) and dose 3 (14.6%) compared with dose 1 (26.9%, $P < 0.001$). No group differences were observed in the percentage of patients requiring rebolusing because of awaking while in the scanner (group 1: 15.6%, group 2: 14.6%, group 3: 19.2%, overall $P = 0.48$) (Figure 1). There was only one patient among the total of 747 study patients who did not complete the scan and this occurred in the lowest dose 1 group. Recovery time was significantly longer for the 55 patients who required pentobarbital for sedation compared with 692 who did not (72 ± 32 min vs. 29 ± 23 min, $P < 0.001$, Student's *t*-test). This longer recovery time for those children needing pentobarbital was consistent for each of the three dexmedetomidine protocols.

Multivariate predictors for rebolusing during the initial sedation included the gender with a higher incidence among males ($P = 0.007$) and the dose of dexmedetomidine with doses 2 and 3 showing significantly lower rates of rebolusing than dose 1 independent of age, gender, weight and ASA level ($P < 0.001$). There were no significant predictors of rebolusing because of the patient awakening in the scanner and no differences between the three dexmedetomidine protocols even after adjusting for age, gender, weight, and ASA. The duration of sedation was significantly longer in patients who were rebolused because of awakening in the scanner, on average 20 min longer, regardless of specific dose ($P < 0.001$).

In the population of 747 patients, there were 120 children (16.1%) with heart rates below the age-specific normal awake ranges (16), however in 28

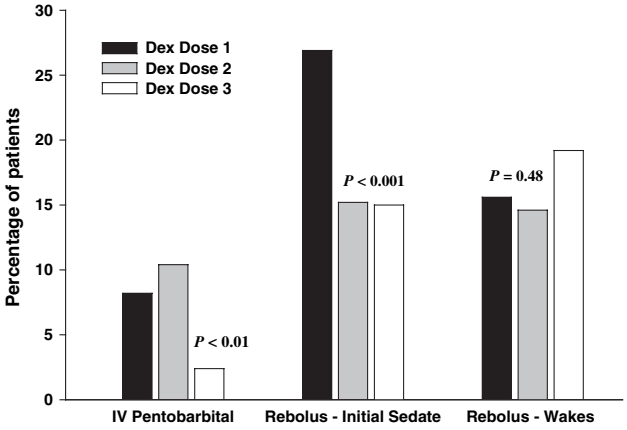

Figure 1
Comparison of rates of pentobarbital for rescue and need for rebolusing between the three dexmedetomidine protocols. The rebolus initial sedate group denotes those patients who required two boluses initially to achieve successful sedation. The rebolus wakes group identifies those who awoke in the magnetic resonance image scanner during acquisition of images, and subsequently required an additional bolus to achieve adequate sedation.

children (<4% of total cohort) the lowest recorded heart rate during sedation fell greater than 20% below the lower normal limits. In several cases, heart rates fell to less than 60 b·min^{-1} for children less than 1 year; these children were assessed by the supervising anesthesiologist and all had MAPs within the normal range and oxygen saturations of 95% or higher during bradycardia and no treatment was given. Among the 120 children in whom bradycardia occurred, the time of occurrence varied. In 15 children (13%), bradycardia occurred during bolus in the MRI scanner, in 36 children (30%) bradycardia occurred during bolus in the sedation room, in 53 children (44%) bradycardia occurred during infusion, and in 16 children bradycardia occurred in the recovery room. The time at which bradycardia occurred was not associated with dexmedetomidine dose. The precise etiology (sinus bradycardia, junctional escape) of the bradycardia observed in this analysis cannot be delineated as electrocardiographic monitoring is not a standard for MRI, especially because during the acquisition of imaging studies, the electrocardiogram (EKG) is often neither reliable nor interpretable. Thus, in the MRI environment, the exact focus of the bradycardia (sinus, nodal, etc) cannot be delineated. The recovery time of children experiencing bradycardia was almost identical to those who did not experience bradycardia.

Mean recovery time among the 120 children with bradycardia was not significantly different than the other 627 children in the study population without bradycardia (34 ± 22 min vs 32 ± 26 min, $P = 0.45$, Student's *t*-test). In addition, no differences in recovery time were observed between patients with and without bradycardia in each of the dexmedetomidine dosing protocols (Table 4).

Table 4
Recovery times for patients with and without bradycardia according to dexmedetomidine dosing protocol

	Bradycardia	n	*No. of bradycardia*	n	*P-value*
Dose 1	38 ± 25	64	34 ± 29	352	0.25
Dose 2	30 ± 17	35	33 ± 21	129	0.56
Dose 3	24 ± 12	21	25 ± 20	146	0.75
All doses	34 ± 22	120	32 ± 26	627	0.45

Plus and minus values are mean ± SD (min).
Dose 1, bolus dose 2 μg·kg^{-1}, infusion rate 1.0 μg·kg^{-1}·h^{-1}; dose 2, bolus dose 3 μg·kg^{-1}, infusion rate 1.5 μg·kg^{-1}·h^{-1}; dose 3, bolus dose 3 μg·kg^{-1}, infusion rate 2.0 μg·kg^{-1}·h^{-1}.

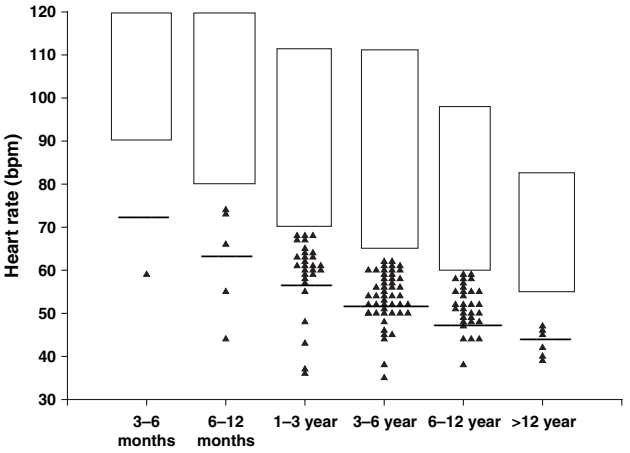

Figure 2
Heart rates for the 120 children below the age-specific normal range. Boxes represent normal range and tick marks denote heart rate values 20% below the normal range (16). Thirty children of the entire cohort of 747 (<4%) were beyond the lower limit of normal by more than 20%.

The distribution of heart rates is shown in Figure 2. The percentage of patients with heart rates below the normal range was not related to dexmedetomidine protocol ($P = 0.10$, chi-square test) nor was it related to ASA level ($P = 0.41$, chi-square test) or age ($P = 0.22$, *t*-test). However, when we examined total dexmedetomidine dose for each patient, there was a moderate inverse correlation between dose and the lowest heart rate observed ($r = -0.34$, $P < 0.01$) suggesting that a higher total dose may be associated with lower heart rate.

We also analyzed the MAP values for the subgroup of 120 children with heart rates below norms (Figure 3). While many children were in fact outside the age-specific normal ranges, none were beyond 20% of normal boundaries in either direction. All of the 120 children with heart rates below normal range had O_2 saturations of 95% or higher and approximately 92% had O_2 saturations between 97% and 100%.

Discussion

We have demonstrated that high dose dexmedetomidine can be used safely as the sole sedative agent for successful acquisition of pediatric MRI scans. Our findings are important, as previous studies have shown that dexmedetomidine, albeit at dosages significantly lower than ours does not

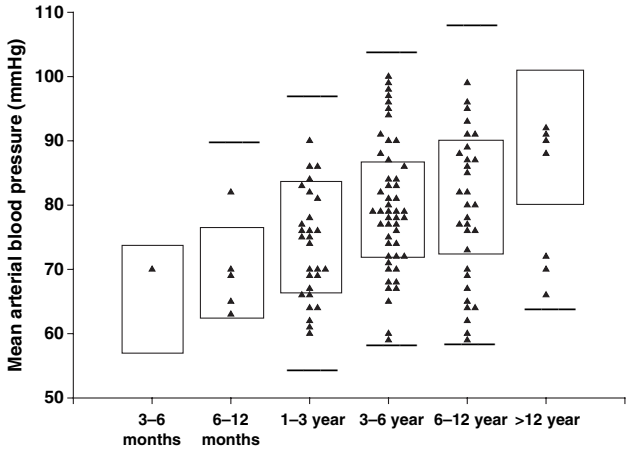

Figure 3
Distribution of mean arterial blood pressures (MAP) among 120 children who were below the age-specific normal range for heart rate (16). Boxes represent normal range and horizontal bars denote MAP values 20% below the normal range. All children were within 20% of the lower normal limits.

reliably provide sufficient sedation for pediatric MRI (19). This is consistent with our experience wherein the dexmedetomidine dosing protocol which had proved efficacious for CT imaging (10) did not provide consistently adequate sedation for MRI.

We have shown that the dosages of dexmedetomidine required to establish adequate sedation for MRI are surprisingly high, and significantly greater than those dosages that were approved by the Food and Drug Administration. For example, the dexmedetomidine dosing protocol which had been implemented successfully for CT imaging by our group proved to be inadequate for consistent success for MRI (10). As a result, a protocol utilizing higher doses of dexmedetomidine as the sole sedative agent for MRI evolved over the ensuing 2 years.

The hemodynamic effects of dexmedetomidine have been well described in healthy adults. There is a biphasic response with an initial increase in systolic blood pressure and a reflex decrease in heart rate followed by stabilization of heart rate and blood pressure below baseline (20). In adults and children, the incidence of significant bradycardia and hypotension is increased when dexmedetomidine is administered in conjunction with other medications which possess negative inotropic or chronotropic effects (21). Sinus arrest has been reported when dexmedetomidine has been administered in conjunction with potent anesthetic agents as well as with digoxin (21).

During dexmedetomidine sedation, the majority of children in our analysis maintained both a heart rate and a MAP that fell within age-specified norms (16). It is important to recognize, however, that age-specified norms represent children who are in an 'awake' state. There are no established MAP and heart rate norms for children during either natural sleep or medication-induced sedation. Our data suggest that the hemodynamic effects of dexmedetomidine on heart rate and MAP are similar to those seen when propofol or inhalation anesthetics are used alone to achieve anesthesia (22).

Although there was a 16% incidence of bradycardia during dexmedetomidine sedation, the majority of bradycardia patients had heart rates that fell within 20% of the age-adjusted values of 'awake' children (16). More importantly, during these periods of bradycardia, the MAP of these patients was always within 20% of age-adjusted values and the oxygen saturation was always between 95% and 100%. A fluctuation in blood pressure of 20% from normal 'awake' values is generally considered acceptable and well within the range that occurs during natural sleep. While the bradycardia seen in a small percentage of patients is important and potentially concerning, the overall effect on blood pressure does not appear significant. Although heart rate was relatively low, oxygen saturation was always well maintained. These patients were all carefully observed in recovery period and were discharged per routine according to our established guidelines utilizing a modified Aldrete score.

Our incidence of sedation failure with the initial attempts at lower dexmedetomidine doses is similar to that previously reported for dexmedetomidine MRI protocols in pediatric patients using a 1.0 $\mu g \cdot kg^{-1}$ bolus followed by an infusion of 0.5 $\mu g \cdot kg^{-1} \cdot h^{-1}$ (23,24). We have demonstrated that when dexmedetomidine is used as the sole sedative agent for acquisition of MRI studies, establishing a low failure rate requires higher dosages than previously reported. By using dexmedetomidine as the sole agent, one may reduce the need for adjuvant sedatives and thereby minimize the risk of critical adverse outcomes, particularly death and permanent neurological injury (25). In the event that pentobarbital is required as an adjuvant to the

dexmedetomidine, the dose of pentobarbital required to achieve sedation with dexmedetomidine is still less than that required when pentobarbital is used alone (8). In our patient population, the need for small doses of pentobarbital did not affect the adverse event rate, although it prolonged recovery time by approximately 1 h.

Currently, at our institution dexmedetomidine has replaced pentobarbital for all radiological imaging sedation and has received widespread approval by patients and parents, in particular, who appreciate the shorter recovery time and faster return to baseline neurological status. Parents, particularly those who have experienced the prolonged recovery times with pentobarbital or experienced the pentobarbital rage return specifically requesting dexmedetomidine for subsequent sedations. An advantage of dexmedetomidine is that its shorter recovery time allows coordination of same day visits with other specialty clinics, particularly oncology. Previously, following pentobarbital sedation for MRI, same day clinic appointments were not scheduled recognizing that neurologic assessments would be compromised by residual sedation.

When utilizing high dose dexmedetomidine as the sole sedative agent for pediatric MRI we have demonstrated that the potential for bradycardia must be anticipated. Although to date we have not observed any concomitant hemodynamic compromise or hypotension, we nonetheless recommend that a designated physician be available to treat hemodynamic instability should it occur. In addition, sedation providers must anticipate that the heart rate and blood pressure may deviate outside the established 'awake' norms. These decreases generally fall within 20% of normal values, and have not been associated with adverse outcomes in our experience.

We conclude from our analysis that dexmedetomidine can be used alone in doses significantly higher than previously reported to achieve sedation conditions which allow successful completion of pediatric MRI studies even for relatively long duration.

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