Dexmedetomidine Infusion for Routine Dental Management of an ASA IV Patient: A Case Report

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Anesthetic management of elderly patients requires numerous physiological considerations. With aging, degenerative changes occur in the structure and functional capacity of tissues and organs. Typically, these patients experience clinical effects with lower doses of medication. Important considerations for the geriatric populations following anesthesia include increased time to recovery and avoidance of functional decline. A case is reported in which an 83-year-old Caucasian female with a complex medical history presented for routine dental treatment under intravenous sedation via dexmedetomidine infusion.

Key Words: Dexmedetomidine infusion; Sedation for elderly; General anesthesia for elderly.

For decades, α -2 adrenergic receptor agonists have been utilized in medicine to treat a variety of conditions including hypertension, pain, panic disorders, opioid dependence, and alcohol withdrawal.¹ In recent years, these α -2 agonists have been explored as adjuncts for sedation and anesthesia. Dexmedetomidine, specifically, has been formulated for clinical use as a sole sedative agent or adjunct to reduce patient anesthetic drug requirements.² In the case presented, dexmedetomidine was selected to avoid the negative effects associated with the more commonly used propofol, while achieving an efficacious level of sedation. This case report presents the use of dexmedetomidine as the primary anesthetic agent, examining both its benefits and consequences of use, for repeated anesthetic management of a severely medically compromised patient for routine dental care.

CASE REPORT

An 83-year old Caucasian female presented at the University of Pittsburgh School of Dental Medicine for restorative dental treatment requiring multiple visits under intravenous sedation in the Center for Patients

with Special Needs. The patient had a medical history significant for cardiovascular disease, angina, atrial fibrillation, coronary artery disease, myocardial infarction, heart murmur, mitral valve prolapse with regurgitation, aspiration pneumonia, epilepsy, hemiplegia, cerebrovascular accident, polycystic kidney disease, and depression.

Medical History

The following narrative is adapted from the husband of the patient, a critical care physician.

In October 1987, the patient had a ruptured berry aneurysm of her right middle cerebral artery, which necessitated emergency neurosurgery with clipping of the aneurysm, approached through the temporal lobe, but which eventually led to complete destruction of the temporal lobe.

In the 1990s, the patient was diagnosed with obstructive cardiomyopathy with a loud systolic murmur over the outflow tract of the left ventricle based on malinsertion of the mural leaflet of the mitral valve. Transthoracic echocardiography confirmed mitral annular calcifications with a moderate mitral valve regurgitation in addition to a thickened aortic valve with associated mild aortic regurgitation. As a result, the patient experienced substantial hypertrophy of the left ventricle and at one time a gradient between the left ventricle and aorta of 100 mmHg.

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In February 2005, the patient had a transvaginal hysterectomy for a mild vaginal prolapse. Unfortunately, the patient was not placed on prophylactic postoperative anticoagulation therapy and on March 25 of the same year, the patient had a sudden ischemic stroke and developed left sided hemiplegia presumably from a mural thrombus in the same cerebral artery where she had her berry aneurysm in 1987. Echocardiography demonstrated atrial fibrillation with a flipping mural left atrial thrombus, suggesting the origin of the cerebral artery embolus. On March 30, the patient embolized the remaining left atrial thrombus into the left anterior descending coronary artery resulting in cardiogenic shock. With a systolic pressure of 20 mmHg, the patient was transported to a cardiac catheterization laboratory. Attempted thrombus removal with suction was unsuccessful. Emergency stent placement successfully reestablished flow to the left ventricular myocardium, but led to development of a large apical left ventricular aneurysm. While in shock, she received intravenous fluid and had a femoral central venous line. When later withdrawn, a pseudoaneurysm formed on the artery in that area and eventually ruptured days later with massive bleeding into the leg and hypovolemic shock necessitating emergency surgery to repair the artery. Resuscitation again proved successful.

In 2007, the patient had an additional stroke episode with worsening hemiplegia leaving the patient unable to walk or stand without assistance. Her condition continued to deteriorate leaving her sometimes disoriented and confused. Nonetheless, the patient continues to indicate positive quality of life, meeting friends and relatives, but is unable to cooperate for routine dental treatment. At the request of her husband, who holds medical power of attorney, the patient is referred to the dental school for care.

Preoperative Evaluation

The patient had a complete work-up by her primary care physician 2 months prior to the first sedative appointment. This included transthoracic echocardiogram, 12-lead electrocardiogram, and full set of laboratory testing (Table 1). At each individual sedation appointment, a medical history, with attention to any interval change and current medications, was obtained (Table 2). The patient and her husband denied any new onset headaches, decreased vision, sore throat, neck pain, shortness of breath, or chest pain at each sedation appointment. An anesthetic physical examination was performed at each visit. Breath sounds were bilaterally clear to auscultation, and heart sounds revealed

irregularly irregular rhythm consistent with atrial fibrillation at each appointment. Airway evaluation revealed Mallampati Grade II, good cervical range of motion, and thyromental distance > 6 cm. The patient was 154.9 cm (61 in) tall and weighed 54.4 kg.

Anesthetic Regimen

Multiple methods of anesthesia induction and airway management were considered in selection of anesthetic regimen for the patient. In view of her age and cardiovascular health history, anesthetic management was planned for minimal to moderate sedation with routine American Society of Anesthesiologists (ASA) monitors. A total of 4 sedation appointments were provided over the course of 8 months.

At the first sedation appointment, the patient was 2-person transferred to the dental operator chair. Patient's baseline vital signs were: blood pressure (BP): 150/92 mmHg left arm sitting (LAS); respiratory rate (RR): 17 breaths per minute; SpO₂: 95% with 3 L/min O₂ via nasal cannula; heart rate (HR): average of 82 beats per minute (bpm) irregularly irregular; and electrocardiogram with atrial fibrillation. Atrial fibrillation with irregularly irregular rhythm was present at each subsequent sedation. A 22-gauge peripheral venous cannula (PVC) was secured in the right antecubital fossa. The patient initially received 1 mg of midazolam and incremental doses of alfentanil beginning with 250 µg and totaling 625 µg over the course of the procedure. The patient appeared mildly to moderately resistant to treatment necessitating administration of propofol to a level of deep sedation. Propofol infusion was set at a rate of 35 µg/kg/min over 2.5 hours for a total of 365 mg. The patient then received 0.45 mL of 4% articaine with epinephrine 1 : 100,000 and 3.6 mL of 3% mepivacaine local anesthetic solution for restorative dentistry. The patient experienced intermittent airway obstruction requiring placement of a 30-Fr nasopharyngeal airway during the case with BP fluctuations from 170/100 mmHg to 100/70 mmHg and oxygen saturation variation from 88 to 95%. The patient emerged from anesthesia without incident. Total intravenous fluid intake was less than 300 mL of 0.9% sodium chloride and estimated blood loss was less than 5 mL, which was consistent with each of the following sedation encounters.

At the second sedation appointment, the anesthetic regimen was shifted to address previous airway maintenance difficulties by administration of dexmedetomidine infusion. The patient's initial vital signs were: BP: 145/90 mmHg LAS; SpO₂: 95% with 3 L/min O₂ via

Table 1. Preoperative Testing

	<i>Cardiovascular System</i>
Transthoracic echocardiogram	Final impressions: 1. Nondilated left ventricle with a small aneurysmal apex and mild to moderately decreased systolic function (ejection fraction 38%) 2. Wall thickness increased consistent with mild left ventricular hypertrophy 3. Thickened aortic valve with mild regurgitation 4. Mitral annular calcifications 5. No significant change from previous echocardiogram
12-lead electrocardiogram	Atrial fibrillation Evidence of anteroseptal myocardial infarction ST and T wave abnormality compatible with inferolateral ischemia No significant change when compared with previous EKG
Laboratory values	Basic metabolic panel Sodium: 135 mEq/L Potassium: 4.2 mEq/L Chloride: 103 mEq/L CO ₂ : 27 mEq/L BUN: 22 mg/dL Creatinine: 1.3 mg/dL Glucose: 107 mg/dL Calcium: 9.0 mg/dL Complete blood count WBC: $5.4 \times 10^3/\mu\text{L}$ RBC: $3.63 \times 10^6/\mu\text{L}$ Hemoglobin: 12.1 g/dL Hematocrit: 37.2% Platelet count: $311 \times 10^3/\mu\text{L}$ MCV: 102.5 fL MCH: 33.2 pg MCHC: 32.4 g/dL RDW: 19.3% Liver function tests Serum albumin: 2.8 g/L Total protein: 6.7 g/L Bilirubin, total: 0.7 mg/dL Alanine transaminase: 43 IU/L Aspartate aminotransferase: 34 IU/L

nasal cannula; HR: average 78 bpm; and RR: 14 breaths per minute. A 22-gauge PVC was secured in the right dorsal hand. The patient received 0.5 mg of midazolam and 2.5 μg of sufentanil at the beginning of the case with 1 $\mu\text{g}/\text{kg}$ loading dose of dexmedetomidine over 10 minutes followed by 0.5 $\mu\text{g}/\text{kg}/\text{h}$ maintenance dose totaling 94 μg over case duration of 1 hour 55 minutes. Patient sedation was supplemented with 5 to 10 mg incremental propofol boluses totaling 30 mg and 5 mg ephedrine. The patient experienced BP fluctuations from 145/95 mmHg to 80/63 mmHg with pressure elevations seeming to coincide with administration of 3.6 mL of 3% mepivacaine for restorative dentistry. The patient tolerated dentistry with mild to moderate sedation and emerged from anesthesia without incident.

At the third sedation appointment, an attempt was made to improve upon the patient's previous sedation level with primarily dexmedetomidine infusion. Patient's initial vital signs were: BP: 147/90 mmHg LAS; SpO₂:

95% with 4 L/min O₂ via nasal cannula; HR: 90 bpm; and RR: 14 breaths per minute. In recognition of the patient's past sedation experience, it was decided to eliminate midazolam from the anesthetic regimen. A 22-gauge PVC was secured in the dorsal right hand. The patient received 2 incremental doses of fentanyl totaling 37.5 μg in addition to a loading dose of dexmedetomidine 1 $\mu\text{g}/\text{kg}$ over 10 minutes followed by 0.5 $\mu\text{g}/\text{kg}/\text{h}$ maintenance dose over 1.5 hours for a total of 72.88 μg . The sedation was supplemented with propofol boluses totaling 30 mg and a total of 25 mg of ephedrine, dosed as necessary to maintain HR and BP. As with previous sedations, the patient's BP fluctuated from 165/100 mmHg to 95/45 mmHg over the course of the procedure. The BP variations did not coincide with administration of 3.6 mL of 2% lidocaine with epinephrine 1 : 100,000. The patient tolerated dentistry with mild to moderate sedation and emerged from anesthesia without incident. Oxygen saturation was

Table 2. Preoperative Evaluation

The patient is an 83-yr-old female presenting for restorative dental treatment under intravenous sedation.

Past medical history

1. Atrial fibrillation
2. Left atrial thrombus diagnosed in 2005 when she presented with a right MCA and left occipital stroke
3. Subarachnoid hemorrhage secondary to right MCA aneurysm status postclipping
4. Coronary artery disease status poststenting
5. Hypercholesterolemia
6. History of depression
7. Hypothyroidism

Social history

No history of tobacco, alcohol, or illicit drug use

Patient lives at home with husband who is a critical care physician and primary care taker

Allergies

No known drug allergies

Current medications

1. Amiodarone (Pacerone) 20 mg
2. Warfarin (Coumadin) 1 mg
3. Levetiracetam (Keppra) 1000 mg
4. Atorvastatin (Lipitor) 10 mg
5. Levothyroxine (Levothroid) 50 µg

Intended use

Arrhythmia
Atrial fibrillation
Seizures
Hypercholesterolemia
Hypothyroidism

maintained at 95% for the duration of the case with 6 L/min O₂ via nasal cannula.

At the fourth sedation appointment, the goal was to reproduce and improve upon the previous sedation technique. The patient's initial vital signs were: BP: 115/60 mmHg LAS; SpO₂: 97% with 2 L/min O₂ via nasal cannula; HR: 82 bpm; and RR: 17 breaths per minute. A 22-gauge PVC was secured in the right wrist. The patient received 25 µg fentanyl and 1 µg/kg loading dose of dexmedetomidine over 10 minutes followed by 0.5 µg/kg/h maintenance dose totaling 67.28 µg over case duration 1 hour 42 minutes. The patient was again supplemented with propofol boluses totaling 25 mg and 15 mg ephedrine in 5-mg doses as necessary to maintain HR and BP. No local anesthesia was administered for dentistry. The patient tolerated mild sedation with minimal fluctuations in BP and oxygen saturation. The patient maintained her airway and oxygenation with minimal respiratory depression and oxygen supplementation via nasal cannula. The patient emerged from anesthesia without delay or cognitive dysfunction.

DISCUSSION

As the human body ages, several physiologic changes occur in the cardiovascular, musculoskeletal, renal, and hepatic systems (Table 3). These changes will influence the effects of several anesthetic medications and must be considered with any anesthetic plan. Significant age-related changes within the cardiovascular system may negatively impact a patient's reserve and his or her ability to respond to perioperative stresses.³ Even in the absence of disease, the aging myocardium hypertrophies

leading to stiffer ventricles and reduced diastolic filling, known as diastolic dysfunction.⁴ Once this process is initiated, the heart begins to rely more heavily on the Frank-Starling mechanism to maintain adequate cardiac output. This in turn places significant importance on optimizing fluid management and maintaining adequate preload.⁵ As a result of connective tissue stiffening in the venous walls, the heart is required to compensate frequently for varying preloads.⁴ With elderly patients, compensation for hypovolemia places additional stress on the myocardium. Connective tissue and smooth muscle growth within muscular arteries thicken the vascular walls, narrow the lumen, and demand greater systolic pressures. To overcome the systolic hypertension, the heart contracts more forcefully,¹ which leads to an increased myocardial oxygen demand further stressing the heart and coronary arteries supplying cardiac tissues. Coronary artery disease coupled with less compliant ventricles places the heart at risk of an ischemic event.⁵

Additional physiologic changes experienced with advanced age place individuals at increased risk during sedation. The normal baroreceptor reflex that provides autoregulation of BP is diminished resulting in poor cardiovascular compensation in the setting of hypotension.⁴ Furthermore, decades of β receptor stimulation on cardiac tissue may desensitize these receptors in an elderly heart and affect its ability to respond during sympathetic stimulation.⁵ Therefore, β receptor stimulation by either endogenous or exogenous catecholamines may generate a reduced chronotropic and inotropic response by the heart.⁴ During sedation, cardiovascular compromise may be less sensitive to pharmacologic intervention. In this case, there was

Table 3. Physiological Changes in the Aging Individual: Pharmacologic and Anesthetic Implications*

<i>Physiologic Changes</i>	<i>Organ System Change</i>	<i>Pharmacokinetic and Pharmacodynamic Changes</i>	<i>Affected Medications</i>	<i>Anesthetic Management</i>
Drug distribution	↑ Body fat ↓ Total body water ↓ Cardiac output	↑ Volume of distribution → ↑ duration lipophilic drugs ↑ Peak drug concentration → ↑ potency hydrophilic drugs ↑ Circulation time → ↑ time to hypnosis	Benzodiazepines, opioids, propofol, dexmedetomidine, inhaled anesthetics Ephedrine	Limit redosing, turn off infusions early Consider dose reduction
Plasma binding protein	↑ α-1 glycoprotein	↑ Binding of basic, neutral drugs → ↓ free drug, ↓ potency	Propofol, etomidate	Anticipate delayed hypnosis, emergence
Drug metabolism	↓ hepatic blood flow → ↑ duration of metabolism	↑ metabolism of anesthetic drugs	Propofol, fentanyl, dexmedetomidine Opioids, ketamine, benzodiazepines, dexmedetomidine	Anticipate reduced initial effect Anticipate limited duration (in patients without liver impairment)
Drug elimination	↓ Renal blood flow and Function	↓ Clearance of renally eliminated drugs → ↑ duration	Opioids, muscle relaxants	Limit redosing
Drug sensitivity	Cerebral atrophy → ↓ white matter, # neurons in brain	↑ Sensitivity to anesthetic drugs	Propofol	Reduce dose by ~50%
		↓ Receptor sites – GABA, NMDA, mu, kappa	↑ Sensitivity to sedatives, benzodiazepines, opioids, especially remifentanyl	Reduce dose

* Aurimi L, White PF. Anesthesia for the elderly outpatient. *Curr Opin Anaesthesiol.* 2014;27:563–575.

Table 4. Dosing Guidelines for Dexmedetomidine (Precedex) Infusion for Procedural Sedation*

<i>Patient Classification</i>	<i>Loading Dose</i>	<i>Infusion</i>
Adults	1 µg/kg over 10 min	0.6 µg/kg/h Titrate to targeted level of sedation with doses from 0.2 to 1 µg/kg/h
Adult undergoing less invasive procedure	0.5 µg/kg over 10 min	0.6 µg/kg/h Titrate to targeted level of sedation with doses from 0.2 to 1 µg/kg/h
Patients over 65 years of age	0.5 µg/kg over 10 min	A dose reduction should be considered
Adults with impaired hepatic function	A dose reduction should be considered	A dose reduction should be considered

* Precedex dosing for procedural sedation. Available at: https://www.hospira.com/en/images/EN4003_tcm81-92504.pdf. Accessed March 20, 2016.

potential concern with dexmedetomidine and central α -2 effects promoting bradycardia and hypotension.

In addition to affecting the cardiovascular system, the aging body becomes increasingly sensitive to anesthetic medications due to musculoskeletal, hepatic, and renal changes. Atrophy of the musculoskeletal system leads to loss of lean muscle and accumulation of body fat. With a larger lipid reservoir, lipophilic anesthetic agents have a larger volume of distribution, increased β half-life elimination, and a potentially prolonged clinical effect. Conversely, total body water decreases, which reduces the central volume of distribution for water soluble drugs.³ Although the majority of anesthetic agents are highly lipid soluble, commonly used sympathomimetic drugs are more hydrophilic and their administration must be considered as well.⁶

The hepatic system, chiefly responsible for metabolism and inactivation of the anesthetic agents, undergoes relatively fewer changes. A healthy, though aged liver has reduced amounts of functional parenchymal tissues; however, enzymatic biotransformation remains unimpaired.⁴ Bradycardia is responsible for decreased hepatic blood flow per unit time, slowing drug metabolism further, particularly for drugs with high hepatic extraction ratio.⁵ Also, production of the plasma binding protein albumin decreases with age.³ Because albumin is the main binding protein for acidic drugs, reduced albumin production leads to less binding and an increased proportion free drug in the plasma. Poor nutrition can exacerbate the decreased albumin levels, further increasing the proportion of unbound, free drug.³ Conversely, the level of α -1 glycoprotein, the plasma binding protein for basic and neutral drugs, increases with age.⁵ Therefore, drugs such as fentanyl, propofol, and dexmedetomidine may have lower unbound concentrations and a reduced potency upon initial administration.^{7–9}

Renal function becomes increasingly impaired with age and presents more challenges to anesthesiologists. Kidney mass, glomerular filtration rate, and renal blood

flow all decrease with age.⁵ As renal function declines, tubular secretory function is impaired leading to poorer elimination of renally excreted drugs and their metabolites.^{3,10} Poor sodium regulation may also create periods of dehydration or fluid overload, both poorly tolerated in the CHF patient.⁵

For this patient, the anesthetic agents were specifically chosen to achieve hypnosis, anxiolysis, amnesia, and analgesia. Propofol was initially selected as the hypnotic agent; however, several cardiovascular implications are associated with its use. Propofol decreases mean arterial pressure and impairs the carotid sinus baroreceptor reflex, a homeostatic mechanism already compromised in elderly patients. Propofol also prevents sympathetic vasoconstriction, which lowers the systemic venous resistance and preload. Variable preload and fluctuations with venous return are further exacerbated by dehydration, which should be considered with established anesthesia guidelines dictating no clear liquids for at least 2 hours prior to the sedation.⁴ Additionally, due to a decrease in central white matter, number of neurons, and active binding sites for the drug, a dose reduction by 40–50% in a patient over 65 years old is recommended to achieve the comparable level of sedation.³ Where propofol interacts centrally, it demonstrates a longer context sensitive half-life in the aged patient. With lengthier sedations requiring longer infusion times, emergence is likely to be delayed.⁴

Midazolam was administered in the first 2 sedations to provide anxiolysis, amnesia, and sedation. Though an effective adjunct for sedation, several considerations must be made when using benzodiazepines, such as midazolam, in elderly patients. Changes within the aging brain show a decreased number of GABA receptors.³ Since the elderly patient is already predisposed to postoperative cognitive dysfunction, such as deficits in memory, information processing, and executive function, midazolam must be used cautiously as it can potentiate the risk.⁴ The enzymatic conversion of midazolam to its active metabolites by the liver may

be reduced by roughly 30% given the loss of functional hepatic tissue and slower circulation time.⁴ Additionally, the kidneys are less efficient at secreting the conjugated metabolites of midazolam, each with 20–30% potency of the parent drug.^{4,5} Given its lipophilic nature at physiologic pH, the increased volume of distribution and its decreased elimination, midazolam has a quick onset of action, greater potency following an intravenous bolus, and a prolonged effect.⁴ Therefore, midazolam has the potential to cause prolonged sedation and interfere with the postoperative cognitive assessment.

For the patient presented in this case, midazolam was used as part of the first 2 sedations, but given as small, single doses to a patient without previous history of renal compromise. Normal renal function was confirmed by pre-existing laboratory testing always within 6 months of each sedation appointment including standard electrolytes, calcium, phosphorus, blood urea nitrogen, creatinine, and glucose. The patient was also carefully monitored and observed following the procedure. Prior to discharge, the patient was further evaluated and determined to be awake, alert, and oriented with stable vital signs appropriate with preanesthetic baselines. However, subsequent sedations eliminated the use of midazolam to avoid potential respiratory depression and postoperative cognitive dysfunction associated with the patient's increasing age.

Alfentanil, fentanyl, and sufentanil were used for initial analgesia and as additional adjuncts to the sedation. However, intraoral administration of local anesthetic served as the primary analgesic. With decreasing numbers of central nervous system neurons and opioid receptors, the elderly have increased sensitivity to opioids.³ These changes cause alfentanil, fentanyl, and sufentanil to become twice as potent in the aged patient.⁵ Interestingly, because the pharmacokinetics of these opioids does not fluctuate with age, the duration of action and elimination half-life should remain unchanged.⁴ Renal implications are less important with fentanyl and its synthetic phenylpiperidine derivatives. The majority of the dose is inactivated in the liver via glucuronidation while less than 7% of the drug remains unchanged when excreted by the kidneys.⁷

Dexmedetomidine

The α -2 agonist, dexmedetomidine, has been shown to serve as an adjunct to procedural sedation with its analgesic and sedative properties while avoiding respiratory depression. For this medically compromised patient, there were several goals of perioperative

management using dexmedetomidine as the primary sedative agent. The primary goal was to provide a safe, moderate sedation while maintaining adequate anxiolysis and analgesia. Based on the distribution of α -2 receptors in the medial dorsal pons and the dorsal horn of the spinal column, dexmedetomidine inhibits post-synaptic neuronal signaling, providing sedation, hypnosis, and analgesia, respectively. Additionally, this highly selective α -2 agonist has been shown to reduce concomitantly administered opioid and benzodiazepine component by 50–75% and 80%, respectively. In an elderly individual where both opioids and benzodiazepines already have increased potencies and are associated with negative side effects, the equipotent dosages may be further reduced when used with dexmedetomidine. Lastly, with its relatively short elimination half-life and predicted context-sensitive half-time, shorter infusions of dexmedetomidine allow for faster return to baseline and subsequent discharge.¹²

Secondly, for this patient, the avoidance of respiratory depression or airway obstruction was preferred. The patient required supplemental oxygen of 2–4 L delivered through nasal cannula at baseline to maintain an initial peripheral capillary oxygen saturation of 95–97%. Any airway obstruction could potentially compromise her ability to adequately exchange gases, increasing oxygen demand and necessitating introduction of an airway adjunct or advanced airway device for mechanical ventilation. Her ability to ventilate unimpeded was desirable given her advanced age, compromised cardiovascular system, and poor physiologic reserves.

Additionally, we aimed to mitigate the sympathetic stress response during the perioperative management given the patient's history of berry aneurysm rupture and left ventricular hypertrophy with left ventricular outflow tract obstruction. Through its analgesic properties, dexmedetomidine promotes stability and resistance to fluctuations in hemodynamics by preventing adrenergic stimulation both centrally and peripherally.¹² A sudden increase in BP could exacerbate an occult cerebral aneurysm or cause its rupture. According to Davies and Cousins,¹³ the hypertrophic heart with left ventricular outflow tract obstruction will benefit from a stable systemic vascular resistance, maintenance of preload, and a reduction in sympathetic activity during an anesthetic. Dexmedetomidine may protect against significant deviations in BP and HR, and lessen the chronotropic and inotropic demands placed on the atherosclerotic heart. However, some caution should be exhibited with dexmedetomidine, especially on initial infusion or with rapid administration (1 μ g/kg in less than 10 minutes), which may result in transient hypertension via peripheral vasoconstriction followed

by hypotension and bradycardia via centrally acting α -2 agonism.¹²

The sedation of an 83-year-old woman with multiple comorbidities for an elective dental work carried inherent risk given her extensive cardiovascular and neurologic history. Over the course of 4 separate sedations, the anesthetic plan and technique was modified to reduce her perioperative risk and provide improved postoperative outcome.

The intent of the first sedation was to provide a moderate level of anesthesia with low-dose opioid and benzodiazepine administration. This did not produce the desired cooperation and necessitated a rapid change in level of sedation by the addition of propofol as the primary hypnotic agent. Though hemodynamic stability was maintained without vasopressor support, intermittent airway obstruction occurred as a consequence to the anesthetic agent requiring intraoperative management. Airway obstruction was rectified with a nasopharyngeal airway without incident; however, given the patient's history of warfarin use for chronic atrial fibrillation, any bleeding induced into the airway as a result of traumatic placement could have significant consequences.

For the second sedation, the anesthetic regimen was modified to address previous airway maintenance difficulties by administration of dexmedetomidine infusion. Small doses of opioid and benzodiazepine were again administered at the beginning of the sedation, but were coupled with dexmedetomidine infusion. An initial loading dose of dexmedetomidine was infused over the first 10 minutes allowing the patient to maintain her own airway without introduction of airway adjuncts or variations of peripheral oxygen saturation. Ephedrine was necessary to maintain normotensive mean arterial pressures, which transiently fluctuated below 20% of baseline. However, elevated mean arterial pressures associated with the sympathetic response to local anesthetic administration were mild, staying within 10% of baseline recordings.

The third and fourth sedations were similar both in the anesthetic regimen and patient hemodynamics. Midazolam was eliminated from the sedation allowing dexmedetomidine infusion to serve as the primary sedative with minimal use of opioids and propofol to maintain an adequate level of anesthesia. In contrast to the first sedation, small intermittent doses of ephedrine were necessary to maintain BP and HR; however, the patient was able to maintain her airway without manipulation. Furthermore, the patient emerged smoothly from each of the sedations without complication or cognitive dysfunction and was discharged home in the care of her husband.

The primary consequence of dexmedetomidine use in this patient was hypotension, necessitating the use of the vasopressor ephedrine to maintain BP. Ephedrine was selected as the therapy of choice for this patient as opposed to fluid bolus due to patient's history of myocardial dysfunction. As the dexmedetomidine worked centrally to prevent the release of norepinephrine, ephedrine provided direct stimulation to the cardiac and peripheral vascular adrenergic receptors and precipitated indirect norepinephrine release.⁶ As discussed previously, optimizing fluid management and maintaining adequate preload is important in elderly patients; however, correcting decreased cardiac output with significant fluid administration has potential to result in electrolyte disturbance, pulmonary congestion, and peripheral edema. The relative hypotension could further be explained by the higher loading dose of 1 μ g/kg used during the 3 infusions rather than the manufacturer suggested dose of 0.5 μ g/kg in patients over 65 years of age.¹⁴ However, a decreased loading dose may have warranted a higher maintenance dose to achieve the similar level of sedation or larger doses of sedation adjuncts such as fentanyl or propofol. Another potential adverse effect is bradycardia, which often accompanies hypotension or can present independently. The severity of these adverse effects is directly related to increasing doses of dexmedetomidine.¹² Modification of both the loading and maintenance dose to examine changes in hemodynamics or adjusting the dose of sedation adjuncts could be beneficial for future anesthetic management (Table 4). Unfortunately, the patient passed away late in 2015 of unrelated natural causes just 2 weeks prior to her next scheduled dental appointment.

CONCLUSION

When considering sedation for elective dental procedures in the nonoperating room setting, a number of risks and benefits must be weighed. Patients with multiple comorbidities and complex medical histories are at additional risk for complications and adverse outcomes. Patient age is a significant risk factor. The probability of death from any cause doubles every 10 years from 70 to 80 and 90 years old. In addition to age, the ASA classification, which stratifies a patient's risk based on total systemic disease, should be considered as risk increases with each higher ASA classification level.^{15–17} The patient discussed in this case report was ascribed an ASA IV physical status given her history of myocardial infarct, cerebrovascular accident, and lack of ambulation with hemiplegia.¹⁶ Although the patient presented was having a low risk dental surgical procedure, patient risk factors have been known to be

stronger predictors of mortality and perioperative complications than type of surgery.^{3,15} Therefore, prior to developing anesthetic management plans, comprehensive medical history and type of surgery must be considered collectively.

For 3 moderate sedations, dexmedetomidine served as the primary sedative agent with minimal secondary drug requirements. While its use did precipitate transient hypotension, larger doses of sedation adjuncts could similarly cause hypotension along with respiratory depression. By employing dexmedetomidine, airway obstruction and respiratory depression were minimized. Its sympatholytic properties helped prevent cardiovascular stimulation and reduce potentially threatening hypertensive episodes for a severely medically compromised octogenarian. With the success of dexmedetomidine infusion for this patient, its use as a sole sedative agent for medically compromised individuals undergoing elective dental procedures may be considered.

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