Dexmedetomidine Infusion as an Adjunct Anesthetic for Tetralogy of Fallot Repair During a Pediatric Cardiac Mission Trip in Jamaica: A Case Report

Kristen L. Hiscox, CRNA, DNP

Dexmedetomidine was used as an adjunct anesthetic for an infant with tetralogy of Fallot (TOF) who underwent complete surgical repair during a mission trip in Jamaica. Anesthetic maintenance was achieved with the concomitant use of dexmedetomidine and remifentanil infusions, as well as inhalational sevoflurane. The dexmedetomidine infusion ranged from 0.3 to 0.5 μg/kg/h and the remifentanil infusion ranged from 0.5 to 2 μg/kg/min, with end-tidal sevoflurane ranging from 0.8% to 6%. The continuous infusion of dexmedetomidine in a complex pediatric cardiac surgical patient provides sedation, decreases the need for narcotics and volatile agents, while also providing improved hemodynamic stability.

This report includes a review of the anatomy and pathophysiology of tetralogy of Fallot, medical and surgical treatments, anesthetic management, as well as global health issues involved in caring for complex cardiac patients in this underserved population. The expertise and dedication of medical mission professionals ensures that children in developing Caribbean countries receive life-saving heart surgery that would otherwise not be available. Collaboration between pediatric cardiac surgery programs in the United States and developing programs in the Caribbean is vital to the future of a self-sustaining cardiac program that will provide the knowledge and resources to care for these complex cardiac patients.

Keywords: Cardiac surgery, dexmedetomidine, pediatric, tetralogy of Fallot.

Dexmedetomidine is an intravenous (IV), centrally acting α₂ selective receptor agonist approved by the US Food and Drug Administration in 1999 for short-term (<24 hours) sedation in mechanically ventilated patients. Although the initial usage of dexmedetomidine was for intensive care unit (ICU) sedation for intubated patients, this unique sedative agent is being widely used in other patient care areas. Dexmedetomidine is being used as a general anesthetic, especially for total IV anesthesia cases, as well as a postoperative sedative/analgesic in postanesthesia care units. Several beneficial physiologic effects have been demonstrated including sedation, anxiolysis, analgesia, a decrease of the minimum alveolar concentration of inhalational anesthetics, decreased renin and vasopressin levels leading to diuresis, lowering of heart rate and blood pressure, and blunting of the sympathetic nervous system response to surgery. Dexmedetomidine use in pediatrics has expanded to include prevention of emergence delirium, to facilitate invasive and noninvasive procedural sedation, to facilitate radiologic procedural sedation, as well as in the management of opioid withdrawal.

Dexmedetomidine is a unique sedative that does not work by the γ-aminobutyric acid (GABA) system, the mechanism common with other sedatives, such as propofol and benzodiazepines. Unlike other commonly used sedatives, dexmedetomidine provides analgesia, in addition to its sedative and anxiolytic effects. The analgesic actions of dexmedetomidine have been examined but are not well understood and need to be further researched. As a sedative agent, its mechanism of action is similar to clonidine, another central α₂ agonist (Figure 1). Dexmedetomidine is shorter acting than clonidine, and therefore easier to titrate as an infusion. Dexmedetomidine has an affinity for α₂ receptors vs α₁ receptors of 1,620 to 1, approximately 8 times that of clonidine. Because of this unique property, dexmedetomidine produces sedation and analgesic effects without respiratory depression, making this drug an excellent choice for pediatric cardiothoracic surgery when these patients are extubated in the operating room or early on in the ICU.

Dexmedetomidine exhibits a linear relationship between dose and plasma concentration in the dosing range of 0.2 to 0.7 μg/kg/h; therefore, increasing the dose should result in proportional increases in its effects. The relatively short distribution half-life (t½ α) of about 6 minutes results in rapid onset and an elimination (t½ β) of approximately 2 hours facilitates clearance in a matter of hours. Dexmedetomidine has a steady-state
volume of distribution (Vss) of 118 L and a clearance of 39 L/h and is 94% protein bound to serum albumin and α1-glycoprotein (Table 1). It undergoes extensive hepatic metabolism through oxidation via the cytochrome–P450 system and direct glucuronidation in the liver, with its metabolites excreted by the kidneys. There is limited unchanged drug excreted in the urine or stool. The pharmacokinetics of dexmedetomidine are heavily affected in patients with severe liver failure, showing increased elimination and decreased clearance; therefore, the manufacturer does warn that lower doses be used in these patients. A pharmacokinetic study done in infants and children following surgery for congenital heart disease reported a median clearance of 27.2 mL/kg/min, peripheral volume of distribution of 2.5 L/kg, and terminal elimination half-life of 83 minutes. The results concluded that infants appear to clear dexmedetomidine more quickly than adults or older children. The recommended dosing for dexmedetomidine consists of a loading dose of 1 µg/kg over 10 minutes, followed by a continuous infusion of 0.2 to 0.7 µg/kg/h (Table 2). The adverse circulatory effects of dexmedetomidine include hypertension, hypotension, and bradycardia (Table 3). Dexmedetomidine shows a biphasic effect, with an initial increase in systolic blood pressure and a reflex decrease in heart rate, followed by a stabilization of systolic blood pressure and heart rate at values below the baseline. The activation of peripheral α2 receptors results in vasoconstriction and the initial increase in systolic blood pressure, whereas the eventual decrease in blood pressure and heart rate results from central presynaptic α2 receptors stimulated sympatholysis in the central nervous system, causing a decrease in norepinephrine release. This central nervous system effect is associated with a decrease in blood pressure. The decrease in heart rate may be caused by both a reflex response at the sinus node to peripheral vasoconstriction and the decrease in sympathetic outflow from the central nervous system. Bradycardia can be treated by the administration of atropine or glycopyrrolate. Hypertension may be a transient, initial circulatory effect after a loading dose of dexmedetomidine and also may be associated with continuous infusions resulting in high plasma concentrations. These adverse circulatory effects can be decreased by administering the loading dose over a longer period of time (20-30 minutes) or by not giving the loading dose and beginning the continuous infusion. Initiation of a maintenance infusion without the loading dose has been shown to achieve similar levels of sedation without the undesirable hemodynamic effects. Hypotension and bradycardia occur more commonly with the initial loading dose, with comorbid cardiovascular disease, and when administered concomitantly with medications that have negative chronotropic effects. During the maintenance infusion, titration at 30-minute intervals or greater than

Table 1. Pharmacokinetics of Dexmedetomidine

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rapid distribution (t1/2α)</td>
<td>6 min</td>
</tr>
<tr>
<td>Steady-state volume of distribution (Vss)</td>
<td>118 L</td>
</tr>
<tr>
<td>Clearance</td>
<td>39 L/h</td>
</tr>
<tr>
<td>Terminal elimination half-life (t1/2 β)</td>
<td>2 h</td>
</tr>
</tbody>
</table>

Table 2. Dexmedetomidine Dosing

**ICU sedation**

**Adult patients:**
- Loading dose of 1 µg/kg over 10 min
- Maintenance infusion generally initiated at 0.4 µg/kg/h
- Titrate to desired clinical effect with doses ranging from 0.2 to 0.7 µg/kg/h

**More than 65 years old/impaired hepatic or renal function:**
- A dose reduction should be considered

**Sedation for surgical or other procedures**

**Adult patients:**
- Loading dose of 1 µg/kg over 10 min
- Maintenance infusion generally initiated at 0.6 µg/kg/h
- Titrate to desired clinical effect with doses ranging from 0.2 to 1 µg/kg/h

**Adult patients undergoing less invasive procedures:**
- Loading infusion of 0.5 µg/kg given over 10 min may be suitable

**More than 65 years old/impaired hepatic or renal function:**
- A dose reduction should be considered

**Awake fiberoptic intubation:**
- Loading infusion of 1 µg/kg over 10 min
- Maintenance infusion of 0.7 µg/kg/h until the endotracheal tube is secured
30-minute intervals will help reduce the incidence of hypotension.

There are conflicting reports in the literature regarding effects on ventilatory functions. Some studies have demonstrated respiratory depression with mild increases of \( \text{Paco}_2 \) (4-5 mm Hg), decreased minute ventilation, decreased response to carbon dioxide challenge using carbon dioxide response curves, or upper airway obstruction following bolus doses \(^3\) (see Table 3). These findings, combined with the potential for transient upper airway obstruction may occur in patients receiving dexmedetomidine while spontaneously ventilating without use of an artificial airway, which necessitates that continuous monitoring and skilled anesthesia personnel be available for these patients.

Potential central nervous system adverse effects include changes in cerebral perfusion pressure and cerebral blood flow. \(^3\) Observed decreases in cerebral perfusion pressure are primarily mediated through decreases in systemic blood pressure and not in alterations in cerebral vascular resistance or intracranial pressure.

If medical professionals have concerns regarding the potential adverse effect profile of dexmedetomidine in critically ill pediatric patients, precautions should be used, including omitting the loading dose, using a lower loading dose infused over a longer period of time, and using a continuous infusion starting at the lower end of the recommended dosage range.

During pediatric cardiac surgery, the use of dexmedetomidine has been shown to reduce plasma concentrations of various perioperative hormonal stress responses. \(^8\) The use of a dexmedetomidine infusion intraoperatively will reduce the narcotic and volatile agent requirement. The perioperative administration of \( \alpha_2 \)-adrenergic ago-

Table 3. Potential Adverse Effects of Dexmedetomidine

<table>
<thead>
<tr>
<th>Cardiovascular</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bradycardia or sinus pause/arrest</td>
</tr>
<tr>
<td>Hypotension</td>
</tr>
<tr>
<td>Decreased cardiac output</td>
</tr>
<tr>
<td>Hypertension</td>
</tr>
<tr>
<td>Sinus arrhythmia</td>
</tr>
<tr>
<td>Decreased central sympathetic nervous system activity</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Respiratory</th>
</tr>
</thead>
<tbody>
<tr>
<td>Increased resting ( \text{Paco}_2 )</td>
</tr>
<tr>
<td>Decreased minute ventilation at rest and during ( \text{CO}_2 ) challenge</td>
</tr>
<tr>
<td>Irregular breathing pattern</td>
</tr>
<tr>
<td>Obstructive apnea</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Central nervous system</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ineffective analgesia/sedation</td>
</tr>
<tr>
<td>Paradoxic agitation</td>
</tr>
<tr>
<td>Decreased cerebral perfusion pressure (2° to lower mean arterial pressure)</td>
</tr>
</tbody>
</table>

Figure 2. Anatomic Findings in Tetralogy of Fallot

Abbreviation: VSD, ventricular septal defect.

Tetralogy of Fallot (TOF) is the most common form of cyanotic congenital heart disease and represents about 10% of cases of congenital heart disease. \(^9\) The anatomy of TOF comprises 4 components: large ventricular septal defect (VSD), overriding aorta, pulmonary stenosis, and right ventricular hypertrophy \(^10\) (Figure 2). The VSD is a large opening between the right ventricle and left ventricle. It is located in the membranous septum and extends anteriorly to the subaortic area, creating varying aortic override. The VSD is usually large enough to be unrestrictive, allowing equalization of right ventricular and left ventricular pressures. Rarely, the VSD is restrictive, producing suprasystemic pressures in the right ventricle. Pulmonary stenosis is narrowing of the pulmonary valve and right ventricular outflow tract (RVOT), which is the area below the valve that creates an obstruction or blockage of blood flow from the right ventricle to the pulmonary artery. The aortic valve is enlarged and appears to arise from both the left ventricle and right ventricle (overriding), instead of only the left ventricle, as in normal anatomy. There is thickening of the muscular walls of the right ventricle occurring because the right ventricle is pumping at high pressures due to the pulmonary stenosis present.

The combination of RVOT obstruction and VSD causes intracardiac right-to-left shunting. The shunting
results from interplay among the RVOT obstruction, systemic pulmonary vascular resistance (SVR), and to some degree pulmonary vascular resistance (PVR). Desired hemodynamic changes for a VSD (right-to-left shunting) include normal preload, decreased PVR, increased SVR, normal heart rate, and normal contractility. The degree of systemic arterial desaturation is dependent on the amount of shunting. Additional pulmonary blood flow, such as from a patent ductus arteriosus, aortopulmonary collateral vessels, and surgically created aortopulmonary shunts can also influence systemic arterial saturation. The RVOT obstruction can be dynamic, depending on the degree of muscular infundibular narrowing. The obstruction can increase with increasing circulating catecholamines, whether endogenous or administered. With decreases in SVR, the right-to-left shunt increases, producing more cyanosis.

The loud systolic murmur typical in infants originates from the dynamic narrowing of the RVOT. The direction of flow through the defect depends on the severity of the obstruction of the RVOT. If the obstruction is severe, or there is atresia, a large right-to-left shunt with low pulmonary blood flow and severe cyanosis will be present at birth. Most patients have adequate pulmonary blood flow at birth but develop increasing cyanosis during the first few weeks and months of life. Over time, the degree of RVOT obstruction progressively increases; therefore, the child may become more cyanotic with time. Chronic cyanosis results in increasing red blood cell mass in an attempt to maintain oxygen-carrying capacity, as well as clubbing of the fingers and toes. (“Clubbing” is a thickening of the flesh under the fingernails and toenails. The nail curves downward, similar to the shape of the round part of an upside-down spoon.)

Patients with TOF can have hypercyanotic spells or “tet spells.” These spells are characterized by severe cyanosis, paroxysmal hyperpnea, and metabolic acidosis. Feeding, crying, or defecation can initiate these spells, and they usually resolve spontaneously or when the child is comforted. Spells are initiated by events that result in increased oxygen demand associated with decreasing arterial PaO₂ and pH and increasing PaCO₂. Hypercyanotic spells are caused by the reduction of pulmonary blood flow, due to spasm of the RVOT or reduction in SVR (hypovolemia), with resulting increased right-to-left shunting across the VSD. Infundibular muscle spasm, partly induced or worsened by catecholamines, can be a component of these spells. These hypercyanotic spells can be terminated by IV β-blockers and palliated in the longer term with oral propranolol. The propranolol decreases right ventricular hypercontractility and heart rate and increases SVR. Other treatments for hypercyanotic spells include administering 100% oxygen, morphine to reduce stress and hyperpnea, and the knee-chest or squatting position. The knee-chest or squatting position produces a calming effect, reduces systemic venous return, and increases SVR. This effect causes the reduced venous return of desaturated blood from the lower extremities resulting in decreased right-to-left shunting.

A single episode of a hypercyanotic spell is an indication for early surgical treatment, whether palliation with a shunt or complete repair. The natural history of untreated TOF is for the tet spells to decrease in frequency over the long term, presumably because of the physiologic adaptation to hypoxia. In countries with well-developed pediatric cardiac services, severe cyanosis, recurrent hypercyanotic spells, squatting, and other consequences of severely reduced pulmonary blood flow are now rare. The diagnosis is seldom delayed, and infants undergo palliative procedures or complete repair within the first few days, weeks, or months of life.

The anesthetic management of patients with TOF requires vigilant care with attention to detail. The anesthesiologist should select agents to maintain/increase SVR relative to PVR, to minimize right-to-left shunting. Alpha stimulation causes pulmonary vasoconstriction, whereas β stimulation causes vasodilation. (Factors that increase or decrease PVR are summarized in Table 4.) Vigorous hyperventilation without positive end-expiratory pressure is one of the most powerful tools available to decrease PVR. Small increases in PaCO₂ are associated with significant increases in resistance, thus it is imperative to maintain normocarbia, avoiding any hypercarbic periods. The avoidance of excessive myocardial depression that could potentiate right ventricular failure is extremely important in TOF patients, which is why minimal inhalational agent concentrations intraoperatively are preferred. It is important to keep the patient with TOF well hydrated. If the patient is polycythemic (hemoglobin levels averaging 15-20 g/dL), a prolonged nothing by mouth period may put the patient at risk for dehydration with increased viscosity and sludging, potentially leading to thrombosis or impaired organ perfusion. Hypovolemia will exacerbate the RVOT obstruction if the patient has infundibular stenosis. Maintaining euvolemia or even a little hypervolemia will help open the RVOT obstruction and improve pulmonary blood flow. Fibrinolysis and clotting abnormalities are common; therefore, the administration of clotting factors, as well as blood products, are frequently necessary to achieve hemostasis. Patients with TOF may be vulnerable to hypercyanotic spells during induction and emergence. Adequate sedation preoperatively, as well as adequate analgesia postoperatively, will minimize catecholamine release and the potential for hypercyanotic spells. If a hypercyanotic spell occurs while under anesthesia, the anesthesiologist should treat accordingly with volume (5% albumin or lactated Ringer’s solution), deepen the anesthetic, administer fentanyl and/or phenylephrine, and hyperventilate with 100% oxygen to decrease PVR.
The surgical repair of patients with TOF can either be palliative with the placement of a Blalock-Taussig (BT) shunt or definitive. Primary repair of TOF in infancy is preferred in most centers. Some centers will use palliation for small, cyanotic infants or for infants who eventually may require conduit repair because of pulmonary valve atresia or coronary anomalies preventing transannular patch-type repairs. Palliative surgery involves the creation of a surgical shunt between the innominate artery or the subclavian artery and the ipsilateral branch pulmonary artery (PA). This is a Gore-Tex graft that varies in size from 3 to 5 mm. The shunt can increase PA size, promoting improved hemodynamics following complete repair. These shunts provide palliation for compromised, intensely cyanotic infants, allowing these infants to stabilize physiologically as they grow in preparation for complete repair of their cardiac defect.

Definitive repair for TOF includes dissection and resection of the infundibular stenosis; visualization of the pulmonary valve and a valvotomy, if necessary; estimation of RVOT dimensions, with a decision of whether transannular patching is appropriate; closure of the VSD; and closure of any atrial septal defect (ASD) or patent foramen ovale (PFO). An ASD/PFO is sometimes left in patients as a “pop-off” valve for the hypertrophied right ventricle and RVOT obstruction. Advocates of early, complete repair point out the benefit of normalizing circulatory pathways at the earliest age possible and avoiding distortions of the pulmonary arteries that can potentially be induced by palliative shunts. Early 1-stage repair can minimize adverse effects of hypoxia, prevent organ damage, reduce the development of severe right ventricular hypertrophy and fibrosis, thus avoiding extensive right ventricular muscle resection, reduce ventricular arrhythmias, encourage the development of normal pulmonary vasculature, and optimize functional outcomes.

Case Summary
Baby boy JM was born October 10, 2008 and was admitted to the Bustamante Hospital for Children in Kingston, Jamaica, because of cyanosis and the discovery of a cardiac murmur. Echocardiogram showed TOF. JM had 3 admissions for cyanotic spells and aspiration pneumonia with the last admission being March 2009. This infant underwent placement of a BT shunt at 6 months of age in April 2009 by a mission team from the United States. The decision at that time to perform a BT shunt vs complete surgical repair was made because of the infant’s small size (2.5 kg). Since his initial surgery, he had been eating well and gaining weight, had minimal hypercyanotic spells, and was maintained with medications: furosemide, 5 mg twice daily; spironolactone, 5 mg twice daily; and aspirin, 81 mg once daily. At 1 year, 5 months of age, JM presented for complete surgical repair of his TOF. His weight is 8 kg, he has no known drug allergies, and has had nothing by mouth for 10 hours. The infant was not in any apparent distress, mucous membranes were pink and moist, and oxygen saturations were 95% on room air. His pulses were regular and he had a grade 3/6 murmur. Laboratory values were as follows: hemoglobin, 15.3 g/dL; hematocrit, 45%; platelets, 145,000/mm; white blood cell count, 8,500/µL; prothrombin time, 13 seconds; partial thromboplastin time, 22 seconds; sodium, 143 mEq/L; potassium, 4.2 mEq/L; chloride, 95 mEq/L; sickle cell, negative; and blood type, O positive. Electrocardiogram showed a rightward QRS axis deviation, right atrial enlargement, and right ventricular hypertrophy. Echocardiography showed the following: left ventricular ejection fraction of 63%, normal coronary artery anatomy, dilated right atrium, intact atrial septum, 2 atroventricular valves, membranous ventricular septal defect, hypertrophied right ventricle, moderate infundibular stenosis of the right ventricular outflow tract, confluent, good size pulmonary arteries, and a patent BT shunt showing flow to the pulmonary artery. This patient was given an ASA physical status score of 4.

Once in the operating room, standard ASA monitors were placed. A mask induction was performed with oxygen, nitrous oxide, and 6% sevoflurane. After induction, 2 peripheral 22-gauge IV lines were placed, one in the left saphenous vein and one in the right hand. To facilitate endotracheal intubation, 8 mg of rocuronium was given intravenously. The infant was intubated orally.
with a 4.0-cuffed endotracheal tube and placed on the ventilator with a maintenance anesthetic of oxygen, air, and 2% sevoflurane. A 22-gauge left radial arterial line was placed, as well as a 4-French, 5.5-cm right internal jugular double lumen central venous catheter. Maintenance IV fluids of Plasma-Lyte were used during the case. A remifentanil infusion was started at 0.5 µg/kg/min and a dexmedetomidine infusion was started at 0.3 µg/kg/min through the central venous catheter. A bolus dose of 5 µg/kg of fentanyl was given intravenously before surgical incision. During the case, the remifentanil infusion ranged from 0.5 to 2 µg/kg/min, the dexmedetomidine infusion ranged from 0.3 to 0.5 µg/kg/h, and the exhaled concentration of sevoflurane ranged from 0.8% to 3%. These infusions were used intraoperatively to decrease the exhaled concentration of sevoflurane (potent myocardial depressant), while also providing sufficient control of blood pressure and response to surgical and painful stimuli. Both infusions were titrated to desired effect and were adjusted based on hemodynamic stability. The remifentanil and dexmedetomidine infusions were continued throughout the surgical procedure, including while the patient was on cardiopulmonary bypass. The cardiopulmonary bypass machine in the operating room does not have an inhalational anesthetic vaporizer (as do the ones in the United States); therefore, the continuous infusions of remifentanil and dexmedetomidine are necessary to keep the patient anesthetized and the blood pressure within a normal range while on cardiopulmonary bypass. Complete surgical repair was achieved with the following: dissection and resection of the infundibular stenosis; visualization of the pulmonary valve with a valvotomy; RVOT dimensions were estimated with dilators and a transannular patch was placed; and pericardial patch closure of the VSD was completed.

Surgical repair was achieved without complications and the patient came off cardiopulmonary bypass successfully without hemodynamic instability. Hemostasis postcardiopulmonary bypass was achieved with the transfusion of blood products, including platelets, fresh frozen plasma, and packed red blood cells. The total volume of IV fluids and blood products used throughout the case were Plasma-Lyte, 375 mL; platelets, 80 mL; fresh frozen plasma, 225 mL; and packed red blood cells, 300 mL. During sternal closure, sevoflurane was decreased to 1%, and ventilator settings were minimized to facilitate spontaneous breathing. After the patient was spontaneously breathing, dexmedetomidine was titrated to 0.5 µg/kg/h. During skin closure, the remifentanil infusion was discontinued. Upon completion of skin closure, the sevoflurane was discontinued. The patient had adequate respiratory effort with an oxygen saturation of 99% and was successfully extubated in the operating room. The dexmedetomidine infusion remained at 0.5 µg/kg/h, and the patient was taken to the cardiac ICU. The dexmedetomidine infusion continued at 0.5 µg/kg/h in the cardiac ICU overnight and into postoperative day 1, providing sedation and comfort. The dexmedetomidine infusion was discontinued late in the afternoon on postoperative day 1.

Discussion

The use of anesthetic adjuncts, such as dexmedetomidine, an α₂-adrenergic agonist mainly used for sedating mechanically ventilated patients in the ICU, is a recent intraoperative technique being used in cardiothoracic surgery. This drug has become increasingly popular because of its ability to (1) block the sympathetic stress response, (2) decrease anesthetic and analgesic requirements, (3) decrease presence of respiratory depression, and (4) provide a low and predictable side-effect profile.13 There is support of the safety and benefit of dexmedetomidine in managing pediatric cardiac patients, as evidenced by the increasing use of this drug by providers who care for pediatric cardiac patients.2,3,8 Conversely, some anesthesia providers still question the use of this drug in children, mainly because of the lack of pharmacologic data, US Food and Drug Administration approval, and the limited understanding of the adverse effects. With the lack of adequate randomized controlled trials to determine the clinical usefulness of a particular drug, medical professionals have the choice of using the drug or excluding it from clinical practice.

Dexmedetomidine is available as a 2 mL, 100-µg/mL solution that can be diluted in 50 mL of normal saline solution, providing a 4-µg/mL solution that can be administered either peripherally or centrally. The average wholesale cost of dexmedetomidine is about $55 per vial. Given the favorable sedative and anxiolytic properties, combined with its limited side-effect profile, there is growing interest in the use of dexmedetomidine in the pediatric population in various clinical scenarios (Table 5).

The pediatric population has a pressing need for improvements in pediatric pain management, and the collective clinical and published data on the use of dexmedetomidine in pediatrics provides a strong argument for its inclusion in this population. Reducing or eliminating a loading dose, administering the loading dose over a longer period of time, or by starting the continuous infusion at a lower rate, can avoid most of the adverse effects of dexmedetomidine. There is a definite need for future studies in pediatrics evaluating the short-term and long-term benefits of using dexmedetomidine as a routine perioperative anesthetic adjunct in infants and children with congenital heart disease.

Since the first procedures in the 1950s, advances in the diagnosis, medical management, preoperative care and surgical treatment, and postoperative care has ensured that almost all children born with TOF can now expect to survive into adulthood. Most also have a relatively
normal childhood. The remarkable progress in the care of children with TOF exemplifies the collaboration between pediatric cardiologists and congenital pediatric cardiac surgeons, the development of new techniques to address problems as they emerge, and the continuous evolution of management that have characterized care of all patients with congenital heart defects in the last 60 years. The care of children with TOF and their transition to adult life has been one of the successes of modern medicine.

The rise in congenital heart disease throughout the Caribbean is especially visible among the indigent children in Jamaica. Jamaica has an increasing list of infants and children with congenital heart disease issues requiring complex medical and surgical care. The waiting list for surgery, as well as for those awaiting evaluation, needs immediate attention and action. Without life-saving cardiac surgery, many of these children will die at a young age. Mission trip volunteer professionals are strong advocates for children's healthcare, having seen the challenges that these children and their families face. Collaboration among these professionals can help build strong communities while providing safe, quality medical care to these children who would otherwise not have access to this life-saving surgery. Continued collaboration between pediatric cardiac surgery programs in the United States and developing programs in Jamaica is essential to reduce the mortality rate of indigent children with heart disease, to facilitate and encourage a process of early detection and treatment of heart disease, as well as to advocate and share expertise for the future of cardiac surgery in developing Caribbean countries.

REFERENCES


AUTHOR

Kristen L. Hiscox, CRNA, DNP, is a staff nurse anesthetist at All Children’s Hospital in St Petersburg, Florida. The author was a student in the Doctorate of Nursing Practice Program at the University of South Florida, Tampa, Florida, when this article was written. Email: khiscox@health.usf.edu.