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Case report
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Dexmedetomidine-facilitated anaesthesia in paediatric single ventricle physiology undergoing dental rehabilitation: two case reports

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Abstract

We describe 2 successful cases of children with single ventricle physiology (SVP) who underwent dental rehabilitation under general anaesthesia, a scenario that carries significant challenges. Both patients received intranasal dexmedetomidine as premedication, which provided effective anxiolysis, facilitated intravenous access, and contributed to perioperative haemodynamic stability. One patient was maintained on sevoflurane, while the other received total intravenous anaesthesia with propofol and remifentanil. In both cases, deep extubation was performed safely, aided by dexmedetomidine's sedative and sympatholytic properties and careful titration of anaesthetic depth. These cases highlight important anaesthetic considerations in SVP, including the role of dexmedetomidine as premedication, careful titration of anaesthetic agents to achieve haemodynamic goals, particularly in reducing pulmonary vascular resistance, the potential to omit muscle relaxants, goal-directed fluid therapy, and the importance of smooth extubation.

Keywords: deep extubation, dexmedetomidine, sevoflurane, single ventricle physiology, total intravenous anaesthesia

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Introduction

Single ventricle defects are a group of complex congenital heart diseases (CHD) characterized by the presence of a single functional ventricular chamber.[1-3] These include conditions such as hypoplastic left heart syndrome, tricuspid atresia, and double-inlet left ventricle. Some anomalies with 2 ventricles, such as unbalanced atrioventricular septal defects, are also functionally single ventricle due to structural limitations that prevent biventricular circulation.[3] Complete mixing of systemic and pulmonary venous return occurs at the atrial or ventricular level, and outflow tract obstruction is commonly present.[3] Standard surgical management involves staged palliative procedures including the bidirectional Glenn shunt and Fontan operation, aimed at optimizing the balance between pulmonary and systemic blood flow.[2-3]

Perioperative risk assessment is essential in this group. The American College of Surgeons National Surgical Quality Improvement Program (ACS NSQIP) classifies children with CHD into minor, major, and severe risk categories. Patients with single ventricle physiology (SVP) fall into the severe category, requiring anaesthesia management in tertiary centres with cardiac expertise and intensive care backup.[2]

These patients pose significant anaesthetic challenges due to limited cardiovascular reserve and dependence on passive pulmonary circulation.[1,2] Despite their complex cardiovascular physiology, these cases contribute uniquely by describing anaesthetic approaches for dental procedures in SVP, with a focus on intraoperative haemodynamic trends and perioperative management considerations.

Case presentation

Case 1

The first patient was a 5-year-old boy with tricuspid atresia, a small ventricular septal defect, and pulmonary stenosis. As part of staged palliation, he had undergone a balloon atrial septostomy in 2019, followed by a right bidirectional Glenn shunt, repeat atrial septostomy and pulmonary artery augmentation in 2021. His baseline oxygen saturation at home ranged from 75% to 80%. An echocardiogram performed in March 2024 confirmed a patent Glenn shunt.

He had recently been hospitalised for pneumonia requiring high-flow nasal cannula oxygen therapy with a 4-day stay. He was not on any anticoagulant or anti-failure medications. Preoperative haemoglobin was 17 g/dl and haematocrit 51%. The patient was kept nil by mouth for 6 hours for solids and allowed clear fluids up to 2 hours preoperatively. EMLA cream was applied to both hands in the ward

prior to transfer. Upon arrival at the operating theatre, intranasal dexmedetomidine 3 mcg/kg was administered as premedication and an intravenous cannula was successfully inserted 20 minutes later.

Standard American Society of Anesthesiologists (ASA) monitoring was applied, including dual pulse oximetry. Induction consisted of intravenous (IV) midazolam 0.1 mg/kg, IV ketamine 1.5 mg/kg, and IV fentanyl 2 mcg/kg, followed by rocuronium 1 mg/kg to facilitate nasal intubation. A 4.5-mm cuffed nasal RAE endotracheal tube was secured 16 cm at the nares. Antibiotic prophylaxis for infective endocarditis was administered preoperatively.

Anaesthesia was maintained with sevoflurane at 1.9% (minimum alveolar concentration [MAC] 0.7). Controlled ventilation was applied (tidal volume 6 ml/kg, PEEP 5 cmH_2O, Fio_2 0.35, $EtCO_2$ 35 mmHg). Intraoperative SpO_2 ranged 85%–88%.

Multimodal analgesia included IV morphine 0.1 mg/kg, IV paracetamol 15 mg/kg, rectal diclofenac 12.5 mg, and local infiltration by the dentist with mepivacaine 2% with adrenaline. IV dexamethasone 0.15 mg/kg was administered as an antiemetic.

Baseline vital signs were blood pressure (BP) 99/57 mmHg (mean arterial pressure [MAP] 71 mmHg), heart rate (HR) 96 bpm, and SpO_2 83%. Post-induction, BP transiently increased to 123/85 mmHg and HR to 114 bpm. Intraoperative vitals remained stable, with BP ranging 86–93/48–57 mmHg (MAP 61–69 mmHg), HR 88–95 bpm, SpO_2 86%–88%, and temperature 36°C. This represented a mild reduction in MAP of approximately 10 mmHg below baseline, which was well tolerated without desaturation or signs of poor perfusion. Fluid management consisted of no maintenance infusion, and a total of 10 ml/kg Hartmann's solution given as replacement boluses. Total procedure time was approximately 1.5 hours.

Toward the end of the procedure, IV sugammadex 2 mg/kg was given to reverse neuromuscular blockade. Sevoflurane was discontinued while the patient was maintained on FiO_2 0.5 with ultra-low-flow anaesthesia to facilitate slow washout. At MAC 0.7, spontaneous respiration with adequate minute ventilation was observed. Anaesthesia was then transitioned to intermittent IV propofol boluses (0.5–1 mg/kg) to maintain depth for emergence. Deep extubation was done smoothly to a face mask at 5 l/min. He remained stable in the recovery area and was discharged to the ward on room air after 15 minutes. The perioperative course was uneventful.

Case 2

The second patient was a 9-year-old boy with autism spectrum disorder and pulmonary atresia with an intact ventricular septum and a unipartite right ventricle. He previously underwent balloon atrial septostomy and patent ductus arteriosus

stenting in 2015 and a bilateral Glenn shunt with pulmonary artery augmentation in 2019. An echocardiography in July 2024 confirmed a patent Glenn shunt.

He was not on any anticoagulant or heart failure medications. Preoperative haemoglobin was 15 g/dl, and haematocrit 44%. He was kept nil by mouth for 6 hours for solid food and allowed clear fluids up to 2 hours before the procedure. EMLA cream was applied to both hands before transfer to the operating theatre.

Upon arrival in the preoperative bay, the patient received intranasal dexmedetomidine 3 mcg/kg as premedication. After 20 minutes, IV cannulation was attempted but unsuccessful; therefore, inhalational induction with sevoflurane was commenced using incremental concentrations of 2% and 4% with standard ASA monitors applied.

Once IV access was secured, boluses of IV ketamine 2 mg/kg and IV remifentanil 1 mcg/kg were given. Smooth intubation was performed without muscle relaxants, facilitated by remifentanil. Target-controlled infusion propofol (Peadfusor model) was commenced at 5 mcg/mL, and remifentanil infusion at 0.2 mcg/kg/min. Nasal intubation was performed using a 5.5-mm cuffed RAE tube, secured 18 cm at the nares. Antibiotic prophylaxis for endocarditis was administered.

Remifentanil infusion was maintained at 0.1 mcg/kg/min intraoperatively, while target-controlled infusion propofol was titrated to 3.5 mcg/ml. Multimodal analgesia included IV morphine 0.1 mg/kg, IV paracetamol 15 mg/kg, and local anaesthesia mepivacaine 2% with adrenaline by the dental team. IV dexamethasone 0.15 mg/kg was given as an antiemetic.

Baseline vital signs were BP 98/61 mmHg (MAP 73 mmHg), HR 78 bpm, and SpO_2 84%. Post-induction, the patient's BP dropped by 20% to 68/38 mmHg (MAP 48 mmHg) and was managed with 2 boluses of IV phenylephrine (10 mcg each). Intraoperatively, vital signs remained stable: BP 70–80/44–46 mmHg (MAP 53–57 mmHg), HR 73–76 bpm, SpO_2 86%–89%, and temperature 36.3°C. Fluid management consisted of 10 ml/kg Hartmann's solution, administered as boluses.

Ventilation was maintained using pressure support ventilation with a tidal volume of 6 ml/kg, PEEP 5 cmH_2O, rate 18 bpm, FiO_2 0.35, and $EtCO_2$ target of 35–40 mmHg. The procedure lasted 1.5 hours. Propofol was discontinued approximately 20 minutes before the end of surgery, and remifentanil was discontinued at the completion of the procedure. This facilitated a smooth deep extubation and the patient was transitioned to a face mask at 5 l/min. He remained stable in the recovery area and was discharged to the general ward on room air. The perioperative course was uneventful.

Discussion

Children with SVP are categorised as severe risk within the ACS NSQIP framework.[2] Nonetheless, this risk is heterogeneous; those with well-compensated Glenn or Fontan physiology may be considered major risk for selected outpatient procedures, provided that thorough preoperative assessment and multidisciplinary planning are undertaken.[2]

Both cases presented practical challenges, including preoperative anxiety, behavioural difficulties, *e.g.*, autism, and lack of IV access. A tailored induction plan, such as the use of intranasal dexmedetomidine, provides the dual advantage of preoperative anxiolysis and reduced anaesthetic maintenance requirements.[4-6] It has an onset of 20–30 minutes and a duration of action up to 135 minutes. In both cases, its use as premedication decreased the doses of sevoflurane and total intravenous anaesthesia (TIVA) agents required, while its sympatholytic profile supported smoother induction and emergence without respiratory compromise.[1] Its growing utility in children with CHD is increasingly recognised in current literature.[1,2,5]

Induction strategies using ketamine remain a reliable induction agent for children with SVP, as it preserves heart rate, systemic vascular resistance, and cardiac output.[1,3] In contrast, propofol can cause vasodilation and hypotension, as seen in Case 2 with a 20% transient drop in MAP requiring vasopressor support (IV phenylephrine 1 mcg/kg) after induction. However, this drop in BP could also have been caused by initial sevoflurane induction for IV access placement. Nevertheless, with a carefully titrated TIVA regimen, propofol combined with remifentanil offers advantages of smoother emergence, haemodynamic predictability, and the possibility of avoiding muscle relaxants.[7]

In the TIVA case, smooth tracheal intubation was achieved without muscle relaxants by administering boluses of ketamine and remifentanil.[1,7] An IV remifentanil bolus of 2–4 mcg/kg following induction has been shown to provide optimal intubating conditions comparable to relaxant use. Intubation performed either after a reduction in heart rate or approximately 30 seconds post-administration of IV remifentanil.[8,9] No significant bradycardia or muscle rigidity was observed in our patient. Avoiding muscle relaxants preserved spontaneous ventilation, which is advantageous in Glenn physiology where passive pulmonary blood flow can be impeded by high intrathoracic pressures.

Maintenance of anaesthesia with sevoflurane, as in Case 1, is commonly employed in paediatric practice because of its familiarity, non-irritating profile, and controllable depth of anaesthesia.[3] However, sevoflurane produces dose-dependent vasodilation and reductions in systemic vascular resistance, which may

be poorly tolerated in SVP if not titrated carefully.[1,3] The use of a muscle relaxant can help limit sevoflurane requirements, thereby reducing the risk of significant systemic vascular resistance depression. At the same time, controlled ventilation with muscle relaxation may increase intrathoracic pressure and impede passive pulmonary blood flow through the Glenn shunt. Ventilation strategies such as applying an optimal PEEP of 4–5 cmH_2O, tidal volumes of 6–8 mL/kg, and targeting an end-tidal CO_2 of 35–45 mmHg is recommended.[1,2]

At our centre, children with SVP and Glenn shunt undergoing short dental procedures (< 2 hours) typically receive a single intraoperative crystalloid bolus of 10 ml/kg. Further fluid administration is restricted to this limit and guided by continuous haemodynamic monitoring. Euvolemia is essential in Glenn circulation, where pulmonary blood flow depends on passive venous return.[1,2] While IV maintenance fluids were traditionally started preoperatively to prevent dehydration, current practice favours allowing clear oral fluids until transfer to the operating theatre.[2,10] This strategy is particularly practical in neurodivergent children who may resist IV cannulation. Perioperative fluid management should be individualised: patients unable to maintain oral intake should receive IV fluids, whereas intraoperative management should prioritise goal-directed therapy.[1,2] Excessive fluid administration risks pulmonary congestion and impaired haemodynamics, underscoring the importance of tailored fluid strategies in this population.

Prophylaxis antibiotics should be given consistent with 2008 American Heart Association (AHA)/American College of Cardiology (ACC) guidelines, which recommend endocarditis prevention in patients with unrepaired cyanotic CHD or palliative shunt undergoing dental procedures involving gingival manipulation.[11]

Both awake and deep extubation techniques are recognised approaches in paediatric anaesthesia, each with distinct advantages and limitations. Deep extubation is often preferred in our practice, as it provides smoother emergence and greater haemodynamic stability, particularly in children with CHD.[12,13] This technique, however, requires experience and vigilance due to the potential risk of airway obstruction compared with awake extubation. [12,13]

TIVA offers advantages over volatile anaesthetics for deep extubation by allowing more precise titration of depth and smoother transitions.[7] In contrast, maintaining adequate spontaneous ventilation at a deep plane of anaesthesia with sevoflurane can be challenging.[3] Traditionally, a MAC above 1.0 is the target, along with established spontaneous ventilation, before proceeding with deep extubation.[12] In our cases, we adapted this recognised approach by discontinuing sevoflurane earlier and maintaining anaesthetic depth with intermittent propofol boluses (0.5–1 mg/kg). This adjustment promoted more reliable spontaneous ventilation

and facilitated a smoother extubation profile. Our experience aligns with emerging evidence that adjuncts such as dexmedetomidine or propofol can enhance the safety of deep extubation in paediatric patients with CHD.[13,14]

This case series illustrates that both volatile and TIVA-based anaesthetic techniques can be applied safely in children with SVP undergoing dental rehabilitation. The consistent factor across both cases was the use of intranasal dexmedetomidine, which provided effective anxiolysis, facilitated IV access, and contributed to perioperative haemodynamic stability. Its sedative and sympatholytic properties also supported smooth deep extubation, minimising the risk of haemodynamic surges and airway complications. These reports demonstrate dexmedetomidine's role as a versatile adjunct in this high-risk population, particularly in supporting anxiolysis and stable deep extubation, areas that remain underrepresented in the existing literature. Larger prospective studies are warranted to further evaluate its safety and efficacy in children with SVP undergoing non-cardiac procedures.

This report is limited by its small sample size (2 cases), which precludes generalisation of findings. Invasive haemodynamic monitoring (*e.g.*, arterial line, central venous pressure) was not used, restricting detailed physiological assessment. Long-term postoperative outcomes were not captured, and only immediate perioperative results are available. Additionally, as both cases were relatively short dental procedures, the applicability of these strategies to longer or more complex non-cardiac surgeries remains uncertain.

Conclusion

Children with SVP require proper perioperative planning with strategies to maintain preload, reduce pulmonary vascular resistance, and ensure smooth recovery for safe outcomes in this high-risk population. Both sevoflurane and TIVA can be used safely for short non-cardiac procedures. Intranasal dexmedetomidine shows promise as an anxiolytic, reducing anaesthetic requirements and facilitating smoother deep extubation.

Declarations

Informed consent for publication

Informed consent was obtained from the patients' legal guardians for the publication of the clinical data contained in this case report.

Competing interests
None to declare.

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