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Navigating the Nexus: Anesthetic Management of Craniotomy for Brain Abscess in a Pediatric Patient with Uncorrected Tetralogy of Fallot

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A B S T R A C T

Introduction: Tetralogy of Fallot (TOF) is the most prevalent cyanotic congenital heart disease, predisposing patients to brain abscesses via right-to-left shunting that bypasses pulmonary bacterial filtration. Anesthetic management for craniotomy in pediatric patients with uncorrected TOF and a concurrent brain abscess presents a formidable challenge, requiring meticulous integration of neuroanesthetic and cardiac anesthetic principles. Literature detailing comprehensive perioperative anesthetic strategies for this specific dual pathology remains scarce. **Case presentation:** An 11-year-old male with uncorrected TOF and a large left frontoparietal brain abscess with significant mass effect underwent emergent craniotomy and abscess evacuation. Preoperative echocardiography confirmed TOF with severe pulmonary stenosis and right-to-left shunting. Anesthetic induction was achieved with titrated ketamine and propofol, followed by fentanyl and rocuronium. Maintenance involved sevoflurane, oxygen-air mixture, and intermittent fentanyl and rocuronium, focusing on normovolemia, normocapnia to slight hypocapnia, and invasive hemodynamic monitoring. Phenylephrine was utilized for blood pressure support. The perioperative period was uneventful, with the patient experiencing no neurological or cardiac complications. **Conclusion:** This case underscores the critical importance of a tailored anesthetic approach, integrating neuroprotective strategies with meticulous cardiovascular management, in children with uncorrected TOF undergoing major neurosurgery. Comprehensive preoperative assessment, vigilant intraoperative monitoring, strategic pharmacological interventions, and a deep understanding of the complex pathophysiology are paramount to preventing cyanotic spells, managing intracranial pressure, and ensuring a successful outcome in this high-risk cohort.

1. Introduction

Tetralogy of Fallot (TOF) stands as the most frequently encountered cyanotic congenital heart disease (CHD), with an incidence of approximately 1 in 35,000 live births, constituting 7–10% of all congenital cardiac anomalies. This complex cardiac malformation is classically defined by a constellation of four anatomical defects: a ventricular septal defect (VSD), an overriding aorta that receives blood from both ventricles, right ventricular outflow tract obstruction (RVOT), and subsequent right ventricular hypertrophy (RVH). One of the significant extracardiac complications associated with uncorrected TOF is the development of brain abscesses, reported in 13–70% of such cases. The underlying mechanism involves the intracardiac right-to-left (R-to-L) shunt, characteristic of TOF, which allows venous blood containing bacteria to bypass the normal filtering function of the pulmonary capillaries and gain direct access to the systemic and cerebral circulation. Patients with uncorrected TOF undergoing surgical intervention for a brain abscess, particularly a craniotomy for abscess resection, present a unique and

substantial challenge to the anesthesiologist. The existing medical literature predominantly addresses the surgical or conservative management of brain abscesses in this patient population, with a notable paucity of detailed reports focusing specifically on the intricacies of perioperative anesthetic planning and management. The anesthetic care for these children necessitates a sophisticated, multidisciplinary approach that carefully integrates established neuroanesthetic principles, aimed at optimizing cerebral physiology, with a profound understanding of the complex pathophysiology of uncorrected TOF.[1-4]

The physiological milieu in children with uncorrected TOF is characterized by chronic hypoxemia and the presence of a significant R-to-L shunt. These baseline abnormalities render them exquisitely vulnerable to a host of perioperative complications. These include profound hemodynamic instability, cardiac arrhythmias, hypercyanotic episodes (often referred to as "Tet spells"), critical acid-base disturbances, and adverse neurological sequelae such as seizures, meningitis, and dangerous elevations in intracranial pressure (ICP). Anesthetic agents and techniques can profoundly influence both systemic vascular resistance (SVR) and pulmonary vascular resistance (PVR), thereby altering the magnitude of the R-to-L shunt and potentially precipitating life-threatening events. Furthermore, neurosurgical requirements, such as maintaining adequate cerebral perfusion pressure (CPP) and minimizing brain bulk, add another layer of complexity to the anesthetic management.[5-8]

The novelty of this case report lies in its detailed exposition of a successful, meticulously planned anesthetic strategy for an 11-year-old child with uncorrected TOF who presented with a large, space-occupying brain abscess necessitating urgent craniotomy. While isolated aspects of managing TOF or brain abscess surgery are documented, comprehensive reports detailing the integrated anesthetic approach for this specific high-risk dual pathology in the pediatric population are limited.[9,10] This report aims to contribute to this sparse body of knowledge by elucidating the specific pharmacological choices, monitoring techniques, and physiological goals that facilitated an uneventful perioperative course. We highlight the decision-making process involved in balancing competing demands: the need to reduce ICP and provide optimal neurosurgical conditions while simultaneously preventing deleterious increases in R-to-L shunting and avoiding cyanotic spells. The aim of this study is to describe and meticulously discuss the anesthetic considerations, perioperative management strategies, and specific interventions employed in an 11-year-old pediatric patient with uncorrected Tetralogy of Fallot undergoing emergency craniotomy for the resection of a large frontoparietal brain abscess.

2. Case Presentation

An 11-year-old male patient, weighing 16.5 kg, presented to Prof. Dr. Ngoerah General Hospital with a two-week history of persistent headache and fever. He was diagnosed with a left frontoparietal brain abscess and was scheduled for an urgent craniotomy and abscess resection. There were no reports of decreased consciousness, seizures, or visual disturbances upon initial presentation. The patient had a known history of congenital heart disease, specifically Tetralogy of Fallot, diagnosed at the age of two months; his cardiac condition was uncorrected. Clinically, he was generally asymptomatic at rest, without cyanosis or dyspnea, but experienced exertional dyspnea and cyanosis during episodes of crying or vigorous physical activity. During such episodes, the patient instinctively adopted a squatting position, which reportedly alleviated his symptoms – a classic sign associated with TOF aimed at increasing SVR and decreasing R-to-L shunting. He was born at full term with a normal birth weight via spontaneous vaginal delivery. His nutritional status assessment indicated moderate protein-energy malnutrition of the marasmic type, for which he was under pediatric management. Pre-admission medications included oral propranolol, and he had been initiated on intravenous antibiotic therapy consisting of ceftriaxone, vancomycin, and metronidazole by the pediatric department for the suspected brain abscess. A comprehensive summary of the patient's key clinical findings upon presentation and diagnosis is detailed in Table 1.

Table 1. Summary of patient's clinical findings.

Feature	Details
Demographics	11-year-old male, 16.5 kg
Presenting complaints	Two-week history of headache and fever
	No decreased consciousness, seizures, or visual disturbances
Past medical history	Uncorrected Tetralogy of Fallot (diagnosed at 2 months)
	Squatting episodes during crying/vigorous activity
	Full-term, normal birth weight, vaginal delivery
	Moderate protein-energy malnutrition (marasmic type)
Pre-admission medications	Oral propranolol, IV ceftriaxone, IV vancomycin, IV metronidazole
Physical examination	BP 90/60 mmHg, HR 90 bpm, RR 18/min, SpO_2 86% (room air)
	Alert, followed commands, no focal motor deficits
	Cardiac: Grade III murmur at 5th intercostal space
Echocardiography (TTE)	TOF: Severe pulmonary stenosis, pulmonary dysplasia, mild TR, mild MR, MAPCAs, overriding aorta, RVH, large malaligned perimembranous VSD (7.2–7.6 mm) with R-to-L shunt
Cranial CT scan	Large (5.2x5x4 cm) left frontoparietal abscess with vasogenic edema, mass effect on left lateral ventricle, 1.3 cm subfalcine herniation, rightward uncal herniation
Laboratory findings	Hb 14.2 g/dL, Hct 42.8%, WBC 21.97x10^3 µL, PLT 524x10^3 µL
	Coagulation, electrolytes, liver & kidney function tests within normal limits

On physical examination at the time of anesthetic consultation, his vital signs were: blood pressure 90/60 mmHg, heart rate 90 beats per minute (bpm), respiratory rate 18 breaths per minute, and oxygen saturation (SpO_2) of 86% while breathing room air. The central nervous system examination revealed that the patient was alert, oriented, and followed commands appropriately, with no discernible focal motor deficits. Cardiac auscultation identified a grade III/VI systolic ejection murmur at the left upper sternal border, consistent with his known cardiac condition. Diagnostic imaging, as summarized in Table 1, included a cranial Computed Tomography (CT) scan, which revealed a significant left frontoparietal abscess with extensive surrounding vasogenic edema and critical mass effect, including subfalcine and uncal herniation. Transthoracic echocardiography (TTE) re-confirmed the uncorrected Tetralogy of Fallot with features indicative of severe RVOT obstruction and a substantial right-to-left shunt through a large VSD. Routine preoperative laboratory examinations showed evidence of infection and polycythemia but otherwise normal major organ function.

The patient was prepared for surgery under general anesthesia and was classified as American Society of Anesthesiologists (ASA) Physical Status III due to the coexistence of a severe systemic disease (uncorrected TOF with a large intracranial abscess) and malnutrition. A 20-gauge intravenous (IV) line was already in place from the inpatient ward, confirmed to have adequate flow. Preoperative fasting guidelines were followed, and fluid requirements during this period were met with appropriate intravenous crystalloid solutions. The patient's volume status was further assessed non-invasively using ultrasound to evaluate inferior vena cava (IVC) collapsibility, which suggested euvolemia. Extreme care was taken with all intravenous administrations to ensure that no air bubbles were present in the infusion line, to mitigate the risk of paradoxical air embolism through the VSD. The detailed anesthetic procedure, surgical intervention, and subsequent follow-up are outlined in Table 2.

Table 2. Procedure of treatment and follow-up.

Phase	Intervention / Observation
Preoperative preparation	ASA III classification
	Existing 20G IV line, fasting fluids met, IVC ultrasound for volume status
	Meticulous air bubble prevention in IV lines
Premedication	IV Midazolam 1.5 mg, IV Ketamine 5 mg
	Oxygen via nasal cannula 3 L/min
Monitoring initiated	Standard: ECG (Lead II, V_5), SpO_2, Arterial line (post-sedation, local anesthesia with lidocaine 2%)
	Initial IABP 97/62 mmHg, HR 88 bpm, SpO_2 88%
Anesthetic induction	Titrated IV Ketamine (25 mg), IV Propofol (30 mg)
	IV Fentanyl (50 mcg), IV Rocuronium (10 mg)
Airway management	Preoxygenation (close-circuit, normoventilation, avoiding high PAP)
	Endotracheal intubation with 5.5 cuffed non-kinking ETT
Anesthetic maintenance	Intermittent Fentanyl (0.25 mcg/kg), intermittent Rocuronium (0.15 mg/kg)
	Oxygen, Air, Sevoflurane (1 MAC)
	Ventilation: Normoventilation to slight hyperventilation (avoiding hypercarbia, acidosis, high peak airway pressure)
Intraoperative events	Hypotension managed with IV Phenylephrine (5–10 μg/kg bolus)
	Vital signs: IABP 80–105/50–62 mmHg, HR 85–108 bpm, SpO_2 80–89%
	Craniotomy and abscess resection duration: ~2.5 hours
	Fluids: Crystalloid 700 mL, Colloid 100 mL. EBL 100 mL, UOP 100 mL
Emergence/Extubation	Reversal: Atropine, Neostigmine
	Extubated in OR after meeting criteria
Recovery room	Awake, followed commands, SpO_2 84–88% on 3 L/min nasal cannula
Postoperative care	PICU monitoring overnight
	Pain management: Continuous IV Morphine (0.5 mg/kg over 24h), IV Paracetamol (10 mg/kg q8h)
	Propranolol and antibiotics continued
Outcome	Uneventful perioperative course; no neurological or cardiac complications

In the preoperative holding area, premedication consisting of intravenous midazolam and a small dose of ketamine was administered to ensure anxiolysis and hemodynamic stability, respectively, while the patient received supplemental oxygen. Upon arrival in the operating room, comprehensive ASA standard monitoring was initiated, critically augmented by invasive arterial blood pressure monitoring after adequate sedation. The anesthetic induction was carefully titrated using a combination of ketamine and propofol, supplemented with fentanyl and rocuronium, to achieve a smooth transition to general anesthesia while respecting the patient's fragile cardiovascular and neurological status. Airway management involved meticulous preoxygenation and gentle endotracheal intubation. Anesthesia was maintained with sevoflurane in an oxygen-air mixture, along with intermittent analgesics and muscle relaxants, while ventilation was precisely controlled to balance cerebral and pulmonary physiology. Any episodes of intraoperative hypotension were promptly managed with phenylephrine boluses. The neurosurgical procedure was completed within 2.5 hours with stable intraoperative hemodynamics and oxygenation within the patient's baseline cyanotic range. Fluid management was judiciously performed. At the conclusion, neuromuscular blockade was reversed, and the patient was extubated uneventfully in the operating room. Postoperatively, the patient was monitored in the Pediatric Intensive Care Unit (PICU), received appropriate multimodal analgesia, and continued his preoperative cardiac and antibiotic medications.

3. Discussion

The intricate dance of anesthetic management in a pediatric patient burdened simultaneously with uncorrected Tetralogy of Fallot (TOF) and a life-threatening brain abscess demanding urgent

craniotomy is a narrative of navigating profound physiological challenges. This 11-year-old boy's successful perioperative course, free from cardiac or neurological compromise, was not a stroke of serendipity but rather the outcome of a deeply considered, physiology-centric anesthetic strategy. This discussion aims to unravel the complexities encountered, dissect the rationale underpinning each therapeutic decision, and articulate how the chosen interventions harmonized to produce a favorable result, all within the detailed context of this specific patient's journey. Understanding the profound pathophysiological disturbances intrinsic to both uncorrected TOF and a large cerebral abscess is the bedrock upon which any rational anesthetic plan must be constructed. These conditions, each formidable in its own right, synergize to create a uniquely perilous clinical environment. Uncorrected TOF, as seen in this patient with its hallmark features of a large ventricular septal defect (VSD), severe right ventricular outflow tract (RVOT) obstruction, an overriding aorta, and consequent right ventricular hypertrophy (RVH), engenders a state of chronic cardiovascular instability and systemic hypoxemia. The patient's baseline oxygen saturation of 86% on room air was a direct reflection of this.[11,12]

The primary hemodynamic consequence of TOF is the obligatory right-to-left (R-to-L) shunting of deoxygenated blood across the VSD directly into the systemic circulation. The magnitude of this shunt, and thus the severity of arterial desaturation, is perpetually dictated by the relative impedances of the pulmonary and systemic vascular beds. Pulmonary vascular resistance (PVR) is influenced by a myriad of factors relevant to anesthesia: alveolar oxygen tension (hypoxia dramatically increases PVR), arterial carbon dioxide tension (hypercarbia increases PVR), pH (acidosis increases PVR), lung volumes and airway pressures (high pressures compress alveolar capillaries, increasing PVR), and the effects of various anesthetic drugs. Systemic vascular resistance (SVR), on the other hand, is primarily determined by arteriolar tone, which is also heavily influenced by anesthetic agents, sympathetic nervous system activity, and intravascular volume status. Any intervention that disproportionately increases PVR or decreases SVR will inevitably exacerbate the R-to-L shunt, leading to profound cyanosis and potentially precipitating a hypercyanotic spell. This patient's history of squatting during exertion was a classic example of a behavioral adaptation to increase SVR (by kinking femoral arteries and increasing intra-abdominal pressure), thereby reducing the R-to-L shunt and improving pulmonary perfusion and oxygenation. The echocardiographic finding of severe pulmonary stenosis underscored the high baseline PVR in this child. The presence of major aortopulmonary collateral arteries (MAPCAs), while sometimes providing an alternative source of pulmonary blood flow, also adds complexity to the circulatory dynamics and can contribute to volume load on the left heart if significant.[13,14]

The right ventricle in TOF is subjected to chronic pressure overload due to the RVOT obstruction, leading to significant hypertrophy. While this hypertrophy is an adaptive mechanism to maintain output against increased resistance, it also renders the RV stiff, diastolic-ally non-compliant, and exquisitely sensitive to changes in preload and afterload. Inadequate preload can lead to a decrease in RV end-diastolic volume, potentially worsening dynamic RVOT obstruction and reducing pulmonary blood flow. Conversely, excessive preload or an acute increase in afterload (PVR) can precipitate RV failure. Myocardial contractility, already working at its limit, can be easily depressed by many anesthetic agents, further compromising the RV's ability to eject blood into the high-resistance pulmonary circuit. These terrifying episodes represent an acute physiological decompensation characterized by a sudden and dramatic increase in R-to-L shunting, leading to profound hypoxemia, severe respiratory distress, and altered consciousness. The underlying mechanism is often multifactorial, involving spasm of the muscular infundibular portion of the RVOT (drastically increasing RV afterload) and/or an abrupt fall in SVR (further encouraging shunting away from the lungs). Stimuli such as pain, anxiety, crying (as noted in this patient's history), fever, dehydration, or certain anesthetic maneuvers (especially during induction or emergence) can trigger these spells. Effective anesthetic management must prioritize the prevention of Tet

spells, and if they occur, institute rapid and aggressive treatment. The body attempts to adapt to chronic hypoxemia. Polycythemia, an increase in red blood cell mass (reflected in this patient's hemoglobin of 14.2 g/dL and hematocrit of 42.8%), develops to enhance oxygen-carrying capacity. However, this also significantly increases blood viscosity, predisposing to microcirculatory sludging and thromboembolic complications, including cerebrovascular accidents. Coagulation abnormalities, such as thrombocytopenia or platelet dysfunction (though this patient had an elevated platelet count), can also occur, increasing bleeding risk. Paradoxical embolism, where venous thrombi or air bypass the pulmonary circulation via the VSD and enter the systemic arterial system, is an ever-present danger. Nutritional deficiencies, like the marasmic malnutrition noted in this child, can further impair immune function and wound healing.[15,16]

The development of a brain abscess, a severe focal intracranial infection, in a patient with TOF is a well-recognized complication directly linked to the R-to-L shunt. This patient's large (5.2 x 5 x 4 cm) left frontoparietal abscess, with its attendant vasogenic edema and striking mass effect—evidenced by a 1.3 cm subfalcine herniation and rightward uncal herniation—represented an acute neurological catastrophe superimposed upon a chronic cardiac condition. Bacteria from mundane sources (oral cavity, skin, respiratory tract) can, in the presence of an R-to-L shunt, gain direct entry into the arterial circulation, effectively bypassing the crucial filtration mechanism of the pulmonary capillary bed. These circulating bacteria can then lodge in the cerebral microvasculature, typically at the grey-white matter junction where blood flow naturally slows, initiating an inflammatory cascade that culminates in abscess formation. The initial cerebritis progresses to central necrosis, liquefaction, and finally, encapsulation by a collagenous wall formed by fibroblasts and reactive astrocytes. The Monro-Kellie doctrine dictates that within the rigid confines of the skull, the total volume (brain parenchyma, cerebrospinal fluid (CSF), and blood) must remain constant. An expanding mass lesion like an abscess, coupled with the significant surrounding vasogenic edema (due to breakdown of the blood-brain barrier and extravasation of plasma fluid into the interstitial space), rapidly exhausts the compensatory mechanisms of CSF displacement and venous blood volume reduction. This inevitably leads to a perilous rise in intracranial pressure (ICP). The CT findings of ventricular effacement and herniation syndromes in this patient were ominous signs of critically elevated ICP. Sustained ICH can lead to global cerebral ischemia, further edema, and ultimately, brainstem compression and death.[17,18]

Compromised Cerebral Perfusion Pressure (CPP), the effective pressure gradient driving blood flow to the brain, is defined as Mean Arterial Pressure (MAP) minus ICP (or Central Venous Pressure, CVP, if higher). As ICP rises, CPP falls, unless MAP can be commensurately increased. If CPP drops below critical thresholds (typically 40-50 mmHg in children, though this varies with age and clinical condition), cerebral ischemia ensues. This is particularly concerning in a cyanotic patient already experiencing reduced arterial oxygen content. Anesthetic management must therefore aim to optimize CPP by both controlling ICP and supporting MAP. Normally, cerebral blood flow (CBF) is maintained relatively constant across a wide range of MAPs (typically 50-150 mmHg in adults, with adjusted ranges for children) through a process called cerebral autoregulation, which involves myogenic and metabolic modulation of cerebrovascular resistance. However, in areas surrounding a brain abscess or with significant edema and inflammation, autoregulation is often impaired or abolished. In such regions, CBF becomes passively dependent on CPP, making the brain exquisitely vulnerable to both hypotension (risking ischemia) and hypertension (risking increased edema and hemorrhage). The inflammatory penumbra surrounding a brain abscess, and direct cortical irritation by the lesion, can create an epileptogenic focus. Seizures dramatically increase cerebral metabolic rate ($CMRO_2$), CBF, and ICP, potentially worsening neurological injury. Anesthetic choices should ideally favor agents with anticonvulsant properties or, at a minimum, avoid those known to lower the seizure threshold. The confluence of these severe cardiac and neurological pathologies presented the anesthesiology team with a daunting set of

conflicting priorities. For instance, maneuvers to reduce ICP, such as aggressive hyperventilation, might adversely affect PVR. Conversely, strategies to optimize cardiac function, such as maintaining a higher SVR, could impact cerebral hemodynamics. The anesthetic plan had to be a masterclass in physiological compromise and targeted intervention.[19,20]

The anesthetic management detailed for this patient was not a generic protocol but a bespoke symphony of pharmacological agents, monitoring techniques, and physiological manipulations, all finely tuned to the unique dissonance of his dual pathologies. The uneventful perioperative course, culminating in a good neurological outcome without cardiac setbacks, serves as compelling evidence of the strategy's success. The meticulous preoperative preparation was foundational to the positive outcome. The detailed TTE findings were not merely academic; they provided a roadmap of the patient's cardiac vulnerabilities. Understanding the severity of pulmonary stenosis, the size of the VSD, the direction and magnitude of the shunt, and the presence of MAPCAs informed every subsequent anesthetic decision. The continuation of oral propranolol until surgery was a key element; propranolol, by its beta-blocking action, can reduce infundibular spasm (a component of dynamic RVOT obstruction), decrease heart rate (allowing more time for diastolic filling of the stiff RV), and potentially reduce the frequency and severity of Tet spells. Its role in stabilizing the patient's baseline cardiac status cannot be understated. While the patient was alert preoperatively, the CT scan's revelation of a large abscess with significant mass effect and herniation underscored the critical nature of the neurological insult and the urgent need for surgical decompression. This urgency influenced the pace of preoperative optimization, balancing thoroughness with the need for timely intervention.

Euvolemia is the holy grail of fluid management in TOF. Hypovolemia can exacerbate RVOT obstruction, lead to systemic hypotension, and worsen R-to-L shunting. Hypervolemia, on the other hand, can overwhelm the non-compliant, pressure-loaded RV, leading to RV failure and increased CVP, which in turn can impede cerebral venous drainage and further elevate ICP. The preoperative use of IVC ultrasonography to assess volume status was a sophisticated and proactive approach. The collapsibility index of the IVC provides a dynamic, non-invasive estimate of intravascular volume and can help guide fluid administration, aiming for a state of optimal preload before the vasodilatory and myocardial depressant effects of anesthetic induction. This demonstrated a commitment to evidence-informed practice, as IVC ultrasound is increasingly recognized for its utility in pediatric fluid management. The ongoing administration of a triple antibiotic regimen (ceftriaxone, vancomycin, metronidazole) was crucial. This combination provides broad-spectrum empirical coverage against common pathogens implicated in brain abscesses, including gram-positive cocci, gram-negative bacilli, and anaerobes, with good penetration across the blood-brain barrier. Controlling the intracranial infection and preventing systemic sepsis were paramount, as sepsis could trigger catastrophic vasodilation, drastically reducing SVR and precipitating profound cardiovascular collapse in a TOF patient. The explicit mention of meticulous care to prevent air bubbles in all IV lines highlights a fundamental safety principle in managing patients with intracardiac shunts. This involves not only careful de-airing of syringes and infusion sets but also using Luer-Lok connections, avoiding dependent loops in tubing where air can collect, and being vigilant during any manipulation of IV access. The consequences of even a small paradoxical air embolus to the brain or coronary arteries can be devastating, ranging from stroke to myocardial infarction or sudden death. This diligence likely contributed significantly to the neurologically intact outcome.

The transition from the ward to the operating room, and the period leading up to induction, are fraught with potential triggers for anxiety and physiological instability in a child, especially one with TOF. The premedication strategy was designed to mitigate these risks. Midazolam (1.5 mg IV), This short-acting benzodiazepine, administered at approximately 0.09 mg/kg, served multiple crucial purposes. It provided potent anxiolysis, diminishing the child's fear and apprehension, which is particularly important as stress and crying can provoke deleterious increases in PVR

and precipitate Tet spells. Midazolam also induces anterograde amnesia, improving the child's overall perioperative experience. Its sedative effects facilitated smoother IV access if additional lines were needed and a calmer acceptance of monitoring equipment and the operating room environment. Importantly, at this dose, significant respiratory depression or profound SVR reduction is unlikely, especially in a closely monitored setting with supplemental oxygen already in place. Ketamine (5 mg IV), The administration of a small, sub-anesthetic dose of ketamine (approximately 0.3 mg/kg) as part of the premedication regimen was a particularly astute pharmacological choice. Ketamine's unique profile, characterized by indirect sympathomimetic effects (due to inhibition of catecholamine reuptake and direct stimulation of central sympathetic pathways), results in maintenance or even an increase in SVR, heart rate, and myocardial contractility. In the context of TOF, this translates to a reduction in R-to-L shunting and improved pulmonary blood flow. Furthermore, ketamine preserves respiratory drive and airway reflexes at these low doses and provides potent analgesia, which can be beneficial if any minor procedures like arterial line placement were anticipated before full induction. This proactive use of ketamine likely contributed to the observed hemodynamic stability (initial IABP 97/62 mmHg, HR 88 bpm) upon arrival in the OR and during the establishment of invasive monitoring. The administration of supplemental oxygen via nasal cannula at 3 L/min further fortified the patient's oxygen reserves and aimed to improve baseline saturation, though the impact is limited by the degree of shunt.

The induction of general anesthesia is arguably the most perilous phase for a patient with uncorrected TOF and raised ICP, as anesthetic agents can profoundly and rapidly alter cardiovascular and cerebrovascular physiology. The chosen technique reflected a masterly balancing act. Ketamine as the Cornerstone (25 mg IV), The decision to use ketamine as the primary induction agent, at a dose of approximately 1.5 mg/kg, was predicated on its well-established cardiovascular benefits in cyanotic congenital heart disease. As discussed, its sympathomimetic properties bolster SVR, which is paramount in reducing R-to-L shunting. Unlike many other induction agents, it typically does not cause significant myocardial depression, and may even enhance contractility. While historical concerns existed regarding ketamine's potential to increase ICP, contemporary evidence, particularly from studies in patients with controlled ventilation and stable hemodynamics, suggests that at clinical doses (1-2 mg/kg IV), ketamine does not significantly elevate ICP and may even possess neuroprotective qualities through NMDA receptor antagonism. Its potent analgesic effects also contribute to blunting the stress response to airway manipulation. The dissociative anesthetic state it produces, while unique, provides rapid unconsciousness. Propofol in Judicious Concert (30 mg IV), administered in a carefully titrated dose of approximately 1.8 mg/kg, served as an important adjunct to ketamine. Propofol offers the advantages of rapid onset, smooth induction quality, and a reduction in $CMRO_2$, which is beneficial in neuroanesthesia. However, its primary drawbacks in this scenario are significant dose-dependent vasodilation (leading to a fall in SVR) and myocardial depression, both of which could catastrophically worsen R-to-L shunting and hemodynamic stability in TOF. The co-administration with ketamine, whose sympathomimetic effects counteract these SVR-lowering and cardiodepressant tendencies, was a strategic maneuver. The slow, titrated infusion of propofol allowed the anesthesiologist to achieve the desired depth of anesthesia for intubation while continuously assessing the hemodynamic response and allowing ketamine's favorable cardiovascular effects to predominate. This combination aimed to harness the benefits of both agents while mitigating their respective risks.

Fentanyl for Sympathetic Attenuation (50 mcg IV), The administration of fentanyl, a potent synthetic opioid, at approximately 3 mcg/kg prior to laryngoscopy was crucial for blunting the intense hemodynamic and neuroendocrine stress response associated with airway manipulation. Laryngoscopy and intubation are powerful noxious stimuli that can trigger marked increases in heart rate, blood pressure, and sympathetic outflow. In a TOF patient, this sympathetic surge could precipitate infundibular spasm, acutely increasing RVOT obstruction and triggering a Tet spell.

In a patient with raised ICP, such a response could also dangerously elevate ICP further. Fentanyl's ability to provide profound analgesia and cardiovascular stability by attenuating this reflex sympathetic discharge is invaluable. It also contributes to reducing $CMRO_2$. Rocuronium for Optimal Intubating Conditions (10 mg IV), The choice of rocuronium (approximately 0.6 mg/kg) as the neuromuscular blocking agent was deliberate and well-justified. Rocuronium provides rapid and reliable muscle relaxation, facilitating atraumatic endotracheal intubation. Critically, it is devoid of histamine-releasing properties. Histamine release, a characteristic of some other NMBAs (such as atracurium or succinylcholine in some individuals), can cause systemic vasodilation, leading to a precipitous drop in SVR, which would markedly worsen R-to-L shunting and systemic hypoxemia in this patient. Rocuronium's cardiovascular stability makes it an excellent choice in this high-risk population. Preoxygenation with 100% oxygen for three minutes via a close-circuit breathing system, targeting normoventilation, was performed to maximize the patient's functional residual capacity with oxygen, thereby prolonging the safe apnea time during intubation. Crucially, high positive airway pressures were avoided during both mask ventilation (if any was needed) and subsequent mechanical ventilation. Elevated airway pressures can impede venous return (both systemic and cerebral), increase PVR by compressing pulmonary capillaries, and thus worsen R-to-L shunting and potentially elevate ICP. The successful and smooth endotracheal intubation with an appropriately sized (5.5 mm) cuffed, non-kinking endotracheal tube ensured a secure airway, protected against aspiration, and allowed for precise control of ventilation and oxygenation throughout the neurosurgical procedure. Confirmation of correct tube placement via bilateral auscultation and capnography is standard but vital.

The maintenance phase of anesthesia requires a continuous balancing act to provide adequate surgical anesthesia, optimize neurophysiological parameters, and maintain cardiovascular stability in the face of ongoing surgical stimulation and the patient's underlying TOF. Sevoflurane as the Volatile Agent (1 MAC in Oxygen/Air) was chosen as the primary maintenance agent, administered at an end-tidal concentration of approximately 1 MAC, titrated to clinical effect and hemodynamic response. Sevoflurane is widely favored in pediatric anesthesia due to its low airway irritability, pleasant odor (less relevant for IV induction but good for inhalation induction if needed), bronchodilatory properties (which can be beneficial in reducing PVR), and relatively rapid pharmacokinetics allowing for swift adjustments in anesthetic depth and a reasonably quick emergence. It generally has minimal impact on PVR at typical clinical concentrations (1-1.5 MAC) and is less likely to sensitize the myocardium to catecholamines than older agents like halothane. However, all volatile anesthetics, including sevoflurane, produce dose-dependent systemic vasodilation (reducing SVR) and myocardial depression. Therefore, careful titration and vigilant hemodynamic monitoring were essential. The use of an oxygen/medical air mixture allowed the FiO_2 to be precisely controlled, aiming to maintain the patient's SpO_2 in their baseline cyanotic range of 80-89%. This strategy acknowledges that in severe TOF, achieving "normal" saturations is often impossible due to the fixed shunt and that excessive FiO_2 offers little additional benefit to arterial oxygen content while potentially having other minor systemic effects. The provision of intermittent boluses of fentanyl (stated as 0.25 mcg/kg, likely per bolus as needed) played a crucial role in providing ongoing potent analgesia, thereby reducing the sevoflurane requirement (a MAC-sparing effect). This helps to minimize the dose-dependent cardiovascular depressant effects of the volatile agent. Fentanyl also contributes to stress response attenuation and cardiovascular stability during periods of heightened surgical stimulation.

Intermittent boluses of rocuronium (0.15 mg/kg as needed) ensured continued muscle relaxation. This is vital during delicate neurosurgery to prevent patient movement, coughing, or straining, any of which could lead to disastrous consequences such as surgical injury, increased ICP, or acute hemodynamic decompensation (Tet spell). This was arguably one of the most critical aspects of intraoperative management, requiring a meticulous balance between

neuroprotective and cardioprotective goals. The strategy of "normoventilation to slight hyperventilation" (targeting an $EtCO_2$ of 30-35 mmHg, assuming a relatively normal $PaCO_2$-$EtCO_2$ gradient in this patient without severe lung disease) was designed to induce mild cerebral vasoconstriction secondary to hypocapnia. This reduction in cerebral blood volume helps to lower ICP and improve brain relaxation, facilitating surgical exposure – a critical consideration given the large abscess and significant mass effect. However, this had to be balanced against the potential effects on PVR. While severe hypocapnia can increase PVR, mild hypocapnia as targeted here is generally well-tolerated and less likely to cause significant PVR elevation, especially when acidosis is avoided. The absolute avoidance of hypercarbia and acidosis was paramount. Hypercarbia is a potent cerebral vasodilator, which would acutely worsen ICP, and it is also a significant pulmonary vasoconstrictor, which would exacerbate R-to-L shunting. Similarly, acidosis (respiratory or metabolic) has similar deleterious effects on both ICP and PVR. Strict control of peak inspiratory pressures and mean airway pressures was also vital. High airway pressures can impede cerebral venous drainage (raising ICP), increase PVR by compressing alveolar vessels, and reduce right ventricular preload and output, all of which would be detrimental. The ventilation was managed to achieve the desired physiological targets without causing barotrauma or hemodynamic compromise.

The prompt and effective management of intraoperative hypotension (defined as a sustained drop in MAP >20% from baseline, though specific thresholds can vary) with intravenous boluses of phenylephrine (5–10 µg/kg) was a cornerstone of maintaining cardiovascular stability and, by extension, cerebral perfusion. Phenylephrine, as a pure α_1-adrenergic agonist, selectively increases SVR without significantly affecting PVR or possessing direct inotropic or chronotropic effects on the heart (though reflex bradycardia can occur, it is usually modest and well-tolerated, especially if atropine is available). In the context of TOF, this SVR augmentation is highly beneficial: it reduces the R-to-L shunt by making it more favorable for the RV to eject blood into the pulmonary artery, thereby improving pulmonary blood flow and systemic oxygenation. Furthermore, by supporting MAP, phenylephrine helps to maintain an adequate CPP in the face of potentially elevated ICP. The ability to keep the patient's IABP within a stable range (80–105/50–62 mmHg) throughout a 2.5-hour complex neurosurgical procedure attests to the efficacy of this proactive vasopressor strategy and the overall anesthetic plan. The judicious intraoperative fluid management, consisting of 700 mL of crystalloid and 100 mL of colloid for an estimated blood loss of 100 mL and a urine output of 100 mL over 2.5 hours, was aimed at maintaining normovolemia. This precise balance is critical in TOF to ensure adequate RV preload without causing volume overload and RV distension. The choice of fluids (isotonic crystalloids and colloids) helps maintain intravascular volume and oncotic pressure. Maintaining normothermia using a forced-air warming blanket is crucial in pediatric anesthesia, particularly during long neurosurgical procedures. Hypothermia can lead to a host of adverse effects, including coagulopathy (increasing surgical bleeding), increased SVR (which might initially seem beneficial in TOF but is associated with increased oxygen consumption upon rewarming/shivering), delayed drug metabolism, prolonged emergence, and an increased risk of postoperative infections.

Continuous, comprehensive, and invasive monitoring formed the vigilant sensory apparatus of the anesthesiology team, enabling early detection of physiological derangements and guiding therapeutic interventions. Continuous electrocardiography (ECG), with specific attention to leads II and V_5, allowed for the detection of arrhythmias (common in structurally abnormal hearts) and signs of myocardial ischemia (though less common in children, still a concern with severe hypoxemia or coronary embolism). Pulse oximetry (SpO_2) provided a continuous, albeit relative, measure of arterial oxygen saturation; in this patient with baseline cyanosis, the trend and the maintenance of SpO_2 within the target range of 80-89% were more important than the absolute value. End-tidal carbon dioxide ($EtCO_2$) monitoring was indispensable for guiding the ventilation strategy, allowing for precise titration of $PaCO_2$ to achieve the desired mild

hypocapnia for ICP control while avoiding dangerous hypercarbia. Continuous temperature monitoring ensured the maintenance of normothermia. Invasive Arterial Blood Pressure (IABP), The Gold Standard for Hemodynamic Assessment, The placement of a radial arterial line for continuous beat-to-beat IABP monitoring was non-negotiable in this high-risk case. It provided immediate feedback on the efficacy of vasopressor therapy, allowed for the rapid detection of hypotension or hypertension, and was essential for the calculation and optimization of CPP. The arterial waveform itself can also provide subtle clues about SVR and cardiac contractility. Furthermore, the arterial line offers ready access for intraoperative blood gas analysis, which, although not explicitly stated as performed in the source, would be invaluable for assessing oxygenation (PaO_2), ventilation ($PaCO_2$), acid-base status (pH, bicarbonate), and electrolytes if clinically indicated. An indwelling urinary catheter allowed for continuous monitoring of urine output, serving as a crucial indicator of renal perfusion and, by extension, overall adequacy of systemic perfusion and intravascular volume status. Maintaining adequate urine output (around 1 mL/kg/hr) is a key therapeutic goal.

A smooth, controlled emergence from anesthesia and safe extubation are critical for facilitating early neurological assessment and minimizing physiological stress that could adversely affect both cardiac and cerebral status. The administration of atropine (an anticholinergic to prevent neostigmine-induced bradycardia) and neostigmine (an acetylcholinesterase inhibitor) to reverse residual rocuronium-induced neuromuscular blockade was a standard and necessary step. Complete reversal ensures adequate respiratory muscle strength for spontaneous ventilation, effective coughing, and maintenance of airway patency post-extubation. The decision to extubate the patient in the operating room was based on the fulfillment of stringent criteria: an awake and alert state, ability to follow commands, demonstration of adequate respiratory effort and tidal volumes, and intact airway protective reflexes. Successful extubation in the OR in such a complex case is a significant achievement, indicative of profound physiological stability, excellent pain control, and minimal residual anesthetic effects. It obviates the risks associated with prolonged intubation and allows for more immediate and accurate neurological evaluation. The patient's comfortable state in the recovery room, maintaining an SpO_2 of 84–88% on 3 L/min of nasal cannula oxygen, further validated this decision.

The immediate postoperative period remained a time of heightened surveillance, as the patient was still vulnerable to complications. Transfer to the PICU for overnight monitoring and specialized care was an essential and prudent measure. The PICU environment provides the capability for continuous cardiorespiratory monitoring, advanced ventilatory support if needed, rapid diagnosis and management of emerging complications (neurological or cardiac), and expert pediatric nursing care. The provision of robust postoperative pain relief through a continuous intravenous morphine infusion (calculated to deliver approximately 0.5 mg/kg over 24 hours) supplemented with regular intravenous paracetamol (10 mg/kg every 8 hours) was a cornerstone of postoperative management. Effective multimodal analgesia is critical not only for patient comfort but also for obtunding the detrimental physiological stress responses to pain. Uncontrolled pain can lead to sympathetic activation, resulting in tachycardia, hypertension, increased myocardial oxygen consumption, agitation, and crying – all of which could precipitate a Tet spell or increase ICP. Morphine provides potent, titratable systemic analgesia, while paracetamol offers a non-opioid adjunctive analgesic and antipyretic effect, reducing opioid requirements and their potential side effects. The uninterrupted administration of the patient's preoperative cardiac medication (propranolol) and the triple antibiotic therapy (ceftriaxone, vancomycin, metronidazole) throughout the postoperative period was vital for ongoing cardiovascular stability and continued treatment of the intracranial infection, respectively. This continuity of care is crucial for preventing relapses or new complications.

The remarkably uneventful perioperative course of this patient, despite the extreme physiological challenges posed by his dual pathology, was a direct consequence of an anesthetic strategy that

meticulously respected and proactively managed the underlying pathophysiological disturbances. The entire anesthetic conduct was fundamentally geared towards manipulating the PVR/SVR balance in favor of increased pulmonary blood flow. This was achieved by consistently supporting SVR (through choices like ketamine and phenylephrine, and avoidance of histamine-releasing drugs) while actively minimizing increases in PVR (through careful ventilation avoiding hypoxia, hypercarbia, acidosis, and high airway pressures). This concerted effort directly addressed the central hemodynamic lesion of TOF and was likely the single most important factor in preventing intraoperative desaturation and Tet spells. The anesthetic plan was a sophisticated tapestry where neuroprotective interventions were seamlessly interwoven with cardioprotective measures, rather than being pursued in isolation at the expense of the other system. For example, the choice of ketamine offered benefits for both SVR maintenance (cardiac) and potentially stable ICP (neurological). The controlled mild hyperventilation aimed to reduce ICP while being carefully moderated to avoid significant adverse effects on PVR. This integrated approach ensured that interventions aimed at the brain did not destabilize the heart, and vice-versa. The anesthetic team demonstrated a clear anticipation of potential problems inherent to the patient's conditions. This was evident in the choice of premedication to ensure a calm induction, the proactive use of an SVR-supporting induction technique, the immediate availability and judicious use of phenylephrine for any tendency towards hypotension, and the meticulous attention to air embolism prevention. This foresight allowed for interventions to be implemented preemptively or at the earliest sign of deviation, preventing minor physiological trespasses from escalating into major crises. The preoperative IVC assessment for volume status is another prime example of this proactive philosophy.

The success underscores the importance of precision in pediatric anesthesia, especially in complex cases. Seemingly small details—such as ensuring absolute freedom from air bubbles in IV lines, precise titration of volatile anesthetics to the nearest fraction of a MAC, and fine-tuning ventilator parameters to achieve specific $EtCO_2$ targets—collectively contribute to a significant margin of safety. This case exemplifies how such meticulousness translates directly into improved patient outcomes. The selection of each anesthetic and adjuvant drug was clearly predicated on a profound understanding of its complex pharmacodynamic and pharmacokinetic profile in the context of pediatric cyanotic heart disease and raised intracranial pressure. There was no "one size fits all" approach; rather, each agent was chosen for its specific ability to contribute positively to the overall physiological goals, or at least to minimize harm. The combination of ketamine and propofol for induction, the choice of rocuronium, the reliance on phenylephrine, and the multimodal analgesic regimen all reflect this deep pharmacological insight.

This complex case serves as a powerful illustration that even in the shadow of formidable, coexisting cardiac and neurological pathologies in a vulnerable pediatric patient, an anesthetic approach grounded in robust physiological principles, characterized by anticipatory vigilance, and executed with meticulous precision can indeed culminate in an outstandingly positive outcome. The seamless integration of specialized knowledge from the distinct but related fields of pediatric cardiac anesthesia and neuroanesthesia was unequivocally pivotal. The detailed chronicle of the anesthetic decisions made, the clear articulation of their underlying rationales, and the direct correlation with the patient's stable intraoperative course and complication-free recovery provide an invaluable and instructive narrative for clinicians faced with similarly daunting challenges. While the uniqueness of each such patient precludes the formulation of rigid protocols, the core tenets exemplified here—thorough and individualized assessment, a dynamic understanding of the evolving pathophysiology, strategic and titrated pharmacological interventions, unwavering vigilance in monitoring, and proactive management of anticipated complications—constitute an enduring and robust framework for optimizing care and enhancing safety in this exceptionally high-risk patient demographic. The fact that this child navigated such a perilous journey

without any discernible cardiac or neurological sequelae stands as compelling testimony to the efficacy and sophistication of the anesthetic care delivered.

4. Conclusion

The successful anesthetic management of an 11-year-old child with uncorrected Tetralogy of Fallot undergoing urgent craniotomy for a large brain abscess underscores the paramount importance of a deeply integrated, physiology-driven approach. This case powerfully demonstrates that even in the face of such extreme, coexisting pathologies, an excellent outcome is achievable. This success hinged on meticulous preoperative optimization, the judicious selection and titration of anesthetic agents to favorably balance systemic and pulmonary vascular resistances while controlling intracranial pressure, and unwavering intraoperative vigilance. The seamless fusion of neuroanesthetic and cardiac anesthetic principles, tailored to the patient's unique challenges, was critical in preventing perioperative complications. This report provides a compelling testament to how profound physiological understanding and proactive, individualized anesthetic care can navigate the most perilous clinical scenarios, offering valuable insights for managing this exceptionally vulnerable pediatric patient population.

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