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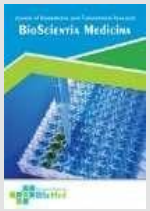

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Spontaneous Respiration Intubation as a Cornerstone of Multidisciplinary Management for Delayed Tracheoesophageal Fistula Repair in a Critically Ill Neonate

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A B S T R A C T

Background: The perioperative management of neonates with esophageal atresia and Type C tracheoesophageal fistula (EA/TEF) is exceptionally challenging, particularly in cases of delayed diagnosis complicated by aspiration pneumonia and congenital heart disease. The primary anesthetic risk is catastrophic gastric insufflation and hemodynamic collapse from positive pressure ventilation (PPV) before fistula control. This report details a successful multidisciplinary strategy centered on an airway technique that preserves spontaneous ventilation. **Case presentation:** A 16-day-old, 2.5 kg female neonate with Type C EA/TEF presented for surgical repair following a delayed referral. The case was critically complicated by severe aspiration pneumonia (cultures positive for *Klebsiella pneumoniae*), which was managed with targeted antibiotic therapy, and hemodynamically significant congenital heart defects (2.5 mm patent ductus arteriosus, 3 mm patent foramen ovale). After 48 hours of intensive cardiorespiratory and nutritional optimization in the neonatal intensive care unit (NICU), the patient underwent surgery. To circumvent the life-threatening risks of PPV, an inhalational induction with sevoflurane was performed, maintaining spontaneous ventilation. The airway was secured via direct laryngoscopy without neuromuscular blockade. A right extrapleural thoracotomy, fistula ligation, and primary esophageal anastomosis were successfully performed. Intraoperative lung retraction-induced desaturation was managed with coordinated surgeon-anesthetist maneuvers. The postoperative course was uneventful. **Conclusion:** In a high-risk neonate with delayed TEF presentation and profound cardiorespiratory compromise, securing the airway while maintaining spontaneous ventilation is a cornerstone of safe anesthetic practice. This approach, integrated within a comprehensive, multidisciplinary management plan, directly mitigates the risk of gastric perforation and cardiovascular collapse, thereby enabling a successful surgical repair and favorable outcome.

1. Introduction

Esophageal atresia with a distal tracheoesophageal fistula (EA/TEF), Gross Type C, represents the most common variant of this congenital anomaly, with an incidence of approximately 1 in 2,500 to 4,500 live births.[1] The condition presents an immediate surgical and anesthetic emergency due to its complex and life-threatening pathophysiology. The direct communication between the respiratory and gastrointestinal tracts precipitates a vicious cycle of clinical deterioration.[2] Oral secretions pool in the blind upper esophageal pouch, leading to continuous

aspiration and chemical pneumonitis, which is often compounded by bacterial infection.[3] Concurrently, air from the trachea passes through the fistula into the stomach, causing progressive gastric and intestinal distension. This diaphragmatic splinting severely impairs ventilation, while compression of the great vessels reduces venous return and compromises cardiac output.[4]

Ideally, definitive surgical repair is performed within the first 24 to 72 hours of life. However, a significant subset of neonates experiences a delay in diagnosis or referral, often due to subtle initial symptoms, remote geographic location, or the overriding need to stabilize more severe concomitant anomalies.[5] Such delays, as seen in the case presented, profoundly amplify perioperative risk. The neonate is frequently in a state of septic shock, suffering from established pneumonia, severe malnutrition, and metabolic derangement, with mortality rates in this high-risk cohort remaining significant. This precarious clinical picture is further complicated in up to 50% of cases by associated congenital anomalies, particularly cardiovascular defects as part of the VACTERL (Vertebral, Anal, Cardiac, Tracheoesophageal, Renal, Limb) association.[6] The presence of a patent ductus arteriosus (PDA) or other cardiac shunts creates a fragile hemodynamic equilibrium that is highly susceptible to disruption by anesthetic agents, surgical stress, and, most notably, positive pressure ventilation (PPV).[7]

The central challenge in anesthetic management is securing the airway without triggering iatrogenic catastrophe. Conventional anesthetic induction involving neuromuscular blockade and PPV is fraught with peril.[8] The positive pressure delivered via a face mask preferentially follows the path of least resistance through the fistula directly into the stomach, risking acute gastric perforation and precipitating profound cardiorespiratory collapse. Consequently, an airway strategy that obviates the need for PPV prior to surgical control of the fistula is not merely an alternative but a fundamental tenet of patient safety.[9]

While various advanced techniques exist, such as bronchoscopic placement of the endotracheal tube (ETT) beyond the fistula or Fogarty catheter occlusion, these are technically demanding and not universally available. An approach centered on maintaining spontaneous ventilation throughout induction and intubation represents a conceptually ideal method to mitigate these risks. This technique prevents gastric insufflation, preserves protective airway reflexes, and maintains physiological airway pressures, thereby preventing hemodynamic collapse.[10]

This case report provides a powerful clinical demonstration of a multidisciplinary strategy for a 16-day-old neonate with a delayed diagnosis of Type C TEF, complicated by severe aspiration pneumonia and congenital heart disease. The aim of this report is to provide a comprehensive, detailed exposition of the perioperative management, with a specific focus on the procedural nuances and pathophysiological rationale for employing intubation on a spontaneously breathing patient as the keystone of the anesthetic plan to enhance safety in this uniquely challenging clinical context.

2. Case Presentation

A 16-day-old, 2.5 kg female neonate was transferred to our tertiary care pediatric surgical center for the management of EA/TEF. A detailed timeline of her clinical course is presented below (Table 1a and 1b): (1) Day of Life 0-2: Born at 38 weeks of gestation (birth weight 2.7 kg) via spontaneous vaginal delivery. Apgar scores were 7 and 8. Initial symptoms included excessive salivation and coughing with feeding attempts, which were initially misinterpreted as feeding intolerance; (2) Day of Life 3-14: Remained at the referring district hospital. An orogastric tube (OGT) repeatedly failed to pass beyond 10 cm. A "babygram" X-ray on Day 14 confirmed a coiled OGT in the upper thorax with significant bowel gas, leading to the diagnosis of Type C EA/TEF. The delay in diagnosis was attributed to a lack of immediate pediatric surgical consultation at the peripheral center. During this period, the infant

developed progressive respiratory distress and signs of sepsis, including thrombocytopenia; (3) Day of Life 15 (Admission to NICU): Transferred to our neonatal intensive care unit (NICU). The patient was in moderate respiratory distress (respiratory rate 58 breaths/min) with subcostal retractions, requiring 1 L/min of humidified oxygen via nasal cannula to maintain SpO_2 >94%. She was tachycardic (145 bpm) with a blood pressure of 68/42 mmHg. Physical examination revealed coarse crackles in the right upper lobe and a distended abdomen; (4) Day of Life 15-16 (Preoperative Optimization): A 48-hour period of intensive stabilization was undertaken; (5) Day of Life 17 (Surgery): The patient underwent definitive surgical repair; (6) Postoperative Day 4 (Extubation): Successfully extubated to a high-flow nasal cannula; (7) Postoperative Day 5 (Enteral Feeding): Enteral feeding initiated via a trans-anastomotic tube; (8) Postoperative Day 16 (Esophagogram): Contrast study confirmed anastomotic integrity; (9) Postoperative Day 25 (Discharge): Discharged home in stable condition.

Upon admission to our NICU, a comprehensive diagnostic workup and stabilization plan were initiated. The patient was classified as American Society of Anesthesiologists (ASA) physical status IV E. Chest x-ray confirmed the OGT coiled in the upper esophageal pouch at the T3 vertebral level. It revealed extensive bilateral patchy infiltrates, most prominent in the right upper lobe, consistent with severe aspiration pneumonia. Marked gastric and intestinal gas distribution confirmed the presence of a distal fistula. A transthoracic echocardiogram identified a structurally normal heart with two hemodynamically significant findings: a 2.5 mm PDA with a continuous left-to-right shunt and a 3 mm PFO, also with a left-to-right shunt. Biventricular function was normal (ejection fraction 65%), with no evidence of pulmonary hypertension. Initial labs revealed leukocytosis, elevated C-reactive protein (CRP) at 8.12 mg/L, and hypoalbuminemia (2.8 g/dL), indicative of sepsis and malnutrition (Table 2). Initial thrombocytopenia (nadir 75,000/μL) at the referring hospital was determined to be sepsis-induced. A tracheal aspirate culture grew *Klebsiella pneumoniae*, which was sensitive to meropenem.

The decision to proceed with surgery was made after a 48-hour optimization period, based on a downward CRP trend (to 4.5 mg/L), normalization of the platelet count (to 285,000/μL), and stable cardiorespiratory parameters on minimal support. For infection control, intravenous meropenem (40 mg/kg q8h) was initiated. Continuous suctioning of the upper esophageal pouch was performed using a Replogle tube. The patient was managed with a humidified high-flow nasal cannula at 2 L/min (estimated FiO_2 0.25-0.30) to maintain SpO_2 >94% and reduce the work of breathing. The pO_2 of 68 mmHg on the initial ABG reflected a significant V/Q mismatch from her pneumonia. Total parenteral nutrition (TPN) was initiated via a peripherally inserted central catheter to address the catabolic state and hypoalbuminemia, providing essential metabolic support for wound healing.

The primary anesthetic goal was to secure the airway while completely avoiding PPV. In the operating room, the infant was placed on a warming blanket under a radiant warmer. Standard ASA monitors, including a three-lead ECG, pulse oximetry, non-invasive blood pressure cuff, and esophageal temperature probe, were applied. A precordial stethoscope was placed. Two 24-gauge peripheral intravenous catheters were secured. Atropine 0.02 mg/kg (50 mcg) was administered as premedication. An inhalational induction was performed with 100% oxygen and sevoflurane, delivered via a Jackson-Rees circuit with the pop-off valve fully open. The sevoflurane concentration was incrementally increased by 0.5% every 3-4 breaths, guided by close observation of the patient's respiratory rate, maintenance of chest wall excursion, and hemodynamic stability. Gentle jaw support was provided, but no positive pressure was delivered via the face mask.

Table 1a. Diagnostic and Admission Phase (Day 0 - 15)

TIMELINE / DAY OF LIFE	LOCATION / SETTING	KEY CLINICAL EVENTS & FINDINGS	INTERVENTIONS & MANAGEMENT	STATUS / OUTCOME
Day 0 - 2	District Hospital	• Born at 38 weeks (2.7 kg). Apgar 7 & 8. • Symptoms: Excessive salivation, coughing with feeds. • Initial diagnosis: Misinterpreted as feeding intolerance.	Standard postnatal care. Feeding attempts continued.	Condition stable but symptomatic; underlying cause missed.
Day 3 - 14	District Hospital	• Repeated failure to pass Orogastric Tube (OGT) beyond 10 cm. • Progressive respiratory distress and signs of sepsis. • "Babygram" X-ray (Day 14) confirms coiled OGT and bowel gas. • Diagnosis: Type C EA/TEF confirmed.	Diagnostic X-ray performed. Pediatric surgical consultation sought.	Significant delay in diagnosis; patient developed severe aspiration pneumonia and sepsis.
Day 15 (Admission)	Tertiary Center NICU	• Weight: 2.5 kg. ASA Status: IV E. • Moderate respiratory distress (RR 58/min), tachycardia (145 bpm). • Chest X-ray: Severe aspiration pneumonia (R upper lobe). • Echocardiogram: 2.5mm PDA, 3mm PFO (L-to-R shunts). • Labs: Elevated CRP (8.12), hypoalbuminemia (2.8). • Culture: Tracheal aspirate positive for *Klebsiella pneumoniae*.	Initiation of 48-hour intensive stabilization protocol. Placed on humidified O_2 via nasal cannula.	Critically ill neonate requiring urgent multidisciplinary care.

Table 1b. Optimization, Surgical, and Recovery Phase (Day 15 - Post-op Day 25)

TIMELINE / DAY OF LIFE	LOCATION / SETTING	KEY CLINICAL EVENTS & FINDINGS	INTERVENTIONS & MANAGEMENT	STATUS / OUTCOME
Day 15 - 16	Tertiary Center NICU	Preoperative Optimization Phase	• **Infection Control:** IV Meropenem, continuous Replogle tube suction. • **Respiratory Support:** High-flow nasal cannula (2 L/min). • **Nutritional Support:** Total Parenteral Nutrition (TPN) via PICC line.	Stabilization achieved: CRP trending down (to 4.5), platelet count normalized, stable cardiorespiratory status. Cleared for surgery.
Day 17	Operating Room	• Definitive surgical repair of EA/TEF. • Anesthetic technique: Spontaneous ventilation inhalational induction. • Surgical approach: Right extrapleural thoracotomy. • Procedure: Fistula ligation and primary esophageal anastomosis. • Intra-op events: Two transient desaturations managed successfully.	Anesthesia maintained with Sevoflurane/Fentanyl. Post-ligation, transitioned to PCV. Trans-anastomotic feeding tube placed.	Successful repair. Hemodynamically stable throughout. Transferred to NICU intubated.
Post-op Day 4	NICU	Postoperative Weaning	Successful extubation to high-flow nasal cannula.	Breathing spontaneously with minimal support.
Post-op Day 5	NICU	Initiation of Nutrition	Enteral feeding started via trans-anastomotic tube (TAT).	Tolerating feeds well, GI function returning.
Post-op Day 16	Radiology Department	Anastomotic Integrity Check	Contrast esophagogram performed.	Study confirmed a patent anastomosis with no leak or residual fistula. Oral feedings initiated.
Post-op Day 25	Discharge	Discharge from Hospital	Enrolled in long-term multidisciplinary follow-up program.	Discharged home in stable condition on full oral feeds.

Table 2. Preoperative Laboratory Results on Admission to NICU

PARAMETER	RESULT	UNIT	REFERENCE RANGE	INTERPRETATION & CLINICAL SIGNIFICANCE
Complete Blood Count				
Hemoglobin (Hb)	13.1 ↑	g/dL	14.0 - 20.0	Slightly low; suggestive of anemia, likely related to sepsis and chronic inflammation.
White Blood Cell Count	17,500	/µL	5,000 - 21,000	High-normal; consistent with a systemic inflammatory response to infection (sepsis).
Platelets	285,000	/µL	150,000 - 450,000	Normal; indicates recovery from prior sepsis-induced thrombocytopenia.
Inflammatory Markers				
C-Reactive Protein (CRP)	8.12 ↑	mg/L	< 5.0	Significantly elevated, confirming an active, severe inflammatory process consistent with sepsis.
Biochemistry				
Albumin	2.8 ↓	g/dL	3.5 - 5.0	Low (Hypoalbuminemia); indicates malnutrition and a catabolic state due to chronic illness and sepsis.
Coagulation Profile (PT, INR, aPTT)	Normal	-	-	Within normal limits, suggesting no overt coagulopathy at the time of admission.
Arterial Blood Gas (on supplemental O_2)				
pH	7.37	-	7.35 - 7.45	Normal acid-base balance.
pCO_2	42	mmHg	35 - 45	Indicates adequate ventilation.
pO_2	68 ↓	mmHg	80 - 100	Low (Hypoxemia); reflects a significant V/Q mismatch from severe aspiration pneumonia.

Once a deep plane of anesthesia was achieved (end-tidal sevoflurane 3.0%), confirmed by central pupillary position and loss of the eyelash reflex, direct laryngoscopy was performed with a Miller size 0 blade. A Cormack-Lehane grade I view was obtained. A 3.0 mm uncuffed ETT was gently advanced 2 cm through the vocal cords. The ETT was purposefully advanced into the right mainstem bronchus and then withdrawn slowly until bilateral breath sounds were confirmed by auscultation, ensuring the tip was positioned above the carina but below the glottis. ETT placement was further confirmed by a persistent capnography waveform, all while the patient continued to breathe spontaneously. The ETT was secured at 9 cm at the lip with its bevel facing anteriorly, a crucial maneuver to direct the tip away from the posteriorly located fistula opening. Anesthesia was maintained with sevoflurane (1.5-2.0 MAC) in an air/oxygen mixture (FiO_2 0.5). Analgesia was provided with fentanyl (2 mcg/kg bolus, followed by 1 mcg/kg/hr infusion). The patient was positioned in the left lateral decubitus position. A right extrapleural thoracotomy through the fourth intercostal space was performed to minimize pleural contamination in the setting of pneumonia.

The surgeon identified a 3 mm fistula located approximately 1 cm proximal to the carina. There was significant inflammatory adhesion of the surrounding tissues. The fistula was meticulously dissected and suture-ligated with 5-0 polydioxanone suture (PDS) before being divided. After fistula ligation, ventilation was transitioned to pressure-controlled ventilation (PCV) with a peak inspiratory pressure of 18 cmH_2O and PEEP of 5 cmH_2O.

The upper esophageal pouch was mobilized for approximately 1.5 cm to achieve a tension-free anastomosis. A single-layer, end-to-end esophageal anastomosis was performed using 12 interrupted 5-0 PDS sutures. A 5 Fr trans-anastomotic feeding tube

was passed by the surgeon into the stomach. An 8 Fr chest tube was placed in the extrapleural space. Two transient desaturation events occurred. The first (SpO_2 to 85%) was during surgical retraction of the right lung. This was managed by immediate communication with the surgeon, who released the retractor, allowing for manual lung recruitment, and a temporary increase in FiO_2 to 1.0. The second (SpO_2 to 88%) occurred post-anastomosis and was attributed to atelectasis, resolving with an increase in PEEP to 6 cmH_2O. Hemodynamics remained stable throughout the 150-minute procedure. Estimated blood loss was less than 10 mL (Figure 1).

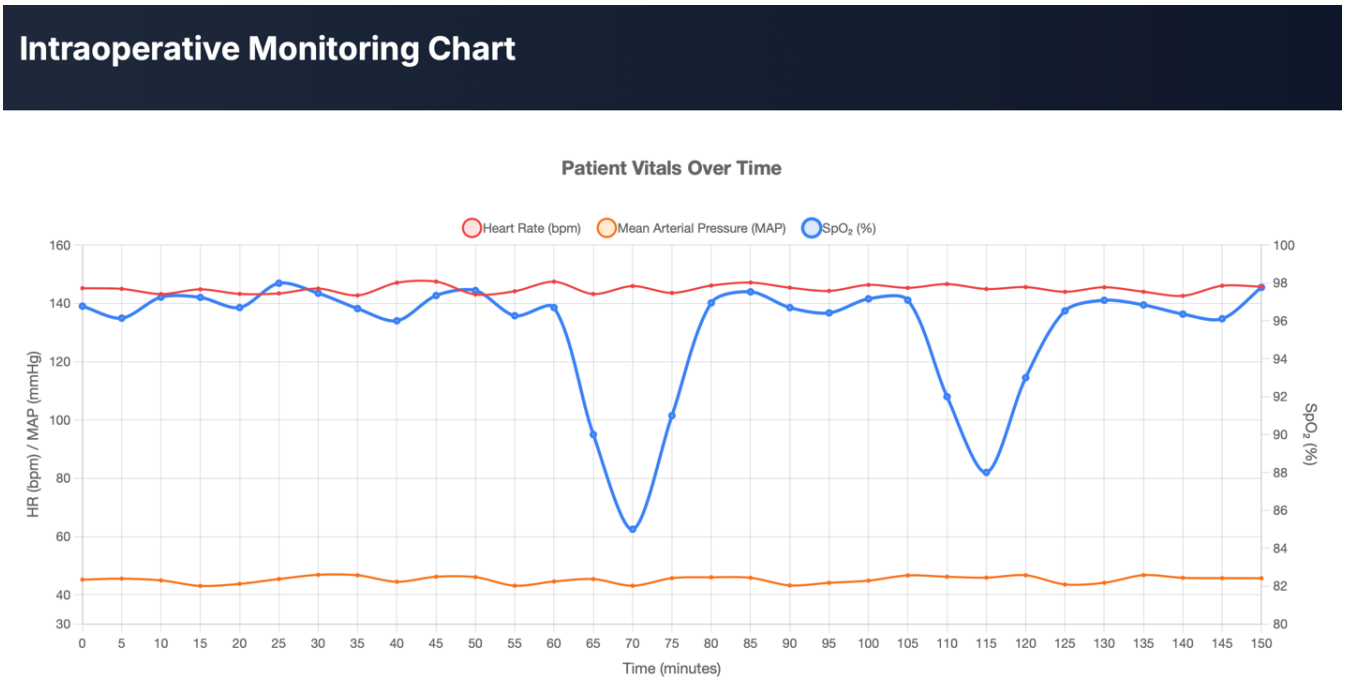

Figure 1. Intraoperative monitoring chart.

The infant was transported intubated to the NICU and was successfully extubated on postoperative day 4. Enteral nutrition via the TAT was started on day 5. The chest tube was removed on day 6. A contrast esophagogram was performed on postoperative day 16, a delay based on institutional protocol for high-risk anastomoses to allow for maximal tissue healing. The study showed a patent anastomosis with no leak. Oral feedings were initiated, and the infant was discharged on day 25. The patient has been enrolled in our institution's multidisciplinary long-term follow-up program for surveillance of potential complications, including gastroesophageal reflux disease, anastomotic stricture, and tracheomalacia. A postoperative esophagogram performed on day 16 showed a smooth, uninterrupted flow of contrast medium through the esophagus and into the stomach. The image demonstrates a widely patent esophageal anastomosis (arrow) with no evidence of contrast extravasation to indicate a leak or residual fistula.

3. Discussion

This case report presents a successful multidisciplinary approach to one of the most challenging scenarios in pediatric anesthesia and surgery: the delayed repair of a Type C EA/TEF in a critically ill neonate with severe cardiorespiratory comorbidities. The favorable outcome was contingent upon meticulous preoperative optimization and a precisely executed perioperative plan, for which the

anesthetic airway strategy was the undeniable cornerstone.[11] This discussion will focus on the complex interplay of pathophysiology that dictated this approach, the specific rationale for the techniques employed, and the integration of anesthetic and surgical management.

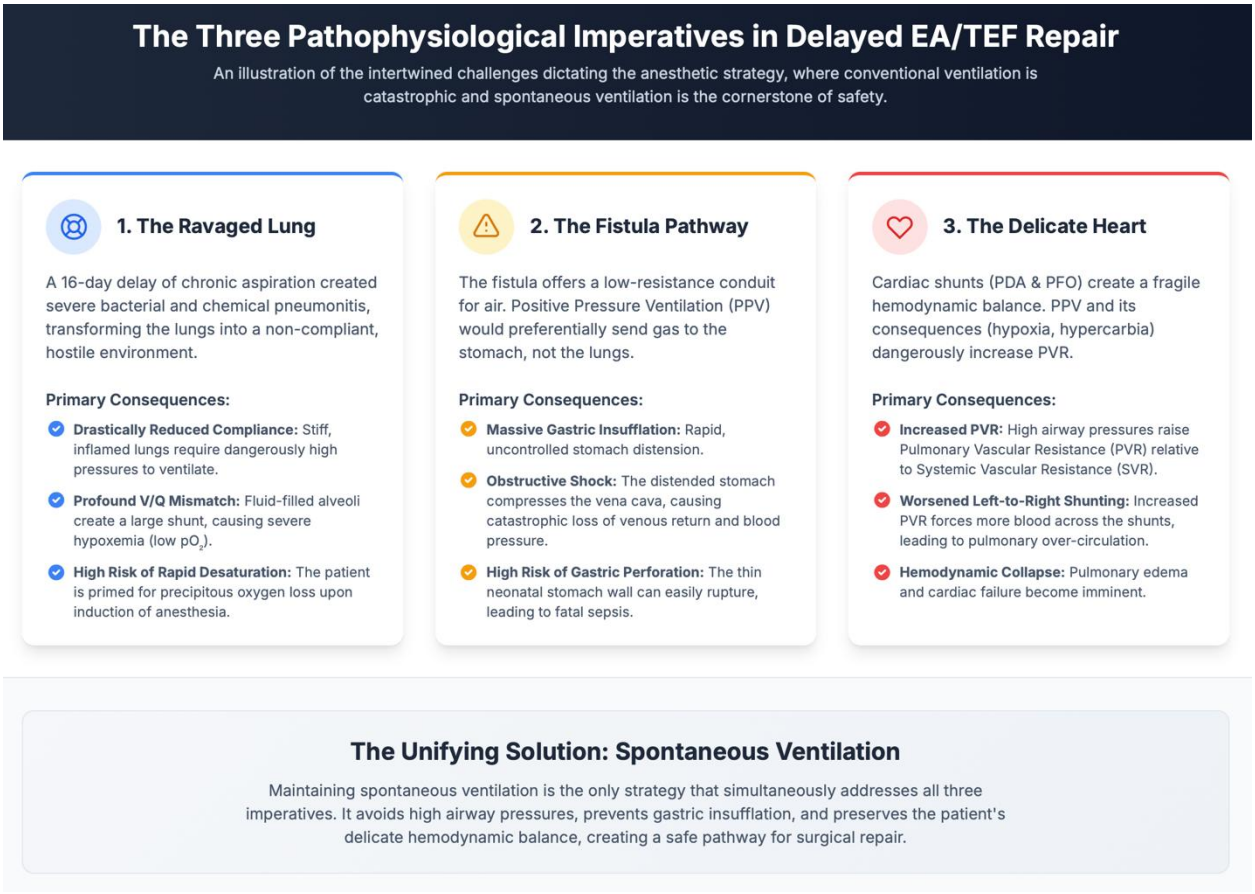

Figure 2. Pathophysiological imperatives in delayed EA/TEF.

In the high-stakes theater of neonatal surgery, every decision is weighed against the gossamer-thin margins of physiological reserve. The case of a 16-day-old neonate with a delayed diagnosis of a Type C tracheoesophageal fistula (EA/TEF) represents an apotheosis of this challenge. The choice of anesthetic technique for securing the airway was not a matter of academic preference or technical elegance; it was a mandate dictated by a cascade of life-threatening pathophysiological derangements that had been amplified by the prolonged delay in diagnosis. This was a clinical scenario where the standard approach to anesthesia—neuromuscular blockade followed by positive pressure ventilation (PPV)—was not merely risky but almost certainly catastrophic. The decision to proceed with an inhalational induction on a spontaneously breathing patient was, therefore, the cornerstone of the entire perioperative strategy, a deliberate and calculated maneuver designed to neutralize three intertwined and formidable threats: a severely compromised pulmonary system, a low-resistance fistula primed for iatrogenic disaster, and a fragile, unstable neonatal circulatory system.[12]

The 16-day delay in diagnosis had transformed the patient's lungs into a hostile environment for anesthesia. For over two weeks, a silent and relentless cycle of aspiration had been underway. Saliva and attempted feeds, unable to drain into the stomach,

pooled in the blind upper esophageal pouch and inevitably spilled over into the trachea.[13] This chronic contamination had two devastating consequences. First, it induced a severe chemical pneumonitis, an inflammatory reaction to the acidic and enzymatic content of the aspirate, which damages the delicate alveolar-capillary membrane. Second, this damaged tissue became a fertile ground for infection, culminating in a bacterial pneumonia, in this case caused by *Klebsiella pneumoniae*.

By the time of surgery, the patient's lungs were stiff, inflamed, and poorly compliant. Pulmonary compliance—the ability of the lungs to stretch and expand—was drastically reduced, meaning that a much higher pressure would be required to deliver a given volume of air.[14] More critically, this widespread inflammation and fluid-filled alveoli created a profound ventilation-perfusion (V/Q) mismatch. Large areas of the lung were still being perfused with blood but were no longer being effectively ventilated, acting as a functional right-to-left shunt where deoxygenated blood passed into the systemic circulation without picking up oxygen. This was evidenced by the patient's preoperative hypoxemia (pO_2 of 68 mmHg) even with supplemental oxygen. This precarious state primed the neonate for precipitous and profound desaturation upon the induction of anesthesia. The vasodilatory effects of anesthetic agents would worsen the V/Q mismatch, and any period of apnea, however brief, could lead to a rapid spiral into severe hypoxia and cardiac arrest. A conventional induction with PPV would have required high inspiratory pressures to ventilate the non-compliant lungs, further increasing the risk of barotrauma and hemodynamic instability.[15]

The anatomical reality of the Type C fistula—a direct communication between the trachea and the distal esophagus—presented the most immediate and dangerous iatrogenic risk. In a normal airway, the glottis and larynx provide the primary resistance to airflow. In this patient, the fistula offered a wide-open, low-resistance conduit directly to the stomach. Air, like any fluid, follows the path of least resistance. Applying positive pressure via a face mask or after intubation would preferentially direct the majority of the gas flow not into the stiff, non-compliant lungs, but through the fistula and into the stomach.

The consequences of this would be immediate and disastrous, leading to a condition known as massive gastric insufflation.[16] The stomach would rapidly distend like a balloon, triggering a cascade of secondary effects. Firstly, the ballooning stomach would splint the diaphragm, physically preventing its downward excursion and thereby severely impeding any attempt at ventilation, exacerbating the already critical pulmonary situation. Secondly, the massively enlarged stomach and intestines would compress the great vessels within the abdomen, particularly the inferior vena cava, drastically reducing venous return to the heart. This collapse in preload would lead to a state of obstructive shock, causing profound hypotension and cardiovascular collapse. Thirdly, the neonatal stomach wall is thin and fragile. The relentless increase in intragastric pressure could easily exceed the perfusion pressure of the gastric mucosa, leading to ischemia and, ultimately, iatrogenic gastric perforation—a surgical catastrophe that would spill gastric contents into the peritoneum, causing overwhelming sepsis and almost certain mortality. The strategy of maintaining spontaneous ventilation completely sidesteps this threat. By allowing the patient to breathe for themselves, airway pressures remain negative during inspiration, precluding any significant gas flow through the fistula and keeping the stomach decompressed.[17]

Compounding the respiratory and gastrointestinal risks was the patient's delicate cardiac physiology. The echocardiogram revealed a 2.5 mm patent ductus arteriosus (PDA) and a 3 mm patent foramen ovale (PFO), both with left-to-right shunting. In the neonatal period, the circulatory system is in a transitional state. Pulmonary vascular resistance (PVR), which is high in utero, begins to fall, while systemic vascular resistance (SVR) rises. The balance between these two resistances dictates the direction and magnitude of blood flow through any existing shunts. The clinical goal in this patient was to maintain a state where SVR

remained significantly higher than PVR. This would limit the volume of the left-to-right shunt, preventing the lungs from being flooded with excess blood flow (pulmonary over-circulation), which would lead to pulmonary edema, worsening respiratory failure, and eventually, cardiac failure. Nearly every aspect of a conventional anesthetic induction poses a threat to this delicate balance. Factors known to acutely increase PVR include hypoxia, hypercarbia (high CO_2), acidosis, and high mean airway pressures associated with PPV. A conventional approach would risk all of these. In contrast, the spontaneous ventilation technique helped to preserve the physiological SVR/PVR balance. By avoiding high airway pressures and maintaining more stable levels of CO_2, it prevented an iatrogenic rise in PVR. This ensured that the left-to-right shunt did not dangerously increase, thereby protecting both the lungs and the heart from hemodynamic collapse.[18]

It is essential to clarify that the term "awake intubation" is a misnomer in this neonatal context. Unlike the adult technique, which involves topical anesthesia and sedation, this procedure requires inducing a deep plane of general anesthesia using an inhalational agent like sevoflurane. The goal is to render the patient completely unresponsive, ablating the powerful airway reflexes of the larynx to allow for instrumentation, all while meticulously preserving the patient's own respiratory drive. This requires a masterful titration of the anesthetic, navigating the narrow therapeutic window between a depth sufficient for laryngoscopy and the onset of sevoflurane-induced apnea.

While technically elegant alternatives exist, such as placing the endotracheal tube tip distal to the fistula under bronchoscopic guidance or occluding the fistula with a Fogarty catheter, they were less ideal for this specific case. The extensive inflammation and copious secretions would have made bronchoscopy extremely difficult in such a small airway. Similarly, blind placement of a Fogarty catheter carries a significant risk of airway trauma or inadvertent occlusion of the trachea or a mainstem bronchus. In contrast, intubation on a spontaneously breathing patient, while demanding intense vigilance, is a more fundamental skill that directly addresses the primary physiological threat—the avoidance of positive pressure—without reliance on highly specialized equipment.[19]

However, this technique is not without its own perils. The primary risks are laryngospasm, coughing ("bucking") on the endotracheal tube, and apnea. A cough would be particularly devastating, as the sudden spike in intrathoracic pressure would act like a piston, forcing a large volume of air through the fistula into the stomach in an instant. The clinical team was therefore in a state of high alert, with a clear crisis management plan. A syringe containing a rescue dose of succinylcholine (a rapid-onset, short-acting muscle relaxant) was drawn up and immediately at hand to treat intractable laryngospasm. The sevoflurane was administered with extreme care, with the anesthesiologist's hand on the vaporizer dial and eyes fixed on the patient's chest excursions and the monitoring waveforms, making beat-to-beat adjustments to maintain the delicate balance.

The success of this case was a testament to the symbiotic relationship between the anesthetic and surgical teams. A delayed repair presents immense surgical challenges; the tissues are no longer pristine but are inflamed, friable, and bound by adhesions, making dissection treacherous and increasing the risk of a leak from the delicate esophageal anastomosis. The stable intraoperative platform provided by the anesthetic management was the foundation upon which surgical success was built. A calm, decompressed abdomen provided the surgeon with optimal exposure, while stable hemodynamics and oxygenation allowed for the meticulous, unhurried dissection required to create a tension-free anastomosis.

This dynamic interplay was vividly illustrated during the two intraoperative desaturation events. These were not moments of panic, but of focused, high-fidelity communication. Upon seeing the SpO_2 dip to 85%, the anesthetist immediately diagnosed the

cause as mechanical compression from the surgical retractor, clearly communicating, "Retraction is causing atelectasis." The surgeon responded instantly, releasing the pressure, allowing the anesthesiologist to perform a gentle recruitment maneuver and temporarily increase the oxygen concentration. The rapid resolution of the hypoxia was a direct result of this seamless, coordinated response. This level of collaboration is not a luxury but an absolute necessity when managing one-lung ventilation in a neonate with pre-existing, severe lung disease. Furthermore, the surgeon's decision to use an extrapleural approach—dissecting outside the pleural lining—was synergistic with the overall goal of minimizing physiological insult. This technique prevents contamination of the pleural space, reducing the risk of empyema and potentially lowering postoperative pulmonary morbidity, aligning perfectly with the protective anesthetic strategy. In conclusion, the entire perioperative course was a masterclass in proactive risk mitigation, where a deep understanding of the patient's complex pathophysiology informed every decision, from the choice of induction technique to the specific surgical approach, culminating in a successful outcome for one of the most fragile of patients.[19,20]

The primary limitation of this report is that it describes the outcome of a single case. The success of this strategy cannot be generalized to all patients and is highly dependent on the experience and skill of the clinical team. Furthermore, our institutional protocol for a delayed postoperative esophagogram, while chosen for safety in this high-risk patient, deviates from more common early-study protocols, and its impact on outcomes like length of stay cannot be determined from this case alone.

4. Conclusion

The management of a neonate with a delayed presentation of EA/TEF and severe cardiorespiratory comorbidities epitomizes a high-stakes clinical challenge requiring an integrated, multidisciplinary approach. This case provides a detailed account of a successful strategy where securing the airway via direct laryngoscopy while meticulously maintaining spontaneous ventilation served as the definitive safety maneuver. This technique directly neutralizes the immediate, life-threatening risks of conventional induction by preventing gastric insufflation and preserving hemodynamic stability. When combined with aggressive preoperative optimization and collaborative intraoperative management, this anesthetic cornerstone provides the critical foundation required to achieve a successful surgical outcome in the most fragile of neonatal patients.

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