

---

# Anesthetic Management of Neonatal Emergency Abdominal Surgery

---

Esra Caliskan

Additional information is available at the end of the chapter

<http://dx.doi.org/10.5772/63567>

---

## Abstract

Anesthetic management of neonatal surgical emergencies presents serious difficulties and challenges for the anesthesiologist and is associated with increased risks of morbidity and mortality. Therefore, in these patients, anesthetic assessment should be directed to the patient's current disease and associated anomalies. The provision of safe anesthesia and perioperative management of these cases also depends on a clear understanding of the physiologic and hemodynamic variables of the preterm and term infants and a precise knowledge of the pathophysiology of disease states likely to affect the neonate. This chapter describes physiology and pathophysiology-based view on the neonatal abdominal emergency surgeries. General principles of surgery and anesthetic management of specific neonatal emergencies that include congenital diaphragmatic hernia (CDH), abdominal wall defects, necrotizing enterocolitis, and esophageal atresia are reviewed and discussed.

**Keywords:** neonatal, emergency abdominal surgery, pediatric anesthesia, neonatal physiology, perioperative anesthetic management

---

## 1. Introduction

Advances in obstetrics and neonatology have greatly improved neonatal survival especially for infants with complex anatomical defect and preterm neonates. Most of these anatomical abnormalities to be corrected by surgical interventions after birth, some of them also corrected by intervention before birth [1, 2]. Advances in prenatal diagnosis and survival rate in these patients have resulted in an increase in the anesthetic and surgical approach. Emergency surgical conditions can be life threatening in the neonatal period. Therefore, anesthesiologists

must know and better understand the distinctive physiological characteristics of neonates than of adults so that they can have information about response to anesthesia and surgery.

The purpose of this chapter is to provide basic information that will help anesthesiologists to better approach the problems of neonates. The present considerations recommend that to provide safe care these patients are also reviewed.

In this context, anesthetic management will be presented in selected neonatal surgical emergencies including congenital diaphragmatic hernia (CDH), abdominal wall defect (gastroschisis and omphalocele) necrotizing enterocolitis, and esophageal atresia. Clinical presentation and associated anomalies, perioperative approach, surgical stabilization, and postoperative follow-up period are explained and discussed.

## 2. Physiological features in neonates

### 2.1. Respiratory system

Lung development can be divided into five stages [3]. In literature and textbook knowledge reviewed, the last stage begins at about 36 weeks' gestation and continues until at least 18 months [4]. Tip II pneumocytes are responsible for surfactant production. These begin to differentiate at 24 weeks' gestation, but surfactant level is not adequate to ensure appropriate pulmonary function until 34–36 weeks of gestation [4, 3]. Therefore, respiratory immaturity and potential for respiratory distress are well-defined entities in the preterm neonate due to a deficiency of surfactant.

One of the characteristics of the fetal circulation is high pulmonary vascular resistance (PVR) [3]. PVR reduces by approximately 10% after labor, but the apparent decrease occurs with the first breath [5]. Pulmonary blood flow and alveolar and arterial  $PO_2$  increase along with the expansion of the lung [5]. These changes are important for modification of the fetal circulation to the adult form of circulation [3, 6]. Over the next 1–2 months, PVR decreases to adult levels.

The differences in the neonatal respiratory system from that of the adult can be summarized as follows:

- Central control of ventilation is not fully developed.
- Response to hypoxia is immature.
- Peripheral feedback mechanisms are not sufficiently mature.
- Neonatal oxygen consumption per kilogram of body weight is twice as much as an adult.
- Lower functional residual capacity (moderate decrease in value of inspired oxygen or ventilation can cause rapid desaturation).

Immature airway and respiratory system can lead to airway obstruction during sedation or mask ventilation. In addition to this, diaphragmatic movement associated with gastric distension can also trigger significant respiratory failure. Infection, airway obstruction,

hypoventilation, hypoxemia, and sepsis are among the reasons of apneic episodes [7]. Therewith, anemia may precipitate postoperative apnea and bradycardia in neonate too. Prolonged period of apnea results in hypoxia and hypercapnia. Our current knowledge shows that the risk of postoperative apnea continues in expremature infants until 60 postconceptual weeks [8]. Frequently, preterm infants are victims of respiratory failure due to the immature and sensitive respiratory structures. Many preterm infants needed increased (mechanical ventilatory support or CPAP, supplemental oxygen, etc.) respiratory support to preserve fundamental oxygenation. In hypoxic circumstances, respiratory rates of preterm and newborns decrease paradoxically.

Numerous neonatal emergency abdominal surgeries can be seen associated with respiratory system insufficiency. Respiratory variables, which are mentioned earlier, must be taken into consideration in these cases.

## **2.2. Cardiovascular system**

The circulation of a newborn infant is a transitional characteristic. This means that, a significant decrease in pulmonary vascular resistance after birth. Patent ductus arteriosus (PDA) on the side interventricular shunts and connections are responsible from this circulation.

The fetal circulation is defined by increased PVR, decreased pulmonary blood flow, low systemic vascular resistance, and right-to-left blood flow through ductus arteriosus and foramen ovale [9, 3]. The onset of spontaneous breathing is the main factor that reduces PVR. This reduction of PVR is followed by an increase in systemic vascular resistance and left atrial pressure. Extrauterine circulation changes to a serial type of circulation as an adult.

Physiological closure of ductus arteriosus may take up to first 24 h after birth in healthy infants. However, ductus arteriosus closes anatomically after several days. Different pathological situations may cause shunting of blood via this structure. Anatomic closure of foramen ovale occurs between 3 months and 1 year of age. However, functionally closure takes place immediately. Foramen ovale may persist in 10% of the adult patients.

Reduction of pulmonary blood flow or pulmonary vasoconstriction can cause a return of the fetal circulation characteristics. This circulatory pattern is called persistent fetal circulation (PFC) or persistent pulmonary hypertension of the newborn (PPHN) [10]. Hypoxemia and acidosis can aggravate PFC. In infants with CDH, meconium aspiration and infection may be seen as PFC.

Therefore, neonatal myocardium is less compliant, and newborn's cardiac output is heart-rate dependent. The sympathetic innervation of the developing heart is decreased. In such a case, neonatal heart variable responds to vasoactive drugs.

## **2.3. Thermoregulation**

The newborn baby has a high ratio surface area to body weight and a thin layer of subcutaneous fat tissue. Therefore, they are prone to lose heat more than older children and adults. Heat loss

in newborn occurs with four different mechanisms, including evaporation, convection, conduction, and radiation.

Nonshivering thermogenesis is a primary mechanism in heat loss and regulated by brown fat [11]. Brown fat is a located adipose tissue settled in the posterior neck along the interscapular and vertebral areas.

For the reason mentioned earlier, severe temperature changes can be seen in the perioperative period. Hypothermia may trigger myocardial depression, acidosis, and delayed recovery from anesthesia. Maximal precautions should be taken to prevent hypothermia in perioperative period. Applications, which required to be done to prevent heat loss, are listed below.

- Keep the ambient temperature in neutral condition.
- Humidify and warm inspired anesthetic gases.
- Use the heated blanket, radiant warmer, and transport the neonate in a heated incubator.
- Warm blood and intravenous solutions.
- Use heating solution for cleansing the skin.

### 3. General principles of anesthesia in neonate

Anesthetic management of neonates that require emergency surgery is challenging and may cause serious problems to the anesthesiologist. The identification of patients at high risk for procedure-related morbidity and mortality should be a basic preventive effort. The practice of a safe anesthesia and perioperative management in such patients depends on a clear understanding of the anatomical, physiological, and hemodynamic differences and variables.

Our knowledge about pain perception in newborns has changed in recent years. Unlike the historical information, premature and term infants can feel pain from painful procedures. In recent literatures and studies, it is shown that preterm and term infants have a perception of pain, and these infants give physiological responses (such as autonomic, endocrine, and metabolic) to painful stimuli [12]. These stimulus and stress can increase pulmonary vascular resistance ergo, increase shunting through the foramen ovale, and ductus arteriosus. This phenomenon may result in hypoxemia.

Because of this, a detailed anesthesia plan should be developed for safe patient care, effective pain control, and outcome in perioperative period.

#### 3.1. Effects of anesthetic agents

The effects of anesthetic agents vary with physical growth and changes, organ maturation, development, and coexisting disease in pediatric patient population. Inhalation anesthesia is the commonly used anesthetic technique in children. In infants, minimal alveolar concentra-

tion (MAC) is an estimate of anesthetic requirements and changes with age [8, 11]. Infants up to 6 months of age require higher MAC value than older neonates [8]. Volatile anesthetics cause dose-dependent cardiac and respiratory depressant effect in neonates. These effects can cause bradycardia, hypotension, and postoperative apnea. This hypotensive effect is more likely in preterm and term neonates than older children, especially in the induction period.

In contrast, an opioid agent causes fewer hemodynamic changes, but it should be noted that increase the risk of postoperative apnea in preterm and term neonates [2].

In the last 15 years, effects of general anesthetic agents on brain development have made a great progress. The recent animal studies have demonstrated that general anesthetics (especially these acting through the N-methyl-D-aspartate (NMDA) and Gama aminobutyric acid (GABA) receptors) can be responsible from neuronal cell death and deterioration of brain development especially immature brain [13]. It is reported that this effect is to be related to drug dosages and exposure time. Many factors affect brain maturation apart from anesthesia. Dinardo et al. emphasized that neuroanatomic abnormalities preexist in neonatal patients with complex congenital cardiac pathologies. The retardation of brain maturation has been observed in these patients [14]. Long-term neurobehavioral effects of sedative and anesthetic agents should be supported by long-term human studies.

### **3.2. Monitoring**

Basic monitorization should include heart rate-electrocardiograph, pulse oximetry, blood pressure, and temperature in neonates. Two oxygen saturation probes must be plugged (preductal and postductal probes). This approach is intended to evaluate possible PDA-mediated shunt development. Invasive arterial blood pressure and central venous pressure can be monitored in special circumstances such as expected ventilation-perfusion and hemodynamic changes and acid-base imbalance.

Precordial-esophageal stethoscope is a very useful method in neonatal monitoring. With this method, changes in heart rate and respiratory parameters can be identified in the early phase. Although end-tidal carbon monoxide monitorization is a routine application, increase in dead space in the breathing system can cause false and inaccurate measurement in infants.

## **4. Anesthetic management of special neonatal surgical emergencies**

### **4.1. Congenital diaphragmatic hernia**

CDH is one of the complex and compelling anomalies in the neonatal period. CDH occurs in about 1:2400 and 1:3000 live births [8, 15]. Incomplete formation of the diaphragm and the inability of the intestines to return to the abdominal cavity result in CDH. Posterolateral foramen originated hernia (foramen of Bochdalek) constitutes 90% of the cases. In patients with CDH because of the displacement of the abdominal contents until the mediastinum, pulmonary hypoplasia (unilateral or bilateral) and possible mediastinal shift are serious

problems. Less than 15% of the remaining lung volume for gas exchange indicates a poor prognosis.

Clinical presentation of CDH includes a scaphoid abdomen, increased anteroposterior diameter of the chest, bowel sounds during chest auscultation, and respiratory distress in variable degrees. Newborn babies with CDH have a 25–30% incidence of cardiac anomalies [11, 15]. Often, gastrointestinal malformations associated with skeletal abnormalities are also possible.

In radiological imaging of the chest, bowel loops in the thoracic cavity and mediastinal shift (due to compression of the lungs) are observed. Therewithal, diagnosis can be made through ultrasonography in utero, but this diagnosis should be confirmed in the postpartum period.

#### *4.1.1. Preoperative evaluation*

In recent years, one of the strongly highlighted issues in CDH is the timing of surgery. There is considerable argument about this topic. Current approaches, after corrective surgery, are intended to provide cardiopulmonary stabilization. CDH study group reported that only one-third of the patients underwent surgical correction in the first 24 h of life in the mid-1990s. Nowadays, the number of patients who underwent surgery within the 24 h is less than 5% [15, 16]. Because of this, it is recommended that surgical repair should be postponed until after initial resuscitation provides preoperative stabilization.

#### *4.1.2. Preoperative ventilation strategies*

The main point in the protective efforts for patients with CDH is minimizing the physiologic instability associated with pulmonary hypoplasia and pulmonary hypertension in the postnatal period. Developing ventilator strategies have played an important role in terms of reducing mortality in the last 20 years.

After delivery (in a neonate who is diagnosed during the intrauterine period) immediate endotracheal intubation (to abstain from positive pressure ventilation and pulmonary barotrauma), ventilation with small tidal volumes, and increased respiratory rates (especially in the presence of pulmonary hypoplasia) are important initial ventilatory maneuvers [8, 15, 16]. Nasogastric tube assists in decompression of gas content of the bowel and protects the lung volume.

Inflating pressures must be kept below 30 cm H<sub>2</sub>O with a continuous monitoring of airway pressure [2]. Pulmonary barotrauma may result in serious complications such as pneumothorax, air embolism, pneumomediastinum, and soft tissue emphysema.

In the past years, perioperative management of these patients was intended to maintain adequate oxygenation and prevention of pulmonary hypertension. As hypoxemia and hypercarbia may increase pulmonary vascular pressure, hyperventilation (to lower PCO<sub>2</sub>) of high oxygen levels was used. However, to reduce the risk of barotrauma and high mortality incidence, today with a gentle approach, permissive hypercapnia and lower percentage of oxygen are preferred [2, 15].

On this basis, the general approach in preoperative period should include reduction of pulmonary vascular resistance, recovery of pulmonary compliance, minimal ventilatory support, and regression of radiological changes.

#### *4.1.3. Different presurgical therapy modalities*

##### *4.1.3.1. Extracorporeal membrane oxygenation*

Some neonates show rapid clinical improvement, if surgery is performed within 48 h. However, some patients may require hemodynamic, respiratory, and medical supportive therapy before definitive surgery. Extracorporeal membrane oxygenation (ECMO) is one of these treatment procedures.

The first reports about ECMO in neonates with CDH were declared in the late 1970s [17]. Oxygenation index (OI) $>40$ , PaCO<sub>2</sub> consistently  $>12$ , and A-a gradient consistently  $>600$  mmHg were accepted indications for initiation of ECMO in the past [18]. However, nowadays, the indications for ECMO use can be summarized as unresponsiveness to treatment. Currently, more than 30% of CDH neonates are now being managed with ECMO, and it is frequently used in patients with severe reduction of pulmonary volume before surgery [2, 19].

Nonetheless, ECMO should not be used in the presence of different serious clinical cases such as intracranial hemorrhage  $>$  Grade I, presence of accompanied congenital anomalies, fatal chromosomal abnormalities, and gestational age  $<34$  weeks [17].

Summarization—ECMO should be considered only on those infants who cannot be treated with conventional medical therapy. So ECMO is used as an additional therapy for patients who have resistant hypoxemia despite inotropic and ventilatory support.

##### *4.1.3.2. Ex utero intrapartum treatment (EXIT) procedure*

EXIT is an intrauterine treatment procedure in some cases of CDH. In this procedure, after fetal head removal from the uterus, fetus is intubated for assessing oxygenation or connected to the ECMO prior to umbilical cord clamping. In literature, some cases have been reported where fetal gas exchange is supported by ex utero placental circulation for over 60 minutes [2].

##### *4.1.4. Surgical approach and intraoperative consideration*

In the conventional approach, diaphragmatic defect is repaired via a thoracotomy incision on the ipsilateral side of the hernia. Thoracic approach is preferred in some cases. During the operation, diaphragmatic defect is repaired depending on the size of the defect. Small defects were closed primarily in which large patch graft may be required. After the closure of abdominal viscera, intraabdominal pressure may increase excessively. This clinical situation can worsen respiratory insufficiency and higher ventilation pressure should be required in the intraoperative and postoperative period. But pulmonary barotaruma should be avoided in this period.

#### 4.1.5. Postoperative period

Severity of pulmonary hypoplasia and hypertension is an important factor that affects overall survival. Several factors affect pulmonary compliance after emergency surgery. These factors (such as increased intraabdominal pressure, lung hypoplasia, and postoperative painful stimulus) frequently cause rapid decrease in compliance. Opioids can reduce the stress response and catecholamine release in response to painful stimuli. In addition, opioids provide effective pain control so decrease the pulmonary vasoconstrictor responses to painful stimuli in neonatal intensive care unit (NICU) [2, 16].

### 4.2. Abdominal wall defect

#### 4.2.1. Gastroschisis and omphalocele

Gastroschisis and omphalocele are characterized by congenital defect in the abdominal wall and herniation or evisceration of abdominal viscera [2, 20, 21]. The incidence of these defects is 1:3000 to 10000 live births [2].

Gastroschisis is defined by the absence of a division in the right abdominal wall if omphalocele is a periumbilical defect of the abdominal wall and a majority in male neonate [2]. Current knowledge supports the fact that omphalocele and gastroschisis arise from separate embryologic processes. Herniation of abdominal contents overlaps with a translucent lamina of peritoneum. The characteristic of the lesion is amniotic membrane. In addition, the intestines, spleen, and liver may be located in a large defect. Both clinical cases should require emergency surgical approach due to rapid and substantial heat and fluid losses by extra abdominal organs (especially bowels). Some congenital anomalies are often associated with omphalocele such as trisomies, congenital cardiac anomalies (e.g., Fallot tetralogy), Bechwith-Wiedemann syndrome, malrotation of the intestine, chromosomal anomalies, and meningocele [21, 2]. Prematurity is an important factor that increases mortality in these patients.

#### 4.2.2. Preoperative period

Although each of the lesions is a different etiology, approaches are similar for both the pathologies. The preoperative fundamental approach to abdominal wall defects primarily includes adequate fluid resuscitation to prevent heat loss and treatment of infection and sepsis to avoid direct trauma to the eviscerated organs [11]. In both the cases for the prevention, the defect (especially large defect) is covered with sterile wet dressing [22].

Dense fluid replacement therapy is required and is vital for these patients. The stomach should be decompressed with a nasogastric tube. This process is intended to reduce regurgitation, aspiration, and bowel distention. Systemic antibiotics should be started primarily. Intense protein loss and translocation of fluid into third space can be seen in ruptured cases. Oncotic pressure is also decreased. Arterial blood gas analysis should be performed.



#### 4.2.3. *Intraoperative management*

Major issues that we need to pay attention in preoperative approach are also important in the intraoperative period. The maintenance of body temperature and adequate fluid resuscitation should be noted. Because of this, adequate intravenous access must be ensured and invasive monitoring is necessary if an associated cardiac defect is present.

Surgical intervention is required to reduce the bowel back into the abdominal cavity and repair the abdominal wall defect. Different surgical techniques (such as primary closure in the operating room or bedside, placement of a prefabricated silastic silos or fashioning of a hand-sewn silo) are applicable [23]. Silo method is a transition repair procedure. Some infants may remain intubated until final closure in the NICU setting. Nowadays, the sutureless umbilical closure has become a widely accepted technique [24]. In the renewed procedure, herniated viscera are reduced into the abdomen. Umbilical cord is used to pack the abdominal wall defect. This technique may be implemented at the bedside in the neonatal intensive care unit. Nevertheless, in patients with larger defect, surgery should be performed in the operating room. Anesthetic management consists of endotracheal intubation in the awakened patients with gastric decompression after preoxygenation. Anesthesia is maintained with inhalational anesthetics and controlled ventilation. Muscle relaxants may be used to provide optimal surgical circumstances for closure of the defect.

It must be kept in mind that if abdominal defects are closed too tightly, abdominal pressure can increase because of underdeveloped abdominal cavity. This situation can lead to respiratory failure, diminish visceral organ and lower extremity perfusion, and decrease venous return due to caval compression [8]. In this situation, postoperative ventilatory support may be necessary. In patients with associated anomalies, close monitoring (especially airway pressure and central venous pressure) is vital and invasive blood pressure monitorization and arterial blood gas analysis may be useful and observational hemodynamic parameters provide optimal oxygenation and fluid therapy. Respiratory parameters should be monitored closely after surgery. Postoperative mechanical ventilatory support is recommended during 24–48 h in NICU. Prolonged ileus after surgery may cause parenteral nutrition in a longer period.

#### 4.3. **Necrotizing enterocolitis**

Necrotizing enterocolitis (NEC) is one of the important cause of morbidity and mortality that particularly affects premature infants under 36 weeks of gestational age (usually premature infants fewer than 32 weeks) [2, 16, 21]. The causes of NEC are still unclear. However, the etiology of NEC is considered multifactorial. Prematurity is a definite risk factor mainly affecting premature infants fewer than 32 weeks' gestation and body weight is less than 1500 gr. In addition, hypoxemia, enteral feedings (especially hyperosmolar solutions), cyanotic heart disease, and PDA have been suggested as risk factors. Although the etiology is different, the result is increased mucosal permeability, intestinal ischemia, and sepsis.

Clinical features of these patients are characterized by abdominal distention, retained gastric secretion, bloody stool, and bile-stained aspirates [2, 25]. In some cases, irritability, lethargia, apnea, and temperature instability may accompany this situation. Hypovolemic shock can be

seen in cases with generalized peritonitis. A hemorrhagic diathesis with thrombocytopenia may emerge in septic infants. Many of these infants who have developing respiratory insufficiency and hyaline membrane syndrome require intubation and mechanical ventilation support.

It is important to know the risk factors, and early diagnosis and initial treatment in these patients are also necessary. Some cases including less severe ones can be treated with broad-spectrum antibiotics, intravenous fluids, and nasogastric decompression. However, acute phase of NEC required emergency surgery, and pneumoperitoneum is an absolute indication [25, 26]. Positive paracentesis, constant abdominal mass, fixed dilated intestinal loop, and progressive metabolic acidosis are among relative surgical indications [2, 16, 21].

#### *4.3.1. Intraoperative management*

Intraoperative approach in patients with NEC should contain close monitoring of invasive arterial and central venous pressure, blood gas, and metabolic analysis. Blood products (such as red blood cells, fresh frozen plasma, and platelets) may be required in infants with coagulopathies in the early period. In addition, severe hypovolemia is a significant problem in these infants and requires intensive treatment with crystalloid and colloid solutions.

Potent inhalational anesthetics can aggravate a hypovolemic situation. Because of this, frequently inhalational anesthetics are utilized in low concentration to supplement opioids. High-dose opioid (especially fentanyl) is a safe and viable option for balanced anesthesia [8]. Nitrous oxide should not be used. Because of the risk of premature retinopathy, inspired oxygen concentration should be calibrated to keep between an arterial oxygen saturation of 85–90% [16].

Neuromuscular blockers may be used to facilitate surgical intervention. Hemodynamic collapse is often in these newborns. In this regard, despite fluid resuscitation in patients who are not provided adequate perfusion, inotropic agents are often necessary to protect cardiovascular stability. Glucose intolerance is a significant problem particularly in septic patients. Intermittently, arterial blood gas analysis and glucose measurement is an important measurement parameter.

#### *4.3.2. Postoperative consideration*

These infants often require postoperative mechanical ventilatory and cardiovascular support. Parenteral nutrition is necessary after sepsis is controlled and metabolic stability is maintained [20].

### **4.4. Esophageal malformations**

#### *4.4.1. Esophageal atresia, tracheoesophageal fistula*

Esophageal atresia is the most common esophageal malformation. The incidence reported is at 1 in 2500–3000 live births [27].

Esophageal malformations can manifest in five different forms according to localization of atresia and presence of fistula. The most common defect (80–90%) consists of a blind, upper esophageal pouch with distal tracheoesophageal fistula.

Severe cardiovascular anomalies (such as ventricular septal defect, coarctation of aorta, and Tetralogy of Fallot,) can be associated with esophageal atresia. This anomaly can also be a part of a syndrome known as VATER [vertebra (V) anal (A), tracheoesophageal (TE), renal (R)] or VATER syndrome associated cardiac (C) and limb (L) anomalies called VACTERL syndrome [8, 27]. These associated anomalies worsen the prognosis.

Prenatal diagnosis can be made with the ultrasound. Inability to swallow, choking attacks, aspiration, and abdominal distention should be preoccupying esophageal atresia in the early postnatal period. The disability of the nasogastric tube to pass through the atretic esophagus passage is also seen in the clinical examination.

Definitive diagnosis is made by contrast radiological examination. Pulmonary aspiration, other causes of abdominal distention, and prematurity-dependent problems should be considered in differential diagnosis. Preoperative echocardiography must be performed owing to the associated congenital cardiac anomalies. Concomitant cardiac anomalies and birth weight are among the factors affecting mortality in patients with Tracheoesophageal Fistula (TEF)/EA.

#### *4.4.2. Anesthetic management*

The operation of TEF is acceptable and urgent, but not immediate, and the time of surgery is determined by the general condition of the infant. Considering the general condition of the patients, surgical approach can be planned—primary closure of the fistula and anastomosis, gastrostomy before or after definitive surgery, or a delayed primary closure [28]. In patients, who provide clinical improvement, surgery can be carried out within 24–72 h [11]. Antibiotic therapy should be initiated immediately in infants with pulmonary infection. In these cases, surgery may be delayed as much as clinically stability. Despite the intensive, care, support, and antibiotic therapy, if there is continuous clinical worsening and respiratory insufficiency, immediate surgery may be necessary.

However, if the scheduled surgery is delayed, usually fistula is closed, and gastrostomy is performed. Gastrostomy is used to decompress the stomach and decrease regurgitation into the lungs. In addition, gastrostomy provides nutritional support in cases in which definitive surgery is delayed. This process can take 3–6 months.

The basic preoperative anesthetic management in patients undergoing surgical approach includes the following:

- Degree of neonatal age (especially prematurity) and associated congenital anomalies
- Interruption of feeding and intermittent aspiration of the upper esophageal pouch
- Prevent excessive gastric distention due to air intake into the stomach through the fistula

- Evaluation of aspiration pneumonia, if necessary, antibiotic therapy to treat pulmonary infection

Before management of anesthesia, aspiration catheter may be useful to drain secretions in the esophagus. In anesthesia, induction should be administered carefully to prevent abdominal distention and pulmonary aspiration. In general, intubation is performed in awake conditions. Sedation can be administered with titrated dose of fentanyl ( $0.5\text{--}1\ \mu\text{g kg}^{-1}$ ) and  $25\text{--}50\ \mu\text{g kg}^{-1}$  midazolam [8]. Also deep inhalational anesthesia to induction may be preferable to endotracheal intubation. In this period, maintenance of spontaneous breathing should be noted. Muscle relaxant may be an addition after the ligation of fistula.

The placement of the endotracheal tube (ETT) is important. ETT tip must be above the carina, but it must be placed below the fistula. It may not be easy to find the correct tube localization. Generally, tube is advanced through the right mainstem bronchus and then pulled back slightly until breath sounds are heard bilaterally. Tube localization can be confirmed by flexible fiberoptic bronchoscope [29]. Even with initial optimal position of the ETT, in some patients, ventilation through the fistula still occurs, and this is particularly seen in patients who require high peak airway pressure. In these cases, the use of low inspiratory pressure will prevent gastric distention until the ligation of the fistula.

In appropriate cases, a primary repair can be performed, the fistula is ligated, and esophagus is primary anastomosed. Intraoperatively, retraction of the lung may be required to provide proper exposure intraoperatively. In this period, deterioration of oxygenation is a common problem due to collapsed lung experience by an anesthesiologist. Intermittent ventilation of collapsed lung and close cooperation of surgical team may improve oxygenation.

In addition, precordial stethoscope should be a part of an intraoperative monitorization during repair of the TEF. Precordial stethoscope is placed in the left axilla to assess the adequacy of ventilation in the dependent lung. In infants with associated anomalies such as congenital cardiac defect or hemodynamic instability, invasive arterial monitoring should be performed.

Two oxygen saturation probes may be helpful to evaluate pre- and postductal oxygen saturation and shunt fraction via patent ductus arteriosus.

#### 4.4.3. Postoperative approach

In patients with TEF, significant pathological finding is a reduced amount of tracheal cartilage. Tracheomalacia or defective tracheal wall can cause airway collapse. Therefore, most patients require postoperative ventilatory support in early postoperative period (at least 24–48 h) in NICU. This period may take up 5–7 days in infants who have long gap anastomosis [8, 16, 29]. Intermittent arterial blood gas analysis and chest radiographs will be useful in the postoperative follow-up period.

Opioid infusion is effective in the treatment of postoperative pain and sedation in this period.

Anastomotic leak and strictures are main complications in early postoperative period. Structural and functional abnormalities (such as gastroesophageal reflux and chronic pulmonary disease) are considerable problems that may be encountered in the long-term period [2, 30].

Therefore, it should be noted that EA or TEF is not a small anatomic problem that completely ameliorates by surgical approach.

## 5. A Special highlight for pediatric spinal anesthesia

In the last 30 decades, pediatric spinal anesthesia has become one of the most attractive and popular topics in anesthesia practice with the increase of our knowledge and experience on this issue. Despite the growing interest, spinal anesthesia requires technical expertise, and it is not still performed in many centers. Notwithstanding, general anesthesia is a standard technique for many pediatric anesthetist.

Spinal anesthesia has become a considerable anesthetic technique for reducing the risk of postoperative apnea in premature and ex-premature infants [31]. Unsupplemented spinal anesthesia may be used in lower abdominal surgery (such as necrotizing enterocolitis, pyloromyotomi, omphalocele, and gastroschisis) in neonatal age group. Abajian et al. in 1984 reported the use of spinal anesthesia in high-risk ex-premature infants and considered regional anesthesia by spinal approach to be safe and effective in these patients [32].

At the same time, spinal anesthesia is characterized by remarkable hemodynamic stability in neonate and infant patients. In reference [33] this physiological characteristic means that minimum or no cardiodepressant effect and is important especially in the neonatal period. Because neonatal heart just after birth is more immature than adult heart and have a lower velocity of contraction and a minimize length-tension correlation.

In infants with limited pulmonary perfusion, it is important to maintain systemic vascular resistance and control of PVR and pulmonary blood flow. Some of these patients require immediate surgical intervention. In these patients, spinal block decreases undesirable adverse effects related to the sympathetic system activity.

Surgical trauma and pain cause a hormonal stress response that is directly related to the severity and urgency of surgery. Spinal anesthesia is an effective pain control technique. Thus, hormonal stress response and catecholamine release reduces in intraoperative and postoperative period [31, 34].

In the previous studies, it has been suggested that when compared with general anesthesia, spinal anesthesia is associated with fewer cardiovascular and respiratory complications, and there is less need for postoperative mechanical ventilation and a shorter hospital stay [35].

Because of these factors, it has to be kept in mind that spinal anesthesia seems reliable and a safe alternative technique in selected cases especially for specific lower abdominal surgery.

## 6. Summary

Preterm and term neonates may need various surgical interventions, but unfortunately, many of them are urgent. Newborn period is a challenging period for the anesthesiologist. This

difficulty is caused by many factors related to neonatal population such as immaturity of physiological processes, organs, and systems, and pathophysiological instability caused by the underlying comorbidities and associated congenital anomalies. For these reasons, anesthesia-associated morbidity and mortality is greater in this group than in older infants and children. A cautious approach to all aspects of perioperative care is vital to minimize the possible side effects. The basics of safe anesthetic management in preterm and term neonates are summarized in this chapter, based on emergency abdominal surgery.

## Author details

Esra Caliskan

Address all correspondence to: [duru5654@gmail.com](mailto:duru5654@gmail.com)

Department of Anesthesiology and Reanimation, Baskent University, Faculty of Medicine, Ankara, Turkey

## References

- [1] Partridge EA, Flake AW. Maternal-fetal surgery for structural malformations. *Best Pract Res Clin Obstet Gynaecol.* 2012; 26:669–682. DOI: 10.1016/j.bpobgyn.2012.03.003.
- [2] Brusseau R, McCann ME. Anaesthesia for urgent and emergency surgery. *Early Hum Dev* 2010; 86:703–714. DOI: 10.1016/j.earlhumdev.2010.08.008.
- [3] Motoyoma EK, Finder JD. Respiratory physiology in Infants and Children. In: Davis PJ, Cladis FP, Motoyoma EK editors. *Smith's Anesthesia for Infants and Children.* 8th ed. Philadelphia: Elsevier; 2011. Pp. 23–41. ISBN: 978-0-323-06612-9. ch3.
- [4] Joshi S, Kotecha S. Lung growth and development. *Early Hum Dev* 2007; 83:789–794. DOI: 10.1016/j.earlhumdev.2007.09.007.
- [5] Finnemore A, Groves A. Physiology of the fetal and transitional circulation. *Semin Fetal Neonatal Med* 2015; 20:210–216. DOI: 10.1016/j.siny.2015.04.003.
- [6] Souza DD, McDaniel GM, Baum VC. Cardiovascular physiology. In: Davis PJ, Cladis FP, Motoyoma EK editors. *Smith's Anesthesia for Infants and Children.* 8th ed. Philadelphia: Elsevier; 2011. Pp. 81–115. ISBN: 978-0-323-06612-9. ch4.
- [7] Sale SM. Neonatal apnoea. *Best Pract Res Clin Anaesthesiol* 2010; 24:323–336. DOI: 10.1016/j.bpa.2010.04.002.
- [8] Cote CJ. Pediatric anesthesiology. In: Miller RD, editor. *Miller's Anesthesia.* 7th ed. Philadelphia: Churchill Livingstone; 2010. Pp. 2559–2597. ISBN: 978-0-443-06959-8.ch.

- [9] Hines MH. Neonatal cardiovascular physiology. *Semin Pediatr Surg* 2013; 22:174–178. DOI: 10.1053/j.sempedsurg.2013.10.004.
- [10] Jain A, McNamara PJ. Persistent pulmonary hypertension of the newborn: advances in diagnosis and treatment. *Semin Fetal Neonat Med* 2015; 20: 262–271. DOI: 10.1016/j.siny.2015.03.001
- [11] Gregory GA, Brett C. Neonatology for anesthesiologist. In: Davis PJ, Cladis FP, Motoyoma EK Editors. *Smith's Anesthesia for Infants and Children*. 8th ed. Philadelphia: Elsevier; 2011, pp. 513–553. ISBN: 978-0-323-06612-9. ch17.
- [12] Anand KJS. Pharmacological approaches to the management of pain in the neonatal intensive care unit. *J Perinatol* 2007; 27:S4–S111. DOI: 10.1038/sj.jp.7211712.
- [13] Sun L. Early childhood general anesthesia exposure and neurocognitive development. *Br J Anaesth* 2010; 105: i61–i68. DOI: 10.1093/bja/aeq302.
- [14] DiNardo JA. Should what we know about neurobehavioral development, complex congenital heart disease, and brain maturation affect the timing of corrective cardiac surgery? *Paediatr Anaesth*. 2011; 21: 781–6. DOI: 10.1111/j.1460-9592.2010.03429.
- [15] Lally KP. Congenital diaphragmatic hernia—the past 25(or so) years. *J Pediatr Surg* 2016. Available from <http://dx.doi.org/10.1016/j.jpedsurg.2016.02.005>.
- [16] Brett C, Davis PJ. Anesthesia for general surgery in the neonate. In: Davis PJ, Cladis FP, Motoyoma EK editors. *Smith's Anesthesia for Infants and Children*. 8th ed. Philadelphia: Elsevier; 2011; pp. 554–588. ISBN: 978-0-323-06612-9. ch18.
- [17] Harting MT, Lally KP. Surgical management of neonates with congenital diaphragmatic hernia. *Semin Ped Surg* 2007; 16:109–114. DOI: 10.1053/j.sempedsurg.2007.01.007.
- [18] Hedrick HL. Evaluation and management of congenital diaphragmatic hernia. *Pediatr Case Rev* 2001; 1:25–36. DOI: 10.1097/00132584-200110000-00004.
- [19] Tiruvoipati R, Vinogradova Y, Faulkner G, Sosnowski AW, Firmin RK, Peek GJ. Predictors of outcome in patients with congenital diaphragmatic hernia requiring extracorporeal membrane oxygenation. *J Pediatr Surg* 2007; 42:98–104. DOI: 10.1016/j.jpedsurg.2007.03.031
- [20] Chesley PM, Letbetter DJ, Meehan JJ, Oron AP, Javid PJ. Contemporary trends in the use of primary repair for gastroschisis in surgical infants. *Am J Surg* 2015; 209: 901–906. DOI: 10.1016/j.amjsurg.2015.01.012.
- [21] Dierdorf SF, Krishna G. Anesthetic Management of Neonatal Surgical Emergencies. *Anesth Analg* 1981; 60: 204–214. ISSN: 0003-2999.
- [22] Mann L, Ferguson-Smith MA, Desai M, Gibson AA, Raine PA. Prenatal assessment of anterior abdominal wall defects and their prognosis. *Prenat Diagn* 1984; 4: 427–435. DOI: 10.1002/pd.1970040606.



- [23] Aldrink HJ, Caniano DA, Nwomeh BC. Variability in gastroschisis management: a survey of North American pediatric surgery training programs. *J Surg Res* 2012; 176:159–163. DOI: 10.1016/j.jss.2011.05.012.
- [24] Riboh J, Abrajano C, Garber K, et al. Outcome of sutureless gastroschisis closure. *J Pediatr Surg* 2009; 44: 1947–1951. DOI: 10.1016/j.jpedsurg.2009.03.027.
- [25] Bayley G. Special considerations in the premature and ex-premature infant. *Anaesth Intensive Care Med* 2014; 15: 107–110. DOI: 10.1016/j.mpaic.2008.01.007.
- [26] Pierro A. The surgical management of necrotising enterocolitis. *Early Hum Dev* 2005; 81(1): 79–85. DOI: 10.1016/j.earlhumdev.2004.10.018.
- [27] Natasha Broemling N, Campbell F. Anesthetic management of congenital tracheoesophageal fistula. *Pediatr Anesth* 2011; 21: 1092–1099. DOI: 10.1111/j.1460-9592.2010.03377.
- [28] Andropoulos DB, Rowe RW, Betts JM. Anaesthetic and surgical airway management during trachea-oesophageal fistula repair. *Pediatr Anesth* 1998; 8:313–319. DOI: 10.1046/j.1460-9592.1998.00734.
- [29] Broemling N, Campbell F. Anesthetic management of congenital tracheoesophageal fistula. *Pediatr Anesth* 2011; 21:1092–1099. DOI: 10.1111/j.1460-9592.2010.03377.
- [30] Marseglia L, Manti S, D'Angelo G, Gitto E, Salpietro C, Centorrino A et al. Gastroesophageal reflux and congenital gastrointestinal malformations. *World J Gastroenterol* 2015; 21: 8508–8515. DOI: 10.3748/wjg.v21.i28.8508.
- [31] Bosenberg A. Benefits of regional anesthesia in children. *Pediatr Anesth* 2012; 22:10–18. DOI: 10.1111/j.1460-9592.2011.03691.
- [32] Abajian JC, Mellish RW, Browne AF, Perkins FM, Lambert DH, Mazuzan JE Jr. Spinal anesthesia for surgery in the high-risk infant. *Anesth Analg* 1984; 63:359-362. ISSN: 0003-2999.
- [33] Shenkman Z, Johnson VM, Zurakowski D, et al. Hemodynamic changes during spinal anesthesia in premature infants with congenital heart disease undergoing inguinal hernia correction. *Pediatr Anesth* 2012; 22:865–870. DOI: 10.1111/j.1460-9592.2012.03873.
- [34] Wolf AR, Doyle E, Thomas E. Modifying infant stress responses to major surgery: spinal vs extradural vs opioid analgesia. *Pediatr Anesth* 1998; 8:305–311. DOI: 10.1046/j.1460-9592.1998.00239.
- [35] White MC. Approach to managing children with heart disease for noncardiac surgery. *Pediatr Anesth* 2011; 21:522–529. DOI: 10.1111/j.1460-9592.2010.03416.