# Lesson 260: PreAnesthetic Assessment of the Newborn With an Abdominal Wall Defect

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## NEEDS STATEMENT

Abdominal wall defects in the newborn present as emergency situations. Critical differences exist depending on the cause of the defect, which affect anesthetic management. Care of these difficult pediatric cases has been identified by committee as required knowledge for the anesthesiologist.

## TARGET AUDIENCE

Anesthesiologists

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## LEARNING OBJECTIVES

*At the end of this activity, the participant should be able to:*

1. Describe the physiologic characteristics of the neonate.
2. Differentiate between gastroschisis and omphalocele.
3. Assess the risk for abdominal wall malformation by the clinical presentation.
4. List the preoperative considerations for anesthetic care of the newborn with an abdominal wall defect.
5. Apply appropriate intraoperative monitoring.
6. Identify correct strategies for fluid management.
7. Outline the difference between primary and secondary closure.
8. Develop an anesthetic plan.
9. Describe possible intraoperative complications.
10. Manage postoperative anesthetic care.

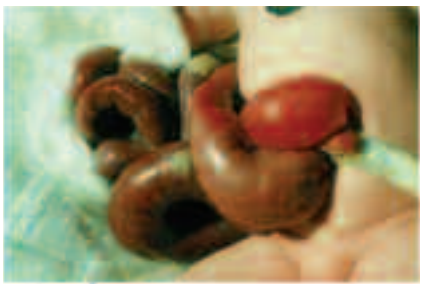
## CASE HISTORY

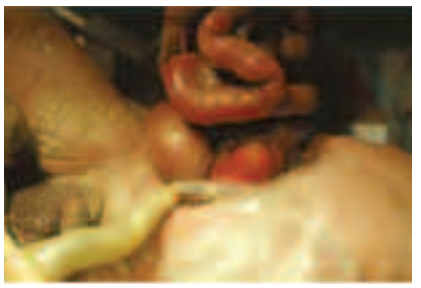
A 4-hour-old girl weighing 2.8 kg was admitted for emergency repair of an abdominal wall defect. The gestational age of the patient was 37 weeks. She was born by vaginal delivery with Apgar scores of 9 at both 1 and 5 minutes. Physical examination findings included heart rate, 154 beats per minute; blood pressure, 59/33 mm Hg; $Spo_2$, 96% on nasal cannula; respiratory rate, 54 breaths per minute; and temperature, 36.5°C. In the supine position, the patient showed no significant respiratory distress. On auscultation, the lungs were clear, and a soft systolic murmur was heard over the precordium. The hernia measured 3 cm in length.

*Gastroschisis* is a defect in the abdominal wall caused by an infarction of the distal segment of the omphalomesenteric artery or umbilical vein.[1] At the site of infarction—more commonly to the right of the umbilicus—the bowel can herniate through the necrotic wall and enter the extra-embryonic coelom. Such a malformation develops relatively late in the fetal period, and these infants are usually born prematurely.

Gastroschisis is usually not associated with other congenital malformations. However, 25% of patients have some vascular impairment of the bowel and related adhesions that can lead to malrotation or atresia. Exposure of the intra-abdominal contents to amniotic fluid results in dilation, inflammation, and thickening of the bowel—factors that lead to a poor prognosis.[2,3] Because of a large loss of fluids and heat along with a possibility for trauma and sepsis, these cases are surgical emergencies.

*Omphalocele* results from a failure of the gut to migrate into the abdominal cavity at about the 10th week of gestation. Omphalocele is more than twice as likely as gastroschisis to be associated with other congenital anomalies.[4] For example, a neonate with an epigastric omphalocele should be evaluated for pentalogy of Cantrell, a syndrome associated with defects in the midline supraumbilical abdominal wall, the lower sternum, and the diaphragmatic pericardium, along with a deficiency in the anterior diaphragm and cardiac anomalies.





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Beckwith-Wiedemann syndrome (omphalocele, macroglossia, gigantism, and hypoglycemia) may also coexist.

In neonates with an omphalocele, the bowel is covered with peritoneum. Therefore, the loss of fluids and heat is significantly less than in gastroschisis. These cases are not considered to be a surgical emergency, but an evaluation for associated congenital abnormalities and medical intervention and optimization should be accomplished before surgical intervention is undertaken (Table).

## PREANESTHETIC ASSESSMENT

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***Table.*** **Differences Between Gastroschisis and Omphalocele**

| | Gastroschisis | Omphalocele |
|---|---|---|
| Surgical emergency | Yes | No |
| Etiology | Occlusion of omphalomesenteric artery | Failure of gut migration from yolk sac into abdomen |
| Age when defect occurs | After 10 wk | 10 wk |
| Congenital anomalies most commonly associated | Sometimes: intestinal atresia, gallbladder agenesis, renal agenesis | Yes: Beckwith-Wiedemann syndrome, congenital heart disease, exstrophy of bladder |
| Fluid/temperature losses | More | Less |
| Location | Periumbilical | Umbilical |

## Epidemiology

The incidence of gastroschisis is 0.94 births per 10,000.[5] It occurs 4 times more frequently in children of women younger than the age of 20 years.[6,7] Young maternal age also appears to be a risk factor for preterm birth and low birth weight—added complications of gastroschisis.[8] The malformation has been linked to the increased prevalence of cigarette smoking and drug abuse in younger women.[9] Certain countries, including England, Japan, and Spain, in addition to Hawaii,[10] have significantly higher incidences of gastroschisis, suggesting an environmental or lifestyle influence on etiology.

Gastroschisis has also been found to be more prevalent in male infants. Familial recurrences may indicate a link to an autosomal-recessive gene. Teratogens and vasoactive substances such as nicotine, cocaine, and pseudoephedrine have been associated with gastroschisis, although more studies are required to determine causality.[7] One study found a 4.2-fold increase in infants of mothers who took pseudoephedrine, a sympathomimetic vasoconstrictor contained in many over-the-counter oral decongestants.[11] Aspirin, unlike the other nonselective nonsteroidal anti-inflammatory drugs and the selective cyclooxygenase-2 inhibitors, has also been linked to an increased occurrence of abdominal wall defects.[12]

## Physiologic Considerations In the Neonate

During the neonatal period (the first 30 days after birth), many physiologic and transitional changes occur. As in the present case, emergencies can develop in the neonate within a few hours after birth. During this period, the newborn is particularly susceptible to the effects of anesthesia and surgical intervention. An understanding of the unique physiology of the neonate during this transitional period is important for developing a safe perioperative plan for the repair of an abdominal wall defect.

### *Central Nervous System*

The central nervous system is the least developed of the major organ systems at birth. The goal perioperatively is to prevent any increase in intracranial pressure and maintain cerebral perfusion pressure. Although neonates have open fontanels and can compensate for small increases in intracranial pressure, several factors make neonates more susceptible to increases in intracranial pressure.

Intracranial compliance is lower in neonates than in adults because of a higher ratio of brain tissue to water content, a lower volume of cerebrospinal fluid, and a higher ratio of brain substance to intracranial capacity.[13] As a result of this lower compliance, neonates with increased intracranial pressure are more susceptible to hemorrhage and seizures. Increases in intracranial pressure should be monitored intraoperatively.

Infants are less sensitive to anesthetic agents, and at 3 months of age, the minimum alveolar concentration (MAC) is higher than in any other age group. However, babies less than 30 days old have been found to be more sensitive to anesthetic agents, and neonates have a MAC about 25% less than that of infants[14] (Figure 1). Although more studies are needed to fully understand the mechanisms, there is evidence that the blood–brain barrier is sensitive to increased levels of maternal progesterone and endogenous endorphins.[15,16]

### *Airway Management*

The anatomic differences between neonatal and adult airways play a big role in airway management. Newborns have smaller nares, a larger tongue, a smaller oral cavity, and a stiffer epiglottis. In addition, the vocal cords are higher—at the C3-4 level compared with the C4-5 level in the adult. Because the larynx is positioned more cephalad in the neck and anatomically closer to the tongue, the vocal cords are at a sharper, more acute anterior plane compared with the plane of the oral axis. This orientation, combined with a broader and stiffer epiglottis, makes the neonatal airway more difficult to secure. The larger occiput of the neonate presents a challenge for positioning. Placement of a shoulder roll facilitates intubation.

### *Cardiovascular System*

At birth, the chambers of the heart begin to work in series, rather than in parallel as before birth. The blood volume of the neonate is increased by a transfusion of placental blood. As the systemic circulation accommodates this blood, systemic vascular resistance increases. During delivery, epinephrine and norepinephrine are rapidly released, secondary to decreased temperature and cord clamping; as a result, cardiac output and contractility increase.[17]

The heart of the neonate is less compliant than the adult heart. Stroke volume is relatively fixed, and cardiac output is heart rate–dependent. In addition, sympathetic innervations are immature, and the supply of catecholamines at neuronal endplates is reduced.[18] The capacity for peripheral vasoconstriction is diminished, as is the ability to adequately compensate for dehydration and blood loss. Neonates, more vagotonic secondary to a less mature sympathetic system, tend to have a bradycardic response to stress insults such as laryngoscopy and hypoxia.

The pulmonary circulation of the fetus, with an open foramen ovale of the heart and a patent ductus arteriosus, is designed for minimal pulmonary function. Pulmonary vascular resistance is high, and right-sided heart pressures are low. At the first inspiration by the newborn, the increase in $Pa_{O_2}$ of the pulmonary vasculature causes vasodilation and a decrease in pulmonary resistance. Vasodilators such as bradykinin, prostaglandins $E_1$ and $E_2$, prostacyclin, nitric oxide, and endothelium-derived relaxing factor also decrease the pulmonary vascular resistance.[19]

The neonatal pulmonary circulation is transitional. Hypoxemia and acidemia must be quickly corrected to prevent the return of increased pulmonary vascular resistance and the reopening of right-to-left shunts.[20] If the neonate reverted to a fetal circulation, hypoxia, pulmonary arterial vasoconstriction, pulmonary hypertension, and eventually reduced pulmonary blood flow would result. The chronic persistence of fetal circulation, called persistent pulmonary hypertension, is incompatible with life under ambient conditions.

### *Respiratory System*

In neonates, peripheral carotid and aortic chemoreceptors sense $Pa_{O_2}$ and $Pa_{CO_2}$ (a similar mechanism exists in adults), while the central chemoreceptors in the medulla are sensitive to pH changes. Control of the respiratory drive is not fully developed. Hyperventilatory efforts during hypoxia cannot be sustained, and initial increases in respiratory rates lead to a decrease in ventilation and oxygen tension[21]

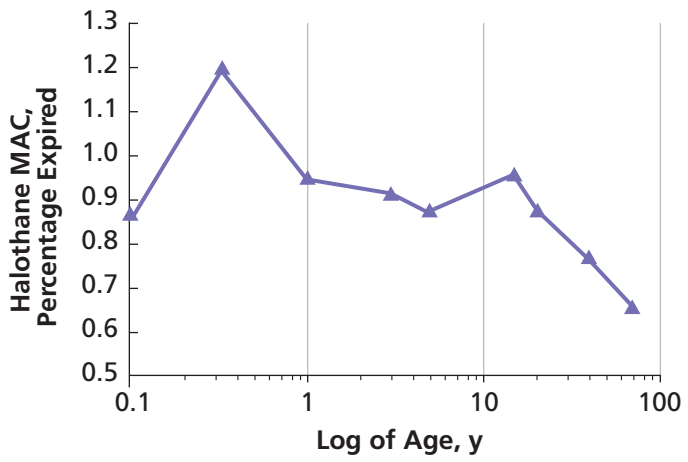


***Figure 1.*** **The minimum alveolar concentration (MAC) of halothane according to patient age.**

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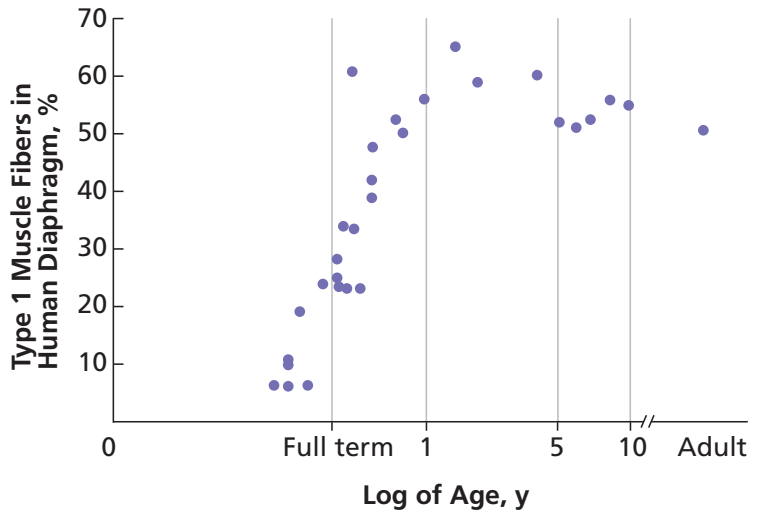


***Figure 2.*** **Type 1 muscle fibers in the diaphragm according to age.**

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caused by weak chemoreceptor output, a central inhibitory effect of hypoxia on ventilation, or immature pulmonary mechanics.[22]

Although the response to carbon dioxide is more developed, the neonate can increase ventilation only 3- to 4-fold, compared with up to 20-fold in the adult. Thus, the neonate is prone to apneic episodes, and in the presence of anesthetic agents, opioids, and sedatives, the response to ventilatory depression is even more exaggerated.

Oxygen balance is more tenuous in the neonate than in the adult. There is a 2- to 3-fold increase in oxygen consumption that must be met by an increase in oxygen supply. Because tidal volume in the neonate is the same as in the adult on a per-kilogram basis, any increase in oxygen demand must be met by an increased respiratory rate. The increase in the ratio of oxygen consumption to functional residual capacity in neonates (5:1) compared with that in adults (1.5:1) explains the rapid desaturation of oxygen when the airway is obstructed or the neonate is apneic. The increased closing volume in neonates results in small-airway closure during resting tidal volume, causing atelectasis and shunt.

The diaphragm of the neonate is composed of 10% type 1 fatigue-resistant muscle fibers—compared with 55% in an adult (Figure 2). As a result, neonates are more likely to tire from the respiratory effort. In addition, the more compliant chest wall secondary to the presence of cartilaginous ribs tends to collapse in neonates at the end of inspiration—which is exacerbated if the airway is obstructed. The work of breathing is increased because greater negative pressure is required to open the airways. In other words, the neonate has to do more work to breathe, and with less strength to accomplish it.

### *Hematology*

Fetal hemoglobin, composed of a pair of $\alpha$ chains and a pair of $\gamma$ chains, constitutes about 75% of the total hemoglobin and is present for the first 3 to 6 months of the infant's life. Fetal hemoglobin has an increased affinity for oxygen because it does not bind 2,3-diphosphoglycerate as readily as does adult hemoglobin. Thus, compared with adult hemoglobin, fetal hemoglobin has a decreased capacity for releasing oxygen. As a result, as the percentage of circulating fetal hemoglobin increases, the oxyhemoglobin dissociation curve shifts to the left. As the age of the infant increases from birth to 6 months, the level of fetal hemoglobin decreases; the oxyhemoglobin dissociation curve shifts to the right as oxygen affinity decreases, and increased unloading is seen with increasing levels of adult hemoglobin (Figure 3).

Fetal hemoglobin in utero is advantageous for taking up oxygen from the mother; however, for the neonate, fetal hemoglobin is disadvantageous. After birth, in periods of distress, acidosis, hypercarbia, or hypothermia, there is less potential for fetal hemoglobin to release bound oxygen, so hypoxia and acidosis worsen. To compensate, the hemoglobin concentration increases; it ranges from 14 g/100 mL to 20 g/100 mL at birth. Adult levels of hemoglobin are reached at about 9 to 12 weeks of age.

Neonates have a relatively large blood volume for their size compared with adults. Term infants have a blood volume of 80 to 100 mL/kg; the preterm volume is 90 to 105 mL/kg. Volume differences are secondary to placental transfusion and gestational age. As the period of gestation lengthens, plasma volume decreases. The characteristics of red blood cells (RBCs) in the neonate are different from those in adults. In neonates, permeability to sodium and potassium is elevated, making RBCs more susceptible to osmotic changes and lysis.

Neonatal RBCs are also at risk for damage in the microcirculation because of a lower level of glutathione peroxidase. As a result, the survival time of RBCs is decreased to 80 to 100 days in term infants, and to 60 to 80 days in preterm infants. An increased turnover of RBCs is seen. In addition, neonatal RBCs have an increased demand for adenosine triphosphate and an increased oxygen consumption.[23,24]

### *Renal Function*

The placenta is the major organ of excretion for the fetus. At birth, the lungs and kidneys must quickly take over the roles of metabolic homeostasis and control of fluid electrolyte balance. Although the maximum number of nephrons is attained by 35 weeks, the nephrons are shorter and less mature. The glomerular filtration rate in a full-term neonate can change rapidly, corresponding to the level of hydration. Reactivity of the renal vascular bed is increased during the perinatal period. In premature neonates less than 28 weeks, the glomerular filtration rate is lower. The maturation of renal tubular function lags behind that of glomerular function. Thus, the neonate can produce a dilute urine, but compared with adults, neonates can concentrate only about half the amount because of decreased tonicity of the medullary interstitium.

The neonate is at risk for overhydration, water intoxication, and the effects of antibiotics, barbiturates, and diuretics, which will have longer half-lives. On the other hand, neonates are considered obligate sodium losers; the immature neonatal kidney has a decreased ability to absorb sodium. As a result, urine cannot be concentrated and dehydration is a concern. The renal functions of acid–base homeostasis and electrolyte balance are immature and tenuous, and therefore neonates are at an increased risk for acidosis and hyperkalemia.

### *Metabolism*

Neonates are prone to metabolic disturbances, especially hypoglycemia and hypocalcemia. Hepatic glucose storage is limited, and metabolic demands for growth are high. Rapid glycogenolysis eliminates most of the glycogen stores within the first 24 to 48 hours after birth, while gluconeogenesis is slow.

Studies have shown that hypoglycemia in the first few hours can be attributed to intrauterine hyperinsulinism.[25] Dextrose-containing maintenance fluids can be started at a dosage of 4 mL/kg per minute; the glucose level should be checked every 30 minutes. Plasma glucose levels below 35 mg/100 mL in the first 3 hours, 45 mg/100 mL between 3 and 24 hours, and below 45 mg/100 mL after 24 hours are of concern[26] for symptomatic hypoglycemia and sequelae such as lethargy and failure to thrive.

Neonatal hypocalcemia develops when ionized calcium levels drop below 1 mmol/L in full-term infants and below 0.75 mmol/L in preterm infants. Hypocalcemia may develop secondary to a decrease in calcium transport across the placenta and an inadequate level of parathyroid hormone.[27] If the neonate becomes symptomatic—exhibiting tremors or seizures—a 100-mg/kg bolus of calcium gluconate can be given. If the neonate is asymptomatic, an infusion of 100 to 200 mg/kg per day can be started.

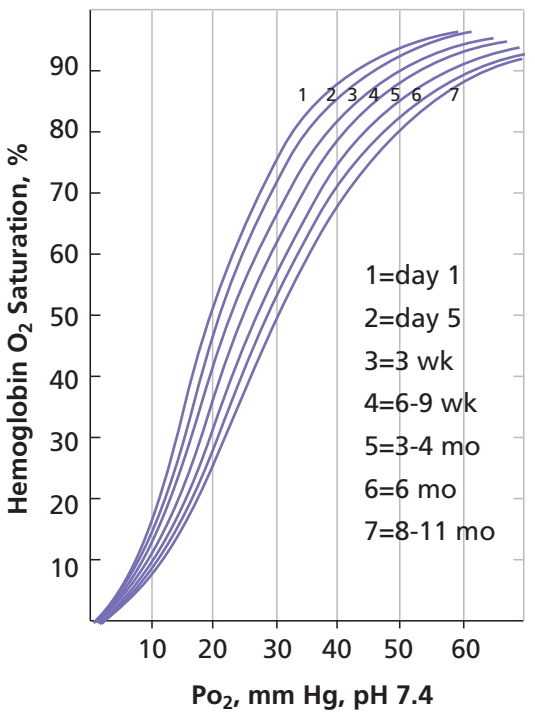


***Figure 3.*** **Hemoglobin concentration in infants of different ages.**

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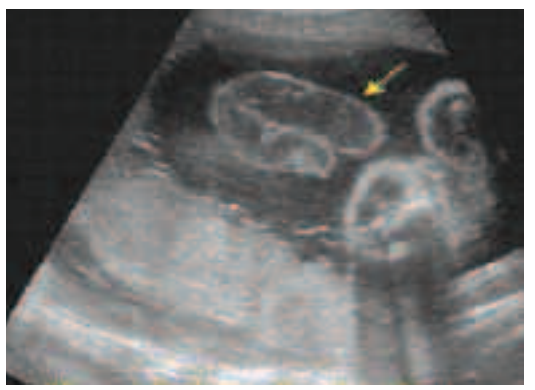

***Figure 4.*** **Bowel gastroschisis.**

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### *Thermoregulation*

Thermoregulation is critical to an infant's survival. The ratio of surface area to body mass is bigger for infants, and infants also have less insulating subcutaneous fat, greater skin permeability to water, and a small radius of curvature of exchange surfaces.[28]

Newborns, and even most preterm infants, have developed some thermoregulatory capacity. At delivery, a stimulation of cutaneous cold receptors activates the sympathetic nervous system to release norepinephrine. Clamping of the umbilical cord removes placental factors that suppress nonshivering thermogenesis and activates brown adipose tissue metabolism and heat production. Brown adipose tissue, which develops at 26 to 30 weeks of gestation, accounts for 2% to 7% of total weight. It is found in the midscapular region, nape of the neck, clavicles and axillae, mediastinum, kidneys, and adrenal glands.[29] Brown adipose tissue may be able to supply nearly 100% of the infant's thermal requirements because it contains many small vacuoles and has a high fat-to-cytoplasm ratio that enhances its rapid metabolism. An abundance of mitochondria, glycogen stores, blood supply, and sympathetic nerve supply in brown fat also facilitates its metabolism by norepinephrine.

## Preoperative Assessment For Gastroschisis

The perioperative management of newborns with gastroschisis begins in the delivery room. Vital signs must be assessed; early stabilization, intubation, oxygenation, ventilation, or I.V. cannulation may be necessary.[27] Maternal health and pregnancy status can reveal important information for the risk stratification of neonates before delivery. A preoperative ultrasound image may show a midline umbilicus with a cauliflower-like mass, usually to the right, that is external to the fetus. No covering sac is identified. The cauliflower appearance is created by echogenic fluid between multiple loops of bowel (Figure 4).

Although it is not standard practice, the use of 3-dimensional magnetic resonance imaging may produce more information, and with greater definition and accuracy, about the bowel both outside and inside the abdomen.[30] For this and other serious congenital disorders, a prenatal diagnosis presents therapeutic options and time for preparation. Even with a later diagnosis, however, efforts to achieve safe and optimal conditions for a full-term pregnancy and delivery are essential.[31]

There has been no report of in utero repair of this type of abdominal defect. However, medical intervention for coexisting complications or comorbidities has improved the outcome for neonates with gastroschisis. One case report described 2 fetuses diagnosed with gastroschisis at 19 weeks of gestation and severe oligohydramnios at 30 to 31 weeks who received serial transabdominal amnioinfusions. With prenatal detection, the potential consequences to the fetus of severe oligohydramnios were avoided.[32]

After stabilization of the newborn, the exposed abdominal contents should be covered with a warm, moist, sterile dressing to minimize fluid and heat losses. Alternatively, the entire lower body can be covered by a sterile bag filled with warm saline.[33] Care should be taken to avoid further trauma to the bowel or incarceration at the exterior site. However, large volumes of fluid may transude or exude from exposed abdominal viscera despite appropriate covering. Therefore, appropriate fluid administration is a quintessential component of preoperative management.

A balanced isotonic solution should be administered to replace expected third-space losses. An infusion of 10 mL/kg per hour must be started based on an assessment of the urine output and the central venous pressure or blood pressure; additional boluses of 3 to 5 mL/kg may be needed.

Electrolyte values—calcium and glucose in particular—should be monitored, especially if large volumes of fluid are given. Such neonates require 3 to 4 times the normal amount of maintenance fluid because of the losses associated with this condition. Because the babies are prone to hypoglycemia, a maintenance infusion of glucose-containing solution should be added.

Dextrose-free solution should be used to replace blood, third-space, or fluid losses, although maintenance requirements can begin with the administration at 3 to 4 mg/kg per minute of dextrose solution for infants weighing less than 1,000 g.[34] If hypoglycemia is detected with glucose monitoring, treatment should be undertaken. Fluids should be replaced cautiously because osmotic diuresis secondary to hyperglycemia causes dehydration.

An orogastric tube should be placed to decompress the stomach and prevent regurgitation and pulmonary aspiration. Although these neonates have probably not been fed, they should be considered to have stomach contents secondary to bowel distention. Because the herniated viscera and intestine have been exposed to amniotic fluid in utero and to air after delivery, the bowels may be inflamed and dilated and are probably functionally abnormal. Extruded bowels increase susceptibility to infection, and therefore broad-spectrum antibiotic coverage with ampicillin (100 mg/kg per day) and gentamicin (5 mg/kg per day) is appropriate.

The site and size of the lesion are important for prenatal risk stratification. A team that has an anesthesiologist, obstetrician, and pediatrician should be readied for a possibly complicated delivery that may require a cesarean section. The type of delivery (vaginal or cesarean section) results in no statistical difference in outcome.[35] There has been no clinical evidence to support the supposition that vaginal delivery of a neonate with exposed bowel results in more complications throughout the perioperative course. Preterm birth, not mode of delivery, was found to affect the survival of infants with gastroschisis.

Because intestinal damage is exacerbated by increased contact with amniotic fluid, the strategy of preterm delivery of neonates with gastroschisis avoids intestinal damage and prevents the formation of peel, an inflammatory response to amniotic fluid. There is evidence to demonstrate that peel formation in gastroschisis is caused by the accumulation and activation of intestinal waste products in the amniotic fluid.[36] A study by de Lagausie et al[37] found that digestive enzymes in amniotic fluid trigger and maintain the process of inflammation in the human fetus. Affected neonates with minimal exposure to amniotic fluid have a lower risk for complications such as intestinal atresia and postoperative hypoperistalsis, which prolong hospital stay.[3] Surgical reduction is easier.

Neonates with gastroschisis should be evaluated for other congenital anomalies, although these occur less frequently than in infants with an omphalocele. A thorough physical examination should be conducted; however, because gastroschisis is a surgical emergency, awaiting the results should not delay surgery. Physical examination findings do, however, have implications for intraoperative management and are worth investigating when preparing the neonate for surgery.

In summary, preoperatively the physician must minimize fluid loss, prevent infection, correct electrolyte abnormalities, and avoid trauma to the exposed intra-abdominal contents.

## Intraoperative Management

Based on results of the preoperative assessment and size of the hernia, the patient can be stratified for risk and an anesthetic plan formulated. For example, standard American Society of Anesthesiologists (ASA) monitoring is adequate for a hemodynamically stable neonate who is able to maintain respiratory homeostasis and whose hernia size is less than 4 cm.

In a patient with an uncomplicated intraoperative course, there is a greater chance for making a primary closure. On the other hand, if the preoperative assessment reveals an unstable neonate—hypotensive, hypoxic, using accessory muscles to maintain saturation, and with a hernia size larger than 4 cm—more invasive pressure monitoring, such as central venous, arterial, intragastric, or intravesical, should be considered. In this case, the surgical risk is greater and a primary closure may not be possible.[38] Intra-abdominal pressure can be reflected by intravesical pressure.

Neonates with gastroschisis are susceptible to regurgitation and aspiration. Therefore, either an awake intubation or a rapid-sequence induction should be performed. The choice is usually based on the anesthesiologist's comfort level with either technique. Bradycardia, as a result of the relative vagotonicity of infants, which is compounded by apneic (breath-holding) episodes, can lead to hypoxia.

Maintenance of anesthesia can be accomplished in several ways. As previously discussed, the anesthesia requirement for maintenance in neonates is less than that in infants.[14] Nitrous oxide must be avoided to minimize bowel distention in anticipation of increased abdominal pressure once the bowels are returned to the abdominal cavity. Inhalation agents much be titrated carefully to avoid hypotension. An oxygen–air mixture is used at concentrations that maintain the $Spo_2$ in the mid-90% range to avoid the risk for retinopathy of prematurity.

Body temperature maintenance and fluid balance are of continuing concern in the OR. Hypothermia is minimized by increasing the room temperature, using warming blankets, warming the I.V. fluids, and keeping the bowel wrapped. Hyperthermia and its sequelae—increased oxygen consumption and evaporative losses—also must be avoided.

Third-space losses continue into the intraoperative period. A bolus of fluid may be given before induction, especially in high-risk patients who have signs of hypovolemia (eg, decreased urine output, tachycardia, decreased skin turgor, and slow capillary refill). With adequate fluid management, urine output should be 1 to 2 mL/kg per hour.

Intraoperative management is different for a neonate with a hernia larger than 4 cm, or with hemodynamic instability. The placement of an arterial cannula permits the monitoring of acid–base status and of electrolyte and glucose levels. Hypovolemia is indicated by hemoconcentration and metabolic acidosis.

Infants who have Beckwith-Wiedemann syndrome—more commonly associated with omphalocele—are particularly prone to hypoglycemia, and their glucose levels must be checked frequently. Measurement of $Pao_2$ is critical in neonates; at a postconception age of less than 44 weeks, neonates are still at risk for retinopathy of prematurity. Optimal $Pao_2$ is from 50 to 70 mm Hg, or saturation between 90% and 95%.

Other invasive monitors measure central venous pressure and intragastric pressure. Central venous pressure monitoring can be used for evaluating blood volume and allows delivery of drugs directly to the central circulation. To measure intragastric pressure, a nasogastric tube can be placed in the stomach with a column of saline. During closure of the abdomen, intragastric pressure can be inferred. Primary closure will be successful with intragastric pressures below 20 mm Hg.[39]

In 1988, a retrospective study by Yaster et al[39] found that values for intragastric pressure and central venous pressure can predict a successful primary closure of the abdominal wall defect. The researchers concluded that if the intragastric pressure increased more than 20 mm Hg or the right atrial pressure increased more than 4 mm Hg, mortality caused by ischemia of the bowel or the lower extremity was increased. If these parameters are exceeded, a staged closure is necessary.

As the bowel is placed into the peritoneal cavity, the increase in intragastric pressure may result in hypotension. A decrease in preload secondary to compression of the inferior vena cava can compound any decrease in pressure. Pulmonary compliance decreases as the abdominal contents are pushed up into the thoracic cavity and diaphragmatic excursion decreases. Oxygenation and ventilation may be compromised. Peak airway pressure should be monitored closely during this part of the surgery, especially when the hernial defect is larger than 4 cm.

The increased intra-abdominal pressure also causes decreased urine output. A pressure above 20 mm Hg results in an increased visceral pressure and decreased renal blood flow.

If the neonate has been able to physiologically tolerate the primary closure, the anesthesiologist may opt to extubate the trachea at the end of surgery. Adherence to recommendations for primary and staged closures has significantly reduced morbidity and mortality. The postoperative maintenance of mechanical ventilation for 24 to 48 hours improves respiratory compliance.

In a staged closure, the abdominal cavity must be covered with a protective silo. The patient is carefully monitored in a critical care setting; after the bowel has been observed to have returned to the abdominal cavity secondary to other factors—including the resolution of edema and third spacing—the infant is returned to the OR for definitive closure.

The anesthesiologist must continue to monitor for possible complications. Pulse oximetry via the big toe can be valuable to assess circulation in the lower extremities. Oliguria may be seen secondary to decreased blood flow to the kidneys. Peak pressures may continue to be elevated, and the neonate may be at increased risk for barotrauma.

A recent study questioned the need for general anesthesia in all cases of gastroschisis repair.[40] The outcome of 24 neonates who underwent conventional reduction under general anesthesia in the OR (group 1) was compared with that of 27 babies who underwent crib-side reduction without general anesthesia (group 2). The groups were matched for age, birth weight, extent of bowel evisceration, and time required for reduction. Admission to the ICU occurred in 92% of the group 1 neonates and in 7% of group 2. Otherwise, similar outcomes were recorded with regard to complications, time to feeding, and total hospital stay. Reduction without general anesthesia was found to be safe and cost effective.

## Postoperative Management

Postoperatively, neonates with gastroschisis have a good prognosis in more than 90% of cases. Monitoring and care in a neonatal ICU is continued with recording of peak airway pressure, signs of increased abdominal pressure, infection prevention, and fluid and electrolyte homeostatic regulation.

A recent study emphasized the detrimental effects of hyperglycemia on the ability to wean an infant from assisted ventilation.[41] The researchers found that a high maximum serum glucose concentration correlated positively with duration of total parenteral nutrition, mechanical ventilation, and hospital stay. Avoidance of excessive nutrition, in addition to tight glycemic control, is recommended.

Extubation, an important goal for the pediatric team, minimizes the consequences of prolonged intubation and bronchopulmonary dysplasia. Unfortunately, despite appropriate management, some patients may exhibit postoperative intestinal hypoperistalsis, malabsorption, and shortened bowel length. Recent research demonstrated the trophic effects of recombinant human erythropoietin in increasing the length and height of villi in the developing small bowel, thus increasing villous surface area. In addition, there is evidence of a prokinetic effect of erythropoietin on hypoperistalsis and reduction in bowel damage.[42]

## Management of the Case Presented

In the case presented, a baby girl was born at term, weighing 2.8 kg, with an uncomplicated perinatal course and delivery. Prenatal ultrasound revealed renal pyelectasia, a benign condition of fluid collection in the kidney. With supplemental nasal oxygen, the neonate was in no apparent distress. An abdominal wall defect measured less than 4 cm. A peripheral I.V. cannula was placed by the pediatric team, and a maintenance infusion of dextrose 10% in water at 12 mL/h was started. The prophylactic antibiotics ampicillin and ceftriaxone were administered. Preoperative laboratory results included a glucose level of 79 mg/dL and a hematocrit of 55%. No congenital abnormalities were suspected.

In preparation for surgery, the room was warmed to 28°C and an age-appropriate setup was completed. A forced-air warming blanket was placed and fluids warmed. Standard ASA monitors were applied. Invasive monitors were deemed unnecessary and not placed. A 30-mL bolus of Ringer's lactate solution was administered, in addition to continuing the fluid infusion of dextrose 10%. An Andersen gastric tube was easily passed to suction the stomach. An additional 20-mL bolus of Ringer's lactate solution along with atropine 0.1 mg/kg were given immediately before a rapid-sequence induction with propofol and succinylcholine. After placement of a 3.0-mm uncuffed endotracheal tube, an air leak was detected at 25 cm $H_2O$.

As surgery proceeded, maintenance fluids were sufficient so that additional boluses were not needed. After removing the bowel from a sterile environment, the surgeons worked expeditiously to return it to the abdominal cavity within 1.5 hours. Peak airway pressures increased less than 10 cm $H_2O$, and hemodynamic stability was maintained without intervention. Primary closure was easily achieved. The baby with her trachea intubated was returned to the neonatal intensive care unit. She was ventilated, sedated, and paralyzed. Extubation was performed on postoperative day 3, and the patient was discharged home 3 weeks later.

## Conclusions

Neonates with gastroschisis require emergency surgery because of their susceptibility to fluid loss, infection, electrolyte disturbances, and trauma. The dynamics between surgery and anesthesia are unpredictable in these neonates, whose physiology, at several hours after birth, is transitional as they adapt from a fetal to a post-birth environment. The goals of the anesthesiologist—to secure the airway, obtain adequate I.V. access, and maintain homeostasis with electrolytes and normothermia—are challenging.

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## Post-test

1. **Cardiac output in the neonate is increased by:**
   a. an increase in heart rate
   b. an increase in stroke volume
   c. an increase in cardiac compliance
   d. peripheral vasoconstriction
2. **Which of the following is true regarding differences between gastroschisis and omphalocele?**
   a. Omphalocele is a surgical emergency and gastroschisis is not.
   b. The defect in gastroschisis is not covered by a peritoneal sac over the eviscerated bowel.
   c. Infants with an omphalocele are more likely to become dehydrated.
   d. Gastroschisis develops earlier in gestation than does omphalocele.
3. **Neonates are more likely to develop hypoxia than are adults because:**
   a. neither the carotid nor aortic chemoreceptors are developed at birth
   b. the neonate's response to hypercarbia can increase ventilation only 3- to 4-fold
   c. the tidal volume of an infant is 3 times that of an adult on a per-kilogram basis
   d. the chest wall of the neonate is less compliant than that of an adult
4. **The central nervous system of a newborn:**
   a. is highly developed at birth
   b. demonstrates increased compliance regarding intracranial dynamics
   c. readily compensates for an increase in intracranial pressure because of open fontanelles
   d. has a decreased sensitivity to anesthetic agents
5. **Fetal hemoglobin:**
   a. has a single $\alpha$ and a single $\gamma$ chain
   b. is present during the first year of life
   c. does not bind 2,3-diphosphoglycerate readily and thus has a decreased ability to release oxygen
   d. causes a rightward shift of the oxyhemoglobin dissociation curve
6. **Characteristics of RBCs in neonates include:**
   a. decreased permeability to sodium and potassium
   b. greater susceptibility to osmotic changes and lysis
   c. increased levels of glutathione peroxidase
   d. a survival time longer than 100 days in term infants
7. **Gastroschisis is more likely to occur in:**
   a. males
   b. children born to teenage mothers
   c. children of mothers who smoke or take over-the-counter decongestants
   d. all of the above
8. **The preoperative management of the neonate with gastroschisis includes:**
   a. covering the exposed bowel with a bag to decrease fluid loss
   b. a thorough workup to uncover associated congenital abnormalities
   c. delaying surgery until electrolyte levels and urine output are within normal limits
   d. avoiding placement of a nasogastic tube because the neonate has not been fed
9. **Which of the following describes appropriate fluid management for a neonate with gastroschisis?**
   a. A balanced salt solution of 5 mL/kg per hour is sufficient.
   b. Enough fluid is needed to maintain urine output above 5 mL/kg per hour.
   c. The amount of fluid required for maintenance is 3 to 4 times that required for a healthy newborn.
   d. Dextrose-containing solutions should be avoided.
10. **Which of the following is true regarding the delivery of anesthetics to patients undergoing repair of an abdominal wall defect?**
    a. It is essential to maintain oxygen saturation above 96% because of the presence of fetal hemoglobin.
    b. Nitrous oxide is useful to prevent awareness during anesthesia.
    c. The anesthetic requirement for newborns is greater than that for infants.
    d. General anesthesia with endotracheal intubation is not always necessary.