Guidelines for the anesthetic management of pyloromyotomy

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The author provides guidelines for the management of pyloromyotomy in the infant preoperatively, intraoperatively, and postoperatively. This article also reviews the pathophysiology of hypertrophic pyloric stenosis, its incidence, symptoms, and possible complications.

Pathology
Having been first reported in 1788, pyloric stenosis occurs in approximately 0.3% of live births. It is associated with a history of nonbilious vomiting following feeding. The pyloric sphincter, a muscle located at the junction of the stomach outlet and the duodenum, hypertrophies and causes an obstruction of the gastric outlet. The muscles of the stomach thicken because of the increased work required to move the gastric contents through the narrowed pyloric channel. Hyperperistaltic waves can be seen moving from left to right during inspection of the patient's abdomen. Reversal of these peristaltic waves is responsible for the projectile vomiting that occurs with pyloric stenosis.

The symptoms associated with pyloric stenosis include delayed emptying, gastric dilatation, regurgitation, and vomiting. Progressive vomiting results in the loss of hydrogen chloride, sodium and potassium ions, leading to dehydration and metabolic alkalosis. Proper preoperative fluid and electrolyte correction is mandatory in infants with pyloric stenosis. Measurements of serum electrolyte levels, arterial blood gas tensions, and pH will help quantify the degree of the patient's metabolic abnormality.

Signs of dehydration in the infant include weight loss, dry tongue and mucous membranes, sunken eyeballs, depressed fontanelles, rapid/thin pulse, scanty urine output, and dry skin with poor tissue turgor.

If the symptomatology of pyloric stenosis is clear, a physical examination will reveal the presence of a palpable mass in the abdomen just above and to the right of the umbilicus. When the symptomatology is not clear, an upper GI series or a barium swallow is performed to confirm the problem prior to surgery.

Incidence
Pyloric stenosis occurs in 1 of 500 live births and is five times more common in males than in females. It usually develops at two to five weeks of age, and is as common in preterm as term infants. In the black population, the incidence of occurrence is 1 in 2000. There has been a striking tendency toward increased incidence in infants whose parents had the lesion, as well as in the first born children. It is most commonly seen within the first six months of life, whereby the autonomic nervous system imbalance and humoral disorders have been suggested as etiologic factors.

The surgical procedure for pyloric stenosis is
called pyloromyotomy or Ramstedt's operation, the latter being named after the technique developed by Conrad Ramstedt in 1911. The procedure consists of an incision made near the upper quadrant of the abdomen. The stomach is then located and the pyloric area of the stomach is exteriorized onto the abdomen. This is followed by a longitudinal incision along an a vascular area of the serosa. The muscle is then spread apart to allow the intact mucosa to bulge into the separated muscle of the pylorus.

Bleeding in the area is minimal, and when it is satisfactorily controlled, the pyloric muscle is returned to the abdomen and closure begins. The duration of surgery should be no more than 45 minutes to one hour.5

Mortality-morbidity

Though pyloric stenosis can be a medical emergency, it is not a surgical emergency. No patient should be taken to the operating room until the fluid, electrolyte, and acid-base imbalances have been corrected. It still carries a mortality of 0.5-2%, an unacceptable figure, and an even higher morbidity.5 The morbidity and mortality in infants with pyloric stenosis results from preoperative dehydration and electrolyte imbalance, intraoperative vomiting and regurgitation that may result in aspiration, and postoperative respiratory arrest and hypoglycemia.1

Preoperative assessment

The anesthetist must be assured that: (1) the patient has been adequately evaluated and prepared for the operation (especially the metabolic considerations), (2) the anesthetic technique, as well as fluid and electrolyte maintenance, is impeccable, and (3) the postoperative care is available, adequate, and utilized totally.5

Definite information is necessary before the infant is accepted for pyloromyotomy. The following information should be obtained from the patient's chart and/or parents:

1. Birth weight, weight prior to onset of vomiting, and present weight.
2. Duration of active vomiting and its characteristics, frequency, and usual quantity.
3. Intake over this period, general effect on urination and stooling, and last voiding.
4. Hospital course, laboratory, blood and electrolyte data as indicated, and therapy administered to date.
5. If there is severe dehydration, the presence of coma or other neurological signs.4

When no oral intake is retained and normal losses via skin, respiratory tract, and kidneys continue, the development of dehydration may reach an advanced state within 3-4 days, with laboratory evidence of metabolic alkalosis appearing in the form of rising serum pH, hyponatremia and hypochloremia.4

As the level of bicarbonate rises, the body tries to compensate through: (1) the respiratory system (that is, through hypoventilation and retention of carbon dioxide), and (2) through the kidney. The renal adjustment consists of excreting \( \text{HCO}_3^- \) in exchange for chloride. Sodium is excreted with \( \text{HCO}_3^- \) as the preferential cation. Later on, as hyponatremia develops, potassium and hydrogen ions are used to excrete bicarbonate. This gives rise to further hypokalemia and alkalosis. Due to excretion of hydrogen, there is a paradoxical acidic urine, although there is systemic alkalosis. Hence, in pyloric stenosis, urine is alkaline initially and acidic later.

The hypokalemia is due to: (1) the loss secondary to vomiting, (2) systemic alkalosis, and (3) renal loss of potassium. The metabolic alkalosis is due to: (1) loss of hydrogen in vomitus, (2) loss of potassium, (3) loss of chloride, and (4) dehydration.1

A normovolemic person, once anesthetized, becomes relatively hypovolemic. The infant with an untreated pyloric stenosis is already depleted in both extracellular fluid and plasma volume and may be stressed beyond his ability to compensate further. In order to correct the acid-base imbalances fully, dehydration and electrolyte deficits (particularly sodium, chloride, and potassium deficits) also have to be fully corrected. In severe dehydration, it can take three to four days for total correction.3

The degree of dehydration may be estimated by physical examination, hematocrit, and history of weight loss. Therapy consists chiefly of restricting oral intake in vomiting patients and of replacing chloride loss with saline solution. Potassium depletion should be calculated; not more than 3 mEq/kg/day should be given and replaced only after urination has provided evidence of initiation of rehydration.4 Glucose, 5 gm/kg/day, not only meets the patient's metabolic needs but also reduces further potassium losses from its neoglucogenic and protein-sparing effects. Alkalosis will remain until sodium and potassium equilibrium has been reestablished.5

The volume of water to be given is usually judged from any evidence of dehydration, remembering that up to 5% of total body water can be lost without there being any evidence of this loss. Usually, 200-400 ml of normal saline with normal
maintenance fluid added while it is being administered is sufficient. Urinary pH and electrolyte levels will indicate the restoration of total body needs. Serial laboratory work should be performed, and the operation should be delayed until the proper criteria of replacement are apparent.5

It is safe to perform the surgery when vital signs are stable, pH is between 7.3 and 7.5, hemoglobin is more than 10 gm, serum chloride is more than 88 mEq/L, bicarbonate is less than 30 mmole/L, potassium is more than 3.2 mEq/L, urine output is satisfactory (1.2 ml/kg/hr), and a specific gravity of the urine is less than 1.020. Only after the infant has met the above criteria should he or she be taken to the operating room.1

**Intraoperative anesthetic management**

The problems the anesthetist encounters with the pyloric stenosis infant include:

1. Those associated with any newborn—hypothermia, labile cardiovascular system, and so forth.
2. Preoperative dehydration and electrolyte imbalance, if uncorrected. (In that case the child should not have been brought to the operating room in the first place.)
3. Upper gastrointestinal tract obstruction.1

All patients, regardless of age, with a diagnosis of pyloric stenosis must be considered at increased risk for aspiration pneumonitis.

Before bringing the child to the operating room, the anesthetist must make sure that the following equipment is available: Jackson Rees™ circuit, suction, different sizes of endotrachial tubes, laryngoscope, warming blanket, and overhead heating lamp. The operating room should be warmed to at least 75 degrees and the gases should be humidified. Monitoring devices should include a precordial stethoscope, blood pressure cuff, ECG, ultrasound flow Doppler, and temperature probe. Additional gastric tubes (sizes Fr 10 and 14), appropriate restraints, and an additional person should be available to restrain the patient for awake intubation.

An intravenous infusion is normally in place when the infant arrives in the operating room. One of the contents frequently added to the fluid is potassium. If so, the fluid and administration set should be changed to a fresh unit. Potassium must be given slowly when given intravenously. If the solution containing potassium is used as a flushing solution during the administration of induction agents, atropine, succinylcholine, or other drugs, the patient is actually being given a bolus of potassium which could cause cardiac arrhythmias or cardiac arrest.2

The patient is placed on the operating room table after the room is warmed and all personnel and equipment are ready. The anesthetist should connect the infant to all monitors, check and record vital signs. The nasogastric tube should be suctioned. If solids or barium are aspirated, the patient’s stomach should be gavaged until the returning solution is clear and then the nasogastric tube should be removed. Removal of the nasogastric tube is advocated to prevent its serving as a gag stimulus. Some sources, however, advocate leaving the nasogastric tube in. The child is then preoxygenated, and 0.02 mg/kg of atropine should be administered intravenously. Given intravenously in the operating room, atropine has the advantage of increasing the tone of the esophagogastric junction.5

An awake intubation should be done with the help of an assistant who stabilizes the body and head of the child. If the child is very vigorous, and awake intubation is unsuccessful, a rapid-sequence induction with cricoid pressure should be performed. In that case, succinylcholine 2 mg/kg may be used for intubation. Normally a 3-mm endotracheal tube should be used for a 5-week-old child.

Anesthesia should be maintained with nitrous oxide/oxygen and halothane. A gastric tube should be reinserted. Usually a single dose of 0.5 m/kg of d-Tubocurarine or 0.06 mg/kg of pancuronium is sufficient to provide adequate relaxation if needed. Light levels of anesthesia are generally adequate but it is extremely important that good relaxation and a quiet surgical field is provided when the pylorus is delivered, when the muscle is split and during the close of the abdomen.

Controlled ventilation should be adequate, but not excessive because one of the compensatory mechanisms for alkalosis is the retention of CO₂, which is in turn negated by the blowing off of CO₂ during hyperventilation.2

**Intraoperative complications are not common.** A celiac reflex (bradycardia, hypotension) can occur from traction upon the pylorus. Perforation at the pyloroduodenal junction can occur. Blood loss is usually minimal.5

Fluid requirements under anesthesia will be minimal if preoperative preparation has been adequate and if the surgery is rapid and deft. The solution used (assuming adequate preparation) for maintenance is isotonic saline or half-strength saline with 2.5% dextrose. This increases extracellular fluid circulating volume, corrects metabolic alkalosis, and restores extracellular osmolarity.5

Following the procedure, the muscle relaxant should be reversed with atropine 0.03 mg/kg and...
neostigmine 0.07 mg/kg. After suctioning the nasogastric tube, the child should be extubated when he is awake with protective reflexes intact.

Postoperative management

Good and experienced nursing care with constant observation in a recovery room or intensive care unit for the first 1-3 hours is mandatory for the patient with pyloromyotomy. The morbidity in infants with pyloromyotomy is mainly postoperative. The most serious and most frequently encountered problem is respiratory depression that may progress to respiratory arrest. These patients may have a high PCO₂ from compensatory mechanisms, and the added stress of anesthesia and surgery further compromises ventilation. Additional hypoventilation may raise hypercarbia to depressant levels. This may occur in a matter of minutes, even after a period that seems to be leading to a satisfactory recovery. Signs of hypoventilation are not readily observable in infants. The patient should be placed naked in the incubator, the atmosphere should be humidified oxygen, and a good stir-up regimen should be followed.

A second complication in the recovery room that the infant is susceptible to is hypoglycemia. Hypoglycemia may cause unexplained respiratory arrest, seizures, and even death. To prevent this, glucose should be administered intravenously until oral intake is adequate. Hypoglycemia can occur if infusion is stopped before oral intake is adequate, particularly if glycogen stores of the liver have been depleted. There have been reports of severe hypoglycemia occurring 2-3 hours after surgery. Oral feedings are started 8 hours after surgery. Some vomiting often occurs at first, but soon subsides. Intravenous infusion should be maintained until oral intake is adequate, usually 24 hours. Feeding should be started with plain water, and if well tolerated, advanced to Pedialyte® or Lytren®, and subsequently to dilute formula. The infants are usually discharged on the first or second postoperative day.

The gastric suction tube should be left in position for 2 hours postoperatively or until the child is completely responsive, the tube being aspirated intermittently.

Summary

In summary, the anesthetic considerations and risks that must be taken into account when anesthetizing the infant for pyloromyotomy, namely, fluid and electrolyte imbalances, have been reviewed. It is crucial to ensure safety and success that the anesthetist recognize and understand the anesthetic implications of pyloromyotomies.

REFERENCES


AUTHOR

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