Continuing Medical Education

Pyloric stenosis

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Infantile pyloric stenosis is the most frequently encountered infant gastrointestinal obstruction in most general hospitals. Although the primary therapy for pyloric stenosis is surgical, it is essential to realize that pyloric stenosis is a medical and not a surgical emergency. Preoperative preparation is the primary factor contributing to the low perioperative complication rates and the necessity to recognize fluid and electrolyte imbalance is the key to successful anaesthetic management. Careful preoperative therapy to correct severe deficits may require several days to ensure safe anaesthesia and surgery. The anaesthetic records of 100 infants with pyloric stenosis were reviewed. Eighty-five per cent of the infants were male (i.e., 5.7:1 male to female ratio) 12% were prematures. Surgical correction was undertaken at an average age of 5.6 wk, and the average weight of the infants at the time of surgery was 4 kg. A clinical diagnosis of pyloric stenosis by history and physical examination alone was made in 73% of the infants presenting to The Hospital for Sick Children. All the infants received general anaesthesia for the surgical procedure and there were no perioperative deaths.

La sténose pylorique demeure la principale cause d'obstruction intestinale survenant chez les bébés. Même si son traitement ultime doit être chirurgical, elle représente d'abord une urgence médicale. Une réanimation préopératoire adéquate est nécessaire afin de prévenir les complications périopératoires et la reconnaissance par l'anesthésiste des perturbations hydro-électrolytiques en est la clef. Il faut souvent passer plusieurs jours à corriger les déficits les plus importants afin d'assurer le succès de l'intervention. Nous avons revu 100 dossiers anesthésiques de pyloromyotomie. On y retrouvait 85% de garçons et 12% de prématurés. L'intervention survenait vers l'âge de 5,6 semaines alors que l'enfant pesait en moyenne 4 kg. Dans 73% des cas, on avait posé le diagnostic sur la base de l'anamnèse et de l'examen physique seulement. On a employé une anesthésie générale dans tous les cas et nous n'avons eu à déplorer aucun décès périopératoire.

Key words

ACID-BASE EQUILIBRIUM: acidosis, alkalosis, metabolic;
ANAESTHESIA: paediatric;
FLUID BALANCE: electrolites, ions;
INTUBATION: technique;
SURGERY: paediatric, pyloric stenosis.

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Accepted for publication 25 January, 1991.

Infantile hypertrophic pyloric stenosis is one of the most common gastrointestinal abnormalities occurring in the first six months of life.[1] Usually its presentation is quite characteristic:

> A four-week-old term infant is admitted to hospital with a five-day history of projectile vomiting. The baby is lethargic and dehydrated. His eyes are sunken and his skin feels cool and rather doughy, and when it is pinched it takes several seconds to return to its former position. The capillary refill time is more than eight seconds. He has worn the same diaper all day because his mother says that he did not produce stool or urine. He is 15% below his birth weight. During examination an olive-sized mass can be felt 3 cm below the right costal margin and gastric peristalsis is observed. He is admitted as a medical emergency due to dehydration and subsequently undergoes pyloromyotomy.

In this article reference is made to an experience in the anaesthetic management of 100 cases of infantile hypertrophic pyloric stenosis during 1986 and 1987. The demographic characteristics of this patient population are analyzed and compared with previous papers. Due to the frequent occurrence of this disease with its associated and potentially severe biochemical complications, it is appropriate to review current clinical practice and suggest improvements in management. A clinically useful relationship between the adequacy of fluid resuscitation and a urinary chloride concentration >20 mEq $\cdot L^{-1}$ is proposed and the anaesthetic management of these infants is discussed.

Aetiology

Hypertrophic pyloric stenosis is one of the most common gastrointestinal defects of infancy in the western hemisphere and surgical intervention is usually required to relieve the obstructed bowel.[1] More recently an apparent increase in the frequency of pyloric stenosis has been noted.[2,3] Frequencies of 1.4 per 1000 to 8.8 per 1000 live births have been reported.[2–4] However, Katz *et al.* reported that the overall incidence may approach 1 in 300 live births with considerable regional variation.[5]

While the condition is often labelled as being congenital, we know little of the true aetiology of this condition, and Spicer stated,[4] "We are little nearer understanding the pathogenesis of infantile pyloric stenosis than we were 100 years ago." Many non-genetic factors have been implicated in the aetiology but scientific evidence for these claims has been difficult to confirm. Proposed causes have included infections (because a seasonal incidence exists), hypoganglionosis or immaturity of ganglion cells within the pyloric muscle, hypergastrinaemia with pylorospasm, and oedema with hypertrophy secondary to the increased work of emptying the stomach. Finally, it has been suggested that milk moving slowly through the pyloric sphincter could cause irritation, oedema, and progressive obstruction.[6]

The condition appears to have some form of polygenic inheritance.[7] First-born male patients are most commonly affected with a higher incidence occurring in the offspring of affected parents. An affected mother is calculated to have a 20% chance of producing an affected son whereas the risk to the son of an affected father is only five per cent.[8] White infants are two and a half times more likely than black infants to develop pyloric stenosis and there is a male predominance of approximately 4:1. In the group under study there were 85 males and 15 females (male: female ratio of 5.7:1). This is a somewhat higher ratio than quoted values of 4:1[5] or 4.9:1,[9] but comparable with that of MacDonald and FitzPatrick who found 84% of their study group of 350 patients to be male.[10]

The clinical manifestations of pyloric stenosis usually present between the third and fifth week of life. It is rare for symptoms to occur before the second week or after five months of age.[1,4] In this review there were some additional findings of interest. Twelve per cent of our cases were premature and a further four per cent were infants from multiple gestations (three unrelated twin infants and one triplet). This somewhat high proportion of premature and multiple gestation infants may be due to the bias caused by the referral of higher risk infants to a specialist hospital from peripheral community hospitals. The conceptual age at the time of surgery was 40 to 49 wk and appeared to be a more consistent finding than the time of birth to surgery. The three twins were delivered at 24 to 36 wk gestation, yet all underwent surgery at 42 to 45 wk post-conceptual age. The triplet infant was delivered at 33 wk gestation and underwent pyloromyotomy at 41 wk conceptual age. The term infants underwent surgical correction at 5.1 wk of age compared with ten weeks of age in the premature group, with an overall average age of 5.6 wk (45 ± 2 wk corrected gestational age) comparable to a previously reported age of 5.4 wk.[11] The mean weight of the infant at the time of surgery was 4.0 ± 0.4 kg with a range from 2.2 to 5.5 kg.

Pathophysiology

The pathology is one of hypertrophy of the gastric outlet musculature. This initially produces regurgitation and eventually projectile vomiting. The aetiology is not precisely understood but the underlying pathological mechanism is known to be one of irritation with increasing oedema which evolves to muscular hypertrophy and subsequent obstruction.

Patients with pyloric stenosis may show a wide range of metabolic disturbances and can be characterized as being a chloride- (or saline)-responsive, hypochloraemic, hypokalaemic, hypovolaemic, and in some degree, hyponatraemic metabolic alkalosis. It is so termed because the metabolic disturbance can be corrected by the administration of Cl^- (NaCl or KCl).[7] Initially, the urine is alkaline with a pH > 7.0 as a result of HCO_3^- losses, with little or no Cl^- loss in the urine. Sodium losses are variable but are generally low in the face of extracellular fluid volume (ECFV) depletion while K^+ losses may be considerable secondary to the effects of aldosterone. Later the urine may become more acidic as HCO_3^- reabsorption by the proximal renal tubule becomes more efficient, and as lactic acidosis and starvation ketosis become significant factors.[12]

The wide spectrum of acid-base disturbances seen in pyloric stenosis has been previously reviewed by Touloukian *et al.*[13] With each mEq of gastric acid secreted, 1 mEq of HCO_3^- is generated. Normally as the gastric acid passes into the duodenum, it is neutralized by the secretion of pancreatic HCO_3^-. However, with obstruction of the pylorus, gastric acid is lost in the vomitus and by gastric suction while the generated HCO_3^-, rather than being secreted by the pancreas, continues to rise in the plasma. As the increased HCO_3^- load is presented to the kidney, it overwhelms the resorptive capacity of the proximal tubule, resulting in increased amounts of $NaHCO_3$ and water being delivered to the distal tubule and the collecting duct. As $NaHCO_3$ cannot be reabsorbed in the distal tubule, the kidney attempts to conserve sodium (in the face of ECFV depletion) by stimulating aldosterone secretion, resulting in a significant kaliuresis. While some potassium is lost in the vomitus, its concentration is

generally <15 mEq·L^{-1}. Of greater importance is the potassium loss in the urine in exchange for hydrogen in an effort to maintain a normal serum pH. The potassium shift intracellularly as the pH of the plasma rises represent only a small portion of the plasma K^+ loss.

Along with considerable H^+ loss from the gastric secretions, Cl^- is also lost. The resulting hypochloraemia results in maximal Cl^- conservation, with urinary $Cl^- <$ 20 mEq·L^{-1}. The urine Cl^- concentration is usually similar to that of Na^+ in hypovolaemic states since Na^+ and Cl^- are reabsorbed together. The finding of a low or absent urinary urine Na^+ concentration is virtually pathognomonic of reduced tissue perfusion and is diagnostic of hypovolaemia.[14] However, an exception occurs when Na^+ is excreted with another anion. A patient who has vomited delivers more HCO_3^- to the distal tubule and collecting duct than can be reabsorbed. As some Na^+ (and K^+) loss is obligatory with the HCO_3^- delivered to the distal tubule and the collecting ducts, the urine contains Na^+ and K^+ which is not reabsorbed despite the contracted ECFV. Therefore, urinary Na^+ cannot be relied on as an indirect measure of volume status. In contrast, since all Cl^- is reabsorbed in exchange for HCO_3^-, the urinary Cl^- concentration provides a much more accurate estimate of the volume status in pyloric stenosis. It should be measured when an apparently hypovolaemic patient has what appears to be an inappropriately high urinary Na^+ concentration. The urine will not contain Cl^- when the ECFV is contracted and severe metabolic alkalosis is present.[7] Patients with ECFV contraction and Cl^- and K^+ depletion respond to Na^+, K^+, and Cl^- replacement. A urine Cl^- concentration >20 mEq·L^{-1}, suggests volume status has been corrected. Urine Cl^- results are more relevant than serum electrolytes when assessing volume status in infants awaiting a pyloromyotomy.[7,15]

Management

Diagnosis

It should be possible to diagnose pyloric stenosis by its clinical features alone in 90% of infants.[16] The cardinal symptom is regurgitation and vomiting which usually starts between three and four weeks of age. Characteristically, the vomiting is forceful and projectile, emerging simultaneously from the mouth and nares.[4] Jaundice is an interesting complication which occurs in about eight per cent of cases and which seems to be associated with a deficiency of hepatic glucuronyl transferase and caloric deprivation. It resolves rapidly after successful treatment.[4,17] Leahy *et al.* reported that 84% of infants with pyloric stenosis had an accurate diagnosis made on clinical examination alone.[18] Visible gastric peristalsis may occur. The confirmatory physical finding is the presence of an olive-sized tumour which may be palpated at the lateral margin of the right rectus abdominus muscle or in the midline of the abdomen. Success in palpating the pyloric tumour varies from 40 to 100% but pride should not prevent the utilisation of an ultrasonographic scan or barium meal if the diagnosis is uncertain.[4] False positive results are rare. However, false negative results may occur with ultrasonography in up to 19% of cases and with contrast studies in ten per cent of cases.[8]

TABLE I Diagnosis of pyloric stenosis

History and physical examination alone	66
With ultrasound (US)	4
With contrast study (barium meal)	14
With both US and contrast study	7
Infant transferred with diagnosis	9
Total	100

The diagnosis of pyloric stenosis in this study was made by history and physical examination alone in 71% of cases (Table I). In two instances, the history, physical, and ultrasound examinations were not conclusive but required a barium meal to provide confirmation of pyloric stenosis. The time from diagnosis (as recorded in the infant's chart) to the time of surgery was 23 ± 12 hr (range 2–72 hr). A majority of these patients had received some initial fluid resuscitation while differential diagnoses were being considered, consultations sought or investigations performed.

Preoperative medical preparation

Pyloric stenosis is a medical emergency. The surgical repair is considered a minor abdominal procedure but effective preoperative rehydration is imperative. The mortality in pyloric stenosis has been greatly reduced by aggressive therapy. The patient's acid–base status, fluid deficit, and electrolyte disturbances must be corrected preoperatively.[19] In this series, the electrolyte status was determined on admission and before fluid resuscitation had commenced. Severe imbalances were found in some cases with chloride concentrations in the range of 72–86 mEq·L^{-1}, urine chloride, ≈ 0 mEq·L^{-1}, and sodium and potassium concentrations as low as 123 mEq·L^{-1} and 2.8 mEq·L^{-1}, respectively. Therefore, replenishment of intravascular volume and correction of electrolyte disturbances are mandatory and should always precede the surgical correction of the obstruction.

In recent years, with earlier diagnosis, we encounter fewer patients who are in a state of severe dehydration, alkalosis and malnutrition.[20] All our infants had electrolyte concentrations within normal limits at the time of surgery. Two patients, however, had their operations delayed because of persistent electrolyte abnormalities.

The typical metabolic disturbance as described above is a hypochloraemic metabolic alkalosis with elevated plasma bicarbonate associated with various degrees of dehydration.[21] Blood gas determination and measurement of urine Cl^- is the most effective way to assess the severity of the alkalosis and contraction of the ECFV. A base excess >15 is not uncommon. Uncorrected alkalosis may delay recovery from anaesthetic agents and cause post-anaesthetic apnoea.[4,22]

Correction of the metabolic alkalosis and the hypochloraemia in infants with pyloric stenosis depends on: (1) reestablishing ECFV, (2) replacing Na^+ and Cl^- to enable the kidney to excrete HCO_3^- and correct the alkalosis, and (3) maintaining urine Cl^- >20 mEq·L^{-1}.

For mild or moderate degrees of dehydration corresponding to 5–15% loss of body weight or a fluid deficit 50–100 ml·kg^{-1} and moderate alkalaemia (HCO_3^- 32–42 mEq·L^{-1}) replacement is achieved preferably with normal saline with half the estimated deficit given over the first six to eight hours.

For severe degrees of dehydration corresponding to a loss in body weight of >15% or a fluid deficit >150 ml·kg^{-1} with severe alkalaemia (HCO_3^- >42 mEq·L^{-1}), fluid resuscitation and correction of the acid–base disorder is achieved initially with 20 ml·kg^{-1} of normal saline. The metabolic alkalosis responds to NaCl therapy even if the K^+ deficit is not restored. Some patients with large K^+ deficit (K^+ < 2.0 mEq·L^{-1}) will not respond to NaCl administration until the K^+ deficit is replenished. Once urine output is established, K^+ is added with a maximum replacement of 3 mEq·L^{-1}·day^{-1}.

Once the deficit is corrected, maintenance fluid (2/3, 1/3 or D5W-0.2N saline and potassium supplementation) is given at a rate of 4 ml·kg^{-1}·hr^{-1}.[9] The plasma chloride concentration is used as a clinical guide in the assessment and correction of the biochemical disorder of these infants based on the rationale that when the hypochloraemia has been corrected, so too the alkalaemia.[23] The median adult Cl^- concentration of 100 mEq·L^{-1} is accepted in clinical practice as a satisfactory concentration at which adequate resuscitation is achieved. In infants, a chloride concentration of 90 mEq·L^{-1} is suggested as normal[24] but it has been found in one series that 38% of infants with a Cl^- concentration of 90 mEq·L^{-1} may remain alkalaemic.[25] It has been proposed recently that plasma chloride is a reliable variable for the assessment and correction of hypochloraemic alkalaemia during resuscitation in infantile hypertrophic pyloric stenosis and recommended that an appropriate target for plasma Cl^- concentration be at least 106 mEq·L^{-1}.[23] A plasma Cl^- concentration >105 mEq·L^{-1} corresponds to a urine Cl^- concentration >20 mEq·L^{-1} and the latter may be used to confirm that ECFV is restored. Once the infant has been properly prepared, surgery should be carried out expeditiously so that the infant may return as soon as possible to his normal environment.

Anaesthesia

Considerations

While it is possible that medical therapy alone can be used to treat this condition in some cases, the most expedient, acceptable and definitive treatment remains the Ramstedt pyloromyotomy.[4] The anaesthetic considerations for pyloromyotomy are:

1 Neonatal anaesthesia (physiological features of the newborn under general anaesthesia, e.g., respiratory and cardiovascular physiology, body temperature homeostasis, monitoring),
2 Restoration of fluid and electrolyte balance,
3 "Full stomach" due to high bowel obstruction (danger of regurgitation and pulmonary aspiration),
4 Adequate surgical muscle relaxation (danger of the duodenal mucosa tear during pyloromyotomy),
5 Postoperative analgesia and related complications.

Monitoring

A precordial stethoscope, blood pressure cuff, radial doppler, ECG, and pulse oximeter should be used routinely for induction. Further intraoperative monitoring should include an oxygen analyzer, airway pressure monitor with low pressure disconnect alarm, peripheral nerve stimulator, end-tidal CO_2 ($ETCO_2$) monitor and temperature probe. The beneficial effect of warm room temperature before incision has been disputed.[26–29] Studies at this institution have demonstrated that, in a warm or cold environment, there is an initial decrease in body temperature due to an internal redistribution of heat.[28–29] It accounted for 0.6° C in the first hour of anaesthesia. We were unable to show differences with regard to complications or recovery from anaesthesia between a cold and warm group of patients undergoing abdominal surgical procedures lasting >90 min.[28–29] There are no differences among tympanic membrane, oesophageal, rectal, and axillary temperatures.[30] Since pyloromyotomy is a short surgical procedure, axillary temperature as monitor of core temperature is very convenient.

Pre-induction

It has been suggested that vomiting is usually not a problem in neonates and infants, as the stomach can be completely emptied by aspiration,[31] whereas Unal *et al.* showed that even after gastric suction there is always a residual gastric volume of 30 to 100 ml.[32] After atropine 20 μg·kg^{-1} *iv* the gastric tube may be removed with continuous suction. With the infant placed on the right

side and after preoxygenation, a red rubber tube sized 8–12 french should be passed into the stomach to further insure reduction of gastric contents.

Either local or general anaesthesia techniques may be used for pyloromyotomy. However, complications have been reported during pyloromyotomy with local anaesthesia in 12.4% of patients compared with 7.5% with general anaesthesia.[33] Thus, general anaesthesia is usually preferred.[4]

Induction and intubation

While the method of tracheal intubation remains controversial (i.e., awake or asleep), the practice of inhalational induction of anaesthesia followed by intubation recommended by Steven *et al.* in 1973[31] appears to have been abandoned. The induction technique that will be chosen will depend upon whether an awake intubation or a rapid sequence induction of anaesthesia is planned. The Sellick manoeuvre is recommended.[34] As these infants usually present for operative correction between their third and fifth weeks of life, some degree of mucosal damage may result from an awake intubation. Indeed, if the infant is not sufficiently vigorous, one should question whether fluid resuscitation has been adequate. A chart review of the 55 infants in this series who were intubated asleep failed to reveal any evidence of trauma on intubation, vomiting on induction, aspiration, or failed intubation.

While intubation following a rapid sequence intravenous induction may be accomplished more quickly and less traumatically, the anaesthetist must assess his experience and skills, as well as the infant's airway, and decide which is the safer method. If the anaesthetist questions his ability to expose the larynx, he can attempt a quick direct laryngoscopy without anaesthesia. If adequate visualization of the larynx is achieved, he can proceed safely with an intravenous induction-intubation technique. If adequate visualization is not achieved, he should obtain more expert assistance or refer the patient to a specialist centre. If a rapid sequence *iv* induction is chosen, with preoxygenation and cricoid pressure, thiopentone 3–5 $mg \cdot kg^{-1}$ and succinylcholine 2 $mg \cdot kg^{-1}$ should be given. It is by far the most popular technique (Table II). Ketamine dissociative anaesthesia has been suggested as an alternative to general tracheal anaesthesia.[35] Chatterjee *et al.* suggested that intubation of neonates even in expert hands can be hazardous and ketamine could obviate this problem especially where expert help is not readily available. However Bush states:[36] "The only acceptable solution is that if general anaesthesia is deemed to be difficult in a particular circumstance, and where there is no emergency, an experienced anaesthetist should be available or the patient should be transferred to a paediatric facility. The safety of the patient is of paramount importance and it should never be compromised because of inexperience or inappropriate environment."

TABLE II Intubation

Thiopentone/atropine/succinylcholine*	53
Atropine/succinylcholine	1
Halothane induction	1
ETT *in situ*	1
Awake intubation	44
Total	100

*Includes one patient after failed awake intubation.

In the cases under review 44% were intubated awake compared with 32% in an earlier review.[1] Awake intubation has continued to be popular in this institution because it is one of the few opportunities available to teach the technique.

Maintenance

The anaesthetic considerations during maintenance of anaesthesia are:

1 Anaesthetic agents,
2 Ventilation,
3 Muscular relaxation,
4 Fluid management,
5 Maintenance of body temperature.

Maintenance of anaesthesia in all but two of the patients in this review was by a mixture of N_2O in oxygen and a volatile agent. Halothane and isoflurane are suitable choices to maintain anaesthesia (Table III).

Intermittent positive pressure ventilation should be used in association with muscle relaxation. The use of muscle relaxation improves the surgical conditions and allows the dose of inhalational agents to be reduced. This not only makes the surgical task easier, but also minimizes the risks of perforation of the duodenal mucosa. The time taken from anaesthetic induction to arrival in the postanaesthetic recovery room was 70 ± 13 min with a range of 45 to 90 min. Atracurium in a dose of 0.3–0.4 $mg \cdot kg^{-1}$ was used to provide satisfactory muscle relaxation in the majority of the cases. Other relaxants included d-tubocurarine, gallamine, and pancuronium.

Bleeding is not usually a problem. Peroperative *iv* fluid should consist of five per cent glucose in 0.2 N saline to

TABLE III Anaesthetic agent used for maintenance

Halothane	45
Isoflurane	47
Halothane/isoflurane	6
Narcotic alone	2
Total	100

N_2O used in all but two cases.

avoid hypoglycaemia. (see postoperative management). If additional fluids are needed to replace lost extracellular fluid a multiple electrolyte solution such as Ringer's lactate is appropriate.

Maintenance of body temperature can be ensured by warming the inspired gases with a heat and moisture exchanger.[29,37]

Emergence

The anaesthetic considerations for emergence of anaesthesia are:

1 Elimination of anaesthetic agents,
2 Reversal of neuromuscular blockade,
3 Stomach contents.

At the end of the operation, time must be allowed for the elimination of anaesthetic agents. The tracheal should be extubated after muscle paralysis has been reversed with neostigmine and atropine (50 $\mu g \cdot kg^{-1}$ and 20 $\mu g \cdot kg^{-1}$ respectively). It is desirable that the trachea should not be extubated until the infant is fully awake, vigorous, and demonstrates purposeful movements, spontaneous respiration, and an active gag reflex.

Postoperative management

The anaesthetic considerations for the immediate postoperative period are:

1 Oxgenation and respiration,
2 Analgesia,
3 Intravenous fluid management,
4 Hypoglycaemia,
5 Vomiting.

All infants should receive supplemental oxygen and be placed on an apnoea monitor for at least two hours in the postanaesthetic recovery room.

The use of opiates in infants is controversial. Opiate analgesics are best avoided because infants <46 wk gestational age (full term plus 6 wk) exhibit an increased sensitivity to these drugs and there is a risk of postoperative apnoea.[8,38] In our series, two infants received fentanyl 1 $\mu g \cdot kg^{-1}$ *iv* post-induction. Despite this small dose in these full-term infants, both demonstrated abnormal respiratory patterns after surgery. As respiratory depression in the postoperative period has been described,[10] the use of opiates in these infants is not recommended. Postoperative analgesia was most commonly provided by rectal acetaminophen (10 $mg \cdot kg^{-1}$). The technique of wound infiltration with bupivacaine is an effective alternative frequency forgotten. The only analgesic required by a large majority of infants is infiltration at the completion of surgery with 0.25% bupivacaine 0.25 to 0.50 $mg \cdot kg^{-1}$.

Intravenous fluid administration should continue at the maintenance rate with five per cent glucose in 0.2 N saline until oral feeding is established. Previously, feeding was attempted 2–4 hr after surgery but this was often associated with vomiting due to depression of gastric motility for 12–18 hr postoperatively.[39] Minor degrees of transient postoperative vomiting are common. More persistent vomiting is unusual and most often due to associated gastroesophageal reflux, which occurs in 10–13% of patients.[10]

Potential fatalities can occur during recovery.[40] Hypoglycaemia is always a serious hazard and is due to hepatic glycogen depletion. The first signs of hypoglycaemia are lethargy and irritability progressing to convulsions and cardiac arrest.[41]

Morbidity and mortality

Although the morbidity of pyloric stenosis is likely to remain small but important, the mortality of this condition has been reduced considerably.[10] The availability of skilled paediatric anaesthetists and the use of general anaesthesia has been of benefit to both surgeon and patient by enhancing the safety of the procedure.[4] The operative mortality of ten per cent has declined steadily to <0.5%.[1,4,20,33]

Recommendations

The essential concerns of the anaesthetist managing the infant with pyloric stenosis include:

1 *Adequate fluid resuscitation before surgery.* This should be confirmed by clinical examination with evidence of good urine output, and may be supported with the measurement of urinary chloride >20 $mEq \cdot L^{-1}$.
2 *Correction of acid–base and electrolyte abnormalities.* It is unsafe to operate with an uncorrected alkalosis because of delayed recovery from anaesthesc agents and the danger of post-anaesthetic apnoea occurring in alkalaemic infants.
3 *Maintain normothermia intraoperatively.*
4 *Prevent gastric aspiration.* Measures include maintaining the infant in a fasting state preoperatively with nasogastric suction, passing a red rubber catheter for gastric suction prior to induction, establishing general tracheal anaesthesia (either with an awake intubation or following a preoxygenation, rapid sequence intravenous intubation technique), and extubating the trachea with the infant awake on his side.
5 *Inhibition of vagal reflexes.* Atropine (20 $\mu g \cdot kg^{-1}$) is given prior to any airway or gastric manipulation.
6 *Adequate muscle relaxation with intermittent positive pressure ventilation* during the surgical division of the hypertrophied pylorus. The effects of the non-depolarizing muscle relaxants must be reversed before extubation.

7 *Risks of parenteral narcotics*. Rectal acetaminophen for postoperative analgesia or wound infiltration with 0.25% bupivacaine 0.25 to 0.50 ml · kg^{-1} are recommended alternatives.

Acknowledgements

The authors would like to thank Drs. Bernard Braude, Lawrence Roy and John E.S. Relton for their advice in the preparation of the manuscript.

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Self-assessment Questionnaire

EACH OF THE QUESTIONS BELOW IS FOLLOWED BY FIVE SUGGESTED ANSWERS OR COMPLETIONS. SELECT THE ONE THAT IS BEST IN EACH CASE.

1 In infants, hypokalaemic alkalosis may
- A shift the oxygen dissociation curve to the right,
- B occur in the presence of pyloric stenosis,
- C is usually well compensated by respiratory acidosis,
- D occurs because the kidney excretes K^+ in order to maintain normal CL^- levels,
- E always require therapy with dilute solutions of HCL.

2 The cardinal symptom of pyloric stenosis is
- A the "olive-sized" tumour felt around the lateral margin of the right rectus abdominus.
- B jaundice associated with failure to thrive,
- C projectile vomiting which usually starts between six and eight wks of age,
- D dehydration,
- E none of the above.

3 Patients with pyloric stenosis usually have
- A normal ECFV,
- B normal urinary sodium,
- C contracted ECFV with normal urinary Na^+ concentration,
- D urinary chloride >20 mEq $\cdot L^{-1}$,
- E normal serum K^+ concentration.

4 Which of the following is not correct concerning anaesthesia for pyloric stenosis?
- A spontaneous ventilation and light anaesthesia should not be used during pyloromyotomy,
- B duodenal mucosa tear is the most common surgical complication,
- C ketamine anaesthesia should be used when the anaesthetist considers that he would not be able to manage the infant's airway,
- D when the infant FCFV has been completely replenished, an intravenous catheter is not necessary,
- E when the infant has been corrected medically the surgery should be done in an expeditious manner to limit the separation time from the mother.

5 Which of the following statements regarding the jaundice associated with pyloric stenosis is true?
- A it is caused by a high caloric intake but a deficiency in hepatic glucoronyl transferase,
- B it is a contraindication to the use of halothane anaesthesia,
- C it is observed in 30% of pyloric stenosis infants,
- D it resolves spontaneously after successful surgical treatment,
- E pyloromyotomy should be delayed until jaundice has been corrected medically.

FOR EACH OF THE QUESTIONS OR INCOMPLETE STATEMENTS BELOW, ONE OR MORE OF THE ANSWERS OR COMPLETION GIVEN IS CORRECT.

A if 1, 2, and 3 are correct
B if 1 and 3 are correct
C if 2 and 4 are correct
D if only 4 iscorrect
E if all are correct

6 Frequently associated with congenital hypertrophic pyloric stenosis
- 1 vomiting starting usually in second or third week of life,

2 more common in female,
3 gastric peristaltic waves are visible,
4 hyperglycaemia is a common sign.

7 Potassium losses are increased in the presence of
1 nasogastric suction,
2 large renal excretion of HCO_3^-,
3 hyperventilation,
4 acidosis.

8 The adverse effects of metabolic alkalosis in infants are
1 increase in pH results in shifting of the oxygen dissociation curve to the left, binding more O_2 to the Hb and unloading less oxygen at the tissue level,
2 associated with high level of fetal Hb, the P_{50} is largely displaced to the right,
3 increased potential for postoperative delay in recovery and respiratory depression with apnoea,
4 if uncorrected increase in blood potassium concentration.

9 Which of the following features are to be considered during post-anaesthesia recovery?
1 reinstitution as soon as possible of oral feeding,
2 incidence of vomiting remains elevated,
3 opiate agents should be used carefully to control pain postoperatively,
4 hypoglycaemia could be fatal.

ANSWERS:
1 A 3 C 5 D 7 A 9 C
2 C 4 C 6 B 8 B