ANAESTHESIA FOR PYLOROMYOTOMY IN INFANCY

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CONGENITAL HYPERTROPHIC PYLORIC STENOSIS is a relatively common condition occurring once in every 250[1] to 300[2] live births. Because it is the most common lesion requiring surgical treatment in infancy,[19] and has certain anaesthetic features of importance, it was decided to review the subject briefly and report our anaesthetic experience. The paper is based on 441 consecutive cases treated by operation at The Hospital for Sick Children, Toronto, in the years 1957 to 1960 inclusive.

PATHOLOGY

Aetiology

The aetiology of this disease is unknown, but at least fourteen different theories have been suggested. The most striking fact is that it is found predominantly in males. In this series, the incidence in males was 79.45 per cent, which is almost identical with the accepted figure of 80 per cent. There is also an increased incidence in the first-born, and a familial tendency.[2] Additional congenital anomalies are frequent and were present in 20 cases (4.5%) in this series. The lesion is present at birth, but there is usually a lag before the onset of symptoms.

Gross Pathology

A post mortem of an infant who has died from this disease reveals only one primary pathological lesion. A firm, gristle-like, sausage-shaped mass about 2–3 cm. in length is present at the pylorus, ending abruptly at the pyloric ring. This is the so-called pyloric tumour. The mass is actually the result of smooth muscle hypertrophy occurring mainly in the fibres of the circular layer, and to a lesser extent in the longtitudinal layer. On section, the pylorus is much thickened, often elongated, and the lower end may project into the duodenal lumen somewhat like the uterine cervix into the vagina. The lumen will admit only a fine probe. Frequently water cannot be forced through the stenosed pylorus, perhaps because the mucous membrane has been thrown into folds. The pyloric canal is obstructed by this mass, which results in gastric dilatation and hypertrophy.

Microscopic Pathology

There is a two- to three-fold increase in both size and number of smooth muscle strands of the pylorus. The other layers of the pylorus are also thickened, but to a much less degree. The stomach wall is thickened by oedema and is infiltrated with chronic inflammatory cells. No neurological abnormality has been discovered that might explain the muscular hypertrophy.[19]

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Pathological Physiology

The pathological disturbances are entirely the result of vomiting, secondary to pyloric obstruction. At birth, the hypertrophied pyloric musculature does not usually cause obstructive symptoms. The lumen is usually adequate for the first week of life, although there may be delayed emptying of the stomach. Perhaps the mechanical effect of passage of curds adds oedema or spasm, because the degree of obstruction gradually increases, gastric dilatation occurs, and vomiting begins. If untreated, the stomach undergoes further dilatation, and all feedings are vomited, either immediately, or after a variable period of time. (See the diagram.) The loss of normal feedings, plus gastric juice, results in reduced caloric intake with water and electrolyte loss as well, producing dehydration, metabolic disturbances, and failure to gain weight. The excessive chloride loss in gastric juice causes a retention of HCO_3 in plasma producing hypochloraemic alkalosis.

Clinical Features

Symptoms

Vomiting. A history of vomiting is invariably present. In this series it was projectile in all cases. It may commence at birth (as in 16.3%), but usually begins at one week to 10 days of age. After the infants have been nursed and gained weight, vomiting begins without evident cause, at first occasionally and then habitually. Emesis becomes forceful and later projectile. Blood is often present, but never bile. Constipation and weight loss follow.[10]

Signs

Physical appearance. Dehydration and evidence of weight loss vary according to the duration and extent of vomiting. Dehydration was absent in 59.7 per cent of our series, indicating an early diagnosis.

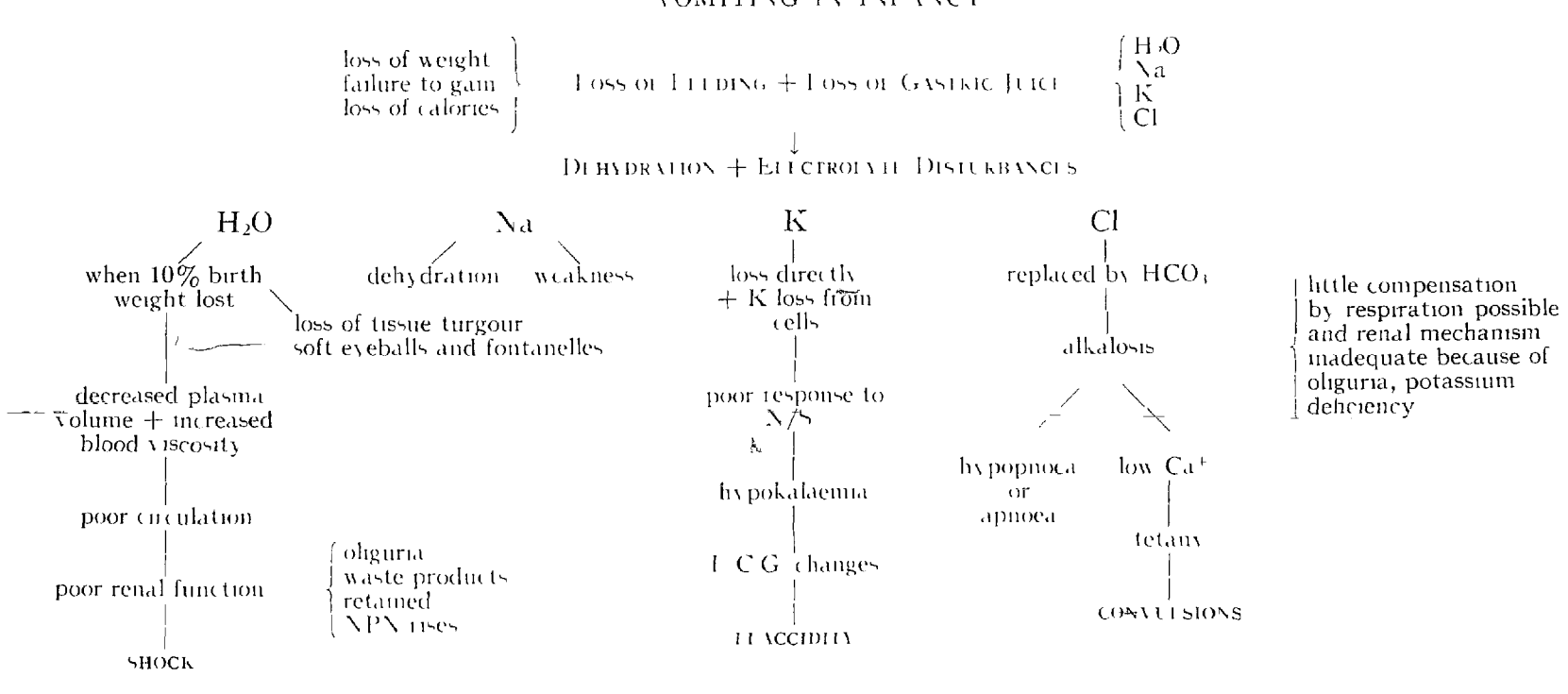
Epigastric distension is observed during or after feeding and visible peristalsis is frequent (56%).

A pyloric tumour, a 2–3 cm. olive-shaped mass, is usually palpable in the upper abdomen halfway between the umbilicus and xiphoid to the right of mid-line (87.9%).

Laboratory

If a tumour cannot be palpated or confirmation is desired, a barium swallow test may be done (20.1% of cases). Typical radiological findings are:

(*a*) Enlarged stomach with a rounded end due to "ballooning," or a curving of the antrum.
(*b*) Greatly increased gastric peristalsis.
(*c*) Passage of barium to pylorus, but escape of only a tiny amount of it into the duodenum.
(*d*) An elongated and greatly narrowed thread-like lumen to the pylorus ("string sign").
(*e*) Abnormally long retention of barium in stomach.


VOMITING IN INFANCY
loss of weight
failure to gain
loss of calories
Loss of Feeding + Loss of Gastric Juice
H2O
Na
K
Cl
Dehydration + Electrolyte Disturbances
H2O
when 10% birth weight lost
loss of tissue turgour
soft eyeballs and fontanelles
decreased plasma volume + increased blood viscosity
poor circulation
poor renal function
SHOCK
oliguria
waste products retained
NPN rises
Na
dehydration
weakness
K
loss directly + K loss from cells
poor response to N/S
hypokalaemia
ECG changes
FLACCIDITY
Cl
replaced by HCO3
alkalosis
little compensation by respiration possible and renal mechanism inadequate because of oliguria, potassium deficiency
hypopnoea or apnoea
low Ca++
tetany
CONVULSIONS

Diagnosis

In a typical case, the diagnosis is not difficult. Holt[14] states that abnormal gastric retention, visible peristalsis, and a palpable pyloric tumour are essential requirements.

Preoperative Preparation

It is generally agreed that these patients are never a surgical emergency. Therefore, the sicker the infant the more care and attention must be given to adequate preoperative preparation. In most centres, this is the responsibility of the paediatrician or surgeon, but the anaesthetist must also be fully conversant with the infant's condition. Inadequate treatment is associated with marked circulatory instability, so that extra hazards await the unwary anaesthetist. Operation need not be delayed for many days while fluids and electrolytes are completely returned to normal, as this is unnecessary and death from aspiration has occurred during such periods.

These infants have been classified by Benson[7] in three groups: mild, moderate, and severe.

Mild. In these cases dehydration is minimal (±) as is metabolic alkalosis (±) with serum bicarbonate levels below 32 mEq./L. Treatment is satisfactory with almost any method, such as stock premature formula q.4.h or intravenous 5 per cent glucose and water.

Moderate. There is moderate dehydration (2+) and metabolic alkalosis in these cases with the serum bicarbonate levels between 32 and 42 mEq./L. An intravenous of ⅝ to ½ plus one-sixth molar ammonium chloride (2 c.c./lb. for each mEq./L. of bicarbonate above 30) will restore normal fluid balance. After hydration, 10–20 mEq./L. of potassium should be given over 24 hours.

Severe. In severe cases malnutrition is present associated with dehydration (4+), metabolic alkalosis (4+), and serum bicarbonate levels greater than 42 mEq./L. Normal saline should be administered at the rate of 60–90 c.c./lb./24 hours, which can be increased if significant weight loss is present. These measures should produce a normal urine output of 8–20 c.c./hr. If the patient is hypokalaemic after hydration, 40 mEq. of potassium should be added per 24 hours. If the haemoglobin is low, 5–10 c.c. of blood should be used instead of the normal saline.

In this series 247 patients (56%) received intravenous therapy pre-operatively, but electrolyte determinations were considered necessary in only 129 cases (29.2%). Eight patients (1.8%) received blood preoperatively, averaging 80 cc. each with a range of 50–140 cc. These findings indicate very early diagnosis and, therefore, a high proportion of the series were in the "mild" group.

Anaesthetic Management

Prior to a discussion of anaesthetic agents and techniques, certain factors relating to the management of these cases should be considered. These factors fall into three groups: (*a*) physiological features, (*b*) pathological disturbances, (*c*) surgical requirements.

(a) Physiological Features

The patient is a neonate or in early infancy (the average age in this series was 31 days with a range of 3 days to 129 days). Therefore, certain qualitative as well as quantitative physiological differences from older children and adults are present.

(*i*) *The circulatory system.* The circulatory system favours a central "pooling" of the blood with a paucity to the peripheral circulation.[4] The arterial blood pressure is kept at a relatively high level (75–80/40–50) by a high peripheral resistance and a relatively high elastic resistance in the aorta and its main branches. The heart rate is rapid, the stroke volume small, and the pulse pressure is narrow. As a result there is a little latitude for regulation or compensation and the blood pressure is very labile. Even a minor stress of anaesthesia or surgery may thus produce exaggerated responses.[3]

(ii) *The respiratory system.* The metabolic rate is at its peak in infancy (at approximately 60 cal./hr./m^2) and the oxygen consumption of 6 ml./kg./min. is approximately one and one-half times the average adult's of 3.5–4.0 ml./kg./min. With high oxygen requirements and rapid circulation, cyanosis occurs promptly with apnoea.

To alter alveolar ventilation, breathing is altered by changes in the rate and/or tidal volume (minute volume = tidal volume × rate). In neonates, respiration is almost entirely diaphragmatic owing to (i) the existing semi-lateral position of the ribs and the costal cartilages and (ii) the shape of the infant's thorax, which makes a piston-like action of the diaphragm more effective than a bellows-like movement of the ribs.[6] Since the extent of diaphragmatic movement is limited, a need for increased ventilation must be met principally by increasing the respiratory rate and not by increasing the tidal volume. The normal rapid respiratory rate (40–50/min.) in infants is well known. When the rate is increased, higher air-flow velocities occur causing increased resistance and the expenditure of more energy. Therefore, the necessity for increased ventilation during anaesthesia may easily lead to tachypnoea with hypoventilation and fatigue.

The dead space/tidal air ratio is 0.31 in adults and 0.32 in neonates.[6] However, in neonates, any additional increase in dead space results in a tremendous change in this ratio. Much anaesthetic equipment used for this age group is too large and readily produces respiratory and anaesthetic difficulties. However, suitable equipment is gradually eliminating these problems.

(iii) *Temperature regulation.* In air-conditioned operating rooms hypothermia is an additional problem. This occurs readily because of the very low absolute heat production, the immature heat regulating centre, the central pooling, the excessive prepping, the cool room, the depression of muscles and metabolism, etc.

(b) Pathophysiological Disturbances

These patients have the equivalent of a "high bowel obstruction" with all its secondary consequences (already mentioned). Complete restoration of water and electrolytes is not essential so that some degree of dehydration and electrolyte imbalance is always present. Gross miscalculation in the restoration of water and electrolytes leads to a circulatory instability, and marked ill-effects may occur during anaesthesia.

(c) Surgical Requirements

Little need be said regarding the surgical procedure as everyone is familiar with the Fredet-Ramstedt operation. It is performed through a right upper quadrant incision. Upper abdominal procedures require more analgesia and relaxation than any other area if optimal surgical requirements are to be met. With adequate anaesthesia and relaxation by any method, the muscles of respiration are inevitably affected and weakened. If, in addition, there is limitation of diaphragmatic movement due to gastric distension, abdominal packs, surgeon's fingers, etc., gross alveolar hypoventilation occurs, ultimately affecting the circulation.

Preoperative Preparation

Assuming that adequate preoperative preparation has been carried out and consideration given to the foregoing points, premedication is ordered. Atropine sulphate (0.02 mg./lb.) is given subcutaneously half an hour pre-operatively. The value of atropine is well known for anaesthesia in this age group[30] and it is considered essential if succinylcholine is to be used. In this series, 82.2 per cent received atropine preoperatively.

Before induction, the stomach must be emptied, whether local or general anaesthesia is used. Even with a gastric tube in place, as much as 100 c.c. of fluid may be removed by syringe. If during this procedure massive regurgitation occurs, aspiration can result even with a conscious infant. Therefore, a mouth suction must be instantly available.

At the time of induction, the infant should be placed on the operating table with a heating blanket or hot water bottle, and later, a rectal thermocouple is inserted. When the stomach is emptied, the blood pressure cuff and stethescope are attached and restraints may be applied. The infant is then ready for induction with either local or general anaesthesia. For completeness, infiltration and regional methods will be included although they were not used in this series.

Local Anaesthesia

Infiltration

Infiltration anaesthesia has been in widespread use for many years. The simplest method is the injection of procaine 0.5 per cent at the operative site while the infant sucks on a nipple. In skilled hands this method produces quite satisfactory anaesthesia.[31] Denis Browne's method[12] consists of preparation with intravenous fluids and gastric lavage. Preoperatively, 2 grains of Chloral are administered. At operation, the infant is tied to a padded frame and 0.5 per cent procaine (containing epinephrine) is injected up to a total of 15 c.c. After waiting five minutes, a right rectus incision is made and pyloromyotomy performed. He reports a 2 per cent mortality in 407 cases at the Hospital for Sick Children, Great Ormond Street (1943–45), with no operative mortality.

After this dose of Chloral, the infant is sometimes markedly depressed for a period of 4 to 8 hours. If severe pain is produced, as with gastric retraction, hypnosis and analgesia are not adequate and regurgitation may occur causing either laryngospasm or aspiration.

Epidural

Ruston[13] in 1954 described an epidural technique for pyloromyotomy using 5–6 c.c. of 1 per cent xylocaine with 1:200,000 epinephrine. Although 7 out of his 20 cases needed supplemental anaesthesia, he described the results in a total of 54 cases as excellent. No endotracheal intubations were performed and the patient's skin colour remained pink throughout the procedures. No mention is made of the extent of anaesthesia or blood pressure changes. In addition to those immediate reactions due to inadvertent subarachnoid or intravenous injection, the possible delayed complications of epidurals have only been described recently.[20] Although the use of epidural anaesthesia remains unconvincing, the author has had no personal experience with it for this operation.

General Anaesthesia

In this series general anaesthesia was administered to all cases by a total of 32 different internes and 9 different staff members. A wide diversity of agents and techniques were employed, as can be seen from Table I (Tubocurare was also administered in some cases but was unfortunately omitted from the table). It is evident that any agent or technique of general anaesthesia can be used. However, certain methods are obviously preferable when considering possible dangers, physiological trespasses,[29] or surgical inadequacies. Therefore, various alternatives will be discussed briefly in a chronological sequence.

Induction

Open drop. (*a*) Smith[22] recommends the use of open-drop divinyl ether followed by ether in strong infants. He admits that members of this group are unusually hard to put to sleep with the open-drop technique and explains it on the basis of "age group." "If they are not anaesthetized after 10 minutes of attempted induction, it has proved expedient to put them on the operating table, restrain their limbs and start the operation, in the meantime continuing to administer the anaesthetic." Divinyl ether induction was not used in this series, but the preceding statement is self-explanatory.

(*b*) Ethyl chloride induction was used in 104 (46.2%) of the patients and was followed by ether in 67 cases (15%). This agent allows a smoother, more rapid induction than divinyl ether, but there is a possibility of circulatory depression in unskilled hands.

The simplicity of the open-drop technique is offset by such factors as the impossibility of assisting or controlling ventilation, and the excessive dead space is difficult to counteract by adding oxygen without reducing anaesthetic concentrations.

Intravenous. (*a*) Thiopentone was used in 34 cases (7.7%) as either a 2.5 per cent or 1.25 per cent solution, but was not satisfactory. Despite the very slow administration, apnoea, laryngospasm, or hypotension occurred in some infants.

(*b*) Relaxants may be administered either intravenously or intramuscularly when an infant is fully conscious. Later intubation is performed and anaesthesia begun. This technique is not aesthetically satisfactory, and during the brief

TABLE I

Anaesthetic agents	No. of cases
Anectine and ether	45
Anectine, ether, and nitrous oxide	51
Anectine, ether, nitrous oxide, and fluothane	35
Anectine, ether, and fluothane	2
Anectine and nitrous oxide	26
Anectine, nitrous oxide, and fluothane	38
Anectine, ether, and ethyl chloride	2
Anectine and fluothane	1
Fluothane, oxygen, and nitrous oxide	54
Fluothane, oxygen, nitrous oxide, and cyclopropane	3
Fluothane, oxygen, and cyclopropane	2
Fluothane and nitrous oxide	2
Fluothane and ether	7
Fluothane and ethyl chloride	1
Ether and cyclopropane	3
Nitrous oxide	2
Nitrous oxide and ether	8
Ethyl chloride, anectine, and nitrous oxide	2
Ethyl chloride	1
Ethyl chloride and ether	67
Ethyl chloride, ether, and nitrous oxide	22
Ethyl chloride and fluothane	2
Ethyl chloride, fluothane, and nitrous oxide	5
Ethyl chloride, ether, trilene, and nitrous oxide	1
Ethyl chloride and nitrous oxide	5
Ethyl chloride, nitrous oxide, and pentothal	1
Ether, trilene, and nitrous oxide	3
Pentothal, ether, anectine, and nitrous oxide	9
Pentothal, anectine, and nitrous oxide	17
Pentothal, anectine, fluothane, and nitrous oxide	1
Pentothal, anectine, nitrous oxide, and syncurine	3
Pentothal, syncurine, and nitrous oxide	2
Pentothal, ether, cyclopropane, and nitrous oxide	2
	427
Unrecorded	14
TOTAL	441

period of struggling, ventilation is difficult and cyanosis or aspiration can occur. During ventilation preceding intubation of a paralysed infant, it is easy to distend the stomach and any remaining gastric contents may be regurgitated.

Closed system. The closed technique allows assisted or controlled ventilation to be carried out easily. Accurate concentrations of gases are possible as well as increased humidity. Its principal disadvantages include the resistance of valves and apparatus, plus the mechanical and chemical problems related to the use of soda lime.

Semi-closed. The use of Ayre's technique as modified by Rees[32] overcomes some of the disadvantages of a closed system as long as high flow rates are used. Any agent or combination of agents which produces a safe, smooth induction can be used.

The following technique has been found to be quite satisfactory. Using Ayre's technique, induction is begun with oxygen and high fluothane concentrations (up to 2%). The infant falls asleep after 5 to 10 respirations and, at

that stage, low concentrations of ether are added. During the next few minutes, the ether concentration is gradually increased while the halothane concentration is gradually diminished. With experience, a smooth transition of agents can be easily accomplished. Hypoventilation from excessive halothane or breath-holding from a too rapid concentration of ether may occur, so that an endotracheal tube and stillette should be available for use if required. Throughout this period, the blood pressure and respirations must be closely monitored.

Intubation

Following induction, it is essential that the patient is intubated. Not only does an endotracheal tube prevent aspiration and laryngospasm, but it also allows light levels of anaesthesia and permits controlled respiration including hyperventilation. Marriott[16] has even recommended intubation for all operative procedures in this age group. It should be emphasized that in infants there is no visible indication of endobronchial intubation during spontaneous respiration. Therefore auscultation of the chest must always be performed.

Intubation may be performed with the infant awake or following the use of relaxants or general anaesthesia.

(*a*) *Awake.* In the fully conscious weak or premature infant, intubation is technically easy and is probably the safest means of producing anaesthesia. However, strong infants may twist and struggle, causing trauma.

(*b*) *Relaxants.* In neonates, the relaxants have been shown to predispose towards postoperative respiratory depression either directly, or in connection with mild hypothermia.[17] Stead[15] states that the neonatal infant shows increased sensitivity to tubocurare, but is relatively resistant to succinylcholine. Since the Fredet-Ramstedt procedure is brief in duration, curare may well outlast the period of operation. Even with the use of an antidote, recurarization is a distinct possibility. Hypokalaemia is present in some patients, which enhances the sensitivity of the myo-neuro junction to curariform drugs. In paediatric anaesthesia, long-acting drugs mean lack of control over the patient and shorter-acting ones should be substituted for them whenever possible.

The brief duration of effect of succinylcholine allows "titration" of this drug to meet rapidly changing requirements. However, it is not free from circulatory effects.[17,18] Tachycardia, bradycardia, or arrhythmias have all been reported. The author witnessed one case when the patient did not receive atropine and the intravenous injection of succinylcholine produced a transient state suggestive of pulmonary oedema. Cardiac arrests have been reported from suxamethonium.*

Decamethonium is not recommended because it offers no advantages over other relaxants and has the disadvantage of uncertain duration and irregular muscle relaxation.

Intubation under general anaesthesia is the simplest and safest technique for most infants. This can be accomplished using oxygen, fluothane, and ether, as already mentioned. When a moderate depth of anaesthesia and relaxation is

*Since this paper was prepared, two infants with pyloric stenosis received 5 and 8 mg. of succinylcholine. After intubation, a period of prolonged apnoea occurred lasting 3 and 5 hours, respectively. Although this complication is infrequent, it is an additional hazard.

reached, ventilation and circulation are not depressed with this method. Unhurried intubation can then be performed without coughing, cyanosis, or regurgitation.

Maintenance

Any anaesthetic technique is satisfactory which produces analgesia and relaxation while avoiding circulatory or ventilatory depression. In practice, oxygen or nitrous oxide – oxygen is administered, to which ether, halothane, cyclopropane, or trilene can be added as required. In most strong infants, halothane in 0.5 per cent concentration produces excellent anaesthesia. In weak or premature infants, cyclopropane may be preferable because of the minimal depression of the circulation. Unfortunately, in this age group, cyclopropane can cause very severe laryngospasm following extubation, unless ether or other agents are also used.

Following intubation, the administration of nitrous oxide – oxygen in a 3:1 ratio is commenced. Halothane is reduced to 0.5 per cent concentration and the ether discontinued. Ether may be added later, to produce relaxation during opening or closing of the peritoneum.

Hypoventilation is to be expected if adequate relaxation is present, and this may be aggravated by retractors, gastric distension, etc. The necessity for controlled respirations has been outlined by Jackson Rees, and this technique was instituted in 129 cases (29.2%) of this series. Vigorous hyperventilation enables the use of extremely light levels of anaesthesia (owing to alkalosis) without harmful biochemical effects[14] and is recommended.

Following peritoneal closure, halothane is discontinued and later, during skin closure, nitrous oxide is also discontinued. Thus, the infant should be well oxygenated and awake at the completion of the operation. Extubation may then be performed and the patient observed for possible laryngospasm. However, this complication is rare in these circumstances.

Recovery

Even when the child appears conscious, the onset of respirations may be delayed following vigorous hyperventilation. This reaction is prone to occur in weak or premature infants especially if associated with hypothermia or excessive depth of anaesthesia. For this reason, anaesthesia is "lightened" towards the end of the procedure allowing surgical stimuli to initiate spontaneous respirations. Following skin closure, if the child is apnoeic but appears fully conscious, nasal or tracheal suction may also be successful in triggering respirations.

Postoperatively

The infant is then returned to the recovery room. Although the infant may be fully conscious and vigorous at the end of the operation, respiratory depression can occur up to half an hour or more postoperatively so that a period of observation is necessary. This complication is particularly associated with prematurity or hypothermia. Circulatory depression is rare in the operative and postoperative stages of a well-prepared infant, so that intravenous therapy is usually unnecessary.

Complications

(*a*) *Mortality*

There were two deaths out of 441 cases (0.45% mortality). One infant died during the procedure and a second died many months postoperatively of septicaemia unrelated to operation. The operative death occurred in a male, aged 17 days, with a history of vomiting since birth. At the age of 12 days, the vomiting became projectile and one pound in weight was lost in the subsequent four days. On examination he was found to be slightly dehydrated with a lax skin, but there was good tissue turgour. A typical pyloric "tumour" was palpated and he received 190 c.c. of ¾:¾ solution during the night preceding the operation. Atropine gr. 1/500 was given half an hour preoperatively and at operation he received succinylcholine 8 mg. for intubation. He then received nitrous oxide – oxygen, oxygen, and 1 per cent halothane, but relaxation was inadequate, so ether was added. During incision of the pyloric tumour, cardiac arrest occurred. Despite thoracotomy, direct cardiac massage, the administration of intracardiac adrenaline and calcium, the heart failed to restart. Many factors may have contributed to produce this complication, but the exact mechanism remains unknown.

(*b*) *Morbidity*

(i) *Anaesthetic.* There were five cases of aspiration of vomitus requiring bronchoscopy, and two cases of difficult intubation were noted. Four cases of apnoea and spasm were recorded on extubation.

(ii) *Surgical.* One child required reoperation and three children were found to have no pyloric tumour. In three cases the infant's duodenal mucosa was inadvertently opened and sutured.

Summary

This paper gives a brief review of congenital hypertrophic pyloric stenosis with a report on 441 consecutive cases coming to operation at The Hospital for Sick Children during 1957–60. The pathology, pathological physiology, and clinical features and preoperative treatment are outlined. The anaesthetic management of these cases is discussed in detail and our results reported.

Résumé

Nous avons présenté un bref aperçu de la stenose congenital hypertrophique du pylore. Quatre cent quarante-et-un cas consecutifs operés a l'Hospital for Sick Children depuis 1957 jusqu'à 1960 sont rapportés. La pathologie, la physiologie pathologie, les signs cliniques et le traitement pré-operatoire ont été décrit. Une discussion detailée de ces cas, du point du vue anésthésique, et leurs résultats ont présentés.

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