ANAESTHESIA FOR CONGENITAL HYPERTROPHIC PYLORIC STENOSIS

A Review of 350 Patients

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Congenital hypertrophic pyloric stenosis, with an incidence of 3 per 1000 live births (Editorial, 1984), is the second commonest reason for surgical intervention in the first 6 months of life. Ramstedt's pyloromyotomy is the treatment of choice.

Bennet (1968) and Touloukain and Higgins (1983) have described the complex electrolyte and metabolic disturbances which occur in this condition, and which stem from the loss of hydrogen, chloride, sodium, potassium and water in the vomitus. Initially, the kidney compensates by excreting sodium and potassium bicarbonate, but when hypokalaemia and hyponatraemia supervene an acid urine is produced, aggravating the hypochloraemic hypokalaemic alkalosis. A compensatory respiratory acidosis is usually present. Lactic acidosis secondary to hypovolaemic shock occurs in severe cases. These starving infants are prone to hypoglycaemia.

Although the management of this condition has improved considerably, some published series show a small but significant mortality which ranges from 0.32% to 2% (Bell, 1968; Daly and Conn, 1969; Kiely et al., 1981; Gray, Gear and Stevens, 1984). We report the experience of the anaesthetic management of congenital hypertrophic pyloric stenosis in 350 consecutive patients over an 8-year period in one hospital.

SUMMARY

The anaesthetic management of 350 consecutive patients with congenital hypertrophic pyloric stenosis over an 8-year period is reviewed. The anaesthetic technique is discussed and the complications encountered reviewed. The morbidity rate was 15.9%. The anaesthetic related morbidity rate was 3.7%. One patient in the series died (0.27%) 8 weeks after pyloromyotomy, as a result of an underlying myopathy.

PATIENTS AND METHODS

The names of all patients who underwent pyloromyotomy between January 1, 1977 and December 31, 1984 were taken from the operating theatre record books. All of the case notes were located and included in the study.

Fourteen patients (4%) had been admitted to hospital for other reasons when the symptoms of pyloric stenosis developed. The diseases present in this group of patients are listed in table I. For the purposes of the study they were designated group A. The statistics for the variables "time from admission to surgery" and "days spent in hospital after operation" were calculated both for all patients (350) and with group A excluded (336).

Management

After admission to hospital the infants were resuscitated with appropriate i.v. fluids and electrolyte solutions. A nasogastric tube (size 8 French gauge) was passed and the stomach kept empty by frequent aspiration. Estimations of blood glucose concentration were made 2-4 hourly using reagent strips (Ames Dextrostix).
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### TABLE I. Conditions present in patients who were in hospital when the symptoms of pyloric stenosis first occurred

<table>
<thead>
<tr>
<th>Condition</th>
<th>No. patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Congenital cardiac anomaly</td>
<td>8</td>
</tr>
<tr>
<td>Tracheo-oesophageal fistula</td>
<td>2</td>
</tr>
<tr>
<td>Cystic fibrosis</td>
<td>1</td>
</tr>
<tr>
<td>Congenital myopathy</td>
<td>1</td>
</tr>
<tr>
<td>Seborrhoeic dermatitis</td>
<td>1</td>
</tr>
<tr>
<td>Viral meningitis</td>
<td>1</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>14</strong></td>
</tr>
</tbody>
</table>

Initial i.v. therapy was always with an electrolyte/dextrose solution, usually 2.5% dextrose in saline 0.45 g at 150 ml kg\(^{-1}\) day\(^{-1}\). Additional i.v. fluid with physiological saline (sodium chloride 0.9 g%) was given to replace nasogastric losses. Potassium chloride 2 mmol dl\(^{-1}\) was added to the i.v. fluids once urine flow had been established. Occasionally, an infusion of plasma (10–15 ml kg\(^{-1}\)) was required for patients presenting in hypovolaemic shock.

Frequent assessments, both clinical and biochemical, were mandatory and resuscitative efforts adjusted accordingly. The infant was deemed fit for surgery when fluid and electrolyte balance was restored to normal. Premedication was with atropine 0.02 mg kg\(^{-1}\) i.m., but was omitted if body temperature exceeded 37.5 °C.

The patient was transferred to the operating theatre in a neutral thermal environment. Heat loss was minimized during surgery by keeping the environmental air temperature at 23–25 °C, and by using a thermostatically-controlled heated water mattress.

Before the induction of anaesthesia the nasogastric tube was aspirated (and left in place). Anaesthesia was induced either by inhalation or with a rapid sequence i.v. technique using a “sleep dose” of thiopentone. Suxamethonium 1–2 mg kg\(^{-1}\) was administered, the trachea intubated and anaesthesia maintained with 66% nitrous oxide in oxygen supplemented with a volatile anaesthetic agent. Muscular paralysis was usually achieved with a competitive neuromuscular blocking agent or with intermittent injections of suxamethonium. Ventilation was controlled mechanically using a suitable ventilator, or manually via the Jackson–Rees modification of Ayres’ T-piece (both techniques are used in this teaching hospital). Arterial pressure, the electrocardiogram and rectal temperature were monitored routinely. A precordial stethoscope was used frequently.

At the end of surgery neuromuscular blockade was reversed and the trachea extubated when the infant was fully awake and moving all four limbs vigorously. The patient remained under close observation in the recovery unit for at least 1 h, and during this period blood glucose concentration was estimated using a “Dextrostix”. The patient returned to an infant ward staffed by nurses experienced in the postoperative care of infants.

I.v. fluids were continued with 2.5% Dextrose in saline 0.45 mg% at 4 ml kg\(^{-1}\) h\(^{-1}\) until oral feeding was re-established.

The current practice is to leave the nasogastric tube in situ for 12 h. If, at 24 h, there is no vomiting, a trial feed of dextrose 100 ml, electrolyte and water is given. If this is tolerated then feeds are recommenced, starting with one-quarter strength and gradually building up to full strength (Leahy and Fitzgerald, 1982).

### RESULTS

Of the 350 patients in the series, 294 (84%) were male and 56 (16%) were female. The mean values, standard deviations and ranges for age (weeks) and weight (kg) are given in table II. Three hundred and fifteen (90%) patients were full term (38–42 weeks gestation) at birth and 11 (3.1%) were born at less than 35 weeks gestation.

Pyloric stenosis developed in eight patients who were admitted to hospital because of congenital heart disease (table I). (This hospital is the

### TABLE II. Mean, SD and range for age, weight, time from admission to surgery, duration of anaesthesia, time on i.v. fluids after operation and days spent in hospital after operation

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean</th>
<th>SD</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (weeks)</td>
<td>6.2</td>
<td>3.4</td>
<td>1–30</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>3.76</td>
<td>0.76</td>
<td>1.8–8.4</td>
</tr>
<tr>
<td>Time from admission to surgery (h)</td>
<td>51.3</td>
<td>59.9</td>
<td>2–200</td>
</tr>
<tr>
<td>All patients</td>
<td>46</td>
<td>36.2</td>
<td>2–336</td>
</tr>
<tr>
<td>Group A excluded</td>
<td>44.3</td>
<td>13.6</td>
<td>15–100</td>
</tr>
<tr>
<td>Duration of anaesthesia (51% of patients) (min)</td>
<td>48.2</td>
<td>25.5</td>
<td>6–240</td>
</tr>
<tr>
<td>Time on i.v. fluids post-op. (h)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>All patients</td>
<td>6.4</td>
<td>6.4</td>
<td>2–66</td>
</tr>
<tr>
<td>Group A excluded</td>
<td>5.9</td>
<td>4.8</td>
<td>2–43</td>
</tr>
</tbody>
</table>
National Centre for paediatric heart disease, with 500–600 referrals per year.

The mean interval between admission and surgery was 51.3 h (table II). One hundred and forty-five (41.4%) infants were not dehydrated on admission. However, of these, 86 (59.3%) had been actively treated in other hospitals before their transfer (table III). One hundred and ninety-three (55.1%) patients had chloride concentrations below normal (table III). Seventy-one (20.3%) had serum potassium values below normal (table III). Atropine was given i.m. in 287 (82%) infants as a premedicant, and omitted in 63 (18%), 26 of whom had a rectal temperature of 37–38 °C. The other 37 were brought to theatre unpremedicated and received atropine i.v. at induction.

Anaesthesia was induced i.v. in 210 (60%) patients and by inhalation in 140 (40%). The relative proportions of i.v. and inhalation inductions in each year are displayed in figure 1. In 1977, anaesthesia was induced i.v. in six (14.6%) patients; in 1984, 35 (85.4%) received an i.v. induction. One hundred and ninety-nine (57%) patients were ventilated automatically and 151 (43%) were manually ventilated using the Jackson-Rees modification of Ayre's T-piece.

The duration of anaesthesia was recorded in 179 (51%) patients and was not available in 171 (49%). The mean value was 44.3 min (SD 13.4, range 15–100). The mean time for which i.v. fluids were required after operation was 48.2 h (SD 25.5, range 6–240) (table II).

### TABLE III. Degree of dehydration, and serum chloride and potassium concentrations, on admission. (Of those not dehydrated, 59.5% had been actively treated in another hospital before transfer here)

<table>
<thead>
<tr>
<th>Degree of dehydration</th>
<th>No. patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>None</td>
<td>145 (41.4%)</td>
</tr>
<tr>
<td>Mild</td>
<td>154 (44%)</td>
</tr>
<tr>
<td>Moderate</td>
<td>40 (11.4%)</td>
</tr>
<tr>
<td>Severe</td>
<td>11 (3.2%)</td>
</tr>
<tr>
<td>Serum chloride (mmol litre⁻¹)</td>
<td></td>
</tr>
<tr>
<td>&gt; 95</td>
<td>157 (44.9%)</td>
</tr>
<tr>
<td>90–95</td>
<td>88 (25.1%)</td>
</tr>
<tr>
<td>80–90</td>
<td>68 (19.5%)</td>
</tr>
<tr>
<td>&lt; 80</td>
<td>32 (10.5%)</td>
</tr>
<tr>
<td>Serum potassium (mmol litre⁻¹)</td>
<td></td>
</tr>
<tr>
<td>&gt; 3.5</td>
<td>276 (78.7%)</td>
</tr>
<tr>
<td>3.0–3.5</td>
<td>42 (12%)</td>
</tr>
<tr>
<td>&lt; 3.0</td>
<td>32 (9.3%)</td>
</tr>
</tbody>
</table>

Morbidity and mortality

One patient (0.27%) known to have a rare progressive muscle disorder died 8 weeks after pyloromyotomy. This patient was the only infant in the series to have his operation performed under local infiltration anaesthesia (using 3 ml of 0.25% lignocaine).

The morbidity rate was 15.9%. This was arbitrarily divided into surgical and anaesthetic morbidity. The surgical morbidity rate was 12.2%; this included wound infections, wound dehiscence, persistent vomiting, delayed feeding and urinary tract infections. The anaesthetic

![Fig. 1. Percentage of inhalation (black columns) and i.v. inductions in each year from 1977 to 1984.](image-url)
related morbidity rate was 3.7%. This included all patients in whom morbidity was definitely or possibly related to anaesthesia. These are listed in table IV. All of these patients made a full recovery.

**DISCUSSION**

No patient died as a result of congenital hypertrophic pyloric stenosis or pyloromyotomy in this hospital during an 8-year period in which 350 pyloromyotomies were performed.

This compares favourably with several reports in which a small but definite mortality was recorded (Bell, 1968; Kiely et al., 1981; Gray, Gear and Stevens, 1984). One patient, suffering from myotubular myopathy and requiring mechanical ventilation from birth, died 8 weeks after pyloromyotomy. The operation was performed under local infiltration analgesia and both surgery and anaesthesia were uneventful.

The morbidity rate was 15.9%. All patients made a full recovery. Gray, Gear and Stevens (1984) report a complication rate of 20%; Dubousset (1973) had two cases of gram-negative septicaemia from which both patients subsequently died, in a series of 109 patients.

Four infants developed postoperative complications which warrant further discussion.

**Patient 1.** A 49-day-old full term male infant weighing 3.6 kg presented with pyloric stenosis. He was mildly dehydrated on admission and biochemical analysis revealed a hypochlolemic alkalosis. Twenty-four hours later, following correction of hydration with i.v. fluids, he was deemed fit for surgery. Atropine 0.06 mg i.m. was given as a premedicant. Before induction of anaesthesia the nasogastric tube was aspirated and anaesthesia induced with halothane and nitrous oxide in oxygen. Following the administration of suxamethonium 6.25 mg, the trachea was intubated. The lungs were ventilated manually using the Jackson-Rees modification of Ayre's T-piece, and anaesthesia was maintained with halothane and nitrous oxide in oxygen. Surgery was uneventful and lasted 20 min. The trachea was extubated once the infant was fully awake and moving all limbs. Shortly after extubation of his trachea he was returned to the ward where, 30 min after anaesthesia, he had an apnoeic attack and became cyanosed. He responded to manual ventilation with 100% oxygen via a face mask.

**Patient 2.** A 17-day-old 4.06-kg male infant presented for Ramstedt's pyloromyotomy. After aspiration of the nasogastric tube, anaesthesia was induced with thiopentone 12.5 mg and tracheal intubation was facilitated with suxamethonium 6.25 mg. Cricoid pressure was maintained from the start of the induction until correct placement of the tracheal tube was confirmed by auscultation. The lungs were ventilated mechanically using a

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**TABLE IV. Causes of morbidity related to anaesthesia**

<table>
<thead>
<tr>
<th>Cause</th>
<th>No. patients</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Upper respiratory tract infection</td>
<td>3</td>
<td>Consisted of mild pyrexia, nasal snuffles, and/or pharyngitis. There was no clinical or radiological evidence of lower respiratory tract infection.</td>
</tr>
<tr>
<td>Lower respiratory tract infection</td>
<td>5</td>
<td>Two patients had a preoperative upper respiratory tract infection. One patient had had respiratory distress syndrome requiring controlled ventilation as a neonate. One patient had congenital heart disease and cardiac failure before operation.</td>
</tr>
<tr>
<td>Delayed recovery from anaesthesia</td>
<td>3</td>
<td>See text</td>
</tr>
<tr>
<td>Postoperative cyanotic attack</td>
<td>1</td>
<td>See text</td>
</tr>
<tr>
<td>Postoperative stridor</td>
<td>1</td>
<td>Persisted for 6 h after operation. Treated with humidified oxygen.</td>
</tr>
</tbody>
</table>

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The morbidity related to anaesthesia was 6.7%. This included all patients in whom morbidity was definitely or possibly related to anaesthesia. These are listed in table IV. All of these patients made a full recovery.
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Servo 900C ventilator. Anaesthesia was maintained with 0.5% halothane and nitrous oxide in oxygen. Pancuronium 0.3 mg was administered. Surgery and anaesthesia were uneventful. The duration of anaesthesia was 45 min. Upon cessation of anaesthesia, and after adequate antagonism of neuromuscular blockade, with neostigmine 0.24 mg and atropine 0.12 mg—as shown by an adequate response to peripheral nerve stimulation—the infant developed a jerky, inadequate ventilatory pattern. Blood-gas analysis was normal. He was transferred to the intensive care unit and required ventilatory assistance. Following 7 h of i.v. fluid therapy and intermittent positive pressure ventilation, he was weaned from IPPV and the trachea was extubated. The patient made an uneventful recovery. Blood sugar concentration was normal throughout the intra- and post-operative periods.

Patient 3. A 42-day-old full-term female who weighed 4.7 kg presented with a 3-day history of vomiting and was mildly dehydrated on admission. She was resuscitated for a 24-h period, before undergoing surgery. Atropine 0.08 mg was given i.m. as a premedicant. A rapid sequence i.v. induction was used in this patient with thiopentone 20 mg and suxamethonium 7.5 mg. After intubation of the trachea, the lungs were ventilated mechanically, anaesthesia was maintained with nitrous oxide in oxygen, and neuromuscular blockade was achieved with pancuronium 0.4 mg. This child was given morphine 0.4 mg during the operation.

After antagonism of neuromuscular blockade with neostigmine 0.28 mg and atropine 0.14 mg, the infant remained drowsy and unresponsive. Spontaneous ventilation was inadequate. Nalorphine 0.5 mg and later naloxone 40 μg failed to produce significant improvement in the level of consciousness, or ventilation.

Blood sugar concentration, serum electrolyte concentrations and the chest x-ray were normal. Blood-gas analysis showed a respiratory acidosis (pH 7.23: \(P_{CO_2} 8\) kPa; \(P_{O_2} 37\) kPa; \(HCO_3\) 24.6 mmol litre\(^{-1}\)). She was transferred to the intensive care unit for controlled ventilation. Eighteen hours after operation spontaneous breathing was adequate and her trachea was extubated. She remained drowsy for a further 24 h. There were no sequelae.

Patient 4. A 3.7-kg male infant presented for Ramstedt's pyloromyotomy. Again, a rapid sequence induction was used with thiopentone 20 mg and suxamethonium 5 mg. Anaesthesia was maintained with nitrous oxide in oxygen, and pancuronium 0.4 mg was the neuromuscular blocking agent used. The lungs were mechanically ventilated. In this patient fentanyl 12.5 μg was given during the operation. On completion of surgery, residual neuromuscular blockade was antagonized with neostigmine 0.2 mg and atropine 0.1 mg. However, the infant failed to resume spontaneous breathing and required ventilatory assistance for 60 min. Subsequent recovery was uneventful.

Three of the patients in this series were in extremis on admission as a result of severe dehydration. They had marked hypochloraemic alkalosis with serum chloride concentrations ranging from 44 to 67 mmol litre\(^{-1}\), and a serum potassium concentration of less than 2.5 mmol litre\(^{-1}\). Admission to the intensive care unit and meticulous correction of metabolic derangement, which took 3–7 days, resulted in uneventful surgery and anaesthesia. Two of these three patients were discharged home within 12 days of surgery. The third developed a septicaemia on the 10th day after operation, but was eventually discharged home well after 5 weeks. It is essential that any underlying metabolic derangement and dehydration are completely corrected before surgery is contemplated. As pointed out by Steven and Allen (1973), Ramstedt's pyloromyotomy is an elective operation. Emergency surgery for this condition is never necessary, nor is it desirable.

Anaesthetic technique

In 1973, Steven and Allen recommended inhalation induction as the method of choice for pyloromyotomy. The widespread use of Sellick's manoeuvre (Sellick, 1961) associated with a rapid sequence i.v. induction in adults, has spread to paediatric anaesthesia and has been recommended as the technique of choice by Dierdorf and Krishna (1981), and Battersby and his colleagues (1984). As can be seen from figure 1, anaesthesia is induced in the vast majority of patients in this unit by this method. In our series both techniques proved safe and no case of aspiration occurred. The safety of induction depends upon ensuring, as far as possible, that the stomach is emptied, by prior aspiration of the nasogastric tube.

The nasogastric tube was left in place. It facilitated the surgeon's testing the integrity of the bowel wall after pyloromyotomy. A small volume of air was injected down the nasogastric tube and the surgeon manipulated it into the
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duodenum and occluded the bowel lumen a short
distance proximal and distal to the incision.
Mucosal perforation would have been indicated
by leakage of air.

Anaesthesia was maintained with 60% nitrous
oxide in oxygen, and supplemented in 171 patients
(48%) with a low concentration of a volatile agent.
Halothane was used in the majority of patients
although, more recently, we have used enfurane
or enflurane. Neuromuscular blockade was main-
tained in the majority of patients with pancu-
ronium, although in 1984 we used atracurium
satisfactorily in nine patients.

The use of opioids in infants is a controversial
issue. In our series 11 patients received an opioid
during the surgical procedure. Of the five patients
who received morphine, three weighed less than
5 kg, and delayed recovery occurred in one of
these. The six patients who received fentanyl all
weighed less than 5 kg and one case of delayed
recovery occurred in this group. Opioid drugs
should be used with caution.

It is important to emphasize the need for close
observation in the postoperative period. Poten-
tially fatal complications can occur during this
time. Hypoglycaemia is always a potential hazard
(Pagliara et al., 1973) and was the cause of fatality
in Kiely’s series (Kiely et al., 1981).

The postoperative feeding regimen, advocated
by Leahy and Fitzgerald in 1982, has resulted in
a smoother postoperative course with less vomit-
ing. Previously, feeding was attempted 4 h after
operation, but this was frequently associated with
vomiting.

The morbidity was evenly distributed over the
first 7 years. However, there was no morbidity
recorded in 1984. There was no significant change
in surgical or anaesthetic management in 1984 and
we attributed this morbidity rate to random-
ization. Both surgical and anaesthetic morbidity
have occurred since completion of the study in
1985.

In summary, we report on 350 consecutive
pyloromyotomies in which there were no deaths
either directly or indirectly attributable to the
procedure. There was, however, a small but
significant morbidity.

The importance of thorough preoperative
preparation, careful anaesthesia and close super-
vision in the postoperative period is stressed.

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