Vomiting after pyloromyotomy for infantile hypertrophic pyloric stenosis

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SUMMARY An analysis of the factors which may predispose towards postoperative vomiting after pyloromyotomy for hypertrophic pyloric stenosis was carried out in 72 infants at this hospital. 26 (36%) infants experienced moderate to severe postoperative vomiting of sufficient intensity to cause the postoperative feeding regimen to be modified or interrupted. Only two parameters were found to be of statistical significance. These were the state of the oesophageal mucous membrane on endoscopical examination and the presence of haematemesis in the preoperative period. No evidence for a gastric mucosal lesion could be found. An advanced oesophageal mucosal lesion was found in 30% of patients, and this was the source of the haemorrhage in all 11 in whom haematemesis was noted preoperatively. The stay in hospital was prolonged (8 days) in those infants with troublesome vomiting postoperatively compared with those with lesser problems (3 days).

Vomiting after surgery for infantile hypertrophic pyloric stenosis is common, yet only vague statements about the problem can be found (Benson, 1969; Swenson, 1969; Cook and Rickham, 1978). In well documented analyses the incidence ranges from 65 to 90% (Table 1).

The precise cause of vomiting has been the subject of numerous reviews, and some authors have even attempted to identify those infants particularly at risk of developing troublesome postoperative vomiting. Haberer (1932) stated that most of the infants with postoperative vomiting 'showed blood in their vomitus preoperatively'. He presupposed a gastric mucosal lesion which required time to heal. Guiseffi (1948) noted that infants over 6 weeks tended to have a 'stormy' postoperative course. Terezis et al. (1959) identified a 3 to 8 times greater incidence of postoperative vomiting among infants having either preoperative alkalosis or a loss of >1·0 kg from their greatest attained preoperative weight. Unlike Guiseffi, they found that vomiting was more likely in infants aged 2 to 3 weeks than in ones who were older. Bell (1968) found no difference in the incidence of postoperative vomiting between infants with slight and those with gross metabolic disturbances. Scharli and Leditschke (1968) showed that gastric peristaltic activity had stopped 4–6 hours postoperatively, and that activity was depressed for an additional 16–24 hours. They concluded that early and frequent oral feeding during the first postoperative day was illogical and could lead to postoperative vomiting. Scharli et al. (1969) further showed that the incidence was unrelated to the duration of symptoms, to the preoperative serum bicarbonate level, or to special postoperative feeding routines. Benson (1971) suggested that postoperative vomiting could be made less likely by adequate preoperative fluid and electrolyte correction, by wide separation of all circular muscle fibres at surgery, and by avoiding oral fluids until 8 hours postoperatively.

Incomplete pyloromyotomy as a cause of persistent vomiting is rare (Hayes and Goldenberg, 1957; Benson, 1971) and it is generally recommended that secondary pyloromyotomies should be postponed for 2 weeks after the first operation. Contrast radiography is of little help, as the radiological appearance of pyloric stenosis may persist post-

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<th>Author</th>
<th>Year</th>
<th>No. of cases</th>
<th>Incidence of post-operative vomiting (%)</th>
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<tbody>
<tr>
<td>Hayes and Goldenberg</td>
<td>1957</td>
<td>122</td>
<td>80</td>
</tr>
<tr>
<td>Pollock et al.</td>
<td>1957</td>
<td>1422</td>
<td>90</td>
</tr>
<tr>
<td>Froser</td>
<td>1965</td>
<td>281</td>
<td>86</td>
</tr>
<tr>
<td>Bell</td>
<td>1968</td>
<td>305</td>
<td>69</td>
</tr>
<tr>
<td>Scharli et al.</td>
<td>1969</td>
<td>300</td>
<td>65</td>
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operatively for several months even if the stenosis has been clinically relieved (Cremin et al., 1973).

Excessive gastric mucus, secondary to diffuse gastritis, has been implicated by many authors (Rickham, 1969; Swenson, 1969; Dodge, 1975; Nixon, 1976; Cook and Rickham, 1978). They recommend gentle gastric washouts with warm normal saline preoperatively in order to evacuate the stomach of excess mucus.

This study was a prospective analysis of a consecutive series of 72 infants with pyloric stenosis at this hospital during a 3-year period 1975–78. The diagnosis was confirmed at operation in all cases.

The patients

The infants were fully assessed preoperatively, and any disturbance of fluid and electrolyte homeostasis or acid-base balance was corrected preoperatively. The stomach was evacuated of milk curds by gentle saline washouts in each. The pyloromyotomy was performed under general endotracheal anaesthesia and under the same anaesthetic an endoscopical examination of the oesophagus and stomach was carried out using the Olympus paediatric oesophagofiberscope, model EP type PA (maximum external diameter 7.2 mm). All the endoscopical examinations were performed by the same observer. A gastric mucosal punch biopsy was taken in 35 patients. No complications from the endoscopical examinations were encountered.

Classification of postoperative vomiting. Four grades of this symptom were recognised.

Grade I (n=10, 14% patients).
No vomiting at all postoperatively.

Grade II. Slight (n=36, 50% patients).
An odd vomit or occasional posset in the first 24–48 hours postoperatively.

Grade III. Moderate (n=14, 19% patients).
More frequent vomiting, sufficient to modify the postoperative feeding regimen.

Grade IV. Severe (n=12, 17% patients).
Excessive vomiting causing prolonged delay in establishing oral nutrition and often necessitating parenteral fluid administration.

The incidence for each grade of postoperative vomiting was assessed according to the following preoperative parameters. (1) Age of the infant on admission. (2) Sex. (3) Duration of symptoms. (4) Birthweight. (5) Degree of weight loss or failure to thrive. (6) Presence of haematemesis. (7) Feeding practice. (8) Size of the pyloric ‘tumour’. (9) Appearance of the oesophageal mucosa on endoscopy. (10) Appearance of the gastric mucosa on endoscopy. (11) Presence of alkalosis. (12) Presence of dehydration.

For the purposes of statistical analysis the two grades with slight problems or none were combined (group 1) as were those with moderate and severe postoperative vomiting (group 2). This appeared to be a logical approach as the practical significance of the vomiting was determined by whether or not the postoperative feeding regimen had to be interrupted.

Statistical analyses used consisted of the chi-squared and Student’s t tests if appropriate.

Results

Age on admission. The mean age on admission was 5.7 weeks, range 2 to 16. There was no statistically significant difference in the age on admission between the two groups (χ² 0.9407, P>0.05). It is noteworthy that all 4 infants aged <3 weeks experienced pronounced postoperative feeding problems.

Sex. There were 55 boys and 17 girls in the series (ratio 3 : 1). This ratio was maintained in the various grades of postoperative vomiting.

Duration of symptoms. The mean duration of symptoms before admission was 15 days. There was no correlation between the duration of symptoms and the incidence of postoperative vomiting. (t 1.0309, P>0.05)

Birthweight. The mean birthweight for the 72 patients was 3200 g. There was no significant difference in birthweight between the two groups (t = 1.5480, P>0.05) and this applied equally to those infants weighing <2500 g at birth.

Degree of weight loss. The degree of failure to thrive was determined by measuring the difference between the expected and the actual weight on admission using the standard Tanner and Whitehouse growth chart. Seven infants displayed a positive growth pattern, 2 remained on their original centiles, while the remaining 63 infants failed to thrive. The average decrease in growth for the last group was assessed at −1.03 SDs from the mean. The degree to which infants in the two groups failed to thrive was not of statistical significance (t = 0.437, P>0.05).

Haematemesis. Eleven (15.3%) patients gave a positive history for blood in the vomitus preoperatively, either in the form of frank blood or 'coffee grounds'. Two had slight postoperative vomiting, while the remaining 9 experienced either moderate (7) or severe (2) vomiting. The difference was statistically significant (χ² 9.5349, P<0.01).
Feeding practice. 24 infants were entirely breast fed while 40 were fed on a variety of proprietary milk formulae. Insufficient data were available in 8 patients. No difference could be determined in the incidence of postoperative vomiting according to feeding practice.

Size of the pyloric ‘tumour’. The size of the pyloric tumour was subjectively estimated at operation to be small, moderate, or large (R. B. Zachary, 1975, personal communication). There was no correlation between the size of the ‘tumour’ and the frequency of postoperative vomiting ($\chi^2 2.9205, P>0.05$).

State of the oesophageal mucous membrane. Endoscopically four types of oesophageal mucosa were recognised: Grade 0—normal appearance (32 patients); I—slight erythema of the distal oesophagus only (18 patients); II—erythematous and friable mucosa (10 patients); III—presence of superficial ulceration of the mucous membrane (12 patients).

Analysis of the incidence of postoperative vomiting according to the above criteria is shown in Table 2. Although there was no statistical difference between the individual subgroups, infants with a normal or only slightly erythematous distal oesophagus experienced significantly less postoperative vomiting than those infants with a more advanced oesophageal mucosal lesion ($\chi^2 18.1983, P<0.001$).

State of the gastric mucosa. No infant showed any evidence of a diffuse gastric mucosal lesion either on endoscopy or on histopathological examination of the mucosal biopsy (Batcup and Spitz, 1979). 19 (26%) infants displayed occasional punctate haemorrhages on endoscopy. There was no correlation between presence of punctate haemorrhages and the incidence of postoperative vomiting.

Alkalosis. 24 (33%) infants were biochemically alkalotic, as defined by a serum bicarbonate level preoperatively $>28 \mu mol/l$. No correlation could be found with the incidence of postoperative vomiting ($\chi^2 2.1747, P>0.05$).

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<tr>
<th>Oesophageal mucosal inflammation</th>
<th>Degree of postoperative vomiting</th>
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<tr>
<td>None</td>
<td>Slight</td>
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<tr>
<td>Grade 0</td>
<td>7</td>
</tr>
<tr>
<td>I</td>
<td>1</td>
</tr>
<tr>
<td>II</td>
<td>1</td>
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<tr>
<td>III</td>
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Dehydration. There was no relation between the incidence and severity of clinical dehydration (28 patients), or azotaemia (11 patients) and postoperative vomiting ($\chi^2 0.7533, P>0.05$).

Discussion

Of the various parameters analysed only two of statistical significance emerged—namely, the appearance of the oesophageal mucosa on endoscopical examination and the history of haematemesis preoperatively. It is noteworthy that of the 22 infants exhibiting a friable or ulcerated oesophageal mucous membrane on endoscopy, 11 (50%) gave a positive history for blood in the vomitus preoperatively. In view of the absence of evidence for gastritis endoscopically and on pathological examination, it can be concluded that the oesophageal, rather than the gastric mucosa, was the source of the haemorrhage in the infants in this series.

This finding is in direct contrast with the views of most authors on the subject (Benson and Lloyd, 1964; Swenson, 1969; Dodge, 1975; Nixon, 1976; Cook and Rickham, 1978). Forshall (1955) and Roviralta (1967) were the first to recognise the association between lower oesophageal sphincter incompetence and hypertrophic pyloric stenosis. Pellerin et al. (1974), in a retrospective study of 627 cases of hypertrophic pyloric stenosis with adequate radiological study of the hiatal region, demonstrated gastro-oesophageal reflux in only 13% of cases. Although it is generally recognised that oesophagitis commonly occurs secondarily to lower oesophageal sphincter incompetence, the converse may equally apply. Lower oesophageal irritation may adversely affect the competence of the gastro-oesophageal mechanism (Eastwood et al., 1975). Manometric studies of the oesophageal peristaltic activity were not undertaken in this series.

The finding of advanced oesophageal mucosal inflammation in 22 (30%) of the 72 infants, indicates that gastro-oesophageal reflux is more common than had been previously recognised. It has furthermore been shown to be a significant factor in the pathogenesis of postoperative vomiting in infants with hypertrophic pyloric stenosis. The effect of posture on the incidence of postoperative vomiting is currently being studied.

It is not my intention to advocate endoscopy on all infants with pyloric stenosis. The finding of oesophagitis in almost one-third of the infants was unexpected, but it has proved to be relevant with regard to the postoperative management of these infants. A positive history of haematemesis, which has been shown to be a consequence of the
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Possibility of an important indicator.

References


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