Pyloric and oesophageal dysfunction in the cat

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ABSTRACT

The paper describes the symptoms, diagnosis and treatment of pyloric stenosis in thirteen cats, associated in some animals, with megalo-oesophagus.

INTRODUCTION

Though vomiting in otherwise normal cats is a frequent presenting symptom, it seldom been attributed to pyloric dysfunction. Joshua (1960) reported that mals with symptoms of projectile vomiting suggestive of pyloric stenosis did not respond permanently to specific surgical treatment. Twaddle (1970) however, dered three cases of vomiting associated with prolonged gastric emptying time, which the symptoms were completely controlled by pyloroplasty over a follow-up period of 1–3 years. Two were Siamese females, one of which subsequently produced a litter in which at least two of the seven kittens were affected with the disorder (Twaddle, 1971). The symptoms were poor growth, respiratory distress and frequent daily vomiting which was completely abolished by pyloroplasty with no relapse over an 18 month period.

Megalo-oesophagus ('achalasia') in the cat unassociated with vascular ring structure, is equally uncommon. Cawley & Gendreau (1969) reported the condition in an adult animal presented with severe dyspnoea but with an earlier history of frequent regurgitation. No specific evidence of neuronal degeneration or primary myogenic atrophy could be detected at autopsy. More recently, congenital oesophageal 'achalasia' in four cats of common ancestry was recorded by Clifford L. (1971).

This paper describes a series of fifteen animals suffering from pyloric dysfunction, galo-oesophagus or a combination of both disorders.
CASE HISTORIES

The clinical data relating to these cases are summarized in Table 1.

Of the thirteen cats in which vomiting was the principal symptom, twelve were Siamese. There were six males and seven females. The duration of the symptom could not always be ascertained but in most cases, vomiting began soon after weaning and persisted, sometimes intermittently, thereafter. The pattern of vomiting was variable. In six cats, it occurred at a fairly constant interval after feeding, varying in different animals from 30 minutes to 8 hours. In other cases, the time of vomiting did not appear to be related to the last meal. In one exceptional case, Case 12, the vomiting initially occurred within 30 minutes of feeding but later took place only every 3rd or 4th day and then comprised debris of food ingested 2 days previously. In five animals, the vomiting was often or always strongly projectile in nature. In some cats, it occurred daily without remission but in others, was confined to bouts of several days' duration at irregular intervals.

The vomitus was never re-ingested. In two animals, vomiting was preceded by salivation and retching. A constant sequel in most cats was immediate hunger. Secondary symptoms were very variable. None of the cases was fat but seven were in reasonably good bodily condition and these animals were active and normal in behaviour except for the vomiting. The remaining six were poorly grown and three to the point of severe emaciation and clinical dehydration. One of this latter group, Case 5, was also dyspnoeic and showed abdominal distension and gastric tympany; a previous rectal prolapse had been reduced.

In the cats whose growth was not adversely affected, the appetite was normal but the emaciated animals ate capriciously. The condition was always afebrile.

DIAGNOSIS

The diagnosis was based on:

(a) the symptoms,
(b) the radiological findings,
(c) the response to specific surgical treatment.

(a) Symptomatology

If vascular ring stricture of the oesophagus can be eliminated, a history of continual vomiting, often projectile in nature and beginning at weaning or soon afterwards, suggests some form of pyloric dysfunction and merits radiological investigation or even specific surgical treatment as test therapy.

(b) Radiology

The results of radiological investigation in 13 cases are summarized in Table 2.
<table>
<thead>
<tr>
<th>Case No.</th>
<th>Oesophagus</th>
<th>Gastric Size</th>
<th>Gastric Clearance</th>
<th>Follow-up</th>
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<tr>
<td></td>
<td>Contrast retention</td>
<td>Degree of dilatation</td>
<td>Degree of dilatation</td>
<td>Small volume</td>
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<td>√</td>
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<td>√</td>
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</tr>
</tbody>
</table>

* = Dilated organ identified on plain films. † = Residual food material in pyloric antrum on plain films. – = Not determined.
The diagnosis of pyloric dysfunction was based initially on radiological evidence from routine barium meal studies on cats presented with a history of persistent or intermittent vomiting. A proportion of such cases showed moderate gastric dilation, delayed passage of contrast medium and prolonged retention of ingesta within the stomach. Any combination of these signs is indicative of pyloric dysfunction (Rhodes & Brodey, 1965; Douglas, 1968; Gibbs & Pearson, 1973).

**Fig. 1.** (Case 5). Lateral and ventro-dorsal plain radiographs showing gastric and oesophageal dilation. In the lateral view the trachea is displaced ventrally by the air-filled oesophagus.
FIG. 2. (Case 8). Lateral and ventro-dorsal plain radiographs. Fragments of radiopaque food material can be seen within the gastric lumen.
FIG. 3. (Case 4). Lateral and ventro-dorsal radiographs taken 5 minutes after administration of barium. The stomach and oesophagus are dilated. Mucosal corrugation is a normal feature of the feline oesophagus.
Fig. 4. (Case 10). Lateral and ventro-dorsal 10 minute radiographs. Contrast medium is retained within the oesophagus which is not significantly dilated. The stomach is large and in the region of the body and fundus its outline is smooth and stretched in the ventro-dorsal view; a small quantity of barium has already entered the duodenum.
FIG. 5. (Case 3). 30 minute radiographs. The small bowel contains only small flecks of barium. Although the stomach is not grossly enlarged on the ventro-dorsal view (B), the lateral radiograph shows marked antral dilatation.
An additional finding in seven of the eleven animals radiographed was retention of contrast medium within the oesophagus, which, in some cases, was dilated, giving a radiological appearance identical to megalo-oesophagus in the dog.

The degree of severity of radiological signs is variable. In advanced cases (Fig. 1), plain radiographs may show evidence of gastric dilation if gas is present. Fragments of radiopaque food material may be visible within the lumen suggesting prolonged retention of gastric contents (Fig. 2). A dilated, gas-filled oesophagus may also be identified at this stage (Fig. 1).

However, should the stomach contain only fluid, contrast medium is required to demonstrate its size and location (Fig. 3a). A consistent finding in all cases except one was increase in gastric volume. The fundus and body are usually involved and tend to become elongated cranio-caudally but remain to the left of the midline. Stretching of the mucosal folds gives a smooth appearance to the gastric margin. On the right recumbent lateral radiographs the pyloric antrum may be listended (Fig. 5).

As in the dog, the time of onset of gastric emptying in the cat is variable. Cases of pyloric dysfunction show no evidence of significant delay, and contrast medium was usually entered the duodenum between 5 and 10 minutes after administration (Fig. 4). However, with the aid of fluoroscopy it has been possible to demonstrate that although peristaltic contractions appear to be normal in rate and intensity, there is marked diminution in the quantity of barium passing through the pyloric canal with each relaxation of the sphincter. This observation would explain poor filling of small intestinal segments seen on later films (Fig. 5) and the fact that contrast medium may be present in the stomach for periods up to 24 hours (Fig. 6).

**Fig. 6.** (Case 10). 24 hour lateral radiograph showing retention of contrast medium in the stomach. The remainder is incorporated within faecal boluses in the large bowel.
The principal radiological abnormality in the two cases of megalo-oeso (Cases 14 and 15) was gross oesophageal dilation with a secondary tympany in the older cat (Fig. 8).

![Diagrams showing follow-up examination 7 months after pyloromyotomy. Lateral and ventro-dorsal radiographs 10 minutes after barium. The stomach is almost empty and segments of small intestine are well filled. The oesophagus still retains contrast material.](Image)
TREATMENT

The thirteen animals tentatively diagnosed as suffering from pyloric dysfunction were all treated by pyloromyotomy. Food and fluids were withheld for 12 hours before surgery.

a) Anaesthesia

Anaesthesia was carried out according to the principles of balanced anaesthesia outlined by Weaver (1964). All the cats were premedicated with 0·3mg atropine sulphate and six cases were also given a sedative, acepromazine maleate, (Acetylpromazine—Crookes Veterinary Ltd) trimetrazine tartrate (Vallergan—May and Baker Ltd) or pethidine hydrochloride.

The premedication was given by intramuscular injection one hour before the induction of anaesthesia, which was carried out by the slow intravenous injection of either a 24% solution of thiopentone sodium (Intravel Sodium—May and Baker Ltd) or the alphaxalone and alphadolone acetate steroid anaesthetic (Saffan (CT 341)—Glaxo Laboratories Ltd). In all cases except one, endotracheal intubation was performed under direct vision, after spraying the vocal cords with lignocaine hydrochloride (Xylocaine Spray—Astra Chemical Ltd). Endotracheal tubes of 1·5 or 5·0 mm (i.d.) were used.

The cats were allowed to breathe spontaneously and anaesthesia was maintained with methoxyflurane (Penthrane—Abbot Laboratories Ltd) or halothane (Fluothane—I.C.I. Ltd) vapourized in an oxygen/nitrous oxide mixture, containing...
66% nitrous oxide, delivered through an Ayre’s T-piece (Mapleson system. The average duration of anaesthesia was 55 minutes and recovery was uneven in all cases.

(b) Surgical technique
Through a mid-line laparotomy, the pylorus was exposed and Ramstedt’s pyloromyotomy was performed through a 2.5 cm incision extending from the pyloroduodenal junction. The gastric mucosa was not incised. Haemorrhage was minimal and no sutures or ligatures were placed.

In all cases, the pylorus was normal in calibre and showed no evidence of muscular hypertrophy. In one cat, a gastric hairball was removed through separate gastrotomy. There were no post-operative complications and food was offered on the second day.

SUBSEQUENT COURSE
Case 7 was critically ill before surgery and its death 8 hours later was unexpected.

Case 4 showed an initial improvement but relapsed and was destroyed 2 months later. Case 11 showed no improvement whatsoever and was subjected, 2 weeks later, to pyloroplasty, again without effect, and was then destroyed. None of the bodies was made available for autopsy. Radiologically, Cases 7 and 4 had shown oesophageal retention and gross dilation before surgery, and the persistence of symptoms was attributed to megalo-oesophagus. It seems likely in retrospect that Case 11 also had oesophageal dysfunction which was not detected.

The remaining ten animals all showed a rapid and remarkable improvement in bodily condition and hair growth. In four (and possibly five) animals, the vomiting stopped completely at once. Four more cats improved dramatically and vomit only occasionally if overfed.

In Case 5, with severe pre-operative megalo-oesophagus and gastric tympani, the improvement was less immediate but within 4 weeks, growth and body weight were noticeably increased and the vomiting was less frequent and no projectile. The appetite, previously poor, was also better. The dyspngeo improved over the next 3 months, and over a 10 month period, the vomiting has stopped and the cat now merely regurgitates fluid occasionally at feeding.

A limited number of follow-up radiological studies have shown that the dimensions of the stomach and rate of gastric emptying return to normal post-operative although evidence of retention of contrast medium within the oesophagus remains (Fig. 7).

DISCUSSION
The response to specific surgical treatment suggests that the vomiting in ten
ese cases was due to pyloric dysfunction developing, as in dogs and in Boxers particularly, at the time of weaning. In the young dog, the condition may properly be described as a congenital hypertrophy of the pyloric musculature which is often ossily thickened. In these cats however, no macroscopic evidence of hypertrophy as detected and some form of functional pylorospasm must be assumed.

The relationship between the pyloric and oesophageal conditions is speculative. Cases 14 and 15 indicate that megalo-oesophagus alone may present with symptoms of dyspnoea rather than regurgitation, and not only in young animals. Moreover, in the remaining cases with radiological evidence of megalo-oesophagus, vomiting was delayed after feeding for longer than would normally occur with an disorder in the dog. The pathological significance of megalo-oesophagus associated with pyloric dysfunction is therefore not clear but the regression of symptoms following pyloromyotomy in most of these animals indicates that the oesophageal disorder, in a moderate degree, contributes but little to the clinical signs and that cats suffering from both conditions should first be treated by pyloric section. On the other hand, the results suggest that gross oesophageal dilations in Cases 4, 5 & 7, adversely affects the prognosis and may account for the persistence of symptoms after pyloromyotomy. The co-existence of megalo-oesophagus and pylorospasm also occurs in the dog (Pearson, unpublished data) and the resultant symptoms may be completely abolished by pyloromyotomy.

The demonstration of functional disturbance at two sites in the upper alimentary tract suggests that the aetiology of the conditions is more likely to be related to autonomic nervous dysfunction than to specific organic lesions. Such a relationship as been demonstrated experimentally in the dog by Okamoto et al. (1967) who produced a non-organic stenosis of the distal colon, pylorus and lower oesophagus by selective destruction of the myenteric plexus, but the pylorus failed to show the muscular tumor which is characteristic of hypertrophic pyloric stenosis in infants and many puppies. These authors cast doubt on the hypothesis that the muscular tumor is a work-hypertrophy resulting from a pre-existing pylorospasm caused by degenerative changes in the myenteric plexus, and conclude that loss or absence of the myenteric plexus of the gut, congenital or acquired, produces a non-organic tenosis caused by a tonic contraction of the denervated smooth muscle fibres. It may therefore be significant that none of the cats in this series showed evidence of gross muscular hypertrophy of the pylorus, a finding also reported by Twaddle (1970; 1971). It is interesting to speculate on the possible effects of cardioplasty in cases with severe megalo-oesophagus.

As a diagnostic procedure, radiological examination may provide corroborative evidence to support a clinical diagnosis based on the symptoms but negative radiological findings do not preclude such a diagnosis. The ultimate proof of diagnosis is response to treatment and immediate pyloric section without prior radiological investigation is justifiable in many cases but co-existent megalo-oesophagus may then go undetected. Pyloromyotomy was performed in preference to pyloroplasty because it is less likely to lead to post-operative fibrotic stenosis.
The exceptionally high proportion of Siamese cats in this series suggests that pylorospasm (and possibly also megalo-oesophagus) may be inherited in this breed. Fig. 9 illustrates the familial relationship of eight of these animals. Case 10 did not appear to be related to these cats and four pedigrees were not available for scrutiny. Without a comparable analysis of the pedigrees of a random sample of normal Siamese cats, the significance of this apparently in-bred relationship is questionable.

The probability of genetic transmission however, should be seriously considered, particularly as Twaddle (1971) recorded a dam/daughters incidence of pyloroplasty in at least three Siamese cats, and in this series also, Case 8 was the sister of Case 13, and siblings of Case 2 were reported to be showing similar symptoms at the same age. Treated animals should obviously not be used for breeding purposes.

ACKNOWLEDGMENTS

The authors are indebted to the veterinary surgeons who referred the cases for...
ther investigation and treatment. The radiographs were taken by Mrs K. E. Angstaff, and photographed by Messrs M. H. C. Parsons and J. Conibear.

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