Clinical features and management

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Cats suffering from this disease were first recognized in late 1981. During the early
months of 1982 the number of cases increased dramatically, and reached a peak in
July, when we saw as many cases as in the previous 6 months put together. There
may have been a drop in numbers during the winter of 1982 but a large number of
cats were affected during 1983. Cases are continuing to occur but I have no accurate
figures on incidence. However, a number of interested colleagues in practice in
various parts of the United Kingdom have agreed to log their cases during the next
year and this should give us a better picture of incidence totals, plus any indications
of regional and seasonal variations.

The pattern of the disease does not appear to have altered since it first appeared.
Usually it occurs sporadically, with one cat in a household affected. However, there
have been a number of instances in which several kittens in a litter have been
affected, and pairs of related or unrelated cats in the same house have become ill at
the same time. In other cases individual cats in adjacent houses have become ill at
the same time, and instances have occurred where another cat in a previously
affected household has developed the disease up to 12 months later. Nevertheless,
these occurrences have tended to be the exception rather than the rule.

Country cats as well as town cats have been affected and the disease has occurred
in animals kept permanently indoors as well as in those allowed total freedom.

Domestic short-haired cats have outnumbered pure-bred cats affected by about
4:1, and males to females by 3:2. Seventy five per cent of affected cats have been
less than 3 years old at the onset of the disease with an overall age range of 2 months
to 11 years. In most cases the owner reports that the disease has developed quite
suddenly. Not infrequently many of the major signs have appeared in less than 12
hours. In other cases (about 30 per cent) the onset is more prolonged, up to one week,
with the cat becoming vaguely ill prior to the onset of recognizable signs. The
presenting signs vary, but generally consist of one or more of dullness, anorexia,
‘vomiting’, and ocular changes become apparent early on. In some cases, the change
from healthy normality to severely ill has occurred so quickly and dramatically that
a road accident has been suspected.
Depression and severe weight loss are early and ongoing features in most cases. Clinical findings have been recorded and monitored in over 50 cases that I have seen and Table 1 summarizes the findings in the first 40 of these cases.

**TABLE 1. Major clinical features in 40 cases of feline dysautonomia**

| Feature                                | Number investigated | Number affected | (%) affected |
|----------------------------------------|---------------------|----------------|--------------|
| Constipation                           | 39                  | 37             | 95           |
| Dry rhinarium                          | 38                  | 36             | 95           |
| Reduced tear production                | 27                  | 25             | 93           |
| Mega-oesophagus                        | 37                  | 34             | 92           |
| Dilated pupils                         | 40                  | 36             | 90           |
| Regurgitation/vomiting                 | 38                  | 31             | 82           |
| Prolapsed membrane                     | 35                  | 25             | 71           |
| Photomotor reflex reduced/absent       | 30                  | 21             | 70           |
| Dry oral mucosa                        | 39                  | 27             | 69           |
| Bradycardia ( < 120/min)               | 37                  | 22             | 59           |
| Areflexic anus                         | 24                  | 7              | 29           |
| Faecal incontinence                    | 35                  | 7              | 20           |
| Urinary incontinence                   | 39                  | 7              | 18           |
| Paresis/collapse                       | 28                  | 5              | 18           |
| Proprioceptive deficits                | 28                  | 4              | 14           |

The most consistent findings have been constipation (95 per cent) and inability to defaecate. Prior to this, some cats have reportedly had transient diarrhoea. A dry, crusted nose has also been a regular feature (95 per cent) and in the early stages there may be some snuffling, though actual sneezing has been rare. In later cases, secondary infection may lead to discharges reminiscent of viral respiratory disease. The appearance of abject misery and nasal discharges are reminders of the clinical similarities to equine grass sickness, although we have not seen evidence of abdominal pain in the cat.

Regurgitation and vomiting of solid and liquid food is often an early sign (82 per cent). This was recognized by Key and Gaskell (1982) as being associated with oesophageal dilation (seen in 92 per cent) which is readily confirmed radiographically with plain film lateral thoracic views or following administration of 5–10 ml of a barium suspension. One kitten had a particularly large dilation which occupied a huge volume of the thorax and compressed the heart. When the kitten died, a large amount of liquid food and milk, forcibly administered by the owner, ran from the mouth and nostrils. A litter-mate was also examined and found to be similarly affected, although it was more mildly ill and made a good recovery. Further radiography four months later revealed that, although it was apparently well, there was still a degree of oesophageal dilation. There was also moderate cardiomegaly and the cat died in congestive cardiac failure at 13 months. The oesophageal dilation
may run the length of the lower cervical and thoracic oesophagus or may be more limited. In a number of cases it appeared as two pouches on either side of the base of the heart.

Ocular changes have featured very commonly, the most frequent being reduced lachrymation (93 per cent). Schirmer tear tests have shown that in the normal cat the tear response is 12–17 mm/min. In many cats with dysautonomia, tear production has been less than 5 mm/min, and often zero. Uni- or bilaterally dilated pupils have also been a fairly consistent feature (90 per cent), although their presence may be masked by prolapse of the membranae nictitans (71 per cent) and half-closed eyelids. Many cats will seek dim light in the early stages of the illness. Photomotor responses are usually markedly reduced or absent (70 per cent). There is a response to miotic drugs applied topically.

The oral mucous membranes are frequently dry (69 per cent) during the first week of the illness and in some cats this has been prolonged. Secondary infection has caused diphtheresis in several cases in spite of encouragement to salivation with the use of parasympathomimetic drugs.

Other signs have been recorded less frequently and included: bradycardia (59 per cent), with heart rates below 100/min in some cases; faecal and urinary incontinence (20 per cent and 18 per cent respectively) and locomotor problems (18 per cent). In some otherwise recovered cats, faecal incontinence—or inability to remain on the litter tray—has developed as a later problem, and has necessitated euthanasia in four cases.

Detailed neurological examinations indicated mild deficits in hind limb proprioception and placing reflexes (four cases). Cranial nerve examination, pedal and patellar reflexes and conscious perception of pain in the extremities were normal in all cases. Electrophysiological examinations were carried out in a small number of cases and revealed occasional evidence of denervation in the sacrococcygeus and external anal sphincter muscles.

Laboratory tests have proved unhelpful. Routine haematology, blood and urine biochemistry results have either been normal or merely a reflection of debilitation and dehydration. Large numbers of Heinz bodies have been reported in a high proportion of cases, especially in the early weeks of the disease, but signs of anaemia have not been found. This finding may be associated with faecal impaction, as Heinz bodies are said to be increased in cases of intestinal obstruction. A chance biochemical finding in three out of four cases tested was that of high levels of plasma aldosterone, (750, 700, 616 and 214 pmol/l, cf values for three normal cats of 300, 270 and 220 pmol/l). This is likely to have been due to a secondary response possibly associated with reduced cardiac output but is worthy of further investigation. Cats have been routinely checked for the presence of Feline Leukaemia Virus and were all negative. Mouth swabs taken for respiratory virus culture have been positive in a few cases but in no more than would be expected in a normal random cat sample. Samples checked for feline coronavirus antibodies were also negative.

Therapy has of necessity been supportive. We have not recommended forced
feeding with liquidized food until cats are able to accept food without frequent regurgitation. Pharyngostomy tube feeding was attempted in three cases but has proved most ineffective. During periods of dehydration, which have been intermittent in some cases, subcutaneous or intravenous normal saline has been administered, usually on an alternate day basis. Liquid paraffin (5–10 ml \textit{per os}) has proved useful in aiding defaecation in many of our cases, again usually given on alternate days. Dorbanex\textregistered liquid laxative has also been recommended. Enemas have not been necessary in any of our cases but have been reported by other workers. Pilocarpine (1 per cent) and physostigmine (0.5 per cent) ophthalmic preparations have been effective in promoting salivation and the latter may aid peristalsis. The added advantage of these drugs is that they produce temporary pupillary constriction and possibly permit lowering of the membrane and thus producing an improvement in the cat's appearance if achieving nothing else. Anabolic steroid preparations may be helpful during the convalescent period.

Of the 40 cases reviewed, 11 (27.5 per cent) are still alive after periods of up to two years. One of the recovered cats died 13 months after diagnosis in congestive cardiac failure. Nine other cats survived for up to 17 months until euthanasia was carried out for reasons including antisocial behaviour (e.g. faecal incontinence) and failure to thrive. Nineteen cats died or were destroyed early on in the course of the illness. Of the 11 survivors, eight are normal but have residually dilated pupils or moderate oesophageal dilation, while the other three are not normal leading a rather dull, unattractive life, and appearing prematurely old. Some cats are now said by their owners to be normal, although only one has really regained its former bodyweight; however, it still has widely dilated, fixed pupils.

Finally, one interesting feature: some of the cats which have recovered their pupillary activity have developed D-shaped pupils. The flat side is always medial. I leave the explanation for this phenomenon to the neuro-ophthalmologists.

**REFERENCE**

*K* E. T \& *Gaskell*, C.J. (1982) \textit{Veterinary Record} \textbf{110}, 160. (c)