# Study of the short- and long-term outcomes of 65 horses with peritonitis

I. S. F. Henderson, T. S. Mair, J. A. Keen, D. J. Shaw, B. C. McGorum

The records of 65 horses with peritonitis examined at two UK referral centres over a period of 12 years were reviewed. Peritonitis was defined in terms of the horse's peritoneal fluid containing more than $5 \times 10^9$ nucleated cells/l. Horses that had developed peritonitis after abdominal surgery or a rupture of the gastrointestinal tract were excluded. Of the 65 horses, 56 (86 per cent) survived to be discharged. Follow-up information was obtained from practice records and telephone calls to the owners for 38 of the horses. Of these, 32 (84 per cent) had survived for at least 12 months and were considered to be long-term survivors; the others six were euthanased within 12 months. Thirteen (34 per cent) of the horses discharged had experienced complications that could have been sequelae to peritonitis and eight of the 13 were euthanased. The cause of the peritonitis was identified in 15 cases; survival rates were lowest in horses with peritonitis secondary to urinary tract involvement or intra-abdominal masses. Of the other 50 cases, 47 (94 per cent) survived to discharge, but two were euthanased owing to recurrent colic.

PERITONITIS in horses is a potentially life-threatening disease that can be induced by many infectious and non-infectious conditions (Hanson 1999). Short-term survival rates of between 40 and 70 per cent have been recorded (Dyson 1983, Parry 1983, Mair and others 1990, Hawkins and others 1993, Golland and others 1994, Feige and others 1997, Mair and Hillyer 1997, Van Hoogmoed and others 1999, Matthews and others 2001), but only two of these papers studied the horses' survival after they were discharged. In a study of 11 horses with peritonitis caused by *Actinobacillus equuli* (Golland and others 1994) the horses survived for between 15 months and 11 years. However, peritonitis caused by this particular organism is generally associated with a good prognosis after appropriate treatment; for example, Matthews and others (2001) reported that 51 horses with peritonitis due to *A equuli* all survived to discharge. Long-term survival rates of between six months and five years and the complication rates after treatment of peritonitis due to other causes have been reported for only 12 horses (Mair and others 1990).

The purpose of this study was to review the periods of survival and complication rates of a larger number of horses diagnosed with peritonitis (excluding cases associated with abdominal surgery or rupture of the gastrointestinal tract) at two referral centres in the UK, and to examine the effects of different causes, clinical signs and treatments on them, in order to provide a more accurate prognosis for such cases, and to evaluate the therapeutic options available.

## MATERIAL AND METHODS

The case records of horses with peritonitis examined at two referral centres over a period of 12 years were reviewed. Peritonitis was defined in terms of a horse's peritoneal fluid containing more than $5 \times 10^9$ nucleated cells/l (Mair 2002b). The published reference ranges from leucocyte counts in normal peritoneal fluid vary from less than $1 \times 10^9$/l (Knottenbelt 2006), less than $5 \times 10^9$/l and often less than $2 \times 10^9$/l (Mair 2002b) to less than $10 \times 10^9$/l, and often less than $5 \times 10^9$/l (Bach 1973, Brownlow 1979, Nelson 1979). A value of $5 \times 10^9$/l was chosen as the cut-off value above which a diagnosis of peritonitis was made, to increase the sensitivity of detection of cases of peritonitis, and on the basis that the texts agreed that counts above $5 \times 10^9$/l are uncommon in healthy horses or are abnormal. However, it is acknowledged that by using a lower cut-off point, the specificity of the test may be lower than when a higher cut-off point, for example, less than $10 \times 10^9$/l, as in the study by Dyson (1983), is used. Horses developing peritonitis after a laparotomy or laparoscopy were excluded from the study because peritonitis was not the primary reason for their examination and the underlying surgical lesion would probably have affected the outcome. Horses with peritonitis secondary to a rupture of the gastrointestinal tract were also excluded because their poor prognosis resulted in them being euthanased when their condition was diagnosed. Sixty-five horses fulfilled the criteria; there was no known history of abdominal surgery or recent castration in any of them.

Information retrieved from the case records included the horses' characteristics and medical history, and the results of clinical and abdominal ultrasonographic examinations. Haematological variables, plasma fibrinogen levels, and the results of analyses of peritoneal fluid were recorded when the horses were admitted, while they were hospitalised and at follow-up visits. The methods and duration of treatment and the short- and long-term outcomes were also recorded. Short-term survival was defined as survival to discharge from the hospital. Long-term survival was defined as survival for 12 months or more after being discharged. The incidence of complications that were considered to be possible consequences of the peritonitis, and whether these were associated with survival or non-survival, were recorded.

Long-term follow-up information was obtained from practice records and telephone calls to owners, and was available for 38 of the 65 horses. Follow-up information for 12 months or longer was obtained in 32 cases.

The data were entered into a statistics program (Minitab for Windows v 14). The effects of different clinical variables and methods of treatment on the horses' survival were evaluated by chi-squared analysis for categorical variables. Statistical significance was set at $P<0.05$, and odds ratios (ORs) and 95 per cent confidence intervals (CI) were calculated. Owing to the small number of cases in each aetiological category, it was not possible to compare the effects of different methods of treatment for the cases in each category. The effects of different methods of treatment were therefore compared only in the group of 50 horses for which no diagnosis could be made. The age difference between the idiopathic and non-idiopathic groups was compared using a two-sample $t$ test. Survival analysis, using Kaplan-Meier estimates of survival probability, was applied to evaluate the long-term survival of the horses in this group and in the 15 for which diagnoses were available.

## RESULTS

The horses ranged in age from two days to 28 years, and their mean and median ages were 11·7 years and 10·5 years, respectively. There were 29 mares, 32 geldings and four entire

**I. S. F. Henderson,** BVSc, CertEM(StudMed), MRCVS,
**J. A. Keen,** BVetMed, BSc, CertEM(IntMed), MSc, DipECEIM, MRCVS,
**D. J. Shaw,** BSc, PhD,
**B. C. McGorum,** BSc, BVM&S, PhD, CertEM(IntMed), DipECEIM, MRCVS,
Division of Veterinary Clinical Science, University of Edinburgh, Easter Bush Veterinary Centre, Easter Bush, Roslin, Midlothian EH25 9RG
**T. S. Mair,** BVSc, PhD, DipECEIM, DEIM, DEST, MRCVS,
Bell Equine Veterinary Clinic, Butchers Lane, Mereworth, Maidstone, Kent ME18 5GS

**TABLE 1: Clinical signs, diagnostic findings and laboratory variables of 65 horses and their association with their short- and long-term survival and the incidence of complications; percentages and 95 per cent confidence intervals (CIs) are included for groups with more than five horses**

| Clinical variable | Number of horses | Short-term survival (%) [95% CI] | Number with follow-up available | Long-term survival (%) [95% CI] | Complications in non-survivors (%) [95% CI] | Complications in survivors (%) [95% CI] |
|---|---|---|---|---|---|---|
| Colic | 33 | 29 (87) [71-97] | 18 | 17 (94) [73-99] | 3 (17) [4-41] | 2 (11) [1-35] |
| Depression | 32 | 26 (18) [64-93] | 20 | 17 (85) [49-87] | 3 (15) [3-37] | 0 |
| Tachycardia* | 21 | 17 (81) [58-95] | 11 | 9 (82) [48-98] | 2 (15) [2-51] | 2 (18) [2-51] |
| Pyrexia† | 18 | 14 (78) [52-93] | 12 | 11 (92) [61-99] | 1 (8) [0-38] | 2 (17) [2-48] |
| Decreased gut sounds | 12 | 10 (83) [51-97] | 5 | 5 (100) [55-100] | 0 | 2 (40) [5-85] |
| Tachypnoea‡ | 11 | 9 (82) [48-97] | 6 | 5 (83) [36-99] | 1 (16) [0-64] | 1 (16) [0-64] |
| Diarrhoea | 8 | 6 (75) [35-97] | 3 | 2 | 1 | 1 |
| Respiratory signs§ | 5 | 4 (80) [28-99] | 3 | 2 | 1 | 1 |
| Weight loss | 5 | 3 (60) [15-95] | 2 | 1 | 1 | 0 |
| Abnormal mucous membrane colour | 3 | 3 | 3 | 2 | 1 | 1 |
| Sweating | 2 | 1 | 1 | 1 | 0 | 0 |
| Urinary tenesmus | 2 | 1 | 1 | 0 | 1 | 0 |
| Polydipsia∞ | 1 | 0 | 0 | – | – | – |
| Gastric reflux | 1 | 1 | 1 | 1 | 0 | 0 |
| Peripheral blood leucocytosis# | 11 | 8 (73) [39-93] | 5 | 3 (60) ]14-94] | 2 (40) [5-85] | 0 |
| Peripheral blood leucopenia△ | 11 | 11 (100) [76-100] | 8 | 7 (88) [47-99] | 1 (13) [0-53] | 2 (25) [3-65] |
| Transabdominal ultrasound | | | | | | |
|   No abnormalities | 11 | 10 (90) [59-99] | 9 | 6 (66) [29-92] | 3 (33) [7-70] | 2 (22) [2-100] |
|   Abnormal peritoneal fluid (increased volume or hyperechoic) | 10 | 9 (90) [55-99] | 5 | 5 (100) [55-100] | 0 | 2 (40) [5-85] |
|   Abnormal peritoneal fluid with other abnormalities | 2 | 1 | 1 | 0 | 1 | 0 |
|   Distended small intestine | 2 | 1 | 1 | 1 | 0 | 0 |
|   Hepatic changes | 2 | 1 | 1 | 0 | 1 | 0 |
|   Abdominal mass | 1 | 1 | 1 | 0 | 1 | 0 |
| Peritoneal fluid (TNCC) | | | | | | |
|   $5 \times 10^9$ to $9 \times 10^9$/l | 9 | 8 (89) [51-99] | 5 | 5 (100) [55-100] | 1 (20) [0-71] | 0 |
|   $10 \times 10^9$ to $20 \times 10^9$/l | 11 | 10 (91) [51-99] | 6 | 4 (67) [22-96] | 2 (33) [4-77] | 1 (16) [0-45] |
|   $21 \times 10^9$ to $50 \times 10^9$/l | 13 | 12 (92) [64-99] | 9 | 7 (77) [39-97] | 2 (22) [2-60] | 1 (11) [0-48] |
|   $51 \times 10^9$ to $100 \times 10^9$/l | 15 | 12 (80) [52-96] | 8 | 7 (88) [47-99] | 2 (25) [3-65] | 2 (25) [3-65] |
|   $>100 \times 10^9$/l | 15 | 14 (93) [68-99] | 10 | 9 (90) [55-99] | 1 (10) [0-45] | 0 |
| Plasma fibrinogen | | | | | | |
|   ≤4 g/l | 18 | 17 (94) [73-99] | 11 | 10 (90) [59-99] | 1 (9) [2-41] | 0 |
|   >4 to 6 g/l | 18 | 17 (94) [73-99] | 13 | 12 (92) [64-99] | 1 (8) [0-36] | 3 (23) [5-54] |
|   >6 g/l | 10 | 10 (100) [74-100] | 6 | 5 (83) [36-99] | 3 (50) [12-88] | 2 (33) [4-78] |
| Duration of clinical signs | | | | | | |
|   24 hours | 33 | 27 (81) [64-93] | 14 | 14 (100) [81-100] | 0 | 10 (33) [0-33] |
|   1 to 3 days | 20 | 18 (90) [68-98] | 13 | 10 (77) [46-95] | 3 (23) [5-54] | 2 (15) [2-45] |
|   4 to 7 days | 5 | 5 (100) [55-100] | 5 | 5 (100) [55-100] | 0 | 0 |
|   >1 week | 1 | 1 | 1 | 0 | 1 | 0 |

\* Heart rate exceeding 45 bpm (Knottenbelt 2006)
† Rectal temperature exceeding 38·5°C (Knottenbelt 2006)
‡ Respiratory rate exceeding 15 breaths/minute (Knottenbelt 2006)
§ Including a history of coughing, nasal discharge, dyspnoea or submandibular lymphadenopathy
∞ History of increased water consumption reported by the owner
\# Leucocyte count >12·5 x $10^9$/l
△ Leucocyte count <5·5 x $10^9$/l
TNCC Total nucleated all count

males, and 16 breeds were represented; 12 were classified as of undefined breeding, and the second largest group consisted of nine thoroughbreds.

A potential predisposing condition was recorded in the history of seven of the 65 horses. Two had had a respiratory infection, two had had dystocia, and two had sustained vaginal lacerations at covering, and one horse had suffered a penetrating wound to the abdominal wall. The other 58 horses had no history of disease or injury associated with the development of peritonitis. The owners reported that some of the horses had shown mild changes in demeanour or body condition over a period of several days or even weeks, but only four had received treatment. One idiopathic case of peritonitis that survived to discharge, but for which long-term follow-up was not available, had received a six-day course of oral trimethoprim-sulfonamides and clenbuterol. A second idiopathic case, which was discharged and had had no complications up to 74 months after its discharge had been treated for a colon impaction, including enteral fluids and liquid paraffin administered via a nasogastric tube for three days before it was referred and before peritonitis was diagnosed. The two mares that sustained vaginal lacerations had also been treated, one with a five-day course of unspecified antimicrobials and one with a 10-day course of penicillin and phenylbutazone, followed by the drainage of a vaginal wall abscess. Sixteen of the 65 horses had been treated with anthelmintics within the previous two months and seven of them were also known to have been treated for encysted cyathostome larvae with either a five-day course of fenbendazole (Panacur Equine Guard; Intervet UK) or a single dose of moxidectin (Equest; Fort Dodge Animal Health) in the preceding autumn or winter. Four of the horses were known to have been overdue for anthelmintic treatment when they were referred. In one horse a recent faecal worm egg count was negative, and in another an ELISA for tapeworms gave a low result. The worming history of the other 45 horses was unknown.

Thirty-three of the 65 horses showed signs of colic, 32 showed signs of depression or lethargy, 21 had tachycardia, and 18 were pyrexic (Table 1).

**TABLE 2: Aetiology of the peritonitis in 65 horses and its association with their short- and long-term survival and the incidence of complications; percentages and 95 per cent confidence intervals (CIs) are included for groups of more than five horses**

| Aetiology | Number of horses | Short-term survival (%) [95% CI] | Number with follow-up available | Long-term survival (%) [95% CI] | Complications in non-survivors (%) [95% CI] | Complications in survivors (%) [95% CI] |
|---|---|---|---|---|---|---|
| Idiopathic | 50 | 47 (94) [83-98] | 31 | 26 (84) [66-94] | 5 (16) [5-33] | 4 (12) [3-29] |
| Vaginal tear | 4 | 4 | 2 | 2 | 0 | 0 |
| Gastrointestinal neoplasia | 2 | 0 | 0 | – | – | – |
| Ileal hypertrophy | 1 | 0 | 0 | – | – | – |
| Penetrating wound | 1 | 0 | 0 | – | – | – |
| Cystitis | 1 | 1 | 1 | 0 | 1 | 0 |
| Urinary calculus | 1 | 0 | – | – | – | – |
| Partial colon torsion and retroflexion of pelvic flexure | 1 | 1 | 1 | 1 | 0 | 1 |
| Granulosa cell tumour | 1 | 1 | 1 | 1 | 0 | 0 |
| Intestinal abscess | 2 | 1 | 1 | 0 | 1 | 0 |
| Liver abscess | 1 | 1 | 1 | 0 | 1 | 0 |

When they were first examined the mean total nucleated cell count in the peritoneal fluid of the horses was $105 \cdot 6 \times 10^9/l$ (range 5·8 to $350 \times 10^9/l$) (reference value $<5 \cdot 0 \times 10^9/l$), and the mean protein concentration was 36·1 g/l (range 5·5 to 80 g/l) (reference value <25 g/l). Their mean peripheral white blood cell count was $9 \cdot 8 \times 10^9/l$ (range $4 \cdot 0 \times 10^9$ to $25 \cdot 9 \times 10^9/l$) (reference range $5 \cdot 5 \times 10^9$ to $12 \cdot 1 \times 10^9/l$), and their mean plasma fibrinogen concentration was 4·9 g/l (range 0·5 to 11·2 g/l) (reference range 0 to 4·0 g/l).

Aerobic and anaerobic cultures were made of samples of peritoneal fluid from five of the horses, and in each case they were negative. However, bacteria were identified on smears of the peritoneal fluid taken from two of the horses.

Transabdominal ultrasonography of 26 of the horses revealed abnormalities in 15, including an increase in the volume or echogenicity of the peritoneal fluid of 12; one of these horses also had a hepatic abnormality, and one had an abdominal mass. Two of the horses had a distended small intestine, and one had hepatic changes without peritoneal fluid abnormalities (Table 1). There were no clinically significant findings in the other 11 cases.

Transrectal ultrasonography of four of the horses identified a mass in three, and fluid-filled foci within the vaginal wall of one mare.

Four of the horses underwent a laparoscopy, which confirmed the presence of vaginal lacerations that had been identified by manual palpation and an examination with a speculum in two of them. An abscess developing in the wall of the rectum secondary to the vaginal tear was identified in one of these cases. In the other two cases, the laparoscopy revealed fibrin deposits on the abdominal viscera but no underlying cause of peritonitis.

There were no statistically significant associations between the clinical variables and the horses' short- or long-term survival, or fatal and non-fatal complications. The only associations that approached significance (P=0·06) were between

the presence of clinical signs for less than 24 hours and long-term survival, and between a plasma fibrinogen concentration exceeding 6 g/l at admission and a higher risk of fatal complications.

The cause of the peritonitis was identified in only 15 of the 65 cases (Table 2). In four of the other 50 cases, a specific cause was suspected, but not confirmed; they were larval cyathostominosis (larvae identified in faeces), *Streptococcus equi* var *equi* infection (history of infection), cholangiohepatitis (high serum activities of hepatocellular enzymes and evidence of biliary stasis on liver biopsy), and chronic hepatitis (hepatic fibrosis identified postmortem). The horses with idiopathic peritonitis were significantly younger than the others (10·3 years [sd 6·6] v 14·1 years [sd 6·1]; P=0·048).

The methods of treatment included broad-spectrum antimicrobials in 63 of the 65 cases, either with or without non-steroidal anti-inflammatory drugs (NSAIDs). The other two horses received only anthelmintics. Seven of the horses were given anthelmintics (ivermectin or a five day course of fenbendazole) in addition to antimicrobials with or without NSAIDs. Nine of the horses were treated with an abdominal lavage, after an exploratory laparotomy in five of them, and while the other four were standing. The treatments continued for one to 44 days (mean 15·3 days, median 14 days). Fifty-six of the 65 horses survived to discharge, and 32 of the 38 horses for which follow-up information was available survived for 12 months or longer. Thirteen of these 38 horses suffered complications after their discharge, and eight of them had to be euthanased as a result. These complications were colic in seven, weight loss in two, and pyrexia, diarrhoea, renal failure and laminitis, each in one horse (Table 3). The owners of the long-term survivors reported that their horses returned to their previous level of use.

Forty-seven of the 50 idiopathic cases were significantly associated with a short-term survival rate (94 per cent, P=0·003, OR=10·4, 95 per cent CI 1·7 to 72·8). The long-term survival rate of idiopathic cases, using a cut-off point of 12 months, was high (84 per cent). Kaplan-Meier survival analysis showed that the idiopathic cases survived for significantly longer periods (P<0·001) than the cases of peritonitis of known aetiology (Fig 1). The two horses with vaginal tears both survived for 12 months or longer. Only one or two of the horses were suffering from ileal hypertrophy, penetrating abdominal wounds, intestinal abscesses, hepatic abscesses and cystic calculi, but these conditions were generally associated with a poor prognosis for short-term survival (Table 2). Both the horses with gastrointestinal tract neoplasia failed to survive to discharge.

There were no statistically significant associations between the different methods of treatment and the survival or complication rates among the 50 idiopathic cases.

**TABLE 3: Complications reported in 65 horses that could have been sequelae to their peritonitis**

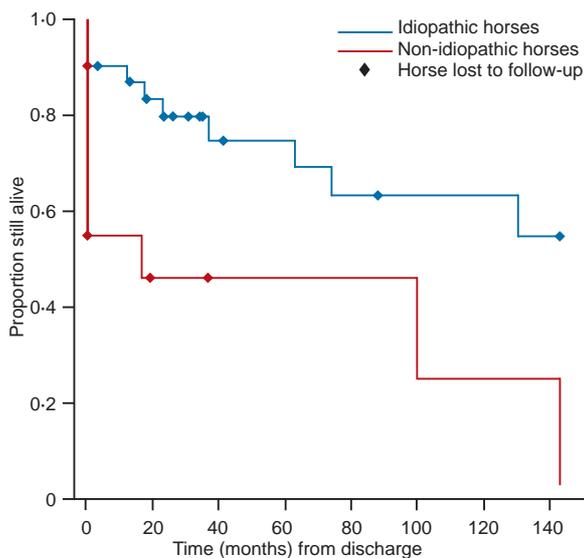| Complication | Number of horses | Time after discharge (months) | Aetiology | Number that survived | Duration of follow-up (months) |
|---|---|---|---|---|---|
| Colic | 7 | 1-36 | Idiopathic (6) | 3 | 0·25-86 |
| | | | Mesenteric abscess (1) | 1 | |
| Weight loss | 2 | 0·25-2 | Idiopathic | 1 | 0·25-26 |
| Diarrhoea | 1 | 0·25 | Idiopathic | 0 | 0·25 |
| Pyrexia | 1 | 63 | Idiopathic | 2 | 88 |
| Laminitis | 1 | 1 | Idiopathic | 0 | 1 |
| Renal failure | 1 | 0·25 | Cystitis | 0 | 0·25 |

**FIG 1: Survival plot in time (months) from discharge for horses with idiopathic or non-idiopathic peritonitis**

## DISCUSSION

The short-term survival rate of the horses in this study (86 per cent) was higher than in previous comparable studies, for example, 70 per cent (Dyson 1983), 57 per cent (Mair and others 1990) and 53 per cent (Feige and others 1997), possibly because of differences in aetiology and/or the management of the cases. Four of the non-surviving horses in the studies by Mair and others (1990) and Dyson (1983) had peritonitis attributable to large strongyles, and the higher survival rate may therefore be associated with a reduction in the frequency of peritonitis associated with large strongyles as a result of the more frequent use of effective modern anthelmintics. The peritonitis of another non-surviving horse in the study by Dyson (1983) was due to *Rhodococcus equi* abscessation. In this study, *R equi* was not identified as the underlying aetiology in any of the horses, a difference which may be due to geographical differences in the prevalence of this disease. Nearly all the horses had been treated with penicillin, gentamicin and metronidazole, with or without NSAIDs. Metronidazole was not used as part of the treatment regimen by Dyson (1983) or Mair and others (1990), but in this study the different combinations of antimicrobials used in the treatment of individual horses had no significant effect on the outcome and it is therefore not possible to attribute any difference in survival between the studies to differences in treatment. A considerably lower survival rate (40 per cent) was reported by Hawkins and others (1993) but this may have been due to the inclusion of horses with an acute rupture of the gastrointestinal tract.

Thirty-two (84 per cent) of the 38 horses for which long-term follow-up was available survived for longer than 12 months. Six short-term survivors were euthanased within 12 months, for complications that may have been related to their peritonitis, including colic, weight loss, diarrhoea, laminitis and renal failure. Five of these horses were euthanased within four weeks of discharge, suggesting that the underlying condition had not fully resolved, and the sixth survived for three months before being euthanased owing to colic. Two horses were euthanased more than 12 months after their discharge because they developed clinical signs that were considered to be related to their episode of peritonitis; one of them developed chronic, recurrent colic over a period of three years, and in the other an ulcerated small intestinal diverticulum perforated 17 months after it had been treated surgically for this condition. Other complications occurring after discharge that did not result in euthanasia were weight loss, colic and pyrexia. Whether these complications were sequelae to peritonitis, such as adhesions or chronic abscessation (Rumbaugh and others 1978, Hillyer and Mair 1997, Mair 2002a), or were unrelated to the peritonitis, is unknown. However, the results of the study provide a useful indication of the frequency of sequelae to peritonitis that may result in the horse requiring either further treatment or to be euthanased.

An analysis of the association between the categorical variables when the horses were admitted and their outcome provided little prognostic information. The 95 per cent CIs were wide for the variables that were significantly associated with survival or complications. Transabdominal ultrasonography identified abnormalities in 15 of the 26 horses to which it was applied, indicating its value in diagnosing and providing information on the aetiology of peritonitis. In the four horses that underwent laparoscopy, no new aetiological information was obtained, but the diagnosis of peritonitis was confirmed by the visualisation of fibrin adherent to the serosal surfaces of them. Laparoscopy was considered useful in the assessment and management of the two cases with vaginal lacerations, and it might have been useful in assessing the horse with a penetrating wound to the abdominal wall, but could not be applied to it. Walmsley (1999) reviewed previous studies and reported that laparoscopy can be a useful diagnostic procedure in horses with various types of abdominal disease, including peritonitis.

Only five of the horses had samples of peritoneal fluid examined by bacterial culture. Such cultures might have yielded further aetiological information in some cases; however, only low positive culture rates have been reported in previous studies, for example, 26 per cent (Dyson 1983) and 15 per cent (Mair and others 1990) and there is a delay in obtaining the culture results. As a result, most of the clinicians involved in the study elected not to have samples cultured, but to provide broad-spectrum antimicrobial cover. The outcome of the cases was not correlated with any particular treatment, such as the use of metronidazole, NSAIDs or abdominal lavage, either standing or at laparotomy. This was probably attributable to the small numbers of horses given each type of treatment, a result of the range of treatment regimens used by different clinicians at the two referral centres.

The short-term and long-term survival rates of the 50 horses with peritonitis due to no known cause were high. However, no single clinical variable was a reliable predictor of their long-term outcome. Although the idiopathic peritonitis group was younger then the non-idiopathic group (P=0·048), this statistical significance is slight and, the authors would argue, does not explain the level of differences observed in the Kaplan-Meier analysis. Furthermore, only one of the non-idiopathic horses was euthanased due to old age and this horse was only 14 years old at admission. There were four cases of peritonitis due to abdominal neoplasia, abdominal abscesses, urinary tract infection or obstruction, penetrating wounds to the body wall and ileal hypertrophy, and these causes were associated with poor outcomes.

## ACKNOWLEDGEMENTS

## References

BACH, L. G (1973) Exfoliative cytology of peritoneal fluid in the horse. In The Veterinary Annual. Eds C. S. G Grunsell, F. W. G Hill. Bristol, John Wright. p 102

BROWNLOW, M. A. (1979) Abdominal paracentesis in the horse: a clinical evaluation. MVetSci thesis, Sydney, University of Sydney

DYSON, S. (1983) Review of 30 cases of peritonitis in the horse. *Equine Veterinary Journal* **15,** 25-30

FEIGE, K., STEIGER, R., GRAF, U. & SCHOBERL, M. (1997) Peritonitis in horses: a retrospective study of 95 cases. *Tierarztliche Praxis* **25,** 55-61

GOLLAND, L. C., HODGSON, D. R., HODGSON, J. L., RAWLINSON, R. J., COLLINS, M. B., MCCLINTOCK, S. A. & RAISIS, A. L. (1994) Peritonitis associated with *Actinobacillus* in horses: 15 cases (1982-1992). *Journal of the American Veterinary Medical Association* **205,** 340-343

HANSON, R. R. (1999) How I treat horses with peritonitis. *Compendium on Continuing Education for the Practising Veterinary Surgeon* **21,** 965-973

HAWKINS, J. F., BOWMAN, K. F., ROBERTS, M. C. & COWAN, P. (1993) Peritonitis in horses: 67 cases (1985-1990). *Journal of the American Veterinary Medical Association* **203,** 284-288

HILLYER, M. H. & MAIR, T. S. (1997) Recurrent colic in the mature horse: a retrospective review of 58 cases. *Equine Veterinary Journal* **29,** 421-424

KNOTTENBELT, D. C. (2006) Part 1 vital signs, normal values. In Saunders' Equine Formulary, Eds J. Rodenhuis, R. Demetriou-Swanick. London, Saunders Elsevier. p 27

MAIR, T. S. (2002a) Additional diagnostic procedures. In Manual of Gastroenterology. Eds T. Mair, T. Divers, N. Ducharme. London, Saunders Elsevier. pp 16-17

MAIR, T. S. (2002b) Colic – other conditions. In Manual of Gastroenterology. Eds T. Mair, T. Divers, N. Ducharme. London, Saunders Elsevier. pp 330-332

MAIR, T. S. & HILLYER, M. H. (1997) Chronic colic in the mature horse: a retrospective review of 106 cases. *Equine Veterinary Journal* **29,** 415-420

MAIR, T. S., HILLYER, M. H. & TAYLOR, F. G. (1990) Peritonitis in adult horses: a review of 21 cases. *Veterinary Record* **126,** 567-570

MATTHEWS, S., DART, A. J., DOWLING, B. A., HODGSON, J. L. & HODGSON, D. R. (2001) Peritonitis associated with *Actinobacillus equuli* in horses: 51 cases. *Australian Veterinary Journal* **79,** 536-539

NELSON, A. W. (1979) Analysis of equine peritoneal fluid. *Veterinary Clinics of North America. Large Animal Practice* **1,** 267-274

PARRY, B. W. (1983) Survey of 79 referral colic cases. *Equine Veterinary Journal* **15,** 345-348

RUMBAUGH, G. E., SMITH, B. P. & CARLSON, G. P. (1978) Internal abdominal abscesses in the horse: a study of 25 cases. *Journal of the American Veterinary Medical Association* **172,** 304-309

VAN HOOGMOED, L., RODGER, L. D., SPIER, S. J., GARDNER, I. A., YARBROUGH, T. B. & SNYDER, J. R. (1999) Evaluation of peritoneal fluid pH, glucose concentration and lactate dehydrogenase activity for detection of septic peritonitis in horses. *Journal of the American Veterinary Medical Association* **241,** 1032-1036

WALMSLEY, J. P. (1999) Review of equine laparoscopy and an analysis of 158 laparoscopies in the horse. *Equine Veterinary Journal* **31,** 456-464