Gastric Dilatation-Volvulus Syndrome: Outcome and Factors Associated with Perioperative Mortality

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Abstract

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Gastric dilatation-volvulus (GDV) or gastric dilatation (GD), were diagnosed in 173 dogs during the period from 1997 to 2001. Risk factors possibly associated with higher mortality in cases of GDV were evaluated. Variables suitable for quantitative evaluation were examined by the Student's *t*- test with regard to the risk of death. We characterised groups of dogs with potential risk factors (age, body weight, time lapsed from the last feeding until presentation, time lapsed from the onset of clinical signs until presentation, time lapsed from the last feeding until the onset of clinical signs), and compared *the groups* with regard to the risk of death, using the χ^2- test. There were no differences between the dying and surviving patients as to their age, body weight and time lapsed from the last feeding until the onset of clinical signs. The time lapsed from the onset of clinical signs until presentation at the clinic was a significant factor associated with a lower survival rate. Mortality in dogs with GDV was 26.3% (36/137). Significantly higher mortality ($p < 0.01$) was found in dogs with gastric necrosis. Gastric necrosis in dogs with GDV is responsible for a 6.5 times higher risk of death compared to patients without gastric necrosis. There was also higher mortality in dogs after splenectomy ($p < 0.05$). Results of our study suggest that the most important prognostic factor is a timely treatment following the onset of clinical signs of GDV.

Dog, retrospective study, gastric necrosis, splenectomy, risk factors, feeding

Gastric dilatation-volvulus (GDV) is a very serious, life-threatening syndrome typically affecting large and giant breeds of dogs. For example, in the United States of America this syndrome affects about 60 000 dogs a year (Burrows and Ignaszewski 1990). The risk of this syndrome in some dog breeds is very high. In the Great Dane it amounts to more than 40% (Glickman et al. 2000). Despite its recognition more than 90 years ago (Cadeac 1906) and considerable advances in diagnosis and treatment, mortality associated with GDV still remains high. Various studies report mortality varying from 10 to 60% (Betts et al. 1974; Muir 1982; Walshaw and Johnson 1976; Matthiesen 1983; Frankquist and Obel 1979; Winfgield et al. 1975; Glickman et al. 1994; Brockman et al. 1995; Brourman 1996; Van Sluijs 1991; Glickman et al. 1998; Eggertsdóttir and Moe 1995; Nagel and Neumann 1992). It is, however, difficult to compare individual results because of differences in the definition of mortality and different therapeutic procedures within individual studies.

On the basis of our current knowledge, therapy of the syndrome consists of three important parts:

1) immediate and aggressive patient stabilisation including gastric decompression and treatment of shock; 2) surgical correction of volvulus including gastropexy to prevent recurrence; and 3) post-operative monitoring and intensive care (Matthiesen 1983; Aronson et al. 2000).

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Recently, a number of factors have been identified in association with higher mortality, such as mobility of the patient, patient behaviour (Glickman et al. 1998; Van Sluijs 1991) and gastric necrosis (Glickman et al. 1998; Brockman et al. 1995; Brourman et al. 1996; Matthiesen 1983).

The aim of this retrospective study was to evaluate the risk factors associated with higher mortality.

Materials and Methods

Our study comprised all dogs diagnosed with gastric dilatation (GD) or gastric dilatation-volvulus (GDV) treated at the Small Animal Clinic of the University of Veterinary and Pharmaceutical Sciences, Brno, Czech Republic, between 1997 and 2001. The analysis was based on the records of individual dogs.

Criteria for including dogs into groups of GD or GDV were based on clinical and radiographic examinations. Dogs that died before presentation at the clinic were excluded from the study. Laparotomy was performed with the consent of the owners to confirm the diagnosis of volvulus in all the cases diagnosed with GDV. When the owners refused the surgery or the dog died before surgery, the final diagnosis was made at autopsy. When the owners refused autopsy, the clinical diagnosis of GDV was considered definitive for the purposes of this study. In one dog suffering from recurrent dilatation, the diagnosis of simple gastric dilatation without volvulus was confirmed by laparotomy. In other cases of GD, the presumptive diagnosis was confirmed by repeated examinations of the patient.

The analysed data on every patient included the date of presentation, age, body weight, sex, address of the owner, time *lapse*d from the last feeding, time of the onset of clinical signs, and time of presentation at the clinic. The last-mentioned *data*, however, were not recorded in all patients. In patients undergoing surgery, other *indices* such as gastric necrosis, splenectomy, gastrotomy and gastrectomy were also recorded.

Patients were subjected to routine physical examination. Typical signs of GDV or GD led to an immediate treatment. It consisted of treatment of shock, gastric decompression, and gastric reposition in cases of GDV. Vascular access was obtained by placing an intravenous catheter into v. cava cranialis or v. cephalica antebrachii. Following the catheter placement, balanced complete isotonic infusion fluids (Ringer's lactate solution) were administered at 40-90ml per kilogram of body weight and hour. Then, a stomach tube was passed and the stomach emptied by careful compression. The stomach was rinsed repeatedly by lukewarm tap water. When it was not possible to pass a stomach tube, percutaneous gastrocentesis was performed. After the successful passage of a stomach tube, we differentiated between GD and GDV by radiography using a laterolateral projection with the animal in right lateral recumbency, and other projections in questionable cases. Patients with GDV were then treated surgically. Surgery followed patient stabilisation, judged by normalisation of the capillary refill time, colour of mucous membranes, quality of pulse and decrease in the heart rate after about 20-120 minutes from the beginning of therapy. A standard midline celiotomy was performed and the stomach repositioned to normal. When reposition was difficult due to gastric dilation, the stomach was first decompressed and then repositioned. After reposition, the gastric wall was inspected and the stomach repeatedly flushed by an orogastric tube, unless it had been done before surgery. The colour of a necrotic gastric wall remained dark red to black, particularly at the gastric fundus. The necrotic part of the stomach was resected until subjectively healthy tissue was found. Total splenectomy was performed in cases of thrombosis of v. lienalis or its branches, avulsion or thrombosis of short gastric vessels, and spleen rupture or torsion. Incision or belt-loop gastropexy was performed in all cases to prevent recurrence.

Intravenous fluids were continued throughout the operation as well as after. Antimicrobial agents used covered Gram-negative bacteria, in particular. H_2-blockers were administered intravenously in the peri-(or pre)operative period as well as post-operatively for the next 5 to 10 days. Water was offered per os 12 to 24 hours after surgery and intravenous fluid therapy was discontinued 24 to 36 hours later if no subsequent vomiting occurred after water intake. Gruel feeds in small doses should be given to patients that drink and do not vomit after gastrectomy after at least 2 days post op. Patients were monitored for cardiac arrhythmias, but infrequent extrasystoles were not treated. Cases of clinically manifested ventricular arrhythmia were treated with lidocaine in a continuous rate infusion at the dose of 25-80 μg/kg/min. Patients tolerating administration of drugs per os were given procainamide at the dose of 10-20 μg/kg q.i.d.

In general, we evaluated mortality and its causes in GD and GDV. In cases of GDV, we evaluated factors based on recorded data, which might be associated with higher mortality.

Variables suitable for a quantitative evaluation were examined by the Student's t-test with regard to the risk of death (age, body weight, time lapsed from the last feeding until presentation, time lapsed from the onset of clinical signs until presentation, time lapsed from the last feeding until the onset of clinical signs). For each of the potential risk factors we characterised groups of dogs to compare with each other by the χ^2-test with regard to the risk of death. The difference between individual groups was considered to be significant at $p < 0.05$. Odds ratio indicated the probability of death as compared to the control group. Values higher than or lower than 1.00 indicated higher or lower risks, respectively. Mortality was determined for each group.

Results

During the study period we treated 173 dogs of GDV and GD. These patients represented a proportion of 0.95% of all the patients treated at the Department of Surgery and Orthopedics of the Small Animal Clinic.

A total of 17 patients were identified as GD. All survived the acute episode and were treated in a conservative way with only one dog undergoing laparotomy and gastropexy.

A total of 156 patients were diagnosed with GDV. Nineteen dogs were excluded from further analyses because they died immediately after arrival at the clinic (3 cases), or their owners disagreed with therapy because of unrelated diseases (mammary gland tumours, cardiomyopathy, chronic renal failure) or financial constraints. The remaining 137 dogs underwent surgery after stabilisation. Three patients died during the operation. Nine other dogs were euthanized during the operation with the consent of the owner because of extensive gastric necrosis. Further 21 dogs died after the operation due to various causes (disseminated intravascular coagulopathy, circulatory collapse, heart arrest or arrhythmia). Another three patients were euthanized because of financial constraints, gastric atony and acute hepatic failure accompanied by hepatoencephalopathy. The remaining 101 patients were clinically healthy within 10-14 days after the operation.

Table 1

Risk Factor	Number	Mean	Standard deviation
Age			
Dogs died	36	7.17	2.97
Dogs survived	101	6.44	2.73
Body Weight			
Dogs died	36	39.89	11.15
Dogs survived	101	43.14	13.71
Time Lapsed from Feeding until Initiation of Treatment↑			
Dogs died	25	9.54	6.00
Dogs survived	82	6.76	5.46
Time Lapsed from Onset of Clinical Signs until Initiation of Treatment↑			
Dogs died	25	5.12	4.12
Dogs survived	82	3.24	3.12
Time Lapsed from Feeding until Onset of Clinical Signs			
Dogs died	23	4.41	3.99
Dogs survived	8	3.50	3.80

↑ difference at level of significance 5% between dogs that died and survived (Student's *t*-test)

Risk factors, which might be associated with higher mortality in cases of GDV, are summarised in Table 1. Time periods lapsed from the last feeding until presentation and from the onset of clinical signs until presentation were considerably longer with statistical significance in patients that died than those that survived (Student's t-test, $p < 0.05$). There were no differences between the dying and surviving patients as to their age, body weight and time period from the last feeding until the onset of clinical signs. The survival rate of dogs with GDV was not dependent on age, body weight, sex, address of the owner, or date of treatment (cf. Table 2). However, we did note a tendency to higher mortality in older dogs. Nine-year-old dogs and older had an up to 4.00 times lower survival rate than dogs under 4 years. The time lapsed from the onset of clinical signs until presentation at the clinic was a significant factor associated with a lower survival rate. Even though the time lapsed from feeding until presentation at the clinic was not a significant factor, as the interval grew,

Table 2

Risk Factor	Number of Dogs Died	Number of Dogs Survived	Odds Ratio	Probability Values (χ^2 – test)	Mortality in the Group of Dogs (%)
Age					
< 4 years ■	3	18	NA	NA	14.29
4 – 6.9 years	14	39	2.15	NS	26.42
7 – 8.9 years	9	29	1.86	NS	23.68
≥ 9 years	10	15	4.00	NS	40.00
Body Weight					
≤ 27 kg ■	4	10	NA	NA	28.57
28 – 35 kg	12	30	1.00	NS	28.57
36 – 47 kg ■	12	26	1.15	NS	31.57
≥ 48 kg	8	35	0.57	NS	18.60
Sex					
Female ■	17	53	NA	NA	24.29
Male	19	48	1.23	NS	28.36
Owner's Address					
Local ■	12	50	NA	NA	19.35
From neighbouring cities and villages	24	51	1.96	NS	32.00
Year of Treatment					
1997 ■	5	13	NA	NA	27.78
1998	4	27	0.39	NS	12.90
1999	7	20	0.91	NS	25.93
2000	14	19	1.92	NS	42.42
2001	6	22	0.71	NS	21.43
Surgeon Number					
1 ■	6	17	NA	NA	26.09
2	1	4	0.71	NS	20.00
3	2	8	0.71	NS	20.00
4	9	10	2.55	NS	47.37
5	1	5	0.57	NS	16.67
6	3	9	0.94	NS	25.00
7	0	6	NA	NS	0.00
8	3	5	1.70	NS	37.50
9	3	10	0.85	NS	23.08
10	0	6	NA	NS	0.00
11	4	6	1.89	NS	40.00
12	1	8	0.35	NS	12.50
Missing data	3	7	-	-	-
Time Lapsed from Feeding until Initiation of Treatment					
0 – 2.9 h ■	2	21	NA	NA	8.70
3 – 4.9 h	5	19	2.76	NS	20.83
5 – 10.9 h	9	22	4.30	NS	29.03
≥ 11 h	9	20	4.73	NS	31.03
Data Missing	11	20	-	-	-
Time Lapsed from Onset of Clinical Signs until Initiation of Treatment					
0 – 2.9 h ■	8	47	NA	NA	14.55
3 – 4.9 h	7	21	1.96	NS	25.00
5 – 10.9 h	7	12	3.48	$p < 0.05$	36.84

■ Reference category   NA Not applicable   NS Non-significant

Table 2 continued

Risk Factor	Number of Dogs Died	Number of Dogs Survived	Odds Ratio	Probability Values (χ^2 – test)	Mortality in the Group of Dogs (%)
≥ 11 h	3	2	8.81	p < 0.05	60.00
Data Missing	11	19	-	-	-
Time Lapsed from Feeding until Onset of Clinical Signs					
0 – 2.9 h ■	9	45	NA	NA	16.67
3 – 4.9 h	7	13	2.69	NS	35.00
5 – 10.9 h	6	15	2.00	NS	28.57
≥ 11 h	1	7	0.71	NS	12.50
Data Missing	13	21	-	-	-
Gastric Necrosis					
No ■	21	91	NA	NA	18.75
Yes	15	10	6.50	p < 0.01	60.00
Gastrectomy					
No ■	20	91	NA	NA	18.02
Yes	4	10	1.82	NS	28.57
Gastrotomy					
No ■	20	94	NA	NA	17.54
Yes	4	7	2.69	NS	36.36
Splenectomy					
No ■	6	51	NA	NA	10.53
Yes	18	50	3.06	p < 0.05	26.47

■ Reference category   NA Not applicable   NS Non-significant

mortality increased many times. Significantly higher mortality ($p < 0.01$) was detected in dogs with gastric necrosis. However, if they were treated by gastrectomy and not euthanized, gastric necrosis was no more associated with higher mortality. Higher mortality was also found in dogs that were splenectomised ($p < 0.05$). Gastrotomy or the operating surgeon had no effect on the survival rate.

Table 3

Time Lapsed from Onset of Clinical Signs until Initiation of Treatment	Number of Dogs with Gastric Necrocis (died/all cases)	Number of Dogs without Gastric Necrocis (died/all cases)	Odds Ratio	Probability Values (χ^2 – test)
0 – 2.9 h ■	2/3	6/52 ■	NA	NA
3 – 4.9 h	4/7	3/18	5.78	p < 0.01
5 – 10.9 h	4/9	3/10	15.60	p < 0.01
≥ 11 h	0/0	3/5	NA	NA
Data Missing	5/6	6/24	-	-

■ Reference category   NA Not applicable   NS Non-significant

As the duration of clinical signs increased, the number of dogs suffering from gastric necrosis and requiring splenectomy (Tables 3 and 4) became significantly higher. The number of splenectomised patients, however, did not increase significantly in dogs with gastric necrosis (Table 5).

Table 4

Time Lapsed from Onset of Clinical Signs until Initiation of Treatment	Number of Dogs with Splenectomy (died/all cases)	Number of Dogs without Splenectomy (died/all cases)	Odds Ratio	Probability Values (χ^2 – test)
0 – 2.9 h ■	3/26	3/27	NA	NA
3 – 4.9 h	4/10	0/14	0.74	NS
5 – 10.9 h	4/13	0/3	4.50	$p < 0.05$
≥ 11 h	3/5	0/0	NA	NA
Data Missing	4/10	3/16	-	-

■   Reference category   NA Not applicable   NS Non-significant

Table 5

Splenectomy	Number of Dogs with Gastric Necrosis (died/all cases)	Number of Dogs without Gastric Necrosis (died/all cases)	Odds Ratio	Probability Values (χ^2 – test)
No ■	9/11	7/56	NA	NA
Yes	6/14	14/56	1.27	NS

■   Reference category   NA Not applicable   NS Non-significant

Discussion

The total of 173 patients with GDV and GD represented a proportion of 0.93% of all patients treated during the study period at the Department of Surgery and Orthopedics of the Small Animal Clinic. The possibility of comparing our findings with published data on the prevalence of GD and GDV is rather limited, because our study was based on patients treated at the Department of Surgery only, while other studies included all the patients at a clinic. The system of administration and data recording at other departments did not allow such evaluation. Dogs with GDV and GD represented approximately 0.3 to 0.4% of all the patients treated at our Small Animal Clinic. This prevalence of the syndrome corresponds with other studies reporting the range of 0.13 to 0.76% of all the patients treated (Strombeck and Guilford 1990; Glickman et al. 1994).

According to previous studies, 22 to 48% of patients with a bloat syndrome suffered from GD (Brockman et al. 1995; Glickman et al. 1994). In our study, there were only about 10% of patients with GD. This might be because of an actually lower prevalence of simple gastric dilatation in the Czech Republic, or due to the pre-selection of patients at other departments of the Small Animal Clinic by referring veterinary surgeons. Another possible explanation is that some cases presented in this study had a relatively long time period from the onset of clinical signs until presentation at the clinic. Most authors share the opinion that in the GDV syndrome, gastric dilatation occurs as a primary event, whereas volvulus occurs secondarily (Matthiesen 1993; Aronson et al. 2000). The longer time lapsed from the onset of clinical signs until arrival at the clinic could thus be responsible for the change from simple dilatation to secondary volvulus of the presented case.

Patients with GD had a 100% survival rate (17/17). This corresponds to the study by Brockman et al. (1995), documenting an approximately 99% survival rate in dogs with GD. Since our clinic employs a similar therapeutic scheme as was used in the above mentioned study, it may be assumed that GD patients treated with this method have an

excellent prognosis. Results of these clinical case studies could thus confirm that in GDV patients, volvulus is a much more important factor, leading to gastric necrosis, considerable alteration of the general condition of the patient, and higher mortality. However, the question remains as to the relatively low survival rate of patients with GD recorded in the Veterinary Medical Data Base at 12 veterinary institutions from 1980 to 1989. These patients had 28.6% mortality (Glickman et al. 1994). Perhaps the difference may be due to different therapeutic procedures or improper selection of cases.

It is difficult to compare the mortality of our GDV patients with the published data because of different definitions of mortality, therapeutic procedures and equipment of individual clinics. In our study we found a 26.3% (36/137) mortality of dogs with GDV. Some authors, however, include all patients into the computation of the survival rate. In that case our study should include the patients euthanized after diagnosis due to unacceptable financial costs or concurrent diseases (16 cases), and the patients dying immediately after arrival at the clinic (3 cases). Such mortality would amount to 35.3%. Compared to the published data, our 26.3% mortality was similar or slightly less successful than the results obtained by specialised or university clinics with mortality rates of 13.2% (Glickman et al. 1998), 13.9% (de Papp et al. 1999), 15% (Brockman et al. 1995), 17.5% (Brourman et al. 1996), 29% (Van Sluijs 1991), 33.3% (Glickman et al. 1994), 33.2% (Nagel and Neumann 1992), 33.3% (Matthiesen 1983), 34% (Eggertsdóttir and Moe 1995), 34.1% (Flanders and Harvey 1984). Differences in survival rates among individual clinics using similar procedures can be due to various levels of pre- and early post-operative care and patient monitoring that allows decreasing the early post-operative mortality by quick and aimed therapy. Such care was not possible at our clinic for patients presenting with GDV and GD overnight due to the lack of most laboratory examinations and personnel. In order to decrease mortality in these cases, it seems inevitable to create an independent intensive care unit of sufficient equipment and competent personnel. At present, however, the survival rate is considerably influenced by early treatment, which depends on adequate public awareness of the syndrome.

A number of factors which might be associated with higher mortality in dogs with GDV (Glickman et al. 1998; van Sluijs 1991; Brockman et al. 1995; Brourman et al. 1996; Matthiesen 1983) have already been studied. As Brockman et al. (1995), we did not include the dogs euthanized after diagnosis or dying on arrival at the clinic into the analysis of factors increasing mortality. It seems more appropriate to exclude these patients from the study (29 cases euthanized before surgery), because it is not clear whether they would have survived or died after treatment. For this reason, the results of the study by Glickman et al. (1998) must be considered with care; they included this kind of patients into the group of dead dogs, which might lead to misrepresentation.

The age of the dog suffering from the GDV syndrome is one of the potential risk factors. In our study, we did not find older age of the dog to be a significant risk factor for the short-term survival rate. Nevertheless, patients that did not survive were older on average than the surviving ones, and 9-year-old patients or older had a 4.00 times higher mortality than dogs under 4 years. Glickman et al. (1998) report similar values and found a 3.11 times higher risk for the same age categories. The cause of this phenomenon remains unexplained. Changes of the cardiovascular apparatus in the form of sub-clinical disorders or age-related lesions leading to the loss of compensation mechanisms may play a role.

The age and sex of the dogs in our study as well as in others (Glickman et al. 1998; van Sluijs 1991; Brockman et al. 1995; Brourman et al. 1996; Matthiesen 1983) did not influence the survival rate. The lower risk of death in dogs weighing 48 kg or more found in our study might be explained by greater awareness of the risk of GDV among the owners of giant breeds, and by the resulting earlier arrival at the clinic after the onset of clinical signs.

Time lapsed from the onset of clinical signs until presentation of the patient and from the last feeding until presentation was a significant risk factor for the survival rate in our study. The time lapsed from the last feeding until presentation is a sum of time periods lapsed from the last feeding until the onset of clinical signs and from the onset of clinical signs until presentation. As the time lapsed from the last feeding until the onset of clinical signs is not a significant factor, the only risk factor is the time period from the onset of clinical signs until presentation. Dogs with GDV arriving at the clinic within three hours from the onset of clinical signs had a nine times lower mortality rate than those arriving after eleven and more hours. Our study thus confirms the general recommendation to treat such patients as soon as possible after the onset of clinical signs (Aronson et al. 2000; Strombeck and Guilford 1990). Previous studies, however, did not strictly determine the time period from the onset of clinical signs until presentation as a significant risk factor (Glickman et al. 1998; Van Sluijs 1991; Brockman et al. 1995). Van Sluijs (thesis) found that in a proportion of dogs, GDV can have a very quick progress and high mortality, associated with a marked and quick onset of signs, whereas in other cases the syndrome can have lower mortality with a slow onset of clinical signs, which allows the owners to arrive at the clinic much later. For purposes of future studies it would therefore be appropriate to consider other factors possibly influencing mortality, such as the degree of volvulus and gastric distension. It is interesting to compare our survival rate with the values published by Glickman et al. (1998) in relation to the time lapsed from the onset of clinical signs until presentation. In our study, dogs treated within two hours from the onset of clinical signs are characterised by 14% mortality (8/55), whereas in the study by Glickman et al. (1998) it is 20% (14/70). The difference in the total survival rates between these two studies depends only on the duration of clinical signs before treatment. Patients in our study arrived and received treatment later. Dog owners' awareness of clinical signs and urgency of treatment, not only in the predisposed breeds, along with a better pre- and post-operative care could contribute to higher survival rates of GDV patients. A number of owners, however, do not have their dog under supervision after feeding. In such dogs the time lapsed from the last feeding until presentation could serve as a prognostic criterion.

Gastric necrosis was a highly significant risk factor in our study ($p < 0.01$). Gastric necrosis in dogs with GDV is responsible for a 6.5 times higher risk of death, compared to patients without gastric necrosis. Mortality in such patients amounts to 60%. These results correspond to other studies reporting gastric necrosis as a significant risk factor with mortality rates as high as 60% (Van Sluijs 1991; Glickman et al. 1998; Brockman et al. 1995; Brourman et al. 1996; Clark and Pavletick 1991). As in other papers, gastric necrosis was the most common reason for patient euthanasia in our study. An interesting finding is that, if patients are left alive during the operation despite gastric necrosis and go through gastrectomy, gastric necrosis becomes an insignificant risk factor and mortality in such patients amounts to about 28%, a value only twice as high as in the patients not requiring gastrectomy. This result leads us to believe that patients with gastric necrosis are euthanized too soon and might survive after gastrectomy. However, the term gastric necrosis is quite general and can mean the affection of only a small part of fundus or the whole gastric wall. It would therefore be appropriate for further studies to specify the extent of gastric necrosis before conclusions about the relationship between gastrectomy and survival in cases of gastric necrosis can be made.

The number of dogs with gastric necrosis in our study significantly increased with the duration of clinical signs. Gastric necrosis was found in 5.5, 24.1 and 47.4% of dogs treated within three hours, after 3 to 4.9 hours and 5 to 10.9 hours from the onset of clinical signs, respectively. Our results of clinical cases confirm previous experimental studies of surgically induced gastric volvulus, in which necrotic changes of gastric fundus were found

after 4 hours (Lantz et al. 1984). In five patients suffering from clinical signs for 11 hours and more, there was no gastric necrosis. In spite of it, three of them died. In our opinion, the absence of gastric necrosis in dogs with ongoing clinical signs for 11 hours and more is simply caused by the fact that dogs suffering from GDV including gastric necrosis would not live long enough to receive treatment.

As in a previous study (Brourman et al. 1996), splenectomy was another risk factor associated with higher mortality. In the GDV syndrome, splenomegaly is commonly caused by congestion of the spleen due to hypertension of vena portae and a. lienalis, or strangulation of vena lienalis secondary to splenic torsion. Long-lasting congestion of the spleen (particularly in spleen torsion) leads to marked systemic hypovolemia, ischemia and subsequent necrosis of spleen parenchyma. It results in the thrombosis of vessels in the spleen and possibly in systemic thromboembolism and development of DIC (Pope and Rochat 1993). If necrosis of the spleen (dark blue or even black colour) or thrombotisation of large vessels become evident, the spleen should not be repositioned but removed because of the possibility of releasing vasoactive mediators, micro-thrombi or degradation products into the systemic circulation (Matthiesen 1993; Aronson et al. 2000). When splenectomy is performed in cases not requiring the procedure, the animal gets stressed by the loss of red blood cells, thrombocytes and some coagulation factors. In this way, splenectomy could initiate the onset of DIC, hypovolemia, shock progression and the loss of compensation mechanisms of the cardiovascular apparatus. It was also found that splenectomy by itself can be the cause of ventricular arrhythmia (Marino et al. 1994). Splenectomy also significantly prolongs the operation. Higher mortality associated with splenectomy could be caused both by disorders of the spleen and the splenectomy itself. The decision to perform splenectomy is thus quite controversial. Matthiesen (1983), Mayer-Lindenberg et al. (1993) and Brockman et al. (1995) report splenectomy in 8, 3.4 and 20.2% of dogs with GDV, respectively. In comparison to the above mentioned studies, we performed splenectomy much more often, i.e. in 53.1% of the operated dogs (68/125). It is questionable whether it was necessary in all the cases, and whether preservation of the spleen might have increased the survival rate.

Splenectomy in our study was performed more often in dogs with a longer duration of clinical signs of GDV. In dogs treated within three hours, after 3 to 4.9 hours, 5 to 10.9 hours and 11 hours and more from the onset of clinical signs, splenectomy was performed in 49.1, 41.7, 81.3 and 100% of cases, respectively. It confirms the fact that some time is necessary for the pathology of the spleen to develop. We did not, however, find any association between gastric necrosis and splenectomy. Since we obtained the data from patient records, it is not possible to generalise or overestimate this finding. The results may possibly be distorted by cases in which pathology of the spleen was diagnosed, but the animals were euthanized due to gastric necrosis and no splenectomy was performed.

We conclude from the results of our study, that the most important prognostic factor is a timely treatment following the onset of clinical signs of GDV. All other risk factors evaluated in this study, such as gastric necrosis and splenectomy are only a consequence of a late treatment. The duration of clinical signs can thus help to formulate a prognosis even on arrival at the clinic. When owners do not know the time of the onset of clinical signs, it is possible to use the time lapsed from the last feeding as a prognostic criterion. Other prognostic clinical signs such as mobility of the patient on arrival, patient behaviour (Glickman et al. 1998; Van Sluijs 1991), heart arrhythmia (Brourman et al. 1996) and plasma lactate level (de Papp et al. 1999) can also be employed. Our study confirmed that better awareness of GDV among the owners of giant breeds of dogs (over 48 kg) led to a timely arrival at the clinic with nearly half the mortality rate in comparison to other breeds (18.6%). It should therefore be the goal of veterinary medicine not only to provide

continuous intensive care for dogs with GDV but also to promote public awareness among dog owners about this syndrome.

Syndrom dilatace a volvulu žaludku - výsledky a faktory spojené s perioperační úmrtností

Syndrom dilatace a volvulu žaludku: výsledky a faktory spojené s perioperační úmrtností Dilatace a volvulus žaludku (GDV) nebo dilatace žaludku (DV) byly diagnostikovány u 173 psů během období od ledna 1997 do srpna 2001. Diskutovány jsou rizikové faktory, které mohou souviset s vyšší mortalitou v případech GDV. Parametry vhodné pro kvantitativní vyhodnocení byly zhodnoceny s ohledem na riziko úmrtí za použití Studentova t-testu. Byly stanoveny skupiny psů s možnými rizikovými faktory (stáří, tělesná hmotnost, časové období od posledního krmení do příchodu na kliniku, časové období od začátku klinických příznaků do příchodu na kliniku, časové období od posledního krmení do začátku klinických příznaků), a tyto skupiny byly porovnány s ohledem na riziko úmrtí za použití χ^2-testu. Nebyl zjištěn rozdíl mezi uhynulými a přežívajícími pacienty s ohledem na stáří, tělesnou hmotnost a období od posledního krmení do začátku klinických příznaků. Signifikantní faktor spojený s nižší mírou přežívání byl časový úsek do začátku klinických příznaků po příchod na kliniku. Úmrtnost u psů s GDV byla 26,3 % (36/137). Signifikantně vyšší úmrtnost ($p < 0,01$) byla u psů s nekrózou žaludku. Nekróza žaludku u psů s GDV představovala 6,5krát vyšší riziko úmrtí v porovnání s pacienty bez nekrózy žaludku. Byla zjištěna také vyšší úmrtnost u psů po splenektomii ($p < 0,05$). Výsledky naší studie vedou k závěru, že nejdůležitějším prognostickým faktorem je včasnost léčby, bezprostředně po projevení se klinických příznaků GDV.

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