tractile integral (PCI; amplitude x duration x length of the contraction wave) and the bolus transit time (BTT). Lower esophageal sphincter (LES) characterisation comprised: baseline pressure and residual pressure (lowest continuous 3 second mean LES pressure relative to intragastric pressure during swallow induced relaxation).The median values for water/food bolus swallows in awake (sedated) dogs were calculated i) UES: baseline pressure 11.5 mmHg (16.1), residual pressure -2.74/2.73 mmHg (-2.03/2.53) and relaxation duration 192.5/245 ms (230.25/324) ii) tubular esophagus function: PCI 478.15/1012 mmHg-cm-s (525.08/1605.4) and BTT 3.7/4.08 ms (4.85/5.82) iii) LES: baseline pressure 36.85 mmHg (16.8), residual pressure 7.2/5.64 mmHg (10.83/7). Significant differences were found for the UES relaxation duration (water) ($p=0.023$) and BTT (food bolus) ($p=0.008$).In conclusion, HRM is a feasible technique for the evaluation of esophageal function in dogs. Patients that require sedation can still be examined, however at this point it is not clear if sedation would affect the assessment of motility disorders in dysphagic dogs.

GA-O-7
WIRELESS AMBULATORY ESOPHAGEAL PH-MONITORING IN HEALTHY DOGS AND DOGS WITH HISTORICAL AND CLINICAL SIGNS INTERPRETED AS GASTRO-ESOPHAGEAL REFLUX. P.H. Kook, J. Kempf, C.E. Reusch. Clinic for Small Animal Internal Medicine, ZURICH, Switzerland

In humans ambulatory intraesophageal pH-monitoring utilizing the Bravo® capsule is the standard test for establishing pathological gastroesophageal reflux (GER). This technique not only provides information on esophageal acid exposure, but is also able to assess symptoms associated with GER. In dogs GER is poorly understood and it is not clear if GER actually represents a clinically relevant problem.The goals of this study were to examine the canine esophageal pH milieu in health and to examine esophageal pH in dogs presenting with signs commonly attributed to GER in the veterinary literature.Thirteen client-owned dogs (COD) of various breeds (median BW 20.3kg, 6.1-45; median age 5y, 1-11) were included. Clinical signs ultimately leading to pH-monitoring comprised: lip-smacking (6), repeated swallowing motions (3), chronic vomiting (3), cough (3), retching (2), regurgitation (2), sudden discomfort (2), excessive surface-licking (2), ptyalism (2), presumed postprandial pain (2), refusal to eat despite interest (2), history of esophageal foreign bodies (1), halitosis (1). Each dog showed a median of 3 (1-4) signs, 3 dogs had additional diarrhea. Six healthy Beagles (median BW 13.9kg, median age 1.5y) with unremarkable gastroduodenal evaluation served as controls (C). No prior antacid or prokinetic treatment was allowed. The capsule was endoscopically placed 4 cm above the lower esophageal sphincter, pH data were transmitted every 4s to a receiver attached to the dog's collar. Owners were instructed to press the individually predefined symptom-buttons on the receiver whenever indicated, and not to change the daily routine. Data were analysed using the RapidpH® software, reflux was defined as a single pH-measurement < 4. Results between groups were compared using non-parametric tests.The median pH-monitoring period (COD/C) was 47.49/43.17h. The following parameters (median, range for COD/C) were evaluated: Number of refluxes: 12 (1-86)/13.5 (1-65), number of longest (> 5min) reflux: 1 (0-14)/1 (0-4), duration of longest reflux (min): 7 (1-18)/6.5 (1-27), and fraction time pH < 4 (%): 0.5 (0.01-5.6)/0.5 (0-3.2). There were no differences between groups. The median number of button pushes was 6 (0-35), 3 dogs had reflux-positive pushes (2.8, 11, and 17.6% of pushes). Mild distal esophagitis was noted in 1 dog. Final diagnoses were: food-responsive IBD (6), steroid-responsive IBD (2), allergic skin disease (2), chronic laryngotracheobronchitis (2), muscular dystrophy (1).Dogs presenting with historical and clinical signs interpreted as GER may not have relevant reflux episodes. Considering normal values established in humans, none of the dogs would have been classified as abnormal.

GA-O-8
LABORATORY AND ULTRASONOGRAPHIC MONITORING OF DOGS WITH ACUTE PANCREATITIS. S. Corradini, A. Diana, F. Bresciani, M. Cipone, F. Fracassi. University of Bologna, Italy, OZZANO DELL'EMILIA, Italy

The aim of the present study was to evaluate the changes of some biochemical and ultrasonographic (US) parameters in a group of dogs with naturally occurring acute pancreatitis (AP) during the therapeutic follow-up.Dogs with clinical signs and abdominal US findings suggestive of AP associated with increased serum canine pancreatic lipase (cPL) activity were included into the study. In these dogs, the serum concentration of C-reactive protein (CRP), amylase and lipase were also measured. Severity indexes were established to semi-quantitatively evaluate the severity of clinical and US findings. In particular, a clinical score (0-3) for each of the following clinical parameters was given: presence and frequency of vomiting, appetite and general condition; an US score (0=normal, 1=abnormal) was assigned per each of the following parameters: pancreas (echogenicity, volume, echotexture and echogenicity of the mesentery), gastrointestinal tract, biliary ducts, lymph nodes and abdominal effusion (total score 0-14). All dogs were treated with fluid therapy, ampicillin-sulbactam, 15mg/kg IV q8h, buprenorphine, 0.01mg/kg q8h, and, if needed, maropitant 1mg/kg SC q24h. The two severity scores, serum CRP, amylase and lipase concentrations were measured at diagnosis (T0) and after 1 (T1), 3 (T3), and 5 (T5) days, and at discharge (Td) and 1 week after discharge (Td1).Nine client-owned dogs were included with a median (range) age of 10 years (8-14 years). Median (range) clinical and US scores were 8 (6-9) and 8.5 (3-13), respectively, at T0, and 0 (0-1) and 1 (0-3), respectively, at Td1. A significant, positive correlation was found between the clinical and US score ($p<0.001$, $r=0.68$). The median (range) serum concentration of CRP (mg/dl), amylase (U/L) and lipase (U/L) was 9.46 (3.36-30.4), 2,995 (776-6,458) and 889 (206-5,270), respectively, at T0, and 0.64 (0.01-3.07), 704 (563-1,002) and 302 (94-566), respectively, at Td1. On admission, serum CRP, amylase and lipase levels were increased in 100%, 77%, and 55% of dogs, respectively while they were increased in 50%, 12.5% and 0% of dogs, respectively, at Td1. Serum CRP and amylase, but not lipase, concentrations decreased during the follow up and were significantly ($p< 0.05$) lower at Td1 compared to T0.Results suggest that US findings, and CRP and amylase concentrations are correlated with the recovery from the AP. Further studies are warranted to evaluate the usefulness of these parameters in the follow-up of AP in a wider population of dogs.

GA-O-9A
BLINDED RANDOMIZED CONTROLLED FIELD TRIAL TO DETERMINE THE EFFECT OF ENTERIC COATING ON THE EFFICACY OF ENZYME REPLACEMENT FOR CANINE EXOCRINE PANCREATIC INSUFFICIENCY. A. Mas[1], P.J.M. Noble[1], P.J. Cripps[1], D.J. Batchelor[1], P.A. Graham[2], A.J. German[1]. [1]University of Liverpool, NESTON, United Kingdom, [2]Nationwide Laboratories, POULTON-LE-FLYDE, United Kingdom

Enzyme replacement therapy is the mainstay therapy for exocrine pancreatic insufficiency (EPI) in dogs. 'Enteric-coated' preparations have been developed to protect the enzyme from degradation in the stomach, but their efficacy has not been critically evaluated. The hypothesis of the current study was that enteric coating would have no effect on the efficacy of pancreatic enzyme supplements for dogs with EPI.Thirty-eight client-owned dogs with naturally occurring EPI were included in this multicentre, blinded, randomised controlled trial. Dogs received either an enteric-coated enzyme preparation (test group) or an identical preparation without the enteric coating (control group) over a period of 56 days.There were no significant differences in baseline characteristics between test and control treatment groups. Body weight and body condition score increased in both groups during the trial ($P<0.001$) but the magnitude of increase was greater for the test treatment compared with the control treatment ($P<0.001$). By day 56, mean body weight increase was 17% (95% confidence interval 11-23%) in the treatment group and 9% (95% confidence