Prevalence of gastrointestinal tract lesions in 73 brachycephalic dogs with upper respiratory syndrome

OBJECTIVES: To determine the prevalence of gastrointestinal tract lesions in brachycephalic dogs with upper respiratory tract disease.

METHODS: The gastrointestinal tract and respiratory disorders of 73 brachycephalic dogs presented with upper respiratory signs were evaluated. Clinical signs and endoscopic and histological anomalies of the upper digestive tract were analysed.

RESULTS: A very high prevalence of gastrointestinal tract problems in brachycephalic dogs presented with upper respiratory problems was observed clinically, endoscopically and histologically. Endoscopic anomalies of the upper digestive tract were present even in dogs without digestive clinical signs. Furthermore, histological evaluation of the digestive tract sometimes showed inflammatory lesions not macroscopically visible at endoscopy. Statistical analysis showed a relationship between the severity of the respiratory and digestive signs. This was significant in French bulldogs, males and heavy brachycephalic dogs.

CLINICAL SIGNIFICANCE: These observations show a correlation between upper respiratory and gastrointestinal tract problems in brachycephalic breeds with upper respiratory disease. Surgical treatment of respiratory disease could improve the digestive clinical signs, and/or gastro-oesophageal medical treatment could improve the outcome for surgically treated brachycephalic dogs.

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INTRODUCTION

Upper respiratory tract disease has been described in brachycephalic dogs (BDs) (Harvey 1982, Wykes 1991, Hendricks 1992, Hobson 1995, Bright and White 1997, Dupre and Freiche 2002, Koch and others 2003). Clinical signs include snoring, inspiratory dyspnoea, exercise intolerance, cyanosis and, in the most severe cases, episodes of syncope.

Dysphagia is defined as difficult or painful swallowing. It can be associated with regurgitation and vomiting (Guilford and Strombeck 1996). Many owners describe signs of dysphagia when BDs get excited or are in respiratory distress. Dysphagia in BDs has also been reported in the literature (Hendricks 1992, Hobson 1995, Bright and White 1997, Ducarouge 2002, Dupre and Freiche 2002, Koch and others 2003), but its prevalence has not been detailed. In one retrospective study conducted at the authors' referral hospital, 27 cases of upper respiratory syndrome in BDs showed a high prevalence of gastrointestinal signs, with 55·6 per cent showing frequent vomiting and 44·5 per cent dysphagia (Ducarouge 2002).

Many anatomical malformations of the gastrointestinal tract have also often been described in BDs, including hiatal hernia (Callan and others 1993), pyloric stenosis (Peeters 1991, Walter and Matthiesen 1993) or oesophageal deviation (Woods and others 1978).

The present prospective study was designed to include fibroscopic examination of the gastrointestinal (GI) tract in the evaluation of all BDs presented with upper respiratory clinical signs in one hospital. The aims of the study were to endoscopically document the prevalence of oesophageal, gastric and duodenal anomalies in BDs and their histological nature; to identify the influence of epidemiological data on the severity of gastric or respiratory clinical signs; and to establish the potential relationship between upper GI tract and upper respiratory tract disease.

MATERIALS AND METHODS

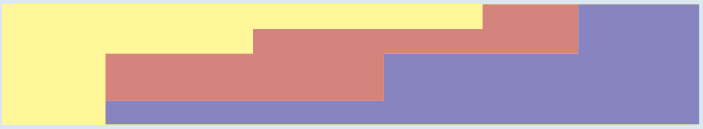
Medical records of all BDs referred to the hospital at the Clinic Fregis, France, with clinical signs of upper respiratory tract disease between November 2000 and June 2003 were reviewed. Dogs presented for GI tract problems without respiratory problems were excluded from the study. Breed, age, sex and history were recorded. A complete clinical examination was performed. The frequency of upper respiratory signs – snoring, inspiratory effort, exercise or stress intolerance, and syncope – was recorded. On the basis of the frequency of each respiratory sign, a global classification of three grades was obtained: absent or minimal (grade 1), moderate (grade 2) or severe (grade 3) (Table 1).

The frequency of gastrointestinal tract signs – ptyalism, regurgitation and vomiting – was also recorded and graded as absent or minimal (grade 1), moderate (grade 2), or severe (grade 3) (Table 2).

A statistical software package (SPSS for windows, Version 11) was used to analyse the data. Four epidemiological variables were introduced into the database: breed (three modalities: French bulldog, English bulldog and other breeds), age (units = month, three equivalent categorised modalities: low, medium, high or metric when possible), weight (units = kg, three equivalent categorised modalities: low, medium, high or metric when possible) and gender (male and female). No distinction was made regarding neutered status as only two dogs in the study had been sterilised. Two variables relating to the severity of clinical signs were introduced into the database: grade of respiratory signs and grade of gastric signs. Due to the low number of grade 1 observations for respiratory diseases (n=2), grades 1 and 2 for both variables were recorded as 'low', with grade 3 'high' being the alternative. The three grades were recorded for the digestive signs.

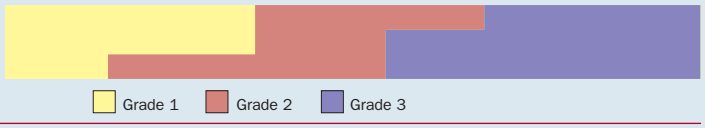
The association of the four epidemiological variables with the severity of the respiratory or digestive clinical signs was analysed using different statistical tests (Table 3). The relationship between respiratory and gastric signs severity was analysed using a Kendall rank correlation test. The association of the four epidemiological variables with this relationship was tested separately via a series of Kendall tests (Table 4). For all analyses, values of $P<0{\cdot}05$ were considered significant.

Table 1. Frequency and nature of respiratory signs and the assigned clinical grade

Nature of the respiratory signs	Frequency: Never	Occasionally (< once monthly)	Regularly (once weekly)	Daily (once daily)	Often (> once daily)	Constantly
Snoring Inspiratory efforts Stress or exercise intolerance Syncope						

The grading of the respiratory disorders is based on the frequency of different clinical signs and is comprised of three grades. Inclusion of at least one sign in a higher grade determines the actual classification. For example, if an animal was presented with regular snoring, inspiratory difficulty, stress or exercise intolerance but occasional syncope, a grade 3 for respiratory signs was assigned

Table 2. Frequency and nature of digestive signs and the assigned clinical grade

Nature of the digestive signs	Frequency: Never	Occasionally (< once monthly)	Regularly (once weekly)	Daily (once daily)	Often (> once daily)	Constantly
Ptyalism Regurgitation Vomiting						

The grading of the digestive disorders is based on the frequency of different clinical signs and is comprised of three grades. Inclusion of at least one sign in a higher grade determines the actual classification. For example, if an animal was presented with regular ptyalism and regurgitation but daily vomiting, a grade 3 for digestive signs was assigned

Clinical evaluations

The upper respiratory and GI tracts of all dogs were evaluated. Food and water were respectively withheld for 24 and 12 hours before anaesthesia. Premedication included 0·05 mg/kg intramuscular acepromazine (Calmivet; Vetoquinol), 0·2 mg/kg intramuscular dexamethasone (Dexadreson; Intervet), 0·5 mg/kg intramuscular metoclopramide (Primperid; Sanofi), 0·01 mg/kg intramuscular glycopyrrolate (Robinul; Vetoquinol). Preoxygenation was provided by an oxygen mask when needed. Following induction with 5 to 10 mg/kg intravenous thiopental (Nesdonal; Merial), the dogs were intubated and anaesthetised with isoflurane or halothane in 100 per cent oxygen. All upper respiratory examinations were performed by the same operator with the dog in sternal recumbency after the endotracheal tube had been removed.

The length and thickness of the soft palate, position of the laryngeal ventricles, and position and motion of the arytenoid cartilages were evaluated. All oesophageal and gastroduodenal fibroscopic examinations were performed by the same operator using a fibroscope (Olympus EVIS GIF-XP 160/8·6 mm) and processor (Olympus CV-100), with the animal in left lateral recumbency.

Oesophageal, gastric and duodenal anomalies were observed and classified grossly as non-inflammatory or inflam-

Table 3. Statistical analysis showing the association of epidemiological variables (gender, age, weight and breed) with the severity of respiratory or gastric clinical signs

Variables	Association with respiratory signs severity (two classes)		Association with gastric signs severity (three classes)	
	Fisher's F	P value	Fisher's F	P value
Age	0·492	0·485	0·395	0·532
Weight	0·057	0·812	**5·077**	**0·027**
	Fisher's exact test	P value	χ^2	P value
Gender	2·890	0·151	0·047	0·977
	χ^2	P value	χ^2	P value
Breed (three classes)	0·498	0·780	**9·947**	**0·041**

χ2 Chi-squared test
Statistically significant associations are shown in bold

Table 4. Statistical analysis showing the association of gender, age, weight or breed with the relationship between respiratory and gastric clinical signs

Variables	Modality	Frequency	Minimum/ maximum	Kendall's tau-b*	P value
Gender	Male	53	N/A	**0·341**	**0·006**
	Female	20	N/A	–0·090	0·670
Age	Low	24	3-16 months	0·265	0·156
	Medium	25	17-27 months	0·298	0·157
	High	24	29-85 months	0·056	0·764
Weight	Low	24	6·50-10·00 kg	0·025	0·894
	Medium	24	10·10-13·70 kg	0·132	0·516
	High	25	13·80-30·30 kg	**0·471**	**0·021**
Breed	English bulldog	13	N/A	0·468	0·146
	French bulldog	49	N/A	**0·284**	**0·034**
	Other breed	11	N/A	**–0·621**	**0·007**

N/A Not applicable
*Kendall's tau-b of the relationship between the severity of the digestive and respiratory clinical signs. The tau-b has been calculated by modality of the variable, which that shows that the relationship can depend on the modality of a variable
Statistically significant associations are shown in bold

matory. Non-inflammatory findings included: oesophageal deviation (tortuous and non-linear oesophagus); cardial atony (abnormally open cardia); gastro-oesophageal reflux (gastric juice entering the oesophagus during the inspiratory phase); axial hiatal hernia (protrusion of the stomach toward the thorax in the oesophageal lumen); gastric stasis (presence of food more than 24 hours after fasting); mucosal hyperplasia of the pylorus (thickened mucosal folds or multiple folds surrounding the pyloric lumen); pyloric stenosis (operator appreciation of narrow pyloric diameter not correlated with the animal's size or inability to pass the pylorus); pyloric atony (abnormally opened pylorus); and duodenogastric reflux (abnormal juice reflux from the duodenum to the stomach).

Inflammatory anomalies included: distal oesophagitis (erosion or ulceration of the distal oesophageal mucosa); inflammation of the corpus or the antrum (diffuse or punctiform); and diffuse inflammation of the duodenum (erythematous coloration and granular aspect of the mucosa).

In the first 33 of 73 cases in the present study, gastric and duodenal biopsies were only performed in the presence of macroscopic anomalies. Given the high prevalence of histological abnormalities, gastroduodenal biopsies were obtained systematically thereafter. No oesophageal biopsies were taken.

Biopsy samples were fixed in a 10 per cent formalin solution. Routine staining with hemalum eosin safran was used. Periodic acid Schiff and Warthin-Starry stains were occasionally used. All histological specimens were interpreted by the same operator.

Gastric biopsy samples were assessed and gastritis was graded as minimally, moderately or severely inflamed according to specific criteria: extension of the lesion in mucosal samples, type and intensity of cellular infiltrate, lamina propria and vessel alterations, and the epithelial changes. Phlogogenic agents were looked for, particularly spiral-shaped bacteria in the stomach. Follicular gastritis was diagnosed in the presence of a lymphoid nodular reaction, including follicular germinal reactivity centres. Gastritis was considered mild when, according to specific criteria, inflammation was limited to superficial subepithelial regions; the number of inflammatory cells was reduced; and collagen production was mild. By contrast, severe gastritis was diagnosed when significant inflammatory infiltrate extended to the mucosa, with fibrosis and atrophy of glands. Moderate gastritis was diagnosed when lesions were intermediate in extension.

The same criteria were applied to evaluate duodenitis, with special attention given to villus architecture and length. Inflammatory cells present at the depth of the lamina propria were also examined to evaluate cell population type and the importance of inflammatory disease. Duodenitis was also graded as mild, moderate or severe by applying the same histological criteria as were used for gastritis. Upper respiratory tract surgeries (rhinoplasty and palatoplasty) were all performed by the same surgeon, immediately after the endoscopic examination, under a single anaesthesia.

RESULTS

Seventy-three dogs met the inclusion criteria. All were purebred. The most common breed was the French bulldog, accounting for 49 of 73 cases (67 per cent). Other breeds included the English bulldog (n=13, 17·8 per cent), pug (n=5, 6·8 per cent), Pekingese (n=2, 2·8 per cent), shar pei (n=1, 1·4 per cent), lhasa apso (n=1, 1·4 per cent), boxer (n=1, 1·4 per cent) and Boston terrier (n=1, 1·4 per cent). Mean age was 30·5 months (median 20 months, range three months to seven years). Seventy-one dogs (97·3 per cent) were intact. Males were over-represented (53 of 73 cases, 72·6 per cent). The chi-squared test showed that there were significantly more males (P=0·042).

Table 5. Distribution of the endoscopic anomalies in 73 dogs

	Oesophagus (n=73)	Stomach (n=73)	Duodenum (n=66)
Non-inflammatory anomalies	Oesophageal deviation 12 (16·4%) Hiatal hernia 3 (4·1%) Cardial atony 28 (38·4%) Gastro-oesophageal reflux 23 (31·5%)	Gastric stasis 23 (31·5%) Pyloric mucosal hyperplasia 63 (86·3%) Pyloric stenosis 22 (30·1%) Pyloric atony 4 (5·4%) Duodenogastric reflux 6 (8·2%)	None
Inflammatory anomalies	Distal oesophagitis 27 (37%)	Diffuse inflammation 65 (89%) Punctiform inflammation 28 (38·4%)	Diffuse inflammation 35 (53%)

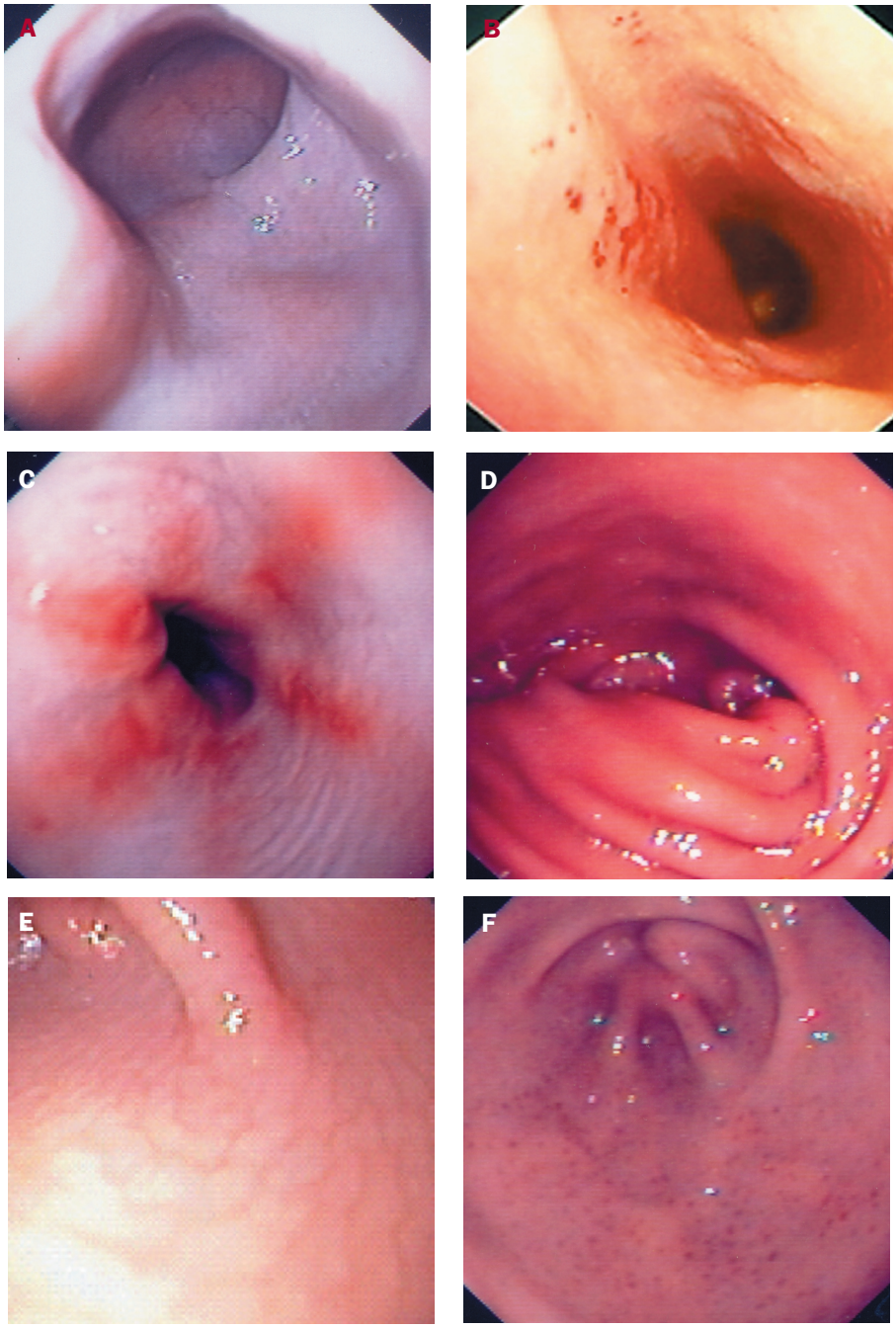


FIG 1. Illustrations of oesophageal and gastroduodenal fibroscopic examinations. (A) Oesophageal deviation: the oesophagus is abnormally long and tortuous. (B) Severe distal oesophagitis: there is congestion and severe erosion of the distal oesophageal mucosa. (C) Gastro-oesophageal anomalies: erosive lesion of the distal oesophageal mucosa with cardial incontinence. A weak cardial tonicity and gastric reflux was noted during the examination. (D) Mucosal hyperplasia of the pylorus: the pyloric lumen is hidden in multiple mucosal folds. (E) Gastric inflammation: the gastric folds have a congested and heterogeneous coloration. These lesions suggest a chronic gastritis. (F) Gastric inflammation: there are multiple punctiform and purplish lesions of the gastric mucosa, suggesting a follicular gastritis

Respiratory signs were classified as grade 1 in two dogs (2·7 per cent), grade 2 in 20 dogs (27·4 per cent) and grade 3 in 51 dogs (69·9 per cent). Nineteen dogs (26 per cent) were presented with grade 1 GI tract disorders, 19 (26 per cent) with grade 2, and 35 (48 per cent) with grade 3. Among the 35 dogs with grade 3 gastro-oesophageal signs, 28 (80 per cent) exhibited grade 3 respiratory disorders, five (14·3 per cent) grade 2 and (5·7 per cent) grade 1.

Statistical analysis showed that weight (heavy BDs) and breed (English bulldog) were significantly associated with the severity of gastric signs (Table 3), and that there was a significant relationship between severity of digestive and respiratory tract clinical signs in French bulldogs, heavy BDs and males (Table 4).

Clinical and endoscopic examination of the upper respiratory tract revealed anomalies in all 73 patients: stenotic nares (n=62, 84·9 per cent), soft palate hyperplasia (n=70, 95·9 per cent), ventricular eversion (n=40, 54·8 per cent), laryngeal collapse (n=51, 69·9 per cent) and amygdalitis (n=15, 20·6 per cent). Among 13 cervicothoracic x-rays, a hypoplastic trachea was evident in six dogs (8·2 per cent). There were five English bulldogs and one French bulldog.

Overall, 71 of 73 dogs (97·3 per cent) showed oesophageal, gastric or duodenal anomalies: 44 cases (60·3 per cent) had one or more oesophageal anomalies; 71 cases (97·3 per cent) had one or more gastric anomalies. Endoscopic visualisation of the duodenum was impossible in seven of 73 dogs (9·6 per cent), because the endoscope could not pass the stenotic pylorus. Among the 66 other cases, 35 (53 per cent) had endoscopic evidence of diffuse inflammation of the duodenum.

Detailed results of endoscopic examination of the GI tract are given in Table 5 and Fig 1.

Histological specimens were available in 51 of 73 cases (69·9 per cent, 15 from the first 33 cases of the study and 36 from the 40 other cases). A chronic diffuse or

Table 6. Microscopic evaluation of 50 dogs with chronic gastritis

Results	n
Depth of the affected mucosa	
Superficial	22
Superficial and deep	28
Type of cellular infiltrate	
Polynuclear (neutrophils, eosinophils)	0
Mononuclear (lymphocytes, plasmacytes, macrophages)	42
Mixed	8
Lamina propria and vessels alterations	
Congestion	25
Glandular atrophy	13
Neovascularisation	0
Neomuscularisation	3
Fibrosis	46
Follicular proliferation	19
Epithelial alterations	
Atrophy	4
Necrosis, erosion, ulceration	1
Epithelial proliferation	5
Intestinal or pseudopyloric metaplasia	0
Presence of phlogogen agents	
Helicobacter species	21
Others	0
Severity of the lesions	
Low	13
Moderate	25
Severe	12

follicular gastritis was found in 50 of 51 specimens (98 per cent). Gastritis was histologically graded as mild in 13 of 51 specimens (25·5 per cent, respectively three of 15 and 10 of 36), moderate in 25 of 51 specimens (49 per cent, respectively seven of 15 and 18 of 36) and severe in 12 of 51 (23·5 per cent, respectively four of 15 and eight of 36) (Table 6 and Fig 2). Among 43 duodenal biopsies, a lymphoplasmocytic duodenitis was evident in 42 cases (97·7 per cent), 13 of which were graded as minimal (30·9 per cent), 23 as moderate (54·8 per cent) and six as severe (14·3 per cent) (Fig 2).

DISCUSSION

Although GI clinical signs have already been described in BDs presented for upper respiratory tract problems (Hendricks 1992, Hobson 1995, Bright and White 1997, Ducarouge 2002, Dupre and Freiche 2002), this is, to the authors' knowledge, the first clinical study specifically describing their prevalence and severity. The authors also believe this to be the first time that endoscopical and histological GI tract anomalies have been systematically studied in these dogs.

Findings showed that three-quarters (74 per cent) of the BDs presented for respiratory problems had grade 2 or grade 3 GI clinical signs. Furthermore, among the 35 dogs with grade 3 GI signs, a large majority (80 per cent) had grade 3 respiratory disorders.

In the present study, a correlation was noted between the severity of digestive and respiratory clinical signs (P=0·065). This relationship can be statistically affirmed in French bulldogs, males and heavy dogs (Table 4). A larger study may have underlined this correlation for all brachycephalic breeds. Nevertheless, it can now be postulated that, in BDs, the severity of respiratory signs influences the severity of digestive signs, and vice versa.

The fibroscopic examination revealed that all animals had upper respiratory anomalies and almost all of them (97·3 per cent) had oesophageal, gastric or duodenal anomalies.

Histological evaluation of the digestive tract revealed inflammatory lesions even in animals that showed no macroscopic lesions on endoscopy.

In this study, the owners of two dogs had never observed GI clinical signs. However, both dogs revealed anomalies of the GI tract during the endoscopic examination, and chronic gastritis on histological examination. From these findings it can be postulated that no dog with respiratory problems is exempt from associated GI tract anomalies.

The findings concerning the upper respiratory system were consistent with other studies (Bright and White 1997, Ducarouge 2002). Stenotic nares and soft palate hyperplasia were the two most common anomalies, present in 62 (85 per cent) and 70 (96 per cent) of 73 cases, respectively. The authors postulate that there are primary lesions due to the relative cutaneous and mucosal hyperplasia encountered in BDs (Ducarouge 2002, Koch and others 2003). According to this assumption, laryngeal collapse, ventricular eversion and amygdalitis are likely to be secondary events induced by high inspira-

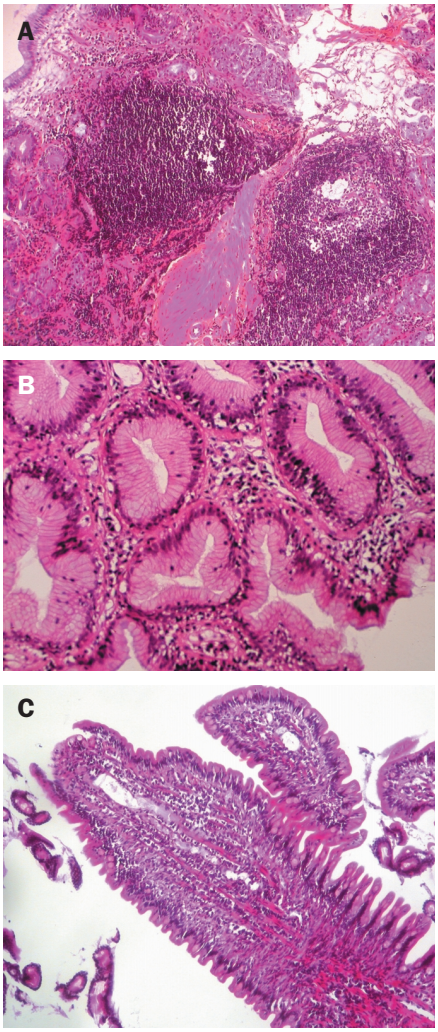


FIG 2. Histological specimens. (A) Follicular gastritis. Photomicrograph of gastric mucosa (fundic) showing two foci of follicular lymphoid hyperplasia with one germinal centre. Hemalum eosin safran (HES). ×10. (B) Pyloric hyperplastic gastritis. Photomicrograph showing hyperplastic epithelium and crypts. Diffuse infiltration of lamina propria by mononuclear cells. HES. ×10. (C) Moderate duodenitis. Photomicrograph of duodenum. Note infiltration of lamina propria of villi by mononuclear cells. HES. ×10

tory depression and chronic inflammation of the pharyngeal area (Ducarouge 2002, Dupre and Freiche 2002).

This study did not permit evaluation of the prevalence of hypoplastic trachea and its association with respiratory insufficiency in BDs, because thoracic radiographs were taken only when there was a history of pneumonia or an occasional cough was reported by the owners. Hypoplastic trachea had been observed in six dogs among 13 radiographed. This result is likely to be an underestimate.

Oesophageal deviation has been previously described in English bulldogs presented with both GI and digestive problems (Woods and others 1978). In the present study it was found in eight of 49 French bulldogs, and in four of 13 English bulldogs (Fig 1A). English bulldogs could be over-represented for oesophageal deviation (Woods and others 1978). No association was found between this anomaly and the severity of the GI signs; among the 12 affected dogs, only two showed a distal oesophagitis and gastro-oesophageal reflux. Nevertheless, as described by Woods and others (1978), the oesophageal deviation can promote retention of saliva and food and can partly explain hypersalivation when an animal is excited. Oesophageal deviation may be associated with shortening of the chest.

The distal oesophagitis (27 cases) was often associated with cardial atony (28 cases) and gastro-oesophageal reflux during the inspiratory phase (23 cases). Although reflux can be influenced by anaesthesia (Roush and others 1990, Galatos and Raptopoulos 1995a,b), the frequent presence of an installed distal oesophagitis seen during the fibroscopic examination suggests a chronic gastro-oesophageal reflux. Besides, in the authors' experience, gastro-oesophageal reflux seldom occurs during endoscopic examination of non-BDs.

Chronic vomiting, slow gastric emptying and hiatal hernias have been classically described to explain gastro-oesophageal reflux (Washabau 2000). In brachycephalic breeds, a possible explanation is the high positive abdominal pressure generated by recurrent vomiting, as well as the negative intrathoracic pressures generated by increased inspiratory effort (Miles and others 1988, Burnie and others 1989, Hardie and others 1998, Hall 2000, Ducarouge 2002, Hunt and others 2002 Sivacolundhu and others 2002). Chronic aerophagia is an additional contributor to increased intragastric pressure (Ducarouge 2002).

Axial hiatal hernia was found in three French bulldogs. Although a congenital form of hiatal hernia has been previously described in the Shar Pei, the English bulldog and the chow chow (Callan and others 1993, Hall 2000), forms found in the three French bulldogs seemed to have an acquired origin relating to positive abdominal and low intra-oesophageal pressures (these three dogs were ranked as grade 3 for both respiratory and digestive disorders).

Finally gastro-oesophageal reflux, whether or not associated with regurgitation and vomiting, can contribute to upper oesophageal, pharyngeal and laryngeal inflammation. These phenomena have been documented experimentally in animals (White and others 2002) and clinically in human infants (Halstead 1999). They can further contribute to upper respiratory problems.

Pyloric mucosal hyperplasia was fibroscopically diagnosed in 63 cases (86·3 per cent), 22 of which showed concomitant pyloric stenosis. A congenital form of pyloric stenosis has been described in boxers and Boston terriers (Hall 2000). The hypertrophy of the pyloric muscular layer would be responsible for gastric stasis and mucosal hyperplasia would occur secondarily to inflammation (Guilford and Strombeck 1996). An acquired form of pyloric stenosis is usually described but its aetiology remains unclear. Many hypotheses have been proposed (Walter and others 1985, Peeters 1991, Leib and others 1993, Walter and Matthiesen 1993, Guilford and Strombeck 1996, Sullivan and Yool 1998, Hall 2000, Ducarouge 2002). The respiratory distress could stimulate the autonomous sympathetic nervous system, which in turn would slow gastric motility and increase the gastric emptying time. Furthermore, the dilated antrum would stimulate gastrin-producing cells responsible for muscular hyperplasia (Peeters 1991, Guilford and Strombeck 1996).

In the present study, only four dogs were less than six months old at the time of endoscopic examination (one three-month-old English bulldog, one five-and-a-half-month-old French bulldog, one five-month-old English bulldog and one 5·8-month-old Shar Pei). All four presented with grade 3 respiratory and digestive signs. In these, the authors observed hypertrophic gastropathy with mucosal hyperplasia and pyloric stenosis likely to be congenital in origin. For most other cases of pyloric mucosal hyperplasia, it is not known whether it was present since birth or if it was due to chronic inflammation and respiratory depression.

GI biopsies were performed on the fundus, the pylorus and the duodenum. Most were characterised by a lymphoplasmocytic infiltrate (Table 6). Since biopsies had been endoscopically performed, no information could be obtained on the muscular layer and it was unknown whether this mucosal inflammation was associated with muscular hypertrophy. No oesophageal sample was taken because of the difficulty of obtaining good quality biopsies from oesophageal mucosa. A follicular gastritis, histologically diagnosed in 19 of 51 dogs, was the most common type of inflammation recorded. Duodenal biopsies were performed in 43 dogs. Almost all samples (42 of 43) showed minimal to severe duodenitis. Endoscopic signs of duodenitis were present only in 35 cases of 66 duodenal examinations. The histological evaluation revealed chronic inflammation, which in many cases did not show any macroscopic anomalies. The same findings applied to the stomach.

Several explanations are plausible. The results of a macroscopic endoscopic exam-

ination may not correlate well with histological results, especially if no severe signs of inflammation are visible (active congestion, exudation) – mild infiltration of mononuclear cells has no macroscopic expression. Also BDs may present with mild digestive inflammation even without clinical respiratory or digestive signs. Therefore, it would be interesting to document the endoscopic and histological aspects of the GI tract in healthy BDs. It would require the compliance of owners or breeders who do not favour anaesthesia in these dogs.

The correlation between respiratory and GI disorders suggests the influence of upper respiratory tract diseases on gastro-oesophageal diseases and vice versa. It supports the common pathophysiological pathway (Dupre and Freiche 2002): the gastro-oesophageal disorders – ptyalism, regurgitation, vomiting and reflux – can aggravate respiratory signs by encumbering the pharyngeal region and stimulating persistent inflammation. Conversely, the chronic respiratory depression promotes gastro-oesophageal reflux. This close relationship between respiratory and digestive problems is sustained by the fact that most of these animals 'vomit' large amounts of saliva when excited, stressed or in respiratory distress.

Conclusions

This study shows that clinical signs of GI dysfunction are frequent in BDs suffering signs of upper respiratory tract disease. Endoscopic lesions of the GI tract are seen even in apparently normal animals. Pathological changes seem even more severe than expected. The results indicate that clinical signs alone cannot be relied upon to diagnose digestive problems in BDs. Both endoscopic and histological examinations are necessary to qualify and grade GI disorders.

Such early and exhaustive diagnosis of GI problems allows the prescription of treatments that could slow the progression of the BD's syndrome. A further study is in progress to find out if surgical treatment of respiratory disease improves GI clinical signs or if treating the gastro-oesophageal signs medically improves the outcome for surgically treated BDs.

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