Digestive disorders associated with the chronic obstructive respiratory syndrome of brachycephalic dogs: 30 cases (1999-2001)

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SUMMARY

This study describes the functional or organic abnormalities of the upper digestive tract observed in Brachycephalic dogs suffering from chronic obstructive respiratory syndrome (CORS) and digestive disorders. The population that was studied includes thirty dogs (22 males, 8 females), 28 months of age on the average, belonging to 4 different breeds (22 French Bulldogs, 5 English Bulldogs, 2 Pugs, 1 Pekingese). Endoscopic examination of the upper respiratory airways confirmed the existence of anatomical abnormalities responsible for CORS in the 30 dogs of the study. Among these abnormalities, an hypertrophy and excessive elongation of the soft palate were constant in all dogs, associated with an eversion of the laryngeal ventricles in 23 cases, nostrils stenosis in 21 cases, laryngeal collapse (11 cases) and tracheal hypoplasia in 5 cases. When examining their upper digestive tract endoscopically, 13 dogs showed a reflux esophagitis at stage I, 9 at stage II, 2 at stage III and 1 at stage IV compounded by a stenosis of the esophagus in 1 case. Sixteen cases of hiatal hernia by sliding were observed. In 13 cases, gastrointestinal examination showed an abnormal hyperplasia of the antral mucosa clearly associated in 10 cases with a duodenogastric reflux. This hypertrophic antropathy was compounded in 6 dogs of the French Bulldog breed by a pyloric hypertrophic stenosis. Five dogs showed no functional or organic abnormalities of the upper digestive tract. Corrective surgery on the respiratory airways was practiced in 20 dogs. This surgery on the respiratory airways was complemented with pyloric surgery (pyloroplasty) in the 6 dogs that showed pyloric stenosis. Nineteen dogs that had surgery on their upper respiratory airways showed marked clinical respiratory and digestive improvement associated with marked improvement in the digestive abnormalities confirmed by a control endoscopy.

KEY-WORDS: Brachycephalic - esophagitis - hiatal hernia - obstructive respiratory syndrome.

RÉSUMÉ


Cette étude décrit les anomalies fonctionnelles ou organiques du tractus digestif supérieur observées chez des chiens Brachycéphales souffrant de syndrome respiratoire obstructif chronique (SROC) et de troubles digestifs. La population étudiée comprend trente chiens (22 mâles, 8 femelles), d’âge moyen 28 mois, appartenant à 4 races différentes (22 Bouledogues Français, 5 Bouledogue Anglais, 2 Carlin, 1 Pékinois). L’examen endoscopique des voies respiratoires supérieures a confirmé chez les 30 chiens de l’étude, l’existence d’anomalies anatomiques responsables du SRO. Parmi ces anomalies, une hypertrophie et un allongement excessif du voile du palais étaient constant chez tous les chiens, associés à une éversion des ventricules laryngés dans 23 cas, une sténose des narines dans 21 cas, un collapsus laryngé (11 cas) et une hypoplasie trachéale dans 5 cas. A l’examen endoscopique du tractus digestif supérieur, 13 chiens ont montré une oesophagite de reflux de stade I, 9 de stade II, 2 de stade III et 1 de stade IV compliqué de sténose de l’oesophage dans 1 cas. Seize cas de hernie hiatale par glissement ont été observés. Dans 13 cas, l’examen gastroscopique montrait une hyperplasie anormale de la muqueuse antrale associée nettement dans 10 cas à un reflux duodénogastrique. Cette antropathie hypertrophique était compliquée chez 6 chiens de race Bouledogue français d’une sténose hypertrophique pylorique. Cinq chiens ne présentaient pas d’anomalies fonctionnelles ou organiques du tractus digestif supérieur. Une chirurgie corrective des voies respiratoires a été pratiquée chez 20 chiens. Cette chirurgie des voies respiratoires a été complétée d’une chirurgie pylorique (pyloroplasty) chez les 6 chiens qui présentaient une sténose pylorique. Dix-neuf chiens opérés des voies respiratoires supérieures ont montré une amélioration clinique respiratoire et digestive associée à une amélioration des anomalies digestives confirmée par une endoscopie de contrôle.

MOTS-CLÉS: Brachycephale - oesophagite - hernie hiatale - syndrome respiratoire obstructif.
The chronic obstructive respiratory syndrome (CORS) of brachycephalic breeds is well described in many publications [8]. These respiratory difficulties are the direct consequence of their anatomical conformation: short and broad head, face shorter than the skull, stenosed nostrils, often narrow and winding nasal cavities with a proportionately wider pharynx to fit an hypertrophied soft palate, an often narrow larynx and, in some cases, tracheal hypoplasia [4, 8].

In the very same breeds, it is not uncommon to observe deglutition disorders, regurgitation or vomiting which may result in false deglutition and sudden respiratory distress [2, 10, 11, 13, 18]. The deglutition of air in large quantity promotes chronic distension of the digestive tract and the appearance of flatulence. Finally, congenital or acquired pyloric hypertrophic gastropathy is often described in brachycephalic breeds and is responsible for the appearance of a gastric retention syndrome («Acquired Antral Pyloric Hypertrophy Syndrome» AAPHS) [9, 13, 14, 18, 19].

While the digestive and respiratory syndromes observed in these breeds are well known to clinicians and often described in the literature, as far as we know, there is no systematic assessment of digestive affections in individuals suffering from obstructive respiratory syndrome. In the dog, a relationship between an obstructive pathology of the respiratory tract and a hiatal hernia or a gastroesophageal reflux (GER) was suggested by PEARSON [16] who described a GER case secondary to an intranasal tumor. ELLISON [5] reported two cases of hiatal hernias associated with laryngeal paralysis. Later, a series of 16 cases associating obstructive respiratory disorders and digestive disorders was published by BRIGHT [2].

The purpose of this study is to determine and assess the lesions and digestive functional disorders which may appear in a brachycephalic suffering from CORS, and determine how these respiratory abnormalities may have digestive functional consequences. Finally, it was important to observe the evolution of these digestive disorders after surgery on the respiratory airways and to determine the therapeutic attitude faced with such affections.

## Equipment and methods

Our study was concerned with 30 brachycephalic dogs brought for consultation between January 1999 and December 2001 for CORS at different evolutionary stages associated or not with digestive syndromes.

All these dogs were assessed by endoscopy at respiratory level in order to precisely determine the anatomical abnormalities responsible for the appearance of this CORS. An upper digestive endoscopy complemented the endoscopy of the respiratory airways in order to determine the functional or organic abnormalities of the upper digestive tract associated with this CORS.

Endoscopic examinations were carried out for every animal according to the same protocol and with identical equipment. This equipment included a videogastroscope (9 mm in diameter) (Olympus GIF XQ140 type), a xenon light source. Examinations were recorded on DigitalVideo cassettes (DV GV-D 300E PAL Sony video recorder) or photographed (VP 1850 EPM Sony color video printer).

To examine the choanae and nasopharynx, a flexible small-diameter fiberscope was used, of the bronchoscope type (Broncho Schott Fiber Optics veterinary VFS-10), 4 mm in diameter.

Patients were tranquilized with a solution of acepromazine to a dosage of 0.1mg/kg as an intramuscular injection (Calmivet ND), then anesthetized by an intravenous injection of ketamin (Imalgene 500ND) to a dosage of 10mg/kg. All animals were first explored at the level of the upper respiratory airways, then intubated before digestive endoscopic examination and a volatile anesthesia was established (halothane, Halothane Belamont ND or isoflurane, Forene ND).

Cardiac and respiratory monitoring were also put in place on animals showing major respiratory difficulties.

For every animal, the anatomical abnormalities of the upper respiratory airways were assessed and noted (nostrils stenosis, elongation and/or thickening of the soft palate, hypertrophy of tonsils, eversion of laryngeal ventricles, laryngeal collapse and tracheal hypoplasia, sagging of the nasopharynx and stenosis of choanae). An assessment of esophagitis lesions was carried out according to the following classification: Stage I showed a more or less marked erythema as a macula or at the top of the folds of the distal thoracic esophagus displaying a characteristic radial image, Stage II corresponded to a worsening of this erythema and to the appearance of superficial erosions which were not converging and not circular, Stage III was characterized by ulcers which could result in Stage IV, characterized by the confluence and circular aspect of such ulcerations. Stage IV could be compounded by cicatricial stenosis. The presence of an abnormally gaping cardia or a hiatal hernia, of a gastroesophageal reflux was noted. During the endoscopy of the stomach, the inflammatory condition of the gastric mucosa, the possible presence of substance loss, an abnormal hypertrophy of the antpyloric mucosa were specified.

Corrective surgery on the upper respiratory airways was proposed in all cases. It associated a rhinoplasty in cases of nostrils stenosis, a partial resection of the soft palate when it was elongated (palatoplasty), an exeresis of laryngeal ventricles in case of significant eversion of these ventricles, a tonsillectomy if an hypertrophy was noted.

All animals in this study were controlled again 3 months after surgery or implementation of the medical treatment.

## Results

### POPULATION STUDIED

The population that was studied included thirty dogs (22 males, 8 females) 28 months of age on the average, belonging to 4 different breeds (22 French Bulldogs, 5 English Bulldogs, 2 Pugs, 1 Pekingese).

All these dogs presented a chronic respiratory obstructive syndrome. Anamnesis allows to show that all these animals had frequently presented digestive symptoms associating
hypersalivation, dysphagia, regurgitation, vomiting. At the time of examinations, 6 dogs showed a gastric retention syndrome. Finally, one should noted that all these dogs suffered from aerophagy.

RESULTS OF ENDOSCOPY OF THE UPPER RESPIRATORY AIRWAYS

Endoscopic examination of the upper respiratory airways confirmed the existence of anatomical abnormalities responsible for CORS in the 30 dogs of the study. Among these abnormalities, an hypertrophy and excessive elongation of the soft palate was constant in all dogs, associated with an eversion of the laryngeal ventricles in 23 cases, nostril stenosis in 21 cases, a laryngeal hypoplasia (11 cases) and a tracheal hypoplasia in 5 cases, especially severe in a dog of the English Bulldog breed. In addition, the retroversion of the soft palate and the examination of the nasopharynx confirmed a substantial thickening of the soft palate resulting in an obstruction of the nasopharynx in 21 cases, a stenosis of choanae in 3 cases. Tonsillitis was constant in all cases.

RESULTS OF ENDOSCOPY OF THE UPPER DIGESTIVE AIRWAYS

Twenty-four dogs in the study showed an hyperplasia of the esophageal mucosa which presented abnormally hypertrophied longitudinal folds, especially in the thoracic distal part of the esophagus. Thirteen dogs showed a reflux esophagitis at stage I, 9 at stage II, 2 at stage III and 1 at stage IV compounded by a stenosis of the esophagus. 5 dogs presented no esophagitis lesions detectable by an endoscopic examination. Sixteen cases of hiatal hernia by sliding or a defective cardiotuberositary position were observed. In 21 cases, the fundic mucosa showed a typical aspect of follicular gastritis. The histology performed in 5 cases confirmed an hyperplasia of lymphoid follicles and an interstitial inflammatory infiltrate. In 13 cases, gastroscope examination showed an abnormal hyperplasia of the antral mucosa. This antropathy was compounded in 6 dogs of the French Bulldog breed by a pyloric hypertrophic stenosis. These 6 Bulldogs were under 2 years of age (6 months to 2 years). In the ten other cases of pyloric hypertrophic stenosis, the pyloroplasty was performed in 2 cases, a pyloroplasty in 2 cases and a pyloric stenosis. The 6 dogs that had surgery on their upper respiratory airways and a pyloroplasty quickly showed a satisfactory clinical improvement with the disappearance of vomiting and a very moderate persistence of signs of gastroesophageal reflux. The other dogs that had surgery on their respiratory airways and a pyloroplasty quickly showed a satisfactory clinical improvement with the disappearance of vomiting and a very moderate persistence of signs of gastroesophageal reflux. The other dogs that had surgery only on their upper respiratory airways also showed a marked improvement in digestive disorders except for a dog of the English Bulldog breed that presented a persistence of snoring but no respiratory discomfort any more. No false deglutition was observed, even in dogs having undergone a very substantial ablation of the soft palate. Out of the 20 dogs that were operated on, 12 were controlled again by endoscopy 6 months after the surgical act. Six dogs showed no inflammatory lesions of the esophagus any more. However, 2 dogs still had an abnormally gaping cardia associated with a stage I esophagitis, 3 dogs of the English Bulldog breed
showed a persistence of their hiatal hernia by sliding, yet without any severe esophagitis and with a very limited protrusion of the stomach into the diaphragmatic hiatus. 1 English Bulldog still showed a severe esophagitis associated with its hiatal hernia. Furthermore, the hyperplasia of the antropyloric mucosa was markedly reduced in all cases, and so was the frequency of the duodenogastroduodenal reflux.

In the 6 dogs that presented significant abnormalities of the upper respiratory airways justifying corrective surgery that was not performed, the medical treatment allowed an improvement in the digestive clinical signs, but a systematic recurrence of symptoms was noted as soon as treatments were stopped.

Discussion

The obstructive respiratory syndrome of brachycephalic breeds (ORS) is well described in many publications [4, 8]. Brachycephalics (Pekingese, English Bulldog, French Bulldog, Boxer, Lhassa Apso, Shih Tzu, Pug...) have a short and broad head with a face shorter than the skull which is convex in all directions. Their stop is clearly marked and their nasal area is centrally depressed. The head shape results from a defect in the growth of the basicranial epiphysyal cartilage. Nostrils are often poorly conformed or even stenosed, nasal cavities are often narrow and winding. The pharynx, proportionately wider than long, fits an abnormally developed soft palate. This soft palate is generally very elongated and thickened and may go up to the point of occluding the glottis when breathing in [8]. The larynx is sometimes narrow, especially in English Bulldogs that also seem to be predisposed to tracheal hypoplasia. These abnormalities of the primary respiratory airways severely aggravate the obstructive respiratory syndrome [4].

In the very same brachycephalic breeds, deglutition disorders, regurgitation or vomiting which can induce false deglutition and sudden respiratory distress may be observed [8, 10, 13]. The deglutition of air in large quantity promotes a chronic distension of the digestive tract and the appearance of flatulence [8]. Finally, pyloric hypertrophic stenosis is often described in brachycephalic breeds and is clinically responsible for the appearance of a gastric retention syndrome («Acquired Antral Pyloric Hypertrophy Syndrome») [9, 11, 14, 17].

While the digestive and respiratory syndromes such as those observed in these breeds are well known to clinicians and often described in the literature, as far as we know, there is no systematic assessment of digestive affections in individuals suffering from obstructive respiratory syndrome.

This study made it possible to show a high prevalence of digestive functional or anatomical abnormalities in dogs suffering from ORS (only 5 dogs in the serie presented no visible abnormalities in an endoscopy of the upper digestive tract). Among these affections, esophagitis were the most frequent ones (25/30 dogs, i.e. 83 %). Inflammatory lesions were chiefly located in the distal third of the thoracic esophagus, but could extend to the whole thoracic esophagus (2 cases). They could result in a mere erythema whose radial layout centered on the cardia is characteristic of lesions induced by a gastroesophageal reflux. Erosions, ulcerations are the result of a worsening of inflammation and may induce secondarily a cicatrical stenosis observed in 1 case. This peptic esophagitis was associated in 16 cases with a hiatal hernia by sliding, generating and aggravating the frequency of the gastroesophageal reflux. In 5 of the 16 cases, endoscopic examination showed very clearly the gastric mucosa rolling onto the diaphragmatic ring during respiratory motions. These cases were observed in dogs of the English Bulldog breed. In the 11 other cases, a small collar of gastric mucosa was observed between the esogastric epithelial junction and the diaphragm neck during the two stages of breathing, often associated with an abnormally gaping cardia. Furthermore, it is remarkable that all these dogs presented a substantial thickening of the esophageal mucosa showing longitudinal folds which were sometimes very protuberant and hampered the progress of the endoscope. A priori, this hyperplasia has no explanation.

The most severe digestive disorders associating in particular a hiatal hernia and an ulcerated esophagitis were observed in this series in dogs of the English Bulldog breed that presented a very important obstructive respiratory syndrome. The high prevalence of hiatal hernias and of their consequences in the English Bulldog was also reported in other series [2, 5, 15]. However, these hiatal hernias were described in other breeds with no obstructive respiratory syndrome [1, 5].

In the dog, a relationship between an obstructive pathology of the respiratory tract and a hiatal hernia or a gastroesophageal reflux (GER) has already been suggested. Thus, PEARSON [16] mentioned a GER case secondary to an obstruction of nasal cavities by a tumor. ELLISON [5] reported two cases of hiatal hernias associated with laryngeal paralysis. Later, BRIGHT described several cases of hiatal hernias associated with obstructive respiratory disorders [2].

Richard P. HACKETT et al. [6] showed that for the racehorse, when exercising, the decrease in resistance in the extrathoracic airways resulted in a sharp decrease in intrathoracic pressures and esophageal intraluminal pressures. This study shows that the esophageal intraluminal pressure of the horse, when it breathes in and out, depends on the degree of obstruction of the upper airways. A recent study in man [3] has shown a higher prevalence of hiatal hernias in asthmatic patients vs. nonasthmatic patients and the existence of a pathological gastroesophageal reflux in 81 % of asthmatics with a hiatal hernia and in 65 % of asthmatics with no hiatal hernia. Furthermore, this study showed an incontinence of the caudal esophageal sphincter in 35 % of asthmatics with or without hiatal hernia.

For these authors, the violent inspiratory efforts observed in these patients increase the intrapleural pressure, thus creating a considerable depression. An effect of aspiration of the digestive structures and of their content results from this. This phenomenon generates regurgitation, vomiting and burning sensations of the lower esophagus in direct relation with GER [3].
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In conclusion, an abnormal inspiratory effort could induce an abnormally low negative intrathoracic pressure. This negative pressure could be sufficient to worsen or even induce a hiatal hernia and/or GER.

The gastric lesions observed in certain dogs of this series contribute to the worsening of GER and of the peptic esophagitis. An hypertrophic antropathy was observed in 13 cases of our study (43 %), a pyloric hypertrophic stenosis in 6 cases, exclusively in dogs of the French Bulldog breed. These different abnormalities modify and delay gastric emptying, leading to a significant increase in the intragastric pressure. This intragastric pressure increase considerably promotes the risks of gastroesophageal reflux by opening of the caudal esophageal sphincter. In the current state of our knowledge, it is difficult to explain the relationship between these gastric or pyloric lesions and ORS. It is certain that dogs suffering from ORS swallow air in large quantity, resulting in a chronic dilatation of the stomach clearly observed in 16 cases of our study. This chronic air dilatation may lead to an increase in the intragastric pressure, thus mimicking a meal. The secretion of gastrin and gastric acid is then stimulated despite the absence of an effective meal. The production of mediators (cholecystokinin, secretin) is then stimulated, leading by their trophic effect to an hyperplasia of the antral and pyloric mucosa and to the appearance of an hypertrophic antropathy. Another explanation could also be suggested in the pathogenesis of this antropathy in these dogs suffering from ORS. Indeed, in 10 cases out of 13 cases of hypertrophic antropathy, a substantial duodenogastric reflux was observed. Highly corrosive bilio-pancreatic secretions could be at the origin of this hyperplasia, similarly to what has been described in man in reflux antritis. However, it is interesting to note that 3 dogs showed a duodenogastric reflux with no hyperplasia of the antral mucosa. This hyperplasia of the antropyloric mucosa certainly has multiple factors.

In the cases of pyloric hypertrophic stenosis observed in our series exclusively in dogs of the French Bulldog breed, it is difficult to state that the respiratory abnormalities these dogs were suffering from contributed to the progress of this affection well described in this breed [13, 14, 17, 18]. For several authors, an antropyloric coordination defect resulting from a congenital functional abnormality of myenteric plexuses could be the cause of this affection leading gradually and more or less quickly to an hypertrophy of the smooth muscle layer of the pyloric channel [9]. Secretory neuronal immaturity has also been suspected, even though the existence of an hypergastrinemia in these situations has not been proven [9]. However, there is no doubt that the gas-
tric hyperpressure induced by the significant deglutition of air observed in these dogs is involved, as we have explained above, in this hyperplasic phenomenon in the antropyloric area and can but aggravate a preexisting abnormality.

Consequently, it seems that complex mechanisms can appear in an ORS and could play a major role in the appearance of the functional disorders and organic affections of the digestive tract observed in this study. It is interesting to note in dogs that had surgery on their upper respiratory airways that the digestive symptoms markedly receded and did not reappear even though they were recurrent in individuals without surgery. Moreover, this disappearance of digestive symptoms was associated with a disappearance of esophagitis lesions in most cases, with a functional improvement in the esophagogastric junction and with a decrease in the hyperplasic aspect of the antropyloric mucosa.

Even though it is difficult to explain these mechanisms and the interrelation between ORS and digestive disorders, this study deserves the credit for showing the high prevalence of these diseases of the upper digestive tract in dogs suffering from ORS and the need not only to deal with the abnormalities of the upper respiratory airways in these animals, but above all not to neglect treating the lesions of the digestive tract associated with ORS.

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References