

Evaluation of respiratory parameters at presentation as clinical indicators of the respiratory localization in dogs and cats with respiratory distress

Nadja E. Sigrist, Dr med vet, DACVECC; Katja N. Adamik, Dr med vet; Marcus G. Doherr, Dr med vet, PhD, DECVPH and David E. Spreng, Dr med vet, DECVS, DACVECC

Abstract

Objective – To describe clinical respiratory parameters in cats and dogs with respiratory distress and identify associations between respiratory signs at presentation and localization of the disease with particular evaluation between the synchrony of abdominal and chest wall movements as a clinical indicators for pleural space disease.

Design – Prospective observational clinical study.

Setting – Emergency service in a university veterinary teaching hospital.

Animals – Cats and dogs with respiratory distress presented to the emergency service between April 2008 and July 2009.

Interventions – None.

Measurements and Main Results – The following parameters were systematically determined at time of admission: respiratory rate, heart rate, temperature, type of breathing, movement of the thoracic and abdominal wall during inspiration, presence of stridor, presence and type of dyspnea, and results of thoracic auscultation. Abdominal and chest wall movement was categorized as synchronous, asynchronous, or inverse. Diagnostic test results, diagnosis, and outcome were subsequently recorded. Based on the final diagnoses, animals were assigned to 1 or more of the following groups regarding the anatomical localization of the respiratory distress: upper airways, lower airways, lung parenchyma, pleural space, thoracic wall, nonrespiratory causes, and normal animals. One hundred and seventy-six animals (103 cats and 73 dogs) were evaluated. Inspiratory dyspnea was associated with upper airway disease in dogs and expiratory dyspnea with lower airway disease in cats. Respiratory noises were significantly associated and highly sensitive and specific for upper airway disease. An asynchronous or inverse breathing pattern and decreased lung auscultation results were significantly associated with pleural space disease in both dogs and cats ($P < 0.001$). The combination is highly sensitive (99%) but not very specific (45%). Fast and shallow breathing was not associated with pleural space disease. Increased or moist pulmonary auscultation findings were associated with parenchymal lung disease.

Conclusions – Cats and dogs with pleural space disease can be identified by an asynchronous or inverse breathing pattern in combination with decreased lung sounds on auscultation.

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From the Departments of Clinical Veterinary Studies (Sigrist, Adamik, Spreng) and Clinical Research and Veterinary Public Health (Doherr), Vetsuisse Faculty of Bern, Laenggassstrasse 120, CH-3012 Bern, Switzerland.

The authors declare no conflict of interest.

Address correspondence and reprint requests to Dr. Nadja Sigrist, Department of Veterinary Clinical Studies, Vetsuisse Faculty of Bern, Laenggassstrasse 128, CH-3012 Bern, Switzerland.
E-mail: nadja.sigrist@kkh.unibe.ch

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Introduction

The purpose of breathing is to provide adequate gas exchange to meet the metabolic demands of the individual animal. In health, this is accomplished effectively and efficiently. With respiratory disease, the animal either minimizes its activity or compensates by altering its breathing pattern or respiratory rate (RR). Many animals effectively hide their illness until

critically low levels of lung reserve remain. These animals deteriorate rapidly when experiencing stress, and stress associated with handling and diagnostic evaluation may cause respiratory arrest.¹ Stabilization of the patient with appropriate drugs or procedures is often required before a diagnostic workup, as procedures such as diagnostic imaging may be too stressful for the patient. Clinical parameters that help distinguish respiratory diseases on presentation may be useful for the stabilization of the emergent patient.

Potential causes of respiratory disease may be identified by simple observation of the patient. In people, the pertinent parameters are the rate, regularity, depth, and effort of breathing.² It has been suggested that cats and dogs demonstrate typical types of breathing depending on the localization of their respiratory problem.^{3,4} With respiratory disease, ventilation or oxygenation or both may become impaired and the respiratory muscles must work harder to compensate. The animal's respiratory pattern may therefore supply important clues as to the anatomical localization and nature of the respiratory tract disorder. Animals with pleural space disease may present with a restrictive respiratory pattern.^{5,6} This pattern has been described as fast and shallow breathing.^{7,8} Others have described paradoxical chest wall movement⁹ or inward movement of the abdomen during inspiration¹⁰ with pleural space disease. The term 'paradoxical' and 'dysynchronous' breathing have been used inconsistently for several abnormal respiratory patterns in the veterinary literature.^{7,11-14} Although respiratory types are commonly evaluated in emergency patients, clinical respiratory parameters at presentation as a diagnostic indicator for localization of the respiratory problem, specifically pleural space disease, have, to our knowledge, not been investigated.

The purpose of this study was to describe clinical parameters of respiration in dogs and cats with respiratory abnormalities and to evaluate associations between clinical signs and the localization of respiratory disease. Particular interest was devoted in assessing the type of breathing and chest wall movements in animals with pleural space disease and we hypothesized that patients suffering from pleural space disease can be identified by an asynchronous type of breathing.

Material and Methods

All dogs and cats presented to the Emergency Service of the Small Animal Clinic of the Vetsuisse Faculty of Bern, Switzerland between April 2008 and July 2009 with clinical signs of respiratory abnormalities at presentation were considered as potential candidates for the study.

The following parameters were evaluated at presentation and after initial stabilization but before diagnostic procedures by the clinician in charge (resident, intern, ACVECC specialist): RR, heart rate (HR), temperature, type of breathing, type of dyspnea, movement of the chest wall during inspiration, movement of the abdominal wall during inspiration, bilateral auscultation results (normal, decreased, increased, moist), and presence of stridor or stertor. Type of breathing was characterized as normal, fast, and shallow (with closed mouth), costoabdominal (increased abdominal breathing effort), panting (fast and shallow open mouth breathing in dogs), and open mouth breathing in cats. The type of dyspnea was characterized as none, inspiratory (inspiration longer than expiration), expiratory (expiration markedly prolonged), and mixed (signs of dyspnea without a clear inspiratory or expiratory component). Animals were allowed to be stabilized before evaluation with oxygen supplementation, analgesic therapy, and IV fluids if indicated. The preferred analgesic agent used was methadone^a 0.2 mg/kg, IV or IM, but the choice of analgesic was at the discretion of the clinician in charge. After evaluation of respiratory parameters, animals were diagnostically evaluated and treated at the discretion of the clinician in charge. Clinician's diagnostic and therapeutic plans were performed and treatment was not withheld for the purposes of the study.

Abdominal and chest wall movements during inspiration were noted by the primary clinician and categorized later by the lead author (N.S.) as synchronous (outward movement of chest and abdomen during inspiration), asynchronous (outward movement of the chest and inward movement of the abdomen during inspiration), or inverse (inward movement of the chest and outward movement of the abdomen during inspiration). The final diagnose(s) and the survival to hospital discharge (death during the hospitalization period or euthanasia due to owners request) were retrospectively extracted from patient records, looking specifically at radiology reports but also ultrasound, CT, endoscopy, surgery, and histopathology reports depending on availability. According to the final diagnose(s), the respiratory abnormalities were localized to 1 or more of the following localizations: upper airway (including nose, larynx, pharynx, trachea, and main bronchi), lower airways (bronchi and bronchioli), lung parenchyma (vascular-interstitial-alveolar problems), pleural space (including mediastinal masses and diaphragmatic problems), chest wall, and nonrespiratory causes (eg, severe anemia [PCV < 15%], severe hyperthermia [$T > 40.0^{\circ}\text{C}$], brainstem disease).

Exclusion criteria included excessive missing data, lack of either thoracic radiographs, CT scan, or

postmortem histopathology, and treatment of patients with furosemide, corticosteroids, bronchodilators, sedatives (other than analgesic agents), or thoracocentesis before the evaluation of respiratory parameters. Based on the preliminary results, cats with painful diseases (eg, fractures) not evaluated after analgesic treatment were also excluded from the study.

Statistical analysis

For the continuous variables, median and range are reported as many variables were not normally distributed. Frequency distributions of the categorical or ordinal variables were derived. Possible associations between respiratory parameters and localizations as well as respiratory parameters before and after analgesic therapy (categorical variables) were evaluated using χ^2 -test. As many cell frequencies were below 5, associations between a specific respiratory parameters and the localization of the respiratory problem were evaluated using a 2-sided Fisher's exact test. RR was correlated with specific locations (affected, not affected) using the Wilcoxon signed-rank test for difference in medians.

All analyses were performed using the statistical software program.^b Statistical significance was set at $P < 0.05$ for all analyses.

Results

One hundred and seventy-six patients (103 cats and 73 dogs) were included in the study. Median age of cats was 49 months (range 4–216 mo) and of dogs 92.5 months (range 2–188 mo), respectively. Thirty-three animals were intact females, 50 spayed females, 25 intact males, and 65 castrated males.

Ninety-one animals were evaluated before and after stabilization with oxygen and analgesic treatment. Strong agreement was demonstrated ($P < 0.001$) between respiratory parameters (type of breathing, type of dyspnea, chest wall movement, auscultation results, and respiratory noises) prior and after stabilization except for RR in cats. In cats, RR decreased significantly from 60/min (20–200/min) to 45/min (28–180/min) ($P < 0.001$) while the RR in dogs was the same prior and after analgesia. If pre- and poststabilization results were available, the poststabilization parameters were used for further statistical analysis.

Cats presented with a median RR of 48/min (range 20–180/min), a median HR of 180/min (range 60–280/min), and a median temperature of 38.0 (range 33.4–40.4)°C while dogs presented with a median RR of 80/min (range 16–180/min), a median HR of 120/min (range 44–200/min), and a median temperature of 38.6 (range 35.5–41.7)°C. Median RR in dogs

Table 1: Frequency distributions of respiratory parameters in dogs ($n = 73$) and cats ($n = 103$) with respiratory distress

Parameter	Cats		Dogs		All animals	
	<i>n</i>	%	<i>n</i>	%	<i>n</i>	%
Type of respiration						
Normal	4	3.9	1	1.4	5	2.9
Superficial	16	15.7	8	11.1	24	13.8
Costoabdominal	77	75.5	38	52.8	115	66.1
Panting	0	0	23	31.9	24	13.8
Open mouth	45	4.9	2	2.8	6	3.4
	102	100	72	100	174	100
Type of dyspnea						
None	33	33	18	26.1	51	30.2
Inspiratory	12	12	4	5.8	16	9.5
Expiratory	11	11	10	14.5	21	12.4
Mixed	44	44	37	53.6	81	47.9
	100	100	69	100	169	100
Synchronicity of thoracic and abdominal wall movements						
Synchronous	56	55.5	53	72.6	109	62.6
Asynchronous	35	34.6	18	24.7	53	30.5
Inverse	10	9.9	2	2.7	12	6.9
	101	100	73	100	174	100
Auscultation results left side						
Normal	35	37.2	25	36.2	60	36.8
Decreased	22	23.4	11	15.9	33	20.2
Increased	26	27.7	30	43.5	56	34.4
Moist	11	11.7	3	4.4	14	8.6
	94	100	69	100	163	100
Auscultation results right side						
Normal	36	38.3	24	34.8	60	36.8
Decreased	21	22.3	11	15.9	32	19.6
Increased	25	26.6	30	43.5	55	33.8
Moist	12	12.8	4	5.8	16	9.8
	94	100	69	100	163	100
Stridor or stertor						
None	88	88.9	61	83.6	149	86.6
Present	8	8.1	10	13.7	18	10.5
Only auscultatory	3	3	2	2.7	5	2.9
	99	100	73	100	172	100

was significantly higher than in cats ($P < 0.001$). One hundred and four animals received analgesic therapy. Analgesia was provided with methadone ($n = 73$), buprenorphine^c ($n = 10$), butorphanol^d ($n = 8$), and fentanyl^e bolus followed by continuous rate infusion ($n = 13$).

Frequencies of evaluated respiratory parameters are presented in Table 1. The most frequent presentation findings were costoabdominal (66.1%), synchronous (62.6%) breathing patterns with a mixed type of dyspnea (47.9%). Lung sounds on auscultation were normal (36.8%) or increased (33.7% on right side and 34.4% on left side) in most animals.

According to the final diagnose(s) the following localizations were identified (Table 2): upper airways ($n = 15$; 7 cats, 8 dogs), lower airways ($n = 10$; 7 cats, 3 dogs), lung parenchyma ($n = 98$; 49 cats, 49 dogs),

Table 2: Localization of respiratory distress in dogs and cats with multiple or single localizations

Localization	Multiple localizations			Single localization		
	Cats (<i>n</i>)	Dogs (<i>n</i>)	All animals (<i>n</i>)	Cats (<i>n</i>)	Dogs (<i>n</i>)	All animals (<i>n</i>)
Upper airways	7	8	15	2	6	8
Lower airways	7	3	10	6	0	6
Lung parenchyma	49	49	98	18	23	41
Pleural space	50	19	69	19	5	24
Chest wall	7	4	11	1	1	2
Nonrespiratory cause	16	13	29	12	3	15
Normal				8	7	15
Total	136	96	232	66	45	111

pleura ($n = 69$; 50 cats, 19 dogs), chest wall ($n = 11$, 7 cats, 4 dogs), and extrapulmonary ($n = 29$; 16 cats, 13 dogs). Fifteen animals (8 cats, 7 dogs) had no identifiable cause despite diagnostic testing and were localized as 'normal.' These 'normal' animals showed no signs of dyspnea (87.5%) or respiratory noises (100%), normal auscultation results (71.4%), a synchronous breathing pattern (100%) but various breathing types.

Of the animals with a pleural localization, 24 of 69 animals had only pleural space disease whereas 45 showed pleural space disease in combination with another respiratory localization. Respiratory parameters associated with pleural space localization are summarized in Table 3. Asynchronous breathing ($P < 0.001$) as well as decreased lung sounds on auscultation ($P < 0.001$) were significantly associated with a pleural space localization in dogs as well as cats. The combination of asynchronous breathing and decreased lung sounds on auscultation was significantly associated ($P < 0.001$) with pleural space localization and was 99% sensitive and 40% specific. Inclusion of inverse breathing increased the specificity (SP) to 45%. Asynchronous breathing was also significantly associated with chest wall localization in cats ($P = 0.047$). Fast and shallow (superficial) breathing was not associated with pleural space localization in either dogs or cats. Associations between respiratory parameters and pleural space localization and appropriate sensitivity (SE) and SP results are summarized in Table 4.

Other significant associations were found between inspiratory dyspnea and upper airway localization in dogs ($P = 0.049$, SE 28.6%, SP 96.8%), expiratory dyspnea with lower airway localization in cats ($P = 0.023$, SE 40%, SP 89.3%), and panting with chest wall localization in dogs ($P = 0.01$, SE 100%, SP 69.1%). A mixed type of dyspnea was significantly associated with lung localization in dogs ($P = 0.036$, SE 62.5%, SP 66.7%) but pleural space disease in cats ($P = 0.026$, SE 56.3%, SP 67.3%). The presence of inspiratory noises was significantly associated with upper airway

localization in dogs ($P < 0.001$, SE 75%, SP 93.8%) as well as cats ($P < 0.001$, SE 71.4%, SP 96.7%), and increased or moist lung sounds on auscultation with lung parenchyma localization in both dogs and cats ($P = 0.001$, SE 60.9%, SP 80%).

One hundred and eleven animals (66 cats, 45 dogs) presented with a single localization (Table 2). When looking only at animals with an isolated localization, inspiratory dyspnea in dogs ($P = 0.012$, SE 40%, SP 100%) and respiratory noises in dogs ($P < 0.001$, SE = 87.5%, SP = 95%) and cats ($P = 0.005$, SE 100%, SP 95%) remained significantly associated with upper airway localization. Expiratory dyspnea remained significantly associated with lower airway localization in cats ($P = 0.003$, SE 66.7%, SP 89.9%) and increased or moist lung sounds on auscultation with pulmonary parenchymal localization in dogs ($P = 0.002$, SE 72.7%, SP 76.2% [left side] and $P < 0.001$, SE 77.3%, SP 76.2% [right side]) as well as cats ($P < 0.001$, SE 70.6%, SP 81.8%). The type of breathing (shallow, costoabdominal, panting) and mixed dyspnea were no longer associated with a specific localization when only animals with an isolated localization were evaluated. Asynchronous breathing in cats was also no longer associated with chest wall localization.

One hundred and fourteen (64.8%) animals survived to hospital discharge while 10 animals (5.7%) died during hospitalization and 52 (29.6%) were euthanized. Of the 103 cats, 71 (69%) survived to hospital discharge, 5 (5%) died and 27 (26%) were euthanized. Forty-three of 73 (59%) dogs survived to hospital discharge, 5 (7%) died and 25 (34%) were euthanized. Survival was not different in dogs versus cats ($P = 0.17$).

Discussion

Respiratory distress can be caused by various diseases of the respiratory tract as well as nonrespiratory causes such as CNS disease, anemia, or heatstroke.^{15,16} Patients with respiratory distress generally present

Table 3: Frequency distributions of respiratory parameters with pleural space localization in dogs ($n = 19$) and cats ($n = 50$)

Parameter	Multiple localizations						Single localization					
	Cats		Dogs		All animals		Cats		Dogs		All animals	
	<i>n</i>	%	<i>n</i>	%	<i>n</i>	%	<i>n</i>	%	<i>n</i>	%	<i>n</i>	%
Type of respiration												
Normal	2	4.1	0	0	2	2.9	1	5.3	0	0	1	4.1
Superficial	5	10.2	2	10.5	7	10.3	3	15.8	1	20.0	4	16.7
Costoabdominal	41	83.7	13	68.4	54	79.4	15	78.9	4	80.0	19	79.2
Panting	0	0	4	21.1	4	5.9	0	0	0	0	0	0
Open mouth	1	2.0	0	0	1	1.5	0	0	0	0	0	0
	49	100	19	100	68	100	19	100	5	100	24	100
Type of dyspnea												
None	9	18.8	1	5.3	10	14.9	3	16.7	1	20.0	4	17.4
Inspiratory	7	14.6	1	5.3	8	11.9	6	33.3	0	0	6	26.1
Expiratory	5	10.4	4	21.0	9	13.4	1	5.6	1	20.0	2	8.7
Mixed	27	56.3	13	68.4	40	59.7	8	44.4	3	60.0	11	47.8
	48	100	19	100	67	100	18	100	5	100	23	100
Synchronicity of thoracic and abdominal wall movements												
Synchronous	16	32.7	6	31.6	22	32.4	4	22.2	0	0	4	17.4
Asynchronous	27	55.1	13	68.4	40	58.8	12	66.7	5	100	17	73.9
Inverse	6	12.2	0	0	6	8.8	2	11.1	0	0	2	8.7
	49	100	19	100	68	100	18	100	5	100	23	100
Auscultation results left side												
Normal	10	20.8	2	11.1	12	18.2	5	26.3	0	0	5	20.8
Decreased	21	43.8	10	55.6	31	47	13	68.4	5	100	18	75.0
Increased	12	25.0	6	33.3	18	27.3	1	5.3	0	0	1	4.2
Moist	5	10.4	0	0	5	7.6	0	0	0	0	0	0
	48	100	18	100	66	100	19	100	5	100	24	100
Auscultation results right side												
Normal	11	22.9	2	11.1	13	19.7	7	36.8	0	0	7	29.2
Decreased	20	41.7	10	55.6	30	45.5	11	57.9	5	100	16	66.6
Increased	12	25.0	6	33.3	18	27.3	1	5.3	0	0	1	4.2
Moist	5	10.4	0	0	5	7.6	0	0	0	0	0	0
	48	100	18	100	66	100	19	100	5	100	24	100
Stridor or stertor												
None	46	93.9	16	84.2	62	91.2	18	94.7	5	100	23	95.8
Present	3	6.1	2	10.5	5	7.4	1	5.3	0	0	1	4.2
Only auscultatory	0	0	1	5.3	1	1.5	0	0	0	0	0	0
	49	100	19	100	68	100	19	100	5	100	24	100

with minimal respiratory reserves. Prompt recognition of the underlying problem and appropriate therapeutic interventions are essential in the successful management of these patients. Radiography or CT scanning are important diagnostic procedures providing a diagnosis in many instances. Unfortunately, most diagnostic procedures are associated with stress and may lead to aggravation of respiratory distress, which could eventually lead to respiratory arrest.¹ A thoracic focused assessment with sonography for trauma (TFAST) protocol has been evaluated in traumatized dogs¹⁷ and TFAST has become increasingly used in veterinary medicine. However, the TFAST protocol described by Lisciandro et al¹⁷ describes patient evaluation in lateral recumbency, which may be too stressful for the patient with severe respiratory distress. TFAST is also

technically demanding and requires training and experience.¹⁷

Patients with severe compromising respiratory distress therefore should be stabilized before performing diagnostic procedures. The presented study aimed to identify clinical respiratory parameters on presentation that may allow the clinician to better localize the disease and begin more specific therapeutic interventions as part of patient stabilization. Localization of the respiratory problem to a specific part of the respiratory tract allows for a reduction of potential differential diagnoses.^{1,3,4,18} Clinical parameters such as chest auscultation are predictive of radiographic chest trauma signs^{19,20} and may therefore also be useful in localizing respiratory signs to a specific part of the respiratory tract. Many textbooks and review articles

Table 4: Significant associations between respiratory parameters and pleural space localization in dogs and cats (nonsignificant associations not shown)

Localization															
Parameter	Pleural space in combination with another localization						Isolated pleural space localization								
	All animals (n = 69)		Cats (n = 50)		Dogs (n = 19)		All animals (n = 24)		Cats (n = 19)		Dogs (n = 5)				
	P-value	SE	SP (%)	P-value	SE	SP (%)	P-value	SE	SP (%)	P-value	SE	SP (%)	P-value	SE	SP (%)
Costoabdominal respiration	0.003	79.4	42.0	0.071		0.180	0.034	79.2	46.5	0.26		0.057			
Mixed type of dyspnea	0.018	59.7	59.8	0.026	56.3	67.3	0.479			0.57		0.65			
Asynchronous breathing	< 0.001	58.8	87.7	< 0.001	55.1	85.6	< 0.001	68.4	90.7	< 0.001	73.9	66.7	< 0.001	89.4	92.5
Asynchronous or inverse breathing	< 0.001	67.6	82.1	< 0.001	67.3	76.9	< 0.001	68.4	87.0	< 0.001	82.6	77.8	< 0.001	80.9	87.5
Auscultation results left side decreased	< 0.001	47.0	97.9	< 0.001	43.8	97.8	< 0.001	55.6	98.0	< 0.001	78	68.4	< 0.001	97.5	97.4
Auscultation results right side decreased	< 0.001	45.5	97.9	< 0.001	41.7	97.8	< 0.001	55.6	98.0	< 0.001	66.7	61.1	< 0.001	97.6	100

Bold: $P < 0.05$.
SE, sensitivity; SP, specificity.

describe presence of respiratory distress or dyspnea and auscultation findings in a specific disease but do not describe the respiratory type related to the anatomical localization.^{21–25}

Only few and inconsistent associations between specific breathing types and localizations or diseases are described in the literature and many of them are only based on clinical impressions.^{1,12,26} Our study confirms some of these associations but also shows significant differences to some commonly used, clinically relevant associations.

A particular focus of the present study was to investigate the clinical signs of respiratory distress in dogs and cats with pleural space disease. Rate and depth of respiration are mediated by central and peripheral chemoreceptors.²⁷ Any changes in PO₂, PCO₂, or insufficient thoracic movement may manifest in abnormal breathing types or dyspnea. Normal chest excursions are a balance between lung compliance and airway resistance.²⁸ Pleural space disease is defined as an accumulation of air, fluid, and soft tissue, or any of them within the pleural cavity.⁶ The reduced functional residual capacity seen with pleural space disease forces the lung to operate on a less compliant portion of the compliance curve. It has been concluded that the patient compensates for the increase in respiratory work with a restrictive breathing pattern seen as rapid and shallow breathing.^{6,28} Short and shallow respirations have therefore been proposed to indicate pleural space disease in most textbooks.^{3,4,8,29} An increased abdominal component with shallow breathing has been suggested as well.²⁹ Others have described inspiratory dyspnea as a sign of pleural space disease.²² In our study, shallow and superficial respiration was not associated with pleural space disease, neither in dogs nor cats, and independent of pleural space disease being an isolated or combined localization. Fast and shallow breathing was significantly more frequent in cats with no respiratory abnormalities compared with cats with respiratory disease. Therefore, fast and shallow breathing may be more a sign of stress than a sign of disease. Pleural space disease was significantly associated with a costoabdominal breathing pattern and asynchronous breathing in both dogs and cats and SE and SP increased when pleural space disease was the single localization. This breathing pattern has been implicated previously but has not been investigated in a clinical setting.^{9,10} In the dog, the parasternal intercostal muscles were identified as the main inspiratory muscles.³⁰ By observing chest wall mechanics in dogs with bilateral phrenectomy leading to diaphragmatic paralysis, the rib cage expands during inspiration while the abdomen moves inward.³⁰ This is explained by the anatomical localization of the intercostal muscles and the

rib cage conformation in dogs. The inspiratory expansion of the rib cage in affected dogs is more substantial than in normal dogs and compensates for the inward motion of the abdomen.³⁰ Transthoracic pressures generated by the inspiratory musculature must be sufficient to overcome both airway resistance and inward elastic recoil of the lungs and thoracic wall.³¹ The asynchronous breathing in dogs with diaphragmatic paralysis has been explained by the lack of a transdiaphragmatic pressure gradient and may also explain the breathing mechanics in animals with an increase in intrapleural pressure seen with pleural space disease.

In the present study, an asynchronous breathing pattern was differentiated from an inverse breathing pattern. The term inverse breathing may be interchangeable with the term paradoxical breathing. We preferred the term inverse breathing for the description of inward movement of the thoracic wall during inspiration as paradoxical breathing has been used inconsistently by several authors. Paradoxical breathing has been used to describe abnormal chest wall movement seen with a flail chest,¹³ pleural space disease,^{14,32} and severe upper airway obstruction.²⁹

Only 12 animals showed inverse breathing and inverse breathing was not associated with a specific localization. Six animals with inverse breathing were diagnosed with pleural space disease. SE to identify pleural space disease was increased when inverse breathing was combined with asynchronous breathing, but SP decreased as parenchymal lung disease was as frequent a localization as pleural space disease. The influence of parenchymal lung disease is also reflected by increased lung sounds on auscultation in 70% of animals with inverse breathing. Inverse breathing should therefore alert the clinician to identify or rule out pleural space disease by evaluating other clinical signs. Auscultation results may be helpful in differentiating between pleural space disease and other causes of an asynchronous or inverse breathing pattern. Pneumothorax causes a reduction in computerized sound amplitude in an experimental canine study.³³ Auscultation results were significantly associated with pneumothorax in a study looking at trauma patients.¹⁹ Our study identified a significant association and high SE of pleural space localization with decreased lung sounds on auscultation, this is in accordance with other studies.^{33,34} Muffled heart sounds have also been described as a clinical sign of pleural space disease.^{22,35} However, 1 study describes increased respiratory sounds in dogs with pleural effusion.³⁵ None of the animals with isolated pleural space disease in our study were reported to have increased lung sounds on auscultation.

Asynchronous breathing was not only significantly associated with pleural space disease but also with chest wall localizations in cats. This may be due to the fact that 8 of 10 chest wall injuries occurred in conjunction with other respiratory tract problems and all included pleural space disease. Only 2 animals with isolated chest wall problems were identified and statistical analysis was not performed. An abnormal (asynchronous) breathing pattern with inward movement of the abdominal wall during inspiration has also been described in chest wall disease with accompanying muscle paralysis.³⁶

The present study allowed assessment of respiratory parameters with other localizations. The findings of this study support the theory that upper airway problems are associated with upper airway noises^{3,29,37,38} and inspiratory respiratory distress.^{14,39–41} An exaggerated inspiratory effort seen as inspiratory dyspnea will increase tidal volume and therefore increases the volume of air delivered to the alveoli.¹⁴ Interestingly, inspiratory dyspnea was only associated with upper airway disease in dogs but not in cats. Holt⁴¹ has described open mouth breathing in cats with upper airway disease. Stress or anxiety may lead to an increase in RR with less time for a prolonged inspiration. The feline group with upper airway disease was also rather small (7 cats) and upper airway localization as an isolated localization was only seen in 2 cats, so further studies are needed to define the type of dyspnea in cats with upper airway disease. Upper airway diseases such as tracheal collapse may present with coughing rather than signs of respiratory distress.^{38,42} Our study did not investigate the influence of the presence of cough but presence and type of coughing may also be of interest and help localize the problem to the trachea.

This study also found an association between expiratory dyspnea and lower airway localization in cats as has been reported previously.^{7,14,39,43} An obstructive respiratory pattern has been described in lower airway disease.^{5,29} Obstructive diseases causing increased airway resistance may be associated with a slow and deep pattern of breathing.²⁸ An obstructive respiratory pattern can be either inspiratory or expiratory, so identification of the affected respiratory phase may be beneficial. Again, the identification of coughing may be helpful in identifying cats with lower airway disease.⁴⁴ Our study identified only 3 dogs with lower airway localization which did not show an association with any clinical sign.

The anatomic localization of lung parenchyma includes many different disease processes such as bleeding, inflammation, infection, and neoplasia.^{24–26,45} Thoracic radiographs often show an infiltrative pattern localizing the problem to the pulmonary parenchyma,

but often do not provide a specific diagnosis.^{46,47} Auscultation findings in trauma patients were significantly associated with radiographic signs of lung contusions.¹⁹ Pulmonary parenchyma localization has been described with clinical signs of open mouth or paradoxical respiration,⁷ increased RR,^{48,49} rapid shallow breathing,⁴⁹ mixed dyspnea,^{29,39} or unspecified respiratory distress.^{26,50,51} In our study, dogs with pulmonary parenchymal disease in combination with another localization showed a mixed type of dyspnea; this was no longer significant when dogs with isolated parenchymal diseases were evaluated. There was no association with a paradoxical respiration or open mouth breathing. In our study, dogs and cats with a pulmonary localization showed increased or moist lungs sounds on auscultation. This is in accordance with retrospective studies investigating clinical signs in animals with pneumonia,^{48,52,53} lung contusions,⁵⁴ pulmonary neoplasia,²⁶ and cardiogenic pulmonary edema.⁵⁵ Other clinical signs such as identification of a heart murmur or cardiac arrhythmia and hypothermia may support the diagnosis.^{51,56}

Chest wall injuries are thought to be painful, leading to a fast and shallow breathing pattern⁵⁷ caused by a lack of diaphragmatic and abdominal movement, as well as a lack of intercostal muscle assistance.⁷ In dogs, the external intercostal and the internal intercartilaginous intercostal muscles elevate the ribs during inspiration.³⁰ In our study, dogs were found to be panting with chest wall disease, which may be a sign of pain or stress, and represents a shallow breathing pattern. Panting in concordance with normal auscultation results has been associated with normal pulmonary radiographs and stress.¹⁹ Other clinical signs, such as abnormal movement of chest segments as seen with flail chest or intercostal muscle disruption will help localize the problem to the chest wall.^{57,58} A flail chest is defined as a segment of ribs that moves independently, typically collapsing inwards during inspiration.^{14,59} None of the animals in our study that had rib fractures visualized on radiographs presented with an open chest wound or other visible signs of chest wall injuries.

Many animals presented with more than 1 respiratory localization. When investigating only animals with an isolated localization, no new significant associations between localization and breathing parameters could be identified. Breathing types such as panting or mixed dyspnea were no longer associated with a specific localization. Both findings may be caused by the small group size. However, many identified breathing parameters remained significant despite small numbers per group, including breathing parameters seen with pleural space disease. As the clinician dealing with an emergency patient does not know if the respiratory

distress is caused by an isolated disease or a combination of localizations, parameters seen only with an isolated localization would need to be interpreted with caution.

The localization of respiratory distress allows the emergency clinician to be more specific with the treatment of a patient without increasing stress by performing diagnostic procedures. If oxygen and rest is not sufficient to stabilize a patient with severe respiratory distress, administration of specific drugs such as furosemide or corticosteroids and emergency procedures such as thoracocentesis or tracheostomy may be life saving. Drugs used for stabilization of patients with respiratory distress have side effects and procedures may be associated with further stress, so clear indications are needed. In the case of pleural space disease, thoracocentesis is not only a therapeutic procedure but may also be diagnostic.⁶⁰ Therapeutic thoracocentesis can remove significant volumes of fluid or air and results in rapid improvement of respiration and a more stable patient for further diagnostic procedures. The results of this study help to identify the patients that could benefit from thoracocentesis. Iatrogenic pneumothorax resulting from thoracocentesis without confirmed pneumothorax or pleural effusion may be a potential complication.⁶¹ The incidence of complications of thoracocentesis in animals has not been reported. The impact of diagnostic and therapeutic thoracocentesis in animals with clinical signs of pleural space disease on survival remains unknown as well.

In 16 animals, no respiratory or extrathoracic problem could be identified despite animals showing respiratory abnormalities at presentation. A recent study evaluating the etiology of 90 cats presenting with dyspnea included only 1 cat presenting with dyspnea without underlying disease.²⁴ Respiratory distress as an inclusion criteria was not strictly defined in our study and any patient with respiratory abnormalities could have been included in the study. Most of these 'normal' animals had either fast and shallow or costoabdominal breathing. Stress may have caused the respiratory signs. Sympathetic stimulation increases the RR and may also influence the type of breathing.⁶² Stress, shock, pain, or other causes of sympathetic stimulation could have caused the respiratory abnormalities in our 'normal' cats. Pain has been shown to influence RR in trauma patients¹⁹ as seen with cats in this study. However, all other respiratory parameters showed a strong agreement prior and after analgesic therapy. Because of ethical reasons, a control group receiving the same treatment with rest and oxygen supplementation but without analgesia was not evaluated. Therefore, the actual influence of analgesia in comparison with other supportive treatment modalities cannot be determined.

Overall, RR has not been shown to be a relevant discriminator of different causes of dyspnea²⁴ and has not been evaluated in this study.

A limitation of the study included patient evaluation being performed by various clinicians, including interns, residents of various specialties, and an ACVECC specialist. This may have influenced the results of presence and type of dyspnea and may be reflected in the large group of animals presenting with a mixed type of dyspnea that was not associated with any localization. However, the questionnaire was designed as objective as possible with description of chest and abdominal wall movement rather than interpretation of dyssynchronous or inverse breathing.

Another limitation is that not all patients received the same initial stabilization protocol. Oxygen therapy or analgesia or both and rest did not influence respiratory parameters (except RR in cats) so the conclusions from the study are useful at presentation as well as during the stabilization phase and may also apply to patients that show changes in respiration during their ICU stay. Furthermore, sample size may have been insufficient to identify specific breathing patterns in the groups of upper and lower airway and chest wall disease and additional clinical studies are indicated.

In conclusion, this study confirmed that dogs and cats with upper airway disease often present with inspiratory noises and dogs with upper airway disease present with inspiratory dyspnea while cats with lower airway disease show expiratory dyspnea. The frequent description of fast and shallow breathing in patients with pleural space disease was not seen in this study population. Animals with pleural space disease showed predominantly an asynchronous breathing type. SE and SP for animals with pleural space disease showing an asynchronous or inverse breathing type in combination with decreased lung sounds on auscultation was 99% and 40%, respectively. These clinical signs should alert the clinician to consider pleural space disease and perform thoracocentesis. Increased or moist lung sounds heard on auscultation are significantly associated with pulmonary parenchymal disease. Various breathing types, especially fast and shallow breathing in cats, can be seen in animals without respiratory problems. However, these 'normal' animals can easily be identified as most of them present without dyspnea and show normal auscultation findings.

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Footnotes

- ^a Methadone, Ketalgin[®], Streuli AG, Uznach, Switzerland.
^b NCSS, Number Cruncher Statistical Systems, Kaysville, UT.
^c Buprenorphine, Temgesic[®], Reckitt Benckiser, Slough, Berkshire, UK.
^d Butorphanol, Morphasol[®], Dr E. Graeub AG, Bern, Switzerland.
^e Fentanyl, Fentanyl-Janssen[®], JANSSEN-CILAG GmbH, Neuss, Germany.

References

- Rozanski EA, Chan DL. Approach to the patient with respiratory distress. *Vet Clin North Am* 2005; 35(3):307–317.
- Snider GL. History and physical examination, In: Baum GL, Wolinsky E. eds. *Textbook of Pulmonary Disease*. Philadelphia, PA: WB Saunders; 1993.
- Tseng LW, Waddell LS. Approach to the patient in respiratory distress. *Clin Tech Small Anim Pract* 2000; 15(2):53–62.
- Silverstein DC, Drobatz KJ. Clinical evaluation of the respiratory tract, In: Ettinger SJ, Feldman EC. eds. *Textbook of Veterinary Internal Medicine*. St Louis, MO: Saunders Elsevier; 2005, pp. 1206–1217.
- Mandell DC. Respiratory distress in cats, In: King LG. ed. *Textbook of Respiratory Disease in Dogs and Cats*. St Louis: Saunders Elsevier; 2004, pp. 12–16.
- Monnet E. Pyothorax, In: King LG. ed. *Textbook of Respiratory Disease in Dogs and Cats*. St Louis: Saunders Elsevier; 2004, pp. 605–610.
- Lee JA, Drobatz KJ. Respiratory distress and cyanosis in dogs, In: King LG. ed. *Textbook of Respiratory Disease in Dogs and Cats*. St Louis: Saunders Elsevier; 2004, pp. 1–11.
- Silverstein DC. Pleural space disease, In: King LG. ed. *Textbook of Respiratory Disease in Dogs and Cats*. St Louis: Saunders Elsevier; 2004, pp. 49–52.
- Sanders NA, Sleeper M. Pleural transudates and modified transudates, In: King LG. ed. *Textbook of Respiratory Disease in Dogs and Cats*. St Louis: Saunders Elsevier; 2004, pp. 587–597.
- Rudloff E. Clinical signs of respiratory distress. In: *Proceedings of the Western Veterinary Conference*; 2002: Las Vegas, USA.
- Ford RB, Mazzaferro EM. Respiratory emergencies, In: Ford RB, Mazzaferro EM. eds. *Kirk and Bistner's Handbook of Veterinary Procedures and Emergency Treatment*. St Louis: Saunders Elsevier; 2006, pp. 253–271.
- Waddell LS, King LG. General approach to dyspnoea, In: King LG, Hammond R. eds. *Manual of Canine and Feline Emergency and Critical Care*. Gloucester: BSAVA; 2003, pp. 65–86.
- Smith MM. Flail chest, In: King LG. ed. *Textbook of Respiratory Disease in Dogs and Cats*. St Louis: Saunders Elsevier; 2004, pp. 647–651.
- Harpster NK. Physical examination of the respiratory tract, In: King LG. ed. *Textbook of Respiratory Disease in Dogs and Cats*. St Louis: Saunders Elsevier; 2004, pp. 67–71.
- Luis-Fuentes V. Differential diagnoses of dyspnoea, In: Luis-Fuentes V, Swift S. eds. *Manual of Small Animal Cardiorespiratory Medicine and Surgery*. Gloucester, UK: BSAVA; 1998, pp. 123–128.
- Hall K, Lee JA. Nonrespiratory look-alikes, In: Silverstein DC, Hopper K. eds. *Small Animal Critical Care Medicine*. St Louis: Saunders Elsevier; 2009, pp. 141–144.
- Lisciandro GR, Lagutchik MS, Mann KA, et al. Evaluation of a thoracic focused assessment with sonography for trauma (TFAST) protocol to detect pneumothorax and concurrent thoracic injury in 145 traumatized dogs. *J Vet Emerg Crit Care* 2008; 18(3):258–269.
- Aldrich J. Global assessment of the emergency patient. *Vet Clin North Am Small Anim Pract* 2005; 35(2):281–305.
- Sigrist NE, Doherr MG, Spreng DE. Clinical findings and diagnostic value of posttraumatic thoracic radiographs in dogs and cats with blunt trauma. *J Vet Emerg Crit Care* 2004; 14(4):259–268.
- Holmes JF, Sokolove PE, Brant WE, Kuppermann N. A clinical decision rule for identifying children with thoracic injuries after blunt torso trauma. *Ann Emerg Med* 2002; 39(5):492–499.
- Rozanski EA, Rush JE. *A Color Handbook of Small Animal Emergency and Critical Care Medicine*, 1st ed. London, UK: Manson publishing; 2007, pp. 66–78.
- Demetriou JL, Foale RD, Ladlow J, et al. Canine and feline pyothorax: a retrospective study of 50 cases in the UK and Ireland. *J Small Anim Pract* 2002; 43(9):388–394.
- Brockman D, Puerto DA. Pneumomediastinum and pneumothorax, In: King LG. ed. *Textbook of Respiratory Diseases in Dogs and Cats*. St Louis: Saunders Elsevier; 2004, pp. 616–624.
- Swift S, Dukes-McEwan J, Fonfara S, et al. Aetiology and outcome in 90 cats presenting with dyspnoea in a referral population. *J Small Anim Pract* 2009; 50(9):466–473.
- Parent C, King LG, Walker LM, Van Winkle TJ. Clinical and clinicopathologic findings in dogs with acute respiratory distress syndrome: 19 cases (1985–1993). *J Am Vet Med Assoc* 1996; 208(9):1419–1427.
- Sauve V, Drobatz KJ, Shokak AB, et al. Clinical course, diagnostic findings and necropsy diagnosis in dyspneic cats with primary pulmonary parenchymal disease: 15 cats (1996–2002). *J Vet Emerg Crit Care* 2005; 15(1):38–47.
- O'Regan RG, Majcherczyk S. Role of peripheral chemoreceptors and central chemosensitivity in the regulation of respiration and circulation. *J Exp Biol* 1982; 100:23–40.
- Orton EC. Respiratory system, In: Wingfield WE, Raffe MR. eds. *The Veterinary ICU Book*. Jackson, WY: Teton New Media; 2002, pp. 281–297.
- Ludwig LL. Surgical emergencies of the respiratory system. *Vet Clin North Am* 2000; 30(3):531–553.
- De Troyer A, Kelly S. Chest wall mechanics in dogs with acute diaphragm paralysis. *J Appl Physiol* 1982; 53(2):373–379.
- Orton EC. Thoracic wall, In: Slatter D. ed. *Textbook of Small Animal Surgery*. Philadelphia, PA: Saunders; 2003, pp. 373–387.
- Hendricks JC. Respiratory muscle fatigue and failure, In: King LG. ed. *Textbook of Respiratory Disease in Dogs and Cats*. St Louis: Saunders Elsevier; 2004, pp. 61–65.
- Mansy HA, Royston TJ, Balk RA, Sandler RH. Pneumothorax detection using computerised analysis of breath sounds. *Med Biol Eng Comput* 2002; 40(5):526–532.
- Davies C, Forrester SD. Pleural effusion in cats: 82 cases (1987 to 1995). *J Small Anim Pract* 1996; 37(5):217–224.
- Mellanby RJ, Villiers E, Herrtage ME. Canine pleural and mediastinal effusions: a retrospective study of 81 cases. *J Small Anim Pract* 2002; 43(10):447–451.
- Donahue S. Chest wall disease, In: Silverstein DC, Hopper K. eds. *Small Animal Critical Care Medicine*. St Louis: Saunders Elsevier; 2008, pp. 138–140.
- Koch DA, Arnold S, Hubler M, Montavon PM. Brachycephalic syndrome in dogs. *Compend Contin Educ Pract Vet* 2003; 25(1):48–55.
- Hedlund CS. Tracheal collapse. *Probl Vet Med* 1991; 3(2):229–238.
- Cohn LA. Recognizing respiratory patterns and signs. In: *Proceedings of the Western Veterinary Conference*; 2002: Las Vegas, USA.
- Holt DE, Brockman D. Laryngeal paralysis, In: King LG. ed. *Textbook of Respiratory Diseases in Dogs and Cats*. St Louis, MO: Saunders Elsevier; 2004, pp. 319–328.
- Holt DE. Upper airway obstruction, stertor, and stridor, In: King LG. ed. *Textbook of Respiratory Disease in Dogs and Cats*. St Louis: Saunders Elsevier; 2004, pp. 35–38.
- Johnson LR, Fales WH. Clinical and microbiologic findings in dogs with bronchoscopically diagnosed tracheal collapse: 37 cases (1990–1995). *J Am Vet Med Assoc* 2001; 219(9):1247–1250.
- Bay JD, Johnson LR. Feline bronchial disease/asthma, In: King LG. ed. *Textbook of Respiratory Disease in Dogs and Cats*. St Louis, MO: Saunders Elsevier; 2004, pp. 388–396.
- Corcoran BM, Foster DJ, Fuentes VL. Feline asthma syndrome: a retrospective study of the clinical presentation in 29 cats. *J Small Anim Pract* 1995; 36(11):481–488.
- Powell LL, Rozanski EA, Tidwell AS, Rush JE. A retrospective analysis of pulmonary contusion secondary to motor vehicular accidents in 143 dogs: 1994–1997. *J Vet Emerg Crit Care* 1999; 9(3):127–136.

46. Myer W. Radiographic review: the alveolar pattern of pulmonary disease. *J Am Vet Radiol Soc* 1979; 13:4–17.
47. Suter PF, Lord PF. Radiographic differentiation of disseminated pulmonary parenchymal diseases in dogs and cats. *Vet Clin North Am* 1974; 4(4):687–710.
48. Kogan DA, Johnson LR, Jandrey KE, et al. Clinical, clinicopathologic, and radiographic findings in dogs with aspiration pneumonia: 88 cases (2004–2006). *J Am Vet Med Assoc* 2008; 233(11):1742–1747.
49. Kallet RH, Hemphill JC III, Dicker RA, et al. The spontaneous breathing pattern and work of breathing of patients with acute respiratory distress syndrome and acute lung injury. *Respir Care* 2007; 52(8):989–995.
50. Johnson LR. Pulmonary thromboembolism. In: Silverstein DC, Hopper K. eds. *Small Animal Critical Care Medicine*. St Louis: Saunders Elsevier; 2009, pp. 114–117.
51. Martin MW, Stafford Johnson MJ, Celona B. Canine dilated cardiomyopathy: a retrospective study of signalment, presentation and clinical findings in 369 cases. *J Small Anim Pract* 2009; 50(1):23–29.
52. Jameson PH, King LA, Lappin MR, Jones RL. Comparison of clinical signs, diagnostic findings, organisms isolated, and clinical outcome in dogs with bacterial pneumonia: 93 cases (1986–1991). *J Am Vet Med Assoc* 1995; 206(2):206–209.
53. Thayer GW, Robinson SK. Bacterial bronchopneumonia in the dog: a review of 42 cases. *J Am Anim Hosp Assoc* 1984; 20:731–735.
54. Crowe DT. Traumatic pulmonary contusions, hematomas, pseudocysts, and acute respiratory distress syndrome: an update-Part I. *Compend Contin Educ Pract Vet* 1983; 5(5):396–402.
55. Hughes D. Pulmonary edema. In: King LA. ed. *Textbook of Respiratory Disease in Dogs and Cats*. St Louis, MO: Saunders Elsevier; 2004, pp. 487–497.
56. Hall DJ, Shofer F, Meier CK, Sleeper MM. Pericardial effusion in cats: a retrospective study of clinical findings and outcome in 146 cats. *J Vet Intern Med* 2007; 21(5):1002–1007.
57. Anderson M, Payne JT, Mann FA. Flail chest: pathophysiology, treatment and prognosis. *Compend Contin Educ Pract Vet* 1993; 15:65–74.
58. Scheepens ET, Peeters ME, L'eplattenier HF, Kirpensteijn J. Thoracic bite trauma in dogs: a comparison of clinical and radiological parameters with surgical results. *J Small Anim Pract* 2006; 47(12):721–726.
59. Cappello M, Legrand A, De Troyer A. Determinants of rib motion in flail chest. *Am J Respir Crit Care Med* 1999; 159(3):886–891.
60. Sigrist NE. Thoracocentesis. In: Silverstein DC, Hopper K. eds. *Small Animal Intensive Care Medicine*, 1st ed. St Louis, MO: Saunders Elsevier; 2009, pp. 131–133.
61. Gordon CE, Feller-Kopman D, Balk EM, Smetana GW. Pneumothorax following thoracocentesis: a systematic review and meta-analysis. *Arch Intern Med* 2010; 170(4):332–339.
62. Miserocchi G. Role of peripheral and central chemosensitive afferents in the control of depth and frequency of breathing. *Respir Physiol* 1976; 26(1):101–111.