THE APPROACH TO THE PATIENT IN RESPIRATORY DISTRESS

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Successfully managing an animal with severe respiratory distress is one of the greatest challenges we face as emergency clinicians. But the magnitude of the challenge means that it is also one of our most rewarding experiences. Successful management demands that we remain acutely aware of the fragility of the dyspnoeic patient. The stress of life-threatening disease coupled with transport and the unfamiliar surroundings of a noisy emergency clinic should never be underestimated. Even a brief evaluation of the patient may prove fatal, especially in cats, so the initial major body system assessment may have to be performed in stages. All dyspnoeic animals should immediately be given supplemental oxygen using the least stressful method available. Furthermore, prior to performing any diagnostic tests on the animal, the risks of any procedure should always be carefully weighed against the potential benefits. To appreciate the significance of this, ask yourself the following question: In your experience, what is the most common cause of death in dyspnoeic cats? Do they die spontaneously? Or do they die when something is being done to them? Unfortunately it is usually the latter which tells us that we all need to be very, very careful with this patient group. Many cases will stabilise to some degree with oxygen and stress reduction alone. So our greatest challenge with these patients is to have the confidence to do nothing other than give oxygen for a time while the animal stabilises even though our impulses are screaming for us to do something!

INITIAL EVALUATION AND PHYSICAL EXAMINATION

As previously mentioned, the dyspnoeic animal is an extremely delicate creature, many of them teetering on the line between life and death and they must be handled with great care. It is imperative that they are not stressed excessively; oxygen should be supplied immediately and examination in the first instance should be limited and directed at identification of the cause of dyspnoea.

On arrival at the practice it is likely that the animal will have suffered a car journey and be stressed at the unfamiliarity of the situation. If possible the patient should be provided with oxygen supplementation and a very brief examination of the respiratory tract should be carried out and then the patient left to relax (as much as possible). Remember that muscle activity greatly increases the oxygen consumption of skeletal muscles. And when that essential oxygen goes to the skeletal muscles instead of the heart and brain that is when you get a respiratory and then cardiac arrest. Do not make them do anything that makes their skeletal muscles work. You can very easily restrain them to death if you are not careful; or radiograph them to death; or blood sample them to death; or IV catheter them to death. You guys with me on this one? If they are really, really bad then you may have to be brave and sedate/anaesthetise/intubate them. Actively taking control of the airway (which often only requires very small doses of sedative) is vastly superior to tubing them following a respiratory arrest!

While they are getting some oxygen have someone get a capsule history with particular reference to pre-existing clinical signs or previous diagnoses, concurrent medication, history of trauma and the onset and progression of the condition. Does their signalment correspond to any breed predisposition? Giant breeds and DCM, old small breed dogs may have tracheal collapse or mitral valve disease especially if they are Cavaliers; older middle size dogs like Labs might have laryngeal paralysis. Recently kennelled is a no brainer for possible kennel cough. Very old animal makes neoplasia ascend your differential list. Most dogs with heart failure have premonitory signs like exercise intolerance, coughing, orthopnoea or overt dyspnoea but most new cases of heart failure in cats do not. Asthmatic cats almost always cough (if the owners can recognise it) but cats with heart disease do not. Many cats with heart disease are young whereas many dogs are old. If they’ve been vomiting then aspiration pneumonia is higher on your list. If they’ve been regurgitating it is higher still. Bangs on the head, pulls on the choke chain, other head or neck trauma and, of course, electric cord bite may result in neurogenic pulmonary oedema. If there’s anticoagulant rodenticide anywhere in the animal’s vicinity them pulmonary haemorrhage and haemothorax may be your cause of dyspnoea. If they’ve licked some paraquat, you know why they are dyspnoic.

Anyway, now for a quick segue into anatomy before we continue our patient assessment:

Remember that the respiratory tract is split into five main regions for the purposes of localisation of the disease process: upper airways, lower airways, parenchyma, pleural space, chest wall and diaphragm. Your aim is to establish where the problem is as quickly as possible. The methods of stabilisation, diagnostic tests required,
underlying conditions, treatments and prognosis are all different for each area so localisation is of paramount importance.

Back to the animal: the first part of the evaluation of the respiratory tract should be to watch and listen without a stethoscope. The patient should be evaluated for: respiratory rate, respiratory effort, respiratory noise, respiratory pattern, abdominal movement.

Normal animals have respiratory rates of 15-30 breaths a minute and the majority of inspiration is due to diaphragmatic contraction, so you see little chest wall movement. As the diaphragm contracts the abdominal contents are pushed caudally and the abdominal wall moves out (passively). That means that in a normal animal the chest and abdomen both move out on inspiration. In cats and small dogs a very slight inward movement of the cranioventral thorax can be a normal finding.

Rate is self evident but don’t forget it. For effort, try to rate their respiratory effort as mild, moderate, severe or imminently life threatening. The postural manifestations of dyspnoea include an extended neck, abducted elbows, open mouth breathing, an anxious facial expression, a glazed-eyed stare, increased abdominal movement and paradoxical abdominal movement. Paradoxical abdominal movement is when the abdomen moves in, instead or out, on inspiration. It means that there is something preventing adequate lung inflation despite the outward movement of the chest. There are only a few possibilities: upper respiratory tract obstruction, diaphragmatic rupture or paralysis, decreased lung compliance and severe pleural effusions. Straightening of the neck and open mouth breathing occur in both dogs and cats, however, some other postural manifestations of more severe dyspnoea vary between species. Dogs prefer to stand with abducted elbows, while cats tend to sit in sternal recumbency. Constantly changing body position in cats implies a much worse degree of dyspnoea than it does in dogs. Lateral recumbency due to dyspnoea is a serious sign in a dog, however, it often means impending respiratory arrest in a cat. If you see a dyspnoeic cat’s pupils dilate significantly then it is respiratory arresting NOW! Also be aware that puppies can lie to you! Sometimes they do not show the same degree of difficulty breathing as an adult dog despite severe lung problems.

To evaluate the respiratory pattern watch the timing of inspiration and expiration (and a pause in between if they are breathing normally). Count: in, in, in, in to yourself (or out loud) as the animal inspires. Continue until you are confident that you have correctly identified when the animal is breathing in and when it is breathing out. Next, compare the time spent on each phase compared to normal. If one particular phase is longer than normal then this is the one that is causing the animal the most difficulty and we can then characterise the dyspnoea as inspiratory, expiratory, or both.

**Inspiratory dyspnoea** (more difficulty breathing in) with a short expiratory phase and with stridor or stertor is associated with a dynamic upper airway obstruction (most commonly in dogs and usually due to laryngeal paralysis). Some cats with severe, chronic, pleural effusions may have an inspiratory dyspnoea but without stertor. An expiratory dyspnoea (an expiratory push) may be seen in some cats with feline allergic airway disease. An increase in both inspiratory and expiratory effort can be seen with a fixed (i.e. not dynamic) upper airway obstruction e.g. granulomatous laryngitis in cats or laryngeal neoplasia in either species or a ball occluding the pharynx. A fixed upper airway obstruction is rare but it is vital that you recognise this pattern because these animals with go from alive to dead very rapidly if you mess with them without sorting out the obstruction first. Most other causes of dyspnoea will be associated with tachypnoea and a mixed respiratory pattern. Short shallow respiration may be seen in some animals with pleural space disease but this finding is obviously not specific for pleural space disease. Be careful because some animals with severe pleural space disease may only show tachypnoea and shallow respiratory movements.

**PULMONARY AUSCULTATION**

Auscultation is one of the true arts of veterinary medicine but it can be learnt and perfected with some diligence and perseverance. It requires a methodical approach and a decent stethoscope. You have to make a serious effort: lackadaisical auscultations are tantamount to useless. But with dedication, many respiratory abnormalities can be differentiated on physical examination alone, especially in cats.

The easiest way to ensure a relatively complete auscultation is to divide the chest into a noughts and crosses board i.e. 9 smaller fields, and then to auscult each square. This enables comparison of dorsal, middle and ventral aspects.
of the cranial, middle and caudal lung fields. For a complete auscultation each individual stethoscope field should be
examined (if the patient is sufficiently stable). Lung sounds should be compared between different areas on the same
side of the chest and to the same area on the opposite side. Lung sounds are normally slightly louder and coarser in
the cranioventral lung fields compared to the dorsocaudal fields. In some large breed dogs and in dogs or cats taking
very shallow breaths, it can be difficult to hear lung sounds in the caudodorsal chest. Normal lung sounds are always
symmetrical when the same area is compared on both sides of the chest except for the area of cardiac dullness in the
cranial portion of the left ventral chest. This means that, regardless of whether one can determine which is the louder
or quieter side, any asymmetry is abnormal. You should also cross reference at all stages with respect to what you
would expect to hear given the tidal volume of the animal. An increased tidal volume per se will cause louder lung
sounds. So an animal that is breathing faster and deeper due to stress for example should have louder lung sounds.
Take a dog that has been hit by a car. If it is just tachypnoeic from pain then it will have increased lung sounds that
are symmetrical and have the normal difference between the dorsocaudal and cranioventral lung fields. If the
dorsocaudal fields are quieter than they should be then there may be a pneumothorax. Contusions make the lung
sounds coarser than normal or cause crackles or both. If you hear coarse lung sounds, are they coarser and louder
than they should be for the dog’s respiratory rate and tidal volume?

In human medicine, adventitious lung sounds are classified as either rales (crackles) or rhonchi (wheezes) and then
subdivided on their acoustic nature and the various subgroups have diagnostic relevance. In small animals, we are
not so lucky. I personally think that the term “wheeze” is rather vague and confusing so I don’t use it. One person’s
wheeze is another person’s whistle! Occasionally, asthmatic cats and animals with other processes which narrow the
conducting airways generate true wheezes but many have only harsh lung sounds. I attempt to classify abnormal
lung sounds into two groups: Harsh lung sounds i.e. louder and coarser than normal and Crackles- which can be
either fine or coarse.

**Harsh lung sounds** can be caused by parenchymal or airway disease. Somewhat surprisingly, many dogs with
pneumonia or pulmonary contusions exhibit harsh lung sounds but not crackles. **Pulmonary crackles:** To hear
crackles, the animal must be taking sufficiently deep breaths to inflate the lung. Consequently, they are usually
loudest at the end of inspiration. **Fine crackles** are usually only heard at the very end of inspiration and are probably
generated by the opening of collapsed small airways. These are the ones you hear in sixteen year old Poodles with
no parenchymal disease! In contrast **coarse crackles** are usually associated with parenchymal disease but
occasionally can be due to airway disease. In my experience the most severe airway crackles occur with eosinophilic
bronchitis in dogs. Nevertheless, if you hear coarse crackles, it is most likely that the animal has a fluid build up of
some sort in its lungs. By auscultation you cannot tell whether that fluid is blood, exudeate from pneumonia,
hydrostatic oedema from left heart failure or fluid overload, neoplasia related fluid, or neurogenic pulmonary
oedema.

The distribution of the abnormal (adventitious) lung sounds can provide supportive evidence as to the cause of the
disease. A cranioventral distribution of crackles or harshness in dogs can be appreciated in many dogs with
aspiration pneumonia. Cardiogenic oedema may sometimes be associated with sounds loudest over the heart base.
Neurogenic oedema (which is seen most commonly after head or cervical trauma, seizures, upper respiratory tract
obstruction and electric cord bite) results in either a caudodorsal or a generalised distribution of harshness/crackles.

Pleural space disease is associated with an absence of lung sounds in the affected area. The pattern of dullness
provides information as to the possible cause:

- Ventral dullness- fluid or soft tissue
- Dorsal dullness- pneumothorax
- Gut sounds may be heard with diaphragmatic rupture
- Decreased thoracic compliance may be apparent with intrathoracic masses and sometimes pleural effusion.

Pleural effusion allows the lungs to float into the dorsal aspect of the chest cavity so there is an absence of ventral
lung sounds and the dorsal sounds are often harsh. Don’t be fooled by the heart sounds in cats with pleural effusion:
they may not be muffled and occasionally can radiate over a larger area of the chest than normal. In contrast to
pleural effusion, pneumothorax results in muffling of the lung sounds in the dorsal pleural space as air accumulates
in this area. Most people find pleural effusion easier to detect by auscultation than pneumothorax because the
distribution of lung sounds (quiet ventrally and harsh dorsally) is the opposite of normal. Many dogs with
pneumothorax after being hit by a car also have pulmonary contusions that can seriously complicate the auscultation. The pneumothorax dampens lung sounds, whereas the pulmonary contusions make them louder and coarser. This can sometimes result in an absolute volume close to normal. With practice, one can appreciate that the lung sounds are both harsh and muffled; however, the severe dyspnoea in such a patient with normal volume lung sounds should point towards concurrent pulmonary contusions and pneumothorax.

PUTTING IT ALL TOGETHER
The ability to establish a working diagnosis and treat on the basis of history and physical examination without additional diagnostics, such as chest radiographs, can mean the difference between life and death in some dyspnoeic animals. As we said earlier, an immense amount of information can be obtained by simply watching the animal breathe in the oxygen cage. Look at the animal’s body condition (for a clue on chronicity) in conjunction with the history. Also watch the degree of distress the animal is experiencing relative to the degree of chest movement. Large chest excursions but a relatively undistressed patient speaks for chronic rather than acute disease. For example, a young cat in good body condition with a history of coughing and a mixed dyspnoea with increased effort on expiration is more likely to have feline asthma. Although chest radiographs would be necessary to be sure, harsh lung sounds in all fields and the absence of a heart murmur or gallop would just about clinch the diagnosis of asthma in most situations. An underweight, old cat with lots of chest movement, an inspiratory dyspnoea without upper airway noise and dull ventral lung sounds has a pleural effusion until proven otherwise.

A WORD ON EMPIRICAL TREATMENT
Some purists may sneer but they will likely be purists with a lower overall survival rate! When empirical treatment must be instituted prior to a definitive diagnosis, good clinical reasoning and maintaining perspective as to the likely differential diagnoses is tantamount. The vast majority of cats that present for dyspnoea have a pleural effusion, heart disease, or asthma. The clinical findings in each of these conditions are often distinct. A severely dyspnoeic cat with a heart murmur or gallop rhythm and diffuse bilateral crackles will usually have cardiomyopathy and the benefits of intravenous or intramuscular furosemide almost always outweigh the potential risks. As previously mentioned, pleural effusion results in quiet ventral lung sounds and harsh dorsal sounds whereas most asthmatic cats have lung sounds which are harsh in all fields and a concurrent history of coughing (and hopefully not an incidental heart murmur!). Some cats may be so dyspnoeic that virtually any handling outside of 100% oxygen proves fatal. In these cases it is not unreasonable to treat for potential pulmonary oedema and asthma with furosemide and an injectable, fast acting corticosteroid such as dexamethasone prior to establishing a definitive diagnosis. Another example of maintaining perspective as to the most likely diagnoses is in the puppy with dyspnoea. Many 2-6 month old puppies have neurogenic oedema, rodenticide intoxication, or occasionally pneumonia following kennel cough or distemper virus infection. Although there is no replacement for following the problem-oriented approach with a complete problem list and all diagnostic differentials, the emergency clinical must always maintain perspective as to what are the most likely probable diagnoses.