Most cases of airway and lung parenchymal disease in horses can be readily managed in general practice. Specialist equipment and facilities for diagnosis and intensive care are rarely required. Animals with non-responsive respiratory distress, hypoxaemia and/or cyanosis, those requiring thoracic radiography for diagnostic purposes and animals where thoracic drainage, pleuroscopy or intrathoracic surgery are indicated will require hospitalisation. In field equine practice, four common scenarios lead to problems causing frustration to both owner/trainer as well as the attending Veterinarian.

1. Poorly or incompletely responsive Recurrent Airway Obstruction (RAO/COPD) or Summer Pasture-Associated Obstructive Disease (SPAOPD)
2. Apparently non-responsive Inflammatory Airway Disease (IAD)
3. The persistent cougher
4. The poorly performing horse where IAD has been diagnosed as the cause
5. Suspected chronic parenchymal and/or pleural disease

GENERAL APPROACHES

1. Make no assumptions; start investigation of the case again from the beginning
2. Take a very detailed history, again
3. Carefully inspect the animal’s environment, feeding and stable management.
4. Are you and the client sure what the problem actually is?
5. Can you see / demonstrate the client’s real or perceived problem?
6. Perform a very thorough clinical examination including use of a rebreathing bag and trust your clinical judgement regarding abnormalities as well listening to the owner’s assessment of the case.
7. Is there evidence of systemic disease as well as respiratory tract disease
8. See the horse lunged and ridden and if necessary worked to its expected level of performance
9. Obtain baseline clinical pathology information as required – haematology, total serum protein, albumin, globulin and acute phase proteins (fibrinogen ± serum amyloid A), tracheal aspirate/wash cytology & bacteriological culture and bronchoalveolar lavage fluid (BALF) cytology (if possible use the same laboratory as any previous sampling and request cell differentials as well as cytological interpretation)
10. REMEMBER NEUTROPHILS IN AIRWAY SECRETIONS DO NOT NECESSARILY EQUATE WITH INFECTION!

I. POORLY OR INCOMPLETELY RESPONSIVE RAO/COPD OR SPAOPD
The animal continues to show compromise to lung function (“heavey”) ± cough ± nasal discharge and/or exercise intolerance despite apparently appropriate management / environment / therapeutic intervention. Diagnosis of RAO must be based on the following criteria:

1. Clinical signs of airway disease – abnormal lung function and cough which is a highly sensitive indicator of lower airway inflammation
2. Recurrent reversible episodes of lower airway obstructive disease induced by environmental challenge.

3. Airway obstruction is reversible by use of bronchodilators or movement to a non-challenge (organic-dust free) environment i.e. pasture in the case of RAO.

4. >5% neutrophils on differential BALF cytology (N.B. % of neutrophils in BALF shows a poor correlation with disease severity). Many horses with active RAO will have very high neutrophil proportions (>90%) but some cases can be very heavy with much lower neutrophil proportions.

5. Disease usually develops during periods of increased organic dust exposure e.g. stabling in winter.

The duration and extent of exposure required to induce disease in a particular animal is highly variable due to variations in magnitude of respirable dust exposure, pro-inflammatory content of dust and inherent susceptibility of animal.

**Reasons for treatment / management failure**

1. Incorrect diagnosis – follow general approach
2. Diagnosis of RAO has not been made
3. Poor environmental management with ongoing organic dust exposure
4. Inappropriate management and treatment (inappropriate use of inhaled medication, failure to use corticosteroid therapy effectively)
5. Chronic irreversible airway remodelling / pathology e.g. smooth muscle hypertrophy, fibrosis, bronchiectasis

**1. Incorrect diagnosis**

The horse does not have RAO despite the presence persistent low grade dyspnoea, increased expiratory effort, cough and/or nasal discharge. Diagnostic clues that RAO is not the problem might include:

- Asymmetrical auscultation findings especially quiet areas of lung particularly when a rebreathing bag is employed
- Change in appetite, demeanour an/or subtle weight loss
- Presence of fever and/or response to inflammation/infection on blood tests (e.g. increases in leucocyte counts, total serum protein, globulin and fibrinogen)
- Other possible causes are chronic pleural or parenchymal disease most commonly affecting the interstitium including interstitial inflammation, fibrosis, neoplasia and rarely occult congestive cardiac failure.

**2. Diagnosis of airway inflammation not made**

Middle-aged and older sport and race horses where RAO was not considered a likely diagnosis. A small sub-group of animals with RAO or on occasion after severe IAD fail to fully gain remission / recovery. They are often presented with poor performance, exercise intolerance or slow recovery from exercise. Clinical examination and tracheal cytology at rest may be unremarkable. Routine cytology of BALF may be equivocal. BUT immediate close inspection of harvested BALF reveals mucus flecks or plugs which may be packed with PMN when examined microscopically. These mucus plugs are often removed during routine BALF preparation in the laboratory or missed on cytological examination.

**3. Poor environmental management**

The owner and sometimes the veterinarian think that air hygiene management is satisfactory but have failed to spot a simple management error. There is great value in personally inspecting the premises, turn out paddocks & stable routine. In RAO cases the most significant source of inhaled antigens / organic dust arises in the 30 cm “breathing zone” around an animal’s muzzle. Common errors in management include:

- Horse is very sensitive to organic dusts i.e. a fast responder; there is great individual variation (hours – weeks)
- Remote organic dust sources (dust, dung, deep litter, communal supplementary dry forage feeding)
- Bedding/forage in adjacent stables
- Use of soaked hay may not be an adequate means of controlling dust exposure as it may dry out; consider a change to haylage.
e. Some SPAOPD cases can only be managed with aggressive corticosteroid therapy and may need to be moved to a completely new environment in another geographic location to see improvement without ongoing corticosteroid therapy.

4. Inappropriate management / treatment

a. Always check the air hygiene management (see above).

b. Manage client expectations; some RAO cases may take 3 months or more to achieve remission – Mucus hypersecretion can persist for at least 3 months and may never be normal in some animals. Some animals may have persistent peripheral airway obstruction due to irreversible pathology (see below).

c. Incorrect delivery of inhaled medication (see below).

d. Inappropriate use of inhaled medications in animals with significant dyspnoea and hence airway obstruction due to bronchoconstriction, airway narrowing and airway plugging with mucus and inflammatory debris i.e. inadequate penetration of inhaled medications.

e. Incorrect dose or drug choice for inhaled medication – with recent evidence of slow but satisfactory clinical improvement with low dose inhaled medication (beclomethasone, 2 puffs, BID) treatment failure is unlikely to be due to incorrect drug selection.

f. Inappropriate spacer use – some animals appear to respond better to use of equine specific spacers vs generic human paediatric spacers.

g. Although clenbuterol has a significant anti-inflammatory effect as well as being a bronchodilator, the clinical response varies at standard doses due to variable bioavailability. The response may improve with upward flexible dosing (clinical response rate reported 25% at 0.8 μg/Kg, 50% at 1.6 μg/Kg 75% at 3.2 μg/Kg) but this can become expensive.

h. Systemic steroids (prednisolone 1-2 mg/Kg, dexamethasone 0.05 – 0.1 IV or 0.053 – 0.16 mg/Kg PO, once daily in the morning) are often required to at least stabilise a patient. Only milder SPAOPD cases can be managed without systemic steroids in my hands.

i. When first presented with a significantly heavy horse an intravenous atropine sulphate response test can be useful both as rescue therapy and to assess the proportion of airway obstruction related to bronchoconstriction and inflammation-associated airway narrowing respectively.

Good practice in metered dose inhaler & spacer use

• Keep spacer clean & dry – static!
• Warm MDI before use (pocket or in shirt)
• Shake well - 30 s
• Waste 1st puff
• Keep vertical during use
• Apply mask / nasal cone tightly to try to achieve an airtight seal
• 1- 2 puffs late exp’n / early insp’n
• ? obstruct other nostril if not using mask
• Allow 2-3 complete breath cycles
• Count actuations used!
• Wash spacer weekly with detergent, do not rinse and drip dry to reduce static.

5. Chronic irreversible lung remodelling / pathology - e.g. smooth muscle hypertrophy, fibrosis, bronchiectasis. Having ruled out all possible causes of treatment / management failure, review diagnosis, then ultrasound visceral pleural surfaces and heart, radiograph chest and consider lung biopsy. These cases are in my experience very rare. Radiography can be helpful but may require a specialist opinion; biopsy can be confusing with regard to prognosis. The absence of a clinical response to aggressive systemic corticosteroid therapy adds support to such a diagnosis.
II. THE PERSISTENT COUGHER

Management of persistently coughing horses is challenging to say the least becoming expensive and frustrating. The most common cases are younger horses (young sport and race horses entering training) with persistent IAD that defies treatment. An attempt to confirm a specific diagnosis or perhaps more importantly to rule out other causes beyond persistent airway inflammation should be made. Recourse to the diagnostic tree above often including lung ultrasonography and radiography may help.

Consideration of the causes of coughing and the response of the lung to inflammatory stimuli may help to develop a suitable treatment plan. The presence or absence of coughing in response to a rebreathing bag is a simple and cheap way of assessing progress. Some degree of inflammatory airway wall remodelling may persist for long periods. Detection of increased bronchial hyperresponsiveness, with for example open plethysmography and inhaled methacholine challenge, may not alter your treatment but may help the client understand the nature of the problem and provide a benchmark for ongoing response to treatment. If persistent IAD remains the working diagnosis I have used the following:

a. Scrupulous air hygiene or turn out to pasture 24/7
b. Prolonged rest or limitation of exercise to a level that does not induce coughing
c. Avoid exercise in cold weather and dry dusty hot conditions
d. Initial prolonged antibiotic therapy (potentiated sulphonamides or tetracyclines)
e. Oral clenbuterol at least in the early weeks - months
f. Inhaled steroids ± salbutamol pre-exercise concurrent with antibiotic therapy if there is great pressure to return to full training rapidly
g. Systemic steroids (Dexamethasone intravenously followed by prednisolone)
h. Potassium Iodide daily PO as required or sodium iodide IV twice weekly particularly if excess mucus present
i. Nebulisation with clenbuterol ± dexamethasone – unsure of value over combined MDI and oral therapy.
j. Interferon - unhelpful in my hands

Other more unusual causes of chronic coughing such as chronic upper respiratory tract inflammation / infection and low grade dysphagia should be ruled out.

III. UNRESPONSIVE POOR PERFORMANCE CASE WHERE IAD DIAGNOSED

80% of Thoroughbred racehorses have more than one abnormality when investigated for poor performance with the usual ranking of musculoskeletal disease followed by respiratory tract disease. Many reported referral populations are heavily biased to particular body systems making the need for a complete and thorough investigation necessary. In high performance speed athletes all abnormal systems should be addressed aggressively if finances allow. Recent work demonstrating significant skeletal muscle pathology in RAO-affected horses vs controls raises the potential for significant airway disease to have effects of skeletal muscle that may effect performance for a period after successful management of the primary respiratory disease. Whether this could occur in severe or protracted IAD cases is unknown. IAD will lead to a significant, if on occasion subtle, reduction in high speed performance and investigation via endoscopy, tracheal and if necessary bronchoalveolar lavage cytology and culture should be followed.

The diagnosis of the cause of poor performance in sport and leisure horse may be more challenging as there may also mild abnormalities of several body systems. Moreover, detailed investigation to facilitate a precise diagnosis is very important as any findings may be significant with reference to future performance in animals with a long athletic career. However in some sport horse populations there is a very high prevalence of IAD even in animals with NO performance problems. Airway disease is a very easy diagnosis
to make in these animals but can be regretted as other body systems may be more significant such as low grade musculoskeletal disease. Especially in pleasure horses, seeing the animal work can be very revealing. If exercise tolerance is poor then unless airway disease is severe, other causes of even dyspnoea during, or prolonged recovery after, exercise should be sought. In my hands most commonly pain (musculoskeletal or gastric in origin) is a more likely explanation. Cardiac problems, although still rare, are more common in the older sport horse.

IV. CHRONIC PARENCHYMAL AND/OR PLEURAL DISEASE

Occasionally lung abscessation, inhaled foreign bodies and chronic or focal pleuropneumonia can be present with only low grade chronic respiratory signs. Malaise, intermittent low grade pyrexia and mild weight loss may be noted. Thorough clinical examination, laboratory testing, endoscopy and thoracic ultrasound will usually reveal the cause. Although seen in all age groups, the more unusual cases of non-septic pleural or parenchymal / interstitial disease are more often seen in middle aged to older horses. Although they may present with cough, nasal discharge, mild fever and/or weight loss, changes in respiratory rate, pattern or effort are typical. Often in the early stages they are misdiagnosed as RAO or IAD. Again careful clinical examination, laboratory testing, endoscopy, thoracic ultrasound and on occasion radiography and transthoracic or transbronchial biopsy may be required. Some cases will be discussed.