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Inflammatory Airway Disease

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Inflammatory airway disease (IAD) is second only to musculoskeletal disorders as the most common cause of poor performance, interruption to training, lost training days, and premature retirement in racehorses. The etiology, pathophysiology and diagnosis of this disorder are currently controversial, and the term probably encompasses a number of different diseases that are associated with lower airway inflammation in young and older performance horses. But what definitely isn't under any doubt is that it is something that is an ongoing battle for veterinarians and trainers alike when treating young horses in training.

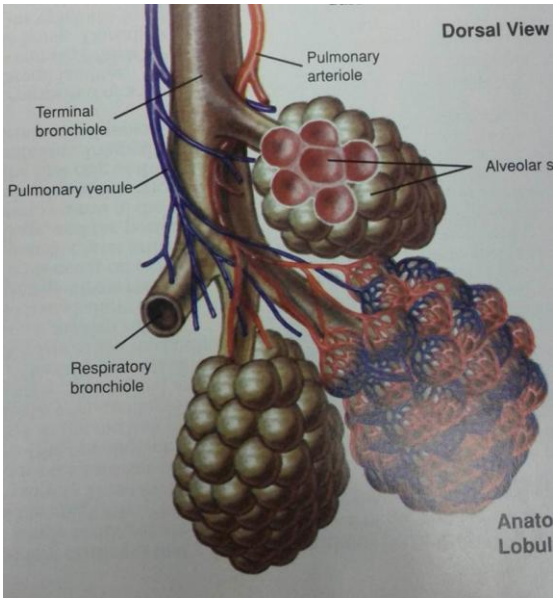
WHAT IS IAD?

Inflammatory airway disease is a broad term, simply referring to inflammation of the lower airways. This inflammation leads to increased mucus production and 'shutting down' of the airways (bronchoconstriction), leading to reduced capacity to take in oxygen. However, not so simple is the relationship between causative factors and the inter relationship between the individual horse, the environment, the involvement of possible infectious agents (bacteria, viruses, mycoplasma, allergens and other inhaled irritants) and the different type of cell populations involved that invade the lower airways.

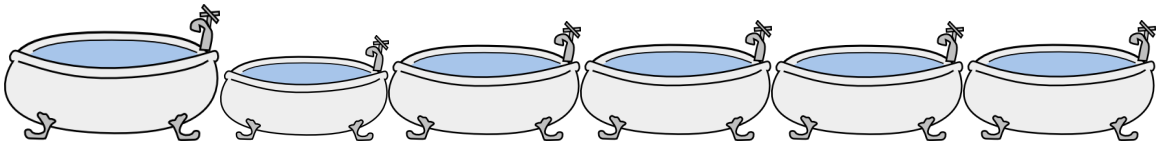
WHY IS A TINY REDUCTION IN ABILITY TO OBTAIN OXYGEN SO IMPORTANT??

THE NORMAL LUNG

The ultimate limiting factor in a horse's ability to perform (albeit everything else being well!) is obtaining oxygen. *THIS IS **THE** RATE LIMITING STEP*. In the healthy lung, tiny end stage capillaries communicate with the end stage air sacs, the alveoli, where gas exchange occurs and carbon dioxide is removed and oxygen supplied.



By the end of a 1000m sprint, the horse will have moved 1800litres of air into and out of the lungs. This is 6 bath tubs!



This is the same as moving 40 litres of air into and out of the lung every second. 70% of the energy needed for the 5 furlongs comes from **AEROBIC** metabolism. The remaining 30% comes from **ANAEROBIC** metabolism. The anaerobic system is fast but very inefficient due to the build up of the waste product lactic acid. The body has a self limiting mechanism to stop the horse running at maximum for long periods under anaerobic metabolism, thus preventing damage to the body.
The harder a horse works, the more oxygen it requires. If a horse doubles its speed, its oxygen requirements double.

If you were to lay out all the tiny airsacs/alveoli of one horse, they would cover 6 tennis courts!

The respiratory system has to move oxygen from the outside world to the lungs, where it is transferred into the blood in exchange for the waste product of metabolism, carbon dioxide. The oxygen binds to the red blood cells via a molecule called haemaglobin. In this way, oxygen is then transported to where it is needed. The cells take up the oxygen where specialized 'mitochondria' utilize the oxygen to produce the unit of energy, ATP.
Aerobic metabolism produces **20 TIMES** more energy from glycogen than anaerobic metabolism. By the time the oxygen reaches the mitochondria it is only approx.1/80th of the level of oxygen in the outside world. Therefore there is not much room for error!!

Being as efficient as possible in oxygen-carbon dioxide exchange is vital to a horse's performance.

As well as gas exchange the lung has several other functions:

Filter: All blood circulation passes through the lungs, therefore the lungs are ideal to act as a filter, removing small blood clots (thrombi) and air bubbles (emboli).

Endocrine function: The lung can activate or deactivate certain hormones that are in circulation.

Thermoregulation: Respiratory heat loss is a very important thermoregulatory mechanism for the horse. Horses' blowing after hard exercise is related to how hot they are, not blood oxygen levels. Blood oxygen levels will fall during periods of intense exercise, but as soon as the horse is pulling up, they will have returned to normal, and I fact, go to just above resting normal values. How hard a horse is blowing is not an indicator of fitness. It is a thermoregulatory issue.

You cannot train the respiratory system. The amount of air moved in and out by an unfit horse is the same 6 months later once the horse is fit. But you can influence and optimize **VO2 MAX** – the maximum oxygen uptake or consumption.

The average man has a VO2 Max of 35-40ml/kg/min.

Miguel Indurain, 5 times Tour De France winner had a value of 88 at his peak.

A thoroughbred horse in training averages 180.

A husky sled dog can peak at 240.

The main way to influence VO2 Max is by being able to supply oxygen, again reiterating how important the respiratory system is to performance.

Interface to the outside world: the lungs, skin and gastrointestinal system are the body's interface with the outside world. Because of this they have highly developed immune systems.

BASIC PATHOLOGY OF IAD

There are many potential causative agents that can trigger IAD. Each ones relative contribution varies amongst different populations of horses, based on feeding, housing, preventative measures taken and the difference in distribution of potentially involved infectious agents. There is also a genetic component.

The end stage result of these factors is **bronchoconstriction**, **decreased functioning of the mucociliary clearance system** and **increased mucus production**. Secondary infection can then also be involved. These all then result in decreased ability to gain oxygen.

Bronchoconstriction

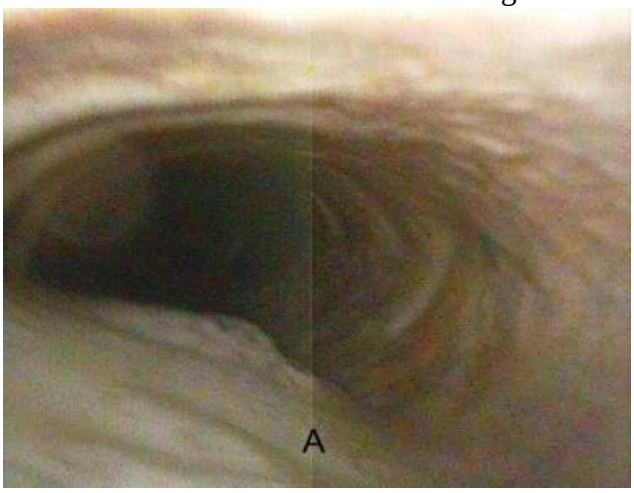
This is narrowing and sometimes complete closure of the small airways within the lungs. They are lined by smooth muscle which is triggered in IAD, by chemical mediators, to contract, leading to bronchoconstriction.

Mucociliary clearance system.

The respiratory system is lined by specialized cells which have hair like protrusions called cilia. These cilia can be imagined to be like sea coral or like people in a stadium performing a 'mexican wave'. Their function is to sweep debris and mucus up the

respiratory tree, to keep the small airways clear. When aggravated, the cilia slow down. Certain infectious and inflammatory agents can also damage the cilia directly.

Mucus production
The whole of the tracheal-bronchial tree is lined by liquid containing mucus. The mucus is produced by special Goblet cells, which are stimulated by respiratory irritants. Long term stimulation leads to increased numbers of Goblet cells. This increased mucus then clogs up the airways and cannot be cleared due to the damage to the cilia.



Increased mucus in the trachea, seen on endoscopic examination

INCIDENCE
Based upon certain criteria, IAD occurs in 12-50% of horses in training. One study in the UK showed a prevalence of 13.8% with an incidence of 8.9 cases per 100 horses per month. The mean duration of the disease was 7.8 weeks. Disease duration can be anywhere from 4-22 weeks. In many cases IAD is subclinical and goes undiagnosed, being the 'Hidden Handicap' of training. Remember IAD can be on going, it isn't always 'cured'. As long as the irritants are present the horse will continue to suffer if not treated, hence why symptoms often return once treatment is stopped.
IAD can account for approx. 20% of lost training days.

RISK FACTORS
Certain management and training practices can predispose horses to IAD.
These include:
High level exercise
Transportation/traveling
Co mingling with other horses
EIPH
Housing in stables/barn design/ airflow in the stables and barns
Age

CAUSATIVE AGENTS

These fall into two main categories, infectious and non-infectious.

Infectious agents

Viruses (Influenza, herpes, rhino virus)

There is very little evidence that there is a direct link between viruses and IAD, but respiratory viruses do alter the respiratory defense mechanisms against bacterial infections, and they can cause prolonged periods of inflammation and hyper reactivity of the airways. But viruses are rarely directly associated with IAD.

Bacteria

These have been shown to be important contributors to IAD. This is why it is important to quickly recognize and treat secondary infection.

Mycoplasma

These have been shown to be involved in IAD in some cases and some regions of the world, but they can also be part of the residential/commensal population of bugs that live in the respiratory tract on a day to day basis, causing no harm.

Parasites

Due to good worming strategies, parasites are unlikely to be involved in IAD, but they must not be ruled out. The main parasites are *Parascaris Equorum* and *Dictyocaulus Arnifieldi.*

Non infectious Agents

Environmental

Horses, when stabled are constantly challenged by airborne dust. The dust may contain bacteria, viruses, moulds, mite debris, plant material, ammonia compounds, to name just a few of the air contaminants. These airborne compounds can potentially induce airway inflammation by initiating infection, by inducing allergies, by direct toxicity, or by overwhelming pulmonary/lung defense mechanisms.

Air hygiene standards for human cotton mill workers, require dust levels to be < 2.5mg/m cubed.

Dust levels in the corridors of horse barns is on average 3mg/m cubed.

Levels within the horses breathing zone can be 20 x this.

When mucking out, levels reach 10-15mg.

Exercise

Exercise alone can cause inflammation. Very strenuous exercise also impairs lung defense mechanisms.

Air Temperature

Cold air causes bronchoconstriction and mucosal sloughing, both leading to inflammation.

CLINICAL SIGNS

IAD signs are often very subtle, and are easily overlooked.
Coughing only occurs in approx. one third of horses affected.
Mild serous or mucupurulent **nasal discharge** can be observed, but again isn't always present. Horses may have prolonged recovery rates also. Some trainers often comment that the horses 'pull' or become anxious in their work, and some claim coat alterations.
Abnormal breathing sounds are rarely picked up by the vet when auscultating the chest. The use of a re-breathing bag though will often elicit a cough response and a rapid increase in inspiratory and expiratory effort. But, **reduced exercise tolerance and poor performance** are often the only signs suggesting IAD. This is when further diagnostic tests are required.

DIAGNOSIS

Endoscopy and retrieval of a tracheal wash is the standard way to diagnose IAD. A tracheal wash or a **bronchoalveolar lavage** may be taken and there are pros and cons for each method.
Whilst scoping the horse, the trachea is examined for the presence of mucus, mucopurulent discharge and blood. Upon retrieval of the sample, it is visually assessed and also sent to the laboratory for cytology.
The tracheal wash should be performed approx one hour after exercise.
In the laboratory the sample is analyzed for the presence of mucus, epithelial cells, the number and ratio of types of different white blood cells present (neutrophils, macrophages, haemosiderophages, mast cells, eosinophils, lymphocytes), bacteria, and fungi. The lab can also test for the presence of DNA of mycoplasma and pneumocystis.
If severe infection is suspected, the sample can be cultured in an attempt to grow and identify the offending organism.

TREATMENT

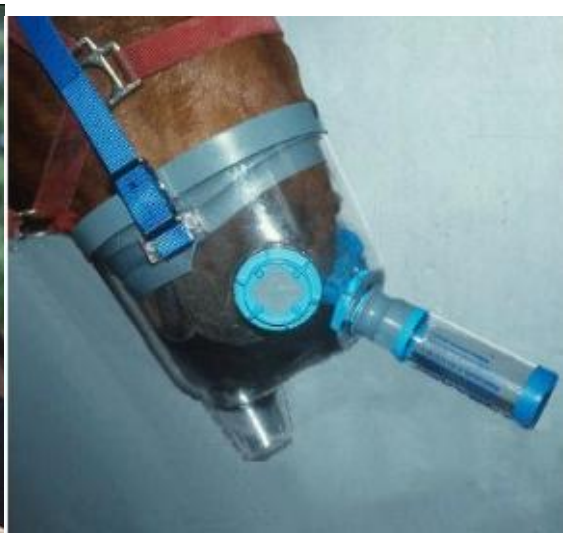
The main aims of treatment are to:
Settle down inflammation
Relieve/prevent bronchoconstriction

Remove excess mucus
Treat secondary infections
Instill management systems to reduce the irritant load that the horse is exposed to

DRUG THERAPY

The mainstay of therapy is to settle down inflammation. Aside from management strategies, treating the inflammation is the crux of successful treatment. If secondary infection is present then this must be recognized and treated with **antimicrobials**. Different yards will have different 'bug' populations and therefore different successes with varying antibiotics.

The most successful anti inflammatories for IAD are **corticosteroids/cortisones**. These may be given intravenously, intramuscularly, orally or by inhalation. Each method has its own pros and cons. Treatment regimes should be discussed with your own vet. A common and very successful approach is to start the horse on a course of reducing injectable cortisone, then to follow this with an inhaled corticosteroid.



Bronchodilators ('Ventipulmin', ipratropium) will provide immediate relief as they relieve the bronchoconstriction. These should be used in conjunction with the cortisones. These can be given orally or inhaled. It may be of benefit to use an inhaled bronchodilator (salbutamol, salmeterol, 'Asthavent') prior to inhaled cortisones, in an attempt to open up the airways so that the steroid can reach a greater surface area of lung.

Mucolytics can be used to help remove the excess mucus. These can be given orally (Potassium iodide, 'Bromhexine') or as intravenous solutions (Sodium iodide drips).

Other treatments including sodium cromoglycate may be required in mast cell inflammation.

The key to successful treatment is early recognition, thorough treatment, and potential ongoing use of inhaled corticosteroids/cortisones, along with good management strategies.

MANAGEMENT STRATEGIES

Avoid dusty bedding.
Dampen feed and roughage down
Remove the horse from the stable when being mucked out
Do not have haystores/ bedding stores upwind from the stables
Remove cobwebs
Work horses with known IAD in later strings if working in a cold climate
Ensure good stabling/barn ventilation
At least once a year, steam clean all the stables and barns with a recognized disinfectant.
REST!!- The horse needs time to recover and time to respond to the drug therapy.