Avian Respiratory Diseases

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INTRODUCTION

The success of the treatment of respiratory diseases in birds is directly related to the speed at which an owner notices a problem and presents the bird to the clinician, who in turn makes a specific diagnosis and instigates appropriate therapy. In view of the inactivity of many pet psittacine birds, birds may often not be presented until severe dyspnoea is already present. It is important that a full clinical history is taken before the patient is examined. Information regarding the source of the bird, type of housing, diet, date of entry of last bird to the collection and any recent medical history of other members of the group, should be collected and considered. The correct diagnosis and treatment of avian respiratory disease is dependent on a full understanding of the anatomy and physiology of the avian respiratory system. This differs greatly from mammals, with the absence of a diaphragm (whose function is replaced by the intercostal and abdominal musculature) and the addition of a fixed lung (facilitating a thinner air - blood barrier), supplied with a constant unidirectional supply of air by the caudal air sacs, (acting as bellows). At rest, respiratory effort should not be noticeable and the mouth should remain closed. Normal resting respiratory rates are as listed in Table 1. Respiratory disease commonly causes excessive chest movement, nasal discharge, head or tail bobbing (frequently with open mouth rather than nasal breathing), abduction of the wings from the body, neck stretching, coughing, alteration of voice or, on occasions, vomition. Birds should be observed for all these signs prior to handling. The presenting signs are determined by the cause and the site of the pathology.

Table 1. Normal resting respiratory rates.

Weight of bird	Respiratory rate/min
100g	40 - 52
200g	35 - 50
300g	30 - 45
400g	25 - 30
500g	20 - 30
1000g	15 - 20

In order for the correct treatment to be given the site and aetiology of the disease must be accurately determined. There are many different causes of respiratory disease, the major causes are listed in Table 2 below.

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Table 2. Major Causes of Avian Respiratory Disease.

Bacterial (typically Gram -ve) *Klebsiella* spp., *Pasteurella* spp., *Pseudomonas* spp., *Bordetella* spp., *Salmonella* spp., *Haemophilus* spp., *Chlamydia psittaci*, *Mycobacterium avium* + others.	**Nutritional** Vitamin A deficiency, iodine deficiency (goitre), obesity
	Fungal *Aspergillus spp., Candida spp., Cryptococcus*spp..
Parasitic Tracheal mites, eg. *Sternostoma* spp.. Gape worms, eg. *Syngamus* spp.. Air sac worms, eg. *Serratospiculum* spp..	
	Toxic Teflon, creosote, tobacco, carbon monoxide, ammonia, aerosols + others.
Protozoa *Sarcocystis spp., Trichomonas spp.*. *Cryptosporidium* spp..	**Viral** Avipoxvirus, adenovirus, influenza virus, infectious bronchitis virus, paramyxovirus, laryngo-tracheitis virus.
Ruptured Air Sac	
Foreign bodies / aspiration pneumonia	*Mycoplasma* spp.
Metabolic / neoplasia / abdominal masses	

Initially one should differentiate between conditions of the upper respiratory tract (URT), including the nares and sinuses, and conditions of the lower respiratory tract (LRT), including the trachea, lung and air sacs (see Table 3).

Table 3. Differentiation between URT and LRT infection.

URT	LRT
Open-mouthed breathing.	Change of voice, dyspnoea, tail bobbing.
Nasal plugging, discharge, sneezing.	Inspiratory / expiratory difficulty, coughing.
Peri-orbital swelling, epiphora, head shaking.	Exercise intolerance.
Dyspnoea, exercise intolerance, yawning.	Inappetance / vomition.
Neck stretching, inflamed cere.	

URT infection in psittacine birds is frequently associated with chronic malnutrition (linked to the feeding of sunflower or peanut based diets), in particular hypovitaminosis A. Correct diagnosis and therapy cannot be carried out without a thorough examination of the affected area. Respiratory signs (either URT or LRT) are one of the commonest presenting signs of chlamydiosis, (psittacosis). Any bird presenting with respiratory or ocular disease should be screened for chlamydiosis.

Chlamydiosis - otherwise known as Psitticosis / parrot fever

Caused by *Chlamydophila psittaci*, an obligate intracellular bacterial parasite.
Source of infection pet birds, ducks or pigeons (159 avian spp). Of all psittacine birds, cockatiels carry the infection most commonly.
Infection from inhalation of aerosol of faeces and possibly feather dust.
Incubation 5 - 14 days

Risk to self, staff, inpatients, owners - Human signs - anorexia, nausea, photophobia, chest pain, vomiting, pneumonia, headaches, spiking fevers, night sweats. Humans typically respond well to therapy so long as the medics have made the correct diagnosis. All vet practices which deal with birds should have the risk of chlamydiosis assessed in the Health and Safety and COSHH Manuals. Any precautions that can be taken should be (testing sick inpatients, isolation accommodation, face masks), staff should be aware of the risks and the clinical signs in humans. A record of staff health status should be kept such that any repeated or uncharacteristic illnesses will become apparent to the partners. The owners or keepers of any infected bird, should be given a 'Chlamydiosis' fact sheet, explaining the disease and advising them of any precautions which they should take.

Biology of the Organism -- The chlamydial organism can survive in the host macrophages and remain viable through mitosis, hence it can be passed onto the next generation of macrophages, without having to leave the host cell, thereby maintaining protection from antibodies and antibiotics. 5 serovars exist (psittacine, pigeon, duck, turkey, pigeon 2). Inter species transmission and virulence does vary. *C psittaci* enters the host via the respiratory or gastrointestinal epithelium. Clinical signs in birds include :- sick / ill bird, fluffed up, any abnormal respiratory signs, ocular disease including conjunctivitis, diarrhoea (often green in colour), or they may be asymptomatic. In the past the presence of lime green faeces have been often reported to be pathognomonic for chlamydiosis, however this observation is a reflection of the concurrent liver pathology which occurs with the infection, but may also arise due to other causes. If birds under 2 years of age are infected they will shed the infective particles at a higher rate, and succumb to the infection more readily.

Diagnosis : Any newly purchased bird, or sick psittacine bird (irrespective of signs), should be tested for chlamydiosis. Any bird with ocular or respiratory signs or diarrhoea should be tested and it may well be considered that in failing to test such a bird one is negligent. When one considers that this disease is a zoonotic condition with the potential to cause fatalities in humans, the thought of negligence acquires a certain gravity.

In simple terms there is no one test which will conclusively prove that a bird is currently infected by the organism. Infected birds only shed the organism in the faeces on an intermittent basis, although **sick** birds are expected to be shedding consistently. The gold standard test is cell culture, however this is normally impractical, as it is only performed by a small number of specialised laboratories and takes about 2 weeks. Faecal ELISA tests such as 'Clearview', 'RapidChlam' 'Kodak Surecell' will detect the organism although one requires a minimum number of elemental bodies to trigger the test, false positives commonly occur due to cross reactions with certain bacteria, and false negatives can occur due to intermittent shedding. A faecal PCR test is far more accurate, it will not suffer false positives (so long as samples are not mixed up), although false negatives are still common place due to the intermittent shedding. Test results are typically not available for approximately 1 - 2 weeks. One can reduce the chances of a false negatives by batching faeces over a period of several days. It should be remembered that all feacal antigen tests will be negative within 48 hours or so of suitable antibiotic therapy.

Alternatively one can use a serological test to determine the level of immunity in the bird's system to the organism. If the titre is high, and the bird has not been treated for the disease recently, then infection must still be present. Such tests can be performed through commercial laboratories, although there is also a simple in house test 'Immunocomb'. Immunocomb is a semi quantitative test, requiring the assessing of shades of grey. This author favours a test which can be used in house, to give an immediate result (infected or not), with minimal false results. In our view Immunocomb is currently the best test available, although it is a changing field, and views could change in the future. No bird that is ever tested by whatever means should ever be stated to be clear of *Chlamydophila* spp, such a statement is insecure, and one opens oneself to the risk of litigation. It should be remembered that chlamydiosis is a very very common disease in pet birds, the detection of chlamydiosis does not prove that this is certainly the cause of the bird's illness, it may in fact just be a significant but incidental finding.

Other tests - most affected birds will have a raised white cell count, elevated fibrinogen, an increase in non specific soft tissue or liver enzymes. On xray almost all cases will have a readily visible enlarged spleen on the lateral view.

Post mortem :- clinicians should not perform psittacine post mortems unless they have appropriate safety facilities (e.g. fume cupboard). If they are to be performed, the bird's feathers should be soaked in suitable disinfectant. The abdomen incised, the spleen located and removed. An impression smear should be made from the spleen and stained with Gimenez or Macchiavello's techniques to show red staining chlamydial inclusions. If these are found the post mortem should be abandoned, if negative one may proceed with a normal post mortem for other causes of death. Apart from splenomegaly and chlamydial inclusions on cytology, air sacculitis and hepatomegaly are typical.

Latent Infection :- is a very common occurrence. As a consequence of the organism's ability to hide in macrophages, many birds will harbour the infection from months to years before clinical disease is ever seen. The more classic chlamydiosis commences within a few to several months following exposure. Birds which are latently infected will usually break down following a period of stress, eg owner on holiday, sold from shop, transported, breeding season, concurrent disease, new bird arrives etc..

Treatment - it is unrealistic to treat a **group** of birds and expect to clear the infection entirely from all of them. Indeed treatment may not rid even an individual bird of infection all together. There is an argument that since infection passes from macrophage to macrophage within the bone marrow, that medication can never clear it from this site. There is no way of ever proving that the organism has been eliminated from a bird's system, although a falling titre following therapy is a good indication. One expects the titre to approximately halve every 3 - 4 months. It is therefore impossible to ever certify a bird as free from chlamydiosis. This particular method is the one utilised by the author.

Many treatment regimes have been recommended over the years. The oxytetracycline group are the drugs of choice, and in particular doxycycline. It is recommended that treatment must be maintained consistently at MIC for a period of 45 days. The most effective way of achieving this is to give weekly injections of doxycycline at 75 - 100 mg/kg (ie a large volume), of long acting injection. This product is vibravenous injection, and is licensed by Pfizer.

It has been shown to be possible to maintain an MIC in cockatiels, by administering doxycycline hyclate, in food or water, thereby achieving efficacy in apparently clearing positive cases. Doxycycline hyclate dose is 15 mg/kg, but in practical terms 1 sachet in 2 litres of water, given fresh daily. Doxycycline does tend to be chelated with calcium so all mineral supplements should be withheld during therapy. Water which is used to dissolve the powder in should be free from mineral

salts and ferrous material. No dairy products or citric acid should be permitted to be consumed whilst on medication. The birds should have no other source of water, and no wet foods should be given as this would reduce water consumption.

Some therapeutic regimes have been suggested by injecting twice daily, oral dosing twice daily, or water medication with baytril. These methods have now fallen out of favour. Although baytril was considered to an effective medication, its efficacy is now questioned.
Azithromycin (*Zithromax* - Pfizer - tabs or oral flavoured suspension) at 40 mg/kg PO SID has now been used in a significant number of cases.

The Practical Problem: All sick birds showing any respiratory, ocular or diarrhoea signs, especially psittacine birds, should be tested for chlamydiosis, and should be assumed to be infected until proven clear, for the sake of staff and in patients alike. Chlamydiosis should be assessed under Health & Safety and COSSH. The risks have to be quantified and minimised. Post mortems in practice are questionable unless you have a fume cupboard. All staff should be made aware of the risk, as should the owners of any positive tested birds, it is prudent to have a prepared client advice sheet on the disease to issue to such owners.

PSITTACOSIS CLIENT ADVICE SHEET

Psittacosis is a very common disease which may affect all members of the psittacine family as well as waterfowl, pigeons, raptors, most other bird groups as well as other animal species, and is found in all areas of the world. The severity of disease will vary with respect to the species which is infected as well as the strain of the chlamydophila involved.

Psittacosis is also a 'ZOONOSIS' ie. it can infect and cause significant disease in humans.
The are a number of important facts that you as the keeper of a positive or suspect bird must be aware of.

Psittacosis is a disease in which birds may be infected, for many years (at least > 15yrs) before any signs of the disease every become apparent. The infection commonly remains 'latent', ie hiding, in the body, then following some period of stress, or change in the bird's life style, the disease may suddenly become active.

Clinical signs of disease range from any respiratory signs, sneezing, coughing, eye discharges, diarrhoea or soft droppings, loss of appetite, inco-ordination or other nervous signs, sudden death or simply being 'fluffed up' and off colour, ie. just a sick bird. In many cases only one of the presenting signs will be present. Any bird with eye or nasal discharge, breathing difficulties or diarrhoea should be tested for chlamydiosis.

Young birds are more prone to the serious form of the disease. Infection of young birds often occurs from their parents who may be shedding the organism, as breeding is such a stressful time for them. The bird may remain healthy in all respects and yet have commenced shedding the infective agent, there endangering both other birds in the vicinity as well as the keeper.

Testing for the disease is difficult, as both false positives and false negatives do occur. Different tests each have their weaknesses, take advice from your avian vet. In order to be certain, it is often necessary to use two separate tests. Disease cannot be proven in a bird who is a carrier, it can only be confirmed in a bird which is shedding the infective agent at that time. However the bird's serological titre (a measure of its own immune resistance to that infection) against the disease may be tested, if this

is high it denotes current or recent infection. If it is high and the bird has not been treated for the condition one can assume it is still present.

Once a bird has been diagnosed as being positive, although the bird can be given therapy which is regarded as effective, there is no way of proving for certain that that bird is not still a carrier, which may start to shed the agent again at some time in the future.

Treatment of birds usually involves, continual medication for 45 days. One major problem with therapy is the limitation of route of drug administration in many birds. Therapy is simplest in birds who will eat soft food, to which medication may be added. Water therapy is rarely effective (except in cockatiels with Doxycycline hyclate). Alternatively birds may be injected every 7 days for 45 days.

All mineral supplements should be removed during therapy, as calcium in particular interferes with the medication used in treating chlamydiosis.

Stress in the bird room should be minimised during therapy.

Infection is spread in dust (from dried faeces), so all birds in the same air space as one which has been diagnosed positive, will have been exposed, and are likely to be infected. The species most commonly affected are cockatiels. Some estimates are that 30% of all budgerigar colonies are infected.

During treatment the owner should disinfect the premises with an effective disinfectant. Ventilation should be improved, feather and faecal dust should be minimised. People over 45 years of age and pregnant ladies should not enter an infected premises.

Once treated the serological titre should gradually drop over a period of 6 months and then remain low. Failure of the titre to decline is generally an indication of persistent infection.
An infected human can infect birds, as well as birds affecting humans.

Prevention of Chlamydiosis
Only buy birds from reputable sources, always quarantine new birds, and have health screens carried out on all new birds by a recognised avian vet, including a chlamydiosis test. Do not buy parrots from shops or stores where they have been close to (in the same air space) as cockatiels or other small birds. Pet stores, or other suppliers of birds should operate an 'all in all out policy', ie if one of a group of birds is sold, the group should not be replenished until they are all sold. If a group of birds is being constantly topped up, birds are being constantly mixed with new birds in a stressful situation, so infectious diseases are common, such stores should be avoided. Never buy a bird from a group of birds where one of the group, or any near by bird is obviously ill. Remember there is no such thing as a 'cheap' bird, buy quality and be prepared to pay for it. One should expect parrots and other larger psittacine birds to be free from chlamydiosis. Cockatiels and budgerigars however are commonly affected, it is unrealistic to expect breeders to test these small, generally low value birds, prior to sale. It is more realistic to consider such birds as being positive on arrival, and hence to keep them separate from any uninfected birds.

HUMAN DISEASE : Although psittacosis can cause severe, even fatal disease in humans, the disease is not difficult to treat effectively, so long as your Doctor knows that you at risk.
Symptoms in humans : Headaches, flu like symptoms, non-productive cough, swollen glands, liver problems, or a rapid severe pneumonia.
If you have had a bird diagnosed positive, please inform your doctor. If you are suffering from any of the above symptoms, and keep birds, please consult your doctor, and inform him that you keep birds.

DISEASES OF THE UPPER RESPIRATORY TRACT

Nares

The external appearance of the nares is often indicative of the type of URT condition. Hypovitaminosis A, as well as excessive dry heat, predisposes to URT infections. Hypovitaminosis A often presents with nasal, choanal, lingual or sublingual (often involving the salivary glands) sterile abscessation. Hypovitaminosis A causes squamous cell metaplasia and hyperkeratosis, which results in these pathological changes.

Rhinoliths are frequently present; these are typically related to hypovitaminosis A, or to bacterial, mycoplasmal, chlamydial, fungal or viral infections. Severe infections with avian pox or *Cnemidocoptes* spp., or mycotic, bacterial or mycoplasmal infections, can all lead to marked proliferation of the cere with resultant occlusion of the nares. The centre of the nares is occupied by the operculum, which is a hard, but vascular, fibrous raised structure; it should be dry , smooth and shiny. Exudate often concentrates around the operculum, blocking the nares. This should be removed with a fine blunt point, eg. endodontic paper points, prior to the cause of the exudate being treated. The nasal cavity is divided into two halves, each being made up of three segments (conchae).

Sinuses

The sinuses are entered from the middle and caudal conchae. Significantly, the entry points into the sinuses are at rostral level. Infection may enter the sinuses from the nasal concha. Infection, together with cellular debris and mucoid secretion produced within the sinuses, fills and extends the sinuses with fluid. In view of the level of the entry points, the resulting mucoid fluid is often unable to drain away. The lack of drainage, together with the resultant reservoir effect of the accumulation of infected debris, has previously rendered sinus infections difficult to treat. The interconnection of the nasal chambers and sinuses of either side, together with the multiple ramifications of the infra-orbital sinus, mean that if the sinuses become infected, many of the surrounding structures of the head may also become affected. Therefore, sinusitis may be indicated by a persistent unilateral or bilateral ocular discharge. The amount of tissue destruction is dependent on the lytic properties of the pathogen involved, or is a consequence of pressure necrosis caused by the production of inspissated caseous material.

Diagnosis

The presence of URT infection is rarely difficult to demonstrate. Visual inspection, scrapes, rhinoscopy sinus flushing, aspiration, choanal swabs and radiography should all be used where relevant. Choanal swabs or sinus aspirates should always be used in preference to direct swabs from the nares. Cytology should be performed on all samples collected. The normal flora of the choana should be predominantly Gram positive, eg. *Lactobacillus* spp., *Streptococcus* spp. and *Micrococcus* spp., with less than 5% Gram negative, only occasional budding yeasts and no fungal hyphae. Culture and sensitivity testing for significant pathogens should be carried out. Fungal infections (*Aspergillus* spp. are frequent, *Candida* spp. are infrequent) of the psittacine URT are commonly found. Normal cytology of the nasal and infra-orbital sinuses reveals occasional non-cornified squamous epithelial cells with low levels of extracellular bacteria and debris. Cytological evidence of sinusitis is denoted by the presence of inflammatory cells in the aspirate. If bacterial infection is present, septic, heterophilic or mixed cell inflammation will be evident. Mycotic infections will typically be demonstrated by mixed cell or macrophagic inflammation with the presence of fungal hyphae or spores. Mycoplasmal organisms have been frequently implicated in URT infection, although authenticated cases are rare. This may be due to the difficulty in culturing these organisms. Mycoplasmal infections respond well to tetracyclines, erythromycin, tylosin, spectinomycin or enrofloxacin.

Adenovirus, herpesvirus (similar to the laryngotracheitis virus of poultry) and reovirus have been implicated in chronic respiratory disease, but are rare in the UK. Diagnosis is by exclusion of other aetiologies and virus isolation from tracheal swabs or washes.

Treatment
If hypovitaminosis A is likely the diet should be improved and parental vitamin A administered (30,000 iu/kg once / week for 3 weeks). Parental therapy may be required for bacterial organisms such as *Pseudomonas* spp. However, sinus flushing or intra-sinus injection of antibiotics is invariably essential. Prior to culture and sensitivity results being available, the author favours sinus flushing with enrofloxacin (*Baytril injection*, Bayer Australia) (0.75 ml of 5% Injection in 20 ml of saline/kg bodyweight. The bird should be held upside down allowing more distant diverticulae of the sinuses to be penetrated. The syringe nozzle (without a needle) is held tight against the nares (the mouth should not be held closed) and the total contents of the syringe evacuated at force through the nasal chambers. This treatment is continued daily for a minimum of ten days. The technique is simple and may be easily demonstrated for the owner to carry out at home. Parenteral antibiotics may be given at the same time.
Candida spp., Aspergillus spp., Trichomonas spp., Mycoplasma spp. and *Chlamydophila psittaci* may be present. These organisms should be tested for and treated as necessary, Table 2 shows some effective therapeutic agents.

Table 2. Treatment of URT infections.

Organism	Therapeutic agent
Bacteria	Effective antibiotics.
*Candida*spp.	Nystatin/ itraconazole.
Chlamydophila psittaci.	Doxycycline / enrofloxacin.
*Mycoplasma*spp.	Tylosin / lincocin / spectinomycin / enrofloxacin / erythromycin.
Aspergillus spp.	Amphoteracin B + itraconazole / 5 flourocytosine + enilconazole.
Cnemidocoptes spp.	Ivermectin.

When debriding oral or nasal abscesses, care should be taken to avoid causing haemorrhage. Chemical agents (silver nitrate or ferric sulphate) or radio-surgical equipment should be available. Trephination of sinuses should only be performed as a last resort; very few cases fail to respond to sinus flushing as described earlier. If nasal aspergillomata occur, the author favours the placement of an indwelling catheter following identification of the affected site. Therapy is continued with parental and topical medication (see later for aspergillosis therapy).

DISEASES OF THE LOWER RESPIRATORY TRACT
The lower respiratory tract, comprises the trachea, bronchi, lungs and air sacs.

Trachea
Diseases of the trachea and primary bronchi are several and varied. Differential diagnosis of these diseases provides one of the most important and potentially rewarding challenges of avian practice. Clinical signs will include loss or change of voice, coughing, rasping or rattling inspiration and/or

expiration, and dyspnoea. The trachea commences at the rima glottis, which is the slit like opening positioned in the posterior segment of the tongue at the rostral end of the trachea and obscured from sight in most conscious psittacine birds by the large fleshy tongue. The glottis is not protected by a soft palate, but has an efficient closing system, which operates whenever the bird swallows. The vocal apparatus is located at the bifurcation of the primary bronchi (syrinx) within the thorax, and not at the larynx as in mammals. The avian trachea differs in two main respects from mammals. Firstly , the tracheal rings are complete and secondly, the tracheal diameter is comparatively larger. The position of the syrinx is similar in all avian species, although the shape differs greatly. This makes radiological interpretation of syringeal pathology difficult in some species. A collection of radiographic normals for each species is useful.

Diagnosis

Clinical diseases affecting the trachea and primary bronchi include parasitic, bacterial (occasionally viral) and fungal infections, nutritional disorders, toxicity problems or consequences of foreign bodies. It is crucial that a specific diagnosis is made prior to instigation of therapy. Diagnostic tests for LRT disease should include cytology, faecal examination, haematology, biochemistry, endoscopy and or radiography. If the bird is to be intubated, tracheal swabs and endoscopy should be performed first. Both these procedures will require general anaesthesia.

Tracheal washes may be carried out by one of two methods. If the bird is severely dyspnoeic the bird should be anaesthetised and an air sac breathing tube placed in the caudal abdominal air sac. A sterile endotracheal tube is placed in the trachea and a plastic respiratory (or male dog urinary) catheter is passed down the tube. The catheter is passed to the level of the syrinx (just caudal to the thoracic inlet) and sterile saline (0.5 - 1 ml/kg body weight), is introduced and then withdrawn. Alternatively, an 18 - 22G plastic intravenous catheter is introduced through the skin and into the trachea in the caudal cervical area; the catheter is advanced to the level of the syrinx. The latter method may occasionally be carried out in a conscious bird. The bird is maintained parallel to the floor and the sample aspirated. The author favours the former method as tracheal endoscopy would routinely be carried out in all cases. The tracheal aspirate in a normal bird should have a low cellular content with minimal macrophages or inflammatory cells. An increase in heterophils, macrophages or other inflammatory cells is clinically significant. In bacterial conditions bacterial phagocytosis will be evident, whereas in fungal infections there are characteristic thick septate hyphae that branch at 45^0.

Radiography may reveal the presence of an aspergilloma at the syrinx. However, careful and experienced interpretation is required in view of the inter-species variation of normal shape and size of the syrinx .

There is no substitute for endoscopy when investigating cases of LRT disease. Endoscopy allows visualisation of tracheal parasites, the degree of inflammation, the presence of hyperaemia or excessive mucoid secretions, and the collection of aspirates. Most importantly, endoscopy gives full visualisation of the proximal primary bronchi.

Aetiological factors involved in LRT disease

Nutrition. See earlier.

Mycoses. *Aspergillus* spp. are the commonest fungal pathogens of psittacine birds; the organism is ubiquitous in nature, and the disease is difficult to treat. (Respiratory infection involving *Candida* spp. does occur on occasions, although it is generally secondary to an enteric *Candida* spp. infection). Aspergillosis is commonest in African Greys, Amazons and cockatoos. Birds that are immune-suppressed, imported, stressed or malnourished, or that have been housed in dirty accommodation, frequently in the proximity of rotting vegetable matter, **or who are fed seed that was stored before it**

was fully dry, are the most susceptible. Where susceptible species have been in a potentially contaminated or stressful situation, prophylactic therapy is justifiable. Every effort should be made to ensure that environmental *A. fumigatus* loading, as well as the level of stress, is minimised. Particular attention should be paid to the quality of the seed and nuts, as well as the nest material supplied.

Infection may be localised, as in mycotic tracheitis (signified by a change or loss of voice and/or a rasping inspiratory noise) and some air sac forms (where one or more aspergillomata form), or it may be multi-focal. Mycotic granulomata may be found within the nasal cavities, oropharynx, glottis, syrinx, lungs or air sacs. Multifocal cases tend to be more commonly associated with poor husbandry; the rate of pathogenesis and deterioration in these cases are faster. Clinical signs will vary greatly and will be dependent on the site and extent of the lesions. Change or loss of voice is highly suggestive of the syringeal form; other signs will include tracheal haemorrhage, weight loss, general malaise, loss of appetite and vomition, polydipsia and polyuria, a drooped wing, or severe dyspnoea in advanced cases. *Aspergillus* spp is not transmissible between birds.

Parasites. *Syngamus trachea* infection is relatively uncommon in psittacine birds. It is most commonly seen in ground dwelling species**. I**nfection usually occurs from eating transport hosts, eg. infected slugs, snails, earthworms or other invertebrates, although direct bird to bird infection can occur. Passage via earthworms renders the parasite more infective to the main host. After ingestion the larvae can pass via the bloodstream into the lungs within six hours, and from there travel into the trachea. The pre-patent period is 17 - 21 days. Air sac mites (*Sternostoma tracheocolum*), and *Trichomonas* spp, *Cyathostoma* spp. *Serratospiculum* spp should also be excluded as causes of parasitic respiratory disease.

Bacteria . These are generally Gram negative. The commonest respiratory bacterial pathogens isolated are *Klebsiella pneumonia*, *Pseudomonas aeroginosa* and *Pasteurella multocida*. Other organisms, such as *Yersinia pseudotuberculosis*, *Eshericia. coli*, *Streptococcus* spp. and *Staphylococcus* spp., have all been isolated on occasions. Although the clinical signs are similar to those seen with parasitic infections, a muco-purulent exudate may be visualised within the trachea, or the trachea may be excessively erythematous with copious mucoid discharge. If bacterial infection is suspected, aspirates or tracheal swabs or washes should be taken, impressions smears should be examined, and culture and sensitivity testing performed.

Aspiration pneumonia. This occurs most frequently following inexperienced use of a crop feed tube, hand feeding of neonates or following anaesthesia (if the crop was not empty and the bird was not intubated). Although it is appreciated, particularly with small birds, that prolonged starvation prior to anaesthesia is potentially dangerous, it is now accepted by most workers that a limited time without food is advantageous. Any bird anaesthetised, but particularly those in which it is known that they do not have an empty crop, or where there is proximal gastrointestinal tract pathology, should be intubated in order to prevent the risk of aspiration.

Treatment of aspiration pneumonia is with antibiotics, and anti-inflammatory therapy systemically and by nebulisation. Prognosis is grave, but not as hopeless as in mammals, as foreign material can pass through the lung into the air sacs.

Polytetrafluoroethylene (PTFE) (the commonest form of which is Teflon) poisoning is the most commonly reported inhalant toxicity in birds (LaBonde 1996). The condition has been recognised and reported for over 20 years (Blandford et al 1975). Many vets and bird keepers are aware of the risk of PTFE from non stick cookware, but are unaware that it is now far more widely used in other domestic utilities or engineering situations. Teflon or similar products are commonly utilised in some domestic boilers, irons, ironing board covers, solid fuel burners, etc..

In the UK there is currently a new 'baking sheet' being marketed, for domestic use. This is a new product and 'bird keeping' users may well not realise that it contains Teflon. If the baking sheet is used flat in an evenly heated oven at the correct temperature, no problems will arise, but if (as is likely), that it comes into close contact with the heat source, poisonous fumes are likely to be generated. In one recent incident 4 birds were affected over a three week period, the last bird dying within 15 minutes of the owner over heating the sides of a baking sheet, as they curled up towards the flames of a gas grill. Gross and histopathological findings were consistent with Teflon toxicosis.

Another relatively new product, is a make of heat lamp which is designed and marketed for use with animals, which is painted on the exterior with teflon. In the case involving the heat lamps, the lamps were being used to prevent chilling of raptors which were being kept in otherwise unheated buildings over night. The lamps appeared to function safely for one year, after which time, with continued use at their normal working temperature, several poisonings occurred. In all 8 birds died over a period of three months. All birds died or were affected by fumes over night. The lamps were proven to be the source of the fumes. Histopathological findings were consistent with Teflon toxicosis.

These products have recently caused the deaths of numbers of birds, in separate incidents, as well as possibly others unknown to the authors. Extreme levels of fumes may lead to acute deaths of all birds in the air space immediately. In contrast low levels can lead to intermittent deaths of birds within a group over a period of weeks or months.

It should be stressed that there have been no reported deaths of birds when cookware has been used correctly. If any of these products is heated to over 280 oC (536 oF), the compound will pyrolyse, resulting in polymer fumes. Humans exposed to polymer fumes experience 'flu like symptoms'. In birds, acute death is most common, mild exposure will cause dyspnoea, wheezing, weakness and ataxia. At post mortem air sacculitis, pulmonary congestion, haemorrhage and necrotising pneumonitis are common findings (Lyman 1986, Wells 1982). These signs are relatively non specific, and the diagnosis may well be missed at either gross postmortem, or on histology.

Diagnosis of aspergillosis

History, clinical examination, haematology, radiology, endoscopy, biopsy, tracheal wash, cytology, culture, protein electrophoresis and serology.

History : species susceptibility, stress (import, transport, quarantine, training, isolation, caging, crowding), food / environmental contamination, chronic nutritional deficiency.

Clinical signs : behaviour, loss of weight or reduced appetite, loss of / altered voice, cyanosis (especially on handling). Signs vary depending on the site, multitude and position of the lesions.

Diagnosis with respect to site of lesion : Tracheal - loss or changed voice, inspiratory stridor, severe dyspnoea. Diagnosis requires a 2.7mm or smaller endoscope, tracheal lavage, swabs (cytology / culture). Air sac cannulation prior to collecting diagnostic material is advisable. Tracheoscopy can be misleading with respect to the size, seveerity and depth of the lesion. Tracheoscopy must be performed under isoflorane GA, the syrinx must be visualised. Dipping the scope in sterile water prior to entering the trachea helps to prevent moisture forming on the lens.

Differential diagnosis : tracheal foreign body, hypovitaminosis A, trichomoniasis, bacterial or viral infections.

Sinus / Beak : may include Candida and or aspergillosis. Diagnosis via choanal swab, aspiration, (or sinus flushing) endoscopy + cytology where required.

Pneumonic form :- Signs will vary with respect to the size, situation and number of lesions, from mild depression or reduced appetite to severe cyanosis and dyspnoea. diagnosis is by radiography, endoscopy, biopsy, culture, cytology, histology.

Air sac form : in either abdominal or thoracic air sacs. these may be few in number, discrete and treatable or extensive and multiple. There is on occasion secondary bacterial infection and abscessation. Diagnosis is by radiology, endoscopy and biopsy.

***Aspergillus* sp affect on the liver** : Many birds affected by aspergillosis have a reduced appetite and hence may show increased bile pigments in the faeces / urates. Others may demonstrate a marked anaemia consequent to erthyrocyte autolysis, leading to increased biliverdin production etc. Others may show a marked hepatotoxaemia which may also lead to increased biliverdin excretion.

Aspergillosis radiological signs : hyperinflation of abdominal air sacs, loss of honey comb pattern of lung field, radiodensities in lung field and or air sacs. Delineation of air sac walls due to air sacculitis. If any of these signs are present the bird must be endoscoped.

Aspergillous endoscopic lesions :- vary from discrete syringeal or air sac lesions, to multiple discrete or even extensive and ubiquitous wall to wall thick walled, hyphae filled lesions. As there is often a concurrent air sacculitis, with thickening and opacity of the air sac walls, visualisation through a wall is likely to be impossible. It may be necessary to penetrate through a solid looking wall, into an area which you can have no premonitory knowledge of. Radiology prior to endoscopy is essential in almost al cases.

Aspergillosis - Haematology : haematology is readily performed rapidly in house and is an invaluable adjunct to the diagnosis of most avian diseases. Raised WBC, initially a heterophilia, changing with chronicity to a monocytosis. Liver and soft tissue enzymes may be elevated but are of little diagnostic value.

Aspergillosis - electrophoesis : Plasma is applied to an electrical field on a gel substrate. Proteins migrate on the gel dependent on their chemical charge. Bands are stained and enumerated by laser. High resolution E - has permitted a much more accurate measurement and more specific interpretations of combinations of alterations.

Results : **Chronic asper cases** - moderate to marked **hyper betaglobulins**, occasionally only hyper gamma globulins

30% of clinically normal birds have an abnormal EPH, ie it is useful for subclinical diagnosis of various diseases. However it should **only be considered as an ancillary test**.

Only 30% of confirmed aspergillosis cases show any EPH abnormality.

Acute asper - decrease albumin, moderate **hyper alpha-2, beta and gamma globulins**.
Immunoelectrophoresis : Immunofixation in gels, involves the use of specific antisera. Antigen-antibody complexes precipitate in bands with respect to plasma proteins. Chickens fractionate IgM, IgA, IgE and IgY. IgG tends to be raised in chronic aspergillosis.

Aspergillosis - serology : many birds contract aspergillosis as they are immune compromised, or for other reasons fail to mount an immune response. radioimmunodiffusion (RID) & ELISA, can be used by selecting a cut off point with respect to sensitivity is a problem. One has to consider on the one side that the organism is ubiquitous and hence many birds have met it and will be sero positive (in the

absence of disease) on the other hand birds infected may well have a primary or secondary immune suppression.

Serology Result Considerations
Most birds have been exposed
+ve titre may be normal and incidental
If +ve test + clinical signs then certainly treat.
Low +ve titres are common in very sick birds
Frequently false -ve titres.
Paired samples are a waste of time for diagnosis, although they can be useful for response to therapy.
Infection may cause immune suppression, or infection may occur as a consequence of it.
Sero conversion often occurs in recovery

Aspergillus sp antigen linked ELISA
results -ve, weak +ve, +ve, strong +ve.
Test is based on sanofi Pasteur (France)
The test is not *A fumigatus* specific.
If antigen +ve but antibody weak **TREAT**
If antigen +ve & antibody +ve **TREAT**
Positive cases may have -ve or weak positive antigen and or antibody.
Weak positive antigen is common in clinically normal cases, due to environmental contamination.

Consideration of EPH + antibody + antigen is most useful, yielding a specificity and sensitivity in excess of 85%.

Conclusion
If treatment is to be effective, early diagnosis and treatment is essential.
Sertology, radiology or haematology alone is inadequate
A 2.7mm scope is essential
Diagnostic and treatment costs will always be high, prognosis will never be better than guarded.

Treatment of Respiratory Disease

The importance of an accurate and specific diagnosis is realised when assessing the therapy and progress of a respiratory case. Treatment of *Syngamus trachea* involves the use of an anthelmintic, eg. fenbendazole (Panacur, Hoescht) (20 mg/kg - should not be used during a moult as it may cause abnormalities of feather growth), or ivermectin (Ivomec , MSD AgVet) (200 µg/kg s/c, p/o or topically). Two doses of either drug should be given at 10 day intervals. A consequence of anthelmintic therapy is that a quantity of worms (the females being up to 2 cm in length) will be killed in the trachea and primary bronchi. This may lead to foreign body pneumonia or tracheal obstruction. Hence, the clinical signs may not be alleviated (for up to six weeks), despite the fact that the primary pathogen has been effectively treated. Birds should be maintained on parenteral or droplet (nebulisation) therapy for the duration of the clinical signs. The treatment of air sac worms (*Serratospiculum* spp) requires avermectin therapy at 1-2 mg/kg (ie above the normally recommended dose). The commoner pathogens, together with advised treatment are listed in Table 4.

Table 4. Therapy for some common pathogens of the LRT.

Pathogen	Medication	Route and duration
Sarcocystis	Pyrimethamine.	Give with sulphonamides 21 days p/o. Control insect vectors.
S*yngamus* spp.	Fenbendazole or ivermectin + antibiosis.	p/o. Repeat in 10 days. p/o, s/c or percutaneous, repeat in 10 days. Parenteral / tracheal / nebulisation. Up to 6 weeks.
Sternostoma tracheocolum	Ivermectin	Dose weekly x3
Bacterial	Antibiosis.	Parenteral +/- tracheal or nebulisation.
Fungal	Amphotericin.	I/v (+ fluids) and / or tracheal or nebulisation.
	Itraconazole.	p/o 10 mg/kg bid (dose varies w/r species)
	Enilconazole.	Tracheal or nebulisation.
Chlamydophila	Doxycycline.	i/m or p/o. for 45 days or Azithromycin at 40 mg/kg po sid.

METHODS OF MEDICATION

Nebulisation

Nebulisation was traditionally thought of as being complicated and requiring expensive equipment. However, it is now within the financial reach of any avian practice. Small, mass-produced nebulisers are now readily available. These may be purchased or even loaned by the week from local human hospitals. Nebulisation is typically used for 15 - 20 minutes, 4 - 5 times daily. If particle size as low as 0.5 microns can be achieved medication can reach the lung and parts of the air sac system. Ionising nebulisers are preferable. Drugs such as amphotericin B, gentimicin, amikacin, polymixin B and tylosin have been shown to be poorly absorbed from the respiratory system. However, they are highly effective at a local level without the risk of toxic systemic effects. This method of medication can be carried out easily without any stress and without any need to handle the patient. This can be of great benefit in sick, dyspnoeic birds. An additional benefit of medication by nebulisation is that the humidity of the bird's immediate environment is raised. Table 5 gives suitable drugs and dilutions for nebulisation.

Table 5. Drugs used in treatment by nebulisation.

Drug	Dosage
*Amphotericin B	100 mg in 15 ml saline.
Chloramphenicol succinate	200 mg in 15 ml saline.
Erythromycin	200 mg in 10 ml saline.
*Gentamicin	50 mg in 10 ml saline.
*Polymixin B	333,000 iu in 5 ml of saline.
Spectinomycin	200 mg in 15 ml saline.
Sulphadimethoxine	200 mg in 15 ml saline.
*Tylosin	100 mg in10 ml saline or 1g in 50 ml DMSO.
*Amikacin	50 mg in 10 ml saline.
Enrofloxacin	100 mg in 10 ml saline.
Doxycycline	200mg in 15 ml saline.

* Poorly absorbed from the respiratory epithelium.
All these drugs can also be used by the intratracheal route (see later).

Intratracheal
General anaesthesia is generally required to carry out this technique because of the large fleshy tongue which obstructs the rima glottis in most psittacine species. The author uses a therapeutic dose of any of the agents listed in Table 5. If the volume of fluid can be restricted to 0.5 ml/kg bodyweight no respiratory embarrassment will be caused. However, nebulisation remains the route of choice as no general anaesthetic is required.

SURGICAL CONDITIONS OF THE TRACHEA AND PRIMARY BRONCHI
Traumatic injuries to the trachea occur occasionally. Punctures, lacerations or avulsions of the trachea may occur. The primary treatment is to maintain a respiratory airway; this is often best achieved by placing an abdominal air sac breathing tube. Following this, reconstructive surgery can be attempted. In the event of a localised tracheal lesion which is not responsive to medical therapy, then a tracheotomy with removal of a small number of tracheal rings and restoration is often feasible and efficacious.

The commonest form of tracheal surgery which is requested is the de-voicing of noisy birds (screamers). This procedure is considered by the Royal College of Veterinary Surgeons to be an unnecessary mutilation. Therefore, it is an unethical procedure and may not be carried out under any circumstances by a veterinary surgeon in the UK. The procedure is also considered to be dangerous and may not have a permanent effect, and hence is not advised.

Placement of an abdominal air sac breathing tube
Acute airway obstruction in birds is typically caused by the formation of a syringeal aspergilloma or the presence of a tracheal foreign body. The former condition is common, and is initially characterised by alteration to or loss of the bird's voice. Time is critical; from the onset of signs, complete respiratory obstruction may occur within 10 - 15 days. Any bird with acute LRT dyspnoea must be examined endoscopically as an emergency procedure. Prior to endoscopy or surgery, an abdominal air sac breathing tube must be placed. A number of different sites have been recommended; each varies in ease of placement and effectiveness. The principal is that a tube with multiple endings is inserted into one of the abdominal air sacs. It is sown in place (into the muscle or around rib). These tubes are

generally very well tolerated and may left *in situ* for several days without a significant incidence of secondary air sacculitis. The lumen of the air sac tube can be used as an access port for endoscopy.

SURGICAL TREATMENT OF SYRINGEAL ASPERGILLOMATA.

Having placed an air sac breathing tube, the trachea may be operated upon more safely. Some authors describe retraction and transection (usually through 75% of the circumference) of the trachea to gain access to the syringeal region (see soft tissue surgery lecture). Alternatively one may use a per-glottis approach, attempting to debulk the lesion endoscopically per os. A 2.7mm (or smaller) endoscope, together with a male dog catheter are passed through the glottis and down the trachea. The aspergilloma is easily visualised on one or both sides of the syrinx. By careful placement of the bird, the bulk or all of the lesion can be removed by a rotational cutting motion with the prepared catheter, whilst simultaneously applying suction using a 60 ml syringe, or a vacuum system. Segments of aspergilloma are sucked up, or impaled within or on the end of the catheter and can be recovered. If the aspergilloma cannot be recovered it may, as a last resort, be pushed down a primary bronchus, from where it will emerge on the ventral aspect of the lung, in the thoracic air sac. It may be either recovered from this new position or treated *in situ*. This latter technique is not an ideal solution, but at least the life threatening obstruction has been removed. Following surgical removal or debulking of an aspergilloma, long-term medical treatment is essential to prevent recrudescence and to eliminate any infection elsewhere in the lung or air sacs (see later).

On occasions tracheal or syringeal lesions cannot be removed by the method as listed above. In such situations a tracheotomy is performed, as close to the lesion as possible. If the cartilage of the tracheal rings is involved, (which is not uncommon). Then the resection of 2-3 tracheal rings is well tolerated in most species and may well be a far quicker and less traumatic technique than prolonged suction.

MEDICAL TREATMENT OF FUNGAL INFECTIONS

In order for an antifungal drug to be effective, the organism must be susceptible to that drug at concentrations achievable at the site of infection. Most importantly, the drug must be able to penetrate to the centre of the infection. Furthermore, the antifungal drug must not be toxic to the patient at the level and duration of treatment that is required to eliminate the infection. It has been reported that the azole compounds do not reach adequate minimum inhibitory concentrations within the first 3 - 5 days of therapy, although this point is refuted by the manufacturer of itraconazole and other members of the azole group. In view of this, amphotericin B (*Fungizone intravenous*, Squibb) (1.5 mg/kg IV TID for 3 - 5 days) is recommended. Because of the potentially nephrotoxic properties of amphotericin B, 15 ml/kg of fluids should be given intravenously with each dose. There are several different medical treatment regimes that have been reported. The method preferred by the author is a combination of surgical removal or debulking of any identifiable lesion, followed by topical (intratracheal) enilconazole (*Imaverol*, Boehringer Ingleheim Pty Ltd) (0.5 ml of 1:10 dilution per kg), concurrently with amphotericin B for 3 - 5 days, as well as either itraconazole (*Sporonox*, Janssen-Cilag) (10 mg/kg BID PO). Topical treatment is maintained for two weeks, and parenteral treatment for two months following visual disappearance of any lesion. The potential efficacy of Terbinafine (*Lamasil tablets*, Novartis) as a new therapeutic agent for avian mycotic infection has recently been investigated.

LOWER RESPIRATORY DISEASE

Air sacculitis

Most species of bird have at least four paired and one non-paired air sacs. Inspired air passes down the trachea, through the lung via the intrapulmonary primary bronchi, and through the neopulmonic parabronchi to the caudal air sacs. The neopulmonic part of the lung possesses a highly efficient and active scavenging system. Any inspired particulate matter should be removed at this point. The caudal air sacs comprise the paired abdominal and caudal thoracic air sacs. No gaseous absorption occurs

within the air sacs, they simply act as a bellows system. Bacterial air sacculitis does occur, although it is rare in comparison with fungal or chlamydial air sacculitis. Fungal infections are by far the commonest condition of the air sacs. It must be appreciated that air sac disease rarely causes respiratory signs until the bird is *in extremis*. The severity of signs caused by air sacculitis does not relate to the severity of the pathology. Aspergillosis of the air sacs most commonly affects the caudal air sacs. As the caudal air sacs are the first part of the air sac system for the air to enter, particulate matter, eg. fungal spores, are most likely to precipitate here. Furthermore, it is the first part of the respiratory system that the air has entered where there is not a scavenging system for the control of particulate or infectious particles. As the air sacs surround the digestive system, cases typically present with lethargy, inappetance or vomiting, and with dramatic loss of condition. Affected birds do not usually show any respiratory signs.

Diagnosis - is made by high quality lateral and dorsoventral radiographs, followed by endoscopy. The value of endoscopy cannot be overemphasised. All parts of the lower respiratory system, from the trachea and lungs to air sacs, can be readily visualised. Not only can the lesions be visualised, but samples (of air sac tissue and lung) can be taken for culture, impression smear or histopathology. It is imperative that the clinician has use of an endoscope and is familiar to all approaches. Practising approaches on cadavers is invaluable, although inter-species variations in anatomy should not be forgotten.

Treatment. Unless an aspergilloma is detected very early drug therapy alone is unlikely to be effective because of the thick leathery caseous encapsulation. In these cases surgical removal should be attempted. Surgery of air sac aspergillomata is heroic, but is often the only possible chance of survival. Surgery usually involve midline and paracostal incisions on one or both sides. the author favours initial medication and patient stabilisation for 3-4 days prior to surgery.

Pneumonia

Pneumonia in psittacine birds is a relatively uncommon condition compared with other diseases of the respiratory system. Table 6 lists the commoner causes of pneumonia.

Table 6. Common causes of pneumonia.

Cause	Comments
Aspiration	Relatively common in hand fed neonates.
Bacterial	Rare; diagnose and treat as for bacterial tracheitis.
Mycotic	Multifocal pneumonic aspergillosis is typically associated with a highly spore ridden environment / food; typically these lead to acute death. Treat as for any fungal infection.
Sarcocystis	Coccidian parasite causes haemorrhagic interstitial pneumonia or peracute death.
Toxic	eg.Teflon, commonly seen as peracute death due to pulmonary oedema.
Neoplasia	Rare, biopsy via caudal thoracic air sac.

Subcutaneous emphysema - It is not uncommon for a rupture to occur in the wall of a cervical, clavicular or other air sac. Any air sac rupture causes large volumes of localised or generalised subcutaneous emphysema. In such cases electrosurgery or cautery should be used to burn an incision in the cutis (3 - 5mm long). This will continue to allow the subcutaneous air to escape, enabling the internal rupture to heal. The external incision will heal within a short period. In persistent cases, a silastic tube may be sutured into the skin wound in order to maintain patency, giving a longer period for healing of the defect in the air sac wall.

NON-RESPIRATORY CAUSES OF RESPIRATORY SIGNS

Table 7 lists a number of non-respiratory diseases which may present with respiratory signs.

Table 7. Non-respiratory diseases presenting with respiratory signs.

Cause	Comments
Ascites	Liver or kidney disease, neoplasia, or any other space occupying lesion producing ascites.
Abdominal haemorrhage	Iatrogenic post-surgery, trauma, neoplasia or coagulation defect.
Malnutrition	General weakness / anaemia (+ other causes of anaemia).
Obesity	Causing space occupying lesions / hepatic disease -> anaemia.
Goitre/trauma	Pressure on trachea or damage to integrity of trachea. The condition is common in budgerigars; birds present with a 'click' on inspiration and often have an altered voice. The condition occurs due to an iodine deficient diet, typically arising due to feeding low grade seed mixes, which leads to hypothyroidism. Treatment is by addition of Lugols Iodine (1 part Lugols to 14 parts water, add one drop of this to 30 ml of drinking water daily for three weeks). Increase dietary iodine content (better quality boxed seed, ground oyster shell, or cod liver oil).
Heart failure	Due to ascites or inefficiency of circulation.
Herpes/reo/paramyxo/adeno viruses	All cause viraemia which may present with respiratory signs.
Egg peritonitis or other causes of peritonitis	Due to effect on coeliomic cavity as a whole.

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