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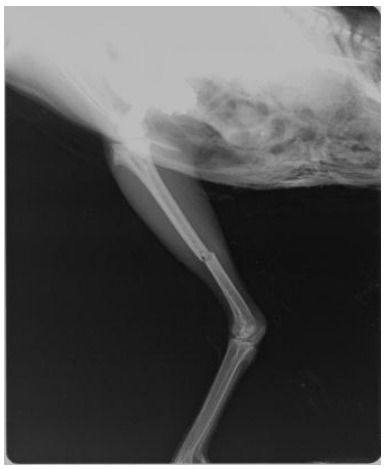
Backyard Poultry Online 'Mini Series'

Session 3: Respiratory, Neurological and Musculo-skeletal Diseases

Aidan Raftery
MVB CertZooMed CBiol MSB MRCVS



The avian respiratory system has several unique features when compared with mammals. Being aware of these differences is important to be able to understand many of the diseases of the bird's respiratory tract, and it is also important in evaluating and maintaining avian patients under anaesthesia which is a much more volatile situation when compared with mammals.
The avian respiratory system is the most efficient of all vertebrates. It is said 10 times more efficient than the mammalian design. However it is also much more complex than the respiratory system of other vertebrate groups. These differences are believed to be adaptations for the demands of flight.
The increased efficiency may explain the increased susceptibility to many environmental insults, nutritional deficiencies and infectious and non-infectious diseases.

The external nares are at the base of the beak protected by a flap called the operculum. From there air enters into the nasal cavities which are divided into channels by the nasal conchae. These are covered in highly vascular epithelium which has a role in warming and filtering the air which flows past. There is a communication from here into the infra-orbital sinus. This is a very large triangular shaped cavity which roughly lies between the external areas and the orbit although there are diverticula off the sinus which extend above and below the eye. In avian medicine this large sinus is very important as chronic infection of this area is common
The air flows from the nasal cavities down through the slit like choana in the hard palate and in through the glottis. When the mouth is shut the glottis is pushed into the choana.
The jugal arch can be palpated here and is an important landmark in the surgical approach to the infraorbital sinus.
The infraorbital sinus is very important as it is a common site of infection.
Less commonly we encounter infection of the frontal sinus. The nasal diverticulum of the frontal sinus extends into the base of the beak. Changes here will be seen as a darkening of the beak on the affected side (unless the beak is pigmented, in which case colour changes to the tissues under the beak may not be obvious).
In cases not responding to medical treatment frontal sinus may be entered trephining either through the base of the beak or just caudal to the nasal-frontal hinge through the frontal bone. In chickens the size of the comb can make this approach more difficult when compared with other groups of birds. Care is needed to avoid injury to the neurocraniun, the eyes or the bony orbit. Once the sinus is entered samples are collected for cytology, histology if abnormal tissue is present, and culture and sensitivity. The sinus should then be flushed with warm isotonic saline. The flash should be seen to enter the oral cavity.
Following the procedure the skin is sutured over the frontal bone. The hole in the frontal bone will heal quite rapidly.
If the sinus has to be approached through the beak, the approach is planned where there is darkening of the beak and this area of beak is reflected caudally. This provides a flap which makes closure easier. Due to the position and size of the external nares in chickens when compared with other groups of birds such as psittacines this approach is more difficult and additionally will not give good access to the sinus as a whole. The deficit in the maxillary bone is likewise not repaired. It's important to make sure that no areas of the horn of the beak are depressed as otherwise they will interfere with the downward growth of the beak. The beak deficit is sealed with PMMA or equivalent.

Infraorbital sinusitis

Symptoms seen include swelling on the side of the head rostral to the eye. Periocular erythema especially rostral to the medial canthus. Ocular discharge and conjunctivitis can also be seen. On oral exam discharge may be seen coming from the choanal slit and the area may appear inflamed. There may also be a, nasal discharge, sneezing, frequent headshaking or yawning in attempts to dislodge discharge, blocked external nares and open beak breathing. Most of these signs are common with rhinitis which is often concurrent.
Occluding the opposite nostril and holding the mouth shut will force the bird to breath through the affected side and air may enter the sinus but not escape so easily so the side of the face will inflate and deflate with each breath or it may not deflate but can be deflated manually by pressing over it.
Head may be held to one side. There may be other signs associated with upper resp infections. Often there is a poor response to antimicrobials

Infraorbital sinusitis is usually an individual bird problem. Differentials would include bacterial and fungal causes as well as neoplasia. Developmental abnormalities such as choanal atresia are occasionally seen.
Haematology will reveal if there is on-going infection and also give an indication of chronicity.
Cytology is used to determine if the normal flora is present or has it been replaced by a pathogenic monoculture. Are normal mucosal cells present or has the picture had been changed by some inflammatory process or neoplasia. If neoplastic cells are present then some indication of prognosis may be given. If the normal microbial flora of the sinus has been disrupted then a culture and sensitivity would be indicated. Acid-fast staining will be necessary to identify mycobacterium. Radiology will help with providing information on the extent of the problem and also on the long-term prognosis.

Treatment of infraorbital sinusitis can be challenging. Most cases are of either bacterial or fungal origin. If there is a build-up of inflammatory exudate this will need to be removed by either flushing or in some cases surgically approaching the sinuses to remove rhinoliths or benign neoplasms such as papillomas. Nebulisation is often used to deliver therapeutic agents directly to the target tissues in high concentrations. However there is no evidence that nebulised drugs will reach the sinus in high concentrations especially if the entrance to the sinus is blocked with inflammatory exudate.
Nebulising with agents that help hydrate the sinuses and facilitate the action of antibacterial drugs is there most common approach.
NSAIDS are used to reduce inflammation which will be contributing to airway obstruction and also disrupting other local tissues such as the eye.

Surgical approach to the infraorbital sinus is more difficult than the approach to the frontal sinus. However infraorbital sinusitis is more commonly diagnosed than a frontal sinusitis
This sinus is a large triangular cavity in the lateral area of the upper mandible and rostral-ventral to the eye. The skin ventral to the jugal arch is incised. The lateral wall of the sinus will be visible just anterior and deep to the adductor mandibulaexternus. This muscle raises the lower mandible. Opening the lower mandible will enlarge the sinus space and provide greater access. The adductor mandibulaexternus and the pseudotemporalissuperficialis are retracted caudally and the ethmomandibularis muscle is deflected rostrally. The sinus is then

incised into. It is then inspected and any relevant samples collected. It is often filled with rubbery inflammatory material which needs to be scooped out.
Sinus is then thoroughly flushed with warm saline. The surgical site is closed. First the lateral wall of the sinus and then the skin is closed.

Lower respiratory tract anatomy
The lower respiratory tract in birds starts at the glottis. There is no epiglottis which accounts for a higher incidence of tracheal foreign bodies. The glottis is not involved in sound production the voice box of birds is the syrinx at the caudal end of the trachea. So voice change in birds is associated with pathology of the syrinx. Relative to mammals the trachea is long and has complete tracheal rings. The mucosal lining of the trachea is very susceptible to damage. During anaesthesia it is important to avoid traumatising the mucosal lining of the trachea. If there is damage to the lining of the trachea it will set up a fibrous reaction and several weeks later the animal will present with breathing problems caused by the reactive tissue obstructing the trachea. This is best investigated endoscopically and usually requires resection of the damaged area in the trachea. The trachea can easily be evaluated endoscopically with a 2.7mm rigid scope or smaller but it must be long enough. A scope with a 0 degree angle of view is best for the trachea as with scopes having for example a 30° viewing angle it is very easy to traumatise the tracheal lining as the scope is been passed. We have a 2.7 mm which is too short to reach the syrinx in chickens, but we also have a 1.9mm scope which is ideal in most cases. We also have a needle scope a 1.2 mm for much smaller birds.

Safe endotracheal intubation
Birds weighing more than 200 grams should always be intubated when anaesthetised. Choose the appropriate sized tube, it should easily slip through the glottis.
The glottis is located behind the tongue and with no epiglottis the glottis can be easily visualised by gently opening the mouth. The endotracheal tube is sparingly lubricated is then inserted into the trachea. Care should be taken not to push the tube too far down as in many species the trachea narrows distally. The tracheal lining is easily traumatised resulting in post intubation tracheal stenosis which usually becomes apparent a few weeks after the anaesthetic. This risk can be minimised by choosing the appropriate sized tube for the bird, careful placement of the tube, never using cuffed tubes (even a deflated cuff can cause damage) and especially important, keeping the trachea straight during the anaesthetic to avoid trauma to the inside of the trachea by the tip of the endotracheal tube. The tube is secured with tape to the mandible so the glottis can be easily examined

The syrinx at the bottom of the trachea, when reached endoscopically the two vibrating tympaniform membranes can be seen as the syrinx is the birds voice box.
Often foreign bodies which have been aspirated come to rest in this area and fungal plaques can be seen on top of the two tympaniform membranes where they have come to rest after sloughing off the wall of the trachea. At the syrinx the airways divide into two primary bronchi that come from the lungs.
Complete tracheal rings make the avian tracheal lumen less flexible and more prone to damage from an over inflated endotracheal tube cuff.
Avian lungs are rigid and do not significantly expand and deflate with inspiration and expiration.

This also means that information that can be gained by use of a stethoscope about the lungs is very limited when compared with its use in mammals.
However occasionally there will be abnormal sounds associated with disease of the airways and air sacs. In birds, oxygen is exchanged as air moves through the lungs. Birds exchange oxygen only when there is a constant flow of air. For this reason, apnoea in birds can result in death more quickly than in mammals. Birds lack a diaphragm and rely on movement of the ribs and sternum for ventilation. The air sacs act as storage for air and are not significantly involved with significant oxygen exchange. Anything that decreases the size of the air sac, including ascites, enlarged organs, internal masses) can reduce the bird's respiratory efficiency causing dyspnoea and may mislead the clinician to diagnosing pathology of the respiratory system also outside pressure (rough handling or restraint devices can have the same effect. Chickens with intracoelomic masses or disease often have significantly reduced abdominal and caudal thoracic air sac space. When handling birds, devices or restraint techniques that prevent normal sternal movement should be avoided. Anesthesia and general handling are much more risky when diseases that directly or indirectly affect the air sacs and lungs are present.

The lungs of domestic fowl are found in the craniodorsal part of the body cavity attached to the ribs.
There are eight air sacs comprising of one cervical one clavicular and the cranial and caudal thoracic air sacs and the abdominal air sacs are paired. They act as temporary storage of air, both on inspiration and expiration. Air sacs are poorly vascularized and account for less than 5% of respiratory system gas exchange. Movement of the sternum acts to create a bellows like effect in the air sacs to draw air in and then as the body cavity is compressed air is pushed out through the lungs. So it takes two ventilation cycles to move air completely through the avian respiratory system.
The air sacs are a common site of disease. Chronic bacterial and fungal infections are most commonly seen in the air sacs, however generalised respiratory infections will also involve the sacs.
The presence of a large number of air sacs in the coelomic cavity enhance the value of both endoscopy and radiography as diagnostic tools in birds. With endoscopy we can make a small puncture into an air sac to insert our scope and evaluate many different areas. Internally we can move around by making small puncture holes in the air sac membranes to move into adjoining air sacs.
The contrast provided by the large air sacs provides very valuable negative contrast for radiography. The very large reproductive tract in laying hens reduces the value of this somewhat compared with other birds.

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Examination of the upper respiratory tract should be an integral part of any clinical examination. Start by observing that the head is bilaterally symmetrical. Note if the plumage is normal, oculonasal discharges can sometimes be seen dried on the feathers. Gently palpate the skull for any asymmetry which may be hidden under the plumage. Similarly palpate the mandible and assess its range of movement.
The eyelids should be observed to be completely open before the head is restrained. Any wounds, swellings or any areas of abnormal pigmentation should be noted. The outer surface of the eyelids should be dry, any discharges or areas of wet feathers is an abnormality. The margin of the eyelid should be symmetrical and should fit smoothly against the curvature of the eye. When the eyes are closed the upper and lower eyelids should meet symmetrically and completely protect the eye.
Check the external ear canals for excess cerumen, inflammation, swelling or any developmental abnormalities such as a narrowed external ear canal.
Check the beak for any discoloured areas especially the area overlying the frontal sinus. The external nares at the base of the beak should be free from any discharge, using the magnification and illumination of an otoscope is usually possible to view a few millimetres in.
Some respiratory conditions will cause colour changes to the comb and wattles. They may become erythematous (generalised or patchy), but there may be paler than normal which may be an indication of poor profusion or anaemia. In cyanotic birds they will be a blueish colour.

Opening the beak we evaluate the oral cavity. First observe the colour of the mucosa and any odours present. The mucosa should be moist with no mucus accumulations. Check for any abnormal swellings, pigmentation or ulceration. White to yellow plagues in the oral cavity may be seen with diseases such as the diphtheritic form of fowlpox and trichomoniasis and also less commonly candida vit a def and with Capillaria plaques may be seen in the oesophagus although this type is rare in chickens and more commonly seen in pheasants. Also T-2 toxicosis, caused by ingestion of Fusarium spp. of fungi, early signs of the condition may include refusal to eat as well as ulceration, crusting, and necrotic lesions in the oral mucosa caused by direct contact with the fungal toxin.
Observe the tongue and underneath it for any abnormalities
The palate of the chicken contains several transverse rows of papillae. Numerous small openings will be seen which are the openings of the salivary glands. There is a slit like opening in the palate into the nasal cavity. This is called the choanal opening.

There should be no discharges visible coming from the choana. If any tissues can be visualised through their choana they should be moist and a normal pink colouration. The infundibular cleft is just caudal to the choana but it is difficult to visualise. This is the opening to the pharyngotympanic tube (auditory tube). If a middle ear infection is present this cleft may appear red and swollen. This area is difficult to evaluate and usually requires endoscopy. It is best seen was a short scope that has a 30° angle of view. This can easily be achieved in the conscious bird.

Different signs are seen if it is a lower respiratory tract infection. Dyspnoea, dysphonia if the syrinx is involved, increased breathing sounds, gasping, tachypnoea, open beak breathing, the neck held extended to straighten the trachea and an oral discharge can sometimes be seen.
With lower respiratory tract disease increased expiratory sounds are more likely on auscultation. The stethoscope is placed over a variety of sites, the trachea, the lungs, the thoracic and abdominal air sacs and the heart.
Auscultation is not as useful in birds as it is in mammals due to the anatomy of the lungs.
Radiographs are very useful for investigating symptoms pointing towards lower respiratory tracked pathology. Don't forget that any condition reducing the size of the air sacs may manifest itself with respiratory signs. The best views are lateral and ventral dorsal.
Sometimes barium studies may add importing information to the radiographs.
Endoscopy is invaluable in birds in the investigation of respiratory and other disease processes in the coelomic cavity. The scope can also be passed down the trachea to investigate tracheal lesions.

Infectious bronchitis

Infectious bronchitis is a widespread acute respiratory infection caused by a coronavirus. Transmission can be by aerosol over long-distance as well as by fomites. It can also be spread by wild birds. All ages of chickens are susceptible and spread within a flock is rapid however the disease is more severe in young chicks less than six weeks of age. However the pathogenicity varies between breeds of chickens is also dependent on the strain of virus.
The virus does not persist very long in the environment however some birds may become carriers intermittently excreting the virus via nasal discharges and in faeces. Chicks less than two weeks of age will have damaged oviducts impairing their egg producing ability.
Clinical signs include upper respiratory signs such as sneezing, gasping, rales, and nasal discharge. The disease spreads rapidly and typically all birds in a flock will develop clinical signs within 48 hours. Laying chickens affected will show a drop in egg production for about two weeks. The eggs often have irregular and roughened eggshells with watery albumin.
There is no treatment however antibiotics are used to control secondary bacterial infections.
The virus is easily destroyed with disinfectants and UV light.
Diagnostic tests include virus neutralization, haemagglutination inhibition and ELISA tests.

Avibacterium paragallinarium

Infectious coryza affects mainly chickens, it can affect pheasants and guinea fowl as well. The causative agent is *Avibacterium paragallinarum* , a gram-negative rod. The incubation period

is 1 to 3 days and the course of the disease is approximately 4 to 12 weeks. Clinical signs include upper respiratory signs and swelling of the face with foul smelling and sticky nasal and ocular discharge, dyspnea and rales. Clinical signs also include decreased egg production. Birds that recover are lifetime carriers. Transmission is through direct or indirect contact. Mortality is about 20% to 50%. The condition can be treated with antibiotics. Recovered birds are lifetime carriers so the only way to control the infection is through depopulation and leaving the premises vacant for at least 30, preferably 60 days after cleaning and disinfecting before repopulating. Avoid mixing flocks or mixing different ages and sources of birds. There is a vaccine available commercially, but it is seldom used in backyard flocks

Avian influenza

Avian influenza affects many species. The causative agent is an Orthomyxovirus. The clinical signs are variable as there are low and high pathogenic forms, but include anorexia, decreased egg production, and respiratory disease with the mild form to respiratory distress, facial swelling, diarrhea and neurological signs with the highly pathogenic form. Usually the mild form of the disease is associated with high morbidity and low mortality. Basically coughing, sneezing, and sudden death are the typical signs of this disease. This a highly contagious disease associated with high mortality of both domestic and wild birds. There is no treatment. Testing usually involves virus isolation. This disease is more commonly known as Avian Flu.
The disease is notifiable: if you suspect the disease, **you must immediately notify the duty vet in your local Animal Health Veterinary Laboratories Agency (AHVLA) office**.

Newcastle disease

Newcastle disease affects many species of birds and is caused by a Paramyxovirus. There are different strains of the virus which produce widely differing forms of the disease.
In its mildest form it can just causes a mild upper respiratory disease with decreased egg-laying, and a low mortality.
The neurologic/velogenic form causes a sudden onset of upper respiratory disease followed by neurological signs such as paralysis/paresis of the legs, clonic spasms/seizures.
After infection with highly pathogenic strains, initial signs include oedema of the conjunctiva, lethargy, inappetence and ruffled feathers. As the disease progresses birds may develop severe dyspnoea and inflammation of the head and neck often with cyanotic discoloration. These symptoms are often associated with enteritis and/or neurologic signs and a dramatic fall in egg production with approximately a 50% to 90% mortality.

This disease is notifiable: if you suspect the disease, **you must immediately notify the duty vet in your local Animal Health Veterinary Laboratories Agency (AHVLA) office.**

Mycoplasma gallisepticum

Mycoplasma gallisepticum causes respiratory disease in chickens, *Mycoplasma synoviae* causes air sacculitis and synovitis/lameness in chickens. *Mycoplasma gallisepticum* is seen in backyard flocks and is of concern because it can easily spread to nearby commercial flocks and cause economic devastation for that commercial flock. Most commercial flocks are free. The best prevention is to depopulate and repopulate with clean stock. Birds remain carriers for life.
Sources of infection are latently and chronically infected birds with spread possible vertically and horizontally both directly from bird to bird (also via semen) and can be indirectly spread by living and inanimate vectors with transmission via aerosol being of special importance. Within a flock the spread of the pathogen is normally slow. Outside the body mycoplasmas can only survive for a few days or even hours only.

Mycoplasmas are very small bacteria lacking a cell wall. Due to their very small genome size they rely on host cell metabolism and are difficult to culture requiring special culture methods. In most cases the infection remains latent due to the ability of mycoplasmas to hide from the host immune system. They act as co-pathogens paving the way for other pathogens and clinical disease usually only develops in combination with them like Newcastle Disease, Infectious bronchitis, Infectious laryngotracheitis, Avibacterium paragallinarum, Ornithobacterium rhinotracheale, and other Mycoplasmas.

Disease is also possible after impairment of the host's defense system by different stressors like transport, bad environmental conditions and sexual maturation.

Once introduced mycoplasma infections may be difficult to eradicate from backyard flocks. Optimal environmental and nutritional conditions may prevent clinical outbreaks of the disease. Alternatively, vaccination with live or inactivated adjuvant vaccines is possible, but if the flock is already infected this will interfere with the vaccination

The clinical signs seen are mostly associated with the respiratory system, nasal discharge, infraorbital sinusitis often filled with caseated pus especially in more chronic cases, severe dyspnoea and rasping breathing sounds.
Reduced hatchability of eggs from affected hens, and chicks that hatch may have curled toes or maybe dwarfed.
Direct detection and identification of the mycoplasma species is possible by cultivation on special media followed by biochemical differentiation, by direct immunofluorescence and by genus and species specific PCR.
Detection of specific antibodies is possible by serum plate agglutination, ELISA, indirect immunofluorescence test and in some strains by haemagglutination inhibition HI.
Simultaneous infections with other pathogen are very common.

Infectious Laryngotracheitis

Infectious laryngotracheitis affects chickens and also pheasants. The causative agent is a herpesvirus. Chickens older than 14 weeks are more affected than younger chickens, so the

disease is usually seen in mature chickens. The disease is associated with decreased egg production, conjunctivitis, nasal discharge, swollen infraorbital sinus, and in more severe cases moist rales. Shaking of the head and flinging haemorrhagic mucoid material from the trachea is a hallmark of this disease including an inspiratory dyspnea.

At gross necropsy a mucoid to haemorrhagic tracheitis is found. Diagnosis is confirmed by virus isolation, ELISA, or indirect fluorescent antibody test or by histopathology where intranuclear inclusion bodies are seen. Prevention is through the use of a live vaccine. The disease can be spread by fomites. Mortality can reach up to 50%. In mild cases the prognosis is good for the individual bird and treatment consists of controlling secondary infections and providing fluid therapy, and nutritional support. Infected birds that recover will be lifelong carriers. Severely affected individuals should be euthanised. Sudden asphyxia from the tracheal inflammatory secretions is the most common cause of death.

Right-sided heart failure

Right-sided heart failure is commonly seen in chickens especially the ex-bats. Exercise intolerance or lethargy are the most common presenting signs. Radiographs will show a large heart. An echocardiogram will show right sided heart enlargement with decreased contractility. Currently the most common treatment used in other avian species is pimobendan. In some cases frusemide has to be added to the treatment regime.

Ornithobacterium rhinotracheale

Natural infections occur not only in other farmed poultry, but also in many wild birds which may also serve as a source of infection.
Transmission is mainly horizontally via direct contact or indirectly by living and inanimate vectors. All ages of birds seem to be susceptible, although signs of disease are more severe in older birds.
Many different serovars are known which vary in their pathogenicity and susceptibility to different antimicrobial agents.
Infection of the mucosa of all parts of the respiratory tract induces exudative inflammatory lesions. The severity of the disease is influenced by the presence of co-pathogens like ND, IB, mycoplasmas and metapneumovirus or E. coli as well as poor environmental conditions.
Clinical signs are very variable and initially include sneezing, nasal discharge followed by oedema around the infraorbital sinus and dyspnea of varying degrees. The birds are usually depressed and there is a decrease in egg production with an increase of abnormal eggs. Mortality may vary between 1-50%. Respiratory symptoms associated with ORT are similar to those caused by numerous other pathogens.
On post-mortem the main findings are pulmonary oedema, consolidation of the lungs, pneumonia, pleuritis and airsacculitis with foamy, white, inflammatory excudate.
Ornithobacterium rhinotracheale does not grow readily on culture media so can easily be missed as the primary cause especially if the secondary pathogen grows more readily.
Tracheal swabs or tissues of lung and airsacs can be sent for culture and sensitivity. Specific antibodies can be detected by ELISA, or AGP (agar gel precipitation) and there is a PCR available.

Prognosis for the flock is usually good and repeated treatment usually leads to success. However, in the individual bird, especially if chronically and severely affected, prognosis is poor as there may be a massive fibrinopurulent aerosacculitis which is may be difficult to treat.
Big differences in susceptibility to antibiotics exist between different strains, it is necessary to test the isolate in a sensitivity test before starting treatment.
Once endemic in a flock, especially in multi-age flocks, eradication is very difficult. Live and inactivated vaccines exist.

Fowl cholera

Fowl cholera (FC) is a highly contagious disease of domestic and wild birds. The disease is caused by *Pasteurella multocida*, There are many serotypes of *Pasteurella multocida* which differ in pathogenicity.

Many avian species are susceptible as well as wild avian species. Some domestic mammals are also susceptible.

FC is spread via horizontal transmission through contact with infected birds, and via fomites. *Pasteurella multocida* can enter through mucous membranes, including oral, nasal, and conjunctival, as well as through cutaneous wounds. Chronically infected carriers play a major role in the spread of this disease and infected birds can remain carriers for life. The prevalence of *Pasteurella multocida* among wild birds, rodents, and other domesticated and non-domesticated species is likely responsible for the introduction of the infection into most domesticated poultry flock outbreaks.

The disease usually manifests as a septicaemia, sometimes with high morbidity and high mortality. However, a more chronic and asymptomatic form of the disease can also occur. Young adults are most susceptible. FC is more prevalent in the cooler parts of the year. Signs vary depending on the course of the disease. In the acute form of FC, infected birds may develop ruffled feathers, anorexia, increased respiratory rate, and cyanosis. Cyanosis is more easily appreciated on the skin of the comb and wattles. Mucoid discharge from the mouth and diarrhea may also occur. Diarrhea often begins as watery and whitish in colour, progressing to a greenish colour with mucus. In the peracute form, signs may be absent and birds may be found dead. Birds that survive the acute septicemia may later die. Birds that survive will remain chronically infected.

In the chronic form of FC, birds that survive acute infection or birds exposed to a low virulence strain, generally exhibit localized infections. The wattles, sinuses, foot pads, sternal bursa, and leg and wing joints may become swollen. Exudative pharyngeal and conjunctival lesions may be present. Sometimes tracheal rales and dyspnea may occur secondary to respiratory tract infections. Infected birds may also exhibit torticollis from middle ear infections and meningeal involvement. The chronic form of FC may last 3 to 4 weeks and may sometimes persist for years.

Culture samples can be taken from the liver, lungs, spleen, wattles or affected joints at necropsy. Additionally, impression smears of the liver and heart blood can also be obtained.

Gram-stained impression smears may reveal the typical bipolar, gram-negative rods suggestive of *P. multocida*. Use of Wright's stain or methylene blue readily demonstrates the bipolar morphology of *P. multocida*. *P. multocida* grows readily on blood agar but does not grow on MacConkey agar. Isolates should be tested for antibiotic sensitivity and resistance.

Aspergillosis

Aspergillosis has been recorded in many species of birds and mammals including man. There are two forms of the disease; the acute form causes outbreaks with high morbidity and high mortality most commonly seen in very young birds. The chronic form is seen in adult birds.

It can be caused by many different types of fungus although *Aspergillus fumigatus* is the most common species involved. *Aspergillus fumigatus* is present everywhere and readily grows on substances such as damp litter, feed if slightly damp and on rotten wood and many similar materials. These are the fungi that are often commonly referred to as mould.

Infection occurs when the spores are inhaled. Large numbers of spores need to be inhaled to cause disease however it will depend on the immune status of the bird. The disease is not contagious and does not spread from bird to bird.

In the acute form the main symptoms seen are gasping, sleepiness, anorexia, leading to convulsions and death. Occasionally if the infection invades the brain paralysis and other forms of central nervous system disease are seen.

In the chronic form there is usually loss of appetite, gasping, coughing and rapid weight loss. Usually only a few birds are affected at one time. Lesions may be seen in the lungs or more commonly as fungal granulomas in the air sacs. In more acute cases in adult birds colonies of the fungus with the appearance of mould can be seen growing on the air sac membranes.

Diagnosis in the acute form is based on the history and symptoms and confirmed by cytology of the lesions during a post-mortem.
Itraconazole terbinafine are commonly used in other avian species but they are not allowed in food producing animals due to the difficulty in providing a safe withdrawal period. Treatment is unlikely to be effective in the acute form.
In chronic cases a presumptive diagnosis is based on history, clinical signs and suggestive abnormalities seen on radiography. The diagnosis is confirmed in the live bird by endoscopy when the lesions can be visualised and samples are collected for cytology and/or culture. Treatment is difficult and currently vorconazole is the most effective treatment although it is very expensive and currently there is no information available to provide withdrawal periods for its use in food producing animals.
The disease can usually be prevented by keeping the litter dry and removing wet litter. This is more concerned if the litter has been damp for a while and as it dries out the fungus sporulates releasing very high numbers of spores. The same is true of feed which has been damp and then dried out with the result that it may obtain large numbers of fungal spores. Dry bedding which has been damp will give off large numbers of fungal spores especially when moved about.

Syngamus trachea

The disease is called “gape” because birds gape or gasp due to heavy infections with the nematode *Syngamus trachea*.
Infections can occur in all kinds of birds with those eating worms especially at risk. Infection is common when they are free ranging and in the same environment as wild birds.
Transmission from bird to bird may be either by ingestion of embryonated worm eggs or larvae or indirect by eating paratenic hosts which is usually earthworms, Gapeworm larvae may remain infective in earthworms for as long as 4 years. For this reason free range birds are more exposed to infection than birds kept indoors.
S. trachea is also called “redworm” because of its noticeable red colour and “forked worm” because of the formation of a Y by male and female worms copulating. Young birds are the most seriously affected by the nematode which can quickly obstruct the lumen of the upper airways by its rapid growth inducing severe dyspnoea and leading to suffocation.
Birds are seen to have heavy gasping and whistling breathing sounds in combination with a stretched neck and head with a beak widely open. Most cases will not present as dramatically and symptoms will be more subtle.
Occasionally they can be seen through the glottis on the oral exam. Tracheoscopy will give a quick diagnosis and worms can be removed endoscopically reducing the risk of a severe reaction if large numbers of worms are killed by treatment in the airways.
At necropsy the worms will be found attached to the trachea. They induce lesions and nodules in the tracheal mucosa as an inflammatory reaction to the permanent attachment of the worms.
Diagnosis is on the basis of typical clinical signs in combination with the detection of the worm eggs in faeces by the flotation method. In severe cases adult worms may be seen in the oral cavity or at necropsy in the trachea allowing a quick diagnosis.
In the individual bird the prognosis is usually good as treatment is usually successful. In heavy infections with severe clinical signs the airway might be blocked and worms can be removed by endoscopy to help breathing and provide time for treatment. Flock treatment is usually successful and has a good prognosis.
Therapy is possible with flubendazol. In case of recurrent disease changing the floor of the cage up to 10cm deep has been recommended however it may not remove all of the infected worms together with regular screening and treatment to prevent build up again. Reintroduction by free ranging birds or worms can occur.

Fractures

Fractures occur relatively commonly in chickens and treatment is the same as in other birds. Chickens are heavy bodied birds and grow rapidly leaving them open to a range of metabolic bone diseases and also susceptible to fractures.

If there is a fracture in one leg or any other cause of lameness then check the contralateral healthy side for signs of pododermatitis developing. Likewise if pododermatitis is identified in one leg always check the other limb for any condition that would reduce weight-bearing.

Bird’s bones have a higher content of inorganic substances (hydroxyapatite) and a thinner cortex than mammals which gives avian bones a relatively brittle consistency with an increased risk of comminuted and exposed fractures. This risk is increased in birds compared to mammals because there is less soft tissue covering the bones in birds. The

bones of the sternum, humerus, pelvis, cervical and thoracic vertebrae are pneumatised. This means that there are extensions of the airsacs into these bones. This has to be considered for the surgical approach, techniques and prognosis during a fracture repairing. Bone structure does not only provide support to the avian body but also stores calcium for the important physiologic processes that occur during egg formation. The most important calcium reservoir for egg development is the medullary bone reserve.
Polyostotic hyperostosis may make fracture repair more difficult due to the bony trebeculae.

In mammals, most long bones have one or more epiphyseal or secondary centres of ossification. The tibiotarsus of birds follows a classic mammalian ossification pattern. The ends of the bones grow rapidly and establish secondary centres of ossification (epiphyses). Avian humerus, radius, ulna and femur develop with endochondral ossification. The growth plates of birds are structurally different from mammalian growth plates but they are controlled with the same physiological processes. Blood vessels coming from the epiphyseal area supply the transitional zone and penetrate the depth of the proliferative zone of the growth cartilage, in mammals blood vessels do not penetrate it.
The majority of the callus tissue during the healing process is derived from the periosteal surface, and the blood supply to the periosteum from surrounding soft tissues is very important therefore it is very important to preserve periosteum and its blood supply during surgery. The intramedullary circulation appears to be of less importance in avian bone healing than in mammals. Healing is faster than in mammals. The healing process starts with the formation of the blood clot that is then substituted with mesenchymal cells and woven bone. If the fracture site is stable the healing process develops without the formation of cartilage and the remodeling with a lamellar bone is complete at 8 weeks. A simple closed fracture can be stable in 3 weeks.
The choice of technique used will reflect the fracture site, the nature of the fracture (for example simple versus compound fractures), the location of the fracture and whether a joint is involved. Other important factors include the function of the bone. The tibialtarsus in a Buff Orphington (a very large breed of chicken) will bear a lot more weight than the humerus or the tibiotarsus of a bantam. Any factors affecting bone strength will influence the repair technique as will age and any associated soft tissue damage.
The principles of orthopaedics in birds are generally modifications of those used for domestic small mammals. However familiarity with their unique surgical anatomy and surgical approaches is important to successfully diagnose and treat those cases that require orthopaedic surgery. The goal of accurate anatomic alignment involves restoration of the bone's original length, axial alignment and rotational orientation, while minimising the trauma to the surrounding tissues and maintaining the blood circulation especially in the case of birds the periosteal blood circulation. This will minimise healing time and reduce the incidence of complications. In backyard poultry an incomplete return to function is sometime acceptable, restoration of flight is not usually necessary.

Rest maybe all that is needed in some cases for example fractures distal to the carpus.

External coaptation is commonly used for fractures of the radius and ulna, especially if there is no displacement.

Intramedullary pinning on its own is rarely used. It is most commonly used in the tie in technique with external fixation.

Plating is rarely if ever used due to damage it causes the avian periosteal blood supply.

External fixation is the most common technique used in avian orthopaedics. It is minimally invasive with little disruption of the fragments their soft tissue attachments and their blood supply. It can be used to bridge areas of shattered bone. External fixation allows a quick return to function reducing the incidence of fracture disease, allowing normal joint movement and more rapid bone healing. Due to avian bone qualities and their thin cortex choice of pin type is very important. Choose pins with a positive profile thread and it may be necessary to drill pilot holes due to their more brittle bone.

Amputation of all or part of the pectoral limb may sometimes be necessary. Amputation of a pelvic limb will rapidly result in severe pododermatitis of the contralateral foot and ambulation on one foot is very difficult.

Fractures of the beak in back yard poultry are not very common. They are most commonly caused by predator attacks. Open fractures are more common in this site with the increased risk of infection leading to malunion. External fixation is commonly used for mandibular fractures. Many small pins can be placed to stabilise many fragments. This allows feeding to continue while the fracture heals. Occasionally depending on the fracture and the size of the mandible, interfragmentary cerclage wiring or pinning may be used as the sole repair method using the principals developed for use in mammals.

Skull fractures can be stabilised by bandaging the mouth in a closed position and placing an oesophageal feeding tube for nutritional support until the dressing is removed. Sometimes cerclage wires need to be used to stabilise skull fractures. Mandibular fractures where the bone will not support external fixation pins can be stabilised by the same method. Ensure that the external nares are exposed and that they are patent, otherwise if the bird is not able to breathe through the nasal cavities the mouth can be left slightly open.

Pododermatitis tends to develop if the chicken is overweight, on a roughened surface, or if one leg and foot bears more of the body weight than the other, or a combination of all these factors. There are varying grades of pododermatitis from mild with hyperaemia of the skin, to severe with osteomyelitis of underlying bone. Radiograph is the best method to determine if there is underlying osteomyelitis, a condition That will require antibacterial therapy and debridement of necrotic tissue. Most cases of ulcerative pododermatitis are somewhere in between mild and severe and consist of a thickened area of skin on the plantar surface of the foot usually over the metatarsal pad, but can also develop on the phalangeal pads. Where surgery is needed it is performed under general anaesthesia with administration of analgesics as this is a painful procedure. An aspirate or tissue sample is collected for culture and sensitivity. The substrate should be made as soft as possible and kept clean. Always try to identify the underlying cause such as lameness on the contralateral limb as this will also need treatment. Following the foot is bandaged to keep the affected part out of wear whilst still maintaining the foor and toes in a normal physiological position A ball bandage similar to what is used in birds of prey but modified for the different foot shape.

Viral arthritis is a condition of chickens most commonly seen between the ages of four and eight weeks but has been seen in younger and older birds. The main signs are swelling of one or both hock joints and associated tendons. Affected birds are lame and reluctant to move. There can also be stunted growth, swelling of the hock joint, gastrocnemius and digital flexor tendonitis. A chronic tendinitis can develop and may result in rupture of the

gastrocnemius tendon. Erosion of the articular cartilage may also occur. Many infected chickens fail to develop clinical signs and remain healthy. It is believed there is a wide variation in virulence and pathogenicity among different virus strains. Serous, sanguinous or purulent joint effusions may be seen, as well as the tendon sheath pathology, damage to articular cartilage and enlarged proximal metatarsal diaphysis. There is often a concurrent myocarditis present.
Diagnosis is based on clinical signs, virus isolation, PCR, and/or serology.
Prevention is by use of the vaccine and cleansing and disinfection of the environment.
Spread of the virus is horizontal mainly by ingestion of faecally contaminated food as the virus can survive many days outside the host. Vertical spread through the egg can also occur. It is shed from the intestinal and respiratory tracts.

Dyschondroplasia sometimes called tibial dyschondroplasia as the tibiotarsus is the most commonly affected bone. It is seen chickens. Clinically affected birds usually have an abnormal gait however some may be symptom free. Radiographically when compared with the normal side or in bilateral cases with a normal bird, there will be an abnormally large area of cartilage at the affected physis. This weakens the bone with the result that pathological fractures occur. Surgical repair is unlikely to be successful due to the lack of bone strength. However if the growth rate is reduced and any nutritional deficiencies corrected, external-coaptation can be applied to give some support while the bone strengthens. Some cases will heal leaving a deformity at the affected area. This will often lead to degenerative joint disease as the birds get older.

Valgus Leg Deformities- Birds with this condition present with twisted and crooked legs, along with long bone distortion, usually less than one month of age. The intertarsal joint is most commonly affected in chickens, the cause is currently unknown. Reducing growth weights reduces the incidence, although this may be purely due to a lower body weight, not improved bone ossification. The condition is worsened with reduced exercise. Valgus deformity is often bilateral, and chronic, and may be associated with displacement of the gastrocnemius tendon.
Differential diagnoses for disorders of cartilage, the growth plate and of ossification include infectious synovitis, dyschondroplasia, valgus/varus deformity, chondrodystrophy, rickets, infectious arthritis, ruptured ligaments and rotated tibia. Corrective osteotomy needs to be done as early as possible to achieve the best outcome. The longer it is left more likely that there are irreversible bony changes of the joints.

Displacement of the gastrocnemius tendon. Chondrodystrophy and valgus deformity are among the metabolic bone diseases which result in long bone deformities of the pelvic limbs. Displacement of the gastrocnemius tendon due to abnormal shape of the bones is common with these conditions. In some early cases it may be possible to surgically replace the gastrocnemius tendon. Corrective osteotomies are often required and the level of success will depend on the degree of joint and long bone deformity that has occurred.

An incision is made over the caudolateral aspect of the joint midway between the lateral condyle of the tibiotarsus and the displaced tendon. Free the tendon of adhesions and return to its correct position in its trochlear groove. The groove may have to be deepened.

The tendon is sutured to the periosteum laterally to prevent reluxation medially. If it is difficult to get a strong anchor in the periosteum then a tunnel through the lateral ridge of the trochlear can be used for the anchor. In very early stages a supportive bandage with or without slint can help to restore the correct anatomy. A corrective osteotomy is often needed which will require careful planning. Correcting any dietary deficiencies is also important.

Spondylolisthesis (Kinky back)- This is seen in chickens mainly broilers between 3 to 6 weeks old. It is due to ventral rotation of the fourth thoracic vertebra causing compression of the spinal cord. The birds are unable to stand and are found sitting on their hocks. Diagnosis is confirmed by radiography. There is no treatment.

Spraddle legs causes the legs to splay laterally from the coxofemoral joint. I have seen this condition most commonly just after hatch, although in the textbooks is described as being more commonly seen around 2-3 weeks of age. In my experience most respond rapidly to hobbling. A tape hobble is fashioned between the two legs. It usually only needs to be worn less than 48-hours. In older birds 2 to 3 weeks of age the differential diagnosis would include disorders of cartilage, the growth plate and of ossification include infectious synovitis, dyschondroplasia, valgus/varus deformity, chondrodystrophy, rickets, infectious arthritis, ruptured ligaments and rotated tibia.
Treatment of advanced cases may be futile.

Crooked neck

Mycoplasma meleagridis causes cervical vertebral osteodystrophy.
Occasional causes of crooked neck in chicks include pathology of the neck muscles, cervical vertebrae and also pathology to the vestibular apparatus. Sometimes using a sponge collar to support the neck in its correct position in rapidly growing chicks would result in a resolution of the problem within two or three days.

Osteoporosis, also known as cage layer fatigue, this is a condition of decreased normal skeletal mineralisation, and is secondary to insufficient weight bearing activity. It is responsible for up to 30% of fractures in commercial poultry. Clinical signs are fractures of the ischium, humerus and keel, spinal fractures leading to paralysis, fractures in other bones, and paresis caused by exposed spinal nerves from bone loss around the spinal column. At necropsy, thin bony cortices and parathyroid enlargement are seen. With the calcium demands of egg-laying, there is continuous remodelling of medullary bone. It seems that laying hens in heavy production start to metabolise structural as well as medullary bone to satisfy the requirements of heavy egg production. This eventually thins the bony cortices. Genetic selection for high egg production, poor nutrition and reduced weight bearing exercise worsens the incidence. Good nutrition is not protective against this disease. Once doing weight bearing exercise (for instance, being changed to free range conditions), bone strength will improve by day 20.

The best prevention is to provide ample exercise, a nutritionally balanced, calcium replete diet and to select only moderate egg producers as pets.

Crooked toes

This condition is most commonly seen in newly hatched chicks there are many possible aetiologies but shortening of flexor tendons is commonly proposed as a cause. Some have proposed a possible heritable component. This condition is much more common in artificially incubated chicks. Poor flooring, brooding with infrared lighting, pyridoxine deficiency and some toxins have been said to worsen the condition.
Treatment is by taping the toes into the correct position. This treatment is very successful if done as soon as the deformity is noticed in very young chicks. The older the chick is the less successful treatment would be.

Ruptured gastrocnemius tendon

Occurring in fast-growing, heavy-bodied birds, clinical signs of a ruptured gastrocnemius tendon are a dropped hock, inability to move, with palpable swelling in the tendon. Birds often sit on the hocks, with toes pointing ventrally. Histopathologically, there can be inflammation due to viral arthritis or staphylococcal or other bacterial osteomyelitis or cellulitis. However, it may be non-inflammatory, and often occurs in the non-vascularised part of the tendon. Differential diagnoses include reovirus infection, staphylococcus, spinal cord injury and tibial dyschondroplasia and displacement of the gastrocnemius tendon. Prevention is via good husbandry, avoiding high impact locomotion.

Calcium Tetany

Whereas more chronic calcium deficiency causes skeletal problems, acute deficiency will present differently. Clinical signs include lethargy, tremors, paralysis and death from cardiorespiratory failure can occur. Diagnosis is made on clinical signs, ionised serum calcium levels, lack of other lesions and response to treatment. Treatment is to supplement with calcium and vitamin D. Prevention is best done via good nutrition, and avoiding excessive supplementation as it is not a substitute for a correct diet.

Avian encephalomyelitis

Signs may include progressive ataxia, incoordination, paralysis, and prostration. Young birds are more susceptible to this disease than older birds. Birds exposed after 2-3 weeks of age may not develop neurologic signs and adults may only experience a slight drop in egg production.
The AE virus is shed into the faeces of infected birds. Shedding occurs for a period of several days to weeks, depending on the age of the infected bird. Ingestion of contaminated faeces, direct bird-to-bird contact, and exposure to contaminated personnel and fomites are the usual sources of horizontal transmission. The virus is resistant to environmental conditions and remains infectious in the environment for long periods of time.
Nearly all chicken flocks worldwide become infected with AE. However, clinical signs are mostly seen in young birds, between 1 to 3 weeks of age. Un-vaccinated adults that become infected after they begin laying eggs, typically do not show signs of clinical disease but do transmit the infection to their offspring.

Serologic assays are commonly be used for a diagnosis. Rising titers in sequential blood samples are highly suggestive of active infection.
Live vaccines are available, they are given in the drinking water.

Newcastle disease cause neurological signs such as paralysis/paresis of the legs, clonic spasms/seizures see above in the respiratory infections.

Mareks disease

This is a very common condition causing neurological signs.
The classic form is asymmetric limb paralysis and the development of pale visceral tumours . These hens is also depressed, there is weight loss, pallor, often diarrhoea, anorexia and death there is no cure.
At necropsy, the affected nerves are often swollen, discoloured and have lost cross-striations.
Most commonly this involves the sciatic nerves and the lungs often have to be peeled back from the body wall to reveal the swellings.
More subtle lesions can be detected by comparing contralateral structures.
Histopathologically, nerves and tumours contain neoplastic lymphocytes.
There is also the acute form in young unvaccinated birds with deaths after three days of depression.

The transient paralysis form where there is a flaccid paralysis of the limbs are neck which results after 24 to 48 hours. Most of these birds go on to develop visceral tumours and die several weeks later.

In the chronic form it is a manifested as immunosuppression with a bird succumbs to other infections.

Mareks disease can also develop as an ocular lymphomatosis.

Some birds were present as a cutaneous leucosis with a lymphocytic infiltration of the feather follicles which is seen as millary lumps in the skin. Marek’s disease affects only chickens. Clinical signs are most commonly seen in birds that are 12 to 20 weeks of age, but can be seen In older and younger birds.
Transmission is via virus shed in skin and feathers, secretions, and droppings. The virus can persist in the environment indefinitely. There is no treatment. Prevention is by administering the vaccine to day old chicks Marek’s disease must be differentiated from lymphoid leukosis based on age of clinical signs, and gross and histopathologic lesions.