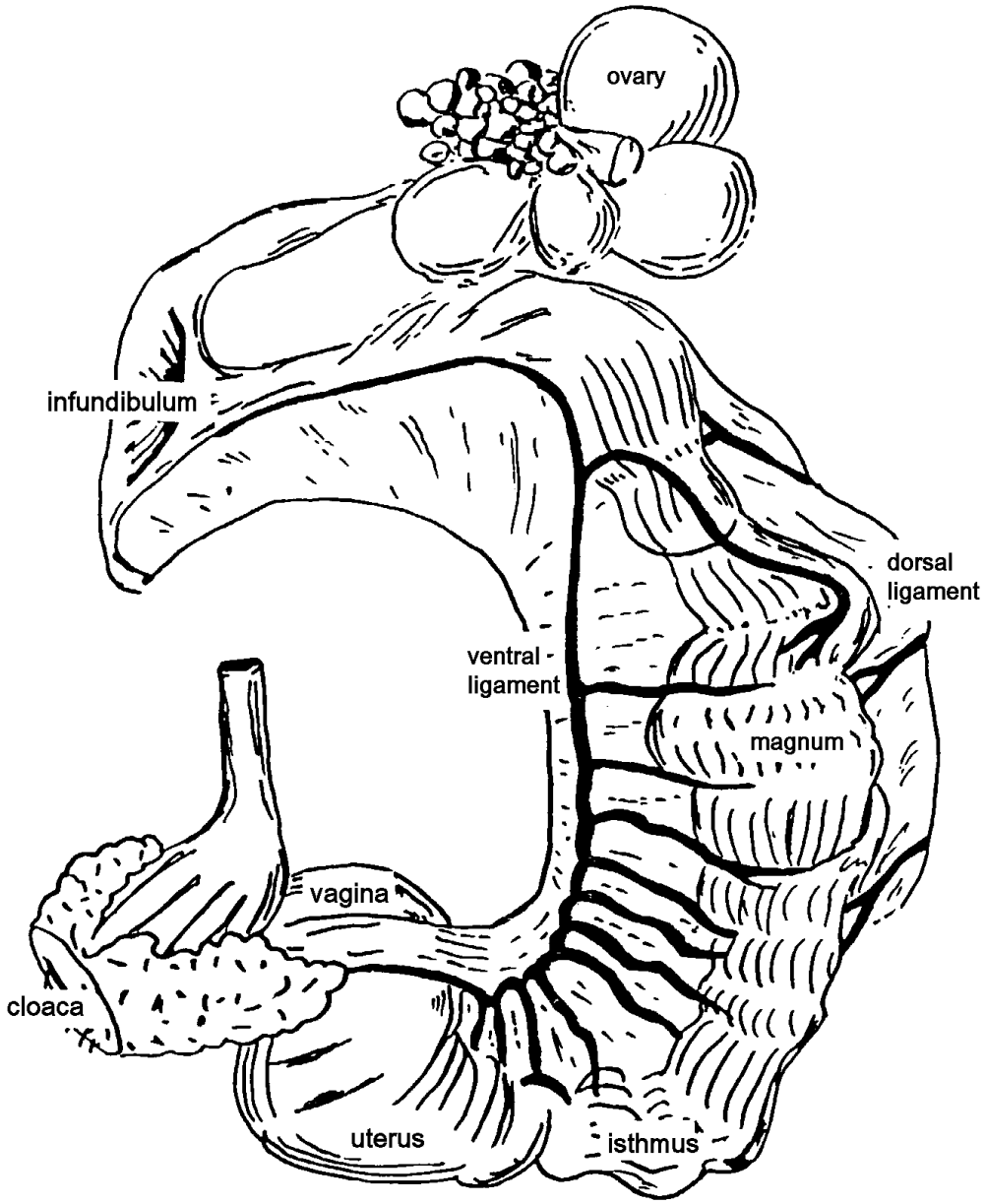


**Figure 1**. Diagrammatic representation of the reproductive tract (left ovary and oviduct, and associated structures) of the Domestic Fowl (courtesy of the Institute of Animal Health, Compton Laboratory, UK).

# Reproductive Diseases of Birds - an Overview

**RL Reece***

## Introduction

Reproductive diseases of avian species pose two quite distinct problems for veterinary practitioners. Firstly, there are disorders or diseases of the reproductive tract of individual birds: these may be life-threatening episodes and are usually presented in a clinical setting[13,14]. Secondly, there are problems of reproduction where veterinary assistance may be sought if either the number of breeding adults of that species is limited or if the birds have considerable economic value as breeders. The latter are likely to be handled on a flock basis. In either situation, it is important for the investigating veterinarian to have a good understanding of the development and function of the "normal" reproductive system of avian species. The scope of this subject is beyond that which can be conveyed in a few pages, and a more lengthy review is available separately with the option to purchase accompanying figures[19]. Diagnosis is one process in a chain of events which hopefully leads to successful treatment, but distinctive clinical signs tend to be rare in avian diseases and recourse to ancillary testings such as clinical pathology, microbiology, radiography, ultrasonic imaging and exploratory laparoscopy, is essential.

The avian inflammatory response is different from that of mammals in several important respects[15]:

1. The predominant granulocyte (heterophil) has eosinophilic rod-shaped granules in the cytoplasm and a basophilic nucleus which may be monomorphic, bilobed, or polymorphic. The term acidophils can be applied to both heterophils and eosinophils, indicating that it can be difficult to differentiate between them! In contrast to mammalian neutrophils, the avian heterophil lacks peroxidase and alkaline phosphatase but they have potent lysosomes and are capable of phagocytosis.
2. The acute inflammatory response can be associated with the formation of syncytial macrophage giant cells 12-72 hrs after stimulation.
3. Suppurative exudates (with masses of heterophils) tend to be inspissated and caseous rather than liquefied.
4. There are no lymph nodes as such in birds, and lymphoid drainage of tissues and organs is not as well developed as in mammals. However, small foci of lymphoid tissue are found in normal tissues: these may comprise lymphoid germinal centres and accumulations of various other lymphoid cells. Many different types of T-cell subsets have been identified in chickens (sometimes designated ChT-cells) and their function is similar to that of equivalent subsets in mammals.
5. Chickens produce three classes of immunoglobulins: IgM, IgG and IgA. Until recent times, some workers preferred to designate the predominant immunoglobulin (IgG) found in yolk and the developing embryo as IgY, because it is slightly different from mammalian IgG. Passive transfer of IgG occurs across the follicular epithelium into the developing ova. All types of immunoglobulins are present in the egg white, but at very low concentrations.

In avian species, contrary to the situation in mammals, the male is homozygous with regard to sex chromosomes (homogametous ZZ) and therefore the neutral sex, whilst the female is heterogametous (ZW) (they are not referred to as X and Y). The sex hormones exert their influence early in development so that the genetic female is demasculinised by her ovarian hormones. This fact allowed young male chickens to be hormonally castrated by implantation with oestradiol. Note: this procedure has been banned in Australia for

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many years; broiler chickens are NOT treated with hormones. Plumage dimorphism and other features used to distinguish males from females in dimorphic species are influenced by gonadal hormones, and many aspects of reproductive behaviour are mediated by sex hormones[16]. Changes in beak colour during the breeding season of some passerines is due to androgens which are produced by both the testes and the ovary. Female Budgerigars have a brown cere during the breeding season but it is blue in males, in immature birds of either sex, and in females with a quiescent ovary.

## The Female Reproductive Tract

### Introduction

In the female of most avian species, only the left ovary and oviduct are functional - there are exceptions: in the Kiwi (*Apteryx australis*) both ovaries are normally functional although there is only one (left) oviduct; and about 25% of the females of some *Falconiiformes* have bilateral ovaries [sparrowhawks (*Accipter* spp), harriers (*Circus* spp) and kestrels (*Falco* spp)][14]. Each ovary contains several thousand ova, and the developing follicles become arranged in a hierarchical order. Ovulation occurs as a result of a surge in pituitary luteinising hormone (LH). In the case of a Domestic Fowl, ovulation may occur on consecutive days for 3-5 days followed by a rest day and then a resumption of lay and so on for a year or more: on the rest day the ripe follicles are not ruptured as there is no surge in LH. The capillaries adjacent to the theca interna of the developing follicle lack basement membranes and are fenestrated. This arrangement allows for the rapid transfer of lipid etc into the developing follicle but also facilitates the movement of septicaemic bacteria into the ovarian stroma.

The oocytes enlarge in response to follicle stimulating hormone (FSH) released by the pituitary. The follicle wall is composed of the oocyte's cell membrane (cytolemma), the perivitelline lamina secreted by the underlying cells, a stratified layer of granulosa cells on a basement membrane, a compact layer of theca interna cells , and a loose layer of fibrous-like cells (theca externa) which merges into the surrounding connective tissue. As maturation of the follicle proceeds, the cytolemma becomes folded and the granulosa cells become cuboidal. The granulosa cells of the developing follicle produce progesterone, the theca interna cells produce oestrogen, and the theca externa cells produce mainly oestrogen and a little progesterone[17]. The primary oocyte undergoes the first reduction division just prior to ovulation.

The oviduct is described as having five parts, namely infundibulum, magnum, isthmus, shell-gland or uterus, and vagina (see Figure 1). The growth, differentiation and function of the normal oviduct are under the control of steroid sex hormones, and these processes are mediated by hormone receptors in mucosal epithelial cells. During post-hatch development the oviduct increases in size in response to oestrogen, and the mucosal cells develop their ability to secrete proteins in response to progesterone.

The ovulated yolk-rich secondary oocyte is received into the ampulla of the infundibulum where fertilisation occurs and an ovum is formed. Passage through the thin walled tubular infundibulum is rapid (15 mins) and the outer layer of the perivitelline membrane is deposited on the developing egg. The magnum has a greatly thickened wall due to longitudinal spiral mucosal folds and a multitude of tubular glands. The egg albumen ("white") is secreted into the developing egg in the 3 hours or so that it takes to pass through this. The beginning of the isthmus may be indicated by a thin white line, and the mucosa is not very folded; the shell-membranes are added to the developing egg in about 1 hour, and the first cleavage division of the ovum occurs here. The developing egg lodges in the shell-gland for about 20 hours in most species, although in the case of the Kiwi, the egg may remain here for several weeks[4]. The complex mucosal folds (lamellae) are pressed flat onto the egg surface and move during secretion thus ensuring even dispersal of the shell secretion. Pigmentation occurs towards the end of shell deposition; the coloration of the egg-shell is due to deposition of porphyrins

in the last few hours of sojourn of the egg in the shell-gland.

During egg laying (oviposition), the sphincter between the shell-gland and the vagina relaxes and the egg is expelled by contractions of the shell-gland. Distension of the vagina initiates a "bearing down" reflex which is associated with an increase in the respiration rate, adoption of an egg-laying posture, and contraction of body muscles. The egg is covered with mucus (the "bloom") during passage through the vagina.

The unincubated but fertilised egg of the Domestic Fowl has a blastoderm which is only 3mm in diameter and almost impossible to detect by unaided visual inspection. Incubation periods for some of the common species are given in Table 1.

The onset of broodiness is associated with an increase in prolactin secretion and a decrease in plasma luteinising hormone and ovarian hormones. It is coupled with regression of the ovary and oviduct. In some altricial species incubation may begin before the clutch is complete.

## Physical Abnormalities

### Atresia

A substantial proportion (20%) of the smaller follicles (<6 mm diameter) normally undergo atresia. The primary oocyte becomes necrotic and the follicle is obliterated by fibrous tissue or becomes cystic by being lined by luteinised granulosa and/or theca cells[27]. Atresia of large yolky follicles is associated with a sudden cessation of lay such as the onset of broodiness, adverse environmental conditions, deprivation of food or water, some metabolic conditions, and acute systemic disease. Affected follicles lose their turgidity and the contents may appear discoloured; the stigma (line of future rupture) becomes prominent. Atresia is more prevalent at the time of nesting. These large atretic follicles are prone to haemorrhage and can be difficult to distinguish from oophoritis.

### Cystic follicles

Cystic follicles have been reported in Canaries and Budgerigars. Their significance is unknown and they need to be differentiated from cystadenocarcinomas.

### Cystic oviduct

Cystic changes in the mucosa of the functional oviduct have been reported but their aetiology and significance are unknown. Ovariohysterectomy is the preferred treatment. Remnants of the embryonic right oviduct can persist in adults and become distended with serous fluid. These may act as a space occupying lesion. Cystic structures may also be found involving the left oviduct.

### Abdominal yolk and eggs

Some ovulated oocytes escape into the abdominal cavity (ectopic ovulation) but the yolk is rapidly resorbed. On examination, either by laparoscopy, laparotomy or at necropsy, the peritoneal membranes are coated in yolky material, but there are no fibrin flocks nor are there any marked inflammatory changes. Abdominal yolk is commonly observed after handling of birds or after other severe disturbance, and is probably associated with premature rupture of turgid follicles. If the yolk material remains sterile there are unlikely to

be any clinical signs. Occasionally, partially or fully formed eggs are found free in the abdominal cavity, necessitating either retropulsion up the oviduct or rupture of the organ.

Infection of 1-day-old maternal antibody-free chickens with infectious bronchitis virus may lead to non-patency of the oviduct lumen, cyst formation and/or hypoplasia. When these birds mature, developing eggs are not able to pass down the oviduct and yolk material is found in the abdominal cavity[7]: these are frequently referred to as internal layers because they do not lay eggs, but that terminology creates an impression of fully formed internal eggs which is not the case with this condition. Presumably, something similar can happen following developmental malformations in any species. In such cases the reproductive tract needs to be examined and if there are predisposing causes such as segmental hypoplasia, obstruction or adenomas of the oviduct, an ovariohysterectomy should be performed.

**Ovarian haemorrhage**

Some haemorrhage into the follicular calyx following ovulation is normal. Occasionally it may be marked and the resultant clotted blood may be incorporated into the developing egg as a "blood spot". The blood vessels associated with turgid follicles are very engorged just prior to ovulation and prone to haemorrhage and congestion.

**Persistent egg laying**

Some passerine and psittacine birds lay eggs on a more-or-less continuous basis, especially solitary caged birds. The diets of such birds are usually inadequate for this effort and the birds' reserves of Ca become depleted; they are then prone to egg binding and prolapse. Treatment with progesterone may be of value, but ovariohysterectomy needs to be considered.

**Egg binding**

This probably occurs as a result of atony or spasms of the oviduct. It is commonly seen in Budgerigars and Cockatiels but that may be a reflection of the numbers kept rather than species susceptibility. Some strains of Domestic Fowl appear to have a higher incidence of egg binding than other strains and other factors are also associated with predisposition to egg binding, namely egg oversize, first egg of a clutch, obesity, young age, pelvic and abdominal tumours, cold weather and hypocalcaemia. It is often a problem in solitary caged birds which have not laid an egg for some time, and those which have never laid an egg previously.

Affected birds show straining and frequent tail wagging in the early stages. They have an abnormal gait, perch unsteadily, sit in the bottom of the cage, or they may adopt a "penguin-like" squatting pose. Respiratory distress may be a feature, and inactivity and inappetence are noted. If the retained egg is not removed the affected bird may become exhausted. A clinical examination and/or radiography can be useful to reveal the location of the egg and relative size. It may be impossible to distinguish by palpation a retained egg from fat deposits in overfat birds. Oversized eggs may be associated with cloacal tearing, haemorrhage and congestion. Egg binding is an acute life-threatening episode in small passerines, and urgent surgical intervention is required. In species such as Budgerigars and Cockatiels there may be opportunity to try more conservative treatments such as lubricating the vent, warming the birds' cage to around 30°C, and gentle manipulation and pressure. Intramuscular injection of Ca borogluconate (0.5ml/100g live weight) will lead to expulsion

of normal sized eggs in cases of hypocalcaemia induced shell-gland atony. It is interesting to note that the egg shells in such cases are thin. The bird should be anaesthetised in urgent or severe cases, and the egg contents aspirated with syringe and needle and the egg shell removed[21,24].

### Trauma

Trauma to the cloaca of female birds may be inflicted by other birds in the same group, or be a consequence of laying a large or misshapen egg, egg binding or prolapse. It is likely that another egg will be laid within 1-2 days so treatment or surgical repair needs to take that into account: ovulation can be suppressed by treatment with progesterone.

### Oviduct herniation

Herniation of the oviduct, often with a fully-formed egg in the shell gland, is sometimes encountered in pigeons and other species. These require surgery.

### Cloacal and/or oviduct prolapse

Prolapse associated with eversion of the cloaca with or without prolapse of the distal oviduct, usually the vagina, is an occasional sequelae to laying oversized eggs or excessive straining. The prolapsed tissue is liable to be traumatised. The affected tissues need to be cleaned and replaced, and held in position with purse-string sutures. The bird must be treated with hormones to stop any further immediate egg production.

## Infectious Diseases

### Oophoritis

Inflammation of the ovary has been associated with several important bacterial diseases of poultry, particularly pullorum and *Salmonella enteritidis*. Characteristically, the ovarian follicles are distended with serosanguinous or fibrinopurulent material and the ovary is congested. Histologically, there is inflammatory cell infiltration and necrosis, with debris in the lumina of affected follicles. The ovary can also be affected in cases of salmonellosis due to other serotypes, coliform septicaemias (chronic respiratory disease, colibacillosis), fowl cholera (*Pasteurella multocida*) pseudotuberculosis (*Yersinia pseudotuberculosis*) and tuberculosis.

**Differential diagnosis will include:**

1. Atresia of the ovarian follicles associated with abrupt cessation of lay.
2. Congestion of the turgid follicles in birds dying of heat stress, or circulatory collapse, suffocation, sudden death syndrome.
3. There is some controversy over the isolation of *S. enteritis* from ovaries: does the isolation of the organism constitute infection even in the absence of inflammation? In some cases, there is overt oophoritis but such birds usually are no longer laying eggs and therefore unlikely to be producing infected/contaminated eggs.
4. Small foci of eosinophils are found in the stroma of normal active ovaries and can be difficult to distinguish from heterophils. In addition, the ovary is a site of predilection for infiltration with myelocytoma cells (heterophil series cells transformed by one of the avian leucosis viruses): mitotic figures are common in these myeloma cells. Extramedullary myelopoiesis can be found in the ovary of

young birds (up to a few weeks of age) and besides only a few mitotic figures there is a gradation of cell type consistent with some maturation.

**Salpingitis**

In bacterial salpingitis, the oviduct may be distended with fibrinopurulent debris, often arranged in concentric rings (lamella) around degenerate developing eggs. Salpingitis may extend from cloacal trauma. It may also be found in young chickens with systemic diseases such as colibacillosis.

There are several viral diseases of poultry [infectious bronchitis virus (IBV) and haemagglutinating adenovirus (EDS'76)] which affect the oviduct and are characterised by a decreased rate of lay, deformed and/or poorly pigmented egg shells, and poor internal egg quality, but there are no obvious gross lesions in the oviduct. Histologically, there may be oedema and inflammatory cell infiltration. In the case of EDS'76, inclusion bodies may be found in the nuclei of epithelial cells lining the infundibulum, magnum and shell gland. Sections of the magnum of hens laying eggs with watery whites following IBV infection can be stained with alcian blue to reveal multiple small foci with a lack of cilia on epithelial cells and an absence of alcian blue positive cytoplasmic granules. A mild salpingitis is associated with *Ureaplasma* spp infection in turkeys.

**"Egg" peritonitis**

Yellow flecks or sheets of fibrin or fibrinopurulent material may be found in the abdominal cavity of adult female birds with an active or recently active ovary: the peritoneal blood vessels are engorged. The yellow material is frequently assumed (wrongly) to be egg yolk. A variety of bacteria can be isolated from these lesions including coliforms, streptococci, salmonellas and *Pasteurella* spp and it is thought that they gain access to the abdominal cavity following cloacal trauma. Affected birds have raised plasma fibrinogen concentrations and heterophilia. Detailed clinical examination, laparoscopy and radiography will be helpful. Abdominal irrigation with antibiotics may be of some value. Fibrinous peritonitis is also a feature in some systemic diseases such as chlamydiosis, paratyphoid, fowl cholera and virulent avian influenza but in these males, and females without active ovaries, may be affected.

Differentiation of "egg" peritonitis from abdominal ovulation can be difficult: in the latter the peritoneal surface is usually covered with oily yolk rather than flecks, and the blood vessels are not very engorged.

**Oviductal parasites**

Trematodes can be found in the oviduct: *Prosthogonimus ovatus* in Domestic Fowls and turkeys, and *P. macrorchis* in waterfowl. The life cycle involves both an aquatic snail and a dragon fly naiad. Ascarids can be detected in shelled eggs but this is extremely rare: presumably the ascarids migrate from the cloaca into the oviduct and are then entrapped in the descending egg.

**Neoplasia**

Gonadal tumours are common in Budgerigars[18]. Excellent descriptions of the more prevalent tumours of the Domestic Fowl are available[5,11] and can be applied to most other avian species.

## Ovary

### Ovarian adenocarcinomas

These may be multiple small white or creamy pedunculated nodules, large fleshy masses or cystic nodules (cystadenocarcinoma). The neoplastic cells arise from the germinal epithelium covering the ovary. Histologically, they may be arranged as well differentiated epithelial lined tubules, or as sheets of relatively anaplastic epithelial-like cells. There may be metastases throughout the abdominal cavity. Cystic follicles have been reported in some species but these lack the mitotic figures found in adenocarcinomas.

### Granulosa cell tumours

These are large yellow or cream coloured nodular masses with a smooth surface. Central areas of necrosis and/or haemorrhage often give these a pink tinge. Metastases to the adjacent viscera can occur. Histologically, these tumours can be extremely varied with follicle-like structures, loose acini with some attempt at pallisading, or sheets of anaplastic cells. The stroma usually contains abundant plump theca cells. These tumours are associated with high plasma oestrogen concentrations yet granulosa cells normally produce progesterone (?).

### Theca cell tumours

These are usually a single cream coloured nodule. The predominate cells are plump and fusiform, often with vacuolation of the cytoplasm. Lobulation may be a feature, and areas of necrosis and haemorrhage are frequent.

### Dysgerminomas

This rare tumour is derived from the seminiferous elements within the left ovary or the vestigial right ovary, and is usually presented as a large pedunculated mass with a thick capsule. Histologically, there are tubules lined by polygonal or round Sertoli-like cells and arranged in a manner somewhat reminiscent of seminiferous tubules.

### Arrhenomas and arrhenoblastomas

The term arrhenoma can be applied to an ovarian tumour which contains testicular elements, or to a diverse group of ovarian tumours associated with virilism because of the production of androgens, however remember that in avian spp the male is the neutral sex and cessation of production of ovarian hormones will invariably lead to a reversion to male type of external features, ie virilism? The histogenesis of these arrhenomas is uncertain: they may be derived from ovarian cortical mesenchymal elements, or the hilar cells of the rete ovary (or its equivalent on the vestigial right ovary), or they may arise in association with granulosa cell tumours. In poultry, these tend to be large and fleshy, pedunculated and malignant tumours. Histologically, they are extremely variable even within the same case. The more differentiated forms consist of adenomatoid tubules and cords lined by columnar epithelial cells often two cells thick and lacking a definite lumen, whereas others are a loose network of fusiform cells, and epithelial cells arranged as nests, cords or rosettes. Scattered amongst these tubules are nests of polyhedral lipoid laden cells resembling Leydig cells. These tumours also contain abnormal theca and granulosa cells, and can be difficult to differentiate from anaplastic adenocarcinomas. Unfortunately, no hormonal studies have been carried out

on affected poultry.

### Other tumours

The ovary is a common site for infiltration of lymphoma cells in Marek's disease or lymphoid leucosis in Domestic Fowls, and lymphomas in other avian spp. It is also involved in other multifocal tumours including myelocytomas, plasma cell myelomas, fibrosarcomas etc, and this feature is not restricted to poultry. In addition, the ovary may be the site of origin of myxomas and fibromas. Teratomas can occur in the ovary or may be found as a large mass seemingly free in the abdominal cavity but attached by a thin pedicle. These contain feathers, bone and/or other tissues.

## Oviduct

### Leiomyomas, leiomyofibromas and fibroleiomyomas

These occur on the anterior free border of the ventral ligament of the oviduct of the Domestic Fowl. These tumours are usually single, round and sharply circumscribed with prominent blood vessels. Histologically, they are composed of interlacing whorls and bundles of smooth muscle with a variable amount of fibrous tissue. A high incidence of these tumours can be induced in Domestic Fowls by treatment with diethylstilboestrol and progesterone but the incidence of spontaneous cases varies enormously between breeds and lines with some indication of a genetic predisposition[5].

### Adenomatosis, adenomas and adenocarcinomas

These conditions merge into each other and are more prevalent in the magnum of adult hens than in other parts of the oviduct. Adenomatosis is found in apparently healthy in-lay hens and turkeys and is characterised by multiple small foci of closely packed columnar epithelial cells in the mucosa: these cells are arranged concentrically rather than being oriented towards the lumen. Single or multiple polyps of adenomatoid tissue (adenomas) can be found protruding into the lumen of the oviduct. Highly invasive adenocarcinomas of the magnum are the most common cause of metastatic abdominal adenocarcinomas in poultry and are reported in most surveys. They are also found in many other avian species. The nodules may be composed of well defined epithelial lined tubules and cords, or sheets of relatively anaplastic epithelial like cells. Emboli can be found in oviductal blood vessels and there may be spread to the lungs.

Adenocarcinomatous cells derived from the magnum retain their oestrogen and progesterone receptors[6], and contain ovalbumen in the cytoplasm, thus confirming their origin from the magnum. There is strong evidence to indicate that ovarian hormones are involved in the induction and maintenance of magnum adenocarcinomas, and these tumours can be suppressed by anti-oestrogenic substances. Abdominal spread of metastatic tumours occurs via tunnels between the coelomic membranes.

### Metastatic abdominal adenocarcinomas

These may originate from the ovary or the magnum of the oviduct and have been reported in many avian spp[18]. Affected birds are usually in poor condition with a pendulous abdomen. There is some ascites (often containing mucus) and multiple small white, grey or yellow nodules on the serosal surface of the viscera, particularly the duodenal loop because it is the

most dependant organ in the abdomen. Sometimes they are erroneously referred to as pancreatic adenocarcinomas. Adenocarcinomatous nodules may also be found on the serosal surface of the spleen and liver, and the ovary is usually affected. The serosal implants tend to be heavily fibrosed, and the neoplastic cells may form discrete acini or occur as sheets of anaplastic epithelial cells. Metastatic abdominal adenocarcinomas need to be differentiated from metastatic abdominal fibrosarcomas and myxosarcomas. These probably originate in the ovary and the latter are characterised by abundant tenacious mucinous ascites. Mesotheliomas are very rare. "Egg" peritonitis and abdominal egg material do not have serosal nodules. Serosal implants of lymphomatous cells can be found in some cases of Marek's disease.

#### Lipomas

Pelvic lipomas although not originating in the reproductive tract may act as a space occupying lesion and be associated with egg binding and other problems.

## Male reproductive system

### Introduction

The testes of an adult male bird are paired spherical or bean shaped organs adjacent to the mid-line and overlying the anterior poles of the kidneys. They may be white, yellow or heavily pigmented with melanin. Histologically, they are composed of seminiferous tubules lined by Sertoli cells and a multi-layered germinal epithelium containing spermatogonia in various stages of development, and interstitial (Leydig) cells: the maturing spermatozoa tend to be arranged in clusters with their heads in Sertoli cells. In those species with a definite breeding season, the testes may enlarge greatly (eg x500 in the House Sparrow) but during the non-breeding season the testes revert to a pre-pubertal state with the deposition of abundant interstitial fat.

There are no accessory sex glands as such in male birds but in some species there is a convolution of the vas deferens near the cloacal wall which creates the seminal glomus. A protrusible phallus is found in ducks, geese and related birds (Anseriformes), penguins, and ratites (emus, ostriches). The flaccid phallus lies on the ventral cloacal wall, and erection results from engorgement of lymphatics. In other species, a non-protrusible phallus is found on the ventral cloacal wall. This engorges with lymph, and semen is ejected from the vas deferens and passes along longitudinal folds between the phallic bodies.

### Physical abnormalities

#### Testicular hypoplasia, atrophy and degeneration

These can be difficult to differentiate especially in those species with a seasonal breeding cycle, and where the normal structure of the testes is not known. In general, hypoplasia is a failure to reach normal structure and is characterised by tubules of small diameter lined only by Sertoli cells and with a thickened basement membrane. Atrophy is a normal event in the non-breeding season of those species with a definite breeding season, and the seminiferous tubules revert to a prepubertal state. There is an increase in stromal fat tissue. Atrophy may occur in association with other generalised conditions such as emaciation: it is generally considered reversible. Degeneration implies a failure of maturation of spermatazoa and a collapse of tubules with an increased thickness of the basement membranes. It may be associated with stromal fibrosis and therefore may be non-reversible. A delayed onset of spermatogenesis can occur and may be associated with poor fertility - this can be significant in commercial poultry.

### Phallic prolapse

Phallic prolapse and ulceration is a common problem in Anseriformes (drakes and ganders). The aetiology and pathogenesis of this condition is not known. There are reports of the isolation of *Neisseria* spp and *Mycoplasma* spp, and there is some evidence of this being a true venereal disease with cloacitis, oophoritis, salpingitis and infertility in the females. In other instances, phallic prolapse and ulceration is secondary to minor trauma and a great variety of bacteria can be isolated from affected phalli, probably as a consequence of gross faecal contamination. Enteric protozoa may also be found on the surface of the prolapsed phallus. Affected birds may not show any clinical signs, and a partially prolapsed phallus can be difficult to detect unless the bird is examined carefully. Flock fertility may be decreased if there is a high incidence of affected males. Broad-spectrum antibiotics may be applied topically, and given systemically in the early stages. In chronic cases, surgical correction and/or amputation need to be considered.

### Infectious diseases

#### Orchitis

The testes may contain granulomas in *S. typhimurium* infections in pigeons and *Y. pseudotuberculosis* infections in any species. Involvement of the testes is also found in tuberculosis, colibacillosis and other bacterial septicaemias.

#### Neoplasia

##### Seminomas

These are usually large unilateral well encapsulated masses containing islands of large round or polyhedral cells with pale nuclei and acidophilic cytoplasm. The stroma is well developed.

##### Sertoli cell tumours

These are large pale fleshy tumours, often with cystic and haemorrhagic foci. They may be comprised of dense sheets of epithelial-like cells, or tubules in a delicate stroma. The cells have a basal nucleus with slightly basophilic cytoplasm which may be vacuolated. Metastases to the adjacent viscera have been recorded. There is a famous case of a Sertoli cell tumour in an incompletely caponised (carstrated?) cockerel which developed secondary sexual characteristics of a hen (?)[25]. There are a few cases in the literature mentioning possible feminisation of Budgerigars affected with this type of tumour[3], but such reports need to be treated with caution as the secondary sexual characteristics which develop are not very pronounced. Grossly and histologically, Sertoli cell tumours resemble granulosa cell tumours.

##### Leydig cell tumours

These are an extremely rare tumour, although they may be found as a component of a seminoma. Neoplastic Leydig cells are large and polygonal with granular acidophilic cytoplasm, and they tend to be arranged as irregular acini.

### Other tumours

As with ovarian tumours, the testes can be involved in Marek's disease, lymphoid leucosis and other multifocal tumours such as myelocytomas and fibrosarcomas, or may be the site of origin of fibromas or myxomas. Teratomas may be attached by a thin pedicle and lie within the abdominal cavity.

## Differentiation of monomorphic species

Many avian species lack external sexual dimorphism. Sexing by laparoscopy is a simple surgical procedure but it is not without risk: post-surgical mortality rates of up to 2% have been reported but after practice it should become less than 1%. Occasionally individuals cannot be confidently sexed using this procedure because of excess fat deposits, sexual immaturity and other problems. The sex of a bird can be determined by direct observation of chromosomes in metaphase but this requires ready access to sophisticated laboratory facilities[8]. Analysis of the faecal oestrogen: testosterone ratio correctly sexes most adult breeding birds but much still needs to be learnt about age and breeding season variations in various species[26]. The phallus of young male ratites and penguins can be detected by cloacal examination with a fibreoptic proctoscope, or by digital examination[22,23].

The sex of a Domestic Fowl can be determined by testing of blood cells with a non-radioactive probe against the W chromosome[28]. In so called "vent sexing", the cloaca of 1-day-old chicks is mildly everted and a trained operator can identify the central phallic papilla (about pin head size) on the inner edge of the cloaca of males. For feather sexing of 1-day-old chicks, the male parents must be homozygous for rapid feathering (kk) and the female parents be hemizygous for slow feathering (K); the gene being carried on the Z (sex) chromosome[9].

### Sex Reversal

Hens which crowed have been recognised since ancient times. There is an example of a Medieval hen pheasant which developed male characteristics in the Hunterian Museum (Royal College of Surgeons of England) and other cases of sex reversal from female to male have been reported in pheasants[12] and pigeons. Most documented cases of sex reversal in avian species have been from female to male, probably because the male is the neutral sex. Observations also tend to be more common in poultry than wild birds, and there has always been a tendency to keep far greater numbers of female poultry than males.

Destruction of the ovary so that oestrogens are no longer produced may cause the affected bird to revert to secondary characteristics typical of males. Masculinisation or defeminisation is noted with some primary ovarian tumours, hence the suspicion that they may be producing male hormones. On the other hand testicular tumours are not normally associated with sex reversal from male to female, probably because such tumours are unilateral with the unaffected testis still secreting testosterone.

Sertoli cells are the male equivalent of granulosa cells, and Sertoli cell tumours in mammals are associated with oestrogen production leading in some cases to demascularisation or feminisation. Clinical histories of demasculinisation or feminisation of Budgerigars, as shown by change in cere colour (from blue to brown), has been associated with seminomas and Sertoli cell tumours[3].

Ovariectomy of young Domestic Fowls is associated with hypertrophy of the vestigial right gonad into an organ resembling an ovo-testis or testis, depending upon the age at which the chickens are treated. Treated birds undergo reversion to male type. Alternatively ovo-testes can arise spontaneously in an apparently normal ovary. They are usually small firm nodules composed of an ovarian cortex and a medulla containing testiculoid-like tubules lined by Sertoli cells. They have a predisposition to neoplasia particularly ovarian Sertoli cell tumours and arrhenomas/arrhenoblastomas. Spermatogenesis is not normally a feature.

A recent survey found that up to 1% of Domestic Fowl hens were triploid (ZZW) and some of these showed reversion to male type after producing eggs. Some such birds developed testes-like gonads with limited spermatogenesis and their blood hormone concentrations were similar to males[10]. In one documented case, a hen which laid eggs then became a cockerel and sired chicks[6].

Hermaphroditism, that is the presence of gonads of both sexes in the one bird, has been reported in pigeons[20] and Domestic Fowl. In dimorphic species, there is a very rare but interesting phenomena known as lateral gynandromorphism where one side of the body has a testes and external male features, whilst the other side has an ovary and external female features. It has been reported in a Bullfinch and a pheasant[29]. There have been several reports of asymmetry with regard to secondary sexual characteristics in the Domestic Fowl.

## The Investigation of Reproductive Problems

The main components of the avian reproduction cycle to be considered in any investigation into reproductive problems are:

1. the laying of eggs;
2. the fertility of eggs which are laid;
3. the hatchability of eggs which are fertile;
4. the survival of hatchlings from eggs which hatch; and
5. the successful breeding from birds which have survived to adulthood.

Disease is often thought of in terms of particular clinical signs or specified pathogens, but it is better to consider that disease is less than anticipated (sub-optimal) performance. This may be expressed as not enough eggs, low fertility, poor hatchability, poor survival of hatchlings and/or unsatisfactory breeding of the progeny. There is an implicit comparison of these to an optimal "figure" but that may be unrealistic or undefined. A common problem is that the performance which is considered sub-optimal may be very difficult to assess or be poorly defined.

## References


1 Anjum AD, Payne LN & Appleby EC (1988). Spontaneous occurrence and experimental induction of leiomyoma of the ventral ligament of the oviduct of the hen. *Research in Veterinary Science*. **45**: 341-348.

2 Anjum AD, Payne LN & Appleby EC (1989). Oestrogen and progesterone receptors and their relationship to histological grades of epithelial tumours of the magnum region of the oviduct of the domestic fowl. *Journal of Comparative Pathology*. **100**: 275-286.

3 Beach JE (1962). Diseases of Budgerigars and other cage birds, a survey of post-mortem findings, parts I and II. *Veterinary Record*. **74**: 10-15 & **74**: 63-68.

4 Calder WA & Rowe BE (1977). Body mass changes and energetics of the Kiwi's egg cycle. *Notornis*. **24**: 129-139.

5 Campbell JG (1969) Ed, Tumours of the Fowl. London, Heineman: 291p.

6 Crew FAE (1923). Sex reversal in the fowl. *Proc Royal Soc B*. **95**: 256-278.

7 Crinion RAP, Ball RA & Holfstad MS (1971). Abnormalities in laying chickens following exposure to infectious bronchitis virus at one-day-old. *Avian Diseases*. **15**: 42-48.

[8] Delhanty JDA (1989). Rapid chromosomal sexing of birds by direct and short term culture techniques. *Veterinary Record*. **125**: 92.

[9] Dun P & Macleod H (1986). Feather sexing in white layers is reliable. *Poultry (Misset)*. **2**: 44-45.

[10] Frankenhuis MT, Walsum J van, Boer LEM de *et al*. (1990). Triploidy and intersexuality in adult commercial layers. *Avian Pathol*. **19**: 3-14.

[11] Fredrickson TN (1987). Ovarian tumours of the hen. *Environ Hlth Perpec*. **73**: 35-51.

[12] Harrison JM (1932). A series of nineteen pheasants (*Colchinus phasianus*) presenting analagous secondary sexual characteristics in association with changes in the ovary. *Proc Zool Soc London*. **1**: 193-203.

[13] Hasholt J (1966). Diseases of the female reproductive organs of pet birds. *J Small Anim Pract*. **7**: 313-320.

[14] Keymer IF (1980). Disorders of the avian female reproductive system. *Avian Pathol*. **9**: 405-419.

[15] Montali,RJ (1988). Comparative pathology of inflammation in the higher vertebrates (reptiles, birds and mammals). *J Comp Path*. **99**: 1-26.

[16] Ottinger MA, Adkins-Regan E, Buntin J *et al*. (1984). Hormonal mediation of reproductive behaviour. *J Exp Zool*. **232**: 605-616.

[17] Porter TE, Hargis BM, Silsby JL & El-Halawani ME (1989). Differential steroid production between theca interna and theca externa cells: a three-cell model for follicular steroidogenesis in avian species. *Endocrinology*. **125**: 109-116.

[18] Reece RL (1992). Observations on some naturally occurring neoplasms in birds in the state of Victoria, Australia. *Avian Pathol.* **21**: 2-32.

[19] Reece RL (1993). Reproductive Diseases of Birds. A monograph. Elizabeth Macarthur Agricultural Institute, Camden: 130pp.

[20] Riddle O, Hollander WF & Schooley JP (1945). A race of hermaphrodite-producing pigeons. *Anat Rec*. **92**: 401-423.

[21] Rosskopf WJ & Woerpel RW (1984). Egg binding in caged and aviary birds. *Mod Vet Pract* **65**: 437-440.

[22] Samour HJ, Stevenson M, Knight JA & Lawrie AJ (1983). Sexing penguins by cloacal examination. *Vet Rec*. **113**: 84-85.

[23] Samour JH, Markham J & Nieva O (1984). Sexing ratite birds by cloacal examination. *Vet Rec*. **115**: 167-169.

[24] Schultz DJ (1981). Reproductive system. In: Refresher Course in Aviary and Caged Birds, Proc 55 Postgrad Comm Vet Sci, Univ Sydney: pp 584-589.

[25] Siller WG (1956). A Sertoli cell tumour causing feminization in a brown leghorn. *J Endocrin*. **14**:

197-203.

26 Stavy M, Gilbert D & Martin RD (1979). Routine determination of sex in monomorphic bird species using faecal steroid analysis. *Int Zoo Yearbook*. **19**: 209-214.

27 Uhrin V (1984). Follicular atresia in the growing ovary of fowl. *Veterinarni Medicina*. **29**: 181-188.

28 Uryu N, Nagata Y, Ito K *et al*. (1989). Determination of the sex of chickens by a biotin-labelled deoxyribonucleic acid probe. *Poult Sci*. **68**: 850-853.

29 Walker KM (1924). The internal secretion of the testis. *Lancet*. Jan 5:16-21.

**Table 1**

The incubation periods for some of the commonly kept avian species.

| Scientific name | Common name | Incubation period (days) |
|---|---|---|
| Anseriformes: | | |
| *Anser anser* | Domestic Goose | 28-34 |
| *Anas platyrhynchos* | Domestic Duck or Mallard | 28 |
| *Cairina moschata* | Muscovy | 35 |
| Columbiformes: | | |
| *Columba livia* | Domestic Pigeon or rock dove | 17 |
| Galliformes: | | |
| *Colinus virginianus* | Bob-white Quail | 24 |
| *Corturnix cortunix japonica* | Japanese Quail | 17 |
| *Gallus gallus domesticus* | Domestic Fowl or bantam | 21 |
| *Meleagris gallopavo* | Turkey | 28 |
| *Phasianus colchicus* | Common Pheasant or Ring-neck Pheasant | 23-28 |
| Passeriformes: | | |
| *Serinus canaria* | Canary | 13-14 |
| *Taeniopygia guttata* | Zebra Finch | 12 |
| Psittaciformes: | | |
| *Cacatua galerita* | Sulphur-crested Cockatoo | 30 |
| *Melopsittacus undulatus* | Budgerigar | 18 |
| Struthioformes: | | |
| *Struthio camelus* | Ostrich | 35-43 |