CHAPTER
18

Evaluating and Treating the Reproductive System

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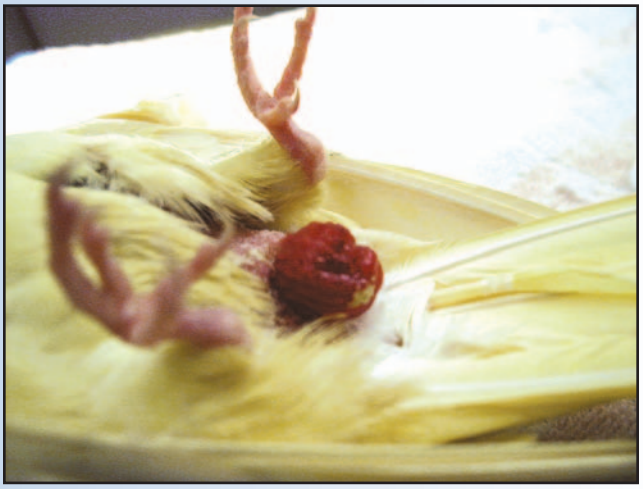


Reproductive Embryology, Anatomy and Physiology

FORMATION OF THE AVIAN GONADS AND REPRODUCTIVE ANATOMY

The avian gonads arise from more than one embryonic source. The medulla or core arises from the mesonephric ducts. The outer cortex arises from a thickening of peritoneum along the root of the dorsal mesentery within the primitive gonadal ridge. Mesodermal germ cells that arise from yolk-sac endoderm migrate into this gonadal ridge, forming the ovary. The cells are initially distributed equally to both sides. In the hen, these germ cells are then preferentially distributed to the left side, and migrate from the right to the left side as well.[58] Some avian species do in fact have 2 ovaries, including the brown kiwi and several raptor species. Sexual differentiation begins by day 5 in passerines and domestic fowl and by day 11 in raptor species. Differentiation of the ovary is characterized by development of the cortex, while the medulla develops into the testis.[30,58]

As the embryo develops, the germ cells undergo three phases of oogenesis. During the first phase, the oogonia actively divide for a defined time period and then stop at the first prophase of the first maturation division. During the second phase, the germ cells grow in size to become primary oocytes. This occurs approximately at the time of hatch in domestic fowl. During the third phase, oocytes complete the first maturation division to

become secondary oocytes. Completion of the second maturation period results in an ovum.[30,34,53,58]

The ovarian medulla consists of blood vessels arranged in irregular vascular zones, interstitial cells, autonomic nerve fibers and smooth muscle. The ova are located peripherally in the cortex of the ovary. The ovarian surface is covered by parietal peritoneum with an underlying layer of dense connective tissue, the tunica albuginea. The ovary is located just caudal to the adrenal gland, near the tip of the cranial division of the kidney. It lies deep to the abdominal air sac, which forms an ovulation pocket near the time of lay. This pocket is thought to help receive the ovulated ovum with its yolk into the oviductal opening. The pocket is suspended by a dorsal mesentery, the mesovarium. Vascular supply to the ovary is through the cranial renal artery, which has several short branches. There are often two ovarian veins that drain blood directly into the caudal vena cava. The ovary has a roughened, granular appearance due to follicular development. As the hen becomes sexually active, the follicles begin to grow in a hierarchal pattern. [30,34,53,58]

The oviduct enlarges to occupy the dorsal aspect of the left intestinal peritoneal portion of the coelomic cavity. Seasonal growth and differentiation of the reproductive tract is under hormonal control. In a mature hen that is not reproductively active, the ovary and oviduct appear similar to that of a juvenile bird: small with no active follicles. The oviduct develops from a thickening in the peritoneal epithelium between the degenerating pronephric ducts and the first mesonephric tubules. These thickenings invaginate to form a tubular structure. This process occurs bilaterally and symmetrically in both sexes, but regression and disappearance of the ducts occurs in the cock and a relative regression of the right duct occurs in the hen. A right oviduct may be present in some raptors. The opening of the oviductal lumen into the cloaca often appears near the time of production of the first egg.[30,34,53,58]

There is a period of time during fetal development when the testes are ambisexual due to extensive covering with a cortical crust. These cortical remnants normally disappear prior to hatch; however, they may remain and further complicate visual sex identification. The seminiferous tubules differentiate in the medullary portion of the gonad. The ductus deferens develops from the mesonephric duct, while the epididymis arises from the mesonephros. The epididymis and ductus deferens initially develop in both sexes, as does the oviduct. The male tubular structures normally regress in the female. However, they have been reported to persist in some passerines and domestic fowl, but are significantly smaller than the oviduct.[23,35,58]

AVIAN REPRODUCTIVE PHYSIOLOGY

Avian reproduction is guided by seasonal physiologic controls such as favorable climate, low predation risk, social interaction, and food and nest site availability. Internal physiologic processes work in combination with external factors to promote gonadal development. This annual cycle involves integration between environmental, physiologic and behavioral conditions. Biologic clocks control the release of hormones and other chemicals that regulate metabolism, reproduction and behavior. Photoperiod plays a key role in this system using environmental light, which stimulates neural receptors, and clock information from an internal circadian cycle. This allows the bird to measure day length. In most of our Neotropical psittacine species, birds normally start to nest when the rains begin, when food is the most available. In temperate zone species, reproduction is stimulated by photoperiod. The lengthening day in early spring stimulates gonadal development. Warmer temperature, rainfall and behavioral displays fine-tune the physiologic events of breeding and the resulting increased secretion of sex hormones.[23,47,55,58]

There is strong evidence that the pineal gland (*hypophysis cerebri*) is largely responsible for photoperiodic control. However, unlike reptiles, the avian pineal gland does not have primary light receptors, but they do have photoreceptors like cells in the brain and retinae. Birds do not monitor day length visually, but rather by means of special receptors in the hypothalamus. After direct stimulation of the photoreceptors, neurosecretory cells in the hypothalamus induce the release of neurohormones in the median eminence (neural portion) of the epiphysis (pituitary), which is linked to the midbrain. These neurohormones are then carried via the bloodstream to the anterior pituitary gland, inducing the synthesis and subsequent release of luteinizing hormone (LH) and follicle-stimulating hormone (FSH). Luteinizing hormone stimulates gonadal activity and in combination with FSH stimulates ovarian development and testicular spermatogenises.[23,37,54,55,70] *See Ed. Note on page 522.*

Each circadian cycle includes a limited time period of each day during which photoreceptors are particularly sensitive to light. This stimulates a series of physiologic reactions. As daylight length increases each year, so does the opportunity for light to stimulate these receptors during this limited time period. In addition, the length of time these receptors are affected increases with increasing daylight length (see Chapter 19, Endocrine Considerations).[23,37,55,70]

During the late summer after breeding season has ended, the shortened daylight length stimulates the main molt. Gonadal hormones and tissue size decreases dramatically. Although the days are still relatively long, there is a photorefractory period that does not stimulate gonadal hormone release and tissue growth. This photorefractory period is best developed in migratory temperate zone species and is weak to absent in most tropical zone species. This may represent an adaptation for scheduling a major molt and preparation for migration by terminating reproductive activity while days are still long. The shorter days of winter inhibit gonadal growth. This is necessary to restore photosensitivity during the spring, as gonadal tissue will not grow in response to increasing day length unless there has been a prior period of short daylight length.[34,35,58]

Birds have developed several mechanisms to reduce body weight, thereby conserving energy expended during flight. The reproductive tract (ovary, oviduct, testes and ductus deferens) is greatly reduced in size during the non-breeding season, and eggs are laid and incubated externally.[34,35,58]

Physiology of the Female

When reproductively active, the ovary enlarges and follicles form in a hierarchal manner. The primary or F1 follicle is the largest and first in line to be ovulated. There are several smaller follicles on the ovarian surface as well. A stalk that contains smooth muscle with a blood and nerve supply suspends each large follicle.[30,34,49,58] Gonadotropin secretion causes follicles to develop on the ovary in a hierarchal manner. As the breeding season approaches, follicles undergo a period of rapid development and growth. There is deposition of yolk protein and lipid from the liver; gonadotropins and steroid hormones regulate this. The primary oocyte is surrounded by six layers of tissues: the oocyte plasma membrane, perivitelline membrane, granulosa cells, basal lamina, and the theca interna and externa. These tissues have an endocrine role, providing communication between the ovary and oviduct with passage of each ovum. The nerve supply is both adrenergic and cholinergic. Ovulation occurs under the influence of several hormonal factors. Meiotic or reduction division occurs approximately 2 hours preovulation, while the primary oocyte is still within the follicle. This yields a secondary oocyte and first polar body, each with a haploid number of chromosomes. Most birds have a meridional band or stigma on large preovulatory follicles. This is where the oocyte breaks through the follicular wall during ovulation.[30,34,58]

Ovulation occurs at a relatively fixed time period after oviposition under several physiologic, neural and hormonal controls. Determinant layers such as budgerigars and crows lay a fixed number of eggs, while indeterminate layers such as domestic fowl and Japanese quail replace eggs that are lost. Continuous breeders lay throughout the year. A rate of lay is the number of eggs laid in a given time period. A sequence is a number of eggs laid on successive days, separated by pause days. A clutch is a number of eggs laid during a sequence. A longer sequence is associated with a shorter oviposition/ovulation cycle.[30,31,34,52]

During the non-breeding season, ovarian follicles normally undergo atresia. Atresia is a process of regression and resorption of a follicle. Two types of atresia have been described: bursting and invasion. Bursting atresia occurs when the follicular wall ruptures and the yolk is released into the coelomic cavity where it is usually absorbed without any harm to the bird. Invasion atresia involves granulosa and thecal cells invading the ovum and subsequent *in situ* yolk absorption. Early atresia is noted when a vesicular lesion appears on the follicular surface. This vesicular formation progresses until the entire follicle is covered. As the largest F1 follicle is absorbed, the other smaller follicles will progress in a similar manner. Small follicles may be covered with connective tissue, occasionally leaving a scar-like area. Large follicles may undergo cystic degeneration. If ovulation ceases suddenly, as may occur during trauma or stress, developing follicles may become hemorrhagic, resulting in regression of the developing follicles. Aflatoxicosis also may cause follicular atresia. Aging hens may exhibit permanent ovarian involution, which is believed to be a normal physiologic process.[30,31,34,73]

Knowledge of the oviduct and its different anatomic regions is important in discerning pathologic conditions of the oviduct and developing egg. The oviduct is divided into five parts: the infundibulum, magnum, isthmus, uterus or shell gland, and vagina. A mucosal layer of ciliated epithelium with unicellular mucous glands or goblet cells lines the wall of the oviduct. The submucosa has mucosal folds that vary in height, thickness and tubular glands. The muscular layer has an inner layer of circular smooth muscle and an outer layer of longitudinal smooth muscle.[30,31,34,73]

The infundibulum is divided into the proximal funnel portion and a distal tubular portion. The funnel portion is where fertilization occurs. It has a thin wall with low mucosal folds. This portion surrounds and engulfs the ovum during ovulation. At the beginning and end of the ovulatory cycle, the oviduct and ovary may not be synchronized, resulting in ectopic ovulation also referred to as internal laying. This yolk and ovum may be resorbed without incident or may lead to coelomitis. The exact mechanism by which coelomitis occurs after ectopic

ovulation is not well understood.[17,30,31,34,73]

The tubular portion of the infundibulum is thicker, with taller branching folds. Underneath these folds are branched, convoluted tubular glands that produce the chalaza, which are fibrous bands that suspend the yolk within the egg. A thin, dense layer of albumin is added to surround the yolk. In some species, sperm host glands maintain sperm for fertilization for a variable time period and are located within this portion of the infundibulum.[17,30,31,34,73]

Large mucosal folds that result from numerous tubular glands distinguish the magnum histologically. It is the longest portion of the oviduct. The majority of albumin as well as sodium, magnesium and calcium are added to the egg by these glands. The release of albumin may be controlled by mechanical, neural and endocrine factors.[17,30,31,34,73]

The isthmus follows and, in domestic fowl, is clearly delineated from the magnum by a narrow translucent band. This band is not present in psittacines. The isthmus is relatively short and the mucosal folds are less prominent. The tubular glands are unique in that they produce sulfur-containing proteins. These proteins are incorporated into the shell membranes that are produced in the isthmus. A small amount of albumin is added to the developing egg. Calcification is initiated in the isthmus.[17,30,31,34,73]

During passage of the egg through the oviduct, the majority of the time is spent in the uterus, or shell gland. There are two portions: a short, narrow region which the egg traverses rapidly, and a pouch-like region where the egg spends the majority of the time. The mucosal lining is characterized by a large number of leaf-like lamellar folds that press against the surface of the egg. This increases the surface area to improve efficiency of calcification and plumping. "Plumping," a process in which a large amount of water and solutes are added to the egg relatively quickly, occurs in the proximal short, narrow region of the oviduct. It is in the pouch-like region that calcification of the shell is completed.[17,19,30,31,34,73]

The vagina is separated from the uterus by the uterovaginal sphincter. In most species the egg passes rapidly through the vagina to exit into the urodeum. However, in some species the egg may remain for a longer time period to allow for hardening of the shell. There are sperm host glands at the uterovaginal sphincter for sperm storage. Sperm from multiple male birds may be stored and remain viable for long time periods. This may be up to several months in the turkey. This allows for the possibility of insemination of different ova of a single clutch by different males.[17,19,30,31,34,58,73]

Physiology of the Male

The testes are paired, ellipsoid- to bean-shaped organs that lie near the cranial pole of the kidney. The surface is covered with a fibrous tunic, the tunica albuginea. Each testicle is suspended by a short mesentery or mesorchium that protrudes into the intestinal peritoneal cavity and is partially surrounded medially by the abdominal air sac. The testes change in size and color in response to hormonal fluctuations that influence sexual activity. The inactive testicles are often white to yellow due to accumulation of lipid in the interstitial cells. In some species, the inactive testicles are black due to a large number of melanocytes. Active testicles are significantly larger and paler due to the increased volume in the seminiferous tubules. The increased size is a result of increased length and diameter of the seminiferous tubules, and numbers of Leydig or interstitial cells. Generally speaking, the increase in testicular size is a result of increasing serum concentrations of FSH and LH. These physiologic processes occur during the nuptial or culmination phase of the reproductive cycle.[35,38,52,73] *(Ed. Note: These results have not been verified since FSH was purified. Prior to the early 1990s, FSH was contaminated with LH [Etches, R, University of Guelph, 2003]).*

Birds do not have septa that divide the testicles and there are no mediastinal testes. Unlike mammals, the seminiferous tubules anastomose with each other. Each seminiferous tubule is composed of a lining of spermatogonia and sustentacular or Sertoli cells. The spermatogonia divide to form primary, then secondary spermatocytes. As these spermatocytes progress toward the lumen, they undergo a maturation process to become spermatids, which then mature into spermatozoa or sperm. This maturation process proceeds with the head of each sperm embedded in the sustentacular cells. The sustentacular cells extend the width of the epithelium to provide support for the developing spermatozoa. They are phagocytic and also may produce steroid hormones and bind testosterone.[35,38,58]

The spermatozoa detach from the Sertoli cells and travel down the seminiferous tubules when mature. In most species, these tubules converge into a smaller number of short, straight tubules that continue as the rete testes. The rete testes is a meshwork of tubules embedded in connective tissue, located dorsomedially to each testicle, adjacent to the epididymis. Both the rete testes and straight tubules are lined by sustentacular cells.[35,38,58]

The Leydig, or interstitial, cells are located between the seminiferous tubules. They are light-colored and stain eosinophilic with hematoxylin-eosin stain because of the large concentration of smooth endoplasmic reticulum and cholesterol. The smooth endoplasmic reticulum is

involved in the conversion of cholesterol to steroid hormones, with testosterone and androstenedione being the major androgens. These hormones stimulate the secondary sex characteristics, including courtship, coloration, song, and the development and maturation of the tubules, particularly the ductus deferens.[35,38,58]

The epididymis of the bird is concealed due to its dorsomedial location on the testicle and its small size compared to mammals. It is not divided into a head, body and tail, but is composed of several efferent ductules that drain the rete testes and straight tubules. Several efferent ductules drain into the main epididymal duct along its length. The epididymal duct is relatively short and straight, and is lined by non-ciliated pseudostratified columnar epithelium. The epithelium is secretory and provides some of the seminal fluid. Sperm may be stored in the epididymis or in the seminal glomus of more seasonal birds. Some species have an appendix epididymis that extends cranially into the adrenal gland. The efferent ductules of this tissue may secrete androgens following castration.[35,38,58]

The epididymis continues distally as the ductus deferens. The ductus deferens is closely associated with the ureter in the dorsomedial coelom. In passerines, each ductus elongates distally during the culmination phase of the reproductive cycle to form the cloacal promontory, which can project into the cloaca. This protrusion gives the male external cloaca a pillar-like prominence compared to a rounded profile in females; therefore, it can be used for sex determination during the breeding season. Birds with a seminal glomus use it as the main storage site for sperm. The ductus is composed of non-ciliated pseudostratified squamous epithelium and has less secretory function when compared with the epididymis. There are no accessory sex glands in birds.[35,38,58]

Avian semen is derived in part from the sustentacular cells and epithelial cells that line the reproductive tract. Lymph-like fluid is produced from lymphatic folds in the floor of the proctodeum. This fluid appears to be harmful to spermatozoa because of the presence of clotting factors and high concentrations of chlorine and calcium. Avian spermatozoa are either complex or simple. Complex sperm is found in passerines and simple sperm in other species. Similar to mammals, each spermatozoon is composed of an acrosome, head and tail. In simple spermatozoa, the acrosome is attached to the head only at its most rostral point. The head is long and slender, and the tail is long and moves in an undulating manner. In complex sperm, the entire sperm is spiral in appearance and moves by rotating along its longitudinal axis.[35,38,58]

See Chapter 14, Evaluating and Treating the Gastrointestinal System for a discussion of the cloaca.

The vent may be either a circular opening as in psittacines or a transverse slit as in Galliformes. The sphincter muscle surrounds the opening and has an outer circular and an inner transverse striated muscle layer. In addition, there is a transverse muscle originating on the pelvic bone and/or caudal vertebrae that interdigitates with the sphincter muscle surrounding the vent. Upon contraction of this transverse muscle, the vent is pulled ventrocranially, which is important during coitus. This muscular action allows the cloaca in the male bird to be directed over the female's cloaca. The levator muscle originates on the ventral tailhead and inserts ventrally onto the vent and/or the phallus. This muscle pulls the vent caudally after copulation and defecation.[15,36,73]

The phallus may be intromittent as in ratites and Anseriformes, non-intromittent as in Galliformes, or absent as in psittacines and passerines. The intromittent phallus may be found in two forms: one form lacks a ventral cavity, and is found in ostriches, kiwis and tinamous; the other form has a cavity and is found in emus, rheas, cassowaries and Anseriformes. The former type of phallus consists of paired fibrolymphatic bodies with a dorsal sulcus to deliver semen. It lies on the floor of the cloaca and partially everts during micturition and defecation. Tumescence occurs by increased lymphatic flow and stasis into an elastic vascular body within the distal end of the phallus. Those phalluses with a ventral cavity also are located on the cloacal floor, but are enclosed in a sac or cavity. The proximal portion stays within the cavity and does not become engorged, while the distal portion everts when engorged with lymphatic fluid.[15,36,73]

The non-intromittent phallus, as found in domestic fowl, is located on the floor near the lip of the vent. It consists of a median and two lateral phallic bodies. Lateral to the phallic bodies are lymphatic folds located on the ventrolateral floor of the proctodeum. A lymphatic meshwork connects these folds and phallic bodies. Tumescence is a result of lymphatic flow through these structures. The lymphatic folds and lateral phallic bodies accumulate a greater amount of fluid than the median phallic body, resulting in eversion of the phallus and creating a groove for delivery of semen. The phallus contacts the everted oviductal opening where semen is deposited.[15,36,73]

Reproductive Disorders

Avian reproductive disorders are a result of complex combinations of hormonal, physiologic and behavioral actions reacting to photoperiods, food availability and availability of nest sites.[30,70] Environmental influences in captivity may result in the induction of reproductive and hormonal activity in several ways. Artificial lighting may

interfere with the normal photoperiod and annual light cycles, resulting in abnormal cycling.[23] Food is typically available ad libitum and is often high-fat, calorically dense seed, or foods high in simple carbohydrates such as corn and fruit. These foods may actually stimulate reproduction. Most pet birds are not intended for breeding and do not have mates. In some environments, pet birds may select an abnormal mate such as their human cohabitants or cage furniture. There may be a genetic predisposition and lack of normal reproductive hormonal balance[30,34,37] (see Chapter 3, Concepts in Behavior: Section III, Pubescent and Adult Psittacine Behavior).

Reproductively driven birds may display instinctual territorial and mate-related behaviors. These behaviors may include, but are not limited to aggression, biting, masturbation and excessive vocalization. These "undesirable" behaviors may jeopardize their value as pets, diminishing the pet-human relationship and even result in these birds losing their homes.[30,34,37]

Reproduction is often not desired in pet birds. Egg production and hormonal cycling may lead to disease processes of the reproductive system or systemic, endocrine and metabolic disorders. Therefore, avian practitioners have sought medical and surgical methods to limit reproductive drive and hormone production.[8,56,59]

CHRONIC EGG LAYING

Chronic egg laying in pet birds occurs when a hen lays repeated clutches or larger than normal clutch size without regard to the presence of a mate or accurate breeding season. This process often physically exhausts the reproductive tract and is a serious metabolic drain, particularly on calcium stores, all of which may predispose the hen to egg binding, yolk coelomitis and osteoporosis. Commonly affected species include cockatiels, finches and lovebirds, however, any species may be affected. Diagnosis of chronic egg laying is based on history and physical examination. There typically is a history of the hen laying large numbers of eggs with or without a pause period in between clutches. A thorough history of the home environment will often reveal several reproductive stimuli and a "mate relationship" with a member of the household or owner. Physical exam may reveal normal findings, a palpable egg in the coelom, or other secondary disease conditions such as a pathologic fracture secondary to osteoporosis. Serum chemistries may reveal hypercalcemia, hypercholesterolemia and hyperglobulinemia supportive of an ovulating hen. There may be a hypocalcemia present if the hen's calcium stores are depleted, and particularly if she is consuming a low-calcium diet such as seed[9,31,33,65] (see Chapter 5, Calcium Metabolism).

Therapy for chronic egg laying focuses on stopping egg production while altering any predisposing stimuli and correcting any secondary diseases that may be present. Pharmacologic, behavioral, nutritional, environmental and surgical options are used alone or in combination depending on the needs of the individual patient. Pharmacologic options have included medroxyprogesterone acetate[a], levonorgestrel[b], human chorionic gonadotropin[c], norethindrone/mestranol[d], testosterone, leuprolide acetate[e] and tamoxifen[f] **(Table 18.1)**. Medroxyprogesterone acetate, though often effective, may cause serious side effects such as polyuria/polydipsia, obesity, lethargy, hepatic lipidosis, diabetes mellitus, hepatic cirrhosis and death.[9,31,63] Levonorgestrel, another synthetic progestin, has been evaluated only in Japanese quail (*Coturnix coturnix japonica*) and may carry the same side effects as medroxyprogesterone acetate.[72,77] Testosterone therapy interrupts the ovulatory cycle, but has variable results and is contraindicated in patients with liver disease.[73] Norethindrone/mestranol has caused severe hypertension in one Rouen duck (D. Zantop, personal communication, 2000). Human chorionic gonadotropin has demonstrated to be a safer alternative with significantly fewer side effects; however, it has not been consistently effective in managing these disorders and patients may become refractory to treatment.[34,44] Tamoxifen is a non-steroidal anti-inflammatory agent used as an estrogen blocker to treat women with breast cancer. Tamoxifen was administered to budgerigars presumed to be hens, but not actively laying for 38 to 46 weeks.[46] Leukopenia was a significant side effect that resolved after therapy was discontinued. An incidental finding in this study was the change in coloration of the hen's cere from brown to pink or blue. This change implies that tamoxifen does have some estrogen-blocking affects in birds. Leuprolide acetate is a long-acting gonadotropin-releasing hormone (GnRH) analog. A single injection in women and studied laboratory rodents results in an initial stimulation followed by a prolonged suppression of pituitary gonadotropins. In rats, this reduction in serum gonadotropin levels is achieved by reducing the number of pituitary GnRH receptors. Repeated monthly injections result in receptor down regulation of GnRH pituitary receptors, which causes a decreased secretion of gonadal steroid hormones. Therefore, tissues and functions that depend on these hormones for maintenance become quiescent, and diseases resulting from reproductive hormone production improve or resolve.[16,33,50,51,78]

Environmental stimuli should be altered, including decreasing the photoperiod to 8-10 hours of daylight per day. Nest sites, toys and other items toward which the bird has a sexual affinity should be removed from

Table 18.1 | Summary of Medical Therapy for Reproductive Disorders

Therapy	Dosages	Comments
Leuprolide Acetate[e]	700-800*[1] µg/kg IM for birds <300 g	Administered every 14 days; 3 doses are usually adequate
	500 µg/kg IM for birds >300 g	Stable in standard freezer 9 months
Human chorionic gonadotropin*[2,c]	250-500 IU/kg IM on days 1, 3 and 7, 500-1000 IU/kg IM	Stable in refrigerator 60 days. If a second egg is laid, repeat dose on day 3; if a third egg is laid, repeat dose on day 7
Levonorgestrel[h]	—	Not recommended
Medroxyprogesterone[a]	—	Not recommended
Tamoxifen[f]	2 mg/kg PO QD	Leukopenia
Arginine vasotocin	0.01-1.0 mg/kg IM	Stable in standard freezer
PGE (Prepidil Gel)[g]	0.025 ml/100 g 0.2 mg/kg applied topically	May freeze into aliquots and thaw prior to administration; relaxes uterovaginal sphincter while inducing uterine contractions
PGF2alpha (Lutalyse)	0.02-1.0 mg/kg IM	Does not relax utero-vaginal sphincter when inducing uterine contractions
	Topically	Applied to prolapsed uterine tissue to stop hemorrhage and shrink tissues

*[1] *Ed. Note: 1000 mg/kg has been used in recalcitrant cases.*
*[2] *Ed. Note: Should be administered with dexamethasone to avoid what appears to be immune-based response to HCG. Can be given with leuprolide acetate.*

the enclosure. Access to nesting environment or materials such as a box, other dark cavities, or shredded papers should be prohibited. In the event that a pet bird is showing nesting behavior and laying eggs in a designated site, removal of eggs from the "nest" should be avoided for the normal incubation period for each species to discourage the hen from laying further eggs to replace those removed. Any perceived or actual mate should be removed from the cage or room. In some species such as the cockatiel, visual and auditory separation from an actual or perceived mate may be necessary. A "one-person bird," which has a single household person who exclusively or primarily handles and cares for it, should potentially be viewed as having a "mate relationship" with that person. This may serve as a trigger for reproductively driven behaviors. Stimulatory petting such as rubbing the pelvis, dorsum and cloacal regions and kissing the beak should be avoided. Feeding calorically dense diets should be avoided, as mentioned previously. Interactive behaviors that simulate a "flock relationship" should be encouraged such as the bird being handled by several people in the household. The cage location and furniture (toys, perches, food dishes) should be changed and rotated periodically to discourage territorial behavior and limit reproductive drive in response to a perceived "nest site." Any nutritional problems should be corrected to improve the hen's dietary plane to reduce the severity of metabolic drain.[31,53,63] Dietary alteration and reduction of caloric intake does appear to anecdotally reduce or stop egg production. This nutritional effect is often achieved by converting the pet bird from a seed-based diet to a formulated one. The exact reason for this effect is unknown, but it is common practice in poultry to reduce feed intake to stop egg production and induce molting.[56] Surgical salpingohysterectomy may be elected or necessary if medical therapy is not successful and if there is no intent to breed the particular hen. Laparoscopic salpingohysterectomy may be performed as a preemptive measure on juvenile birds to prevent egg production and its associated diseases. Any secondary disease conditions also should be appropriately treated.[6,59,71]

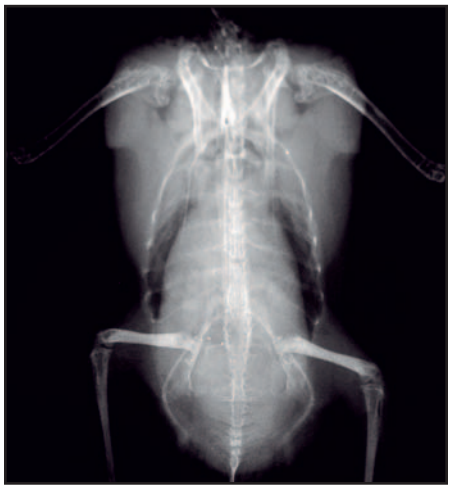
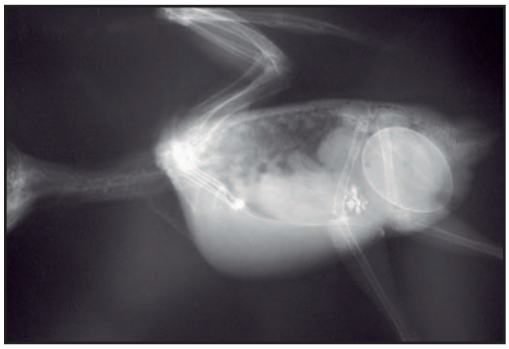
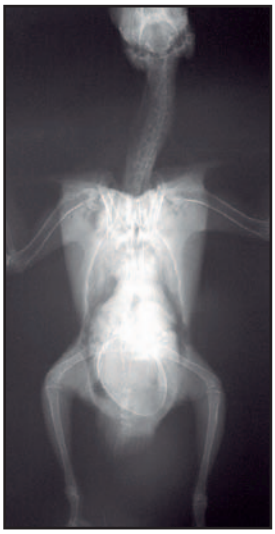


Fig 18.1 | Ventrodorsal radiograph of a budgerigar hen with polyostotic hyperostosis. Note the increased density of both femurs.

POLYOSTOTIC HYPEROSTOSIS

Polyostotic hyperostosis differs from physiologic osteomyelosclerosis in that the latter condition occurs in nonlaying hens and cocks as a result of pathologic conditions. Typically, radiographs reveal significantly increased bone density of the long bones and occasionally the vertebrae (Fig 18.1). The pathogenesis of polyostotic hyperostosis is still unclear. Many affected birds exhibit concurrent reproductive-associated activity or may suffer from reproductive-associated disease conditions. In addition, increased medullary bone density does appear to resolve radiographically with resolution of reproductive drive or disease. Hepatic disease may play a role in this condition due to the liver's role in the inactivation of estrogen. However, a recent study does not support this theory, stating that budgerigars affected by polyostotic hyperostosis had no evidence of estrogen secretion or other endocrine disease.[5,29,58,74]





Figs 18.2a,b | Left lateral and ventrodorsal radiographs of a 3-year-old egg-bound cockatiel. The hen had been consuming a seed diet and was unable to perch for several days prior to presentation. The bird was thin (68 g), dehydrated and hypocalcemic (6.2 mg/dl), and responded to supportive care and arginine vasotocin. Egg production was controlled after egg expulsion with leuprolide acetate (750 μg/kg IM every 14 days for 3 injections) and alteration of environmental stimuli. Diet was modified to include a 90% formulated diet.

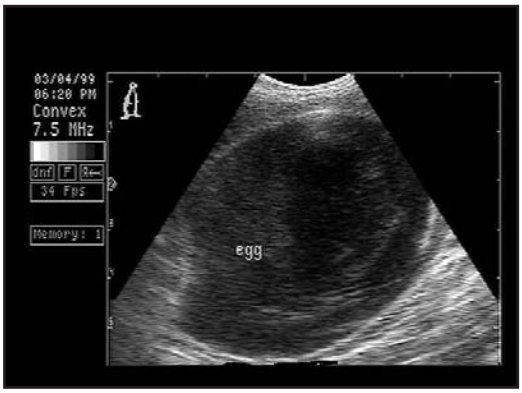


Fig 18.3 | Coelomic ultrasound of the same cockatiel. Note the well-calcified egg in normal presentation for oviposition (7.5-MHz probe).

EGG BINDING AND DYSTOCIA

Egg binding is defined as the failure of an egg to pass through the oviduct within a normal period of time. Most companion birds lay eggs at intervals of greater than 24 hours, and individuals may vary further. This variability may make it difficult to determine if there is a problem in the early stages of this disease. Dystocia involves the mechanical impedance to oviposition. The most common anatomic areas for this to occur are the distal uterus, vagina and vaginal-cloacal junction.[31,63,73]

Causes of egg binding may include chronic egg laying, oviductal muscle dysfunction secondary to excessive egg laying, calcium metabolic disease, vitamin E and selenium deficiencies, malnutrition, obesity, inadequate exercise and muscle strength, malformed eggs, mechanical tears or damage to the oviduct, oviductal infections, systemic disease, genetic predisposition and environmental stressors. Dystocia also may result when a developing egg in the distal oviduct obstructs the cloaca or causes oviductal tissue to prolapse. Oviductal torsion and oviductal or abdominal masses compressing the oviduct also may obstruct passage of an egg and result in dystocia. Breeding birds out of their natural season, egg-producing virginal hens and hens with a persistent right oviduct may be predisposed to egg binding or dystocia.[31,63,73]

Diagnosis

Cockatiels, lovebirds, canaries and finches are most commonly reported to be affected and seem to present with more severe clinical signs, possibly due to their small size. Clinical signs associated with egg binding and dystocia vary according to severity, size of the bird affected and degree of secondary complications. Common signs include acute depression, abdominal straining, persistent tail wagging, a wide stance, failure to perch, abdominal distension, dyspnea, and/or sudden death (Fig 18.3). An egg lodged in the pelvic canal may compress the pelvic blood vessels, kidneys and ischiatic nerves, causing circulatory disorders, lameness, paresis or paralysis. Pressure necrosis of the oviductal wall may occur. Dystocia may cause metabolic disturbances by interfering with normal defecation and micturition, and cause ileus and renal disease, respectively. The severity of the patient's condition can be estimated by the degree of depression and the length of time clinical signs have been present.[31,63,73]

Diagnosis of egg binding or dystocia in a severely compromised patient may be made based on history and physical examination alone, and the patient may not be stable enough to survive other diagnostic procedures. Rapid diagnosis and therapy are crucial for a successful outcome. Physical examination may reveal depression, lethargy, a thin or normal body condition, and dehydration. There may be dyspnea or an increased respiratory rate due to compression of the caudal thoracic and abdominal air sacs. The hen may not be able to perch, and may demonstrate pelvic limb paresis, paralysis or cyanosis. An egg typically, but not always, is palpable in the caudal abdomen. Cranially located, soft-shelled and non-shelled eggs may not be detected on abdominal palpation. Palpable eggs may be located within the oviduct or ectopically within the coelom, and careful abdominal palpation, cloacal examination, radiographs, coelomic ultrasound, laparoscopy and/or laparotomy may be required to determine the egg's position.[31,63,73]

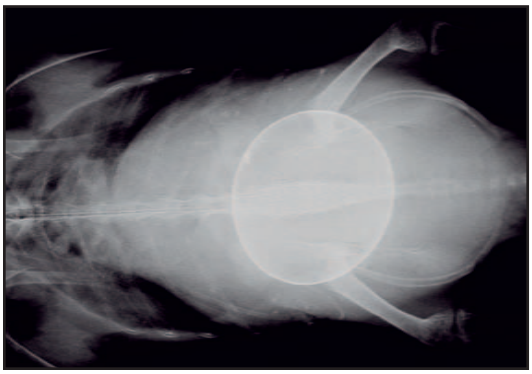


Fig 18.4a | Ventrodorsal radiograph of an eclectus hen with a history of depression and inappetence. Physical examination revealed a distended abdomen and palpable egg in the coelom. Radiographs revealed a calcified egg in the mid coelom.

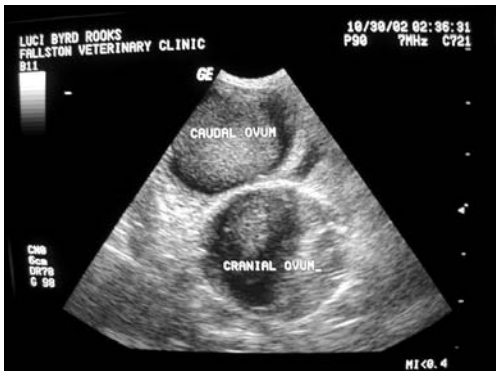


Fig 18.4b | Ultrasound of the same bird revealed a non shelled egg cranial to the shelled egg noted on the radiograph in Fig 18.4a.

Radiography and ultrasonography aid in evaluation of the position and characterization of the egg(s). There may be multiple eggs identified in the coelom due to an obstruction distally or secondary to motility disorders. Radiographs may reveal an egg in the coelom if the egg has a visible shell. The egg is typically located in the distal oviduct, in the region of the uterus (see Figs 18.2a,b, Fig 18.3). Osteomyelosclerosis of the femurs, tibiotarsi, radii, ulnas and/or spine may be visible, and a soft tissue density suggestive of an enlarged ovary in the region of the ovary may be noted, and is supportive of a reproductively active hen. A coelomic ultrasound will often reveal an egg and may identify soft-shelled or non-shelled egg(s) that may not be identifiable on radiographs (Figs 18.4a,b). Again, there may be several eggs visible within the coelom. Follicles may be visible on the ovary, indicating the potential for further ovulation and egg formation. A hematologic analysis and serum chemistries are useful to identify any predisposing and secondary diseases. A complete blood count may reveal a leukocytosis with a relative heterophilia if there is a concurrent inflammatory or infectious process. Serum chemistries may demonstrate elevated aminotransferase and creatinine phosphokinase due to skeletal muscle enzyme leakage from tissue damage, or as a result of reduced food consumption and a hypermetabolic state. Hypercholesterolemia, hyperglobulinemia are supportive of an ovulating hen. Elevated total and ionized calcium may be indicative of a cycling hen. Hypocalcemia may be observed if a hen has been consuming a calcium-poor diet or has been laying excessive numbers of eggs, resulting in depletion of calcium stores[31,40,60,73] (see Chapter 5, Calcium Metabolism).

Treatment

Therapy varies with history, severity of clinical signs and diagnostic test results. Supportive care should include elevated environmental temperature, parenteral calcium (only if indicated), fluid therapy and nutritional support. Broad-spectrum antibiotics are indicated if it is suspected that the integrity of the oviduct has been compromised. Analgesics are indicated if the patient appears to be in pain or if clinical knowledge of the patient's condition suggests that pain may be a part of the pathologic state. Supportive care alone is often enough to allow oviposition, although the hen should be monitored closely for deterioration of her condition, which may require further intervention.[40,63,73]

Prostaglandin and hormonal therapy may be used to induce oviductal contractions. This may result in expulsion of the egg if the contractility of the oviduct is sufficient to expel the egg, the uterus is intact, the egg is within the oviduct, and there is no obstruction such as a neoplastic mass, granuloma or egg adhered to the oviduct. Studies performed in poultry have found that prostaglandin E2 (PGE2) and prostaglandin F2alpha (PGF2alpha) bind at specific receptor sites in the uterus and vagina. The uterine myometrium appears to preferentially bind PGF2alpha because it contains low-affinity and some high-affinity binding sites for PGE2 and specific high-affinity for PGF2alpha. Prostaglandin F2alpha binds at the shell receptor sites to cause a time- and dose-dependent mobilization of cellular calcium in the presence of extracellular calcium, thereby causing uterine muscle contraction. It has been demonstrated in vitro that PGE2 is itself ineffective in calcium ion mobilization, but will enhance PGF2alpha-induced calcium mobilization. This suggests that PGE2 may potentiate the ability of PGF2alpha to cause uterine contraction. In the vagina, high-affinity binding sites for PGE2 predominate. It is possible that a high PGE2 concentration in the vagina is needed to saturate high-affinity binding sites and block PGF2alpha-binding sites. This allows for relaxation of the uterovaginal sphincter and vagina. Due to the fact that fewer PGE2 high-affinity binding sites are present in the uterus, they are not likely to interfere

with PGF2alpha binding and may potentiate the action of PGF2alpha.[27,28,63,67,68]

When an egg is present in the uterus, the administration of PGF2alpha and PGE2 will cause the concentration of arginine vasotocin (AVT) to increase in systemic circulation, however, PGF2alpha is the more potent stimulator of AVT release. It is suggested that prostaglandins stimulate uterine contractions, which in turn stimulate the release of AVT from the neurohypophysis, and that AVT probably acts synergistically with PGF2alpha to increase uterine contractions. Oxytocin and AVT appear to specifically affect the uterus, inducing contractions. It is important to note that PGF2alpha, oxytocin and AVT do not cause relaxation of the uterovaginal sphincter while inducing oviductal contractions. This may result in peristalsis of the egg, severe pain and/or rupture of the uterus. Prior to their use it should be determined if the utero- vaginal sphincter is open. In addition, prostaglandin and hormonal therapy does require adequate calcium to be effective. As many of these patients are severely hypocalcemic due to either malnutrition or chronic egg laying, supplemental calcium may be required prior to administration of these medications.[27,28,63,67,68]

PGE2 gel[g] may be applied to the uterovaginal sphincter at a dose of 0.1 ml per 100-g bird. PGE2 causes relaxation of the uterovaginal sphincter while causing oviductal contractions and may be applied topically, thereby decreasing the incidence of systemic side effects. These contractions may expel the egg within 15 minutes. Contact with PGE2 gel may cause altered menses and induce spontaneous abortion in women. Therefore, it is important to flush any excess from the cloaca after egg expulsion, and to caution staff and clients regarding contact with any stool and/or urine produced. Prostaglandin F2alpha, oxytocin and AVT also will cause powerful uterine contractions. Prostaglandin F2 alpha is administered parenterally, rather than locally, and is more likely to cause systemic reactions such as hypertension, bronchoconstriction and general smooth-muscle stimulation.[27,28,63,67,68]

If supportive care and medical therapy fail to induce oviposition, then manual manipulation may be necessary. Massaging the abdomen and vaginal opening may relax the vaginal sphincter and allow passage of the egg. It may be helpful to infuse lubricants into the cloaca to moisten the tissues. Careful digital pressure applied to the cranial portion of the egg and directed caudally may encourage movement through the distal oviduct and cloaca. Using a cloacal speculum, the vaginal opening of the oviduct can be dilated by inserting a blunt probe (eg, lubricated cotton-tipped swab) that is gently advanced in a twirling motion. Potential complications may include retroperistalsis of the egg out of the oviduct into an ectopic position within the coelom, rupture of the egg, oviductal trauma, oviductal laceration, oviductal avulsion, hemorrhage, and displacement of the egg or fragments into an ectopic position. If fertilization may have occurred and the egg may be fertile, it may be incubated if successfully removed intact.[31,63,73]

Fig 18.5 | Transabdominal ovocentesis and aspiration of egg contents in a 7-year-old egg-bound cockatiel. Patient was anesthetized with isoflurane by mask induction. A 22-gauge needle and 3-ml syringe were used to aspirate approximately 1 ml of egg contents. The shell was passed approximately 4 hours postovocentesis. Complete passage of the shell was confirmed by radiography.

Ovocentesis may be performed to facilitate passage of an egg. Aspiration may be performed through the cloacal opening if the egg is distally located, or transabdominally if the egg is more cranially positioned. The egg is manipulated so that it is visible through the cloaca and a needle is inserted into the egg through the cloaca. The contents of the egg are aspirated into a syringe, while the shell is manually collapsed and the pieces expelled through the cloaca. (see Chapter 7, Emergency and Critical Care and Chapter 24, Diagnostic Value of Endoscopy and Biopsy). If the egg cannot be visualized through the cloaca due to a more cranial location, transabdominal ovocentesis may be performed (Fig 18.5). The egg is manually placed directly against the abdominal wall so that other abdominal organs are displaced and not damaged during aspiration. A needle is inserted through the skin and abdominal wall into the egg. The egg contents are aspirated into the syringe while the egg is manually collapsed. The eggshell remnants are expelled through the cloaca, either naturally or with clinical assistance. It is important to confirm radiographically that these eggshell pieces have been completely expelled. If these pieces are not expelled within a reasonable amount of time, approximately 36 hours, it may be necessary to irrigate the oviduct through the cloaca or laparotomy approach. A salpingohysterectomy may be performed if egg remnants are retained and the hen is not required for breeding.

Some clinicians advocate flushing the uterus postoviposition with saline, chlorhexidine or iodine to remove any shell fragments and decrease the incidence of metritis. Oviductal rupture, resulting in an ectopic egg, shell fragments and yolk coelomitis, are possible complications of ovocentesis.[8,31,63,73] See Chapter 24, Diagnostic Value of Endoscopy and Biopsy.

Prostaglandin treatment, manual delivery and ovocentesis are contraindicated in cases of ectopic eggs, oviductal rupture, oviductal torsion and mechanical obstruction. Complications that may require surgical intervention include oviductal rupture with or without an ectopic egg, oviductal necrosis, oviductal torsion, abdominal hernia or if the condition is interfering with defecation and/or micturition. Medical therapy to reduce reproductive hormone levels and reproductive activity should be utilized to temporarily prevent further egg production. Surgical removal of an egg is required in cases of ectopic eggs and dystocia, including oviductal rupture, oviductal torsion, or mechanical obstruction, or if medical treatment is not successful. If surgical intervention is necessary, bacterial culture and sensitivity and histopathology should be performed on oviductal tissue samples. Salpingohysterectomy may be considered to prevent further reproductive complications, and any predisposing and secondary diseases should be corrected.[6,24,40,63,73]

OVIDUCTAL PROLAPSE

Oviductal prolapse may occur secondary to any condition that causes chronic, excessive abdominal straining such as normal physiologic hyperplasia, egg laying or dystocia. An intracoelomic space-occupying mass also may induce prolapse of the oviduct. Predisposing factors may include abnormal or soft-shelled eggs, malnutrition, obesity, salpingitis and cloacitis. Typically, the uterus protrudes through the cloaca, often with a partial prolapse of the vagina and cloaca (Fig 18.6).[31,63,73]

Rapid management is necessary to prevent necrosis of these tissues. Any egg that may be present should be removed, all exposed tissues cleaned, irrigated and kept well moistened to prevent desiccation. Topical anti-inflammatories such as dimethyl sulfoxide may be applied, any lacerations should be repaired and all tissues should be gently replaced. Temporary stay sutures may be indicated to aid in preventing recurrence, as prolapse of the oviduct may recur and repeated replacement is often required. Bacterial culture and sensitivity of the prolapsed tissue should be performed to aid in appropriate antibiotic therapy. Complete blood count, serum chemistries, radiographs, ultrasonography and laparoscopy should be included in a complete diagnostic evaluation to identify any predisposing and secondary disease conditions. Treatment should be directed at

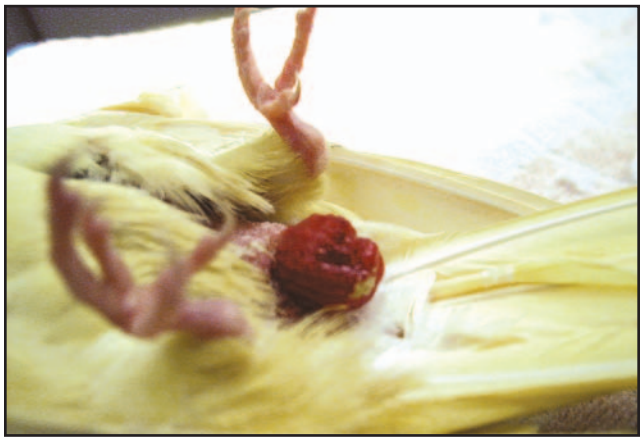


Fig 18.6 | Uterine prolapse, including partial prolapse of the vagina and cloaca, in a 7-year-old cockatiel with a history of chronic egg laying. Samples of affected tissues were taken for bacterial culture and sensitivity. Prolapsed tissues were irrigated with sterile saline, dimethyl sulfoxide was applied and tissues were reduced manually. Therapy included supportive care, enrofloxacin (15 mg/kg PO q 12 h x 14 days), carprofen (2 mg/kg PO q 12 h x 3 days). Further egg production was controlled with leuprolide acetate (750 μg/kg IM every 14 days for three injections) and alteration of environmental stimuli.

clearing any bacterial infection and preventing further prolapse. It also is important to decrease reproductive hormone levels to prevent further egg formation, decrease the size of oviductal tissue and allow the reproductive tract to rest. Broad-spectrum antibiotics and anti-fungals should be initiated while bacterial and fungal cultures are pending.[8,31,63,69,73]

Salpingohysterectomy may be considered to prevent recurrence. Predisposing factors should be corrected to prevent recurrence and secondary diseases addressed (see Chapter 35, Surgical Resolution of Soft Tissue Disorders).

UTERINE TORSION

Uterine torsion is usually diagnosed in the later stages of the disease. Birds typically present with abdominal distension secondary to coelomitis. Early clinical signs may include depression and anorexia following recent oviposition. A complete blood count often demonstrates a leukocytosis with a relative heterophilia, and serum chemistries show an elevated aspartate transferase and creatinine kinase. Diagnosis is usually made at exploratory laparotomy or laparoscopy. Many times, severe vascular compromise and necrosis of the oviduct is found, which requires salpingohysterectomy.[1,6,24,71,73]

OVIDUCTAL IMPACTION

Oviductal impaction may occur following salpingitis, metritis or dystocia. Impactions may occur due to excess mucin or albumin, secondary to cystic hyperplasia of the

oviduct. Inspissated egg material also may cause obstruction. Clinical signs may be vague and can include cessation of egg production, broody behavior without egg production, weight loss, anorexia, depression, constipation, diarrhea, abdominal distension, and reluctance to walk or fly. A tentative diagnosis is made through history, physical examination and supporting diagnostic tests. A leukocytosis with or without a relative heterophilia may be noted. Serum chemistries may be supportive of an ovulating hen. Radiographs and coelomic ultrasound may demonstrate a soft tissue density in the region of the oviduct, displacement of other coelomic viscera, loss of coelomic visceral detail or coelomic fluid if there is a concurrent coelomitis. Definitive diagnosis of oviductal impaction is often made at laparoscopy or laparotomy, revealing an abnormal-appearing, enlarged oviduct with or without coelomitis and adhesions. In many cases, it is necessary to clean and repair or surgically remove the oviduct. Surgery may be complicated if coelomic fluid and/or adhesions are present.[6,24,31,40,63,69,73]

Bacterial culture and sensitivity should be performed on specimens from the affected oviduct, and histopathologic examination should be performed on biopsy samples. Treatment includes parenteral fluids, nutritional support, warmth and broad-spectrum antibiotics, pending culture and sensitivity results. Medical or surgical therapy to reduce reproductive hormone production and reproductive activity should be initiated, and environmental stimuli altered as discussed with chronic egg laying, to prevent recurrence.

SALPINGITIS AND METRITIS

Salpingitis is defined as inflammation of the oviduct either by an infectious or non-infectious etiology, the latter being far less commonly reported. It is generally seen associated with airsacculitis, liver disease, pneumonia, systemic infections, and ascending infections of the oviduct from the uterus or cloaca. Excessive abdominal fat has been associated with salpingitis in domestic fowl. Some of the most commonly identified pathogens are *Escherichia coli*, *Salmonella*, *Mycoplasma*, *Pasteurella* and *Streptococcus* spp. Newcastle disease also has been associated with salpingitis in several species. In ground-nesting species such as Anseriformes and emus, non-lactose-fermenting, gram-negative bacteria such as *Pseudomonas aeruginosa*, *Proteus mirabilis* and *Proteus vulgaris* are commonly identified. Noninfectious causes of salpingitis include trauma and inflammation secondary to oviposition disorders, malnutrition and foreign bodies. Salpingitis is most common in adult hens but may occasionally occur in young birds as well.[8,63,73,75]

Metritis is a localized infection or inflammatory process within the uterine portion of the oviduct. Metritis may occur secondary to dystocia, egg binding, oviductal impaction, systemic bacterial infection and ascending infection. Salpingitis and metritis may cause abnormal shell formation and impaired uterine contractions, and may cause infections in chicks and embryos including embryonic death. Fatalities are often associated with ovulation, egg binding or dystocia, oviductal rupture, coelomitis and septicemia.[8,63,73,75]

Clinical signs of salpingitis and metritis may be vague and difficult to detect initially. In pet birds, these include decreased egg production, infertility, abnormally shaped eggs and mild depression. More advanced cases may exhibit anorexia, lethargy, abdominal distension, oviductal rupture, coelomitis and septicemia. There may be a leukocytosis with a relative heterophilia. Serum chemistries may or may not be supportive of an ovulating hen. Radiographs and ultrasonography may reveal an enlarged oviduct. Laparoscopy may or may not identify inflammation of the serosal surface of the oviduct. The oviduct may be thin-walled, decreased in length or have vascular congestion. The lumen may contain fluid or fibrinous exudates. Definitive diagnosis of salpingitis and metritis is based on cytology, bacterial and fungal culture and sensitivity, and biopsy with histopathologic analysis of a specimen from the oviduct.[8,24,40,63,73]

Therapy of salpingitis and metritis is focused on correcting any underlying or contributing causes. Antibiotic therapy for identified or suspected bacterial organisms should be initiated pending results of bacterial culture and sensitivity. Pharmacologic treatment and husbandry-related intervention, as discussed with chronic egg laying, should be initiated to prevent further hormonal stimulation with subsequent egg production, which may perpetuate or contribute to this disease. There should be close follow-up including bacterial culture and sensitivity and fertility monitoring after treatment, as many cases are difficult to resolve completely. It is important to note that bacteria isolates from the cloaca are not equivalent to oviductal infectants, and cloacal bacterial cultures should be interpreted carefully. Severe refractory cases may require laparotomy to remove necrotic tissue and flushing of the oviduct with fluids and antibiotics. Patients suffering from severe salpingitis may require salpingohysterectomy, or this may be elected in milder cases to resolve disease and prevent recurrence if the hen is not intended for breeding.[6,63,71,73]

CYSTIC HYPERPLASIA OF THE OVIDUCT

Cystic hyperplasia of the oviduct may occur from improper formation of the left oviduct or secondary to an endocrine abnormality. Additionally, the vestigial right

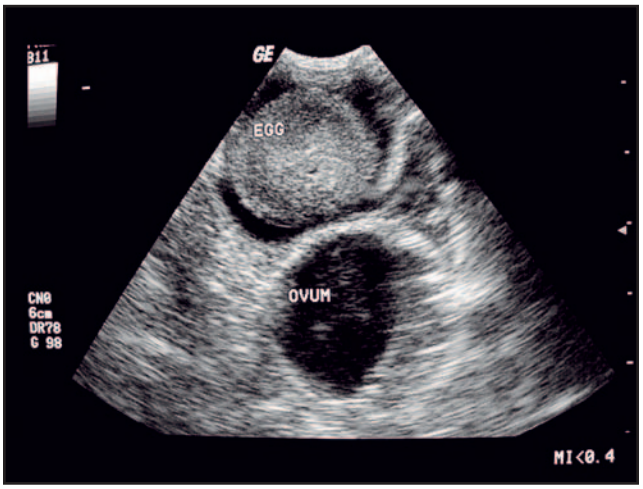


Fig 18.7 | Coelomic ultrasound. An ectopic egg and uterine laceration were noted on laparotomy, and a salpingohysterectomy performed. Note the calcified egg in the caudal coelom and the ovum located more cranially (7.5-MHz probe). This ovum could not be detected on the radiographs. The hen recovered fully.

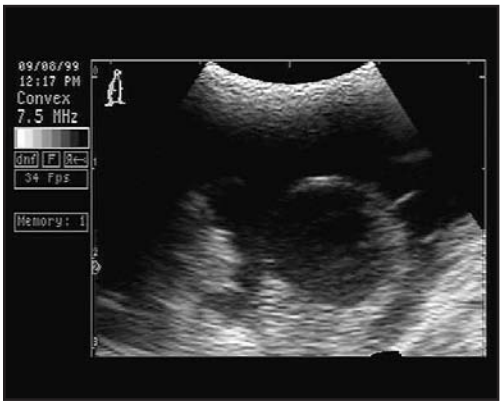


Fig 18.8 | Coelomic ultrasound of an 8-year-old African grey parrot. The patient had a history of depression and inappetence. Physical examination revealed abdominal distension. Note the coelomic fluid and large ovarian cyst (7.5-MHz probe).

oviduct may become cystic and the associated ovary often has cystic changes as well. Cystic hyperplasia often contributes to salpingitis and egg binding. Clinical signs may include depression, anorexia, abdominal distension, ascites and dyspnea. A tentative diagnosis is made through history, physical examination and supporting laboratory tests, similarly to that of salpingitis and metritis. Radiographs may demonstrate an enlarged soft tissue density in the region of the oviduct. Ultrasonography may reveal an enlarged oviduct that may be fluid-filled or have obvious cysts present, with or without concurrent ovarian follicles or cysts. Laparoscopy may show a dilated oviduct filled with a white or brown mucoid fluid. Definitive diagnosis requires laparotomy with biopsy, cytology, histopathology, and bacterial culture and sensitivity.[8,24,31,40,63,73]

Therapy to stop ovulation should be initiated due to increased risk of oviductal rupture during ovulation, oviposition and possible hormonal contribution to the cystic state of the oviduct. If bacterial infection is suspected or documented by cytology and bacterial culture and sensitivity, appropriate antibiotic treatment is indicated. Salpingohysterectomy may be required to resolve the current problem or prevent future recurrences and should be considered if the hen is not intended for breeding, as complete resolution with medical therapy alone may be difficult.[6,8,31,63,73]

OVIDUCTAL RUPTURE

Oviductal rupture may occur secondary to dystocia or oviductal disease. Prostaglandins, oxytocin, arginine vasotocin and ovocentesis may cause traumatic rupture of the oviduct. Clinical signs may include depression, anorexia, and abdominal distension secondary to coelomitis or the deposition of egg or oviductal contents. Radiographs and ultrasonography may reveal osteomyelosclerosis, polyostotic hyperostosis, a soft tissue density in the region of the ovary, ovarian follicles, an enlarged or cystic oviduct, a shelled or non-shelled egg, and coelomic fluid if a concurrent coelomitis is present (Figs 18.7, 18.8). Diagnosis is confirmed at laparoscopy or laparotomy. The laceration may be repaired, depending on the integrity of the tissue, or salpingohysterectomy may be performed as a therapeutic and preventive technique.[6,24,31,40,63,73]

ECTOPIC OVULATION

Ectopic ovulation may result from failure of the infundibulum to retrieve an ovulated ovum, reverse peristalsis of the oviduct or oviductal rupture. Ectopic ovulation does not necessarily result in coelomitis. Internal laying is actually a common occurrence in many avian species, and the ova are usually resorbed without any problems whatsoever. Reverse peristalsis may be triggered by obstruction of the oviduct, cystic hyperplasia, neoplasia, malnutrition, trauma and stress. The ectopic ova may be resorbed without incident or may induce a severe coelomitis.[30,31,56,63,73]

Clinical signs of ectopic ovulation may include transient or persistent depression, inappetence and abdominal distension, especially if there is an associated coelomitis. There may be a leukocytosis with a mature heterophilia. Serum chemistries may demonstrate an ovulating hen. Radiographs may reveal polyostotic hyperostosis and one or multiple eggs in the abdomen. It is important to

Greg J Harrison

Fig 18.9 | A cockatiel hen with a history of egg laying 3 months previous is showing depression and dyspnea. Note the severely distended abdomen. Diagnosis was cystic ovarian disease.

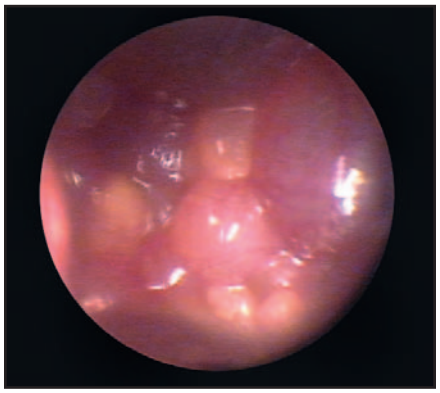


Fig 18.10 | Laparoscopic examination of the African grey hen in Fig 18.8. Note the two ovarian cysts and several smaller follicles.

note that an ectopic ova detected by ultrasound in the absence of clinical signs may resolve on its own with no treatment, and any medical or surgical intervention may be contraindicated. It may be difficult or impossible to determine if an egg is located within the oviduct or is ectopic without laparoscopy or laparotomy, depending on its location within the coelom. If the egg is not laid within a reasonable time period and/or the patient's condition is declining, a laparotomy is indicated as an exploratory procedure. Ectopic eggs are removed by laparotomy, and any oviductal tear should be surgically repaired or a salpingohysterectomy performed. Cytology, culture and sensitivity, and histopathology should be performed in cases of oviductal rupture, cystic hyperplasia and neoplasia.[8,31,40,63,73]

CYSTIC OVARIAN DISEASE

Ovarian cysts have been known to occur in several pet bird species including cockatiels, canaries, budgerigars, macaws, pheasants and domestic ducks. Cyst development may be caused by endocrine disorders, anatomic abnormalities on the ovary itself and pathologic conditions of the ovary. A thorough history may reveal current or previous egg production, with an abrupt halt. Owners may even report chronic reproductive behavior without egg production or impaired reproductive performance in breeding hens.[8,14,73]

Advanced cystic ovarian disease may cause depression, inappetence and weight loss. Abdominal distension, often due to secondary coelomitis, and related clinical signs may be noted as well (Fig 18.9). A leukocytosis with a relative heterophilia, as well as a peripheral hypercalcemia, hyperglobulinemia and hypercholesterolemia are common findings. Radiographs may demonstrate polyostotic hyperostosis, a soft tissue density in the area of the ovary and/or oviduct, coelomic fluid and displacement of coelomic viscera. Ultrasound may reveal a fluid-filled cyst(s) in the area of the ovary or simply coelomic fluid of an undetermined source. An ovarian cyst may be quite large and may actually fold onto itself as it grows. There may be normal ovarian follicles present as well (Fig 18.10). Abdominocentesis with cytology and bacterial culture and sensitivity should be performed in those patients suffering from associated coelomitis. It is beneficial and often necessary to perform a laparoscopic exam or celiotomy with ovarian biopsy, especially for those patients with cysts that do not resolve with medical therapy, as it is not uncommon for hens to develop ovarian cysts secondary to neoplasia and oophoritis. Laparoscopy will reveal an ovarian cyst(s), and the contents may be aspirated during this procedure. Cytology of fluid aspirated from these cysts is clear to straw-colored and of low cellularity. However, it is important to practice extreme caution during a laparoscopic exam and aspiration, as fluid from the cyst or coelom may gain access to the respiratory system through the entry hole in the abdominal air sac. Ovarian biopsy with cytologic analysis, histopathologic exam, and bacterial culture and sensitivity should be performed to identify any primary or secondary disease processes.[14,40,63,73]

Treatment goals include resolution of the cyst(s) and associated disease conditions such as coelomitis, oophoritis, ovarian granuloma and neoplasia. Abdominocentesis often improves related dyspnea if there is coelomic fluid compressing the air sacs. Pharmacologic, behavioral, environmental and dietary intervention to reduce ovarian activity are indicated as production of reproductive hormones may perpetuate ovarian cysts.[14] Aspiration of cysts, salpingohysterectomy and partial ovariectomy may be beneficial for complete resolution.[6,8,60,63,73] Cryosurgical destruction may be beneficial. Long-term resolution may be difficult and patients suffering from cystic ovarian disease should be regularly monitored for recurrence.[8,14,63,73,78]

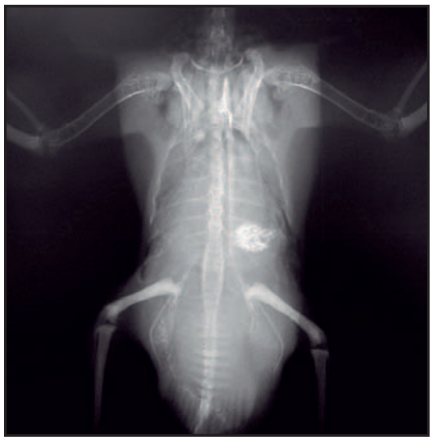


Fig 18.11 | Ventrodorsal radiograph of a cockatiel hen with reproductive-associated coelomitis. Note the severely enlarged fluid/soft tissue density, cranial displacement of the grit-filled ventriculus, obliteration/compression of caudal thoracic and abdominal air sacs, and increased density of both femurs.

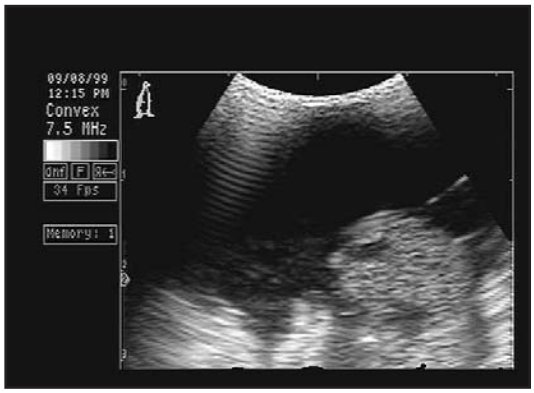


Fig 18.12 | Coelomic ultrasound of the cockatiel hen from Fig 18.11. Note the coelomic fluid and soft tissue density. Laparotomy revealed an oviductal granuloma, and the hen recovered fully in response to salpingohysterectomy and antibiotic therapy.

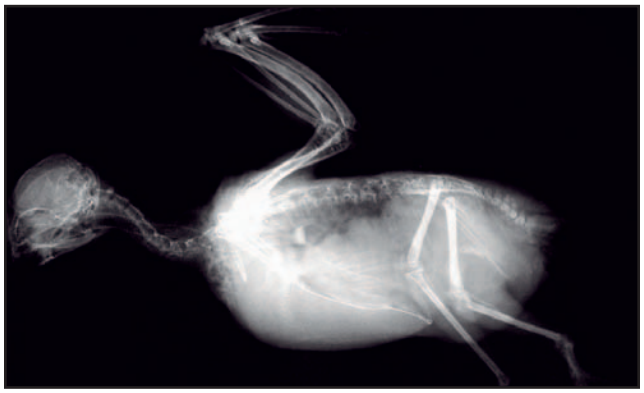
REPRODUCTIVE-ASSOCIATED COELOMITIS

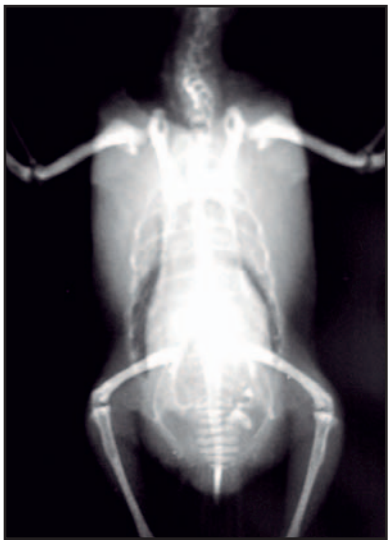
Reproductive-associated coelomitis may encompass egg yolk coelomitis, (previous egg related peritonitis) ectopic ovulation-associated coelomitis and septic coelomitis. Coelomitis may be found in association with other diseases such as malnutrition, metabolic disorders and systemic infections. Cystic ovarian disease, salpingitis, metritis, cystic hyperplasia, oviductal rupture, oviductal and ovarian granulomas, septicemia, intestinal rupture and neoplasia may cause associated coelomitis as well.[8,31,73]

Tentative diagnosis of reproductive-associated coelomitis is made through history, physical examination and supporting laboratory tests. The hen may have a history of egg production, which may have abruptly stopped. Clinical signs may include transient or persistent depression, lethargy and inappetence. Patients with more advanced disease may suffer from weight loss, abdominal distension, and dyspnea associated with coelomic fluid and air sac compression (Fig 18.11). It is important to note that not all patients with coelomitis will have identifiable fluid present. Supportive diagnostic tests include a complete blood count, serum chemistries, radiographs, coelomic ultrasound and analysis of any fluid recovered from abdominocentesis including cytology, culture and sensitivity. A leukocytosis with a relative heterophilia, as well as a peripheral hypercalcemia, hyperglobulinemia and hypercholesterolemia compatible with pre- and immediate postovulation may be noted. Many birds will actually be hypocalcemic due to calcium depletion subsequent to malnutrition or chronic egg laying. Some birds may have egg yolk visible in their peripheral blood smears as well as above the buffy coat in separated blood samples and lipemia is common. Radiographs may demonstrate polyostotic hyperostosis, soft tissue density in the region of the ovary and/or oviduct, coelomic fluid, abdominal and caudal thoracic air sac compression, or even an obvious shelled or non-shelled egg, or egg remnants leading to oviductal granuloma (Fig 18.12). Contrast radiography may be helpful to illustrate organ displacement and locate any suspected space-occupying mass. Ultrasound may reveal coelomic fluid, ovarian follicle(s), ovarian cyst(s), an ovarian mass, and oviductal masses such as granuloma or neoplasia (Figs 18.13a,b). Cytology of coelomic fluid may demonstrate a septic or non-septic exudate, a transudate, or yolk or fat globules if such material is present. Bacterial culture and sensitivity should be performed on samples of coelomic fluid. Laparoscopy and/or laparotomy may be necessary to identify the causative etiology of coelomitis.[8,31,40,63,73]

Treatment of reproductive-associated coelomitis varies with type and severity of clinical signs. Many birds respond well to supportive care alone. Abdominocentesis is not only supportive of diagnosis but therapeutic as well, to relieve dyspnea due to air sac compression. Broad-spectrum antibiotics should be initiated in cases of suspected or confirmed infectious coelomitis while waiting for sensitivity results on fluid obtained from abdominocentesis. Corticosteroids may be indicated in cases where an infectious etiology has been excluded, but should be used judiciously due to potential serious side effects. Pharmacologic therapy may be used to stop further ovulation, reproductive hormone production and to reduce the size of the reproductive tract, which may perpetuate this condition.

Once the coelomic fluid has decreased and the patient is stable, it is beneficial to perform a laparoscopic





Figs 18.13a,b | Left lateral and ventrodorsal views of a 6-year-old male budgerigar with a Sertoli cell tumor. Note the soft tissue mass in the midcoelom and polyostotic hyperostosis of the long bones.

examination. This allows direct visualization of the ovary, oviduct and other organs to help confirm etiology such as a cyst, granuloma and/or tumor. Cytology and biopsy with histopathologic examination, and bacterial culture and sensitivity should be performed on abnormal tissue. A celiotomy with or without a salpingohysterectomy may be necessary to biopsy or remove a mass, cystic oviduct or remove inflammatory debris from the abdomen, particularly if medical therapy alone is not effective. It is important to note that during laparoscopy or laparotomy there is a risk of fluid gaining access to the respiratory system via the incision through the abdominal air sacs, and often there are significant adhesions between the oviduct and neighboring viscera due to chronic inflammation.[8,31,63,73]

OOPHORITIS

Inflammation of the ovary results from neoplastic, mechanical or infectious causes. Infectious oophoritis often occurs as a result of spread from adjacent organs or septicemia, and is frequently bacterial in origin. Clinical signs may be vague and include anorexia, weight loss, depression, cessation of egg production, egg binding and sudden death. A diagnosis of oophoritis is made through history, physical examination, radiography, ultrasonography, abdominocentesis with coelomic fluid analysis, laparoscopy, laparotomy, and biopsy of the ovary with bacterial culture and sensitivity and histopathologic analysis. Hematology may demonstrate a leukocytosis with a relative heterophilia. Radiographs and ultrasound may demonstrate an egg or an enlarged soft tissue density in the region of the ovary, ovarian follicle(s) and ovarian cyst(s), and there may be coelomic fluid present if there is a concurrent coelomitis. If coelomic fluid is present, abdominocentesis is beneficial both therapeutically and diagnostically. Cytologic analysis as well as bacterial culture and sensitivity should be performed on fluid recovered. Laparoscopy may demonstrate an enlarged, abnormal-appearing ovary, which may have associated hypervascularization. Persistent or chronic oophoritis may progress to granulomatous disease, which may be evident on ultrasound, laparoscopy and laparotomy. Definitive diagnosis is based on ovarian biopsy with histopathologic examination along with bacterial culture and sensitivity.[8,31,40,63,73]

Treatment of oophoritis includes broad-spectrum antibiotics, pending sensitivity results. Egg binding is handled as previously described. As discussed for chronic egg laying, pharmacologic therapy to temporarily stop ovulation should be initiated, as it does appear clinically that ovulation may perpetuate inflammation of the ovary. Laparoscopic exam, bacterial culture and sensitivity, and complete blood count should be repeated until culture results are negative and any leukocytosis has resolved. Reproductive performance and general condition should be carefully followed, as complete resolution may be difficult. Partial ovariectomy, usually performed with salpingohysterectomy, may be beneficial in refractory cases if the hen is not intended for breeding.[8,31,63,73]

OVARIAN AND OVIDUCTAL NEOPLASIA

Ovarian and oviductal neoplasia is most commonly seen in the budgerigar (*Melopsittacus undulatus*), cockatiel (*Nymphicus hollandicus)* and gallinaceous species. Clinical signs may include abdominal distension, coelomic fluid, lameness, dyspnea, depression, inappetence and chronic reproductive-associated behavior with or without egg production. Egg binding, oviductal impaction, ovarian cysts, abdominal hernia and coelomic fluid may be seen in conjunction with reproductive tract neoplasia. This fact demonstrates the extreme impor-

tance of a complete diagnostic work-up. Alteration of secondary sex characteristics such as a cere color change may occur as well. Diagnosis is supported by history and physical examination, demonstration of enlargement in the area of the ovary or oviduct on radiographs and ultrasound, and biopsy with histopathologic examination of abnormal tissues. Lymphomatosis, adenocarcinoma, leiomyosarcoma, leiomyomas, adenomas and granulosa cell tumors have been reported.[3,10,31,40,41,63,73] There have been anecdotal reports of treatment with chemotherapeutic drugs such as carboplatin (D. Zantop, personal communication, 2000); however, no consistent results have been documented to date. Prognosis for long-term recovery is grave, with no refereed reports of successful treatment. Salpingohysterectomy with partial or complete ovariectomy may have value in select patients.[31,63,73] Cryosurgical ablation of the ovary and anti-angiogenesis therapy may prove beneficial.

PARASITES

Ascarids and flukes have been reported to infect the oviduct from the cloaca by reverse peristalsis. Heavy infestation may cause soft-shelled and shell-less eggs, and may result in salpingitis. Anseriformes are most commonly affected. Ascarids and small flukes reportedly have been passed in eggs. Diagnosis is made by finding adult worms in the oviduct on celiotomy or necropsy, or by finding adult worms in eggs laid by affected hens. Fecal floatations should be performed, especially on ground-dwelling species, and prophylactic anthelmintic programs may be helpful in preventing severe infestations. If these parasites obstruct the oviduct, they require surgical removal or salpingohysterectomy.[73]

OVERPRODUCTION OF EGGS

Safe numbers for egg production for different species are not definitively documented. Nutrition and environmental conditions affect safe production levels. Free-ranging psittacines typically produce one to two clutches per year; however, many captive psittacines produce far more eggs than this. While many birds show no obvious side effects, chronically overproducing hens may develop reproductive tract disorders, as well as poor body condition and feather quality. To improve long-term health in producing birds, egg production should be limited to two clutches per year in birds that show any signs of poor health secondary to overproduction. It also is recommended that all birds receive some rest period each year to prevent reproductive disorders from developing.[73]

ORCHITIS

Infectious orchitis may occur from ascending infections, hematogenous spread or infected adjacent organs. Rarely, non-infectious causes are associated with inflammation of the testicles. Early clinical signs are vague and difficult to detect, and may include infertility, mild depression and decreased appetite. As the disease progresses, the patient may develop lethargy, inappetence and abdominal distension if a secondary coelomitis develops. Leukocytosis with a relative heterophilia may be noted. Testicular enlargement may be noted on radiographs and ultrasonography, which is a normal condition for a normal male. There also may be notable enlargement, inflammation and hypervascularization on laparoscopy. Definitive diagnosis is made by cytology, bacterial culture and sensitivity, and histopathologic examination of samples from affected testis. Therapy includes broad-spectrum antibiotics, pending sensitivity results.[12,31,73]

TESTICULAR NEOPLASIA

Testicular neoplasia has been commonly documented in the budgerigar (*Melopsittacus undulatus*) and is often unilateral. Clinical signs include abdominal distension and one-sided paresis, paralysis, or cyanosis and hypothermia of the pelvic limb due to compression of the ischiatic nerve and blood vessels. The disease is often advanced once clinical signs are evident. Definitive diagnosis is made by testicular biopsy and histopathologic examination.[13] Alterations of secondary sex characteristics such as cere color change from blue to brown may occur. Neoplasms reported include Sertoli cell tumor, seminoma, interstitial cell tumor and lymphosarcoma. Leiomyosarcoma and carcinoma have been reported to arise from the epididymis and ductus deferens.[3,10] Radiographs may reveal a soft tissue mass in the region of the testicles, air sac compression, and secondary sex changes such as polyostotic hyperostosis (see Figs 18.13a,b). Treatment includes orchiectomy. Cryosurgical ablation may be beneficial. Chemotherapy with carboplatin and O,P'-DDD (mitotane)[21] has been anecdotally reported if the tumor is deemed incompletely or non-resectable, or if the patient is not a good surgical candidate. However, no conclusive data have been reported to date regarding efficacy of chemotherapy.[31,73]

CLOACAL PAPILLOMAS

Cloacal papillomas have been noted in New World psittacine species with green-wing macaws over-represented. To date the cause is unknown, but a herpes virus etiology is strongly suspected[7,22,39,57,62] (see Chapter 32, Implications of Viruses in Clinical Disorders). Clinical signs may include infertility due to mechanical obstruction, hematochezia, and straining to urinate and defecate. Examination of the cloaca reveals one or several fleshy masses at the mucosal border. Inserting a lubricated swab into the cloaca to evert the tissue facilitates cloacal examination (Fig 18.14). In addition, white vinegar applied to

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Fig 18.14 | A 22-year-old yellow-naped Amazon parrot with a history of depression, weakness and inappetence. Note the papillomatous masses at the mucocutaneous junction of the cloaca.

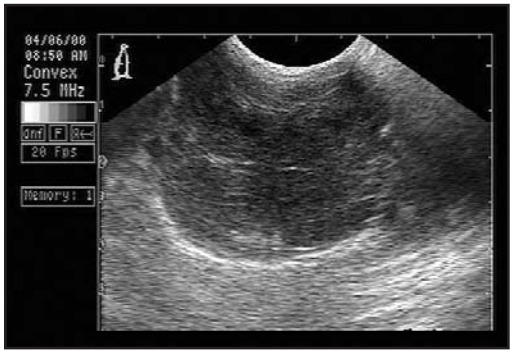

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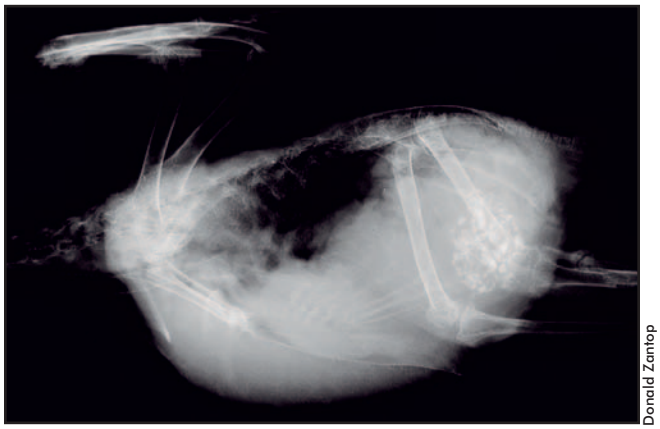
Fig 18.16 | Coelomic ultrasound of the same Amazon parrot. Note the severely enlarged liver with increased echogenicity. Liver biopsy and histopathologic examination revealed bile duct adenocarcinoma (7.5-MHz probe).



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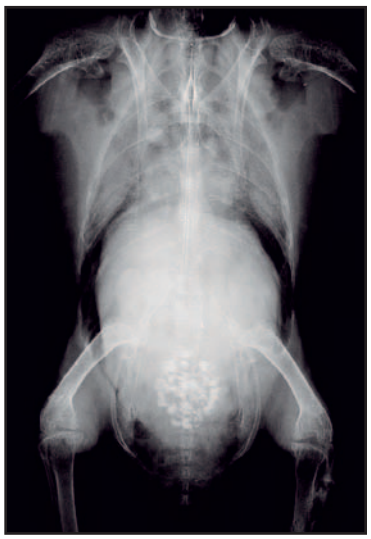
Figs 18.15a,b | Ventrodorsal and right lateral radiographs of the Amazon parrot in Fig 18.16. Note the severely enlarged liver and caudal displacement of the grit-filled ventriculus.



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the cloacal wall will significantly blanch papillomatous tissue, further assisting in identification. A thorough endoscopic exam of the cloaca should be performed to rule out obstruction of the gastrointestinal, urinary and reproductive tracts. It is important to perform a full examination to detect oropharyngeal and laryngeal papillomas and hepatomegaly. It is equally as important to perform complete blood count, serum chemistries, bile acids evaluation, radiographs including contrast studies, coelomic ultrasound, and endoscopic exam of the coelom and cloaca. These tests are crucial to fully evaluate the patient, as papillomas may occur at any site of the gastrointestinal tract. Furthermore, bile duct and pancreatic adenocarcinoma are associated with papillomatous disease.[25,31,32,73,76] It is necessary to perform a liver and pancreatic biopsy for histopathologic examination to rule out early bile duct and pancreatic adenocarcinoma upon diagnosis of cloacal papilloma (Figs 18.15a,b, 18.16). Cytology, culture and sensitivity are beneficial to diagnose a secondary cloacitis. Histopathology of excised or biopsied tissue will confirm the diagnosis.[31,73]

Therapy includes cauterization of papillomas with silver nitrate, cryosurgery, cloacotomy and laser surgical removal.[18,61] Autogenous vaccines and cloacal mucosal stripping have not met with consistent success.[2,64] Imiquimod[h], an immune response modifier used in humans for the treatment of anogenital warts, has been shown to decrease lesion mass and tenesmus, but it did not cause remission of papillomatous tissue.[43] It is important when cauterizing papillomatous tissue with silver nitrate to flush the area profusely with saline when sufficient tissue is cauterized, otherwise normal cloacal tissue will be cauterized. Topical medications such as dimethyl sulfoxide and silver sulfadiazine cream should be applied to affected tissue after cauterization. Analgesics should be administered postoperatively as well as antibiotics if there is an associated cloacitis. Temporary spontaneous remission has been reported. Recurrence is extremely common and frequent re-examinations are necessary.[31,73] Spontaneous regression has been reported.

Carboplatin chemotherapy has been reported to benefit patients suffering from bile duct and pancreatic adenocarcinoma, and does appear to be well tolerated.[20,79]

Affected birds should be separated from the non-affected to prevent possible transmission.[8,31,73] It is beneficial to

Fig 18.17 | Prolapsed cloaca in a 5-year-old male umbrella cockatoo. History included chronic masturbation and intermittent partial cloacal prolapse.

Fig 18.18 | The same umbrella cockatoo after manual cloacal reduction and cloacopexy.

perform a thorough cloacal examination, particularly on New World psittacines, during routine health examinations, as affected birds should be isolated to prevent potential spread to non-affected individuals. Healthy chicks have been raised from artificially incubated or fostered eggs from affected pairs.[31,73]

CLOACAL PROLAPSE

Cloacal prolapse may occur secondary to chronic straining from masturbation, egg laying, space-occupying abdominal masses, and inappropriate weaning and social behavior. Physical examination will reveal prolapsed tissue through the vent that may be intermittent or persistent (Fig 18.17). Careful cleaning, irrigation and lubrication of prolapsed tissue are a necessity. Affected tissue should be examined for necrosis, and any adhered egg should be removed. Cytology and bacterial culture and sensitivity should be performed on prolapsed tissue to aid in antibiotic therapy. A complete blood count, serum chemistries, radiographs, ultrasound and endoscopic exam of the coelom and cloaca are useful to determine any other predisposing cause.[31,73] Chronic reproductive-associated behavior and straining secondary to masturbation may respond to pharmacologic therapy such as leuprolide acetate or environmental manipulation to decrease reproductive stimuli. Cloacopexy and the use of temporary stay sutures may be helpful in temporary or permanent reduction. However, those procedures interfere with movement of the cloaca and may alter defecation and micturition[4,66] (Fig 18.18). Ventplasty may decrease the vent opening and prevent further prolapse if the vent has become flaccid (see Chapter 35, Surgical Resolution of Soft Tissue Disorders). Clomipramine hydrochloride and phenylpropanolamine administration has been anecdotally reported to contract the vent orifice and assist in the resolution of prolapse of the cloaca (D. Zantop, personal communication, 2003). Salpingohysterectomy with partial ovariectomy or orchiectomy may be beneficial in those patients refractory to medical therapy. Broad-spectrum antibiotics should be initiated, pending bacterial culture and sensitivity, because primary and secondary bacterial infections are common.[8,31,63] See Chapter 3, Concepts in Behavior.

CLOACITIS

Cloacitis may result from both infectious and non-infectious processes. Cloacal prolapse, cloacal papillomas, cloacoliths and bacterial infections may cause inflammation of these tissues. This may result in secondary urogenital and/or gastrointestinal disease due to the anatomic relationship to the cloaca. Cytology with bacterial culture and sensitivity should be performed. Appropriate antibiotics or anti-inflammatory therapy may be indicated. Dimethyl sulfoxide may be used to reduce inflammation with no systemic side effects, and swabbing the cloaca with petroleum jelly will prevent fecal and urate accumulation on the cloacal surface with subsequent irritation.[73]

CLOACOLITHIASIS

Cloacolithiasis is infrequently noted in pet birds. It may result from previous egg binding, infectious cloacitis, malnutrition or neurologic disease of the cloaca. Cloacoliths should be manually or surgically removed. Cloacal cytology with bacterial culture and sensitivity should be performed. The identification of anaerobic bacteria, notably *Clostridia* sp., is a common finding often associated with a fetid odor. Since routine aerobic cultures will not isolate these organisms, Gram's stains of the feces should be performed. Broad-spectrum antibiotics should be initiated, pending culture results.

Improved nutrition through diet change should be initiated. Patients should be monitored closely for recurrence, and prognosis for return to normal breeding performance is poor.[73]

CLOACAL NEOPLASIA

Cloacal carcinomas are infrequently reported in pet birds. An endoscopic examination of the cloaca should be performed, and the patency of openings into the urodeum, proctodeum and coprodeum should be noted. Definitive diagnosis is made from histopathologic analysis of biopsy samples. Neoplastic masses should be surgically removed, with caution not to damage the openings to the gastrointestinal, urinary and reproductive tracts.[3,41,42,73]

OTHER CLOACAL DISEASES

Other cloacal diseases include cloacal strictures and excessive vent feathering. Cloacal strictures may be gently manually dilated with the use of a speculum. Excessive feathering around the vent may cause infertility; prior to breeding season, these feathers should be removed by trimming or pulling.[73]

REPRODUCTIVE HORMONE-RELATED FEATHER PICKING

Birds pick their feathers for several different reasons. Avian veterinarians often cannot elicit a cause from a complete history, physical examination and diagnostic work-up. In some avian patients, history may reveal reproductive-related behaviors such as breeding, masturbating, regurgitation and failure to molt. Physical exam may demonstrate removal of one or several types of feathers, rough feather condition of any remaining feathers, and the patient may become reproductively stimulated during the examination. Serum chemistries panels may reveal elevated serum calcium in hens (provided they are not calcium deficient) and hypercholesterolemia. Radiography may demonstrate an enlarged gonad(s) and/or polyostotic hyperostosis. Ultrasonography and laparoscopy may reveal ovarian follicles, ovarian cysts, an enlarged oviduct or enlarged testicles. The diagnostic work-up must be extensive to rule out any other possible diseases. Serum estradiol, progesterone and testosterone level tests are available, although normal values are not known for every avian species. Pharmacologic treatment, behavior counseling and environmental changes to reduce reproductive drive and hormone levels as discussed with chronic egg laying are recommended. Response to treatment is important therapeutically and diagnostically. Treatment failures may be due to the presence of non-reproductive-related disease and concurrent disease. It is important to note that a full history and diagnostic work-up must be performed prior to establishing a diagnosis of reproductive-related feather disease. Many of these birds molt heavily after starting therapy and this should not be interpreted as a deleterious side effect.[8,11,48]

Products Mentioned in the Text

a. Depo-Provera, Upjohn C., Kalamazoo, MI, USA
b. Levonorgestrel, Sigma Chemical, St. Louis, MO, USA
c. HCG, Pregnyl Organon, Inc, West Orange, NJ, USA
d. Ortho-McNeal Pharmaceutical, Rantan, NJ, USA
e. Lupron Depot, TAP Pharmaceuticals Inc
f. Tamofen, Phone-Poulenc Rorer Canada Inc, Montreal Quebec, Canada
g. Prepidil, Pharmacia and Upjohn, Kalamazoo, MI, USA
h. Imiquimod Aldara 5%, 3M Pharmaceuticals

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