Physiology, diagnosis, and diseases of the avian reproductive tract

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Although the reproductive cycle and its pathologies have been thoroughly investigated in many species, this is not the case in avian medicine, where there is a great discrepancy between the knowledge of sexual and reproductive aspects of some of the commonly and commercially farmed species, such as chickens, turkeys, and ducks, and the less is understood exotic or wild birds.

This may be due to several reasons:

1. General knowledge of avian medicine is more superficial with over 8000 species, compared to medicine of companion animals.
2. The reproductive physiology of birds has been less investigated, compared to mammalian reproductive physiology.
3. Inhibiting and/or stimulating factors of the reproductive cycle (external, like day length, as well as internal factors like hormonal levels) vary depending on different avian species/groups.
4. The reproduction of birds until recently has been extrapolated from species of relevant commercial interest (chicken, turkey, goose, duck, etc.)

Thus, until recently, the study of reproduction in pet birds had not been approached scientifically. Even more recent is the advent of attempting to treat infertility/hypofertility in these species. This is due to the growing interest for these animals [1]
Anatomy of the male avian reproductive tract

Testis

The bean-shaped testes are located laterally to the Vena cava caudalis, caudally to the adrenal glands and lungs and medially to the cranial kidney lobes [1,2]. Each testis is suspended by the mesorchium, is partially surrounded by the abdominal air sac, and is encapsulated by two fibrous layers [3,4]. This peritoneal fold serves not only as an attachment for the testis, but also as a conduit for nerves and blood vessels [5]. Size, color, and shape of the testes may vary with age and also between species. Dark coloration is due to the presence of melanoblasts in the interstitium, so that melanistic testes, like melanistic ovaries, can occur in some species [3,6]. Bicolored testes are not considered abnormal (Figs. 1–3).

The dimensions of the testis increase greatly with sexual activity. In seasonal birds and especially in some passerines, testicular mass can increase 300 to 500 times. This is due to the increased length and diameter of the seminiferous tubules and to a greater number of interstitial cells, in response to LH and FSH [2,4]. The surface of the testis is covered by a very thin

Fig. 1. Crimson-bellied conure (Pyrrhura perlata). Endoscopic view of a normally developed adult testicle. (From Crosta L. Endoscopia chirurgica negli Uccelli. In Croce A, editor. Animali esotici da compagnia. Gaggiano, Italy: Poletto Editore, in press.)
tunica albuginea, but septa, and hence lobulation, are absent. Also, there is no mediastinum testis, an incomplete vertical septum formed by a portion of the tunica albuginea that, in mammals, is reflected into the interior of the testis [2]. In the domestic fowl, most of the blood reaches the testis by the testicular artery, a branch of the cranial renal artery. The testis is drained by several short testicular veins into the Vena cava caudalis [2].

The bulk of the testis is composed of thousands of seminiferous tubules. These tubules resemble mammalian tubules, but in the avian testis there is a greater number of anastomoses. The seminiferous tubule is lined by a spermatogenetic epithelium consisting of germ cells and Sertoli cells. Spermatogenesis proceeds through three phases: first a period of multiplication, in which the cells are known as spermatogonia; in the second phase, the cells (primary spermatocytes) grow and enlarge; finally, in the third period, the first maturation division forms the secondary spermatocytes and the second maturation division forms the spermatids. Each spermatid will develop into a spermatozoon. Spermatogenesis is shorter in birds than in mammals [2].

When spermatozoa are mature they detach themselves from the Sertoli cells and pass towards the very short, outgoing straight tubules, which end...
by opening into the rete testis. This is a meshwork of tubules embedded in connective tissue [4]. This may not be present in all avian species, but in the domestic fowl consists of thin-walled irregular channels, embedded in connective tissue on the dorsomedial surface of the testis. The interstitial, or Leydig, cells, occupy some of the interstitial space between the tubules. They contain cholesterol-rich lipid droplets that are believed to be the precursor material involved in the production of androgenic steroids [2].

**Epididymis**

It is relatively short, when compared to the mammalian counterpart, and lies on the dorsomedial surface of the testis. A head, body, and tail cannot be distinguished. The efferent tubules are initially of a wide diameter, but
then taper and finally open, via short connecting tubules, into the epididymal duct. When the testis is removed surgically, the main part of the epididymis is resected with the gonad, but the appendix remains attached to the adrenal gland. Here it can sprout nodules that secrete androgens [2].

**Ductus deferens**

The anatomy of the deferent duct seems similar in the domestic fowl and wild species. The duct proceeds parallel with the ureter in a zigzag course. Its diameter is increased progressively by smooth musculature, and it enters into the dorsal wall of the urodeum. In passerine birds, the caudal end of the ductus deferens forms a mass of convolutions called the seminal glomus. This is believed to be the main storage site for the spermatozoa, because in the glomus the temperature can be 4°C below core body temperature [2].

**Accessory organs**

Although birds do not have organs homologous to the mammalian prostate, seminal vesicle, bulbourethral glands, and ampulla, accessory reproductive organs do exist and include the paracloacal vascular bodies, the dorsal proctodeal gland, and lymphatic folds [2,6]. The accessory reproductive organs are either in proximity to, or are an integral part of, the cloaca [5].

**Semen**

There are two kinds of avian spermatozoon: a complex type characterizing the Passeriformes, and a simple form typifying all the remaining orders. The complex type is typified by a predominantly spiral structure. The simple type is approximately 100 μm long [2]. Like mammals, each spermatozoon is composed of an acrosome, head, and tail [4]. Seminal plasma composition can vary among species, but is similar in the domestic fowl and the budgerigar. A lymph-like fluid, called transparent fluid, originates in the proctodeum and mixes with the semen during ejaculation.

**Phallus**

Most avian species do not have any penis-like organ, but in those species that do have such a structure, it can be of two types: the truly intromittent phallus that occurs in the ratites, tinamous, anseriformes, and cracidae, and the nonprotruding (nonintromittent) phallus observed in some galliformes [2]. When present, the phallus is located ventrally in the proctodeum. Dysfunction or disease of the phallus can cause reproductive failure. Psittacine birds do not have a phallus, and copulation is accomplished by an eversion of the cloacal wall, which contains the slightly raised papilla that transfers semen to the everted orifice of the oviduct [3].
Anatomy of the female avian reproductive tract

In general, female birds have a functioning left ovary and oviduct, while the right side of the reproductive tract only develops in the beginning of the embryonic phase and remains vestigial. In chickens, the distribution of primordial germ cells becomes asymmetrical by day 4 of incubation [7]. Exceptions to this rule are some Falconiformes (Falconidae, Accipitridae, and Cathartidae), in which development of both ovaries (and more rarely both oviducts), may happen, and the Brown kiwi (Apteryx australis), in which both ovaries are functional, but only the left oviduct develops [1]. The reason for this unilateral development is still unknown, but one advantage might be reduction of body weight to improve the ability to fly. Because the Falconiformes are known to be excellent flyers, this theory remains questionable [8].

The left ovary is located craniodorsal in the coelomic cavity and has a close contact with the cranial pole of the left kidney, the caudal part of the left lung, the left and sometimes the right adrenal gland (Figs. 4–6). Due to the close contact with the peritoneum, its suspensor ligament (mesovarium) measures only a few millimeters [8]. The oviduct can be divided in five portions: infundibulum, magnum, isthmus, uterus, and vagina. The mature oocyte is caught by the infundibulum, and in its longer part, the Tubus infundibularis, fertilization of the oocyte occurs. While passing down the oviduct, the yolk is covered with the various layers of albumen to make up the

Fig. 4. Musk lorikeet (Glossopsitta concinna). Endoscopic view of a normal, young left ovary.
content of the egg. In the domestic fowl the egg needs about 24–25 hours to pass along the oviduct, and the majority of this time (about 20 hours) is spent in the uterus, where it is covered by the eggshell [8,9] (Fig. 7). Female birds also have special spermatic crypts (Fossulae spermaticae), in the Lamina propria of the most distal portion of the oviduct (vagina), where the sperm can be stored up to several weeks and be present for subsequent fertilizations [9]. The ovary receives blood from the Arteria ovarica. The blood supply for the oviduct is brought by the Arteria oviductalis cranialis, A. oviductalis cranialis accessoria, A. oviductalis media, and the A. oviductalis caudalis [8].

General avian reproductive physiology and endocrinology

Hypothalamus–pituitary–gonadal axis

Endocrine and behavioral components of reproduction in birds are directed by the hypothalamus in response to environmental triggers and internal factors [10]. As in other vertebrates, the reproductive system is regulated by the hypothalamus–pituitary–gonadal axis. The hypothalamus produces a gonadotropin-releasing hormone (GnRH), that stimulates the pituitary gland
to produce luteinizing hormone (LH) and probably also in birds, follicle-stimulating hormone (FSH). These regulate gonadal function. In turn, gonadal steroids returns to the central nervous system by the bloodstream and provide a feedback regulation of the GnRH production and release. In birds, the process is complicated by the fact that two forms of avian GnRH are known to exist. These types of GnRH were first described in the chicken, and were called cGnRH-1 and cGnRH-2, but since then, several avian species have been shown to have both cGnRH-1 and cGnRH-2 [10].

GnRH is secreted into the portal veins of the hypophysis, where it is transported to the adenohypophysis. Norepinephrine is thought to play the major role for the release of GnRH [11]. Although there is evidence for the separate functions of the different forms of GnRH, the exact function of cGnRH-2 is not fully understood [10]. cGnRH-1 is known to be involved in the release of gonadotropins [12]. cGnRH-2 is several times more potent than cGnRH-1 in stimulating LH release from chicken pituitary cells in vitro, but there is little difference in in vivo activity. It is likely that cGnRH-1 exerts a more important role than cGnRH-2 in the control of LH secretion [13]. The release of FSH is thought to be under hypothalamic control, it is not completely understood if GnRH is really involved in FSH release. There is also evidence that the whole GnRH system is directly involved in social and behavioral interactions [10].

Fig. 6. Desmarest’s Fig parrot (*Psittaculirostris desmarestii*). Necroscopic gross appearance of a normally developed, active female sexual apparatus.
Physiology and endocrinology of the male avian reproductive tract

Avian gonadotropins (LH and FSH) are glycoproteins produced in the adenohypophysis [11]. They are composed by two subunits: an alpha-subunit (common to LH, FSH, and TSH) and a beta-subunit (hormone specific). Both LH and FSH are of primary importance for the control of testicular function in the male bird. As in mammals, LH stimulates the Leydig cells to produce sexual hormones, whereas the major role of FSH is stimulation of spermatogenesis [13]. Leydig cells respond rapidly to LH stimulation and produce testosterone and androstenedione. Although it is clear that FSH acts on the Sertoli cells, the way in which it works is poorly understood. However the effect of FSH is potentiated by testosterone [5].

Spermatogenesis

To date, spermatogenesis in birds has primarily been studied in the Japanese quail (Coturnix c. japonica). Four types of spermatogonia have been
described. They are followed by primary and secondary spermatocytes and spermatids. Spermiogenesis, which can be defined as the transformation of spermatids into spermatozoa without further cell division, is reported to entail 8–12 steps in the most studied Galliformes. One of the most striking features of this process is the reduction of cytoplasm and cell volume: mature sperms embody only the 3% of the initial spermatid cell volume [5]. In the domestic fowl, the spermatozoa undergo maturation in the proximal reproductive tract and must reach the ductus deferens before becoming fully fertile. This journey takes from 1 to 4 days [2].

Following spermiation, sperm are suspended within fluid secreted by Sertoli cells. Sperm passage through the labyrinth of seminiferous tubules most likely depends upon the hydrostatic pressure of the fluid in the seminiferous tubules and the contractility of the myoepithelial cells overlying the surface of the tubules. After this, sperm passage through the distal excurrent ducts (efferent ducts and connecting ducts) and deferent duct, depends upon peristalsis. The most evident change during sperm transport along the excurrent ducts is the absorption of seminiferous tubule fluid and subsequent concentration of sperm. The efferent ducts, in the epididymis, may be a critical site for sperm maturation. Due to extensive mucosal folding and the presence of apocrine secretion, ciliated cells, and cells with abundant microvilli, the efferent ducts appear to be the place where sperm are mixed with secretions. The resulting medium is the seminal plasma. Its chemical composition differs from blood plasma in electrolytes, free amino acids, and protein composition. Sperm are immotile prior to ejaculation. A proposed theory for a factor that stimulates sperm motility is the temperature change accompanying ejaculation. However, more recent research has shown that accessory reproductive fluids may play a role in this process. In this respect, Ca^{2+} and HCO_3^- have been shown to stimulate motility in vitro [5].

**Physiology of the avian female avian reproductive tract**

The physiology of the female sexual apparatus is not yet fully understood. Seventeen different hormones are involved in the ovulatory cycle of the hen, and at least two forms of gonatropin releasing hormone (GnRH-I and GnRH-II) are known to exist. A constant exposure to GnRH leads to decreased gonadotropin output while pulsatile GnRH stimulation increases gonadotropin production [14]. Four to 6 hours prior to ovulation, a peak of luteinizing hormone (LH) and progesterone plasma concentration occurs. LH and progesterone have a close relation and interaction. While progesterone naturally increases during ovulation, an injection of progesterone can induce a preovulatory surge of LH and premature ovulation. Several findings indicate that progesterone induces the preovulatory LH peak. This preovulatory LH peak likely provides a stimulus for germinal vesicle breakdown and for subsequent ovulation [7]. The ovulatory response to external LH depends on the stage within the cycle. LH treatment 11–14
hours prior to ovulation results in premature ovulation, while LH treatment before mid-sequence ovulation results in follicle atresia [15]. The role of Follicle-Stimulating-Hormone (FSH) in the female reproductive cycle remains unclear [16]. The mechanism for the release of FSH in the female reproductive has not been clarified. It has been reported that GNRH-1 induces FSH secretion, while other studies could not find any stimulation of FSH by GNRH-1 [17,18].

The majority of estrogen is produced by prehierarchal follicles [7]. Like testosterone, estrogen is also unlikely to be involved in the induction of LH secretion or ovulation. On the other hand, estrogen, together with progesterone, is responsible for priming the hypothalamus and pituitary for progesterone, which itself induces LH release [19]. Other functions of estrogen are the regulation of the calcium metabolism and the control of sexual behavior [7,20].

**Factors affecting the sexual activity of birds**

Reproduction in birds involves several aspects, each of which, like in a puzzle, must all fit perfectly to result in balanced physiologic well-being and successful breeding. Aside from macro- and microanatomical perfection, in the absence of which reproduction would not be possible, environmental events play an important role in this process.

**Seasonal cycles**

The annual cycle of most avian species revolve around three events: breeding, molting, and survival until the following breeding season. Variations in this routine may be migration, and breeding after molting, as happens in King and Emperor penguins. This annual cycle involves a sequence of complex integrated behavioral and physiologic conditions. An elaborate biologic clock synchronizes birds biologic changes with the environmental cycles [21].

Day length, or photoperiod, plays a key role in this control system. In bird species that evolved in temperate zones, the gonadal cycles are predominantly controlled by photoperiod. These species use the increasing day lengths of late winter and spring as a initial cue for the stimulation of gonadal growth. The pineal gland is believed to be the location of the biologic clock and the mechanism of photosensitivity in birds, but the pineal gland of birds differs from the reptilian one, as it does not house the primary light receptors. Furthermore, birds do not measure the day length visually, as do mammals, but have special receptors in the hypothalamus. Indeed, it is interesting that birds require neither eyes nor a pineal gland to detect changes in day length [14]. When these receptors are stimulated by an increasing day length, neurosecretory cells in the hypothalamus induce the release of GnRH, which will promote the synthesis and release of LH and, probably, FSH [13,21].
Prolonged exposure to different day lengths does not continue to stimulate birds to breed, but instead they become photorefractory. A relatively long (5 weeks in some studied species) period of short day length is needed to eliminate photorefractory state [13]. The exact physiologic mechanism of photorefractoriness is not fully understood, but it is best developed in migratory species from the temperate zones [21]. In addition, it is believed that the development of photorefractoriness is mediated by the secretion of thyroid hormones and is associated to the GnRH system in some species [14].

Humidity and rainfall

Although few experimental studies exist on the relationship of environmental humidity and/or rainfall, with avian breeding behavior, it is clear that some species, especially desert ones, use the rainy season as an indicator for the arrival of the breeding season. Many anecdotal reports suggest that birds appear to enjoy being sprayed with water or lay eggs after exposure to misting [14,22].

Nutrition

There is no doubt that nutrition influences gonadotropic drive [14]. Whether nutritional factors can act as breeding cues for birds that are already feeding on a nutritional complete diet is not known, even if examples do exist. Two bird species well known for having the breeding season induced by the availability of fresh foodstuff are the budgerigar (Melopsittacus undulatus) and zebra finch (Taeniopygia g. castanotis). Both of these species start breeding according to rainfall, and thus the direct availability of fresh vegetation, which they use both for nest building (zebra finch) and chick rearing. The specific appetite for calcium is the most dramatic example, at least in poultry, of a specific nutritional requirement for reproduction [14]. Malnourished birds will probably not court or mate at all, or their breeding performance will be poor [6].

Housing and nesting

Several aspects of the aviary design may influence the sexual activity of birds. In many species of parrots, nest availability serves as a crucial denominator in initiation of reproductive activities [23]. Although all captive birds should be provided with safe, spacious housing, several aspects of caging are of particular importance in the breeding facility. Disturbing the breeding pair with noises or any other stressors, is a certain way to reduce production levels [6].

Birds must be housed according to their size, number, environmental needs, and any other known factor specific for the species [24].
Social contacts

Social cues can contribute to sexual stimulation, particularly in monogamous and colonial birds. In some species, ovarian development is stimulated by specific calls, to the extent that sexual relevant calls may have direct input to cGnRH-1 neurons [14]. The presence of one partner or several cage mates, according to the species, may upset the sexual activity in birds. The avian veterinarian must concentrate on the suitability of avian and nonavian species kept nearby, to avoid mistakes that will lead to poor breeding performance [24].

Male reproductive disorders

Although there are likely several diseases affecting the male avian sexual apparatus, they are not all currently known. Documented diseases affecting the male reproductive system are largely limited to the testis [24]. Diseases of the female reproductive tract tend to be more obvious, and have been studied in greater detail [1].

Orchitis

Orchitis is an inflammatory process that occurs to the testis. Orchitis can be uni- or bilateral, and understanding this can be extremely important for maintaining the breeding capabilities of the affected animal. Orchitis can be primary (neoplastic, bacterial), secondary to other diseases, or originate from adjacent organ systems. A variety of bacteria can cause avian orchitis, including *Escherichia coli*, *Salmonella* spp., and *Pasteurella multocida* [3]. A common cause of orchitis, in sexually active males, is *Chlamyphila psittaci*. Orchitis or epididymitis may follow a general psittacosis infection [25]. Infection can originate from cloacitis, prolapsed phalli, or septicemia [3]. In most cases bacterial orchitis can lead to permanent sterility [25]. Diagnosis is not easy, because the early signs of disease are often vague, and may be similar to those expected for many other conditions [3,26]. One of the signs that often leads to a diagnosis of orchitis is infertility, but this applies only to breeding birds. Pet animals may also suffer from this disease, but typically presents as a generalized infection [26].

Often, the diagnosis of orchitis is derived from an endoscopic examination, either because a breeding pair is underproducing, or because there are other indications for celioscopy as a proper diagnosis of a symptomatic bird, which cannot be reached by other means. A confirmed diagnosis is often achieved with a testicular biopsy [24,27] (Fig. 8). Treatment depends on etiology, and the primary cause should be addressed whenever possible. Aggressive antibiotic treatment is generally indicated in cases of bacterial infection, but often does not result in a functional recovery of the testicle(s).
Testicular neoplasms

Testicular tumors have been frequently observed in budgerigars (Melopsittacus undulatus), but have been sporadically seen in other bird species. Although a large percent of these tumors have not been diagnosed histologically, some specific neoplasms that have been conformed with histopathology include: seminomas, Sertoli cell tumors, Leydig cell tumors, tubular adenomas, teratocarcinomas, carcinomatoid embryomas, anaplastic tumors, sarcomas, and spindle cell sarcomas [28–32].

Testicular tumors may cause signs similar to those seen in case of a renal mass. Sertoli cell tumors may be responsible for the secretion of estrogen-like substances, causing alterations related to the neoplasm-induced endocrine imbalance [33]. A possible sign of a Sertoli cell tumor, is the development of the estrogenic bone, which is typical of the laying female and can have diagnostic value. A definitive diagnosis is often made on postmortem examination. In the living patient, a diagnosis can be reached by testicular biopsy [34].

Treatment, if the neoplasm is diagnosed early, can be surgical, and includes orchidectomy [26]. In the author’s experience, a palliative treatment and a presurgical measure is megestrole acetate, administered orally at 1–3 mg/kg, orally, every 24 hours, for 7–10 days before surgery. This will reduce
testicular (and tumor) size and limit the blood supply to the organ, making surgery easier.

**Testicular degeneration**

Secondary forms of testis degeneration, due to malnutrition, bacteriemia, vitamin E deficiency, or toxins (such as lead and cadmium), have been reported in some bird species. Spermatogenesis ceases, and if a severe fibrotic process develops, it is considered permanent [6,35].

**Phallus prolapse**

This condition is obviously linked to the presence of a phallus, so that a phallus prolapse is generally observed in Anseriformes and Ratites [1]. In the first group, the cause is generally traumatic: the erected phallus is traumatized when birds try to copulate on the ground. This happens when duck-like birds are kept in the absence of water [36]. The traumatized phallus becomes infected, and the bird is unable to retract it into its cloacal pouch. Bacterial, mycoplasmal infections, and extreme weather fluctuations may also play a role in phallic prolapse [26]. *Neisseria* spp. have been isolated from erosions of the phallus, cloaca, and oviduct of geese, and are believed to be sexually transmitted [3].

In Ratites, especially the ostrich, the primary cause is also attributed to trauma. The primary lesion depends on the length of time the phallus is exposed, which is common in the active male ostrich. The exposed phallus is traumatized when the animal lays down and displays or “dances” in front of the female. In addition, debilitation at the end of the breeding season and temperatures of 0°C, in conjunction with high humidity, have also been advocated as possible causes [37].

Treatment is based on disinfecting and daily flushing with chlorohexidine solution, application of antibiotic ointments, and/or Dimethyl Sulfoxide and systemic treatment with broad-spectrum antibiotics and antimycoplasmal drugs. Whenever necessary, the necrotic debris should be removed and the organ kept in situ with a purse-string suture. Rarely phallus amputation is necessary, and this is acceptable only if the animal is not intended for breeding purposes.

**Female reproductive disorders**

**Oophoritis**

The status of the ovary in the adult female can be an indicator of the general health of the animal. Infections of the ovary are relatively common and might be caused by bacteria, *mycoplasma, Chlamyphila*, or virus. Furthermore, oophoritis often evolves into peritonitis. Clinical signs are not specific and can include depression, anorexia, weight loss, distended abdomen, or
sudden death [38]. They may be chronic (depression, loss of appetite, and progressive wasting), acute, or even peracute, with sudden death. Therefore, diagnosing this disease can be challenging, and may include radiology, endoscopy, hematology, and bacterial and fungal cultures from the cloaca. Antibacterial therapy must be aggressive (ceftriaxone sodium, cefotaxime sodium, enrofloxacin) and combined with hormonal therapy, to decrease ovarian activity (megestrole acetate, leuprolide) [1].

Salpingitis/metritis

The term salpingitis indicates an inflammatory process involving the proximal portion of the oviduct (infundibulum, magnum, and isthmus), while metritis relates to an inflammatory lesion of the distal oviduct (uterus). Both salpingitis and metritis are often bacterial in origin, and frequently produce an oviductal impaction. Although metritis can occur as an ascending infection from cloacitis, or a consequence of a previous dystocia, salpingitis may be secondary to pneumonia, aerosacculitis, or septicemia/bacteremia [1,38]. Microorganisms causing salpingitis/metritis include: Enterobacteriaceae (E. coli, Klebsiella, or Salmonella spp.), Pasteurellaceae, Staphylococcus, or Streptococcus and Mycoplasma or Chlamydophila. In addition, viruses such as Newcastle Disease virus (Paramyxovirus-1), Infectious Bronchitis Virus, and the Avian Adenovirus Salpingitis (AAVS) are known to cause salpingitis in adult birds [38]. Clinicals signs are vague and include abdominal distension, tenesmus, depression, weight loss, infertility, and abnormally shaped or abnormally colored eggs. Diagnosis is generally based on clinical signs, hematology, radiology, and ultrasound. This is one of the few cases in which a laparoscopy may be contraindicated, due to abdominal distention. In the larger species it is possible to perform an ascending cloacoscopy to visualize this condition. Treatment is based on broad-spectrum antibiotics and general supportive therapy. Oviduct flushing is possible in the larger birds such as Ratites, but if the bird does not improve quickly, salpingohysterectomy may be needed [1,39].

Ectopic eggs

The presence of eggs in the coelomic cavity may depend on several factors, including uterine rupture or reverse peristalsis of the oviduct [39]. Reverse peristalsis can be caused by stress, nutritional imbalances, obstructions, or salpingitis [38,39]. Diagnosis is aided by palpation, radiology, or ultrasonography. Ectopic eggs must be surgically removed.

Egg yolk peritonitis

Whatever the cause, the presence of ectopic eggs often evolves into egg peritonitis. Although egg peritonitis may be an aseptic condition, similar to a foreign body reaction, the majority of egg peritonitis cases are septic
and may result in septicemia [1]. It is important to distinguish whether only egg yolk from an ovulated ovum is present, or if there is also albumen or infectious material from the oviduct. Even sterile albumen can cause a more severe reaction than the yolk alone. Among pet birds, budgerigars (Melopsittacus undulatus), cockatiels (Nymphicus hollandicus), and lovebirds (Agapornis spp.) are more often affected than other species. Patients are depressed, loose weight, are dyspneic (or display labored breathing), and show a dilated abdomen. Ascites and abnormal postures might also be present. Hematology often shows leucocytosis (>30,000/μL) with a relative heterophilia and biochemistry may show hypercholesterolemia, hyperglobulinemia, and hypercalcemia [38,39]. A tentative diagnosis can be based upon the clinical presentation, but it should be confirmed by radiology and/or ultrasound. Cytology of the coelomic contents, often differentiates between septic and nonseptic inflammation [1,38]. Treatment depends on the severity of clinical signs, but does not vary from treatment for salpingitis/metritis [38]. In addition, it might be necessary to surgically remove the egg material from the coelomic cavity. Coelomocentesis may be helpful in unstable patients with a severe ascites to improve respiration [39].

Egg retention and dystocia

Both of these conditions, often linked together, are among the most frequent obstetric disorders of birds. They are also the most frequent avian emergencies. Egg retention may be described as an egg that is not laid within the normal time [1]. Egg retention is a frequent complication in laying birds. The normal transit time through the oviduct varies and depends on the species; 24 hours in the domestic chicken, 48 hours in Psittaciformes, and about 44 days in the Brown kiwi (Apteryx australis). Dystocia can be described as the mechanical obstruction of the egg passage in the caudal part of the oviduct (uterus and vagina) [38]. Causes of egg retention are multifactorial, and involve oversized or misshapen eggs, thin eggshell, excessive egg-laying calcium deficiency, Vitamin A, E, and selenium deficiency abdominal hernia, dysfunction of the oviduct, obesity, torsion of the uterus, neoplasia, infectious diseases of the oviduct, and genetic predisposition [1,38,39]. Small bird species, like canaries, finches, and budgerigars, are most commonly affected [39]. Diagnosis is complicated by the variation of laying of two subsequent eggs in the different avian species, and within different individuals. Therefore, it is difficult to distinguish between normal and disease [1]. The diagnosis of egg retention should be based on clinical signs, physical examination, radiography, and/or ultrasonography. Suspicion should arise if the clinician is presented with a female patient, with distended coelom and tenesmus. Other signs might include lameness, leg paralysis, or respiratory difficulty [39]. The presence of an egg should be ascertained by radiographs or ultrasound, as coelomic palpation is not sensitive enough. In the case of egg retention with a soft egg with noncalcified eggshell ultrasound is the preferred diagnostic tool [1].
Therapy depends on the physical status of the patient and the severity of the clinical signs [39]. Patients that are still able to pass feces and urine should be given antibiotics, fluids, vitamin E/selenium, Vitamin D, and calcium gluconate (40 mg/kg body weight). Calcium gluconate is given by slow intravenous injection or diluted with sterile saline and injected intramuscularly in small amounts and several sites [40]. The birds should be placed in a warmed and humidified cage [1]. In minimally depressed birds, the egg will pass without further treatment [3]. To promote oviduct contractions Prostaglandin E2-gel (0.2 mg/kg body weight) (Prepidil gel; Upjohn Co, Kalamazoo, MI; Sigma Chemical Co., St.Louis, MO), can be inserted in the cloaca, at the uterovaginal sphincter level [38]. This normally promotes egg expulsion within 15–20 minutes of application, if the egg is not adhered to the oviduct mucosa. It is important to remember that PGF$_{2\alpha}$ does not relax the uterovaginal sphincter and can produce serious side effects, including hypertension, uterine rupture, bronchoconstriction, and generalized smooth muscles stimulation [1,38]. Oxytocin (0.2–2 IU/bird) has no relaxing effect on the uterovaginal sphincter, and should be used only when it can be ascertained that there is no adhesion between the eggshell and the oviductal mucosa.

In birds that are not able to pass feces and urine, and run the risk of kidney damage and intestinal impaction, more aggressive therapy should be considered. After supportive therapy with fluids, calcium, and antibiotics, ovoce- nesis should be performed, through the cloaca or by transcoelomic access [39]. If the condition of the patient will allow, the bird should be anesthetized, otherwise ovoce- nesis has to be performed in the conscious patient. After removing the egg contents with a 16–18-gauge needle, the eggshell can be broken with a gentle laterolateral compression and the bird returned to a warm and humidified cage. Lubricating the cloaca helps the broken eggshell to pass [1]. The shell fragments should pass within a few days. In some cases, such as eggshell fragments are not delivered, or the removal through the cloaca is not possible, or if the adhesions between the egg and the uterus are severe, a coeliotomy might be necessary [3]. To prevent subsequent egg laying, which is extremely risky for the recently egg-bound hen, megestrol acetate (1–3 mg/kg body weight, orally, Solum In Die [once a day] (SID)) can be given. The formerly used medroxyprogesterone may have serious side effects, and is not recommended [1,41].

**Oviduct prolapse**

This is probably the second most common avian emergency. The uterus is the most distal portion and thus the one to prolapse most commonly [1]. This condition occurs secondary to normal physiologic egg laying or as a sequela to dystocia [3]. Predisposing factors for a prolapsed uterus include malnutrition, salpingitis, cloacitis, and soft shelled or otherwise abnormal eggs [39]. Treatment must be aggressive and include fluids and antibiotics. As soon as the condition of the patient allows it, the prolapsed uterus must
be cleaned and, if possible, reduced [1]. Addition of a sterile 40–50% glucose solution to a lubricant might help to reduce edema and facilitate replacement of the prolapse [38]. Different kinds of suture can be used to keep the prolapsed uterus temporarily in situ. It is very important to confirm the capability of the patient to pass stools [1].

**Chronic egg laying**

This disorder, relatively common in budgerigars, lovebirds, and cockatiels, can be observed in other bird species. Chronic egg laying is more common in hand reared females, especially if imprinted and single. Predisposing factors also include an unbalanced diet, psychogenic factors, genetic problems, inadequate environmental management, and anything that alters the normal hormonal balance [1,26]. In the aforementioned psittacine species, the process of domestication is reported as another possible reason for chronic egg laying [38]. The problem itself is not serious, but overtime can lead to dangerous mineral imbalances, especially related to calcium metabolism [1,3,39].

Treatment should be based on different steps, which involve:

1. **Behavior**: laid eggs should be left in the nest to avoid the “double clutch phenomenon.” Remove all possible sexual stimuli like toys and mirrors [3,26]
2. **Environmental changes**, including the removal of the nest, if it is empty. The cage should be moved to another, unfamiliar environment, and the photoperiod should be decreased to a maximum of 8–10 hours a day [3,26]
3. **Nutritional changes** should first focus on an adequate supply of calcium and vitamins to avoid nutritional imbalances. In budgerigars and cockatiels limiting their access to water for a couple of hours in the morning and in the afternoon, for a period of 3 or 4 days might interrupt the egg laying. But this should be done only under an intensive medical control [3,38,39]
4. **Pharmacologic management** can produce varying results. The most commonly used drugs are:
   - **Medroxyprogesterone acetate**: suppresses ovarian activity for a time ranging between 2 weeks and 2 months. Reported dosing is from 0.05 mg/kg body weight for birds up to 150 g body weight, and 0.025 mg/kg body weight for birds over 700 g body weight. However, it is no longer recommended, due to the potential side effects.
   - **Megestrole acetate**: has a similar action to the former drug, but it is eliminated more rapidly, from the organism, so that if undesired side effects occur, it can be withdrawn and its effects will disappear. Dosing is 1–3 mg/kg body weight, orally, SID, for the first week, than the frequency is decreased.
Testosterone: no longer used because of its side effects.

Human Chorionic Gonadotrophin: this product is a mixture of several hormones and it is thought that its LH (alpha-subunit) content acts as the active compound. Human chorionic gonadotrophin inhibits egg laying by stimulation of progesterone secretion, which directly influences egg production [38]. Reported dosing for chronic egg laying is 500–1000 IU/kg body weight [42], on days 1, 3, 7, 14, and 21, then at 2-week intervals.

Leuprolide acetate: is a “depot” synthetic gonadorelin (GnRH analog). It is used in human medicine for the treatment of reproductive tract neoplasia because of its “paradox” effect. Leuprolide first stimulates the hypophysis, but later inhibits the production of LH and FSH. It is used in avian medicine for its capacity to stop ovulation. Dosing is not yet standardized, but some protocols exist. According to some authors, total dose can be calculated using the following formula: 1000 lµg/kg body weight, intramuscularly, multiplied by the desired number of days of effectiveness [43]. For example: Amazona aestiva, 450 g/body weight. Desired treatment: 4 weeks; total dose intramuscularly: 0.45 kg × 1000 lµg × 28 days = 12,600 lµg. Some others report dosages from 100 to 1000 lµg/kg body weight, every 2–4 weeks. A protocol for this drug is currently being investigated at Utrecht University.

5. Surgical treatment: the only possible treatment is salpingohysterectomy.

Cystic hyperplasia of the oviduct

This disease is frequently observed in the budgerigar and domestic fowl but has been reported in other psittacine species [3]. It is characterized by the presence of white to brown mucous exudate, and isolated masses of white caseous matter in the oviduct, that results so dilated [1]. Because the ovary in affected hens might also show cystic changes, hormonal abnormalities are suspected [3]. Clinically, a distended abdomen, dyspnea, and ascites are observed. Because conservative treatment with antibiotics often fails, a salpingohysterectomy might be indicated [38].

Cystic ovary

The etiology of this disease is unknown, but a hormonal disturbance seems likely. The syndrome is mostly observed in cockatiels and less frequently in pheasants, budgerigars, and cockatoos. Symptoms might include coelomic distention, resulting from large cysts and, in breeding birds, an increased number of copulations without egg laying. In those birds radiographs often show hyperostosis. Cystic ovarian disease is often observed during a routine coelioscopy in asymptomatic birds [38,39].
Treatment includes surgical resection or transcoelomic aspiration of the cyst. Hormonal treatments with leuprolide acetate (Lupron Depot, TAP Pharmaceuticals, Inc, Deerfield, IL) were successful in 76% of 18 cases [44]. Treatment with Human Chorionic Gonadotrophin (500–1000 IU/kg body weight) might be useful [38]. The breeding performance of those hens should be regarded as suboptimal [39].

_Torsion of the uterus_

Torsion of the uterus is often diagnosed during diagnostic coeliotomy [39]. This condition is most likely caused by peritonitis [38]. In acute cases, replacement of the uterus might be possible, but often a salpingohysterectomy is required due to severe vascular compromise [39].

_Neoplasia_

In birds, spontaneous tumors of the ovary and, less frequently, of the oviduct, are fairly common. The highest tumor rate is reported in the budgerigar [29–31,33]. The most commonly reported female tumors are adenocarcinomas of the ovary, leiomyomas of the mesovarium, and adenocarcinomas of the oviduct [38]. Further, there are reports of cloacal carcinomas. All of these neoplasms have a variety of symptoms, but generally coelomic distension, dyspnea, and ascites are present. Diagnosis is made by biopsy, but often, sexual tumors are diagnosed at necropsy. Treatment is surgical [1].

_Disorders involving both sexes_

The final aspect of the disorders affecting the avian reproductive tract, are the diseases of both the male and female bird. Some of these disorders might be infectious and are eventually able to cause disease in various parts of the reproductive tract. Others can be referred to as “sexual disorders,” as they may be simple management problems that affect the breeding performance of one or more bird pairs.

_Generalized diseases_

It is of primary importance, in the case of poor breeding performance, to carefully analyze the general health status of both the hen and the cock birds. Any systemic disease may limit fertility or inhibit normal sexual activity. It is also important to take into consideration previous diseases (ie, psittacosis), which might have induced permanent fertility problems. In the authors’ experience, testicular biopsy often diagnoses postinfectious chronic orchitis [34].
Cloacitis

All the disorders involving the cloaca can lead to fertility problems. Therefore, the rough examination of the cloaca is indicated at presentation. Cloacitis can result from infectious as well as noninfectious causes, and can result in secondary urogenital diseases [26]. The most common causes of cloacitis include bacterial infections, mycotic infections, neoplasia, cloacolith, prolapses, and excessive fat deposits around the vent. Treatment of cloacitis is based on identification of the specific cause, that is, culturing the involved infectious agents. General and local antibiotic treatment may be indicated, as well as careful cleaning and lubrication of the cloaca and vent to avoid fecal accumulation.

Papillomatous disease

Although this disease has been reported in many psittacine species, it is most commonly found in the genera *Ara*, *Amazona*, and *Deroptyus*, and in other South American parrots. A papillomavirus has never been isolated from affected birds, but a link with a herpesvirus, possibly related to Pacheco disease virus, has been made. Additionally, a significant percent of the affected parrots develop bile duct and pancreatic malignancies [45].

The affected cloaca is often infected with secondary invaders such as bacteria and fungi. This represents a physical distress for the animal, which is less prone to copulate. Secondary bacterial and fungal infections lead to a change in the normal cloacal pH and bacterial flora, which may limit spermatozoa vitality. Therefore, although birds with cloacal papillomas may not necessarily be infertile, this disease is among the most frequent causes of reduced fertility in Psittacines [1].

Diagnosis is based on clinical observation of the typical lesions (Fig. 9). Many treatments have been reported, including chemical burning with silver nitrate of focal lesions, cryosurgery, radiosurgery, laser, and autogenous vaccines, but no treatment guarantees a complete cure of cloacal papillomatosis [36].

Cloacolithiasis

Cloacoliths are not frequently observed in birds. They may result from previous egg binding, cloacitis, or neurologic alterations of the cloaca. Treatment can be either medical or surgical, but the long-term prognosis is often guarded, as the primary cause cannot always be identified and cured [26].

Behavioral alteration

Birds respond to complex sexual stimuli that involve important behavioral patterns, before, during, and after copulation. Any alteration of normal sexual behavior may be considered as a sexual disease, as it may result in a
lack of normal breeding. It is important to understand the normal sexual behavior of any given species in order to recognize behavioral alterations. These can include:

- a bird’s inability to recognize itself as belonging to the proper species;
- homosexuality;
- aggressive behavior towards the partner;
- aggressive behavior towards the eggs and/or offspring.

**Locomotion disorders**

An inability to move properly can limit the breeding performance of a bird greatly. This may depend on:

- an inappropriate enclosure—one that the bird does not feel comfortable in, and is unable to move properly or sufficiently;
- a previous disease or mutilating surgeries, that physically inhibit the animal’s movements.

The following situations may represent a limiting factor for breeding, and in the extreme cases they have to be considered as primary infertility causes:

- lameness;
- missing limb;

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Fig. 9. Buffon’s macaw (*Ara ambigua*). Cloacal papilloma.
• inability to fly;
• impaired vision;
• balance alterations.

Incorrect sex determination

This is obviously not a disease, but it is important to take into consideration that a bird was perhaps not identified properly, especially when faced with reproductive failures in monomorphic species. In many bird species the gender can be determined by different means: different color (most ducks, pheasants, grouses), different size (birds of prey), different call (most finches), or different behavior (pigeons). Unfortunately, the most commonly presented avian patients (ie, psittaciformes), do not show any of these differences, or display them in subtle ways, which makes sex determination difficult and doubtful. Although several techniques have historically been used to determine the gender of a bird, nowadays this is almost exclusively done by endoscopic examination and DNA analysis. Generally speaking, endoscopy offers an immediate and accurate sex determination, and allows for the evaluation of the gross appearance of many intracoelomatic organs. However, DNA sexing is considered safer, but takes a longer time to produce a result. Also, DNA techniques also allow the opportunity to screen birds for some relevant diseases, such as Psittacosis, Psittacine Beak and Feather Disease Virus (PBFDV) and Polyomavirus, with the same blood collected for the determination of the gender. Endoscopy is greatly preferred by the authors, for the determination of gender, but one key point is the experience and equipment of the operator. In the hands of an experienced avian endoscopist, gender is safely and accurately determined and, if the veterinarian can use one of the most advanced equipment (ie, the Storz rigid system, with a operating channel sheath; Karl Storz GmbH, Tuttlingen, Germany), he/she will also have the chance to take biopsy samples during the procedure.

Inappropriate management

Any situation that limits the normal behavior and the natural activity of a bird, can cause, or contribute to breeding failure:

• improperly built aviary/cage (size, shape, light exposure, material, presence of disturbing factors);
• inappropriate nest (size, shape, location, material);
• inappropriate perches (too slender, too slippery, too unbalanced);
• inappropriate feeders and drinkers;
• inappropriate diet;
• poor hygiene;
• poor health evaluation.
Summary

Disorders of the reproductive system represent a large portion of both large and small domestic animal medicine. Although some disorders of this system have been extensively studied in birds, this science is still in its infancy, when compared to mammalian reproductive medicine.

This may be due to several reasons, but the simple fact that birds are oviparous renders knowledge of mammalian reproductive anatomy, histology, physiology, and disease process, inapplicable to avian patients. Nevertheless, several specific diseases or conditions affecting the reproductive system of birds have been described.

By integrating information about the comparative anatomy and physiology of birds, reports of the most common reproductive diseases of birds and utilizing the latest diagnostic techniques, especially videoendoscopy, the avian practitioner should be able to diagnose and properly treat a high percentage of the avian reproductive diseases.

References