CHAPTER
39

Management of Canaries, Finches and Mynahs

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Johan Van der Maelen

Fig 39.1 | A white recessive color canary is shown.

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Fig 39.2 | A red color canary is shown.

The order Passeriformes (Table 39.1) is the largest of all avian orders and consists of 63 families comprising 5206 different species. The order is extremely heterogeneous, containing granivorous, insectivorous, frugivorous, omnivorous and carnivorous species ranging in size from a few grams to over 1 kilogram.

The species commonly seen in private veterinary practice include canaries, New World finches, Old World finches, waxbills, cardinals and mynahs (see Table 39.1).

The canary (*Serinus canarius*) can be considered a domesticated species. Spanish monks in monasteries as far back as 1402 achieved first breeding. Following a French expedition to the Canary Islands, this bird was introduced to France and Italy and later into the rest of Europe.[17] Nowadays, canary fanciers have a wide range of activities, including preservation of old and rare breeds as well as breeding new color mutations.[32] An important aspect of their hobby is showing and judging their birds. There are three groups of canaries: song canaries, color canaries and form canaries.

Song canaries include Harzer (Germany), Malinois (Belgium), Timbrado (Spain) or American singers (USA). Color canaries are divided into two groups. The melanin group includes black, brown, agate and isabel birds. The lipochrome group includes white (Fig 39.1), red (Fig 39.2) and yellow (Fig 39.3) canaries.

Form canaries are a diverse group, distributed among "frill," "type," "shape," "crested" (and "crest-bred") and "feather pattern" birds. Frills include Parisian frill, Frisé du nord, Frisé du sud, Frisé Suisse, Gibber Italicus, Giboso Espagnol, Padovano and Fiorino (Fig 39.4). Type


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Fig 39.3 | A yellow color canary is shown.

canaries include Belgian bult, Scotch fancy, Münchener, Japan hoso, Lancashire, Yorkshire, Berner and Rheinländer. Shape canaries include border, Fife fancy, Norwich, Raza Espagnol and Irish fancy. Crested and crest-bred birds include Gloster consort, Gloster corona and German crest. The Lady Gouldian Finch is a popular finch amongst fanciers (Fig 39.5).

Anatomy and Physiology

Some anatomic and physiologic characteristics observed in canaries and finches include an extremely high basal metabolic rate and high body temperature (42° C, 108° F). The foot of the passerine bird is anisodactylous: 3 digits forward and 1 digit backward.[105] All passerine species have a crop, but in canaries and finches, the crop is much smaller than in psittacines, pigeons or chickens. There is no production of crop milk, but crop contents are regurgitated to feed the young. A proventriculus and ventriculus are present; the cecae are rudimentary. The spleen is oblong rather than spherical. Nestling Estrildidae finches have species-specific luminous mouth patterns to attract their feeding parents.[71] The right and left nasal sinuses do not communicate as they do in Psittaciformes. Song canaries and other species are able to produce two sounds simultaneously by using both bronchial ends of the syrinx. The neopulmo as well as the paleopulmo divisions of the lungs are well developed in Passeriformes. Most Passeriformes have seven air sacs as opposed to the nine air sacs in Psittaciformes. The cranial thoracic air sacs are fused to a single air sac, as are the clavicular air sacs.[71]


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Fig 39.4 | A form canary, Parisian frill, is shown.


Greg J. Harrison

Fig 39.5 | Lady Gouldian finches are dimorphic with the male in front showing the most and brightest color. The female in the background has less color especially on the head.

Table 39.1 | Selected Passeriformes Families

Family	Scientific name	English name	German name
Corvidae		Crow, jay, magpie	Krähen
Sturnidae		Starling	Stare
	Gracula religiosa	Hill mynah	Beo
Passeridae		Sparrow	Sperlinge
Ploceidae		Weaver	Webervögel
Estrildidae		Waxbill	Prachtfinken
	Poephila guttata	Zebra finch	Zebrafink
	Chloebia gouldiae	Lady Gouldian finch	Gouldsamadine
	Erythrura spp.	Parrot finch	Papageienamadine
	Lonchura striata var. domestica	Society or Bengalese finch	Japanisches Mövchen
Fringillidae		Finch	
	Serinus canarius	Canary	Kanarienvogel
	Carduelis spinus	Siskin	Zeisig
	Carduelis carduelis	Goldfinch	Stieglitz
	Carduelis chloris	Greenfinch	Grünfink
	Fringilla coelebs	Chaffinch	Buchfink
	Pyrrhula pyrrhula	Bullfinch	Gimpel
Emberizidae	*Melopyrrha nigra*	Cuban finch	Kubafink
	Cardinalis	American Cardinal	Kardinale

Reproduction[19]

BREEDING FACILITIES

Nowadays, canaries are mostly bred indoors, often within a building in the garden or close to the house (Fig 39.6), sometimes inside the house in a cellar or an attic. The birds are kept in pairs in breeding cages (50 x 40 x 40 cm) and require artificial lighting. In a traditional European facility, each cage would have drinking water, seed mixture, soft food, cuttlefish bone, grit, nesting material, conditioning seeds and bath water that is changed on a regular basis. The most suitable substrates for the cage bottom are newspaper, plain brown paper or small pieces of wood. Some breeders use cages with wire bottoms. Wire is easy to clean but can be dangerous for the youngsters sitting on the bottom of the cage as entanglement of the feet may occur.

Some breeding facilities have cages with nest boxes inside the breeding cage; others have breeding cages with the nest boxes outside (Figs 39.7-39.9). In order to collect or candle the eggs and leg band the youngsters, it is easier to have outside hanging nest boxes. Additionally, breeders have found that leaving a clutch with the parents until weaning leads to fewer babies produced each year. If they try to let the parents breed with the babies present, the subsequent eggs may be infertile because the babies interfere with the breeding process in some way. The parents may be disturbed and not incubate the eggs properly or the parents may abuse and pluck the youngsters, trying to drive them away. To overcome this, the babies are maintained in accessible but separate enclosures. Where outside hanging "baby cages" are used, the youngsters are fed through the wire. The youngsters of the first clutch are put in a cage next to the parents, separated by a wall containing 2-cm-diameter openings (Fig 39.10). The parents can prepare for the next round of eggs and babies more successfully, while continuing to feed the first clutch through the holes until they are completely weaned.

After weaning, the youngsters are put together in large cages so they can exercise their flight (Fig 39.11). Individual perches are very important to prevent picking. Often an older male is placed together with the youngsters to feed those that still beg for food.

BREEDING PERIOD

In Europe most breeders artificially extend the daily photoperiod. The reasons for using this technique of light manipulation are: 1) to control the breeding period and start reproduction before the natural breeding season (so most intensive work is done before July summer holidays); 2) to prepare the youngsters for the exhibitions at

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Fig 39.6 | A canary breeding facility with netting on the windows helps prevent mosquito problems.

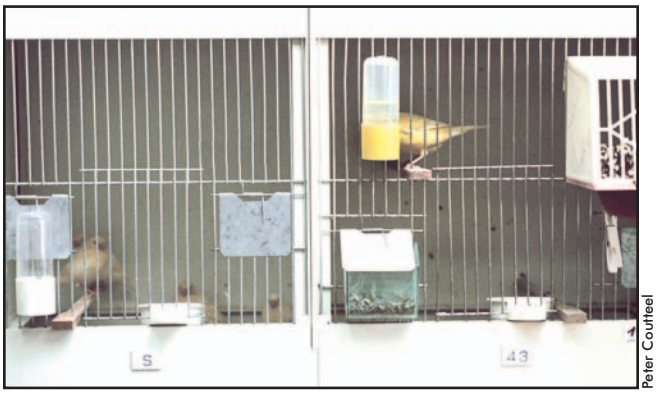

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Fig 39.7 | Breeding cage separates youngsters of the first clutch in a baby cage (left) from the breeding pair preparing the second round (right).

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Fig 39.8 | Between every two breeding cages (with inside hanging nest pans), there is a common baby cage for the youngsters in a canary breeding facility.

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Fig 39.9 | A nest box, outside the main cage, makes access easier.


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Fig 39.10 | Youngsters are fed by their parents on the other side through little holes in the separation wall of the cages.

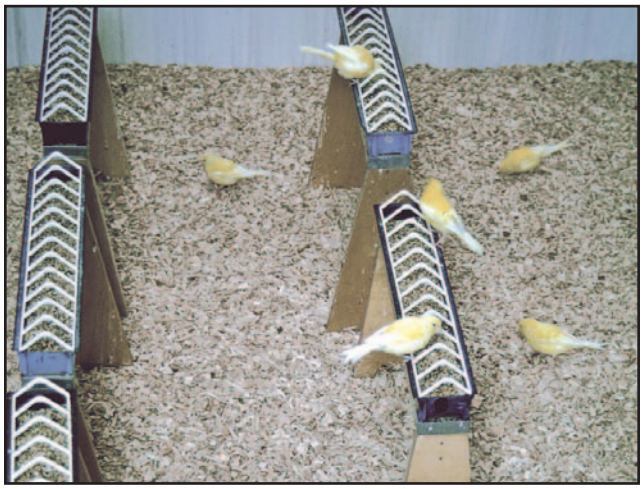
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Fig 39.11 | Young canaries are housed in a large cage for flight exercise. Often an older male is placed together with the youngsters to feed them. The substrate is small pieces of wood.

the end of the year (molt period has to be completed prior to exhibition); and 3) to produce more clutches and youngsters. (*Ed. Note: These techniques are used to improve profit and may not be ideal for the parent birds' over-all health).*

TECHNICAL ASPECTS OF LIGHT

The spectrum of optical radiation lies between 10^2 nm in the ultraviolet (UV) and 10^6 nm in the infrared (IR) spectrums. This wavelength range is subdivided into seven bands: UV-C, UV-B, UV-A, visible light, IR-A, IR-B and IR-C. The UV-B band in natural sunlight allows for the multistep conversion of 7-dehydrocholesterol into 1,25 dihydrocholecalciferol (vitamin D_3), which is then reabsorbed by the skin or ingested during preening.[98] Vitamin D_3 is very important for calcium metabolism. If there is a lack of incoming sunlight, full spectrum lamps can be used to induce the internal vitamin D production in the skin. Also, environmental contamination can be reduced by UV light. The action spectrum for inactivation of microorganisms reaches its maximum at about 265 nm (DNA absorption). If artificial light is provided for breeding birds, the kind of radiation produced by the lamps is an important factor.

Luminance

A minimum luminance (500 to 1000 lux) is needed (lux = lumen/m^2). Lumen gives an indication of the total amount of light produced by a light source per second. Adding the lumen of all lamps present in the breeding room and dividing it by the square meters of the room will give an idea of the quantity of lux. Sunlight produces approximately 100,000 lux.

Frequency

Normal fluorescent lamps do not give continuous light, but flicker like a stroboscope at approximately 50 times per second (50 Hz). The human eye does not discern this frequency and the effect on the behavior of birds is not completely known. However, recent studies reveal that birds may have a spatial difference of 160 frames per second.[63]

The stroboscopic effect of fluorescent light may lead to stress and may negatively influence the general condition of the bird. If many lamps are used at the same time or a combination of bulb lamps and fluorescent light is used, the stroboscopic effects will be less marked. The latest development is the HF (high frequency) lamp. These lamps have a frequency of 28,000 Hz, have a longer life and make dimming possible. When using artificial light, a dimmer should be used to simulate dawn and twilight. In Belgium, 92% of the breeders are using fluorescent lamps and 8% are using bulb lamps.[18]

Color Temperature

Color temperature is the measure for descriptions of "warm" or "cold" light and is expressed in degrees Kelvin. Examples of color temperature include incandescent light (2700° K), warm white (3000° K), cool white (4000° K), daylight (5000° K), and cool daylight (6500° K). The higher the color temperature of the light (higher degrees Kelvin), the more blue the color of the light and the more UV light produced. Cool daylight[b] is ideal.

Ambient Light Temperature

Fluorescent lamps give the highest light output at a temperature of 20° C (68° F). These lamps are very sensitive to temperature. At a low ambient temperature, the buffer gases inside the lamp will disintegrate and there will be

less light output. Therefore, the use of multiple lamps simultaneously is recommended, in case of defects.

Nightlights

A small amount of light (7 W) during the night has a calming effect. If a bird is startled or otherwise disturbed, this lighting will allow it to regain its perch.

Photoperiodic Stimulation

The photoperiod is the ratio of light to dark. A 15:9 ratio means 15 hours of light and 9 hours of darkness. The photoperiod is very important in those species of birds that originate from zones with seasonal changes. In temperate-zone birds such as canaries, ovarian development and testicular growth are synchronized by a combination of increasing day length, the presence of a partner and an available nesting site.[93]

The normal physiologic pathway for photoperiod stimulation has been described.[93] Retinal photoreceptors give stimulation through the optic nerve to the hypothalamus. Releasing hormones from the hypothalamus stimulates the adenohypophysis to produce gonadotropins, which, in turn, stimulate the gonads to release sex hormones. Another factor that can stimulate the hypothalamus is the presence of light rays that penetrate the spongy bones of the head.[93]

These stimulating factors encourage testicular growth in the male bird (up to five times in size) and production of fertile sperm. In the female bird, strong development of the ovaries and follicular growth occur. Experience shows that male canaries need a slightly longer exposure to extended daylight than females. This can be obtained by placing the males in isolation 2 weeks in advance, so that light stimulation may begin earlier. The reason that males need a longer preparation period is probably due to the fact that, in nature, males receive extra incentives, such as the song of another male while marking out their territories and trying to attract a partner. These incentives are often not present in an artificial breeding situation.

The Role of Daylight Length

Most problems in canary breeding are due to errors in the manipulation of the light cycle. Canaries need a minimum of 14 to 15 hours of daylight to begin breeding (nest building and production of eggs). With this amount of light, they are also able to feed their youngsters adequately and raise them properly. The cycle of light is also a major factor in determining whether breeding is sustained. If the length of daylight is submitted to fluctuations, the birds may receive conflicting hormonal incentives and negative endocrine feedback. The result can be an early molt and the birds may cease breeding. There are two methods of manipulating the length of daylight.[18]

Gradually Increasing Daylight Length:

Using this technique, the amount of daylight is gradually increased on a weekly basis. Depending on how quickly this is done, it may take a period of 2 months to extend the 8 to 10 hours of natural daylight to 15 hours. If a weekly addition of 30 minutes (5 additional minutes per day) is used, it will take approximately 10 weeks to obtain this result. This means that the fancier needs 2 to 3 months preparation before breeding can begin. Gradually increasing the length of the day is closest to natural stimuli and is used by more than 80% of the fanciers. Fifteen hours of daylight length appears to be ideal. Poor annual breeding results with higher chick mortality occur when the daylight length exceeds 17 hours.

Immediate Increase to Full Daylight Length:

The daylight length can also be increased suddenly from 10 to 15 hours. In this case, the birds reach breeding condition after 3 to 4 weeks, but most are unable to maintain good results throughout the full breeding season. However, some fanciers do have good results with this method. This method of sudden increase, used by approximately 10% of the breeders, often leads to poor fertilization of the first clutch, which normalizes subsequently, and higher mortality of females.

BREEDING CONDITIONS

Normally, canaries will begin breeding when the following conditions are fulfilled: photoperiod stimulation and appropriate minimum daylight length (as discussed), maturity, good health, an acceptable partner, presence of nest and nesting material and a minimum temperature of 15° C (59° F), during daytime.

Maturity

Most canaries will reach sexual maturity in a few months but good fertility will only start at approximately 10 months of age. Sometimes there are fertility problems when birds born in the last clutch of the former year are bred too early in the following year.

General Health

Many diseases and breeding failures in captive Passeriformes are the result of husbandry problems. Medication is often incorrectly used to attempt correction of husbandry-based problems. Primary infectious disease is less commonly encountered. The ideal avian veterinarian/aviculturist relationship starts before the breeding season when the birds are routinely examined. A physical examination is performed to determine whether each bird is in good condition. Examination includes the head (eyes, ears and nares), abdomen (liver, gastrointestinal tract),


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Fig 39.12 | A nesting material ("sharpie") of small white fibers of cotton is well accepted by canaries.


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Fig 39.13 | Small white fibers of cactus (*Agave rigidus*) are another source used for nesting material.

feathers (state of molt, external parasites and discolorations), wings (lacerations, feather cysts), feet and digits (hyperkeratosis, pox lesions and pododermatitis). Crop swabs are collected to determine if trichomoniasis or candidiasis is present. Fecal samples are taken for bacteriology, and blood samples are collected by venipuncture for serologic diagnosis of paramyxovirus and avian influenza. Finally, a fecal examination is performed to assess for coccidiosis, macrorhabdosis, helminth infections, yeast, protozoal cysts (*Giardia* sp.) and cochlosomiasis (diagnosis of cochlosomiasis requires fresh, warm stool). Sometimes a fecal Gram's stain is performed to interpret the levels of gram-positive and gram-negative bacteria and for the detection of *Candida* and *Macrorhabdus* spp.

Acceptable Partner

In canary breeding, there are generally few problems with the acceptance of the partner. Even during the breeding season, the female bird allows copulation with a different male for each round. If necessary, male birds can be used for several females simultaneously, with a maximum of three females for each male.

Nest and Nesting Material

Breeders use several different types of nest pans. Plastic nest pans have a very smooth surface that is easy to clean and makes movement of the nest possible. Often, holes are drilled in the bottom of the nest in order to attach the nest with wire. Stone nest pans can be well impregnated with a water-soluble insecticide. Sometimes self-made wooden or bamboo nest pans are used. They have the disadvantages of being difficult to clean and disinfect and must be changed after every clutch. Several types of nesting material are available. "Sharpie" consists of small white fibers of cotton, washed and cut in small pieces of approximately 3 to 4 cm (Fig 39.12). This nesting material is well accepted by canaries, and most Belgian breeders utilize it. Sisal fiber, of the plant *Agave rigidus*, is a very fine organic nesting material (Fig 39.13). It is important to note that constriction of a digit can occur due to entanglement in the fine fibers. If not treated, vascular necrosis and digit loss can result. Horsehair and moss are no longer used because they cannot be disinfected.

Temperature and Humidity

Most fanciers maintain the temperature in the breeding room at approximately 15° C (59° F) at the start of the breeding season. If the temperature is higher, the females start laying eggs even before pairing. Temperature is regulated with various heating devices (electrical, central heating, gas and fuel oil). During the breeding season and summer, the temperature may fluctuate. Therefore, good ventilation should be provided to remove exhaust gas and to eliminate temperature extremes. The temperatures should range from 15° to 25° C (59-77° F). In the authors' experience the temperature should not exceed 35° C (95° F).

The humidity in the breeding room should be kept within the range of 60 to 80%. Maintaining the humidity at the lower end of this range minimizes the development of pathogens. It is important to have sufficient humidity at the time of hatching. Therefore, breeders often moisturize the eggs just prior to hatching with a spray of warm tap water in the nest or by plunging the eggs for a second into a cup of warm water (40° C, 104° F).

PREPARATION FOR THE BREEDING PERIOD

Trimming

Before pairing birds, each bird should be physically

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Fig 39.14 | The pericloacal region of a canary (Gloster corona) is trimmed 1 cm ventral to the cloaca to prevent the feathers from interfering in semen transfer.

examined and eventually "trimmed". Because of the heavy plumage of some canary varieties (especially type canaries), it is preferable to trim the plumage surrounding the pericloacal region approximately 1 cm above the cloaca and horizontally prior to breeding (Fig 39.14). This reduces the potential for the caking of excrement around the vent and may increase the possibility of copulation. The nails should be checked and cut shorter if necessary so that good positioning is possible during copulation. Loose perches should be repaired prior to nail trimming. Trimming of the eye feathers can also be important so that the male can see the female. Sometimes there is an overgrowth of the upper beak, which can be trimmed with an electric grinding tool.[a] Traditionally, breeders used cuttlebone for that purpose, but recent nutritional experience shows the problem is more complex than just calcium deficiency and a lack of beak exercise. Nutritional assessment is important in birds with overgrown beaks.

Disinfecting

The breeding cages should be disinfected before introducing birds. No single disinfectant is active against all microorganisms or useful in all situations, so the chemical must be carefully chosen. Commonly used and readily available disinfectants include sodium hypochlorite (household bleach), quaternary ammonium products, chlorhexidine, glutaraldehyde and formalin.

Cleaning and disinfecting the breeding cages, perches and nest pans should be performed prior to introducing the birds. Also, prevention of red mites, northern mites and lice with an appropriate insecticide is advised. A common product utilized in Belgium is permethrin. Alternative pest control agents include insecticidal soaps used for plants, fresh garlic and diatomaceous earth. When using diatomaceous earth, the unpolished form (used for swimming pool filters) should be selected. When this form of diatomaceous earth is dusted onto the birds, the needle-like points penetrate the insect's exoskeleton causing desiccation. To reduce mosquitoes, mosquito netting or screened windows may be necessary. The carbon dioxide generator has advanced outdoor control. Female mosquitoes feed on blood, to which they are attracted by exhaled carbon dioxide. A new machine uses bottled gas to generate the CO_2 bait. The mosquitoes are then trapped in an inescapable collection bottle. If used as directed and combined with traditional preventive measures, mosquito population control is achieved.

Some canaries are housed outside in open aviaries, in which case, disease control and medicating individuals can be impractical. It is difficult to completely eliminate infectious agents once they are introduced into a planted aviary. Free-living birds and rodents may be transmitters of protozoal diseases, helminth infestations and bacterial infections.

PAIRING BIRDS

It is important to predetermine the sex of the individual birds in order to ensure they are paired appropriately. In males, the caudal end of the ductus deferens forms a mass called the seminal glomerulus. During the breeding season, the seminal glomerula push the cloaca walls into a "cloacal promontory" (Fig 39.15). Females do not develop this projection and have a flatter vent (Fig 39.16). The male is placed into the female's cage when the birds are ready for pairing. In the classic situation, one male and one female are together for the whole breeding season and rear the youngsters together. Alternatively, one high-quality cock may be used for pairing with several females. After copulation, the male is separated, and each female will rear her youngsters alone. Fertile sperm may be stored in the female's sperm glands (tubular glands within the oviductal wall located at the uterovaginal junction) and may fertilize eggs for approximately 8 to 10 days.[93]

The regulation of sexual behavior is altered by the effects of social interactions (eg, song of other male birds), which can exert powerful effects on reproductive success and overall well-being of the individual birds.[80]

Genetic Considerations

There should also be appropriate genetic selection of breeding birds regarding origin, color, type and feathers. Feather cysts are usually seen in the Frill, Norwich and other soft feather breeds and are believed to be a genetic disorder. Likewise, cataracts in canaries may have a genetic origin.[33]


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Fig 39.15 | A male canary is trimmed in the pericloacal region. The cloaca is perpendicular to the tail and points down, a "cloacal promontory".


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Fig 39.16 | A female canary is also trimmed in the pericloacal region, but the cloaca points in the same direction as the tail.

Other genetic abnormalities may cause mortality. One example of this is seen when pairing crested canaries such as the gloster (with crest = corona, without crest = consort).[71] The corona gloster is heterozygous for the autosomal crest gene. Birds that are homozygous for the crest gene die. Pairing a corona with a consort yields 50% corona and 50% consort. Pairing two corona phenotypes will yield 25% consort, 50% corona and 25% dead chicks. Another example of genetic mortality occurs with the dominant white color gene. Canaries that are homozygous for the white gene die,[71] while heterozygous birds are white. There is 100% mortality in chicks if two heterozygous dominant white birds are paired. If white birds are paired with other colors, 50% of the chicks will be heterozygous for the dominant white gene and 50% will be the other color.

EGG COLLECTION

During the egg-laying period, eggs are collected and replaced with artificial eggs while the female continues to lay her clutch. The eggs are kept in a cool place (15 to 20° C, 59 to 68° F, humidity 60 to 80%), in numbered boxes with seed at the bottom and put back into the nest when the female starts sitting. This is done to achieve uniform growth in the youngsters. The average clutch size of type canaries is 4 eggs. Color canaries often have a clutch size of 4 to 7 eggs. One should avoid writing on the eggs, especially with an alcohol pencil. Also, soft cloth or rubber gloves should be used while handling eggs in order to avoid contamination (eg, hand creams and cosmetics), which can result in the death of the chick.

Nutrition

Most passerines are primarily seed-eating birds. They may consume up to 30% of their body weight in food daily (compared with 10% for larger parrots). The basal metabolic rate is approximately 65% higher than in non-passerines. Granivorous birds are believed to need grit for digestion.[32]

The majority of commercially available seed mixtures are multideficient.[32] Seeds are deficient in vitamins A, D_3, E, K, lysine and methionine and have a poor Ca/P ratio. In Belgium, seed mixtures for canaries contain canary seed (62%), Niger seed (2%), rapeseed (22%), hemp seed (3%), peeled oats (8%) and linseed (3%). During the winter, 50% canary seed is mixed with this seed mixture to prevent obesity. Research has shown the white canary has a much higher vitamin A requirement level than other canaries.[37]

For good health and breeding results, canaries are traditionally fed soft food (egg food). The formulas of available commercial egg foods vary greatly. The current recommendation is to follow the advice of an experienced passerine veterinarian, as several formulas are used with good breeding results. They contain essential vitamins, amino acids and minerals. During the breeding period, soft food should be given on a daily basis and offered fresh three times a day. Most of the available soft food contains 16 to 18% protein. Soft food is often moisturized just before administration to make it more acceptable. Sometimes soaked seed or couscous is mixed with the soft food to improve the taste. Soaked, germinated seeds contain many additional vitamins.

Soluble grit sources, such as cuttlefish bone (*Sepia* spp.),

oyster shell, limestone (calcium carbonate), marble (crystalline limestone) and gypsum (calcium sulfate), are used as calcium supplements and are usually completely digested by birds. Insoluble grit consists of items such as sand, quartz and granite and can lead to health problems (eg, impaction of the crop, proventriculus and gizzard) if it is overconsumed.[109] Beach sand is shell (and soluble), but contamination with salt excludes its use.

Fruits, vegetables and many free-growing plants such as dandelion, chickweed and parsley are good sources of fiber, vitamins and minerals.

Sometimes color additives are used to manipulate the color of the plumage. For example, red-colored canaries are fed canthaxantines or ß-carotene 2 weeks before the breeding season until the end of the molt period. Spirulina algae is known as a natural source of coloration, however, it may not be effective to acquire the level of coloration desired. Yellow-colored canaries are supplemented with lutein for enhancing the desired "yellow color" for exhibition.

Because of the poor rate of breeder performance and a high rate of nutritional disorders on the seed-supplemented diet, trials offering pellets or mash containing a balanced proportion of the required nutrients are proving a good alternative to a traditional diet mentioned previously. One should not indiscriminately supplement a formulated diet with calcium, vitamins or minerals.

Most small passerines drink from 250 to 300 ml/kg body weight of water each day (desert birds such as zebra finches are an exception).[71]

Diagnostic Procedures

HISTORY AND PHYSICAL EXAMINATION

A careful consideration of the history is essential because the physical examination and other diagnostic procedures are often not rewarding in Passeriformes due to the size and nature of the birds (Table 39.2).

Examination of Cage or Aviary

Pet birds will often be presented individually or in a small group in their own cage. Points to observe include size, hygiene status, perches, substrate, food and water containers, toys and cuttlefish bone or other supplements. Although this might not give much information about a certain disease, it will help assess the owners and their standard of knowledge regarding hygiene, nutrition and general requirements of the birds.

Table 39.2 | Checklist of Questions to Obtain Patient History

- Number of birds owned, number of sick birds
- Clinical Signs
- Age of affected birds
- Species, breeds involved
- Diet: seed mixture, soft food or formulated diet
- Food supplements (eggs, soaked seeds, vitamins, trace elements, amino acids, live food)
- Drinking water supplements (vitamins, trace elements, amino acids; frequency)
- Breeding method (artificial light, foster parents, baby cages)
- Vaccinations (poxvirus)
- Contact with birds (newly acquired, shows)
- Medications already given

When dealing with a breeding facility, the aviary should be visited. This is the only way to assess important aspects such as general hygiene, nutrition and management procedures.

Observation of the Bird

Before handling and restraining a sick bird for a physical exam, it should be observed from a distance, either in its own cage or, if it is presented in a cardboard box, in a small cage that can be placed directly on a scale.

Restraint and Physical Examination

A systematic approach with good restraint of the bird is important. This allows the physical exam to become routine, which shortens the procedure and reduces the stress factor to the often already compromised patient. The systematic approach assures that nothing is overlooked.

The necessary equipment and basic procedure for performing a physical examination are listed in Table 39.3.

DIAGNOSTIC SAMPLING

Fecal Examination

A direct wet mount of fresh, warm stool should be examined for *Cochlosoma, Giardia, Candida, Macrorhabdus,* bacteria, plant material, chitin skeletons, urates and powderdown feathers. Gram's stains should not contain *Macrorhabdus*, yeast (*Candida*) or bacteria (or only low numbers of gram-positive rods or cocci). Flotation can reveal coccidian oocysts (common), or helminth eggs (seldom seen in canaries and finches).

Crop Swabs

A crop swab can be obtained by using a cotton-tipped applicator moistened in saline (Fig 39.17) or by administration of approximately 0.2 ml saline into the crop with a syringe and small feeding needle and then re-aspirating


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Fig 39.17 | A cotton-tipped applicator in saline is used for a crop swab.

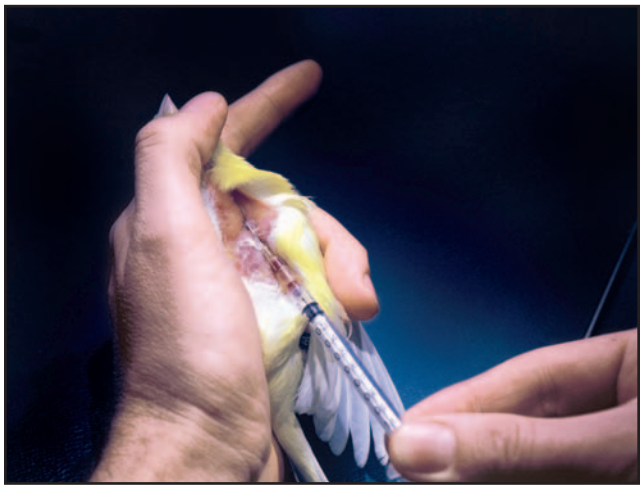
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Fig 39.18 | The right jugular vein in a canary is used for blood sampling.

Table 39.3 | Physical Examination Checklist

Equipment
- Paper towel for capture and restraint
- Jeweler's loupe (for magnification)
- Bright light source (otoscope or endoscope light source)
- Alcohol
- Aural thermometer
- Digital gram scale
- Small observation cage

Procedure
- Prepare all necessary equipment.
- Capture and restrain bird with a paper towel.
- Listen for respiratory sounds.
- Extreme respiratory distress warrants releasing the bird and considering delay of further procedures. Exceeding the oxygen reserve of a small passerine with respiratory impairment can be rapidly fatal.
- Examine the eyes, ears and nares. Look for exudates, crusts, pox lesions, cataracts, sinusitis.
- Examine the oral mucosa and tongue. Look for white plaques (candidiasis, bacterial infections, trichomoniasis).
- Measure body temperature using aural thermometer, which is a fast, low-stress method. Use three readings for an average temperature. In sick small passerines the body temperature is below 40° C (104° F).
- Assess the pectoral muscle mass and color (anemia) and the presence of fat. Look at the skin for signs of dehydration (red, dry skin).
- Examine the abdomen. Assess the internal organs, which can be observed easily through the abdominal wall for signs of hepatomegaly, dilatation of the intestines, ascites or presence of urine in the cloaca. (If the feathers cannot be parted correctly consider applying a drop of alcohol over the abdomen. Be aware that this can cause hypothermia).
- If necessary wet feathers over neck and observe trachea for mites.
- Examine the feathers for the state of molt, ectoparasites, broken feathers, discoloration, alopecia or dirty feathers around the vent and along abdomen.
- Examine the wings and legs for skeletal deformities, fractures, wounds and feather cysts.
- Examine the feet and toes for signs of hyperkeratosis, pox lesions and pododermatitis.
- Place bird in a small cage standing on a digital scale. Register weight. If necessary, cover with a towel.
- Only at this stage (after the bird is no longer in the hand), discuss findings and further testing with the owner.

it. A direct warm wet mount can reveal the presence of *Trichomonas, Candida, Macrorhabdus* or bacteria.

Samples for Bacteriology

Samples for bacteriology may include feces, a cloacal swab, nasal discharge or a skin swab.

Blood Samples

A blood sample may be obtained by venipuncture of the jugular vein (Fig 39.18). No more than 1% of a bird's body weight should be collected, unless this is a terminal procedure for serology prior to euthanasia and necropsy. Hematology includes hematocrit, total protein and blood smear for blood parasites. Serology may indicate paramyxoviruses (PMV), *Chlamydophila* spp. or *Toxoplasma* spp. Table 39.4 lists selected normal hematologic and serum biochemical values.[33]

NECROPSY

Necropsy is a good procedure to find the cause of death or to confirm a diagnosis, especially when dealing with flock problems. The carcass must be refrigerated imme-

Table 39.4 | Hematologic and Chemistries Reference Ranges

Parameter	Canary	Finch	Mynah
PCV (%)	45-60	45-62	44-55
RBC ($10^6/\mu l$)	2.5-4.5	2.5-4.6	2.4-4.0
WBC ($10^3/\mu l$)	4-9	3-8	6-11
Heterophils (%)	20-50	20-65	25-65
Lymphocytes (%)	40-75	20-65	20-60
Monocytes (%)	0-1	0-1	0-3
Eosinophils (%)	0-1	0-1	0-3
Basophils (%)	0-5	0-5	0-5
AP (IU/L)	146-397	—	—
AST=SGOT (IU/L)	145-345	150-350	130-350
LDH (IU/L)	120-350	—	600-1000
Ca (mmol/L)	1.28-3.35	—	2.25-3.25
P (mmol/L)	0.52-1.81	—	—
Glucose (mmol/L)	11-22	11-25	10.5-19.4
TP (g/L)	20-45	30-50	23-45
Creatinine (mmol/L)	8.8-188	—	8.8-53.0
Uric acid (μmol/L)	—	—	237-595
K (mEql/L)	2.7-4.8	—	3.0-5.1
Na (mEql/L)	125-154	—	136-152

Data reprinted with permission from Dorrestein and Elsevier Science.[32]

Table 39.5 | Necropsy Procedure

History:

- Number of birds owned, number of sick birds.
- Identification of carcass: species, leg band.
- Inspect packing and feathers for ectoparasites.
- External examination: feathers, eyes, ears, nostrils, vent; condition of pectoral muscles.
- Wet carcass with alcohol and pluck feathers. Open carcass from sternum to cloaca as well as from sternum up to thoracic inlet and on along neck up to the mandible.
- Assess air sacs and serosal surfaces.
- Remove heart, then the liver and intestinal tract, by cutting through the esophagus and bending the viscera away from the body cavity at the cloaca, leaving lungs, kidneys and gonads in the body.
- Assess all organs macroscopically.
- Swab obvious lesions as well as liver and heart blood for bacteriological cultures.
- Slice all parenchymatous organs (liver, spleen, kidney, pancreas, lung, heart, gonads).
- Open all tubular organs (gastrointestinal tract, trachea).
- Direct wet preparation and flotation of gastrointestinal content.
- Cytology of impression smears of freshly cut surface of liver, spleen, lung, etc, and scrapings of the mucosa of crop, proventriculus and intestine.
- Collect tissues for histopathology in buffered formalin.
- Freeze tissue for virology.

diately after death, and necropsy must be performed within 72 hours. A systematic approach is essential (Table 39.5).

Treatment Techniques

DRUG ADMINISTRATION

Parenteral Administration

As in all avian species, drugs can be administered by intravenous, intramuscular or subcutaneous injection. Drug dosing should be based on the exact weight determined by an electronic gram scale and administered using a precise 0.3-ml insulin syringe, which permits dosing as little as 0.005 ml. Due to the size and nervous character of small passerines, most injectable drugs are administered by intramuscular injection into the cranial third of the pectoral muscle. Applying pressure on the injection site with a dry cotton-tipped applicator can control bleeding. In dehydrated patients, fluids can be administered subcutaneously in the inguinal skin fold.

Oral Administration

Direct oral administration allows accurate and regular dosing but is feasible only in individually diseased birds housed in a small hospital-sized cage. Expecting an owner to individually treat a flock of small passerines housed in a larger cage or aviary will result only in poor owner compliance and a counterproductive stressful situation for the birds being chased through the aviary twice a day.

Medication of the drinking water and soft food is often the only feasible option for flock treatment. Drawbacks of which to be aware include: 1) reduced and irregular water intake, resulting in inadequate or irregular blood levels with therapeutic failure and development of drug resistance; 2) no measurable blood levels during the night due to the high metabolic rate and rapid drug elimination; and 3) the potential for drug toxicoses in dry, hot weather (eg, dimetridazole or furazolidones). Oral drug administration should be via drinking water and soft food simultaneously. Dosages are listed in Table 39.6.[34]

ANESTHESIA

The anesthetic of choice is isoflurane (or sevoflurane) gas, as for other avian species. The bird may be captured and restrained using a paper towel and masked down with 4 to 5% isoflurane. Anesthesia can be maintained at approximately 1.5 to 2.0% isoflurane; the anesthetic depth can be adjusted by monitoring the respiration rate and reflex status. A suitable mask may include a syringe case, syringe or a dropper bottle with the base cut off.

Table 39.6 | Therapeutics Administered via Soft Food and Water in Flock Treatment of Canaries and Small Passerines

Drug	Concentration in Drinking Water (mg/L)	Concentration in Soft Food (mg/kg)
Amoxicillin	200-400	300-500
Amphotericin B	100-200	100-200
Ampicillin	1000-2000	2000-3000
Chlortetracycline	1000-1500	1500
Dimetridazole	125	—
Doxycycline	250	1000
Enrofloxacin	200	200
Fenbendazole	25	25
Furazolidone	200-300	300
Ivermectin	9	—
Metronidazole	100	100
Neomycin	200	200
Nystatin	200,000 IU	200,000 IU
Polymyxin	100,000 IU	100,000 IU
Ronidazole	350	350
Spectinomycin	200-400	400
Spiramycin	200-400	400
Sulfachloropyridazine	150-300	—
Sulfadimidine	150	—
Toltrazuril	180	—
Trimethoprim/sulfonamide	200	200
Tylosin	250-400	400

Note: The birds should be treated at the same time through the drinking water and through the food.
Data reprinted with permission from Dorrestein and Elsevier Science.[32]

Use of a heating pad as well as humidified anesthesia gas will minimize drying out of the mucous membranes and loss of body heat.

SURGERY

Surgery techniques are the same as in other avian species. Special considerations due to the small blood volume and the high metabolic rate include: the use of minimally invasive techniques (eg, removing egg-bound eggs from the cloaca and not by abdominal surgery), bipolar radiosurgical forceps (which allows for hemorrhage control), binocular magnification loupes and a good light source. Postoperative recovery should occur in a dark, warm incubator at 30° to 32° C (86-98° F).

FRACTURE SPLINTING

Fractures of the wing are relatively uncommon and may be treated by external splinting and bandaging with a minimal amount of padding. Fractures of the tarsometatarsus and tibiotarsus are more common. A sandwich adhesive or masking tape splint gives excellent results. The knee and tarsal joint are positioned in a moderately flexed position, and tape is applied to the medial and lateral sides of the fractured leg. The tape is molded to the contour of the leg using hemostat forceps. Instant glue is applied to the outer surface of the tape and then a second layer of tape is applied over the glue-covered surface. The tape is trimmed to make it as small and light as possible. The patient is maintained under anesthesia for another few minutes to allow the glue to dry and form a hard, stable cast. This glue-hardened masking tape splint can be removed with scissors, with the patient under isoflurane anesthesia to avoid refracturing the leg.

Viral Diseases

POXVIRUS

Roughly 232 avian species in 23 orders have been reported to acquire a natural poxvirus infection. It is likely that many more species are susceptible to avipoxviruses. Many of these reported avian species belong to the order of Passeriformes.[9] Poxvirus infections are particularly common in canaries (*Serinus canarius*) and other Fringillidae.[32] Of clinical importance to the avian practitioner is canary pox.[51] The disease is mainly transmitted from latently infected birds by bloodsucking insects, including mosquitoes and red mites.[59]

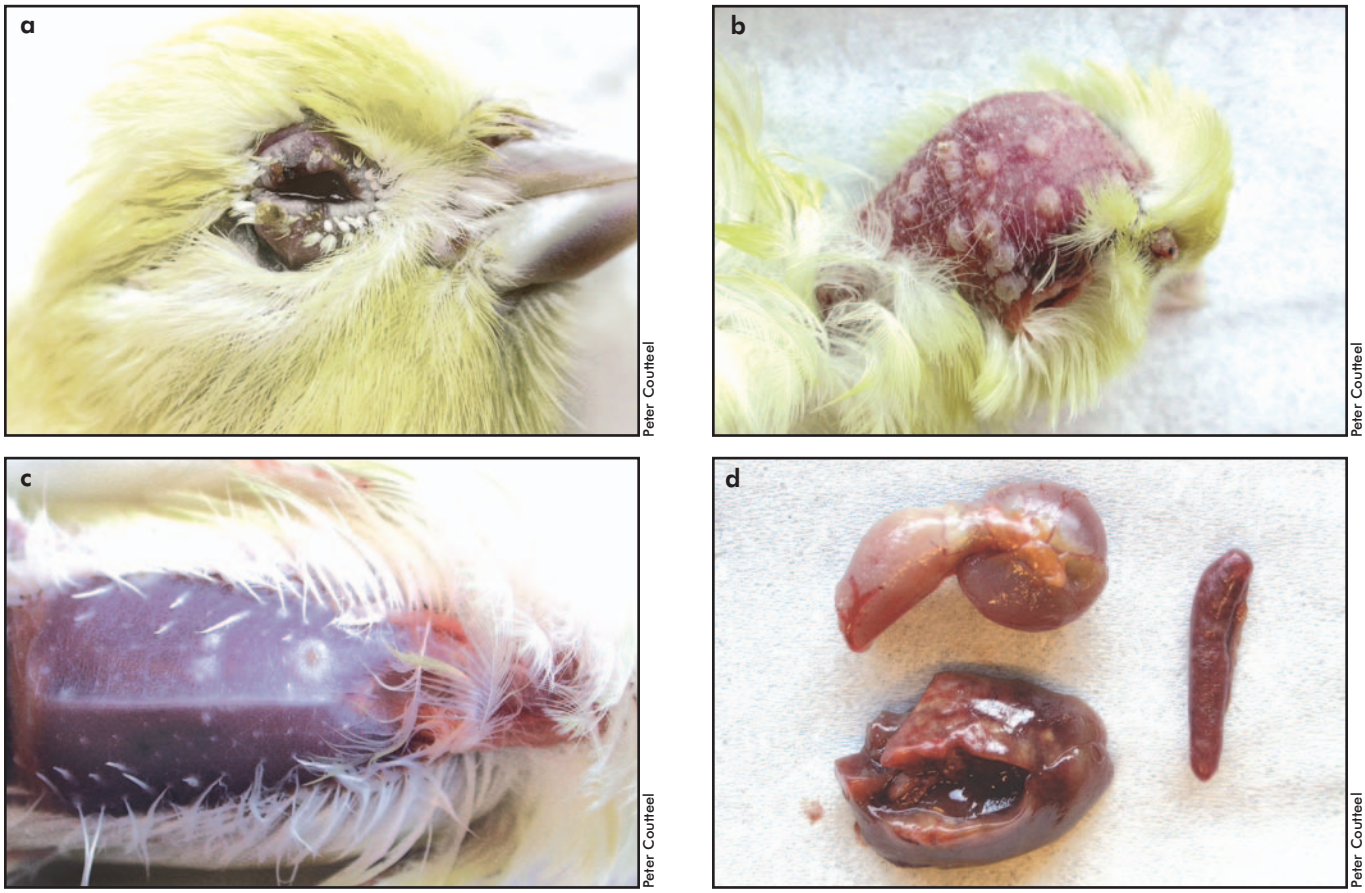
Transmission can also occur through direct contact with an infected bird or indirect contact with contaminated objects such as gloves or hands. Because poxviruses are not capable of penetrating intact epithelium, they must enter the skin through abrasions (eg, caused by cannibalism, territorial behavior, feather picking, aggressive preening or handling and consumption of scabs).[97] Theoretically, pox infections can occur during the whole year. However, most outbreaks are diagnosed in the late summer and autumn, because of the prevalence of mosquitoes. Birds of all ages can develop disease, but young birds (older than 3 weeks) are especially susceptible.

Clinical Disease

The three different clinically recognized symptoms are the cutaneous or external form, the internal diphtheroid form and the septicemic form.[59] Whereas finches often develop the cutaneous form, canaries will often develop the internal diphtheroid or septicemic form, making pox infections in canaries a disease with a high mortality.[17]

Cutaneous Form

This form is localized on the toes and legs and around the eyes, nares and beak (Figs 39.19a-c). These unfeathered body regions are easily accessible to blood-sucking insects. As the lesions progress they develop from papules of roughly 2 to 4 mm diameter to vesicles that open spontaneously, dry out and then become crusts. Tumor-like pox lesions that do not develop vesicles and



Figs 39.19 | a,b,c) Shown are pox lesions on the eyelids, back of head and sternum. **d)** A hemorrhage in the liver and enlarged spleen is shown in a bird with pox.

crusts have also been described.[38,112] The contaminated birds rub their eyes and beaks against the perches. They also may pick the lesions on their legs until they start bleeding. The lesions can be treated locally with iodine, but often the bird will lose a nail or even a digit. The mortality rate, however, is low in this form and the lesions will heal spontaneously after 3 to 4 weeks.[17]

Diphtheroid Form

Lesions occur on the mucosa of the tongue, pharynx and larynx. The fibrinous lesions are gray to brown and caseous.[51] In severe cases birds have difficulty swallowing or exhibit dyspnea.

Septicemic Form

This is the predominant form diagnosed in canaries. Birds show severe dyspnea and become apathetic and cyanotic. They are unable to eat or drink, and mortality can be as high as 90 to 100%. In contrast to other disorders, such as tracheal mites or bacterial infections of the upper respiratory system, the breathing is silent without clicking or growling sounds.

Diagnosis

Antemortem in-house cytology and postmortem histopathology often reveal intracytoplasmic inclusions (Bollinger bodies) in epithelial cells.[59] Macroscopic postmortem findings are often unspectacular (Fig 39.19d).

Prophylaxis

Attenuated live vaccines[c] that are applied using the wing-web method are commercially available for canaries and crossbreeds. A whitish swelling or scab at the injection site observed 8 to 10 days post-vaccination indicates a successful vaccination. Protection lasts approximately 6 months. Vaccination should be performed early in the year, prior to mosquito season. Solid immunity develops 2 to 4 weeks after vaccination. Fledglings should be at least 4 weeks old. Vaccination can be repeated without any risk if vaccination status is unknown. A new needle should be used for every bird so that blood-borne diseases, including poxvirus, are not transmitted. Vaccinated birds should be separate from newly acquired non-vaccinated birds.[17]

Treatment

There is no therapy for the septicemic poxvirus infection. Only a preventive vaccination offers solid protection. In case of an outbreak, the following measures

should be taken: separate diseased birds (gasping birds and birds with cutaneous lesions), consider emergency vaccination and begin antimicrobial therapy against secondary bacterial and fungal infections. Multivitamin (especially vitamins A and C) supplementation may help with epithelial turnover and immune system support. Iodine should be applied locally to cutaneous and mucosal pox lesions. Non-steroidal antibiotic eye ointment may be applied. Access to blood-sucking insects must be prevented, and cages and perches much be thoroughly disinfected. An emergency vaccination in the face of an outbreak is controversial. This may result in the recombination between field and vaccine virus strains, inducing severe disease in the entire flock.[51] Handling of the birds can also induce skin abrasions, creating a port of entry for the poxviruses.

Differential Diagnoses

The following should be considered as differentials for the cutaneous form of poxvirus infection: bacterial and mycotic dermatitis, *Knemidokoptes* mites, fiber constriction and conjunctivitis. *Trichomonas* and *Candida* should be considered as differentials for the diphtheroid form, and for the septicemic form, consider bacterial upper respiratory disease, *Atoxoplasma, Trichomonas, Sternostoma, Chlamydophila, Aspergillus, Syngamus* and *Paramyxovirus* (PMV) infections.

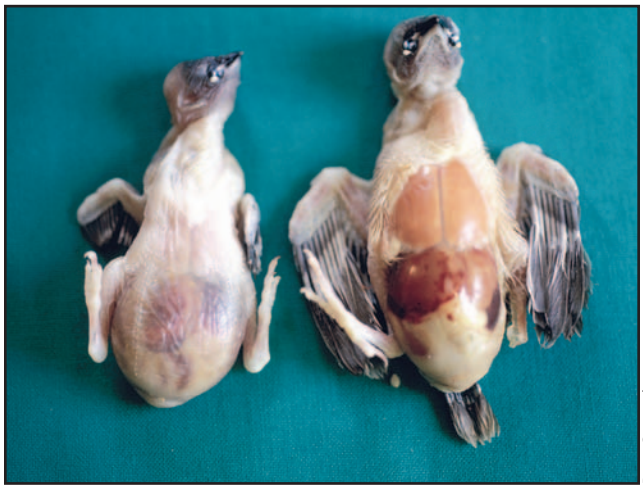
POLYOMAVIRUS

Avian polyomavirus (APV) has been associated with mortality in Passeriformes of all ages. Reports include disease in canaries, greenfinches (*Carduelis chloris*), goldfinches (*Carduelis carduelis*), Gouldian finches (*Erythrura gouldiae*), painted finches, golden-breasted starlings (*Cosmopsarus regius*), seedcrackers (*Pyrenestes* sp.) and bluebills (*Spermophaga haematina*).[22,48,71,99]

Recently, 20 different avian polyomavirus variants have been determined.[85] A parsimony tree was constructed containing three major branches. All European viruses were confined to Branch I, but APVs from the USA represented all three branches. Polyomaviruses from different avian species were also on each of the three branches, suggesting that species-specific pathotypes have not developed.

Clinical Disease

No specific clinical entity is recognized in birds with APV, but reported clinical signs include acute mortality, nonspecific signs of disease (fluffed bird), delayed fledging, poor feather development, abdominal hemorrhages (Fig 39.20) and long, tubular, misshapen beaks in surviving fledglings.[17,95]



Peter Coufteel

Fig 39.20 | Abdominal hemorrhages in these Lady Gouldian finch (*Erythrura gouldiae*) chicks was due to polyomavirus.

Diagnosis

Serodiagnosis of APV has traditionally been performed using a serum neutralization test. Recently a blocked enzyme-linked immunosorbent assay for the detection of avian polyomavirus-specific antibodies was developed in Germany.[61]

DNA probes used to detect avian polyomavirus in psittacines are commercially available. This probe, however, will not detect the virus in seedcrackers.[48] It does not appear clear in which passerine cases these probes are capable of detecting avian polyomavirus. A DNA in situ hybridization analysis using a probe designed to recognize nucleotide sequences coding for a major viral structural protein was highly sensitive in the detection of APV infection in seedcrackers and bluebills.[48]

Necropsy

Avian polyomavirus lesions that can be expected on gross postmortem examination include a pale swollen liver, splenomegaly, and perirenal, serosal, intestinal and hepatic hemorrhage.[51] Histologic changes include inflammation and necrosis of the liver, spleen, myocardium, intestinal tract and bone marrow. These organs also will demonstrate intranuclear inclusion bodies.[95]

Prophylaxis and Treatment

A commercially available inactivated avian polyomavirus vaccine for parrots has not been shown to induce protection in Passeriformes. In one case, over 80 Passeriformes of various species were vaccinated. The nestling mortality caused by polyomavirus did not decrease in the breeding season following the vaccination trial.[99] Other than supportive measures, there is no treatment for diseased Passeriformes. In aviaries experiencing high nestling mortality, discontinuing all breeding during one season can often control the outbreak of clinical disease.

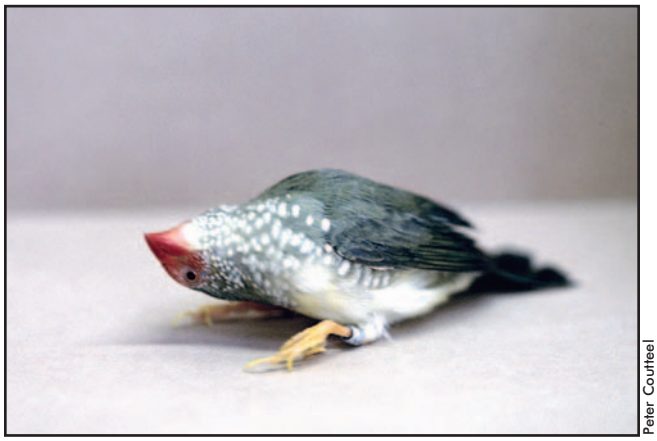

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Fig 39.21 | This melba finch (*Melba astrilde*) is exhibiting torticollis as a result of paramyxovirus (PMV-3).


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Fig 39.22 | Papillomavirus with wart-like skin masses is shown in a siskin (*Carduelis spinus*).

Differential Diagnoses

Differential diagnoses include all pathogens causing sudden death in Passeriformes (Table 39.7).

PARAMYXOVIRUS

Of the nine known serotypes of paramyxoviruses (PMV-1 through PMV-9), only PMV-1, -2 and -3 are known to cause disease in Passeriformes.[96,104]

PMV-1

As in all avian species, PMV-1 can cause a variety of disease signs in Passeriformes. These include watery diarrhea, respiratory signs, sudden death and, less commonly, neurologic signs.[32,71] Most species of free ranging birds have been found to be susceptible to some strain on PMV-1. PMV-1 is a reportable disease in most countries.

PMV-2

PMV-2 infections in Passeriformes appear to be mild and self-limited. They are endemic in finches originating from northern Africa. Experimentally, they cause mild upper respiratory tract disease.[51]

PMV-3

The most common serotype observed in Passeriformes is PMV-3. Commonly affected species are African and Australian finches.[17,32] Clinical signs include conjunctivitis, anorexia, diarrhea, voluminous starchy stools, dyspnea and torticollis (Fig 39.21).[70,102,104] Diagnosis is based on serology or virus isolation. Serologically, there are cross-reactions with PMV-1. An exact differentiation is possible with monoclonal antibodies.[51] Virus isolation is performed by hemagglutination inhibition using antisera specific for PMV-3 on inoculated cell cultures.[59,104] Gross postmortem lesions can be minimal and are nonspecific. Histologic lesions consist of encephalitis, myocarditis and pancreatitis associated with intranuclear and intracytoplasmic inclusion bodies.[104] Although no commercial PMV-3 vaccination is available, an inactivated vaccine was shown to produce sufficient immunity to withstand challenge in canaries.[8] Differential diagnoses include toxins (organophosphates, dimetridazole), trauma, atoxoplasma and bacterial meningoencephalitis (Table 39.8).

HERPESVIRUS

Herpesviruses are much less a clinical problem in Passeriformes than in many other avian orders. Uncharacterized herpesviruses causing necrosis of the liver, spleen and bone marrow have been reported in finches (Estrildidae), canaries and weavers (Ploceidae).[60]

CYTOMEGALOVIRUSES

Cytomegalovirus can cause severe conjunctivitis, pseudosymblepharon (crust sealed eyelids) and acute illness, often with a high mortality rate, in Australian and African finches.[17,24,103] Diagnosis is based on histopathology with cytomegaly and karyomegaly of epithelial cells, hemorrhage in the lung and bronchi and diphtheroid lesions in the esophagus and choana. Basophilic intranuclear inclusion bodies within epithelial cells of the conjunctiva and respiratory tract can be observed.[17,94] Treatment consists of supportive measures including tube-feeding and keeping the eyelids free of forming crusts. Differential diagnoses of conjunctival lesions include infections with poxvirus, *Chlamydophila, Mycoplasma* or other bacteria.[24] For differential diagnoses of sudden death, see Table 39.7.

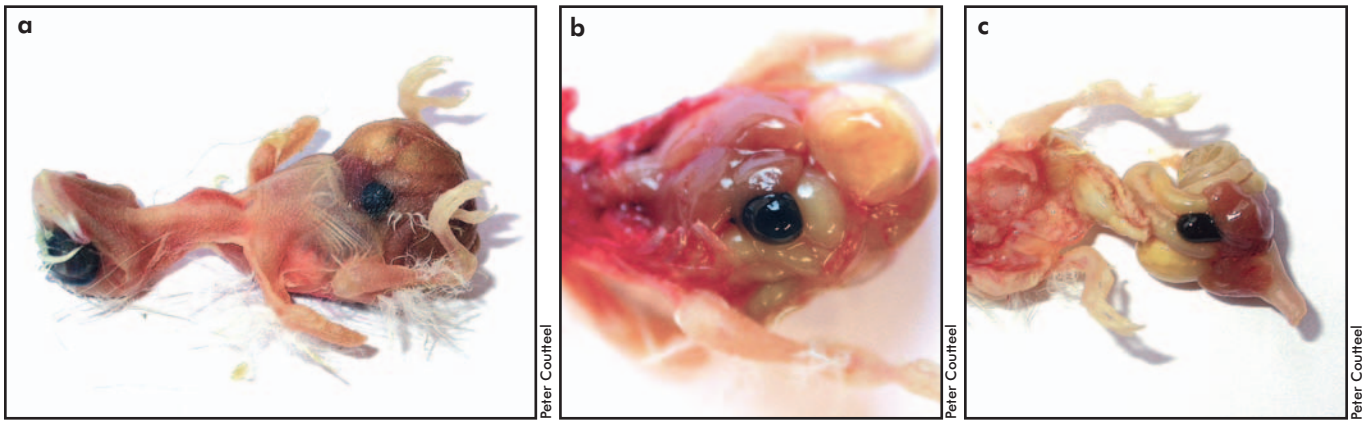
PAPILLOMAVIRUS

Proliferative wart-like skin masses observed on the legs and feet of Fringillidae (especially siskins [*Carduelis spinus*] and European chaffinches [*Fringilla coelebs*]) are caused by papillomaviruses (Fig 39.22). In other passerine species, however, lesions appear to be rare.[95]

Table 39.7 | Causes of Sudden Death and Increased Mortality in Passiformes

	Infectious	Parasitic	Other
Canaries and Finches (Fringillidae)	★ *E. coli* (nestlings) ★ Mycobacteria (adults) ★ *Macrorhabdus* (all ages) ★ *Yersinia pseudotuberculosis* (adults) ★ Poxvirus (fledglings) ● Herpesvirus (all ages) ● Polyomavirus (all ages) ● Circovirus (nestlings) ● *Chlamydophila* (all ages) ● *Salmonella* (all ages) ● Other bacteria (nestlings) ● *Candida* (nestlings) ● *Aspergillus* (adults) ❍ PMV-1 (all ages) ❍ *Campylobacter* (nestlings)	★ *Atoxoplasma* (fledglings) ★ *Dermanyssus* (adults) ● *Isospora* (all ages) ● *Sternostoma* (adults) ❍ *Cryptosporidium* ❍ *Sarcocystis* ❍ *Toxoplasma* ❍ *Cochlosoma* ❍ Blood parasites ❍ Microsporidia ❍ *Syngamus* ❍ Spiruroids	● Inhalant toxins (PTFE, CO) ● Ingested toxins ● Cranial trauma ● Starvation ● Visceral gout ● Blood loss ● Predators (eg, cats) ● Neoplasia ● Diseases of the urogenital tract ❍ Ingested foreign bodies ❍ Iron storage disease ❍ Hepatic lipidosis ❍ Cardiac disease
Waxbills (Estrildidae)	★ *E. coli* (nestlings) ★ *Campylobacter* (nestlings) ● Polyomavirus (all ages) ● Herpesvirus-cytomegalovirus (all ages) ● *Chlamydophila* (all ages) ● *Salmonella* (all ages) ● Mycobacteria (adults) ● *Yersinia pseudotuberculosis* (adults) ● Other bacteria (nestlings) ● *Macrorhabdus* (all ages) ● *Candida* (all ages) ● *Aspergillus* (adults) ❍ PMV-1 (all ages) ❍ Poxvirus (fledgling)	● *Cochlosoma* (nestlings, fledglings) ● *Isospora* (all ages) ● *Sternostoma* (adults) ● *Dermanyssus* (nestlings) ❍ *Cryptosporidium* ❍ *Sarcocystis* ❍ *Toxoplasma* ❍ Blood parasites ❍ Microsporidia ❍ *Syngamus* ❍ Spiruroids	● Inhalant toxins (PTFE, CO) ● Ingested toxins ● Cranial trauma ● Starvation ● Visceral gout ● Blood loss ● Predators (eg, cats) ● Neoplasia ● Diseases of the urogenital tract ❍ Ingested foreign bodies ❍ Iron storage disease ❍ Hepatic lipidosis ❍ Cardiac disease
Mynah (Sturnidae)	★ *Aspergillus* ● *Chlamydophila* ● *E. coli* ● Mycobacteria ● *Salmonella* ● *Yersinia pseudotuberculosis* ● Other bacteria ● *Macrorhabdus* ● *Candida* ❍ PMV-1 ❍ Poxvirus	● *Coccidia* ● Spiruroids ❍ *Dermanyssus* ❍ *Cryptosporidium* ❍ *Sarcocystis* ❍ *Toxoplasma* ❍ Blood parasites ❍ Microsporidia	★ Iron storage disease ★ Ingested foreign bodies ● See canaries ❍ Hepatic lipidosis ❍ Cardiac disease

★ Common ● Occasional ❍ Seldom



Figs 39.23a-c | Circovirus, congestion of the gall bladder, is expressed as "black spot disease," which is shown in a 2-day-old canary prior to and after necropsy.

Table 39.8 | Causes of Central Nervous System Signs

	Infectious	Parasitic	Other
Canaries and Finches (Fringillidae)	● PMV-3 ❍ PMV-1 ❍ Mycobacteria ❍ *Listeria* ❍ *Aspergillus* ❍ *Sarcocystis*	● *Atoxoplasma* (fledglings) ❍ *Toxoplasma*	● Trauma (cranium, spinal cord) ● Toxins (organophosphates) ● Pharmacologic agents (dimetridazole) ❍ Hepatoencephalopathy ❍ CNS neoplasia ❍ CNS abscess ❍ Hypovitaminosis B_1 ❍ Vitamin E/selenium deficiency ❍ Hypocalcemia ❍ Epilepsy ❍ Age - improper diet (arteriosclerosis, infarct)
Waxbills (Estrildidae)	★ PMV-3 ❍ PMV-1 ❍ Mycobacteria ❍ *Listeria* ❍ *Aspergillus* ❍ *Sarcocystis*	❍ *Toxoplasma*	● Trauma (cranium, spinal cord) ● Toxins (organophosphates) ● Pharmacologic agents (dimetridazole) ❍ Hepatoencephalopathy ❍ CNS neoplasia ❍ CNS abscess ❍ Hypovitaminosis B_1 ❍ Vitamin E/selenium deficiency ❍ Hypocalcemia ❍ Epilepsy
Mynah (Sturnidae)	❍ PMV-1, -3 ❍ Mycobacteria ❍ *Listeria* ❍ *Aspergillus* ❍ *Sarcocystis*	❍ *Toxoplasma*	● Trauma (cranium, spinal cord) ● Toxins (organophosphates) ● Pharmacologic agents (dimetridazole) ❍ Hepatoencephalopathy ❍ CNS neoplasia ❍ CNS abscess ❍ Hypovitaminosis B_1 ❍ Vitamin E/selenium deficiency ❍ Hypocalcemia ❍ Epilepsy

★ Common ● Occasional ❍ Seldom

Papillomavirus lesions can bleed profusely and can often cause leg band constriction.[17] Papillomavirus-like infections causing epithelial proliferations at the commissure of the beak and on the head in canaries have been described in Belgium.[28] Treatment consists of local disinfection. An autogenous vaccine can be considered, although no data exist to prove the efficacy in Passeriformes.[17] Differential diagnoses include hyperkeratosis, poxvirus, *Knemidokoptes* mites and trauma.

CIRCOVIRUS

A disease called "black spot" by European canary breeders has been proven to be caused by a circovirus. The disease is observed in hatchlings and nestlings and has a high mortality. Signs include abdominal enlargement and congestion of the gall bladder (visible as a black spot through the skin) (Figs 39.23 a-c).[17] Feather loss and lethargy in finches also have been associated with circovirus.[78] Diagnosis is based on recognizing inclusion bodies on histopathology of the bursa of Fabricius or the presence of 18-nm viral particles on electron microscopy. PCR techniques for psittacine circovirus (psittacine beak and feather disease—PBFD virus) fail to demonstrate viral presence, indicating that the canary circovirus differs genetically from the PBFD virus.[52] Nucleotide sequencing showed the virus to be more closely related to the Columbid circovirus than to the PBFD virus.[111] Differential diagnoses include *Atoxoplasma, Isospora, E. coli* and other causes of mortality in nestlings (see Table 39.7).

Bacterial Diseases

The general principles for diagnosing, treating and controlling bacterial disease in Passeriformes are similar to those in other avian orders.[71] There is normally no bacterial growth on routine aerobic microbiologic cultures taken from passerines. Stained fecal smears collected from normal canaries and finches reveal either no bacteria or low levels of gram-positive rods or cocci.[17,71]

CHLAMYDOPHILA PSITTACI

The causative agent of chlamydiosis is *Chlamydophila psittaci*.[113] Passeriformes appear to be less susceptible than Psittaciformes to chlamydiosis.[71] Clinical signs are nonspecific and include general apathy, diarrhea and nasal and ocular discharge. Mortality is usually low.[32]

Diagnosis is normally made at necropsy. Identification of the organism within macrophages in impression smears


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Fig 39.24 | This canary has *Mycoplasma* conjunctivitis.


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Fig 39.25 | This breeding female looks dirty and wet and has sweating disease.

of internal organs or air sac walls can be made by use of a Macchiavello, Gimenez or Stamp stain.[113] Antigen may be identified from a swab of the cut surface of internal organs or air sac walls by use of an ELISA or PCR.[30,77]

For treatment, water-soluble doxycycline hyclate, at a dose of 280 mg/L, has been proven to achieve plasma doxycycline concentrations >1 μg/ml in cockatiels.[44,88] Doxycycline hyclate at 280 mg/L in the drinking water and simultaneously 280 mg/kg in soft food are clinically effective for the treatment of chlamydiosis in canaries and finches and cause no side effects. Often citric acid is added to the drinking water. In Europe a pharmaceutical company produces water soluble doxycyclinum hyclatum[d] in combination with citric acid. This combination makes the product more water soluble and less sensitive to light.

MYCOPLASMA spp.

Mycoplasma spp. are often associated with conjunctivitis and other signs of upper respiratory disease in canaries and finches (Fig 39.24). Epizootic conjunctivitis caused by *Mycoplasma gallisepticum* has been reported in house finches (*Carpodacus mexicanus*) in the eastern US.[27] Diagnosis is based on PCR or culture of conjunctival swab samples, but isolation of this organism is often difficult.

Treatment with ciprofloxacin ophthalmic solution and tylosin at 1 mg/ml in the drinking water resolved mycoplasma infections in house finches.[73] Suspected cases of mycoplasma infections should be treated with tylosin, tetracycline or enrofloxacin.[71]

Differential diagnoses include *Enterococcus faecalis* and other bacterial causes of upper respiratory disease (see Table 39.9), *Mycoplasma, Atoxoplasma, Isospora,* toxins, liver disease, *Sternostoma* and *Macrorhabdus*.

GRAM-NEGATIVE BACTERIA

Escherichia coli

E. coli is probably the most important bacterial cause of diarrhea and nestling mortality in canaries and finches. Various other Enterobacteriaceae also can be involved. Apparently healthy adult birds can be carriers of *E. coli,* resulting in clinical problems during the breeding season.[17] *E. coli* is a secondary pathogen and should be considered the sequel to a primary problem, such as poor hygiene, unsuitable housing, unbalanced diet or other management-related problems. Primary pathogens such as *Atoxoplasma*, circovirus or polyomavirus also may be present.[32]

The typical clinical presentation in a breeding flock includes diarrhea, dehydration and cachexia. The nests as well as the breeding females are dirty, wet and yellowish (sweating disease) (Fig 39.25). The youngsters die before they can be leg-banded on day 6 or 7.[17] Diagnosis is based on aerobic cultures of fecal samples or internal organs in septicemic disease.

Treatment consists of antibiotics, chosen on the basis of culture and sensitivity results, and should be administered in drinking water and egg food from one day before hatching until 6 days after hatching. At the same time, other management-related problems must be addressed.[17]

Differential diagnoses include other bacterial infections that cause diarrhea and mortality, *Atoxoplasma, Isospora,* polyomavirus, circovirus, *Chlamydophila,* toxic enteritis and PMV-1.

Salmonella

Salmonella typhimurium is the most commonly isolated *Salmonella* species in Passeriformes.[47] Salmonellosis is clinically (and at necropsy) very similar to *Yersinia*

pseudotuberculosis infections.[59] Many birds will die without preliminary signs, but chronic disease can also occur. The existence of clinically healthy carriers in canaries and finches has not been researched.[32] On necropsy, small yellow miliary bacterial granulomas can be observed on a dark and swollen liver and spleen. Sometimes focal necrosis in heart, lung and pectoral muscle can be seen.[17] Diagnosis is confirmed on culture.

Treatment consists of antibiotics chosen on the basis of culture and sensitivity. Antibiotics commonly used include sulfonamid-trimethoprim, enrofloxacin and marbofloxacin. Elimination of salmonellosis is often difficult, and an antibiotic treatment should be combined with counseling on good hygiene and disinfection. The success of a flock treatment should be monitored in serial bacterial cultures at 3 to 6 weeks after therapy by examining pooled fecal samples in enrichment medium.[32]

Differential diagnoses of necropsy lesions include *Yersinia pseudotuberculosis* and mycobacterial infections. A large number of viral, bacterial and parasitic pathogens must be considered in the differential diagnoses of the clinical presentation of unspecific disease and flock mortality (see Table 39.7).

Yersinia pseudotuberculosis

Yersinia pseudotuberculosis infects a wide range of avian and mammalian species, including humans. It is particularly common in a variety of passerine species and also in rodents.[49] The pathogen is thought to be indigenous to central and northern Europe, but is now diagnosed throughout the world. The disease is most common in autumn, winter and spring months and is believed to be transmitted by rodents and migratory birds.[17,47]

The clinical signs are nonspecific but normally include peracute mortality in adult birds.[17] Diagnosis is based on the typical small, yellow, miliary granulomas on a dark and swollen liver and spleen (Fig 39.26) and impression smears and bacteriological cultures revealing gram-negative coccoid rods.[49] Clinically diseased birds usually die before treatment can be initiated, but the rest of the flock should be treated based on culture and sensitivity. Enrofloxacin has been shown to completely protect canaries from experimental infection with *Yersinia pseudotuberculosis,* whereas groups of canaries treated with doxycycline, chloramphenicol, ampicillin and sulphamerazine-trimethoprim suffered mortality rates between 30 and 44%.[53]

Differential diagnoses of the typical necropsy lesions include *Salmonella typhimurium* and mycobacterial infections.

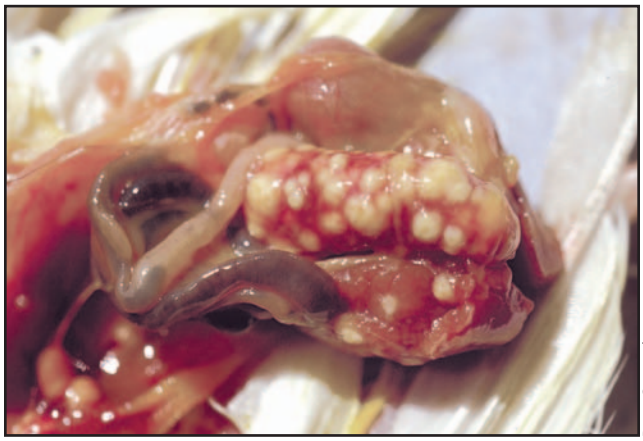

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Fig 39.26 | Miliary granulomas as seen on this canary's dark and swollen spleen are indicative of *Yersinia pseudotuberculosis*.

Campylobacter spp.

Campylobacter fetus subsp. *jejuni* is common in tropical finches and canaries. Society finches can be asymptomatic carriers.[17,59] Clinical signs include apathy in adult birds and high mortality in nestlings. Diseased birds develop yellow diarrhea and pale, voluminous droppings.[17] A fecal Gram's stain reveals comma- to S-shaped gram-negative rods. Culture requires special media and a microaerophilic environment.[47] Campylobacter are often resistant to multiple classes of antibiotics, and the disease frequently recurs despite antibiotic treatment.[47,49] Doxycycline, erythromycin or enrofloxacin could be good choices for flock treatment.[17,47] Thorough cleaning and disinfecting of the aviary may help prevent reinfection.[49]

Pseudomonas spp.

Pseudomonas aeruginosa is the most important pathogen in this genus. Infections often originate from contaminated drinking water, misting bottles or inappropriately prepared sprouted seeds.[71] Clinical signs of disease commonly include foul-smelling diarrhea, but air sacculitis and infections of the oropharynx may also be present. Diagnosis is based on culture, using routine aerobic media.

Treatment is often difficult because *Pseudomonas aeruginosa* is resistant to many antibiotics. Quinolones often offer an adequate treatment.[47] Effective treatment is based on antibiotics (selected by sensitivity testing) combined with elimination of the source of contamination.[32] Differential diagnoses to consider are listed in Tables 39.9 and 39.12.

Other Gram-negative Bacteria

Other gram-negative bacteria such as *Citrobacter, Klebsiella, Serratia, Pasteurella, Bordetella, Borrelia, Actinobacillus* and *Haemophilus* spp. also have been isolated from diseased Passeriformes.[49,71]

Table 39.9 | Causes of Respiratory Clinical Signs

	Infectious	Parasitic	Other
Canaries and Finches (Fringillidae)	★ Poxvirus ★ *Enterococcus faecalis* ★ *Streptococcus* spp. ● *Chlamydophila* ● *Mycoplasma* ● *Pseudomonas* ● Other bacteria ● *Aspergillus* ❍ PMV-1, -2	★ *Atoxoplasma* ★ *Trichomonas* ● *Sternostoma* ❍ *Syngamus* ❍ *Cryptosporidium* ❍ *Sarcocystis* ❍ *Toxoplasma* ❍ *Dermanyssus*	★ Abdominal distension (various causes) (see Table 39.10) ● Inhalant toxins ❍ Foreign body inhalation ❍ Neoplasia
Waxbills (Estrildidae)	★ *Chlamydophila* ● *Mycoplasma* ● *Pseudomonas* ● Other bacteria ● *Aspergillus* ● Cytomegalovirus ● Poxvirus ❍ PMV-1, -2	★ *Sternostoma* ● *Syngamus* ● *Trichomonas* ❍ *Cryptosporidium* ❍ *Sarcocystis* ❍ *Toxoplasma* ❍ *Dermanyssus* (anemia)	★ Abdominal distension (various causes) (see Table 39.10) ● Inhalant toxins ❍ Foreign body inhalation ❍ Neoplasia
Mynah (Sturnidae)	★ *Aspergillus* ● *Chlamydophila* ● *Mycoplasma* ● *Pseudomonas* ● Other bacteria ❍ Poxvirus ❍ PMV-1	● *Syngamus* ● *Trichomonas* ❍ *Cryptosporidium* ❍ *Toxoplasma*	★ Iron storage disease ● Abdominal distension - various causes (see Table 39.10) ● Inhalant toxins ❍ Foreign body inhalation ❍ Neoplasia

★ Common ● Occasional ❍ Seldom

GRAM-POSITIVE BACTERIA

Enterococcus faecalis

Enterococcus faecalis (formerly *Streptococcus bovis*) is a frequent inhabitant of the passerine alimentary tract.[47] It also is associated with chronic tracheitis, pneumonia and air sacculitis.[25] Affected birds have increased respiratory sounds, voice changes and dyspnea. Form canaries are especially sensitive. Treatment with antibiotics will improve the clinical signs, but individual birds can seldom be completely healed. Differential diagnoses include *Sternostoma tracheacolum*, other bacterial infections of the upper respiratory tract, poxvirus, PMV-1, *Chlamydophila, Aspergillus, Atoxoplasma,* and *Trichomonas* and causes of abdomen distension (Table 39.10). Concurrent infections with *Enterococcus faecalis* and *Sternostoma tracheacolum* (tracheal mites) can occur.

Mycobacterium spp.

Mycobacterial infections were the most commonly diagnosed bacterial diseases in a review of 546 necropsies performed between 1991 and 1997 in Passeriformes in Switzerland. Over 8% of all necropsied Passeriformes suffered from a mycobacterial infection, and 40% of all diagnosed bacterial infections in this avian order were caused by *Mycobacterium* spp.[1]

Clinical signs are nonspecific but include chronic "sick bird", diarrhea, weight loss and death.[36] The classic disease with tubercles in the organs is seldom seen in Passeriformes. These results suggest that mycobacterial disease is often overlooked in passerine necropsies. Acid-fast-stained impression smears of the intestinal wall and liver should be performed on every necropsied passerine.[1] Although classically described as being caused by *Mycobacterium avium,* the advent of PCR techniques for species identification has (at least in Europe) shown *Mycobacterium genavense* to be the most commonly isolated species.[56,57,83,86,90,100,110] *Mycobacterium tuberculosis* has recently been diagnosed in a canary.[55]

In Passeriformes, mycobacterial infections are almost exclusively diagnosed postmortem. Gross necropsy is often unrewarding, with nonspecific hepato- and splenomegaly and thickened intestinal walls. Acid-fast bacilli can, however, be found in the eyelids and many internal organs including liver, jejunum, ceca and/or colon, spleen, lung, air sac, iris, brain, bone marrow, muscle and kidney.[57,83,100] No treatment of mycobacterial diseases in Passeriformes has been described.

Other Gram-positive Bacteria

In addition to the gram-positive species described above, infections with *Erysipelothrix rhusiopathiae, Listeria monocytogenes, Clostridium perfringens,* as well as various *Staphylococcus* spp. and *Streptococcus* spp. have been described in Passeriformes.[71]

Table 39.10 | Causes of Abdominal Enlargement

	Hepatomegaly	Reproductive Tract	Other
Canaries and Finches (Fringillidae)	★ *Atoxoplasma* ★ *Isospora* ★ *Yersinia pseudotuberculosis* ★ Mycobacteria ● Circovirus ● *Chlamydophila* ● Other bacteria ❍ Metabolic liver disease	★ Egg binding ● Salpingitis/Impacted oviduct ❍ Egg-related peritonitis ❍ Ovarian cysts	★ Coccidiosis ● Neoplasia ● Adipositas ● Ascites, hepatic ❍ Ascites, heart ❍ Ascites, hypoproteinemia ❍ Peritonitis
Waxbills (Estrildidae)	● *Yersinia pseudotuberculosis* ● Mycobacteria ● *Chlamydophila* ● Other bacteria ❍ Metabolic liver diseases	● Egg binding ❍ Salpingitis/Impacted oviduct ❍ Egg-related peritonitis ❍ Ovarian cysts	● Coccidiosis ● Neoplasia ● Adipositas ● Ascites, hepatic ❍ Ascites, heart ❍ Ascites, hypoproteinemia ❍ Peritonitis
Mynah (Sturnidae)	★ Iron storage disease ● *Chlamydophila* ● Other bacteria ❍ Other metabolic liver diseases	● Egg binding ❍ Salpingitis/Impacted oviduct ❍ Egg-related peritonitis ❍ Ovarian cysts	★ Ascites, iron storage disease Other causes of ascites ❍ Coccidiosis ❍ Neoplasia ❍ Peritonitis

★ Common ● Occasional ❍ Seldom

Fungal Diseases

MACRORHABDOSIS

Classically known as a common disease in budgerigars ("going light"), this organism was previously termed "megabacteria" or "avian gastric yeast". Observation has been made of this organism in a wide range of passerine species.[23,50,43,84] Historically described as "megabacteriosis" during the last 20 years, there has been frequent debate on the description of this as a large gram-positive bacterium. Recent investigations in Germany proved that the so-called megabacteria are indeed fungi,[91,92] and Phalen has now renamed the pathogen, *Macrorhabdus ornithogaster*.

Clinical Disease

Chronic depression and weight loss are typical of macrorhabdosis. Birds are always hungry and stay close to the food bowl, eating large quantities of soft food. Regurgitation is not a clinical sign in passerines. Droppings often contain undigested seeds. The patient may be anemic with pale muscles. The liver becomes visible due to the proventricular dilatation. Other diseases that either may have triggered macrorhabdosis or developed as secondary diseases following macrorhabdosis must be considered.

Diagnosis

Diagnosis is based on microscopic examination of a fecal sample. The organism is easily recognized on a wet mount or following a Gram's stain using a 1000 magnification[17,50] (Fig 39.27). Failure to find Macrorhabdus organisms does not prove that the bird is not infected, as shedding begins only after a certain stage of disease and then may occur irregularly. Microscopic examination of sequential fecal samples will increase the sensitivity. Pooled fecal samples from an aviary will give good information on the status of infection within a group of birds. The organism appears in proventricular scrapings after necropsy. Note that routine fungal culture will not yield growth of this fastidious organism.

Necropsy

A distended, thick-walled proventriculus, often with white mucus on the mucosal surface, is revealed upon necropsy (Fig 39.28). Sometimes, ulcerations and small petechial bleedings of the mucosa can be detected.

Prophylaxis and Treatment

Traditional therapy consists of supportive care, including a warm, controlled environment and administration of amphotericin B. Antibiotics for secondary infections can be given. The patient should have access to soft food ad libitum. Concurrent diseases should be treated accordingly.

Differential Diagnoses

Chronic bacterial infections such as mycobacteriosis and *Chlamydophila* as well as parasitic infections such as *Atoxoplasma, Isospora,* and *Cochlosoma* may be suspected. Toxins and nutritional changes should also be considered.

CANDIDIASIS

Candida is a common environmental contaminant and in low numbers is considered a normal inhabitant of the

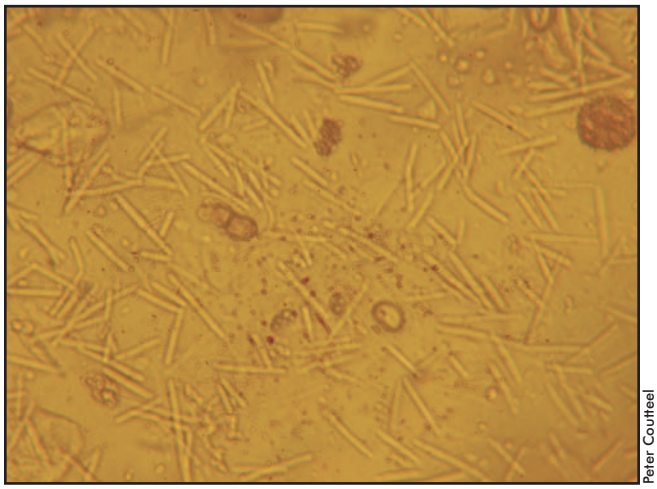
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Fig 39.27 | Macrorhabdus (400x, unstained) is easily recognized on a wet mount or following a Gram's stain.

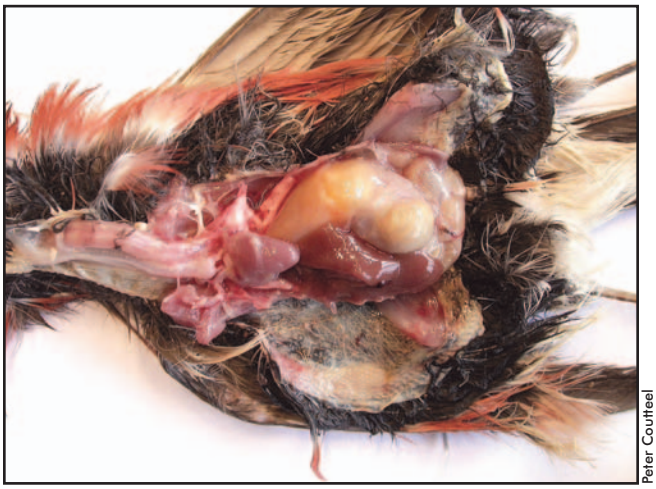
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Fig 39.28 | Macrorhabdosis, distended proventriculus, is shown in this canary.

gastrointestinal tract.[59,115] It is considered an opportunistic pathogen. Predisposing factors include prolonged antibiotic therapy, malnutrition, spoiled food, hand feeding, stress (crowding) and the juvenile immune system.[17] Disease is not overly common in Passeriformes but is observed more often in tropical finches than in canaries.[32] Clinical signs include regurgitation, anorexia, diarrhea, whole seeds in the feces, crop stasis, gas formation and tilted appearance with elevation of the abdomen and tail.[108] Diagnosis is based on demonstrating large numbers of budding yeast, which can be visualized on a crop swab or in a fecal sample. At necropsy, a thickened crop wall and white coating of the mucosa, as well as possible white plaques in the oropharynx, can be observed.

Prophylaxis and Treatment

Prophylaxis is based on prevention of predisposing factors (listed above). Treatment may include antimycotic drugs, such as nystatin (not absorbed from the gastrointestinal tract), fluconazole or ketoconazole (systemic drugs). Medicated drinking water and soft food may be offered.[17] Differential diagnoses include *Trichomonas*, poxvirus, bacterial stomatitis, ingluvitis or enteritis, macrorhabdosis and hypovitaminosis A.

ASPERGILLOSIS

Aspergillosis is one of the most commonly diagnosed diseases in mynahs.[58,62] It is occasionally diagnosed in canaries and finches. Predisposing factors include a warm and humid environment, overpopulation and contaminated food and environment. Recently imported birds are especially susceptible.

Clinical Disease

Mynahs may experience loss or change of voice, peracute dyspnea caused by a syrinx granuloma, chronic dyspnea and depression. In finches, aspergillosis is an acute disease with respiratory distress.[17]

Diagnosis

Definitive diagnosis of aspergillosis in a live bird is difficult. Diagnostic procedures in mynahs may include tracheoscopy, endoscopy and culture of suspicious lesions.

Necropsy

Typical aspergillosis lesions within the lung and air sac walls and syrinx granulomas may be seen. Culture, Gram's stain, wet mount or histopathology can provide a definitive diagnosis.

Prophylaxis and Treatment

Concentration of Aspergillus spores in the environment (moldy fruits on bottom of cage, general poor hygiene) should be reduced as well as susceptibility of the bird by providing a balanced diet (ensuring adequate vitamin A, in particular), improving the room climate, increasing ventilation and reducing stressors. Treatment consists of giving antimycotics orally and via inhalation, as in psittacines.

Differential Diagnoses

Iron storage disease and other causes of respiratory disease in mynahs (see Table 39.9) and dyspnea due to abdominal enlargement (see Table 39.10) must be considered.

DERMATOMYCOSES

The most common etiologic agents are *Microsporum gallinae* and *Trichophyton* spp.[71] Clinical signs include feather loss and hyperkeratosis predominantly on the head and neck (Figs 39.29, 39.30).[17] Diagnosis is based on culture or biopsy and histopathology. Treatment with topical miconazole or several sprays with enilconazole


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Fig 39.29 | Dermatomycosis is shown on this canary's head.


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Fig 39.30 | Dermatomycosis is shown on the dorsal surface of this canary's wing.

are effective. Systemic antimycotics will provide improvement but probably not eliminate the infection. Differential diagnoses include *Knemidokoptes* mites, bacterial dermatitis, feather picking, cannibalism and hyperkeratosis (Table 39.11).

Parasitic Diseases

COCCIDIAL DISEASES

Atoxoplasma

The taxonomy of *Atoxoplasma* spp. is controversial. Some authors suggest that this genus should be placed in the *Isospora* genus.[68,114] This disease is also called "Lankesterella" or "big liver disease".[17] *Atoxoplasma* spp. appear to be host specific.[68] The species affecting canaries has been named *Atoxoplasma* or *Isospora serini*.[17,32]

Unlike other Eimeriidae species, the asexual life cycle of Atoxoplasma takes place in internal organs and not in the intestinal mucosa.[59] The life cycle of the organism begins with the host's oral ingestion of oocysts.[81] Oocysts excyst the sporozoites within the intestinal tract. Sporozoites penetrate the intestinal wall and spread in lymphocytes and macrophages to parenchymal organs. Affected organs include lung, liver, spleen, pancreas, pericardium and intestinal epithelium. Several generations of asexual schizogony in these organs produce merozoites. Merozoites migrate back to the intestinal mucosa. Gametogony (sexual cycle) of the merozoites produce oocysts. Oocysts are excreted with the feces.

This is a common flock disease in canaries but only occasionally diagnosed in exotic finches. It has been a devastating disease for the captive population of the endangered Bali mynah (*Leucospar rothschildi*).[79,81,114]

Clinical Disease

Typically, this is a disease of young canaries aged 2 to 9 months.[32] The affected bird will appear fluffed up and will be debilitated and anorectic. It will have diarrhea and a red, swollen vent. Hepatomegaly is visible through the abdominal wall caudal to the sternum. Mortality is variable, but up to 80%.[32] Occasionally, a patient will exhibit neurologic signs, such as epileptiform seizures and intermittent weakness. It may exhibit respiratory distress.[5,121]

Diagnosis

Definitive antemortem diagnosis is difficult because after the acute phase, only a few *Atoxoplasma* oocysts are excreted.[32,59] Fecal flotation shows oocysts with 2 sporocysts, each containing 4 sporozoites. Microscopic differentiation from Isospora is not easy: *Atoxoplasma serini* oocysts = 20.1 x 19.2 μm, *Isospora canaria* oocysts = 24.6 x 21.8 μm. A PCR assay[c] has been developed that will detect an 18S rDNA fragment of *Atoxoplasma* species in feces, blood and tissues of infected birds.[68]

Necropsy

Necropsy reveals severe splenomegaly, hepatomegaly and dilated bowel loops.[17] Intracytoplasmic inclusion bodies will appear in mononuclear cells in impression smears or on histopathology of the lung, liver and spleen.[5,17,72]

Prophylaxis

Sound husbandry practices must be observed: avoid overcrowding, practice good hygiene and provide proper nutrition.[81] Newly acquired birds must be quarantined and screened with multiple fecal flotations for the presence of *Atoxoplasma*. Adult canaries can be asymptomatic carriers and will shed oocysts sporadically. In collections with recurrent disease, consider annual coccidial treatment prior to the breeding season.

Treatment

Clinically diseased individuals usually die before they respond to treatment. Anticoccidial drugs such as toltrazuril, sulfachloropyridazine (Esb3 30%) or other sulfonamides may be given. Atoxoplasmosis is considered resistant to treatment; however, Esb3 30% at 150

Table 39.11 | Possible Causes of Diseases of the Skin, Feathers and Extremities

	Extremities	Feathers	Skin/Subcutis/Eye/Ear
Canaries and Finches (Fringillidae)	★ Fiber constriction ★ Hyperkeratosis ★ Pododermatitis ★ Fractures ★ Leg band constriction ● Cutaneous pox ● Papillomavirus ● *Knemidokoptes* mites ● Burns ● Frost bite ● Luxations ● Bite wounds ● Arthritis ❍ Leg deformities (malnutrition) ❍ Degenerative joint disease	★ Feather cysts ★ *Dermanyssus* (red mites) ★ Molt (light, temperature) ★ Spoiled feathers (diarrhea, sitting on floor) ★ Poor husbandry ● Polyomavirus ● Ectoparasites ● Feather picking, cannibalism ● Loss of color (nutritional) ● Baldness (hormonal, malnutrition?) ❍ Circovirus	● Cutaneous pox ● *Knemidokoptes* mites ● Bacterial dermatitis ● Dermatomycoses ● Neoplasia ● Abscesses ● Adiposity ● Otitis ● Conjunctivitis/Blepharitis ● Uropygial gland impaction or neoplasia ● Wounds
Waxbills (Estrildidae)	★ Fiber constriction ★ Hyperkeratosis ★ Pododermatitis ★ Fractures ★ Leg band constriction ● Cutaneous pox ● Papillomavirus ● *Knemidokoptes* mites ● Burns ● Frost bite ● Luxations ● Bite wounds ● Arthritis ❍ Leg deformities (malnutrition) ❍ Degenerative joint disease	★ *Dermanyssus* (red mites) ★ Molt ★ Spoiled feathers (diarrhea, sitting on floor) ★ Poor husbandry ● Polyomavirus ● Ectoparasites ● Feather picking, cannibalism ● Feather cysts ● Loss of color (nutritional) ● Baldness (hormonal?)	● Cutaneous pox ● *Knemidokoptes* mites ● Bacterial dermatitis ● Dermatomycoses ● Neoplasia ● Abscesses ● Adiposity ● Otitis ● Conjunctivitis/Blepharitis ● Uropygial gland impaction or neoplasia ● Wounds
Mynah (Sturnidae)	★ Fiber constriction ★ Hyperkeratosis ★ Pododermatitis ★ Fractures ★ Leg band constriction ● Cutaneous pox ● Papillomavirus ● *Knemidokoptes* mites ● Burns ● Frostbite ● Luxations ● Bites from psittacines ● Arthritis ❍ Leg deformities (malnutrition) ❍ Degenerative joint disease	★ *Dermanyssus* (red mites) ★ Molt ★ Spoiled feathers (diarrhea, sitting on floor) ★ Poor husbandry ● Polyomavirus ● Ectoparasites ● Feather picking, cannibalism ● Feather cysts ● Loss of color (nutritional) ● Baldness (hormonal?)	★ Chronic ulcerative dermatitis ● Cutaneous pox ● *Knemidokoptes* mites ● Bacterial dermatitis ● Dermatomycoses ● Neoplasia ● Abscesses ● Adiposis ● Otitis ● Conjunctivitis/Blepharitis ● Uropygial gland impaction or neoplasia ● Wounds

★ Common ● Occasional ❍ Seldom

mg/L of drinking water 5 days a week every week from the moment of diagnosis until after molting has proven to stop production of oocysts, although it will not influence the intracellular stages.[32]

Differential Diagnoses

E. coli, mycobacteria and other bacterial pathogens, *Isospora, Macrorhabdus, Chlamydophila,* polyomavirus, circovirus and toxins must be considered as differentials.

Isospora

The life cycle is completed in the intestinal tract (unlike atoxoplasmosis but like all other Eimeriidae species). Species-specific *Isospora canaria* is common in canaries. Isolates from over 50 species of passerines other than canaries represent different *Isospora* species.[17] Clinical signs are diarrhea and emaciation (Fig 39.31)[32] as well as dilated bowel loops causing abdominal distension. *Isospora* does not always cause clinical disease.

Diagnosis is based on large amounts of oocysts observed on fecal flotation, which, unlike in atoxoplasmosis, are secreted on a continuous basis. Treatment with anticoccidial drugs will be more successful than against *Atoxoplasma* spp. Differential diagnoses include *Atoxoplasma* spp., *E. coli,* mycobacteria, *Macrorhabdus,* other enteric bacteria, toxic enteritis and sudden nutritional change (Table 39.12).

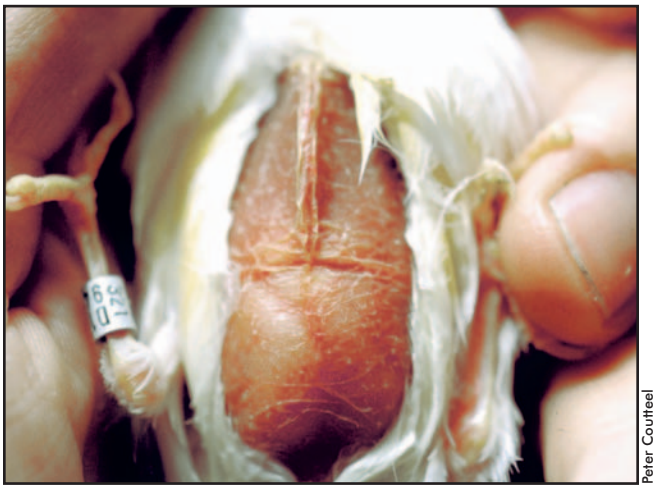

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Fig 39.31 | A canary presented with coccidiosis had emaciation and abdominal swelling.

Cryptosporidium

The clinical significance is not fully understood, but cryptosporidiosis appears to be emerging as a serious disease threat in many avian species, including canaries and finches.[17,81] Autoinfection can occur because of the endogenous sporulation.[13] It may infect and cause disease in the mucosal epithelial cells of the gastrointestinal, respiratory and urinary tracts.[17] The very small oocysts (4 x 8 μm) with 4 sporozoites can be found in fecal flotations. The small size and low numbers of excreted oocysts make diagnosis difficult. Additional diagnostic methods include acid-fast staining, direct immunofluorescence staining or ELISA of fecal samples.[20] Differential diagnoses include other causes of gastrointestinal (see Table 39.12) and respiratory (see Table 39.9) disease.

Sarcocystis

Sarcocystis spp. have an obligatory two-host life cycle. The definitive host is the opossum, whereas a wide variety of avian species can be intermediate hosts.[81] In addition, cockroaches, rats and flies can serve as transport hosts. In the avian intermediate host, merozoites enter striated muscle, where they can be observed macroscopically. Clinical signs can range from inapparent infection to anorexia, diarrhea, weakness, dyspnea, ataxia and death. Diagnosis is based on demonstrating sarcocysts on histopathology of muscle biopsies or after necropsy.

Toxoplasma

Cats and other felids are the only definitive hosts for *Toxoplasma gondii*. Essentially, any warm-blooded animal can be infected as an intermediate host by ingestion of oocysts excreted by cats. Most infections in birds are subclinical and asymptomatic.[81] In the acute phase of infection, birds may show severe respiratory signs.[59] In chronic infections, typical signs include blindness, ataxia and torticollis.[32,67,116] Antemortem diagnosis is difficult and based on an increasing antibody titer. Prevention is based on eliminating direct and indirect contact with cats. Treatment described includes pyrimethamine at 0.5 mg/kg orally every 12 hours for 14 to 28 days or diclazuril at 10 mg/kg orally every 24 hours on days 0, 1, 2, 4, 6, 8 and 10.[59] Differential diagnoses include other causes of central nervous system signs (see Table 39.8).

FLAGELLATE DISEASES

An unidentified flagellate found in the crop has been described as a possible cause of a dermatologic disease in canaries. Clinical signs include feather loss on the crown and neck, often accompanied by signs of dermatitis.[16]

Cochlosoma

Motile flagellates found in the feces of finches have been identified as *Cochlosoma* sp. or *Cochlosoma anatis*-like protozoa, which are classically transmitted by asymptomatically infected society finches, often used as foster parents.[17,42,87] Most commonly affected species include red-headed parrot finches, Bengalese and Lady Gouldian finches.[42]

Clinical Disease

Adult birds are usually asymptomatic carriers. Disease most commonly affects young birds from 10 days to 6 weeks of age.[32] Clinical signs include dehydration, diarrhea, whole seeds in the droppings, yellow staining of nestlings due to contact with their droppings, or death.

Diagnosis

Diagnosis requires demonstration of motile flagellates in saline smears from fresh warm feces.[12] Cochlosoma are smaller than giardia and hard to find in a fecal exam. They can be detected histologically in the intestinal wall (JMM Cornelissen, personal communication).

Prophylaxis and Treatment

All newly introduced adult finches should be screened via fresh fecal smears and, if necessary, treated during quarantine. All breeding finches should be screened and treated, especially foster parents, prior to breeding. Ronidazole can be administered at 50 mg/L drinking water and at 50 mg/kg in soft food for 7 days.[17] Dimetridazole (100 mg/L drinking water for 5 days) may also be helpful. Overdosage of dimetridazole or metronidazole results in reversible CNS signs. Treatment often involves an antibiotic for secondary intestinal infections.

Differential Diagnoses

Differential diagnoses include *Isospora* and other enteric parasites (see Table 39.12), *E. coli,* and other bacterial infections that cause diarrhea and juvenile mortality, *Macrorhabdus* and toxic enteritis.

Table 39.12 | Causes of Diarrhea

	Infectious	Parasitic	Other
Canaries and Finches (Fringillidae)	★ *E. coli* ★ *Macrorhabdus* ★ Mycobacteria ● *Chlamydophila* ● *Yersinia pseudotuberculosis* ● *Campylobacter* ● *Pseudomonas* ● Other bacteria ● *Candida* ❍ PMV-1, -3	★ *Atoxoplasma* ★ *Isospora* ● *Trichomonas* ❍ *Cochlosoma* ❍ *Cryptosporidium* ❍ *Giardia* ❍ *Ascaridia* ❍ *Capillaria* ❍ Cestodes	★ Sudden nutritional change (fruit, vegetables) ● Starvation ● Liver disease ● Toxins ● Kidney disease (polyuria, increased urate excretion)* ❍ Pancreatitis ❍ Neoplasia ❍ Grit over-consumption ❍ Cloacal disease ❍ Diseases of the salpinx
Waxbills (Estrildidae)	★ *E. coli* ★ *Macrorhabdus* ★ Mycobacteria ● PMV-3 ● *Chlamydophila* ● *Yersinia pseudotuberculosis* ● *Campylobacter* (more common than in canaries) ● *Pseudomonas* ● Other bacteria ● *Candida* ❍ PMV-1	★ *Isospora* ★ *Cochlosoma* ● *Trichomonas* ● Cestodes ❍ *Cryptosporidium* ❍ *Giardia* ❍ *Ascaridia* ❍ *Capillaria*	● Starvation ● Liver disease ● Toxins ● Kidney disease (polyuria, increased urate excretion)* ❍ Pancreatitis ❍ Neoplasia ❍ Grit over-consumption ❍ Cloacal disease ❍ Diseases of the salpinx
Mynah (Sturnidae)	★ *E. coli* ● *Chlamydophila* ● *Yersinia pseudotuberculosis* ● *Campylobacter* ● *Pseudomonas* ● Other bacteria ● *Candida* ❍ PMV-1	● *Coccidia* ● *Trichomonas* ● *Ascaridia* ● *Capillaria* ● Cestodes ❍ *Cryptosporidium* ❍ *Giardia*	★ Iron storage disease ● Intestinal foreign body ● Starvation ● Liver disease ● Toxins ● Kidney disease (polyuria, increased urate excretion)* ❍ Pancreatitis ❍ Neoplasia ❍ Grit over-consumption ❍ Cloacal disease ❍ Diseases of the salpinx

★ Common ● Occasional ❍ Seldom *Polyuria and polyurates can create a loose stool but that is not a true diarrhea

Trichomonas

Infections with *T. gallinae* are seen sporadically in canaries and finches.[12,32] Birds of all ages can be affected, but the infection is most predominant in younger birds. Clinical signs include respiratory changes, regurgitation and emaciation. Trichomoniasis can cause thick, yellow caseous lesions of the infraorbital sinus.[66] Diagnosis is based on a fresh wet mount of a crop swab. At necropsy, a thickened opaque crop wall with plaque development is noted (Fig 39.32). Imidazoles are used for treatment as for *Cochlosoma* spp. Differential diagnoses include poxvirus, *Candida* and other causes of respiratory signs (see Table 39.9).

Giardia

Giardia has also been reported in association with gastrointestinal disease in Passeriformes.[71]

BLOOD PROTOZOA

Protozoa are regularly observed in blood smears of clinically healthy captive and free-ranging Passeriformes species and are occasionally associated with primary disease. The most commonly encountered blood protozoa include *Plasmodium* spp., *Hemoproteus* spp., *Leucocytozoon* spp., *Trypanosoma* spp. and *Piroplasma* spp.

MICROSPORIDIA

Encephalitozoon hellem has been diagnosed in a variety of avian species from different orders. Although it has not yet been described in Passeriformes, there is no reason why it should not infect birds of this order.[11]

NEMATODES

Syngamus trachea (Gapeworm or Red Worm)

Syngamus trachea is common in Sturnidae (starlings) and Corvidae (crows) and must be considered in all Passeriformes housed in outdoor aviaries. The life cycle is direct, but earthworms can act as transport and accumulation hosts. Adult worms live in permanent copulation within the lumen of the trachea.[17]

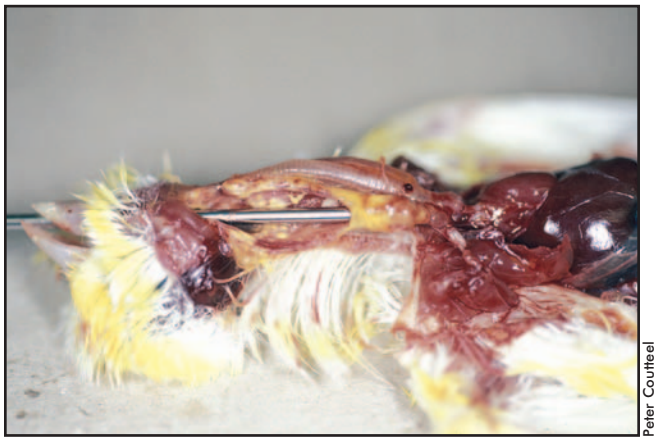
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Fig 39.32 | An oral probe is passed down the opened esophagus in this canary with trichomoniasis. The canary has a thickened crop wall with plaque development.

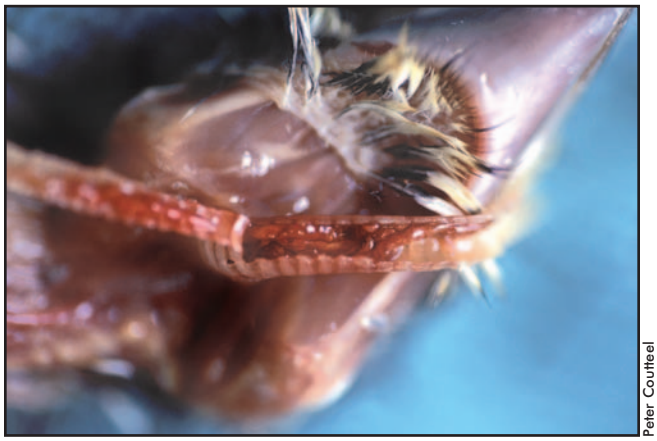
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Fig 39.33 | *Syngamus trachea* (gapeworm or red worm) is shown in a canary's opened trachea.

Clinical Disease

Respiratory distress, gasping, coughing or sneezing is commonly observed in affected birds.[17] Head shaking and dried blood at the beak commissure may also be observed.

Diagnosis

Tracheoscopy or tracheal transillumination reveals adult worms (Fig 39.33). Fecal flotation will reveal ovoid eggs (70 to 100 μm x 43 to 46 μm) with a thick operculum at each pole similar to eggs of *Capillaria* spp. The number of excreted eggs does not correlate with the presence or severity of clinical signs.

Prophylaxis and Treatment

All newly introduced birds must be quarantined and screened by fecal flotation and, if necessary, treated. Contact with natural soil and earthworms must be prevented. A regular annual or biannual worming program should be instituted. Oxygen supplementation should be considered, and dexamethasone administered intratracheally or intramuscularly may be needed to reduce inflammation and mucosal swelling. If size allows, adult worms can be removed with an endoscope and biopsy forceps. Flubendazole, fenbendazole or ivermectin can be used to kill the adult worms that will then be coughed up or swallowed.

Differential Diagnoses

Differential diagnoses include *Sternostoma tracheacolum, Chlamydophila psittaci, Mycoplasma* spp., bacterial rhinitis, tracheitis, pneumonia, aspergillosis, *Trichomonas* spp., *Cryptosporidium* spp. and causes of abdominal distension.

Spiruroidae

Acuaria spp. and *Geopetitia* spp. are most commonly described.[46,64,68] Development proceeds via arthropodal intermediate hosts, such as cockroaches, crickets and mealworm beetles.[46] In the bird, Spiruroidae inhabit the proventricular and ventricular mucosa under the koilin lining.[17] There are no typical clinical signs beyond general malaise and increased mortality. Diagnosis is based on fecal flotation. On necropsy, inflammation of the proventricular and ventricular mucosa is noted. Adult spirurids can be observed under the koilin lining of the ventriculus. Spiruroidae are difficult to treat, with ivermectin most likely to be effective. Differential diagnoses include chronic bacterial enteritis, macrorhabdosis, proventricular candidiasis, coccidiosis, *Cochlosoma* spp. and other helminths.

Other Nematodes

Ascaridia spp. and *Capillaria* spp., commonly observed in Psittaciformes, are of less significance in canaries and finches. *Capillaria* spp. are not only found in the intestines but can also inhabit the oropharynx and crop, causing white plaques. Filarial nematodes, such as *Serratospiculum* spp., *Diplotriaena* spp. and *Splendidofilaria* spp., have been reported in Passeriformes. Most infections are not associated with clinical disease.[71]

CESTODES

All tapeworms require arthropods as intermediate hosts and are most common in finches (eg, parrot finches) that are fed live food.[17,71] Many different species have been described, but in most cases they cause no clinical disease.[71] Clinical signs of disease include emaciation, diarrhea and debilitation. Proglottids or eggs can be demonstrated in fecal flotation samples, but excretion is irregular. Prophylaxis includes limiting access to intermediate hosts and using insect-proof screening. Treatment with praziquantel can be effective.

Fig 39.34 | Tracheal mites (*Sternostoma* spp.) are seen as small black dots in this opened trachea.

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Fig 39.35 | Red mites (*Dermanyssus gallinae*) are shown in the dampened feathers of this canary.

TRACHEAL MITES

Sternostoma tracheacolum should be called tracheal mites and not air sac mites because the mites are normally present in the trachea and syrinx.[71] Common in Lady Gouldian finches and other exotic finches, they are less common in canaries.[17]

Clinical Disease

Clinical signs include wheezing, gasping, open-mouthed breathing, head shaking, loss of voice, cessation of singing and respiratory distress. Mortality is low.

Diagnosis

Tracheal illumination after wetting the feathers may show the mites, recognized as small black spots, in the lumen of the trachea. Lack of visualization does not rule out their presence.[17] The use of the 1.2 mm endoscope is possible, even in the unanesthetized patient. The authors warn that the inexperienced practitioner may kill the bird with this procedure, either by puncturing the trachea or suffocating the bird.

On necropsy, mites can readily be recognized macroscopically within the opened trachea (Fig 39.34).

Prophylaxis and Treatment

Prophylaxis is difficult, as recognition of carriers with a low burden is not possible. One should consider treating all newly acquired birds during quarantine. Individual birds can be treated with ivermectin or doramectin. Ivermectin or doramectin can be diluted with 1:10 with propylene glycol or sesame oil, respectively. The drug is applied as a "spot on" to the bare skin dorsolateral to the thoracic inlet (at the site of jugular blood collection), at the rate of 1 drop per bird up to 50 g and repeated after 7 to 10 days. The potential of propylene glycol toxicity can be avoided by using doramectin that contains sesame oil. It may be helpful to hang a dichlorvos strip near (but out of reach of) the birds.[17]

Differential Diagnoses

For differential diagnoses, consider *Enterococcus faecalis* and other bacterial causes of upper respiratory disease, poxvirus, *Chlamydophila, Atoxoplasma, Trichomonas, Aspergillus* and *Syngamus* (see Table 39.9).

SCALY MITES

Knemidokoptes pilae cause hyperkeratotic lesions on the feet and the base of the beak.[17] Pruritus is noted only by attentive owners. Diagnosis is based on recognizing the small bore holes within the hyperkeratotic lesions or demonstrating the scaly mites in scrapings of the lesions. One may treat locally with plant oil to suffocate the mites in conjunction with ivermectin "spot on". Differential diagnoses include hyperkeratosis, dermatomycoses, papillomavirus, fiber constriction or poxvirus (see Table 39.11).

BLOOD-SUCKING MITES

Dermanyssus gallinae (red mites) hide in nests and other dark areas in the aviary and attack the birds only at night[17] (Fig 39.35). *Ornithonyssus sylviarum* (northern or fowl mite) spends its entire life cycle on the host.[32] This mite can cause high mortality, especially in nestlings. Clinical signs include general depression, anemia, respiratory distress and pruritus. Diagnosis is based on recognizing the mites on the bird or in nests or under perches. The dark excrement of the mites can be found under the nests. During hot weather, the mite population can explode within days. Preventive measures should be initiated before the breeding season. New


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Fig 39.36 | Feather mite (*Analges* spp., *Megninia* spp.) eggs (nits) are shown in the tail feathers of a canary.


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Fig 39.37 | The effects of quill mites (*Syringophilus* spp., *Dermoglyphus* spp.) are fault lines and hemorrhages in the feather's shaft.

nests and perches should be installed and the breeding cages thoroughly cleaned. In aviaries with a known history of red mites, prophylactic use of an insecticide prior to the breeding season may be considered.

The birds may be dusted with a permethrin or carbaryl powder using a salt shaker or sprayed (along with the environment, including the nesting material) with a dilution of carbaryl (5 g 85% carbaryl powder/L water). Fipronil is also effective and well tolerated. Following application of any insecticide, the cage or room should be vacated and thoroughly cleaned with nests and perches replaced with new ones.

OTHER ECTOPARASITES

Quill mites, epidermotic mites (Fig 39.36), and lice of a variety of species have been described in Passeriformes.[32,71] These mites cause general nervousness and feather damage. Quill mites are a major problem for exhibition birds.[17] Many flight and tail feathers develop horizontal lines and hemorrhages in the shaft (Fig 39.37). Diagnosis can be made by opening the base of the shaft of a growing feather and detecting the parasite microscopically. Discovery should cause the owner to rethink general hygiene and management practices. Treatment is similar to that used for blood sucking mites.

Metabolic Diseases

IRON STORAGE DISEASE

Together with aspergillosis, iron storage disease is the most common disorder diagnosed in mynah birds.[1,62] Passerine families with higher incidence of iron storage disease include starlings (Sturnidae), tanagers (Emberizidae), bulbuls (Pycnonotidae) and birds of paradise (Paradisaedae).[36] (Toucans [Rhamphastidae] are not Passeriformes but belong to the order Piciformes and are also commonly diagnosed with iron storage disease).

Various theories exist concerning the pathogenesis of iron storage disease. High iron absorption from the intestines, regardless of body iron stores (similar to hereditary hemochromatosis in humans) is one possibility.[75] High or toxic levels of iron in the diet may be a cause or contributing factor.[21] The incidence of iron storage disease in mynahs has, however, not decreased despite the development of controlled-iron diets. Ascorbic acid in citrus fruits and green leafy plants may enhance the bioavailability of iron; thus, high levels of ascorbic acid (vitamin C) in the diet of captive birds may be a factor.[4] Iron storage disease can, however, also develop in birds specifically fed a diet lacking citrus fruits. Lack of substances such as tannin in the diet of captive birds compared to the high tannin content of water in the rain forests is another possibility. Tannin acts as a natural mineral chelating agent and therefore decreases the bioavailability of iron in the intestinal tract.[119] Concurrent disease conditions could be responsible for the degree and nature of the pathologic changes described in cases of iron storage disease. In avian species not classically considered predisposed to iron storage disease, such as psittacines, the disease is observed histologically in many individuals that succumb to a concurrent disease.[14]

Clinical Disease

Clinical signs include abdominal swelling due to ascites and hepatomegaly, dyspnea due to abdominal swelling, apathy and cachexia, loss of intense coloring of beak and periocular skin in mynahs and sudden death.

Diagnosis

Definitive diagnosis is possible only by histopathology of a liver biopsy sample or quantitative determination of the hepatic iron concentration.[15] It is important to know

the species of a diseased patient. Radiography will show hepatomegaly with or without ascites. Values of liver enzymes, such as AST, LDH and AP as well as bile acids, can be increased. Many mynahs will develop a hypoproteinemia.[62] Total serum iron and total iron binding capacity do not correlate with the presence or degree of iron storage disease in rhamphastids.[118]

Necropsy

A swollen liver with an orange-brown or marbled appearance and transudate within the abdomen may be observed on necropsy. On histopathology, deposition of iron pigment may be seen in hepatocytes and hepatic Kupffer cells, as well as in the spleen, gut wall, myocardium, kidney and pancreas.

Prophylaxis and Treatment

A low-iron diet with less than 60 to 100 ppm (mg/kg) of iron should be fed.[119] Food items containing ascorbic acid, such as citrus fruits, should not be offered, while tannin-rich tea such as oak bark tea may be used to replace drinking water several days a week.

Acute respiratory distress and apathy due to ascites must be relieved by abdominocentesis. Consider diuretics to help resolve ascites. In stable patients, phlebotomies at the rate of 1% of the bird's body weight may be performed once a week. This should induce mobilization of hepatic iron for erythrocyte production. Deferoxamine at 100 mg/kg SC q24h over a 4-month period has been proven to normalize the liver iron concentration in toucans.[15] General supportive liver therapy should be considered, using phytotherapeutics such as dandelion, milk thistle or artichoke, possibly in combination with lactulose (see Chapter 4, Nutritional Considerations).

Differential Diagnoses

Differential diagnoses include *Aspergillus* and other causes of respiratory distress in mynahs (see Table 39.9) and primary hepatic or cardiac disease. For other causes of abdominal distension (see Table 39.10).

OTHER METABOLIC DISORDERS

Amyloidosis of the liver can be observed on necropsy and histopathology of finches such as Lady Gouldian finches.[71] Hepatic lipidosis can be observed in canaries and finches fed a high-fat diet and lacking exercise.

Visceral gout due to hyperuricemia secondary to renal disease is a regular necropsy finding. Diseased birds may show signs of polyuria/polydipsia and apathy, but the most common clinical sign is sudden death. Articular gout is less commonly diagnosed in Passeriformes than in psittacines.

Skeletal malformation caused by hypovitaminosis D, in conjunction with calcium and phosphorus deficiency or imbalances, can be observed in Passeriformes as in all other avian species.

Toxic Diseases

INHALANT TOXINS

Canaries and finches are particularly susceptible to inhalant toxins because they exchange more air per gram of body weight than do larger birds. Dangers include carbon monoxide exposure (cages in car garages, leaks from gas heaters), overheated polytetrafluoroethylene (non-stick cookware), carpet freshener,[29] hair spray, glues, paints and smoke. Disinfectants used in the breeding cages, such as formaldehyde, also pose a hazard. Necropsy reveals nonspecific pulmonary congestion, pulmonary edema and/or hemorrhage (Fig 39.38). Diagnosis is based on a careful history of the bird and its environment.

PLANTS

Plant toxicoses are rare. The majority of ingested plants will merely cause mild gastrointestinal signs. Plants that have proven to result in toxic reactions in canaries include avocado (*Persea americana*), dieffenbachia (*Dieffenbachia* spp.), foxglove (*Digitalis pupurea*), lupine (*Lupinus* spp.), oleander (*Nerium oleander*) and yew (*Taxus media*).[2]

PESTICIDES

Organophosphates inhibit acetylcholinesterase and cause clinical signs such as anorexia, diarrhea, ataxia, tremors and seizures.[3,7] The main source of organophosphates is the inappropriate use of insecticides that are used to combat ectoparasites, such as insecticide spray with an alcohol base administered directly on the bird, insecticide spray used in the nest pans and the addition of insecticide strips to the cage.

PHARMACOLOGIC AGENTS

Many drugs used at doses higher than recommended will cause toxicity in passerines as in other avian species. Drugs with a low therapeutic range that can easily induce signs of toxicity (neurologic signs) in canaries and finches include dimetridazole and furans.[40]

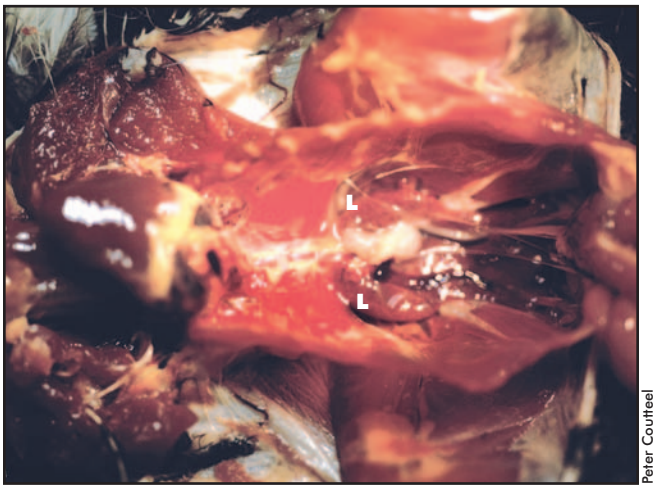

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Fig 39.38 | A canary presented with PTFE (polytetrafluoroethylene) intoxication had acute erythema and edema of lungs (L).


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Fig 39.39 | A feather cyst (arrow) is shown in a canary.

Selected Miscellaneous Diseases

NEOPLASTIC DISEASES

Passeriformes have a low incidence of tumors compared to other avian orders.[71] Reported neoplasms include tumor-like pox lesions,[38] leukosis in canaries[71] and thymoma in a finch.[65]

FEATHER CYSTS

Feather cysts are common in heavily feathered canaries (Norwich, crest and crestbred) and new color canaries.[17] Due to the histologic features, it has recently been suggested that feather cysts in canaries should be considered benign tumors, and the name "plumafolliculoma" has been proposed.[122] The condition appears to be hereditary but other factors may be involved. The most common localizations include the wings (Fig 39.39), on the back, in the shoulder region and on the chest. The cysts can occur individually or in multiple locations. Depending on the stage of the molt, the cysts will contain blood and gelatinous material or dry keratinous material.[17] Individual cysts can be lanced and curetted but will likely recur. A better option is the surgical removal of the entire cyst.[71] Typically, a hemostat is placed at the base of the cyst. The entire cyst is then removed using a radiosurgical unit. Minimal bleeding is controlled using a ferric subsulfate stick or tissue glue. If necessary, a few stitches can be placed. This procedure works well for cutaneous cysts but fails in wing cysts because the origin of the cyst is the germinal epithelium at the base of the feather follicle. On the wing, this occurs at the insertion on a bone.

CHRONIC ULCERATIVE DERMATITIS (CUD)

Chronic ulcerative dermatitis (CUD) is regularly observed in mynahs.[62] The etiology is unknown, but possibilities include metabolic diseases of the liver, malnutrition, polyomavirus, bacterial dermatitis, fungal dermatitis, lipoma, squamous cell carcinoma and giardiasis. Clinical disease is similar to CUD observed in *Agapornis* spp. and budgerigars. It is most common in the axillary body region, but the entire trunk and upper legs can be affected. It is normally pruritic. Treatment is frustrating and includes controlling secondary bacterial and fungal infections, using collars, improving diet and controlling pruritus with local anesthetics or local phytotherapeutic agents such as calendula gel. Adding an antiseptic, such as chlorhexidine in glycerol, to the bath water can help control secondary infections.

FIBER CONSTRICTION OF THE EXTREMITIES

The fibers derived from nesting material are a common cause of digital swelling, inflammation and necrosis of extremities in canaries and finches. Swelling of the feet and legs should be examined using magnification and a good light source to determine if fibers are involved. Fibers should be cut using a pointed scalpel blade or a 26- to 30-gauge needle, and the incisions should be made parallel to the long axis of the legs. Necrotic or mummified digits must be amputated. Hemorrhage is a complication after removal. Local antibiotics and bandaging may be applied.

HYPERKERATOSIS OF THE EXTREMITIES

Hyperkeratosis, which is common in canaries (Fig 39.40), may be caused by genetics (soft feather breeds),

old age, malnutrition and hormonal imbalances.[36] Treatment consists of softening and removing the hyperkeratotic lesions with water soluble creams and addressing predisposing factors, such as malnutrition, and removing leg bands. The differential diagnoses include *Knemidokoptes* mites and papillomavirus-induced warts.

CONSTRICTIONS OF THE EXTREMITIES

Possible causes of constriction of the extremities include leg bands that are too small, hyperkeratosis, swelling due to trauma or inflammation, *Knemidokoptes* mites or pox lesions.[17] Bands may be removed by application of oil and then gentle removal using a special leg band scissors or the drill of a dental unit. The primary disease is treated as necessary.

OTHER DISORDERS OF THE EXTREMITIES

Other disorders of the extremities may include frostbite, injury from wire netting or burns (eg, from hot stove plates, candles or heaters). Missing toenails or toes (bitten off by small psittacines kept in mixed aviaries or overgrown toenails that become entrapped) are common.

EGG BINDING

Pathogenesis and predisposing factors to egg binding are the same as in other avian species. Clinical signs include fluffed-up appearance of a female bird during the breeding period or a female bird sitting continuously in the nest or on the bottom of the cage. The egg can normally be palpated but definitive diagnosis is based on radiology. Conservative treatment, consisting of stabilization of the patient in a warm, humid environment, subcutaneous infusions, parenteral calcium, and PGE_{1or2} applied to the cervix may lead to spontaneous laying of the egg. If this does not occur, manual removal of the egg should be considered as soon as the bird is stable.

Procedure

The bird is induced with isoflurane anesthesia and placed in dorsal recumbency (being cognizant of the increased respiratory effort often present in the egg-bound patient). Warmed lubrication jelly is infused into the cloaca using a 2-ml syringe and a cow teat cannula. Careful digital pressure is applied until the egg can be visualized at the vagino/cloacal junction through the vent. More lubrication jelly is applied between the mucosa of the vagina and the eggshell. The ovocentesis is performed using a 5-ml syringe and an 18-gauge needle. Negative pressure is applied via the syringe at the same time the egg is collapsed by digital pressure through the abdominal wall. Constant, caudally directed digital pressure is applied so the collapsed eggshell can be visualized through the cloaca. Lubrication jelly is continually applied as necessary. Using hemostats, the collapsed eggshell is grasped, and the entire collapsed egg is gently removed. This is normally possible because the broken eggshell is still attached to the shell membrane. Any additional eggshell fragments are removed as necessary. Although this procedure can cause small lacerations, it is still considerably less traumatic than a surgical cesarean. Recovery in a warm humid environment is usually rapid and uneventful.

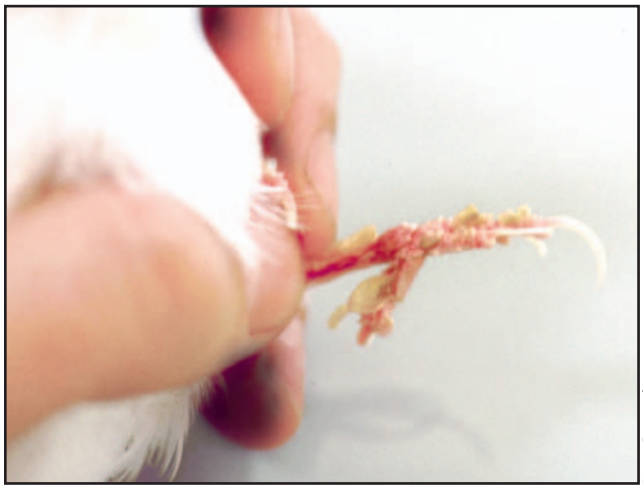

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Fig 39.40 | Hyperkeratosis is shown in the feet of a canary.

OTHER DISEASES OF THE FEMALE REPRODUCTIVE TRACT

Other disorders of the female passerine may include prolapse of the oviduct, excessive egg laying, salpingitis or egg-related peritonitis.

CONCUSSIVE HEAD TRAUMA

A panicked bird flying into a window or wall (often due to the detection of a predator) can induce concussive head trauma (Fig 39.41). Clinical signs include: a depressed bird sitting on the floor; neurological signs such as torticollis, opisthotonos, seizuring, paresis of a wing or leg; blood in the oral cavity, nares or ear canal; ophthalmologic lesions such as blindness, hemorrhage into the anterior or posterior chamber along with uveitis and possibly anisocoria; and sudden death. On necropsy, a hematoma on the skull can be observed. Treatment consists of: controlling swelling and inflammation with manitol; oxygen; controlling seizures (eg, with diazepam); providing supportive care, including adequate fluids (5% dextrose); and maintaining the bird in a cold, dark and quiet environment. Prognosis is guarded to poor, depending on the extent of cranial trauma.

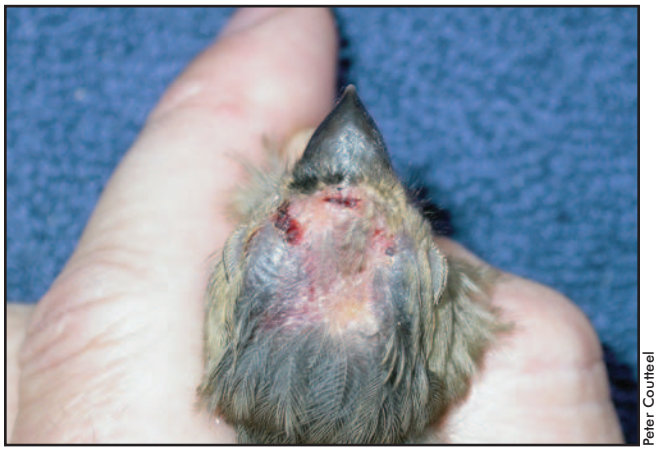
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Fig 39.41 | Trauma on the head of a bullfinch (*Pyrrhula pyrrhula*), due to its head impacting against the wire, is commonly seen with young birds in new cages.


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Fig 39.42 | A rupture of the air sac is shown in this canary.

AIR SAC RUPTURE

Air sac rupture may be caused by trauma (bird flying into a window or wall) or chronic obstructive pulmonary disease. Clinical signs include swelling in the dorsolateral neck area due to rupture of the clavicular air sac (Fig 39.42). Treatment consists of puncturing the air sac swelling with a needle and addressing any primary disease. The likelihood of recurrence after puncturing is high.

Resources Mentioned in the Text

a. Dremel, Racine, WI, USA, 1-800-437-3635, www.dremel.com
b. True-light, Philips Lighting TLD 96 or TL950 series, Osram Biolux, www.lighting.philips.com
c. Poximune, Canary Pox Vaccine, 1-913-894-0230, www.Biomunecompany.com
d. Soludox® 15% water soluble doxycycline in citric acid. Eurovet, www.eurovetanimalhealth.com
e. PCR test for atoxoplasmosis, Department of Medical Microbiology and Parasitology, College of Veterinary Medicine, University of Georgia

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