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7. Diseases of kiwi

7.1 GASTROINTESTINAL TRACT DISORDERS

7.1.1 Foreign body ingestion—traumatic gastritis

Foreign body ingestion is not an uncommon finding in captive kiwi. Metallic foreign bodies (nails, wire, screws, etc.) may cause hardware disease, especially in captive facilities where construction work is being undertaken within enclosures, or where materials are brought in with leaf litter or other substrate. The powerful contractions of the gizzard muscles can easily force sharp objects through the muscular wall (Lumeij 1994). This causes a reduction in ventricular contraction and insufficient digestion of food (Lumeij 1994). Commonly, the foreign body may penetrate the proventricular wall, leading to an acute, generalised purulent peritonitis, or to a local peritonitis with abscess formation on the surface of the proventriculus or gizzard, or duodenum (Lumeij 1994). Occasionally, the foreign object may travel through into the muscles of the thigh. In these cases, lameness or pathological limb fractures may occur (B.D. Gartrell, Massey University, pers. comm.).

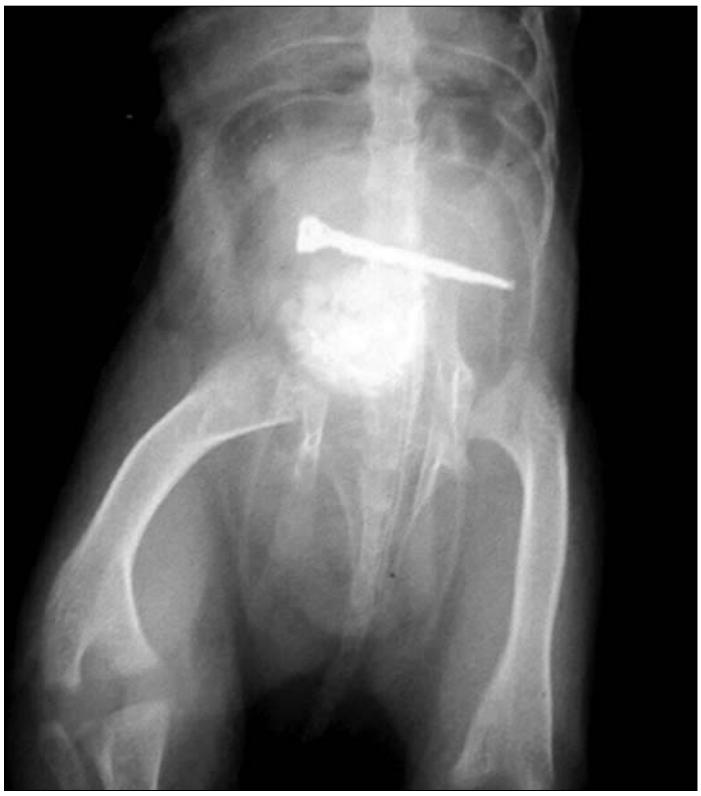


Figure 20. Ventrodorsal radiograph of an adult North Island brown kiwi with a penetrating metallic gastric foreign body. *Photo: T. Kelly.*

Clinical signs of traumatic gastritis include anorexia, weight loss, depression and the passing of undigested food.

Foreign body ingestion may be diagnosed by plain and contrast radiography and gastric endoscopy (Fig. 20). The treatment for large, sharp foreign bodies is removal via proventriculotomy.

To prevent captive kiwi from ingesting metallic foreign bodies, metal detectors can be used to screen substrate material before it is brought into their enclosures.

7.1.2 Gastric impaction

Gizzard and proventricular impaction commonly occurs in other ratites, typically as a result of over-consumption of substrate, including sand and stones (Lumeij 1994). Predisposing factors include gastroenteritis.

Clinical signs of gastric impaction include anorexia, depression, weight loss and scant faeces (Gelis 2006).

Diagnosis includes identification of a full gizzard by palpation, and radiographs demonstrating an excess of grit and stones in the gizzard and proventriculus. A definitive diagnosis of impaction requires the use of contrast radiography, including barium sulphate meals and BIPS™ (barium impregnated polyethylene spheres). Other diagnostics include endoscopy or an exploratory laparotomy (Gelis 2006).

Treatment depends upon the severity of the impaction (Gelis 2006). Medical therapy using psyllium seed husks (Metamucil™) may resolve the impaction by encouraging the movement of gizzard contents. Metoclopramide may also help stimulate small intestinal motility and thus assist in ventricular/proventricular emptying (Gelis 2006). Proventricular flushing, as described for ostriches (Gelis 2006), may help loosen the impaction. This should be performed under general anaesthesia with an endotracheal tube in place to prevent aspiration. Surgical removal of impacted material via a ventriculotomy may sometimes be needed (Gelis 2006).

7.1.3 Candidiasis

Candidiasis (*Candida albicans*) in birds is also known as thrush or sour crop (Dahlhausen 2006). *Candida albicans* is an opportunistic yeast and is not regarded as a primary pathogen (Dahlhausen 2006). In other avian species, including pigeons, the non-budding organism is considered part of the normal gastrointestinal flora (Rupiper 1998).

When severe suppression of the normal gastrointestinal flora occurs, *Candida* spp. can proliferate and cause disease (Dahlhausen 2006). Predisposing factors for candidiasis include antibiotic therapy, stress, poor nutrition, poor hygiene, debilitation or immunosuppression (Dahlhausen 2006). However, infection may be spontaneous in immature animals, or may be secondary to weak or broken mucous membranes (Bauck 1994).

Candidiasis affects the mucocutaneous areas of the body and gastrointestinal mucosa; in particular, the oropharynx and oesophagus (Dahlhausen 2006). The proventriculus and gizzard may also be affected (Bauck 1994). Clinical signs of disease include white or yellow diphtheritic membranes in the oral cavity covered in adhesive mucous (Bauck 1994). Underlying mucous membranes are often inflamed. Infection of the oesophagus, proventriculur or gizzard may cause vomiting or regurgitation, depression, anorexia and poor digestion of food (Bauck 1994; Dahlhausen 2006).

Diagnosis of candidiasis includes cytological examination of affected areas with either Gram's stain or Dif-Quik (Dahlhausen 2006). These may be smears from lesions, or crop washes or faecal samples (Dahlhausen 2006). Candidiasis is characterised by narrowly based budding yeast, oval in shape, measuring 3.5–6.0 × 6.0–10.0 µm (Dahlhausen 2006). The presence of pseudohyphae is indicative of invasive disease (Dahlhausen 2006). Sample swabs may be submitted for culture, and biopsies for histopathology, if necessary.

Treatment includes correction of predisposing factors in combination with antifungal medication. If infection is non-invasive (i.e. no presence of hyphae), oral nystatin (Mycostatin™) can be used at a dose rate of 300 000 IU/kg BID-TID (two to three times daily) for 5–10 days (Dahlhausen 2006). Nystatin is not absorbed from the gastrointestinal tract and thus relies on direct contact by oral or topical absorption. For this reason, lesions in the oral cavity require contact with the nystatin and will not respond to administration of the drug by gavage tube (Dahlhausen 2006).

Severe infections or those invading the gastrointestinal wall may be refractory (noncompliant) to nystatin treatment, and systemic antifungals are indicated. Fluconazole or ketoconazole are the systemic drugs of choice. Itraconazole may also be effective, although some *Candida* spp. are extremely resistant to itraconazole (Dahlhausen 2006).

7.1.4 Bacterial enteritis

Aerobic gram-negative bacteria, in particular those belonging to the Enterobactericeae family, are the most frequently cultured enteric pathogens in birds (Taylor 2000). All produce endotoxins. Potentially pathogenic isolates include *Pseudomonas aeruginosa, Salmonella* spp., *Yersinia* sp., *Klebsiella* sp. and *Proteus* sp. (Taylor 2000). *Salmonella* spp. have been documented in two captive kiwi but not, to date, in wild kiwi (B.D. Gartrell, Massey University, pers. comm.). *Yersinia enterocolitica* has been isolated from several apparently clinically healthy kiwi, both wild and captive, during routine health screening for translocation (C. Travers, Kiwi Encounter, Rainbow Springs, pers. comm.; pers. obs.). *Escherischia coli* causes bacterial enteritis in other avian species. However, when it is present in low numbers it may be considered to be part of the normal intestinal flora of kiwi (B.D. Gartrell, Massey University, pers. comm.).

Gram-positive bacteria, including *Clostridium* spp. (Gelis 2006) also have potential to cause enteritis in kiwi. *Campylobacter* spp. are frequently cultured from clinically normal kiwi during routine health screening, and appear to be non-pathogenic (B.D. Gartrell, Massey University, pers. comm.).

Transmission of pathogenic enteric bacteria occurs by the faecal-oral route. Sources of infection include contamination from other wild birds and overgrowth of a commensal organism secondary to an immunosuppressive disease or antibiotic administration. Infection with *Pastuerella multocida* is a common sequela to predator attacks on birds, as mammalian saliva contains pathogenic bacteria which are then ingested by birds during preening (Quesenberry & Hillyer 1994).

Clinical signs of bacterial enteritis include lethargy, anorexia, diarrhoea (with or without blood), cachexia (weakened body state) and emaciation (Gelis 2006). However, signs are not restricted to the gastrointestinal tract, as septicaemia often follows bacterial enteritis (Gerlach 1994). Joint swelling may indicate infectious arthritis, and ocular lesions, including keratitis and corneal ulceration, may be apparent. Multiple organ infection may occur, including hepatitis (Bauck 1994).

Methods of diagnosing bacterial enteritis include a Gram's stain of a faecal smear and culture and sensitivity of a cloacal or faecal swab.

Treatment of bacterial enteritis involves supportive care, including fluids, warmth and nutritional support. Antibiotics should be selected on the basis of sensitivity testing and in consultation with an avian veterinarian. The treatment of salmonellosis is controversial, as chronic infections are refractory to treatment, and birds may become subclinical latent carriers of the disease (Gerlach 1994). Clinically affected birds should be treated, as should birds being translocated or in contact with humans because of the potential for humans to catch the disease (zoonotic potential) (Gerlach 1994). Clinically healthy kiwi diagnosed with *Yersinia* spp. have previously been left untreated, and remained free of clinical disease (T. Kelly, The Vet Centre, Rotorua, pers. comm.).

Control of enteric bacterial pathogens consists of good hygiene practices, and screening of birds prior to translocation. Access of wild birds to captive kiwi enclosures should be restricted.

7.1.5 Mycobacteriosis

One case of mycobacteriosis has been reported in a captive adult female North Island brown kiwi. Post-mortem examination showed that this bird had classical mycobacteriosis lesions in the liver, spleen, gizzard and intestine (Davis et al. 1984).

Diagnosis of avian mycobacteriosis can be made by demonstration of acid-fast organisms on faecal smears (Gerlach 1994). Repeated samples must be taken because of intermittent shedding of the bacteria (Gerlach 1994). Culture is required for a definitive diagnosis and speciation of the organism, as non-pathogenic mycobacteria may be transient inhabitants of the gastrointestinal tract (Gerlach 1994). Histopathology of mycobacterial granulomas reveals a necrotic foci containing occasional acid-fast organisms (Pollock 2006).

Birds with confirmed mycobacteriosis should be euthanased as there is no effective treatment and there is zoonotic potential (Gerlach 1994; Pollock 2006). Control should focus on identification of affected birds through quarantine and the use of appropriate screening techniques.

7.1.6 Cloacal prolapse

Cloacal prolapses may contain intestines, oviduct or one or both ureters (Lumeij 1994). They may be associated with cloacal masses, neurogenic disorders or conditions causing tenesmus (difficulty passing a stool). These include enteritis, cloacitis, and egg binding. Clinical signs include prolapse of the smooth, glistening pink cloacal mucosa (Lumeij 1994). Examination to rule out involvement of the ureters or uterus should be performed.

Diagnosis should include palpation of the abdomen of female kiwi for the presence of an egg. Examination of the prolapse is best performed under general anaesthesia. Gentle manual massage of the caudal abdominal and cloacal regions may aid in evacuation of faecal material (Lumeij 1994). The cloaca should be examined with (ideally) a rigid endoscope or, alternatively, a speculum and strong light source. Radiographs are useful for identifying masses within the cloaca. A faecal wet mount as well as a Gram's stain and faecal culture may aid in identification of enteritis.

Supportive treatment includes fluid therapy and antibiotics for septic shock. Tissues should be cleaned with a saline solution, covered with a sterile lubricating jelly and replacement of the tissue should be attempted (Lumeij 1994). The prolapse may require retention sutures or a purse-string suture, but only if there is no possibility of egg retention. Care must be taken to ensure that stay sutures do not interfere with evacuation of the cloaca (Lumeij 1994). Chronic cases of cloacal prolapse may require a surgical cloacapexy (not described here).

7.1.7 Cloacal impaction

Impaction of the cloaca has been seen in at least one kiwi chick (pers. obs.). Cloacaliths are typically a cloacal mass of desiccated urates and faecal material, and they may inhibit the passing of excrement, causing congestion of the ureters and intestinal dilatation (Lumeij 1994). Renal failure and visceral gout may occur if the ureters are blocked (Lumeij 1994). Cloacal impactions may be associated with previous egg binding, infectious cloacitis, malnutrition, or neurological disease of the cloaca (Bowles 2006).

Clinical signs of cloacal impaction include depression and anorexia, with scant or no droppings. Abdominal palpation may determine a mass in the caudal abdomen, and radiographs will show a distended cloaca filled with faecal material.

This is an emergency requiring prompt treatment. Therapy includes fluids, antibiotics, anti-inflammatory agents, and gentle manual removal of the cloacolith (Gelis 2006). Manual removal involves the infusion of sterile saline into the cloaca, in combination with manual massage to gently break up the bulk of the impaction which can then be flushed out or manually removed.

Diagnostic procedures for the identification of underlying disease may include cloacal culture and sensitivity, and a thorough neurological examination, including assessment of the vent reflex.

7.2 RESPIRATORY TRACT DISORDERS

7.2.1 Aspergillosis

Aspergillosis has been infrequently diagnosed in kiwi, usually in captive birds. It is most commonly caused by *Aspergillus fumigatus*, although *A. flavis* or *A. niger* may occasionally be isolated (Dahlhausen 2006). *Aspergillus* spp. are ubiquitous in the environment and (for captive birds) are present at particularly high levels in damp, poorly ventilated enclosures, and in mouldy feed or bedding (Dahlhausen 2006). Infection is generally secondary to immunosuppression, and predisposing factors include stress, poor ventilation, malnutrition, antibiotic therapy, concomitant disease and respiratory irritants (Bauck 1994; Dahlhausen 2006). Very young birds are most susceptible, although adults can also be affected.

Transmission occurs via inhalation of infective spores, and the type of disease induced is dependent upon the source and number of spores inhaled, and the immunecompetency of the individual bird. Disease may be local or systemic. Typically, infection occurs within the respiratory tract, including trachea, syrinx, lungs and air sacs. Advancement of lesions to adjacent organs and systemically may also occur (Dahlhausen 2006).

Clinical signs of aspergillosis depend upon the site of infection. Infection involving the lungs and air sacs is the most common presentation, and affected birds may show depression, emaciation, open-mouth breathing, dyspnoea, respiratory distress, wheezing and a change in vocalisation (Bauck 1994). Syringeal aspergillosis has been seen clinically in a captive kiwi (pers. obs.). Renal aspergillosis may present as paralysis or paresis of the hindlimbs

due to compression of pelvic nerve roots by fungal granulomas (Greenacre et al. 1992). Other sites for *Aspergillus* spp. granulomas include the heart muscle, liver, abdominal viscera and the central nervous system (Bauck 1994; Dahlhausen 2006).

Haematology often reveals a marked heterophilia (often 25–100 × 10^9/L) and anaemia (Bauck 1994; Dahlhausen 2006). However, a normal white blood cell count does not exclude aspergillosis. An elevation in liver enzymes and bile acids may also be seen, in combination with biliverdinuria (Dahlhausen 2006). Radiographs may show focal densities in lungs or air sacs, a loss of air sac wall definition, a reduction in coelomic cavity details, or asymmetrical opacities of abdominal air sacs (Bauck 1994).

Endoscopic coelioscopy or tracheal endoscopy may be indicated, and biopsy for culture or histopathology will aid in diagnosis. Cytology of air sac washes may also be performed. Post-mortem examination including cytology, histopathology and culture of lesions will definitively diagnose aspergillus infection (Bauck 1994).

Treatment is dependent upon the location and extent of disease. Itraconazole (Sporanox™) is currently the systemic antifungal medication of choice (10 mg/kg BID PO) (Dahlhausen 2006). Air sac aspergillosis should be treated with nebulisation of an antifungal agent. Treatment options include amphotericin B, enilconazole, terbinafine, clotrimazole and voriconazole. Currently, lack of drug availability in New Zealand restricts our choices to Amphotericin B only. Care should be taken when using amphotericin B, as it is potentially nephrotoxic. Intratracheal flushes with amphotericin B may be indicated for tracheal or syringeal disease (Bauck 1994; Dahlhausen 2006). Surgical debridement of air sac lesions may be required (Dahlhausen 2006). Underlying predisposing factors need to be determined and treated accordingly.

Control of aspergillosis includes reduction of predisposing factors (e.g. stress) (Bauck 1994). Contact with substrates with potential for mould and spore contamination should be reduced, and damp, poorly ventilated enclosures should be avoided (Bauck 1994). Good hygiene practices should be followed at all times, including the feeding of mould-free products (Dahlhausen 2006).

7.2.2 Cryptococcosis

Avian cryptococcosis is a rare disease of birds, and infections may involve the respiratory tract, digestive tract and central nervous systems (Dahlhausen 2006). Cryptococcus bacillisporus (formerly *Cryptococcus neoformans* var. *gatti*) has been reported as causing both pulmonary and disseminated disease in four captive kiwi (Hill et al. 1995; Malik et al. 2003). This particular organism has an ecological association with eucalyptus trees (Dahlhausen 2006), and in one of the reported cases, eucalyptus leaves and twigs had been spread throughout the kiwi enclosure some months prior to the development of disease (Hill et al. 1995).

In the case reported by Hill et al. (1995), the kiwi showed clinical signs 2 hours prior to death, including lethargy, anorexia and dyspnoea. The post mortem showed disseminated disease affecting the heart, kidneys, liver,

pancreas and oviducts (Hill et al. 1995). Histopathology demonstrated a multifocal to diffuse inflammatory reaction associated with cryptococcal organisms (Hill et al. 1995). Unfortunately, lung tissue from this bird was not submitted for examination. In the other cases, all three kiwi died, and at post mortem were found to have extensive granulamatous pneumonia (two cases) or disseminated disease with involvement of the heart, kidneys and proventriculus (one case) (Malik et al. 2003).

It has been proposed that the feeding habits of kiwi would be likely to predispose them to initial pulmonary infection by this disease (Hill et al. 1995).

Diagnosis of cryptococcosis should be based on cytology and histopathology in combination with culture of nasochoanal swabs or washes (Raidal & Butler 2001). Wright's stained smears of gelatinous material obtained from swabs often show aggregates of encapsulated yeast 6–10 μm in length within 8–12-μm non-staining capsules (Raidal & Butler 2001). Histopathology is usually required to confirm cryptococcosis, as it may be carried asymptomatically.

Whether or not to treat a bird with cryptococcosis needs to be considered carefully, as there is significant zoonotic potential associated with this infection (Dahlhausen 2006). Extreme caution is necessary when handling materials that may contain *Cryptococcus* spp. spores. Long-term antifungal treatment of infected birds is required (e.g. daily for 2 months) using amphotericin B, fluconazole or itraconazole (Bauck 1994; Dahlhausen 2006).

7.2.3 Pneumoconiosis

Pneumoconiosis is caused by inhalation of dust particles containing silica, iron and plant material (Boardman 1995). This condition was first reported in kiwi in 1973 (Smith et al. 1973), and is commonly found incidentally in kiwi at post mortem, especially those in captivity. The disease is characterised by the focal accumulation of dust-laden macrophages in bronchial walls (NZWHC 2006). Birds most likely to be affected are those kept on dry sandy substrates containing a large proportion of silica. The olfactory behaviour of the kiwi and the distal placement of their nostrils on the upper bill presumably predispose kiwi to this disease (Smith et al. 1973; Boardman 1995).

7.2.4 Aspiration pneumonia

Birds may aspirate food during tube feeding or following regurgitation (Flammer & Clubb 1994). The lack of a crop and the small capacity of the gizzard and proventriculus make kiwi vulnerable to regurgitation during tube feeding. If large amounts of food are inhaled, rapid placement of an air sac cannula is necessary to prevent asphyxiation (Flammer & Clubb 1994). The prognosis for a bird with this disease is poor. Treatment consists of aggressive antibiotic and antifungal therapy, preferably systemically and via nebulisation (Flammer 1994; Flammer & Clubb 1994). Clinical signs of delayed aspiration pneumonia from inhalation of small amounts of food include poor weight gain, and a persistent leukopenia with or without respiratory signs (Flammer & Clubb 1994). Radiography may assist in diagnosis.

7.2.5 Bacterial pneumonia and air sacculitis

Gram-negative bacteria are commonly isolated from birds with respiratory tract infections (Tully & Harrison 1994). Gram-positive bacteria may also be implicated (Flammer & Clubb 1994). Techniques required for diagnosing pneumonia include auscultation, radiography and endoscopy with or without biopsy.

To date, *Chlamydophilia psittaci* has not been diagnosed in kiwi (B.D. Gartrell, Massey University, pers. comm.). A study of captive kiwi failed to identify *Mycoplasma* spp. by both culture and serum agglutination tests (Christiansen 1996).

7.2.6 Parasitic pneumonia

Migration of parasitic larvae through pulmonary parenchyma has been demonstrated post-mortem in a single kiwi (NZWHC 2006).

7.2.7 Rhinitis

Bacterial rhinitis has been reported post-mortem in a single kiwi (NZWHC 2006). Bacteria commonly isolated from avian species with upper respiratory tract infections include *Pseudomonas* spp., *E. coli, Klebsiella* spp., *Pasteurella* spp. and *Salmonella* spp. (Tully & Harrison 1994). *Aspergillus* spp. granulomas and nasal cryptococcus may cause upper respiratory signs (Dahlhausen 2006).

Clinical signs of upper respiratory tract disease include serous to mucopurulent nasal and/or ocular discharge and sneezing (Dahlhausen 2006).

Diagnosis of rhinitis includes nasal and sinus flushing for cytology and culture. Treatment is dependent upon aetiology, but typically includes antibiotic and/or antifungal medication.

7.3 REPRODUCTIVE DISORDERS

7.3.1 Normal reproduction in kiwi

This section was prepared by Dr B.D. Gartrell, Massey University.

The egg produced by the kiwi is the largest of any bird for its body size, weighing approximately 400 g and comprising 20% of the mass of the adult bird (Fig. 21) (Deeming 1991).

While the laying interval between eggs in kiwi can be 21 days or more, the egg is not carried in the uterus for this length of time. Time from ovulation to oviposition is 7–10 days (Jensen & Durant 2006). The bulk of this time will be used to develop the large, energy-rich yolk follicle on the ovary (Jensen & Durant 2006).

In the wild, kiwi eggs are incubated in a small burrow, and eggs are turned regularly (contrary to early reports; Colbourne 2002). At approximately 80 days, incubation length is the longest for any bird (Vleck & Hoyt 1991). Mean incubation temperature in the wild is 36.5°C at the top of the egg, while the bottom of the egg may be up to 10°C colder (Colbourne 2002). The kiwi egg

has the highest energy content of any bird, with the yolk fraction comprising 65% of egg content and having an energy density of 12.4 kJ/g (wet) (Starck & Ricklefs 1998). The yolk also has the lowest water fraction of any bird, at 61% of egg content (Jensen & Durant 2006). It has been suggested that this is related to the extremely humid conditions of incubation (Vleck 1991). The kiwi embryo expends only approximately 17% of the energy stored in the egg during incubation and 48% of the yolk weight is found in the yolk sac of the hatchling (Prinzinger & Dietz 2002). This yolk is used as the chick's sole source of energy and substrate for tissue production for up to 17 days after hatching (Prinzinger & Dietz 2002).

Incubation behaviour of the parents varies amongst kiwi species (Colbourne 2002). For North Island brown kiwi (*Apteryx mantelli*) and little spotted kiwi (*Apteryx owenii*), the male alone incubates the egg, except for the first week (Colbourne 2002). However, in rowi/Okarito brown kiwi (formerly *A. mantelli* 'Okarito', now *A. rowi*) and tokoeka (*Apteryx australis*), incubation is shared by both sexes (Colbourne 2002). Clutch size ranges from 1 to 3, with eggs being laid from 21 to 66 days apart (Marchant et al. 1991). Multiple clutches are possible, and some North Island brown kiwi are known to lay up to seven eggs a year (Colbourne 2002).

Chicks are precocial, hatching fully feathered and active with a weight of around 300 g for brown kiwi (Marchant et al. 1991). No information is available on hatching weights for great or little spotted kiwi.

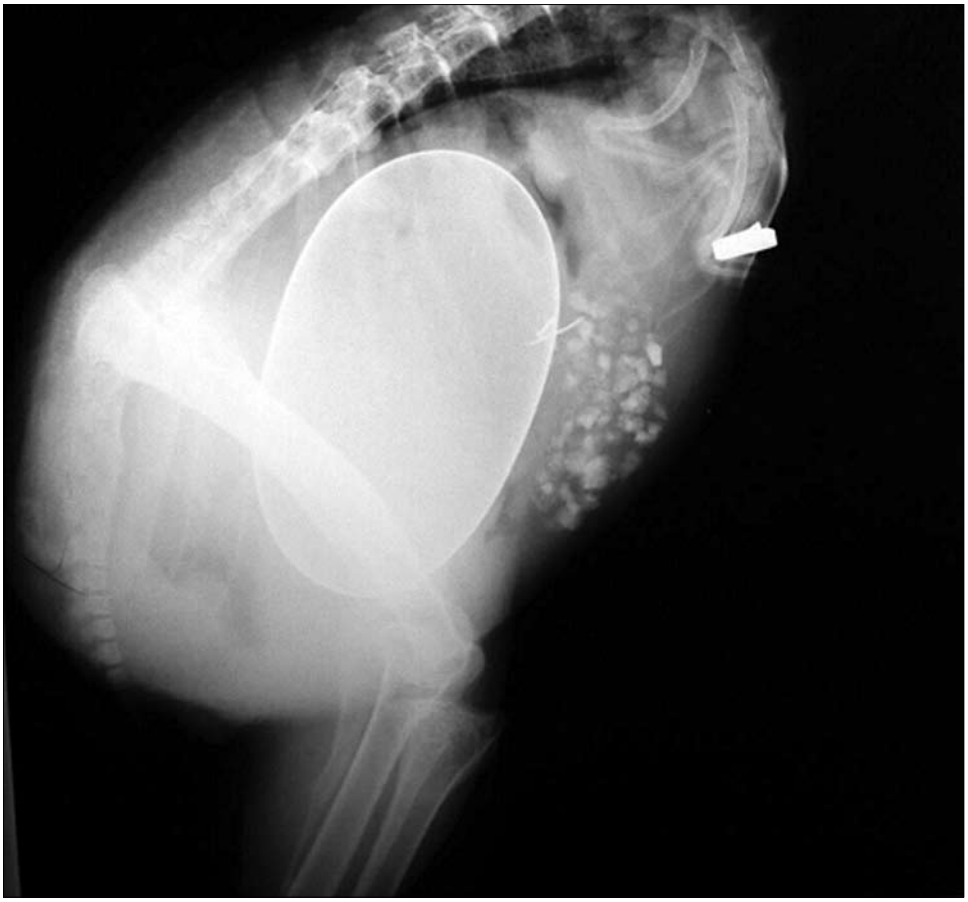


Figure 21. Lateral radiograph of a gravid North Island brown kiwi. *Photo: T. Kelly.*

7.3.2 Egg binding and dystocia

Egg binding is defined as failure of an egg to pass through the oviduct at a normal rate. Dystocia defines a condition in which the developing egg is in the caudal oviduct and is either obstructing the cloaca, or has caused prolapse of oviductal tissue (Joyner 1994).

Given the large relative size of kiwi eggs, problems with egg laying are less common than could be expected. However, dystocia has been observed in captive kiwi and, occasionally, in wild kiwi (NZWHC 2006), and occurs when the developing egg is retained in the oviduct or cloaca.

Common causes of egg binding and dystocia in birds include (Joyner 1994; Bowles 2006):

- Oviduct muscle dysfunction secondary to excessive egg laying
- Calcium-related metabolic disease
- Malformed eggs
- Previous oviduct damage or infection
- Nutritional insufficiencies, including vitamin E and selenium deficiencies and malnutrition
- Obesity
- Lack of exercise and poor muscle strength
- Concurrent stress
- Systemic disease
- Genetic predisposition

Clinical signs of egg binding include depression, anorexia, dyspnoea, an abnormally wide stance and abdominal straining (Joyner 1994). Hindlimb paresis or paralysis may be present due to compression of pelvic nerves by the large egg, and compression of the pelvic blood vessels and kidneys may also occur (Bowles 2006). Sudden death may also be observed (Bowles 2006).

Egg binding may be diagnosed on history and physical examination alone (Bowles 2006). Cloacal examination and radiography may help in diagnosis if the patient's condition is stable enough to allow it to be carried out. Patient stabilisation is the priority of treatment for dystocia (Joyner 1994). This includes fluid therapy and provision of warmth. The cloaca can be lubricated with a water-soluble lubricant (e.g. KY™ jelly), and intramuscular calcium may be given. Analgesia and antibiotics should be considered. Supportive care alone is often enough to allow oviposition, although the bird's condition must be closely monitored for deterioration, in which case further intervention may be required (Bowles 2006).

Once the bird's condition is stabilised, and if the oviduct is believed to be intact and it has been determined that the utero-vaginal sphincter is open, a parenteral prostaglandin may be given to induce oviductal contractions (Bowles 2006). Dinoprost tromethamine (a PGF2alpha) is available in New Zealand. This can be given at a dose rate of 0.02–0.1 mg/kg IM once only (Bowles 2006) and may result in expulsion of the egg. Oxytocin should be used with caution, as complications include oviductal rupture (Joyner 1994).

If medical therapies fail to elict oviposition, manual manipulation of the egg can be attempted in conjunction with lubrication. Gentle pressure should be applied in a persistent, caudal direction. This needs to be gentle to prevent oviduct rupture (Joyner 1994).

Lack of oviposition after an inappropriate period of time, or deterioration in the bird's condition, may indicate the need for more aggressive therapy such as ovocentesis. Ovocentesis involves the aspiration of the contents of the egg with a large needle (18 gauge) (Joyner 1994). Ideally, the egg should be manipulated with the use of a speculum so that it is observable and tapped through the cloaca. If this is not possible, the egg should be brought into juxtaposition to the abdominal wall so that other organs are not damaged during a transabdominal aspiration procedure. After aspiration of the egg contents, the egg can be gently collapsed and the shell fragments and remaining contents of the egg will pass within several days (Joyner 1994). There is some risk of tearing the oviduct resulting in peritonitis, but this appears to be minor. Any shell fragments visible through the cloaca can be gently removed. The uterus may be flushed with an iodine, chlorhexidine or saline flush to help remove egg fragments, and this may decrease the risk of metritis (uterine infection). A course of antibiotics should follow (Joyner 1994).

Other options for treatment of dystocia include an episiotomy if the egg is lodged in the caudal oviduct or cloaca, especially if egg survival is critical. A hysterotomy may be indicated or, alternatively, a hysterectomy in cases where the uterus is ruptured or severe adhesions exist (Joyner 1994). Hysterotomy under isoflurane general anaesthesia has been used to successfully remove a retained egg in a captive kiwi (Figs 22, 23) (T. Kelly, The Vet Centre, Rotorua, pers. comm.).

Post-dystocia complications include a ruptured or necrotic oviduct, peritonitis or abdominal herniation. On at least one occasion, a captive kiwi laying its third egg for the season presented with acute paralysis due to a synsacral spinal fracture (pers. obs.). It is proposed that this may have been secondary to hypocalcaemia (low calcium) with resultant osteopenia (decreased bone density).

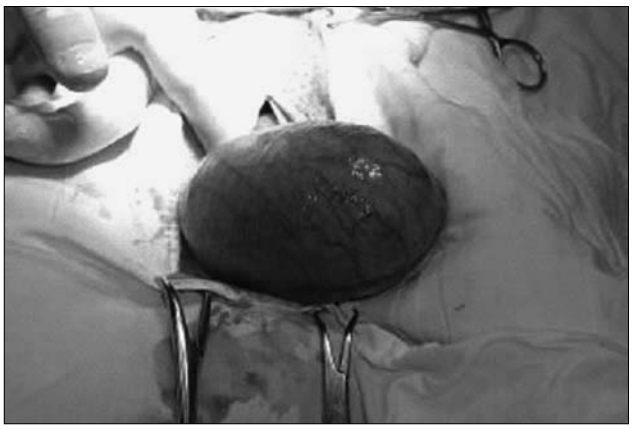


Figure 22. Intra-operative image of kiwi egg within the oviduct during a hysterotomy.
Photo: T. Kelly.

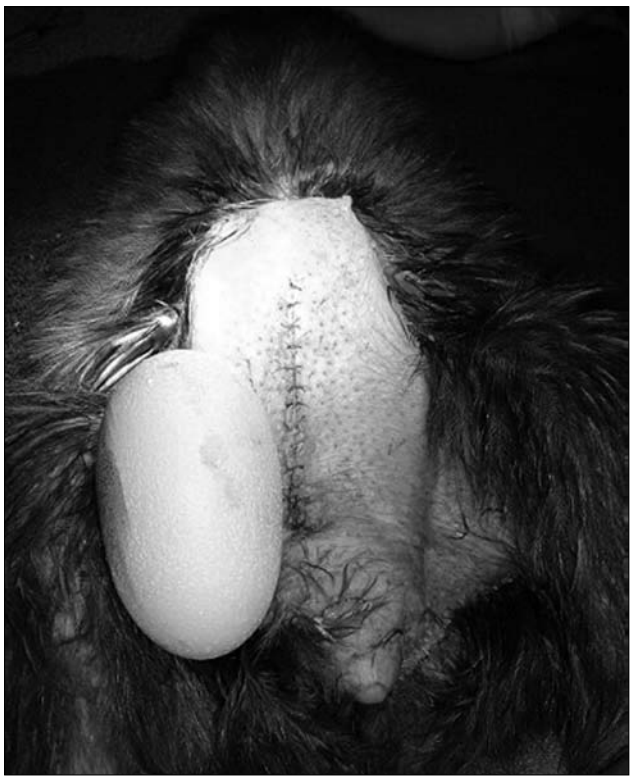


Figure 23. Post-operative image of the bird and egg from Fig. 22.
Photo: T. Kelly.

7.3.3 Oviduct and cloacal prolapse

Prolapse of the oviduct and cloaca can occur secondary to 'normal' egg laying, or as a sequela to dystocia (Joyner 1994). Predisposing factors for prolapse include excessive abdominal muscle contractions, which may be exacerbated by poor physical condition and malnutrition, abnormal or soft-shelled eggs, obesity, salpingitis (oviduct infection) and cloacitis (inflammation of the cloaca) (Joyner 1994; Bowles 2006).

Clinical signs of prolapse include protrusion of the oviduct through the cloaca, often with partial prolapse of the vagina and cloaca (Bowles 2006).

In these situations, rapid aggressive therapy is necessary to prevent tissue devitalisation and secondary infections (Joyner 1994; Bowles 2006). Tissue should be moistened and cleaned with sterile saline solution, and replaced using a lubricated swab and gentle pressure. Stay sutures or a purse-string suture may be required to prevent recurrence. With immediate treatment the prognosis is good (Joyner 1994).

If egg is present in prolapsed tissue, it must be removed before replacing tissue in the abdomen. Surgical debridement may be required to remove the egg if there are adhesions between the shell and the uterus (Joyner 1994).

7.3.4 Salpingitis and metritis

Salpingitis (oviduct infection) and metritis (uterine infection) in birds may occur secondary to air sacculitis (inflammation of the air sacs), pneumonia, liver disease or retrograde infections of the lower uterus, vagina or cloaca (Joyner 1994; Bowles 2006). Predisposing factors include excessive abdominal fat, oviduct impaction and egg-related peritonitis. Most frequently isolated bacterial pathogens include *E. coli*, as well as *Salmonella* spp. and *Pasteurella* spp. (Joyner 1994; Bowles 2006).

Non-specific clinical signs of salpingitis and metritis include depression, anorexia and weight loss. Abdominal enlargement may be visible, and a cloacal discharge may be present. An increased white blood cell count is seen on haematology (Bowles 2006).

Treatment requires aggressive antibiotic therapy and supportive care. The oviduct may be flushed with lactated Ringers solution (LRS). Predisposing factors or underlying causes must be corrected also (Joyner 1994; Bowles 2006).

7.3.5 Egg-related coelomitis

Yolk-related coelomitis (previously called yolk peritonitis) and ectopic eggs have been seen in both captive and wild kiwi (Boardman 1995; NZWHC 2006) (Fig. 24). Egg-related coelomitis may be septic or non-septic. Causes of yolk-related coelomitis include ectopic ovulation, oviduct rupture and salpingitis (Joyner 1994). Ectopic ovulation relates to a failure of the oviductal fimbrium to pick up the ovulating follicle with a subsequent spread of yolk material through the abdomen (Joyner 1994). The yolk may be reabsorbed if it is free of pathogens. However, yolk is an ideal environment for bacterial invasion, in particular with coliform bacteria, creating a septic coelomitis (Joyner 1994).

Figure 24. Ectopic yolk from a North Island brown kiwi, diagnosed at post-mortem. *Photo: B.D. Gartrell.*

Clinical signs of egg-related septic coelomitis include abdominal swelling, respiratory distress from air sac compression, depression and anorexia (Joyner 1994; Bowles 2006). Sudden death may occur. A severe leucocytosis (increased white cell count) is seen on haematology (Joyner 1994).

Survey radiographs may aid in diagnosis of yolk-related coelomitis. Coelomocentesis may reveal a turbid yellow, green or brown yolky fluid, or cheese-like masses of inspissated yolk material (Joyner 1994). Cytology may reveal a septic or non-septic exudate, a transudate, or yolk or fat globules, and bacteriology should be performed on coelomic fluid samples (Bowles 2006). Coelomotomy may be necessary to obtain a definitive diagnosis (Bowles 2006).

Treatment involves supportive care with fluids, broad spectrum antibiotics and analgesia (Joyner 1994). Coelomocentesis is not only diagnostic, but it may also be therapeutic by relieving dyspnoea (Bowles 2006). In other avian species, treatment of severe cases of yolk-related coelomitis may include exploratory coelomotomy and removal of yolk material (Bowles 2006).

7.3.6 Oophoritis

Oophoritis refers to inflammation of the ovary, and it may result from neoplastic (abnormal cell growth), mechanical or infectious causes (Bowles 2006). Clinical signs associated with oophoritis may include depression, anorexia, chronic wasting and sudden death (Bowles 2006). Diagnosis requires endoscopic evaluation of the ovaries, and treatment may include supportive care and antibiotic therapy, if indicated (Joyner 1994).

7.4 YOLK SAC DISORDERS IN CHICKS

The yolk sac is a diverticulum (bulge or pouch) of the intestine, situated at the junction of the jejunum and the ileum (O'Malley 2005). The yolk sac is normally internalised into the abdomen just prior to hatch, after which the yolk is absorbed through the yolk stalk, providing the chick with nutrition, minerals, fat-soluble vitamins as well as maternal antibodies (Flammer & Clubb 1994). Kiwi are precocial birds, meaning that they are born covered in feathers and able to see, walk and feed themselves at hatching (Flammer & Clubb 1994). They have to fend for themselves from early on and thus rely entirely on absorption of yolk to provide nutrition until they have learned to obtain adequate food from foraging (O'Malley 2005). Once the yolk is absorbed, usually from 10 days post hatching, only a small remnant of scar tissue should remain (the vitelline or Meckel's diverticulum) (O'Malley 2005).

7.4.1 Yolk sacculitis and yolk sac retention

Yolk sacculitis (bacterial infection of the yolk sac), is a multifactorial problem seen in ratite chicks usually less than one week old (Doneley 2006). Yolk sac retention is a failure of the yolk sac to be resorbed in the absence of infection (Doneley 2006). Yolk sacculitis and yolk sac retention have been seen in kiwi chicks artificially incubated and reared in the Operation Nest Egg programme (C. Travers, Kiwi Encounter, Rainbow Springs, pers. comm.).

In captive-raised ratites, yolk sacculitis usually arises from poor hatcher or brooder hygiene, and chicks that have been stressed by incorrect incubator/hatcher temperature and/or humidity are predisposed to bacterial infection (Doneley 2006). Weak or undersized chicks are most at risk (Doneley 2006). Transovarial and transoviductal infections may occur, as well as infection from shell contamination (Doneley 2006). Yolk sacculitis often occurs if the yolk sac is poorly internalised prior to hatching, and is primarily caused by gram-negative bacteria, including *E. coli*, *Proteus* spp., and *Streptococcus fecalis* (Flammer & Clubb 1994). *Clostridium* spp. have also been implicated (Flammer & Clubb 1994). Clinical signs of yolk sacculitis include a thickened, prominent, inflamed and necrotic umbilicus, and affected chicks lose weight and become systemically ill (Stewart 1994).

Yolk sac retention in ratites is usually associated with management problems, including incubator or hatcher problems, or chick nutrition and exercise (Doneley 2006). Clinical signs of failure to thrive and weight loss are usually not evident until the chick is 2–6 weeks old, and are a result of the release of toxic substances by the autolysing yolk (Flammer & Clubb 1994; Doneley 2006). Kiwi chicks will normally lose weight for the first 10 days after hatching and then begin to grow rapidly (C. Travers, Kiwi Encounter, Rainbow Springs, pers. comm.). Kiwi chicks with a retained yolk sac fail to thrive and any chick which does not steadily increase in body weight should be investigated for yolk sac retention. In ratites, the retained yolk sac feels enormous, is usually palpable as a doughy structure, and may be 15–40% of the total body weight (Stewart 1994). Other clinical signs of yolk sac retention include dyspnoea and exercise intolerance resulting from air sac compression, depression, anorexia and recumbency (Flammer & Clubb 1994).

Diagnosis of a yolk sac disorder may include radiographs showing a visibly enlarged yolk sac, or ultrasonographic demonstration of the large yolk sac within the chick's abdomen (Doneley 2006). Contrast radiographs with a barium meal are particularly helpful in diagnosing a retained yolk sac. Fine needle aspiration of the yolk sac is contraindicated as the yolk sac wall is very thin, and the needle puncture will cause leakage of yolk into the peritoneal cavity, causing yolk-related peritonitis (Flammer & Clubb 1994).

Initial treatment of yolk sacculitis involves aggressive supportive care. As antibiotics do not penetrate the yolk sac, treatment for both yolk sacculitis and yolk sac retention involves surgical excision of the yolk sac (Doneley 2006). The technique in ostriches (from Stewart 1994) is described below (see also Fig. 25A–F).

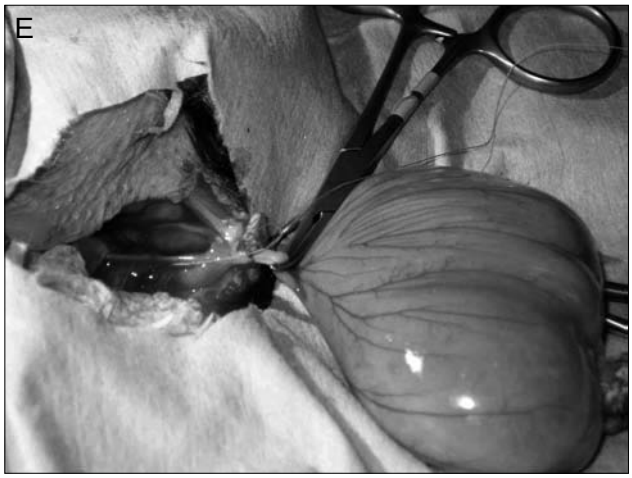
Pre-anaesthetic considerations should include patient stabilisation with fluid therapy, antibiotics, and correction of hypothermia and hypoglycaemia. Pre-surgical analgesia should be considered. Anaesthetic considerations should

A. Kiwi chick prepared for yolk sac surgery.

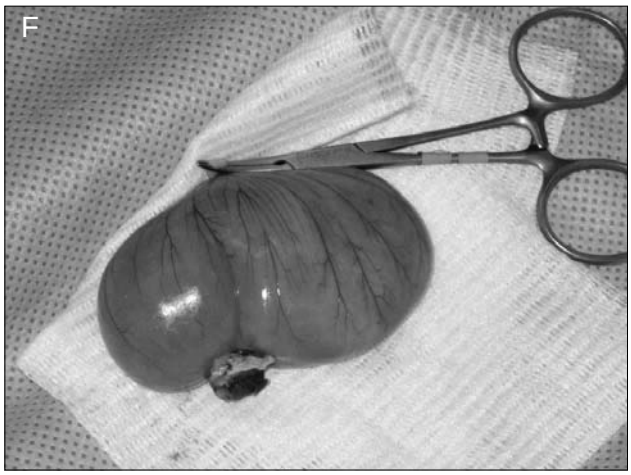
B. The skin incision is made adjacent to the umbilicus.

C. Resection of the umbilicus to expose the underlying yolk sac.

D. Exteriorisation of the yolk sac.



E. Ligation of the yolk stalk.



F. The removed entire yolk sac.

Figure 25. Removal of a retained yolk sac in a North Island brown kiwi chick. *Photos: V. Gray.*

include fluid and heat therapy and, very importantly, minimising surgical time. This requires appropriate organisation prior to anaesthetising the bird.

The bird should be placed in dorsal recumbency. Feathers are plucked over the abdomen at the level of the umbilicus, and the area is prepared for surgery. Alcohol-based surgical preps must not be used, as these cause heat loss. Chlorhexidine aqueous solution may be used instead.

A circumferential incision should be made around the umbilicus, extending transversely at 3 and 9 o'clock to a lateral distance required for easy removal of the intact yolk sac. Alternatively, a longitudinal incision can be made, as shown in Fig. 25. The body wall should be incised in a corresponding pattern, taking care not to damage the underlying yolk sac. The yolk sac is exteriorised by applying gentle traction on the umbilical stump. The yolk stalk should be clamped and clipped or ligated just distal to the intestines, and the yolk sac removed. The body wall should be closed with a monofilament absorbable suture (e.g. Vicryl™ 5-0) in a simple continuous pattern (to minimise surgical time).

Post-surgical antibiotics should be continued for at least 7 days. Broad spectrum antibiotics should be initiated pending the results of culture and sensitivity. These should be effective against gram-negative bacteria, and first choice antibiotics may include amoxicillin/clavulonic acid, enrofloxacin and trimethoprim-sulfa (Stewart 1994). Butorphanol analgesia should be continued post-operatively. Chicks can be offered a small amount of food in the evening following surgery. Most kiwi chicks will be eating within 2–3 days after the operation, but others may require assist feeding for some time (C. Travers, Kiwi Encounter, Rainbow Springs, pers. comm.).

7.4.2 External yolk sac

Yolk sac externalisation occurs if the umbilicus does not close properly by the point of hatch and a portion of the yolk is exposed (Doneley 2006). Causes of this include high incubation temperature, resulting in premature hatching of the chick, poor gas exchange (as with high humidity), or egg infections (Stewart 1994).

Treatment of mild cases involves gentle cleaning of the yolk sac with dilute iodine, and replacement of the yolk in the abdomen. The umbilicus should be covered with an antibiotic ointment and a light gauze dressing, and the abdomen should be wrapped in a self-adherent bandage. (Stewart 1994; Doneley 2006). Severe yolk sac externalisations require surgical removal as described above. Prognosis is poor (Stewart 1994).

Broad-spectrum antibiotics are necessary, and affected chicks should be carefully monitored for yolk sacculitis or yolk sac retention (Doneley 2006).

7.5 NEUROLOGICAL DISEASES

7.5.1 Heavy metal toxicosis

Ingestion of metallic objects, particularly galvanised nails and screws, can result in zinc, copper or lead intoxication, as well as problems associated with the foreign body penetrating tissue (see section 7.1.4). The metal object is degraded by grinding in the muscular gizzard, and by leaching in gastric

acids, with subsequent absorption of the heavy metals into the bloodstream where they become a toxin, causing pansystemic damage (Dumonceaux & Harrison 1994).

Non-specific clinical signs of heavy metal toxicosis include lethargy, weakness, anorexia and emaciation. Regurgitation and diarrhoea often occur, and birds may be polyuric. Neurological signs may include ataxia, blindness, a head tilt, circling, paresis/paralysis, head tremors and convulsions (Dumonceaux & Harrison 1994). Untreated, heavy metal toxicosis is often fatal.

Radiography of the gastrointestinal tract may reveal a metallic foreign body in the gizzard. A commercial veterinary laboratory can detect the presence of blood lead—the presence of any lead is abnormal. Serum or plasma zinc and copper assays can also be determined. Both these are essential dietary trace elements, and results should be compared with reference ranges. Tissue heavy metal levels can also be determined at post mortem.

Treatment of heavy metal toxicosis requires chelation with CaEDTA (35–50 mg/kg BID IM) (Carpenter 2005). It is recommended that affected birds be treated for 5 days, treatment stopped for 2 days, and blood analysed again. Treatment may need to be repeated if subsequent blood analysis indicates that it is necessary (Carpenter 2005). As chelated metals are excreted renally, concurrent fluid therapy is essential to prevent nephrotoxicity (Richardson 2006). Passage of small, smooth metal particles may be assisted by gavage-feeding of psyllium seed husks (Metamucil™) in combination with oral fluids (Dumonceaux & Harrison 1994). Larger or sharp metal objects may require endoscopic retrieval. Often, a ventriculotomy is required, especially if the foreign body is sharp and has penetrated the muscular wall of the gizzard (Dumonceaux & Harrison 1994).

7.5.2 Neural larval migrans

A clinical case of neural larval migrans was seen in an adult kiwi at Auckland Zoo, and several further cases have been identified on post-mortem in free-living birds (Morgan et al. 2005). Neural larval migrans is generally caused by the ingestion of ascarid larvae by an accidental host. *Toxocara cati* is a possibility, as this ascarid has been identified in a kiwi from Hauturu/Little Barrier Island (Clark & McKenzie 1982).

Clinical signs of neural larval migrans may include torticollis, ataxia, depression and death (Bennett 1994). Diagnosis is made on demonstration of migrating larvae within neural tissue at histopathology.

7.5.3 Karaka berry toxicosis

Karaka (*Corynocarpus laevigatus*) berries have been implicated as a potential cause of neurological disease in captive kiwi (Shaw & Billing 2006). Karaka fruits contain a toxin (karakin), and clinical signs of karakin toxicity are weakness, hind leg paralysis and convulsions (Parton et al. 2001). Karaka toxicosis was not definitively diagnosed as the cause of the neurological symptoms in these cases.

7.6 NUTRITIONAL DISEASES

7.6.1 Obesity

Obesity is a common problem in captive kiwi. Many kiwi are fed a diet too high in calories for their sedentary lifestyle. It is recommended that fat be removed from ox heart prior to making up the kiwi artificial diet (see Appendix 4). Obesity can lead to reproductive problems; in particular, predisposing females to egg binding.

7.6.2 Vitamin B responsive dermatitis

A suspected vitamin B responsive dermatitis has been seen in captive kiwi (B.D. Gartrell, Massey University, pers. comm.). A similar abnormality has been recorded in wild-hatched North Island brown kiwi chicks simultaneously parasitised with *Babesia*, and resolved with the addition of biotin to the diet (Doneley 2006).

Treatment of this dermatitis includes dietary alteration and appropriate antibiotics if indicated. Supplementation of the captive diet with a vitamin B rich formulation (i.e. kiwi premix) may prevent this disorder.

7.7 MUSCULOSKELETAL DISORDERS

7.7.1 Tibiotarsal rotation

Also called angular limb deformity, this is one of the most common and serious limb deformities seen in ratite chicks, including kiwi chicks (Figs 26, 27) (Doneley 2006). In severe cases, tibiotarsal rotation may be such that the chick has difficulty in walking and standing. In other ratites, this condition most likely has a multifactorial aetiology that may include diets high in protein and/or energy, calcium/phosphorous imbalances, leg injuries and lack of exercise (Doneley 2006).

7.7.2 Splay leg

In kiwi chicks, splayed legs may occur when they are brooded on a slippery surface that does not afford adequate traction (Figs 26, 27). This condition can be corrected with hobbles if identified in its early stages, and is prevented by the use of non-slip surfaces Doneley 2006.

Figure 26. Bilateral tibiotarsal rotation in a North Island brown kiwi chick. *Photo: C. Travers.*

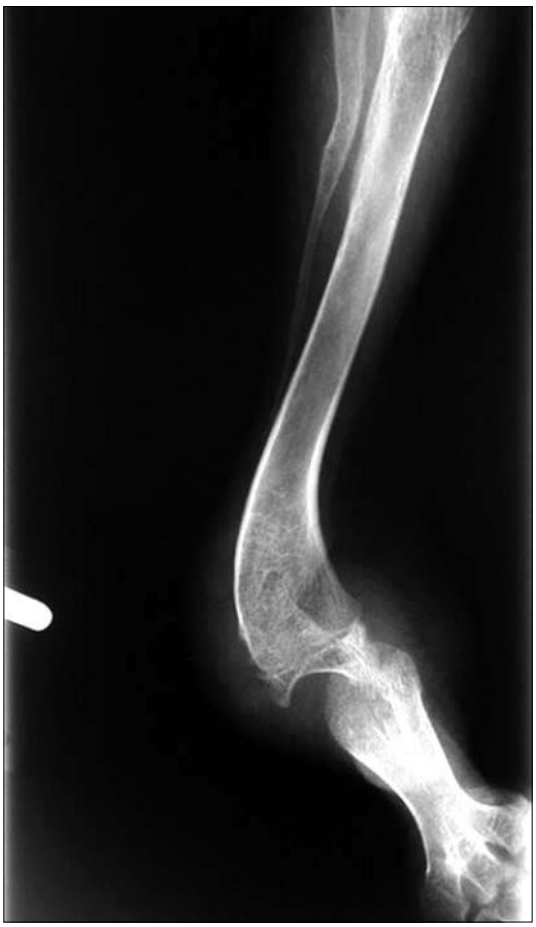


Figure 27. Hindlimb radiograph from the chick in Fig. 26, showing rotation of the tibiotarsus. *Photo: B.D. Gartrell.*

7.8 PARASITES

There have been numerous helminth and protozoal parasites described in kiwi, mostly in scant detail (Table 2).

7.8.1 Endoparasites

Helminths

Helminths described in the kiwi include nematodes, cestodes and trematodes (McKenna 1998). Very little work has been done to determine the relative pathogenicity of these parasites to kiwi. Many parasites coexist with their avian host without causing pathological changes (Greiner & Ritchie 1994). In larger numbers, especially in immunosuppressed or immature animals, infection has the potential to cause disease. This may manifest as anorexia, weight loss, diarrhoea, malabsorption, intestinal obstruction, intussusception and death (Greiner & Ritchie 1994).

Diagnosis by a faecal floatation in saturated salt solution will enable a faecal egg count. Wet faecal smears may detect the presence of eggs without quantification (Greiner & Ritchie 1994).

As in mammals, eosinophilia (high numbers of eosinophil white blood cells in the blood) is occasionally reported to be associated with gastrointestinal parasitic infections in birds. However, this association appears to be inconsistent and it has been difficult to induce the condition experimentally in avian species (Maxwell 1980).

TABLE 2. ENDOPARASITES IDENTIFIED FROM KIWI.

PARASITE SPECIES	PARASITE CLASSIFICATION	KIWI SPECIES PARASITE HAS BEEN ISOLATED FROM	DISTRIBUTION IN HOST SPECIES
Acuaria sp. (Orr 1994)	Nematoda	*Apteryx* sp.	
Ascaris apterycis (Chahn 1884)*	Nematoda	*Apteryx australis mantelli*	Intestine
Capillaria sp. (Clark 1983)	Nematoda	*Apteryx* sp.	
Cyrnea (*Cyrnea*) *apterycis* (Clark & McKenzie 1982)*	Nematoda	*Apteryx australis mantelli*; *Apteryx* sp.	Gizzard
Heterakis gracilicauda (Clark & McKenzie 1982)*	Nematoda	*Apteryx australis mantelli; Apteryx* sp.	Caecum
Porrocaecum ensicaudatum (Clark 1983)	Nematoda	*Apteryx* sp.	Intestine
Porrocaecum sp. (Clark & McKenzie 1982)*	Nematoda	*Apteryx australis mantelli*	Gizzard
Tetrameres sp. (Clark 1983)	Nematoda	*Apteryx* sp.	
Toxocaria cati (Clark & McKenzie 1982)*	Nematoda	*Apteryx australis mantelli*	Small intestine
Anomotaenia minuta (Benham 1900)*	Cestoda	*Apteryx australis mantelli*	Duodenum
Davainea sp. (Reid & Williams 1975)*	Cestoda	*Apteryx australis mantelli*	Intestine
Paricterotaenia apterygis (Benham 1900)*	Cestoda	*Apteryx australis mantelli*	Intestine
Rallietina sp. (Reid & Williams 1975)*	Cestoda	*Apteryx australis mantelli*	Intestine
Taenia apterycis (Chahn 1884)*	Cestoda	*Apteryx australis mantelli*	Intestine
Lyperosomum megacotylosum (Andrews 1977)*	Trematoda	*Apteryx australis mantelli*	Small intestine
Echinorhynchus sp. (Benham 1900)*	Acanthocephala	*Apteryx australis mantelli*	Intestine
Eimeria sp. (Thompson & Wright 1978; Clemence 1995; Hartley 1995; Morgan 2004; W.A. Charleston, pers. comm. 2005)	Protozoa	*Apteryx australis mantelli*; *Apteryx* sp.	Intestine, kidneys, liver, pancreas, bile duct
Toxoplasma sp. (Orr & Black 1996)	Protozoa	*Apteryx owenii* (little spotted kiwi)	Intestine, kidneys, liver

* References are cited from Weekes 1982.

Anthelmintic treatment for helminth parasites may include moxidectin, ivermectin, fenbendazole and pyrantel/praziquantel combinations (see Appendix 6).

The use of levamisole has been associated with a high level of mortality in kiwi. Kiwi appear to be sensitive to levamisole toxicity at doses that are well within the safe range for domestic poultry. Levamisole should not be used as an anthelmintic in kiwi (Gartrell et al. 2004).

Coccidia

Coccidiosis is a naturally occurring disease in North Island brown kiwi, having been described in both free-living and captive birds (Jakob-Hoff et al. 1999; Morgan 2004; NZWHC 2006). It was first reported in 1978 (Thompson & Wright 1978), and both intestinal and renal coccidial forms have been identified (Morgan 2004).

Coccidiosis, which infects primarily juvenile birds, is the main disease affecting kiwi in captivity (Morgan 2004). Several species of coccidia, including *Eimeria* spp., infect kiwi gastrointestinal and renal systems (Morgan 2004; W.A Charleston, Massey University, pers. comm.). Hepatic, biliary and pancreatic coccidial infections have been seen in severe infections (Clemence 1995; Hartley 1995; Morgan 2004).

Transmission of coccidia is by the faecal-oral route. Oocysts are highly resistant to most environmental conditions and to disinfectants, and may survive for extended periods in the environment (Morgan 2004).

Birds infected with small numbers of coccidia may be asymptomatic. Coccidia are commonly shed in small numbers by free-living kiwi (Jakob-Hoff et al. 1999). Higher burdens of the parasite may result in birds developing severe clinical disease, including diarrhoea, anorexia and weight loss, and can result in sudden death (Greiner & Ritchie 1994; Morgan 2004).

Diagnosis requires evaluation of excrement to detect oocysts shed from either the gastrointestinal or renal tract. There is some evidence to suggest that there is a diurnal variation in oocyst shed (pers. obs). For this reason, pooled samples from throughout at least a full 24-hour period are recommended for examination via a faecal floatation (collection over 3–4 days is a commonly used technique) (pers. obs). If there are high numbers of organisms, oocysts may be detectable on a wet faceal preparation. Oocysts of coccidia infecting kiwi are either round or oval, diameter 12–15 µm, length 18–20 µm (unpubl. data).

Treatment with oral toltrazuril (Baycox™) at 20–25 mg/kg is recommended in birds with clinical disease attributable to coccidial infections (Carpenter 2005).

Prevention of infection requires reduction of environmental contamination and sound hygiene practices.

7.8.2 Haemoparasites

Babesia kiwiensis and *Hepatozoon kiwi* have recently been diagnosed in North Island brown kiwi (Pierce et al. 2003).

Babesia kiwiensis is an intracytoplasmic erythrocyte (red blood cell) parasite. To date there has been little study of this haemoparasite; but currently, there is no indication that *B. kiwiensis* is pathogenic in adult kiwi (Pierce et al. 2003). However, in kiwi chicks concomitantly debilitated by heavy burdens of other external and internal parasites, *B. kiwiensis* appears to have caused a pyrexia characteristic of babesiosis in other species (Pierce et al. 2003). Parasitaemia (parasites present in the red blood cells) is usually less than 1% in adult carriers. The vector is likely to be the kiwi tick, *Ixodes anatis* (Pierce et al. 2003).

Hepatozoon kiwi infects mononuclear leucocytes (usually monocytes, but also lymphocytes) and has not been associated with pathology to date (Pierce et al. 2003).

7.8.3 Ectoparasites

Many kiwi, especially those in the north of the North Island, are infected with ticks. *Ixodes anatis* is the most commonly found tick species on brown kiwi, although the cattle tick, *Haemophysalis longicornis*, can also infect kiwi. Ticks are frequently found around the head region of the birds, including the ear canals (pers. obs.). Table 3 lists ectoparasites recorded from kiwi.

TABLE 3. ECTOPARASITES RECORDED FROM KIWI.
Data in this table were compiled by A.C.G. Heath, AgResearch, Wallaceville, November 2006.

PARASITE SPECIES	PARASITE CLASSIFICATION	KIWI SPECIES PARASITE HAS BEEN ISOLATED FROM
Kiwialges phalagotrichus (Gaud & Atyeo 1970)	Feather mite	*Apteryx australis* (brown kiwi)[a] *Apteryx mantelli* (North I. brown kiwi) *Apteryx haasti* (great spotted kiwi) *Apteryx australis australis* (South I. brown kiwi) *Apteryx australis lawryi* (Stewart I. brown kiwi) *Apteryx owenii* (little spotted kiwi)
Kiwilichus delosikyus (Gaud & Atyeo 1970)	Feather mite	*Apteryx australis* (brown kiwi)[a]
Kiwialges haastii (Bishop 1985)	Feather mite	*Apteryx haasti* (great spotted kiwi)
Kiwialges palametrichus (Gaud & Atyeo 1970)	Feather mite	*Apteryx mantelli* (North I. brown kiwi) *Apteryx haasti* (great spotted kiwi) *Apteryx australis lawryi* (Stewart I. brown kiwi) *Apteryx owenii* (little spotted kiwi)
Kiwilichus cryptosikyus (Gaud & Atyeo 1970)[b]	Feather mite	*Apteryx haasti* (great spotted kiwi) *Apteryx australis australis* (South I. brown kiwi) *Apteryx australis lawryi* (Stewart I. brown kiwi)
Apterygon hintoni (Clay 1966)	Chewing louse	*Apteryx haasti* (great spotted kiwi)
Rallicola (*Aptericola*) *gracilentus* (Clay 1953)	Chewing louse	*Apteryx haasti* (great spotted kiwi)
Rallicola (*Aptericola*) *gadowi* (Harrison 1915)[c]	Chewing louse	*Apteryx australis australis* (South I. brown kiwi) *Apteryx australis lawryi* (Stewart I. brown kiwi) *Apteryx owenii* (little spotted kiwi) *Apteryx rowi* (Okarito brown kiwi)
Rallicola (*Aptericola*) *pilgrimi* (Clay 1972)	Chewing louse	*Apteryx owenii* (little spotted kiwi)
Rallicola (*Aptericola*) *rodericki* (Palma 1991)	Chewing louse	*Apteryx mantelli* (North I. brown kiwi)
Apterygon okarito (Palma & Price 2004)	Chewing louse	*Apteryx rowi* (Okarito brown kiwi)
Apterygon mirum (Clay 1961)	Chewing louse	*Apteryx mantelli* (North I. brown kiwi)
Apterygon dumosum (Tandan 1972)[c]	Chewing louse	*Apteryx australis australis* (South I. brown kiwi) *Apteryx australis lawryi* (Stewart I. brown kiwi) *Apteryx owenii* (little spotted kiwi)
Guntheria (*?apteryxi*) (Loomis & Goff 1983)	Trombiculid mite	*Apteryx haasti* (great spotted kiwi)
Guntheria [=*Derrickiella*] *apteryxi* (Loomis & Goff 1983)	Trombiculid mite	*Apteryx mantelli* (North I. brown kiwi)
Haemaphysalis longicornis (Neumann 1901)	Tick	*Apteryx mantelli* (North I. brown kiwi)
Ixodes anatis (Chilton 1904)	Tick	*Apteryx mantelli* (North I. brown kiwi) *Apteryx australis lawryi* (Stewart I. brown kiwi)
Ixodes eudyptidis (Maskell 1885)	Tick	*Apteryx mantelli* (North I. brown kiwi)
Parapsyllus nestoris nestoris (Smit 1965)	Flea	*Apteryx australis lawryi* (Stewart I. brown kiwi)

[a] Gaud & Atyeo (1970) have one set of specimens of *K. phalogotrichus* from Stewart Island, but no locality is given for the *Kiwilichus delosikyus* material, so no current name can be given to the host.

[b] Sales (2005) incorrectly gives the genus as *Kiwialges*, and also omits *Kiwilichus delosikyus*.

[c] See Pilgrim & Palma 1982.

8. Wildlife Diagnostic Service

This section was prepared by Dr Maurice Alley, New Zealand Wildlife Health Centre, Massey University, and is also available from http://wildlife.massey.ac.nz.

Massey University provides a diagnostic service to the Department of Conservation and other wildlife conservation and management institutions. Guidelines on submission of specimens for post-mortem are detailed below.

The three important points to consider are:

- Preservation
- Documentation
- Packaging

8.1 PRESERVATION

Time, heat, autolysis, microbial multiplication, maggots, and scavengers all obscure the information that can be obtained from a dead body. Preservation techniques to counteract these may also introduce artefacts or limit the range of examinations which can be possible on the remains. A dead animal should be chilled to refrigerator temperature (approximately 4°C) as soon after death as possible, and despatched for diagnosis on the earliest available transport. Freezing interferes with gross and microscopic examination of tissues, and some aspects of microbiological culture and should be a last resort if the dead body cannot be delivered within approximately 24 to 36 hours. Freezing a rotten body will not improve its diagnostic value even if it does slightly improve its smell. Fixing bodies whole in alcohol or formalin, and field dissection and submission of formalin-fixed tissues for histopathology is an alternative which can be used in some circumstances—talk to us before undertaking this. However if you are collecting material into fixatives, remember skin contact and inhalation of formalin is hazardous; you need to use a volume of formalin 10 times the amount of tissue you are fixing (i.e. 100 g of tissue needs 1 L of formalin); and formalin does not penetrate quickly enough for proper fixation for histopathology more than about 1 cm deep into tissues.

8.2 DOCUMENTATION

All bodies should be sent with a wildlife submission form (see Appendix 7, or download from http://wildlife.massey.ac.nz).

The objectives of documentation are to:

Identify the animal, tissues or specimens

Providing a detailed provenance may assist the diagnosis, and will also allow a complete data entry in the wildlife disease database Huia. We want to know the individual animal ID if it has one, the species, the geographical location, the timepoint of collection (who, what, where, when?)

Specify what you want

Our routine practice is to try to establish a cause of death and other intercurrent diseases when a whole body is submitted. You may want to know something else either instead or as well as these things.

Disposition of remains and samples

Our routine practice is to retain a range of tissue samples for histopathology and microbiology after a gross necropsy. Do you want anything else kept or handled in a specific way? Is the body important as a natural history specimen? We can modify our examination techniques to be in sympathy with further uses of the remains if you let us know.

8.3 PACKAGING

The objectives of packaging are to:

Retain and maintain the identification of the submission

A legible label in permanent ink or pencil firmly associated with the body either attached as a tag or applied to a bag immediately containing it.

Contain the body and any leakage

Use multiple tear and puncture resistant sealed plastic bags, or plastic containers with firmly screwed down tight-fitting lids. Surround with enough absorbent material such as paper towel to mop up any effusions and seepage which might be anticipated. Do not use glass. We all have a duty to prevent contaminaton of people and equipment with potentially infectious or hazardous substances.

Protect the samples from the transport environment (heat, water) and damage in transport (impact and crush-proof)

A suitably sized polystyrene foam chilly bin is best, but alternatives can include a cardboard box with newspaper and bubble-wrap protecting the well-wrapped and bagged body. Proprietary freezer blocks, gel paks, etc. can be improvised using 500-mL plastic PET drink-bottles—partially fill some with water and put them in your freezer so thay will be ready when you need them.

The physical address for specimens to the wildlife diagnostic service is:

Attention: Maurice Alley/Brett Gartrell/Kerri Morgan
Room 8.28
Vet Tower
IVABS
Massey University
Fitzherbert Road
Palmerston North

Mark the package: **Urgent**, **Perishable** or **Keep Cool, Do Not Freeze**.

It is useful to inform us by email, phone or fax so we know to expect a parcel. The Huia database submission form (see Appendix 7) can be faxed to 06 350 5636.

The following courier companies have been used successfully in the past:

NZ Couriers

Tranzlink

8.4 IN SUMMARY

- Chill and despatch as soon as possible.
- Identify and specify what you want in the documentation.
- Contain, preserve and protect in transit by appropriate packaging.

Continue to next file: Part 5