# Kiwi first aid and veterinary care

Kerri J. Morgan

Department of Conservation
*Te Papa Atawhai*

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Cover: Okarito brown kiwi (rowi) having a computer tomography scan to diagnose a fractured acetabulum.
*Photo: B.D. Gartrell.*

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# Kiwi first aid and veterinary care

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## ABSTRACT

This document provides information about the treatment of sick or injured kiwi (*Apteryx* spp.) for veterinarians, conservation field workers and wildlife park staff. It incorporates basic techniques to stabilise sick or injured kiwi. Specific diseases and common injuries that have been seen in kiwi are also addressed. Diagnostic and treatment techniques specific to each condition and, in some cases, specific to kiwi, are included. The information given is not intended to be a complete reference for the veterinary treatment of kiwi, and is subject to change as more information on diseases or injuries and their treatment becomes available.

Keywords: kiwi, *Apteryx*, first aid, veterinary care, injuries, disease, parasites, New Zealand


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# 1. Introduction

## 1.1 BIRDS—THE PRESERVATION REFLEX AND STRESS

In many circumstances, it is immediately obvious when a bird is sick or injured. However, in other instances it is less obvious and disease processes may be challenging to detect.

Birds go to great lengths to hide clinical signs of illness. In the wild, sick birds attract the attention of predators and, in flocking species, a sick bird will be shunned by flock mates (Hume 2000). This masking of signs of illness is known as the 'preservation reflex'. Birds generally do not look sick until they are in an advanced state of illness and near collapse (Cannon 1991).

Identifying that birds are sick when they are in the early stages of disease requires detection of subtle clinical signs. Initial warning signs may include a reduction in food intake and a drop in body weight. A bird with intermittent clinical signs of illness is definitely unwell, and a bird with constant signs is seriously sick.

Stress can play a significant role in the pathogenesis of avian disease. Healthy birds can accommodate some degree of stress, but sick birds are universally intolerant of all types of stress (Cannon 1991). Birds with subclinical problems (i.e. diseases without detectable manifestations) often become sick when stressed. In the wild, stressors include starvation, territorial aggression, and physiological stress such as breeding. In captivity, stress may be induced by malnutrition, poor hygiene and sanitation, poor handling, and overcrowding. Furthermore, it must be remembered that captivity removes the birds' natural escape mechanisms (Cannon 1991). In captive birds, malnutrition caused by an imbalanced diet is one of the most common forms of chronic stress, and this may contribute to disease (see section 7.6).

## 1.2 LEGAL ASPECTS OF TREATING PROTECTED WILDLIFE

**The Department of Conservation (DOC) has a Captive Management Policy (DOC 2003). Veterinarians undertaking wildlife rehabilitation are required under this policy to obtain an authority (permit) from DOC to hold absolutely protected wildlife.** Contact your local DOC office for more information. Contact details for DOC offices can be found on the DOC website (www.doc.govt.nz > Publications > About DOC > Role > Policies & plans > Captive Management Policy).

When a kiwi is brought in for treatment, there are two priorities. The first is to administer emergency treatment and pain relief and to assess the bird's injuries/disease status. The second is to inform DOC.

According to the Captive Management Policy, DOC will:

> 'Support the treatment of injured absolutely protected wildlife only in cases where it is likely that the animal will: make a good recovery and be suitable for release into the wild; become part of an approved Species Recovery Programme. If the animal's full recovery is unlikely and it cannot be placed in an authorised programme, treatment should be withheld and the animal euthanised.' (DOC 2003: section 2.2.10, p. 7.)

Consult with DOC regarding the prognosis of treatment and possible options for release of the bird or its use in the Kiwi Recovery Programme.

If further care is required after initial treatment has been completed, the kiwi may only be transferred (after consultation with DOC) to a facility which possesses an authority to hold absolutely protected wildlife. If/when the kiwi is suitable for release into the wild, DOC must be consulted to determine the appropriate location and process for release.

# 2. Assessment, first aid and stabilisation

## 2.1 INITIAL ASSESSMENT

Initial assessment of a sick kiwi involves taking a history and performing a distance examination prior to handling the bird. This may give the assessor an indication of the appropriateness of further handling for physical examination and initiation of therapeutics. For example, a bird in respiratory distress may substantially benefit from a period of pre-oxygenation and warmth prior to further handling.

### 2.1.1 History

Gaining a thorough history is an important part of assessment of the avian patient. The ability to obtain a complete history is highly dependent upon the bird's situation, which will vary from captivity, where birds are visually assessed by their keepers daily, to completely wild, with very little known history.

***Important points to note***

- What is the age and sex of the bird?
- Is it a wild or captive bird?

***Captive birds***

- What is the origin of this bird (e.g. captive-raised, wild-caught, transferred from another captive institution)?
- How many birds (in particular, other kiwi) are in contact with this bird?
- How long has the bird been in the particular kiwi house or other captive facility?
- Have any new birds been introduced recently?
- Is the bird in contact with people?
- Is the bird housed indoors or outdoors? Obtain a description of the enclosure, including substrate. Is there any potential for exposure to foreign objects (nails, screws, etc.)?
- Have there been any recent changes in substrate, sources of leaf litter, etc.?
- What is the bird's diet? Have there been any recent changes in diet? What is the water source for the bird?
- How many birds are affected?
- When was the bird noticed to be sick?
- What are the observed abnormalities?
- What is the breeding history and current status of the bird? Is there a possibility the bird is gravid (carrying an egg)?
- Are there any changes in the consistency, frequency or appearance of the bird's droppings?

- Are there any changes in respiration, e.g. open-mouth breathing, wheezing, sneezing, dyspnoea (respiratory exaggeration)?
- Has the bird had any treatment to date, such as fluids, medications (including, but not limited to, antibiotics, antifungals, analgesics, corticosteroids, antiparasitics)?
- Does the bird have any possible access to toxic materials such as rat bait, snail bait, herbicides, toxic plants (e.g. karaka (*Corynocarpus laevigatus*) berries)

***Wild birds***

- Where was the bird found?
- What are the observed abnormalities?
- When was it found? If there has been a delay between the bird being found and the current presentation, where has it been previously?
- Has it had any treatment (treatment may include fluids and medications including, but not limited to, antibiotics, antifungals, analgesics, corticosteroids, antiparasitics)?
- Has the bird been offered food? Has it eaten?
- Is the breeding history of this bird available?
- Has it passed any droppings? If so how did they look (volume, colour, consistency of faeces, urates and urine)?

### 2.1.2 The distance examination

A distance examination should ideally be undertaken prior to a bird's capture and restraint, i.e. prior to the induction of further stress. This will allow the handler to get some idea of the severity of the disease, and how well the bird may cope with the stress of further restraint at this stage. However, a distance examination will not always be possible. If the bird is presented in a transport box, observe the bird for a few moments in the box before picking it up.

A normal bird should be alert, standing straight and even on both feet, with barely visible respiratory efforts at rest (Hume 2000).

Points to note include:

***Mental status***

- Is the bird bright, alert and reactive to stimuli?
- Or is it quiet and dull?
- Or moribund?

***Posture***

- Is the bird standing?
- If so, is it standing evenly on both legs?
- Is it using its bill for balance?

***Gait***

- Is the bird ambulating normally?

***Respiration***

This should be barely visible. Abnormalities include:

- Open-mouth breathing
- Exaggerated respiratory effort
- Respiratory noise or click on inspiration/expiration
- Ocular or nasal discharge

***Feathering***

- Is there any obvious damaged or misshapen feathering?
- Are feathers fluffed up?

***Wounds***

- Are there any signs of injury (fresh or dry blood, cuts, open wounds, matted feathers, bald patches, etc.)?

***Body symmetry***

- Are there any obviously damaged or misshapen parts of the body?

### 2.1.3 Handling and restraint

Once these visual assessments have been completed, the decision whether to handle and restrain the bird for further diagnostics and supportive therapy should be made. A bird in respiratory distress will invariably benefit from supplemental oxygen, warmth and rest prior to further handling.

In many cases, there will probably be an experienced kiwi handler present. However, if not, the following description of handling kiwi by R. Jakob-Hoff may be useful:

> 'Grasp both legs above the hock joint between the thumb and middle finger of the right hand with the index finger between the two legs. The left hand supports the ventral and lateral body into a sitting position in the crook of the right elbow. Direct the bird's bill and eyes under cover of the left arm.
>
> 'An alternative hold is often used in the field with the holder in a sitting position. Grasp the two legs as previously described, hold the bird in an upside-down position with the dorsal body placed on the lap of holder and the kiwi's head under the left arm.' (Doneley 2006)

Taping the legs of kiwi together during handling should occur only if absolutely necessary, and then just for short periods. Kiwi should never be transported with legs taped. There is evidence that taping legs together causes severe muscle damage, as indicated by marked elevations in creatine kinase in great spotted kiwi (*Apteryx haastii*) that were transported with legs strapped together (unpubl. data). If taping is necessary for weighing or transmitter changes in the field, current recommendations are for periods no longer than 15–20 minutes. However, the impact of leg taping on kiwi physiology requires further study. (B.D. Gartrell, Massey University, pers. comm.).

### 2.1.4 Physical examination

A systematic and thorough physical examination is important to ensure that subtle abnormalities are not overshadowed by obvious injuries. Doneley (2006) gives the following summary as a guide to examination of ostriches.

A similar approach can be applied to examination of kiwi. The extent to which the described examination can be done depends upon expertise and equipment available.

### *Head examination*

Examine the eyes, ears (caudo-lateral to the lateral canthus of the eye), bill and nostrils (at the tip of the bill), looking for defects and discharges.

### *Oral cavity*

Examine the choana (slit on the roof of the mouth which communicates with the upper respiratory tract), glottis (opening into the trachea), tongue, and mucosa lining the oral cavity.

### *Body condition*

As kiwi are flightless, it is not possible to determine the body condition score by palpation of the pectoral muscles. Palpation of the epaxial muscles (adjacent to the spine) and assessing the prominence of the ribs will give a subjective indication of kiwi body condition.

### *Heart and respiratory system*

Auscultate (preferably with a paediatric stethoscope) for abnormal cardiac and respiratory sounds. The rigid avian lungs lie in the dorsal cranial body wall, and are best listened to over the dorsum, halfway between the vestigial wings. [Although less developed in kiwi than in flighted avian species (B.D. Gartrell, Massey University, pers. comm.), the air sac system extends throughout the entire coelomic cavity.]

Normal kiwi heart rate: 70–240 beats per minute

Normal kiwi respiratory rate:12–60 breathes per minute (Doneley 2006)

### *Palpate the abdomen*

The firm gizzard lies in a caudoventral position, slightly to the left of the midline.

### *Legs and feet*

Observe the legs and feet for gross abnormalities. Limbs should be palpated for obvious fractures and joint effusion (abnormal fluid). In chicks, tibiotarsal alignment should be evaluated for rotational defects (see section 7.7.1).

### *Skin and feathers*

The skin and feathers should be evaluated for external parasites. Many kiwi carry ticks. These are often found in large numbers over the head region, where the bird is unable to preen them off. They are also common in the external ear canal (see section 7.8.3). Kiwi also carry lice, and it has been observed (in birds in general) that debilitated individuals tend to have increased numbers of lice.

The skin should also be evaluated for traumatic injuries and other lesions, including dermatitis, which may be indicative of a vitamin B deficiency (see section 7.6.2).

### *Vent*

The vent should be examined for prolapse, trauma and vent soiling with faeces and urates.

### *Neurological examination*

Compared with other avian species, kiwi show minimal reliance upon vision, as indicated by eye structure, visual field topography and reduced visual centres in the brain (Martin et al. 2007). Functionally blind kiwi have been found in the wild. These birds were surviving well, and for this reason blindness should not be grounds for non-release. Instead of eyesight, kiwi have an increased reliance upon olfactory and tactile information as an adaptation to their nocturnal behaviour (Martin et al. 2007). However, despite the fact that kiwi might not need good vision to survive, cranial nerve evaluation should include assessment of the pupillary light reflex, the menace reflex and the corneal reflex. In performing these examinations it is important to note some differences that are particular to birds. There is no consensual light reflex, and birds are extremely sensitive to movement of air across the surface of their eye, which may confuse results of the menace reflex. Birds also have some voluntary control over their pupil size because of skeletal muscles within the iris. Anisocoria (asymmetrical pupil size) may be indicative of cranial trauma. Other abnormalities may include strabismus (deviation of the eye) and nystagmus (involuntary movement of the eyeballs in unison). Other cranial nerve abnormalities may cause reductions in olfactory ability, torticollis (head tilt), dysphagia (difficulty in swallowing), and alterations in facial sensation.

Segmental reflexes in kiwi include the leg withdrawal and the vent reflex. These reflexes require intact peripheral nerves without the need for an intact spinal cord. Withdrawal of the leg when stimulated (e.g. a toe pinch) indicates an intact peripheral nerve supply to that limb. Perception of pain requires an intact spinal cord in addition to an intact peripheral nervous system. Indications that a bird can perceive a painful stimulus may include an attempt to escape, or the bird looking at the painful area in response to the stimulus.

The vent reflex can be elicited by pinching the skin adjacent to the vent. The vent should normally contract in response to this stimulus.

It should be noted that many reflexes and responses may be confounded by pain, and the mechanical inability to move severely fractured limbs. The neurological examination is most useful for initial assessment, and to evaluate changes in neurological function over time.

## 2.2 EMERGENCY STABILISATION OF A DEBILITATED KIWI

The priority in the initial 24 hours after presentation of a sick or injured kiwi is to stabilise the patient. Stabilisation involves attempting to regain physiological homeostasis in what are often hypovolaemic, hypothermic, malnourished and septic patients. The extent to which stabilisation can be achieved is dependent upon the available equipment and expertise.

It may be advisable for field staff working with kiwi to carry a first aid kit containing useful items for provision of at least warmth and fluid therapy (Appendix 1).

The following text details the achievable priorities for treating sick or injured kiwi in the field and in the veterinary hospital. It is recognised that there will be many occasions and situations where the procedures that are achievable will fall somewhere between these two extremes.

The goals of treatment in the first 24 hours include rehydration, provision of warmth, stabilisation of fractures, dressing of open wounds, pain relief, and placement of the kiwi in a warm, stress-free environment.

### 2.2.1 In the field

Field workers should be able to provide the debilitated kiwi with the following:

***Fluid therapy*** (see section 2.2.5)

- Fluids may be administered orally or by subcutaneous injection.

***Warmth*** (see section 2.2.4)

- In-the-field heat therapy may be provided with heat pads or hot water bottles.

***Fracture and wound management*** (see section 2.3)

- Lower limb fractures should be dressed and bandaged in order to prevent further injury to the limb and contamination of wounds, as well as reducing pain associated with the injury.

***Quiet, stress-free environment*** (see section 5.2)

- The bird should be placed in a warm, quiet, dark and stress-free environment if there is a delay in its transportation out of the field.

### 2.2.2 In the veterinary clinic

In the veterinary clinic, the first priority must be to address life-threatening dyspnoea (breathing difficulties) if present.

***Provision of oxygen*** (see section 2.2.3)

- Dyspnoeic patients will benefit from the provision of supplemental oxygen, either via a mask or in an oxygen tent, prior to further handling.
- An upper airway obstruction requires immediate intervention by placement of an air sac cannula through which oxygen can be supplied.

***Fluid therapy*** (see section 2.2.5)

- Intravenous (IV) access should be prepared primarily for fluid therapy, as well as IV administration of analgesics and anti-infective medication. Oral and subcutaneous fluid therapy may also be used, but these are less than optimal in the compromised patient.
- Intraosseous cannulation provides access for critical fluid and medical therapy in the event that intravenous access is not possible. The suggested site in kiwi is the tibiotarsus via the tibial crest.

***Warmth*** (see section 2.2.4)

- Warming hypothermic kiwi is critical, and can be achieved by a variety of methods.

***Analgesia*** (see section 2.2.7)

- Opioids (butorphanol) may be given for analgesia. Non-steroidal anti-inflammatory drugs (e.g. carprofen, meloxicam) should only be used in well-hydrated patients.

***Anti-infective medications*** (see section 2.2.8)

- Antibiotics should be started immediately if the patient is suspected to be septic, or has injuries that are likely to become infected.

***Bandaging and wound management*** (see section 2.3)

- The goal of initial bandaging and wound management is to prevent further desiccation of the wound, and to provide external stability for lower limb fractures. This is important in preventing further tissue damage and relieving pain associated with unstable fractures.

***Initial diagnostics*** (see sections 4.1 and 4.2)

- Ideally, a blood sample should be taken in the initial stages of treatment to enable investigation of haematological and biochemical parameters before any treatment is given. At the very least, the packed cell volume (PCV) and total plasma protein (TPP) should be assessed.
- A faecal sample should also be examined to investigate the presence of parasites, including coccidia.

***Quiet, dark, stress-free environment*** (see section 5.2.1)

- The bird should be placed in a warm, quiet, dark and stress-free environment, away from foot traffic and other animals.

**N.B. Corticosteroids are contraindicated in the stressed or septic avian patient, as they may significantly suppress the hypothalamic pituitary axis, and may result in a severe and lasting immunosuppression.**

### 2.2.3 Oxygen therapy

Stress resulting from handling and other procedures can be fatal in a severely debilitated bird. Clinical signs suggestive that handling for examination is contraindicated until the bird is stabilised include prolonged dyspnoea, and prolonged panting and gasping for air (Harrison et al. 2006). These patients benefit significantly from oxygen therapy prior to handling.

Prior to examination, oxygen may be administered in a chamber where the air comprises 40–50% oxygen. An intensive care unit or incubator can be used or, more simply, a cage covered with a plastic bag (ensure an outflow is present) (Harrison et al. 2006). A facemask fashioned for kiwi (see section 3.2.3) is effective for short-term treatment if an oxygen enclosure is unavailable, but the stress resulting from handling the bird to attach the facemask may outweigh any benefit from the extra oxygen.

Air sac cannulation is well described for other avian species. This procedure is indicated for upper respiratory tract obstructions at the level of the trachea or syrinx (Harrison et al. 2006), providing the bird with the ability to inspire and expire through the caudal air sacs, bypassing the trachea. Air sac cannulation is an emergency procedure, and involves placement of a cannula into the caudal thoracic or abdominal air sacs (Harrison et al. 2006).

However, adaptations for flightlessness have resulted in a reduction in air sac development in kiwi, making this a difficult procedure to perform in these species (B.D. Gartrell, Massey University, pers. comm.). Several attempts at using this procedure with kiwi have been made, with varying success, and its use in kiwi is still at an early stage. For further information about this procedure, contact the New Zealand Wildlife Health Centre, Massey University (see Appendix 2 for contact details).

### 2.2.4 Warmth

Debilitated birds are unable to thermoregulate as effectively as healthy birds, and are often hypothermic. Provision of warmth is indicated for most sick and injured kiwi. Options for providing heat vary depending on the situation and availability of equipment.

The gold standard for the provision of warmth is an intensive care unit specifically designed for small animal use. Alternatively, a human paediatric incubator works well. Both pieces of equipment allow thermostatic and humidity control of the air within them. Other options include hot water bottles, wheat sacs and heat pads, heat lamps or, simply, a heater in a small room. All approaches have advantages and disadvantages.

Hot water bottles and heat pads are readily accessible and cheap and are ideal for taking into the field. However, care must be taken to ensure that birds do not get burnt from direct contact with them, and it is recommended that heated objects such as hot water bottles be wrapped in a towel before use. A major disadvantage of hot water bottles, heating pads and wheat bags is that they cool down and need to be removed for reheating, which causes disturbance and further stress to the bird.

Heat lamps are a good source of overhead heat. They can be purchased from a pet store or lighting store. Bulbs are either ceramic (without glow) or infrared. Heat lamps should never be left unattended, and care must be taken to not burn the bird, especially with recumbent birds that cannot move away from the heat source.

A heater in a small room may work well also. Ideally, the heater should have a thermostat which can be set to regulate the temperature in the room.

The desirable ambient temperature for most sick birds is 29–30°C, with 70% humidity (Harrison et al. 2006). However, kiwi have inherent lower body temperatures than other avian species, and it may be more appropriate to house them at lower temperatures (e.g. 25–26°C). Birds should be monitored closely for signs of heat stress. Clinical signs of hyperthermia may include open-mouth breathing and the flattening of feathers to the body. Once the bird has reached its normal body temperature (c. 38°C), the ambient temperature should be reduced, as kiwi do not tolerate high ambient temperatures well. If the heat source is not under thermostat control, a maximum-minimum thermometer should be installed in the enclosure to monitor any fluctuations in temperature. If an ICU or incubator is unavailable, placing a small dish of water or a moist heated towel in the bottom of the enclosure will provide some humidity with the warm air (Harrison et al. 2006).

### 2.2.5 Fluid therapy

It should be assumed that debilitated birds will be hypovolaemic upon presentation. Hypovolaemia refers to a reduction in circulating blood flow, with a resultant reduction in hydration of body tissue. Hypovolaemia may be caused by a combination of blood loss, shock (including septic shock), and severe dehydration due to a prolonged lack of food and water intake or excess loss of body fluids.

Hypovolaemic animals require rigorous fluid therapy. Goals of the fluid therapy plan include:

- Replacement of lost fluids.
- Provision of maintenance fluids to cover normal daily losses, estimated at 50 mL/kg/day.
- Replacement of any ongoing abnormal losses (e.g. diarrhoea, continued bleeding).

#### *Assessment of dehydration and hypovolaemia*

The bird's history may assist determination of the likelihood of hypovolaemia. The likely route and duration of any fluid loss should be assessed (e.g. blood loss, diarrhoea), as well as any indications suggestive of septic shock. Fluid and food intake immediately prior to presentation should also be taken into account.

Assessment of hydration status in a bird is subjective and requires practice; however, in general, many workers assume a loss of 10% of total blood volume in a debilitated bird (Monks 1996).

Below is a guide to a more detailed subjective assessment of hypovolaemia in the avian patient (Redig 1984; Abiu-Madi & Kollias 1992; Monks 1996):

| | |
|---|---|
| < 5% dehydrated | No detectable clinical signs. |
| 5–6% dehydrated | Subtle clinical changes—subtle loss of skin elasticity. |
| 6–8% dehydrated | Moderate dehydration—skin tenting is visible over the dorsal tarsometatarsus (i.e. when the skin is pinched, it takes longer than normal to return to its normal position).<br>Dry mucous membranes (oral, cloacal, conjunctival).<br>Decreased sliding of the skin over the sternum. |
| 10–12% dehydrated | Severe dehydration—all of the above plus skin turgor, sunken eyes, thick and stringy mucous in caudal pharynx, central nervous system depression, lethargy, weakness. |
| 12–15% dehydrated | SHOCK. Death is imminent unless therapy is rapidly established. |

Objective assessment of hydration status can be achieved by evaluation of haematological and biochemical parameters. Dehydration may increase the packed cell volume (PCV) by 15–30%, and the total plasma protein (TPP) by 20–40% (Lumeij 1987; Martin & Kollias 1989).

It should be remembered, however, that TPP may be low in a starving bird regardless of hydration status (Kaufman 1992). Also, PCV may be low due to any chronic disease, and this may mask any rise due to dehydration (Martin & Kollias 1989; Hoefer 1992).

Initially, acute blood loss results in no change in PCV and TPP. As the time between haemorrhage and presentation increases, a reduction in PCV and TPP becomes more likely (Forsyth et al. 1999).

### *Routes of administration of fluids*

Regardless of the route of administration, all fluids should be warmed to body temperature (38–39°C) (Quesenberry & Hillyer 1994). Using warm fluids is particularly important with neonates and with intravenous or intraosseous administration of fluids for hypothermia or shock (Abou-Madi & Kollias 1992).

#### Oral fluids

Oral fluids are indicated for treatment of mild dehydration, or for daily maintenance fluids only (Quesenberry & Hillyer 1994). They are inadequate in birds with sudden or excessive fluid losses. Oral fluids should not be given to birds that are seizuring, laterally recumbent, regurgitating, in shock or have gastrointestinal stasis (Quesenberry & Hillyer 1994). For effective rehydration, oral fluids need to be readministered within 60 to 90 minutes of the first treatment (Quesenberry & Hillyer 1994).

Types of oral fluids include:

1. Oral electrolytes
- Calf electrolytes, e.g. Revive™, Vytrate™, Dexolyte™.
- Human electrolytes, e.g. Gastrolyte™, Powerade™ (though not ideal because of high levels of artificial colours and flavour).
- Bird-specific electrolytes, e.g. Polyaid™ (includes nutritional support).

2. Intravenous solutions which may be given orally
- LRS, 0.9% NaCl, 2.5–5% dextrose.

The relatively small capacity of the kiwi gastrointestinal tract limits the volume of fluids that can be administered orally. The kiwi lacks a distinct crop and has only a small proventriculus (Fergus et al. 1995). This limits the capacity for storage of food and fluids. Administration of excessive fluids may result in regurgitation and possible aspiration into the trachea. The author recommends that no more than 20 mL of oral fluids be given to an adult kiwi at any administration, and no more than 3–4 mL to a kiwi chick. This should be given slowly. To account for individual variation, it may be safer to give a smaller amount than this initially, and work up to this larger volume over subsequent administrations.

Oral fluids are administered into the distal oesophagus using either a metal avian crop tube or a silicone rubber feeding tube attached to a syringe. With the head well restrained and the neck extended, the tube should be passed beside the tongue on either the left or right side of the oral cavity, avoiding the glottis (opening to the trachea) (Fig. 1). The tube is then passed down into the oesophagus which lies on the right side of the neck. The glottis is clearly visible as an opening at the base of the tongue, identifiable by movement of the glottic cartilages as the bird breathes. When the tube is in place, the glottis should be observed to ensure that the apparatus is clear of this structure. Fluid should then be slowly syringed through the tube while the back of the oral cavity is observed for reflux. If reflux occurs, the tube should be removed and the bird's head released to allow it to swallow and shake its head to clear the glottis.

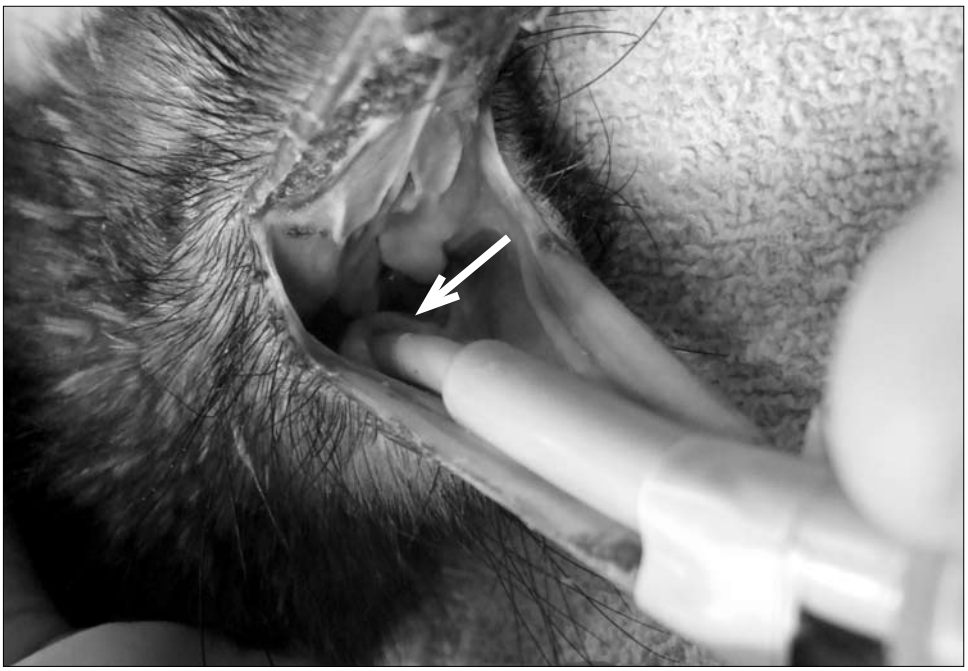


Figure 1. Intubation of a kiwi with a Cole™ endotracheal tube. The stepped portion of the endotracheal tube will be advanced to sit against the glottis (arrowed) to form a seal. Uncuffed endotracheal tubes are used in birds because of the complete cartilaginous rings in their trachea. If cuffed tubes are used, the cuff should never be inflated, as this can cause pressure necrosis and subsequent stricture of the trachea.
*Photo: K. Morgan.*

**Subcutaneous fluids**

Subcutaneous fluids should be utilised for mild dehydration and for maintenance fluid therapy only (Quesenberry & Hillyer 1994). This route of administration is ineffective at treating hypovolaemia and moderate to severe dehydration, because associated peripheral vasoconstriction reduces the absorption of fluid in hypothermic or shocked patients (Forsyth et al. 1999).

Types of subcutaneous fluids include:

- Lactated Ringers solution (LRS)
- NaCl 0.9%

Fluids for subcutaneous fluid administration need to be isotonic (Forsyth et al. 1999). Subcutaneous administration of 5% dextrose should be avoided because equilibration of the extracellular fluid with a pool of electrolyte-free solution may result in an aggravation of an electrolyte imbalance (Forsyth et al. 1999). Sterile abscesses and local fluid accumulation may also occur at the site of subcutaneous dextrose administration (Forsyth et al. 1999). Fluids for subcutaneous administration must be sterile to avoid causing infection at the site of injection.

Sites for administration of subcutaneous fluids are the:

- Intrascapular region—this is the skin over the dorsum (back) of the bird, halfway between the vestigial wings.
- Inguinal fold—this is the fold of skin cranial to (in front of) the stifle (knee), between the stifle and the body wall.

Fluids administered at each site should not exceed 5–10 mL/kg bodyweight, with only one puncture hole per administration site (Quesenberry & Hillyer 1994). Use small needles (25–27 gauge); a butterfly catheter may assist administration.

### Intraperitoneal fluids

**Never** administer intraperitoneal fluids to birds. This is likely to result in fluid getting into the air sacs, and there is a risk of organ laceration (Monks 1996).

### Intravenous fluids

Intravenous fluid therapy is indicated in cases of severe debilitation or severe hypovolaemia (Harrison et al. 2006). The author recommends intravenous fluid administration in the majority of cases of debilitated kiwi, and for all surgical cases.

Intravenous access also allows the administration of antibiotic and analgesic medication with minimal stress on the bird.

To minimise stress, the author recommends placement of the IV catheter while the bird is under general anaesthesia. In kiwi, intravenous access points are limited to the bilateral medial metatarsal veins, and the right jugular vein as a last resort. The medial metatarsal vein is most accessible proximal to the first phalange (medial) and courses in a caudal direction (Fig. 2). In some cases, the right jugular vein may be useful, but it is difficult to maintain a catheter in this position.

A 20- or 22-gauge catheter is recommended for adult kiwi, and a 24-gauge catheter for chicks. The catheter should be securely fixed with tape, and covered in a self-adherent dressing. Extension sets are useful (Fig. 3).

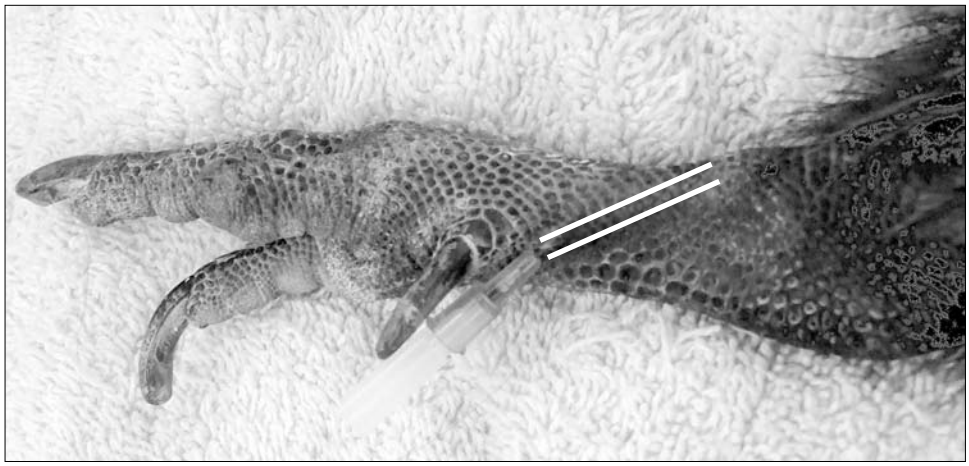


Figure 2. Medial distal limb of a kiwi (with a 22-gauge catheter in place). White lines show position of the medial metatarsal vein. *Photo: K. Morgan.*

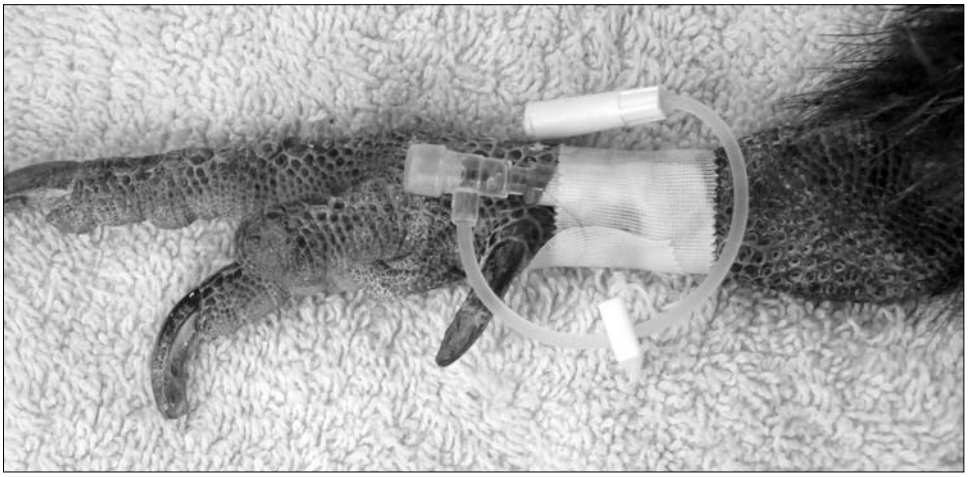


Figure 3. Extension set connected to a 22-gauge catheter in medial metatarsal vein of the right leg of a kiwi. *Photo: K. Morgan.*

Kiwi tolerate intravenous catheters, and they can be maintained for up to 7 days with an aseptic technique. Complications of catheterisation include thrombosis, cellulitis and loss of patency (Monks 1996).

Intravenous fluids can be given either as an intermittent bolus of fluid, or by constant infusion in an IV drip, ideally with a fluid pump.

If a fluid pump is unavailable, it is wise to administer fluids as a bolus to prevent accidental overhydration which may occur with a normal drip set-up. Bolus fluid administrations should be given slowly intravenously, at a rate of 10–25 mL/kg over 5–7 minutes. This should be repeated at 3–4 hourly intervals for the first 12 hours, then every 8 hours for the next 48 hours. Thereafter, IV boluses can be given twice daily for maintenance therapy (Harrison 1986).

**Intraosseous fluids**

Intraosseous fluid therapy is indicated in severely debilitated birds where it is not possible to gain intravenous access. It allows the parenteral administration of fluids (including blood), nutritional support and glucose, analgesia, antimicrobials and drugs for cardiovascular resuscitation. Hypertonic or alkaline solutions should be avoided, as they are painful on administration (Quesenberry & Hillyer 1994).

Intraosseous catheterisation is a painful procedure, and in most cases should be done under general anaesthesia. Exceptions to this include very moribund birds that are unlikely to survive an anaesthetic.

Because they lack significant wing bones, intraosseous catheter sites in kiwi are limited to the tibial crest (Fig. 4). In birds, the femur should be avoided for intraosseous cannulation as this bone is pneumatic, having communications with the air sac system (Quesenberry & Hillyer 1994).

Figure 4. Intraosseous catheterisation in the left tibial crest of the tibiotarsus of a kiwi, using a 20-g intravenous catheter and butterfly catheter. *Photo: J. Youl.*

In young birds, an 18- to 22-gauge, 1.5- to 2.5-inch spinal needle is ideal for cannulation (Quesenberry & Hillyer 1994). An 18- to 22-gauge hypodermic needle will also suffice. In adult kiwi, a kirschner wire and chuck may be used to drill a hole for cannulation. Aseptic preparation of the site is extremely important to avoid introducing infection to the area. The needle/kirschner wire should be advanced into the bone marrow via the tibial crest, passing distally. Pressure should be applied with a slight rotating motion. Once the cortex is penetrated, the needle/kirschner wire should slide easily, with little resistance. If predrilling the cannulation hole with a kirschner wire, the wire is removed and the needle inserted. Bone marrow occluding the needle lumen should be aspirated and flushed with a small amount of heparinised saline. The catheter should be secured with a light padded bandage. Initial fluids should be given slowly while checking for subcutaneous swelling, which indicates improper catheter placement (Quesenberry & Hillyer 1994).

Intraosseous fluid rates of 10 mL/h have been administered to pigeons using an infusion pump (Lamberski & Daniel 1992). Bolus administration rates are limited, so boluses need to be given frequently.

In pigeons, it has been demonstrated that 50% of fluids given into the ulna enters the systemic circulation within 30 seconds (Lamberski & Daniel 1992). Over a 2-hour period, flow into the systemic circulation approximates the administration rate (Lamberski & Daniel 1992).

Intraosseous therapy is most successful if it is used during the first 24–48 hours of treatment for initial rehydration and shock therapy (Quesenberry & Hillyer 1994). The catheter may be maintained for up to 72 hours without complications if it is placed aseptically and maintained with heparin flush every 6 hours (Otto & Crowe 1992). Clinically, after 2–3 days many birds develop a painful response when fluids are administered through an intraosseous catheter. This may be in response to local oedema or extravasation of fluids around the marrow cavity (Quesenberry & Hillyer 1994).

### *Rates of fluid administration for dehydration*

#### Replacement fluids

10% dehydration may be assumed in all debilitated birds (Harrison 1986). Fluid deficits in avian species may be calculated using the following formula (Harrison 1986):

Fluid deficit (mL) = estimated dehydration (%) × body weight (g)

Half of the required fluid should be given over the initial 12–24 hours, with the remaining half divided over the following 48 hours.

#### Maintenance fluids

Maintenance fluids are estimated to be 50 mL/kg/day (Redig 1984). If the bird is not eating or drinking on its own, fluids need to be administered either parenterally or orally to ensure the bird is adequately hydrated. Paediatric birds require 2–3 times the maintenance volume on a weight basis (per kg) compared with adult birds (Bauck & Kupersmith 1991).

#### Ongoing losses

This includes fluids lost due to diarrhoea, polyuria, etc. If such losses are occurring, the fluid rate should be adjusted to compensate for them.

Example of a fluid therapy plan:

A 2.3-kg kiwi, estimated to be 10% dehydrated, with no ongoing fluid losses.

Replacement fluids: fluid deficit (mL) = 10% × 2300 g = 230 mL

Maintenance fluids: daily fluid (mL) = 50 mL × 2300 g = 115 mL/day

*First 12 (–24) hours*:

Fluid (mL) = replacement + maintenance fluids

= (½ × 230 mL) + 115 mL = 230 mL

*Next 24 hours*:

Fluid (mL) = (¼ × 230 mL) + 115 mL

= 57.5 + 115 mL = 172.5 mL

*Next 24 hours*:

Fluid (mL) = 172.5 mL

From here on, all of the replacement fluids have been given, and maintenance fluids of 115 mL per day need to be administered. If the bird is eating and drinking on its own, it may or may not require supplementation, depending on the individual case.

### *Selection of fluids for intravenous rehydration*

#### Replacement fluids

Fluid and electrolyte deficits most commonly result from inadequate water intake, or from excessive loss of extracellular fluid due to haemorrhage or diarrhoea. Replacement fluids are required to contain the same electrolytes as those found in the extracellular fluid (Forsyth et al. 1999). Crystalloids are ideal as replacement fluids. They are capable of distributing to all body fluid compartments.

Lactated Ringer's solution (LRS) warmed to 38–39°C is recommended for fluid replacement and shock therapy (Quesenberry & Hillyer 1994). This most closely approximates the extra-cellular fluid (Forsyth et al. 1999), and is a good choice in most instances of fluid replacement. 0.9% NaCl (sodium chloride) may also be used for fluid replacement.

Total body potassium loss occurs through a lack of intake (anorexia) or through excessive loss (e.g. diarrhoea). Compared with other electrolytes, potassium losses are relatively greater, so supplementation with KCl (potassium chloride) should be considered (20 mEq/L) (Forsyth et al. 1999).

#### Maintenance fluids

Solutions used for replacement therapy are not suitable for long-term maintenance because of variations in electrolyte requirements (Quesenberry & Hillyer 1994). Fluid losses occurring through the gastrointestinal tract, urinary tract, lungs and skin contain approximately half of the sodium and chloride concentrations found in serum and LRS (Forsyth et al. 1999). Maintenance fluids should reflect these electrolyte concentrations by containing much less sodium (40–60 mEq/L) and more potassium (15–30 mEq/L) than replacement fluids (Forsyth et al. 1999; Lichtenberger 2004).

Options for intravenous maintenance fluids include:

1. Half-strength LRS plus 2.5% dextrose plus 20 mEq/L KCl
   e.g. 500 mL LRS plus 500 mL × 5% dextrose plus 20 mEq KCl
   This is an adequate maintenance solution (Forsyth et al. 1999).

2. Lactated Ringers solution (LRS)
   If used alone for maintenance fluid requirements, LRS can result in hypokalaemia (low potassium) and hypernatraemia (excessive sodium) (Forsyth et al. 1999).

3. 0.9% NaCl
   This is unsuitable for maintenance fluid requirements as it is not a balanced electrolyte solution (Forsyth et al. 1999).

### *Hypovolaemic shock*

Rapid or extensive fluid loss causing hypovolaemic shock requires rapid replacement of blood volume. The most common cause of hypovolaemic shock in birds is haemorrhage (Lichtenberger 2004).

Boluses of warmed LRS may be given by slow intravenous or intraosseous administration at 10–30 mL/kg (Harrison 1986; Lichtenberger 2004). This amount should be administered over 5–7 minutes (Harrison 1986). There is usually an associated transient bradycardia (Harrison 1986). Fluid overloading rarely occurs, but may be indicated by tachypnoea (rapid breathing), cardiac dysrhythmias, agitation and collapse (Abou-Madi & Kollias 1992).

It is difficult to determine the exact fluid requirements of birds in shock (Quesenberry & Hillyer 1994). Thirty minutes after treatment with IV fluids, only 25% of isotonic crystalloid fluids remain in the vascular compartment, as the remainder of the fluid has redistributed to the interstitial fluid compartment (Haskens 1992). Therefore, improvement in circulation may be transient, requiring additional fluid therapy to prevent recurrence of hypotension and vasoconstriction (Quesenberry & Hillyer 1994).

Haemodilution is the primary limitation to crystalloid therapy, making the administration of colloids or blood necessary for effective shock therapy (Quesenberry & Hillyer 1994).

Hypertonic saline (7.5%) can be used as an intravenous bolus at a rate of 3–4 mL/kg, followed by intravenous crystalloid solutions (Firth 1995).

Colloids have not been used to any great extent in birds (Quesenberry & Hillyer 1994). Harrison (2006) recommends intravenous hetastarch at a dose rate of 10–15 mL/kg every 8 hours for one to four treatments for the treatment of hypoproteinaemia. Other reports suggest colloids should be given at a maximal dose rate of 20 mL/kg (Monks 1996). Colloids are large molecular weight substances that are unable to pass through capillary membranes, thus remaining in the intravascular space. They act by drawing fluid from the interstitium, and are thus more effective blood volume expanders than crystalloids (Abou-Madi & Kollias 1992; Haskins 1992). They are particularly useful at restoring circulating blood volume without aggravating hypoproteinaemia or causing pulmonary oedema in birds with low oncotic pressure and hypoproteinaemia (Quesenberry & Hillyer 1994).

Whole blood should be considered for severe blood loss.

### 2.2.6 Blood transfusions

Severe blood loss is much better tolerated in birds than in mammals, especially in flighted birds (Quesenberry & Hillyer 1994). This tolerance is the result of an increased rate of absorption of tissue fluids to replace lost blood volume, and baroreceptor reflexes that maintain normal blood pressure (Quesenberry & Hillyer 1994).

Indications for transfusion of whole blood include clinical signs of severe anaemia (tachypnoea, tachycardia, weakness, pallor), a PCV of less than 15–20%, (normal: 38–54%, Appendix 3) or acute blood loss of greater than 25% of the total blood volume (Hoefer 1992). Birds with a more chronic anaemia may be able to tolerate a lower PCV, and it is suggested that a PCV of below 12% may be indicative of the need for transfusion in chronically anaemic birds (B.D. Gartrell, Massey University, pers. comm.). However, these recommendations are anecdotal, especially in kiwi, and are useful as guidelines only.

There is little information on blood typing in avian species (Gilmour 1971), and blood transfusion in avian species is controversial and poorly understood (Degernes et al. 1999). In clinical practice, heterologous (between different species) transfusions are often used because homologous (of the same species) donors are seldom available (Degernes et al. 1999). However, recent consultation with iwi has determined that, at the time of writing, only homologous blood transfusions (i.e. kiwi to kiwi) are culturally acceptable, and heterologous transfusions should not be performed (K. McInnes, DOC, pers. comm.).

Studies of both homologous and heterologous transfusions in cockatiels (*Nymphicus hollandicus*) showed no apparent transfusion reactions or other complications during the study (Degernes et al. 1999). Single, whole-blood transfusions have been performed on kiwi on at least three occasions (to the author's knowledge), without any apparent complications.

The donor kiwi should be healthy at the time of blood collection, and should be anaesthetised for the collection procedure. A suggested maximum blood volume for collection is between 1 and 2% of the bird's body weight, with the assumption that 1 g of body weight equates to 1 mL of blood. On this basis, a 2-kg bird is able to donate between 20 mL (1%) and 40 mL (2%) of blood. The donar bird should receive three times the volume of collected blood of an isotonic crystalloid fluid such as LRS or 0.9% NaCl (N. Smith, Massey University, pers. comm.). Collection of whole blood from the donor should be done via the right jugular vein and the blood should be collected into a citrate-filled syringe using a relatively large-bore needle or catheter (20 gauge). A concentration of 0.14 mL CPD (citrate phosphate dextrose) is recommended per 1 mL of blood collected (V. Walsh, Massey University, pers. comm.). Blood should be filtered using a blood filter fitted to the syringe (see Fig. 5), and administered to the recipient via a peripheral vein. The medial metatarsal vein and the right jugular vein are common sites for blood administration, but intraosseous administration can also be used. For further information on this procedure in kiwi, contact the New Zealand Wildlife Health Centre (see Appendix 2).

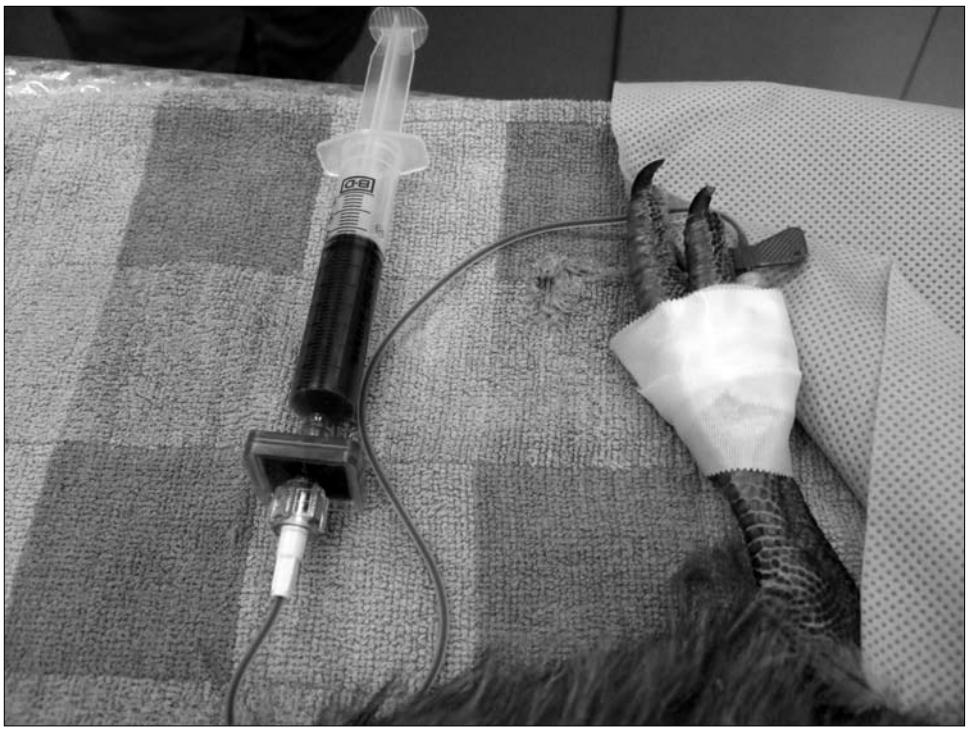


Figure 5. Transfusing whole blood to a kiwi. Note the use of a blood filter attached to the syringe.
*Photo: J. Youl.*

### 2.2.7 Analgesia

Not only do birds perceive and respond to noxious stimuli, but they also feel pain (Paul-Murphy 2006). Misconceptions about the ability of birds to perceive pain arise because of difficulties in recognising bird behaviour associated with both acute and chronic pain (Paul-Murphy 2006). These challenges are probably a result of the preservation reflex that birds demonstrate, which may have evolved as a way of minimising the attention of predators (Paul-Murphy 2006). If the bird demonstrates changes in posture, temperament or behaviour, or if a procedure or injury involves tissue damage, it should be assumed that the bird is in pain (Machin 2005).

Timely administration of analgesics is important, as persistent pain perception can have a negative effect on homeostasis and healing (Wright et al. 1985; Clyde 1994).

Pain management in birds includes the use of analgesics in combination with non-pharmacological methods of analgesia, such as supporting or bandaging the traumatised area. Provision of a dry, warm, quiet and non-stressful environment is also important for pain control (Machin 2005).

There is much uncertainty amongst non-avian veterinary practitioners as to the most appropriate analgesic therapy to use in birds (Hawkins & Machin 2004). Choices of analgesics for birds include opioids and non-steroidal anti-inflammatory drugs.

The current recommendation for opioid analgesia in parrots is butorphanol given at 1–3 mg/kg IM (intramuscularly) (Paul-Murphy 2006). In one study, plasma concentrations of 2 mg/kg butorphanol in African grey parrots (*Psittacus erithacus*) had a mean residence time of less than 2 hours (Paul-

Murphy 2006), and there are reports suggesting that butorphanol should be readministered every 2–4 hours (Clyde 1994; Clyde & Paul-Murphy 1999). Other references for the frequency of medication give values that vary from every three hours to once daily (Marx 2006).

The New Zealand Wildlife Health Centre uses a dose rate for butorphanol of 4 mg/kg IM or IV at least twice daily in kiwi (pers. obs.). Because of their flightlessness, kiwi lack large pectoral muscles for intramuscular administration of therapeutics, and intramuscular injections are usually given in the hindlimbs. To minimise muscular damage, the intravenous route is preferred to intramuscular injection for any more than short-term parenteral administration of therapeutics.

Pre-anaesthetic use of butorphanol will also allow a reduction in the concentration of isoflurane needed for induction and maintenance of anaesthesia (Paul-Murphy 2006).

Pigeons possess a higher proportion of κ receptors in their forebrains than mammals (Hawkins & Machin 2004; Paul-Murphy 2006). For this reason, birds do not appear to respond to μ agonists in the same manner as mammals, and there are conflicting reports of the efficacy of other opioids in avian species (Hawkins & Machin 2004).

Non-steroidal anti-inflammatory drugs (NSAIDs) for use in birds include meloxicam (Metacam™) and carprofen (Rimadyl™). NSAIDs are useful for relief of musculoskeletal and visceral pain, acute pain associated with trauma and surgery, and chronic pain such as that associated with osteoarthritis (Paul-Murphy 2006). These drugs are contraindicated when severe hypovolaemia, renal or hepatic dysfunction and gastric ulceration are present (Paul-Murphy 2006). It is absolutely imperative that the kiwi is well hydrated prior to NSAID administration. For this reason, the author does not recommend the use of NSAIDs in the initial medical treatment of injured kiwi (i.e. during the first 24 hours). Butorphanol should be used for analgesia until the bird is stable, with a restored circulatory system. To reduce the risk of renal side effects, NSAIDs should always be given in conjunction with fluid therapy.

Suggested dosage regimes for meloxicam in avian species are variable and are, in general, anecdotal or published without reference. Frequency of administration is generally recommended as once daily. Intravenous administration of meloxicam in ostriches (*Struthio camelus*) demonstrated a rapid half-life compared with other avian species (Hawkins 2004), and Wilson et al. (2004) make the suggestion that the larger the bird, the shorter the half-life. The New Zealand Wildlife Health Centre uses a regime of once-daily administration of oral meloxicam in kiwi at a dose rate of 0.1–0.2 mg/kg, with concurrent fluid therapy.

Carprofen may be given to birds at a dose rate of 2–10 mg/kg intravenously, intramuscularly, subcutaneously or orally (Marx 2006). Dosing intervals are also variable, and are reported as once to twice daily (Marx 2006).

Paul-Murphy (2006) advocates the use of multimodal therapy, using a combination of both butorphanol and a NSAID. This may provide a wider spectrum of analgesia, and usually allows the dosages of each drug to be reduced, minimising side effects (Paul-Murphy 2006).

### 2.2.8 Antimicrobial therapy

The use of antimicrobial therapy in injured or sick kiwi should be considered. In many situations, it is immediately obvious when antimicrobial medication is warranted, e.g. possum trap injuries, open fractures, and yolk sacculitis in chicks. Septicaemia and bacteriaemia should be considered in any bird that is severely depressed, and prophylactic antibiotics are often used in birds that are immunocompromised from a non-infectious disease (Quesenberry & Hillyer 1994).

In such cases, treatment with antibiotics and, possibly, antifungals in combination with supportive care, should begin without delay. In other instances, the decision to start antimicrobial therapy will depend upon results of diagnostic testing, including faecal Gram stains, and culture and sensitivity of body fluids and tissue.

Antimicrobials should be used cautiously, as there are adverse effects associated with their use. These include the direct toxic effects of some antimicrobial drugs (e.g. nephrotoxicity associated with gentamicin and amphotericin B), and inhibition of normal gastrointestinal flora, resulting in yeast and bacterial overgrowths. It is important to consider possible side effects and toxicities before beginning antimicrobial therapy.

#### *Antibiotics*

Parenteral (injected) antibiotics are recommended for the initial treatment of birds that are weak, sick, debilitated or in shock (Ritchie 1991). Intravenous administration of antibiotics gives a peak plasma concentration within seconds, intramuscular injection takes 30 to 60 minutes to reach peak plasma concentration, and oral administration takes between 60 and 120 minutes (Quesenberry & Hillyer 1994).

If an IV line is set up for fluid therapy, antibiotics can be given intravenously. This is ideal, as it saves handling and stress on the bird when further medications are administered. Alternatively, subcutaneous or intramuscular routes can be used. Repeated intramuscular injections are not recommended because of the possibility of injection-site muscle necrosis (Quesenberry & Hillyer 1994). In addition, kiwi lack pectoral muscles, requiring intramuscular injections to be given into the hindlimbs. The renal portal blood flow system of birds may allow passage of the drug through the kidney prior to systemic circulation. If the drug used is renally excreted, this may mean that it is cleared prior to distribution to target tissues (Flammer 1994). Disadvantages associated with subcutaneous administration include the possibility of leakage from the injection site and poor absorption (Quesenberry & Hillyer 1994). Oral medications can be used once the bird's condition is stable.

Initially, before the results of culture and sensitivity testing are available, antibiotics should be chosen depending upon the clinical signs and history of the bird, and any initial diagnostic results. Birds with suspected gram-negative septicaemia should be treated with a bactericidal antibiotic effective against the most common avian pathogens, including *Escherichia coli, Enterobacter* spp., *Klebsiella* spp. and *Pseudomonas* spp. (Ritchie 1991). Examples of commonly used first-choice broad-spectrum antibiotics in avian medicine effective against Enterobacteriaceae include amoxicillin/clavulonic acid, enrofloxacin and trimethoprim-sulfa (Quesenberry & Hillyer 1994).

### *Antifungals*

Antifungal therapy should be instigated when fungal infections are suspected, and may be used prophylactically during antibiotic therapy to decrease the risk of secondary yeast overgrowths in the gastrointestinal system, especially in immunosuppressed birds (Harrison et al. 2006).

Yeast infections confined to the gastrointestinal tract, candidiasis in particular, can be treated with nystatin (Flammer 1994). This drug is not absorbed from the gastrointestinal tract, and relies on contact for effect (Flammer 1994).

Itraconazole is currently the first-choice oral antifungal for treatment of aspergillus infections in birds (Dahlhausen 2006). Concurrent nebulisation with an antifungal (e.g. fluconazole, amphotericin B) is recommended for treatment of fungal air sacculitis.

Dahlhausen (2006) recommends the use of amphotericin B for aspergillosis. It may be administered intratracheally, intravenously, in sinus flushes and via nebulisation. However, this drug should be used for short durations only, as it is eliminated renally and there is an associated risk of nephrotoxicity (Dahlhausen 2006). For this reason, amphotericin B should be used with extreme caution in dehydrated birds or those suspected of having renal disease.

### *Nebulisation*

Nebulisation may be beneficial for birds with air sacculitis. Blood supply to the air sacs is extremely limited (King & McLelland 1984), and parenteral and oral antimicrobial therapeutics are thus ineffective in treatment of air sacculitis. Nebulisation provides topical, localised treatment of the internal air sacs and is not dependent upon absorption. In general, most parenteral medications formulated for intravenous use (i.e. particle size less than 3 μm) can be suspended in saline and used for nebulisation therapy (Quesenberry & Hillyer 1994). The suggested protocol for nebulisation is for 10 to 30 minutes, two to four times daily.

Agents that are commonly used for nebulisation at the New Zealand Wildlife Health Centre include amphotericin B and enrofloxacin. Diluted forms of the disinfectant F-10™ are also being used by avian practitioners for nebulisation. However, recent histopathological evidence suggests that this may cause a chemically-induced pneumonia (B.D. Gartrell, Massey University, pers. comm.).

### 2.2.9 Other medications

#### *Corticosteroids*

The use of corticosteroids in birds has been widely debated because of the potential complications arising from their use (Orcutt & Flinchum 2001). Potential side effects of corticosteroid use include immunosuppression, adrenal suppression, delayed wound healing, and gastrointestinal ulceration (Harrison et al. 2006). Corticosteroids are contraindicated in birds with a history of immunosuppression or fungal disease. The author recommends that corticosteroids are not used in routine therapy of injured or sick kiwi.

#### *Glucose therapy*

Hypoglycaemia may occur in cases of starvation or malnutrition, sepsis or hepatic dysfunction. Intravenous dextrose alone should not be used in dehydrated patients, and should be given in conjunction with IV fluids (e.g. half-strength LRS and 2.5–5% dextrose). Alternatively, 25% dextrose can be given as an intravenous bolus at a rate of 1–2 mL/kg, slowly to effect (Harrison et al. 2006). This can be continued in fluids at 2.5–10% IV or IO (intraosseous). Oral glucose solutions can be used in birds that are not prone to aspiration (Harrison et al. 2006).

#### *Nutritional support*

A sick or debilitated bird should always have its hydration corrected prior to any attempt to initiate oral gavage feeding (Harrison et al. 2006). In practice, nutritional support is rarely provided to debilitated kiwi in the first 24–48 hours of care. Useful nutritional formulas for kiwi include tube feeding of Hill's a/d™ diet or Wombaroo™ Insectivore Mix, and various preparations of a captive kiwi mince mix (see section 5.1 and Appendices 4 and 5).

Figure 6. Kiwi with a modified external fixator stabilising multiple mandibular fractures. An oesophagostomy tube was placed to allow feeding in the initial phase after surgery. *Photo: K. Morgan.*

An oesophagostomy feeding tube may be placed in birds with bill injuries (see Fig. 6). However, it is difficult to maintain the bird's body weight when feeding by this method (see section 5.1).

## 2.3 INITIAL WOUND AND FRACTURE MANAGEMENT

### 2.3.1 Wound management

During patient stabilisation, and prior to thorough wound assessment and management, Burke et al. (2002) recommend protecting the wound with a temporary bandage. This can be achieved by packing the wound with moistened sterile gauze swabs or by filling the wound cavity with a water-soluble lubricating gel (e.g. KY™ jelly).

Once the bird is in a stable condition, the wound should be thoroughly assessed for location, extent and age (Degernes 1994). Evaluation of underlying orthopaedic injuries and the vascular and nerve supply to the tissue is especially important (Degernes 1994; Burke et al. 2002). Development of a greenish discolouration of traumatised skin is normal in birds. This often occurs 2–3 days post-injury as a result of the accumulation of biliverdin pigment following the breakdown of haemoglobin, and it may persist for over a week (Degernes 1994). Necrotic tissue is black or blanched white (Burke et al. 2002).

Initially, feathers surrounding the wound should be plucked or trimmed to allow the full extent of the wound to be clearly visible. Feather plucking in birds is extremely painful, and it should be done under general anaesthesia. Alternatively, a water-based lubricant (KY™ jelly) can be applied to flatten-down surrounding feathers and allow better visualisation and assessment of the wound.

The wound should be lavaged with copious amounts of warmed sterile saline (with or without 0.05% chlorhexidine or 0.5–1.0% povidone iodine) to remove debris (Degernes 1994). This helps to reduce the number of bacteria present, as well as providing tissue rehydration. It is recommended that samples for cultures be obtained after surface contaminants have been removed from the wound and before any antiseptics have been applied (Degernes 1994; Burke et al. 2002).

Surgical debridement of dead and devitalised tissue under general anaesthesia (isoflurane with oxygen) is recommended once the bird is stable (Burke et al. 2002). The goals of wound debridement include removing as much of the devitalised and necrotic tissue as possible until viable, vascularised tissue can be seen (Degernes 1994).

Application of topical wound medication is indicated when treating infected wounds (Burke et al. 2002). These medications need to be water soluble to avoid the loss of insulation resulting from soiled feathers and to prevent feather contamination with oily substances (Degernes 1994). Useful topical medications include iodine, antibiotic creams, Solosite™ and silver sulfadiazine.

Suturing of wounds is only indicated if the wound is clean and less than 8 hours old (Degernes 1994). This is very rarely the case in wildlife medicine. Most wounds will need to be managed as open wounds and allowed to heal via secondary intention, requiring the application of dressings and bandages.

### *Bandaging principles*

The primary layer is in direct contact with the wound, and is the most critical layer for optimal wound healing. Functions of the primary layer include provision of a moist wound environment and debridement of the wound (Degernes 1994). This layer should be sterile and remain in place even when the bird moves. Adherent and non-adherent dressings can be used, depending upon the stage of wound healing.

Adherent dressings, such as sterile saline-soaked gauze swabs, are useful in the initial stages of wound management when there is a large amount of necrotic debris that cannot be surgically debrided (Degernes 1994; Burke et al. 2002). They act to absorb exudates, and contribute to wound debridement during dressing changes. These adherent dressings need to be changed on a daily basis, and should only be used for 2–4 days before being replaced with a non-adherent bandage. Prolonged use of adherent dressings will interfere with granulating epithelium. Other disadvantages of such wet-to-dry bandages include tissue maceration and bacterial colonisation in the moist environment (Degernes 1994).

Non-adherent bandages do not adhere to the healing wound surface (Degernes 1994). Semi-occlusive, non-adherent dressings (e.g. Melolin™) are the most useful for avian medicine. These are indicated after tissue debridement, or for use on clean fresh wounds that do not require debridement. They should be changed at least every 48–72 hours.

Occlusive hydrocolloidal bandages (e.g. Duoderm™) have been used for management of wounds in raptors (Aguilar 2004). These bandages keep the wound surface moist, preventing the formation of a scab and increasing the rate of re-epithelisation and wound healing. They should only be used on non-infected wounds. Initially, dressings should be changed every 72 hours, then replaced weekly thereafter (Aguilar 2004).

The secondary layer of the bandage consists of soft padding (e.g. Sofban™) that acts to absorb fluids and exudates, as well as providing protection and immobilisation of the wound.

Self-adhesive bandaging (i.e. which sticks only to itself) (e.g. Vetrap™, Coflex™) should be used as the tertiary layer. Adhesive bandaging materials (i.e. those with a sticky surface) should not be used, as they adhere to and damage feathers.

## 2.3.2 Fracture management—external coaptation

### *Distal hindlimb fractures*

Bandaging techniques for fracture management in kiwi are limited to fractures of the lower hindlimb. The modified Robert Jones bandage (Fig. 7) is the most useful and practical form of external coaptation (joining the bone edges together).

Indications for the Robert Jones bandage include fractures to the distal third of the tibiotarsus, fractures of the tarsometatarsus, injuries involving the hock joint, soft tissue wounds of the tibiotarsus or tarsometatarsus, and following orthopaedic repair of the distal two-thirds of the limb (Degernes 1994).

Figure 7. Modified Robert Jones bandage on a North Island brown kiwi with a trapping injury to the distal hindlimb.
*Photo: J Youl.*

The Robert Jones bandage is contraindicated for fractures of the femur and the proximal two-thirds of the tibiotarsus (Degernes 1994) and may cause further tissue disruption and discomfort to the bird (pers. obs.)

A thick layer of cotton wool casting material (Sofban™) is wrapped from the most proximal part of the leg (i.e. the part closest to the body) (Degernes 1994). The leg should be slightly flexed in a normal standing position. Conforming gauze material is tightly applied around the padding, and a self-adherent material is used to cover the bandage (Degernes 1994). The toes of the bandaged limb should be monitored for swelling and discolouration if they are not incorporated within the bandage (Degernes 1994).

### *Proximal hindlimb fractures*

There are no effective bandaging techniques for upper tibiotarsal, femoral or pelvic fractures in kiwi. Any attempts to bandage high up the limb results in poor immobilisation of the stifle, and no immobilisation of the hip, and may cause further tissue disruption and discomfort to the bird (pers. obs.). In this instance, provision of soft bedding and confinement in a small enclosure is indicated to minimise further trauma and pain associated with the fractures until surgical stabilisation (if indicated) is achieved.

# 3. General anaesthesia

## 3.1 IMPORTANT CONSIDERATIONS IN AVIAN ANAESTHESIA

### 3.1.1 Air sacs and positioning

The respiratory system in birds differs from mammals in that the lungs are small and undergo little change in volume during breathing, and birds do not have a diaphragm (O'Malley 2005). Instead, birds possess a system of airsacs which act as bellows to provide airflow to the rigid avian lung during both inspiration and expiration (O'Malley 2005; Edling 2006). The air sac system of the flightless kiwi appears to be much less extensive than those of other avian species (pers. obs).

How a bird is positioned during anaesthesia can significantly alter its ventilation. In dorsal recumbency, the weight of the visceral organs can compress caudal air sacs, reducing their effective volume (O'Malley 2005). For this reason, tidal volume (the amount of gas passing through the lungs on each breath) may be reduced by as much as half when the bird is lying on its back (O'Malley 2005). Birds should be maintained in sternal (upright) or lateral (side) recumbency as much as possible. If a bird must be kept in dorsal recumbency for a period, adequate ventilation can be achieved by the use of intermittent positive pressure ventilation (IPPV) (Edling 2006).

### 3.1.2 Patient stabilisation

During the early stages of assessing a sick or injured kiwi, it may not be possible to perform basic necessary procedures on a conscious bird. A brief period of anaesthesia may be required in order to establish intravenous or intraosseous access for fluid therapy, to address any wounds or fractures, and to obtain a blood sample for haematology and biochemistry. This anaesthesia should be kept as light and short as possible to allow the necessary procedures to be carried out without compromising an already debilitated patient. Once the bird's body temperature and hydration have been stabilised, it will be able to tolerate longer periods of anaesthesia. This is generally 24 hours or more after initial presentation of the bird.

#### *Thermal regulation*

Birds are not efficient homeotherms and they rapidly lose heat when they do not remain metabolically active (Forsyth et al. 1999). The low surface area to volume ratio of birds facilitates heat loss, and anaesthesia reduces a bird's physiologic response to the reduction in body temperature (Edling 2006). The use of dry anaesthetic gases further increases heat loss, as does the removal of feathers for surgical procedures and the use of surgical skin preparations (Edling 2006).

Thermal regulation in anaethetised birds includes minimising anaesthetic time, as well as providing an external heat source during anaesthesia. Heat sources may be heated surgery tables (e.g. heat pads), overhead heat lamps,

warm towels and warm IV fluids (Edling 2006). Warming the room prior to anaesthesia will also help. Radiant heat sources are more effective than under-bird heating because of the insulating properties of the bird's feathers. Insulation is necessary between the patient and the surgical table, and may be provided with towels or plastic bubble wrap.

### 3.1.4 Fasting

In general, for large birds in good physical condition, removing food the night before and water 2–3 hours prior to anaesthesia does not appear to be harmful (Franchetti & Kilde 1978). In contrast, it has been recommended that fasting be limited to no more than 2–3 hours in smaller bird species because of their high metabolic rate and poor hepatic glycogen storage (Edling 2006).

In kiwi, the author recommends a fasting period of 12–24 hours prior to anaesthesia in order to reduce the hazards associated with regurgitation. Kiwi do not possess a crop or a large proventriculus for storage of food (Fergus et al. 1995), and for this reason, if a bird is fasted appropriately before anaesthesia, the risk of regurgitation and aspiration is minimal.

### 3.1.5 Restraint

During induction of gaseous anaesthesia, using a towel to encircle the patient enables careful restraint of the bird while allowing the sternum freedom of movement for respiration (Edling 2006).

### 3.1.6 Surgical preparation

Feathers should be plucked for surgical preparation. This is an extremely painful procedure, and should only be done under general anaesthesia. Feathers should be pulled 1–2 at a time in the direction the shaft is growing (usually caudally) (Cannon 2001). Cutting feathers is not recommended as the bird will need to go through a normal moult to replace those feathers, and this prolongs the rehabilitation process.

Skin should be prepared for surgery using a chlorhexidine or iodine scrub and aqueous chlorhexidine (0.05%). Alcohol should not be used in birds as it has a significant cooling effect, contributing to the risk of hypothermia (Cannon 2001).

## 3.2 ANAESTHETIC EQUIPMENT

### 3.2.1 Inhalant anaesthetics

The author advocates the sole use of inhalational anaesthetic agents for the induction and maintenance of anaesthesia in kiwi. Isoflurane is the anaesthetic agent of choice for avian anaesthesia (Cannon 2001). Sevoflurane is also an excellent anaesthetic agent for use in birds (Edling 2006), although it is not regularly available in New Zealand practice.

Halothane is no longer considered safe for use in avian species as there is a close time interval between apnoea (cessation of unassisted breathing) and cardiac arrest (Cannon 2001), and halothane sensitises the heart to catecholamine-induced cardiac dysrhythmias (Edling 2006). There have been fatalities associated with use of halothane for anaesthesia in stressed birds with high pre-existing levels of catecholamines (Edling 2006).

### 3.2.2 Breathing circuits

Non-rebreathing circuits, such as the Ayre's T-piece and the Bain circuit, are indicated for use in avian species as they have the least dead space and the lowest resistance (Forsyth et al. 1999; Edling 2006).

### 3.2.3 Induction of anaesthesia

A mask for the induction of anaesthesia in kiwi can be fashioned from a 20-mL syringe barrel or a syringe case. A disposable latex glove can be taped over the end of the mask opening with a small central hole for placement of the kiwi bill (Fig. 8).

Induction techniques include a period of pre-oxygenation, after which the concentration of gaseous anaesthetic agent is slowly increased until the desired effect has been attained (Edling 2006). This is the author's preferred method, especially for inexperienced avian anaesthetists, as it allows a more gradual attainment of anaesthesia.

The oxygen flow rate should be approximately 1.5–2 L/minute, and a period of pre-oxygenation of approximately 30 seconds is recommended. The concentration of isoflurane should be increased in 0.5% increments, with a period of approximately 20–30 seconds between each increase. The vaporiser setting can then be reduced to a setting near the minimum anaesthetic concentration (MAC) for maintenance (Edling 2006). The author finds that a light to surgical plane of anaesthesia in kiwi is usually reached at 3–4% isoflurane, and can be maintained at around 1.5–2%.


Figure 8. Induction of isoflurane anaesthesia using a modified syringe case with a latex glove over the end for a mask.
*Photo: K. Morgan.*

### 3.2.4 Intubation

Intubation is usually not necessary for short (less than 10 minutes), non-invasive procedures (Edling 2006). For longer periods of anaesthesia, intubation can be crucial.

The glottis can be easily seen in kiwi at the base of the tongue (Fig. 1). Avian species do not possess an epiglottis, making intubation relatively easy.

The endotracheal tube should provide a good seal with the glottis but should not fit tightly. Birds have complete cartilaginous tracheal rings, contraindicating inflation of cuffed endotracheal tubes. Inflation of the cuff can cause pressure necrosis and consequential stricture of the tracheal mucosa (Edling 2006). Non-cuffed avian tubes are available (Cole™ tubes). In adult kiwi, size 25–35 Cole's tubes are typically adequate. If a Cole tube is unavailable, normal endotracheal tubes (sizes 2.5–3.5) may be used without inflating the cuff. Silicone tubes are preferable to the more rigid plastic ET tubes, as they are softer and are less likely to cause injury to the tracheal mucosa.

Once a kiwi is intubated, the endotracheal tube should be securely attached to the lower and/or upper bill, with Sleek™ or similar tape (Fig. 9).

### 3.2.5 Pre-anaesthetic agents

The use of butorphanol prior to anaesthesia has been found to reduce the concentration of isoflurane needed to maintain anaesthesia in other avian species (Edling 2006; Paul-Murphy 2006). (See section 2.2.7 for further details.)


Figure 9. Intubated kiwi connected to anaesthetic machine.
*Photo: V. Gray.*

## 3.3 PATIENT MONITORING

The most common problems associated with avian anaesthesia include apnoea, hypoventilation, hypothermia and regurgitation (Edling 2006). Veterinary equipment, including a Doppler flow monitor and/or pulse oximeter attached to a toe or lower leg can provide a useful audio tool for monitoring pulse rate.

### 3.3.1 Respiratory system

Respiration during anaesthesia should be monitored both visually and by auscultation. Ideally, end tidal $CO_2$ (i.e. that expired at the end of each breath) would also be measured via capnography (monitoring the pressure of $CO_2$ in respiratory gases). However, this is often not possible because of the lack of appropriate monitoring equipment in many general veterinary practices.

### 3.3.2 Cardiovascular system

The aneasthetised bird should be regularly monitored via auscultation to observe changes in heart rate and rhythm. The heart rate should be quantified to monitor for change. The normal kiwi heart rate has been documented as 70–240 beats per minute (Doneley 2006).

### 3.3.3 Central nervous system

Parameters that can be used to assess anaesthetic depth in birds include eye reflexes, jaw tone, cloacal reflex, pedal reflex and muscle relaxation. Muscle tone is assessed by progressive relaxation of the leg, and appropriate anaesthesia is generally achieved once muscle tone in the limb is absent. One study described the ideal anaesthesia level as when the patient's eyelids were closed, pupils mydriatic, the pupillary light reflex was delayed, the nictitating membrane moved slowly over the entire cornea, all muscles were relaxed and all pain reflexes were absent (Korbel et al. 1998).

Cannon (2001) gives the following guide to stages of anaesthesia (commonly used reflexes in larger birds include the eye reflexes (corneal, palpebral) and the interdigital (toe pinch) reflex):

| | |
|---|---|
| Light plane of anaesthesia | Reflexes present<br>Deep, rapid respiration<br>No voluntary movement<br>Legs flexed |
| Medium plane of anaesthesia | Sluggish reflexes<br>Slow, deep, regular respiration<br>Legs relaxed |
| Deep plane of anaesthesia | No reflexes<br>Slow, shallow respiration<br>Close to cardiac/respiratory arrest |

### 3.3.4 Oxygenation

The colour of the mucous membrane can be monitored for signs of change, but it is not an effective monitoring tool for patients in critical condition (Edling 2006). Pulse oximetry (using red and infrared light sources) is not consistently accurate for measuring oxygen saturation in birds (Schmitt et al. 1998).

### 3.3.5 Temperature

Without thermal support, birds under anaesthesia rapidly lose heat (Edling et al. 2006). A long, flexible, oesophageal thermometer inserted to the level of the heart can be used to reliably monitor body temperature (Edling et al. 2006). Cloacal temperature may be monitored during anaesthesia. However, this is less reliable than using an oesophageal thermometer, as reported temperature can change depending on body position and cloacal activity (Edling et al. 2006). The normal body temperature of kiwi has been documented as being lower than that of other avian species at 38°C ±1.75°C (Farner et al. 1956).

## 3.4 ANAESTHETIC EMERGENCIES

Table 1 lists emergency drug treatments extrapolated from those commonly used for other anaesthetised avian species (Edling 2006; Marx 2006).

TABLE 1. AVIAN EMERGENCY DRUGS FORMULARY.

| DRUG | WHAT ACTION DOES DRUG PERFORM? | DOSAGE* |
|---|---|---|
| Adrenaline | Adrenaline is a positive inotrope and chronotrope. It initiates heartbeats, and increases heart rate and cardiac output. | 0.1 mg/kg (10000IU/mL) IO/IV/IP/IT |
| Atropine | Atropine has parasympatholytic effects. It may correct supraventricular bradycardia or a slow ventricular rhythm by stimulating supraventricular pacemakers. | 0.01–0.02 mg/kg IM, IV |
| Doxapram HCl | Doxapram HCl is a positive inotrope and stimulates breathing. | 5–20 mg/kg IV/IO/IM/IT; can also place a drop on tongue for cardiac arrest |
| Isotonic crystalloids | These expand blood volume and increase tissue perfusion during hypotension. | (See section 2.2.5) |
| Diazepam | For control of seizures. | 0.5–1 mg/kg IM |

* IV = intravenous, IO = intraosseous, IP = intraperitoneal, IT = intratracheal, IM = intramuscular.

# 4. Diagnostics

## 4.1 EVALUATION OF DROPPINGS

The droppings of a bird consist of three compartments—the faecal fraction, the white urates and the liquid urine. Gross evaluation of the colour, texture, consistency and volume of each of these will provide information about a bird's appetite, and gastrointestinal, renal and hepatic functions (Harrison & Ritchie 1994).

A reduction in the frequency of defecation and/or volume of excrement can be an indication of decreased food intake, decreased gastrointestinal transit time, or an obstruction. Food and water deprivation may be indicated by dry, scant droppings (Harrison & Ritchie 1994).

### 4.1.1 Faeces

Normal faeces should be well formed, homogenous and brown. They have a strong odour, and the colour may depend on the diet. Kiwi that have been eating berries may have pigmented faeces. Normally, faeces are smooth and present as a tight-gelled cylindrical shape. However, a stressed bird may have stress-related polyuria indicated by an increased urinary fraction, and/or diarrhoea.

Diarrhoea may be stress related, or it may indicate bacterial or parasitic enteritis, or a dietary change. The physical characteristics of the faecal fraction of a bird's faeces may be influenced by any medications administered (Harrison & Ritchie 1994).

Meleana has been described as looking like coffee granules, and is indicated by dark-coloured faeces. Meleana suggests the presence of old blood. The colouration may originate from the gastrointestinal tract, the oviduct/testes, kidneys or the cloaca. The presence of frank blood may be associated with coagulopathies (blood clotting problems), liver disease, cloacal pathology, pre/post-ovulation, malnutrition, enteritis or traumatic gastritis (Harrison & Ritchie 1994).

#### *Faecal diagnostics*

**Direct wet mount**

Direct wet mounts require ultra-fresh faeces (<15 minutes old) to enable diagnosis of motile protozoa, and are also useful for diagnosing helminths and coccidia. A drop of faeces is placed on a (preferably warm) glass slide, a drop of 0.9% saline is added, a coverslip is placed on top, and the wet mount is evaluated microscopically.

**Gram's stain (or Dif-Quik)**

A faecal Gram's stain is useful for determining the type and relative number of each microbial organism present in a faecal sample (i.e. gram positive v. gram negative bacteria, yeasts). Dif-Quik is useful for evaluating yeasts and cytology, but this stain does not enable gram-positive and gram-negative bacteria to be distinguished.

A small amount of fresh faeces is applied to a pre-cleaned glass slide using the wooden end of a cotton-tipped applicator. The sample is spread into a uniform, thin film, and heat fixed prior to staining. Once stained, the slide should be scanned under low microscopic power to determine a suitable evaluation site; then, using oil immersion, several fields should be scanned.

Normal faecal cytology of kiwi includes mixed gram-negative and gram-positive bacteria, with scant, non-budding yeasts. An abnormal faecal cytology includes a low bacterial count, an increased number and percentage of budding yeasts, and a relative overgrowth of a particular bacterial type.

Evaluation of the Gram's stain requires the clinician to determine if the organism detected is pathologically colonising a mucosal surface. Unnecessary antibiotic therapy instituted from an improperly evaluated Gram's stain can precipitate the colonisation of opportunistic pathogens (Harrison & Ritchie 1994).

**Cloacal culture**

The New Zealand Wildlife Health Centre advocates routine screening by cloacal cultures for *Campylobacter* spp., *Salmonella* spp. and *Yersinia* spp. during translocation of kiwi. Recent evidence suggests that *Campylobacter* spp. may not be pathogenic for kiwi and many other New Zealand bird species, and this bacteria may be able to be removed from the screening programme (B.D. Gartrell, Massey University, pers. comm.). However, *Campylobacter* can cause severe gastrointestinal disease in humans and care should be taken to prevent human infection from birds. *Escherischia coli* are present in most cloacal/faecal samples from kiwi, suggesting that (as with other omnivorous and insectivorous avian species) this organism is part of the normal enteric flora of the kiwi (B.D. Gartrell, Massey University, pers. comm.).

Cloacal cultures are also indicated when a bacterial enteritis is suspected. Commonly, the gram negative Enterobacteriaceae are the causative organisms of bacterial enteritis in avian species (Gelis 2006).

Routine sterile transport culture swabs are used for insertion into the cloaca. It is best to moisten the swab with sterile transport media or LRS before insertion to prevent the dry swab from causing tissue damage. Transportation media is required for transportation of the swab for culture, and it should be sent immediately to prevent bacterial overgrowth. If there is a delay in transportation, the swab should be refrigerated.

**Faecal floatation**

A faecal floatation should be performed to detect the presence of endoparasite eggs and coccidial oocysts. These can be done in-house or at a commercial laboratory.

### 4.1.2 Urates/urine

Urates are normally pasty white-yellow and slightly moist. Uric acid is synthesised in the liver and excreted through the kidneys by glomerular filtration as insoluble nitrogenous waste (Harrison & Ritchie 1994).

Yellow-green discolouration of the urates is indicative of haemolysis or hepatitis, or may indicate a recent traumatic event with the breakdown of red blood cells. Fresh droppings need to be evaluated, as urates may

become green-stained due to leaching of pigments from the faecal fraction. Haematuria or bleeding from the gastrointestinal tract, cloaca, urinary tract or genital tract may be evident as red staining of the urates or urine (Harrison & Ritchie 1994).

Urine is usually clear, colourless and small in volume, forming the outer portion of the excrement. It is difficult to collect urine on its own for urinalysis due to contamination by faeces and urates. Birds often develop a stress polyuria.

## 4.2 HAEMATOLOGY AND BIOCHEMISTRY

A blood sample should be collected as soon as possible from injured or sick kiwi, ideally prior to any form of treatment. This will ensure optimal diagnostic value as well as providing a good tool for monitoring progression of treatment.

One of the most important points to note is that kiwi whole blood lyses rapidly in EDTA (see section 4.2.1).

### 4.2.1 Blood collection

A suggested protocol for blood sampling in kiwi is as follows:

- Fresh smear (without anticoagulant)
- > 0.25 mL into lithium heparin microtainer for haematology
- > 0.5 mL into a second lithium heparin microtainer for biochemistry

#### *Blood volume*

It is recommended that no more than 1% of a healthy bird's total body weight is taken in a blood sample (Hume 1995; Fudge 2000). For example, in a healthy 1-kg bird, 10 ml is the maximum blood volume recommended to be taken at any one time. In an unhealthy bird, 0.5% of the total body weight (i.e. 5 ml from a 1-kg bird) may be safer.

#### *Collection equipment*

A fine needle (23–25 gauge) and a 1–3-mL syringe are ideal. Avian veins are subject to collapse if a high negative pressure is applied to a large syringe. The use of a 1-mL syringe can minimise this.

#### *Blood sampling sites*

The preferred sites for blood collection in kiwi are the medial metatarsal vein (see Fig. 3) or the right jugular vein. The medial metatarsal vein is located above the hock joint on the medial side of the leg. Haematoma formation at this site is uncommon because it is substantially immobilised by surrounding tissues (Fudge 2000). The site should be aseptically prepared with an alcohol swab prior to venipuncture.

The right jugular vein is larger than the left, and is located in a featherless tract along the jugular furrow. The jugular vein is located dorsal to the trachea, which should be manipulated ventrally to allow the vein to be located. The bird must be securely manually restrained, or under general anaesthetic.

For both sites, haematoma formation can be minimised by careful compression of the site after venipuncture.

### *Sample containers*

Because the sample sizes required for haematology and biochemistry are small, it is recommended that micro collection tubes are used (Microtainers™, Becton-Dickinson). These are 0.6-mL blood tubes that are available containing lithium heparin anticoagulant (also available plain and with EDTA). Alternatively, standard collection Vacutainers™ may be used. However, these should be filled to at least half of their capacity to minimise the effects of excessive anticoagulant (Hume 1995).

### *Anticoagulants*

With kiwi blood, it is recommended that lithium heparin anticoagulant is used for both haematology and biochemistry. **Kiwi blood shows an unusual reaction to EDTA, rapidly haemolysing after 15–30 minutes**. This reaction is similar to that of some other avian species, including ostriches (Hume 1995). For this reason, kiwi blood should not be collected into EDTA tubes.

### *Smears*

Hume (1995) stated that the ideal technique for preparing avian blood slides is the coverslip technique. This is described as follows and shown in Fig. 10:

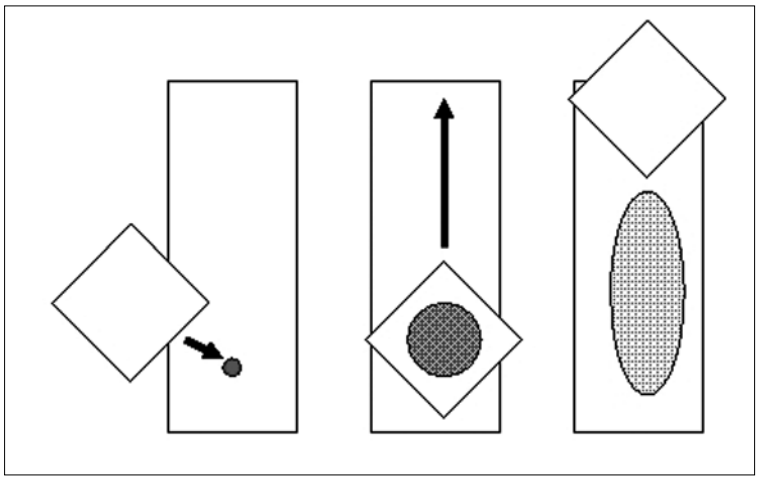


Figure 10. The coverslip method for preparation of avian blood smears.
*Figure: K. McInnes, DOC.*

'A small drop of blood (without anticoagulant) is placed near one end of the slide. If the slide is frosted, place the blood drop at the frosted end of the slide. Place a coverslip on the blood, allowing the blood to fan out. Just before the blood reaches the end of the slide, the two slides should be pulled apart without lifting the coverslip. This technique greatly reduces leucocyte margination and the incidence of smudge cells' (Hume 1995).

The two-slide wedge technique, as commonly used in mammalian medicine, is the least acceptable technique for avian blood. This technique produces an unacceptable number of smudge cells (ruptured white and red blood cells) as well as margination of white blood cells (Hume 1995).

Fixing blood smears in methanol or any other fixative is not recommended. A delay between fixation and staining can result in the reduction in quality of leucocyte granule staining (Hume 1995).

## 4.2.2 Laboratory testing

Most commercial laboratories have standardised methods for analysing avian haematology. A selection of biochemical analytes is often offered as a routine biochemical panel for avian species. Not all of these standard panels include uric acid, and most do not include bile acids. Both of these metabolites should be requested whenever avian blood is sent for analysis, as they are two of the most important biochemical parameters for birds. Alternatives for biochemical analysis of avian blood include using a VetScan™ analyser.

Biochemical artefacts may include an increase in potassium and total plasma protein if the blood is not spun down immediately after collection (Hume 1995). This is because the erythrocytes leak potassium across their cell membranes. Glucose should be analysed within 2 hours on whole blood.

Normal haematological parameters for North Island brown kiwi (*Apteryx australis mantelli*), Okarito brown kiwi (*Apteryx rowi*) and the great spotted kiwi (*Apteryx haastii*) can be found in Appendix 3.

## 4.3 IMAGING

### 4.3.1 Radiography

Radiographs are indicated in all cases of trauma, and they are necessary as part of a routine diagnostic work-up of a sick bird. Survey radiographs should include both laterolateral and ventrodorsal views of the entire bird, and areas of particular interest can be focused on in subsequent radiographs. This ensures that injuries that are less obvious during physical examination are not missed.

#### *Equipment and techniques*

If possible, it is recommended that fine screens be used for radiographing kiwi, as these provide better detail. However, if these are not available, factors and plates used for radiography of small cats are adequate.

Mammography cassettes and film provide excellent high-detail radiographs, and may be useful for fine detail of the extremities of kiwi, or for kiwi chicks.

#### *Restraint*

In order to achieve diagnostic radiographs, to minimise stress on the patient, and to prevent exposure of veterinary personnel to radiation, it is essential that kiwi are restrained under general anaesthesia. Taking radiographs of conscious birds places them under stress and can cause further injuries. It is also very difficult to obtain radiographs that are diagnostically useful if bird positioning is not appropriate. Poor positioning is the most frequently encountered factor compromising a radiography study and hampers the interpretation of subtle lesions in avian radiographs (McMillan 1994).

Respiratory motion is largely unaffected by appropriate planes of anaesthesia, and motion artefact is eliminated by the use of relatively short exposure times (0.01–0.05 seconds) (Helmer 2006).

#### *Positioning*

It is recommended that at least two views of the bird are taken—laterolateral and ventrodorsal.

**Laterolateral view** Ideally, the kiwi should be positioned on the radiograph plate so that the entire bird is in the radiographic field (Fig. 11). Both legs should be pulled in a caudal direction, away from the abdomen, to avoid obscuring the visceral organs. The legs should be splayed apart, with one in front of the other, and taped to the plate using sticky tape. The cranial (forward) leg should be marked as left or right. In order to obtain consistency between radiographs, it is recommended that a system of bird positioning

is developed. At the New Zealand Wildlife Health Centre, the protocol is to consistently put the bird into left lateral recumbency, always with the left leg forward. When assessing appropriate positioning for the laterolateral view, the two femoral heads should overlie one another, the legs should be pulled caudally, and the sternum should be parallel to the film (Helmer 2006).

**Ventrodorsal view** In the ventrodorsal view, the bird is positioned on its back with its legs pulled caudally and secured with tape (Fig. 12). Because of their lack of wings, kiwi often require support to stay in dorsal recumbency. The use of a trough for the bird to lie in, which can be fashioned from a rolled-up towel, may assist. Alternatively, the use of foam wedges should stop the bird from rolling laterally.

Correct positioning will be apparent, as the spine will be superimposed over the centre of the sternum (N.B. kiwi lack a keel), and the acetabula and femurs will be parallel (McMillan 1994).

#### *Contrast studies*

Contrast studies of the upper and lower gastrointestinal tract are often indicated if there is suspicion of a space-occupying mass, gastrointestinal ulceration, abnormalities in size or shape of coelomic organs, gastrointestinal foreign bodies, alterations in gastrointestinal motility or body wall abnormalities (Helmer 2006). Barium sulphate suspension is the most commonly used contrast media. The amount of barium sulphate solution to use may be calculated on the basis of 5 mL per kg of body weight (B.D. Gartrell, Massey University, pers. comm.). The barium solution is introduced into the bird's stomach by gavage. The quantity recommended is much less than that usually recommended for psittacine gastrointestinal barium studies, and relates to the small holding capacity of the kiwi's upper gastrointestinal tract. Care must be taken to avoid aspiration of the contrast media, especially in the early stages of the study when the liquid is in the upper gastrointestinal tract.

BIPS™ (barium impregnated polyethylene spheres) may also be used for evaluating some gastrointestinal motility. These may be a safer form of contrast media in some instances, as there is no risk of aspiration. BIPS have been used on one occasion (dose given 12 hours prior to radiographic examination under anaesthesia) to detect a gizzard impaction (unpubl. data).

Normal gastrointestinal transit time for psittacine species is approximately 3 hours (Helmer 2006). However, this varies depending on species and individual diets, and gastrointestinal transit time in kiwi is unknown. Results of contrast studies should be interpreted in light of associated clinical signs.

### 4.3.2 Advanced avian imaging

#### *Ultrasonography*

Ultrasonography has limited use in birds because the ultrasound waves do not penetrate the gas-filled air sac system, so that organs cannot be clearly seen (Helmer 2006). Interpretation of abnormal findings is also difficult without prior knowledge of normal structures particular to the species. In kiwi, ultrasonography has been useful in identifying yolk sac retention in kiwi chicks, and has been used to diagnose a partial intestinal obstruction in an adult kiwi (unpubl. data).

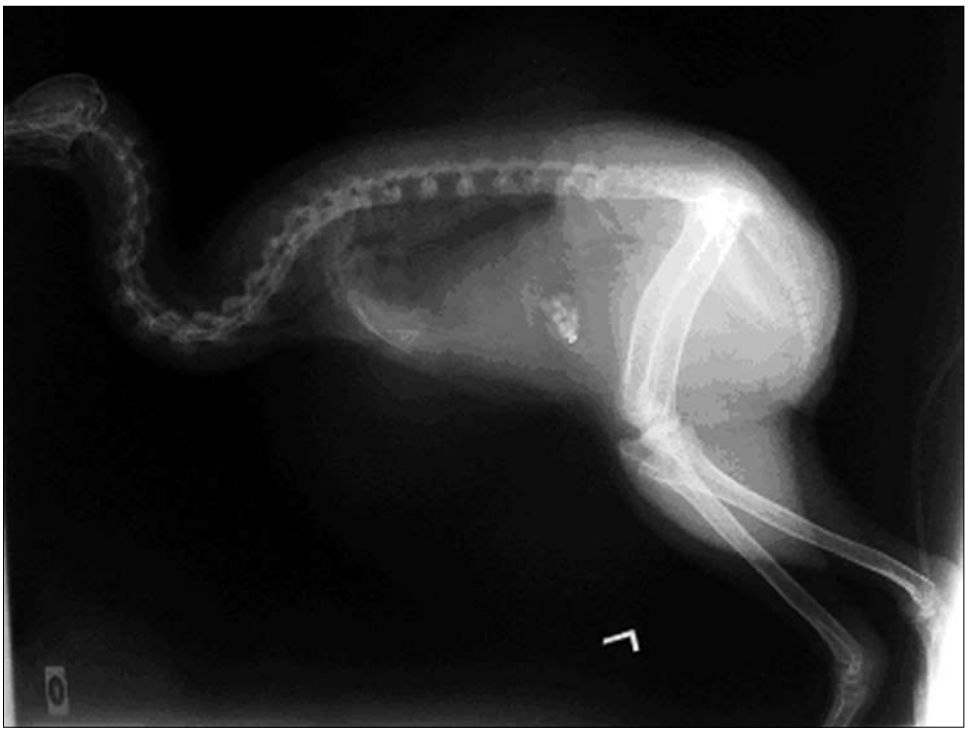


Figure 11. Lateral radiograph of a normal adult North Island brown kiwi. *Photo: B. Gartrell.*

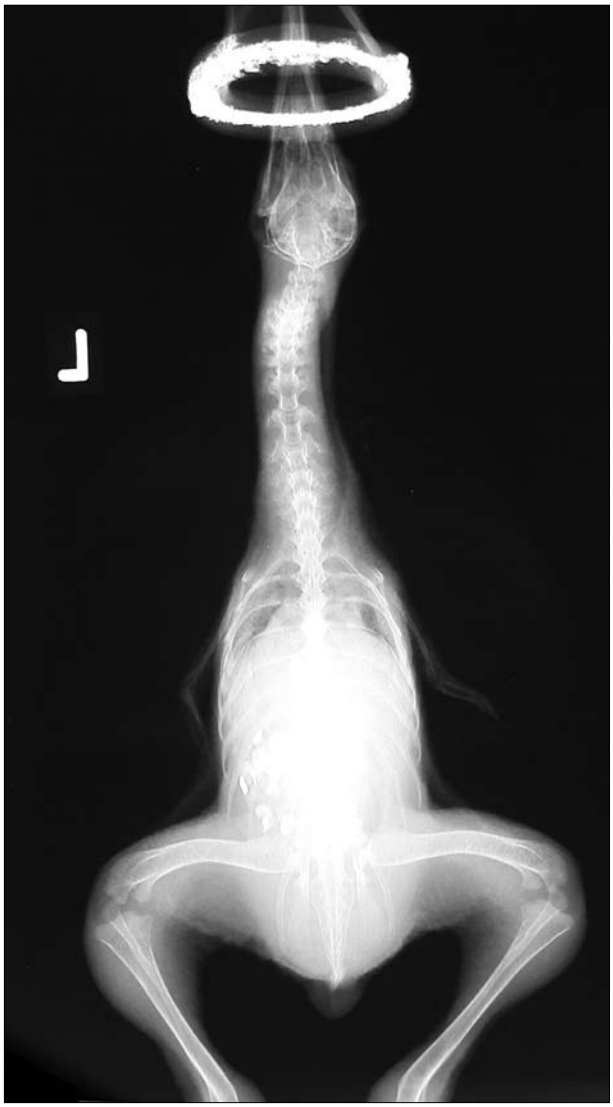


Figure 12. Ventrodorsal radiograph of a normal adult North Island brown kiwi. *Photo: B. Gartrell.*

### *Endoscopy*

Endoscopy has become a routine diagnostic procedure in avian medicine. Rigid 2.7-mm scopes or small flexible scopes are of use in kiwi.

**Tracheoscopy** Endoscopic examination of the tracheal lumen may be indicated in cases of partial tracheal obstruction, such as those caused by *Aspergillus granulomas*, changes in tracheal structure or foreign bodies (Lierz 2006). Endoscopic visualisation helps in lesion diagnosis, with the option of subsequent mechanical debridement of the granuloma using endoscopic instruments. However, the granuloma may be further down the trachea (e.g. the syrinx) than can be reached by the rigid endoscope.

**Gastroscopy** The use of the rigid 2.7-mm or a small flexible endoscope allows the mucosa lining the oesophagus, proventriculus and the gizzard of the kiwi to be examined. This is often indicated as part of a diagnostic work-up for upper gastrointestinal abnormalities, and for endoscopic-guided removal of foreign bodies from the gizzard.

**Coelioscopy** Compared with flighted avian species, the air sac system in kiwi is underdeveloped, making coelioscopic examination more difficult than that described in many avian texts. Only small numbers of kiwi have been examined using coelioscopy and normal statistics have not been developed (B.D. Gartrell, Massey University, pers. comm.).

Coelioscopy is performed using the rigid 2.7-mm endoscope, and allows examination of the serosal surface of internal organs, including the air sacs, lungs, heart, liver, spleen, pancreas, gastrointestinal tract, gonads, and ventral surface of the kidneys and associated ureters. It also allows biopsy of organs using biopsy forceps within a working channel. The biopsy can be submitted for histopathology or microbiology.

**Cloacoscopy** The 2.7-mm rigid scope can also be used for examination of the cloaca and its associated structures, including rectum, ureteral openings and uterus.

### *Computed tomography (CT) and magnetic resonance imaging (MRI)*

Magnetic resonance imaging (MRI) is useful for imaging soft tissue structures, including the brain, spinal cord, coelomic organs and the upper respiratory tract (Helmer 2006). Computed tomography (CT) is best suited for evaluation of bone and air-filled structures (Helmer 2006). Computed tomography has been used to successfully diagnose an acetabular fracture in a kiwi that was not visible on a plain radiograph (Figs 13, 14,& 15) (Gartrell et al. 2006).

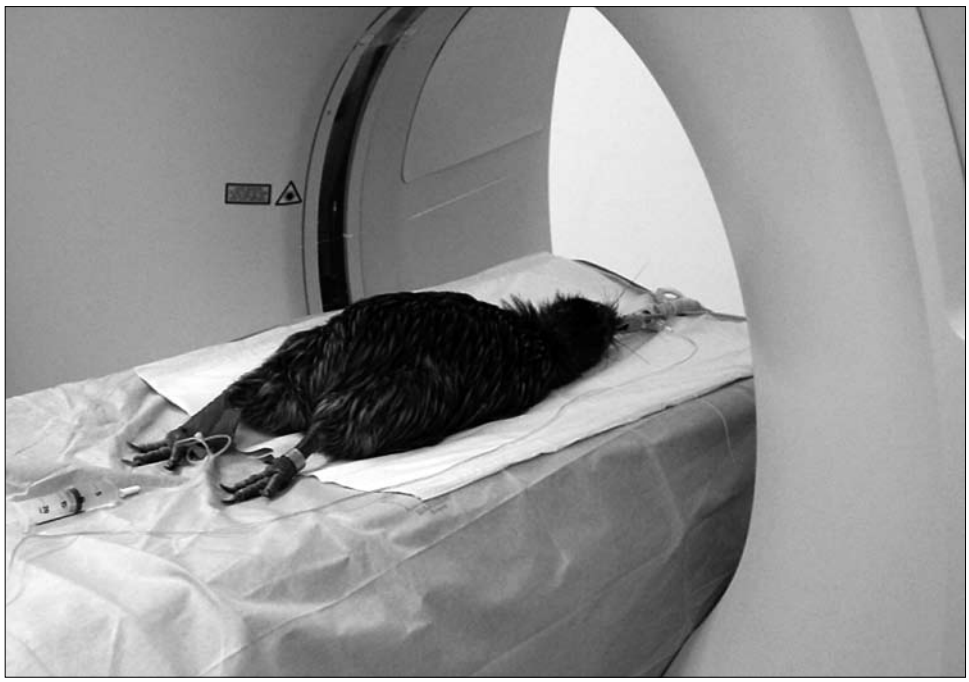


Figure 13. CT scanning of a rowi (Okarito brown kiwi) (to diagnose an acetabular fracture). *Photo: B. Gartrell.*

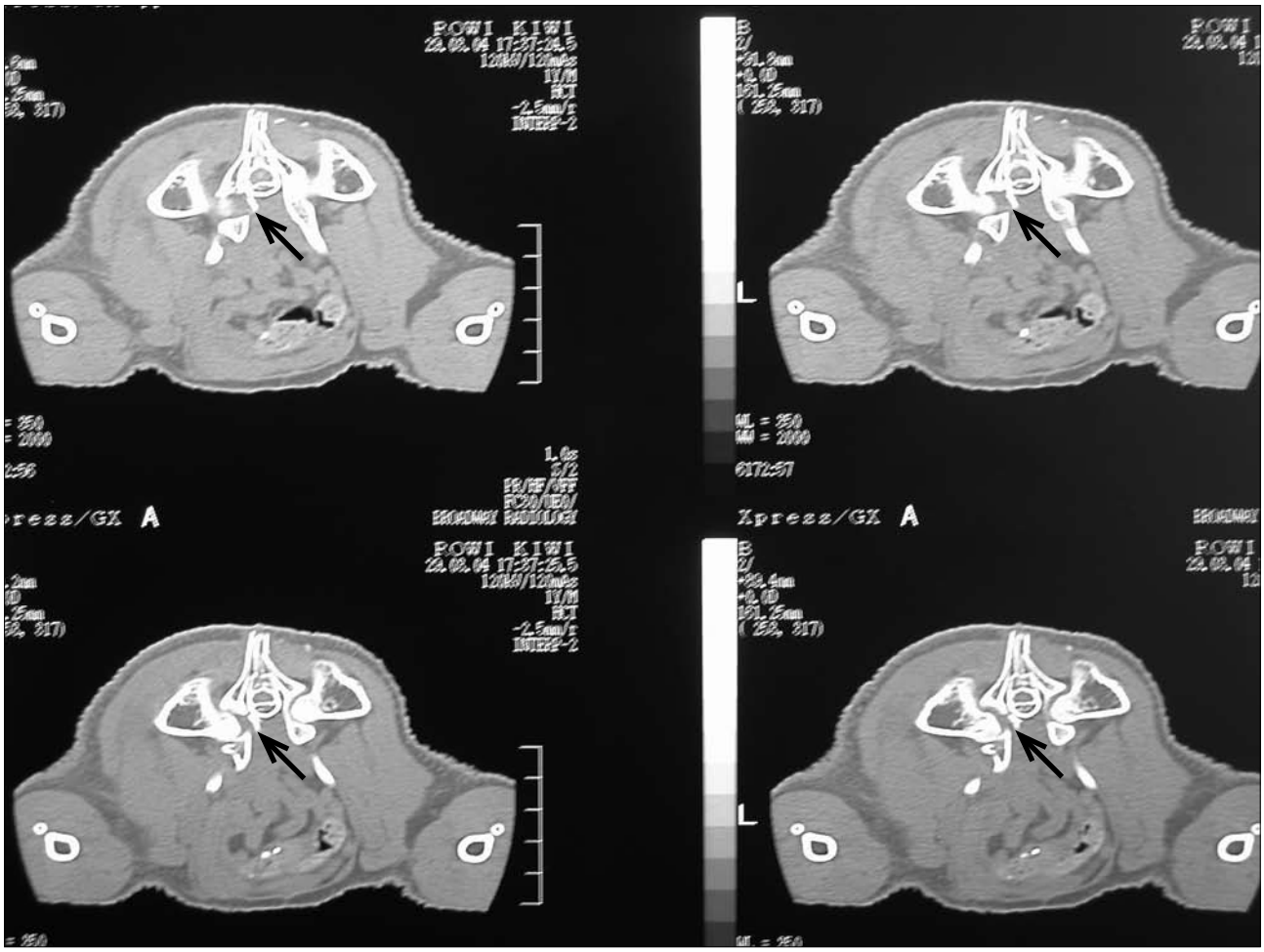



Figure 14. CT images showing fracture to the left acetabulum (arrows). *Photo: B. Gartrell.*

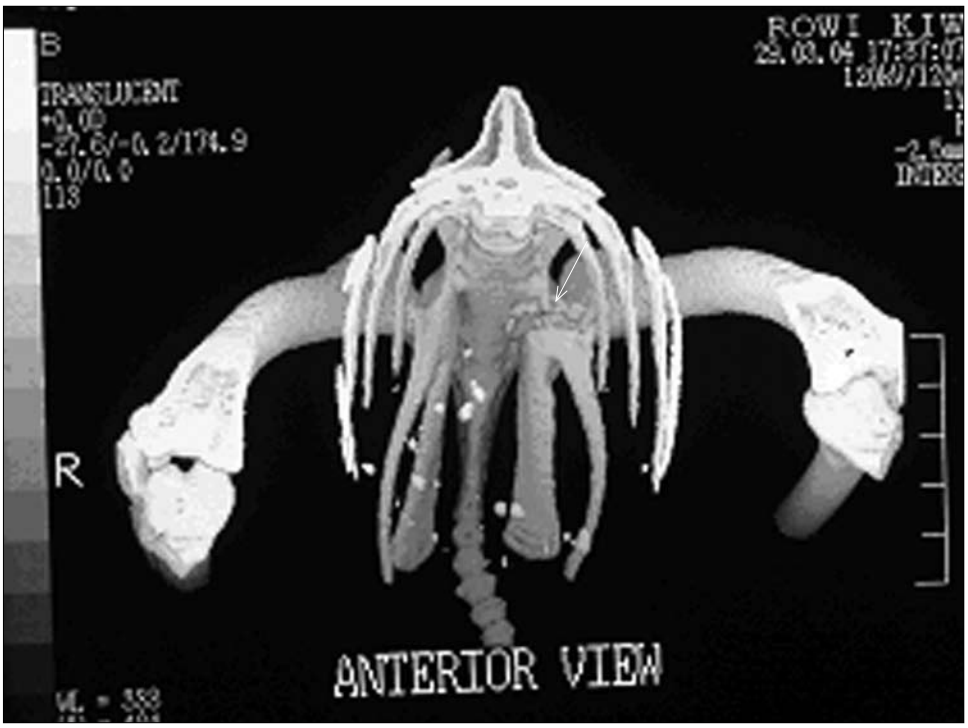



Figure 15. 3D reconstruction of images in Fig. 14 showing a comminuted fracture of the left acetabulum (white arrow). N.B. the floating radiodense objects are stones within the gizzard. *Photo: B. Gartrell.*

# 5. Husbandry

## 5.1 NUTRITION

In the wild, kiwi eat mainly invertebrates, but they will also eat some fallen fruits and, occasionally, leaves. Their main prey are earthworms, beetle larvae, cicadas, moths, spiders, weta, crickets and centipedes. The relative proportion of each dietary component varies from place to place and with season (Heather & Robertson 2000).

Captive birds are generally fed an artificial diet. The captive kiwi diet varies slightly between kiwi houses, but is mainly made up of meat (usually ox heart) and various fruits, vegetables and other ingredients (see Appendix 4). A vitamin and mineral premix supplement designed for kiwi should be added (see Appendix 5). Supplementation with invertebrates is often offered to captive kiwi as a form of environmental enrichment. Feeding solely invertebrates in captivity is difficult to sustain, as an adult kiwi has been estimated to require more than 300 native earthworms a night (S. Bassett, Kiwi Encounter, Rainbow Springs, pers. comm.).

The artificial diet is by no means appropriate, given what kiwi eat in the wild. At the time of writing, alternative captive diets which conform more closely to the natural diet of kiwi were being investigated (C. Minson, Massey University, pers. comm.).

Food requirements and methods of feeding a debilitated kiwi depend on the status of the bird, and whether it is of captive or wild origin. In the hospital situation, it will obviously be easier to initiate self-feeding in a captive kiwi that is used to eating an artificial diet than it will in a wild kiwi that is used to foraging for food. Very sick birds may require assisted feeding in the short term.

### 5.1.1 Intensive care and short-term feeding

The first priority in dealing with an injured kiwi is to address dehydration. A large bird such as a kiwi usually has body reserves to manage for at least 24–48 hours without food.

If the bird is not eating on its own, it may be tube-fed with a formulated diet. Hill's a/d™ may be used for this purpose (see Appendix 5). This needs to be made into a slurry with warm water and gavage-fed using a crop needle or feeding tube in the same way as oral fluids are given. The mixture temperature should be around 38°C (i.e. body temperature). Gavage-feeding food heated to temperatures ≥ 45°C risks oesophageal burns. The limited capacity of the upper gastrointestinal system of the kiwi limits the volume of tube-fed nutritional support that can be given. It is recommended that small amounts of food are initially fed to establish the volume appropriate for the size and status of the kiwi. Adults may be able to ingest up to 20 mL, but chicks may only be able to take 3–5 mL. There is a risk of regurgitation and subsequent aspiration, so care must be taken to deliver food slowly and in small quantities. The limited volumes of nutritional support deliverable

by this method have been found to be insufficient at maintaining weight of kiwi (pers. obs.) This is probably the result of the formula having to be diluted with water to enable it to be administered by tube. Also, the stress of repeated handling for gavage feeding is likely to have a negative effect on the energy balance of the bird.

Kiwi with bill injuries may require that an oesophagostomy feeding tube be installed (Fig. 6). This allows feeding to bypass the oral cavity. However, this method is also unsuitable for maintenance of body weight, and is useful only for short-term nutritional support.

An alternative method of feeding kiwi in intensive care situations is to hand-feed the artificial captive diet. This can be rolled into small balls that are placed in the caudal oral cavity of the kiwi, which stimulates swallowing. An adult kiwi can be fed 100 g or more of this mixture at a time. Alternatively, in the very short term (i.e. no more than a few days), strips of ox heart may be used in a similar way. However, this diet is seriously nutritionally imbalanced. For example, the calcium to phosphorus ratio in ox heart is inappropriate for kiwi, and longer-term feeding of meat alone may lead to nutritional skeletal disorders, including metabolic bone disease.

### 5.1.2 Medium- to long-term care

Supply of a nutritionally balanced diet is imperative for medium- to long-term feeding of kiwi in order to avoid dietary deficiencies and imbalances. Many kiwi, even those of wild origin, will convert to a captive diet within days to weeks. Some remain resistant, and may require prolonged assist-feeding.

Because they are nocturnal, kiwi are inherently more active and inclined to feed for several hours at dusk. Fresh food should be offered in the late afternoon. An adult kiwi may eat 100–200 g of the kiwi artificial captive diet each night. Native earthworms may be offered in soil to encourage probing and some behavioural enrichment for the bird. Other commercially available insects that can be offered include mealworms and wax-moth larvae. Tiger worms (compost worms) appear to be unpalatable to kiwi.

A dish of water should be made available at all times.

## 5.2 HOUSING

Wild birds need to be appropriately housed to provide the most stress-free environment possible. They must be housed away from cats and dogs and other potential ‘predators’. Examples of avoidable stressors include human traffic, loud noise (radios, television, people talking) and excessive handling. Birds should be placed in a quiet, dark, draught-free and warm area, and they should be handled as infrequently as possible.

Housing requirements vary depending upon the situation and status of the bird.

### 5.2.1 Intensive care

Very sick and debilitated birds, and those recovering from general anaesthesia, should be housed in an intensive care situation. Ideally, this would be in an intensive care unit or paediatric incubator with temperature and humidity control. If this is not available, small cages with external heat sources may be used (see section 2.2.4). In many cases (e.g. trauma), it may be necessary to minimise bird movement by providing a small enclosure. In small hospital cages, a towel may be placed over the front of the cage to reduce visual stimulation and provide darkness. Similarly, incubators and intensive care units should be covered.

### 5.2.2 Non-critical hospitalisation

Once the bird is stablised and no longer requiring critical care, it may be placed in a larger container. This may be a specially-designed hospital cage, or simply a large cardboard box (e.g. from a refrigerator or television). These work well, as they can be used for one patient and then thrown out, and can easily be moved to quiet, warm places, away from other animals and sources of negative stimulation. The birds should be provided with a burrow, which may be made from a smaller cardboard or plastic box with an entry hole cut in the front, and turned upside down. Cardboard boxes used for burrows can be discarded and plastic tubs can be cleaned with a disinfectant (e.g. Virkon™, Trigene™) before use by other patients.

A shallow tray of water should also be provided—cat litter trays are ideal for this. Food should be provided in bowls in the enclosure. Generally, kiwi are not active during the day, and will not venture out of their burrow. In the wild, they become active at dusk for several hours for feeding, so it is important to provide fresh food in the late afternoon in preparation for this. Providing live earthworms in a dish of soil and fresh, clean, leaf-litter (with invertebrates) once or twice weekly provides an additional source of nutrition as well as environmental enrichment for the birds.

### 5.2.3 Longer-term care

It is not the intention of this publication to provide information on appropriate housing for longer-term care. It is advisable to refer to the The Kiwi Best Practice manual: www.savethekiwi.org.nz/InformationToolkit/Kiwi+Best+Practice (viewed January 2008) for recommendations on husbandry.

## 5.3 TRANSPORTATION OF KIWI

Ideally, kiwi should be transported in transport boxes specially designed for this species. Considerations when choosing an appropriate transport box include the provision of adequate ventilation, and room sufficient to allow the bird to move to reposition itself, but not to move excessively, which might worsen injuries.

Care must be taken to ensure that the bird's long bill cannot protrude from the box where it would become susceptible to trauma when the box is moved. If the box is hinged at the top, it is important to make sure that the bird cannot get its bill caught in the hinged lid as the box is closed.

Adequate ventilation must be provided via air vents—these can be covered with a fine mesh (e.g. shade cloth) to prevent protrusion of the bill. The box should be well padded with a substrate suitable for absorbing excrement. Clean towels are an ideal covering for the box substrate.

Overheating should be prevented if there is adequate ventilation and the box is not placed in direct sunlight. Small chicks should never be transported in plastic containers, as this risks overheating.

Kiwi tolerate air travel if they are stabilised prior to the flight, and this method of transporting kiwi has been carried out successfully on many occasions. It is important to ensure that the bird to be transported is rehydrated and appropriately treated first. Usually, it is recommended that debilitated birds be given a stabilisation period of at least 24 hours before transportation. Contact Air New Zealand cargo for information on air transportation of live kiwi.

Kiwi should never be transported with their legs taped together (see section 2.1.3).

Refer to the The Kiwi Best Practice manual: www.savethekiwi.org.nz/InformationToolkit/Kiwi+Best+Practice (viewed January 2008) for further information.

# 6. Traumatic injuries

## 6.1 TRAP INJURIES

Trap injuries are the cause of one of the most common presentations of injured kiwi. Leg or bill entrapment can occur when a kiwi inadvertently steps on a trap intended for possums. Traps are supposed to be placed up off the forest floor, making them inaccessible to ground-dwelling birds, but some traps are still placed on the ground.

Often, the birds are found more than 24 hours after they have become trapped in the device. Leg entrapment often involves crushing injuries to the lower tarsometatarsus or foot, resulting in contaminated, open, comminuted fractures with severe neurological and vascular compromise. As the bird moves about attempting to free itself from the trap, the nerves and blood vessels supplying the fracture site and distal limb become further damaged, and the birds often have complete loss of neurological and vascular function. Often, these birds have severe blood loss and are septicaemic as a result of heavy wound contamination.

Bill entrapment may also occur, and these injuries usually have a grave prognosis (see section 6.2).

Treatment principles for leg trap injuries include patient stabilisation as previously described. Immediate consideration must be given to aggressive fluid and antimicrobial therapy, analgesia, and appropriate wound management.

If there is no remaining vascular or neurological function to the entire distal limb, amputation or euthanasia may be the only treatment options. Euthanasia is recommended for birds with crushing injuries above the hock, with a lack of blood and/or nervous supply.

Distal limb amputee birds have only recently been released back to the wild and survival of two birds for over a year suggests a positive outcome. However, breeding has not been observed so far, and careful consideration of the long-term future of such birds must be made in consultation with the Kiwi Recovery Group and DOC (see section 1.1) before surgery is undertaken. Limb amputee kiwi have yet to be proven as breeders in captivity (B.D. Gartrell, Massey University, pers. comm.).

Toe amputations may be performed, and these birds have been successfully returned to the wild. A female kiwi with an entire digit amputation was released back into the wild. Monitoring over the following 6-month period showed that she was able to move successfully over long distances, and examination on subsequent capture for transmitter removal showed her to be in good body condition (D. King, DOC, pers. comm.).

Other methods of fixation for distal limb fractures with intact nervous and vascular supply include the placement of a modified external skeletal fixator (see Fig. 16). Complications associated with this procedure include fracture malunion, tenosynovitis and osteomyelitis.

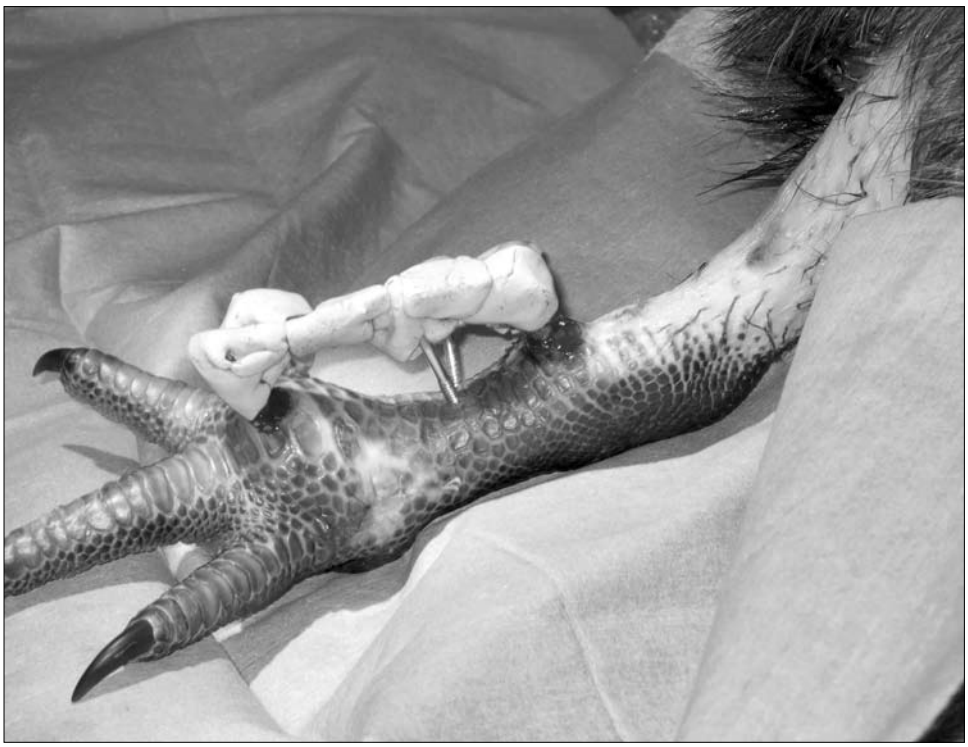


Figure 16. Modified external fixation of an open, comminuted tarsometatarsal fracture as a result of a trapping injury. *Photo: J. Youl.*

## 6.2 BILL INJURIES

Bill injuries may have a variety of causes including traps (as described above), transportation and dog attacks. Bill tip injuries generally have a grave prognosis, especially if the upper bill is injured. Just how important the bill tip is to kiwi survival is becoming clear. Recent research has demonstrated that kiwi detect their buried prey using vibration- and pressure-sensitive mechanoreceptors embedded in pits in the bill tip. It is believed that these specialised structures function as a detector of movement of invertebrates during feeding (Cunningham et al. 2007). Olfaction is thought to play a minor role in prey detection but an important role in social and territorial behaviour. This finding correlates with clinical observations of a female great spotted kiwi which had an upper bill tip amputation after a transport box injury. This bird failed to feed on her own after more than 24 months in captivity (Fig. 17) (pers. obs). Euthanasia should be seriously considered in any bird with a bill tip amputation.

An injury to the proximal upper bill, or to the lower bill, may have a slightly more favourable prognosis than an upper bill tip injury. A sub-adult female North Island brown kiwi injured by a muzzled dog was presented to the New Zealand Wildlife Health Centre. It had multiple fractures of the proximal mandible. The mandible was repaired using a modified external skeletal fixator (Figs 18, 19). The growth plates of the mandible were affected. As the kiwi continued to grow, it developed an undershot lower bill, which prevented it from probing naturally for food. However, the bird is able to eat the captive diet well, and has now been included in the captive breeding programme.

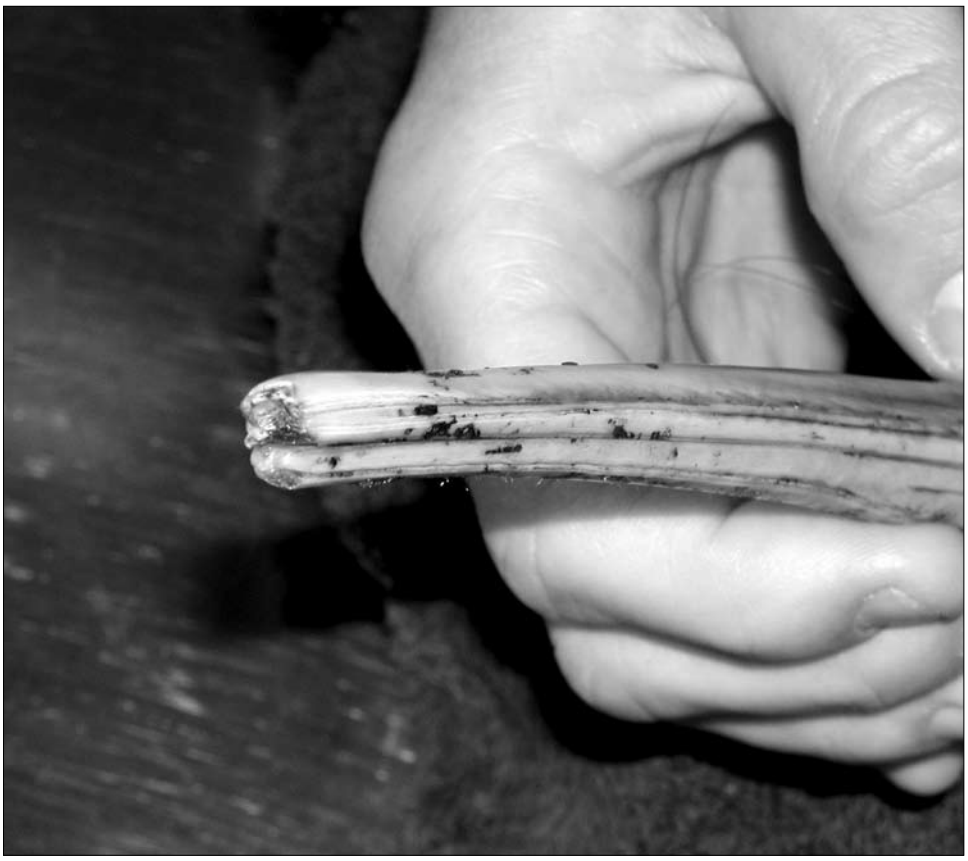


Figure 17. Fractured tip of the bill of a female great spotted kiwi. The distal 10 mm of the bill was broken off during a transportation injury.
*Photo: B Gartrell.*

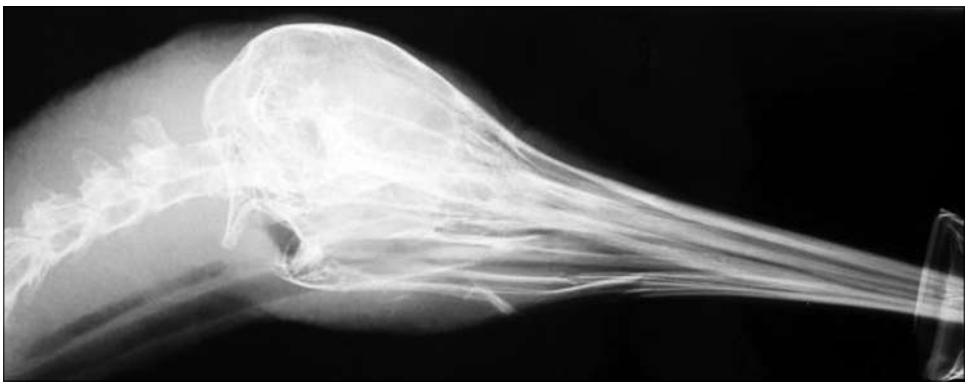


Figure 18. Lateral radiograph of a North Island brown kiwi with multiple mandibular fractures.
*Photo: K. Morgan.*

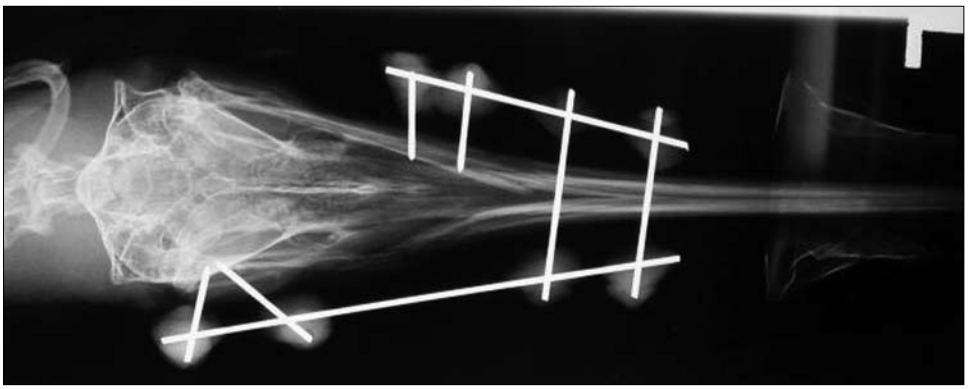


Figure 19. Ventrodorsal radiograph of kiwi in Fig. 18 after placement of a modified external skeletal fixator in the mandible.
*Photo: K. Morgan.*

## 6.3 MOTOR VEHICLE ACCIDENTS

Motor vehicle accidents are another common cause of injuries to kiwi, and birds may present with limb fractures, bill injuries, pelvic and spinal fractures, and internal injuries.

Again, treatment relies upon stabilisation of the patient, as previously described. Once the bird's condition has stabilised, radiographs can be carried out. These may reveal limb fractures and other internal injuries. Pelvic and spinal fractures are more difficult to diagnose, and often require ancillary diagnostics such as CT scans (see section 4.3.2). Myelography (an X-ray examination using contrast material to highlight the spinal cord) has been described in the avian patient (Harr et al. 1997; Naeini et al. 2006). However, it has limited use in clinical situations, because of lack of familiarity with the technique, fear of iatrogenic injury to the spinal cord, and concern about potentially fatal complications with the procedure (Harr et al. 1997; Naeini et al. 2006). In birds, the fused lumbar and sacral vertebrae and pelvis (the synsacrum) allows accessibility to the atlanto-occipital space only (Harr et al. 1997; Naeini et al. 2006). In many species, the atlanto-occipital space overlies a large venous plexus (Harr et al. 1997) and the myelography procedure may be fatal. MRI should be strongly considered as an alternative to myelography, if feasible (Helmer 2006).

Due to the anatomic location of the lungs and kidneys, birds with pelvic and spinal fractures will often have associated renal and pulmonary contusions. The kidneys lie in a fossa within the fused synsacrum, making them susceptible to damage during trauma to the overlying structures.

# 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.).

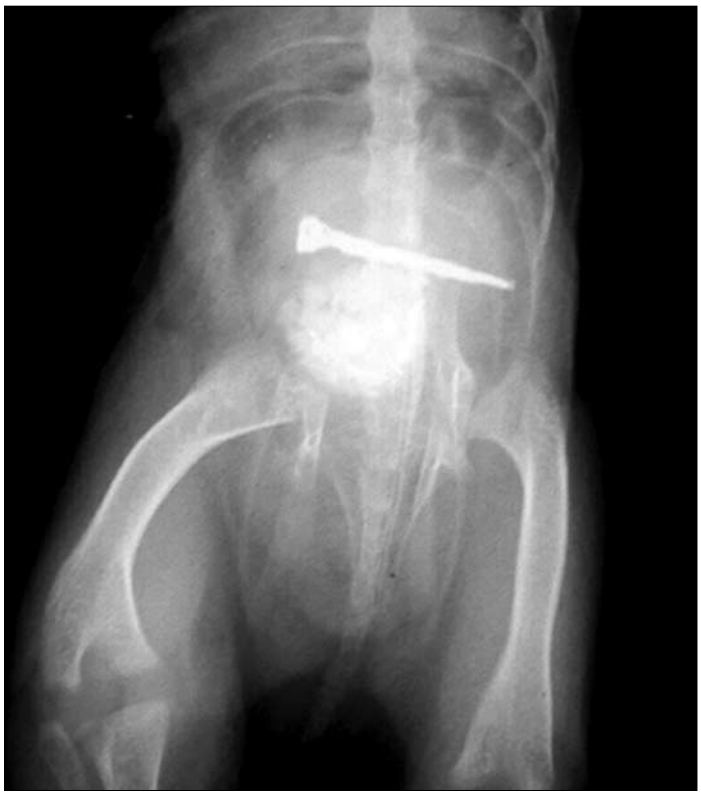


Figure 20. Ventrodorsal radiograph of an adult North Island brown kiwi with a penetrating metallic gastric foreign body. *Photo: T. Kelly.*

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.

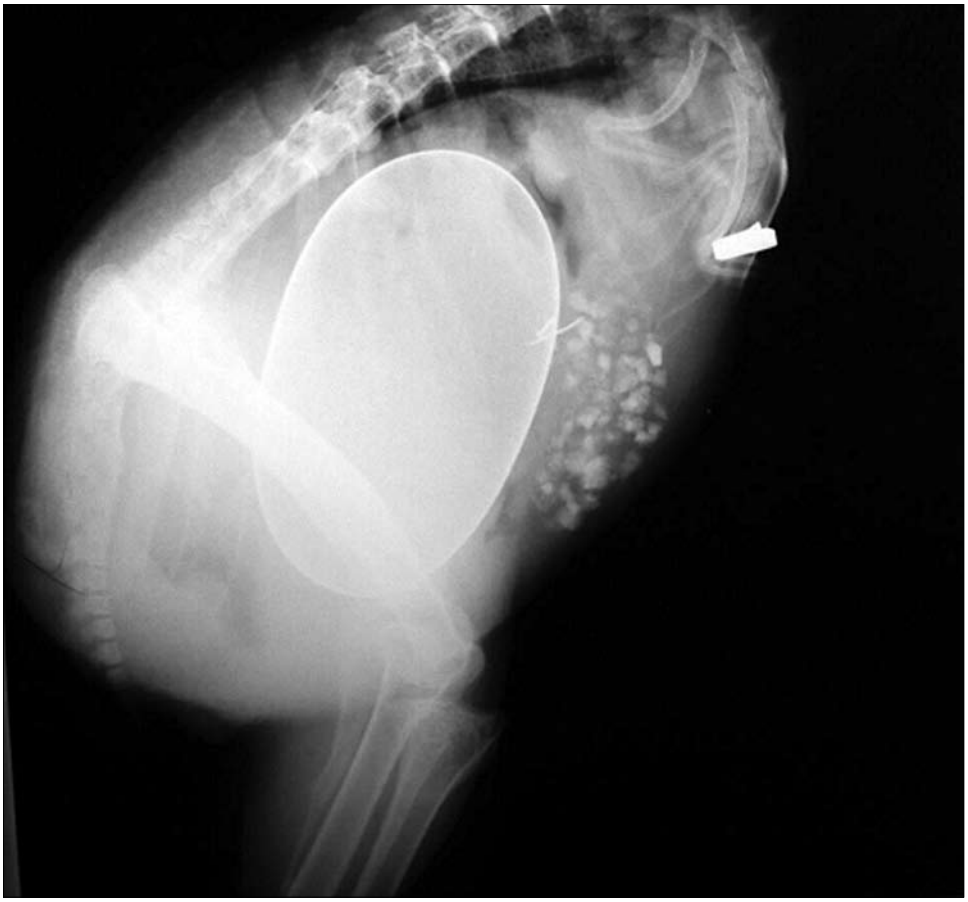


Figure 21. Lateral radiograph of a gravid North Island brown kiwi. *Photo: T. Kelly.*

### 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).

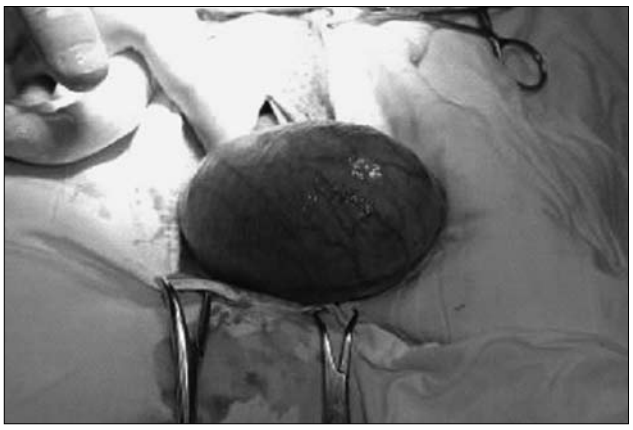


Figure 22. Intra-operative image of kiwi egg within the oviduct during a hysterotomy.
*Photo: T. Kelly.*

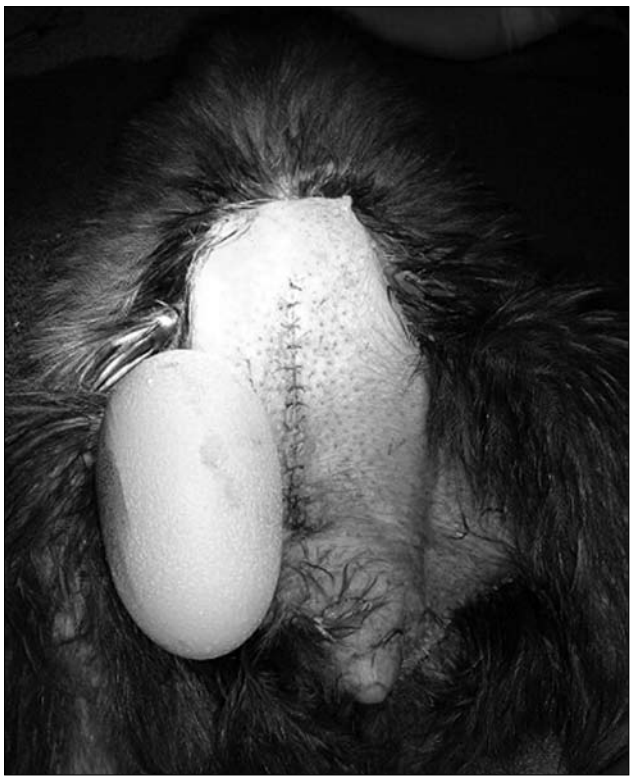


Figure 23. Post-operative image of the bird and egg from Fig. 22.
*Photo: T. Kelly.*

### 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).

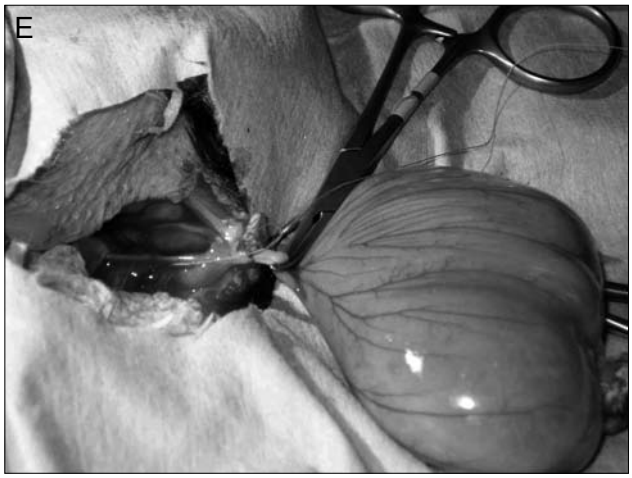
Pre-anaesthetic considerations should include patient stabilisation with fluid therapy, antibiotics, and correction of hypothermia and hypoglycaemia. Pre-surgical analgesia should be considered. Anaesthetic considerations should


A. Kiwi chick prepared for yolk sac surgery.

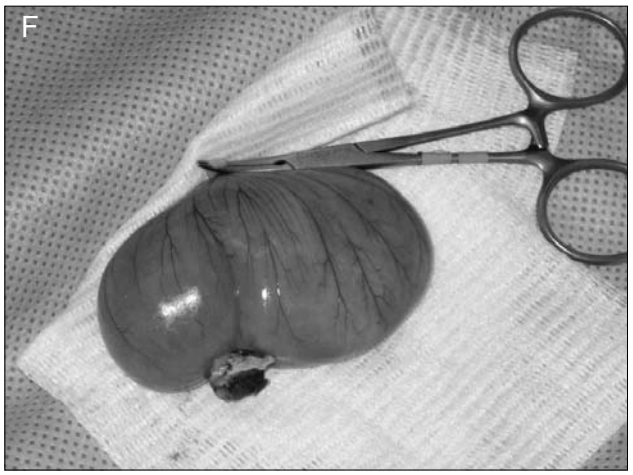

B. The skin incision is made adjacent to the umbilicus.


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.*

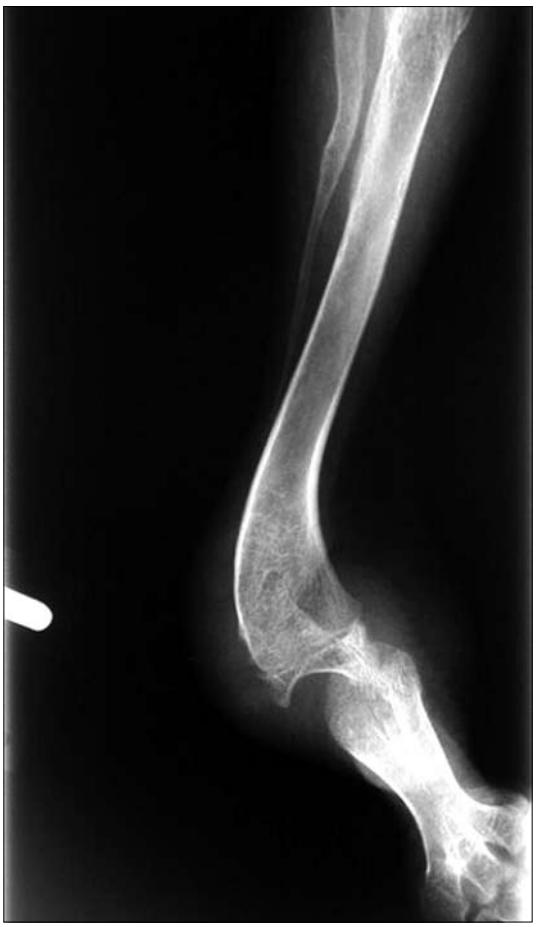


Figure 27. Hindlimb radiograph from the chick in Fig. 26, showing rotation of the tibiotarsus. *Photo: B.D. Gartrell.*

## 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]<br>*Apteryx mantelli* (North I. brown kiwi)<br>*Apteryx haasti* (great spotted kiwi)<br>*Apteryx australis australis* (South I. brown kiwi)<br>*Apteryx australis lawryi* (Stewart I. brown kiwi)<br>*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)<br>*Apteryx haasti* (great spotted kiwi)<br>*Apteryx australis lawryi* (Stewart I. brown kiwi)<br>*Apteryx owenii* (little spotted kiwi) |
| *Kiwilichus cryptosikyus* (Gaud & Atyeo 1970)[b] | Feather mite | *Apteryx haasti* (great spotted kiwi)<br>*Apteryx australis australis* (South I. brown kiwi)<br>*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)<br>*Apteryx australis lawryi* (Stewart I. brown kiwi)<br>*Apteryx owenii* (little spotted kiwi)<br>*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)<br>*Apteryx australis lawryi* (Stewart I. brown kiwi)<br>*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)<br>*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.

# 9. Acknowledgements

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# Appendix 1

## KIWI FIRST AID FIELD KIT

A first aid kit useful for birds can be constructed from a modified human first aid kit. The following table lists suggested essential items:

| ITEM | QUANTITY (MINIMUM) |
|---|---|
| Sterile saline solution for injection (0.9% NaCl) vials (10 mL, 5 mL) | 50–100 mL |
| **Or** lactated Ringer's solution (LRS) or saline (0.9% NaCl) flexibag (For wound lavage, oral or subcutaneous fluids) | 500 mL |
| Melolin™ non-adherent dressings | 3 |
| Sterile gauze swabs (pkt of 5) | 2 |
| Vetrap™—5 cm width | 2 |
| Sofban™—5 cm width | 2 |
| Syringes—5 mL | 3 |
| Syringes—20 mL | 1 |
| Needles—25 gauge, 22 gauge | 6 |
| Snap-type heat pack | 2 |
| Scissors | 1 |
| Crop tubes (silicone) | 1 |

Veterinarians undertaking field work could also consider taking:

- Analgesia (Butorphanol)
- Antibiotics (e.g. 2.5% injectable enrofloxacin (Baytril™), Clavulox™ palatable drops)
- Equipment for an IV fluid set up

# Appendix 2

## VETERINARY ADVICE AND REFERRAL CENTRES

The following contacts are available for advice when dealing with injured or sick kiwi, and may include the option of referral for further diagnostics and treatment.

### New Zealand Wildlife Health Centre (NZWHC)

Veterinary Teaching Hospital
Institute of Veterinary, Animal and Biomedical Sciences
Massey University
Palmerston North
Phone: (06) 350 5329 (Weekdays)
(06) 350 5955 (Weekends)

#### Veterinarians

Dr Brett Gartrell, BVSc, MACVSc (Avian Health), PhD
Senior Lecturer in Avian and Wildlife Health, Director NZWHC
Email: B.Gartrell@massey.ac.nz

Kerri Morgan, BVSc, MACVSc (Avian Health), PGDipVCS (dist.)
Lecturer in Avian and Wildlife Health
Email: K.J.Morgan@massey.ac.nz

Facilities available through the NZWHC include radiography, CT and MRI, endoscopy, anaesthesia and a full surgical suite. The wildlife clinic at the NZWHC is geared towards treating threatened and endangered species on a national scale, as well as local common native species. The NZWHC is currently contracted to the Department of Conservation to provide the diagnostic pathology service for endangered and threatened species (see section 8).

Kiwi from throughout New Zealand may be referred to the NZWHC for treatment. Sponsorship covers costs of treatment of endangered species.

Contact the NZWHC at the Massey University Veterinary Teaching hospital (phone numbers above) for advice and to arrange transportation of kiwi to the wildlife clinic.

### New Zealand Centre for Conservation Medicine (NZCCM)

Auckland Zoo
Motions Road
Western Springs
Auckland

Phone: (09) 360 3814 or (09) 360 4704 (Weekdays)
(09) 360 3800 (Weekends)

#### Veterinarians

Richard Jakob-Hoff, BVMS (Hons)
Senior Veterinarian

Email: Richard.jakob-hoff@aucklandcity.govt.nz

John Potter, BVSc
Associate Veterinarian
Email: John.potter@aucklandcity.govt.nz

Consultant Veterinarians:
Berend Westera, BVSc
Maureen Forsyth, DVM, MACVSc

The Auckland Zoo is an active participant in the Operation Nest Egg Programme, breeds North Island brown kiwi on site and has a progressive public education and advocacy programme for kiwi.

Veterinary facilities at the NZCCM at Auckland Zoo include a fully equipped wildlife hospital and quarantine facility. Partnerships with local veterinary and human medical specialists enable NZCCM to utilise the most up-to-date diagnostic and treatment options.

Staff veterinarians at NZCCM provide clinical, diagnostic, pathological and research services for both captive and free-living wildlife. Kiwi are a major species of interest. Technical advice is provided through the Department of Conservation's Wildlife Health Co-ordinator.

## Other avian veterinarians

The Wildlife Society of the New Zealand Veterinary Association has a database listing veterinarians with avian and wildlife experience. This database may be accessed by contacting the New Zealand Wildlife Health Centre.

# Appendix 3

## KIWI HAEMATOLOGY AND BIOCHEMISTRY REFERENCE RANGES

| HAEMATOLOGICAL/ BIOCHEMICAL VALUES | NORTH ISLAND BROWN KIWI (*Apteryx mantelli*)[a] | OKARITO BROWN KIWI (*Apteryx rowi*)[b] | *n* | GREAT SPOTTED KIWI (*Apteryx haasti*)[c] | *n* |
|---|---|---|---|---|---|
| PCV (%) | 46 (38–54) | 40.1 (37.1–43.1) | 9 | 39.4 (35.3–43.5) | 25 |
| Hb (g/L) | | | | | |
| MCHC (g/L) | 250 (110–333) | | | | |
| WBC ($\times 10^9$/L) | 11.6 (8.7–14.5) | | | 15.7 (7.9–23.5) | 29 |
| Heterophils ($\times 10^9$/L) | 6.0 (4.0–8.2) | | | 8.7 (4.3–13.1) | 29 |
| Heterophils (%) | | | | 55.0 (45.0–65.0) | 29 |
| Lymphocytes ($\times 10^9$/L) | 4.2 (2.5–5.9) | | | 4.8 (1.9–7.7) | 29 |
| Lymphocytes (%) | | | | 32.0 (22.0–42.0) | 29 |
| Eosinophils ($\times 10^9$/L) | 0.18 (0.7–1.29) | | | 0.8 (0.0–1.9) | 28 |
| Eosinophils (%) | | | | 4.0 (0.0–8.0) | 28 |
| Monocytes ($\times 10^9$/L) | 0.3 (0.1–0.5) | | | 0.5 (0.0–1.1) | 28 |
| Monocytes (%) | | | | 2.0 (0.0–5.0) | 28 |
| Basophils ($\times 10^9$/L) | 0.56 (0.09–1.3) | | | 0.9 (0.4–1.4) | 29 |
| Basophils (%) | | | | 6.0 (3.0–9.0) | |
| CK (IU/L) | 521–971 | 758.8 (446.5–1071.1) | 9 | | |
| AST (IU/L) | 64–138 | 204.2 (132.4–276.1) | 9 | | |
| Bile acids (µmol/L) | | <35 | 9 | | |
| Serum protein (g/L) | 54–62 | 52.1 (47.0–57.2) | 9 | 46 (39–53) | 25 |
| Uric acid (µmol/L) | 300–380 | 476.6 (343.4–609.7) | 9 | | |
| Ca (mmol/L) | 1.85–3.1 | 2.49 (2.45–2.54) | 9 | | |
| Glucose (mmol/L) | 3.0–3.9 | | | | |
| LDH (IU/L) | 2380 | | | | |
| Phosphorus (mmol/L) | | 2.02 (1.80–2.23) | 9 | | |
| Sodium (mmol/L) | | 147.1 (145.2–149.0) | 9 | | |

[a] Doneley 2006.

[b] Unpubl. data 2007.

[c] Robertson 2006.

# Appendix 4

## CAPTIVE DIET FORMULATION

(Courtesy of Kiwi Encounter, Rainbow Springs, Rotorua)

3 kg minced ox heart (N.B. remove fat first)

2 cups rolled oats cooked with 4 cups water, allow to cool

1 cup wheat germ

2 cups cat biscuits (premium quality), soaked and mashed

2 mashed bananas

2 pieces grated fruit (e.g. kiwifruit, nectarine, peach, pear)

250 g grated veges (e.g. corn, peas, beans, silverbeet, broccoli)

Mix together. Immediately prior to feeding, add 1 g kiwi pre-mix (see Appendix 5) per 100 g of food. Food should be presented in bowls on the ground.

## FOOD PRESENTATION FOR LONG-TERM CARE

(Courtesy of Auckland Zoo)

Food is presented in flat stainless steel trays on the ground in the late afternoon for birds held outdoors. Food presented in tubes placed into the ground in the morning for birds held in nocturnal exhibits. Tubes used to encourage probing action when feeding and allow for the feeding sites to be moved easily for enrichment. Earthworms are provided daily as available scattered throughout enclosure for the birds housed in nocturnal exhibits. Rotten logs should be provided as often as possible. Birds should have access to small pebbles (2–3 mm).

# Appendix 5

## AVAILABILITY OF DIETARY PRODUCTS MENTIONED IN THE TEXT

Kiwi Vitamin and mineral Premix
Contact Tony Billing
Westshore Wildlide Reserve, Napier
Phone (06) 834 4136
Email tonyb@napier.govt.nz

Hills a/d™
Available in cans from most veterinary clinics

Wombaroo Insectivore Mix
Contact: Karen Wiley
Native Bird Rescue Trust, Wellington
Phone/fax (04) 479 2936
Email nativebirdrescue@actrix.co.nz

# Appendix 6

## AVIAN THERAPEUTICS FORMULARY

Unfortunately, there are relatively few pharmacokinetic studies of medications for use in avian species, and most dosages used in avian medicine are based on empirical data, observations and experience. Dosages given here are extrapolated from published data applicable to avian species other than kiwi.

### Therapeutic contraindications in kiwi

| | |
|---|---|
| Levamisole | Can cause death. Kiwi appear to be acutely 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[a]. |
| Organophosphates | Birds are extremely sensitive to organophosphates, and these products should not be used on birds as they may cause death[b]. |
| Corticosteroids | Corticosteroids may be contraindicated for use in birds (B.D. Gartrell, Massey University, pers. comm.) (see section 2.2.9). |

### Antibiotics

| AGENT | COMMENTS | PREPARATION AND PRODUCT EXAMPLES | SUGGESTED DOSAGES |
|---|---|---|---|
| Amikacin sulfate | Least nephrotoxic of the aminoglysosides; active against gram-negative bacteria including *Pseudomonas* spp., and gram-positive bacteria including *Staphylococcus* spp. and *Streptococcus* spp. Maintain hydration during use[c]. Pain on injection; causes myositis in ostriches[c]. | Injectable (Amikin™). | 10–15 mg/kg IV, IM, SQ bid-tid (most species)[c,d]; Nebulisation: 5–6 mg/mL in sterile water or saline, × 15 min bid-tid[c]. |
| Amoxicillin | Broad spectrum bactericidal penicillin antibiotic, minimal activity for common gram-negative infections of birds[c]. | Oral suspension (Amoxil™ paediatric drops, Ranbaxy-Amoxi™); injectable (Moxylan™, Betamox LA™). | 100–150 mg/kg PO, IV, IM bid-tid (most species)[c,d]. 15–22 mg/kg PO tid (ratites)[c]. |
| Amoxycillin/clavulanate | β-Lactamase inhibitor—use with allopurinol is contraindicated[c]. Intramuscular injections can be very irritant. | Oral suspension (Clavulox™ palatable drops); tablets (Clavulox™, Noroclav™); injectable (Clavulox™); IV powder (Augmentin™). | 70 mg/kg PO, IV, IM bid (kiwi, pers. obs.). 10–15 mg/kg PO bid (ratites)[c]. 125–150 mg/kg PO bid (most species)[c]. |
| Cefotaxime | Third-generation cephalosporin with broad-spectrum activity for many gram-positive and gram-negative pathogens, penetrates cerebrospinal fluid[c]. | IV powder (Cefotaxime sodium). | 75–100 mg/kg IM, IV bid-tid (most species)[c]. 25 mg/kg IM tid (ratites)[c]. Nebulisation: 10 mg/ml saline × 10–30 min bid-qid[c]. |

*Antiobiotics continued on next page*

*Antiobiotics continued from previous page*

| AGENT | COMMENTS | PREPARATION AND PRODUCT EXAMPLES | SUGGESTED DOSAGES |
|---|---|---|---|
| Cephalexin | First-generation cephalosporin; active against many gram-positive and gram-negative bacteria, including *E. coli* and *Proteus* spp., but not *Pseudomonas* spp; useful for *Staphylococcus* spp. dermatitis[c]. | Oral suspension; tablets (Keflex™); Injectable (Ceporex™). | 40–100 mg/kg PO, IM tid-qid (most species)[c]. 15–22 mg/kg PO tid (ratites)[c]. |
| Ciprofloxacin | Broad-spectrum quinolone[c]. | Oral suspension, tablets (Ciproxin™). | 15–40 mg/kg PO bid (most species)[c]. 10 mg/kg PO bid (ostrich chicks)[c]. 3–6 mg/kg PO bid (ratites)[c]. |
| Clindamycin | Lincosamide: indicated for bone, joint and tendon sheath infections; may be used for up to 12 weeks without ill effects; monitor kidneys and liver with long-term use as well as for yeast overgrowth. | Oral suspension, capsules (Antirobe™). | 50–150 mg/kg PO sid-tid[c]. |
| Doxycycline | Drug of choice for *Chlamydophilia* spp. and *Mycoplasma* spp.[c] (N.B. neither of these have previously been identified in kiwi). | Oral paste, tablets (Vibravet™). | 25–50 mg/kg PO sid-bid (most species)[c]. 2.0–3.5 mg/kg PO bid (ratites)[c]. |
| Enrofloxacin | Given orally, the IM formulation (2.5%) produces therapeutic plasma concentration. IM formulation is extremely painful and should not be given repeatedly. In general, avoid IV use in birds.[c] | Tablet, injectable (Baytril™). | 10–15 mg/kg IM, PO sid-bid. (most species)[c]. 5 mg/kg IM bid (ratites)[c]. Nebulisation: 10 mg/ml saline[c]. |
| Gentamicin | Aminogylcoside—not generally recommended, narrow margin of safety, nephrotoxic [c]. Bird should be well hydrated, avoid doses higher than 2.5–5.0 mg/kg q8-12h [c]. | Injection (Gentamax 100™, Genta 50™). | 3–5 mg/kg IM sid-bid (most species, including ostriches and emus)[c]. 1–2 mg/kg IM tid (ratites, use only as last resort)[c]. Nebulisation: 5 mg/mL saline × 15 min tid[c]. |
| Metronidazole | Active against most anaerobes, also antiprotozoal[c]. | Tablet (Stomorgyl™); oral suspension (Flagyl™). | 10–50 mg/kg PO bid (most species)[c]. |
| Trimethoprim/ Sulfamethoxazole | Broad spectrum, contraindicated with dehydration, liver disease or bone marrow suppression. Side effects include GI upset, regurgitation. Resistance to *Pseudomonas* spp. common[c]. | Oral suspension; tablets (Trisul™). | 20–100 mg/kg PO bid (most species)[c]. 21 mg/kg PO bid (ostriches)[c]. |

## Antifungals

| AGENT | COMMENTS | PREPARATION AND PRODUCT EXAMPLES | SUGGESTED DOSAGES |
|---|---|---|---|
| Amphotericin B | Fungicidal, preferred IV agent for aspergillosis. IT administration for syringeal aspergilloma may cause tracheitis. Potentially nephrotoxic, maintain good patient hydration. Resistance may develop[c]. | IV powder (Fungizone™); oral lozenges (Fungilin™). | 1.5 mg/kg IV tid 3–7 days (most species)[c]. 1 mg/kg IT bid-tid (syringeal aspergilloma, raptors)[c]. Nebulisation: 1 mg/ml sterile water or saline, 15 min bid (most species)[c]. 100–200 mg/kg PO tid-qid[d] (for GI candidiasis, avian gastric yeast). |
| Fluconazole | Fungistatic. Penetrates well into brain, CSF and eyes. Only indicated if topical treatment (e.g. nystatin) not feasible. Effective against *Candida* spp., but may be ineffective against aspergillosis[c]. | Capsules (Diflucan™, Flucazole™). Syrup (Diflucan™ suspension). | 2–5 mg/kg PO sid (most species)[c]. |
| Flucytosine | Fungistatic agent. May be administered as adjunctive treatment for aspergillosis, but about 50% *Aspergillus* spp. strains are resistant[c]. | Flucytosine capsules. | 20–100 mg/kg PO bid (most species)[c]. 80–100 mg/kg PO bid (ratites)[c]. |
| Itraconazole | Fungistatic, indicated for systemic mycoses including aspergillosis (currently the drug of choice). Commonly used for prophylaxis. Suspension is first choice, if using capsules, each granule is approximately 0.05–0.39 mg (approximately 285–290 granules/capsule but highly variable number and drug concentration). Method of compounding with strong acid and orange juice has been reported[c]. May cause anorexia. | Oral suspension, capsules (Sporanox™). | 5–10 mg/kg PO sid-bid[c]. |
| Ketaconazole | Fungistatic. Indicated for systemic mycoses, including aspergillosis, and candidiasis. Less toxic than amphotericin B, more toxic than itroconazole. Side effects include regurgitation[c]. | Tablets (Nizoral™). | 15–30 mg/kg PO sid-bid (most species, including ratites)[c]. |
| Nystatin | Drug of choice for treatment of candidiasis, not systemically absorbed across intact GI tract, oral lesions must be treated by direct contact with medication[c]. | Oral suspension (Nilstat Oral™). | 250 000–500 000 IU/kg PO bid (ratites)[c]. |
| Voriconazole | Indicated for aspergillosis, can be used in conjunction with amphotericin B[c]. | Tablets (Vfend™). | 10 mg/kg PO bid[c]. |

## Analgesics

| AGENT | COMMENTS | PREPARATION AND PRODUCT EXAMPLES | SUGGESTED DOSAGES |
|---|---|---|---|
| Butorphanol | Opioid agonist-antagonist. | Injection (Dolorex™, Torbugesic™). | 1–4 mg/kg IM, IV bid-qid (most species)[c]. |
| Carprofen | Analgesic, anti-inflammatory. Use in conjunction with fluid supplementation. | Injectable, tablets (Rimadyl™). | 1–2 mg/kg PO, IM, IV sid-bid (most species)[c]. 2–10 mg/kg IM, SQ, PO sid-bid (psittacines, passerines, raptors)[c]. |
| Meloxicam | Analgesic, antiinflammatory. Use in conjunction with fluid supplementation. | Oral suspension, injectable (Metacam™). | 0.1–0.2 mg/kg IM, PO sid (psittacines, raptors)[c]. |

## Antidotes

| AGENT | COMMENTS | PREPARATION AND PRODUCT EXAMPLES | SUGGESTED DOSAGES |
|---|---|---|---|
| Atropine | Anticholinergic agent. Rarely indicated as a preanaesthetic[c]. | Injection. | 0.01–0.02 mg/kg IM, IV (most species)[c]. |
| Calcium EDTA | Preferred initial chelator for lead and zinc toxicity. May cause renal tubular necrosis in mammals, maintain good hydration and monitor for PU/PD. Do not give PO as this may increase lead absorption from the GIT[c]. | Injection (calcium disodium versenate). | 35–50 mg/kg IM bid 5 days, off 2 days, repeat prn[c] (as indicated by biochemical analysis). |
| D-Penicillamine | Preferred chelator for copper toxicity. Can be used after/in conjunction with initial chelation with CaEDTA for lead and zinc toxicity[c]. | Tablet (D-Penamine™). | 30–55 mg/kg PO bid (minimum 7 days)[c]. |
| Vitamin K1 | Rodenticide toxicity. | Injection, tablets (Konakion™). | 0.2–2.2 mg/kg IM tid-qid until stable, then sid PO, IM, 14–28 days[c]. |

## Obstetric drugs

| AGENT | COMMENTS | PREPARATION AND PRODUCT EXAMPLES | SUGGESTED DOSAGES |
|---|---|---|---|
| Calcium gluconate | Hypocalcaemia—dilute 1:1 with saline or sterile water for IM or IV injections[c]. | Injection. | 5–10 mg/kg slow IV to effect for hypocalcaemic tetany, also SQ, IM, once[c]. 50–100 mg/kg IM, slow IV, once[c]. |
| Calcium syrup | | Oral suspension (Troy Calcium Syrup™—calcium glubionate and calcium lactobionate). | 25–150 mg/kg PO sid-bid (most species)[c]. |

*Obstetric drugs continued on next page*

*Obstetric drugs continued from previous page*

| AGENT | COMMENTS | PREPARATION AND PRODUCT EXAMPLES | SUGGESTED DOSAGES |
|---|---|---|---|
| Prostaglandin $F_{2\alpha}$ (Dinoprost tromethamine) | Dystocia. May be helpful when the egg is located distally and the uterovaginal sphincter is dilated. Can result in uterine bronchoconstriction, rupture, hypertension, death[c]. | Injectable (Lutalyse™). | 0.02–0.1 mg/kg IM, intracloacal once only[c]. |

## Anthelmintics

| AGENT | COMMENTS | PREPARATION AND PRODUCT EXAMPLES | SUGGESTED DOSAGES |
|---|---|---|---|
| Fenbendazole | Effective against cestodes, nematodes, trematodes, *Giardia* spp. Toxicities recorded in some species[c]. Use 2.5%. | Oral liquid (Panacur™). | 20–50 mg/kg PO sid for 3–5 days (most species)[c]. 15–45 mg/kg PO (ostriches)[c]. |
| Ivermectin | Most nematodes and ecto-parasites. Can dilute (e.g. 1:10) with water or saline for immediate use, dilute with propylene glycol for extended use[c]. | Sheep oral formulation (Ivomec™ 0.8 g/L). | 0.2 mg/kg PO, SQ, IM once[c]. |
| Moxidectin | Effective against most nematodes and ectoparasites. Can dilute 1:10 with water. | Sheep oral formulation (Cydectin™ 0.1%). | 0.2 mg/kg PO[c]. |
| Praziquantel | Effective against cestodes and trematodes[c]. | Tablet (Droncit™ tablets, 50 mg). | 5–10 mg/kg PO, repeat in 2–4 weeks (most species)[c]. N.B. can combine with pyrantel (see below). |
| Pyrantel | Effective against intestinal nematodes. | Oral suspension (Combantrin™ children's wormer—50mg/mL, Canex™ puppy suspension—14.4 mg/mL. | 7 mg/kg PO (most species, including ostriches)[c]. |
| Praziquantel/pyrantel combination | | | Combine crushed 50-mg praziquantel tablet with 5 mL of 50 mg/mL pyrantel, give 1 mL/kg |
| Toltrazuril | Coccidiosis. | Oral suspension Baycox™. (50 mg/mL, 25 mg/mL). | 20–25 mg/kg PO once (kiwi, pers. obs.). |

## Miscellaneous drugs

| AGENT | COMMENTS | PREPARATION AND PRODUCT EXAMPLES | SUGGESTED DOSAGES |
|---|---|---|---|
| Furosemide | Diuretic—overdose can cause dehydration and electrolyte abnormalities; toxicity can result in neurological signs and death[c]. | Injectable.<br>Oral suspension (Lasix™), tablets (Lasix™, Diurin™). | 0.15–2 mg/kg PO, IM sid-bid (most species)[c]. |
| Lactulose | Increases gram positive bacteria in GIT, reduces blood ammonia levels, exerts osmotic effect in birds with caeca through fermentation to acetic and lactic acid[c]. | Oral syrup (Duphalac™, Laevolac™). | 150–650 mg/kg PO bid-tid[c]. |
| Metoclopramide | Indicated for GIT motility disorders, regurgitation, ileus[c]. | Injection, oral suspension, tablets (Maxalon™). | 0.5–2 mg/kg PO, IM, IV bid-tid (most species)[c]. |

a Gartrell, B.D.; Alley, M.R.; Mitchell, A.H. 2004: Fatal levamisole toxicosis in captive kiwi. *New Zealand Veterinary Journal 53(1)*: 84–86.

b Alley, M.R.; Morgan, K.J.; Robertson, C.J.R. 2005: Organophosphate toxicity in Northern Royal albatross chicks, *Diomedea sanfordi*. *Kokako 12(2)*: 19–22.

c (Cited in) Carpenter, J. 2005: Exotic animal formulary. Elsevier Saunders, St Louis.

d (Cited in) Marx, K.L. 2006: Therapeutic agents. Pp. 241–342 in Harrison, G.; Lightfoot, T. (Eds): Clinical avian medicine. Spix Publishing, Florida.

### Key to abbreviations

IV = Intravenous

IM = Intramuscular

SQ = Subcutaneous

PO = Per os

IT = Intratracheal

SID = Once daily

BID = Twice daily

TID = Three times daily

QID = Four times daily

prn = As needed

# Appendix 7

## WILDLIFE SUBMISSION FORM

# WILDLIFE SUBMISSION FORM

## Forwarding Instructions

This animal is the property of the Department of Conservation. Please send a copy of test results to: Wildlife Mortality Database Manager, c/- Pathobiology, IVABS, Massey University, Private Bag 11-222, PALMERSTON NORTH

## Submitter Details

Surname: _____
First name: _____
Organisation: _____
Address/Box: _____
Suburb: _____
City/Town: _____
Phone (bus.): _____
Phone (home): _____
Mobile: _____
Fax: _____
Email: _____

## Submission Details

Date submitted: ____/____/____ Submitter ref: _____
Date found: ____/____/____ Number dead: _____
Number at risk: _____ Number sick: _____
(In-contacts)

## Mortality

Date animal died: ____/____/____

Death circumstances:

- ☐ Found dead
- ☐ Found alive and died
- ☐ Treated and died
- ☐ Capture or release
- ☐ Infertile
- ☐ Euthanased
- ☐ By-catch

## Specimen Details

### Animal Details

(Please use separate page for additional animals)

Species/common name: _____
Animal ID: _____
Identification type: _____
(Leg band, microchip implant, rig tag, attoo toe clip etc.) _____
Individual name: _____

Sex: ☐ Male ☐ Female ☐ Unknown
Age Classification: ☐ Adult ☐ Subadult ☐ Juvenile
☐ Neonate ☐ Foetus ☐ Embryo ☐ Egg

Date of birth/mating: ____/____/____

Age/incubation/gestation: _____ _____ _____ _____
period / period Years Months Weeks Days

Where born/hatched Wild ☐ Captivity ☐

Weight: _____ g m/kg

## Location Type

**Wild**

- ☐ Mainland National Park
- ☐ Mainland Reserve
- ☐ Mainland Private Land
- ☐ Maritime Park
- ☐ Island
- ☐ Coastline
- ☐ Sea
- ☐ River
- ☐ Other: _____

**Captive**

- ☐ DoC Facility
- ☐ Private Breeding Facility
- ☐ Rehabilitation Facility
- ☐ Zoological/Wildlife Park
- ☐ Other: _____

Location name: _____
Conservancy: _____
Description: _____

☐ Poisons are being used in the area. Please include details of the toxin.

_____

☐ Special requirements for disposal of body parts, e.g. return to submitter for iwi requirements, genetics, or forward to Te Papa etc.

Please state details of which body parts required and invoice submitter for carrier costs.

_____

## History

Include any information which you think may be relevant to this case.

### Previous health history:

Clinical signs; external examination; individual treaments; abnormal behaviours (feeding, reproductive, agnostic); breeding history; diet with any changes; exposure to toxins; translocation details; previous clinical pathology (attach relevant reports).

_____
_____
_____

### Environmental Conditions (including climate):

Enclosure substrate/size/type; group treatments; in-contacts; clutch details if relevant - sire ID/name, dam ID/name, number of eggs, egg lay interval, season number, season clutch number, incubation temperature and humidity.

_____
_____
_____

Copntinue over leaf

## Invoice Instructions

Invoice: Submitter ☐ National Wildlife Surveillance Fund ☐

(Refer to 'Guidelines for the use of the National Wildlife Surveillance Fund' for eligibility on the WILDLIFE HEALTH PAGE - WGNCR-37176)

New Zealand Government