INTRODUCTION

While pigeons are commonly kept around the world, pigeon fanciers rarely seek veterinary advice. While in part this is often a commercial decision, based on the value of a racing pigeon, there is also a strong feeling that veterinarians don’t understand pigeons (and perhaps pigeon fanciers). This paper seeks to redress this situation by making veterinarians aware of the health requirements and diseases of pigeons. However, it should be noted that this field is too broad to cover it in detail in the time and space available, and the author hopes that those who wish to learn more will access the references provided to avail themselves of the information readily available in the literature.

PIGEONS

Pigeons are members of the order Columbiforme that includes pigeons and doves (and incidentally, the now extinct Dodo *Raphus cucullatus*). There are approximately 300 members of this order, arbitrarily divided by body size into the pigeons and the smaller doves.

*Columba livia*, the domestic pigeon, is thought to have been derived from European Rock Doves. They were amongst the earliest animals to be domesticated, and are even mentioned in the Old Testament of the Bible. Over thousands of years they have been used for a multitude of purposes – message carrying, a food source, sport (racing), aviary specimens, show animals and household pets. Selective breeding has produced many varieties in size and color, resulting in the pigeons we see in practice today.

This paper will focus primarily on the racing pigeon, the most common form seen today. Racing exploits two aspects of pigeon behaviour – their desire to return to their roost, and their ability to navigate over long distances in order to do so.

### Table 1. Biological Data, Domestic Pigeon

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bodyweight</td>
<td>350 – 500 g</td>
</tr>
<tr>
<td>Respiratory rate</td>
<td>25-30 per minute</td>
</tr>
<tr>
<td>Heart rate</td>
<td>240 bpm</td>
</tr>
<tr>
<td>Life span</td>
<td>Up to 30 years, although rarely breed over 10 years. Racing life is usually 7 years</td>
</tr>
<tr>
<td>Food consumption</td>
<td>30 g/bird/day</td>
</tr>
<tr>
<td>Water consumption</td>
<td>30-60 mls/bird/day</td>
</tr>
<tr>
<td>Sex determination</td>
<td>Monomorphic, although subtle differences occur in beak shape, plumage and behaviour</td>
</tr>
<tr>
<td>Egg data</td>
<td>2 eggs laid 44 hours apart; incubation 18-20 days, shared by both parents</td>
</tr>
<tr>
<td>Fledging</td>
<td>Nestlings (squabs) fledge at 24 days; then known as ‘squeakers’ until voice changes at 8 weeks</td>
</tr>
<tr>
<td>Moulting</td>
<td>First moult at 6 weeks of age. Primary moult occurs in early autumn</td>
</tr>
</tbody>
</table>

HUSBANDRY

**Housing**

Racing pigeons are housed in lofts, ideally with 0.25m² floor space per bird. The design of the loft must take into consideration four basic design criteria: the loft must be dry; it must be well ventilated; it must minimise extreme variations in temperature and humidity; and it must be easy to clean.

As well as these design criteria, the loft must meet the birds’ requirement for an environment that it wants to return to. The loft must be safe and secure; it must be a place where they can rest, groom, and interact with other pigeons in a calm, stress-free atmosphere; food and water must be readily available; and the birds should be provided with room and materials to breed if desired.

There are almost as designs and management systems as there are fanciers, but these basic criteria must be assessed by the veterinarian when inspecting premises.

Most fanciers have different lofts for different life stages, ie. young bird lofts, stock lofts, and racing lofts. Each of these presents different problems in husbandry and disease control, and must be assessed separately.

**Feeding**

Pigeons are traditionally fed a mix of grains and legumes. Some greens are offered and usually relished. Various vitamin-mineral tonics and blocks are provided, as is grit.

Fanciers will vary the proportions of different foodstuffs to meet perceived differing needs for racing, breeding and rearing young.

**Breeding**

Pigeons reach maturity at approximately 6 months of age, but are usually not bred till they are 1 year old. Fanciers keep the cocks and hens separate for much of the year, pairing them only when breeding is desired, usually in late winter - early spring. The first egg is laid about 10 days after pairing; the second egg approximately 2 days later. Both parents share the incubation, which takes 18-20 days.
The newly hatched chick (squab) is fed by both parents on crop milk. Crop milk is unique to pigeons; it is produced by the desquamation of fat-laden epithelial cells from the stratified squamous epithelium lining the crop in both sexes. Proliferation of crop epithelium begins at about the sixth day of incubation, and secretion begins at about the sixteenth day of incubation lasting until about two weeks after hatching. The composition of crop milk resembles that of mammalian milk, being rich in fat and protein; fat (6.9-12.7%), protein 13.3-18.6% ash 1.5%, and water 65-81%; unlike mammalian milk, however, it lacks carbohydrates and calcium. These are provided from about the 4th day of feeding in the form of regurgitated food; by the 7th day nearly all the feed provided to the chick is regurgitated food.

The squab grows rapidly. Weighing only 14g at hatch, it reaches 400g by 20 days. It weans at 24 days, at which time it becomes known as a ‘squeaker’ until its voice changes at about 8 weeks.

COMMON MEDICAL PROBLEMS

SKIN AND FEATHERS

Ectoparasites are common in pigeons. Lice (Columbicola columbae, Menapon latum), mites (Knemidocoptes laevis laevis, Knemidocoptes mutans, Syringophilus columbae, Dermanyssus gallinae, Ornithonyssus sylvarium) and pigeon flies (Pseudolynchia canariensis) are all seen from time to time, causing feather damage, skin irritation and restlessness within a flock. Treatments with pyrethrin sprays, ivermectin or moxidectin are effective remedies.

Pigeon pox, caused by the pigeon poxvirus, presents as two syndromes in pigeons. The dry form is seen as discrete scabby lesions on unfeathered parts of the body especially the beak and eyelids. The wet form - fibronecrotic diphtheritic lesions in the oropharynx – is less commonly seen. The presenting syndrome is determined by the strain of the virus, the mode of transmission and the age, species & health of infected bird. Most lesions will heal in 3-4 weeks, although diphtheritic lesions may persist for several months. Although the disease is usually self-limiting, the lesions may become infected and painful, and may interfere with eating, respiration & vision. If lesions are infected, antibiotics and gentle cleansing are indicated. Forceful removal of scabs may result in scarring & deformity.

The virus is environmentally stable, and can survive for years in dried organic debris. It must enter body through the mucous membranes or abraded skin, as it is unable to penetrate intact epithelium. It can be transmitted directly through fighting, feather picking, preening, etc, or indirectly by blood sucking insects eg mosquitoes. The virus will either remain at point of entry, causing localised infection, or spread haematogenously to the liver and bone marrow, producing a systemic infection. The incubation period is 7-9 days.

Prevention revolves around isolation of affected birds, minimising fighting, and prevention of biting insect access. An attenuated live vaccine is available. Only birds older than 6 weeks should be vaccinated. After vaccination, birds are infectious to other birds for 4-6 weeks, and are excluded from racing (hence, vaccination must be given at least 6 weeks prior to the onset of the racing season). If vaccinating in the face of an outbreak, birds already showing lesions should not be vaccinated.

Dermatophytes (usually Trichophyton megnini) typically affect the non-feathered skin, causing thickened, grey patches. Systemic therapy (eg griseofulvin) can be complemented by twice weekly washing with enilconazole or miconazole.

Cutaneous mycobacteriosis is occasionally seen as caseous nodules, usually in the skin of a thin bird.

Iatrogenic feather lesions are seen when pigeons are treated with benzimidazole anthelmintics (such as fenbendazole) or pyrimethamine potentiated sulphonamides when feathers are actively growing. Affected feathers fail to unsheathe, and become brittle and break readily. Cortisone (occasionally given by misguided fanciers as a ‘steroid’) may cause ‘stress lines’ to appear in feathers.

Traumatic injuries are occasionally seen after a hawk attack while flying, or after colliding with wire or other birds in the loft. These injuries are usually amenable to surgery or conservative treatment at the veterinarian’s discretion.

Cutaneous neoplasia is occasionally seen. Fibrolipomas appear to be particularly common.

DIGESTIVE SYSTEM

Trichomoniasis (Canker) is the most common internal parasite seen in pigeons. Trichomonas gallinae is a motile protozoan parasite with a clear, narrow, longitudinal axial rod (the axostyle), an undulating membrane and four anterior flagella. It has a direct life cycle; adults pass it to squabs via crop milk, or it can use faeces, saliva, or crop secretions as the vehicles of dissemination. There may be lentogenic and velogenic forms. It causes necrotic ulceration of the mouth, esophagus, crop and proventriculus; velogenic forms may cause a visceral form of disease involving the liver, gastrointestinal tract and navel. Clinical signs include unthriftiness, regurgitation, diarrhoea, and high mortality. There is often yellow necrotic material in oral cavity and sinuses. Swabbing the crop is usually diagnostic – the characteristic shape and jerky motion of the parasite is readily identifiable. It is thought that infection by lentogenic strains of T gallinae may provide some protective immunity against velogenic strains. However, treatment with the nitroimidazoles (ronidazole, ornidazole, metronidazole and dimetridazole) is usually highly effective.

Worms are also common in pigeons. Ascaridia columbae and Capillaria obesigna, both with direct life cycles, are frequently found in poorly performing birds or those losing weight. Diagnosis is made on fecal examination, and treatment with ivermectin or moxidectin appears quite effective in most cases. Tapeworms are occasionally seen in birds that were have returned to the loft after having been out for some time. Praziquantel is effective.
Coccidiosis is extremely common in many lofts. As with trichomoniasis, lentogenic strains may confer some degree of immunity against more velogenic strains. *Eimeria labbeana* and *E. columbarum* are the most commonly encountered species. Enteritis and weight loss in young birds are the more important clinical signs, and diagnosis is readily made on fecal examination. Very high oocyst counts generally warrant treatment with toltrazuril (Baycox®) or sulfadimethoxine (Albon®).

Salmonellosis (Paratyphoid) is common in some areas. *Salmonella typhimurium var. copenhageni* is the most common isolate, although *S. arizonae* is occasionally reported. The feces of chronically infected carrier birds are the most common source of infection. Contamination of the environment results in build-up of the pathogen, which is then ingested or inhaled. The incubation period can be as short as 3-5 days. There are several forms of this disease; in young birds with a still-developing immune system it frequently presents as an acute onset of lethargy, diarrhoea, weight loss and death. Older birds tend to localise the disease in particular sites e.g., in the meninges around the brain (causing neurological signs) or in the joints (causing swollen, red, painful joints in the wings and legs). A more generalised form is also seen, with multi-organ involvement leading to death. Infertility can result from orchitis or oophoritis. Treatment with antibiotics is based on sensitivity testing, but enrofloxacin (Baytril®) is commonly used. Treatment for 3-8 weeks is often required, but elimination of the bacteria from carrier birds cannot be guaranteed. A killed vaccine is available; two doses, 4 weeks apart, followed by annual re-vaccination is required. Birds under 12 weeks should not be vaccinated.

Other bacterial infections are frequently reported in pigeon lofts *Escherichia coli*, *Yersinia pseudotuberculosis*, *Pseudomonas spp*, *Streptococcus faecalis*, *Mycobacterium avium* and many other bacteria have been isolated from birds affected with diarrhoea. Diagnosis and treatment is based on culture and sensitivity. As with any bacterial infection, the clinician must determine why the bird has the infection, and not just be satisfied with the initial diagnosis.

Candidiasis is commonly reported as a cause of infulvitis, especially in juveniles. Birds that are vomiting should have a crop swab stained or cultured to determine if *Candida albicans* is present. Treatment with an appropriate anti-fungal drug is usually effective.

Adenoviral Inclusion Body Hepatitis has been described as a significant cause of diarrhoea in the UK and Europe. It is most common in 2-4 month old pigeons, with mortality highest 3-4 days after infection. Affected birds adopt a 'crouching position,' and become anorexic. Vomiting, green diarrhoea and polyuria/polydipsia are commonly seen. The virus is shed in feces and ingested. Characteristic inclusion bodies are seen in the liver and intestines. No vaccine is available, but the virus is inactivated by exposure for 1 hour to formalin, aldehydes and iodophors.

Pigeon Circovirus is seen in pigeons in Australia, North America, & Europe. It is morphologically similar, but antigenically different, to PBFD virus. Although transmission is believed to be primarily horizontal (through inhalation/ingestion), histological evidence has been seen in 1-day-old chicks, suggesting vertical transmission. Clinical signs indicate immunosuppression, particularly in young squabs aged 6 weeks – 12 months (before involution of cloacal bursa). They include lethargy, anorexia, diarrhoea, and decreased growth. Numerous secondary diseases are seen, and there is poor response to vaccination. Feather disorders rare but are similar to parrots. Inclusion bodies can be found in the cloacal bursa. As with PBFD in parrots, there is no vaccine or treatment available.

**RESPIRATORY SYSTEM**

Chlamydiosis, due to *Chlamydia psittaci*, is very common in pigeon lofts. A 1983 survey in England and Wales showed that 83% of the surveyed lofts had serological evidence of exposure. Fortunately, the virulence of this disease appears to be lower in pigeons than it is in psittacines – lower grade, chronic infections are more common than explosive outbreaks with high mortality. Transmission is by inhalation or ingested of infected feces or respiratory secretions. Clinical signs include swollen eyelids, ocular discharge, discoloration of the cere, conjunctivitis, keratoconjunctivitis and dyspnoea. More acute cases may present with wasting and green diarrhoea. Concurrent infections (eg PMV-1, Inclusion Body Hepatitis, and Salmonellosis) are common. Often the primary presenting complaint is decreased racing performance. A variety of serological tests and PCR are available for diagnosis. Treatment with doxycycline is usually efficacious, but attention must be given to loft hygiene and ventilation. The zoonotic aspects of this disease must be stressed to the fancier.

**One-Eyed Cold** is the lay term given to a (usually) unilateral conjunctivitis and infra-orbital sinusitis in pigeons. Despite a tendency for doxycycline to be prescribed on the assumption that this is caused by either *Chlamydia psittaci* or *Mycoplasma*, other pathogens may be involved. Herpesvirus, *Trichomonas*, or bacterial infections have all been implicated. The recommended treatment is doxycycline (20mg/bird SID 5 days) and chlorotetracycline eye ointment. Enrofloxacin is also used by some pigeon veterinarians. Affected birds should be screened for trichomoniasis.

*Mycoplasma* is often regarded by fanciers as a major cause of respiratory disease. In contrast however, researchers have yet to determine the role *Mycoplasma* plays in respiratory infections. Three species have been isolated from both sick and healthy pigeons – *M. columboreale*, *M. columbinum* and *M. columbiasale*. *Mycoplasma* is shed in feces and respiratory secretions, and transmission is by inhalation or ingestion. Treatment with doxycycline or enrofloxacin is recommended.

**Aspergillosis** is not uncommon in poorly ventilated lofts. Affected birds usually present for poor performance, weight loss and dyspnoea. Occasionally a caseous sinusitis is present. Diagnosis is based on autopsy findings, histopathology and culture of affected tissues. While individual treatment is feasible, it is not
usually carried out by fanciers as complete recovery and return to racing form is unlikely. Underlying factors, such as immunosuppressive diseases and poor ventilation, should be identified and eliminated.

**Paramyxovirus-1 (PMV-1)** in pigeons is not the classical Newcastle Disease seen in poultry, although it can cause disease in poultry. It usually presents as a neurological disease, although rapid breathing and facial oedema can occur. This disease will be discussed more fully under neurological diseases (below).

**Pigeon herpesvirus (inclusion body hepatitis, Infectious Esophagitis)** is caused by *Columbidae Herpesvirus* 1 and 2, a betaherpesvirus. The pigeon is the primary host and reservoir, with squabs aged 4-16 weeks the most susceptible age group (older birds show milder signs that may go unnoticed and resolve within 1-2 weeks). The virus is spread by fecal and pharyngeal discharges; latently infected carriers are common and transmission occurs during periods of stress. The mild form is seen as a respiratory disease (mild rhinitis, tracheitis and conjunctivitis) with small ulcerations on the mucous membranes of larynx, pharynx, cere and commissure of the beak. The more severe form of the disease is seen as dyspnoea, anorexia, biliverdinuria, diarrhoea, vomiting, protrusion of the third eyelid, and neurological signs. Pathology reveals tracheitis, multiple focal, degenerative lesions of liver (diffuse hepatic necrosis with intranuclear inclusions) and diphtheroid foci on mucosa of upper airways, pharynx and occasionally crop and intestines. Diagnosis is usually based on finding intranuclear inclusion bodies in the liver and other organs. There are no reliable means of ante-mortem diagnosis. A vaccine is not commercially available; trials indicate that it does not provide protection, but may reduce the degree and duration of viral shedding. The virus is unstable outside of the host and is susceptible to most disinfectants.

**Parasites** occasionally occur in the respiratory system. Trichomonas can be found in the sinuses of some birds. Nasal mites (*Neonyssus columbae, N. mellori* and *Sternostoma striatius*) and air sac mites (*Cyodites nudus*) have also been found, although the nasal mites are generally regarded as non-pathogenic. Heavy infestations with any mites could lead to decreased performance. *Syngamus trachea* (gapeworm) has been recorded, but is regarded as rare. Typical Y-shaped paired worms are found in the trachea of affected birds, which present for gaping and sneezing. Treatment with ivermectin or fenbendazole is recommended.

**Ammonia toxicosis** occurs in poorly ventilated, unhygienic lofts. The build-up of ammonia from the droppings initially causes irritation of the conjunctiva and respiratory epithelium, causing ocular discharge, head shaking, sneezing and coughing. If not identified and remedied, affected birds become lethargic and perform poorly. Diagnosis is usually made on examination of the loft; if the clinician’s eyes begin to water and the smell of ammonia is strong, a presumptive diagnosis of ammonia toxicosis can be made. Ammonia toxicosis can be a predisposing factor for other respiratory infections.

**NEUROLOGICAL SYSTEM**

**Paramyxovirus-1** is the causative agent of Newcastle Disease in poultry. In the 1980’s a strain of this virus that appeared to be primarily restricted to pigeons spread from the Middle East into Europe and the UK. It has since spread to Africa, SE Asia and the USA and Canada. It has an incubation period of a few days to a few weeks. In an infected loft new cases can appear for 5-8 weeks after first diagnosed. Affected birds display polydipsia, then watery to haemorrhagic diarrhoea and then neurological signs (head tremor, torticollis, paralysis of wings or legs, and blindness). Respiratory signs are minimal (see above). The mortality rate in adults is low, with birds recovering in about 6 months (although they can have persistent diarrhoea for several months). In young birds malnutrition and renal damage can lead to higher mortality rates. This disease is transmissible to poultry. Vaccination is available and recommended. Racing pigeons in the UK are required to be vaccinated.

**Salmonellosis (Paratyphoid)** can cause neurological signs if the infection localises in the meninges. In these cases affected birds may be ataxic, unable to hold their head in a normal position, lie on their side, and difficulty eating. See the gastrointestinal section for more details.

**Poisoning** commonly occurs around feed mills and farms when poisoned grain is used in an attempt to control feral pigeons. It can also be iatrogenic eg overdosage of dimetridazole (Emtryl®) during treatment for trichomoniasis. Poisoning should be suspected when large numbers of birds are affected simultaneously, or when no other cause of neurological signs is apparent.

**Hypocalcaemia** occurs in breeding hens fed a nutritionally marginal diet. Paresis of the wings and feet, especially after the first egg is laid, is the most common syndrome. Affected birds are unable to fly and often drag themselves along using their beak and their wings for support. Supplementation with Vitamin D3 and calcium, along with supportive care, is usually effective.

**Thiamine (Vitamin B1) deficiency** has been reported to cause neurological signs (weakness, tremors, and opisthotonus) in pigeons that are anorexic for other reasons. Anorexic pigeons should be supplemented with multi-vitamins to prevent this possibility.

**WINGS AND LEGS**

**Congenital abnormalities** are occasionally seen in squabs. They are usually mild, as so-called monstrosities rarely survive hatching or the neonatal period. They include webbed feet (syndactylysm), extra legs, and abnormal feathers (‘porcupine’ quills).

**Metabolic bone disease** is not uncommon in squabs fed marginal diets or when denied access to sunlight or Vitamin D3 supplementation. Curved keels and pathological fractures of the long bones are the most common presentations.

‘**Angel Wing**’ is the term given to a condition where the distal end of the wing is turned up and out. It occurs when chicks are fed a high-energy diet and grow rapidly. The weight of the blood-filled quills becomes too great for the still-developing wing bones, and this abnormality
develops. In the early stages it can be treated by strapping the wingtips into a normal position.

**Pinwheel or Splay Leg** is an outward deviation of the long bones of one or both legs in squabs. The leg is rotated and the femoro-tibiotarsal joint may fuse. Affected chicks are unable to draw the leg under them and the chick is unable to stand. Instead it scrabbles (pinwheels) on the floor of the loft. Various causes have been postulated including tightly-sitting hens, slippery nest surfaces, and nutritional deficiencies. It may be more common in single-chick nests. Hobbling has been tried with varying degrees of success.

**Infectious arthritis** has been reported with Staphylococcal infections and salmonellosis. Staphylococcal infections are usually a problem of individual birds, whereas salmonellosis usually presents as a flock problem.

**Pododermatitis** (‘bumblefoot’) – infection of the plantar surface of the foot by *E. coli* or *Staphylococcus* – is occasionally seen in pigeons housed in less than ideal conditions. The infection may extend up the tendon sheaths and localise in joints and bone. Occasionally the claw on the posterior digit is lost.

**Trauma** from collisions or hawk attack can present as broken bones, bleeding quills and bruising.

**ANAESTHESIA**

As with other birds, inhalation anaesthesia using Isoflurane is the preferred anaesthetic in pigeons. Mask induction, followed by intubation and IPPV, provides a relatively safe anaesthesia with rapid recovery.

Where inhalation anaesthesia is not available, injectable anaesthetics have been used with varying degrees of success.

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dose and Route</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ketamine + Medetomidine</td>
<td>1.5-2 mg/kg i/m 60-88ug/kg i/m</td>
<td>Can be reversed with atipamezole (250-380 ug/kg i/m).</td>
</tr>
<tr>
<td>Ketamine + Xylazine</td>
<td>4.4 mg/kg i/m 2.2 mg/kg i/m</td>
<td>Can be reversed with yohimbine (0.1 mg) or atipamezole; Failure to reverse leads to prolonged recovery</td>
</tr>
<tr>
<td>Propofol</td>
<td>1.33 mg/kg i/v</td>
<td>Can cause initial apnoea; very short duration of effect</td>
</tr>
<tr>
<td>Tiletamine / zolazepam</td>
<td>5-10 mg/kg i/m</td>
<td></td>
</tr>
<tr>
<td>Alphaxalone / alphadalone</td>
<td>5-10 mg/kg iv or 36 mg/kg i/m (iv route preferred because of large volume)</td>
<td>Occasional short period of apnoea on induction; effect lasts 10-20 minutes</td>
</tr>
</tbody>
</table>

Analgesia with butorphanol (1-3 mg/kg) or meloxicam (0.2 mg/kg) should also be given for any potentially painful procedures. Buprenorphine appears to be relatively ineffective in birds.

Other anaesthetic considerations include maintaining body temperature and providing fluid support. These are basic requirements for avian anaesthesia and surgery and will not be discussed further here.

Anaesthetised patients should be closely monitored. Respiratory rate, heart rate, oxygen saturation and blood pressure should be closely monitored. The endotracheal tube should be closely monitored for the accumulation of mucus in the lumen. Warning signs of impending problems include a decrease in respiratory rate to less than 6 breaths/minute and/or a decrease in heart rate to less than 2/3 of initial rate. If these events occur, the anaesthetist should decrease anaesthesia and support respiration (ventilate at 10-12 breaths/minute, with a peak inspiratory pressure- $8-12$ cm H$_2$O).

**SURGERY**

Surgical considerations for pigeons are similar to those of other avian species. The relatively low economic value of the average racing pigeon makes it unlikely that many are presented for surgery; however, valuable breeders, high-performing racing birds and pet birds may well qualify for surgery. Most procedures are trauma repair following hawk attack, although fractures, tumour removal and other procedures are occasionally performed.

Basic surgical principles follow those in other species:

a. Speed is essential to minimise heat loss. The surgeon must be well prepared and have his/her surgery planned before beginning
b. An aseptic technique must be used; antibiotics are not a substitute for poor hygiene
c. Gentle handling of tissue reduces post-surgical pain and speeds wound healing
d. Minimise blood loss

Pre-surgical evaluation requires a physical exam, examining the patient’s general condition, weight and respiratory recovery time (return to normal respirations in 3-5 minutes following handling). Other evaluations may include blood work, radiographs, EKG, and a crude estimate of clotting time (pin prick of Basilic Vein – in a normal bird it should clot after one minute’s application of direct pressure). If time permits, the patient should be pre-conditioned. Vitamins may be of some help with patients on nutritionally marginal diets, but must be given well in advance. If appropriate, antibiotics should be given pre-operatively. If the patient’s haematocrit is < 25%, the surgeon may need to consider a blood transfusion. If the serum glucose < 200 mg/dl (considered hypoglycaemic in most birds) give 5% dextrose IV intra-operatively.

Pre-anaesthesia fasting should only be long enough to empty the crop – usually only about 3-4 hours at a maximum. Longer fasting periods can result in depletion of the liver glycogen stores.
Suture material choice depends on the surgeon’s preferences, although the following must be taken into consideration:

a. Chromic gut causes a marked, granulocytic inflammatory response in pigeons. It has a prolonged presence (>120 days)
b. Polyglactin 910 (Vicryl) has the most intense inflammatory reaction but is most readily absorbed (60 days)
c. Polydioxanone (PDS) is the least reactive and retains its integrity the longest
d. Stainless steel and nylon causes a greater degree of fibrosis, haematoma, seroma and caseogranuloma formation.

References and Recommended Reading