

It Died! I think I killed it! I feel guilty! (The Value of a Post-mortem)

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ABSTRACT: Wildlife carers put hundreds of hours into rescuing and rehabilitating wildlife. They also invest a huge amount of emotional love and dedication while doing it and it is often devastating when an animal dies for some unknown reason. Many feel that they must have done something wrong with their feeding or care and often have feelings of guilt or failure. In Townsville, North Queensland Wildlife Care have had the advantage of having a good working relationship with the James Cook University Veterinary School and have for the last 15 years been paying for native animal post-mortems and for histopathology of all major organs.

Not every animal is sent for post mortem. Resources are not unlimited and this service is reserved for cases where a likely diagnosis has not been reached or needs confirming to help treat others at risk. This paper will look at a summary of results of 15 years of cases.

Introduction


In Townsville, North Queensland Wildlife Care have the advantage of having a good working relationship with the James Cook University Veterinary School and have for the last 13 years been paying the fees for members post-mortems and for histopathology of all major organs when wildlife has died for unexplained reasons.

Not every animal is sent for post mortem. Resources are not unlimited and this service is reserved for cases where a likely diagnosis has not been reached or there is a need to confirm a diagnosis to help treat others at risk.

As the pathologists don't work on weekends there are times when animals die and it is not possible to get the postmortems done in a timely manner. Bodies can be kept under refrigeration for 24 hours at a pinch but it is not ideal. Refrigeration should be used even to keep a body overnight till it can be autopsied next morning. Freezing virtually destroys any chance of a diagnosis and no histopathology is possible. One must remember that the feathers of birds can delay chilling because of their good insulating properties. Consequently birds are wet down with water with a detergent in it so that they will chill quickly before placing them in a plastic bag in a refrigerator to hold overnight for autopsy next day.

In all 189 animals were submitted in this 13 year period comprising 86 macropods, 38 brushtail possums, 36 birds, 12 koalas, 9 Flying Foxes, 4 reptiles, 3 echidnas and 1 bandicoot.

There were 15 animals, namely 11 koalas, 2 reptiles, 1 Bird and 1 fruit Bat, included in this talk that did not come through our wildlife group but came from EPA or a wildlife park. All the rest came from members of North Queensland Wildlife Care.



Macropods

Deaths in macropods are particularly upsetting for carers because they have often been raised from a small pinky up through the various stages and in the case of the larger species may have been in care for 12-15 months. Many grey and red kangaroos become quite tame and people become emotionally attached so that a sudden death is devastating.


This analysis of those deaths may help us understand the causes and may lead to a change in people's beliefs about what husbandry practices are desirable.

Agile Wallaby.

This is the most common macropod in our area and 47 cases were submitted for post-mortem: 21 Males and 19 females with the sex of 7 unrecorded. Causes were many and varied.

- **9 Acute Enterotoxaemia** most likely bacterial – *C perfringens*?
- **7 Acute Septicaemia** (*E coli* isolated in one case) probably associated with gut infection
Acute bacterial. pericarditis? Myocarditis?
- **4 Pneumonia** -- often per-acute
- **5 Trauma** MVA x 3, 1 Dog attack and 1 Ran into fence – neck, spinal injury.
- **4 Haemorrhagic Coccidiosis**, two with *Salmonella*
- **7 Undetermined**
- **2 Snakebite**, one was definite with fang marks on shoulder
- **1 Acute Meningo-Encephalitis** caused by *Toxoplasma gondi*
- **1 Acute Pneumonitis** caused by *Toxoplasma gondi*
- **1 metabolic bone disease**
- **1 Acute necrotizing stricture** of pylorus
- **1 Hairball obstruction** (trichobezoars) many 20mm x 50mm hairballs
- **2 Myocardial degeneration** one with nephritis.
- **1 Ingested Toxin** (Fungus)
- **1 Lumpy jaw** *Actinobacillus* infection

Most were presented as a sudden death – cause unknown. Some are found collapsed and die shortly after. Some are seen with neurological signs that are thought to be Toxoplasmosis but many animals that have seizures/convulsions turn out to be toxaeemias due to bacteria causing a bacterial enterotoxaemia or a bacterial septicemia



Eastern Grey Kangaroo

Nineteen Eastern Grey Kangaroos were submitted. When reviewing these results it was obvious that as a group they were very susceptible to Coccidiosis which in this species has been well known for a long time.

- Six died of acute haemorrhagic gastro enteritis due to Coccidiosis **that could have been complicated with *Clostridium perfringens***
- Three died of acute haemorrhagic gastro enteritis due to **Salmonellosis** Two of these had concurrent Coccidiosis
- Three died of acute enteritis and **enterotoxaemia** probably bacterial *C. perfringens*

All of these animals, 59% of the total, died in less than 48 hrs

- There was one suspect Toxo that turned out to be viral encephalitis
- Two suspect Toxoplasmosis were in the endotoxaemia group above.
- There was one animal sick and lethargic for 3 days, turned out to be an Interstitial Nephritis suspected of being due to Leptospirosis
- Three were undetermined – these died very rapidly – in a few hours. **Enterotoxaemia?**
- One animal died of toxic shock—haemorrhagic pneumonia, septicemia. Its symptoms were of pyrexia and convulsions.
- There was only one Trauma case.

Looking at all the grey Kangaroo cases, it is obvious from the above that in this breed of macropod we must prevent coccidiosis causing any damage to the gut that can then allow secondary infection with either *Salmonella* or *Clostridium perfringens* as these organisms can kill very rapidly...

Red Kangaroos


Eight were submitted for autopsy and one was a faecal submission for diarrhea that was +ve for Coccidiosis -ve Salmonella

Of the 8 autopsies 4 died of Acute Haemorrhagic Gastro/enteritis Two were sick for less than 24 hrs and in one that had no signs of diarrhoea they cultured *Pseudomonas aeruginosa* which had caused an acute Septicaemia. The other two were sick for 1-2 days all were thought to be due to bacterial toxins. *C. perfringens*? One red that had been sick for 21 days with neurological signs was found to have a Granulomatous Encephalitis caused by *Coccidioides immitis*. Try diagnosing that before death!

One other with suspected neurological disease had Liver disease and mineralization of the kidneys which would have caused the symptoms. One other was sick for 10 days and was thought to have Toxo (mother had Toxo) and it died of multifocal pneumonia.

Again we see that suspected disease is not always confirmed on postmortem.

Our most recent case was a Red with weight loss that on post-mortem was diagnosed with having an acute pancreatitis that was associated with rupture of the pancreatic duct causing acute peritonitis in the immediate area. There was a concurrent colitis.



Refous Bettong

Three bettongs were autopsied. Two died of acute bronchopneumonia; one due to inhalation of plant material and the third animal was euthanased for a suspect tumour of the pouch which turned out to be purulent bacterial lymphadenitis of the pouch.

Allied Rock Wallaby

Three were submitted, all following sudden deaths of unknown origin. Two had acute pneumonia and kidney damage of unknown origin and the third which was suspected of dying from snakebite (as good a guess as any) was found to have some nephritis and it was thought that the diagnosis may have been correct.

Common Euro

Only two were submitted. One was thought to have died of trauma and was in fact found to have multiple haemorrhages in the abdomen due to trauma. Probably MVA. The other had been suffering from chronic cystitis for many weeks and when eventually post-mortemed was found to have >100 small stones in the bladder 0.5-2.0 mm in diameter. Male macropods are impossible to catheterize and urine samples are usually collected by centesis. They would probably have shown up on Xray. I imagine urethral obstruction would be a problem in this male.

Common Wallaroo


Only two submissions. One was a sudden unexplained death which was found to be due to an Acute Enterotoxaemia, *C.perfringens*? The other was sick for 4 days with epistaxis and haematuria which was found to have per acute septicemia and DIC (Disseminated Intravascular Coagulation –a simultaneous coagulation and haemorrhagic event) again probably due to bacterial toxin.

Whip Tail Wallaby

Only one submitted and it died of acute coccidiosis

Summary of All Species of Macropods

Macropods are very prone to die rapidly mostly from enterotoxaemia probably due to *C perfringens* but often associated with Coccidiosis and Salmonellosis. Pneumonia is also an important cause of death. Of 86 macropods submitted 36 (41%) died in less than 24hrs sometimes in only 2-4 hrs. 15 (17%) died in 24hrs and 12 (14%) died in 2 days. That is nearly 72% died within 48 hrs of getting sick. Even if we implemented antibiotic therapy within the first few hours we generally accept that antibiotics take 24-36 hrs to start a response so how effective will the treatment be? Ask yourself how many of these cases could have been prevented by vaccinating with 5 in 1 vaccine to prevent enterotoxaemia/Septicaemia caused by *Clostridia perfringens* and having regular coccidioprophylaxis?



Common Brushtail Possum

40 Submissions 38 of which were post-mortems and two were faecal cultures (1 positive for Salmonella) The 38 Post mortems (15 Male 15 Female 5 unrecorded) can be summarized as follows

- 8 Cases died of **gastro enteritis** caused by a variety of agents some coccidia, some parasitic, candidiasis and bacterial. In one case Enterobacter cloacae, E. faecalis and Klebsiella sp, all gram negative bacteria, were cultured.
- 7 Possums died of **pneumonia** which were mostly acute and often complicated by kidney problems such as interstitial nephritis thought to be due to Leptospirosis.
- 5 possums died with **renal failure** and 3 of these had crystals in the kidneys Some of the mineral deposits were identified as Oxylate crystals that probably came from plant food high in oxylates.
- 3 died of **septicaemias** of bacterial origin
- 2 died due to **trauma** both after dog attacks
- 3 died of acute **poisonings** two of these were thought to be from a bacterial toxin causing liver necrosis. One was thought to be a plant toxin causing liver necrosis.
- 2 died of **Gross abdominal catastrophe**. One was an intersusception of the ileum into the colon and one an intersusception of the colon with perforation and peritonitis.
- 2 had a **myopathy**
- 1 had a **demyelination of the spinal cord** thought to be due to a virus – It had paresis
- 1 had **non suppurative meningo-encephalitis** Had a head tilt
- 4 **Undetermined**

Summary

Again we can see that many animals die of gastroenteritis. As most of these deaths are rapid i.e. less than 24 hrs then it is a fair assumption that though we found coccidia, parasites and candida, these by themselves would not usually kill that quickly.

How important is *C. perfringens* in this species? It is difficult to diagnose as this bacteria belong to the Clostridia class that are recognized for their ability to produce highly lethal toxins that can kill in minute amounts. Most times these bacteria gain ascendancy once there has been something to upset the balance within the GI tract such as coccidiosis, dietary change, parasites etc. I believe all possums should be vaccinated with 5 in 1 vaccine to prevent these sudden deaths.

Following the analysis of deaths in macropods I have no problem convincing macropod carers in our group to start vaccinating with Ultravac 5 in 1 vaccine

Clostridium perfringens

Exhibiting frighteningly violent and rapidly progressing symptoms, *Clostridium perfringens*-induced intestinal diseases have confounded livestock producers and veterinarians around the world, along with medical doctors who at times have been stunned to diagnose in humans what is more commonly considered an animal disease.

C. perfringens is ubiquitous in nature and can be found as a normal component of decaying vegetation, marine sediment, the intestinal tract of humans and other vertebrates, insects, and soil. Virtually every soil sample ever examined, with the exception of the sands of the Sahara, has contained *C. perfringens*.

"It makes a lot of toxins, and it's almost always lethal."

Clostridium perfringens is divided into five types based on production of four toxins. These four main toxins--known as alpha, beta, epsilon and iota toxins--combined with many other toxic substances created by the bacteria, produce nearly 25 different diseases.

Once an animal contracts a disease caused by *Clostridium perfringens* it's often too late to do anything about it. Death comes quickly and violently. This type of bacteria, occurring in five different strains identified by toxin type, produces a host of toxic proteins; nearly twenty have been described scientifically and there may be more. These toxins can act rapidly in the body, causing severe diarrhoea, dysentery, gangrene, muscle infections and various other forms of enteric (gut) disease. The symptoms vary in intensity and variety depending on the individual toxin and its host.

C. perfringens can cause disease in most domestic animals including horses, poultry, sheep, birds, rabbits, goats, pigs, cattle, mink, ostrich, emu, dogs, cats and some wildlife,

Humans have also become infected, although cases of enteritis have been localized, most notably in the highlands of Papua New Guinea where it occurs as a severe, usually fatal form of food poisoning that kills the small intestine.

In spite of its potential danger as an infectious agent, the avirulent forms of bacillus are commonly found in the intestinal tracts of warm-blooded animals, and it also inhabits terrestrial, marine and aquatic environments. *The trouble starts when the balance of bacteria in the gut is disrupted, giving C. perfringens a chance to proliferate unchecked.* It may contaminate soil, animal feed and litter, or be transmitted directly from infected to healthy animals.


The most practical way to handle perfringens-related illnesses in animals is to prevent them in the first place by vaccination

<http://ag.arizona.edu/pubs/general/resrpt1998/clostridium.html>
Vaccination protocols for 5 in 1 Ultravac Vaccine (Pfizer)

(*Clostridium perfringens* type D, *Cl. tetani*, *Cl. novyi* type B, *Cl. septicum*, *Cl. chauvoie*.) The first two are the ones that are most important to marsupials.

The latest protocols I can find are those recommended by David Blyde BVSc

For orphan hand reared marsupials including macropods, possums, gliders, echidnas and wombats

- The first vaccination should be given as soon as the animal is stable, certainly within 48 hrs
 - Vaccination should be repeated every week for the next 4 weeks i.e. on day 2,9,16 and 23
 - Vaccination should then be repeated fortnightly for a further month i.e. on day 37 and 51
 - Vaccination should then be given every 6 months with the last vaccine given some days before release
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The Dose rates used for these juveniles is as follows;-

- Less than 30 grams give 0.25ml subcutaneously.
- 30 - 100 grams give 0.30ml subcutaneously.
- 100 – 250 grams 0.5ml subcutaneously.
- 250 – 500grams 0.75ml subcutaneously.
- >500 grams 1.00ml subcutaneously.

For adults the dose rate is 1ml given twice, four to six weeks apart, with a booster every year. The above dose rates can be used for macropods, possums, gliders, echidnas and wombats.


Birds.

There is such a great diversity of species in this group that it will be no surprise that there was a great variation in the diseases seen. Birds probably make up the highest number of wildlife admissions for most wildlife groups. As a group they probably spend less time in care than other species. Orphan rearing is usually measured in weeks rather than months. Birds have a survival mechanism that they don't usually show outward signs of illness until they are so weak that they can't perch or fly. Feathers often act as a cloak that hides an emaciated body that has been ravaged by illness and disease.

This means that birds often appear to die suddenly, are found dead in the cage in the morning. As these birds are wild we don't catch and handle them very often as that is stressful, it means that we are often unaware that there is a disease process going on that is causing the bird to lose weight. Every time a bird comes to hand we immediately assess the muscle coverage of the Keel bone to give us some immediate assessment of the bird's general physical health and condition. When wild adult birds come into care it is generally because there is some physical or disease problem. If there is no trauma to bones etc that would prevent them flying away they are only on the ground able to be caught because they are sick or dying and most times they are well along on the path to death.

The reason that there are less bird autopsies proportionally to the number of admissions is the fact that rehabilitators handle much larger numbers and accept that many coming into care without physical injuries are already severely ill and will probably die. Many are common species and we recognize their problems eg. beak and feather disease in sulphur crested cockatoos and lorikeets that would not necessitate an autopsy. More importance is placed on autopsies if there is a group of mortalities. We encountered two such events. One was when about 15 galahs died on Magnetic Island and autopsies showed demyelination in the CNS cause unknown. The other event was the sudden death of 10 birds of 5 different species in one residential location. These were caused by carbamate poisoning, an insecticide used for lawn grubs. It was not known whether this was accidental or a deliberate poisoning. Sometimes people try to get rid of Indian Mynah birds with tragic results for non target species.

I have looked at the results of all 36 autopsies and there is more variation of cause of death than in any other group. In most cases it would not have been possible to diagnose the cause let alone treat the condition. I tried sorting them statistically by species in alphabetical order we had:



- 1 Cassowary - died of cane toad poisoning (the only bird not submitted by us)
- 2 Common Koel - pneumonia; Trauma.
- 1 Crested Hawk - parasitic enteritis and bacterial infection of many organs
- 1 Crimson Wing Parrot - Bilateral bacterial ophthalmitis
- 2 Cookoo Shrikes - Acute Pneumonia; Severe Pox virus – multifocal
- 4 Curlew - Ricketts x3 and 1 undetermined Calcium deficiency in diet of orphans
- 1 Fig Bird – undetermined.
- 2 Zebra Finches – undetermined but they had a nematode burden.
- 3 Galahs - CNS demyelination - unknown cause in 15 dead Galahs on Magnetic Is.; malnutrition due to beak deformity and one undetermined.
- 2 Kestral northern- Acute bacterial Pneumonia, splenitis and peritonitis; undetermined
- 1 Kite Brahmany - acute pericarditis and necrosis of myocardium
- 1 Kite whistling – haemorrhage due to possible trauma
- 2 Kookaburras Bluewing – respiratory aspergillosis; Undetermined
- 1 Kookaburra Laughing - necrotizing oropharyngitis due to fungal infection.
- 1 Mangrove Kingfisher – undetermined
- 1 Nightjar – Anaemia – Heavy metal?
- 1 Owl – acute fungal pneumonia and sacculitis
- 2 Peewee – Acute pectoral and myocardial myopathy; the other was undetermined.
- 1 Pelican – acute parasitic gastritis
- 2 Pheasant Coucal – acute enteric septicaemic salmonellosis; Toxic nephrosis
- 2 Rainbow lorikeets – Focal mite infection; one of .10 birds that died of deliberate carbamate poisoning
- 1 Redwing Parrot – undetermined
- 1 sparrow - carbamate poisoning one of multiple deaths

Put another way, cause of death was 4 Pneumonias, 5 Parasites, 3 Poisonings, 3 Ricketts, 2 Neurological, 2 fungal infections and 6 were undetermined. All the rest were individual fatal problems. It is interesting to note that both fungal infections were found in birds that eat small rodents reptiles etc (Kookaburra and an Owl). The curlews with ricketts were all from a very good carer that was raising a lot of Curlew fledglings in 1996/7 and once we corrected the diet no further animals died. You can see from the above that although we live by the sea very few sea birds of any sort are handled.

I am sure that many hundreds of birds died and were not autopsied, so this is only a small sample of what really occurs.

Koalas.

In my area we have very few koalas and only one has ever come into care for rearing. North Queensland Wildlife Care have only submitted one animal for post mortem that was inappetent for 2 days before dieing. However I found 12 cases submitted to JCU for PM and histo 7 were from a local wildlife sanctuary one from us and the rest were submitted by Parks and Wildlife ranger.

There were 4 pneumonias one of which was fungal. Two had myocardial degeneration. Two died of Lymphosarcoma. Two were undetermined. One died of suspected malnutrition and one died of malnutrition, vector borne. 50% of these animals were sick for more than a week and 30% for more than 3 weeks which contrasts with our other marsupials.

Fruit Bats. (Flying Fox)

A total of 9 fruit bats were submitted. 7 Little Reds *P.scapulatus*, 1 Black *P.alecto* and 1 Spectacle Flying Fox, *P.conspicollatus*. It is interesting to note that in August 1996 the first Lyssa Virus case (in a Little Red Flying Fox) was diagnosed from a submission of our Bat co-ordinator.

Since that time two people have died from Australian Bat Lyssavirus. From 1996 to 2003 four cases of lyssavirus were confirmed and one case suspected. Three bats died of pneumonia and one of a fatal infection causing focal necrosis in the liver. After 2003 all suspect bat autopsies have to be carried out by the Oonoonba Laboratory of the DPI and there have been more than a dozen confirmed lyssavirus cases.

This probably explains why many veterinary clinics will not handle bats at all. Our wildlife group offers a 50% subsidy of up to \$50 on the cost of each rabies vaccine and we have a medical practice that will bulk bill the visits for vaccination. Some carers pay for their vaccination themselves. Our bat carers must have a blood test to check immunity titre every 2 years and have a booster if it falls below 0.5.

Echidna

Three cases reared by my wife and I were submitted.


Case 1. back in 95, was a young puggle about 800g that we were rearing and trying to get onto more solid food with insectivore mix. It died of impaction obstruction and bloat (possibly with secondary *C.perfringens*).

Case 2. 97. Was a small puggle of 200 grams that was being stomach tube fed (on advice from so called experts) that developed a fever and died. It had septicaemia, nephritis and choroiditis. I have never stomach tubed an echidna since and have never needed to.

Case 3. 2003 was a puggle that was being successfully reared when it was accidentally dropped on its head from a height of 1.4 meters. The nurse pulled a towel out of a high cage not realizing that the puggle was temporarily wrapped in it) Despite treatment it developed neurological signs that later necessitated euthanasia as it was not going to be able to be released. Autopsy showed atrophy of the right Cortex due to previous injury.

Faun Footed Bandicoot

Only one submitted and the cause of death was undetermined.



Conclusion

Autopsies are important to determine the cause of death. Many acute or sudden deaths are unexplainable to the carer who has followed established feeding protocols and husbandry and is at a complete loss on the death of their animal. The speed of some illnesses gives no real indication of the cause. In the 86 macropods the suspected disease was confirmed in only 25 cases which is 29%. Some of these suspect diseases were as broad a diagnosis as "Diarrhoea" that was confirmed as coccidiosis or salmonella or endotoxaemia. Sudden deaths were often found to be acute pneumonias.

While I know it is often difficult to get autopsies and histopathology in many country areas and even larger towns in a timely manner, it must be remembered that once the animal is dead it can still tell us a lot with just a simple autopsy by your local vet.

Be prepared to accept that with any rapid deaths there may be nothing to see in a simple post mortem and only histopathology of the major organs will give some insight into the cause. Animals that have been sick for some time are often more rewarding.

I would like to express my gratitude to Prof Phil Summers for permission to spend many hours in the pathology registry gaining access to the original hard copy request forms so I could determine the length of illness and suspected diagnosis data.

BIOGRAPHY:

Jim Pollock BVSc graduated from University of Queensland 1967 and spent 4 years in Commonwealth DPI. Had a mixed practice in Bowen NQ from 1972 to 1985 when he moved to Townsville. Built an ASAVA Accredited Small Animal Hospital in Townsville in 1991. He retired from full time general practice in 2002.

All his professional life, he has treated wildlife and provided drugs free of charge. He was an early member of North Queensland Wildlife Care Inc. which was formed in 1989, and has been President the last few years.

On Australia Day, January 2005 he was awarded Citizen of the Year for Townsville for his contribution to Wildlife and the Arts. He continues to work from home helping wildlife daily and occasionally does locums and orthopaedic surgery for other veterinary practices. He and his wife Eleanor (Vice President) are presently overseeing the refurbishment of a building that will become a Wildlife Education and Assessment Centre and home for North Queensland Wildlife Care that will include large lecture room for 40 people, office, utility room, two toilets, kitchen, reception area, treatment area and a surgery.

