Cryptic Trauma and its role in Lorikeet Paralysis Syndrome – A summary of information presented at Science Week 2021

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Lorikeet Paralysis Syndrome (or LPS) is traditionally defined as "a sudden onset of paralysis in both legs (bilateral) and/or clenched feet". This syndrome has been observed since the early 1970's . The condition has most commonly been described from affected birds in Sydney and the central New South Wales coast area, south east Queensland and Victoria (WHA 2014).

The incidence of this classical form LPS in our facility is approximately 5%. This consistent with historical records showing between five and ten percent of lorikeets rescued annually in south-east Queensland and coastal NSW are presented with this syndrome (Booth et al 2001).

Recently, the definition of LPS has inadvertently evolved to include any lorikeet that is found on the ground and is unable to fly and/or walk. This evolution has been partly brought about by a misunderstanding of the original defined syndrome resulting in overdiagnosis coupled with the routine use of the term by laypersons, particularly on social media. The end result is a belief that LPS is currently in an epidemic form and is more common that previously described. Recent official documentation (WHA 2021) has also muddied the understanding of the condition by changing the traditional definition to include new clinical signs and exclude traditional identifying features such as clenched claws. The role of this paper is to describe a cryptic clinical process that is actually affecting a large proportion of these birds and is a common reason for presentation of birds labelled as LPS.

To understand the problem we must redefine the clinical components of the syndrome.

Classical LPS as has been defined historically involves birds that have: Unilateral or, more commonly bilateral, flexed hocks and clenched feet and are unable to perch, usually resting on their hocks. A head tilt, intention tremor or voice change may also be present. Typically there are no signs of trauma. The aetiology is not specifically known but may include non-suppurative encephalomyelitis, lead poisoning, thiamine deficiency, viral infections

Modern LPS definitions now include birds that may walk normally or with lateral or sternal recumbency, falling forward when attempting to move, evidence of beak trauma, bilateral legs in fixed extension with or without clenched feet. They are usually unable to fly but may attempt to fly but cannot gain height. Voice change may be present. In many cases, the aetiology can be determined to be associated with various spinal injuries and pelvic injuries whilst other cases may have no obvious causes. These have often been attributed to toxic, infectious or dietary causes and are the subject of current studies which have not yet revealed a satisfactory explanation.

It is the latter birds that I have been working towards finding a causative agent/condition as this group of birds represents a large proportion of our clinical case load. I have been particularly focussing on birds that show no palpable spinal fractures and no radiographic signs of skeletal damage.

WHA eWHIS Surveillance Data shows 594 entries for Rainbow lorikeets of which 129 (22%) were entered as LPS. Of these 129 birds , only 11% were described clinically as **classical LPS** with the remaining 89% being typically described as “ataxic, unable to fly, and not blinking properly. No abnormality on x-rays”.

Clinically, these birds may be bright and alert or may show various degrees of neurological changes consistent with concussive head trauma. In some birds, a distinct voice change or loss of voice may be observed. The beak often shows evidence of keratin fractures (pinpoint or linear) or haemorrhage. The legs and

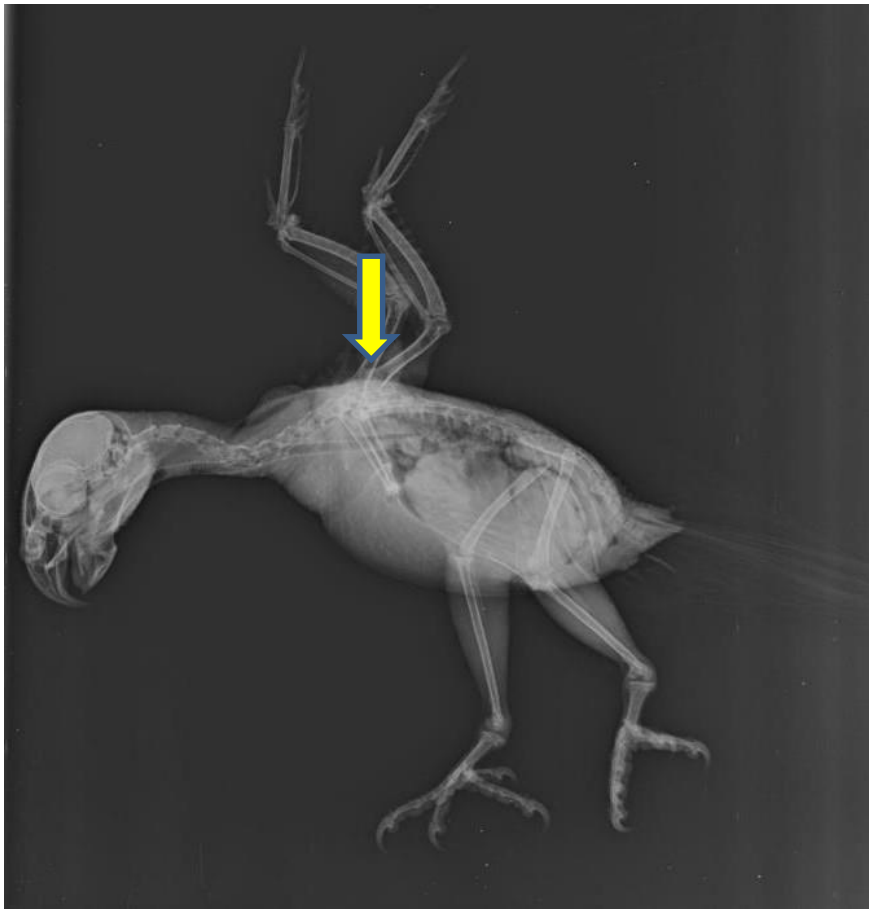
wings typically show no evidence of trauma and the birds show no typical signs of Psittacine Circovirus changes in the feathers. Birds of all ages may be affected. Radiographically, there are no obvious changes to be noted.

Based on clinical signs, it was assumed that the primary cause of the problem was likely to be related to head trauma but a large proportion of cases show no head trauma signs or improve dramatically in their neurological clinical signs but remain unable to use their wings . In all cases where leg function was normal at presentation, other than ataxic movements, leg function remained normal.

Understanding the basis of any syndrome usually requires the identification of a commonality between individuals with the same clinical picture.

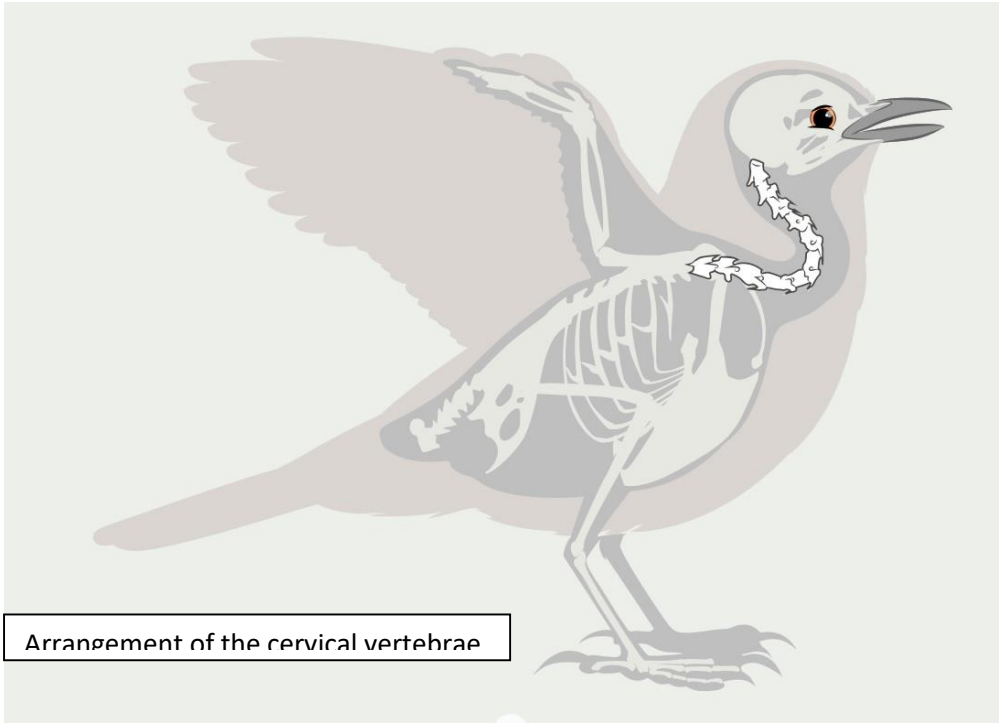
In early 2019, it was noticed that a characteristic palpable difference existed between normal non-affected birds and those fitting the above syndrome. On palpation of the cervical vertebrae of these birds when first anaesthetised and before manipulation of the body, a common change was noted in the vicinity of the most caudal cervical vertebra. The second last cervical vertebra, when the neck was extended ventrally, was ventrally displaced. This allows the sharp cranial edge of the last cervical vertebra to be easily felt. When the neck is extended dorsally whilst supporting the caudal cervical area, a distinct click is heard or obvious crepitus is felt as the second last cervical vertebrae shifts dorsally into its correct orientation. Following this, the sharp projection is then unable to be felt. The clinical perception is that what is being palpated is a ventral subluxation of the caudal cervical vertebrae secondary to head/neck trauma.

Radiographically, this area is always overlaid and obscured by the condyles of the humerus and proximal coracoids so abnormalities cannot be seen on plain radiographs.

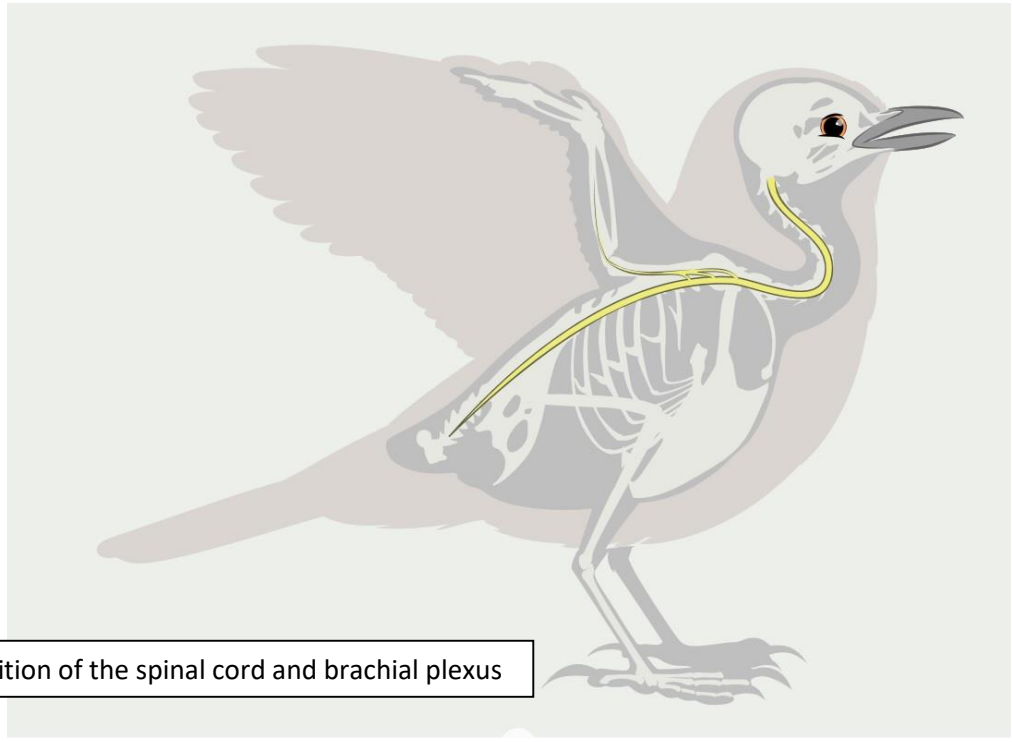


How does this potentially affect the ability to fly?

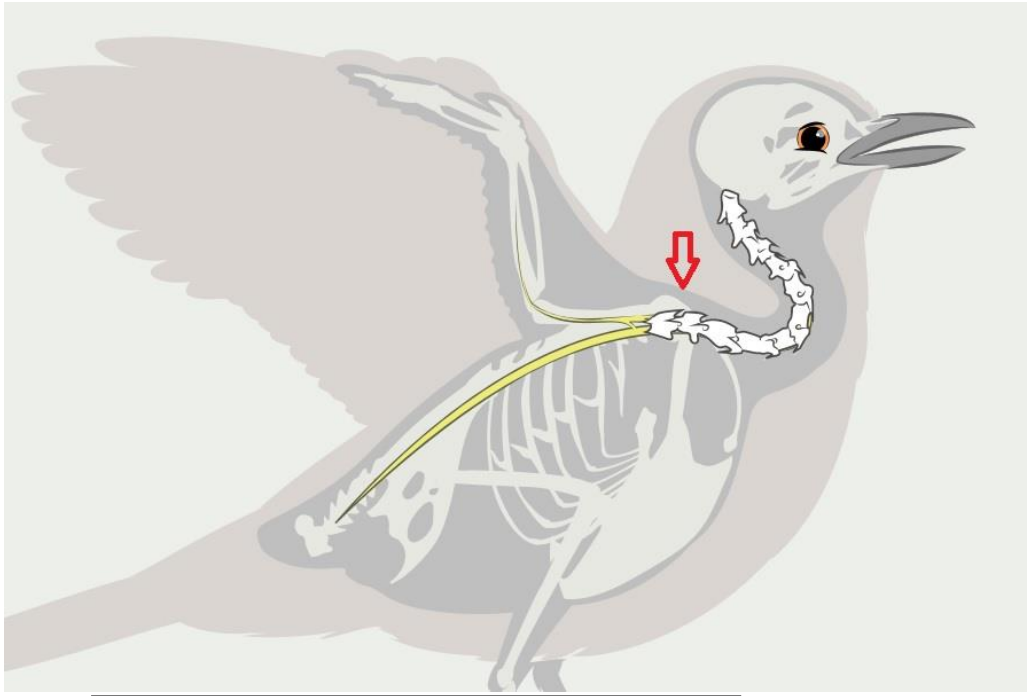
In the vicinity of the last two caudal cervical vertebrae is the emergence point of the brachial plexus (Cornell 2019). My theory was that what we are seeing in these birds is a simple brachial plexus crush injury brought about by a cervical vertebral luxation. This may be independent of or in association with other skeletal injuries.



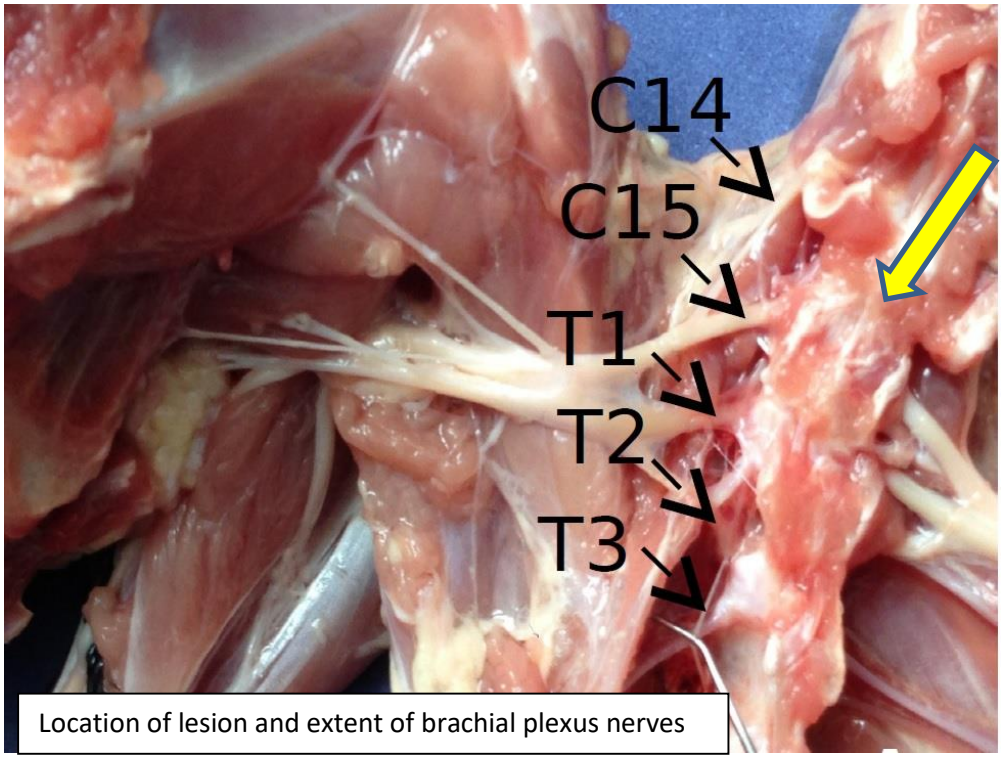
Arrangement of the cervical vertebrae



Position of the spinal cord and brachial plexus



Location of the lesion as seen in Rainbow lorikeets



Location of lesion and extent of brachial plexus nerves

The typical flight posture of the rainbow lorikeet is with the beak held slightly below the horizontal. This posture means that in comparison to other species, the rainbow lorikeet strikes objects at a peculiar and consistent angle quite capable of causing the injuries that we are seeing in these birds, particularly when striking at high speed.

If we examine the physical appearance of these birds we can see the gross changes described quite easily.

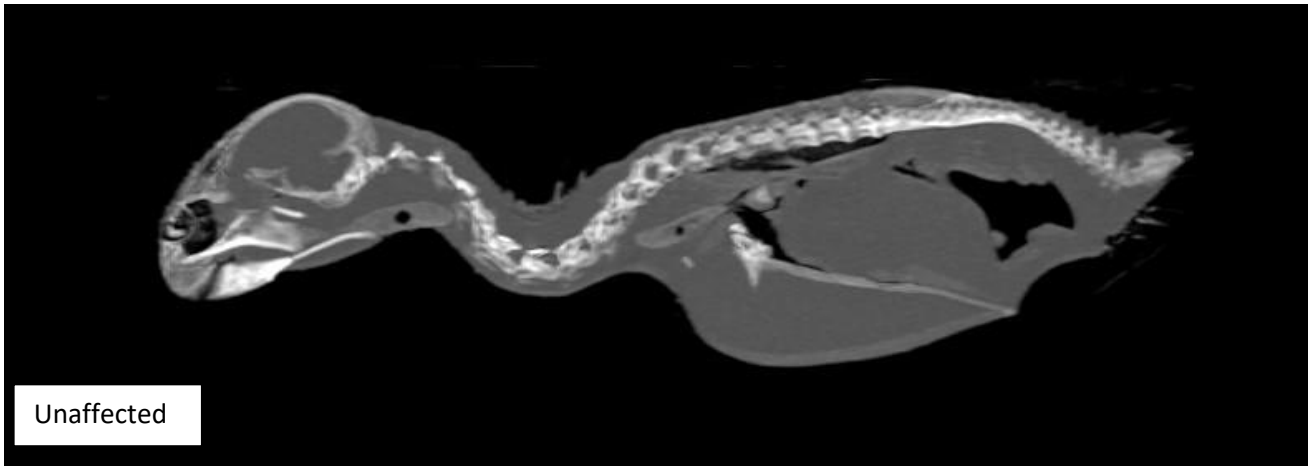
The sharp point of the cranial edge of the cervical vertebral body can be easily seen

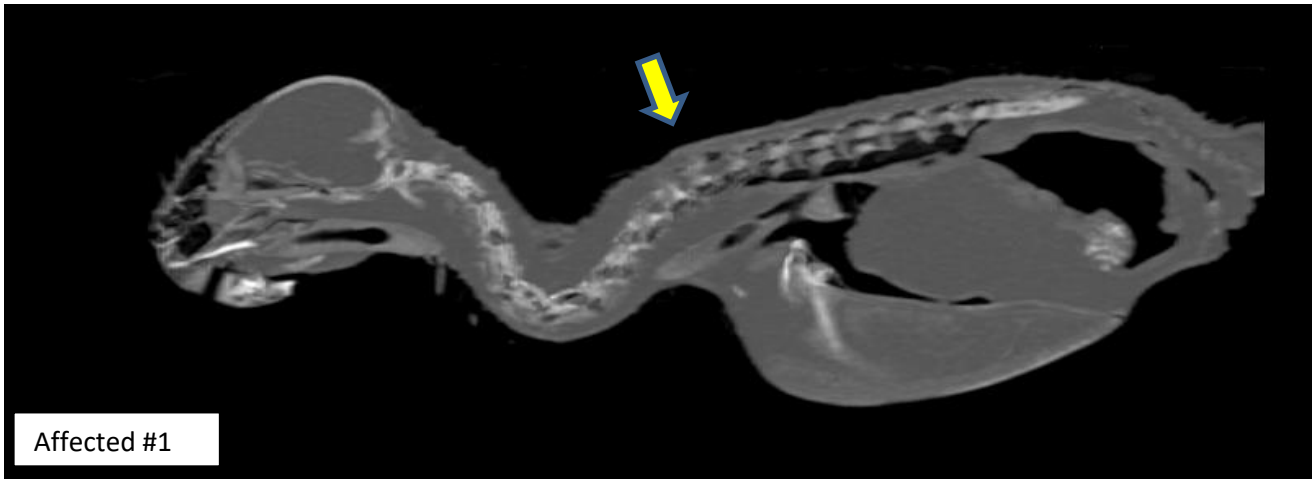
After adjustment, the point is no longer visible or palpable

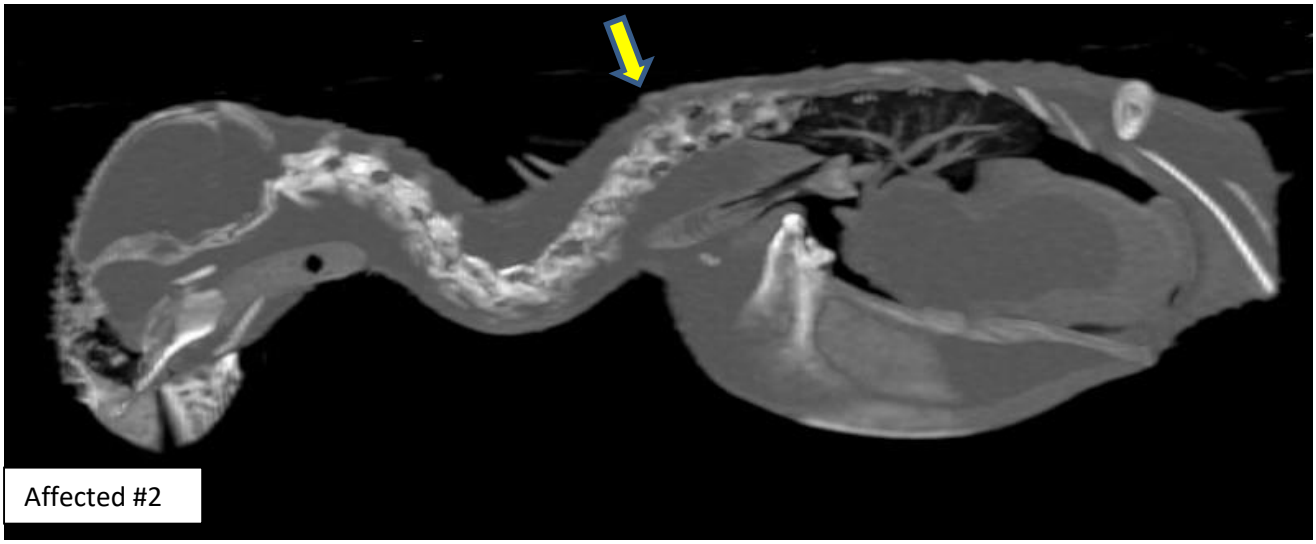
In addition to the palpable changes, an audible click is often evident as the luxation is corrected.

Changes in voice often accompany this injury and I believe this is due to additional damage to the hypoglossal nerve (cranial nerve XII) which innervates the syrinx. This nerve emerges from the brain via the hypoglossal foramen just lateral to the occipital condyle and accompanies the cervical nerves to the level of the thoracic vertebrae. It is not unreasonable to consider that this nerve may be damaged during the same process.

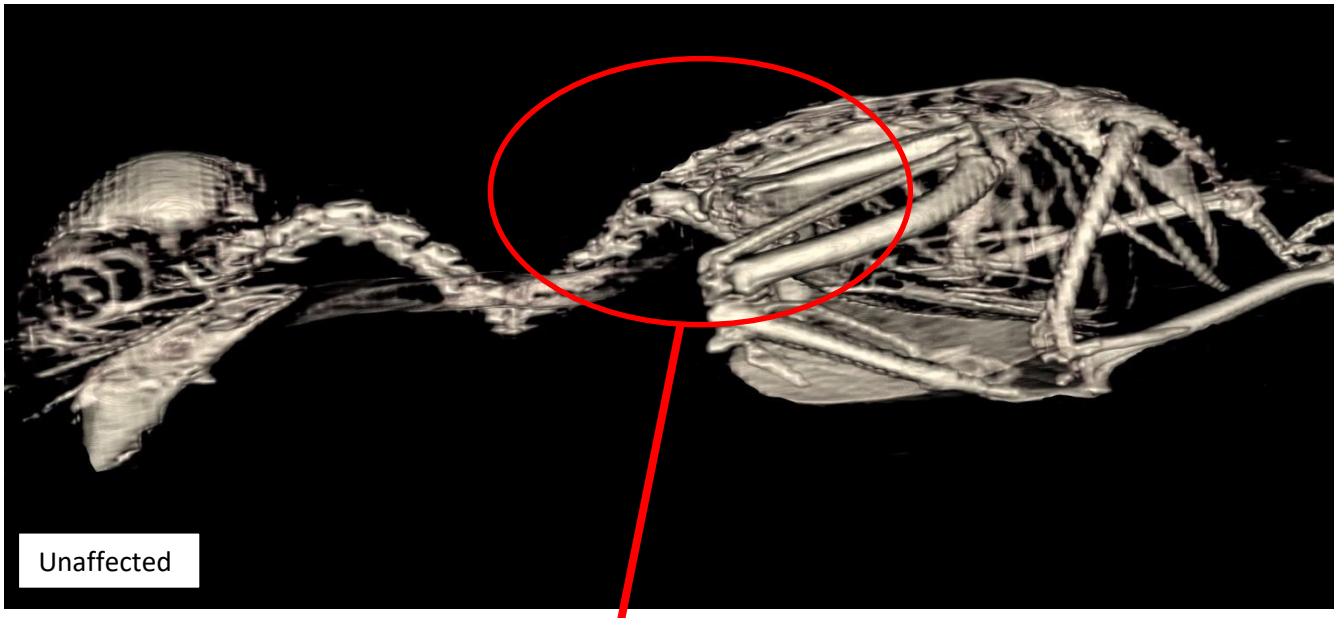
As mentioned previously, radiographically, the area of interest is obscured due to overlying structures. Examining this area for changes in bony alignment requires use of CT scans. This allowed a sagittal section through the cervical spine to be examined for any deviation to support my theory and 3D rendering to physically observe the changes present. The very small size of the specimens did limit considerably the achievable resolution of the CT scans but visible deviations in the spine at the expected site were visible on scans of all affected birds and absent from all control specimens

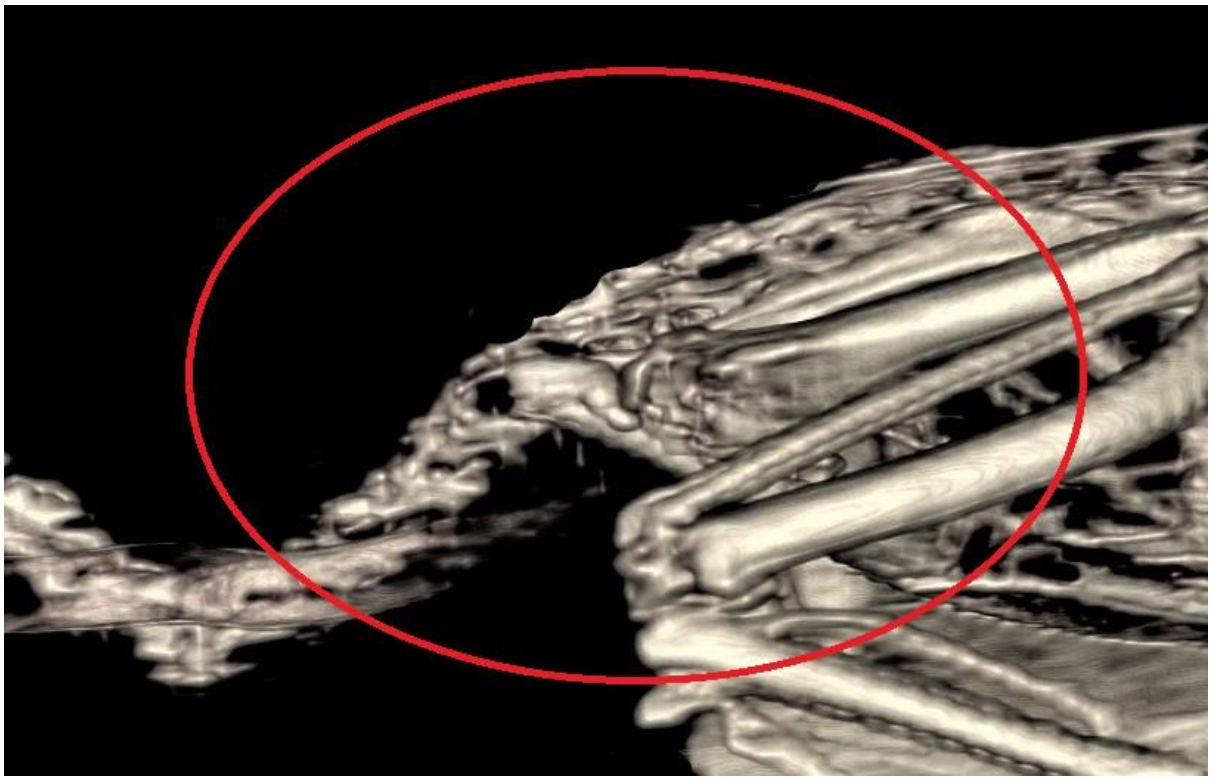

Unaffected

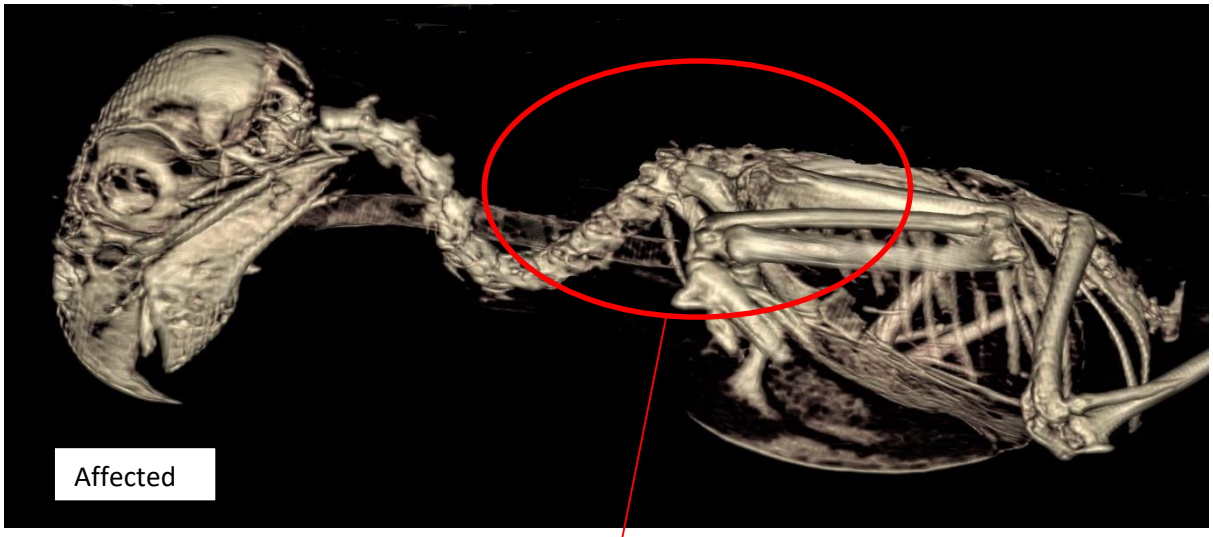

Affected #1

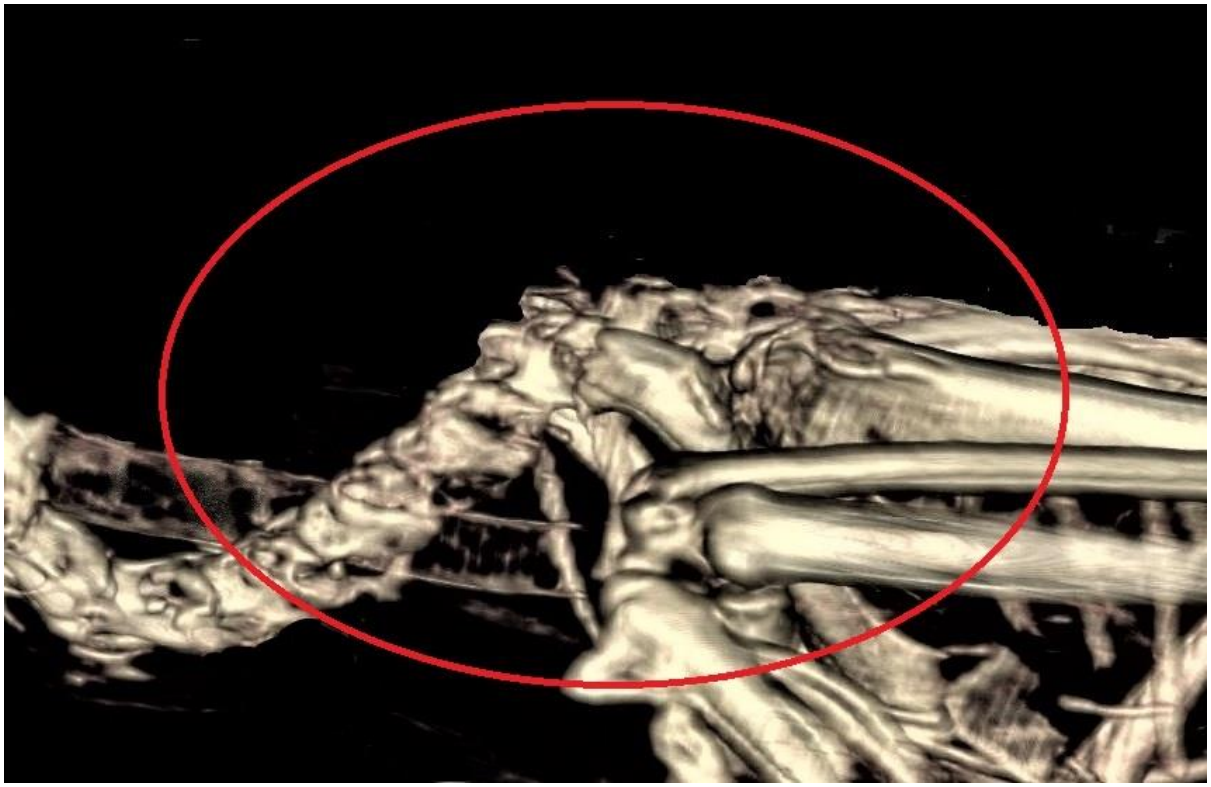


Three dimensional rendering gave much better resolution and allowed the visualisation of dramatic changes in spinal alignment in affected birds vs the control (unaffected) birds.






Affected



Clinical implications of Cervical Subluxation

Being able to specifically allocate a cause to a large proportion of cases of what was formerly referred to as LPS means that this term should be returned to describe those classical cases only as originally described.

In some cases, due to rough handling, secondary trauma or inadvertent manipulation during handling (particularly when palpating for caracoid fractures), it is possible for the cervical subluxation to be repositioned before clinical examination. These birds will behave the same clinically but diagnostic features are absent. These birds, though affected by the damage done to the brachial plexus, will be unrecognised as such. In our facility, I estimate that of those cases where other trauma can be ruled out, at least 70% of the remaining birds show clinical signs of cervical subluxation but as explained above this may be an underestimation if repositioning has occurred.

As with all nerve injuries, time is a factor. Birds that are presented with severe emaciation and cervical subluxation, suggesting injury that is not recent, are unlikely to ever recover as the crush injury has occurred over a considerable time period. Birds that are presented immediately after the injury may have a better prognostic outcome. I have noted however that the neurological damage in these birds becomes worse in the 24 hrs after correction of the luxation, even when on anti-inflammatory medication. It is unknown how long recovery may typically take as there are numerous factors to consider. Some facilities have recorded successful recover after several months of rest with initial anti-inflammatory treatment. A large proportion will never recover regardless of treatment regimes. It is unreasonable to expect birds to recover WITHOUT correction of the subluxation and removal of the compressive nerve injury.

Criteria for management will vary between facilities depending on their available space for recovering birds and the severity of the birds that are presented. Further study is needed to better categorise those birds with a likelihood of recovery.

Epidemics vs. Trauma Clusters

The primary reason why this collection of clinical signs has been aggressively pursued as being caused by an infectious or toxic process is that a large number of cases are frequently seen at the same time. This often results in a media fuelled misrepresentation of an 'epidemic' of a paralysis disease. We certainly do often see a large number of cases together throughout the year but clinically there is rarely any difference in what we see diagnostically. A genuine small percentage of these (<5%) are due to viral disease but the remainder fall into what I consider to be 'trauma clusters' . The commonest causes of trauma clusters in Rainbow lorikeets are :

- Local flowering plant activity. Mass planting of median strip and roadside flowering plants creates an opportunity for mass trauma incidents when these suddenly flower en masse resulting in increased number of birds crossing roads to access them
- Extended rain periods. Wet wings and weak hungry birds (due to nectar washout) results in slower and lower flying birds. Good samaritans are also less inclined to find roadside injured birds until after the rain has stopped, resulting in sudden presentation of large numbers 2-3 days after rain events.
- Flower intoxication. Mass flowering of species such as *Schlefferia* and *Schotia* during hot periods results in fermentation of nectar. The subsequent effect on the mental capacity and alertness of local nectar feeders results in increase trauma related accidents
- Increased vehicle traffic. Increased mortalities often occur associated with public holidays and long weekends due to increased vehicle presence on the road.
- Building improvements. The increased use of glass as a primary structural component of buildings and fencing (particularly pool fencing) increases impact related trauma around these structures.

When multiple causes combine such as increased traffic during flowering periods, a presentation of dozens of birds daily is not at all uncommon.

SUMMARY

- Lorikeet Paralysis Syndrome is NOT a specific disease.
- LPS in its classical form , typically with viral encephalomyelitis, occurs in < 5% of cases.
- Non-traumatic cases attributed to LPS (toxic, infectious, nutritional) account for roughly 20-30% of cases.
- Trauma is the primary cause of clinical presentation
- Cervical vertebral subluxation is present in a large proportion of trauma related cases but may be difficult to diagnose in a routine examination
- Specific examination of the caudal cervical vertebrae area under general anaesthetic reveals typical repeatable changes in the majority of cases.
- These changes cannot be demonstrated radiographically but are readily palpable
- Adjunct diagnostics such as CT scans show the presence of described changes in all affected birds and the absence of changes in non-affected birds (although this is not necessary for diagnosis).
- Cervical vertebral subluxation leads to a gradient of neurological clinical signs from isolated brachial plexus injury to severe spinal cord disease.
- Further work to determine criteria for treatment is required.
- Individuals with a body score <3/5 , sternal recumbency, ataxia are unlikely to recover regardless of treatment.
- Individuals with body scores >4, able to ambulate normally but unable to fly, with corrected subluxation, may be worth treating with anti-inflammatories. Return to flight is possible but this does not equate to releaseability as many still have weakened flight after apparent recovery.

Acknowledgements

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