



# **CORONERS COURT OF QUEENSLAND**

## **FINDINGS OF INVESTIGATION**

|                 |                                                                                                                                                                                                                                                       |
|-----------------|-------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| CITATION:       | <b>Non-inquest findings into the death of Connor Dean</b>                                                                                                                                                                                             |
| TITLE OF COURT: | Coroners Court                                                                                                                                                                                                                                        |
| JURISDICTION:   | BRISBANE                                                                                                                                                                                                                                              |
| DATE:           | 28 August 2026                                                                                                                                                                                                                                        |
| FILE NO(s):     | 2024/3919                                                                                                                                                                                                                                             |
| FINDINGS OF:    | Stephanie Gallagher, Deputy State Coroner                                                                                                                                                                                                             |
| CATCHWORDS:     | CORONERS: child with Trisomy 21(Down Syndrome), severe vitamin C deficiency, multivitamin taken by child, contents label of multivitamin did not list Vit C, cardiac arrest following general anaesthetic, likely underlying factor, Vit C deficiency |

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## **Introduction**

Connor Dean was born on 23 March 2018 and died on 10 August 2024 at Queensland Children's Hospital (QCH).

Connor Dean's death was reported to the Coroner, because the death appeared to be a health care related death and fell within the definition of a reportable death in the *Coroners Act 2003*.

The role of a Coroner is to investigate reportable deaths to establish, if possible, the cause of death and how the person died. The purpose of a coronial investigation is to establish the facts, not to cast blame or determine criminal or civil liability.

## **Identification**

Connor Dean was identified by those caring for him at QCH. That identification was accepted by me.

## **Circumstances**

Connor Dean was a 6 year old boy who had a history of Trisomy 21 (Down Syndrome). He was born with an atrioventricular septal defect which required complete surgical repair in June 2018. He had global developmental delay with slow developmental progress: communicate using mostly single words, follow one step commands, and walk and run with a wide based gait.

On 11 July 2024, he was reviewed by his general practitioner (GP) for a 3-week history of worsening symptoms. His parents reported an onset of knee pain and shuffling gait which progressed to limping, and eventual refusal to weight bear, commencing 3 days prior to review. His pain was managed with paracetamol before school and Nurofen prior to bed. Recent possible trauma included 'flung off a spinning playground', following which he had cried, and then mobilised/played as normal.

On assessment he was afebrile with no recent cold/flu like symptoms, and no bruising/swelling of knee/ankle joints with full range of motion and normal reflexes of the knee. No spinal tenderness. He appeared more distressed on hip movement (left greater than right). Urgent imaging was arranged.

On 16 July 2024, at follow up, his GP requested an urgent consultation at QCH advising of no change in his condition or examination, and no abnormality detected on x-rays of the pelvis, right & left knee & femur.

On 18 July 2024 he was admitted via the orthopaedic outpatients for further investigation including a brain and spine magnetic resonance imaging (MRI). His assessment revealed no spinal tenderness on palpation with range of motion preserved in lower limbs. Discomfort was noted with right knee rotation. Bloods showed a slightly elevated creatinine level (kidney dysfunction), normal full blood count and low inflammatory markers. X-rays of the cervical spine, ankles, and foot showed no fractures or bony abnormalities, and no appreciable soft tissue swelling. His mother advised he was taking levothyroxine and a multivitamin, and his immunisations were up to date.

Overnight he was noted to be coughing excessively by nursing staff with a respiratory swab later confirming Parainfluenza. The following day, he required oxygen therapy for low oxygen saturations and a fluid bolus for minimal oral intake and no urine output until 1900hrs. A subsequent bladder scan revealed 190mls with haematuria identified on urine dipstick.

On the morning of 20 July 2024, he remained nil by mouth in preparation for an MRI later that day under general anaesthetic (GA). He required ongoing supplemental oxygen with no

increased work of breathing or fevers identified. His kidney dysfunction and dehydration were improving despite ongoing minimal urine output. A positive urine cell count was noted (culture pending) with the plan to monitor before commencing antibiotics.

Later that day during the MRI, he required progressively increasing cardiovascular support and following the completion of imaging he rapidly deteriorated in recovery with a weak thready pulse and low cardiac output. Cardiopulmonary resuscitation was commenced, and he was transferred in active resuscitation to the paediatric intensive care unit with sustained return of circulation achieved at approximately at 8-10 minutes. The intensivist was of the opinion that there was a low flow state rather than asystolic state. He was transferred to the intensive care where a bedside echocardiogram (ECHO) showed reasonable ventricular function, right atrial regurgitation, and high pulmonary pressures. His pupils were equal but sluggish in reacting to light. A mobile chest x-ray showed diffuse airspace opacification with some sparing of the lung apices and large bilateral pleural effusions. The anaesthetist reported a grade 1 airway view on induction with reduced bilateral air entry.

The MRI showed non-specific multifocal bone marrow oedema (proximal humerus, around knee and most severe in the pelvis) with infection, infiltration, and metabolic causes such as scurvy within the considered differential. Other findings included bilateral cervical lymphadenopathy, very large bilateral pleural effusions and probable perihilar col/apse/consolidation - presumed infective. Loss of flow void was also identified in the right pulmonary artery which was not definitive but raised the possibility of pulmonary embolus.

He was commenced on broad spectrum intravenous antibiotics for likely multifocal septic shock, with likely multifocal osteomyelitis (no drainable collections). Consideration was also given to an early new leukaemia based on the MR findings. On the evening of 20 July 2024, he was reviewed by the haematologist who advised it was difficult to exclude leukaemia, and in the absence of cytopenias it would be unusual.

A bone marrow aspirate and trephine (BMAT) the following day showed preliminary results that were not suggestive of acute leukaemia. The haematologist considered the anaemia, thrombocytopenia and coagulopathy were likely secondary to bone marrow suppression in the context of cardiac arrest. The full report on 24 July 2024 showed a mildly hypocellular marrow with adequate trilineage haematopoiesis.

An ECHO on 21 July 2024 showed moderate left ventricular systolic dysfunction (LVEF 56%). His vitamin C level was noted to be <1 umol/L (reference range 20-120). He remained in a shocked state requiring triple inotropic support and additional blood products for coagulopathy.

On 23 July 2024, the cause of his deterioration remained unclear. His sedation was weaned with residual extensive posturing, sluggish pupils and bilateral lower limb clonus evident. Widespread petechiae were observed on all limbs and abdomen and he was becoming oliguric with a poor response to diuretics. The renal ultrasound showed features of renal disease evident consistent with sepsis and multiorgan dysfunction. Fluid from his pleural effusion on 23 July 2024 was mononuclear predominant (no growth after 5 days).

The brain and lower extremities MRI on 28 July 2024 showed severe global hypoxic ischaemic injury involving almost the entire cerebral white matter and the posterior cerebral cortex, globus pallidus and caudate nucleus. The metaphyseal abnormalities involving the lower limbs remained unchanged in keeping with scurvy.

Opinions were sought from various specialties. Orthopaedics did not consider the follow up imaging was conclusive for infection, with multiple other aetiologies more likely (e.g. scurvy). Rheumatology advised an autoimmune contribution was unlikely and that his post arrest profound multiorgan dysfunction could potentially be ischemic in origin and the bone marrow changes explained by musculoskeletal scurvy/Vitamin D deficiency which can contribute to coagulopathy features but would not explain the large pleural effusions.

A neurological opinion on 29 July 2024 for prognostication purposes advised that the MRI findings were consistent with severe global injury. These findings were discussed with the family including that severe life-threatening scurvy (Vitamin C deficiency) seemed a likely unifying diagnosis causing the initial presentation of bone pain and bone marrow oedema. The cardiorespiratory arrest may or may not have been related to the deficiency with a low cortisol level as one of the possibilities.

On 7 August 2024 he was compassionately extubated and died on 10 August 2024 at approximately 2203hrs. The Death Certificate issued by QCH said the cause of death was as follows:

- 1(a). Hypoxic ischaemic encephalopathy, due to
- 1(b). Cardiac arrest, due to
- 1(c). Vitamin C deficiency.
2. Down Syndrome.

### **Forensic Pathologist's Examination**

An external examination, and imaging, document review and toxicology studies, were undertaken.

The opinion of the forensic pathologist as to the cause of death is based on consideration of the circumstances of death and a post-mortem examination including associated imaging and testing.

The forensic pathologist summarised the findings on examination as follows:

Post mortem CT (Reported by Paediatric Radiologist)

1. Brain: Bilateral symmetrical calcification of the basal ganglia, and gyriform calcification of the cerebral hemispheres bilaterally. This is consistent with sequelae of prior hypoxic ischaemic injury.
2. Musculoskeletal system: There is subjectively reduced bone density. There are multiple recent fractures of the anterior aspects of the ribs at the costochondral Junctions bilaterally. While scurvy may result in fractures in this location, there are no specific radiographic features of scurvy.

External examination

1. Very small for age:
  - a. Height 104cm (<1st percentile). Weight 14.19kg (<3rd percentile).
  - b. Head circumference 46cm (<2nd percentile).
2. Features of Down syndrome.
3. Signs of recent medical treatment.
4. No bruises or other injuries.

The forensic pathologist commented:

While rare, there are reports of similar incidents in the medical literature in which children with Vitamin C deficiency/scurvy develop pulmonary hypertension +/- cardiac arrest in the context of sedation/anaesthesia.

The forensic pathologist concluded:

This child was extensively investigated in hospital with no definitive cause of the arrest determined. Reviewing the clinical history and medical literature, the favoured cause

of death is as per the attached certificate, which is in agreement with the clinical opinion.

Based on the Post Mortem CT report, it is also possible that some of his developmental delay was due to a previous hypoxic- ischaemic episode.

In the opinion of the forensic pathologist, the cause of death was:

- 1(a). Hypoxic ischaemic encephalopathy
- 1(b). Cardiac arrest (post general anaesthetic)
- 1(c). Vitamin C deficiency
2. Down syndrome.

### **Statement of Connor Dean's Paediatrician**

Connor Dean's paediatrician had approximately 20 years' experience as a paediatrician, when treating Connor Dean.

The paediatrician said:

I have been Connor's paediatrician since he was first admitted to QCH PICU on 10/05/2018.

I, along with the dietetic staff, have had long standing concerns regarding Connor's growth with his weight and height well below the 3rd centile on the standard growth chart for boys. These measurements are below the 5th centile for height and weight on the Trisomy 21 growth chart. Connor was partially fed by nasogastric tube until February 2020. His mother reported to me that Connor ate a wide variety of foods including meat, fruit and vegetables up until and including his June 2022 review

I became concerned with Connor's diet during my review on 10 May 2023. He was reported to have become very fussy with his intake and his diet was said to contain no fruit or vegetables at that time. He had only gained 300 grams in the last 11 months (expect 1.5-2kg). I told his mother that I was very concerned about Connor's diet, specifically that it did not contain enough calories or the vitamins and minerals he needed to grow and be healthy. I referred Connor to the dietician and requested his parents commence a multivitamin. I recommended 'Pentavite with iron' at the standard dose for his age (my usual practice is to show a photo of this from Google, available as a liquid or as blackcurrant gummies). I also organised a blood test for nutrition screening and Connor was found to have iron deficiency, Vitamin B12 deficiency and Vitamin A deficiency. Additionally, his Vitamin D level was borderline low. I emailed Connor's mother with these results requesting she start Iron supplements in addition to the multivitamin, plus get a Vitamin B12 injection at the GP.

Connor was admitted to QCH on 18 September 2023 with a febrile illness. Group A Streptococcus grew on a throat swab. His nutritional screen was repeated and his Vitamin B12 had normalised. However, he was still iron, Vitamin A and now Vitamin D deficient. Vitamin E was checked and normal. He was coagulopathic and this was assumed to be due to Vitamin K deficiency which was treated with a phytomenadione injection. The admission notes do not document whether any iron or multivitamin was being taken. I did not see Connor during this illness.

I saw Connor again in outpatients on 27 November 2023. His diet was still very poor with lots of white bread, some beef sausage, custard and chocolate. He was being offered vegetables at day care but I don't believe he was eating them. He was drinking apple and blackcurrant 100% juice poppers which would contain Vitamin C. He was

taking a ... multivitamin and I checked that it contained Vitamin A, D, B12 and iron to cover his known deficiencies. It also had small amounts of other vitamins including Vitamin C. I don't recall which ... product he was taking in November 2023 ... It was not (the Product) that he was taking in July 2024 as I did not know of this Product's existence at this time. Given Connor's Vitamin D had not improved on the multivitamin I started him on Ostelin Vitamin D3 liquid in addition to the multivitamin. I restarted Connor on Ferroliquid. I also organised for a Vitamin A injection. I discussed with Connor's mother that Connor's diet was inadequate to meet his vitamin and mineral requirements but that I believed that we were covering these requirements with the multivitamin, Vitamin D3, Ferroliquid and Vitamin A injection. I encouraged Connor's mother to give Connor one of the 100% juice poppers every day.

A follow up blood test was collected on 25/01/2024 which showed that Connor's Iron, Vitamin B12 and Vitamin D had normalised. His coagulation was normal. His Vitamin A levels had improved but were still low and this was treated with another Vitamin A injection.

Connor was admitted to QCH on 18/03/2024 with mycoplasma pneumonia. The admission notes document that Connor was taking a multivitamin at this time. There were no micronutrient levels checked during this admission. He was reviewed by the dietitian during this admission. I did not see Connor during this admission.

Connor was admitted to QCH on 18/07/2024 after attending orthopaedic clinic with difficulty weight bearing. MRI of the skeleton showed multifocal bone marrow oedema consistent with scurvy. His Vitamin C level was <1 (20-120). He was also deficient in Vitamin A and Vitamin E. His Vitamin D level was borderline low. He was coagulopathic suggesting Vitamin K deficiency. His Vitamin B12 and folate levels were normal. His Ferritin was not checked prior to his cardiac arrest however his haemoglobin and MCV were normal, suggesting normal iron status. On reviewing Connor's multivitamin during his PICU admission I became aware that the product he was taking was (the Product). This Product does not contain any Vitamin C, Vitamin K or Vitamin E. At the recommended dose it provides only half of the recommended daily intake of Vitamin A.

I have had significant concerns regarding Connor's growth and nutritional deficiencies for a long time...

I am unsure as to the family's compliance with the multivitamin however in PICU his mother told me they had been giving it every day.

Dietitian reviews occurred on 23/07/2018, 5/11/2018, 26/11/2018, 17/12/2018, 07/01/2019, 04/03/2019, 10/06/2019, 01/07/2019, 23/09/2019, 27/12/2019, 20/05/2020, 10/8/2020, 04/01/2021, 24/01/2022, 06/06/2023, 04/09/2023, 11/03/2024, 21/03/2024 and 18/07/2024.

Reviews were also scheduled on 12/02/2024 and 24/06/2024 but are noted in the record as Failure to Attend (FTA). I have taken these dates from ieMR as relevant information as they attest to the ongoing concern by the dietetic staff regarding Connor's diet. They also illustrate the significant dietetic expertise provided to Connor's family. I was not involved in these reviews.

I am concerned that a product such as (the Product) is available in Australia I believe that it is misleading to call this product a multivitamin when it misses essential vitamins such as Vitamin C. Additionally, the absorption of non-haem iron is greatly enhanced by Vitamin C so it is illogical to have an iron containing product in combination with multiple vitamins but not Vitamin C. It is my clinical opinion that Connor would not have had Vitamin C deficiency, and the clinical features of scurvy that lead to his death, if he had been taking a different multivitamin that contained Vitamin C.

## **Statements of Connor Dean's parents in relation to Vitamin C deficiency**

Connor Dean's parents confirmed that he had become a very fussy eater at about 4.5 to 5 years of age, he had consulted a dietician, had a Vitamin A injection and that they gave him the multivitamin (referred to above) and iron each day and then changed the multivitamin to (the Product).

Each said that were not aware he had a Vitamin C deficiency until after his arrest.

## **Response of the manufacturer of the multivitamin provided to Connor Dean by his parents (the Product as referred to above).**

The manufacturer of the Product taken by Conner Dean said, in part:

- First and foremost, we extend our sincere condolences to Connor's family.
- (The company) takes the safety and wellbeing of all consumers — and in particular children — with the utmost seriousness.

In response to the statement of the paediatrician, it advised:

The treating paediatrician's opinion that "it is misleading to call this product a multivitamin when it misses essential vitamins such as Vitamin C". With respect, this is incorrect.

"Multivitamin" is well understood to mean multiple nutrients are included in a dietary supplement product. Multivitamin products do not generally contain all essential vitamins and nor are they indicated for treatment of a severe deficiency, they are indicated to help supplement when dietary intake is inadequate.

The active ingredients in the Product and their quantities are clearly listed on the back of the Product as required by the Therapeutic Goods Administration. Vitamin C is not included in that list. While Vitamin C may assist with iron absorption, it is not necessary for iron absorption. There is otherwise no statement on the Product that would lead to the conclusion that the Product contains Vitamin C, nor can any such implication be inferred from the packaging which complies with all relevant laws and regulations.

The fact that the Product does not contain Vitamin C is not itself a basis to conclude that the Product cannot be called a multivitamin. Further the simple fact that some multivitamins contain Vitamin C, albeit in varying doses is not a basis to conclude that all multivitamins must contain Vitamin C.

(The company) rejected the treating paediatrician's opinion that "Connor would not have had Vitamin C deficiency, and the clinical features of scurvy that lead to his death, if he had been taking a different multivitamin that contained Vitamin C" as:

- a. Connor's paediatrician had long-standing concerns regarding his growth and nutritional deficiencies, noting that by May 2023 his diet contained no fruit or vegetables, and that he had only gained 300 grams in 11 months. As early as September 2023, Connor was found to be iron, Vitamin A, and Vitamin D deficient. The report states that Connor's parents "said that they were not aware he had a Vitamin C deficiency until after his arrest." Unfortunately, vitamin C deficiency does not therefore appear to have been a focal point of Connor's clinical plan. Had Connor's Vitamin C deficiency been a focus in his management, then it is reasonable to assume that a different treatment plan would have been adopted that specifically treated that deficiency.
- b. Multiple other contributing factors also appear to be present. Treating clinicians acknowledged that the cardiorespiratory arrest "may or may not have been related to the deficiency." Connor had a history of Trisomy 21 (Down



Syndrome), had been born with an atrioventricular septal defect requiring complete surgical repair, and had global developmental delay. MRI findings included non-specific multifocal bone marrow oedema with infection, infiltration, and metabolic causes, including scurvy, within the considered differential, as well as bilateral cervical lymphadenopathy, large bilateral pleural effusions, and possible pulmonary embolus.

- c. Given this uncertainty and the apparent lack of monitoring of Connor's Vitamin C deficiency prior to his arrest, there is no basis on which to draw the conclusion that the Product has contributed to Connor's death. Further, any such uncertainty should not be resolved by the adoption of a single clinical opinion by a non-independent clinician in the circumstances.

## Conclusions

Having considered all of the material obtained during the investigation, including the clinical records, statements, and medical opinion, I am satisfied that the available evidence is sufficient to make the necessary findings in relation to Connor Dean's death. The evidence establishes that Connor Dean had severe vitamin C deficiency, with associated clinical and radiological features consistent with scurvy, and that he suffered a cardiac arrest following the administration of a general anaesthetic on 20 July 2024. Although the precise mechanism leading to the cardiac arrest could not be definitively determined, the treating clinicians and forensic pathologist considered vitamin C deficiency to be the most likely underlying factor.

I accept the forensic pathologist's opinion as to the cause of death and find that Connor Dean died on 10 August 2024 from hypoxic ischaemic encephalopathy due to cardiac arrest following general anaesthetic, in the setting of vitamin C deficiency, with Down syndrome being a contributing condition.

Having regard to the matters set out in the Coroners Act 2003, I am satisfied that the circumstances of Connor's death have been adequately investigated and that no further coronial inquiry, including an inquest, is required. I am of the view that, publishing these findings may be sufficient mechanism to draw attention to this death so as to prevent deaths in similar circumstances happening in the future.

I extend my condolences to Connor's family and loved ones for their loss.

## Findings required by s.45

|                                   |                                                                                                                                                                                                                                                                                                                                                                                                                                                                  |
|-----------------------------------|------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| <b>Identity of the deceased –</b> | Connor Dean                                                                                                                                                                                                                                                                                                                                                                                                                                                      |
| <b>How he died –</b>              | Connor Dean had severe vitamin C deficiency, with associated clinical and radiological features consistent with scurvy, and that he suffered a cardiac arrest following the administration of a general anaesthetic on 20 July 2024. Although the precise mechanism leading to the cardiac arrest could not be definitively determined, the treating clinicians and forensic pathologist considered vitamin C deficiency to be the most likely underlying factor |
| <b>Place of death –</b>           | Queensland Children's Hospital SOUTH BRISBANE QLD 4101 AUSTRALIA                                                                                                                                                                                                                                                                                                                                                                                                 |
| <b>Date of death –</b>            | 10/08/2024                                                                                                                                                                                                                                                                                                                                                                                                                                                       |

**Cause of death –**

- 1(a). Hypoxic-ischaemic encephalopathy
- 1(b). Cardiac arrest (post general anaesthetic)
- 1(c). Vitamin C deficiency
- 2. Down syndrome

I close the investigation.



Stephanie Gallagher  
Deputy State Coroner  
CORONERS COURT OF QUEENSLAND - BRISBANE OFFICE  
28 August 2026