Coroners Act, 1996
[Section 26(1)]

Western *Australia*

RECORD OF INVESTIGATION INTO DEATH

Ref No: 47/2014

I, Evelyn Felicia Vicker, Deputy State Coroner, having investigated the death of ***Christopher Ling TAO****, with an Inquest held at Perth Coroners Court, Court 58 Central Law Courts, 501 Hay Street, Perth on 12-16 December 2014 find the identity of the deceased child was* ***Christopher Ling TAO*** *and that death occurred on 10 September 2010 at Princess Margaret Hospital Emergency Department as a result of Myocarditis in the following circumstances -*

Counsel Appearing :

Kate Ellson assisted the Deputy State Coroner
Carolyn Thatcher and Nicholas Damnjanovic (State Solicitors Office) appeared on behalf of Child and Adolescent Health Services (CAHS)
Geoff Bourhill (instructed by MDA Insurance) appeared on behalf Dr Lovegrove
Dominic Bourke and **Anita de Villiers** (Clayton Utz) appeared on behalf of Dr Tan

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INTRODUCTION

The deceased child, Christopher Ling Tao (Christopher), first showed signs of being unwell very early on 3 September 2010. By late 4 September 2010 he was so unwell his parents took him to Princess Margaret Hospital (PMH). He was assessed and discharged home with antibiotics, pain relief and a need for increased fluids.

On Monday 6 September 2010 Christopher was taken to his family General Practitioner (GP) who asked he no longer be managed with the antibiotics and Nurofen. He had developed a rash which the GP believed may relate to allergies.

On 7 September 2010 Christopher remained unwell and was returned to PMH. He was again assessed and discharged home with a diagnosis of a viral illness. He remained unwell, but on review by his GP on 8 September 2010 his parents were assured he would improve, and a follow-up appointment was made for 10 September 2010.

Christopher remained unwell and late on 9 September 2010 his father found him unresponsive in bed. Resuscitation was commenced and an ambulance called. Christopher was transferred to PMH Emergency Department but could not be resuscitated and died early on 10 September 2010.

Christopher was two years and nine months of age.

BACKGROUND

Christopher was born on 3 December 2007 in Newcastle, New South Wales. Christopher's mother was unwell at the time of his birth and they both developed a fever. However, both Christopher and his mother recovered and Christopher was a healthy child, suffering nothing more than normal childhood illnesses.

Christopher's parents had worked as health practitioners in China before immigrating to Australia. His mother is a nurse at Royal Perth Hospital, while his father is now a podiatrist, having changed his career since leaving China, where he was an orthopaedic surgeon.

The family moved to Perth in February 2009 and Christopher remained a happy, healthy, alert toddler. His immunisations were all up to date.

He attended day-care for three days a week and his parents arranged their commitments so there was always at least one parent with him on the days he was not at child care. In September 2010 Mr Tao was sitting for exams and his mother stayed with the family to help with Christopher.

THURSDAY 2 SEPTEMBER 2010

On Thursday 2 September 2010 Christopher's day-care centre had a party in preparation for Father's Day. Christopher attended and there were no concerns or reported difficulties with his health. His father collected him at 4pm that afternoon, but by the time his mother arrived home from work at 6pm, Christopher was asleep on the floor which was unusual. He woke when his mother came home but remained looking distressed and tired. Christopher usually went to bed between 9 and 9:30pm and prior to going to bed Christopher had not eaten his dinner.

At approximately midnight Christopher woke up, crying. He had a high fever with a temperature of 38.7^{0}C.[1] His parents assumed he was suffering from a cold so they gave him some water and Panadol and attempted to cool him with a flannel.

FRIDAY 3 SEPTEMBER 2010

Christopher remained unsettled and distressed for the remainder of the night and in the morning still had a temperature which his parents continued to treat with Panadol. Christopher was not sent to day-care that Friday and his grandmother looked after him at home due to his illness. Christopher did not eat and was very tired and slept whenever he could. He woke up frequently complaining of

[1] t 15.12.14, p.5

pain. His temperature dropped to about 38^{0}C for a short while but Christopher remained quiet, refusing all food, milk or water. This was unusual because Christopher liked and drank a lot of milk. His parents are not sure whether he vomited or had diarrhoea on the Friday.

SATURDAY 4 SEPTEMBER 2010

On the Saturday Christopher's temperature remained high at 39.5^{0}C and he continued to refuse food and water. Each time his parents attempted to persuade him to eat food he vomited. Christopher's parents were concerned because he now had a high fever for over 48 hours, and he was continuing to complain of pain which they believed originated in the back of his head due to his indicating that was the source of his pain. Panadol was not effective in reducing his temperature significantly. Christopher's parents decided to take him to PMH.

At PMH Christopher was triaged at 8:19pm by Registered Nurse (RN) Lindsay Abbot. RN Abbot took a brief history from Christopher's parents and made a visual assessment that Christopher was *"alert, miserable, mouth moist, dry lips"* and was complaining of a sore throat.[2] She gave him an Australasian Triage System (ATS) score of 4 which required he be seen within approximately one hour for medical assessment.

[2] Ex 1, tab 12, tab 29, p.10014

Christopher was assessed at 8:45pm by Clinical Nurse Amber Louise Scott. CN Scott noted Christopher's observations were elevated with a temperature of 38.6^{0}C, pulse 157, respirations 32, oxygen saturation 95%. CN Scott asked Christopher's parents to remove some of his clothing as she believed he was hot and that removing some of his layers of clothing would help him keep cool.[3]

Following that assessment Christopher was seen by RN Trainer who took another set of observations for Christopher at 10pm. His temperature had decreased to 36.8^{0}C and was considered to be in the normal range. His respiratory rate and oxygen saturation had also returned to normal. RN Trainer believed the doctor may wish to take blood samples because of a lump on his neck Christopher's parents had noticed, so she applied an anaesthetic cream to Christopher's arm[4].

Christopher was then seen by Dr Alicia Ai Wei Lim, a Paediatric Registrar in the Emergency Department. According to the Hospital Progress Notes Dr Lim saw Christopher at 10:12pm. Dr Lim noted that on presentation Christopher's temperature, pulse and respiration rates had all been elevated, however, by the time she saw him, his temperature and other observations had returned to within

[3] Ex 1, tab 9, tab 29, p.10014
[4] Ex 1, tab 10

a normal range. Dr Lim checked Christopher had not been in contact with any other unwell people.

On her examination Dr Lim found Christopher to be alert, well and not septic looking. She did not consider him to be unwell or lethargic and his extremities were normal. Dr Lim found Christopher had an inflamed left ear drum, his tonsils were infected but his chest was clear.[5] His heart sounds were normal and she had no specific concerns about his presentation.

Dr Lim examined his left neck and noticed a swelling of two to three large lymph glands, firm to touch. They were tender but the overlying skin was not red. Dr Lim recorded Christopher as suffering from cervical lymphadenitis and tonsillitis. She prescribed pain relief by way of Nurofen, fluids and Cephalexin (antibiotics). This was because she believed the ear infection and tonsillitis were bacterial infections. Dr Lim asked about Christopher's fluid intake and on being told it was reduced she told Christopher he needed to drink more water. In response to this Christopher drank more water than his mother had seen him drink all day.[6]

[5] Ex 1, tab 29, 010015
[6] t. 15.12.14 p.48

Dr Lim advised Christopher's parents he should be returned to PMH Emergency Department if there was any increase in his swelling or they had other concerns.[7]

Christopher's parents asked Dr Lim about a blood test but she advised she did not believe Christopher was sick enough for blood tests to be warranted but did advise them to give Christopher Hydralyte to rehydrate him.

Christopher and his parents returned home.

SUNDAY 5 SEPTEMBER 2010

On Sunday 5 September 2010 Christopher had again deteriorated with his temperature remaining over 39^{0}C. By late on the afternoon of Sunday his parents noticed he had developed a rash on his face, and on his body, especially around the vicinity of his nappy. He was refusing all food, would not drink the Hydralyte, milk or water and slept very little, complaining all the time that he was in a lot of pain. Every time his parents persuaded him to drink he vomited, and he continued to have diarrhoea.[8]

MONDAY 6 SEPTEMBER 2010

Christopher remained unwell and his parents became increasingly concerned. On this occasion they took him to

[7] Ex 1, tab 17
[8] t 15.12.14, p.12-13

see their family GP, Dr Chin Huat Tan, and advised Dr Tan of Christopher's symptoms and continuing high temperature, with the addition of the rash.

Dr Tan had received a discharge letter from PMH advising Christopher had been diagnosed with cervical lymphadenitis, with additional diagnoses of "Ent and Mouth Tonsillitis".[9]

Dr Tan examined Christopher's rash which he observed on Christopher's face and eye lids with a generalised rash on his body. Dr Tan listened to Christopher's chest through his clothes and did not notice any difficulties with his heart sounds. His ear drums were red and Christopher was recorded by Dr Tan as being *"grizzly but alert"*. Dr Tan was not particularly concerned about Christopher's condition but did consider that he may be allergic to the antibiotics or the Nurofen. Dr Tan had been advised by Christopher's parents this was the first occasion upon which he had Nurofen. Dr Tan advised Ms Ling to take Christopher off all medication, keep him hydrated, and to return to providing him with Panadol and Phenergan[10].

Christopher and his parents returned home, but Christopher did not improve and he started suffering noticeably from discoloured diarrhoea.

[9] Ex 1, tab19
[10] Ex 1, tab 19, t. 15.12.14, p.52

TUESDAY 7 SEPTEMBER 2010

By Tuesday 7 September 2010 Christopher's parents became extremely worried about his presentation. His temperature remained high at over 38^{0}C and had now been elevated for five days. They noted that he had dried cracked lips and returned with him to PMH.

Again there was a long wait for assessment.

Christopher was triaged by RN Samantha Tubb at 12:20pm. He was given an ATS of 3 which required him to be seen within 30 minutes. RN Tubb recorded Christopher as having a swelling to the left side of his neck, with a fever, and that this was a re-presentation from the Saturday. He had been seen by his GP on the previous day with a rash. RN Tubb recorded Christopher as alert, now suffering from vomiting and diarrhoea, with a coated tongue, cracked lips and an occasional cough[11].

From triage Christopher was seen by RN Diane Cropper at 12:50pm in the Emergency Department. RN Cropper recorded Christopher's temperature as 39.5^{0}C, pulse at 160bpm, a respiratory rate of 44 and oxygen saturations of 97%. His weight was 14.8kg, down 0.2kg since the weighing on Saturday 4 September 2010.

[11] Ex 1, tab 11

RN Cropper noted his temperature, pulse and respiratory rate were elevated, confirming his febrile illness and need for medical review. RN Cropper took a history from the previous presentation and recorded that Christopher had developed a rash, visited his GP and been advised to cease the antibiotics and Nurofen. RN Cropper was told Christopher was eating and drinking very little, but would also vomit. She recorded he had a slight cough. She noted him to be *"alert, miserable, lethargic, with dry cracked lips, a widespread blanching rash and occasional cough"*. Again RN Cropper applied anaesthetic cream to Christopher's arm in case the medical review indicated blood tests should be taken. Christopher was given Panadol at 1.10pm and later RN Cropper took a throat swab.

Christopher was then reviewed by the paediatric registrar, Dr Nerissa Pecache. She first saw Christopher at 12:57pm.

Dr Pecache trained as a paediatrician in the Philippines and came to Australia in 1996. She is accredited in Australia and moved to Perth in 2000. She had been at PMH for 10 years on 7 September 2010.

Dr Pecache advised it was her practice, when reviewing patients, to write up their notes at the conclusion of her review while it was fresh in her mind. She could not recall very much about Christopher, but was confident her notes had been written at the time and would have followed her

normal processes. Dr Pecache said she would have reviewed the notes relating to Christopher's earlier presentation on 4 September 2010 and noted Dr Lim believed Christopher had a sore throat with fever and had vomited. He had been diagnosed with tonsillitis and provided with antibiotics and Nurofen as a painkiller.

Dr Pecache could see from the triage history Christopher had then developed a rash on his palms, trunk and lower extremities, and at the suggestion of his GP, had stopped taking the antibiotics and Nurofen. Dr Pecache noted a viral infection could also produce the rash that Christopher had developed.

Mr Tao advised Dr Pecache Christopher had now been suffering from his high fever and sore throat for five days. Dr Pecache noted Christopher was miserable, had low fluid intake, vomited whenever he drank and had a loose stool on 7 September 2010. Dr Pecache did not consider Christopher to be lethargic or sleepy, she observed him as alert. Dr Pecache noted Christopher had been encouraged to drink by his father, but because there was no note by the nurses he had vomited, she did not believe he had vomited while in emergency.

On examination Dr Pecache noted Christopher still had a temperature of 39.5^{0}C, that he was tachycardic with a pulse of 160bpm, an elevated respiration rate of 44, but without

signs of respiratory distress. His oxygen saturation had been recorded as 97%. Dr Pecache assessed Christopher as mildly dehydrated, but she did notice his lips were peeling and his skin was dry. Dr Pecache noticed Christopher had conjunctivitis, redness of the eyes without discharge or infection, that he had a generalised rash on his face and a white coated tongue. She also noted a spot of exudate on Christopher's left tonsil. Her attention was drawn to his inflamed lymph nodes with a large inflammation on his left side (2 x 1cm) and a more generalised swelling on his right side (2 x 0.5cm). Dr Pecache listened to Christopher's heart sounds which she found to be normal, with clear dual sounds and no murmur. Christopher's abdomen was normal and she noted his rash was on both palms and in between his toes.

In her statement, and in court, Dr Pecache advised she considered whether Christopher was suffering from Kawasaki Disease (KD). There is no mention in the notes anyone had considered KD as a differential diagnosis.[12]

Dr Pecache stated she had comparatively good experience with KD in the Philippines, and had written a research paper on a small cohort she had studied.[13]

Dr Pecache consulted with the paediatric consultant on the emergency ward at the time, Dr Michael Lovegrove. It was

[12] t 12.12.14, p.22
[13] t. 12.12.14, p.11-12

the practice in PMH for any re-presentation to be checked by a senior doctor during the course of the consultation[14].

Dr Pecache said her reason for considering KD was the length of time Christopher had been suffering from a high fever. Her reasons for discounting the diagnosis of KD were because some of the clinical signs were, in her view, not present. KD is a clinical diagnosis only, it cannot be positively confirmed by way of any tests or investigations. Dr Pecache said she did not consider Christopher's skin to be *"peeling"* which countered against KD and she could not see any rash in his peri-anal area. She did not believe the spot on Christopher's tonsil supported a diagnosis of KD over tonsillitis.[15]

Dr Lovegrove also examined Christopher and in evidence stated he rejected KD in Christopher's case because Christopher did not present as severely unwell. In his view Christopher was more likely suffering a viral illness. There were many similar presentations in the emergency department at the same time. He did not believe KD was a likely diagnosis on 7 September 2010 despite the fact Christopher was exhibiting all five clinical diagnostic signs.

[14] t.12.12.14, p.33
[15] t 12.12.14, p.20

The clinical diagnostic signs for KD are:

- high fever persisting for five or more days;
- cervical lymphadenitis;
- rash especially in the lower abdominal area around the peri-anal and genital areas;
- dry red mouth surfaces; and
- conjunctivitis without infection;

Dr Lovegrove said there were a number of children in the Emergency Department on 7 September 2010, each of whom were exhibiting some of those signs. When asked whether any others were presenting with all five diagnostic features in the same presentation Dr Lovegrove agreed there were not[16].

Dr Pecache and Dr Lovegrove consulted together over a diagnosis for Christopher and it was suggested a *"rapid test"* streptococcal swab (bacterial) be taken. This was done and the doctors waited for the result. It was negative and confirmed for Dr Lovegrove Christopher was suffering a viral illness. That was the diagnosis considered most likely.

Christopher had been in PMH Emergency Department with his father, and while waiting for the doctors to make a decision, Ms Ling joined them.

[16] t.16.12.14 p.131

Christopher was extremely anxious during this time and when his mother arrived he was held by her because he did not wish to be lain down due to pain. Christopher was on a monitor which alarmed at 177bpm. Christopher's parents were assured that was within range for a sick child with a high temperature. Christopher's parents were repeatedly advised he was suffering a viral illness which was not dangerous and he was healthy enough to fight the infection.

On receipt of the results for the *"rapid test"* swab as negative, Dr Pecache consulted with Dr Lovegrove and between them they decided Christopher was able to be discharged home. It was their view he had informed parents who would take good care of him and ensure he was looked after at home. Dr Pecache advised his parents antibiotics were not effective against viral illness, which is correct, and that he would soon start to recover.

On being told they could take Christopher home Christopher's mother became very distressed because she believed he was very sick and should be admitted to ensure his adequate hydration and proper investigation. To each of her queries a response was given as an explanation, including the extent of the pain Christopher was reporting.[17] The assurance of the doctors gave Christopher's parents confidence there was nothing seriously wrong with

[17] t 15.12.14, p.19

Christopher and they took him home hoping he would soon be feeling better and telling Christopher he would get well.

Neither of Christopher's parents believed they had been encouraged to return Christopher to PMH if they continued to have concerns.

WEDNESDAY 8 SEPTEMBER 2010

Christopher did not sleep well over night because he was complaining of pain and he continued to refuse all food and drink and vomited whenever he was encouraged to sip water. His parents noted he had offensive diarrhoea and they could not persuade him to sleep or stand up. By morning his rash had not reduced, his fever continued and his occasional cough had become severe.

Christopher's parents again took him to the family doctor, Dr Tan.

Dr Tan advised the court that approximately a third of his practice would be paediatric cases. He estimated he saw about 50 children per week and had been practising since 1975. With respect to viral illnesses Dr Tan advised the court he believed at least 80-90% of his child patients would attend his practice with viral infections.[18] He had never

[18] t 15.12.14, p.51

diagnosed a case of KD, or myocarditis, as far as he was aware.

His recollection of Christopher's second visit to his surgery was that Christopher was miserable and coughing and sitting on his mother's lap. Dr Tan reviewed the new discharge letter from PMH and asked about the continued concerns with Christopher's presentation. Dr Tan was told the severity of Christopher's coughing had increased. This did not dissuade Dr Tan from his view the diagnosis of a viral illness was correct. He reassured Christopher's parents that coughing was a good sign because it meant his lungs were clearing mucus from the airways.

Dr Tan examined Christopher and looked into Christopher's mouth to see whether he had Koplik spots which is a clinical sign of measles. He did not see any evidence of measles, although he did have a discussion with Christopher's parents about the possibility of a child suffering from measles despite vaccinations. Dr Tan agreed he discussed measles with Christopher's parents and outlined the fact that measles is quite infectious and needs to be isolated. Dr Tan denied telling Ms Ling he had diagnosed measles and she should keep Christopher isolated and not take him to hospital. Dr Tan agreed Christopher's coughing was severe and the extent of his coughing made Dr Tan look for signs of whooping cough, which he also did not see. In evidence Dr Tan agreed

whooping cough was an extreme and unlikely diagnosis, but considered he had appropriately examined Christopher for evidence of conditions which were possible on Christopher's presentation in his practice and the discharge information from PMH.[19]

When asked specifically about his examination of Christopher's chest through his clothing and from his back, Dr Tan advised that due to the small amount of muscle mass in young children it was acceptable to listen to their heart through their clothing. It was only if there was a concern it became necessary to remove clothing and apply the stethoscope directly to the skin. He agreed he had listened to Christopher's chest through his clothing but he had not heard anything abnormal to necessitate a more detailed examination.[20]

Again, to all of Ms Ling's concerns, Dr Tan responded with a logical explanation for her concerns in an attempt to reassure her Christopher was not significantly unwell.

None of the medical practitioners examining Christopher over this period believed Christopher looked as unwell as his parents clearly knew he was. Christopher had been a very healthy child and was appropriately developed. There was a lack of deterioration or weight loss in Christopher to

[19] t 15.12.14, p.53
[20] t 15.12.14, p.57

emphasise to the examining clinicians his loss of condition, as recognised by his parents.

Dr Tan agreed Christopher was irritable and miserable, but did not believe either his heart sounds or respiration indicated anything more serious underlying his fever and cough.

Dr Tan's attempts to reassure Christopher's parents led them to believe Dr Tan was discouraging them from returning Christopher to PMH if they believed he was deteriorating.

In evidence, Dr Tan stated his view that if parents believed children were sick enough they should always return them for medical review, either to a doctor or to PMH.[21]

Following his attendance on his general practitioner Christopher returned home with his parents. Christopher's parents made a follow up appointment with Dr Tan for the afternoon of Friday 10 September 2010.

Christopher did not improve that evening.

[21] t 15.12.14, p.51

THURSDAY 9 SEPTEMBER 2010

After another restless night Christopher appeared to be worse on the Thursday. Ms Ling stated Christopher did not speak at all but was extremely hungry because he had not eaten. She had bought some formula for babies with the idea of mixing some up to give him some sustenance. He was so hungry he tried to eat the powder without it being mixed. Christopher kept complaining about his pain. He was saying *"inside pain, mummy pain"*.[22]

Christopher now had swelling of his face, feet and ankles, continued very high temperature, serious cough, and his diarrhoea continued despite his lack of fluids and food. He had diarrhoea so badly, and his nappy required changing so frequently, the family ran out of wipes and had to obtain more at late night shopping. Ms Ling advised the court that when she returned with the additional wipes for Christopher he had been put to bed and was asleep.[23]

Mr Tao was studying for exams that evening. Christopher was in bed and Ms Ling had also gone to bed. At approximately 11:30pm Mr Tao heard a gurgling sound coming from Christopher's bedroom and he and Ms Ling ran into Christopher's room and found him unconscious on the bed. Mr Tao and his mother commenced CPR on Christopher and Ms Ling rang for an ambulance. Ms Ling

[22] t 15.12.14, p.24
[23] t 15.12.14, p.24

said she saw Christopher open his eyes briefly and that was the last response she saw.

During the time Mr Tao was conducting CPR on Christopher he attempted to perform a percutaneous tracheostomy of the airway when he realised his son did not appear to be taking in air. This was an attempt to bypass his airways and put air straight into Christopher's lungs. The laceration was superficial and had not penetrated the trachea. CPR was continued to keep Christopher perfused whilst awaiting the arrival of the ambulance.

On the arrival of the St John Ambulance paramedics Christopher was asystolic and apnoeic, he had fixed dilated pupils and a Glasgow Coma Scale of 3. The ambulance officers did not obtain any cardiac output throughout the transfer to PMH but his colour improved with the insertion of a laryngeal mask airway and bag and mask ventilation.

The ambulance records indicate St John Ambulance Service received the call at 11:54pm on Thursday 9 September 2010 and arrived at Christopher's home at 12:02am on 10 September 2010. Attempts were made to stabilise Christopher and the ambulance left Christopher's home at 12:21am. They arrived at PMH Emergency Department at 12:27am on 10 September 2010.[24]

[24] Ex 1, tab 27

PMH EMERGENCY 10 SEPTEMBER 2010

On arrival at PMH Christopher was asystolic, mottled and had a stiff jaw when intubation was attempted. CPR had been continued on route and Christopher had an intraosseous cannula in his left tibia through which he received adrenalin. He was provided with normal saline with bicarbonate, but his initial gas analysis indicated a pH of 6.87 with potassium at 3.9 and lactate at 15.

Christopher had had no cardiac output for over 40 minutes and the intensive care consultant considered there would be no successful outcome in any further attempts at resuscitation.[25]

Christopher's father was very distressed. He was eventually left to continue resuscitation of Christopher until he had satisfied himself Christopher could not be revived.

The official record of Christopher's death is at 58 minutes past midnight on 10 September 2010. By that time Christopher had been unwell for seven whole days and had exhibited a fever in excess of 38°C for over five days, cervical lymphadenitis, a rash including his lower abdominal area, diarrhoea with vomiting, a red mouth with dry cracked lips and conjunctivitis without infection. He had been recorded with an elevated heart rate on the occasion he had been

[25] Ex 1, tab 18

monitored in PMH on 7 September 2010, and in the following 2 days remained increasingly unwell with swollen hands and feet and a severe cough.

POST MORTEM EXAMINATION[26]

The post mortem examination of Christopher was performed on 13 September 2010 at the PathWest Laboratory by Forensic Pathologist, Dr Jodie White.

Dr White found Christopher had a soft dilated heart with a pericardial infusion, and heavy fluid laden lungs and an underlying, probable pneumonia. She confirmed enlarged lymph nodes within his neck area and around the airway. His liver appeared fatty and his kidney's mottled and soft. Christopher had no evidence of external injuries for trauma other than those associated with medical intervention and resuscitation.

Dr White ordered extensive investigations including toxicology, histopathology, microbiology, virology, and neuropathology.

Virology originally isolated adenovirus from the pericardial fluid only. On repeat testing this could not be confirmed and all further testing of samples were negative. Likewise

[26] Ex 1, tab 20

no specific bacterial pathogens were isolated from culture of any of the samples provided.

Gross neuropathology demonstrated cerebral swelling and congestion, but microscopy revealed no abnormalities with Christopher's brain development. Toxicology did not isolate any common drugs.

Histopathology revealed a diffuse myocarditis, without evident vasculitis, involving the heart, with patchy, more chronic appearing interstitial inflammation within the lungs, with acute mucopurulent changes involving the branching and smaller airways, a mild acute hepatitis, and inflammatory changes focally within the interstitium of the kidneys and the pelvic area. The lymph nodes, spleen and bone marrow showed acute stress and reactive changes.

Dr White discussed her findings with a virologist and paediatric pathologist and formed the view Christopher's cause of death was myocarditis.

Myocarditis

Myocarditis is a condition in the heart which shows inflammatory changes within the muscle, but may be caused by many different conditions, including viral illness. A viral illness is the most common cause of myocarditis and presents as a' flu like illness, with some cardiac symptoms in some patients. Usually patients recover.

It is a well-recognised cause of sudden death due to the development of fatal arrhythmias in the inflamed or scarred muscle or conduction areas of the heart. Where patients recover from a spell of myocarditis they may later develop chronic problems such as heart failure.

While Dr White could not be sure of the origin of Christopher's myocarditis she did venture an opinion in an explanatory letter it was likely to be a systemic viral infection. She did not include that in Christopher's cause of death because she was unable to confirm a viral infection. In evidence Dr White outlined she had ventured the opinion as a suggestion only as the result of the discovery of the adenovirus in one post mortem sample only. She emphasised this had not been repeated and was one of her reasons for not giving a cause for Christopher's myocarditis.[27]

In evidence Dr White outlined her initial examination of Christopher's heart, being enlarged and softened with pericardial infusion, indicated to her there was some pathology in the heart. This was supported by the pulmonary oedema which can often be seen as a consequence of heart failure or a poorly functioning heart. In addition the purulent secretions in his airway also suggested an infection related to his respiratory function.

[27] t 15.12.14, p.64-65

The fact of Christopher's enlarged lymph nodes were supportive of an inflammatory infective process although she had not been able to confirm that with microbiology.

Dr White stated she had sampled very widely because of her belief the indicators supported an infective process but could find no evidence of an infective process.[28] Again, Christopher's liver also looked ischaemic which indicated inflammatory changes. His kidneys were mottled and softened, which persuaded Dr White, Christopher was probably in shock before he died, and that was supported by evidence of poor peripheral perfusion relating to kidney failure.

With respect to myocarditis, Dr White emphasised it was wide spread throughout the heart, and extended from the inner surface of the heart and throughout the heart wall to the outer surfaces. Dr White said on histopathology the infiltrate:

> *"or the kinds of cells that were in the heart muscle, included both acute phase cells, such as neutrophils, which are the white blood cells, and also histiocytes, which is like a macrophage which tend to come in a little bit later to clean up in most circumstances. There was also a scattering of lymphocytes as well. There wasn't any vasculitis. So vasculitis is inflammation within the wall and surrounding vessels. Within one section I did see a few inflammatory cells around a small vein which was close to the surface of the outer*

[28] t 15.12.14, p.61

surface of the heart. And there was also ischaemic changes within the heart muscle. So in areas, again, you get an infiltrate of polymorph neutrophils, so acute inflammatory cells. They tend to trickle out of the vessels into the injured muscle which is being deprived of blood and oxygen….
So both of these changes were occurring concurrently in the heart muscle."[29]

Dr White went on to describe her histology of various other organs but emphasised that during her examination she found no inflammation of any of the arteries, including coronary arteries on the heart.

At the time of producing her original report in February 2011 Dr White could find no origin for the myocarditis.

In 2012 Dr David Roberts, Consultant Paediatrician, was asked to review the clinical management surrounding Christopher's death. Dr Roberts contacted Dr White to ask for clarification of her post mortem examination. Dr White re-examined her histology with Kawasaki's Disease (KD) in mind. KD is a clinical diagnosis, but there are some late cellular changes which might support a diagnosis of KD on histology. The difficulty from a forensic pathologist's perspective is most of the changes occur in the later stages of KD and are not present in the acute early stages.

[29] t 15.12.14, p.62-63

Kawasaki's Disease (KD)[30]

KD is an acute, febrile, vasculitis of childhood that affects medium sized arteries, particularly the coronary arteries. Consequently, it is the leading cause of paediatric-acquired heart disease in developed countries. It is important to have a high index of suspicion for KD in any child with prolonged fever of unknown origin and to refer to a paediatric facility promptly, as timely treatment reduces coronary artery damage. It is most common in children between 6 months to 4 years of age and a high degree of suspicion is needed to consider the diagnosis.

It is a clinical diagnosis only and there are no tests or investigations which may confirm the presence of KD. This is further exacerbated by incomplete KD where not all diagnostic criteria are present. It is further confounded by the fact there may be coexisting illnesses which make the diagnosis more difficult. Persistent fever, skin manifestations and extreme irritability are some clues to consider the clinical diagnosis. It is well recognised that if there is a clinical suspicion the child is suffering KD it should be referred to hospital as early treatment significantly decreases the risk of long term artery damage.

[30] Ex 2 *"Kawasaki Disease the importance of prompt recognition and early referral"* Daniel Golshevsky, Michael Chung and David Berukner Australian Family Physician Vol 42, No.6, June 2013, p.473.

Currently the treatment of KD is done with intravenous immunoglobulin (IVIG) and is the only proven therapy that improves coronary artery outcomes by reducing the incidence of coronary aneurysm.[31]

Children with suspected KD are often treated with aspirin to reduce fever and improve cardiac function. Dr Pecache indicated in many instances aspirin alone is used because IVIG may be too expensive or unavailable.[32] It is not clear whether aspirin alone is effective because KD is usually a self-limiting disease and due to its tendency to self-limit it is not clear whether the aspirin is merely assisting in management rather than improving outcomes.[33] The IVIG is a proven treatment to improve outcomes.

KD exists in an:

- acute phase, usually lasting up to less than ten days;
- subacute phase as the condition improves; and
- convalescent phase.

Patients reaching the convalescent stage are those exhibiting a self-limiting condition and it is at this stage that some of the post mortem pathology results can identify KD.

[31] t 12.12.14, p.12
[32] t 12.12.14, p.12
[33] t 12.12.14, p.29

Myocarditis is a very rare outcome of KD and usually occurs in the acute or subacute phase, often before there are any diagnostic features other than the presence of the clinical signs. So while KD is a relatively rare disease, myocarditis is an even rarer ramification of KD.

The medical literature documents clear benefit from IVIG therapy as a way of reducing morbidity and mortality from the complications of KD which are usually coronary artery aneurysm. It is unknown whether KD related cardiac arrhythmias, secondary to myocarditis, or myocarditis itself, responds to IVIG treatment.

As a result of Dr Roberts' query, Dr White returned to her histology and the literature in an attempt to determine whether she could find some evidence which may support a diagnosis of KD from histopathology.

In evidence, Dr White stated on re-examining her slides she found a small venule in the heart, and changes in the kidneys, which, with the presence of the infiltrate, made it likely that those changes were consistent with the acute phase myocarditis that you can see with KD. The literature indicates the histological changes tend to lag behind the clinical findings in KD and it is difficult to diagnose post mortem on histology in the acute phase.

Dr White did find a study which indicated that children who died up to 40 days after being diagnosed, on post mortem examination, revealed myocarditis was one of the first changes seen in those cases. The inflammation of the blood vessels normally associated with KD, vasculitis, didn't occur until the second week of the illness. The earliest time myocarditis had been seen in this series was at day six.[34]

Dr White concluded Christopher's histology supported the beginnings of vasculitis, as with the small venules she had observed, and that because Christopher had died in these early stages it was not as evident as in children surviving for a longer period. Overall, Dr White was of the view the myocarditis she saw in Christopher was ultimately more consistent with originating from KD than from a systemic viral illness.[35]

EXPERT REVIEW

Dr David Roberts

Dr Roberts' review of the medical issues surrounding Christopher's death raised the probability of Christopher having KD due to the presence of all the clinical diagnostic indicators for the disease.

[34] t 15.12.14, p.64-65
[35] t 15.12.14, p.66

Dr Roberts described KD as an acute and usually self-limiting vasculitis of childhood, characterised by:

- fever of at least five days duration;
- bilateral non-exudative conjunctivitis;
- erythema of the lips and/or oral mucosa;
- changes in the extremities;
- rash; and
- cervical lymphadenopathy.

It is associated with significant systemic complications, the most common and worrisome being cardiac complications.

Dr Roberts outlined the most common cardiac complication is coronary artery aneurysm, which has significant morbidity and mortality if left untreated. This complication usually occurs in the subacute phase while myocarditis is a well-recognised but more uncommon cardiac complication, which occurs in the acute phase.[36]

To ensure the best outcome it is preferable KD be diagnosed before the five days of high fever, even though not all diagnostic criteria are met. In those cases the patient should be treated with IVIG and aspirin for the best outcome.

Christopher's presentation to PMH ED on 7 September 2010 was consistent with all the diagnostic features of KD.

[36] Ex 1, tab 24 at para 1.1.9

Dr Roberts had consulted with both Dr White, as to her post mortem histology slides, and Dr Philip Roberts Paediatric Cardiologist at the Adolf Brasier Cardiology institute at Westmead Children's Hospital, as to the appropriate diagnosis, management and treatment of KD.

It was Dr Roberts' view Christopher had been suffering from KD at the time he presented to PMH, definitively by 7 September 2010, but most likely the preceding days. He considered it was understandable it may not have been picked up on earlier presentations due to the developing diagnostic features and the prevalence of viral illnesses in the general community at that time.

Dr Roberts concluded that following 7 September 2010 Christopher was suffering acute myocarditis, secondary to KD, and that the myocarditis precipitated a cardiac arrhythmia, from which Christopher effectively died shortly after midnight on 10 September 2010.

Dr Roberts was of the opinion an experienced paediatric consultant should have been in a position to diagnose KD on 7 September 2010. Dr Roberts considered it understandable it had not been diagnosed on 4 September 2010, and did not believe a GP would diagnose KD after review by a paediatric facility.

Dr Edward Oakley[37]

Dr Oakely, Consultant Paediatric Emergency Physician and the Director of Emergency Medicine at the Royal Children's Hospital, Melbourne, provided a report after review of Christopher's case with respect to the diagnosis of myocarditis. Dr Oakley indicated the symptoms and signs of myocarditis are variable, but the most common symptoms are shortness of breath, vomiting, poor feeding, upper respiratory symptoms, fever, lethargy, with tachypnoea and abnormal lung expansion being the most common. He indicated tachycardia is a very variable sign with 40% seen on clinical evaluation and 70% on ECG.[38]

On 4 September 2010 Dr Oakley considered Christopher was suffering tachycardia but it settled during his time in the ED to normal. The notes reflected poor drinking and vomiting, with clear lung and heart sounds. Dr Oakley was not of the view myocarditis was a probable diagnosis at that stage and those proposed by Dr Lim were reasonable as was the management by discharging Christopher home.[39].

By 7 September 2010 Dr Oakley outlined Christopher's description as being miserable and lethargic but still with a clear chest and heart sounds. He thought Christopher's observations generally were of concern and with respect to the monitor alarming at 177bpm Dr Oakley thought that

[37] Ex 1, tab 24A
[38] Ex 1, tab 24A
[39] t 16.12.14, p.133

any pulse of over 170bpm would warrant a period of observation to help determine the cause of tachycardia which may be early indicators of myocarditis.[40] Dr Oakley agreed this would have been a difficult diagnosis to make at that time but would still have expected a child presenting as Christopher presented on 7 September 2010 to have been admitted for observation.

Dr Oakley did not believe that tachycardia could be said to make myocarditis likely because it is nonspecific, however, he thought a period of observation would be justified. Other causes of tachycardia could also be sepsis, pain and dehydration all of which were possibilities in Christopher's case on 7 September 2010.

With respect to a diagnosis of KD, Dr Oakley considered there were a number of features consistent with a clinical diagnosis of KD. Dr Oakley pointed out the reasons for excluding KD on the 7th were all features which appear in the subacute or convalescent phase of KD and would not be expected to be diagnostic in the acute phase. Nevertheless, Christopher did exhibit those signs.

Dr Oakley considered that if KD had been considered rather than excluded, on 7 September 2010, then Christopher should have been admitted. There were some investigations which may have supported such a diagnosis even though it

[40] t 16.12.14, p.114

is the clinical signs which are diagnostic. He indicated looking for an elevated platelet count may have assisted, and ensuring Christopher's electrolyte balance was sufficient, despite his poor oral intake. Consideration should be given to an echocardiogram and while there are no diagnostic tests that confirm or refute KD it is useful to check for inflammatory markers to aid in the diagnosis.

Overall, Dr Oakley was supportive of the fact KD had been considered, but did not believe it should have been excluded on diagnostic signs which are known to appear in the subacute and convalescent phases. He was concerned more significance was not placed upon Christopher's significant tachycardia, increasing from 160-177bpm while on the monitors. In hindsight, Dr Oakley believed KD was supportable on all the clinical signs on 7 September 2010.

In evidence Dr Oakley confirmed his view KD could have been diagnosed on 7 September 2010 but was less clear about the myocarditis. He did comment however Christopher's pulse rate at over 160bpm was significantly abnormal, and that while it was not specific for myocarditis, in retrospect it is possible it was a sign he was in the early stages.[41]

[41] t 16.12.14, p.114

Dr Oakley was relatively supportive of Christopher being sent home on 7 September 2010 in view of the fact the treating clinicians did not believe he appeared significantly unwell. While he believed Christopher's signs and symptoms were consistent with KD they were also consistent with a viral illness which can cause a myocarditis. He believed children frequently did better at home in a familiar environment with informed parents without a definitive diagnosis.

However, if KD had been diagnosed then Christopher should not have been sent home because the treatment available is IVIG which significantly reduces the consequent coronary artery aneurysm and sudden death in children. IVIG needs to be provided in hospital, along with oral aspirin, as an antiplatelet drug, to prevent thrombosis in any of the inflamed or dilated coronary arteries.

Dr Oakley also confirmed that if myocarditis had been diagnosed on the 7th Christopher should also have been admitted to hospital to ensure adequate monitoring and rehydration. While there was no treatment, supportive care was necessary to ensure appropriate electrolyte and metabolic functions.

In hindsight, with the elucidation of the histology findings at post mortem, if KD and myocarditis had been identified on 7 September 2010 there would be no question Christopher

would have been admitted and treated for KD. Dr Oakley pointed out there is inadequate evidence to indicate whether management with IVIG would have influenced the outcome or progression of myocarditis. While myocarditis is a recognised consequence of KD, it is very uncommon and there is no evidence treatment with IVIG affects inflammation of the heart muscle (myocarditis). It is unknown whether treatment with the IVIG would reduce the incidence of sudden death from myocarditis, only that it will reduce the incidence of coronary artery aneurysms and of sudden death from that cause.[42]

Dr Alexander Hopper

Evidence was also heard from Dr Hopper, a Paediatric Emergency Physician in the Emergency Department of Royal Children's Hospital in Melbourne. Dr Hopper also reviewed the matter for the Coroner's Court, both in discussion with Dr Oakley and independently.

Dr Hopper stated that in 10 years in senior practice he had only diagnosed between 10-20 cases of KD. He had only had a high clinical suspicion for the disease of viral myocarditis on two or three occasions. He was only positive that one of those had been the correct diagnosis, but pointed out that viral myocarditis represents a spectrum of disease and he may well have seen it, but not diagnosed it.

[42] t 16.12.14, p.118

There are many features of myocarditis which clinically present in the same way as KD, particularly the fever, rash, inflamed throat, cough, vomiting, diarrhoea, but that with KD one also saw red eyes, inflamed mucus membranes and enlarged lymph glands.[43]

Dr Hopper confirmed his view Christopher's presentation on 7 September 2010 could have been diagnosed as either a viral myocarditis or KD. All the diagnostic criteria for KD were present on 7 September 2010, although there was significant overlap with symptoms of viral illnesses.

Dr Hopper stated it was not always the role in emergency departments for emergency physicians to arrive at a specific diagnosis. The aim in an emergency department is to create a differential diagnosis and formulate an action plan which involves sufficient risk management to account for the differential diagnosis.[44] It was his view that on 7 September 2010, Christopher's diagnosis would have contained a viral illness at the top end of the differential diagnosis.

Dr Hopper confirmed that prevalent illnesses at the time of presentation do influence diagnostic thinking. He agreed emergency department clinicians recognised clinical patterns which inevitably influenced their diagnostic thinking. It is very difficult to exclude an illness in the emergency department context. Specifically, *"one needs to*

[43] t 16.12.14, p.144
[44] t 16.12. 14, p.145

satisfy oneself that the probability of a serious diagnosis is sufficiently low to justify your management plan"[45] If the risk of a very serious illness is below 1%, then in the emergency context, that was probably reasonable because one cannot be 100% certain.

Dr Hopper agreed KD was a clinical diagnosis; there were no tests or investigations which could be done in the emergency department context to definitively diagnose KD, especially in the acute phase. That said there were five clinical signs of KD for Christopher on 7 September 2010. Dr Hopper agreed it is an illusive entity and that some of the symptoms can come and go. It is difficult to diagnose but also difficult to exclude, but was relatively uncommon.

Had Christopher been suspected of having KD Dr Hopper was sure he would have been admitted and an echocardiogram would have been performed over the following days. It is likely the developing myocarditis would have been picked up and treatment commenced with IVIG. There is no guarantee that management would have been effective in preventing an arrhythmia as a result of myocarditis.

[45] t 16.12.14, p.146

It was impossible to say with certainty whether Christopher's admission on the 7th would have changed the outcome, but there was a possibility it may have.[46]

CONCLUSION AS TO THE DEATH OF CHRISTOPHER

I am satisfied Christopher was a 2 year 9 month old toddler. He was bright, alert, healthy and developmentally normal.

On the evening of 2 September 2010 he had appeared quieter than usual which developed by midnight to his feeling significantly unwell with a high temperature.

Christopher's parents were well informed parents and initially treated him with fluids and Panadol assuming he was developing a cold.

Christopher remained unwell the following day, with a high temperature. His parents became concerned when he refused all food and fluids.

By Saturday 4 September 2010 Christopher was not improving and continued to refuse fluids. Due to his continuing unwellness his parents presented with him to PMH Emergency Department.

[46] t 16.12.14, p.151

Christopher was triaged at 8:19pm with a score of 4 and provided with secondary triage at 8:45pm and 10pm. Whilst in the emergency department awaiting medical assessment Christopher's parents noticed a swelling on the left side of his neck.

Christopher was seen by Dr Lim at 10:12pm and on her examination of Christopher she found him to be alert, well and not septic looking. She did not consider him to be unwell although he had an inflamed ear drum and his tonsils were infected. His chest and heart sounds were normal and she recorded Christopher as suffering from cervical lymphadenitis and tonsillitis. She prescribed Nurofen, fluids and Cephalexin. She advised Christopher's parents she did not consider Christopher to be unwell enough to warrant blood tests.

Christopher's parents returned home with him, but he remained unwell to the extent that on Monday 6 September 2010, they took him to their family GP, Dr Tan. Dr Tan had been advised by PMH of Christopher's attendance on the 4th. Dr Tan examined Christopher and noted his parents' concern he had now developed a rash.

Dr Tan did not disagree with the diagnosis from PMH and thought Christopher presented consistently with their management plan, however, was concerned the rash signified an allergy to either the Cephalexin or the Nurofen.

Consequently he advised Christopher's parents to stop giving him those medications, but to continue to provide him with Panadol and Phenergan which it was known he could tolerate.

By 7 September 2010 Christopher continued to have a high temperature, had dried cracked lips, cervical lymphadenopathy, a rash and conjunctivitis. He was also under 5 years of age, of Asian descent, and male. While the latter three features are not diagnostic, they feature in the literature as reflecting a cohort for which there is good reason to have a high level of suspicion especially where all other clinical diagnostic features of KD are present.

I accept a number of children presenting to the PMH Emergency Department were exhibiting similar symptoms during that period of time but I am satisfied Christopher was diagnosable with KD on 7 September 2010. I note, however, both Dr Lovegrove and Dr Hopper confirm prevalent community illnesses do play a part in emergency department diagnoses and value judgements around management of different diagnoses may be conflicted.

The factors considered by Dr Lovegrove and Dr Pecache in rejecting the diagnosis of KD were, however, symptoms associated with the subacute and convalescent phases of KD. In view of the fact KD is considered to be self-limiting these were not good indicators of Christopher's diagnosis in

the acute stage. In hindsight, admission and monitoring were a preferable option.

Whether the early stages of myocarditis were present at that time remains unclear, but I accept Dr Oakley's view 177bpm was an extremely abnormal reading.[47]

I accept to those who did not know Christopher, he did not appear as unwell as his parents, informed and alert professionals, knew him to be. I understand the majority of parents are concerned about their children, however, parents do know their children and the level of anxiety of Christopher's parents should have given pause there was something more to Christopher's presentation than an illness which should have been improving by day five of a fever.

Christopher's parents were reassured by the doctors' apparent explanation for their concerns and returned home with Christopher, albeit with some reluctance in view of their level of concern. I accept there were no diagnostic tests which could be conducted to validate the presence or otherwise of KD. Maybe that alone warranted his admission for monitoring, particularly in view of the elevated pulse rate, and the sorts of supportive investigations mentioned by Dr Oakley.

[47] t 16.12.14, p.114

On return home Christopher's parents followed the advice of the doctors, but Christopher did not improve and they returned to Dr Tan on 8 September 2010.

Dr Tan had the second discharge letter from PMH and reviewed that prior to examining Christopher. Dr Tan examined Christopher with the view to considering differential diagnoses. Essentially, Dr Tan rejected all differential diagnoses he considered fitted Christopher's symptoms and agreed he was most likely suffering a viral illness.

There is some dispute as to whether Dr Tan diagnosed measles, but certainly Ms Ling took that discussion as a diagnosis, although Dr Tan had seen no Koplik spots which would be diagnostic and advised Mr Tao there were none. Dr Tan agreed Christopher's occasional cough was now a severe cough which was why he looked at, and rejected, whooping cough.

Overall, Dr Tan could find no explanation for Christopher's symptoms other than the diagnosis made by PMH the previous day.

In evidence, Dr Lovegrove noted the development of the severe cough and the swelling in Christopher's extremities would have alerted PMH staff to the possibility of the

involvement of a pathology with the heart had they been aware of the development of those symptoms.[48]

However, Christopher's parents did not feel they had been encouraged to return to PMH and were accepting of the advice from the clinicians that Christopher, although unwell, would get better. As a result of the reassurance the clinicians attempted to give Mr Tao and Ms Ling there was some concern by Christopher's parents they were seen as overreacting and they did not return Christopher to PMH.[49] They had quite simply lost confidence in the medical profession to help their child.

Ms Ling and Mr Tao returned home with Christopher and cared for him as best they could while remaining very concerned. Christopher remained unwell, but extremely hungry, throughout 9 September 2010 and that night at around midnight I am satisfied Christopher suffered a cardiac arrhythmia as a result of myocarditis, a consequence of his being in the acute phase of KD.

Despite prompt medical attention from his father and continued input from the St John Ambulance Paramedics and the clinicians in PMH ED Christopher was irretrievable and pronounced dead in the early minutes of 10 September 2010.

[48] t 16.12.14, p.137
[49] t 15.12.14, p.19

I am satisfied Christopher died as a result of myocarditis induced by KD.

I find death arose by way of Natural Causes.

COMMENTS RELATED TO PUBLIC HEALTH

The most concerning aspect of Christopher's interaction with the public health system was the perception by his parents they were not welcome to return Christopher to the PMH ED after their attendance on 7 September 2010.

Ms Ling felt she had been discouraged from taking Christopher to hospital and does not accept any of the medical reviewers of Christopher on either the 7th or the 8th encouraged her to return Christopher to PMH.[50]

I accept it is PMH policy and the practice of all emergency department doctors to emphasise to parents that they need to return with either continued deterioration in a child, or any indication the child is not improving. However, suspect there is a fine line between trying to reassure parents and appearing to dismiss their concerns.

Parents, especially informed parents, have a very good sense of the extent of their child's unwellness. I accept experienced clinicians also develop a sense, but in busy

[50] t 15.12.14, p.20-22

times in emergency departments with many children, appreciate it must be difficult to maintain a heightened sensitivity where the local epidemiology is strengthening the probability of a common illness as opposed to a rare event. As stated by Dr Oakley Christopher's death was a rare event in view of the fact KD is not common and myocarditis as a complication of KD is even less common.[51]

All practising clinicians agreed it was necessary paediatric consultants maintain a high degree of suspicion for KD.

In Dr Hopper's view the diagnosis of KD was a real possibility on 7 September 2010, but that of myocarditis much less likely. If KD had been suspected then Christopher would have been admitted and tests run over the intervening days to evaluate his cardiac status. Provided KD had been diagnosed as a result of the clinical signs then it is likely he would have been treated with IVIG. There is no empirical evidence treatment with IVIG would have prevented his death as a result of myocarditis.[52] It is also not clear an ECG would have been performed early enough, following admission on 7 September 2010, to have influenced his fatal arrhythmia on 9 September 2010.

Dr Lovegrove listened to Ms Ling's evidence as to the progression of Christopher's illness following the review on 7 September 2010 and believed it was apparent Christopher

[51] t 16.12.14, p.125
[52] t 16.12.14, p. 151

was exhibiting some signs of cardiac pathology. This was confirmed at post mortem examination. Had that progression occurred following an admission it is possible the outcome for the family would have been more understandable. This makes it even more important clinicians remember distressed parents are very vulnerable to the wrong messages from clinicians.

Both Christopher's father and mother felt their concerns had been underestimated by Dr Lovegrove, and Ms Ling felt Dr Pecache did not appreciate how sick she believed, as an informed practitioner, Christopher was feeling. Both parents felt dismissed by the emergency department personnel and discouraged about returning Christopher to their care. Clinicians need to be extremely careful about interactions with parents who are clearly distressed and well informed. Ms Ling was asking a number of relevant questions which emphasised her level of concern.

There was some discussion at the conclusion of the inquest as to ways clinicians and PMH emergency department may improve their communication with carers for re-presentation of paediatric patients. I have since received submissions from PMH as to how the methods discussed in court would not achieve the desired result.[53] PMH feel there are now systems in place to encourage the re-presentation of a child with ongoing signs and symptoms.

[53] Submissions on behalf of PMH 23 December 2014

On 7 September 2010 Dr Pecache was not in a position to discharge Christopher without input from the consultant, Dr Lovegrove, due to the fact it was a re-presentation. This system was in place and it is not clear the revised system (consultation with senior doctors) would improve that perception by parents.

Ms Ling and Mr Tao were very supportive of a 'gold card system' as suggested in court. They remained supportive of a 'gold card system' even after reading PMH submissions it was unlikely to improve communication. They did, however, advise the court they had experienced very good interactions with PMH staff more recently. In view of their history with Christopher that is entirely appropriate.

PMH is intending to improve its discharge letters to GPs. There is not always the opportunity to provide a discharge letter when the ED is busy. In Christopher's case timely discharge letters were provided to Dr Tan, and he was aware of the fact Christopher had presented twice to PMH. The proposed action by the emergency department to "*improve the quality of the discharge letter and ensure there was a clear message to the general practitioner that the child had been seen in two or more occasions at PMH and to ensure the GP was alert to the fact that contact with the senior doctor on duty in the emergency department at PMH has recommended*

if the child remains unwell or is presenting with new signs and symptoms that may require further review at PMH".[54]

Dr Tan was aware of those facts, however, did not view it necessary Christopher re-present as he saw him on the 8th, unless things deteriorated. Christopher had developed additional signs on the 8th when he saw Dr Tan and I doubt a discharge letter specifying those facts would have made any difference to Dr Tan's review on 8 September 2010, but it may have encouraged a suggestion to re-present to PMH.

PMH has sent a memo to all clinical staff working in the emergency department as a result of Christopher's death, to encourage the insertion into discharge letters for GPs the suggestion a child re-present, but would point out the GP would still need to be convinced it was necessary. Dr Tan was not and none of the experts giving evidence believed it unacceptable Dr Tan relied on the PMH diagnosis, after considering and rejecting differential diagnoses.

It is to be hoped Christopher's death has refreshed the need to have an elevated suspicion for KD in paediatric emergency departments, with the emphasis on admission for observation and monitoring. This may not have changed the outcome in the rare circumstances where a fatal arrhythmia is the mechanism of death, but it would have

[54] Submissions from PMH dated 23 December 2014

ensured improved hydration and electrolyte control which may have affected the outcome.

E F Vicker
Deputy State Coroner
13 February 2015