

**IN THE CORONERS COURT  
HELD AT HAMILTON**

**IN THE MATTER** of the Coroners Act 2006

**AND**

**IN THE MATTER** of an Inquiry into the  
Death of David Nicholas JACOBSON

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**POLICE SUMMARY OF INVESTIGATION**

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My full name is Garry Wayne PATON. I am a Senior Constable stationed at Hamilton.

**Introduction:**

1. The death of David JACOBSON was the subject of a Police investigation. As a result Police are satisfied that there were no circumstances of a criminal nature relating to this death.
2. Police then carried out a further investigation as directed by the Coroner. This investigation has now been completed, and the following matters were established:

**Background:**

3. Mr David JACOBSON, aged 31 years old, resided at 34 Gainsford Road, RD2, Hamilton.

4. He suffered a chronic neurological condition since his teenage years and had an extensive past neurological history.
5. Mr JACOBSON had become severely disabled with no significant movements of his upper and lower limbs and problems with swallowing and articulation.
6. He had become rest home dependent as it was no longer appropriate to be cared for at home.

**Events:**

7. On 1st September 2008 Mr JACOBSON was admitted to Auckland Hospital where further specialist opinions would be obtained.
8. On 19th September 2008 he was transferred back for ongoing care of the Waikato Hospital. He was being fed via a nasogastric tube.
9. Due to his unreliable oral intake and weight loss, a decision was made to commence Percutaneous Endoscope Gastrostomy (PEG) feeding for optimisation of nutrition.
10. On 15th October 2008 PEG insertion was performed at the Waikato Hospital.
11. Shortly after Mr JACOBSON developed a rare complication of the PEG placement.
12. This involved a dislodgement of the PEG from the stomach causing infusion of food and leakage of gastric contents into the abdominal cavity.

13. Complications were identified and a laporotomy performed on 27th October 2008.
14. Further surgery was later required however Mr JACOBSON's condition continued to deteriorate until his death on 1st December 2008.
15. Police were advised of this death at the Waikato Hospital and attended.
16. Life extinct was certified by Dr KAH WEI TEH.
17. The deceased was identified as David Nicholas JACOBSON by his mother. This was completed by way of statement.
18. Normal mortuary procedures were carried out.
19. Circumstances of this death were reported to the Coroner and a post mortem was performed by Dr LAMONT, a Pathologist.
20. Further enquiries were carried out by way of the Coronial Services obtaining reports from medical personnel involved in the care of Mr JACOBSON along with hospital medical records and family concerns.
21. This information was forwarded to Mr Jonathon Barnes KOEA at the Auckland District Health Board to review and make comment.
22. Following his review Mr KOEA completed a report for the Coroner.

**Production of Exhibits:**

23. I now produce as exhibits;

- (a) Deceased Person Certificate completed by Dr KAH WEI TEH.
- (b) Statement of Identification completed by Mrs Anne JACOBSON.

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Garry W PATON.

Taken and sworn before me at HAMILTON

this                      day of    2011

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Coroner

**CERTIFICATE OF FINDINGS****Section 94, Coroners Act 2006****IN THE MATTER of David Nicholas JACOBSON**

**The Secretary, Ministry of Justice, Wellington**

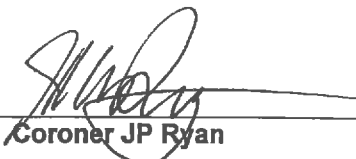
As the Coroner conducting the inquiry into the death of the deceased, after considering all the evidence admitted to date for its purposes, and in the light of the purposes stated in section 57 of the Coroners Act 2006, I make the following findings:

Full Name of deceased: David Nicholas JACOBSON  
Late of: 34 Gainsford Road  
R D 2  
Hamilton  
Occupation: Unknown  
Sex: Male  
Date of Birth: 12 January 1977  
Place of Death: Waikato Hospital  
ICU Unit  
Pembroke St  
Hamilton  
New Zealand  
Date of Death: 01 December 2008  
Cause(s) of Death  
(a). Direct cause: Abdominal sepsis  
(b). Antecedent cause (if known):  
(c). Underlying condition (if known):  
(d). Other significant conditions contributing to death, but not related to disease or condition causing it (if known): Unspecified neurologic disorder

Circumstances of death: David suffered from a very complex and severe neurologic disease. He underwent a PEG procedure in Waikato Hospital on 15 October 2008. Following this, David's clinical course involved numerous complications stemming from this and subsequent procedures, resulting in David undergoing multiple surgeries. In addition, David was in a malnourished and immunosuppressed state. He developed severe sepsis which, in his compromised state, he was unable to overcome. His condition progressively declined despite intensive medical treatment. He suffered a cardiac arrest and was not resuscitated due to the severity of his condition

Those findings, and my reasons for making them, are also set out in my written findings dated: 6 June 2012.

Signed at Hamilton on 6th day of June 2012.

  
Coroner JP Ryan

IN THE CORONERS COURT  
AT HAMILTON

CSU-2008-HAM-000638

UNDER THE CORONERS ACT 2006

AND

IN THE MATTER of an Inquiry into the death of  
DAVID NICHOLAS JACOBSON

Date(s) of Hearing: 27 – 29 September 2011

Appearances: E Staples, Counsel to Assist the Coroner  
M Duggal for Dr Wright, Ms Flevill and Ms Wallwork  
C Garvey for Dr Lynch and Dr Hill  
M Parker and M Cowen for Dr Rafique and Dr French  
Mrs M Jacobson, family representative

Specialist Advisor: Professor Mark Lane

Date of Findings: 6 June 2012

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FINDINGS OF CORONER JP RYAN

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## INTRODUCTION

[1] I decided pursuant to section 80(b) of the Coroners Act 2006 to hold an inquest for the purposes of this inquiry into the death of David Jacobson ("David"). My reason for that decision is that I wished to hear additional evidence in relation to this death in order to properly establish the matters required to be established under section 57(2) of the Coroners Act 2006 ("the Act").

[2] Due to the complex nature of the medical evidence relating to David's condition and clinical course, the Chief Coroner, pursuant to section 83(1) of the Act, appointed a Specialist Advisor to sit with and advise me at this inquest. Professor Mark Lane, who holds an honorary post of Clinical Associate Professor with the University of Auckland and whose speciality is gastroenterology, accepted the appointment as Specialist Advisor. Professor Lane is a fellow of the Royal Australasian College of Physicians, and currently works as a Senior Medical Officer at the Auckland District Health Board in the Department of Gastroenterology and Hepatology. I am satisfied that Professor Lane is eminently qualified and suitably experienced to be able to act as Specialist Advisor.

## FACTS

[3] David was thirty-one years of age at the time of his death on 1 December 2008. During his relatively short life span, he had an extremely complex and difficult medical history, which is succinctly summarized in a report provided by Dr McKenzie.<sup>1</sup> David had been diagnosed with a severe progressive peripheral polyneuropathy, and relapsing, remitting, transverse myelitis (severe disorder of nerves in the spinal cord and peripheries, which wax and wane in severity but are generally progressive). In addition, David also had a marfanoid connective tissue disorder, mild to moderate intellectual impairment, and mitral valve prolapse.

[4] David had his first episode of peripheral weakness as a fourteen-year-old boy. By 2004 he had had four neurological episodes which had responded to intravenous and oral steroids. He had further relapses in 2004 and 2006, and in 2007 he was noted to have progressive deterioration. By mid 2008, despite further interventions, his condition had progressed to the point that he was wheelchair-bound and then by August of that year he had become bed-bound.

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<sup>1</sup> *Report to the Coroner Re: David Nicholas Jacobson*, Dr Jane McKenzie, dated 5 July 2011

[5] On 1 September 2008 he was transferred to Auckland Public Hospital for a second opinion. The clinicians there concluded that David had a relapsing remitting demyelinating peripheral neuropathy consistent with chronic inflammatory demyelinating polyneuropathy, as well as a relapsing-remitting lower brainstem and spinal cord inflammatory disease consistent with multiple sclerosis.

[6] David returned to Waikato Hospital on 19 September 2008. It is noted that his swallowing was impaired and he had become malnourished. To assist with feeding, he underwent a percutaneous enteral gastrostomy ("PEG") on 15 October. This procedure involves the insertion of a flexible tube into the stomach through the abdominal wall, and enables a patient to receive nutrition, fluids and medication directly into the stomach, thereby circumventing any swallowing difficulty the patient may be experiencing.

[7] On 25 October David was noted to be febrile, and investigations demonstrated that the tip of the PEG tube had become dislodged into the peritoneal cavity. He underwent a laparotomy, where the tube was removed and replaced. He was also noted to be infected with several different bacteria. On 6 November David underwent a repeat laparotomy, involving the closure of the two gastrostomies, and a jejunostomy (insertion of a feeding tube into his small bowel). He had become colonised with methicillin resistant *Staphylococcus aureus* ("MRSA"), more commonly known within the lay community as a super-bug.

[8] On 15 November, David was treated for a bleeding gastric ulcer, which required a further laparotomy, gastrostomy (hole made in stomach) and over-sewing of the bleeding ulcer. Then on 27 November a CT scan of his abdomen showed extensive free fluid and gas suggestive of a perforation of the bowel. He underwent a further laparotomy, where a large amount of infected peritoneal fluid was washed out and the jejunostomy was re-sutured.

[9] By this stage of his clinical course, David was in a dire state. On 1 December late in the evening, David suffered a sudden and severe episode of hypotension associated with tachycardia progressing to bradycardia with pulseless electrical activity. Clinicians, in consultation with family, had previously determined that David should not undergo cardio-pulmonary resuscitation in the event of a cardiac arrest, and consequently no resuscitative efforts were made. David was subsequently pronounced dead later that evening.

## ISSUES

[10] The main issues to be considered in this inquiry are:  
(a). Was the initial PEG necessary;



- (b). Was David a suitable candidate for a PEG;
- (c). The PEG procedure on 15 October;
- (d). David's clinical care and treatment following the initial PEG; and
- (e). David's clinical course leading up to his death.

### **Issue 1: Was the initial PEG procedure necessary**

[11] Mrs Margaret Jacobson, David's mother, expressed serious concerns regarding David's treatment at Waikato Hospital during his admission commencing on 19 September 2008. Mrs Jacobson has spent the intervening years between David's death and this inquest conducting considerable research into the risks of a PEG. Without wishing to minimise the extent of her concerns and the depth of her research, in my view Mrs Jacobson's primary concern is that the PEG procedure was not necessary in David's case. She considers that David's death is the direct result of the complications associated with the PEG, and therefore if the procedure had not been performed in the first place, there is a reasonable expectation that David would have survived his admission to hospital.

### **Discussion**

[12] Mrs Jacobson gave evidence at the inquest that David had been living in a rest home for two months prior to his admission to Waikato Hospital in August 2008. While in the rest home, David lost 20 kilogrammes ("kgs") of weight. As to the reasons for this weight loss, Mrs Jacobson was unable to give a definitive answer but she alluded to problems David had with the diet provided to him by the rest home and obliquely acknowledged that he had some difficulties swallowing. When asked why David was admitted to Waikato Hospital in August 2008, Mrs Jacobson stated that it was basically because he could not swallow. Mrs Jacobson further stated that, when David was in Auckland Hospital during September, he was fed by means of a nasogastric tube ("NGT"), and as a result he put on a good amount of weight. Towards the end of his stay in Auckland Hospital, his ability to swallow was tested and found to be capable of handling water and pureed apple.

[13] Mrs Jacobson informed that, in her opinion, David was eating and drinking well in Waikato Hospital subsequent to his return from Auckland Hospital. David's nursing notes from Waikato Hospital contain entries from 20 September 2008 stating that David was on a pureed diet and NGT feeds. On 22 September, David was reviewed by a speech and language therapist, Amanda Flevill. Her entry for that review in the nursing notes state that there was an improvement in swallowing since David's last admission to Waikato Hospital. Her plan was to continue the alternative feeding until David was able to display adequate and consistent oral intake.

[14] Ms Flevill gave evidence that in the last week of September David was reported to be eating and drinking well with encouragement, but that the dietitian documents that David's oral intake remained unreliable. Although an assessment of David's swallow with water taken through a straw revealed a well-timed functional swallow with no indications of airway compromise, Ms Flevill questioned David's strength of swallow and a possible deterioration of the function of swallow as a meal progressed. She noted that David is documented to be unable to meet his nutritional requirements orally, and that *"oral intake is likely to remain low secondary to fatigue, a general muscle weakness, and a fluctuating dysphagia. Long-term feeding options are strongly recommended."*<sup>2</sup>

[15] Although David's ability to swallow appears to have improved early in October, Ms Flevill's report notes that it was the opinion of the team caring for David that he was likely to require ongoing NGT or PEG feeding to keep up his caloric intake. David's earlier well weight had been around 83 kgs. His weight recorded in his notes from Auckland Hospital between the 1<sup>st</sup> and 10<sup>th</sup> September was 71 kgs. His body mass index ("BMI") was recorded as being 17.6, which was below the normal range. Around 14 October, David was weighed and reported to be 67 kgs. David had sustained progressive weight loss that had continued from early September through to mid October despite supplemental feeding.

[16] When consideration was first given to David undergoing a PEG, Mrs Jacobson was strongly opposed to this treatment. As a result, the medical team agreed to continue the current feeding regime while monitoring David's nutritional progress. At a meeting held on 3 October, the decision was made that a PEG was an appropriate treatment option. On 7 October, a gastroenterology referral was made by a clinician for consideration of a PEG. Ms Flevill made an entry in the nursing notes for that day to the effect that David continues to be unable to meet his nutritional requirements by oral intake alone. The entry goes on to say that there is a high reliance on NG feeds to meet his needs, and that he is at risk of compromising his swallow with increasing fatigue during the course of the meal. On 9 October, the clinical notes contain an entry stating that PEG is appropriate for David to improve the quality of life in terms of access for feeding.

[17] Ms Flevill, in her evidence given at the inquest, confirmed her opinion that the PEG was an appropriate option for David because he required long-term feeding options. She stated that without alternative feeding, David would have continued to lose weight and lose energy which would have weakened him more and compromised his swallow more. She was quite clear that she did not consider David could sustain himself on his oral intake alone.

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<sup>2</sup> Report to the Coroner Re: David Nicholas Jacobson, Amanda Flevill, dated 11 August 2011, para 11.6

[18] Helen Wallwork was the dietitian involved in David's care at Waikato Hospital from 20 September 2008. In her report, Ms Wallwork informs that she was suggesting a PEG from around the beginning of October for long-term feeding, and this was a follow on from longer term NG feeding. She estimated from the notes that David was only managing to eat between 1000 and 1500 calories orally per day, with a further 1420 calories via NG feeding. She notes that she considered David was "*unlikely to be able to improve his malnourished state through oral food and fluids alone.*"<sup>3</sup> She also refers to David's recorded weight loss from 74 kgs down to 67 kgs as an indication that "*David's weight was continuing to drop despite high calorie NG feeding plus oral food.*"<sup>4</sup> In her evidence at the inquest, Ms Wallwork stated that this weight loss was a big concern to her.

[19] Doctor Peter Wright, a neurologist at Waikato Hospital who had a long involvement in treating David, outlined at the inquest the process that would normally occur before a decision for PEG was made. Although Dr Wright was on leave until 13 October 2008 and was not therefore involved in the decision, he stated that the decision would be managed by the multidisciplinary team, which would include the nurses, the speech therapist, the dietitian, the family and the neurologist. This team would collectively make a judgement as to whether it was appropriate for David to undergo a PEG.

[20] Dr Christopher Lynch was the neurologist caring for David in early October during Dr Wright's absence. In his evidence at the inquest he stated that there was an extensive meeting with David and Mrs Jacobson held on 3 October 2008 which covered the investigations and treatment options including the PEG. Dr Lynch informed that he considers Mrs Jacobson understood why the PEG was to be performed and the risks and benefits of the procedure. Dr Lynch stated that it was his professional judgement that the PEG was a good option for David given his condition and difficulties in maintaining adequate nutrition by oral intake. He noted that David's weight loss was serious, and that he had failed to meet his nutritional requirements with NGT feeding supplementing oral feeding. He stated that the PEG would optimise the outcome for David.

[21] Dr Wright, in his evidence, stated that a PEG is performed for long-term nutritional support where it is felt that, at least in the intermediate to long term, the patient would otherwise need a nasogastric tube. He further explained that a nasogastric tube is not an appropriate long-term solution because of social issues, the prospect of it being dislodged, irritability of the nasal and oral passages, and ulceration.

[22] Professor Lane, having reviewed all of the reports provided by the clinicians, speech and language therapist and dietitian involved in David's care as well as having listened to

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<sup>3</sup> Report to the Coroner Re: David Nicholas Jacobson, Helen Wallwork, dated 13 May 2011, para 6.5

<sup>4</sup> Ibid. para 6.6

the evidence during the inquest, concludes that it was unreasonable to expect that there would be any further increase in David's ability to take adequate amounts of oral intake.<sup>5</sup> Full oral intake alone was therefore not a viable option for David's nutritional requirements. Professor Lane then goes on to consider the options available to supplement oral intake. He notes that NGT feeding will typically be continued for a period of up to four weeks in the hope or expectation that normal swallowing and ability to take adequate nutrition will be reinstated, and increased metabolic requirements will be reversed. He states that in general only a minority of patients will continue on long-term NGT feeding for the above indications, and he notes the associated risks with prolonged NGT feeding primarily in terms of patient quality of life. He notes also that David received NGT tube feeding for a period of between six and seven weeks in total, from 28 August to 15 October. Professor Lane concurs with Dr Wright's list of associated risks with NGT feeding stated above, and points out that David experienced a dislodgement of the nasogastric tube with food being infused into his oesophagus, thereby placing him at significant risk of aspiration pneumonia.

[23] Another alternative to NGT feeding was parenteral nutrition (intravenous feeding), but Professor Lane points out that the presence of an intact and normal functioning gut would have meant that this option should not and would not have been considered.

[24] The only other feasible option was PEG feeding. Professor Lane states:

*"In broad general terms, PEG feeding will be considered in a patient who requires supplemental enteral feeding via a tube because of inability to take adequate oral intake. It would be considered after the tube has been present for at least 4 to 6 weeks and where the expectation is that supplemental feeding by tube will be required for at least four weeks thereafter. The common neurological scenarios for which PEG tubes are placed include progressive neurological disorders such as motor neurone disease. While David did not specifically fall into this group it is clear that he had a severe, disabling and (while relapsing remitting) progressive neurological disorder that was significantly impacting on his ability to take adequate calorie intake. There is no expectation that it would show a substantial improvement within a reasonable period of time (weeks and months).*

*The significant downside of PEG tube placements, in general, are the requirement for an invasive procedure to have this tube placed (an endoscopy with associated sedation, the discomfort of having the tube placed), and potential complications of placement including wound infection and tube dislodgement. As such in general terms David meets the criteria under which one would consider placement of a PEG as being a reasonable option to consider."*<sup>6</sup>

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<sup>5</sup> Report on Coroner's Inquest on the death of David Nicholas Jacobson, Dr Mark Lane dated 2 November 2011, page 3

<sup>6</sup> Ibid, page 4

[25] Prior to the inquest, David's clinical file was reviewed by Professor Jonathan Koea. At the time he wrote his review, Professor Koea was employed as a Hepato-Biliary and General Surgeon in the Department of Surgery at Auckland Hospital and as an Associate Professor in Surgery by the University of Auckland. In his report, Professor Koea confirms that the clinical notes "*indicate that there was significant concern regarding [David's] nutritional intake and subsequent weight loss*", together with "*... repeated and clear evidence of increasing concern over Mr Jacobson's swallow and some objective evidence of its impairment (admission with aspiration pneumonia).*"<sup>7</sup> On the question of whether a PEG was an appropriate and necessary procedure, Professor Koea concludes that "*... in patients with neurological impairment who require long-term nutritional support, PEG feeding is regarded as the standard of care and was an acceptable intervention.*"<sup>8</sup>

### **Conclusions**

[26] I am satisfied from the evidence provided that the decision to proceed with the PEG was appropriate in the background of David's sustained weight loss on a combination of oral intake and supplemental NGT feeding, and that the decision was made with proper consideration for his clinical status and ongoing nutritional needs. The decision was made by the team rather than by any one individual. The recommendation for PEG was made by the dietitian on the basis of nutritional deficiency and supported by the speech and language therapist, whose evidence is that David had difficulty with oral intake. She was particularly concerned that this difficulty was causing David to expend significant amounts of energy to eat a meal, thereby causing him to lose even more weight. At the same time, the dietitian was also considering that a PEG would most likely be required for long-term feeding. The dietitian and the clinicians were concerned about David's continuing weight loss and the fact that he was unable to sustain adequate nutrition with NGT feeding and some oral intake. This recommendation was considered by the clinical team. The decision to proceed was made after due consideration of the benefits of the procedure in terms of improving his nutritional status and quality of life balanced against the known risks, including David's particular risk profile due to his immunosuppressed state.

[27] I note that this decision was not made hastily; both the speech language therapist and the dietitian were considering the need for a PEG from around the end of September. A discussion regarding a PEG was held by the team on 3 October, and Mrs Jacobson was present. It was agreed that this procedure was appropriate for David. A referral to the gastroenterology service was made on 7 October, and a gastroenterology review on that date confirmed that PEG insertion was appropriate and that there were no physical contraindications to the procedure. Dr Hill also reviewed David's case on 9 October, and

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<sup>7</sup> Re: David Nicholas Jacobson, Professor J Koea, page 1

<sup>8</sup> Ibid. page 2

documented his agreement for the procedure following a discussion with Ms Wallwork. From the initial discussion on 3 October to the date the procedure was performed, approximately 12 days elapsed. During this period, if it had become apparent that David was improving in his ability to swallow and sustain himself through oral intake alone, then there **was** ample opportunity to reverse the decision to proceed with a PEG.

#### **Secondary Issue: Consent to PEG procedure**

[28] Mrs Jacobson raised a secondary issue in relation to the PEG procedure, specifically whether informed consent was granted to the procedure being performed. Although this issue is not strictly within my jurisdiction, I consider that it is a matter so intricately interwoven with the decision-making process that is appropriate I examine the issue. By doing so, I may also be able to obviate the need for this issue to be considered by the Health and Disability Commissioner. My intention here is not to circumvent the function of the Commissioner in any way; it is simply to alleviate the stress on Mrs Jacobson by avoiding (if possible) the need to go through this process a second time on one particular issue. Mrs Jacobson is, of course, perfectly entitled to pursue a complaint to the Health and Disability Commissioner regardless of my finding.

[29] Mrs Jacobson was adamant in her evidence that she was never informed fully of the risks associated with the PEG. She also considers that she was presented with the decision regarding the PEG as a 'fait accompli', and was not part of the decision-making process. In her view, she was told that it was necessary and that she simply had to accept that decision. She points out that the consent form for the procedure has a place for her signature, and also a place for her initials in a box specifically relating to information on the risks of the procedure. The consent form bears her signature; the box relating to information on the risks of the procedure has no initials. She relies on the absence of the initials to support her argument that she was not informed of the risks.

[30] Dr Lynch was equally clear in his evidence that he recalled discussing the procedure with Mrs Jacobson in a series of discussions. This is consistent with evidence from the speech and language therapist and the dietitian who recall discussing the procedure with Mrs Jacobson at various times, as well as being discussed in a family meeting on 3 October. Mrs Jacobson impresses as a person who does not accept things at face value when it comes to the treatment of her son. I therefore find it difficult to accept Mrs Jacobson's recollection that she did not take part in the decision-making process, and that she was not informed of the risks associated with the procedure.

[31] I find it more likely that Mrs Jacobson would have demanded to know both the benefits and the risks of the procedure before agreeing to it. This may not have occurred

just at the time that Mrs Jacobson was asked to sign the consent form, but I am satisfied that such a discussion took place at least on 3 October 2008, and that other discussions regarding the procedure most likely occurred from time to time as different practitioners met with Mrs Jacobson. The failure to have Mrs Jacobson initial the box relating to the risks associated with the procedure appears to have been an oversight on the part of Dr Chang, who presented the form to Mrs Jacobson to sign.

[32] In reaching this conclusion I am not suggesting that Mrs Jacobson is attempting to mislead me in any way. Mrs Jacobson has been an outstanding advocate for her son's medical treatment, and was no doubt under an enormous amount of stress during his last admission to Waikato Hospital. In my view, Mrs Jacobson's memory of events around the time that the decision for the PEG was made is likely to be impaired by the stress she was under in October 2008, and the passage of time since then.

[33] Mrs Jacobson's view may also be coloured by her conviction that the PEG was unnecessary, and she may feel that she failed in her advocacy role by consenting to the procedure. If this is Mrs Jacobson's view, then I cannot stress enough that I disagree with it. The clinicians and the allied health professionals caring for David collectively reached a decision based on their training and experience that the PEG was a proper option for David's nutritional requirements. I consider that their professional judgement was exercised with due consideration; I trust that Mrs Jacobson will come to the same conclusion as a result of all of the evidence presented at the inquest. Mrs Jacobson did not fail her son by agreeing to the procedure. She did what any parent would and should do, when she relied on the clinical judgement of the health professionals with regard to treatment options. She should not hold herself responsible for an adverse outcome attributable to the known risks of the procedure in the background of David's very complex clinical condition.

#### **Issue 2: Was David a suitable candidate for the PEG procedure**

[34] David was suffering from immunosuppression in early October 2008, which potentially increased the risk of infection. Dr Wright also mentioned that the steroids that David was on can also impair wound healing to some degree. In addition, Professor Lane notes that David was significantly malnourished, was undergoing plasmapheresis and his neurological condition would have increased the risk of potential sedation related issues during endoscopic procedure. David was colonised with MRSA which would have made a wound infection more difficult to treat. Dr Wright agreed David had significant risks over and above the standard risks associated with PEG, and that David was at the higher end of the risk continuum.

## Discussion

[35] Professor Lane states that, in his opinion, these potential risk factors in themselves do not represent absolute contra-indications to the procedure but did potentially increase the risks of the procedure itself, and if complications ensued, would significantly increase the challenge of managing them. Dr Jason Hill, the gastroenterologist at Waikato Hospital who performed the PEG on David on 15 October, stated that there was no physical contra-indication to this procedure. He stated that he regularly inserts gastrostomies in patients who are immunosuppressed, and that the presence of MRSA and steroids would not be contra-indications to a gastrostomy insertion.

[36] Professor Koea concludes in his report that "*PEG placement was justified.*"<sup>9</sup> Professor Lane states "... *in general terms David met the criteria under which one would consider placement of a PEG as being a reasonable option to consider.*"<sup>10</sup>

[37] Dr Hill describes part of his role as being a 'gatekeeper', meaning he was more than just a technician carrying out the wishes of the referring service. He applied his professional judgement to the suitability of the candidate for the procedure, and found that David was a suitable candidate notwithstanding his elevated risk level.

## Conclusions

[38] I am satisfied from the evidence provided that the clinicians involved in the decision to proceed with the PEG properly considered the usual risks associated with such a procedure and also David's specific, elevated, risk profile. They then exercised their collective clinical judgement that David was an appropriate candidate for the procedure. I accept that this decision is supported by both Professor Koea and Professor Lane as being clinically justifiable as an appropriate treatment for David.

## Issue 3: The PEG procedure on 15 October

[39] Dr Hill performed the procedure on 15 October 2008. In his report he details the steps taken during the procedure. There were no complications during the procedure itself, and David was transferred back to the ward at the completion of the procedure. Given David's immunosuppressed state and his existing colonisation with MRSA, antibiotic prophylaxis was crucial to prevent the onset of infection as a result of the procedure.

[40] At the inquest, Dr Hill gave evidence that antibiotics would normally be given by the ward staff prior to the procedure. Dr Hill stated that he was convinced that the antibiotic had

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<sup>9</sup> Ibid. page 5

<sup>10</sup> Report on Coroner's Inquest on the death of David Nicholas Jacobson, Professor Mark Lane dated 2 November 2011, page 4



been administered to David prior to the procedure, based on the computer record which indicated this had been done. This is in contrast to the written record, the PEG worksheet, where the box for ticking administration of antibiotics has not been ticked. In his view, the computer record takes precedence over the written record. Mrs Jacobson, in her questioning of Dr Hill at the inquest, has anecdotally confirmed that an antibiotic was administered to David around the time of the procedure.

[41] However, Mrs Jacobson has an issue with the particular antibiotic administered to David. She informs that David was administered Cefazolin, whereas she believes Vancomycin was the more appropriate antibiotic due to David's colonisation with MRSA. Dr Hill's evidence is that, regardless of which antibiotic was administered, David was always going to be at high risk of developing bacteraemia. He goes on to state that David's subsequent infection was not related to the tracts that were created during the procedure, and therefore the issue of the antibiotic is not implicated in the subsequent events.

[42] Professor Lane reviewed the clinical notes, Dr Hill's report, and listened to his evidence given at the inquest. In his opinion, the PEG procedure was carried out in a standard manner. He had no concerns about any technical aspects related to this procedure.

### **Conclusions**

[43] I am satisfied that there were no complications during the PEG procedure, and that the procedure itself was done in a professional manner to an appropriate standard. I also accept that an antibiotic was administered to David prior to the procedure in accordance with best practice for such a procedure. As to whether or not a more effective antibiotic should have been administered, I have no direct evidence on this point and am therefore unable to make a finding. In any event, I take Dr Hill's point that nothing turns on this matter because David's subsequent infection, which ultimately resulted in his death, was unrelated to the incision created during the procedure.

### **Issue 4: David's clinical care and treatment following the initial PEG procedure**

[44] For the first 10 days following the PEG procedure, the tube was functioning well with feeding progressing as anticipated and with no observable complications. On 25 October, David was noted to be febrile. Investigations revealed that the tip of the PEG tube had become dislodged into the peritoneal cavity. On 27 October, David underwent a laparotomy which confirmed that the PEG tube was displaced. This procedure was performed by Dr Mohammad Rafique, and in his report he states that there was not much contamination other than mild inflammatory exudate around the liver close to the PEG tube. He further states that the abdominal cavity was washed out and he left two drains in the abdominal

cavity. A replacement gastrostomy tube was then fixed in place. The report also notes that the drains did not drain anything for the 48 hours following the operation, indicating that there was no leakage.

[45] At some stage prior to 6 November, David's gastrostomy tube began to leak. A further laparotomy was performed on 6 November by Dr Al Askari. In this procedure, the previous two gastrostomy sites were closed, the leaking tube was removed and replaced with a jejunostomy tube. Following completion of this procedure, David began a slow but promising recovery.

[46] On 15 November, a gastrointestinal endoscopy was performed under general anaesthetic to address a large gastric ulcer with probable vessel bleeding. This was completed successfully, but on 19 November David developed a further major gastrointestinal bleed. An emergency laparotomy was performed by Dr Rowan French, a surgeon at Waikato Hospital, to stop the bleeding. The bleeding gastric ulcer was over-sown through a gastrotomy. Slow jejunal feeds were recommenced on 20 November.

[47] On 22 November, further bleeding was noted. The possibility of further jejunostomy leak was considered and so the tube was spigotted and NGT feeds commenced. David was experiencing severe epigastric pain and tachycardia. Investigations showed that the jejunostomy was not leaking, but a surgical review on 27 November notes wound dehiscence and poor healing. By this stage, David's clinical course was on a downward spiral. David underwent a further operation that day, and Dr French informs that he found tertiary peritonitis with infected ascitic fluid throughout the abdomen. Despite an exhaustive search, no hollow viscus perforation or leak was found. David remained critically unwell with severe hypoglycaemia and thrombocytopenia.

### **Discussion**

[48] The clinicians in their evidence state that displacement of a PEG tube is a known risk of the procedure. It was the displacement which necessitated the laparotomy performed on 27 October. Dr Hill stated that displacement occurs because excessive traction pressure is applied to the device externally. This could be due to the patient pulling on the device or if the device becomes caught in bedding when the patient is turned or rolls over.

[49] Professor Lane informs that the actual cause for the displacement in David's case cannot be determined, but agrees that it is most commonly due to traction on the tube. It can, however, be due to over-tightening of the buffer which would usually happen over days or weeks. He goes on to state that if traction on the tube occurs, the tube simply falls out (sudden traction) or slowly erodes into the wall of the stomach (buried bumper syndrome).

He notes that it is rare for the tube to finish up free in the abdominal cavity, as occurred in David's case.

[50] Dr Hill notes that periodic rotation of the tube is the normal post-operative care plan for people with a PEG, and this is necessary to "*prevent the PEG sticking inside the tract and reduce pressure complications from the skin disc or the internal bolster.*"<sup>11</sup> Professor Lane informs that the nursing notes indicate that the tube was being rotated and cleaned on a regular basis. He states that a tube which is being frequently manipulated is at risk of being over-tightened. He recognises that inadvertent pulling is also possible while changing the position of a patient requiring full cares.

[51] The second laparotomy, performed on 6 November, was necessitated by leakage. Dr Rafique, who performed this procedure, notes in his report that leakage around a gastrostomy tube is a known risk of the PEG procedure. He also notes that there was no leakage for the first 48 hours following the procedure, as evidenced by the lack of any drainage coming from the two abdominal drains. Dr Hill supports Dr Rafique's opinion on the known risk of leakage, and affirms that the abdominal drains being clear indicates that the leak did not start during the first 48 hours following this procedure.

### **Conclusions**

[52] The evidence indicates that the displacement of the PEG tube has occurred at some point following the initial PEG, probably around 25 October when David was first noted to be febrile. The cause of the displacement cannot be ascertained, as there is no evidence to support one particular cause over another. Displacement of the PEG tube is a known complication and risk of the procedure. Unfortunately, this complication occurred in David's case, for whatever reason.

[53] I note that Dr Hill in his report refers to a change to procedures for PEG patients, whereby a daily chart for recording skin-disc measurement is now maintained as part of the current post-insertion instructions.<sup>12</sup> If, in the future, displacement of a tube is occurring due to the periodic manipulation of the tube, then hopefully the new procedure will alert clinicians to the complication before there are any adverse consequences.

[54] I accept Dr Rafique's conclusion that there was no leakage around the gastrostomy site following the laparotomy he performed on 6 November. The leak has clearly developed at some point after the first two days following the procedure. The reason for the leak is not obvious, but leakage is a known complication and risk of the procedure. Unfortunately, this

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<sup>11</sup> *Report to the Coroner Re David Nicholas Jacobson*, Dr Jason Hill, undated, para 41.

<sup>12</sup> *Ibid.* para 46

complication also occurred in David's case. In the absence of any evidence to the contrary, I must take the view that this leakage was a naturally occurring complication.

#### **Issue 5: David's clinical course leading up to his death**

[55] By 15 November, David was significantly physiologically stressed with malnutrition, infection, the effects of one procedure (PEG) and two major operations (laparotomies), and had a lengthy history of immunosuppression. On that date he was vomiting blood, and underwent an upper gastrointestinal endoscopy to address a bleeding gastric ulcer. On 19 November he had a further episode vomiting blood, and a further laparotomy was performed to enable over-sewing of the bleeding ulcer.

[56] Following further abdominal pain and fever on 27 November, David underwent a CT scan examination of his abdomen. The scan showed extensive free fluid and gas suggestive of perforation of a hollow viscus (bowel). As a result, David required a further laparotomy where a large amount of infected peritoneal fluid was washed out and the jejunostomy was re-sutured. He was admitted to the intensive care unit for support of his severe sepsis, and was commenced on ventilatory and inotropic support. He was also given blood products for haematological failure, antibiotics and parenteral nutrition. David made surprisingly good progress over 29 and 30 November. He was weaned from inotropic support, and extubated, on 30 November but still required ventilation via a face mask.

[57] Dr McKenzie, in her report, details David's last few hours as follows:

37. *"During the afternoon of 1<sup>st</sup> of December David had further malaena (altered bloody faeces suggestive of further gastro-intestinal bleeding).*
38. *At 2245 [hours] David suffered a sudden severe episode of hypotension (fall in blood pressure) associated with tachycardia (fast heart rate) progressing to bradycardia (low heart rate) with pulseless electrical activity (heart beating but not producing a blood pressure). In light of the previously determined decision for him to not undergo CPR in the event of a cardiac arrest resuscitative efforts were not be done and he was pronounced dead at 2255 [hours].*
40. *In light of the nature of his demise, associated with his earlier malaena, and thrombocytopenia, a further massive gastrointestinal bleed was considered the most likely cause of his final demise."*<sup>13</sup>

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<sup>13</sup> Report to the Coroner re David Nicholas Jacobson, Dr Jane McKenzie, dated 5 July 2011, paras 37, 38, 40

## **Discussion**

[58] Professor Lane notes that stress ulceration is not unusual in this circumstance, and bleeding may occur. The risk of ulceration and bleeding was being mitigated, but it is not completely preventable. When David suffered from a bleeding ulcer, endoscopic intervention was attempted but was not fully successful. The need for a further operation increased his risk of complications.

[59] The clinicians' view, following the further laparotomy on 27 November, was that David had a very poor prognosis. Surgical interventions were not being effective, and the clinical decision was made that his treatment would not be escalated beyond those measures currently established in the event of a further deterioration. David's clinical notes, and the evidence provided by Dr French, show there was considerable discussion with Mrs Jacobson regarding David's prognosis. In his evidence at the inquest, Dr French stated that it may have been difficult for Mrs Jacobson to fully grasp just how serious things were on 27 November. He stated that the length of his entry in the notes reflects the extent that he went to, to try and convey the severity of the situation.

[60] Mrs Jacobson, during her questioning of Dr French at the inquest, indicated very strongly that she believes she never agreed to refuse resuscitative efforts for David. However, she intimated that if she had been told that if David's heart stopped it would be extremely unlikely the medical team would be able to re-start it given how sick he was at the time, then she would have agreed to no resuscitative efforts. She went on to say that if his heart had stopped she would not have wanted resuscitation. In response to a question from Professor Lane, Dr French agreed that the terminal event for David was a cardiac arrest.

## **Conclusions**

[61] I accept the medical evidence establishes that David's clinical course, from around the laparotomy performed on 6 November, was on a perilous downward spiral. Complications flowed continuously from every medical intervention, no doubt due in the latter stages to his very complex underlying condition, his malnourished state, the severe sepsis, the physiological stress caused by four major surgeries, and long-term immunosuppression. There is no evidence to establish that this would lead inevitably to his death, but from around 15 November his condition, in my view, was such that it would have taken a miracle for him to survive.

[62] I accept Professor Lane's conclusion to his consideration of David's clinical course, when he states that the migration of the PEG into the peritoneal cavity caused peritonitis requiring surgery, and that this initiated a cataclysmic chain of events which culminated in David's death. In Professor Lane's opinion, when David suffered a cardiac arrest on 1 December, cardio-pulmonary resuscitation ("CPR") in these circumstances was futile

because success never occurs. He considers that the decision not to do active CPR was clinically appropriate and unquestionably correct. I am satisfied from the evidence provided to me that the medical decisions and procedures made during David's clinical course, are consistent with general medical practice.

[63] The sad reality is that, despite the current level of sophistication in the practice of medicine, there are still complications arising from routine surgical procedures which, in a particularly complex and multi-factorial medical background, can result in the worst outcome. In my view, David's case fits into this category.

#### **FORMAL FINDING**

[64] I find that David Nicholas Jacobson, of Gainsford Road, Hamilton, died at Waikato Hospital, Pembroke Street, Hamilton, on 1 December 2008.

[65] The cause of death was:

Direct cause: Abdominal sepsis

Antecedent cause:

Underlying condition: Unspecified neurologic disorder

Other conditions:


[66] The circumstances of the death are that David suffered from a very complex and severe neurologic disease. He underwent a PEG procedure in Waikato Hospital on 15 October 2008. Following this, David's clinical course involved numerous complications stemming from this and subsequent procedures, resulting in David undergoing multiple surgeries. In addition, David was in a malnourished and immunosuppressed state. He developed severe sepsis which, in his compromised state, he was unable to overcome. His condition progressively declined despite intensive medical treatment. He suffered a cardiac arrest and was not resuscitated due to the severity of his condition.

#### **CONCLUSION**

[67] I acknowledge the assistance provided to me in this case by Professor Lane in his capacity as Specialist Advisor, and also by Professor Koea in his review of David's case history.

[68] I would also like to acknowledge the assistance provided to me by Ms Staples, in her role as counsel to assist the Coroner, as well as all counsel representing various interested parties. I am also grateful for the evidence provided by all witnesses at the inquest.

[69] I would particularly like to acknowledge Mrs Jacobson's extensive and commendable efforts in her role as advocate for David, both during his life and after his death during this coronial process.



Coroner JP Ryan