CORONERS ACT, 2003

SOUTH  *AUSTRALIA*

FINDING OF INQUEST

An Inquest taken on behalf of our Sovereign Lady the Queen at Adelaide in the State of South Australia, on the 25th, 26th and 27th days of February 2008 and the 15th day of August 2008, by the Coroner's Court of the said State, constituted of Mark Frederick Johns, State Coroner, into the death of Natasha Anne Edwards.

The said Court finds that Natasha Anne Edwards aged 31 years, late of Lot 601, Angle Vale Road, Angle Vale, South Australia died at the Royal Adelaide Hospital, North Terrace, Adelaide, South Australia on the 11th day of July 2005 as a result of a right cerebella cerebrovascular accident. The said Court finds that the circumstances of her death were as follows:

1. Introduction

1.1. Natasha Anne Edwards was 31 years of age when she died in the Royal Adelaide Hospital on 11 July 2005. Unusually for such a young person, the cause of death as given by the treating physicians at the Royal Adelaide Hospital was right cerebella cerebrovascular accident. Ms Edwards had been a generally healthy person until the signs and symptoms of this fatal episode manifested themselves only some three days prior to her death.

1.2. Her death was reported to the Coroner as required by the Coroners Act 2003. There was no autopsy. Her case was reviewed by Dr Jeremy Hallpike, Emeritus Neurologist at the Royal Adelaide Hospital, at the request of the Court. In a report dated 30

March 2006 which was admitted as Exhibit C11a in these proceedings, Dr Hallpike gave the cause of death as:

> 'Cerebellar herniation downward through the foramen magnum (pressure coning) → brainstem compression → rapidly progressive reflex and autonomic failure → brain death.' [1]

Dr Hallpike gave the cause of the pressure cone as:

> 'Obstructive hydrocephalus from swelling and brainstem displacement associated with acute right cerebellar infarction (stroke).' [2]

Dr Hallpike stated in his report that the clinically reported symptoms suffered by Ms Edwards, including headache, neck pain, right facial symptoms, dysgeusia[3], vertigo, visual scintillations and loss of balance[4] are very suggestive of vertebral artery dissection. Later in his report, Dr Hallpike stated :

> 'I believe that most neurologists would support the view that the available information in this case points most strongly towards a vertebral artery dissection as the likeliest cause of the cerebellar infarction.' [5]

He acknowledged that this had not been definitively established by autopsy.

1.3. Dr Hallpike also gave evidence at the Inquest. He summarised the case in the following words:

> 'There's some important lessons to be learnt but I don't feel that anyone really did anything that was egregiously wrong. I hope I've conveyed that because there are lessons.' [6]

With respect, I adopt those words. This Finding is presented in the hope that, by a full and detailed recitation of the events, the Court may assist in the promulgation of the lessons so ably conveyed by Dr Hallpike in his report[7] and his evidence[8]. To that end I will describe as best I can the events that happened to Ms Edwards from 8 July to 10 July 2005.

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[1] Exhibit C11a, page 3
[2] Exhibit C11a, page 3
[3] Metallic Taste
[4] Which Dr Hallpike described as a clinical indication of a cerebellum lesion
[5] Exhibit C11a, page 3
[6] Transcript, page 172
[7] Exhibit C11a
[8] Transcript, pages 89-172

2. The Lyell McEwin Hospital – Radiology and Neurology facilities

2.1. Ms Edwards was admitted to the Lyell McEwin Hospital under the care of Consultant Physician Dr Christopher Beare. Dr Beare provided a useful description of the facilities as they existed in the Lyell McEwin Hospital both in 2005 and today.

2.2. Dr Beare described the reporting of radiology out of hours in 2005. He said that radiology undertaken at the Lyell McEwin Hospital would be reported by the on-call radiology registrars at The Queen Elizabeth Hospital. The reports would be ratified either the next day, or if it were a weekend, sometime the following Monday by a radiology consultant. That consultant might be based either at The Queen Elizabeth Hospital or at the Lyell McEwin Hospital. Dr Beare noted that CT head scans could be performed out of hours at the Lyell McEwin Hospital in 2005 using an on-call radiographer. He understood that the scans would then be viewed electronically by the reporter at The Queen Elizabeth Hospital. A verbal report would be made and would be available for retrieval using the computer system at the Lyell McEwin Hospital. The images could also be viewed via the computer[9].

2.3. In 2005 the Lyell McEwin Hospital had an MRI scanner but it was not available except during office hours, Monday to Friday. Thus, there was no weekend scanning. Dr Beare said that the Lyell McEwin Hospital was not funded to run the unit after hours. He said that in 2008 the Lyell McEwin Hospital has a 24 hour on-call MRI service. However, in 2005 if he thought there was an urgent need for an MRI scan after hours he had to try and contact another hospital and arrange for an urgent MRI scan to be carried out at that hospital[10].

2.4. In 2005, there was no Neurological Department at the Lyell McEwin Hospital[11]. Dr Beare said that a neurologist visited for two sessions per week on Tuesday and Thursday mornings. He said that if a neurological opinion was required out of hours at that time it was necessary to telephone around and try to find someone who was available. He said that the situation in this respect remains unchanged in 2008.

2.5. Dr Beare said that in 2005 there was no neurosurgical service at the Lyell McEwin Hospital. He said that if it were necessary to obtain a neurosurgical opinion from

[9] Transcript, pages 15-16
[10] Transcript, page 18
[11] Transcript, page 19

Lyell McEwin Hospital at that time, whether within hours or out of hours, it was necessary to contact the Royal Adelaide Hospital to speak to the on-call neurosurgeon at that hospital. He said that in this respect the situation remains the same in 2008.

3. The events of 8 July 2005

3.1. The evidence shows that late in the afternoon of that day Ms Edwards was driving in her car with her two year old daughter when she felt ill. She stopped her vehicle and called for an ambulance. The ambulance received the call at 1714 hours and arrived at the scene at 1728 hours to find Ms Edwards sitting in the back seat of her car with her daughter. The ambulance officers appear to have stayed with Ms Edwards until 1812 when they departed from the scene arriving at the Lyell McEwin Hospital at 1833 hours. It is not entirely clear why so much time was spent at the scene. The ambulance officers recorded that Ms Edwards was complaining of being dizzy, having had nausea and having vomited. She reported that she had had a flu for the last week and had only just got over the symptoms fully that day. Vomiting was noted, GCS[12] was 15/15, BP[13] 105 systolic. An intravenous access was established and she was given Maxalon[14].

3.2. Ms Edwards' Lyell McEwin Hospital medical notes were admitted as Exhibit C3 in these proceedings. According to a note made at 8:20pm on 8 July 2005 by a Dr Jaber, he examined Ms Edwards at that time. He noted that she had been driving and had felt dizzy during the afternoon. His note proceeded to record that she had expressive dysphasia[15] and felt weak all over, started to vomit half an hour later and could not walk. She felt a right-sided headache with the pain level being 8 to 9 out of 10. Her face felt numb and tingly and she could taste metal. One to 1.5 hours after this episode most symptoms resolved. She continued to have a loss of balance and still felt dizzy and vomiting. She had experienced no similar illness in the past and had no family history of stroke, diabetes, hypertension or ischaemic heart disease. She personally had no history of ischaemic heart disease, diabetes, hypertension or stroke. She experienced no loss of consciousness but felt tired. She had had the flu the week previously. Her GCS score was 15. She was not tachycardic and her pupils were equally reacting to light and accommodation. There was minimal neck stiffness and

[12] Glasgow Coma Scale - a scoring method used to record the conscious state of a person
[13] Blood Pressure
[14] Antiemetic, 10mg
[15] A state in which someone can understand what is being told to them and they can formulate what they want to say but they cannot say it, see Transcript, page 24

there were no abnormalities detected with ear, nose and throat. A chest examination was normal and there was a normal abdominal examination. Heart sounds were normal. Finally, there was ataxia towards the right side. From this I deduce that Ms Edwards was at that stage able to stand and walk for the doctor. The doctor recorded under the heading 'central nervous system' that the cranial was intact, power normal, reflexes increased in the lower limbs, tone normal, coordination impaired and sensation was not equal. Then under 'assessment', the doctor wrote query transient ischemic attack, query internal ear infection or benign positional vertigo query cerebrovascular accident. Under 'plan' he wrote intravenous therapy, bloods including full blood count, urine and electrolytes, protein, calcium, magnesium and blood sugar level. He directed chest X-ray, electrocardiogram and brain CT scan. At the end of the note Dr Jaber wrote:

> 'Brain CT scan: verbal report
>
> Hypo dense area in the left temporal area, greater than 1 cm square size (artefact cannot be ruled out) MRI suggested by radiologist.' [16]

3.3. Later that night Ms Edwards was seen again by a medical registrar (Dr Rehman) working on the night shift on 8 July 2005. His note was recorded at 11pm and reads as follows:

> 'Med Reg notes
>
> 31 yrs female, married living with family
>
> Presenting Complaint: Dizziness and Headache
>
> Past Medical History: Migraine – Childhood
>
> History of Present Complaint:
>
> Was feeling fine till today evening when started feeling dizzy while driving with severe occipital headache radiating to neck, + generalised weakness with tending to fall towards right. + Numbness of the face
>
> Was unable to talk for an hour
>
> No specific limb weakness
>
> No loss of consciousness, no jerky movements
>
> Never had this pain and dizziness before
>
> No family history of premature coronary artery disease/stroke. Occasional smoker 1-2 per day, not on oral contraceptive pill. No cancer, no diabetes mellitus, no hypertension, no increased cholesterol
>
> Allergies nil
>
> Social: occasional smoker 1-2/day, no alcohol

[16] Exhibit C3

Denies drug abuse, married, works as caregiver to handicapped children

On examination: Young lady, sick looking, eyes closed, no abnormality detected

Temperature - 37.5ºC, heart rate 82-90, blood pressure 110/80, oxygen saturation 99% on room air

Head, ears, eyes, nose and throat: pupils equal reactive to light and accommodation, extra-occular movements intact

Neck: no stiffness, no signs of meningeal irritation

Chest: clear clinically

Cardio Vascular System: Dual heart sound, no gallop or S3

Central Nervous System: Alert and oriented by 3 (to time, place and person)

Non-focal and sensory examination, cranial nerves – grossly intact

- pronator drift, planters on the right down going on the left equivocal

+ hyper reflexive in all limbs

+ impaired coordination on right

Unable to stand up because of dizziness

Labs: $\frac{136}{4.0} \left| \frac{102}{26} \right| \frac{5.5}{0.07}$ < 5.9

Ca 2.26, Ionised Ca 1.16, Alb 36, T Prot 77, GGT 53, A Phos 53, Mg 0.88, ALT 45

Hb 133, WBC 6.84 with N 74% and L 21%

EKG: Unremarkable

CT brain: Apparently unremarkable

Reported left temporal ill defined hypodense area, query artefact, further evaluation

CXR: Slightly rotated to right, no apparent abnormality

IMP: Most likely acute migraine episode/benign positional vertigo

Less likely to be transient ischaemic attack/stroke/subarachnoid haemorrhage with no risk factors / family history

But keep in view of worse headache and dizziness with query hypodense and ill defined left temporal abnormality → well

Keep her in house for neuro observations for 24 hours

Plan: Admit under Dr Beare
Neuro observations
Antiemetics
✓ lipids
Review by Consultant in morning
Query MRI → for post fossa pathology.' [17]

[17] Exhibit C3, pages 26 and 26a

Dr Rehman added:

> 'Vomited 10 times in 6 hours
> Feeling dizzy all the time, no relation to movement, no tinnitus, no hearing deficit
> Does not feel the need for thrombophilia workup for now.' [18]

3.4. Dr Rehman made the following entry on the 'Non Drug Treatment Orders' sheet:

> 'Neuro check every four hours, 4 hourly observations, ward diet, bloods for morning.'[19]

He prescribed Paracetamol, Panadeine Forte, Stemetil and Tramadol as required.

4. The events of 9 July 2005

4.1. As noted, Ms Edwards was admitted under the care of Dr Beare as her Consultant. He saw her on the morning of 9 July 2005. Dr Beare gave evidence at the Inquest. He said that before examining Ms Edwards he would have checked all of the earlier notes[20]. He said that he looked at the CT scan and then listened to the report of the scan. He said that the scan was done the previous night at 2153 hours. Dr Beare produced films of those scans which were tendered as Exhibit C10. Further CT scans taken on 10 July 2005 were also tendered and these were marked Exhibit C10a. Dr Beare referred to the plain CT scans made on 8 July 2005[21] in his evidence. He expressed the view on looking at the scan that he regarded it as normal[22]. Dr Beare was asked whether he could identify any indication on the scan of a diffused, ill defined hypodensity involving the grey and white matter of the left temporal lobe and he replied in the negative[23]. Dr Beare also referred to the written report of the scan which was not available to him that morning. However, because it assumes a degree of significance in this matter, I will set it out in full:

> 'CT BRAIN:
>
> CLINICAL DETAILS: Dizziness, nausea, vomiting, headache, increased reflexes in the lower limbs rule out CV A or TIA. TECHNIQUE: Contiguous non contrast helical axial scans were obtained from the skull base to the vertex.
>
> FINDINGS: There is evidence of a diffuse ill defined hypodensity involving the grey and white matter of the left temporal lobe. It is not possible to be dogmatic about the fact

[18] Exhibit C3, pages 26 and 26a
[19] Exhibit C3, page 38
[20] Transcript, page 32
[21] Exhibit C10
[22] Transcript, page 36
[23] Transcript, page 36

> that the hypodensity could be secondary to streak artefacts from the temporal bone. Clinical correlation would be required with respect to this. The rest of the supra and infra tentorial neural parenchyma demonstrate a normal grey white differentiation. There is no evidence of any other intra or extra axial space occupying lesions/haemorrhagic focus. The ventricles and extra axial CSF spaces are normal. There is no midline shift. The bony windows do not reveal any abnormality. Windows appropriate for the paranasal sinuses and the maxillary antra are unremarkable.
>
> IMPRESSION: The hypodensity in the left temporal lobe could be suggestive of an infarct.
>
> If there are toxic symptoms and a proceeding viral illness a possibility of herpes simplex infection needs consideration. Clinical correlation is important to distinguish between the hypodensity due to an infarct/hypodensity due to streak artefacts from the temporal bone.
>
> Suggestion: An MRI is strongly recommended.
>
> If an MRI is not possible over the week-end, a pre and post contrast enhanced CT scan of the brain should be performed. Report conveyed to the treating team.
>
> Consultant review: Sinus disease noted. Intracranial finding is agreed with as per above report.
>
> REPORTED AND AUTHORISED BY: DR A T ARANATH / DR I THOMAS
>
> TYPIST: MS B TULK
>
> TYPED: 11/07/2005' [24]

4.2. Dr Beare gave evidence that there were certain aspects of that report which he did not recall from listening to the verbal report via the computer on the morning of 9 July 2005. He said that to him the scan of the temporal lobe looked normal but as the report said otherwise he had to accept it at face value[25]. He recalled that part of the report which referred to the herpes simplex infection. However, he did not have a recollection about the sentence immediately following which referred to clinical correlation being important to distinguish between the hypodensity due to an infarct/hypodensity due to streak artefacts from the temporal bone. Nor did he have recollection of hearing the suggestion that an MRI was strongly recommended, nor that if an MRI were not possible over the weekend a pre and post contrast enhanced CT scan of the brain should be performed.

4.3. Having viewed the scans via the computer and listened to those parts of the report that he recalled having heard on that morning, Dr Beare's next step was to see Ms

[24] Exhibit C3, page 11
[25] Transcript, page 38

Edwards for a physical examination. The notes of that examination appear in the Lyell McEwin Hospital case notes and I set them out in full:

> 'Seen by Dr Beare
>
> Symptoms: cannot stand up due to loss of balance
>
> States right leg feels lite
>
> Has had cold for last week. Denies urinary symptoms, denies sweats
>
> On examination – CNS – no nystagmus, no drift, finger nose ok, co-ordination good including rapid alternating movements. Lower limb power – right/left power equal. Reflexes +++ bilaterally, plantars ↓↓ bilaterally
>
> Further history:
>
> Driving – whole face became paralysed on right, shooting pain up into neck. Metallic taste in mouth. Speech slurred, body tingling, flashing lights in right eye.
>
> A/ ? encephalitis
>
> ? seizure activity from temporal lobe
>
> P/ 400mg Brufen
>
> 12.5mg Dolasetron
>
> Lumbar puncture
>
> Review tomorrow with results' [26]

4.4. In his evidence, Dr Beare commented that the cerebellar examination was normal and the rest of the neurological examination was only abnormal from the point of view of the hyper reflexia[27]. He said he was able to take a history from Ms Edwards and so there was no continuing evidence of expressive dysphasia. He found no evidence of the incoordination of the right side of the body as found by Dr Rehman. He said that the effect of his examination was that the initial signs and symptoms had almost completely resolved apart from the unsteadiness of gait and the hyper reflexia[28]. He said that some of the earlier symptoms such as the paralysis of the whole face implied whole brain involvement but that when he was speaking to Ms Edwards there were no signs of anything as catastrophic as that because she was talking to him. Furthermore, he had seen what he assessed as being a reasonably normal CT head scan[29]. Thus the first of his differential diagnoses was 'query encephalitis' and the second was 'query seizure activity from temporal lobe'.

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[26] Exhibit C3
[27] Transcript, page 41
[28] Transcript, page 41
[29] Transcript, page 42

4.5. Dr Beare said that the references in the CT scan to an ill defined hypodensity in the left temporal lobe meant that, whatever ensued, Ms Edwards would be sent for an MRI scan[30]. As to the timing of the MRI scan, he decided that it could wait until the following Monday (9 July 2005 being a Saturday). He did not feel there was a particular urgency about the situation because most of the signs and symptoms had improved and he could see no specific urgency for an MRI to be done that day[31]. Dr Beare was asked by his own counsel whether he gave any consideration to the possibility that Ms Edwards may have suffered a stroke. He responded that whenever he hears the word hypodensity on a CT scan report 'it's always part of my differential'[32]. Dr Beare continued that because Ms Edwards was a 31 year old and because the younger a person is the rarer it is for a stroke to occur, he would be thinking about other things before a stroke as a favoured diagnosis while maintaining stroke as part of the differential because of the mention of hypodensity[33]. Dr Beare noted that he would have considered a brain tumour over and above a stroke but that it was not mentioned in the report, nor did he see it in the scan[34].

4.6. Dr Beare reviewed a second group of CT scans which were taken on 10 July 2005 after Ms Edwards' collapse. These were the scans that were admitted as Exhibit C10a. Dr Beare compared these later scans with those of 8 July 2005[35] and commented that, so far as they depicted the left temporal lobe, they looked normal to him, just as the earlier scans had. He said that the second scan showed that the right cerebellum was a lot darker than the left cerebellum and that the midline of the cerebellum had been pushed over to the left-hand side. He said that the later scans indicated that there was an obstructive hydrocephalus of which there was absolutely no evidence on the CT scans taken on 8 July 2005[36]. He repeated that there was no sign of an infarct in the right cerebellar hemisphere on the scan of 8 July 2005[37].

4.7. Dr Beare noted that an MRI scan, had it been performed sometime on 9 July 2005 before Ms Edwards collapsed, would probably have shown the cerebellar stroke if it

[30] Transcript, page 44 - I did not take Dr Beare to be linking this to the recommendation contained in the written report previously quoted
[31] Transcript, page 45
[32] But note that Dr Beare's notes as quoted above did not include stroke as a part of the differential diagnoses
[33] Transcript, page 45
[34] Transcript, page 46
[35] Exhibit C10
[36] Transcript, pages 49-50
[37] Transcript, page 51

was occurring at the time the scan was taken[38]. Dr Beare rejected the suggestion that the lumbar puncture taken on the morning of 9 July 2005 might in any way have affected the progression of the pressure coning. He asserted that Ms Edwards' stroke followed the natural progression of cerebellar strokes and that if the lumbar puncture had been a contributing factor in any way, the acute collapse suffered by Ms Edwards would have occurred much more closely to the time of the lumbar puncture rather than in the early hours of the following morning as it did[39].

4.8. Dr Beare was asked whether he thought there was cause for a neurological consultation on the afternoon of 9 July 2005 when the lumbar puncture result was returned as negative. He said that he acknowledged that there was information to show that something unusual was going on but that even with the benefit of hindsight, he would not depart from his plan for Ms Edwards, which was to observe the situation and keep her in hospital. He maintained that there was no indication for a neurological consultation that afternoon[40]. He acknowledged that the lumbar puncture result effectively ruled out encephalitis as an explanation for Ms Edwards' symptoms and that Ms Edwards was essentially an undiagnosed patient after the receipt of the lumbar puncture result[41].

4.9. Dr Beare commented that Ms Edwards' symptom of ataxia could be attributed to the fact that people with headache can be unsteady of gait[42]. He said that the metallic taste in her mouth could be regarded as an aura of temporal lobe epilepsy. He described her signs and symptoms as diffuse, different and 'difficult to add up but most indicative of encephalitis'[43]. Dr Beare acknowledged that he did not mention cerebrovascular accident as a part of his differential diagnosis in Ms Edwards' case notes, although maintained that he did include cerebrovascular accident as part of his differential diagnosis[44]. Dr Beare regarded the situation as one in which Ms Edwards' signs and symptoms had resolved and she was getting better and thus there was no need to transfer her[45].

[38] Transcript, page 52
[39] Transcript, page 55
[40] Transcript, page 59
[41] Transcript, page 60
[42] Transcript, page 63
[43] Transcript, page 63
[44] Transcript, page 64
[45] Transcript, page 68

4.10. Dr Beare was interviewed by Senior Constable McLean on two occasions. Records of interview were produced and were admitted as Exhibits C9a and C9b in these proceedings. Dr Beare was referred to the second record of interview, Exhibit C9b, in which he speculated that he may not have seen or heard the whole of the report of the first CT scans taken on 8 July 2005 because he did not actually listen to the whole report. However, when giving evidence at the Inquest, Dr Beare was reluctant to acknowledge this as a possibility, preferring to maintain that it was his practice to listen to the whole of the verbal report but that at the time of giving evidence he could not recall one way or another whether he did in fact listen to it. Dr Beare was asked about the suggestion in the radiology report that there was a need to do something by way of further imaging that weekend. He responded, with a hint of petulance, that he had great difficulty in understanding why the reporter thought an urgent CT scan should be performed on his (Dr Beare's) interpretation of the scan itself[46]. On the matter of whether an MRI scan could have been arranged for that weekend, he acknowledged that he made no attempt to organise an MRI scan for that weekend. Dr Beare suggested that any person who had thought that they might be able to arrange an MRI scan after hours would be 'better than I at organising things after hours'[47]. He thought that she did not need an MRI scan at the time and, again with a hint of stubbornness, maintained even when giving evidence that he adhered to that view[48].

5. Evidence of Dr Hallpike

5.1. I have already mentioned that Dr Hallpike provided an expert report on behalf of the Court in this matter which was admitted as Exhibit C11a. Dr Hallpike's eminence as a neurologist and his expertise were acknowledged by counsel representing Central Northern Adelaide Health Service, and Drs Beare and Rehman.

5.2. Dr Hallpike made the following comments in the course of his comprehensive evidence:

1) Headache and vomiting and such symptoms of unsteadiness, an alteration of facial feeling, disturbance of taste would suggest some affection or disorder in

[46] Transcript, page 72
[47] Transcript, page 73
[48] Transcript, page 75

the back part of the brain, potentially what is referred to as the posterior cerebral circulation[49];

2) Dr Hallpike did not think that the signs and symptoms with which Ms Edwards presented were indicative of temporal frontal and parietal lobe pathology;

3) Dr Hallpike commented that the registrar's suggestion of a possible acute migraine was reasonable but that it depended on how long the symptom continued: if a neurological deficit continues for more than an hour it rapidly becomes less likely to be a migraine[50];

4) In the young age group that includes Ms Edwards (31 years) the differential diagnosis is much wider and although migraine as a first thought is very reasonable, it becomes less tenable as time passes and the clinician must move on to consider the differential diagnosis of stroke[51];

5) Of the three possible diagnoses - encephalitis, cerebrovascular accident and migraine, Dr Hallpike did not think that a conclusion that encephalitis was the most likely was readily supportable having regard to Ms Edwards' reported signs and symptoms[52];

6) Dr Hallpike said that with encephalitis one is typically referring to an acute illness with fever, impairment of consciousness and headache, sometimes with generalised seizures; on the other hand, the picture of Ms Edwards was of someone who was conscious, could be talked to, was afebrile and had a normal white blood cell count;

7) The presentation of Ms Edwards on the morning of 9 July 2005 did not display any gross neurological deficit, but the symptoms pointed to an acute vascular process; the brunt of the signs and symptoms are in the brain stem cerebellum[53];

8) Dr Hallpike did not think it reasonable for viral encephalitis to be 'put out' as the sole or overwhelming or 'relatively definitive management diagnosis' and to leave matters for the weekend[54];

9) A neurologist in 2005 would be well aware of a process of arterial dissection as the most common form of acute vascular presentation with varying stroke-like

[49] Transcript, page 92
[50] Transcript, page 94
[51] Transcript, page 96
[52] Transcript, page 99
[53] Transcript, pages 99-100
[54] Transcript, page 103

features in people under the age of 50 and certainly under the age of 40, but a less experienced general physician would probably not be expected to have that knowledge[55];

10) There were sufficient signs and symptoms on the Saturday morning to suggest that the advice of a neurologist should have been sought but only if one was aware of the existence of the wide differential diagnosis in young people presenting with acute vascular presentations. If the clinician's concept of stroke is as for an older person, where it is highly likely to be atherothrombotic, then Dr Hallpike thought it was explicable that one would not seek a neurological consult at that point[56];

11) There is no inherent difficulty about making a telephone call to a neurologist provided that the clinician has good questions to ask and there is always an on-call neurologist at the Royal Adelaide Hospital for that purpose[57];

12) The presentation was that of a vascular illness; Ms Edwards' persistent trouble in standing up and walking was not transient and this suggested a cerebellar lesion[58];

13) Unsteadiness of gait out of proportion to weakness of limbs is indicative of a cerebellar problem[59];

14) Ms Edwards' signs and symptoms pointed towards vascular disease and not infective disease[60];

15) On the Saturday morning the idea of encephalitis was being favoured as the principal management diagnosis and the purpose of the lumbar puncture was to confirm that diagnosis. However, the lumbar puncture result provided no support for a diagnosis of encephalitis[61];

16) Dr Hallpike was unable to find, on his reading of the scan of 8 July 2005, evidence of diffuse, ill defined hypodensity involving the grey and white matter

[55] Transcript, page 105
[56] Transcript, page 108
[57] Transcript, pages 110-111
[58] Transcript, page 111
[59] Transcript, page 112
[60] Transcript, page 112
[61] Transcript, page 113

of the left temporal lobe, but he commented that he was not a professional radiologist[62];

17) The CT report of slight changes in the temporal lobe[63] would justify empirical treatment for an encephalitis but other aspects of Ms Edwards' presentation pointed to vascular disturbances and these should have been kept alive as part of the differential diagnosis[64];

18) The lumbar puncture to test for evidence of encephalitis was quite reasonable but in Ms Edwards' particular circumstances in the period after having the lumbar puncture she was in the process of developing a cerebellar infarct that was not shown on the CT scan performed on 8 July 2005. She was evolving a cerebellar infarct that was causing an obstructive hydrocephalitis, and it should be admitted that the lumbar puncture would have made some contribution to Ms Edwards' final demise[65];

19) However, this does not mean that the lumbar puncture was inappropriately ordered or carried out because the evolving cerebellar infarct could not reasonably have been anticipated by the clinicians involved[66];

20) There is absolutely no justification for doing a lumbar puncture in an acute situation and not acting promptly to obtain the results of the puncture to sort out an important differential diagnosis[67]. Dr Hallpike was not happy with the approach of leaving the review of the lumbar puncture results to the following day[68];

21) The lumbar puncture results should have been followed up on the Saturday afternoon[69]. The afternoon provided the opportunity for an objective review of all of the evidence once the lumbar puncture was returned as providing no support for an encephalitis and support or further assistance sought from a neurologist or neurosurgeon[70];

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[62] Transcript, page 114
[63] CT report for 8 July 2005
[64] Transcript, page 115
[65] Transcript, pages 125-126
[66] Transcript, page 126
[67] Transcript, page 217
[68] Transcript, page 217
[69] Transcript, pages 130-131
[70] Transcript, page 134

22) The general mortality from cervical artery dissection is 5%[71] so with prompt recognition and anticoagulant treatment[72] there is a good outcome rate[73];

23) A range of treatments were available including heparinisation, surgical drainage of the ventricle[74] and the drug mannitol[75];

24) There was a 60% to 70% likelihood, had Ms Edwards' condition been properly recognised and treated within 24 hours of her presentation at the Lyell McEwin Hospital, that she would have survived the illness[76];

25) Pain in the neck is a very strong pointer towards the process of vertebral artery dissection[77];

26) The pathology of cervical artery dissection may involve a connective tissue disorder that makes the innermost lining of the artery more susceptible to micro rupture. If there is any hole in the lumen, the blood will go in under high pressure and begin to separate the layers of the artery which is the dissection process. The blood flows through the innermost layer of the artery into the fibro-muscular layer and under pressure it will track up and down the wall of the artery enclosing the lumen of the artery thus obstructing the blood flow. Furthermore, because of the reduction in blood flow there is a risk of clot formation with artery to artery embolism[78];

27) The management is acute heparinisation for 4 or 5 days then substitution of heparin with oral warfarin for a period of 3 to 6 months after which the dissection is likely to repair itself [79];

28) On the Saturday afternoon discussion with a radiologist would be likely to have led to dissection emerging as a possible explanation for the symptoms. MRI angiography would have identified the vertebral artery dissection[80].

5.3. Dr Hallpike summarised the symptomology by saying that from Ms Edwards' first presentation the symptoms of headache, neck pain, dizziness, nausea, vomiting, troubles with speech that came and went and facial numbness formed a history of

[71] Transcript, page 136
[72] Heparinisation
[73] Transcript, page 136
[74] Transcript, page 138
[75] Transcript, page 140
[76] Transcript, page 141
[77] Transcript, pages 146, 155
[78] Transcript, pages 150-153
[79] Transcript, page 164
[80] Transcript, page 159

fluctuating symptomology of a vascular nature in the vertebro-basilar arterial distribution as well as evidence of a cerebellar lesion[81]. The lumbar puncture greatly accelerated the decompensation process with coning, but Dr Hallpike was unable to quantify the exacerbation as a consequence of the lumbar puncture[82]. Dr Hallpike said that carotid and vertebral artery dissection or 'cervicocephalic arterial dissection' is now known to be an important cause of stroke in younger adults. The most common mechanism of cerebellar infarction was arterial occlusion as a result of intracranial vertebral artery dissection, mainly with posterior and inferior cerebellar artery involvement. Dr Hallpike provided, for the assistance of the Court, several articles which were admitted as Exhibit C11b in these proceedings[83].

6. Summary

6.1. The first CT scan confronted Dr Beare with quite a confusing situation. Dr Hallpike was certainly not seriously critical of Dr Beare in this matter. It was reasonable to entertain encephalitis as a possible explanation for Ms Edwards' signs and symptoms, although Dr Hallpike maintained that a vascular explanation should also have been considered. That said, Dr Hallpike acknowledged that a physician in Dr Beare's position may not be expected to have the necessary knowledge to entertain vertebral artery dissection. Dr Hallpike's main criticism was of the failure promptly to follow-up the results of the lumbar puncture carried out on the Saturday morning which would have been available certainly by the early afternoon of the Saturday. At that point, Ms Edwards had no working diagnosis. However, no step was taken to quickly reconsider the situation - instead matters were left to rest until Sunday morning so far as Dr Beare was concerned.

6.2. Dr Beare was negative about the likelihood of his obtaining assistance of a neurological nature. Indeed his pessimism about that resulted in him making no attempt to seek such assistance. In my opinion, the fact that Dr Beare anticipated that he would not succeed did not justify his decision not to try. Had he tried and failed, appropriate explanations might have been sought at Inquest from those he approached. It is concerning that a senior clinician should have a negative view of the

[81] Transcript, page 169
[82] Transcript, page 171

[83] Cervicocephalic Arterial Dissections from Uncommon Causes of Stroke, edited by J Bogousslavsky, Université de Lausanne, Switzerland and Louis Caplan, Harvard Medical School, Boston, CUP 2001; Scopus - Stroke: Causes and mechanisms of cerebellar infarction in young patients, http://www.scopus.com; Identification and Management of Difficult Stroke and TIA Syndromes, Martin M Brown, J Neurol Neurosurg Psychiatry 2001;70 (suppl 1):i17-i22

likelihood of assistance from his senior medical colleagues. Dr Hallpike displayed a much more optimistic outlook. It is possible that Dr Beare contemplated that neurological assistance would inevitably involve him transferring the patient without further involvement. On the other hand, Dr Hallpike envisaged further active consideration of the possible diagnoses with consultation of the literature - indeed he suggested that a 'Google' search employing the keywords 'stroke in young people' would likely have put Dr Beare on the right track. He was correct - such a search would have been fruitful. Thus armed with some information and, perhaps a theoretical diagnosis, an approach to a neurological consultant may have been more successful than a bare request to transfer the patient.

7. Recommendations

7.1. Pursuant to section 25(2) of the Coroner's Act 2003 I am empowered to make recommendations that in the opinion of the Court might prevent, or reduce the likelihood of, a recurrence of an event similar to the event that was the subject of the inquest.

7.2. I therefore recommend that the Department of Health give consideration to the provision of further education to all emergency doctors and specialist physicians in relation to the signs and symptoms and appropriate clinical management of acute brain attacks in young adults.

Key Words: Cerebrovascular Accident; Incorrect Diagnosis

In witness whereof the said Coroner has hereunto set and subscribed his hand and Seal the 15th day of August, 2008.

State Coroner